



Cardiovascular System E01

Special class

PATHOLOGY CRASH COURSE

NEET PG, FMGE, INI CET
Dr. PRIYANKA SACHDEV , MD

SYSTEMIC PATHOLOGY

Dr. PRIYANKA SACHDEV , MD

CVS

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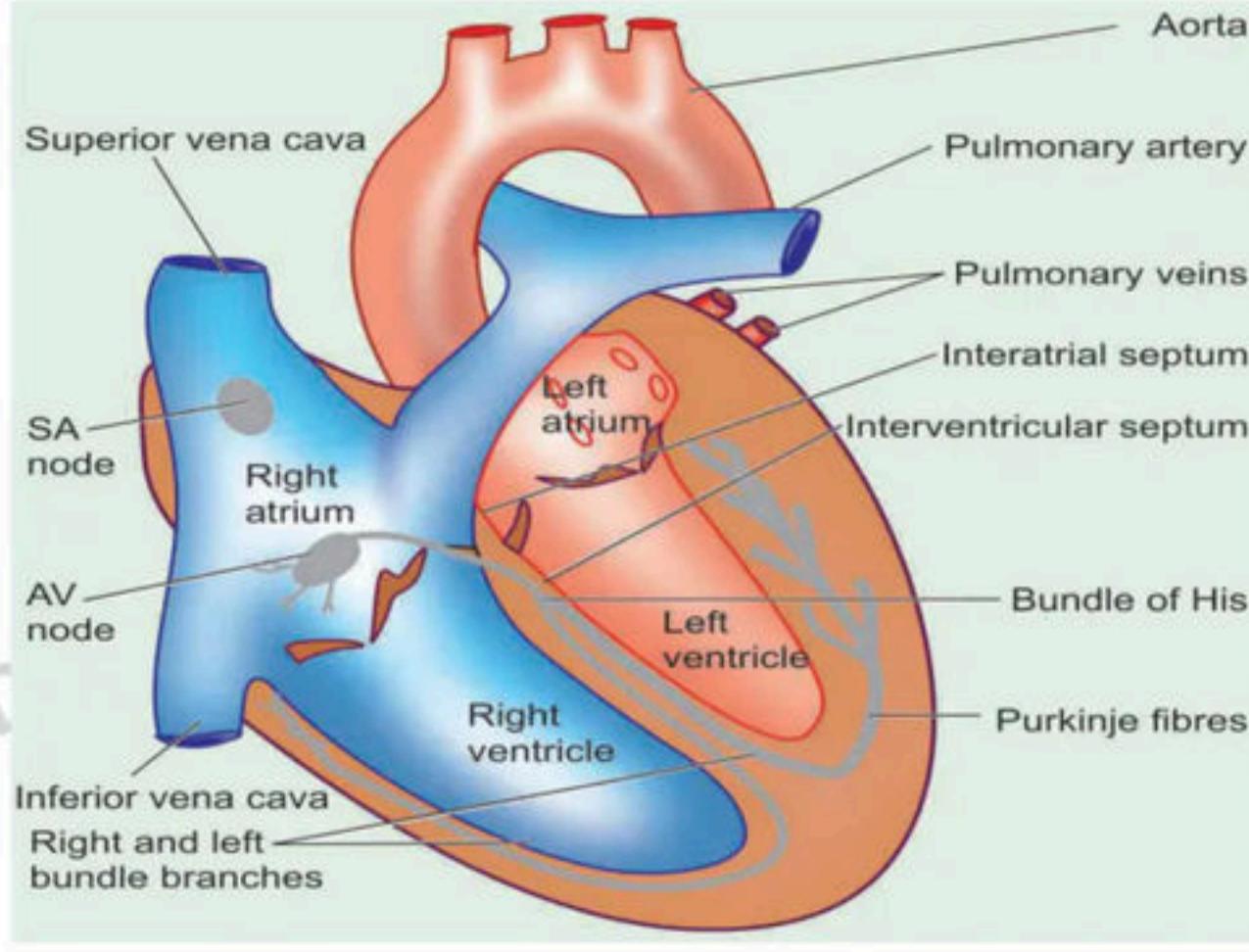


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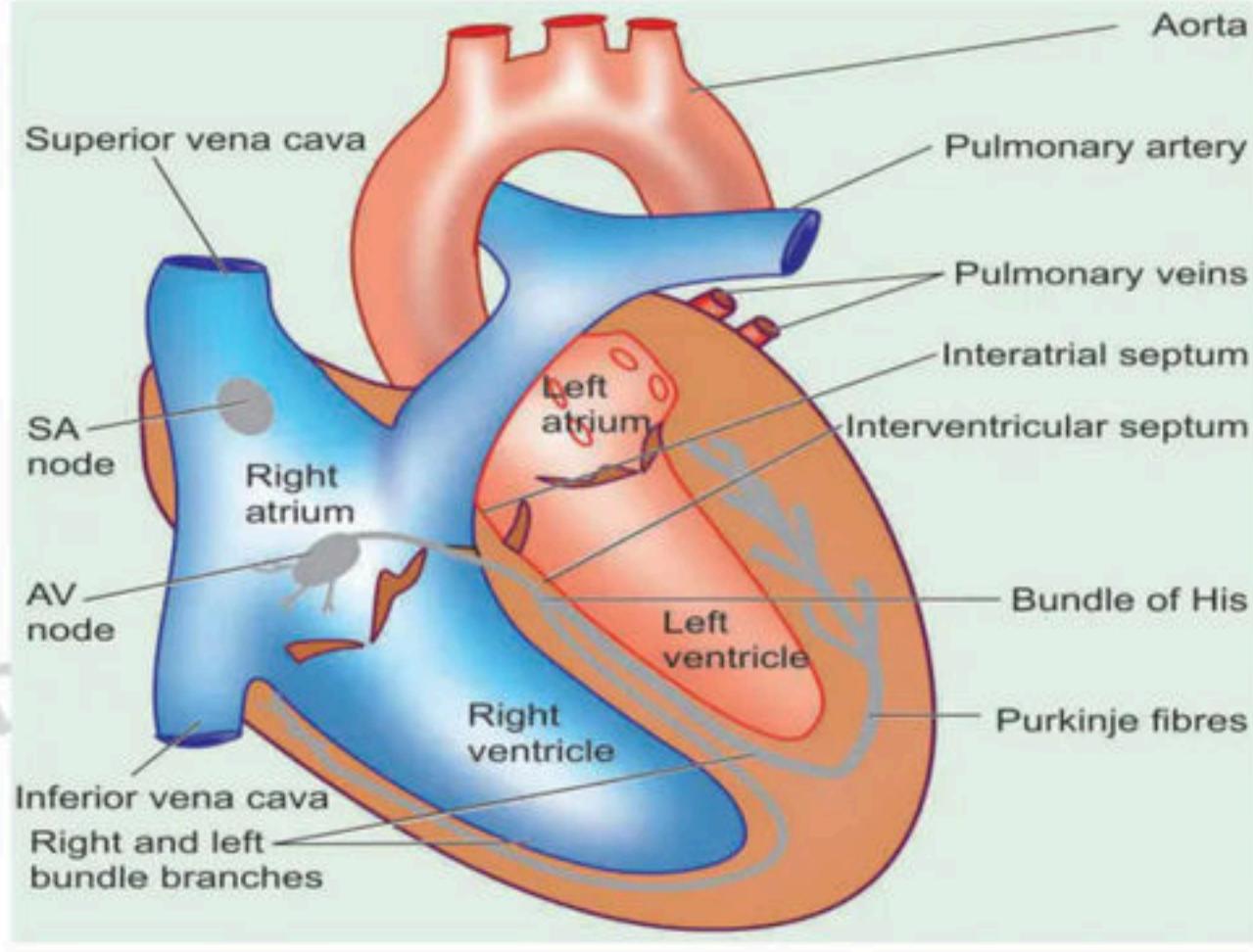


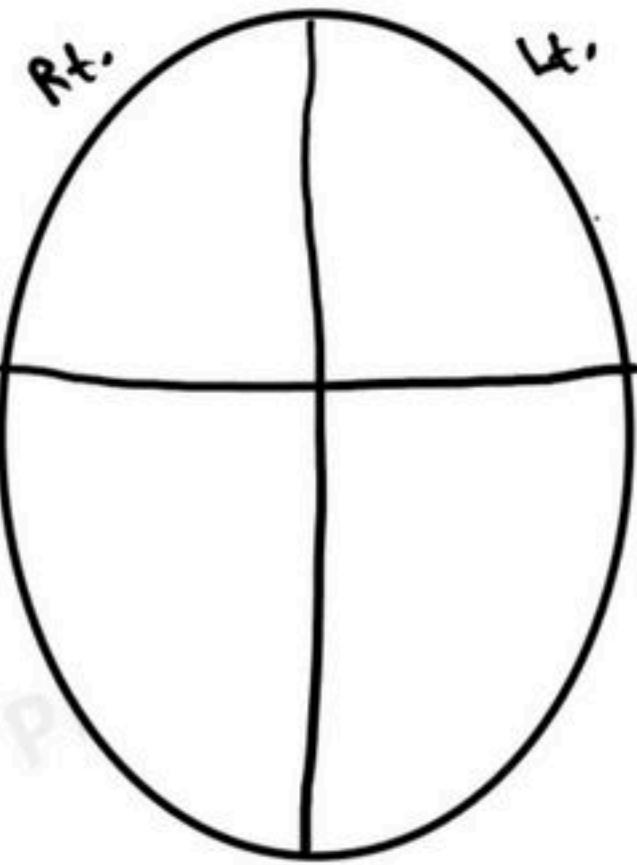
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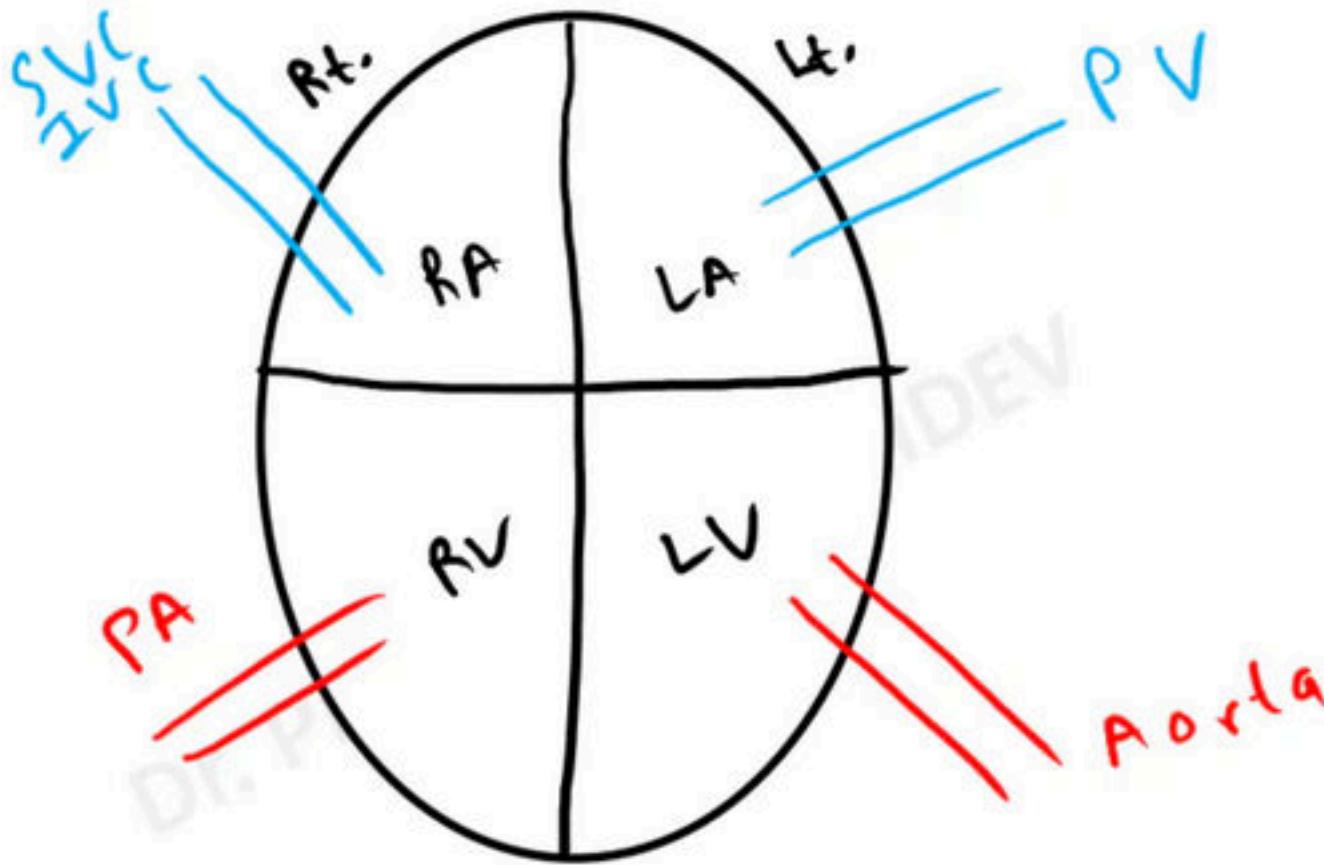


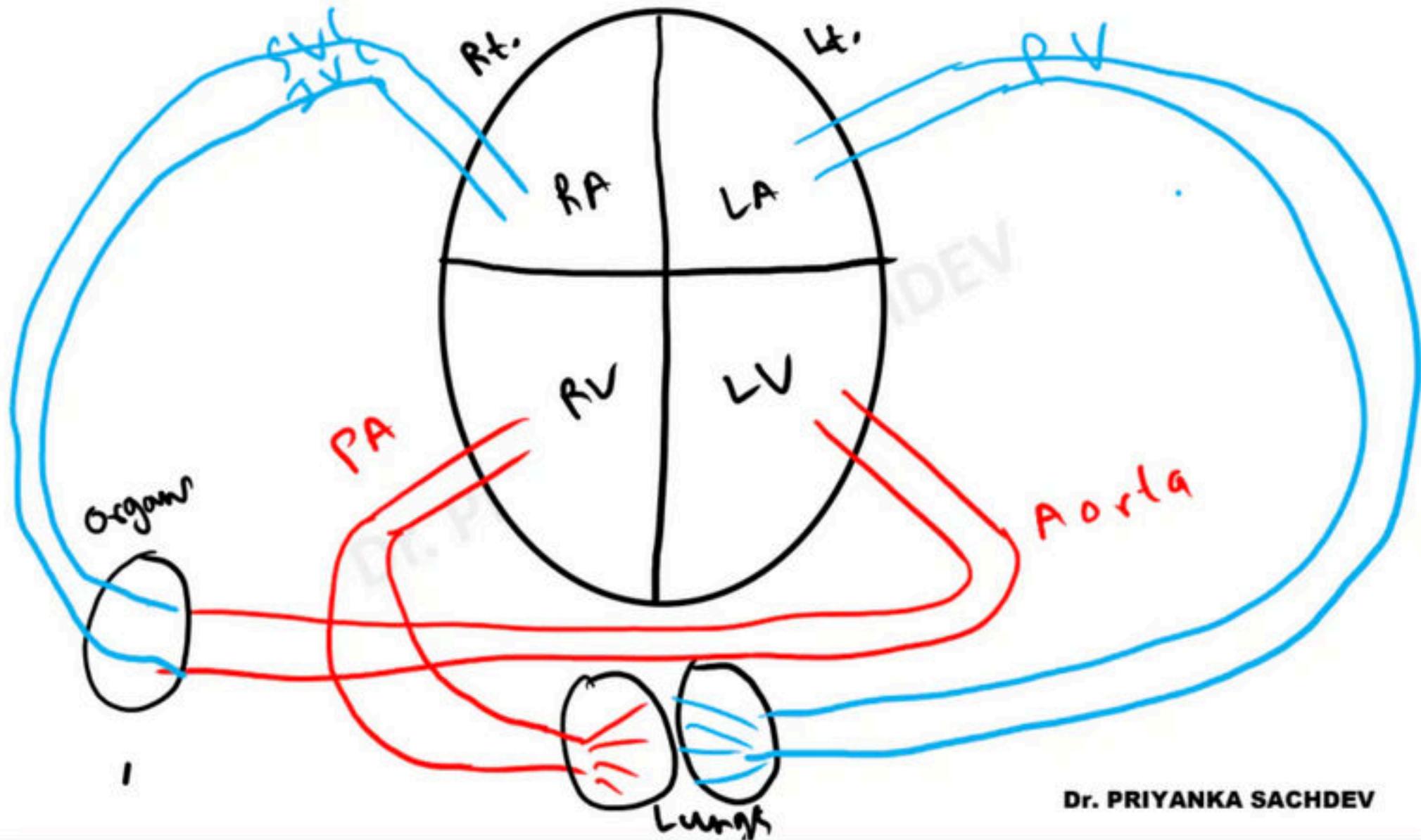


- 4 chambers
- 4 valves
- 2 arteries
- 2 veins









- **Stenosis → opening defect**
- **Regurgitation → Closure defect**

CARDIAC ADAPTATIONS

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TYPES OF CELLS

1. Labile cells
2. Stable cells
3. Permanent cells

5 TYPES OF ADAPTATIONS

Hypertrophy → Increase in the size of cells

Hyperplasia → Increased number of cells

Atrophy → Decrease in the size of cells / shrinkage of cells

Metaplasia → Transformation or replacement of one adult cell type with another

Dysplasia → means 'disordered cellular development'

CARDIAC ADAPTATIONS

- **Definition**
- **Types**

CARDIAC ADAPTATIONS

- The cardiac myocyte is **terminally differentiated cell (permanent cell)** that is not able to divide.
- Myocardium **cannot undergo hyperplasia**, i.e. increase in the number of myocyte.
- So, myocardium can adapt by increasing the size (i.e. **hypertrophy**) of the myocyte in response to stress.

CARDIAC ADAPTATIONS

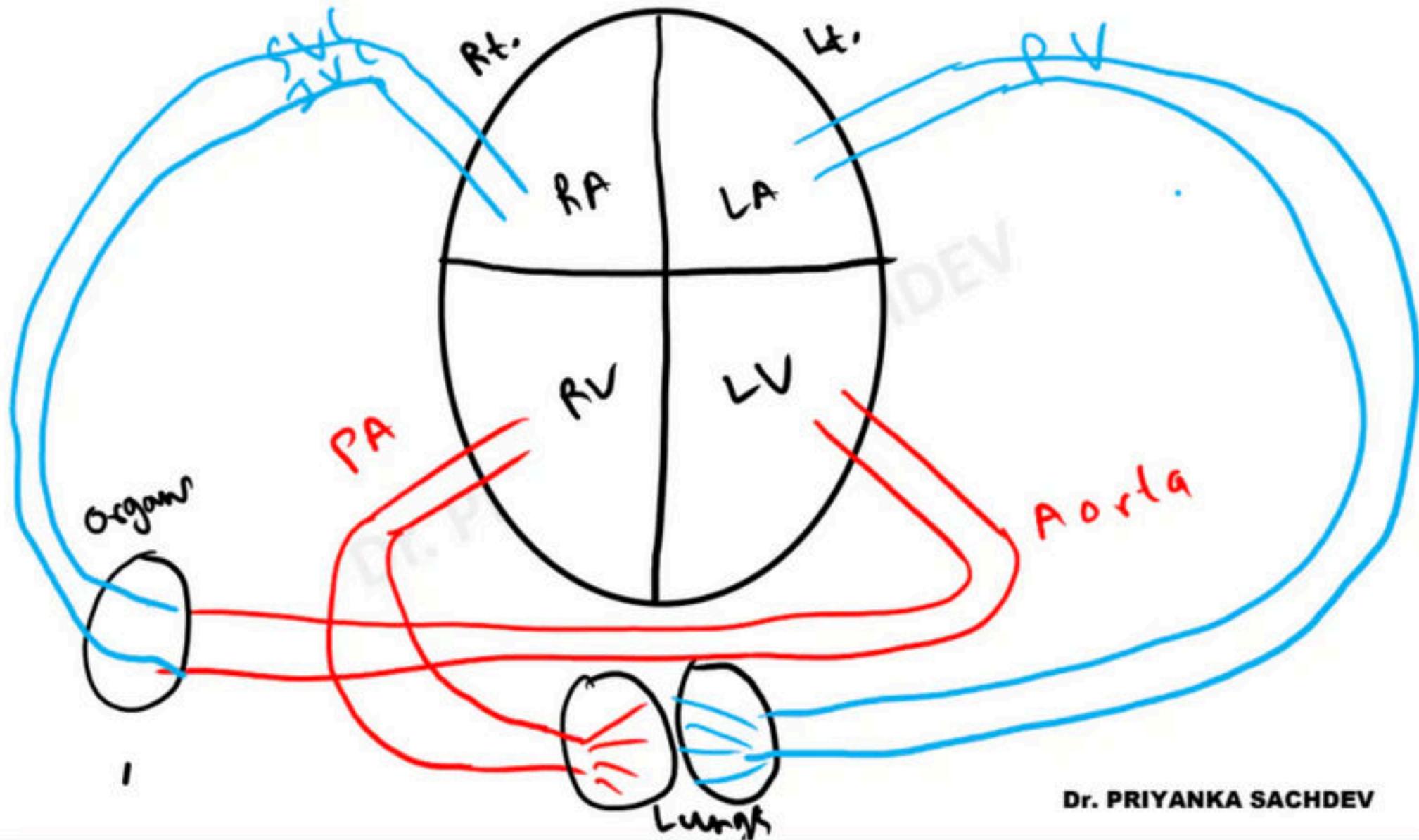
- **Definition**
- **Types**

Types of stress on heart

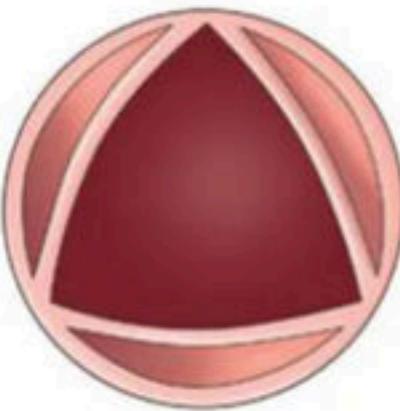
- **1. Pressure overload**
- **2. Volume overload**

1. Pressure overload

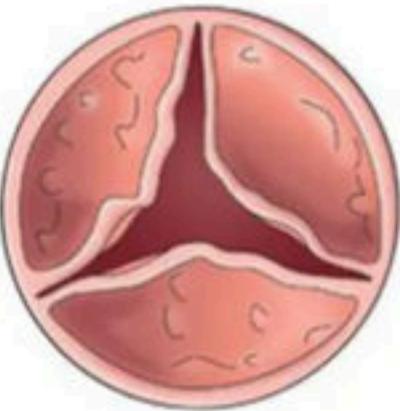
- It occurs in **hypertension or aortic stenosis or congenital bicuspid aortic valve**
- Pressure overloaded ventricles develop **concentric hypertrophy** of the left ventricle, with increased in wall thickness → **Heart size may increase.**
- **The increase in wall thickness may reduce the cavity diameter** → ratio of cavity size to wall thickness decreases.
- **There is increase in the transverse diameter (width) of myocytes, but cell length remains the same.**



**Normal
aortic
valve**



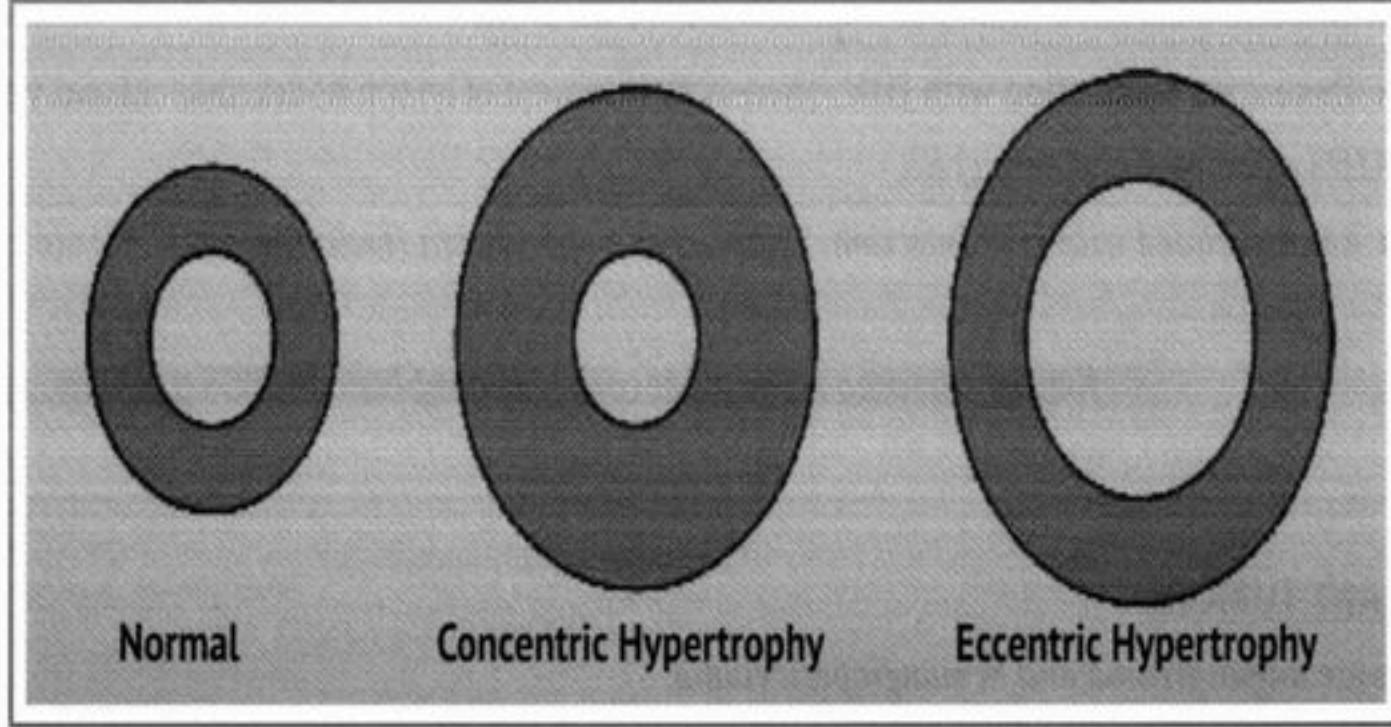
**Stenotic
aortic
valve**

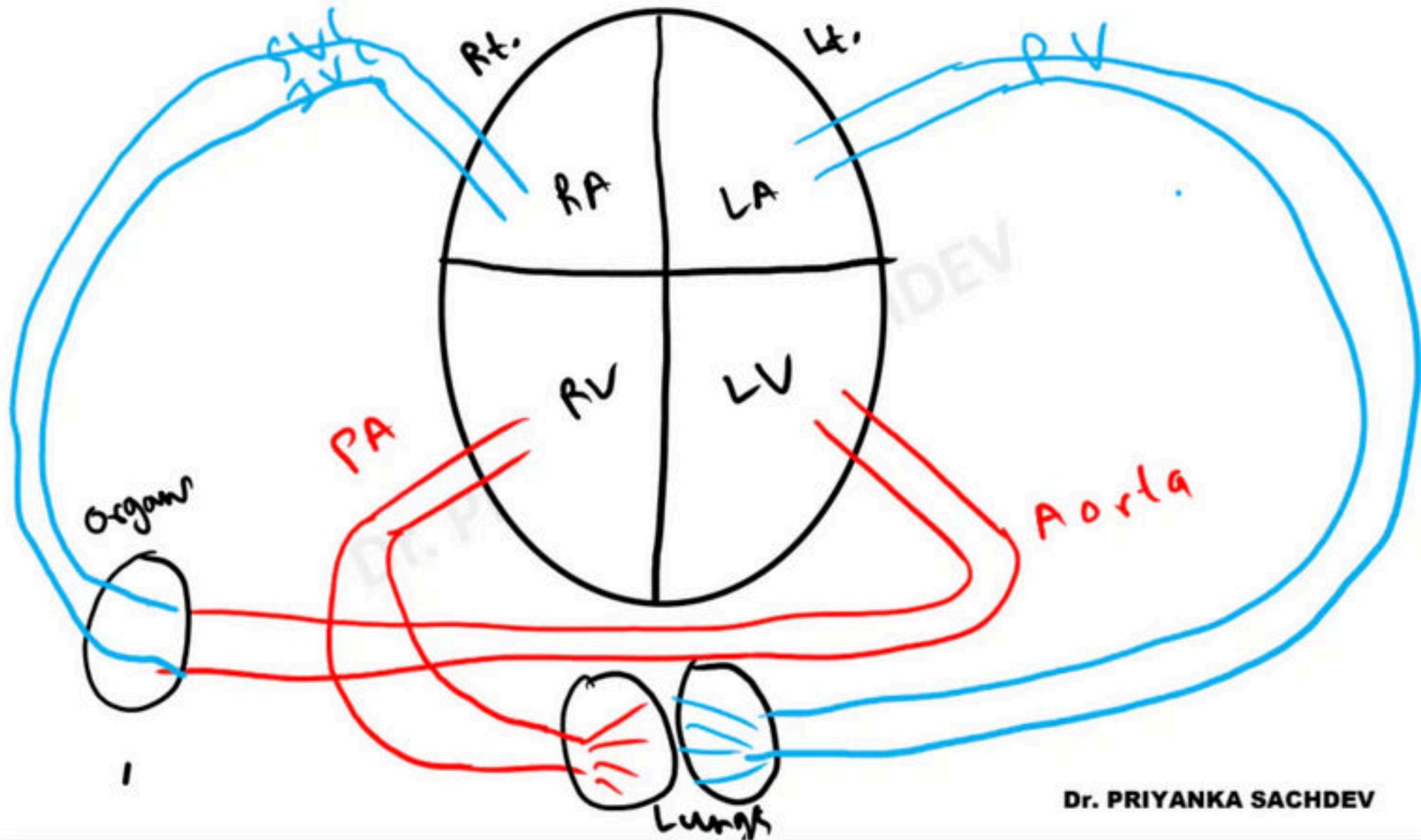


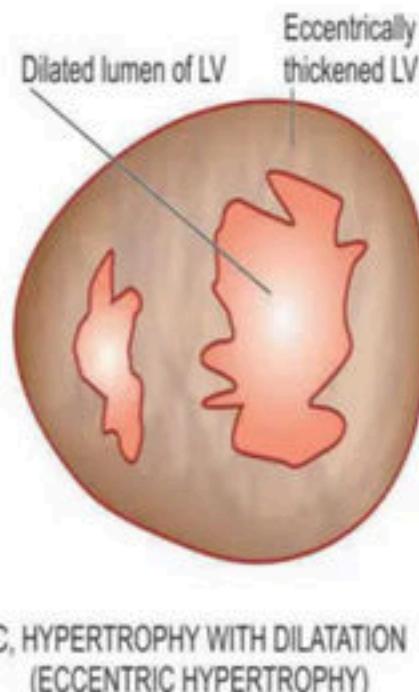
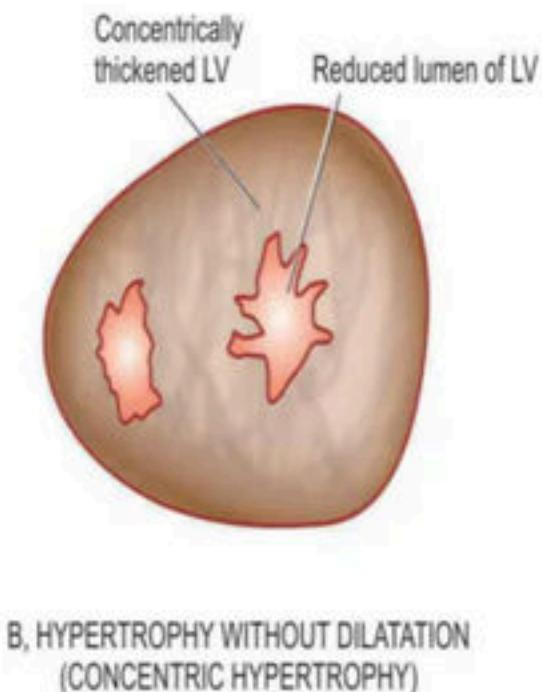
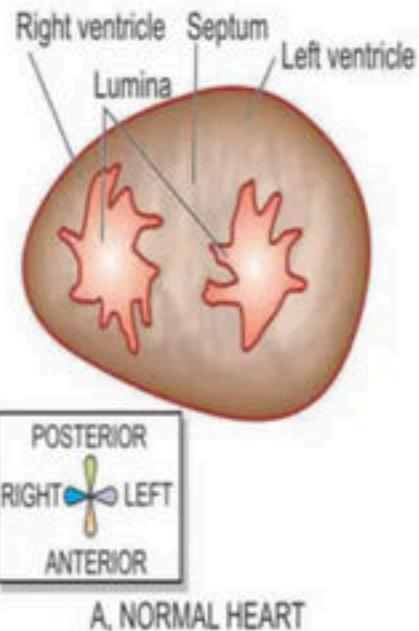
Open



Closed









B

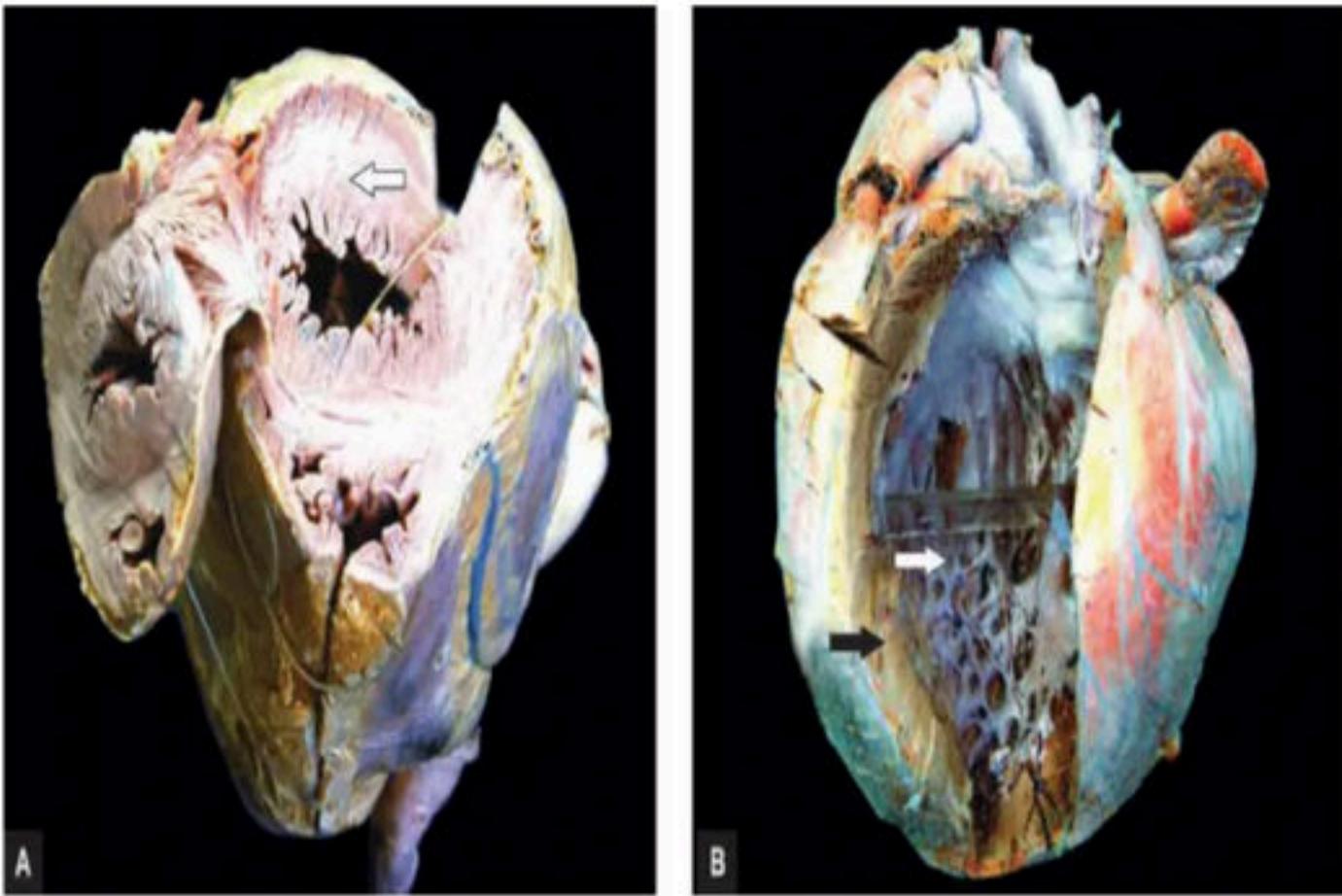
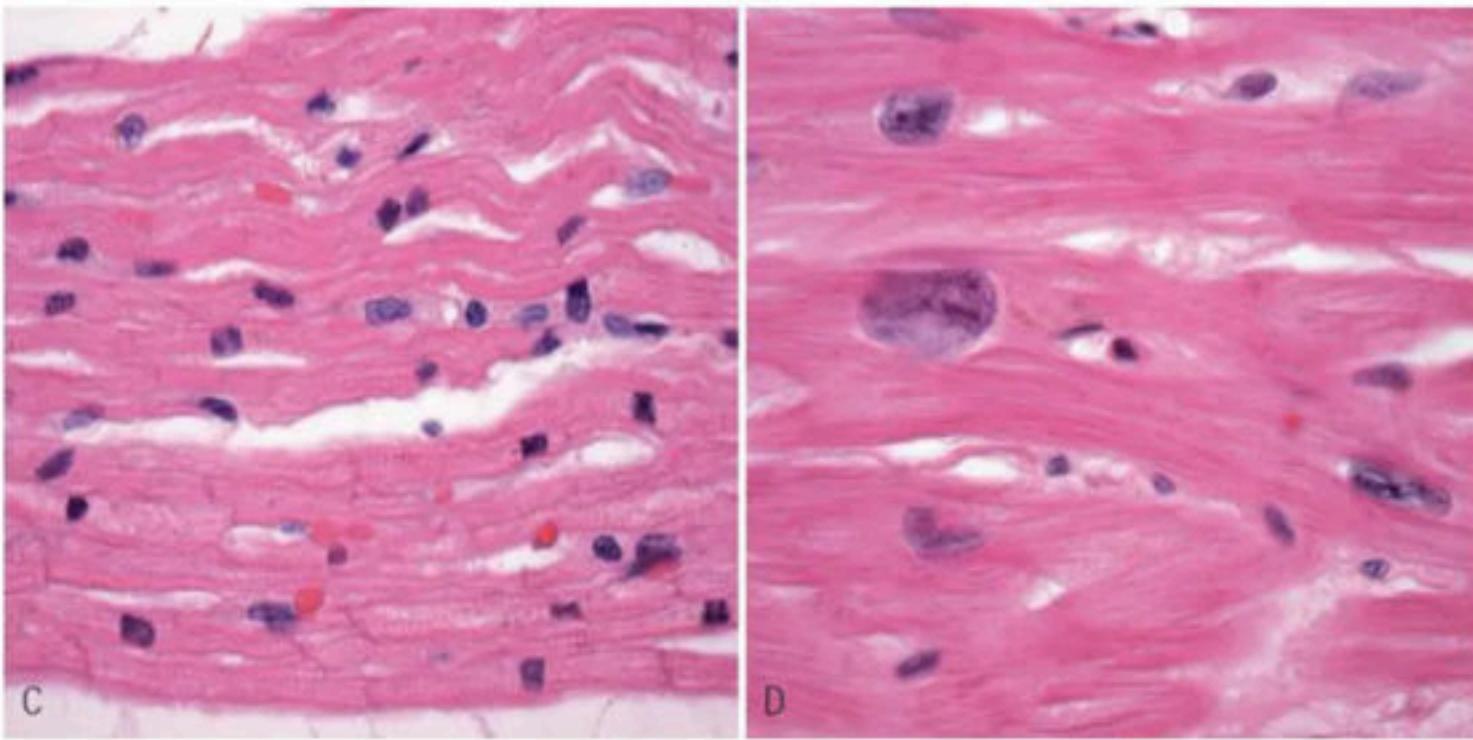


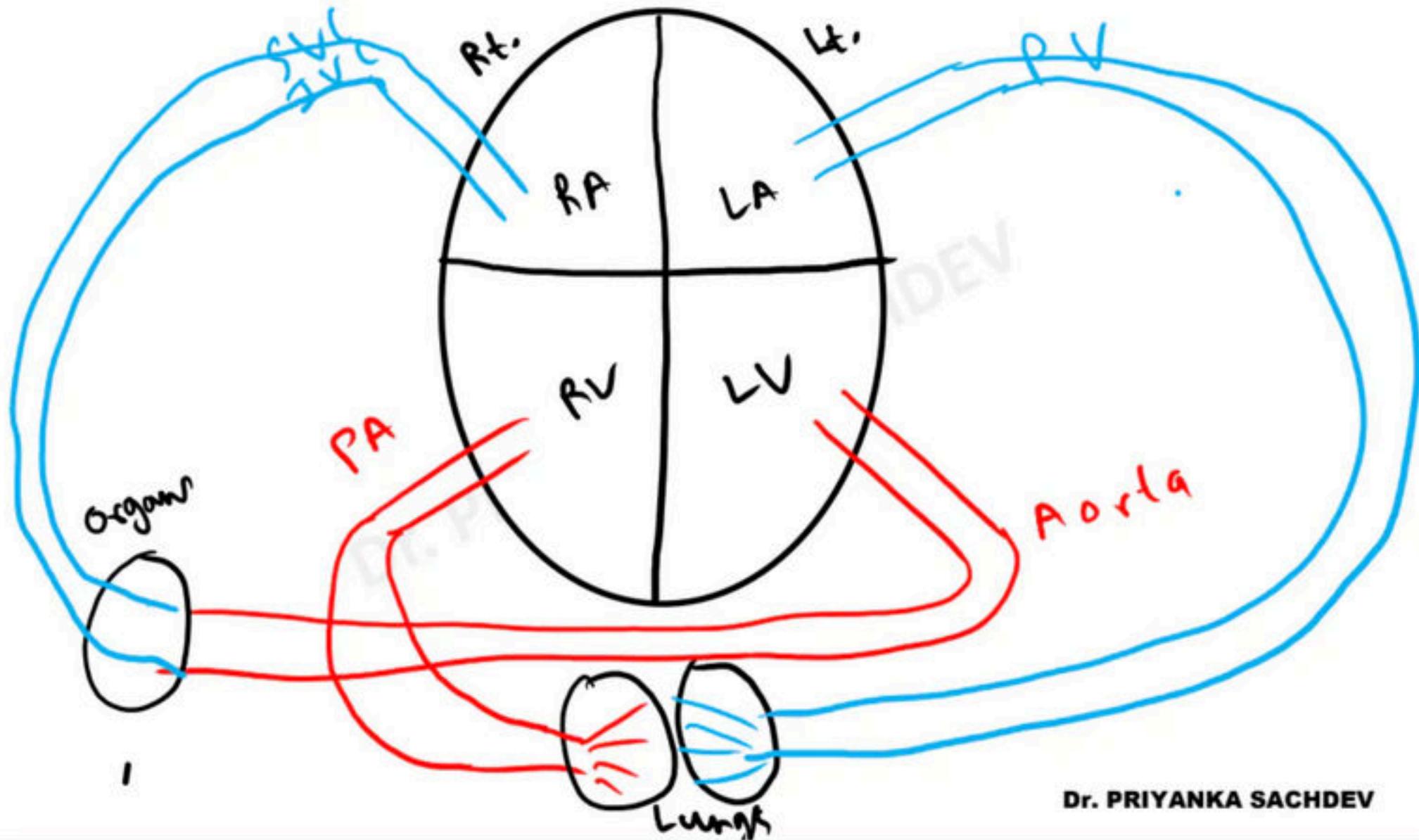
Figure 14.6 A, Concentric cardiac hypertrophy. Weight of the heart is increased. The chambers opened up at the apex show concentric thickening of left ventricular wall (white arrow) with obliterated lumen (hypertrophy without dilatation). B, Eccentric cardiac hypertrophy. The heart is heavier. The free left ventricular wall is thickened (black arrow) while the lumen is dilated (white arrow) (hypertrophy with dilatation).

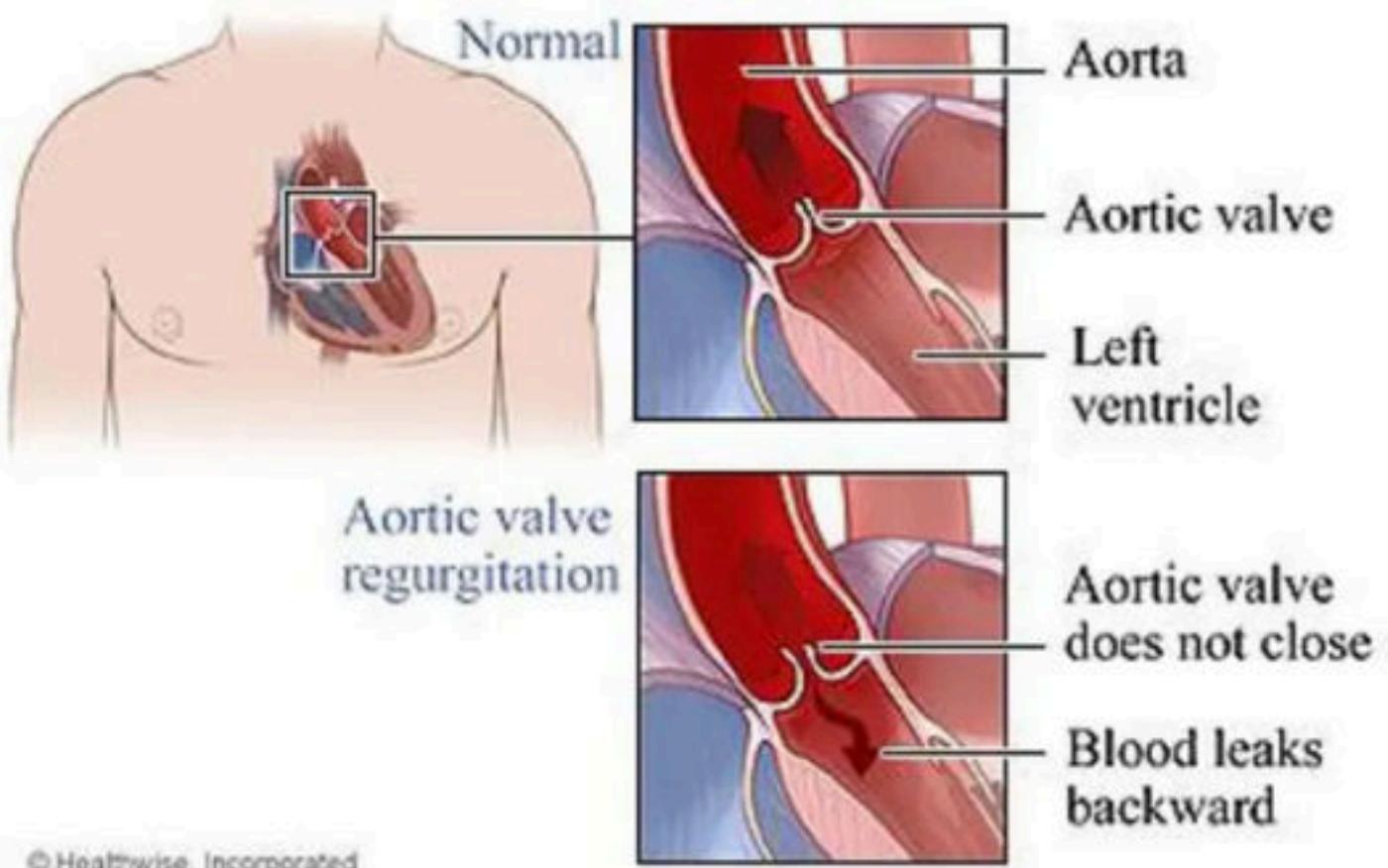


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2. Volume overload

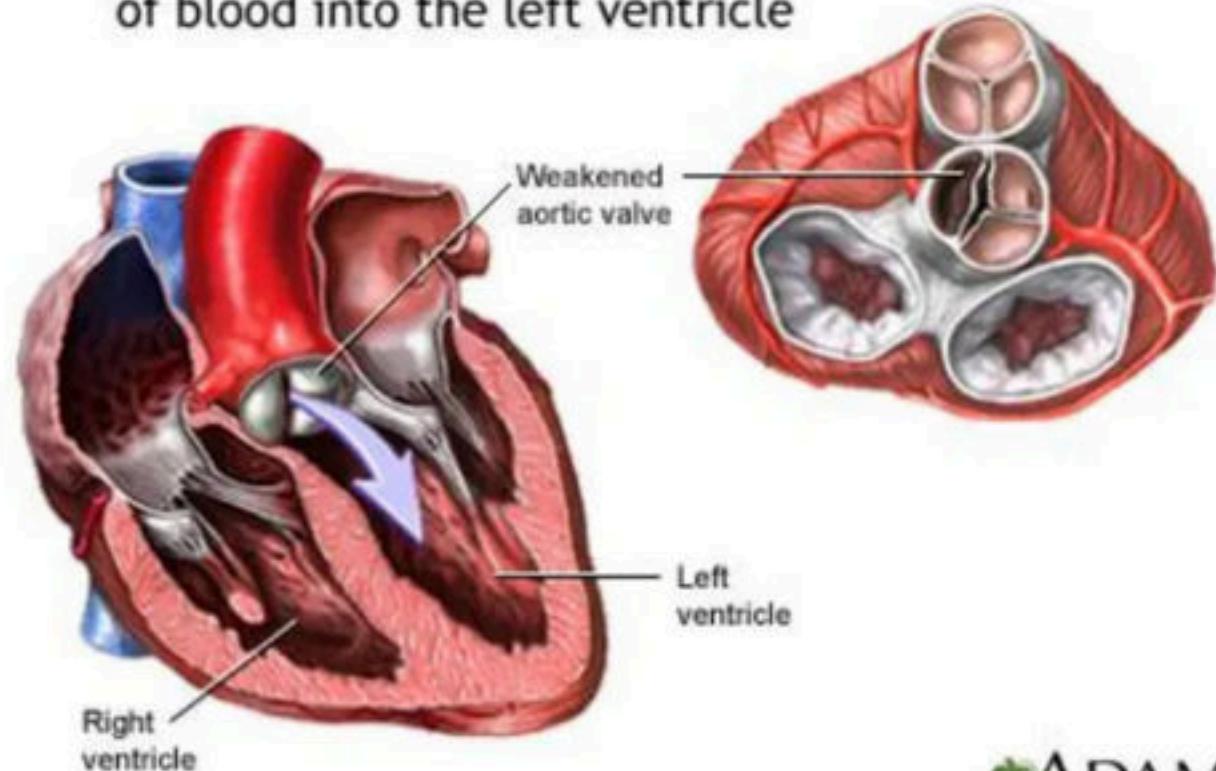
- It occurs in **aortic regurgitation**
- There is **dilatation of ventricular cavity along with increased thickness of ventricular wall → Eccentric hypertrophy.**
- There is **increase both in the transverse diameter (width) and the length of myocytes.**





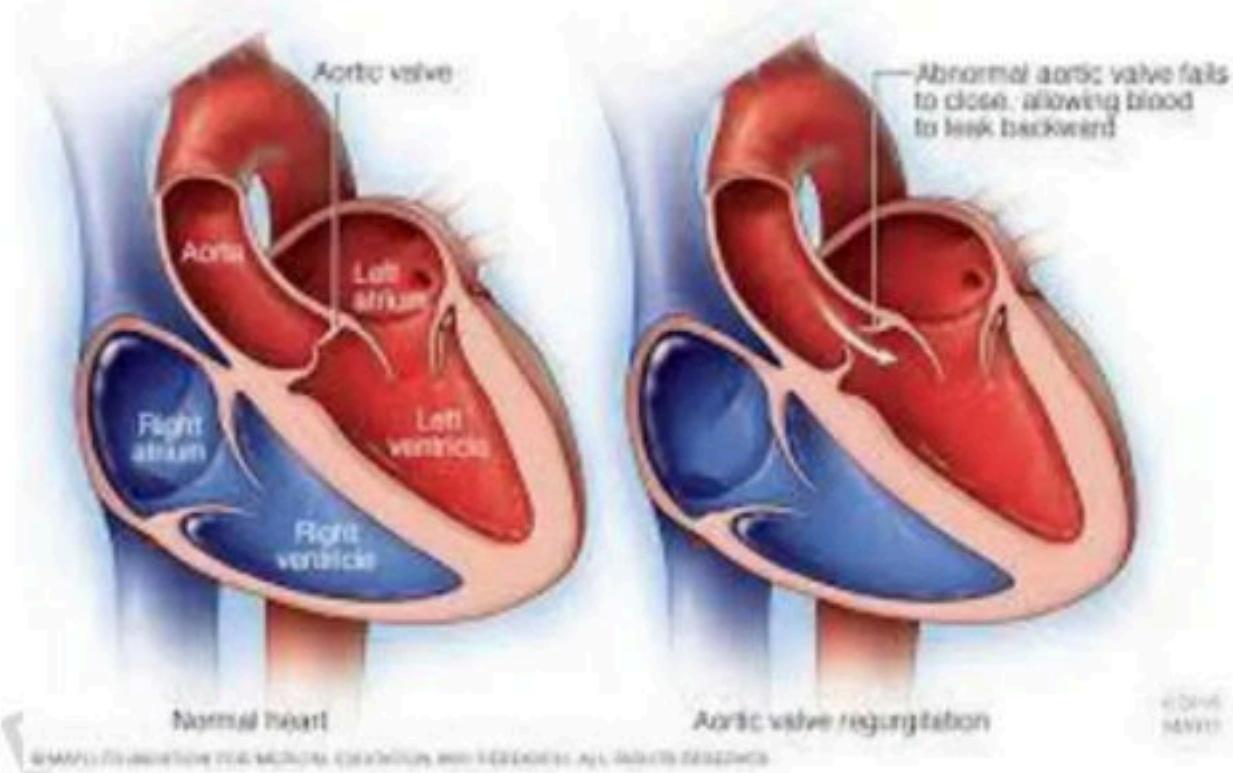
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Failure of the aortic valve to close tightly causes back flow of blood into the left ventricle

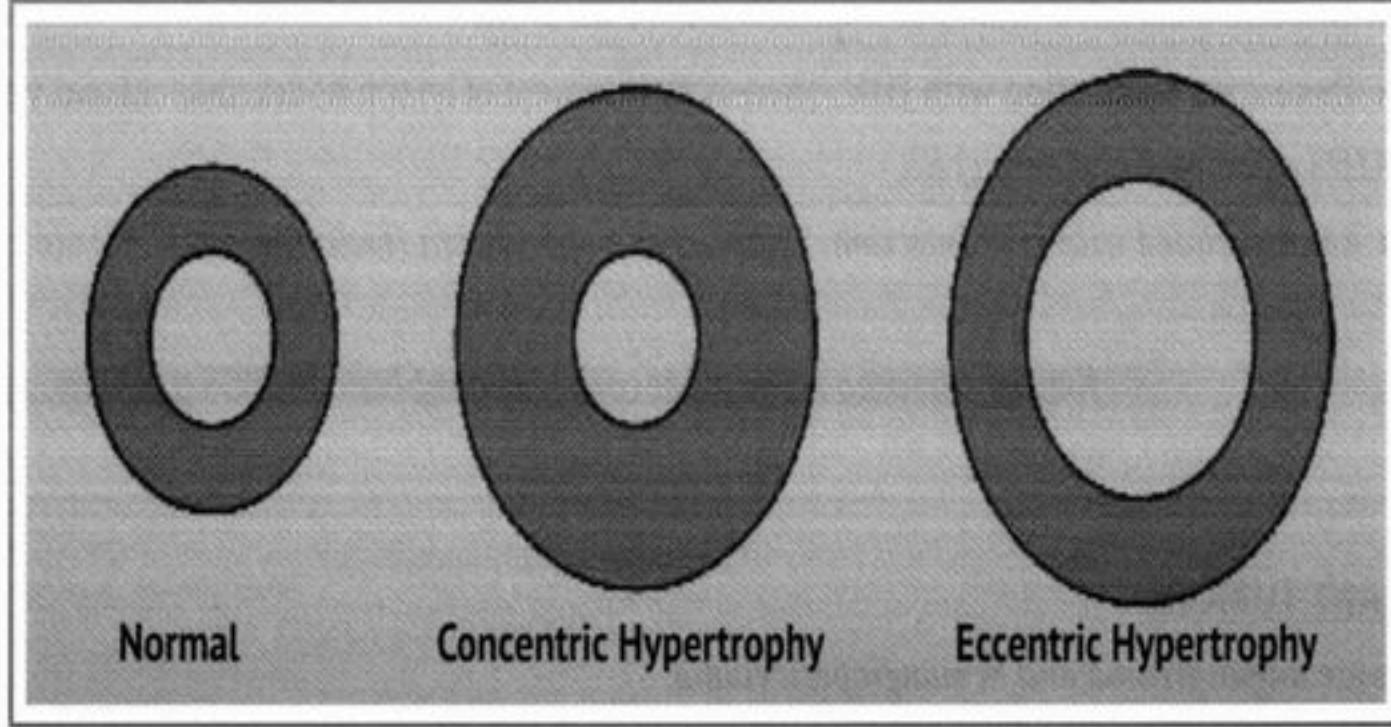


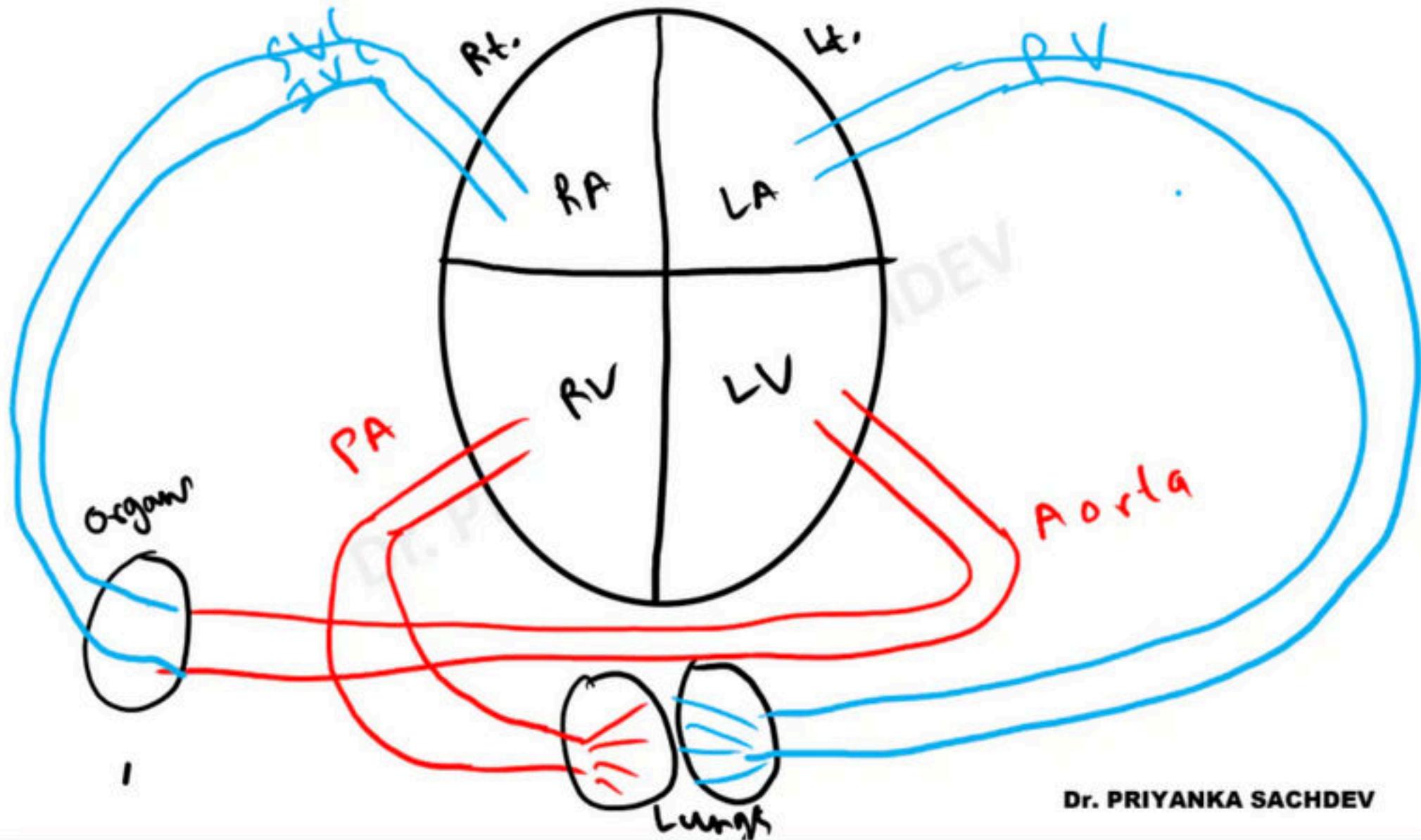
ADAM.

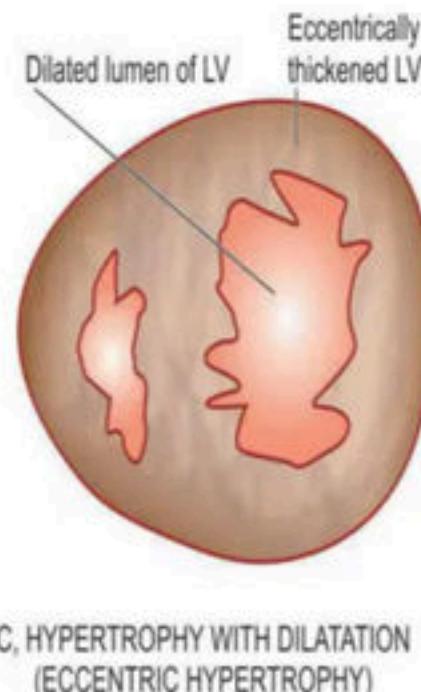
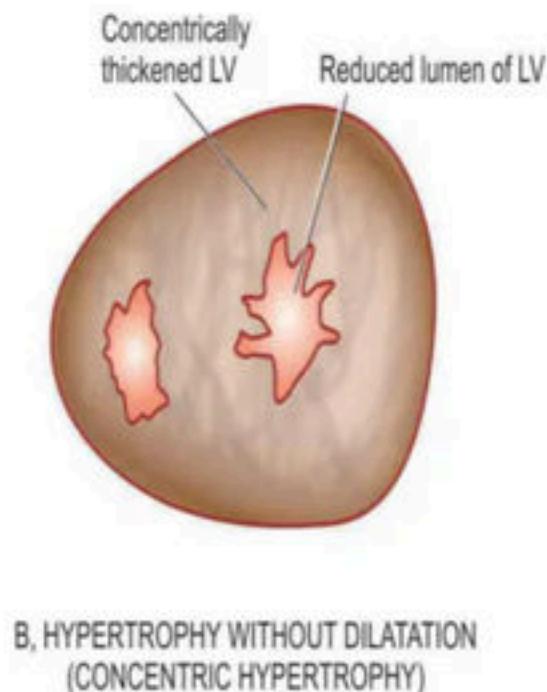
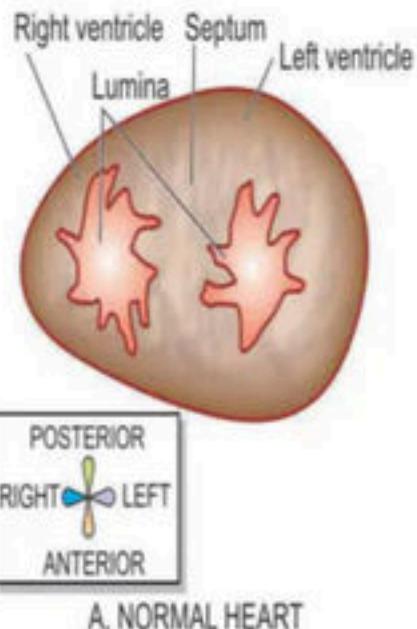
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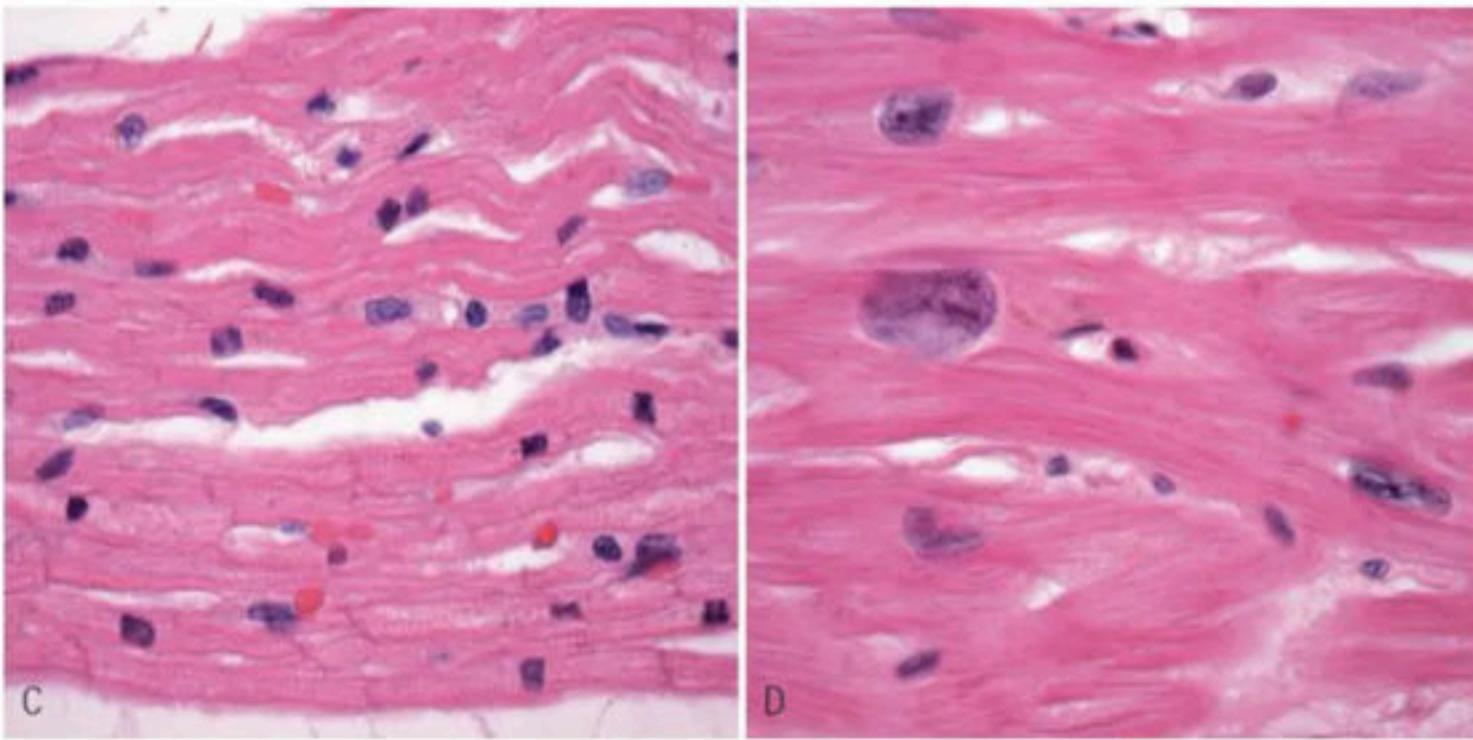








B



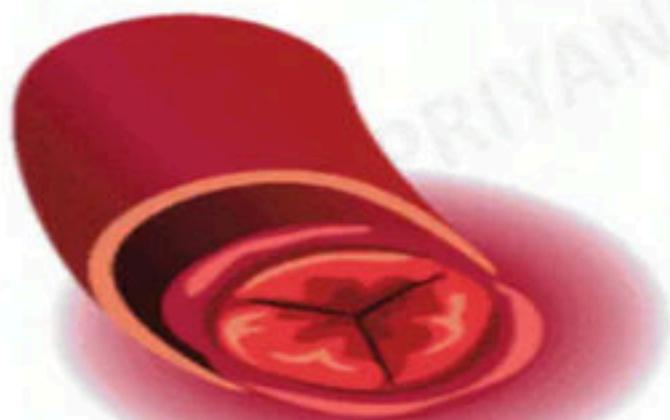
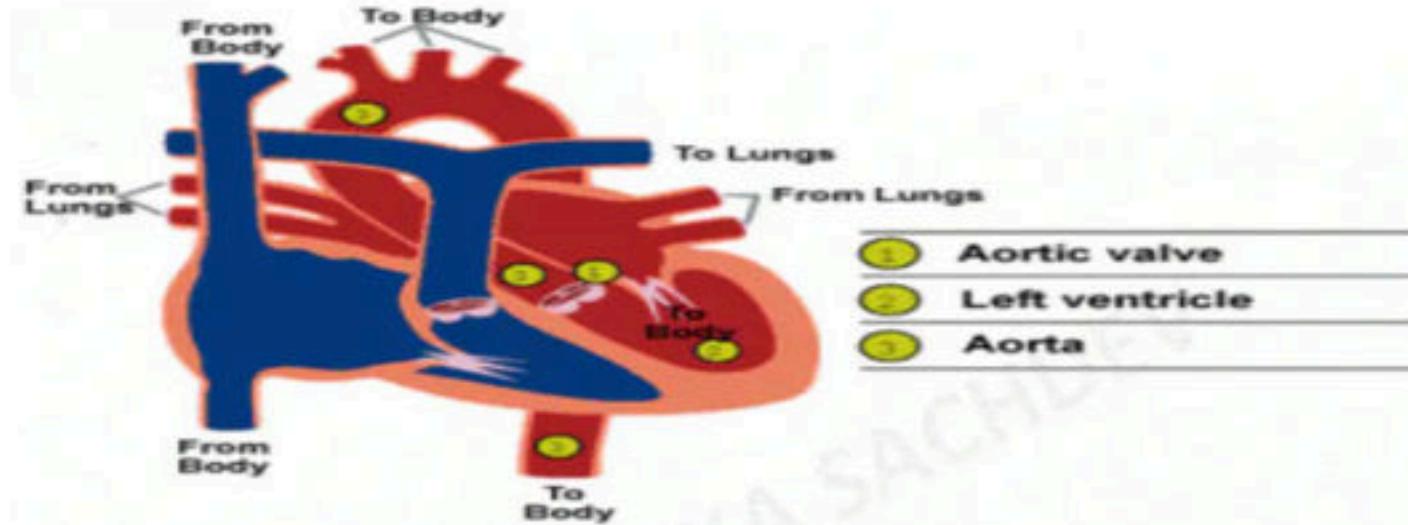
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Pressure overload

- Concentric hypertrophy
- Increase in wall thickness
- Reduce the cavity diameter
- Increase in the transverse diameter (width) of myocytes, but cell length remains the same.

Volume overload

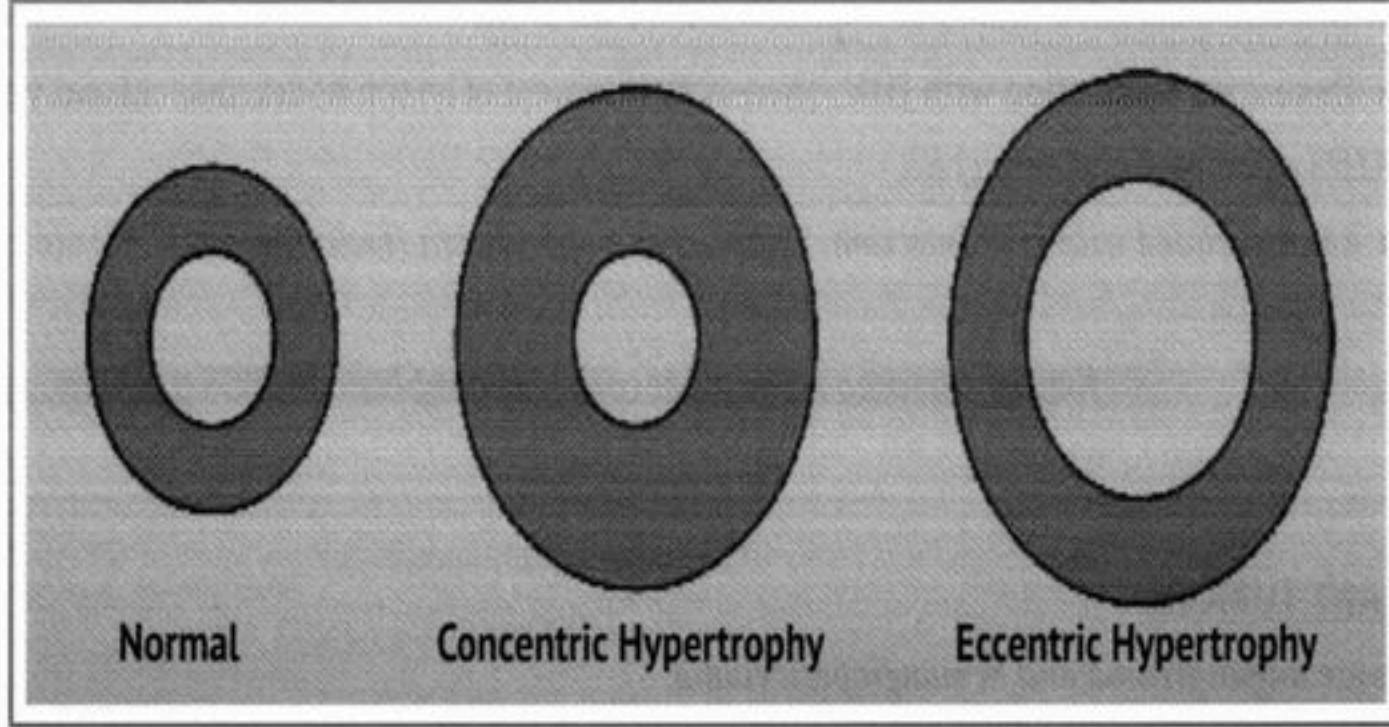
- Eccentric hypertrophy
- Increase in wall thickness
- Dilatation of ventricular cavity
- Increase both in the transverse diameter (width) and the length of myocytes.



④ Three leaflet
normal aortic valve



⑤ Two leaflet
Bicuspid aortic valve



REMEMBER

- Maximum stress to left ventricle is seen when there is **combined pressure overload (aortic stenosis) and volume overload (aortic regurgitation)**

POLLS 1

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Concentric hypertrophy of left ventricle is seen in -

- a) Mitral stenosis
- b) Hypertension
- c) Aortic regurgitation
- d) None

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B

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Concentric hypertrophy of left ventricle is seen in-

- a) Cong. bicuspid aortic valve
- b) MS
- c) AR
- d) HOCM

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A

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HEART FAILURE

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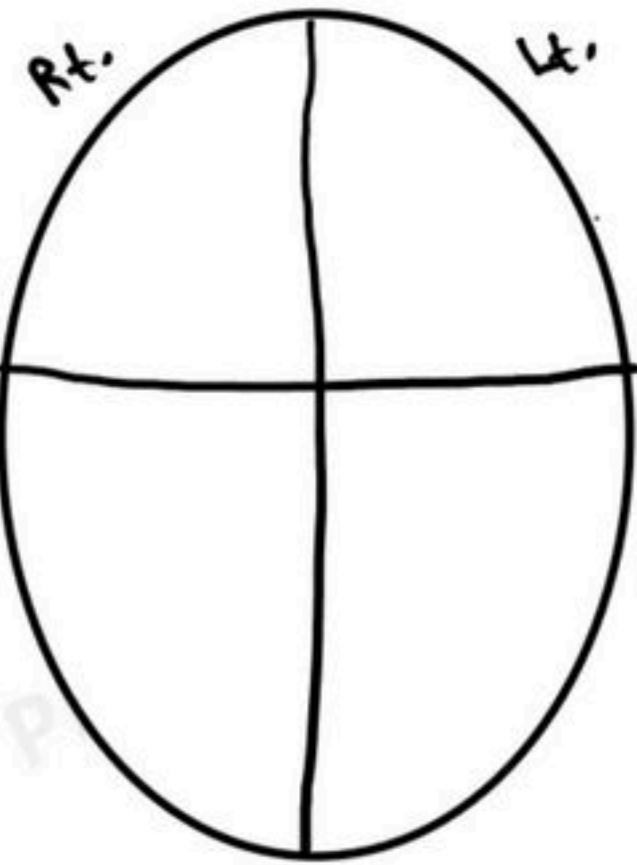
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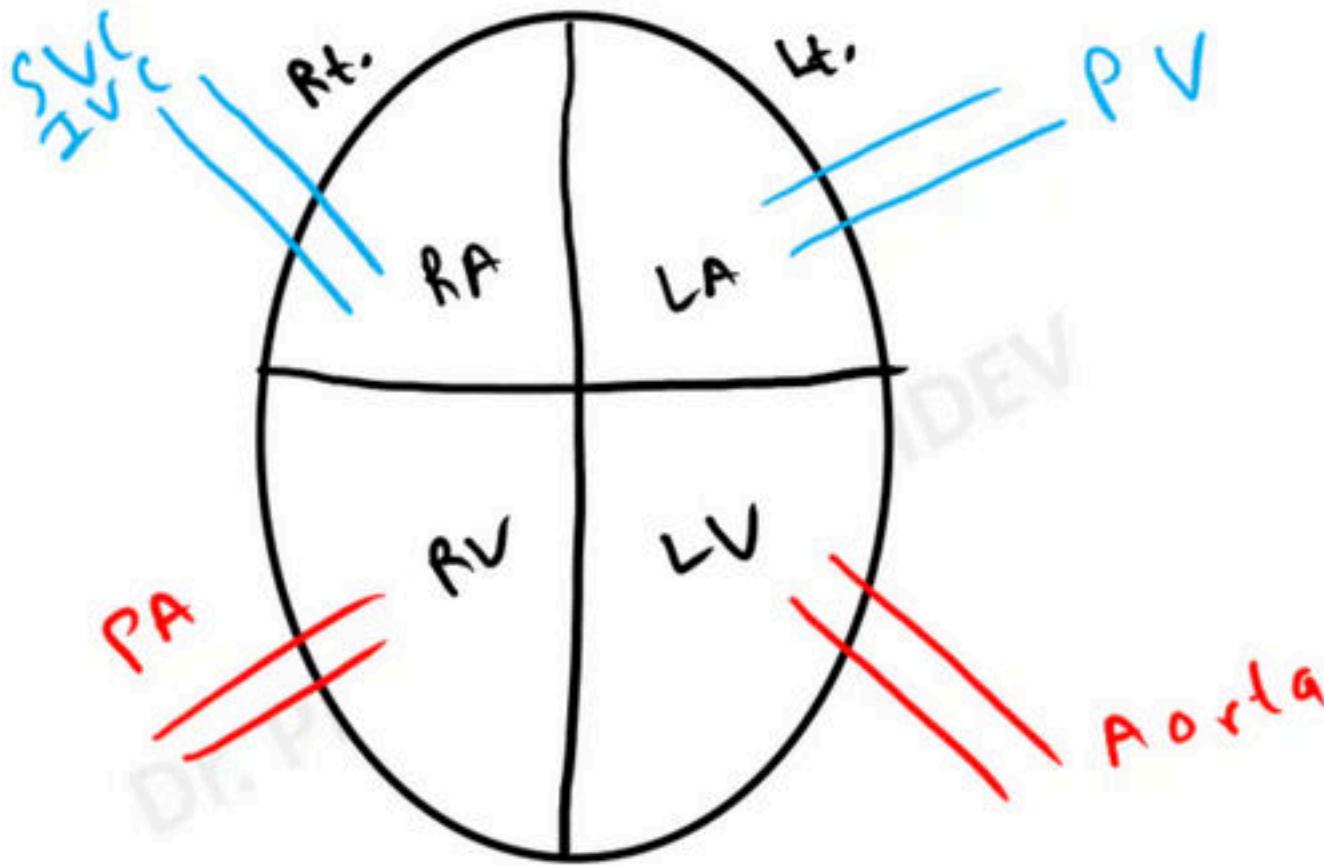
OVERVIEW

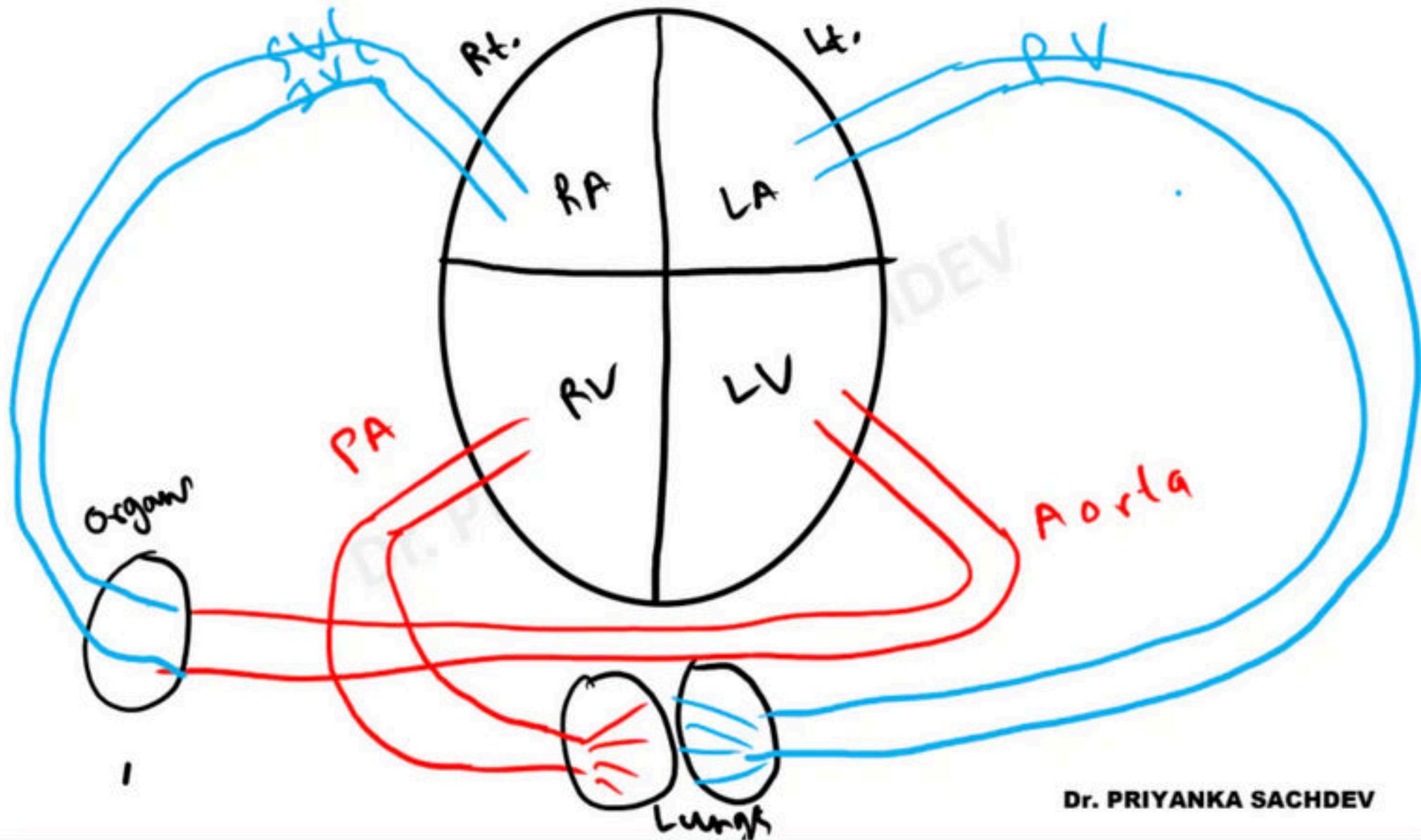
- **Definition**
- **Etiology**
- **Classification**
- **Forward and backward heart failure**
- **Left and right heart failure → Cause and C/F**

Definition

- Heart failure is defined as the pathophysiologic state in which **impaired cardiac function is unable to maintain an adequate circulation for the metabolic needs of the tissues of the body.**







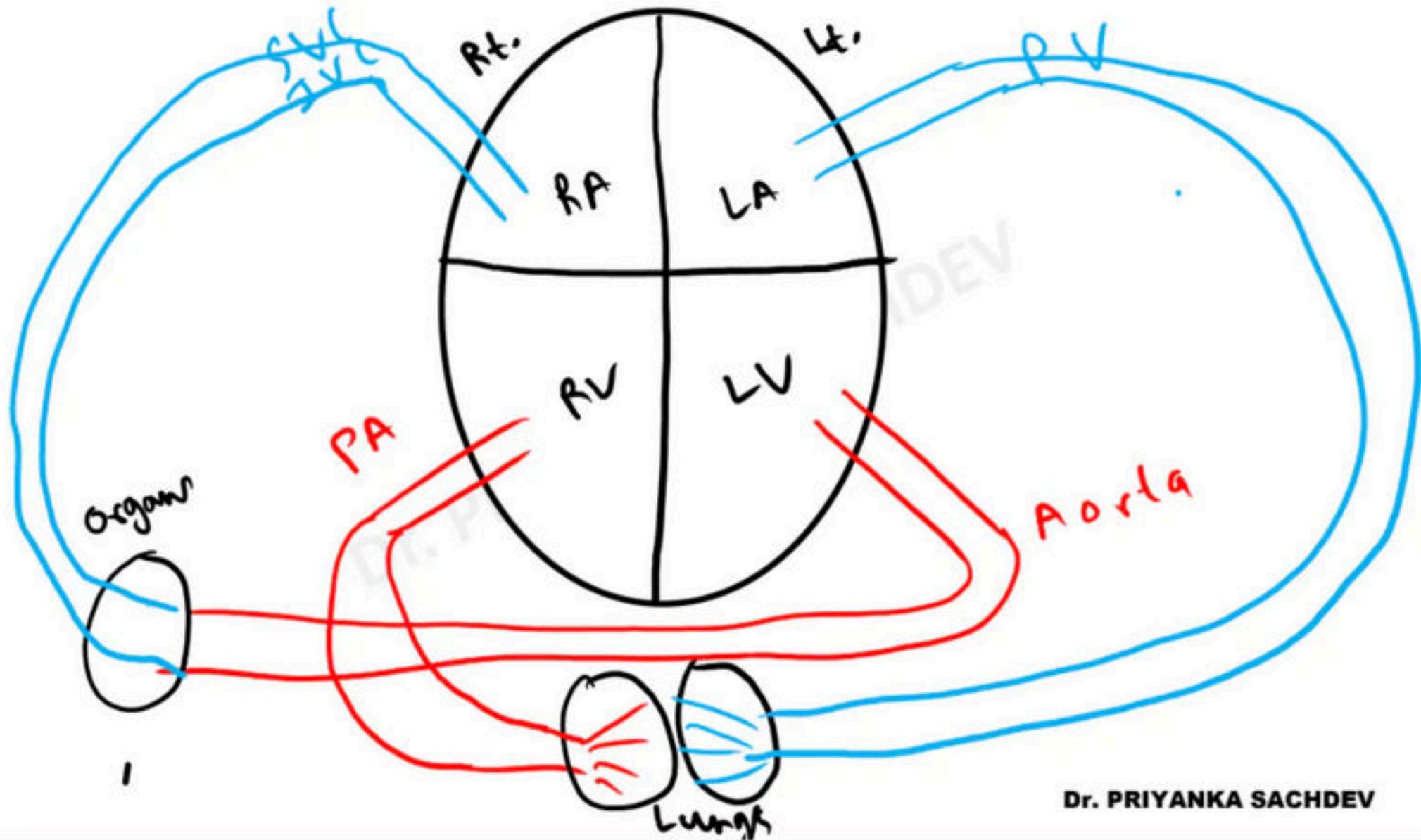
ETIOLOGY

- 1. INTRINSIC PUMP FAILURE**
- 2. INCREASED WORKLOAD ON THE HEART**
- 3. IMPAIRED FILLING OF CARDIAC CHAMBERS**

1. INTRINSIC PUMP FAILURE

The most common and most important cause of heart failure is **weakening of the ventricular muscle** due to disease so that the heart fails to act as an efficient pump.

- i) Ischaemic heart disease
- ii) Myocarditis
- iii) Cardiomyopathies
- iv) Metabolic disorders e.g. beriberi
- v) Disorders of the rhythm e.g. atrial fibrillation and flutter.



2. INCREASED WORKLOAD ON THE HEART

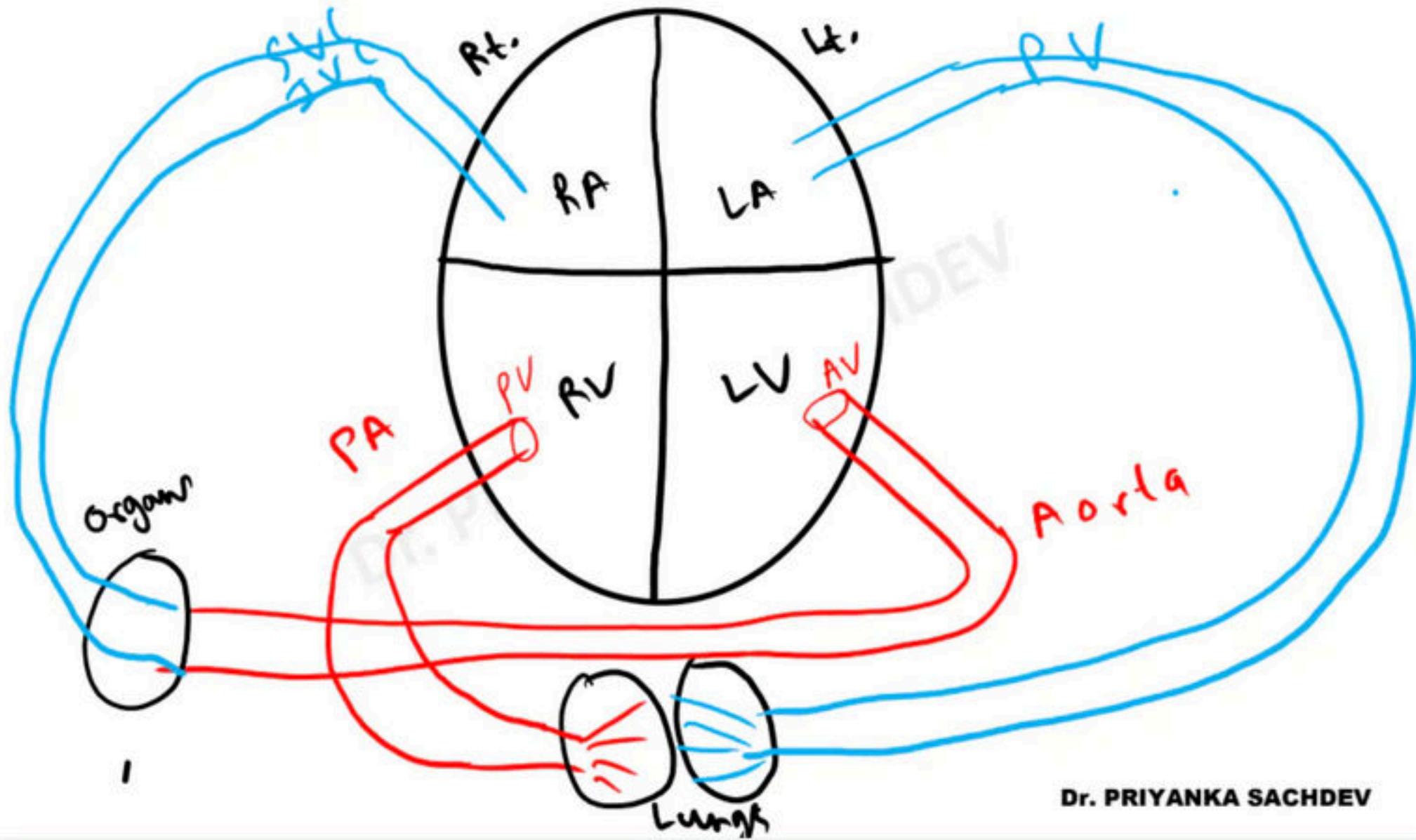
- Increased mechanical load on the heart results in increased myocardial demand resulting in myocardial failure.

i) Increased pressure load may occur in the following states:

- a) Hypertension.
- b) Valvular disease e.g. mitral stenosis, aortic stenosis, pulmonary stenosis

ii) Increased volume load

- a) Valvular insufficiency / regurgitation



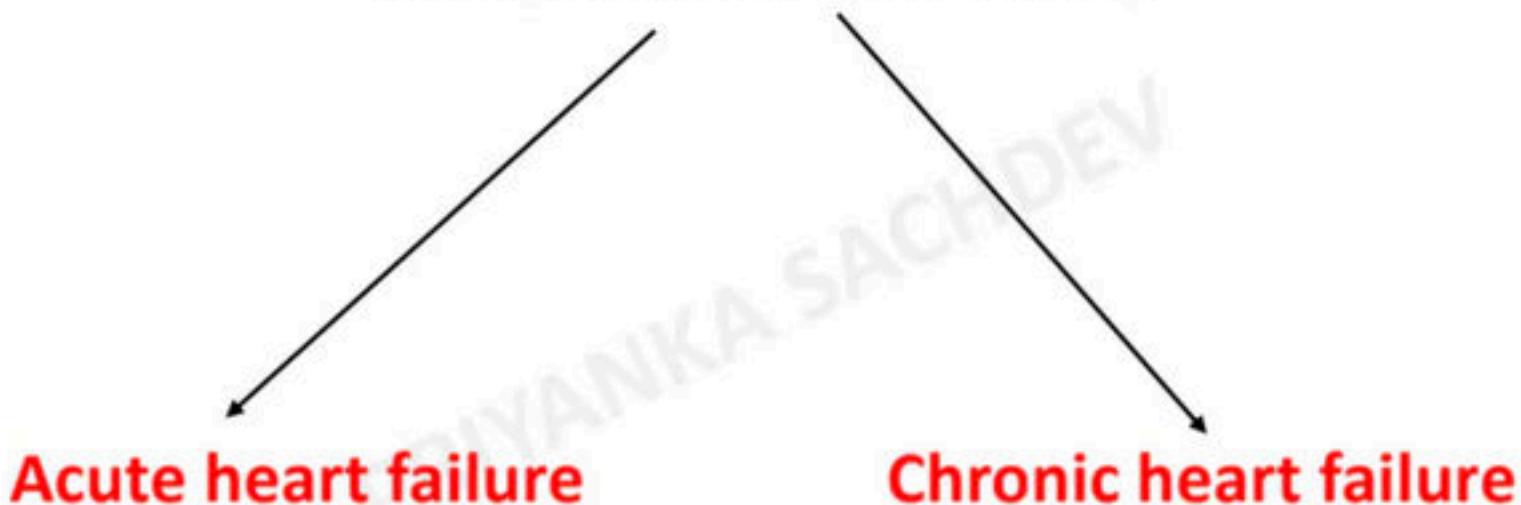
3. IMPAIRED FILLING OF CARDIAC CHAMBERS

- a) Cardiac tamponade e.g.
haemopericardium, hydropericardium
- b) Constrictive pericarditis.

OVERVIEW

- **Definition**
- **Etiology**
- **Classification**
- **Forward and backward heart failure**
- **Left and right heart failure → Cause and C/F**

HEART FAILURE **(congestive heart failure)**

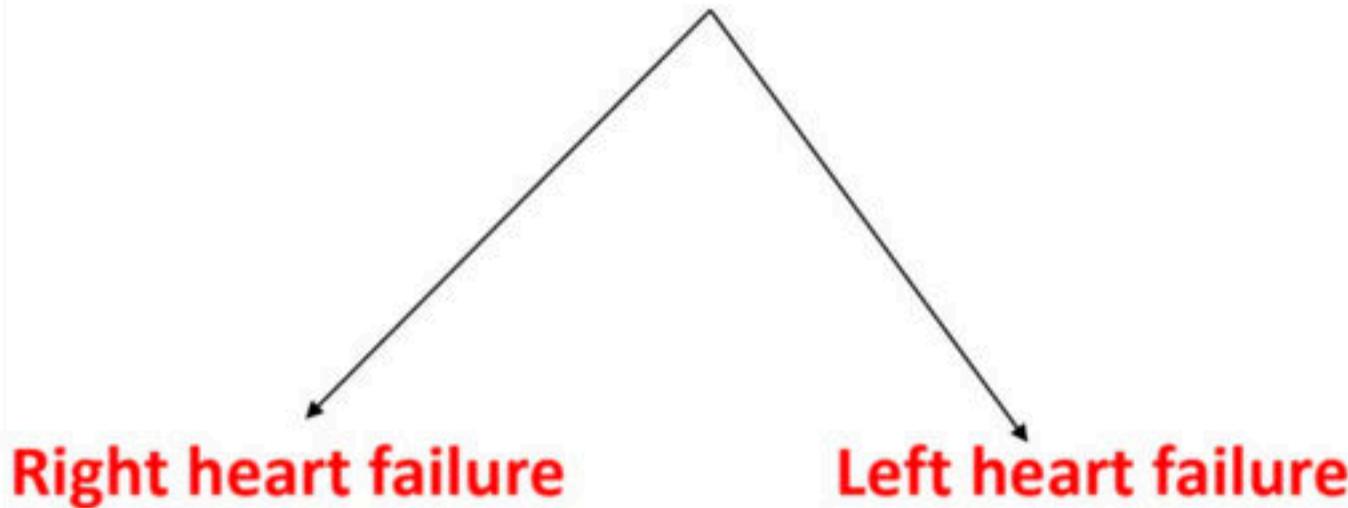


HEART FAILURE

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graph TD; A[HEART FAILURE] --> B[Forward heart failure]; A --> C[Backward heart failure]
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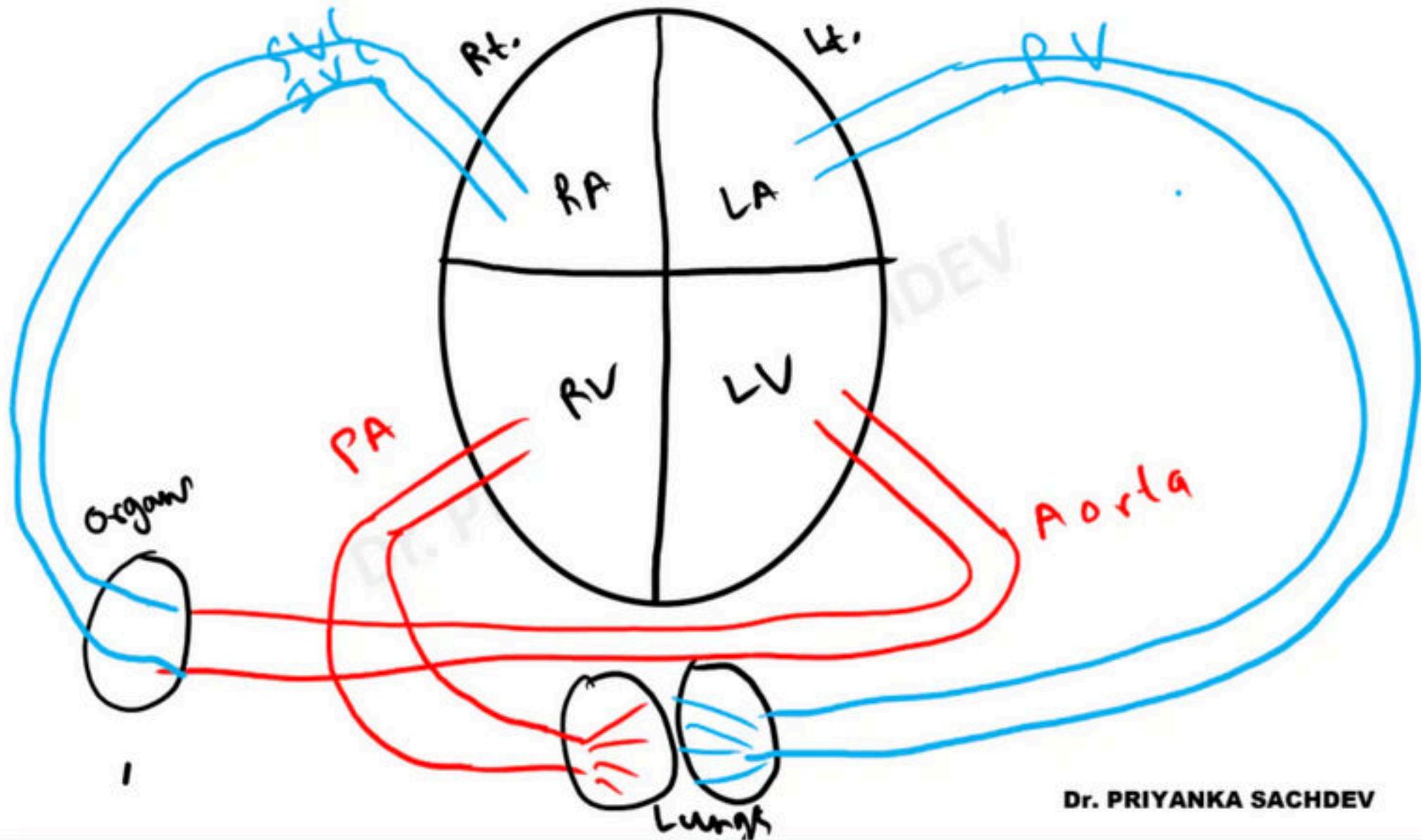
Forward heart failure Backward heart failure

HEART FAILURE



BACKWARD AND FORWARD HEART FAILURE

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Backward heart failure

- According to this concept, either of the ventricles fails to eject blood normally, resulting in rise of end-diastolic volume in the ventricle and increase in volume and pressure in the atrium which is **transmitted backward** producing elevated pressure in the veins

Either of the ventricles fails to eject blood normally



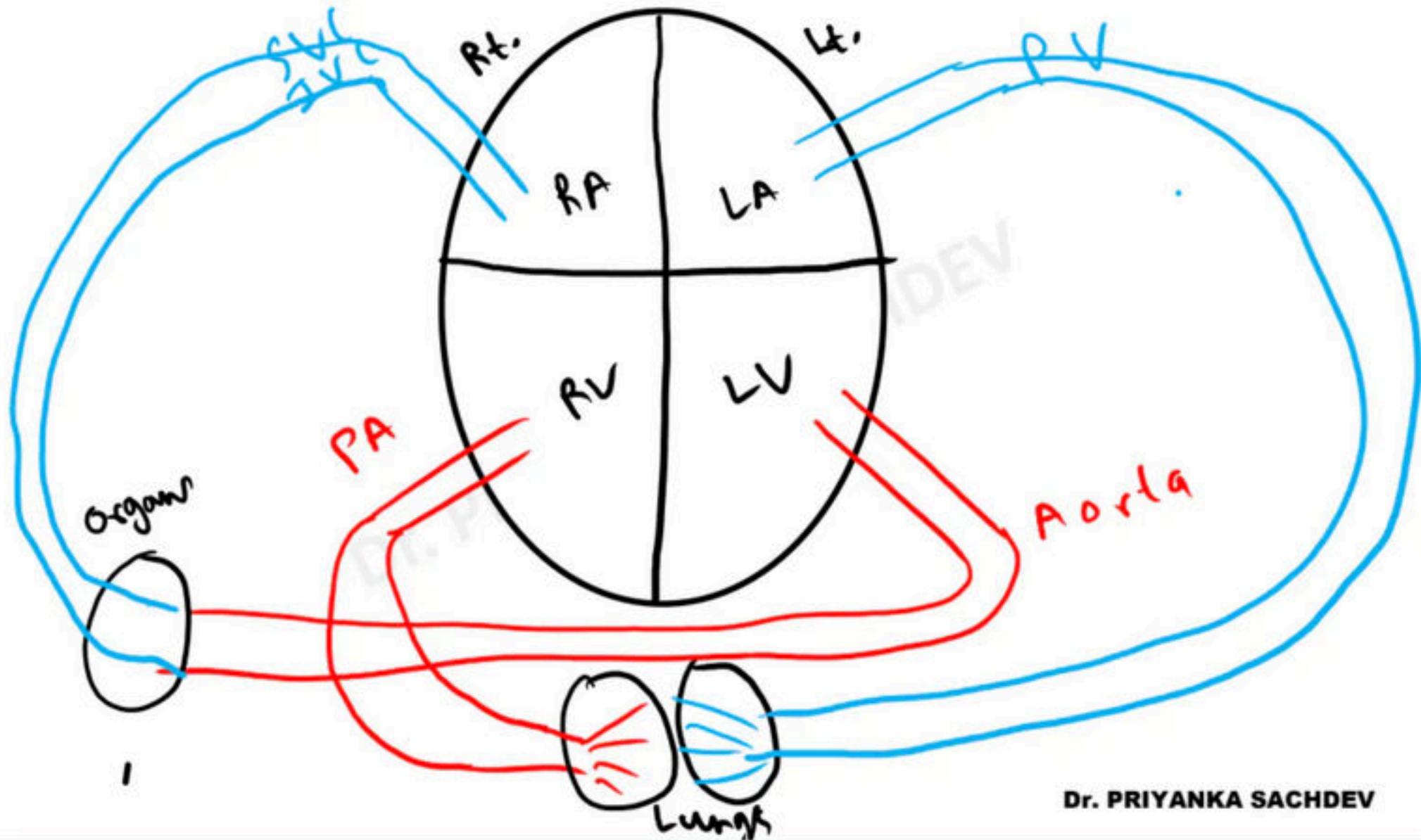
Rise of end-diastolic volume in the ventricle



Increase in volume and pressure in the atrium



This pressure is transmitted backward producing elevated pressure in the veins



Forward heart failure

- According to this hypothesis, clinical manifestations result directly from **failure of the heart to pump blood** causing diminished flow of blood to the tissues

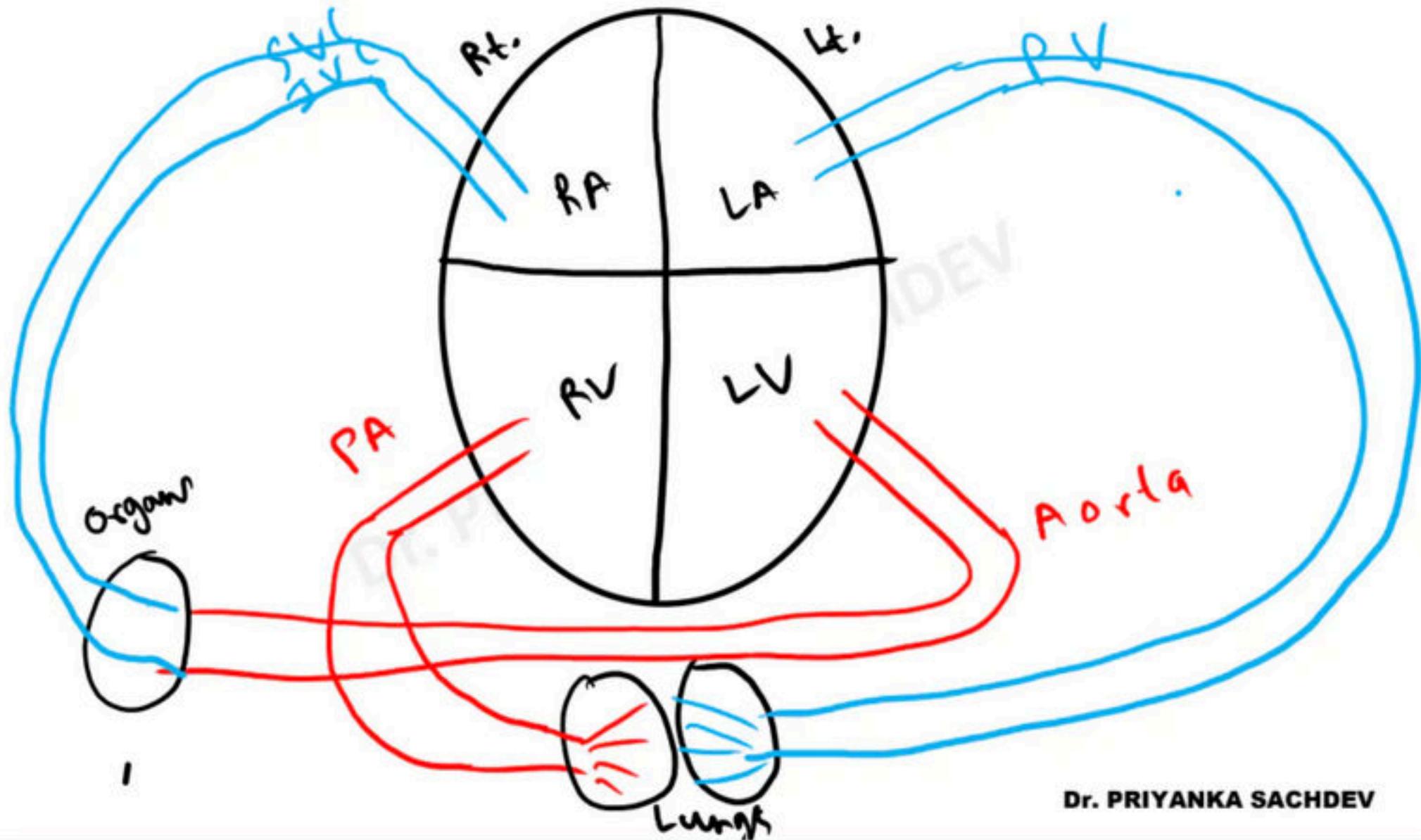
Failure of the heart to pump blood

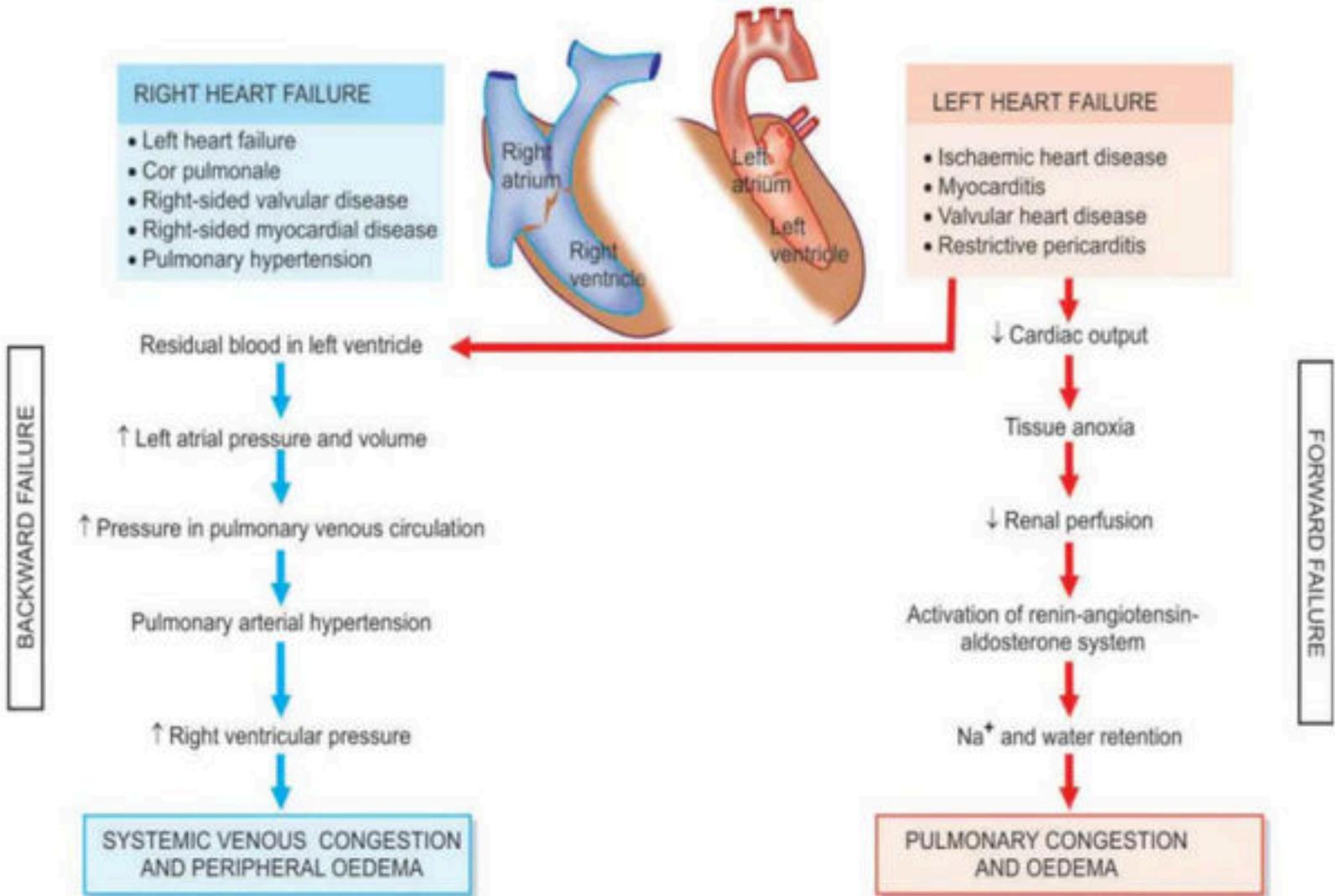


Diminished flow of blood to the tissues



Clinical manifestations





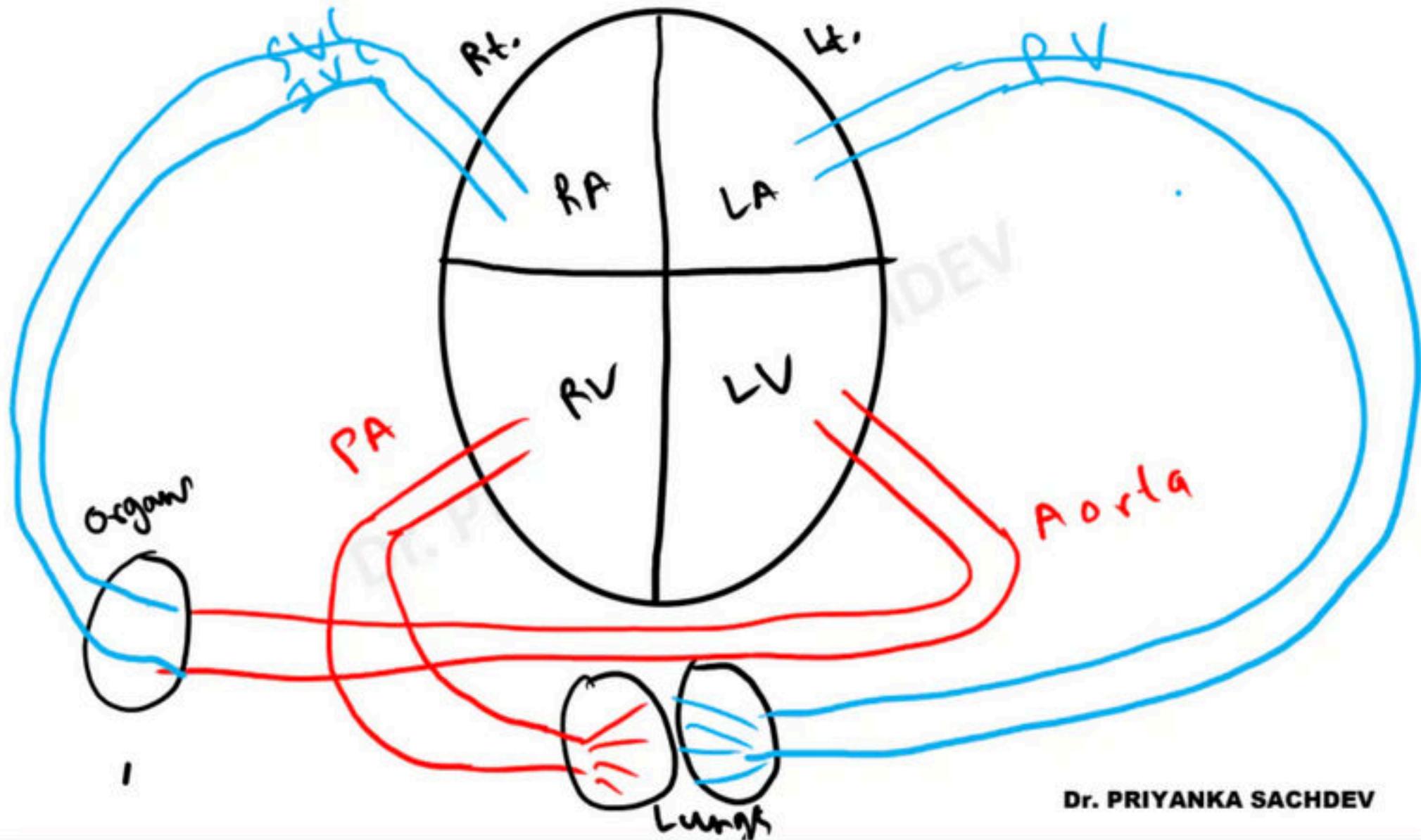
Left-sided heart failure

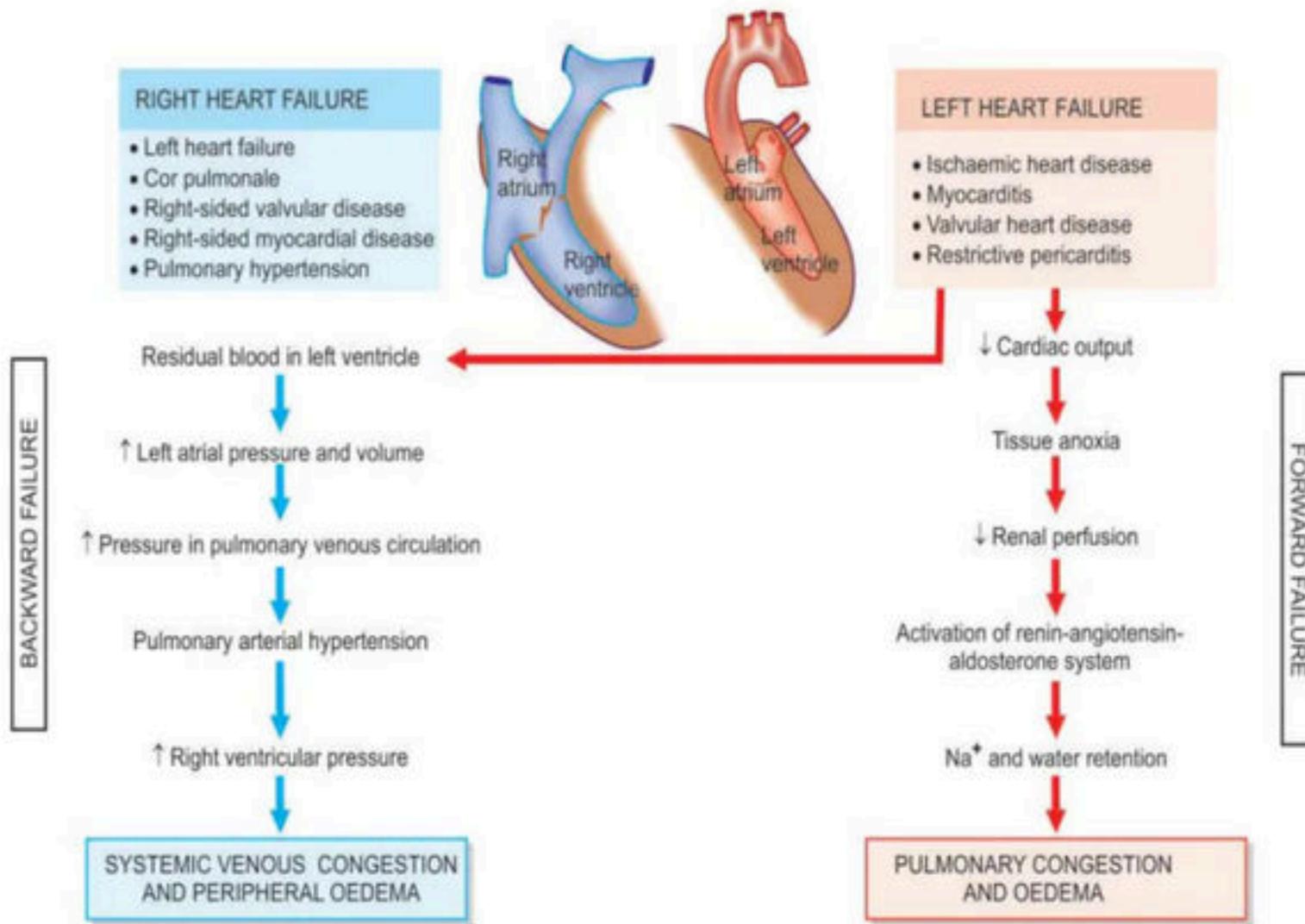
Right-sided heart failure

Left-sided heart failure

Causes :

- i) Systemic hypertension
- ii) Mitral or aortic valve disease (stenosis)
- iii) Ischaemic heart disease
- iv) Myocardial diseases e.g.
cardiomyopathies, myocarditis.
- v) Restrictive pericarditis.

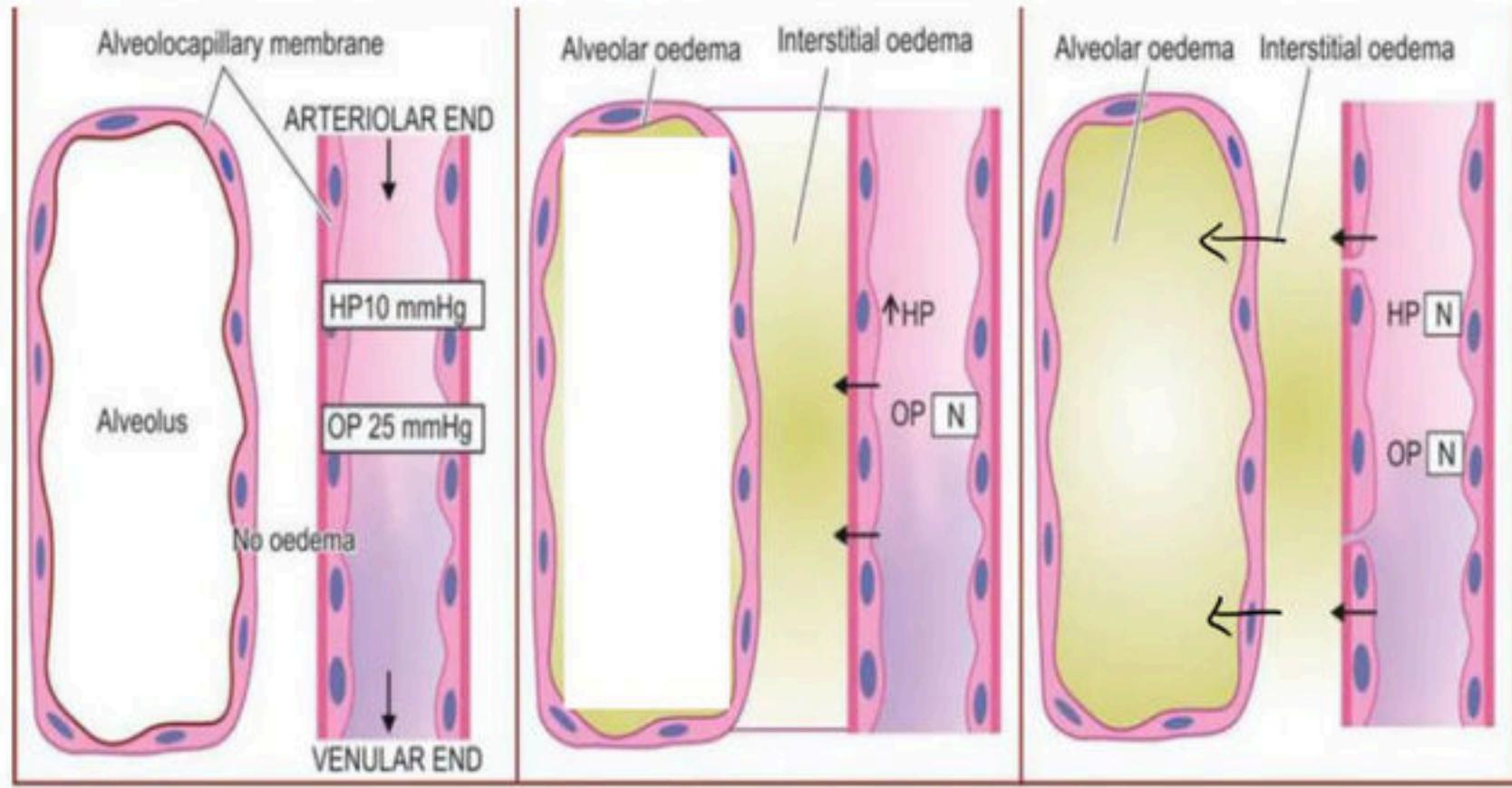




Clinical features of left heart failure

1) Lungs

- Lungs are **most commonly affected organs** in LVF.
- There is pulmonary congestion and pulmonary edema.
- Clinical manifestations due to these changes in lungs : Dyspnea (earliest feature), orthopnea



A, NORMAL

B, ↑ PULMONARY HP

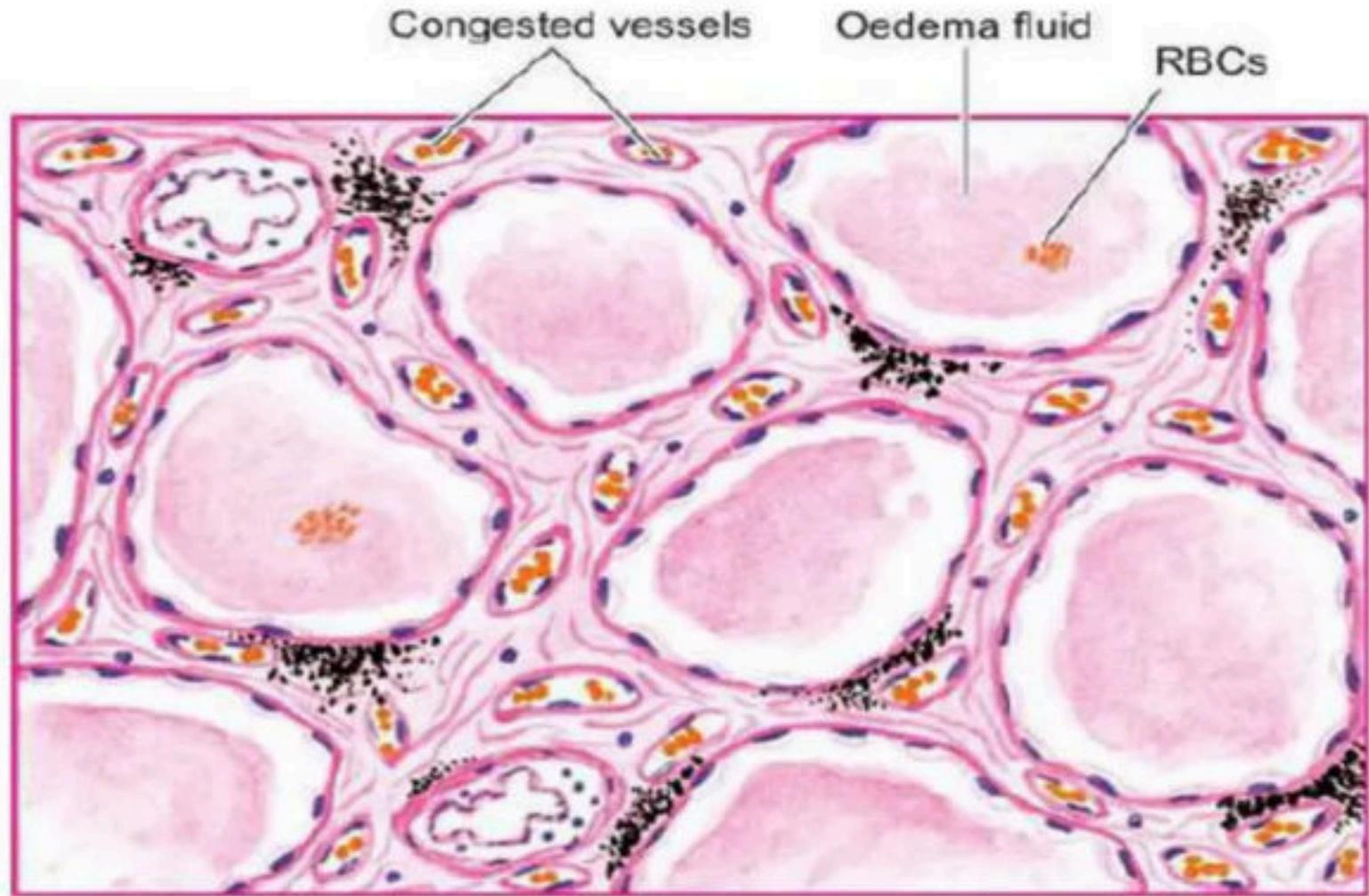
C, ↑ PERMEABILITY

Interstitial
oedema

Alveolar
oedema

Microscopically

- The alveolar capillaries are congested.
- Initially, the excess fluid collects in the interstitial lung spaces in the septal walls (**interstitial oedema**).
- Later, the fluid fills the alveolar spaces (**alveolar oedema**).
- There are hemosiderin-containing macrophages in lung alveoli. These macrophages are called **heart failure Cells**



2) Kidney

- Reduced renal perfusion may cause pre-renal ARF(pre-renal azotemia).

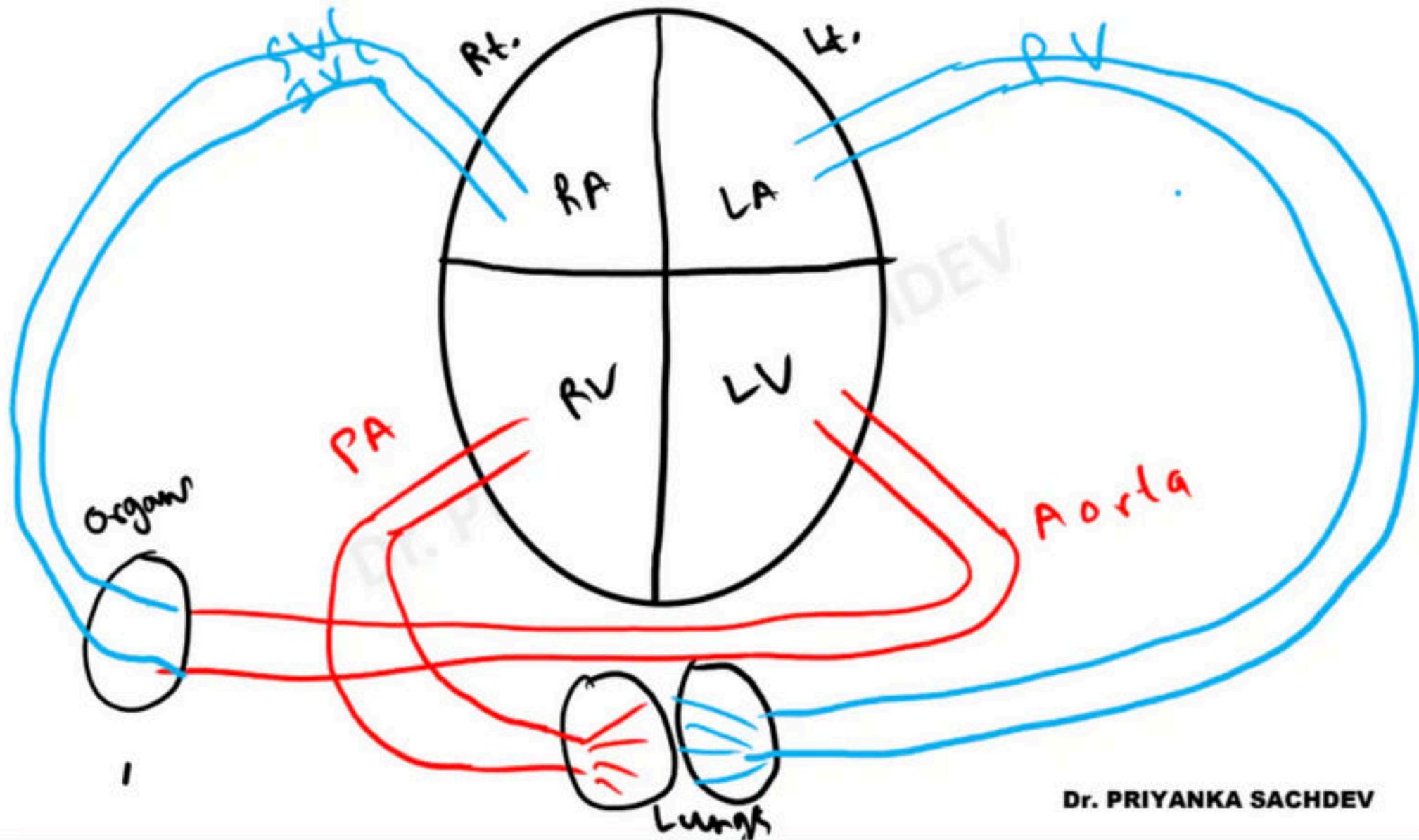
3) Brain

- Reduced cerebral perfusion may cause hypoxic-ischemic encephalopathy

Right-sided heart failure

CAUSES

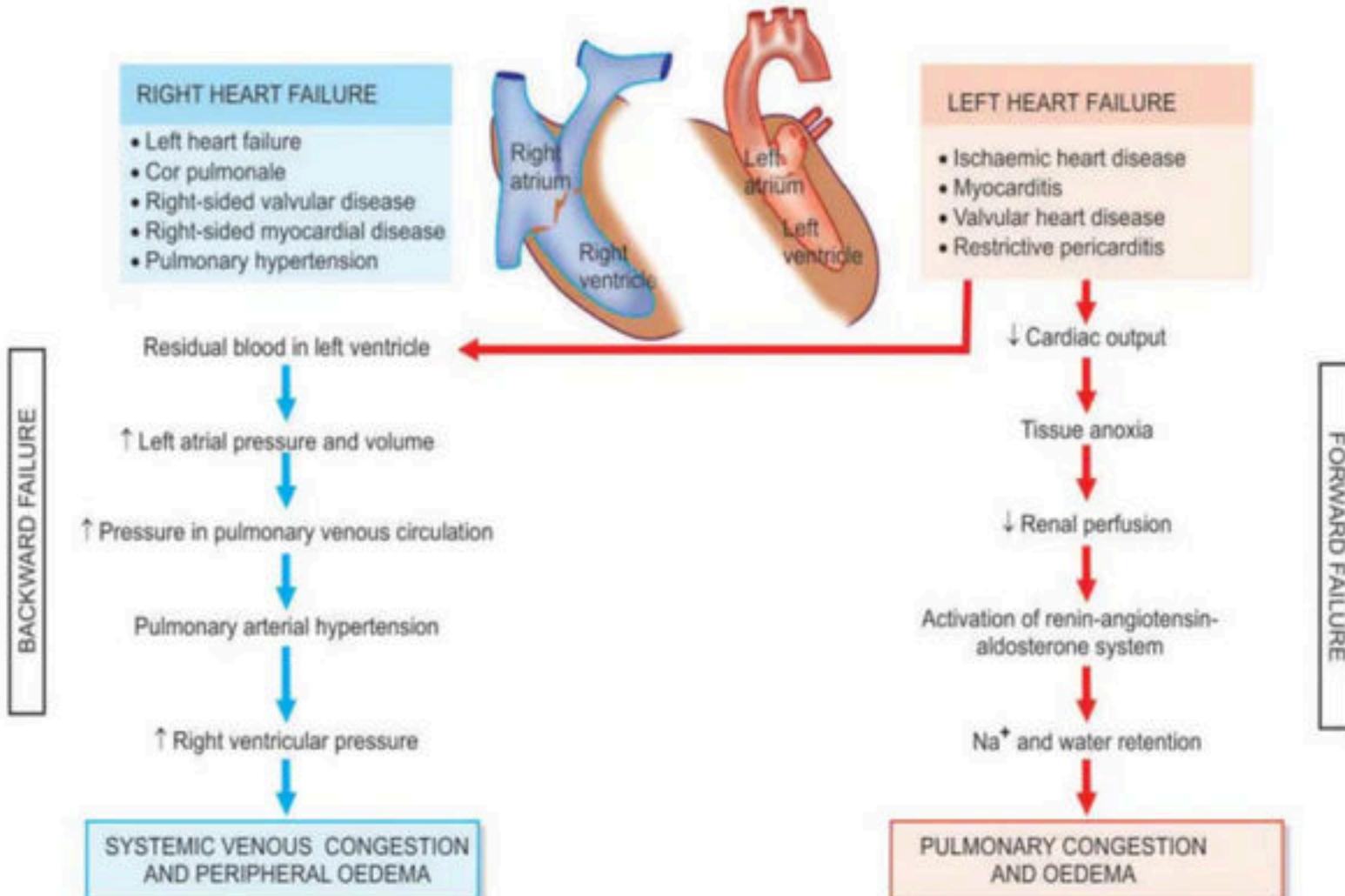
- i) As a consequence of left ventricular failure.
- ii) Cor pulmonale in which right heart failure occurs due to intrinsic lung diseases
- iii) Pulmonary or tricuspid valvular disease.
- iv) Pulmonary hypertension



- **Most common cause of right sided heart failure is left sided heart failure**
i.e. right heart failure occurs as a consequence of left heart failure

Clinical features of right heart failure

- 1) **Peripheral edema of dependent portion**, especially ankle (pedal) and pretibial edema is a hallmark of right sided heart failure. Generalized massive edema may occur → Anasarca.
- 2) Hepatic enlargement with centrilobular necrosis that may progress to cirrhosis → **Cardiac cirrhosis**
- 3) **Congestive splenomegaly**
- 4) **Pleural effusion** (in contrast to left sided heart failure where pulmonary edema occurs).
- 5) **Pericardial effusion**
- 6) **Ascites**



POLLS 2

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Heart failure cells are -

- a) Lipofuscin granules in cardiac cells
- b) Pigmented alveolar macrophages
- c) Pigmented pancreatic acinar cells
- d) Pigment cells seen in liver

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B

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Commonest cause of right ventricular failure is -

- a) Corpulmonale
- b) Pulmonary involvement
- c) Endomyocardial fibrosis
- d) Left ventricular failure

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D

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Heart failure cells are seen in -

- a) Kidney
- b) Heart
- c) Lungs
- d) Brain

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C

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ISCHAEMIC HEART DISEASE/ Coronary artery disease (CAD)

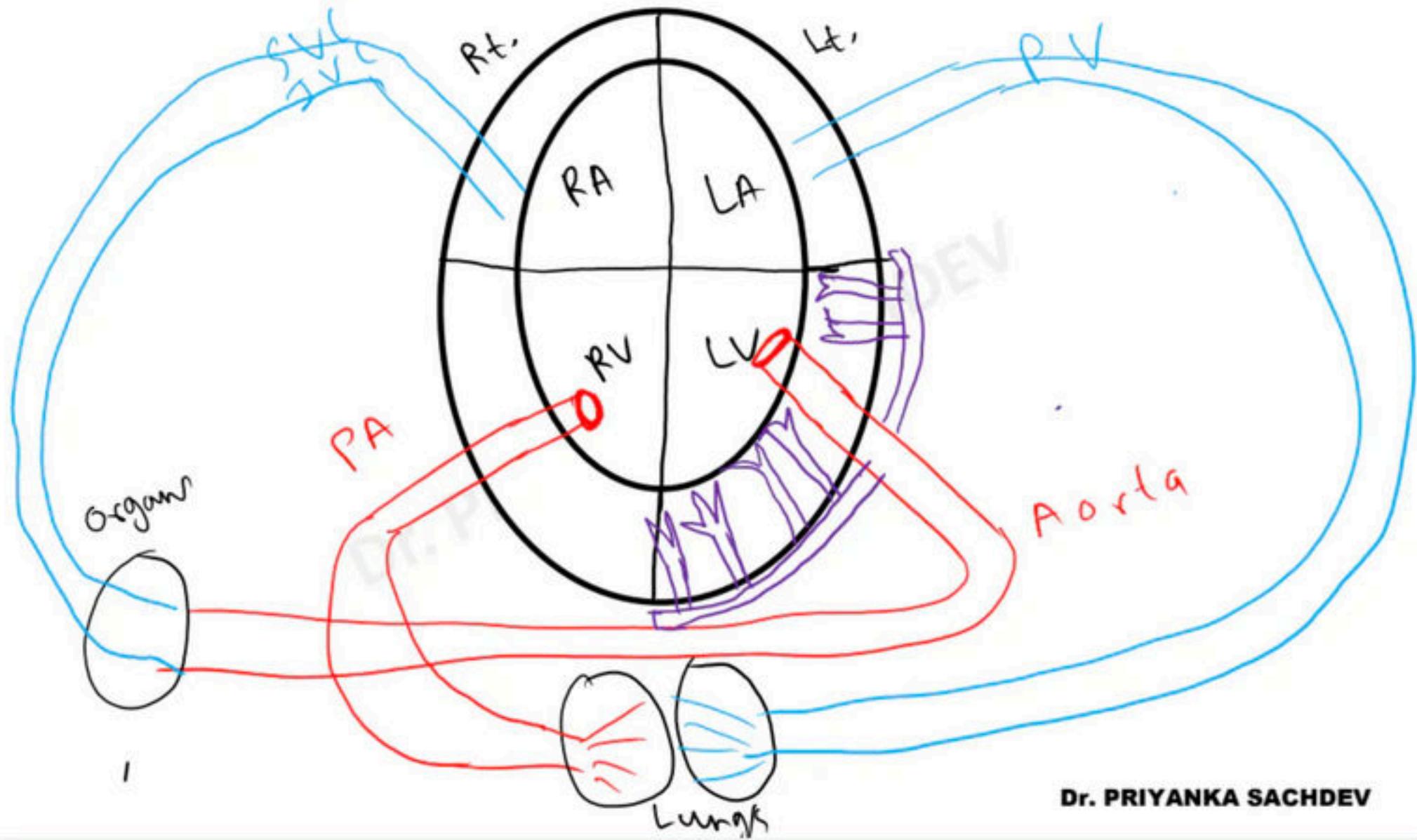
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HEADINGS

- Introduction
- Blood supply of heart
- Etiopathogenesis
- Effects

Introduction

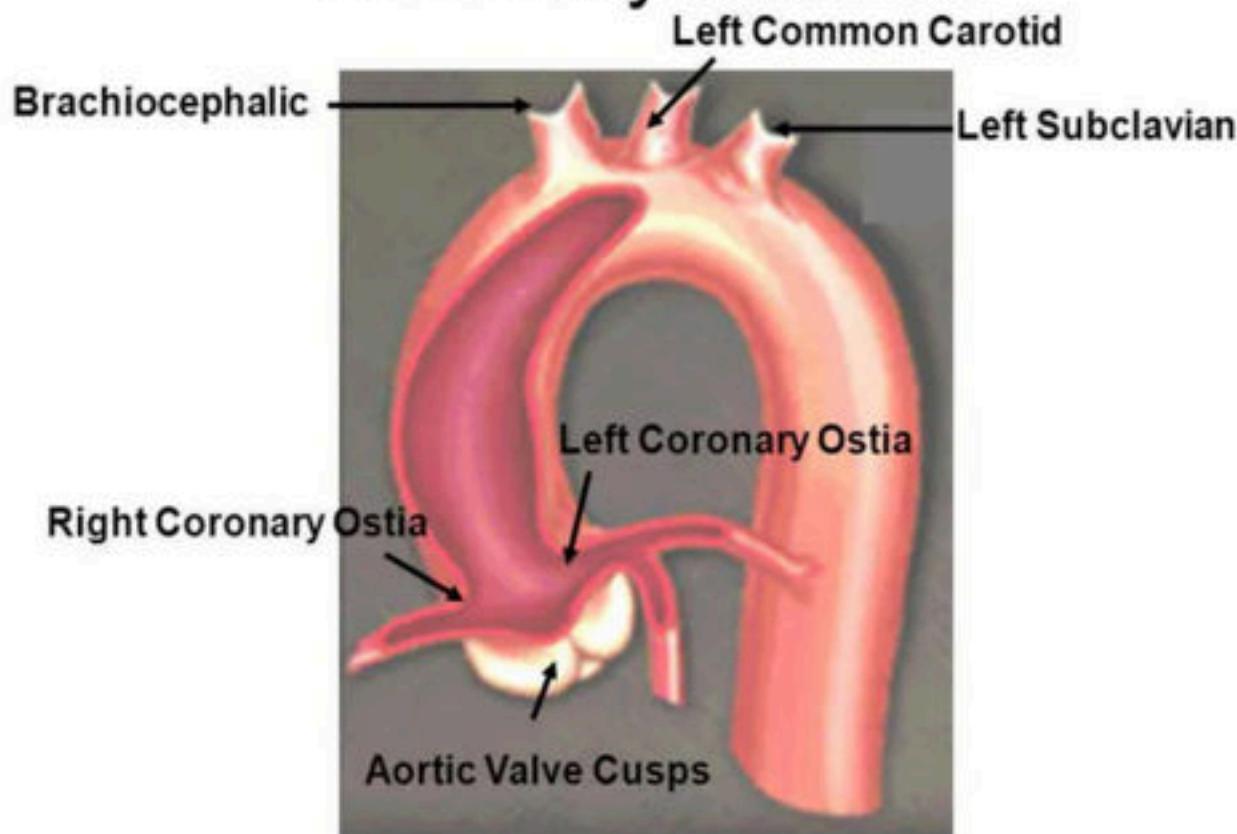
- Ischaemic heart disease (IHD) arise from **Imbalance between the myocardial supply and demand for oxygenated blood.**

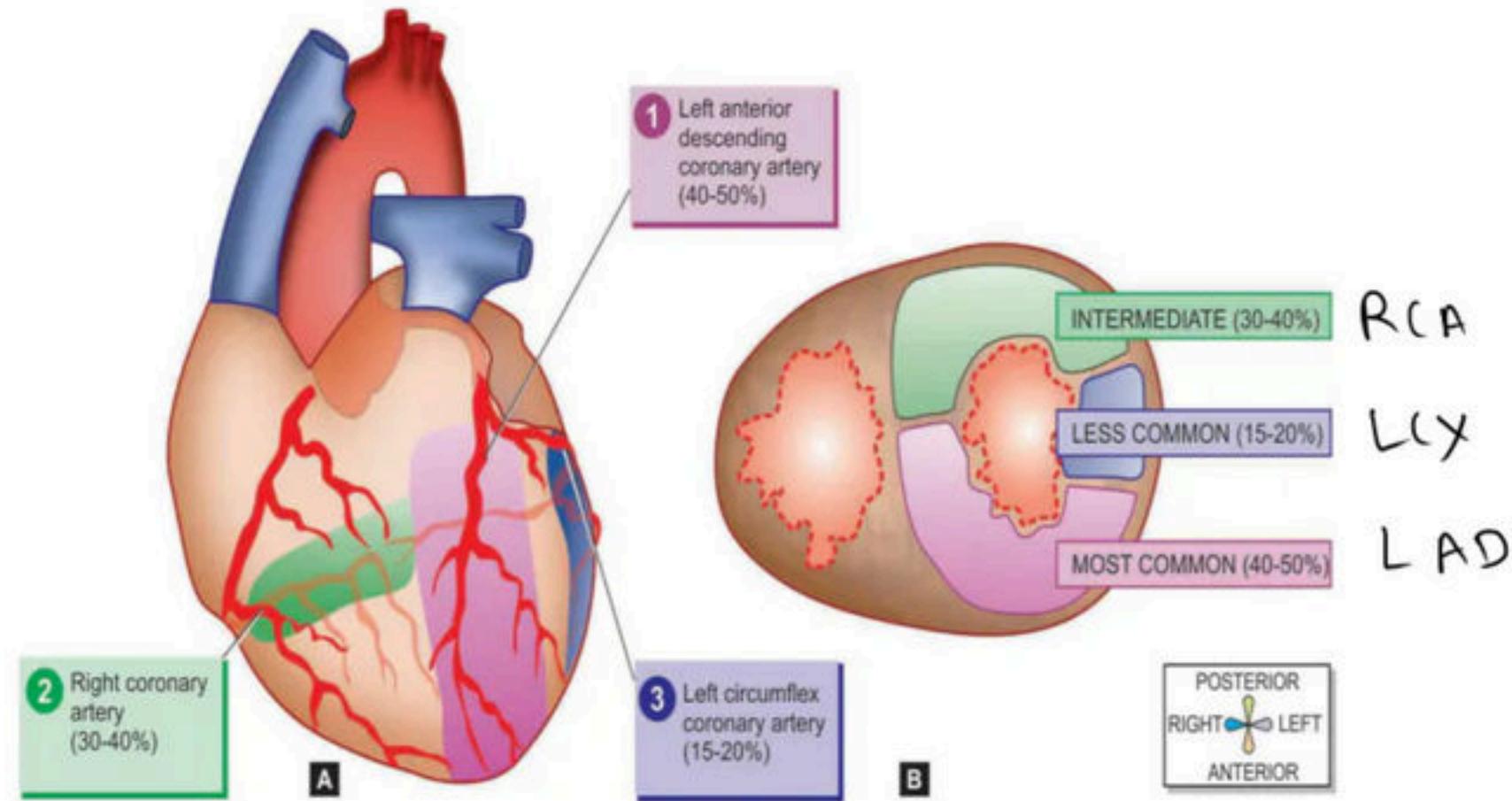


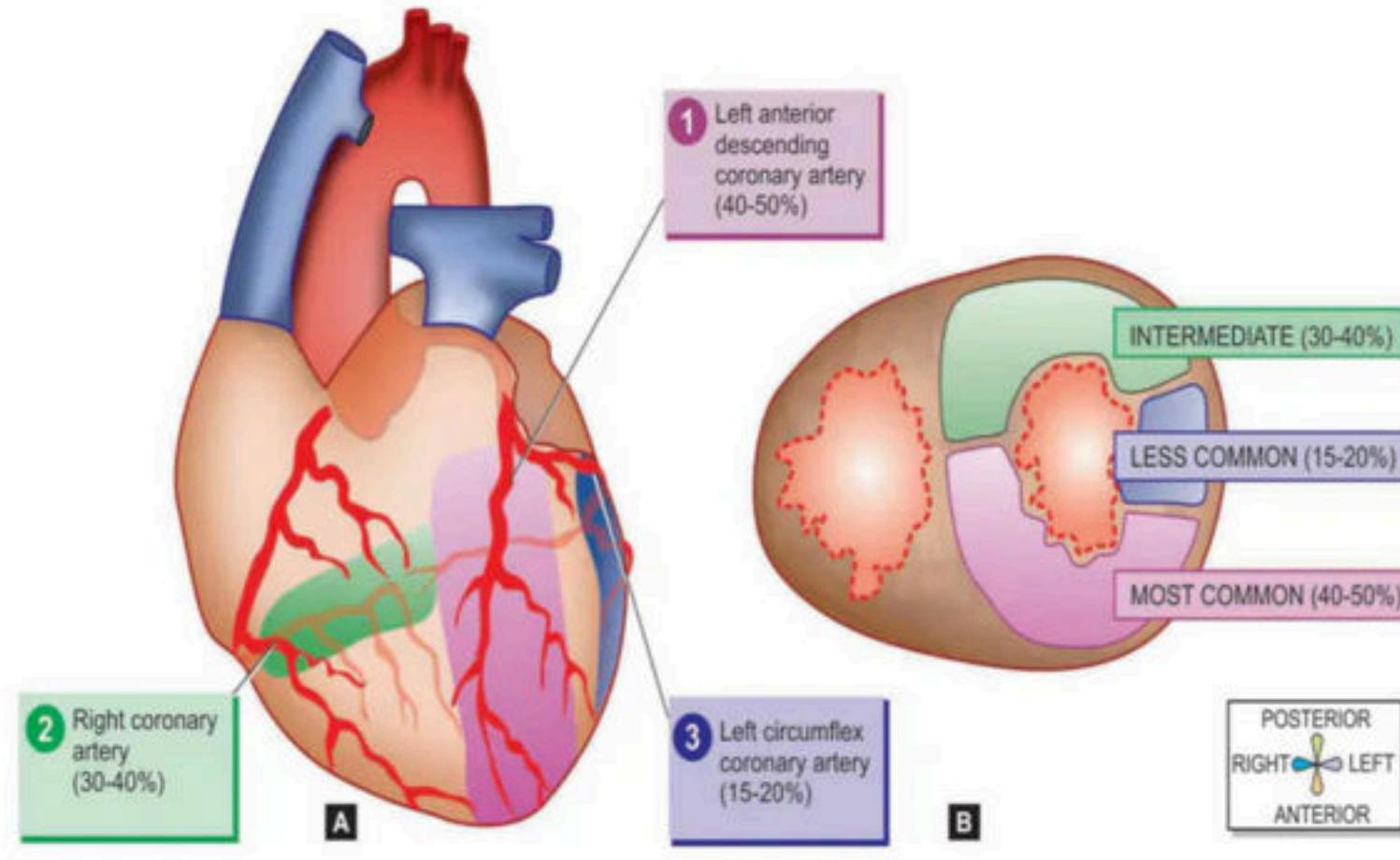
Blood supply of heart

- 1) Left anterior descending (LAD) artery**
- 2) Left circumflex (LCX) artery**
- 3) Right coronary artery**

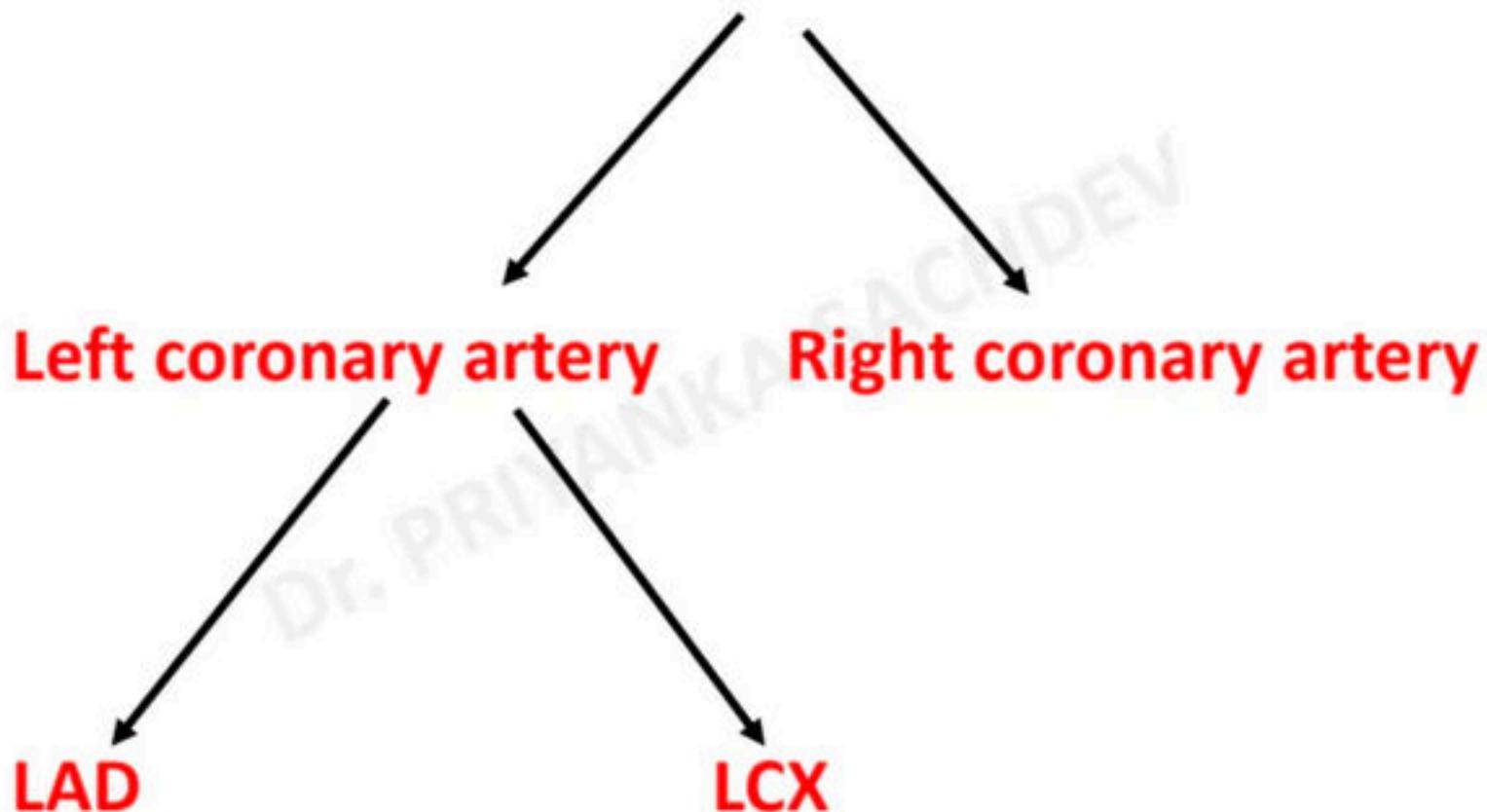
Coronary Arteries

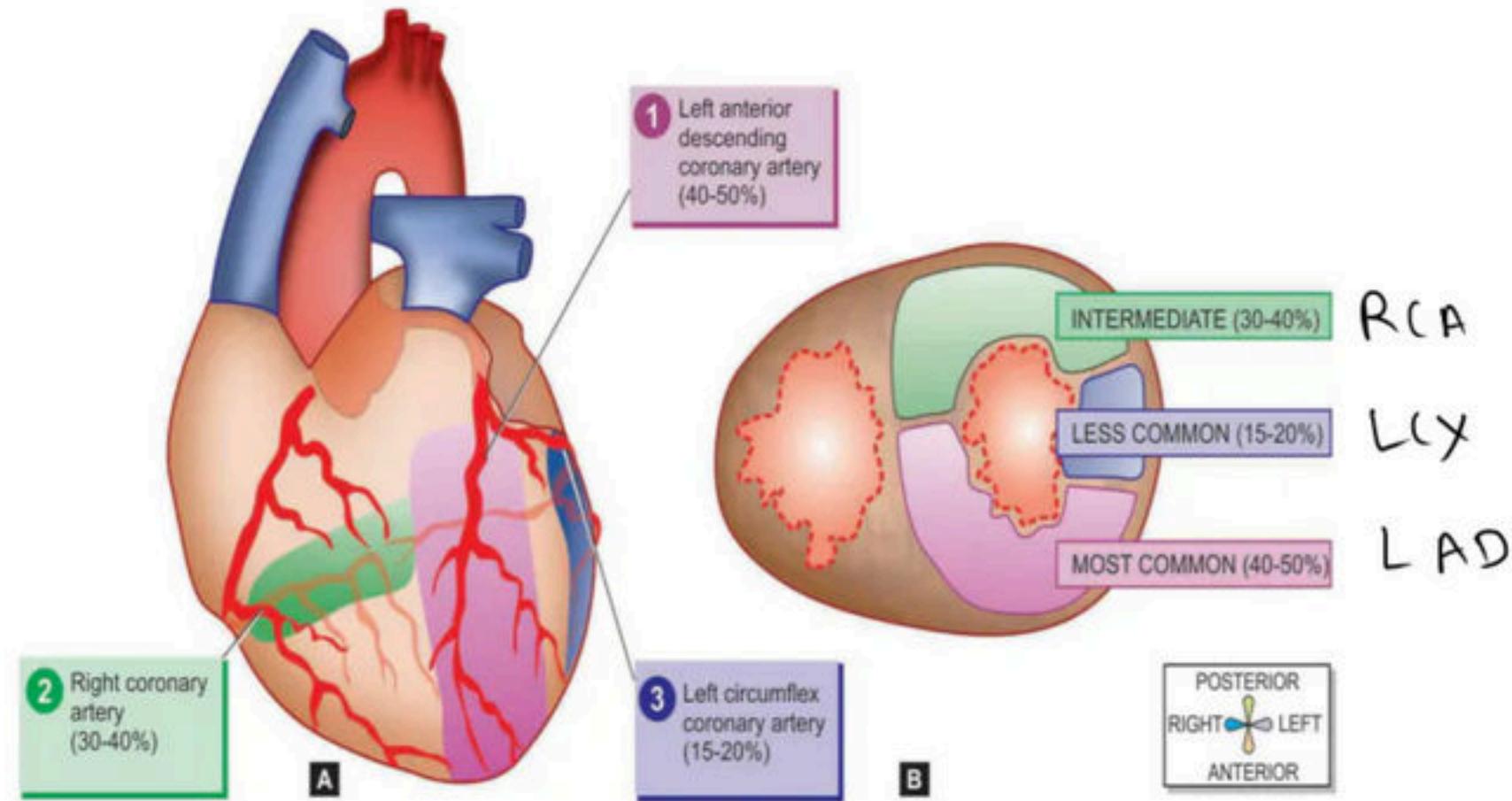






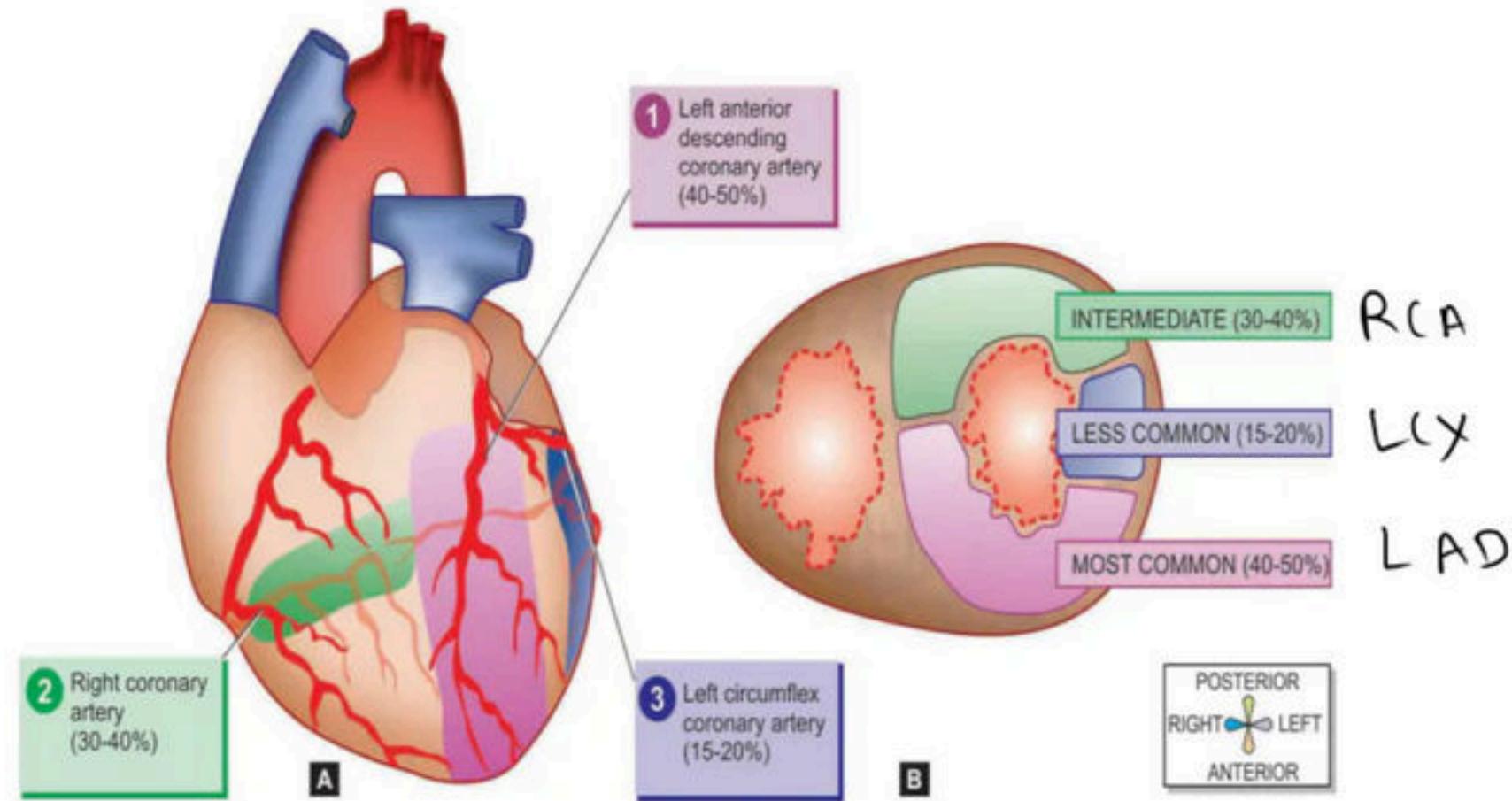
Coronary artery





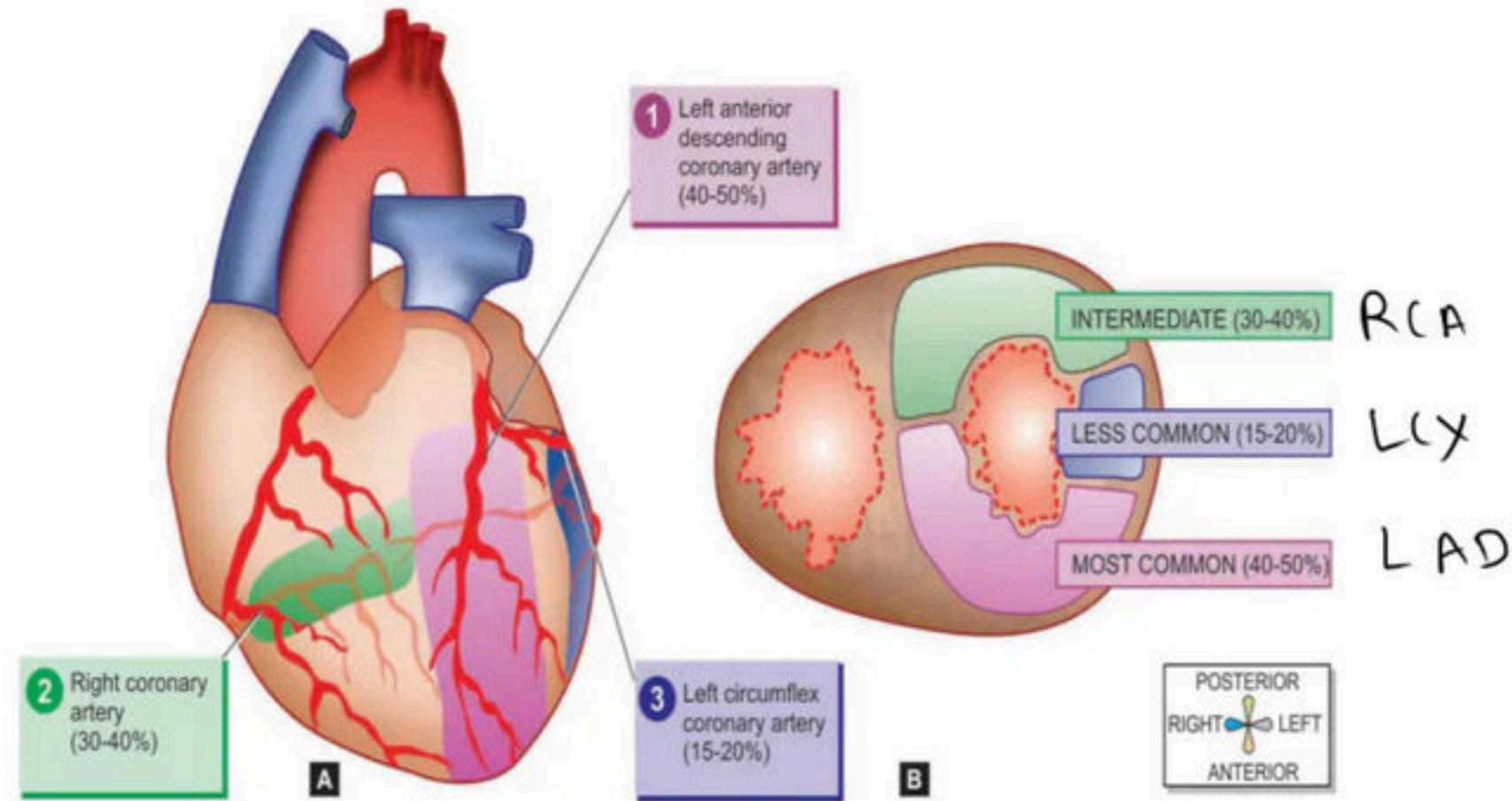
1) Left anterior descending (LAD) artery

- It is a branch of left coronary artery
- supplies →
 - 1. Apex**
 - 2. Anterior wall of left ventricle**
 - 3. Anterior two-third of ventricular septum.**
- LAD artery is the **most commonly** involved artery in atherosclerosis thus these sites are the most common site of myocardial infarction



2) Left circumflex (LCX) artery

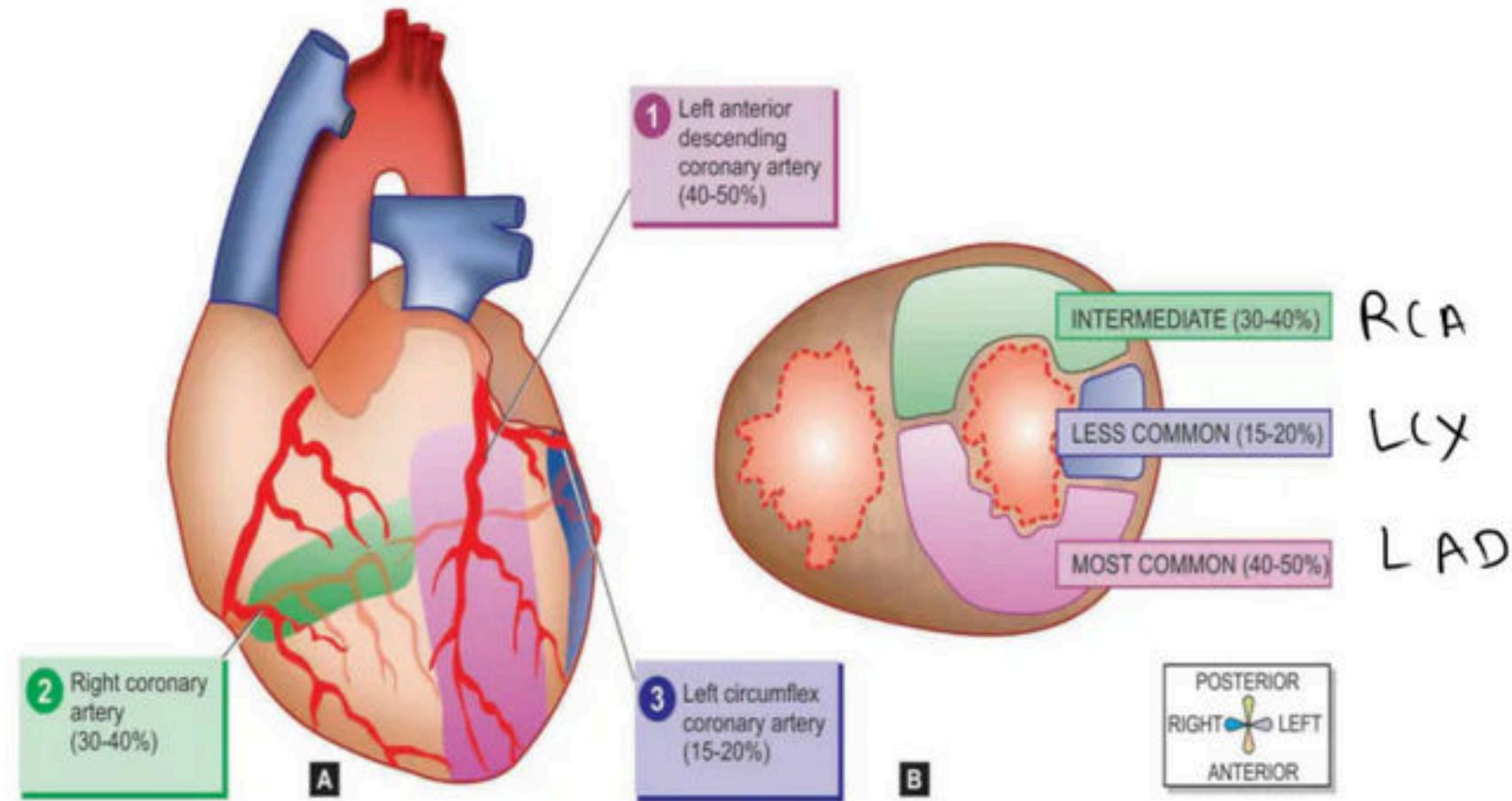
- It is a branch of left coronary artery
- supplies →
 - 1. Lateral wall of left ventricle**
- It is the **least commonly** involved site for MI



3) Right coronary artery (RCA)

It supplies →

- 1. Posterior wall of left ventricle**
 - 2. Right ventricular free wall**
 - 3. Posterior one-third of ventricular septum**
-
- It is the **second most commonly** involved vessel in atherosclerosis and MI.

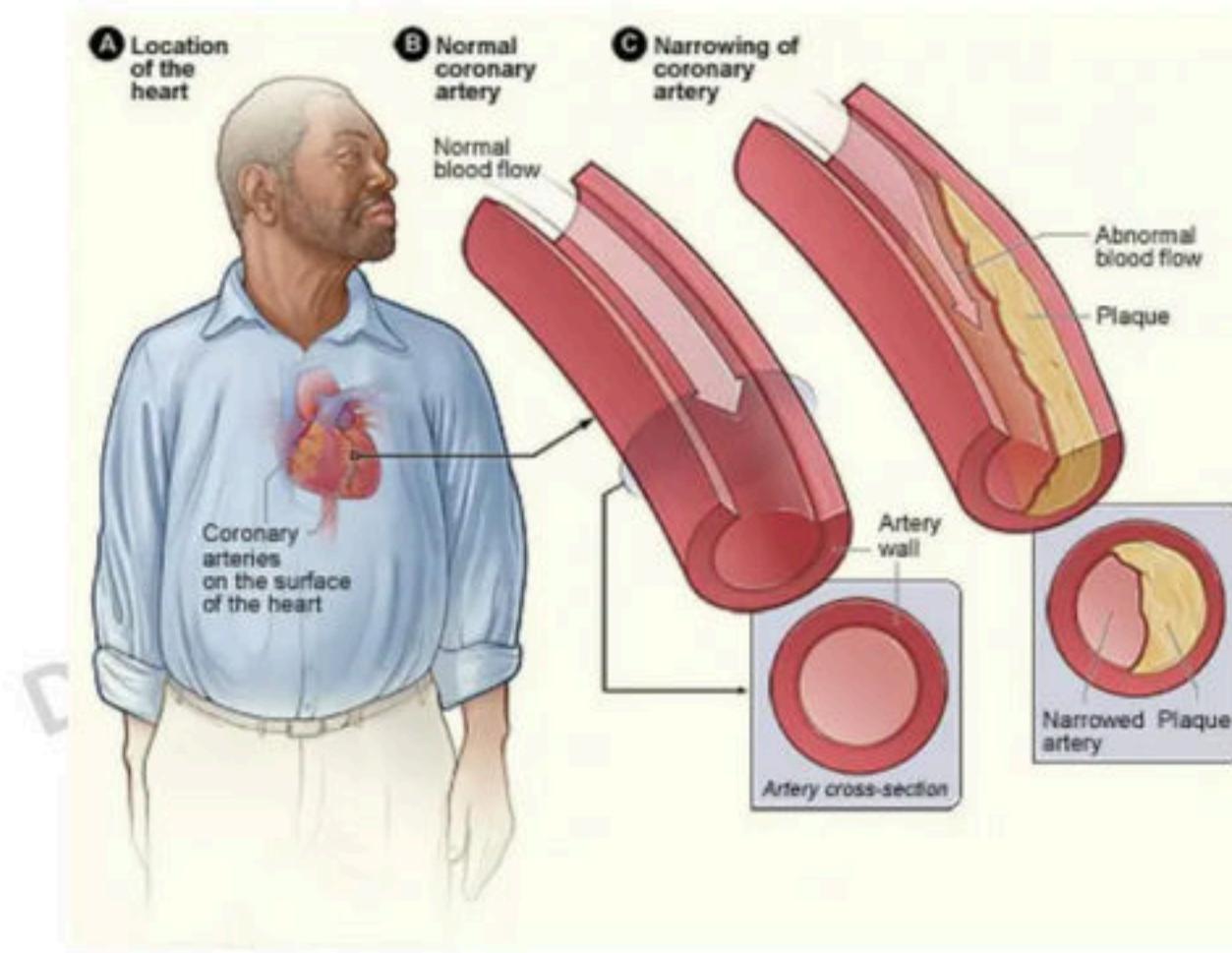


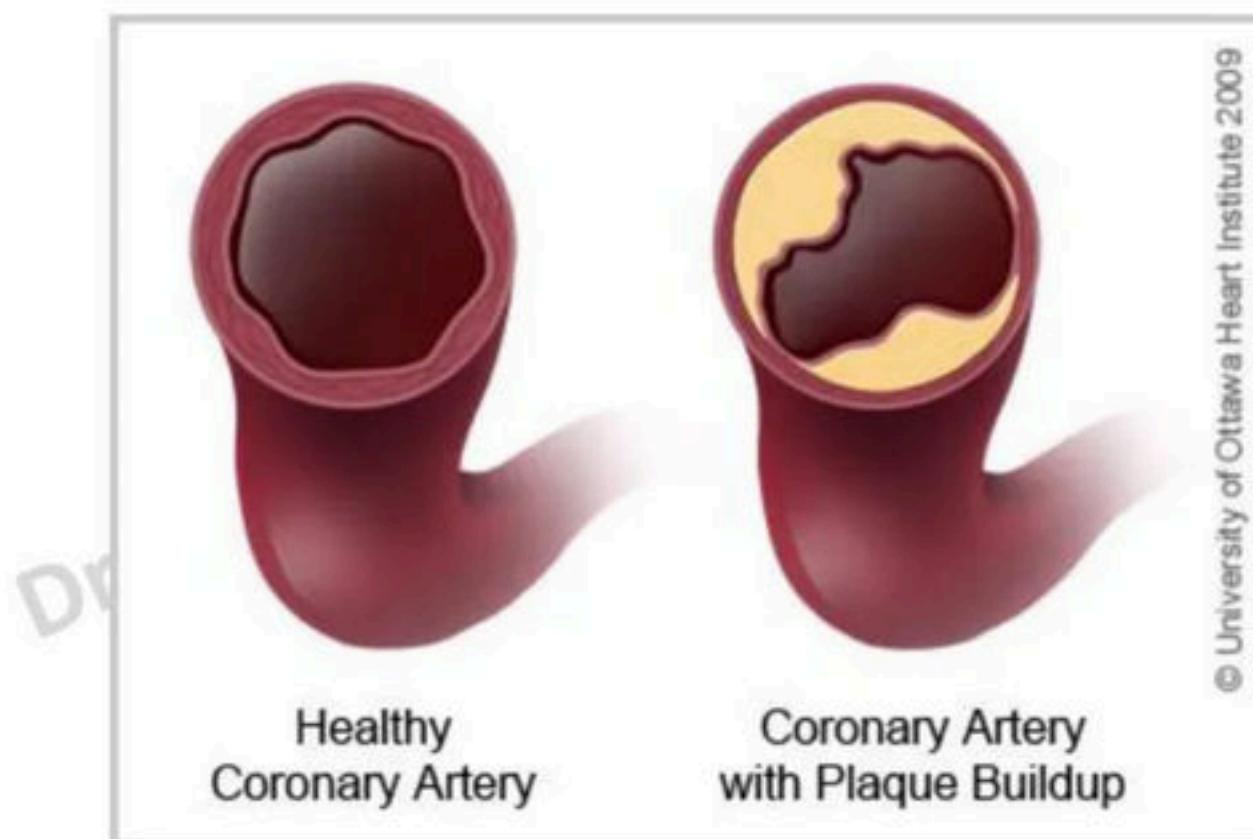
Distribution → (LAD) > (RCA) > (CXA)

- One-third of cases have **single-vessel disease** ie. LAD involvement;
- Another one-third have **two-vessel disease**
- The remainder has **three major vessel disease**

ETIOPATHOGENESIS

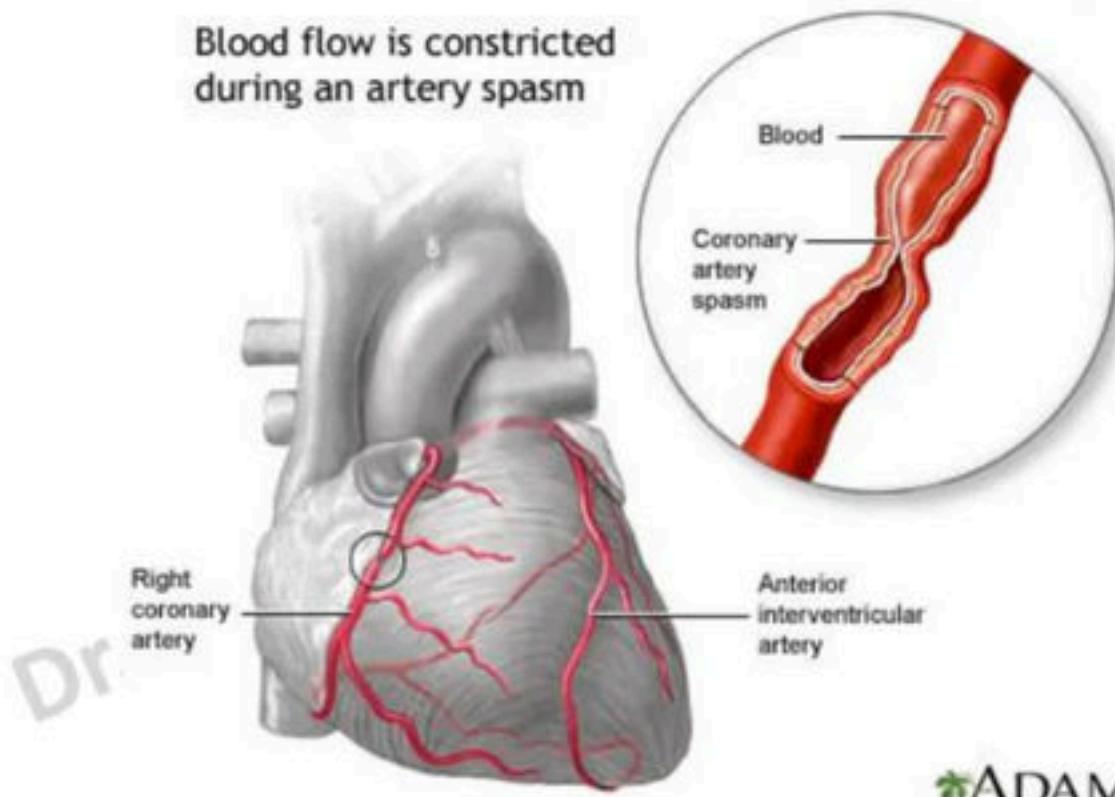
- i) Coronary atherosclerosis
- ii) Superadded changes in coronary atherosclerosis
- iii) Non-atherosclerotic causes





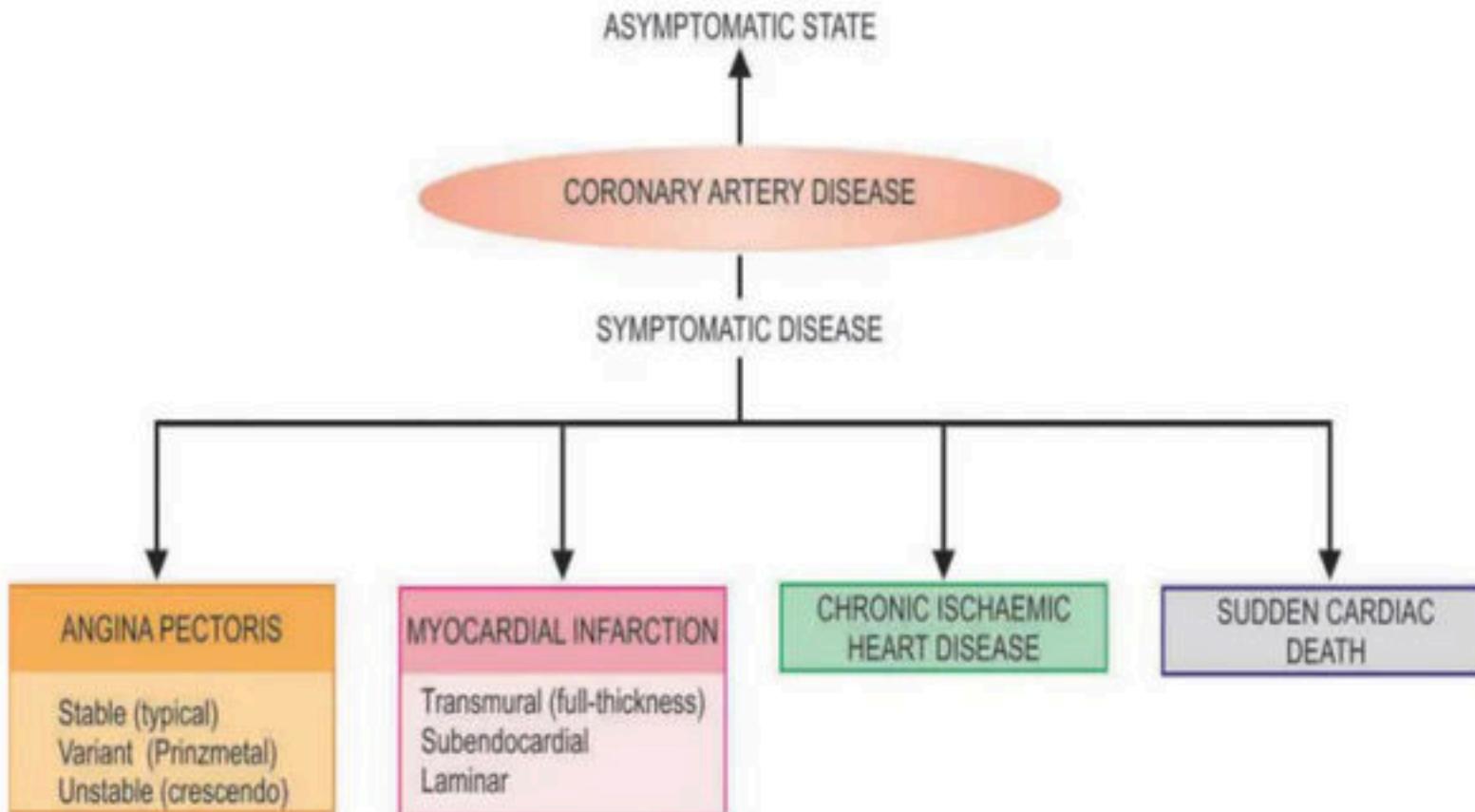
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Blood flow is constricted during an artery spasm



ADAM.

EFFECTS OF IHD



POLLS 3

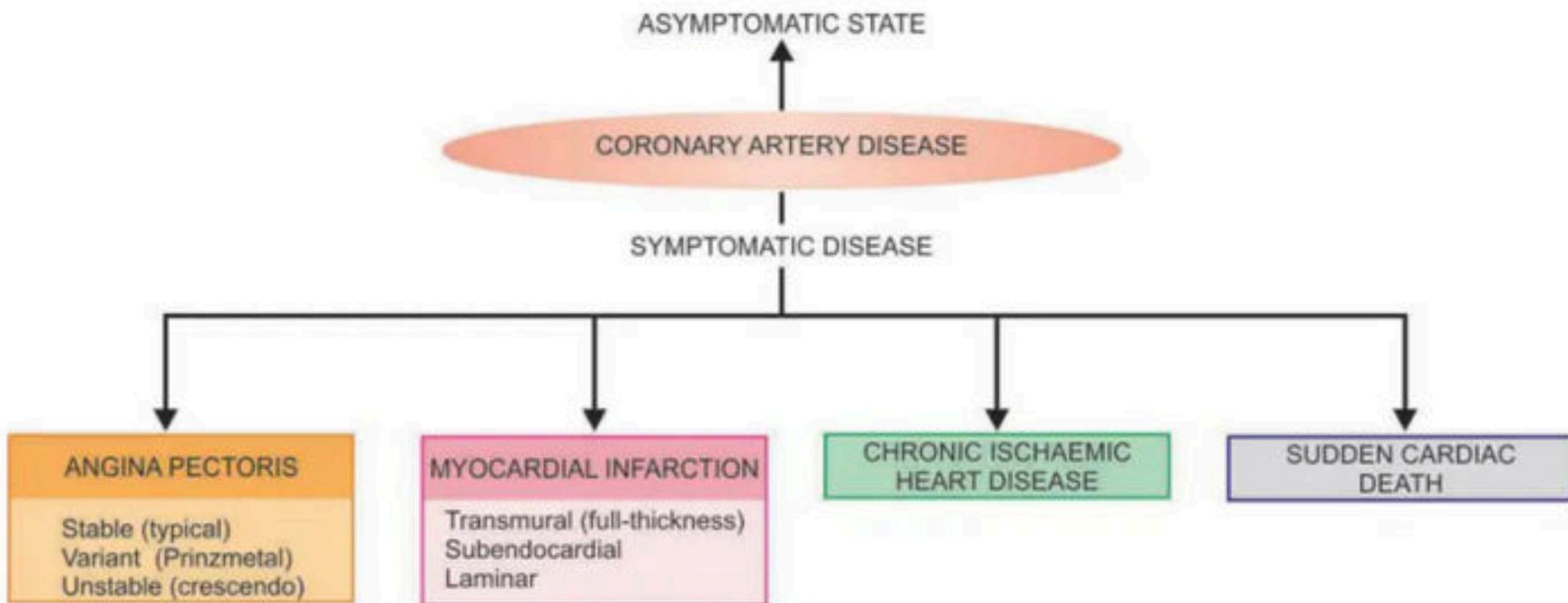
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Coronary heart disease may manifest as all, except -

- a) Angina on effort
- b) Cardiomyopathies
- c) Myocardial infarction
- d) Sudden death

Dr. PRIYANKA

B



Most common site of artery of atherosclerosis -

- a) LAD
- b) RCA
- c) LCX
- d) Diagonal branch of LAD

Dr. PRIYANKA
SACHDEV

A

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Most common site of myocardial infarction is -

- a) Ant. wall of left ventricle
- b) Post. wall of right ventricle
- c) Post. wall of left ventricle
- d) Inf. wall of left ventricle

Dr. PRIYANKA SACHDEV

A

Dr. PRIYANKA SACHDEV

ANGINA PECTORIS

Dr. PRIYANKA SACHDEV

HEADINGS

- **Definition**
- **Types**

ANGINA PECTORIS

- Angina pectoris is a symptom complex of IHD characterized by **paroxysmal and recurrent attacks of substernal or precordial chest discomfort caused by transient myocardial ischemia**
- The levels of cardiac markers remains unchanged

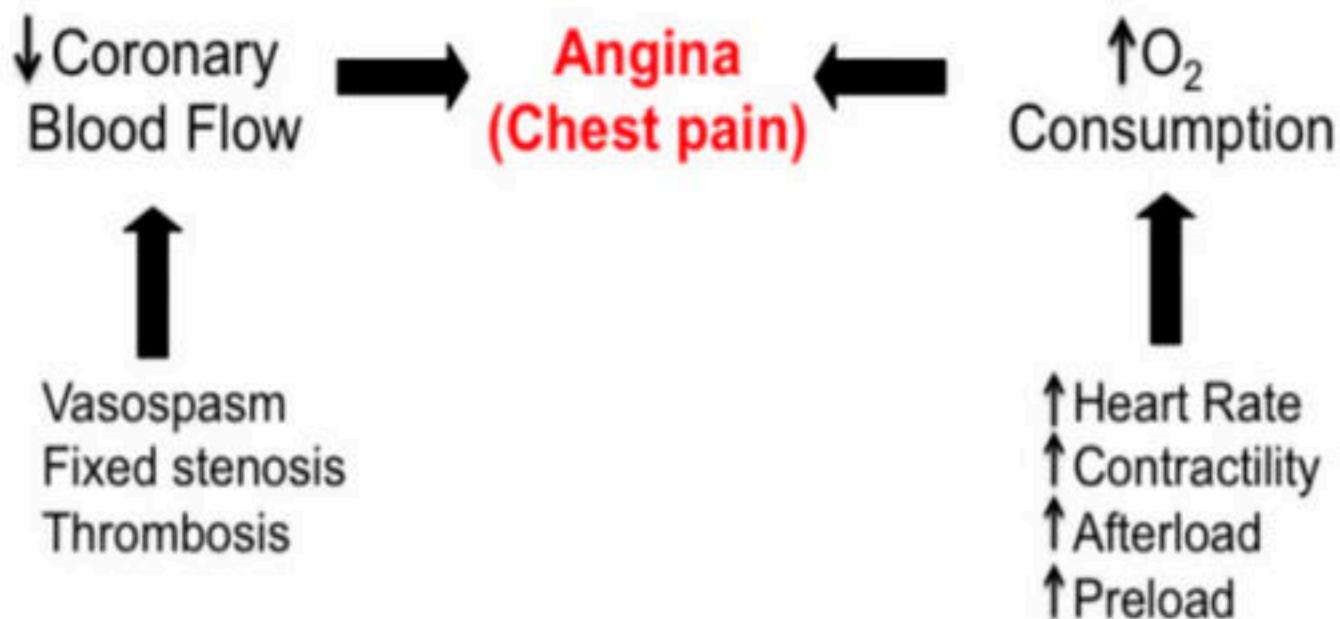


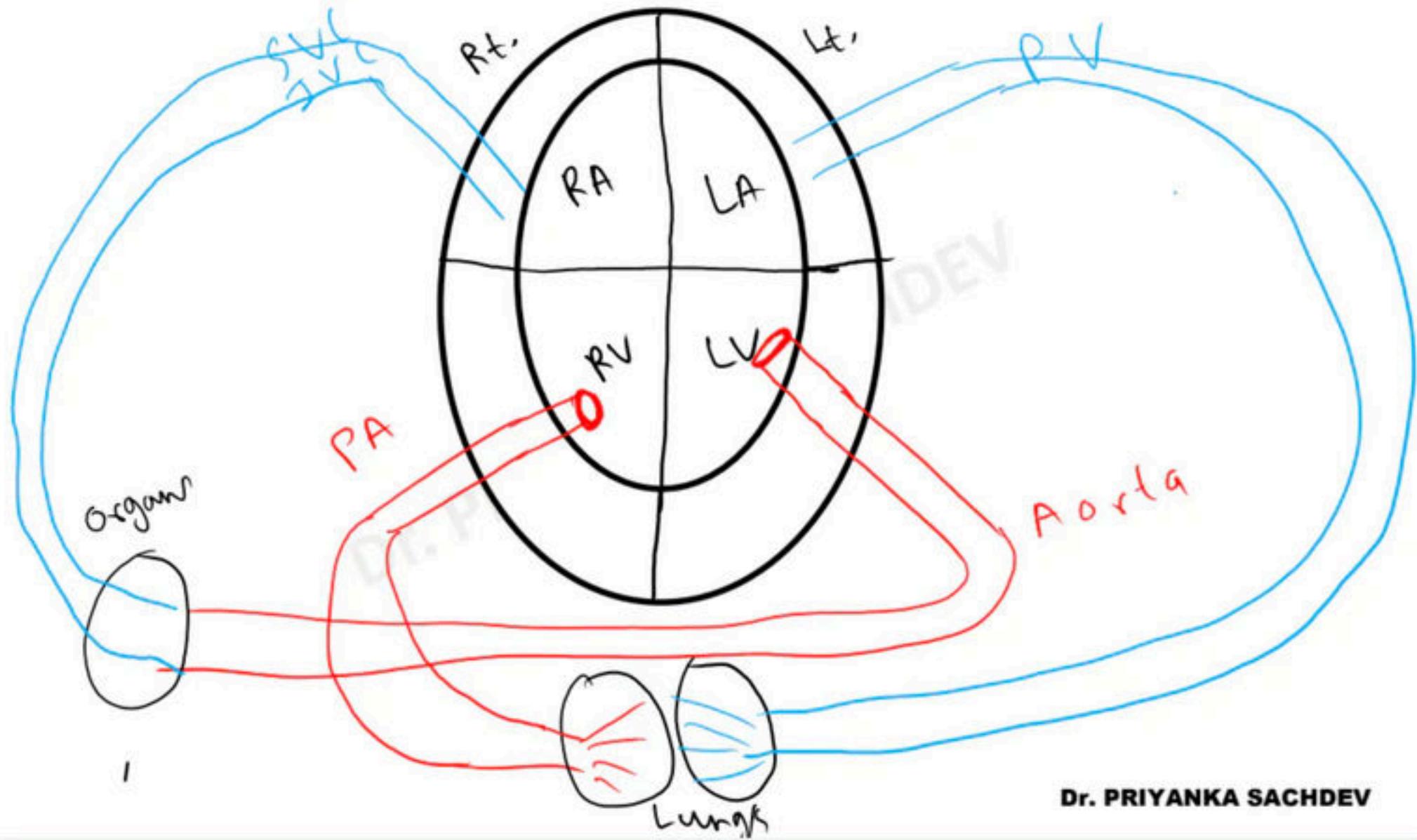
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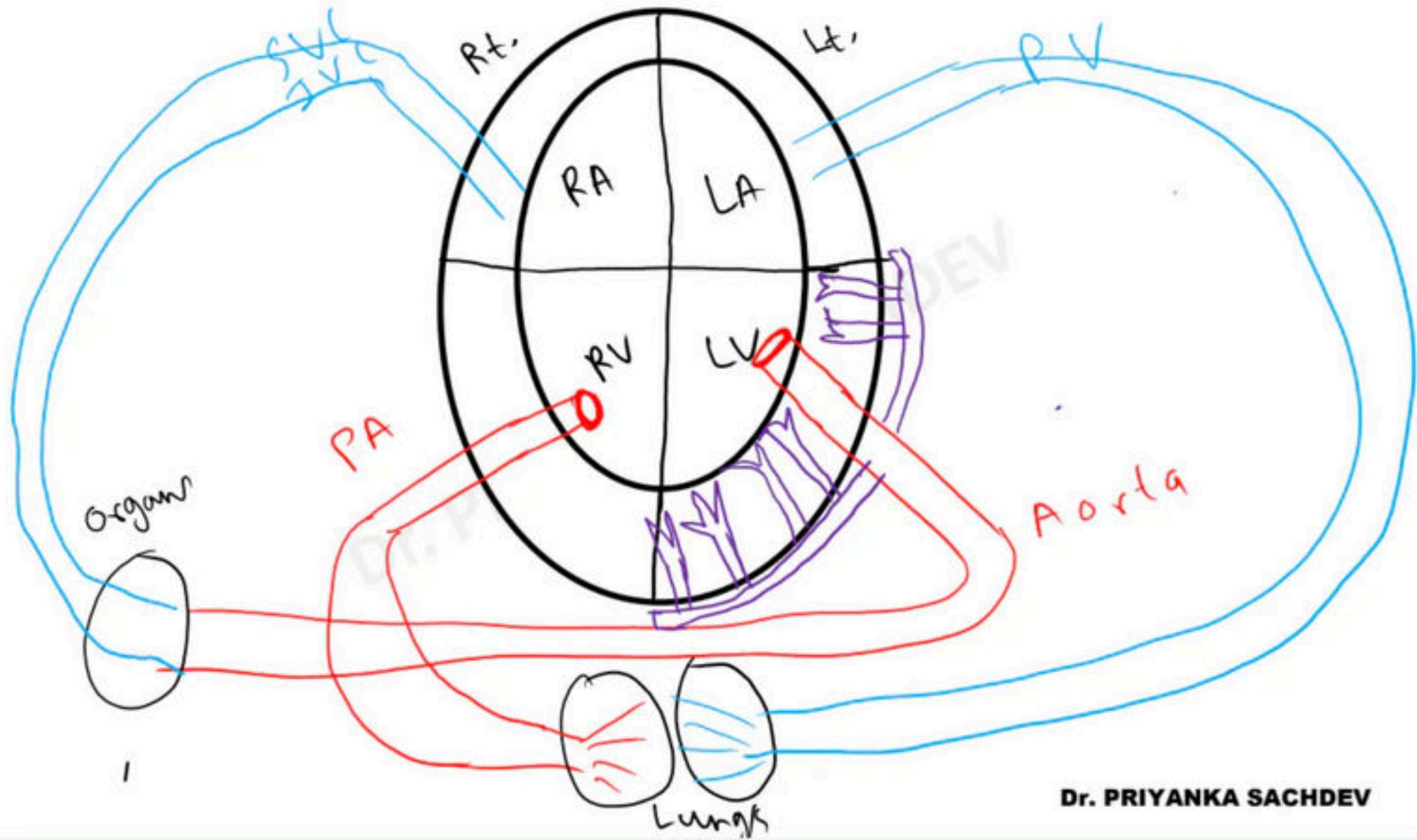


Angina Pectoris
A Chest Pain

Supply vs. Demand







TYPES

There are three patterns of angina →

- i) Stable or Classical angina
- ii) Unstable or crescendo angina
- iii) Prinzmetal's variant angina

Angina

Printzmetal's
Variant Angina
(vasospasm)

*Supply
Ischemia*

Chronic
Stable Angina
(fixed stenosis)

*Demand
Ischemia*

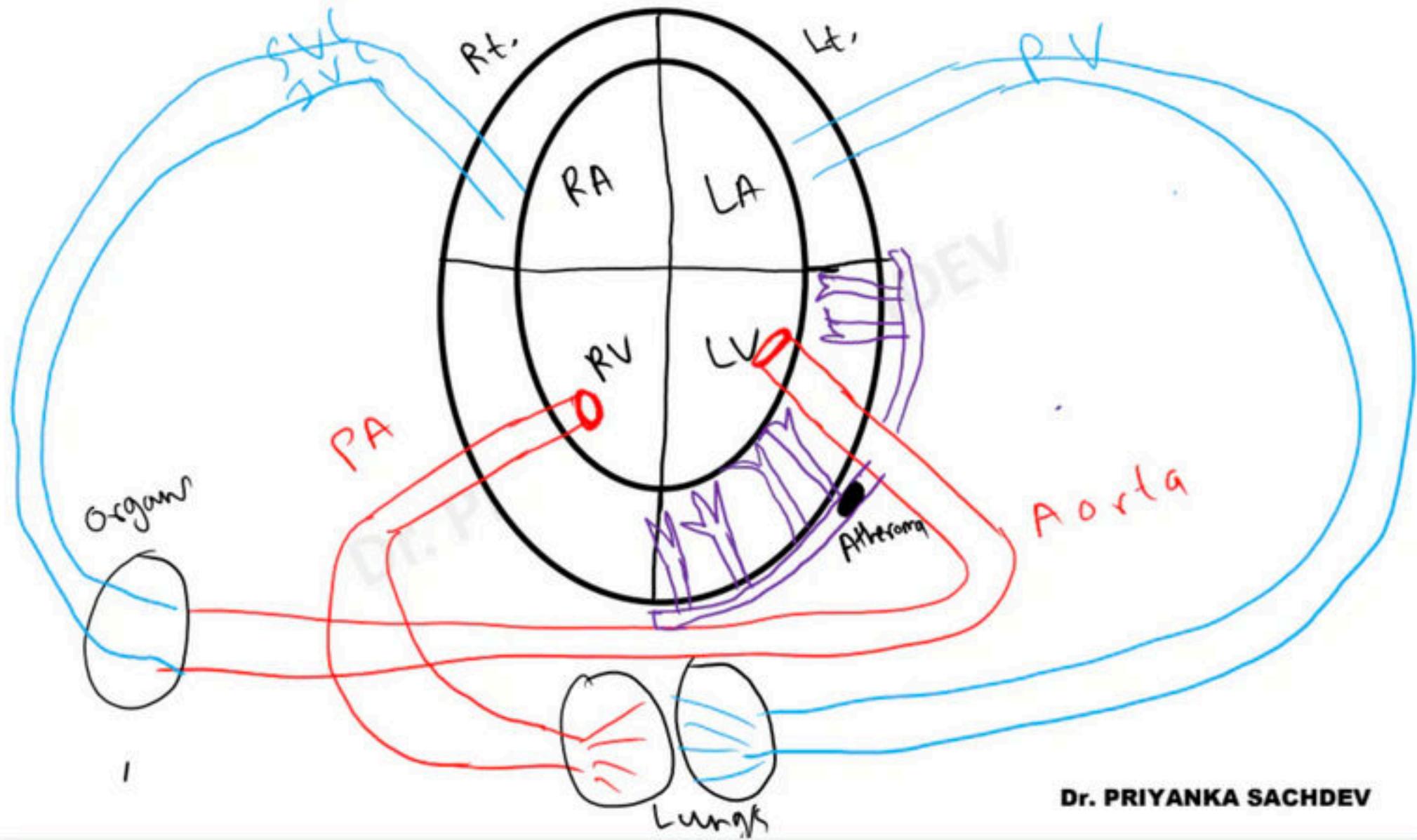
Unstable
Angina
(thrombus)

*Supply
Ischemia*

Dr. V.

Classical (stable) angina

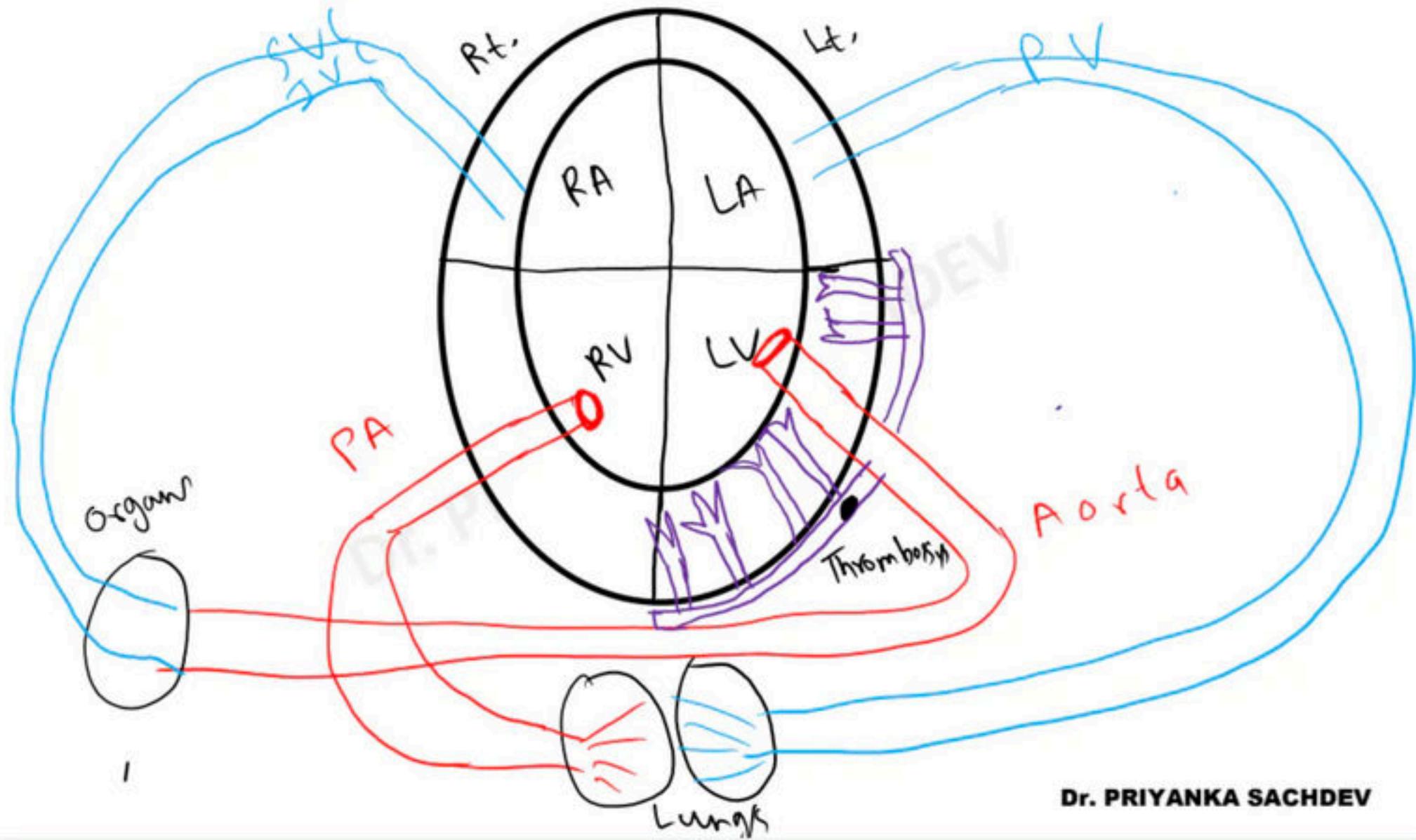
- It is the **most common form of angina**
- It is caused by the **reduction of coronary perfusion to a critical level due to coronary atherosclerosis without plaque rupture.**
- Chest discomfort occurs on **exertion**



- Patient complains **chest discomfort**, usually described as **heaviness, pressure, squeezing, chocking or smothering.**
- It is not described as frank pain
- Chest discomfort occurs on **exertion**
- relieved by **rest.**
- When patient is asked to localize the sensation, he/she will typically place their hand over the sternum with clenched fist → **Levine's sign**
- Chest discomfort typically lasts **2-5 minutes.**
- Pain can radiate to shoulder, arm, back, interscapular region, neck, jaw, teeth, epigastrium.
- Stable angina is usually **crescendo - decrescendo** in nature.

Unstable angina

- Unstable angina is due to **disruption of atherosclerotic plaque with thromosis.**

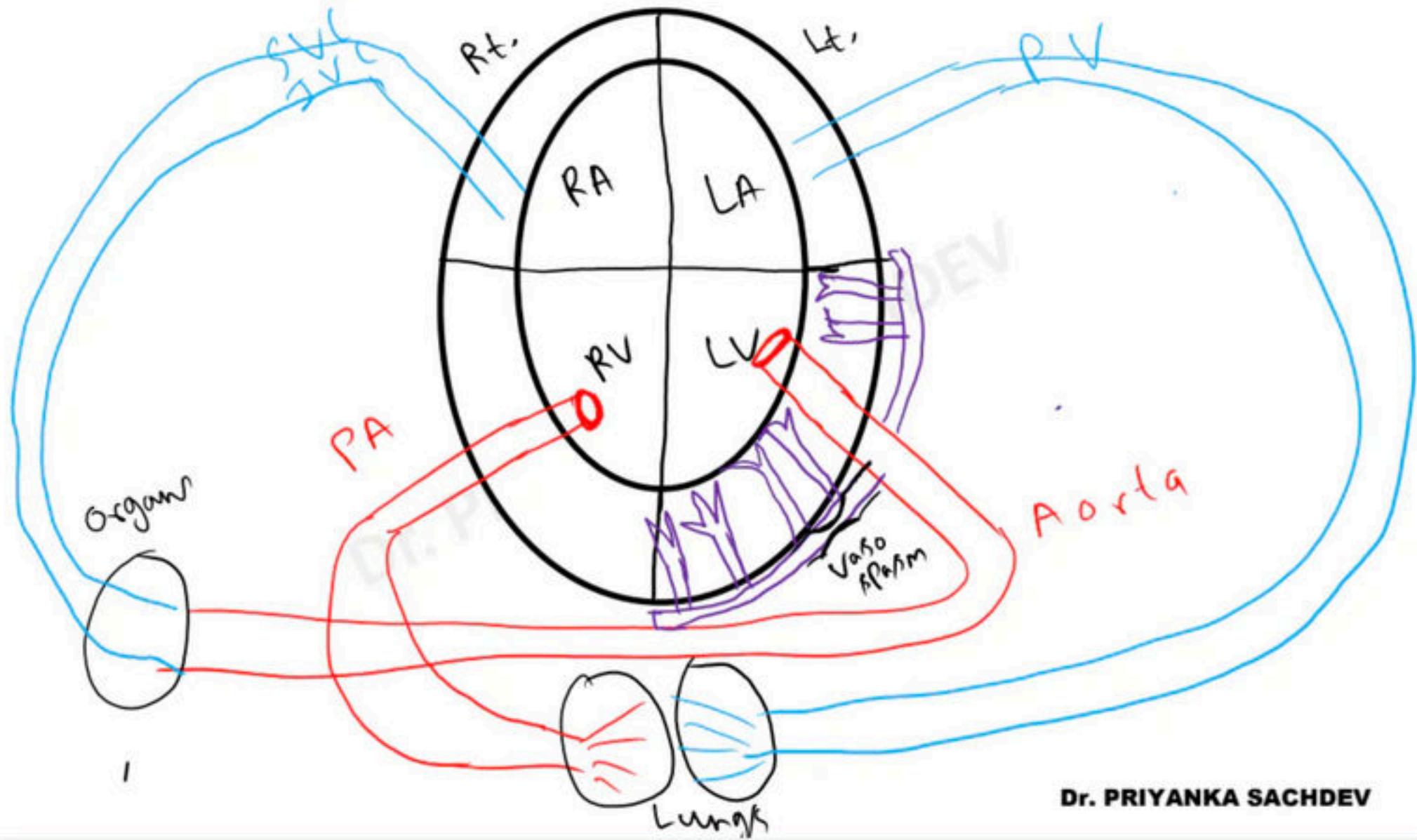


Unstable angina is defined as **chest discomfort** that has at least one of the three features →

- It occurs at **rest** and lasting **> 10 min.**
- It is **severe and of new onset** (i.e. within prior 4-6 weeks).
- It occurs with a **crescendo pattern**
- The chest discomfort of US is described as **pain** (in contrast to stable angina).

Prinzmetal variant angina

- Due to **focal spasm in right coronary artery**
- Pain occurs **at rest.**



Angina

Printzmetal's
Variant Angina
(vasospasm)

*Supply
Ischemia*

Chronic
Stable Angina
(fixed stenosis)

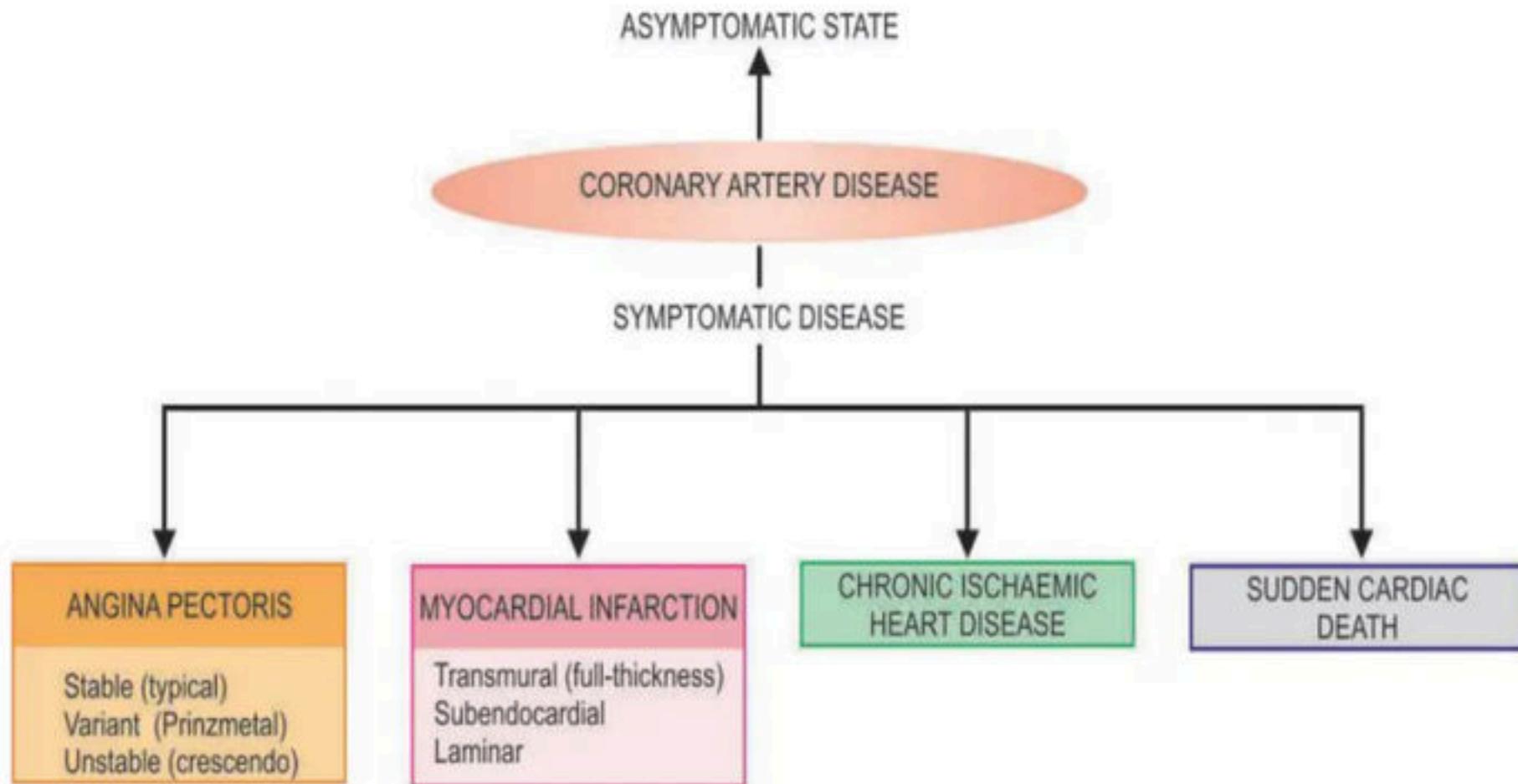
*Demand
Ischemia*

Unstable
Angina
(thrombus)

*Supply
Ischemia*

Dr. V.

EFFECTS OF IHD



MYOCARDIAL INFARCTION

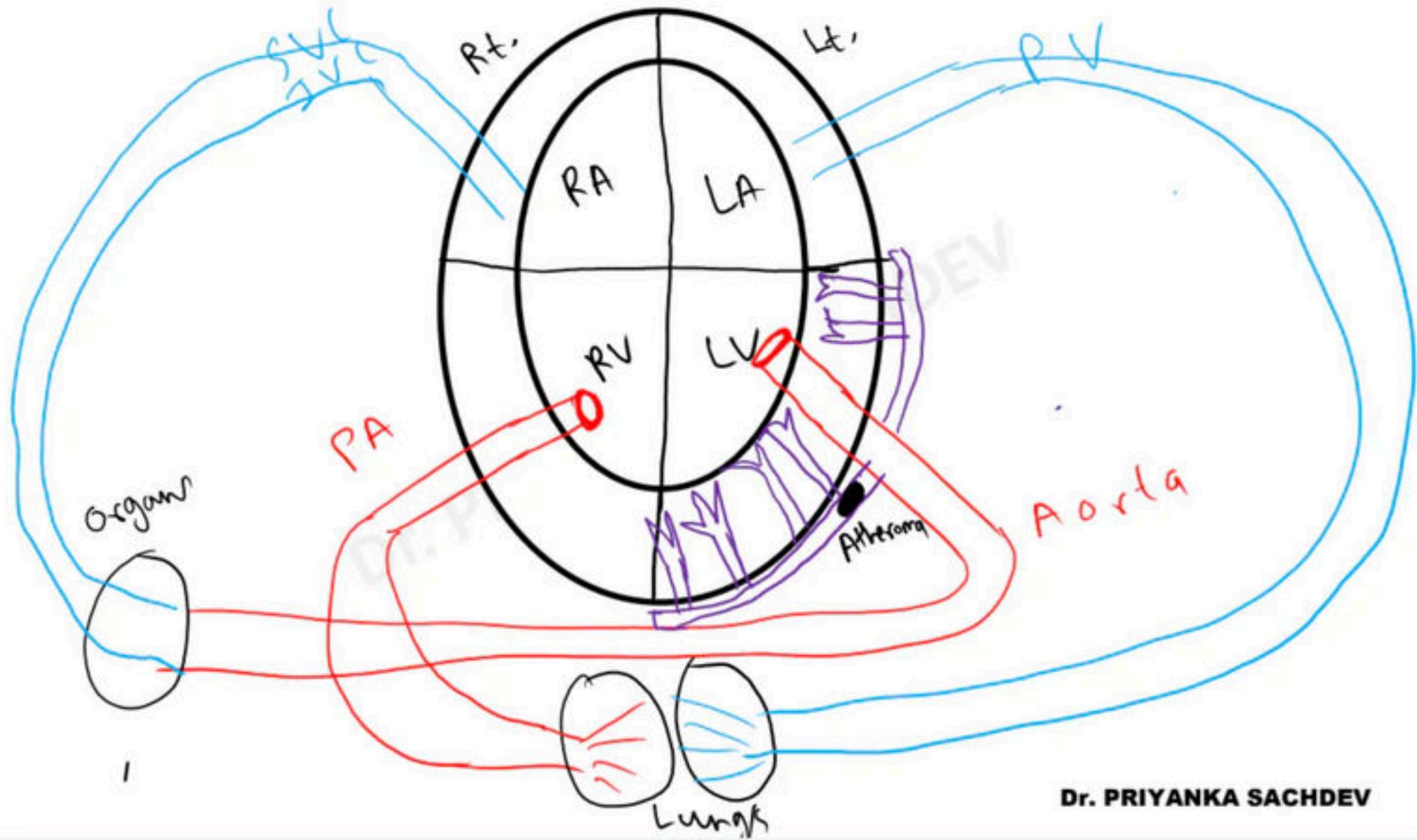
Dr. PRIYANKA SACHDEV

HEADINGS

- **Definition**
- **Etiopathogenesis**
- **Types of infacts**
- **Location of infacts**
- **Morphologic features (Gross and Microscopy)**
- **Clinical features**
- **Diagnosis**
- **Complications**
- **Salvage in early infacts**

MYOCARDIAL INFARCTION

- Infaction (ischemic necrosis) of myocardium of heart due to decreased blood supply



ETIOPATHOGENESIS

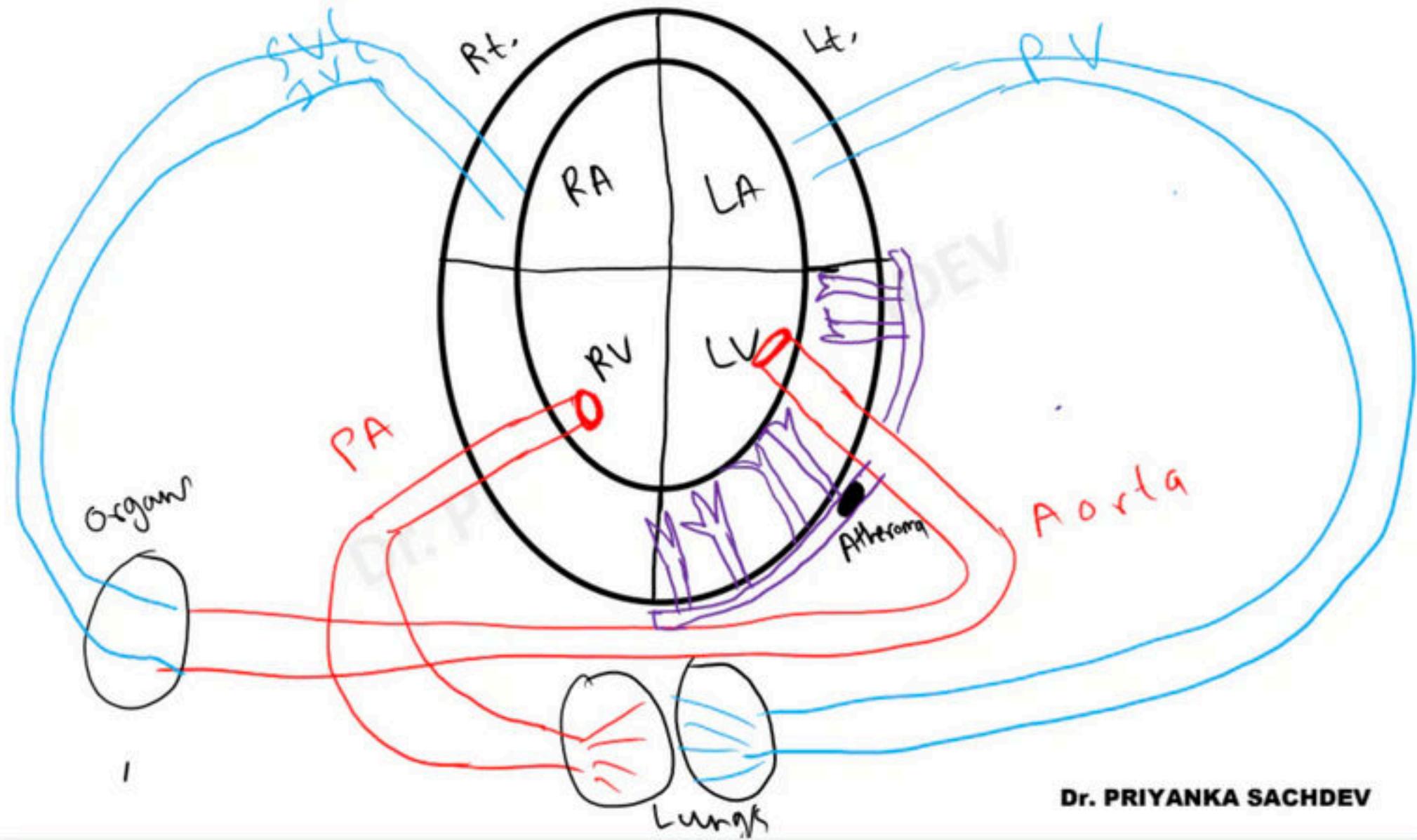
- i) Coronary atherosclerosis
- ii) Superadded changes in coronary atherosclerosis
- iii) Non-atherosclerotic causes

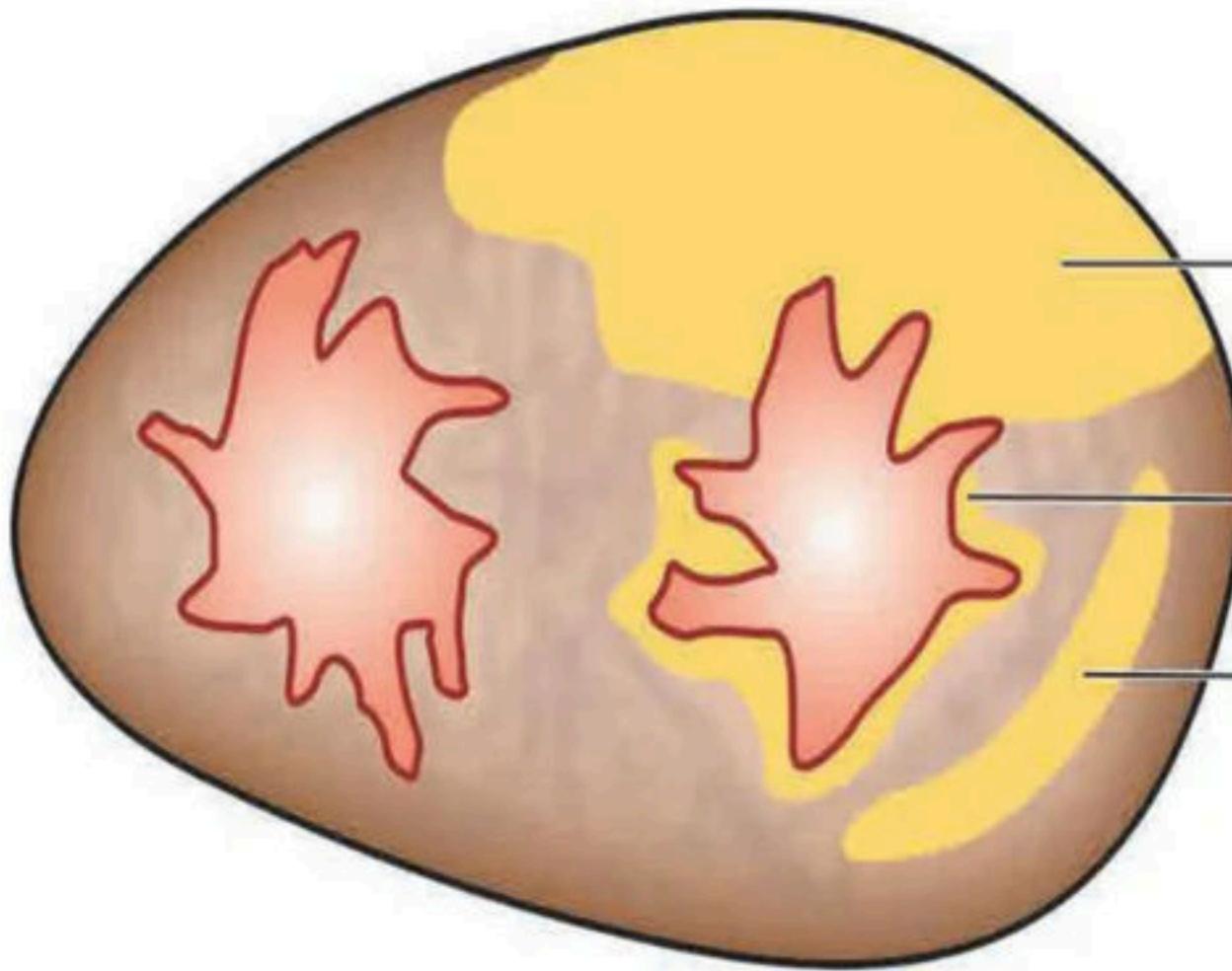
TYPES OF INFARCTS

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According to the degree of thickness of the ventricular wall involved

- i) **Full-thickness or transmural**, when they involve the entire thickness of the ventricular wall.
- ii) **Subendocardial** when they occupy the inner subendocardial half of the myocardium

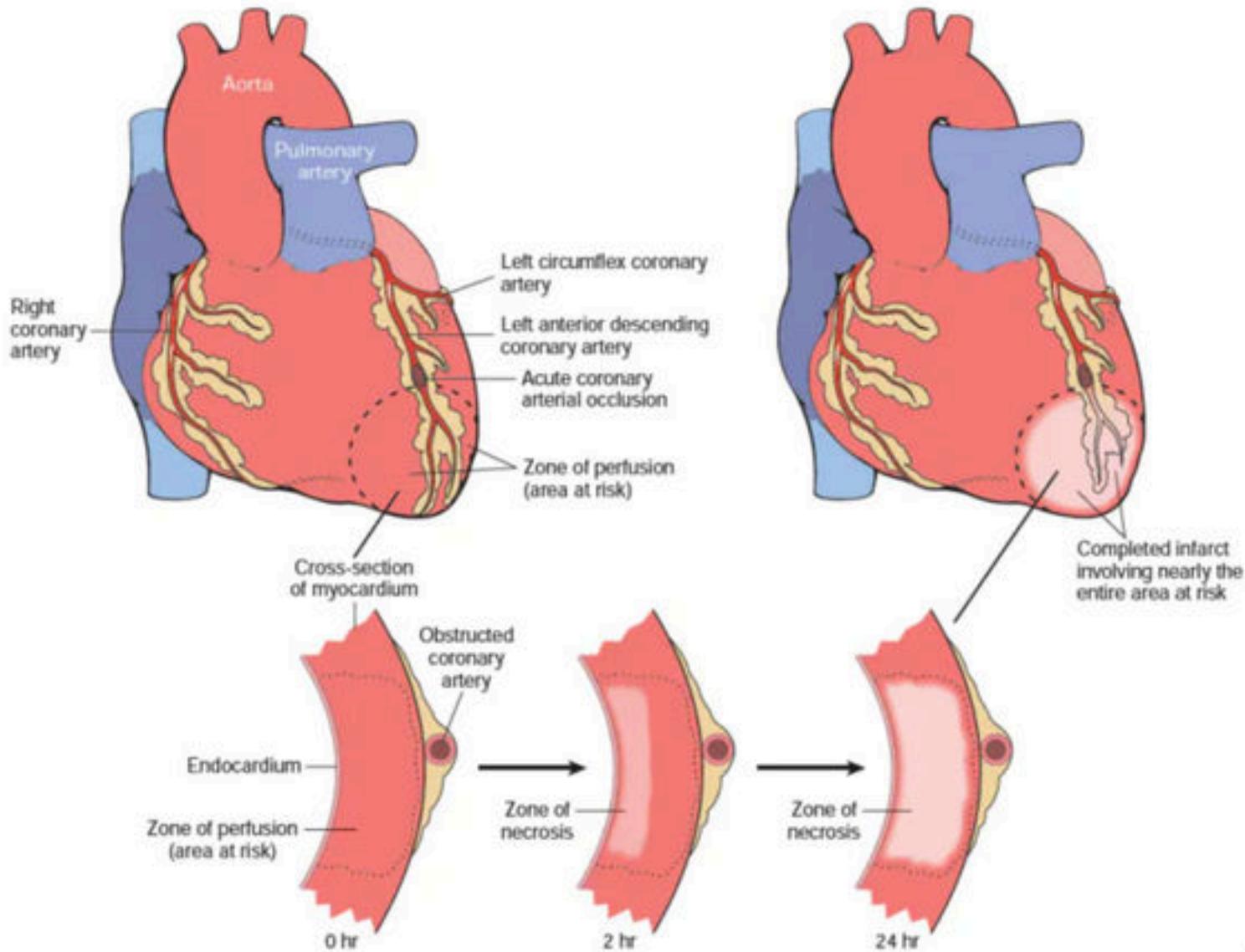




Transmural

Subendocardial

Laminar



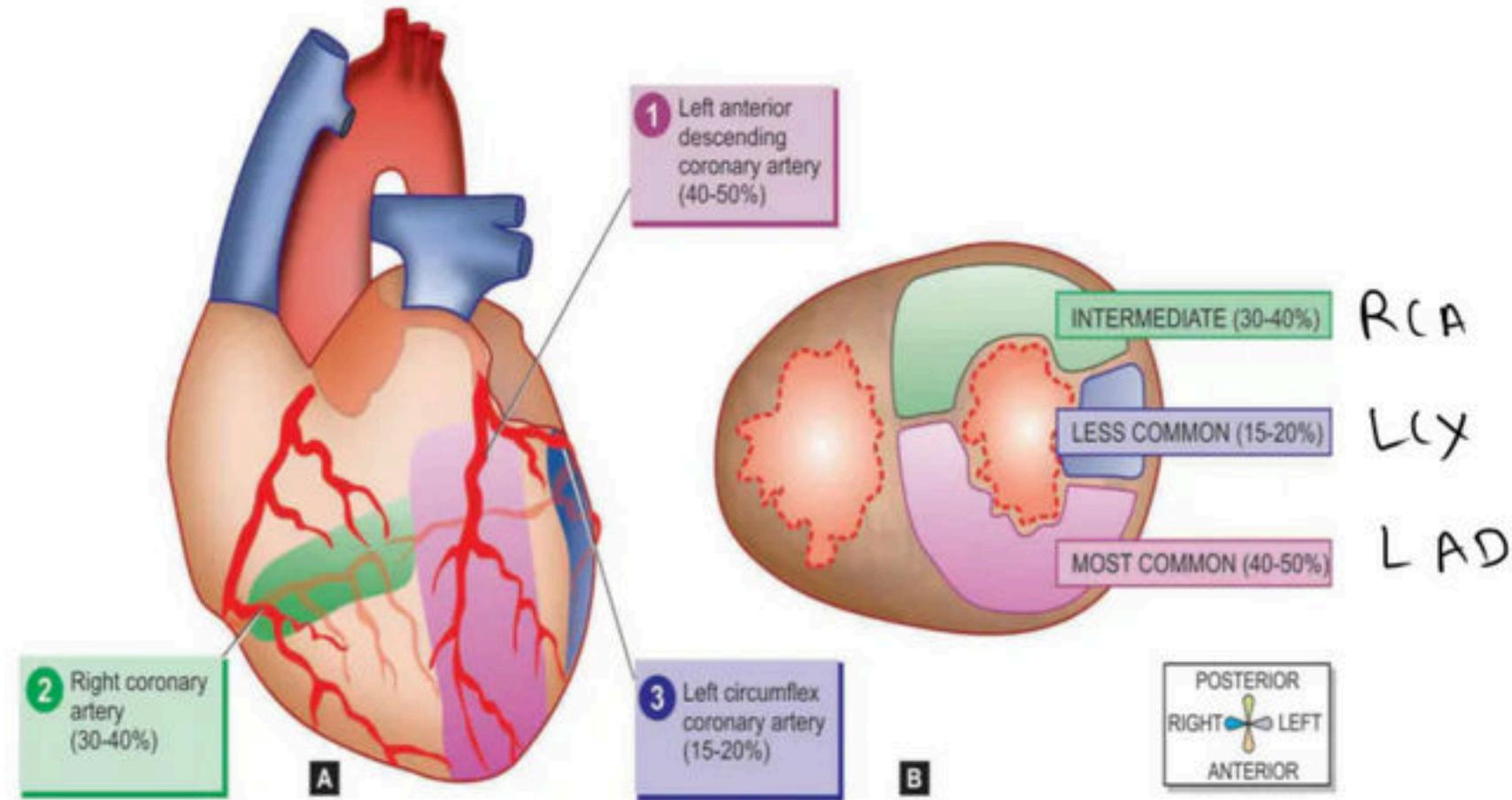
FEATURE	TRANSMURAL INFARCT	SUBENDOCARDIAL INFARCT
1. <i>Definition</i>	Full-thickness, solid	Inner third to half, patchy
2. <i>Frequency</i>	Most frequent (95%)	Less frequent
3. <i>Distribution</i>	Specific area of coronary supply	Circumferential
4. <i>Pathogenesis</i>	> 75% coronary stenosis	Hypoperfusion of myocardium
5. <i>Coronary thrombosis</i>	Common	Rare
6. <i>Epicarditis</i>	Common	None

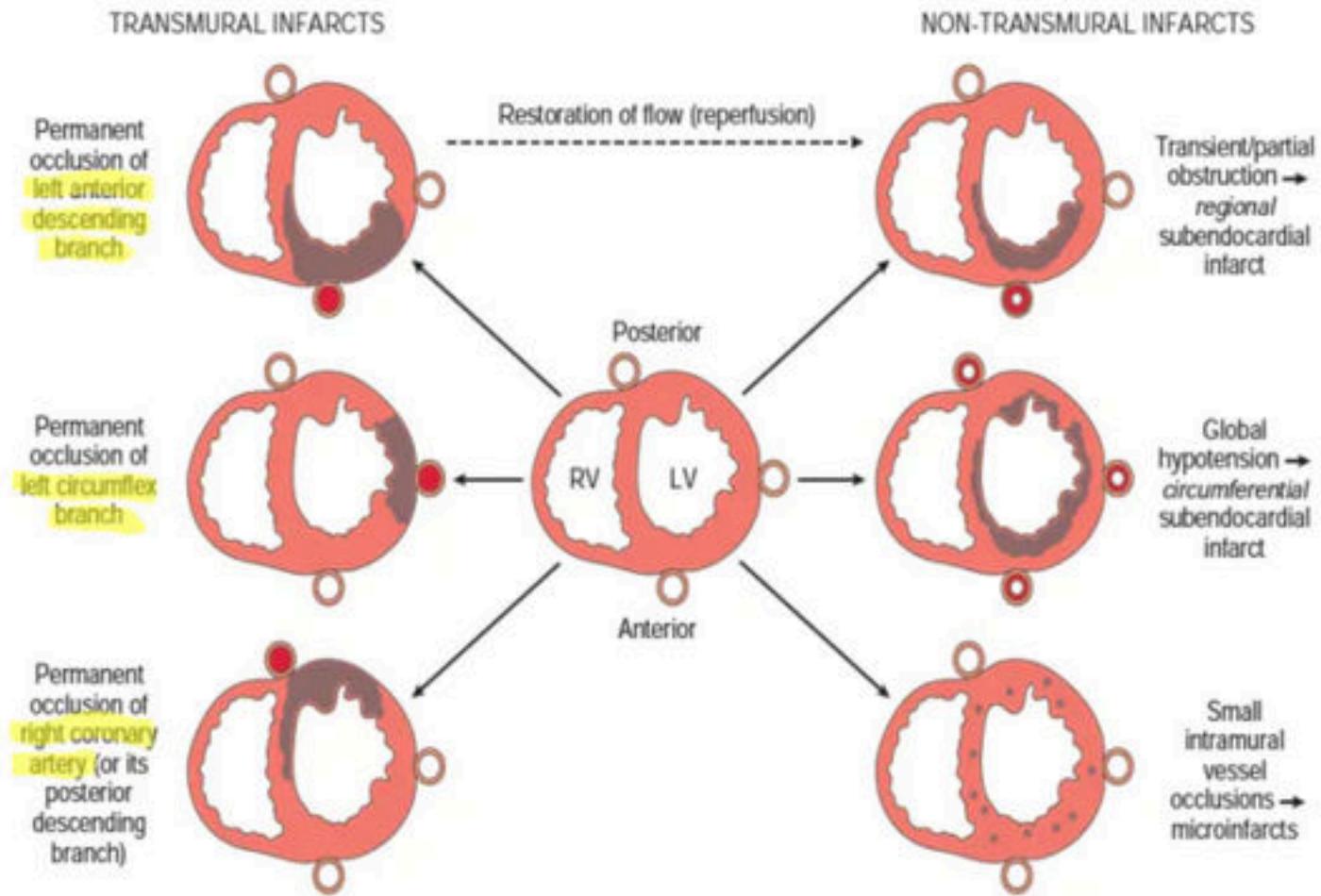
LOCATION OF INFARCTS

- Depends on which branch of coronary artery is obstructed

1. Stenosis of the left anterior descending coronary artery

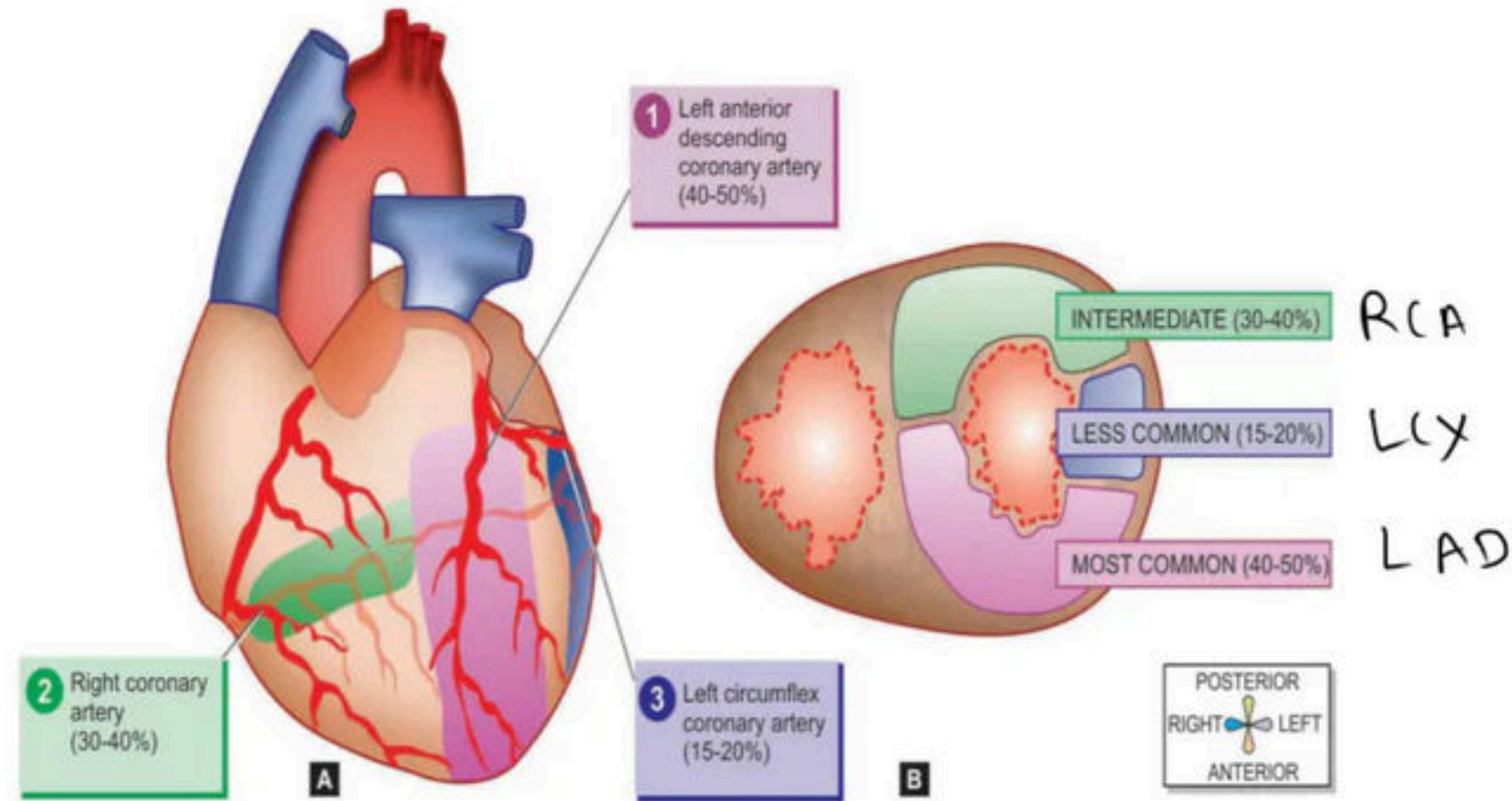
- It is the **most common (40-50%)**.
- The region of infarction is the **anterior part of the left ventricle including the apex and the anterior two-thirds of the interventricular septum**

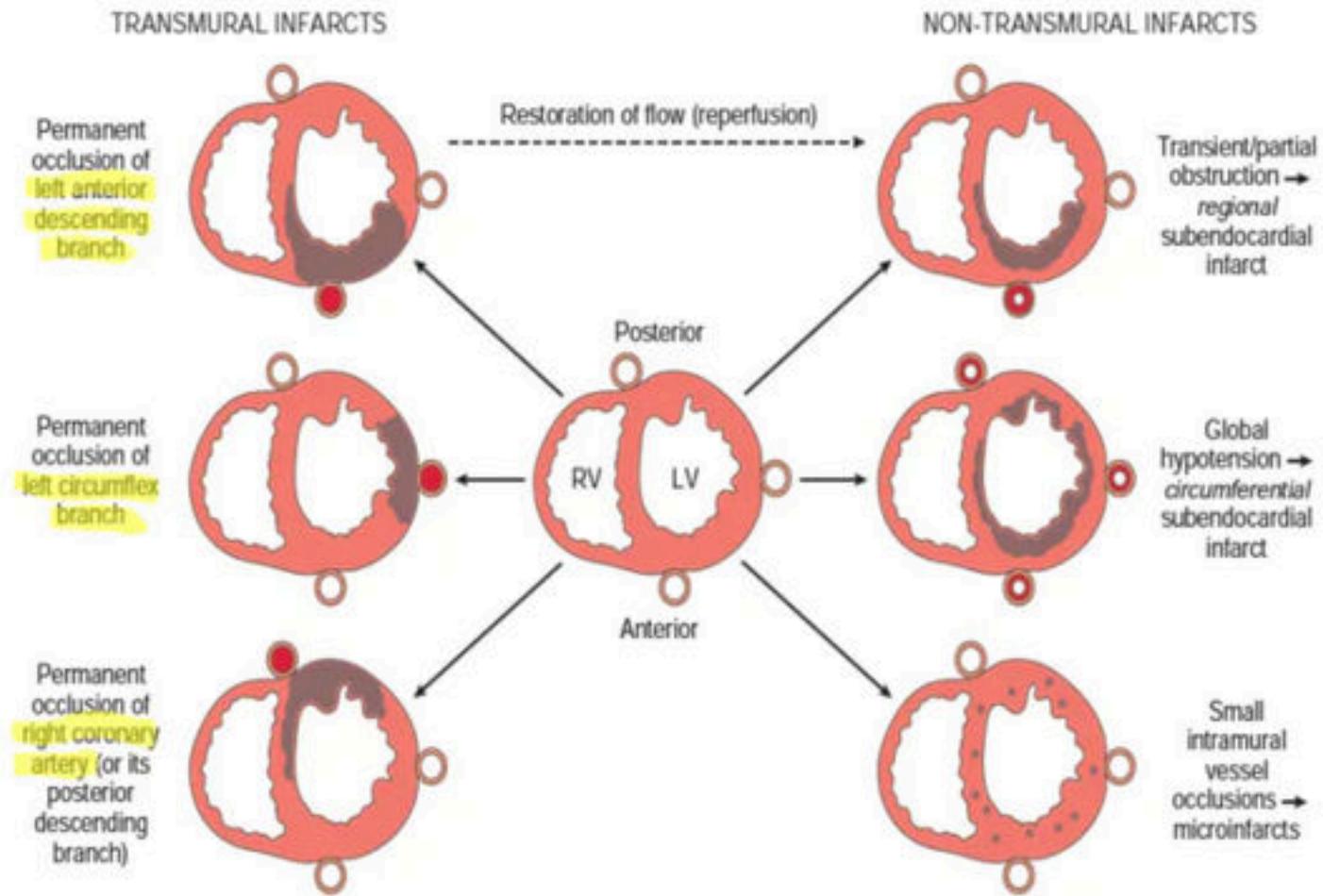




2. Stenosis of the right coronary artery

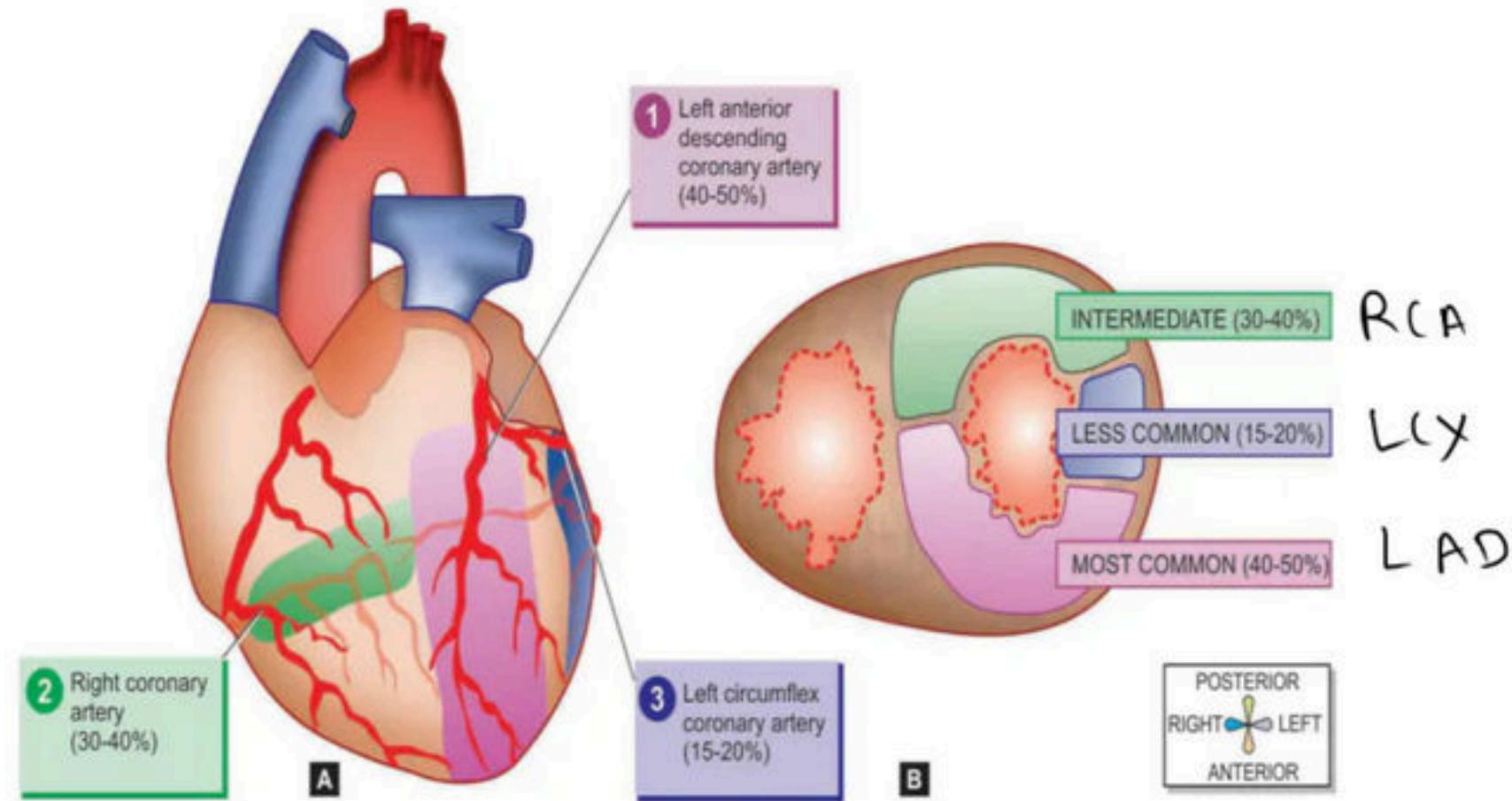
- It is the **next most frequent (30-40%)**.
- It involves **the posterior part of the left ventricle and the posterior one-third of the interventricular septum.**

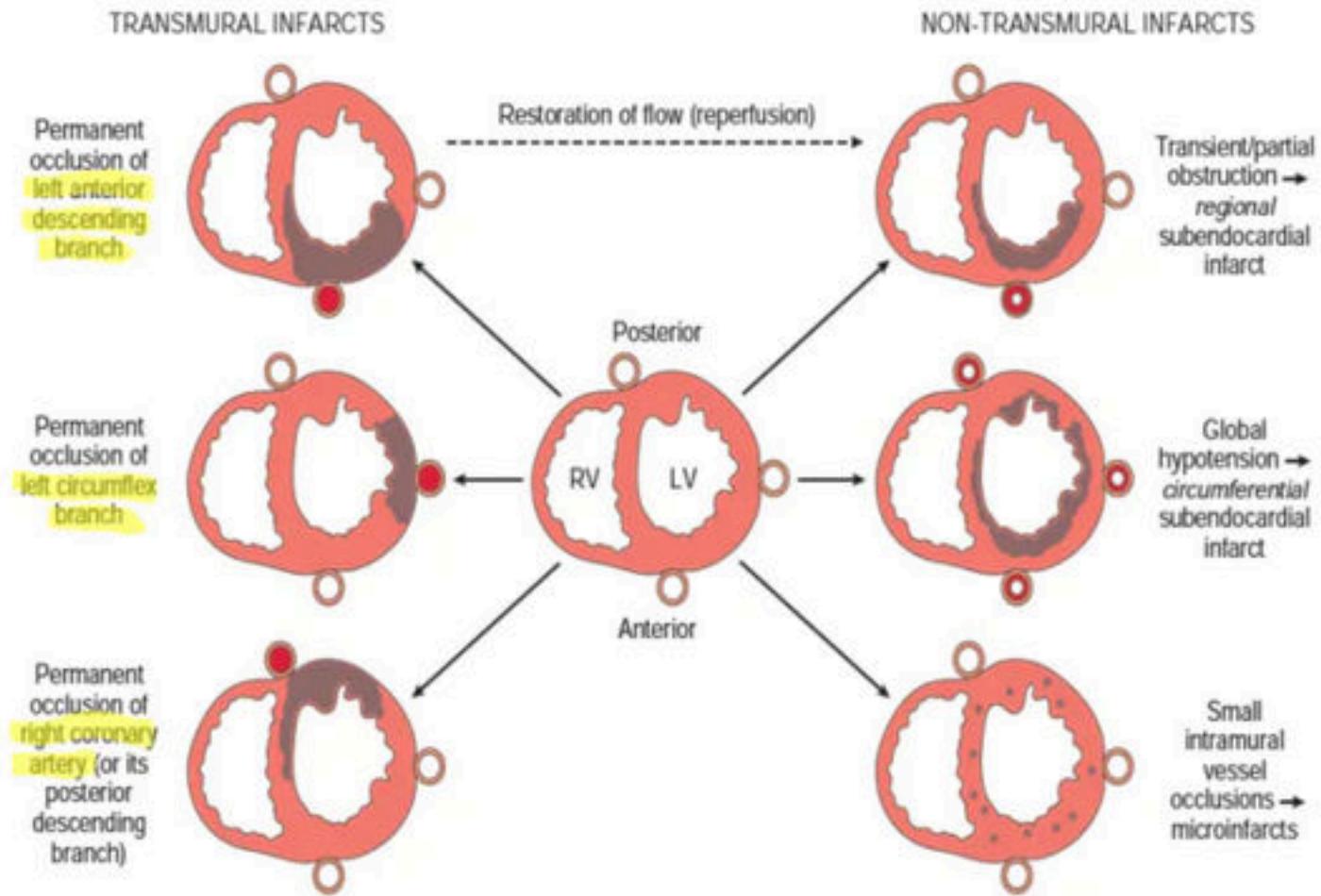




3. Stenosis of the left circumflex coronary artery

- It is seen least frequently (15-20%). Its area of involvement is **the lateral wall of the left ventricle**





HEADINGS

- **Definition**
- **Etiopathogenesis**
- **Types of infacts**
- **Location of infacts**
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- **Clinical features**
- **Diagnosis**
- **Complications**
- **Salvage in early infacts**

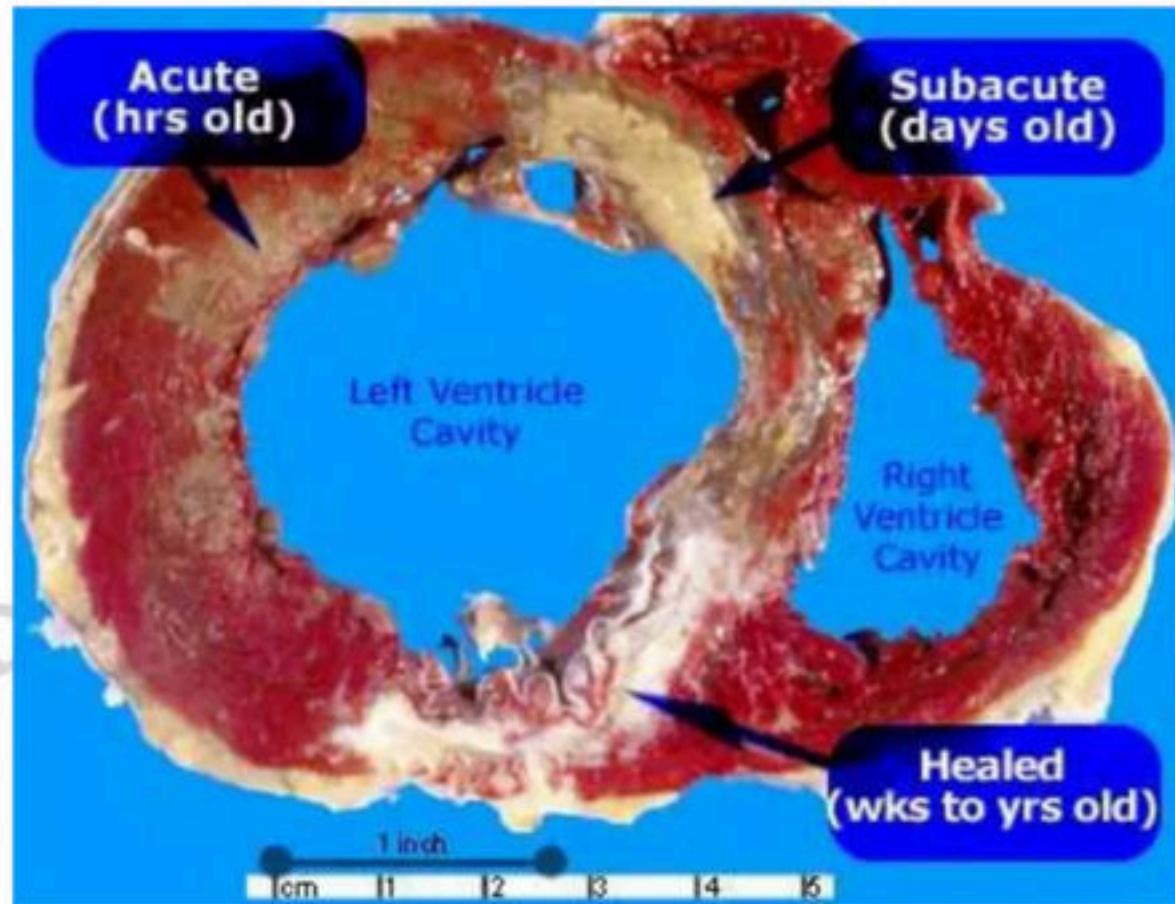
MORPHOLOGIC FEATURES

Dr. PRIYANKA SACHDEV

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4-12 hr	Dark mottling (occasional)	Early coagulation necrosis; edema; hemorrhage	
12-24 hr	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate	
1-3 days	Mottling with yellow-tan infarct center	Coagulation necrosis, with loss of nuclei and striations; brisk interstitial infiltrate of neutrophils	
3-7 days	Hyperemic border; central yellow-tan softening	Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border	
7-10 days	Maximally yellow-tan and soft, with depressed red-tan margins	Well-developed phagocytosis of dead cells; granulation tissue at margins	
10-14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	

GROSS

- Myocardial infarcts less than 12 hours old are usually not apparent on gross examination.
- But, necrotic area can be visualized after 2-3 hours by immersion of tissue slices in a solution of **triphenyltetrazolium chloride (TTC)**
 - **Non infarcted myocardium** → TTC imparts brick red color to it where the dehydrogenases enzymes are preserved.
 - **Infarcted area** appears as an unstained pale zone



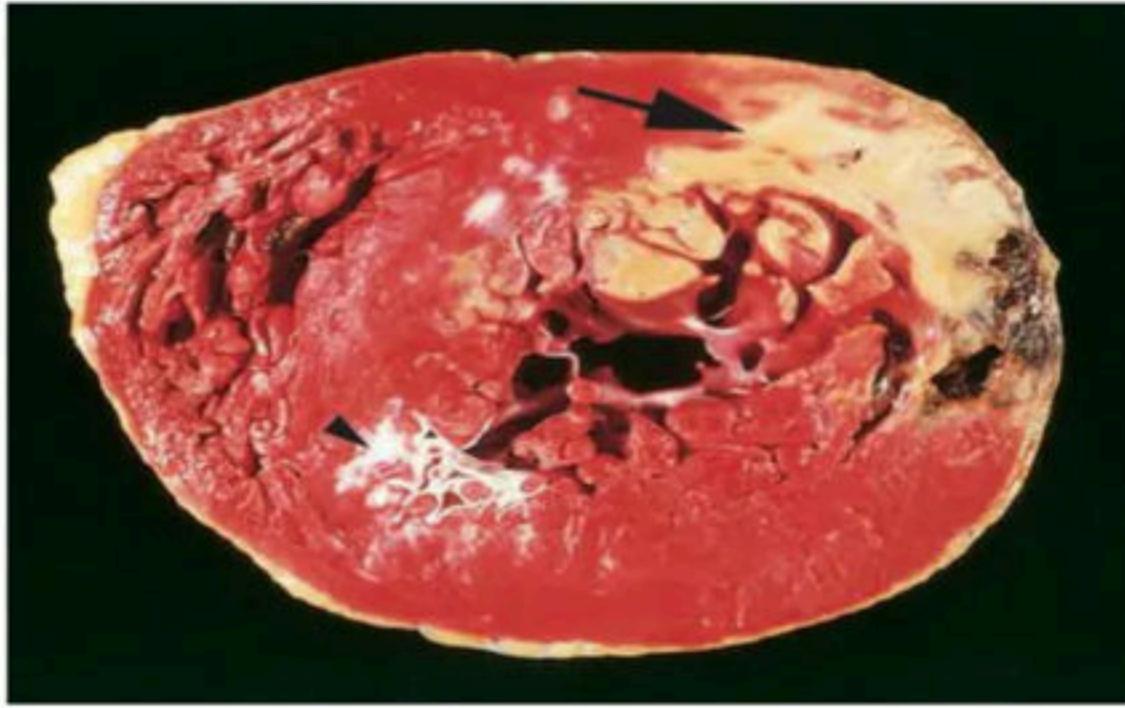
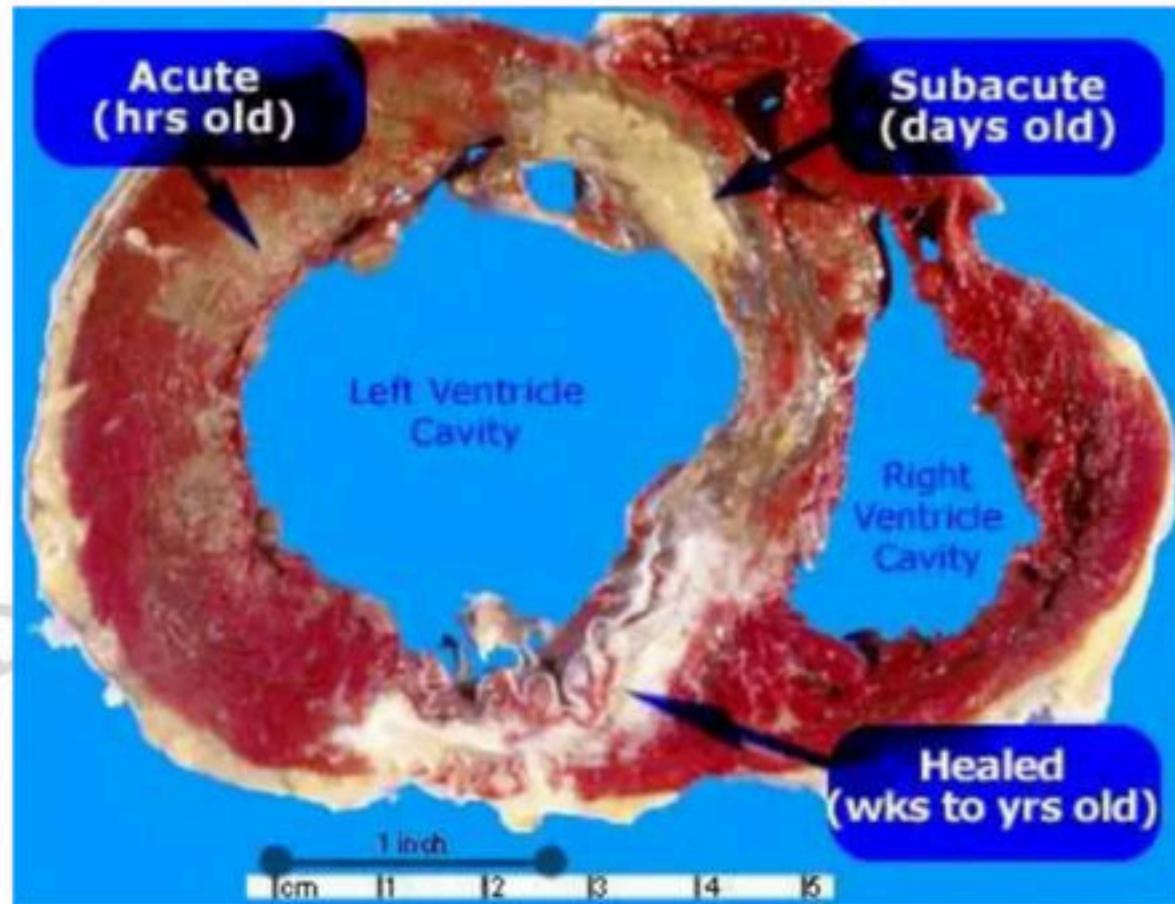


Figure 12-13 Acute myocardial infarct, predominantly of the posterolateral left ventricle, demonstrated histochemically by a lack of staining by triphenyltetrazolium chloride in areas of necrosis (arrow). The staining defect is due to the lactate dehydrogenase leakage that follows cell death. Note the myocardial hemorrhage at one edge of the infarct that was associated with cardiac rupture, and the anterior scar (arrowhead), indicative of old infarct. Specimen is oriented with the posterior wall at the top.

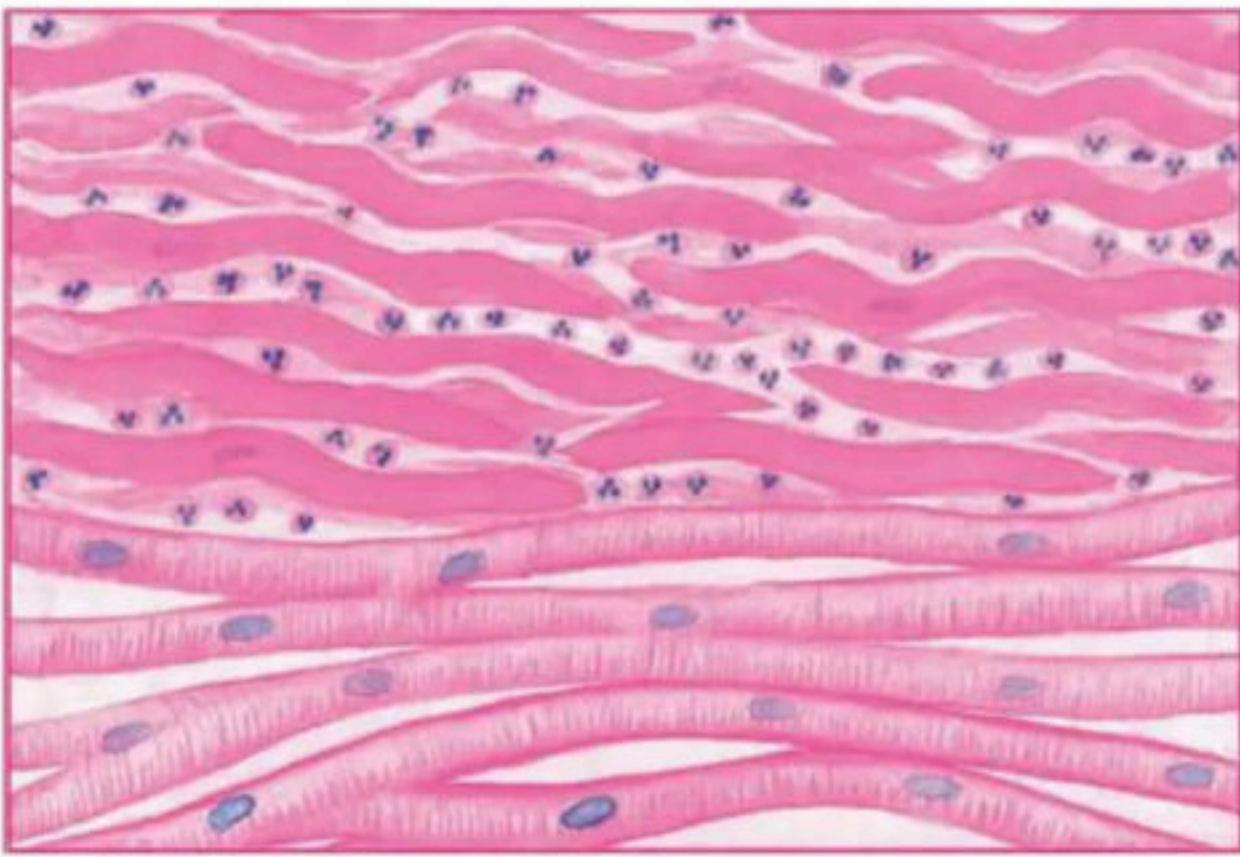


Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
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2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	

MICROSCOPY

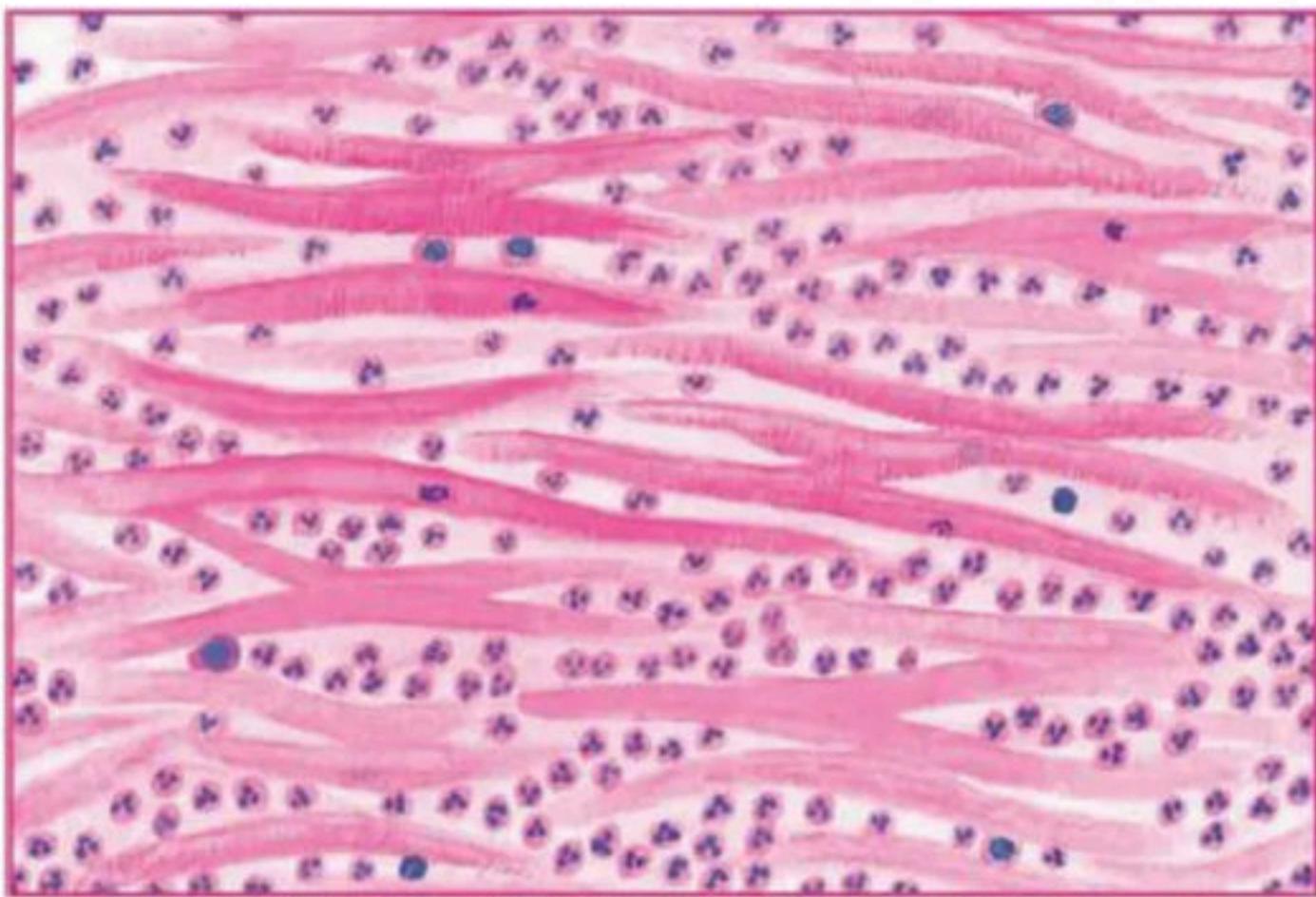
- The predominant mechanism of cell death in heart is **coagulative necrosis**
- Coagulative necrosis starts between **4-12 hours after onset of ischemia.**

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
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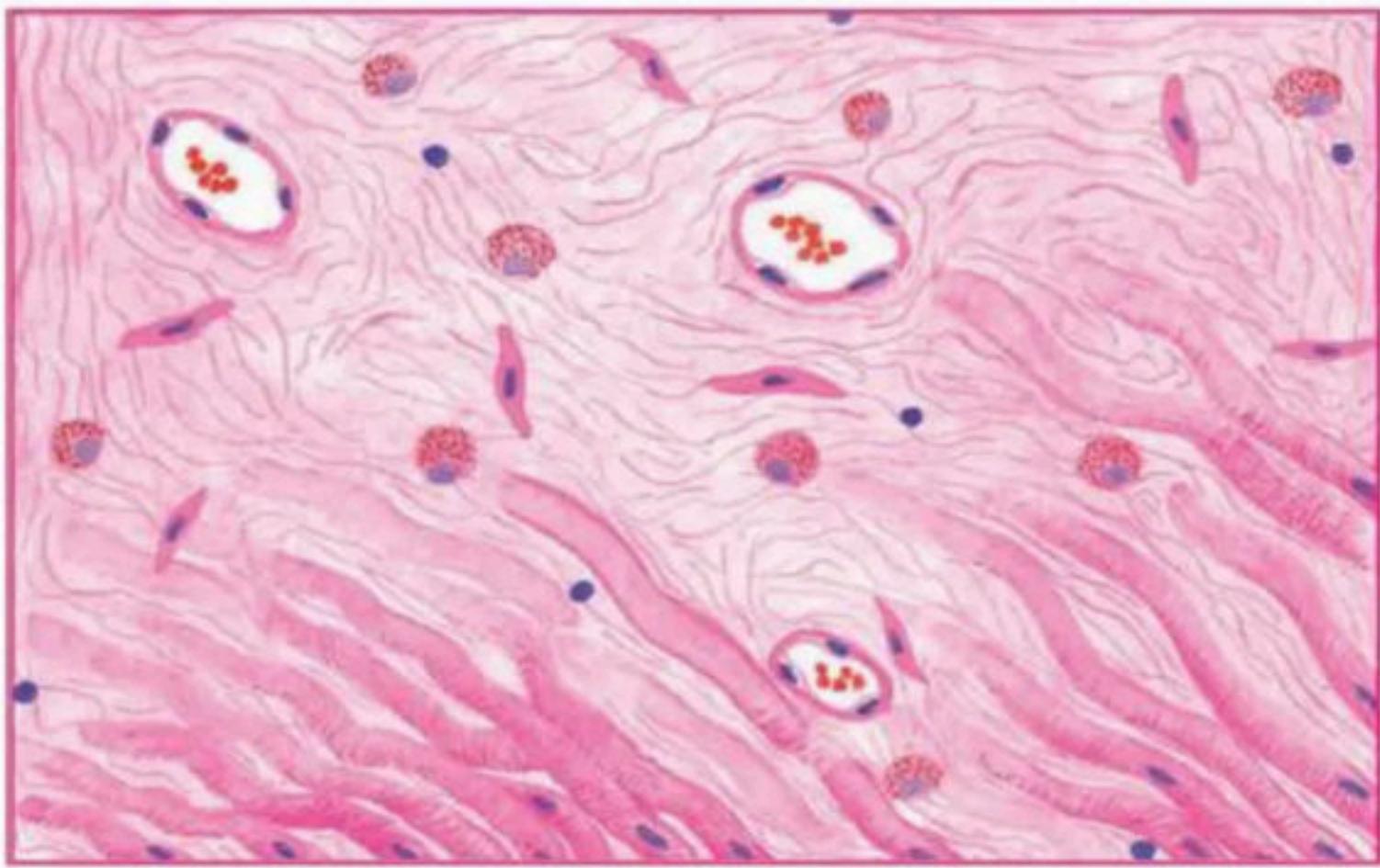
A, CHANGES DURING THE FIRST 24 HOURS

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4-12 hr	Dark mottling (occasional)	Early coagulation necrosis; edema; hemorrhage	
12-24 hr	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate	
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10-14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	



B, CHANGES DURING THE FIRST 48-72 HOURS

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4-12 hr	Dark mottling (occasional)	Early coagulation necrosis; edema; hemorrhage	
12-24 hr	Dark mottling	Ongoing coagulation necrosis; pyknosis of nuclei; myocyte hypereosinophilia; marginal contraction band necrosis; early neutrophilic infiltrate	
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7-10 days	Maximally yellow-tan and soft, with depressed red-tan margins	Well-developed phagocytosis of dead cells; granulation tissue at margins	
10-14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	



C, CHANGES BY THE END OF FIRST WEEK

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
4-12 hr	Dark mottling (occasional)	Early coagulation necrosis; edema; hemorrhage	
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10-14 days	Red-gray depressed infarct borders	Well-established granulation tissue with new blood vessels and collagen deposition	
2-8 wk	Gray-white scar, progressive from border toward core of infarct	Increased collagen deposition, with decreased cellularity	
>2 mo	Scarring complete	Dense collagenous scar	

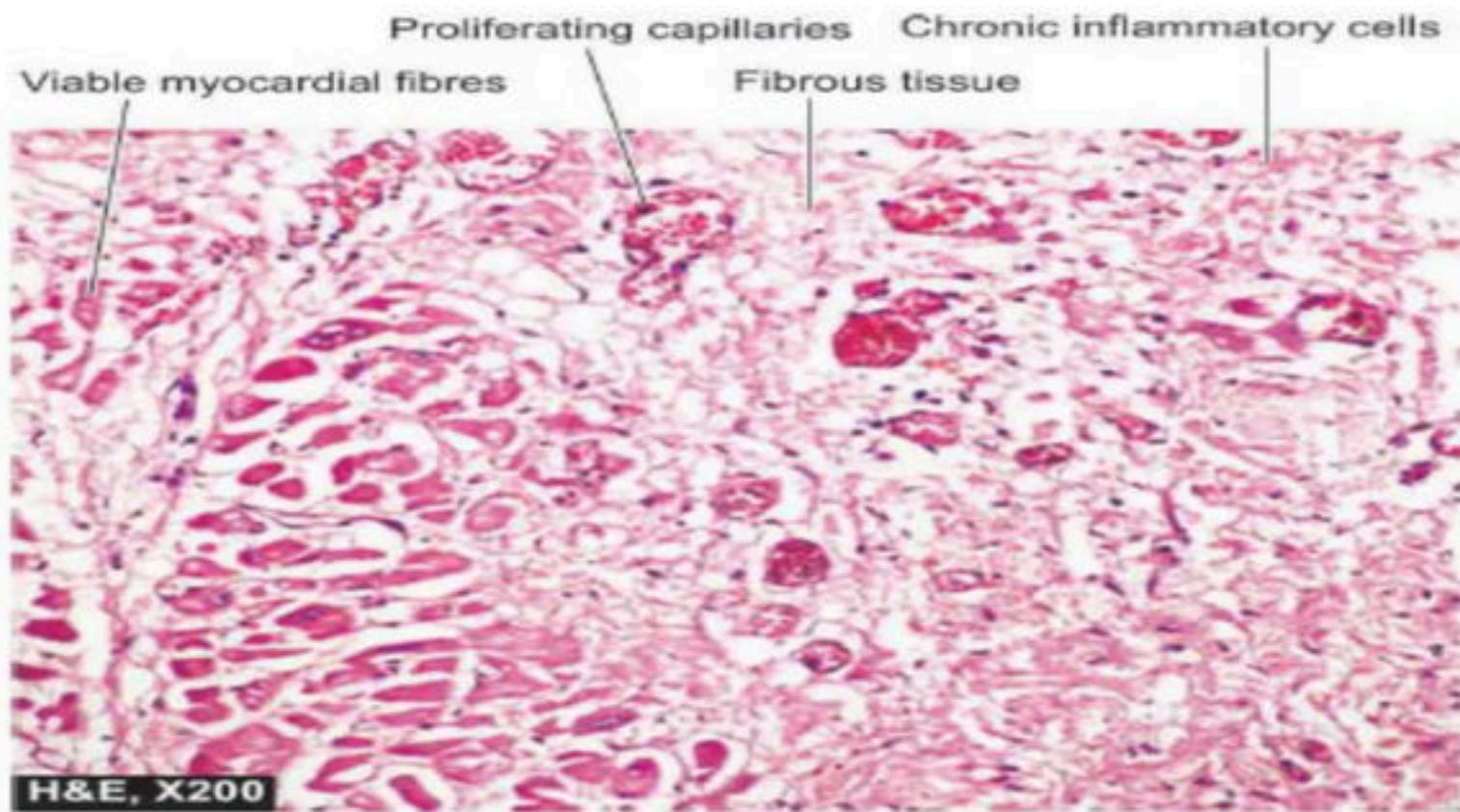


Figure 14.19 Old myocardial infarct. The infarcted area shows ingrowth of inflammatory granulation tissue.

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
Irreversible Injury			
½-4 hr	None	Usually none; variable waviness of fibers at border	Sarcolemmal disruption; mitochondrial amorphous densities
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>2 mo	Scarring complete	Dense collagenous scar	

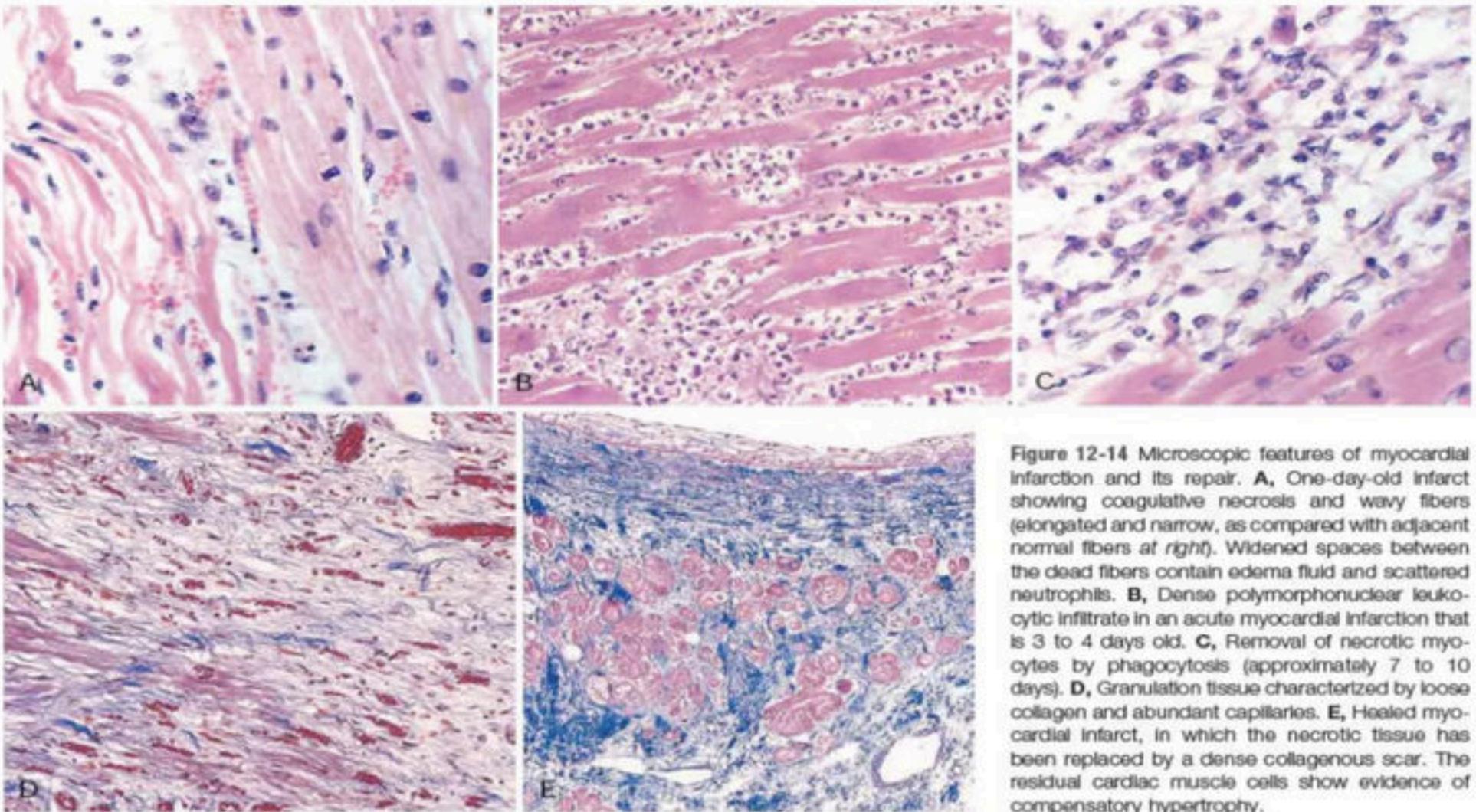


Figure 12-14 Microscopic features of myocardial infarction and its repair. **A**, One-day-old infarct showing coagulative necrosis and wavy fibers (elongated and narrow, as compared with adjacent normal fibers at right). Widened spaces between the dead fibers contain edema fluid and scattered neutrophils. **B**, Dense polymorphonuclear leukocytic infiltrate in an acute myocardial infarct that is 3 to 4 days old. **C**, Removal of necrotic myocytes by phagocytosis (approximately 7 to 10 days). **D**, Granulation tissue characterized by loose collagen and abundant capillaries. **E**, Healed myocardial infarct, in which the necrotic tissue has been replaced by a dense collagenous scar. The residual cardiac muscle cells show evidence of compensatory hypertrophy.

Time	Gross Features	Light Microscope	Electron Microscope
Reversible Injury			
0-½ hr	None	None	Relaxation of myofibrils; glycogen loss; mitochondrial swelling
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>2 mo	Scarring complete	Dense collagenous scar	

POLLS 4

Dr. PRIYANKA SACHDEV

Reversible injury in myocardium occurs at -

- a) 2 minutes
- b) 30 minutes
- c) 2 hours
- d) 5 hours

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B

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During autopsy of a patient died due to suspected Myocardial infarction, the heart was stained with triphenyltetrazolium tetrachloride dye. What will be the color of the viable myocardium?

- a) Blue
- b) White
- c) Red
- d) Dark brown

C

Dr. PRIYANKA SACHDEV

Dr. PRIYANKA SACHDEV

A 45 year old male had severe chest pain and was admitted to the hospital with a diagnosis of acute myocardial infarction. Four days later he died and autopsy showed transmural coagulative necrosis. Which of the following microscopic features will be seen on further examination?

- a) Fibroblast and collagen
- b) Granulation tissue
- c) Neutrophilic infiltration surrounding coagulative necrosis
- d) Granulomatous inflammation

C

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Dr. PRIYANKA SACHDEV

Earliest histological change in MI-

- a) Macrophage infiltration
- b) Neutrophilic infiltration
- c) Waviness of fibers
- d) Coagulative necrosis

Dr. P.P.

C

Dr. PRIYANKA SACHDEV

Dr. PRIYANKA SACHDEV

A myocardial infarct showing early granulation tissue has most likely occurred -

- a) Less than 1 hours
- b) Within 24 hrs
- c) Within 1 week
- d) Within 1 month

Dr. PRIYANKA SACHDEV

C

Dr. PRIYANKA SACHDEV

Dr. PRIYANKA SACHDEV

In myocardial infarction, microscopic picture of coagulation necrosis with neutrophilic infiltration is seen in -

- a) 4-12 hrs
- b) 12-24 hr
- c) 1-3 days
- d) 3-7 days

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C

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Dr. PRIYANKA SACHDEV

The cells seen after 72 hours in the infarcted area in MI are -

- a) Neutrophils
- b) Lymphocytes
- c) Macrophages
- d) Monocytes



C

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Cells seen in MI at 48 hours are -

- a) Polymorphs
- b) Fibroblasts
- c) Lymphocytes
- d) Macrophages



A

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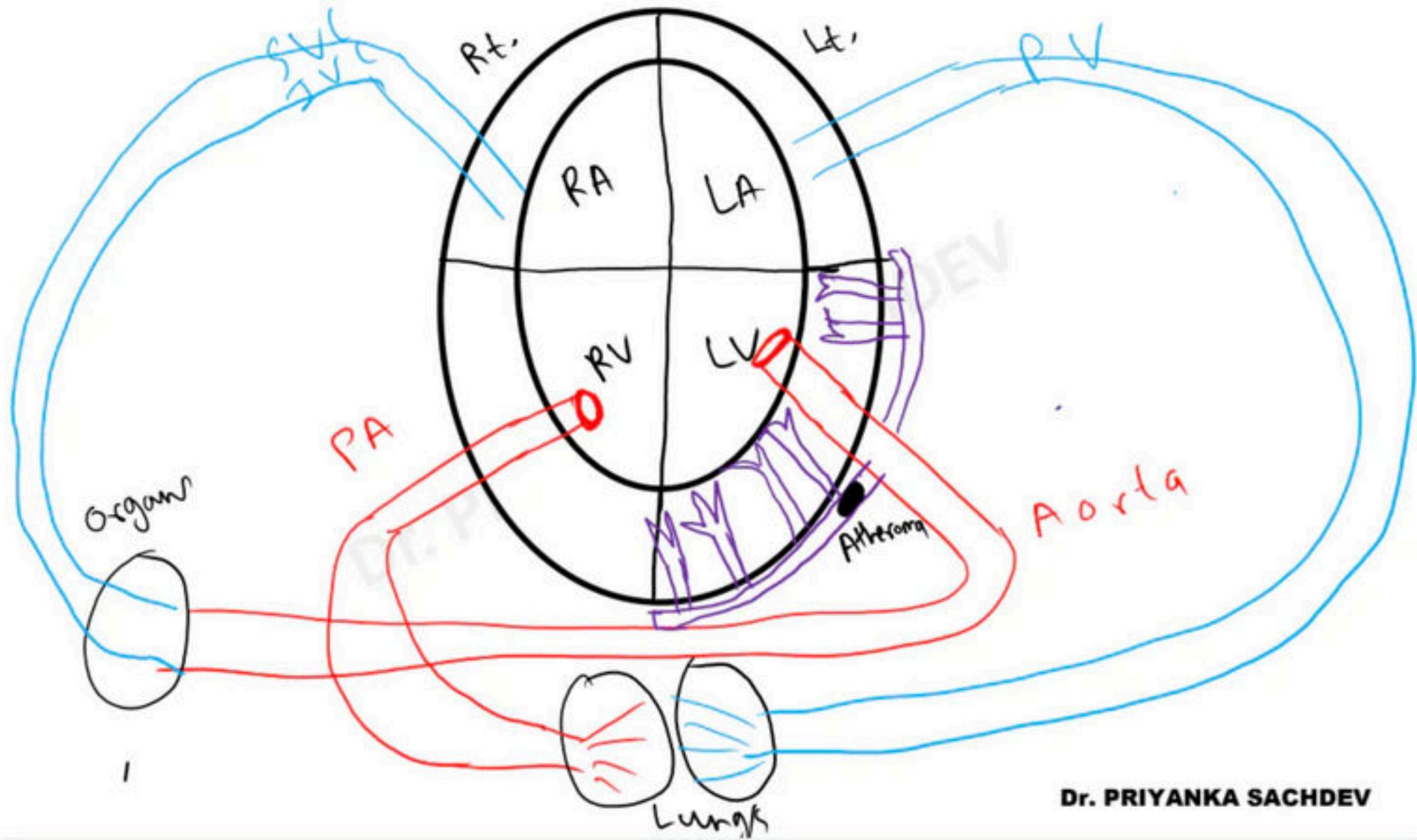
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- **Morphologic features (Gross and Microscopy)**
- **Clinical features**
- **Diagnosis**
- **Complications**
- **Salvage in early infacts**

Clinical features

- i) **Pain**: Usually sudden, severe, crushing and prolonged, substernal or precordial in location, unrelieved by rest or nitroglycerin, often radiating to one or both the arms, neck and back.
- ii) **Indigestion**: Pain is often accompanied by epigastric or substernal discomfort interpreted as 'heartburn' with nausea and vomiting.
- iii) **Apprehension**: The patient is often terrified, restless and apprehensive due to great fear of death.

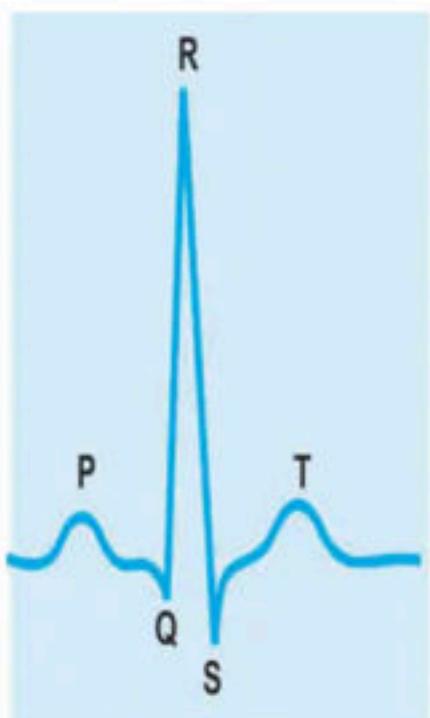
- iv) **Shock**: Systolic blood pressure is below 80 mmHg; lethargy, cold clammy limbs, peripheral cyanosis, weak pulse, tachycardia or bradycardia are often present.
- v) **Oliguria**: Urine flow is usually less than 20 ml per hour.
- vii) **Acute pulmonary oedema**: Some cases develop severe pulmonary congestion due to left ventricular failure and develop suffocation, dyspnoea, orthopnoea



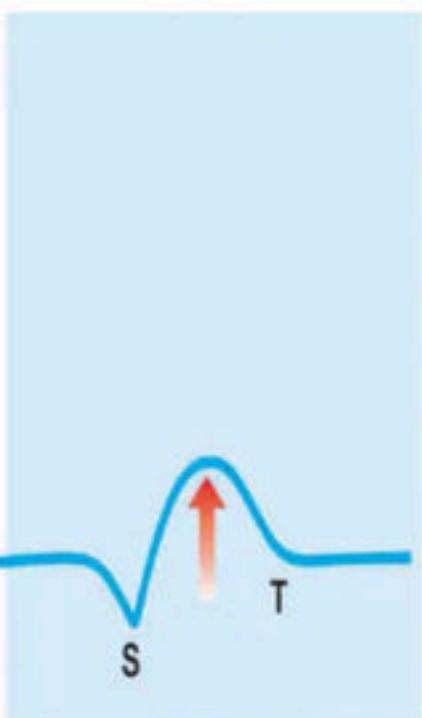
DIAGNOSIS

- 1. ECG changes**
- 2. Serum enzyme determinations/
Serum cardiac markers**

ECG changes



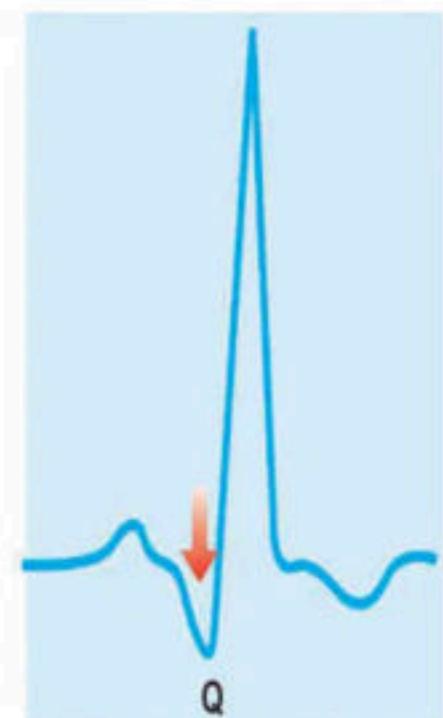
NORMAL ECG



ST-SEGMENT ELEVATION



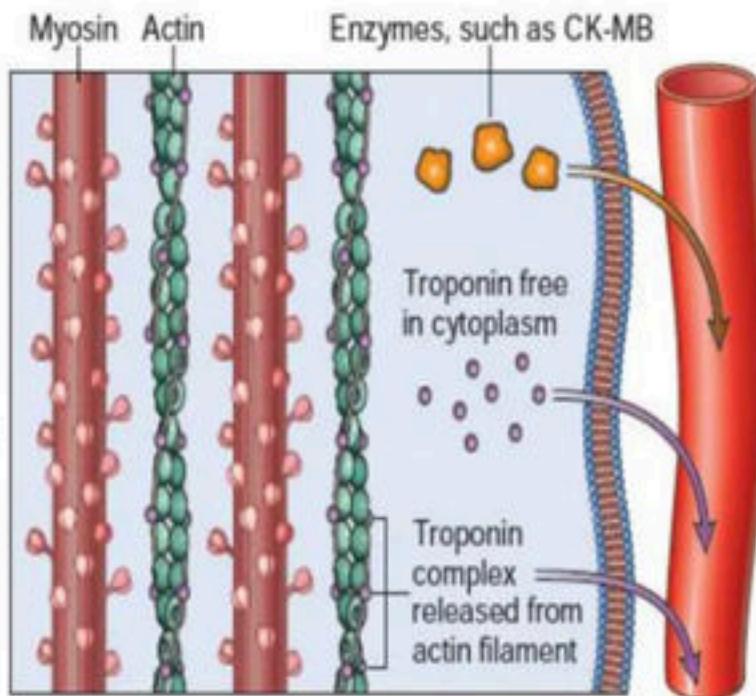
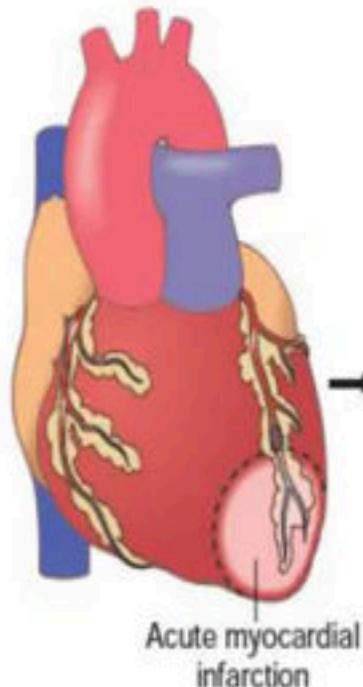
T-WAVE INVERSION



DEEP Q-WAVE

Serum cardiac markers

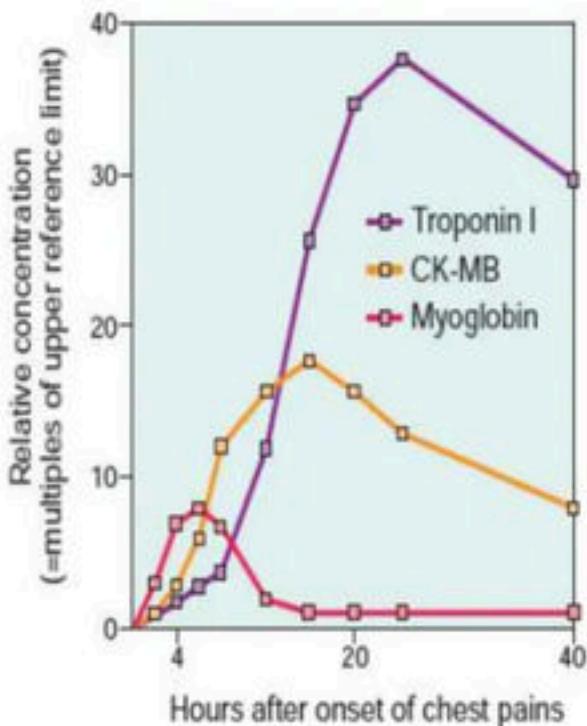
- Cardiac markers are **intracellular enzymes that leak out of dead necrotic cells**



1. Onset of myocardial infarction

2. Plasma membrane of necrotic myocytes becomes leaky

3. Molecules leak out of cell into circulation



4. These molecules can be used as biomarkers for diagnosis of myocardial infarction

Enzyme	Seen at	Peak	Return to normal	Comment
Myoglobin	2 hours		24 hours	Earliest marker to be detected^(AI 13), not specific
CPK-MB	2-4 hours^(AI 13)	24 hours	48-72 hours^(AIIMS 98)	Sensitive and specific; Marker of choice for reinfarction^(AI 03).
Troponin (T or I)	2-4 hours	48 hours	7-10 days^(NEET)	Most sensitive and specific marker^(NEET)
AST/ SGOT	Within 12 hours	48 hours	4-5 days	Not specific
LDH	24 hours	4-5 days	After 10 days^(NEET)	Only marker raised after 10 days^(NEET), Not specific.

i) Creatine phosphokinase (CPK)

3 forms—

- a) CK-MM** derived from skeletal muscle;
- b) CK-BB** derived from brain
- c) CK-MB** derived from cardiac muscles

- CK-MB has further 2 forms—
 1. **CK-MB2** is the myocardial form
 2. **CK-MB1** is extracardiac form.
- A ratio of **CK-MB2: CK-MB1 > 1.5** is highly sensitive for the diagnosis of acute MI after 4-6 hours of onset of myocardial ischaemia.
- CK-MB disappears from blood by **48 hours**

Enzyme	Seen at	Peak	Return to normal	Comment
Myoglobin	2 hours		24 hours	Earliest marker to be detected^(AI 13), not specific
CPK-MB	2-4 hours^(AI 13)	24 hours	48-72 hours^(AIIMS 98)	Sensitive and specific; Marker of choice for reinfarction^(AI 03).
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LDH	24 hours	4-5 days	After 10 days^(NEET)	Only marker raised after 10 days^(NEET), Not specific.

ii) Lactate dehydrogenase (LDH)

- Total LDH estimation also **lacks specificity** since this enzyme is present in various tissues besides myocardium such as in skeletal muscle, kidneys, liver, lungs and red blood cells.
- LDH levels begin to **rise after 24 hours, reach peak in 3 to 6 days and return to normal in 14 days.**

Enzyme	Seen at	Peak	Return to normal	Comment
<i>Myoglobin</i>	2 hours		24 hours	<i>Earliest marker to be detected</i> ^(AI 13) , not specific
<i>CPK-MB</i>	2-4 hours ^(AI 13)	24 hours	48-72 hours ^(AIIMS 98)	Sensitive and specific; <i>Marker of choice for reinfarction</i> ^(AI 03) .
<i>Troponin (T or I)</i>	2-4 hours	48 hours	7-10 days ^(NEET)	<i>Most sensitive and specific marker</i> ^(NEET)
<i>AST/ SGOT</i>	Within 12 hours	48 hours	4-5 days	Not specific
<i>LDH</i>	24 hours	4-5 days	After 10 days ^(NEET)	<i>Only marker raised after 10 days</i> ^(NEET) , Not specific.

iii) Cardiac-specific troponins (cTn)

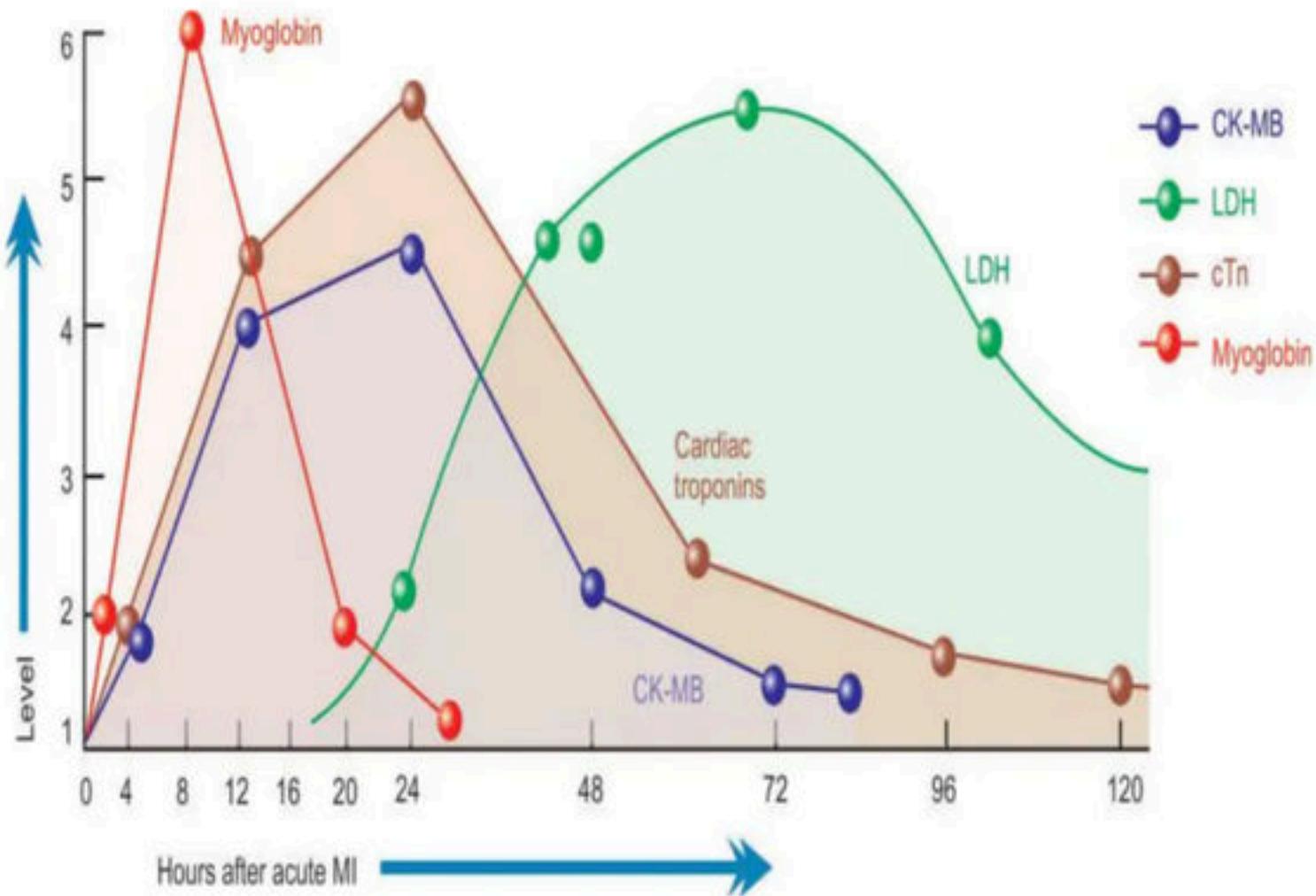
There are two types of cTn →

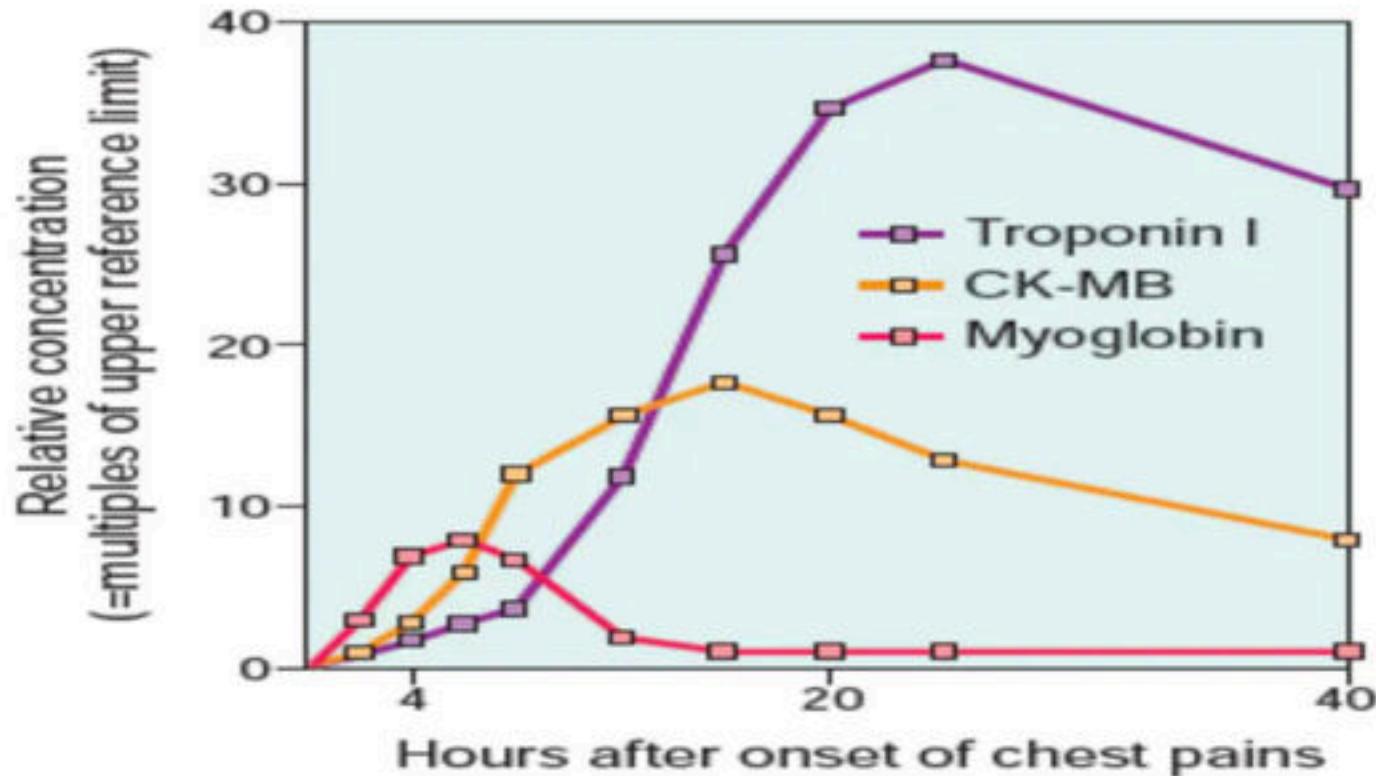
- a) cardiac troponin T (**cTnT**)
 - b) cardiac troponin I (**cTnI**)
-
- **Most sensitive and specific marker for MI**

Enzyme	Seen at	Peak	Return to normal	Comment
Myoglobin	2 hours		24 hours	Earliest marker to be detected^(AI 13), not specific
CPK-MB	2-4 hours^(AI 13)	24 hours	48-72 hours^(AIIMS 98)	Sensitive and specific; Marker of choice for reinfarction^(AI 03).
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AST/ SGOT	Within 12 hours	48 hours	4-5 days	Not specific
LDH	24 hours	4-5 days	After 10 days^(NEET)	Only marker raised after 10 days^(NEET), Not specific.

iv) Myoglobin

- Though myoglobin is the **first cardiac marker** to become elevated after myocardial infarction
- It **lacks cardiac specificity**
- It is excreted in the urine rapidly. Its levels, thus, **return to normal within 24 hours of attack of acute MI.**





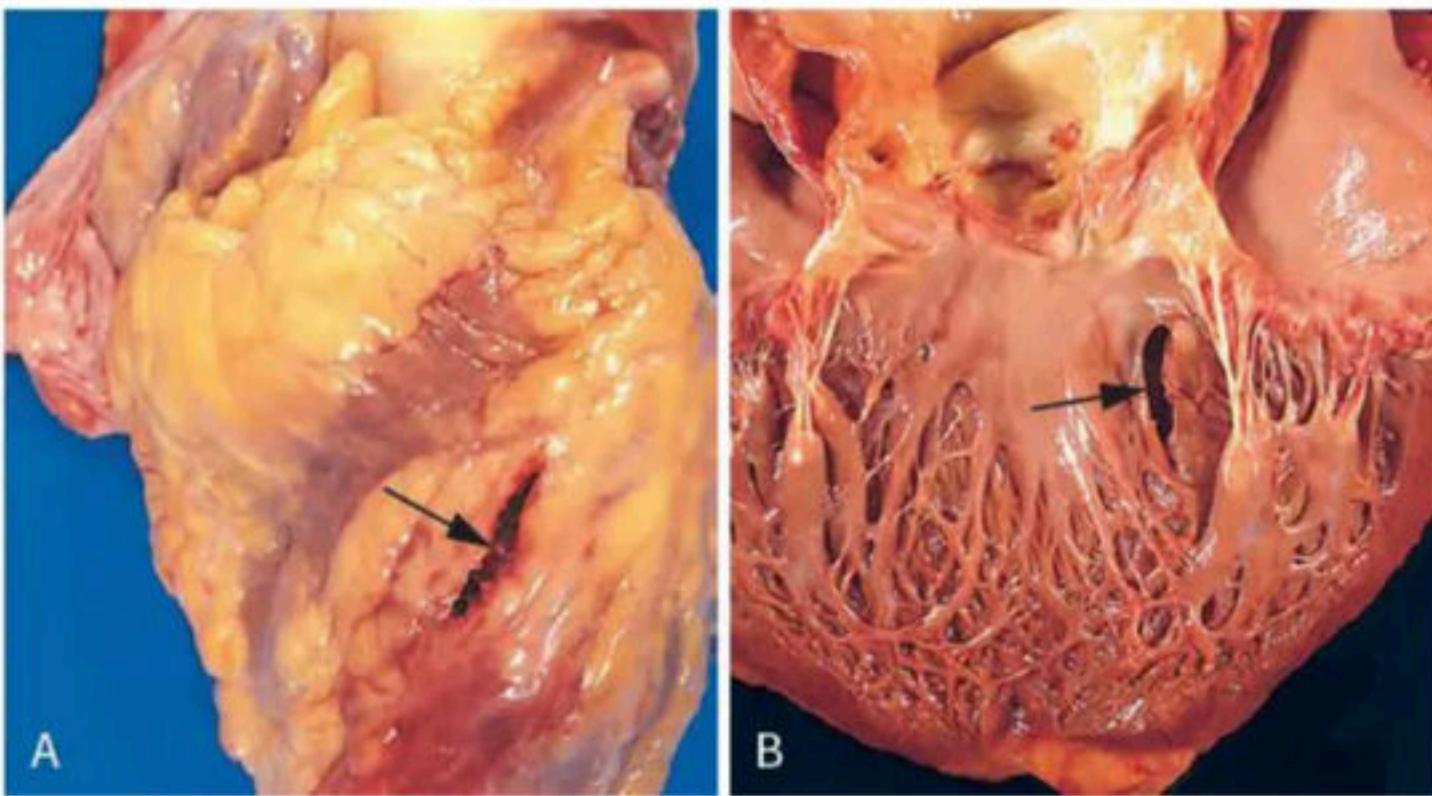
1k
o

4. These molecules can be used as biomarkers for diagnosis of myocardial infarction

COMPLICATIONS

- **1. Arrhythmias** are the most common complication in acute MI.
- These occur due to ischaemic injury or irritation to the conduction system, resulting in abnormal rhythm

- 2. Heart failure (Right or left)
- 3. Cardiogenic shock
- 4. Rupture → most often from the infarcted ventricular wall into the pericardial cavity causing haemopericardium

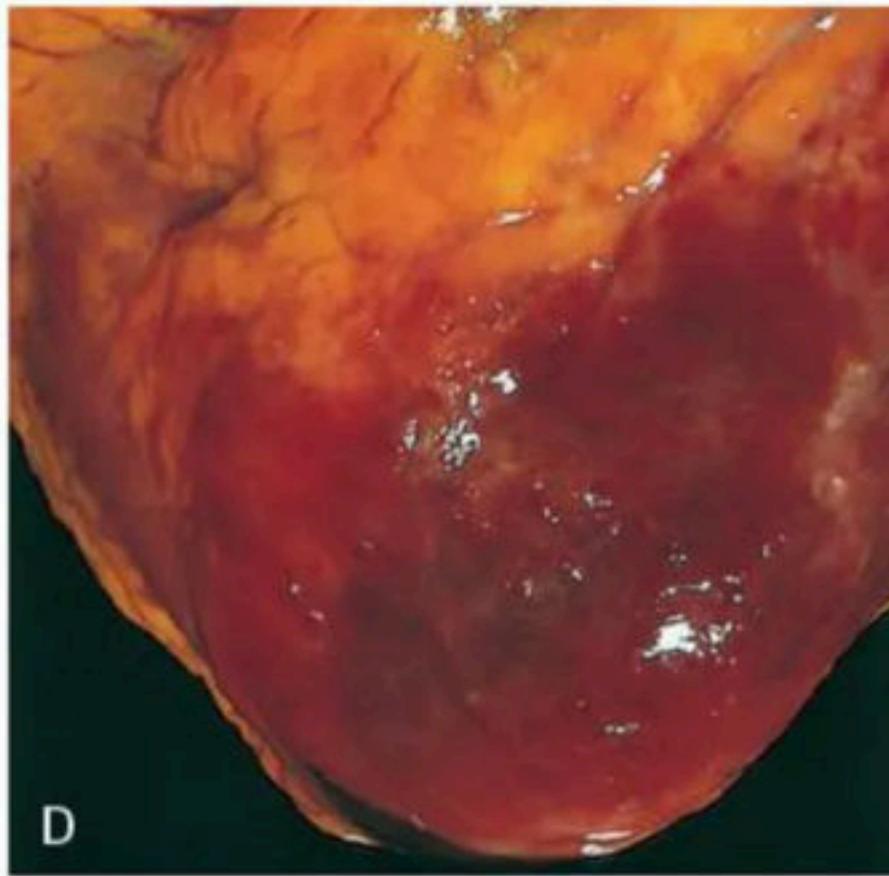


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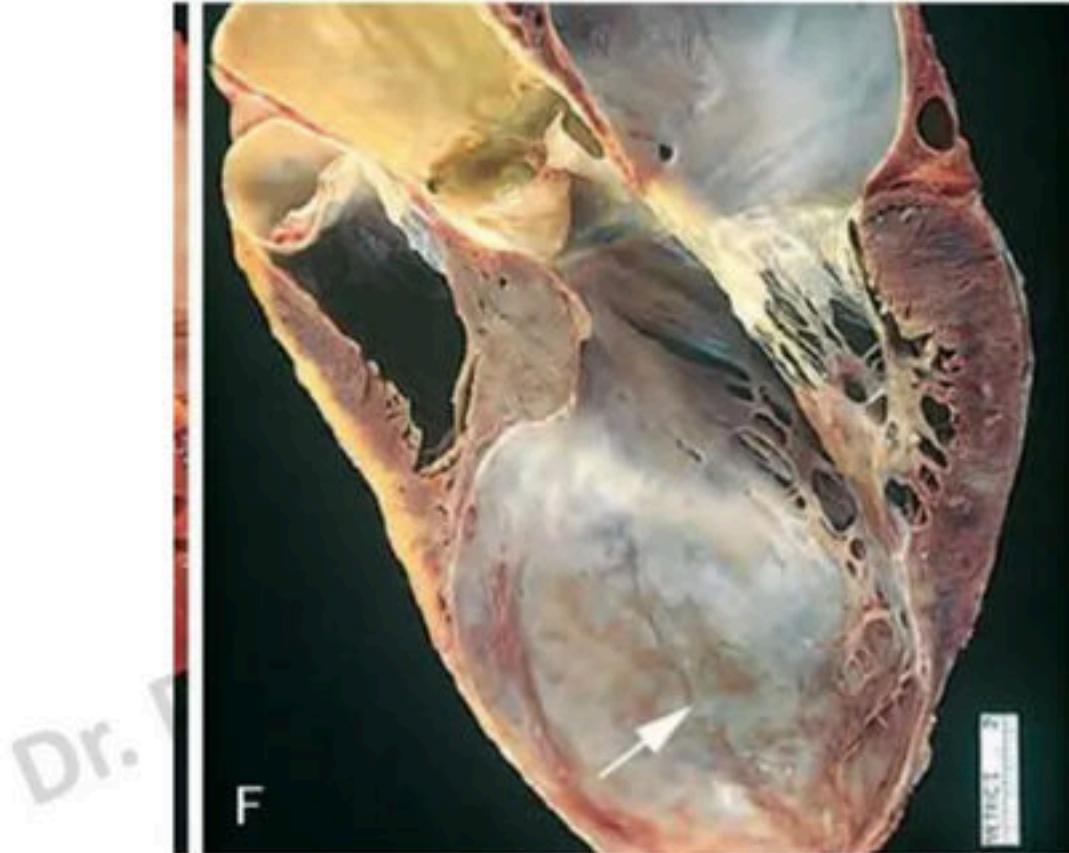
- **6. Cardiac aneurysm** → It occurs in healed infarcts through thin, fibrous, non-elastic scar tissue
- **7. Pericarditis** Sterile pericarditis appearing on about the second day is common over transmural infarcts
- **8. Postmyocardial infarction syndrome/ Dressler's syndrome**
→ It usually occurs 1 to 6 weeks after the attack of MI. It is characterised by pneumonitis.

Dr.

D



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HEADINGS

- **Definition**
- **Etiopathogenesis**
- **Types of infacts**
- **Location of infacts**
- **Morphologic features (Gross and Microscopy)**
- **Clinical features**
- **Diagnosis**
- **Complications**
- **Salvage in early infacts**

POLLS 5

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First enzyme to be raised in MI is -

- a) CPK-MB
- b) LDH
- c) Myoglobin
- d) Troponin-I

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C

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Post MI day 10 which enzyme is raised -

- a) LDH
- b) CPK
- c) Troponin
- d) Myoglobin

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A

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In stable angina -

- a) CK-MB is elevated
- b) Troponin T is elevated
- c) Myoglobin is elevated
- d) The level of cardiac markers remain unchanged

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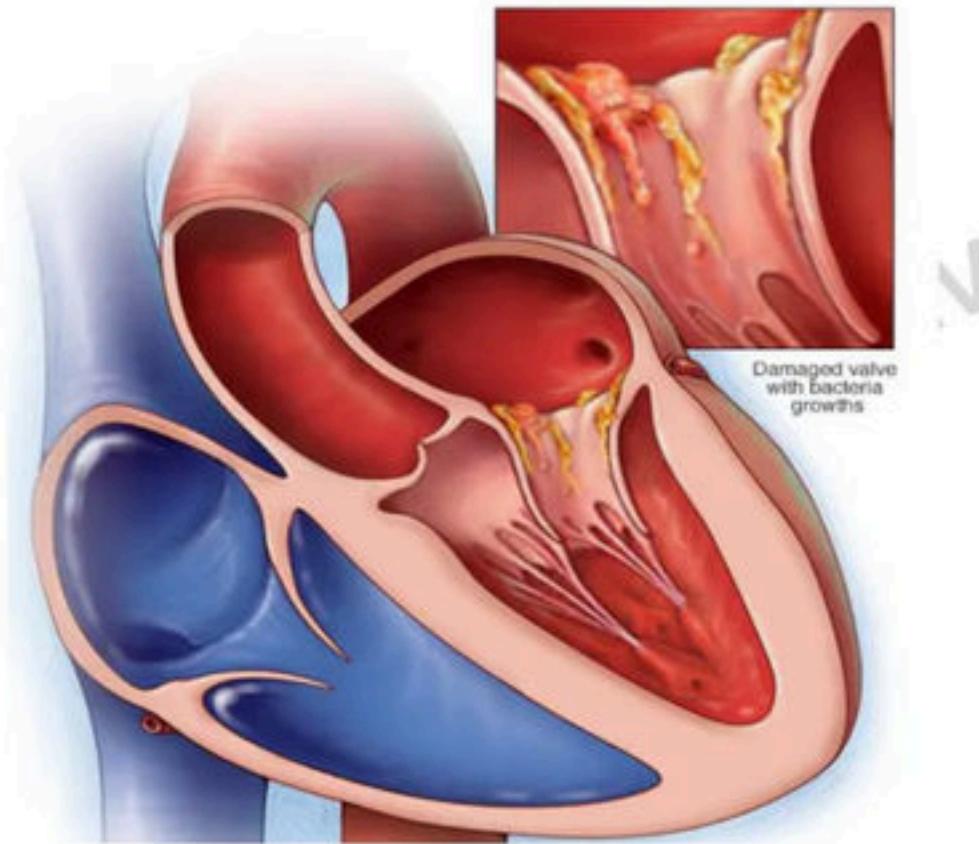
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ENDOCARDITIS

- Endocarditis is an **inflammation of the inner layer of the heart, i.e. endocardium**
- Usually involve the **heart valves (native or prosthetic)**.
- Other structures which may be involved are interventricular septum, chordae tendinae, the mural endocardium, and intracardiac devices



Damaged valve
with bacteria
growths

TYPES

A. NON-INFECTIVE

1. Rheumatic endocarditis (page 420)
2. Atypical verrucous (Libman-Sacks) endocarditis
3. Non-bacterial thrombotic (cachectic, marantic) endocarditis

B. INFECTIVE

1. Bacterial endocarditis
2. Other infective types (tuberculous, syphilitic, fungal, viral, rickettsial)

1. Rheumatic fever and rheumatic heart disease (Endocarditis)

2.

- 2. Atypical verrucous (Libman-Sacks) endocarditis
- 3. Non-bacterial thrombotic (cachectic, marantic) endocarditis

INFECTIVE

- 1. Bacterial endocarditis
- 2. Other infective types (tuberculous, syphilitic, fungal, viral, rickettsial)

FEATURE	RHEUMATIC	LIBMAN-SACKS	NON-BACTERIAL THROMBOTIC	BACTERIAL (INFECTIVE)
1. Valves commonly affected	Mitral alone; mitral and aortic combined	Mitral, tricuspid	Mainly mitral; less often aortic and tricuspid	Mitral; aortic; combined mitral and aortic
2. Location on valve cusps or leaflets	Occur along the line of closure, atrial surface of atrioventricular valves and ventricular surface of semilunar valves	Occur on both surfaces of valve leaflets or cusps, in the valve pockets	Occur along the line of closure	SABE more often on diseased valves; ABE on previously normal valves; location same as in RHD
3. Macroscopy				
	Small, multiple, warty, grey brown, translucent, firmly attached, generally produce permanent valvular deformity	Medium-sized, multiple, generally do not produce significant valvular deformity	Small but larger than those of rheumatic, single or multiple, brownish, firm, but more friable than those of rheumatic	Often large, grey-tawny to greenish, irregular, single or multiple, typically friable
4. Microscopy	Composed of fibrin with superimposed platelet thrombi and no bacteria. Adjacent and underlying endocardium shows oedema, proliferation of capillaries, mononuclear inflammatory infiltrate and occasional Aschoff bodies.	Composed of fibrinoid material with superimposed fibrin and platelet thrombi and no bacteria. The underlying endocardium shows fibrinoid necrosis, proliferation of capillaries and acute and chronic inflammatory infiltrate including the haematoxylin bodies of Gross.	Composed of degenerated valvular tissue, fibrin-platelets thrombi and no bacteria. The underlying valve shows swelling of collagen, fibrinoid change, proliferation of capillaries but no significant inflammatory cell infiltrate.	Composed of outer eosinophilic zone of fibrin and platelets, covering colonies of bacteria and deeper zone of non-specific acute and chronic inflammatory cells. The underlying endocardium may show abscesses in ABE and inflammatory granulation tissue in the SABE.

Rheumatic fever & rheumatic heart disease

Click or Scan QR code to join
Telegram group discussion



Rheumatic fever

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OVERVIEW

- Introduction
 - Etiology
 - Pathogenesis
 - Jones criteria
 - Revised Jones criteria
-
- 1. Rheumatic heart disease (Heart)
 - 2. Polyarthritis (Joints)
 - 3. Sydenham's chorea (Brain)
 - 4. Erythema marginatum (Skin)
 - 5. Subcutaneous nodules (Subcutaneous tissue)

Rheumatic fever

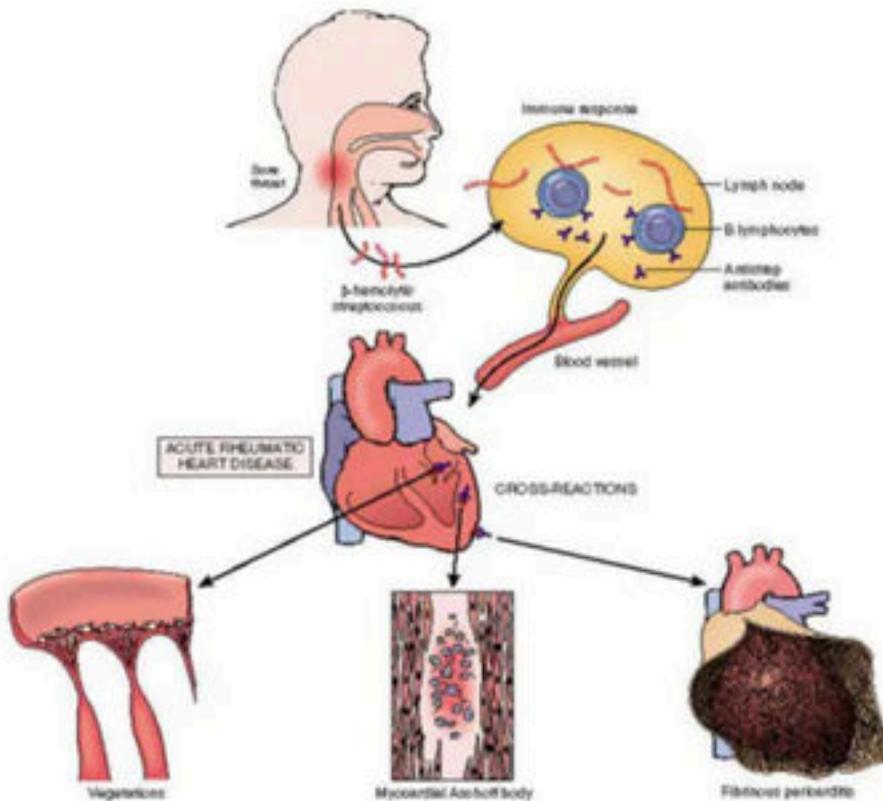
- Rheumatic fever (RF) is a **systemic, post-streptococcal, nonsuppurative inflammatory disease**, principally affecting the **heart, joints, central nervous system, skin and subcutaneous tissues**.

Rheumatic Fever

- Rheumatic fever (RF) is an acute, immunologically mediated, multisystem inflammatory disease that occurs a few weeks following an episode of **group A streptococcal pharyngitis.**
- Major involvement of systemic connective tissue, it often violate connective tissue of **heart, joint, skin, and subcutaneous and CNS**
- Key pathologic features is **Rheumatic Granuloma.**

Pathogenesis and Key Morphologic Changes of Acute Rheumatic Heart Disease

Hypersensitivity reaction induced by group A strept. (ab. Against protein M)
Cross-reaction /
Autoimmune response



Epidemiology

- Ages **5-15 yrs** are most susceptible
Rare <3 yrs
- **Girls>boys**
- Common in 3rd world countries
- Environmental factors→ **over crowding, poor sanitation, poverty,**
- Incidence more during fall ,winter & early spring
- Incubation time **2-5 weeks**

PATHOGENESIS

It is an **autoimmune response** associated with streptococcal infection, but it is **not caused by bacteria directly** effects

- Streptococcal epitopes present on the bacterial cell wall, cell membrane
- These streptococcal epitopes, are immunologically identical to human molecules on myosin, keratin, actin, laminin, vimentin and N-acetylglucosamine (ie on heart, skin,cns,subcutaneous tissue and joints) → **Molecular mimicry**

A susceptible host



Group A Streptococcus infection (pharyngitis)



Antibodies are formed



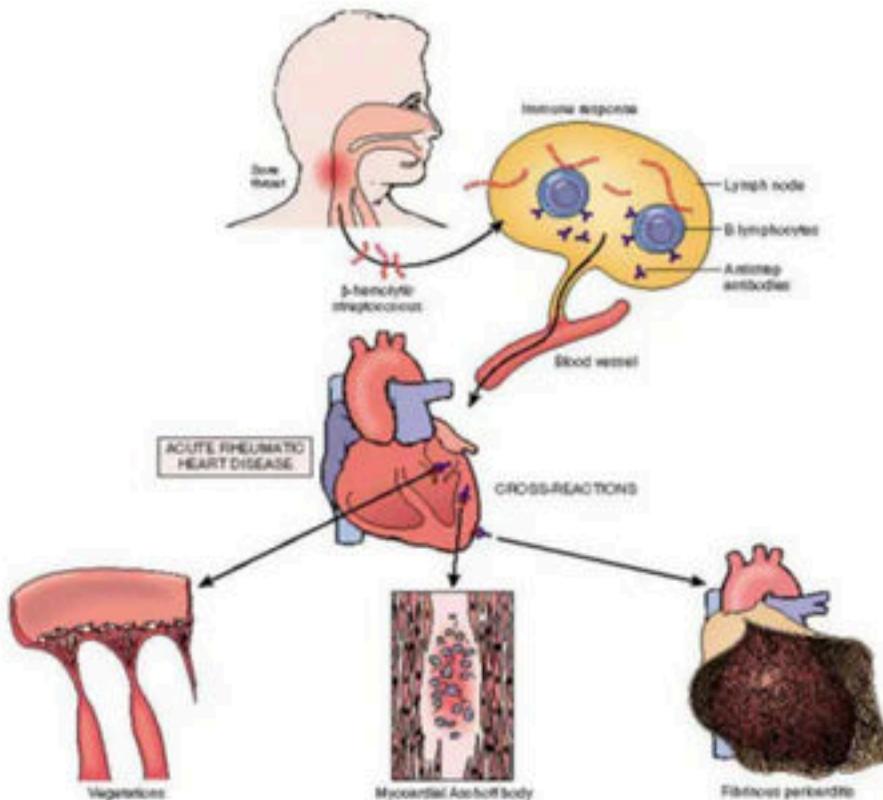
These act as autoantibodies



These antibodies cause damage to human tissues due to **cross-reactivity** between epitopes in bacteria and the host tissue
(N-acetyl glucosamine)

Pathogenesis and Key Morphologic Changes of Acute Rheumatic Heart Disease

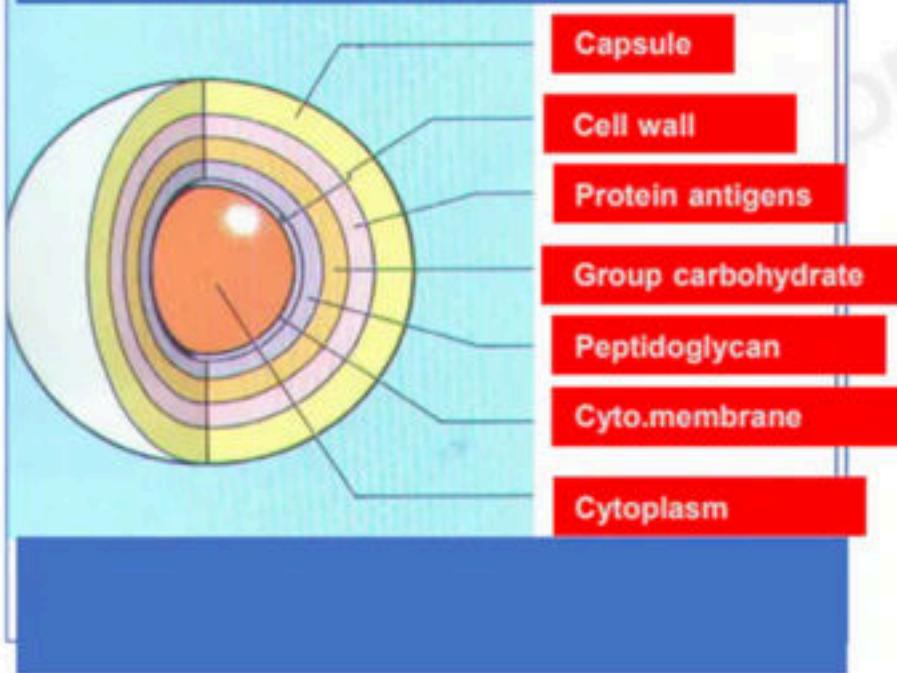
Hypersensitivity reaction induced by group A strept. (ab. Against protein M)
Cross-reaction /
Autoimmune response



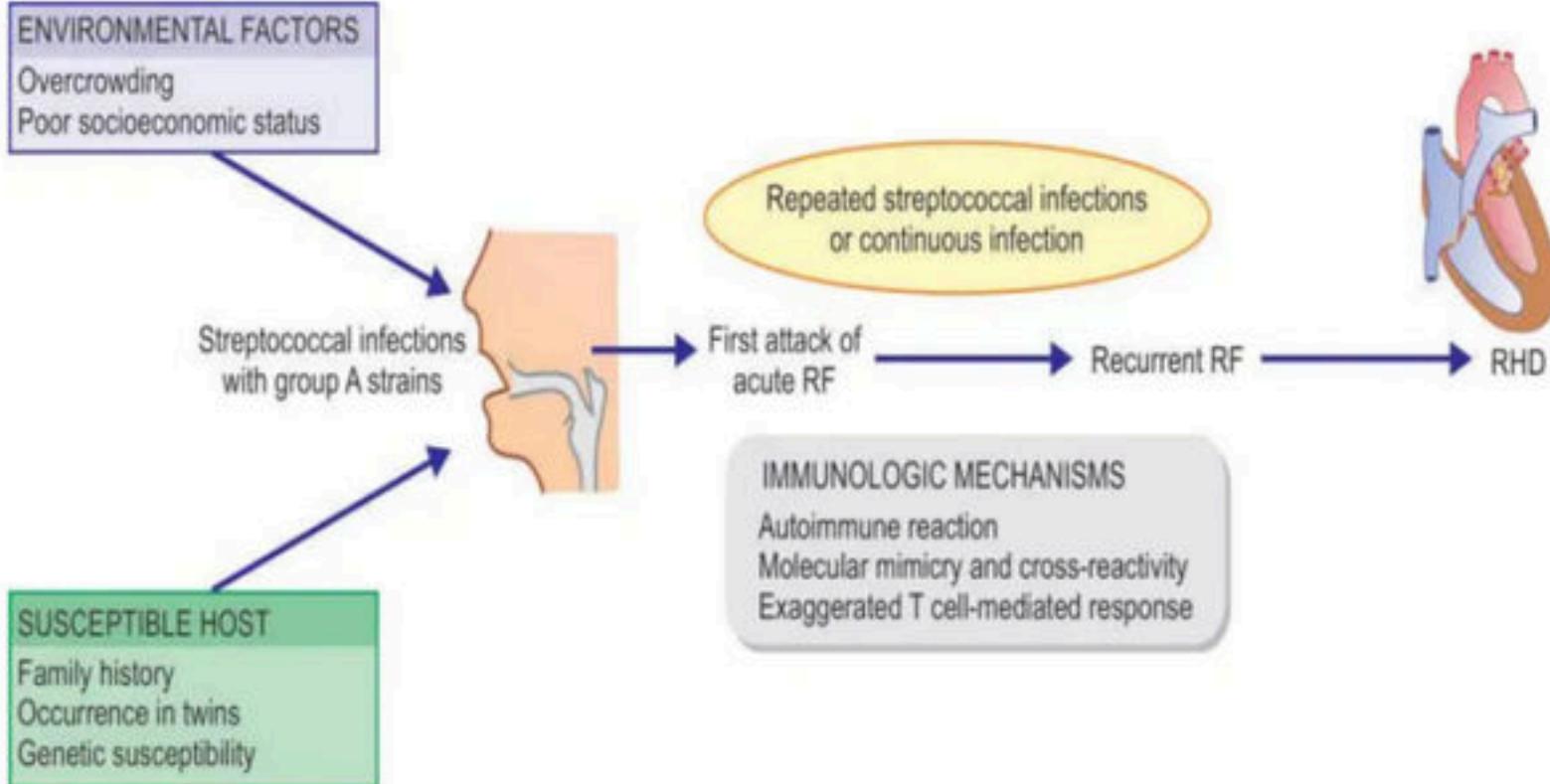
- **Molecular mimicry and cross-reactivity** between streptococcal M protein and the human endogenous molecules forms the basis of autoimmune damage to human target tissues in RHD

- Only a small proportion of patients with streptococcal pharyngeal infection develops Rheumatic fever.
- Attack rate is **less than 3%**

Diagrammatic structure of the group A beta hemolytic streptococcus



Antigen of outer protein cell wall of GABHS induces antibody response in victim which result in autoimmune damage to heart valves, subcutaneous tissue, tendons, joints & basal ganglia of brain



JONES CRITERIA

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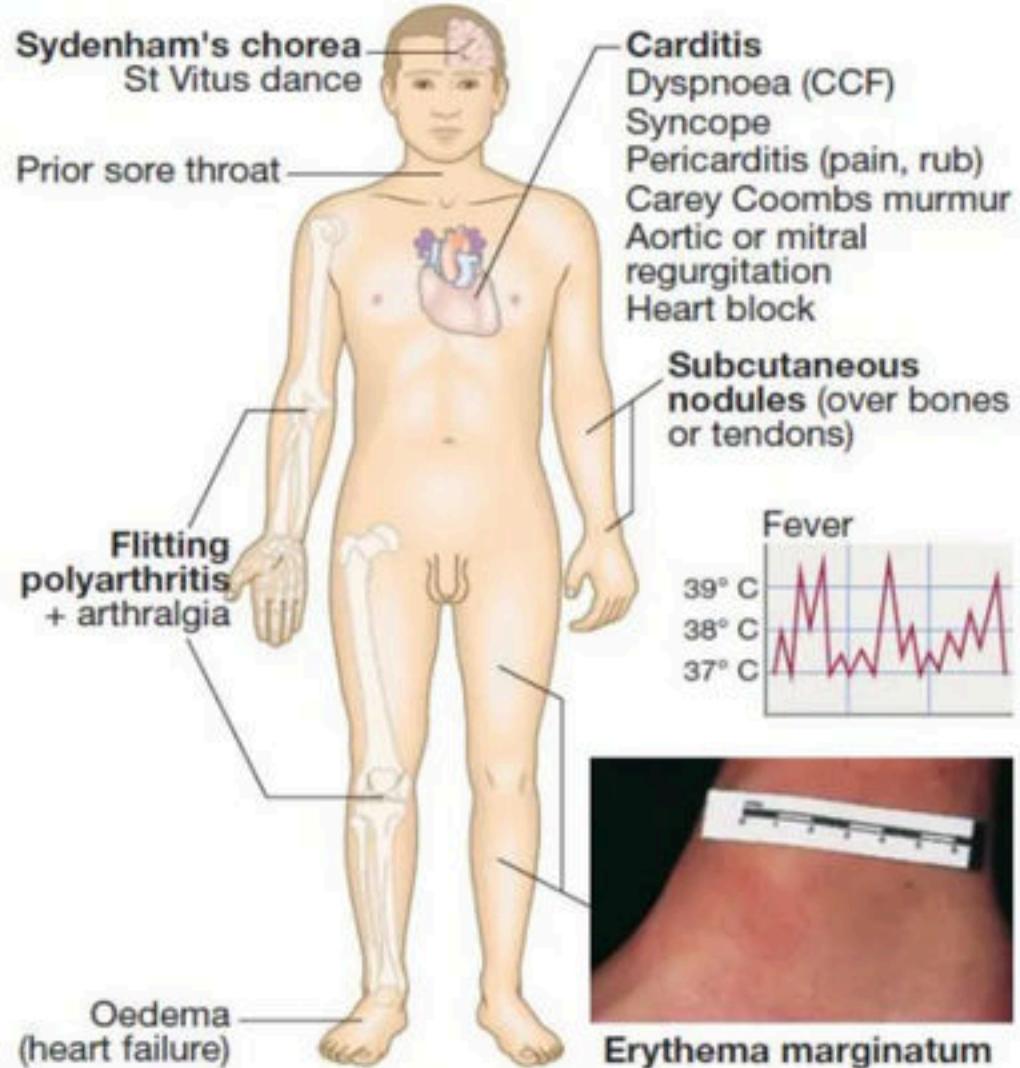
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Major criteria:

- 1. **Carditis → Rheumatic heart disease (Heart)**
- 2. **Polyarthritis (Joints)**
- 3. **Sydenham's chorea (Brain)**
- 4. **Erythema marginatum (Skin)**
- 5. **Subcutaneous nodules (Subcutaneous tissue)**



Minor criteria

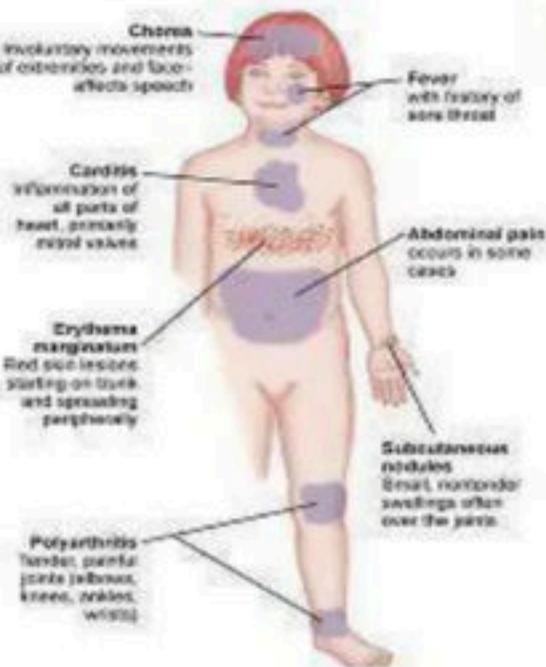
- 1. Fever
- 2. Arthralgia
- 3. Previous history of RF
- 4. Laboratory findings of elevated ESR,
raised C-reactive protein, and leucocytosis
- 5. ECG finding of prolonged PR interval.



Rheumatic Fever - Assessment

Major

- **Carditis**
- **Polyarthritis**
- **Chorea**
- **Erythema marginatum**
- **Subcutaneous nodules**



Minor

- **Arthralgia**
- **Fever**
- **Laboratory Findings:**
 - ↑ Erythrocyte sedimentation rate
 - ↑ C-reactive protein
- **Prolonged PR interval**

Jones Criteria

Presentation

Recent Strep infection

J \heartsuit NES Criteria

- J –Joints (Polyarthritis)
- \heartsuit –Carditis (Pancarditis)
- N –Nodules
- E –Erythema marginatum
- S –Sydenham's chorea

Minor criteria

- Fever, ESR, Arthralgia,
long PR interval



Jones Criteria

- **2 MAJOR manifestations**
- OR
- **1 MAJOR and 2 MINOR manifestations**

Revised Jones Criteria 2015

MAJOR Manifestations	MINOR Manifestations	GAS	Infection
Carditis	Fever	GAS on Throat swab (Culture)	
Arthritis	Arthralgia	Anti-streptolysin O titre (ASOT)	
Sydenham's Chorea	↑ PR interval on ECG	Anti-deoxyribonuclease B (Anti-DNase B)	
Erythema marginatum	ESR \geq 30mm/hr or CRP \geq 30mg/L		
Subcutaneous nodules			

MAJOR Criteria - signs and symptoms more often associated with ARF

MINOR Criteria - signs and symptoms that help support the diagnosis

Evidence of recent **GAS** Infection is required

World Health Criteria for the diagnosis of Rheumatic fever and rheumatic heart disease

Major manifestations

- *Carditis*
- « *Polyarthritis*
- *Chorea*
- *Erythema marginatum*
- ® *Subcutaneous nodules*

Minor manifestations

- ® Clinical: fever, polyarthralgia
- Laboratory: elevated erythrocyte sedimentation rate or leukocyte count
- Electrocardiogram: prolonged P-R interval

@1992 Revised Jones criteria do not include elevated leucocyte count as a laboratory minor manifestation (but do include elevated C-reactive protein)

Supporting evidence of a preceding streptococcal infection within the last 45 days

- Elevated or rising anti-streptolysin O or other streptococcal antibody, or
- A positive throat culture, or
- Rapid antigen test for group A streptococcus, or
- « Recent scarlet fever

@1992 Revised Jones criteria do not include recent scarlet fever as supporting evidence of a recent streptococcal infection

Revised Jones Criteria

- **2 MAJOR manifestations**
- OR
- **1 MAJOR and 2 MINOR manifestations**
- +
- **Evidence of preceding Group A streptococcal infection (within 3 weeks before ARF symptoms)**

POLLS 6

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The probable interval between throat infection and onset of rheumatic fever is -

- a) 2-4 hours
- b) 2-4 days
- c) 2-4 weeks
- d) 2-4 months



C



med[LIVE]

What is the mechanism of rheumatic fever -

- a) Cross reactivity with endogenous antigen
- b) Innocent bystander effect
- c) Due to toxin secretion by streptococci
- d) Release of pyrogenic cytokines



A



Which is not major criteria for diagnosis of rheumatic fever ?

- a) Carditis
 - b) Subcutaneous nodules
 - c) Increased ASLO
 - d) Arthritis
-
-

C



med[LIVE]

The % of coincidence between sore throat and acute rheumatic fever is -

- a) 3%
- b) 5%
- c) 7%
- d) 9%



A



Which is a minor criteria for diagnosis of RF according to modified Jones criteria?

- A. ASO titre
- B. Past History of Rheumatic Fever
- C. Fever
- D. Subcutaneous nodules



A



Which of the following is not included in Jone's Major Criteria

- A. Pancarditis
- B. Chorea
- C. Subcutaneous nodule
- D. High ESR**



D



All of the following are major criteria in the diagnosis of Acute Rheumatic Fever, Except:

- A. Migratory Polyarthralgia
- B. Subcutaneous Nodules
- C. Chorea
- D. Carditis



A



Rheumatic heart disease

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OVERVIEW

- **Introduction**

- 1. Rheumatic endocarditis**

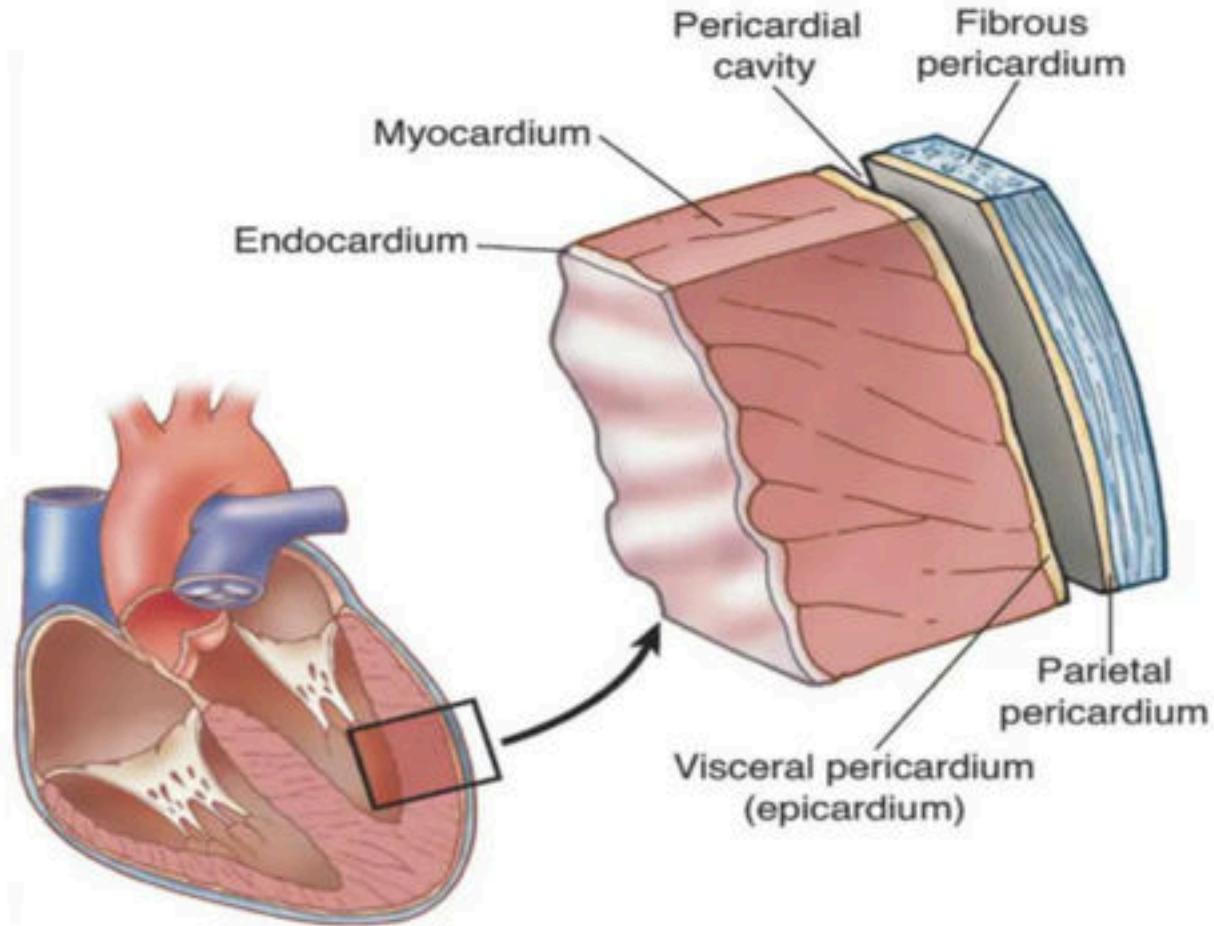
- a) Rheumatic valvulitis**
- b) Rheumatic mural endocarditis**

- 2. Rheumatic myocarditis**

- 3. Rheumatic pericarditis**

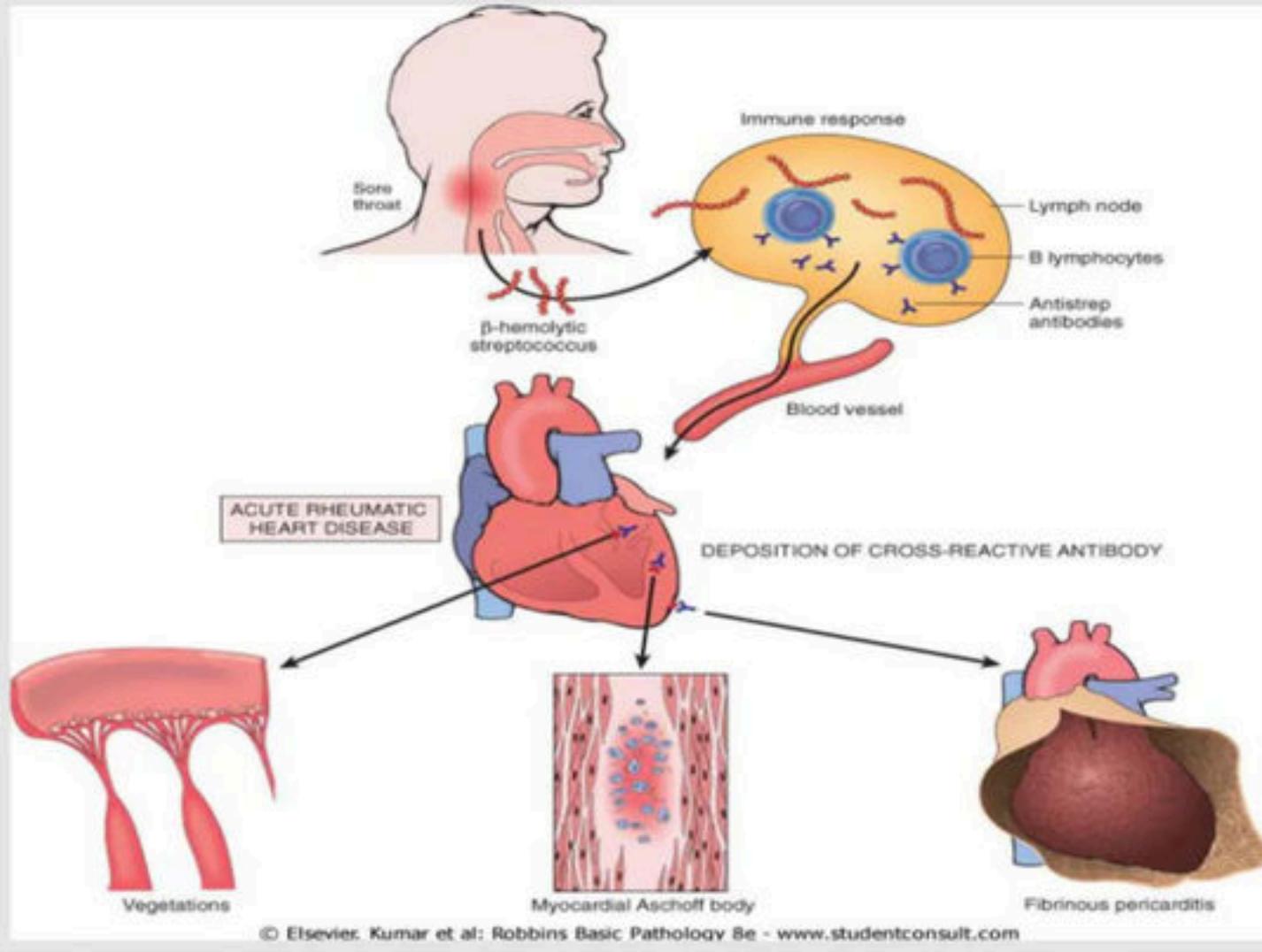
Introduction

- The chronic stage of RF involves **all the layers** of the heart (pancarditis) causing major cardiac sequelae referred to as rheumatic heart disease (RHD).
- The cardiac lesions of RF in the form of **pancarditis**



- Rheumatic **endocarditis**
- Rheumatic **myocarditis**
- Rheumatic **pericarditis**

60% to 80% children associated with
pancarditis



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OVERVIEW

- **Introduction**

1. **Rheumatic endocarditis**

- a) **Rheumatic valvulitis**
- b) **Rheumatic mural endocarditis**

2. **Rheumatic myocarditis**

3. **Rheumatic pericarditis**

RHEUMATIC PANCARDITIS

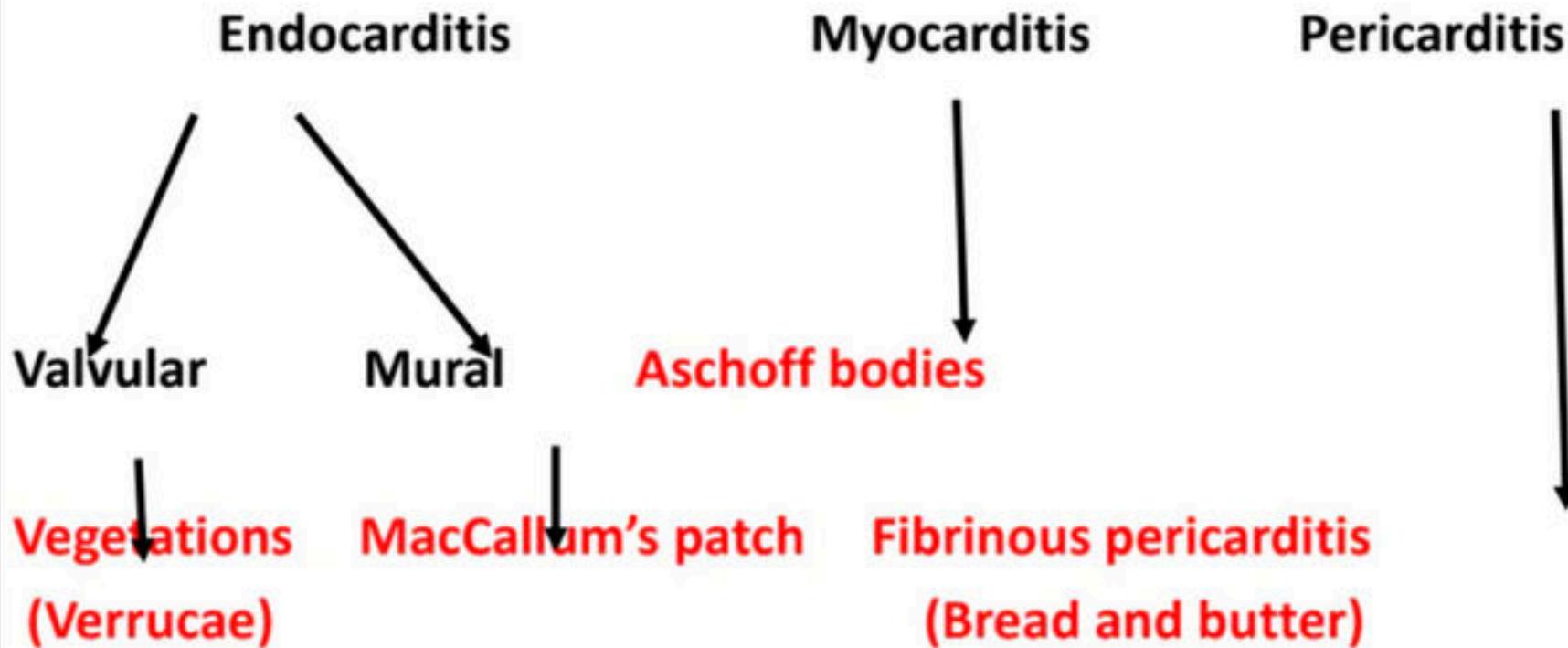


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PANCARDITIS



RHEUMATIC ENDOCARDITIS

- Most common manifestation of caritas
- 2 forms →
 1. Rheumatic valvulitis
 2. Rheumatic mural endocarditis

RHEUMATIC VALVULITIS

- The valves show **thickening and loss of translucency**
- This is followed by the formation of characteristic **vegetations or verrucae**

1. Gross→

- a) Vegetations or Verrucae**
- b) Valves**
- c) Location**
- d) Deformity**

2. Microscopy

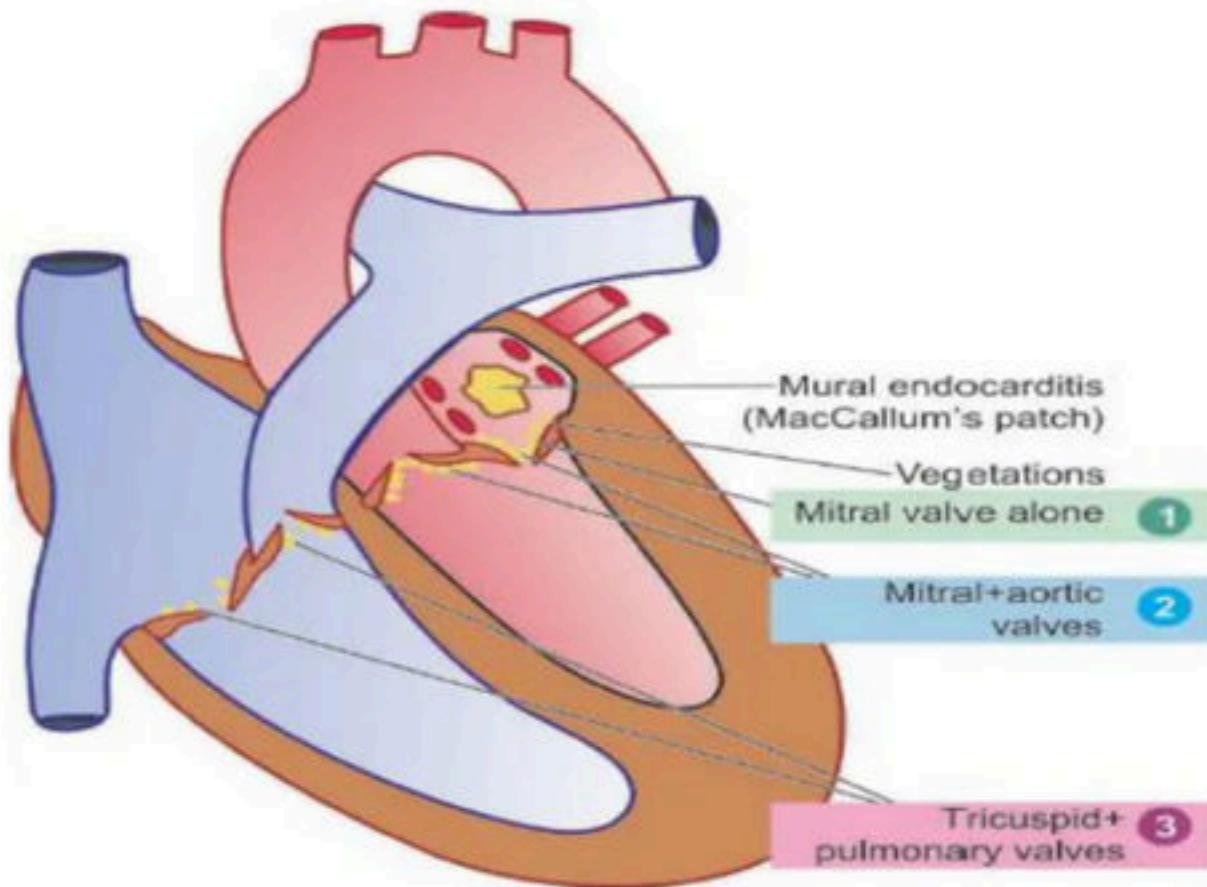
Gross

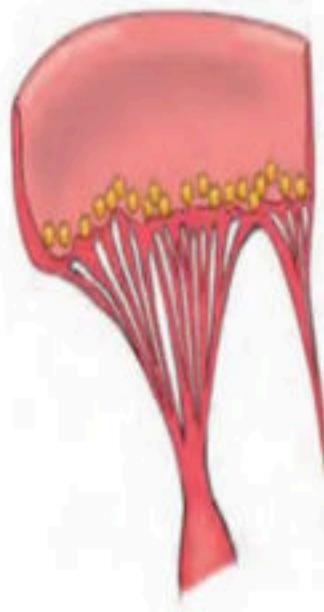
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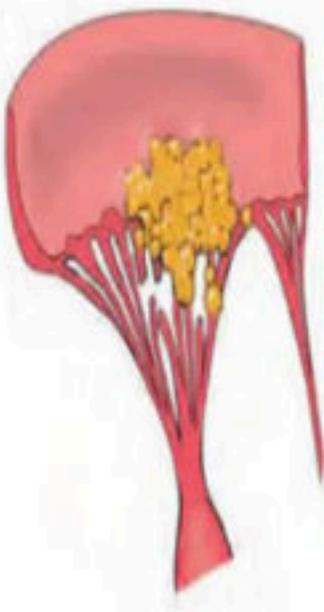
Vegetations or Verrucae

- Small (1 to 3 mm in diameter)
- Multiple
- warty
- Soft and firm
- Sterile, bland

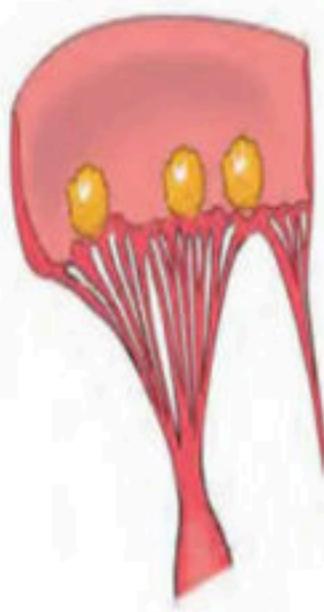




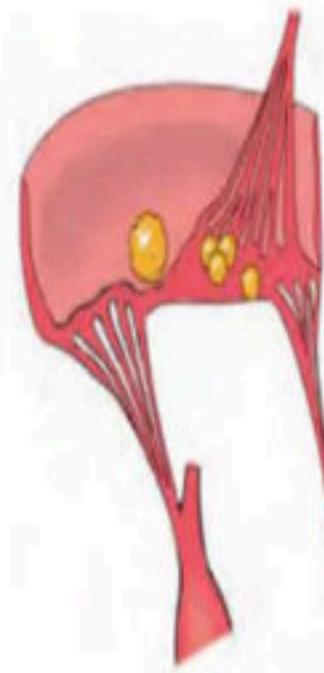
RHD



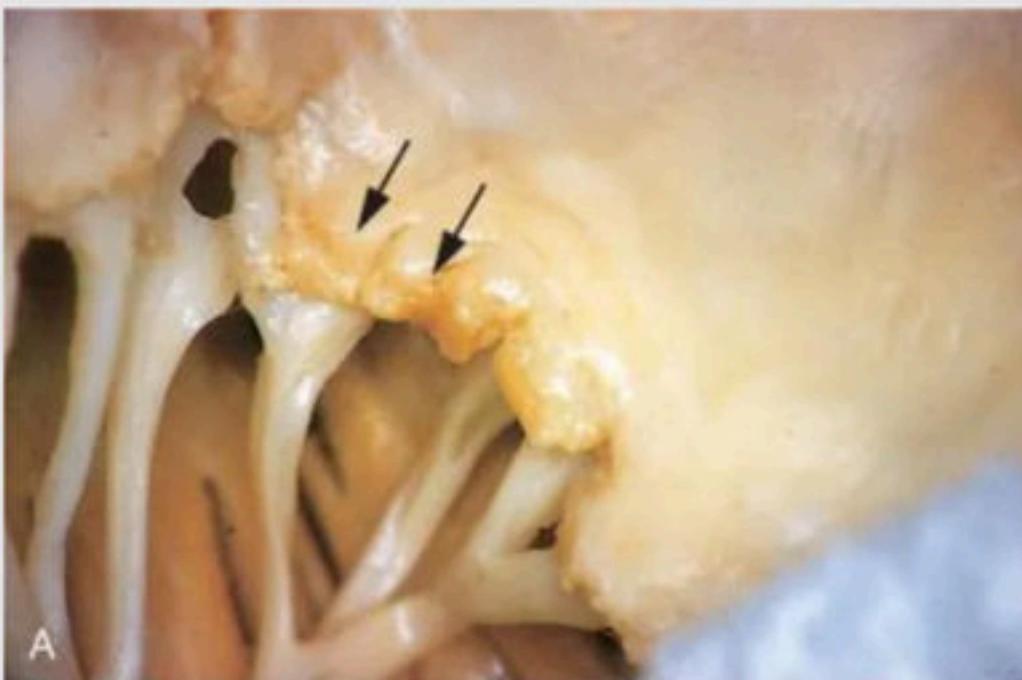
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NBTE

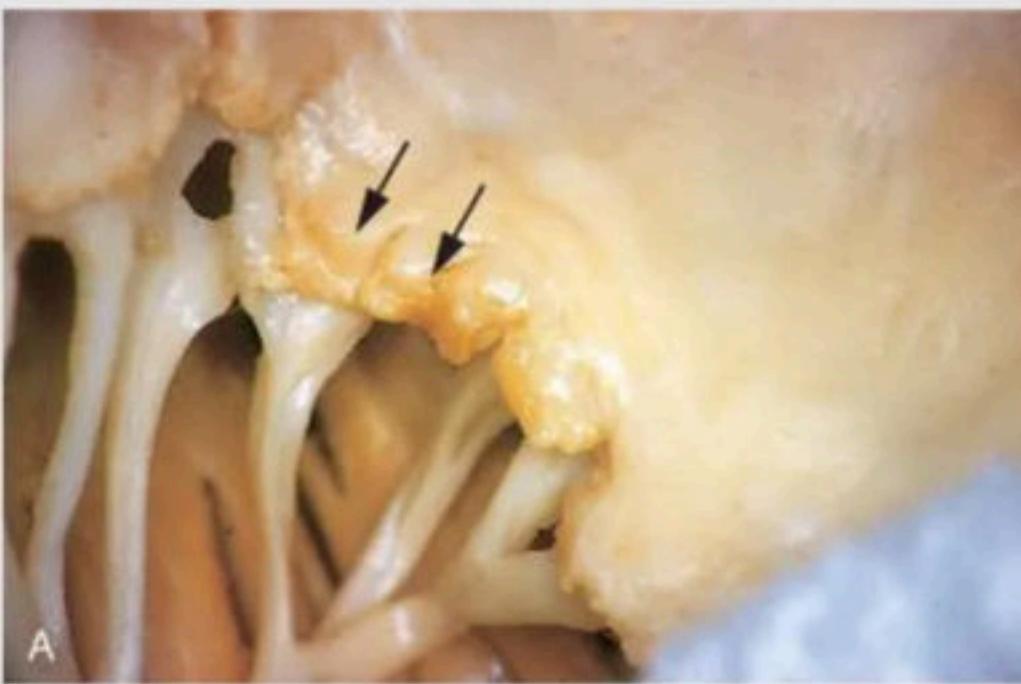


LSE



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Small vegetations (verruca) are visible along the line of closure of the mitral valve leaflet (*arrows*).



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Small vegetations (verruca) are visible along the line of closure of the mitral valve leaflet (*arrows*).

1. Gross→

- a) Vegetations or Verrucae**
- b) Valves**
- c) Location**
- d) Deformity**

2. Microscopy

Valves

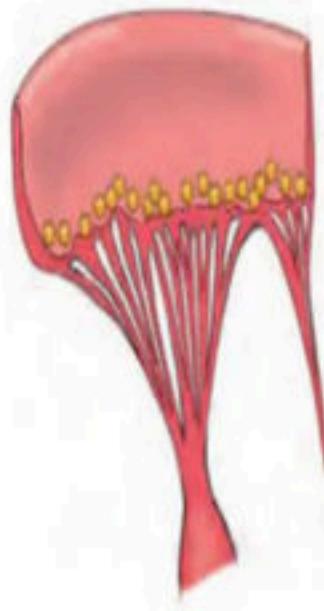
- **Mitral alone = 37% cases.**
- Mitral + aortic = 27% cases.
- Mitral + aortic + tricuspid = 22% cases.
- Mitral + tricuspid = 11% cases.
- Aortic alone = 2%.
- Mitral + aortic + tricuspid + pulmonary = less than 1%

REMEMBER

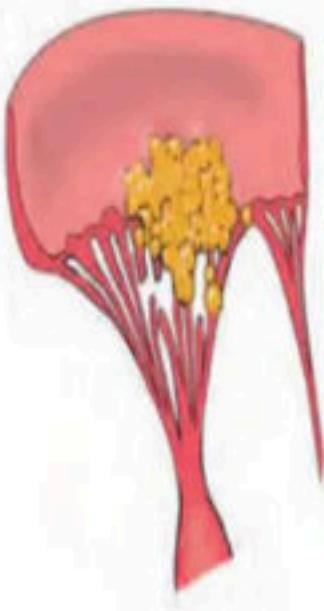
In Rheumatic fever

- Commonest valve to involve → Mitral valve
- 2nd commonest valve to involve → Aortic valve
- ***Least frequently involved valve*** → ***Pulmonary valve***
- Most common valvular deformity produced → Mitral regurgitation

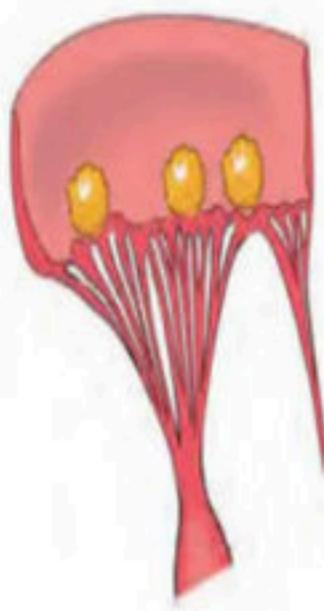
- **Location** → chiefly **along the line of closure of the leaflets and cusps.**
- **Continuous** so that the free margin of the cusps or leaflets appears as a rough and irregular ridge



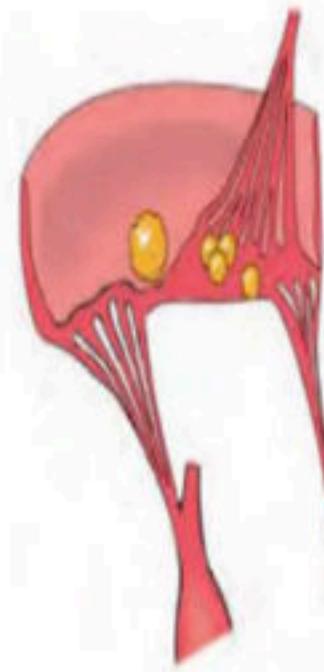
RHD



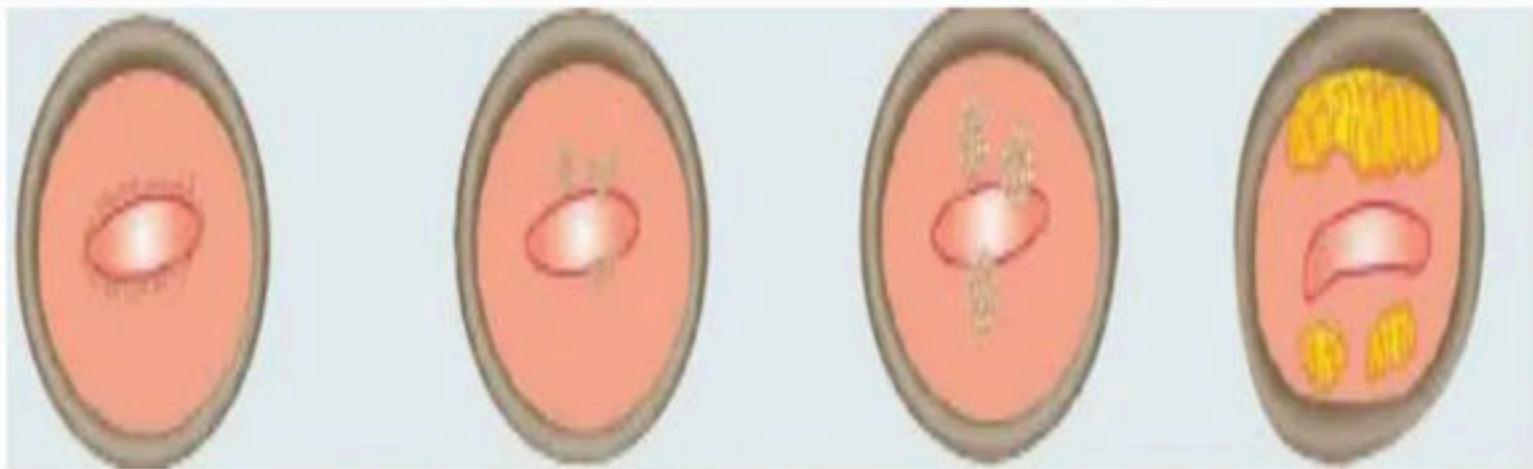
IE



NBTE



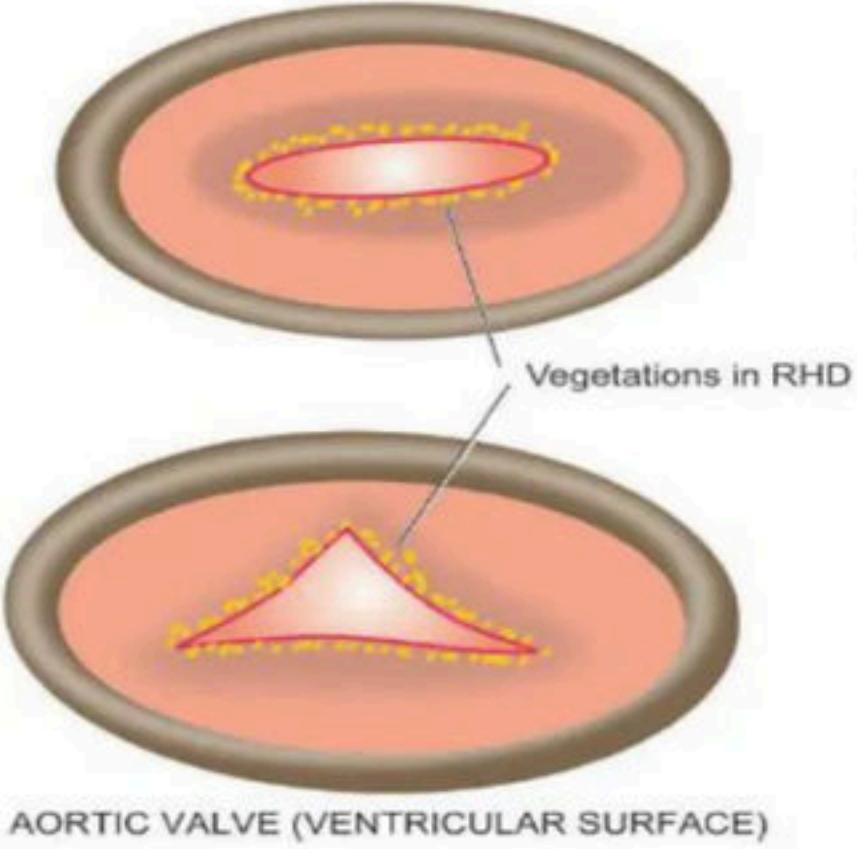
LSE



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FEATURE	RHEUMATIC	LIBMAN-SACKS	NON-BACTERIAL THROMBOTIC	BACTERIAL (INFECTIVE)
1. Valves commonly affected	Mitral alone; mitral and aortic combined	Mitral, tricuspid	Mainly mitral; less often aortic and tricuspid	Mitral; aortic; combined mitral and aortic
2. Location on valve cusps or leaflets	Occur along the line of closure, atrial surface of atrioventricular valves and ventricular surface of semilunar valves	Occur on both surfaces of valve leaflets or cusps, in the valve pockets	Occur along the line of closure	SABE more often on diseased valves; ABE on previously normal valves; location same as in RHD
3. Macroscopy				
	Small, multiple, warty, grey brown, translucent, firmly attached, generally produce permanent valvular deformity	Medium-sized, multiple, generally do not produce significant valvular deformity	Small but larger than those of rheumatic, single or multiple, brownish, firm, but more friable than those of rheumatic	Often large, grey-tawny to greenish, irregular, single or multiple, typically friable
4. Microscopy	Composed of fibrin with superimposed platelet thrombi and no bacteria. Adjacent and underlying endocardium shows oedema, proliferation of capillaries, mononuclear inflammatory infiltrate and occasional Aschoff bodies.	Composed of fibrinoid material with superimposed fibrin and platelet thrombi and no bacteria. The underlying endocardium shows fibrinoid necrosis, proliferation of capillaries and acute and chronic inflammatory infiltrate including the haematoxylin bodies of Gross.	Composed of degenerated valvular tissue, fibrin-platelets thrombi and no bacteria. The underlying valve shows swelling of collagen, fibrinoid change, proliferation of capillaries but no significant inflammatory cell infiltrate.	Composed of outer eosinophilic zone of fibrin and platelets, covering colonies of bacteria and deeper zone of non-specific acute and chronic inflammatory cells. The underlying endocardium may show abscesses in ABE and inflammatory granulation tissue in the SABE.

MITRAL VALVE (atrial surface)



- **Deformity** → Chronic healed mitral valve in RHD is characteristically '**fish mouth**' or '**button hole**' stenosis.



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Mitral stenosis with diffuse fibrous thickening and distortion of the valve leaflets, commissural fusion (*arrows*), and thickening and shortening of the chordae tendinae.



Advanced: vegetations organization, recurrent organization cause
chronic heart valve disease (valvular stenosis and / or valvular
insufficiency)

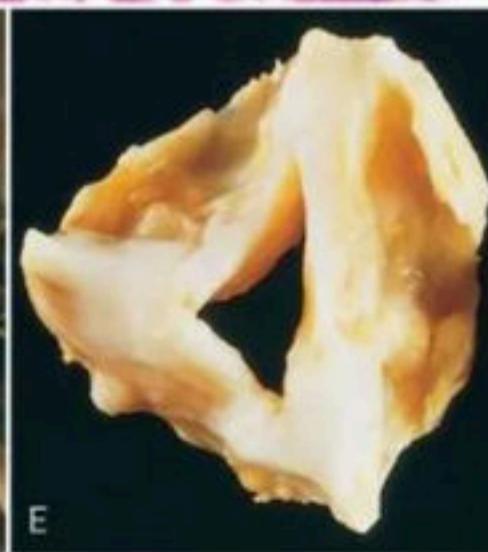
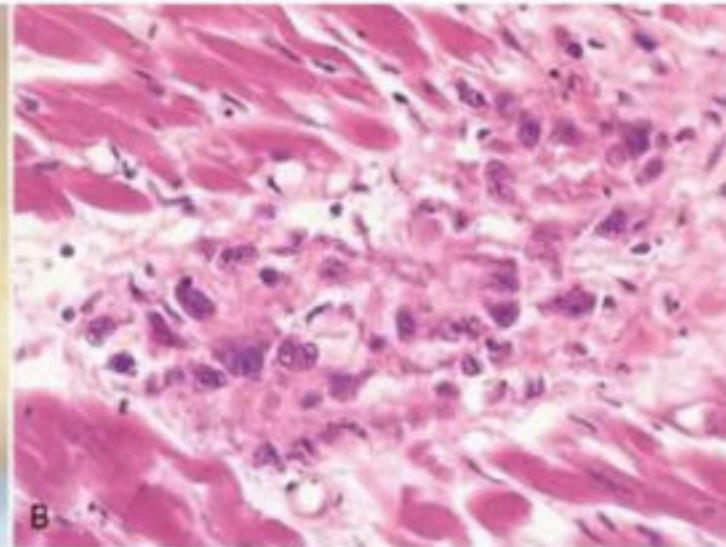


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Mitral stenosis with diffuse fibrous thickening and distortion of the valve leaflets, commissural fusion (*arrows*), and thickening and shortening of the chordae tendinae.



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REMEMBER

- Most commonly involved valve in rheumatic carditis is mitral valve.
- Most common lesion in acute rheumatic carditis is mitral regurgitation
- Most common lesion in chronic rheumatic carditis is mitral stenosis
- Overall most common lesion is mitral regurgitation.
- Least frequently involved valve is Pulmonary valve

1. Gross→

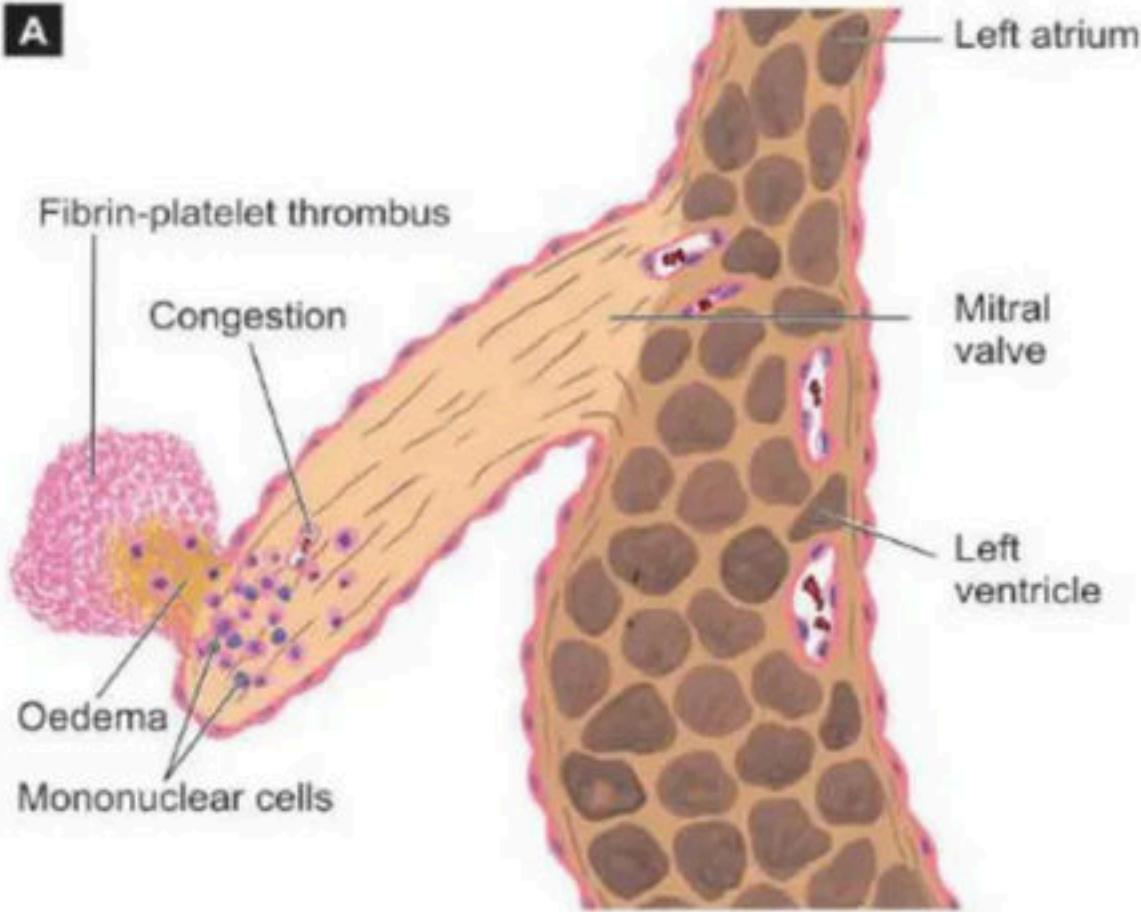
- a) Vegetations or Verrucae**
- b) Valves**
- c) Location**
- d) Deformity**

2. Microscopy

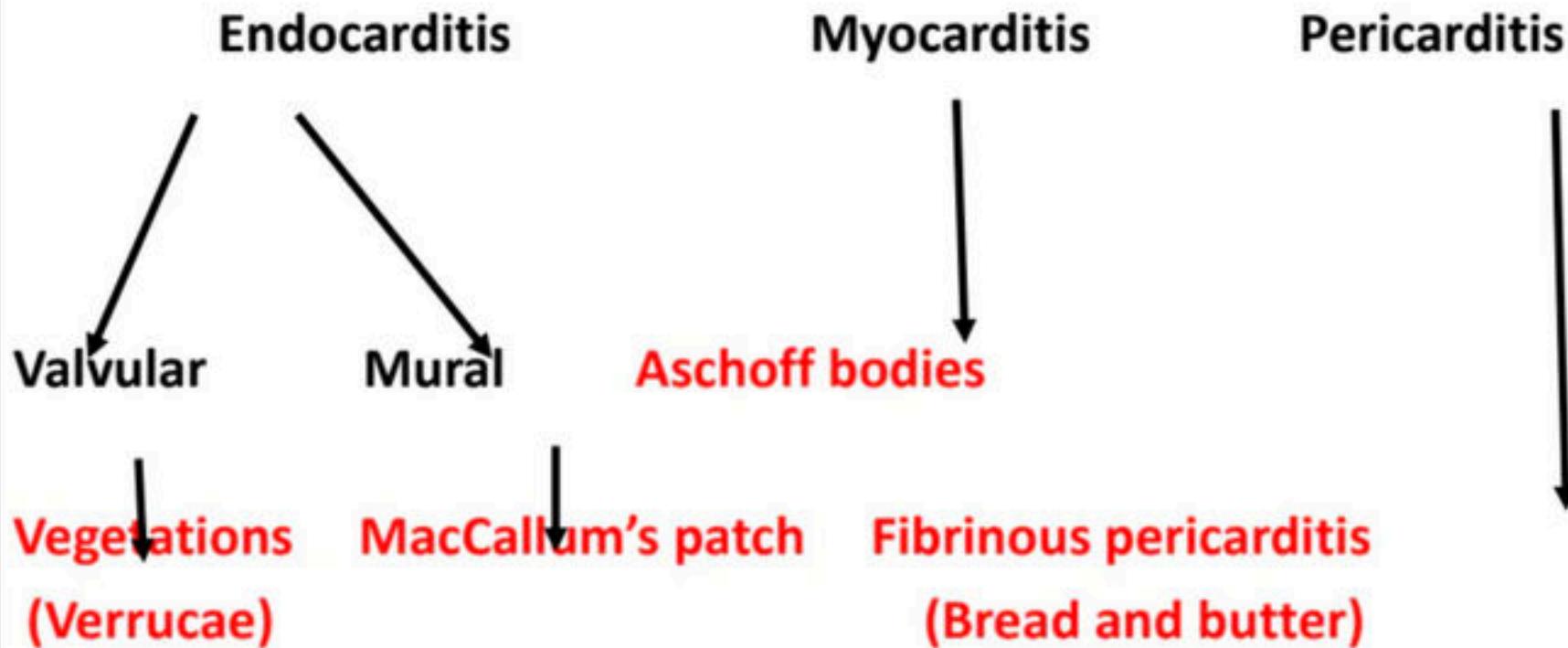
Microscopically

- **Vegetations** → Composed of **fibrin** with superimposed **platelet thrombi** and no **bacteria** (sterile)
- **Adjacent and underlying endocardium** → shows **oedema**, **proliferation of capillaries**, **mononuclear inflammatory infiltrate** and occasional **Aschoff bodies**

A

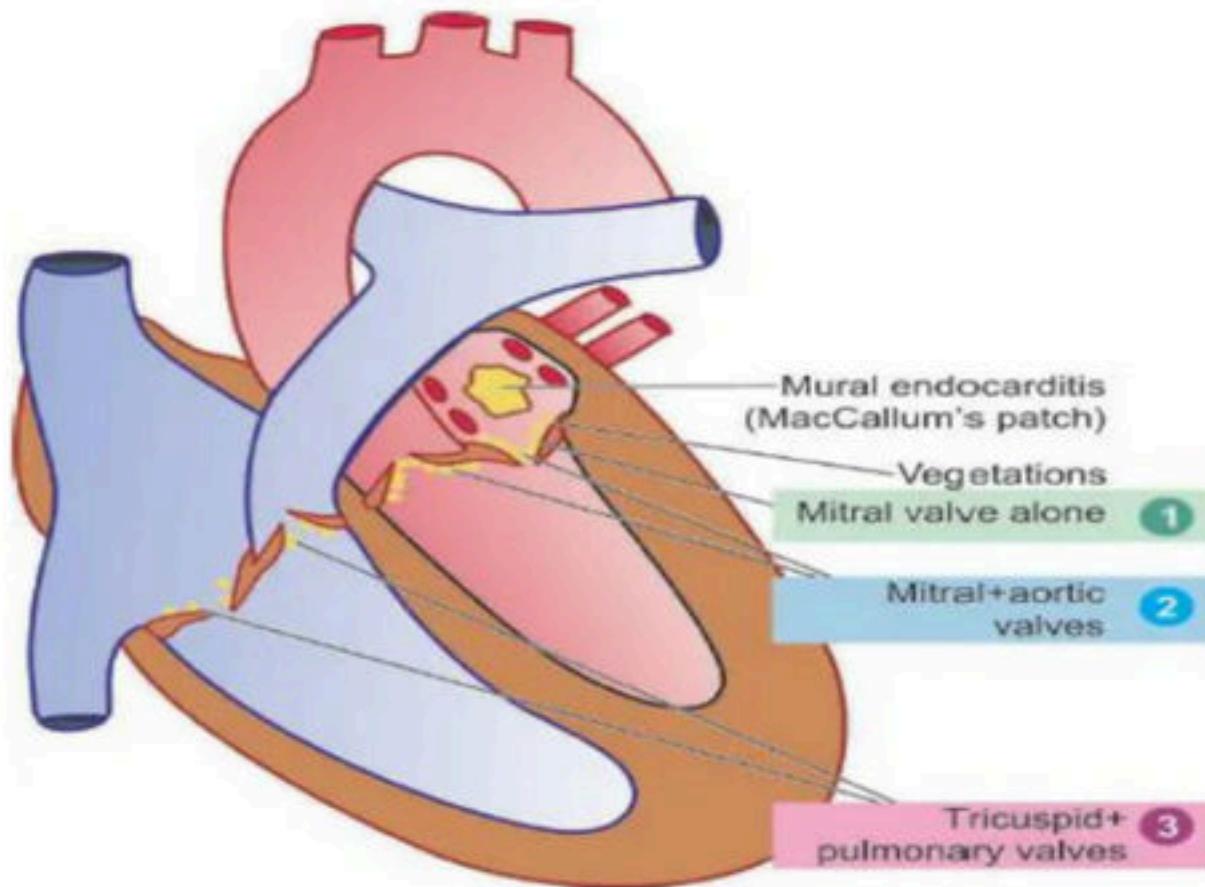


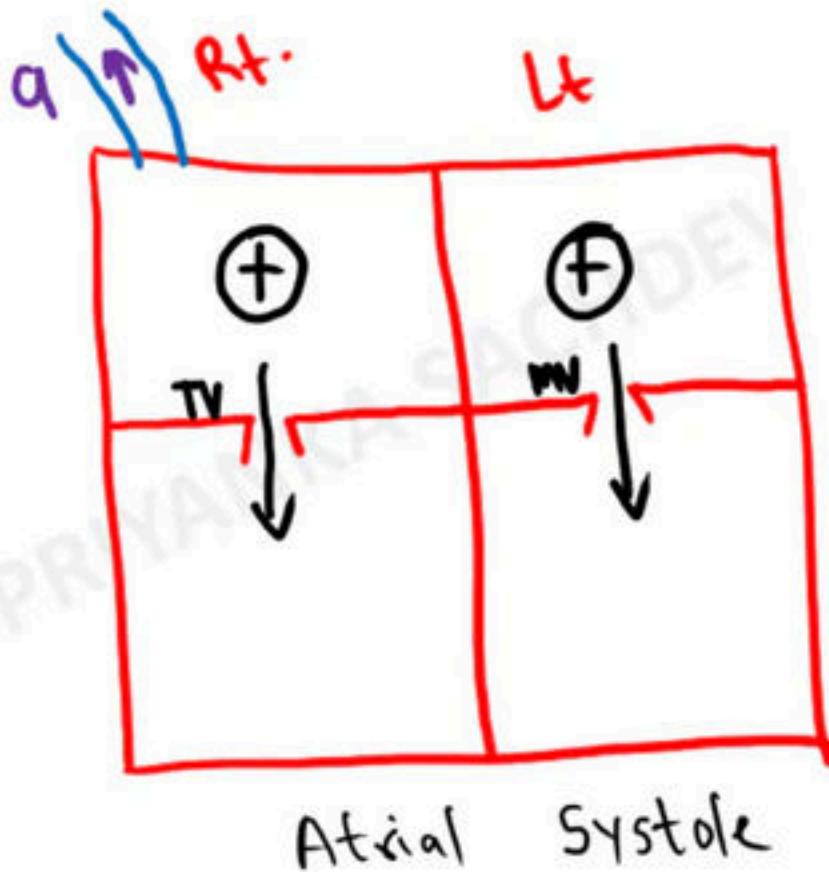
PANCARDITIS



RHEUMATIC MURAL ENDOCARDITIS

- The lesions are seen most commonly as **MacCallum's patch**
- It is **endocardial surface in the posterior wall of the left atrium just above the posterior leaflet of the mitral valve**
- MacCallum's patch appears as a map-like area of thickened, roughened and wrinkled part of the endocardium





OVERVIEW

- **Introduction**

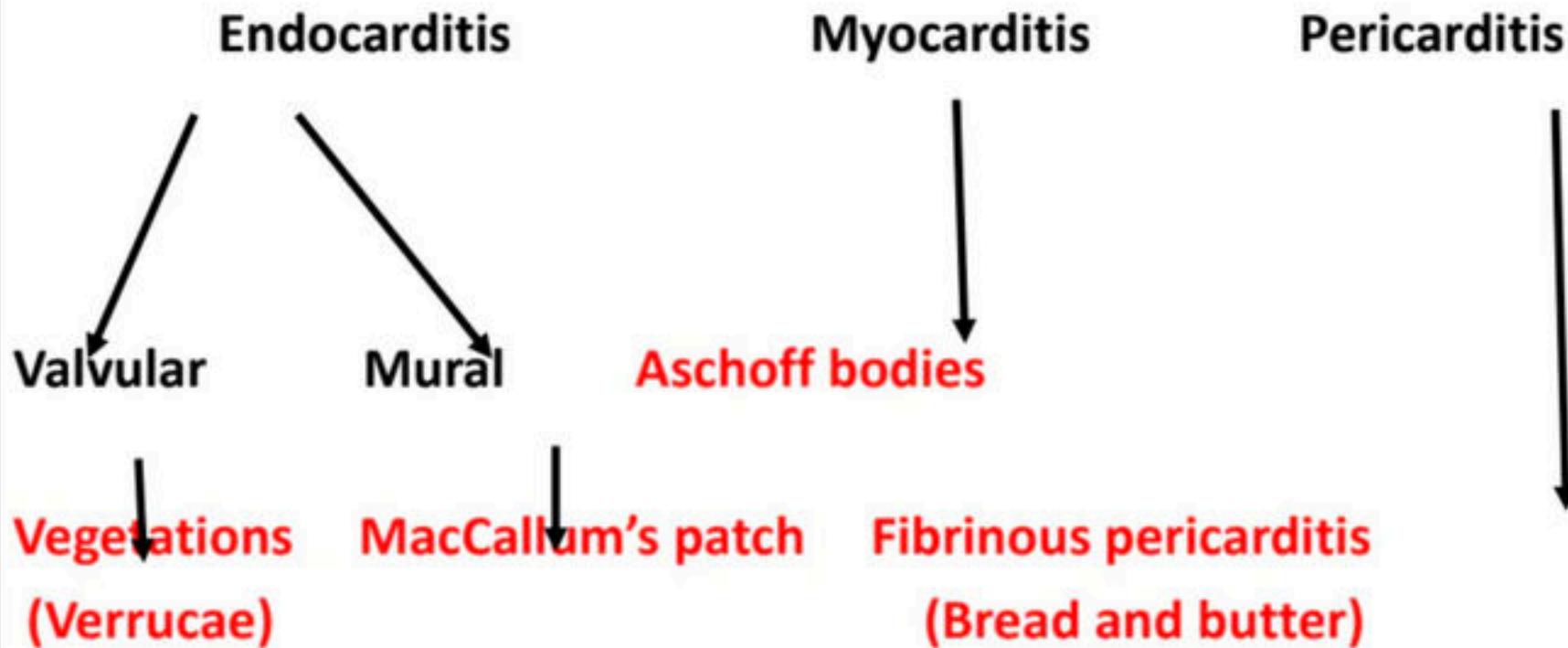
1. **Rheumatic endocarditis**

- a) **Rheumatic valvulitis**
- b) **Rheumatic mural endocarditis**

2. **Rheumatic myocarditis**

3. **Rheumatic pericarditis**

PANCARDITIS



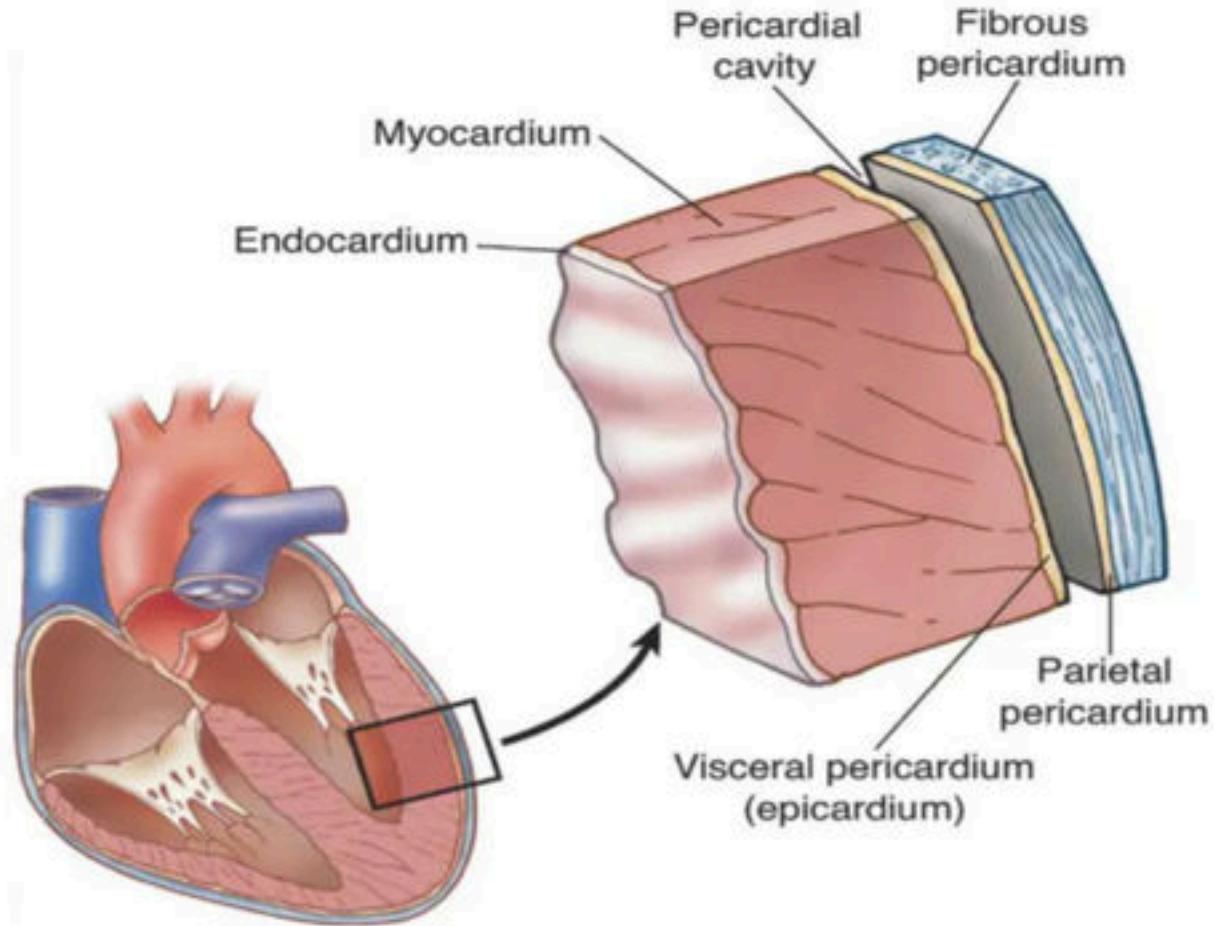
RHEUMATIC MYOCARDITIS

- **Grossly**, in the early (acute) stage, the myocardium, especially of the left ventricle, is **soft and flabby**.

Microscopically

- The most characteristic feature is the presence of distinctive **Aschoff bodies**.
- **Aschoff bodies are pathognomonic of RHD**

- Found in **all three layers of heart**, but mostly seen in **myocardium**
- Scattered throughout the interstitial tissue of the myocardium and are most frequent in the **interventricular septum, left ventricle and left atrium**



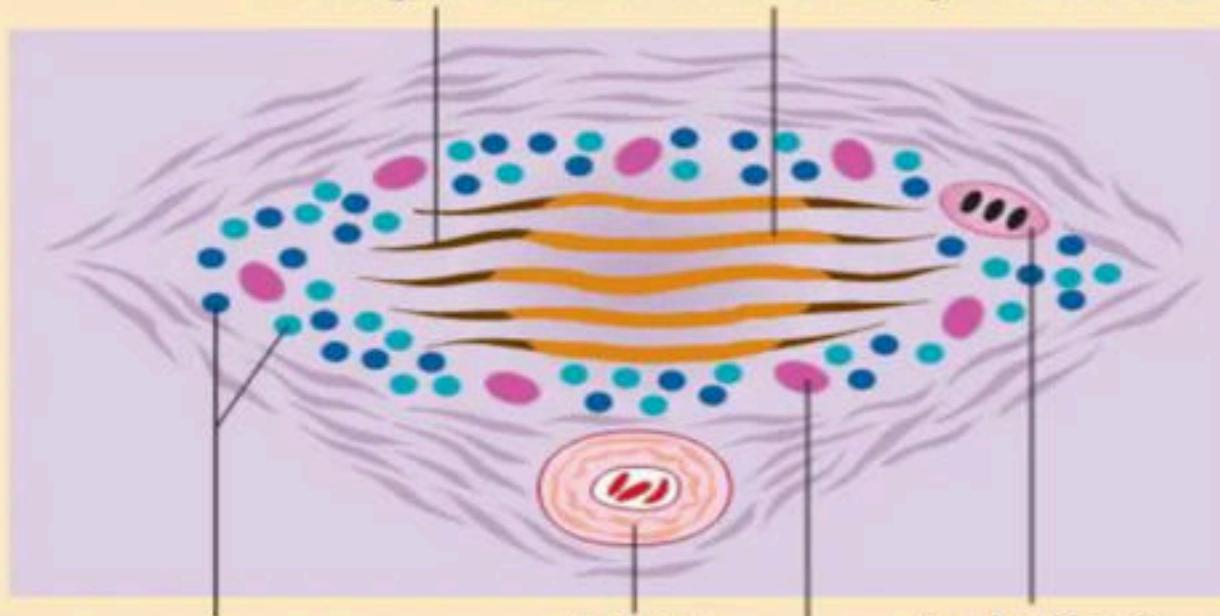
Aschoff bodies→

Granulomas with →

1. Central fibrinoid necrosis
 2. Surrounded by palisade of Anitschkow cells and multinucleate Aschoff cells.
 3. There is infiltration by lymphocytes, plasma cells and fibroblast
-
- Neutrophils (polymorphonuclear cells) are characteristically absent

A**Rheumatic granuloma**

Collagen fibers Fibrinoid collagen necrosis



Plasma cells
Lymphocytes

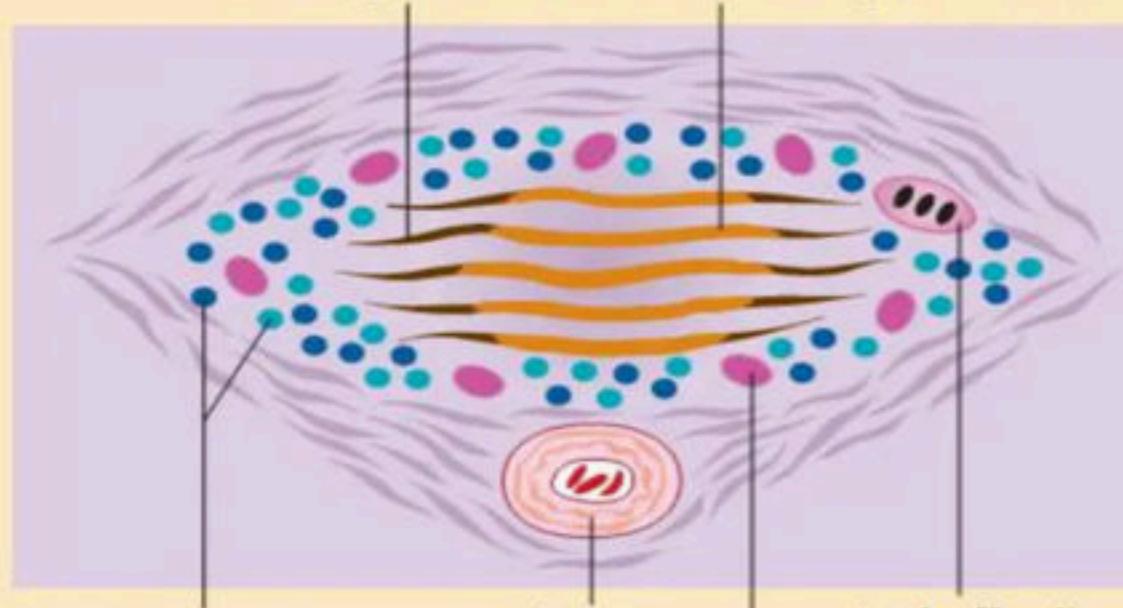
Artery

Aschoff cell
Anitchkov cell
(histiocyte)

- **Aschoff cells** → Some of the larger **macrophages** become multinucleated to form **inflammatory giant cells**
- **Anitschkow cells** → Modified **macrophages** having abundant cytoplasm and central round-to-ovoid nuclei in which the chromatin is disposed in a central, slender, wavy ribbon (hence the designation "**caterpillar cells/ Owl 's eye cells**).

A Rheumatic granuloma

Collagen fibers Fibrinoid collagen necrosis

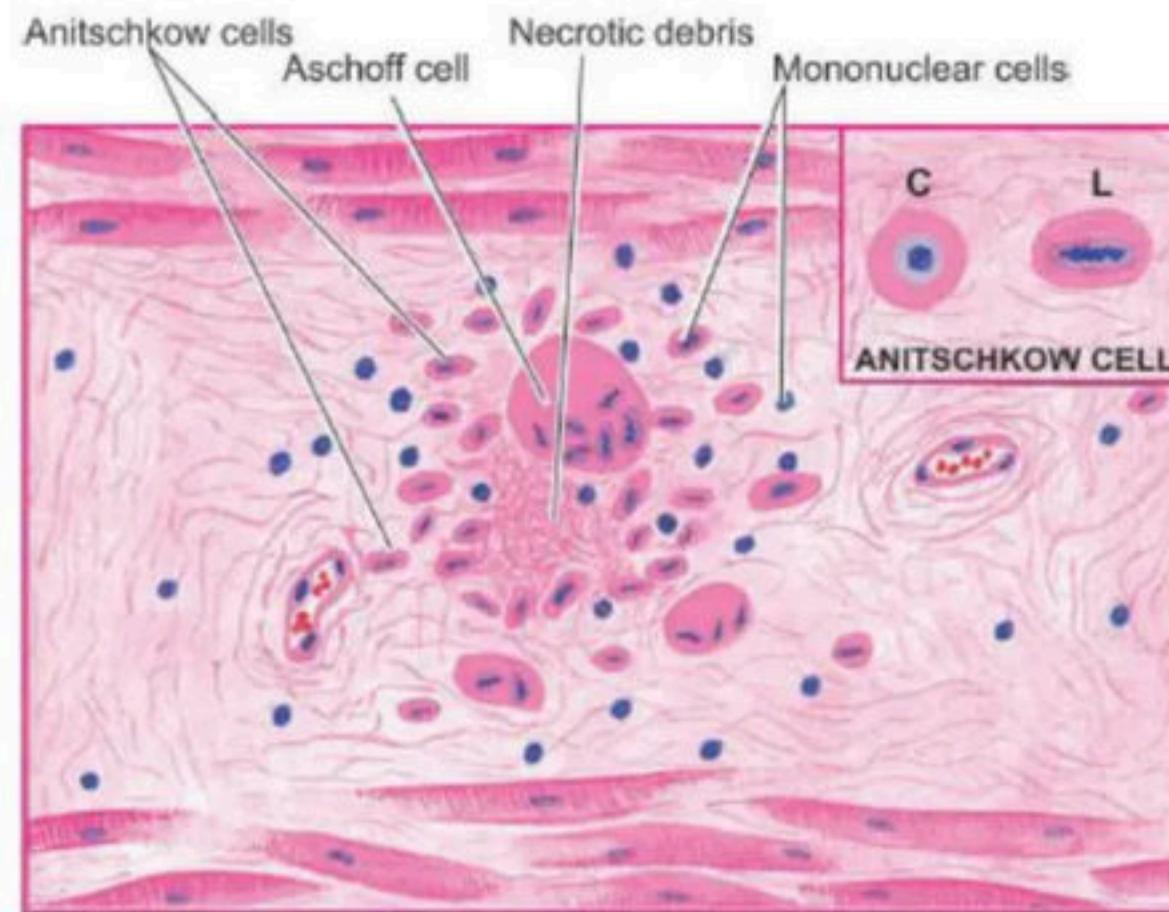


Plasma cells
Lymphocytes

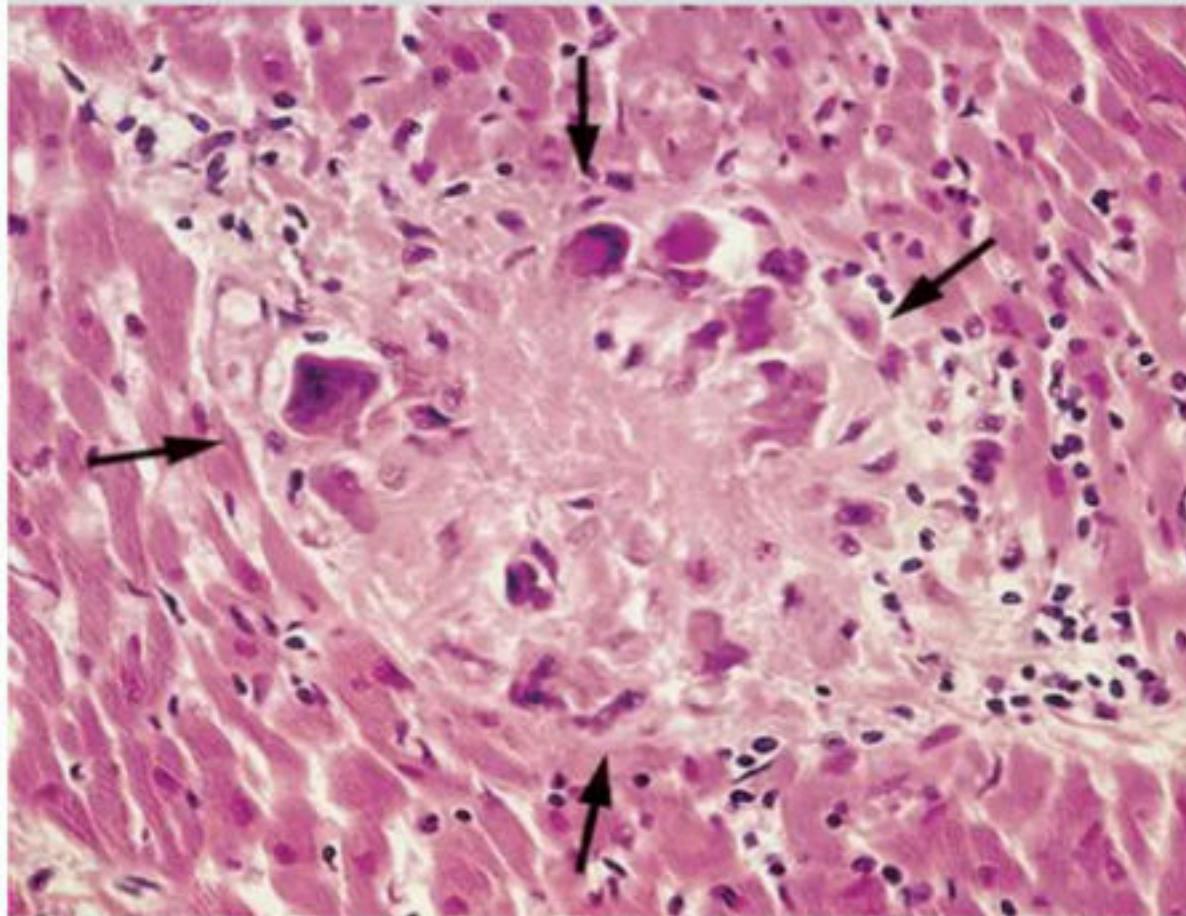
Artery

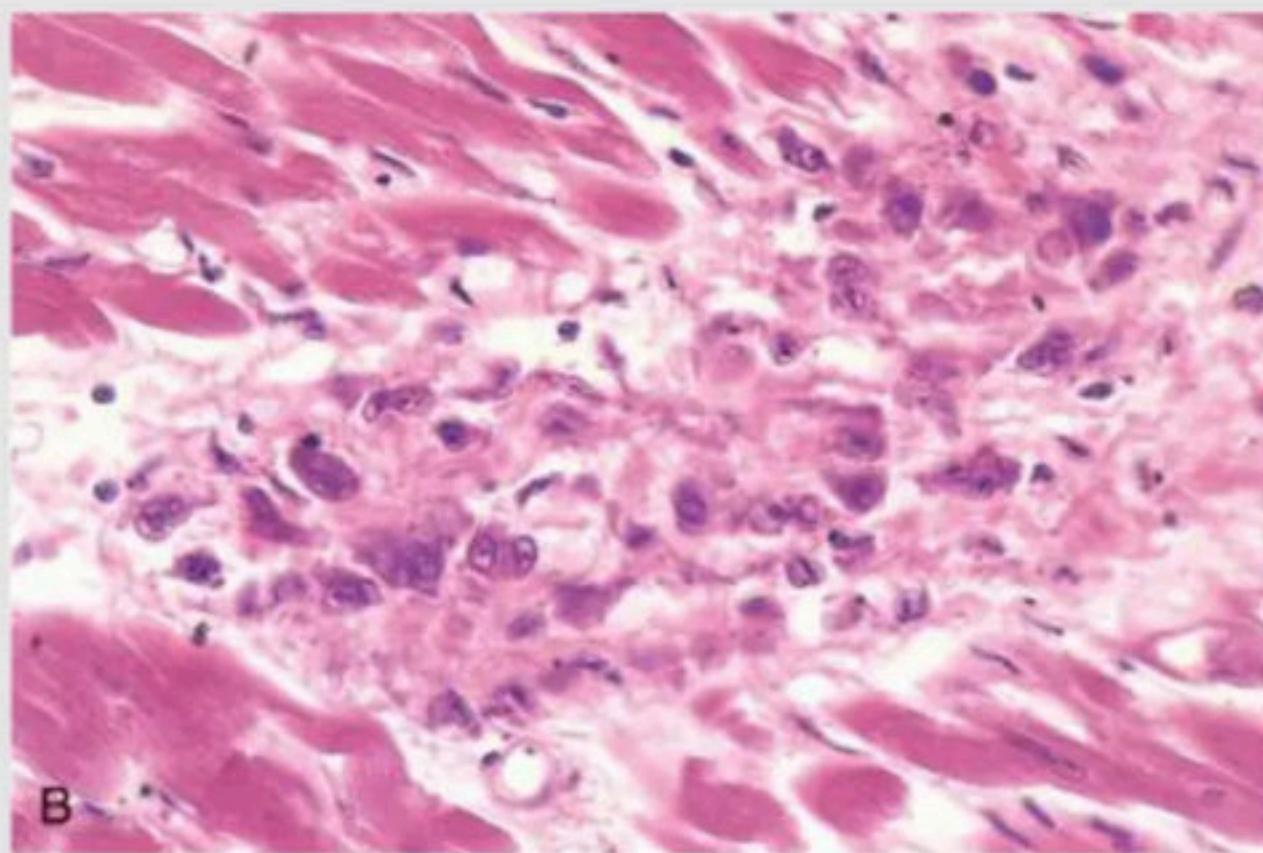
Aschoff cell

Anitschkov cell
(histiocyte)



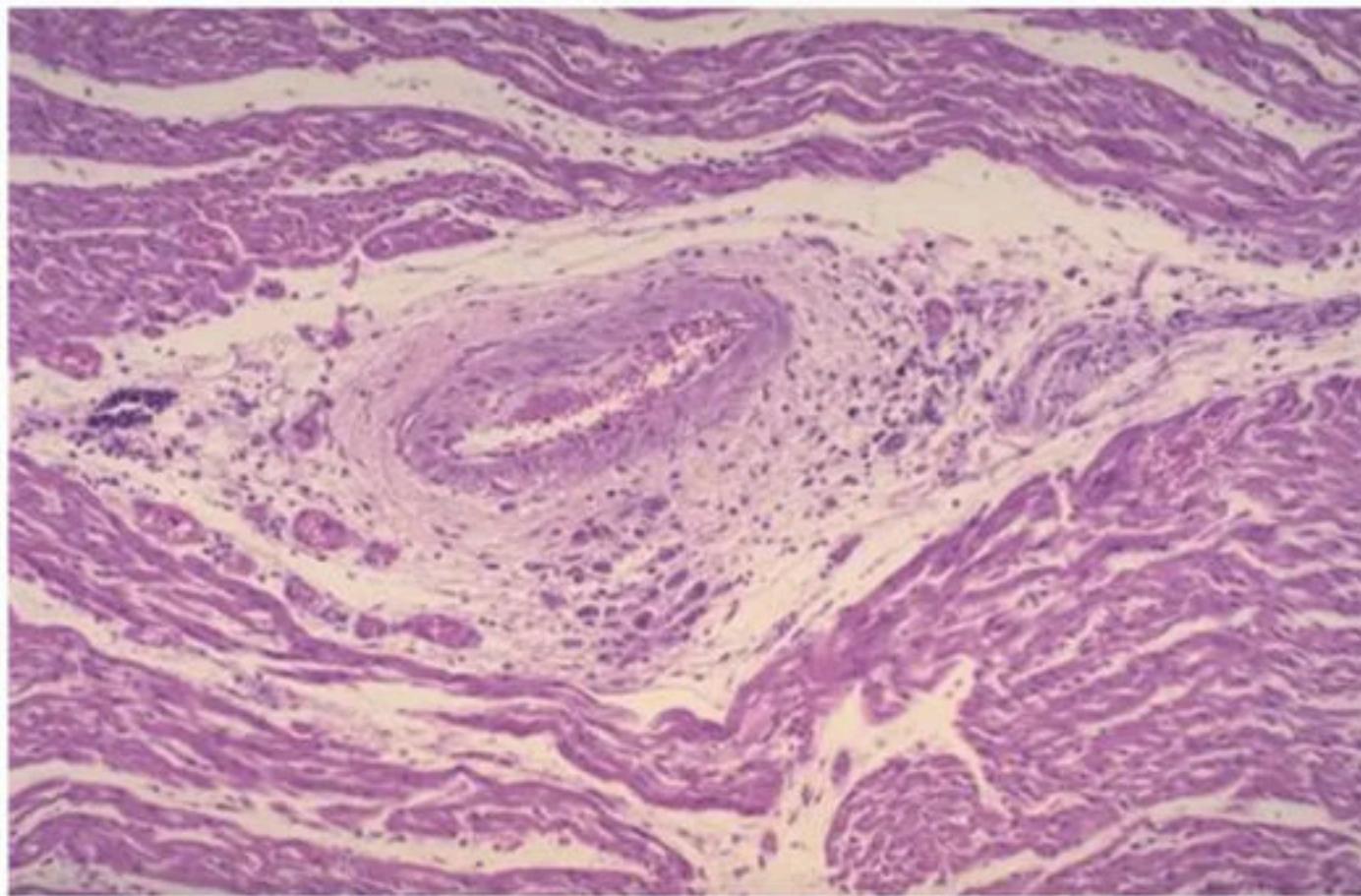
B Rheumatic granuloma
(HE) x 100



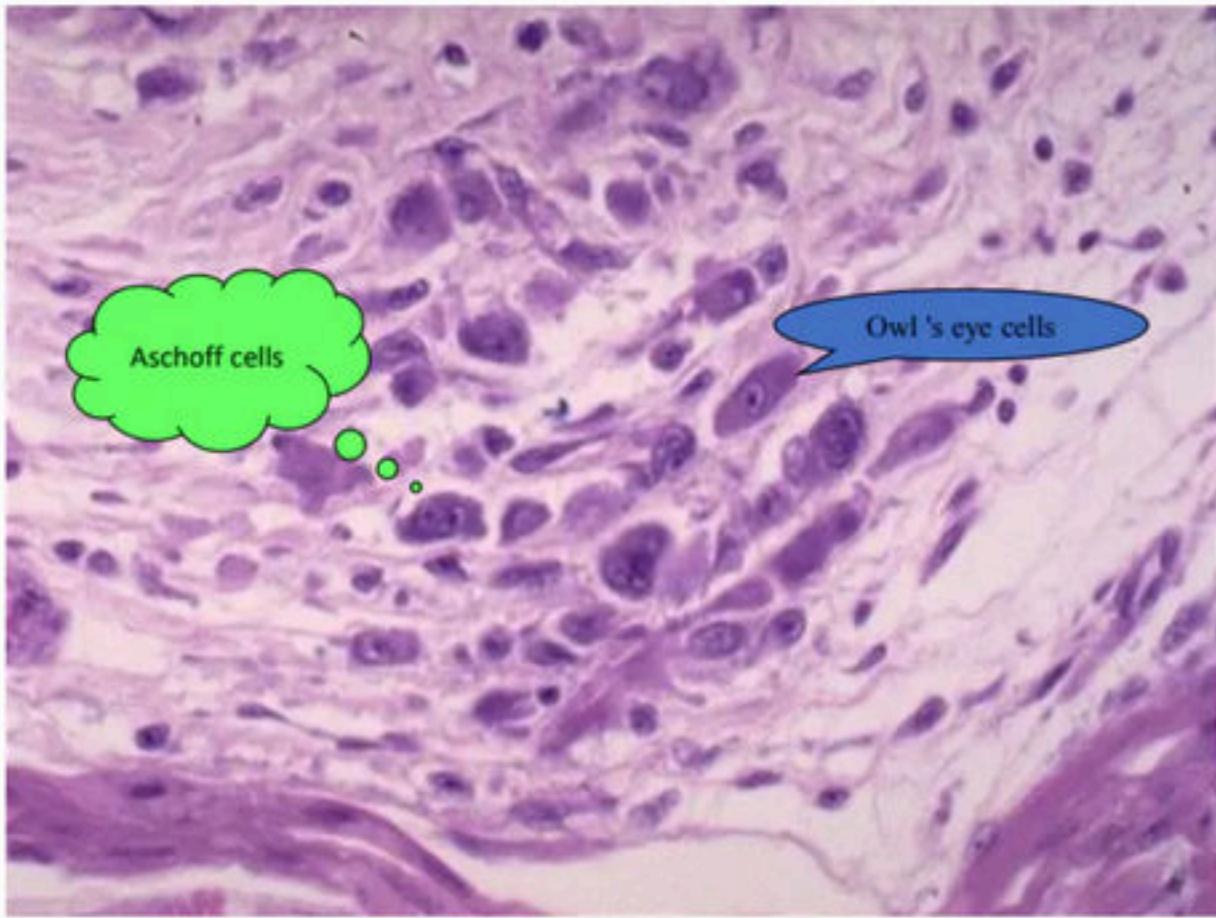


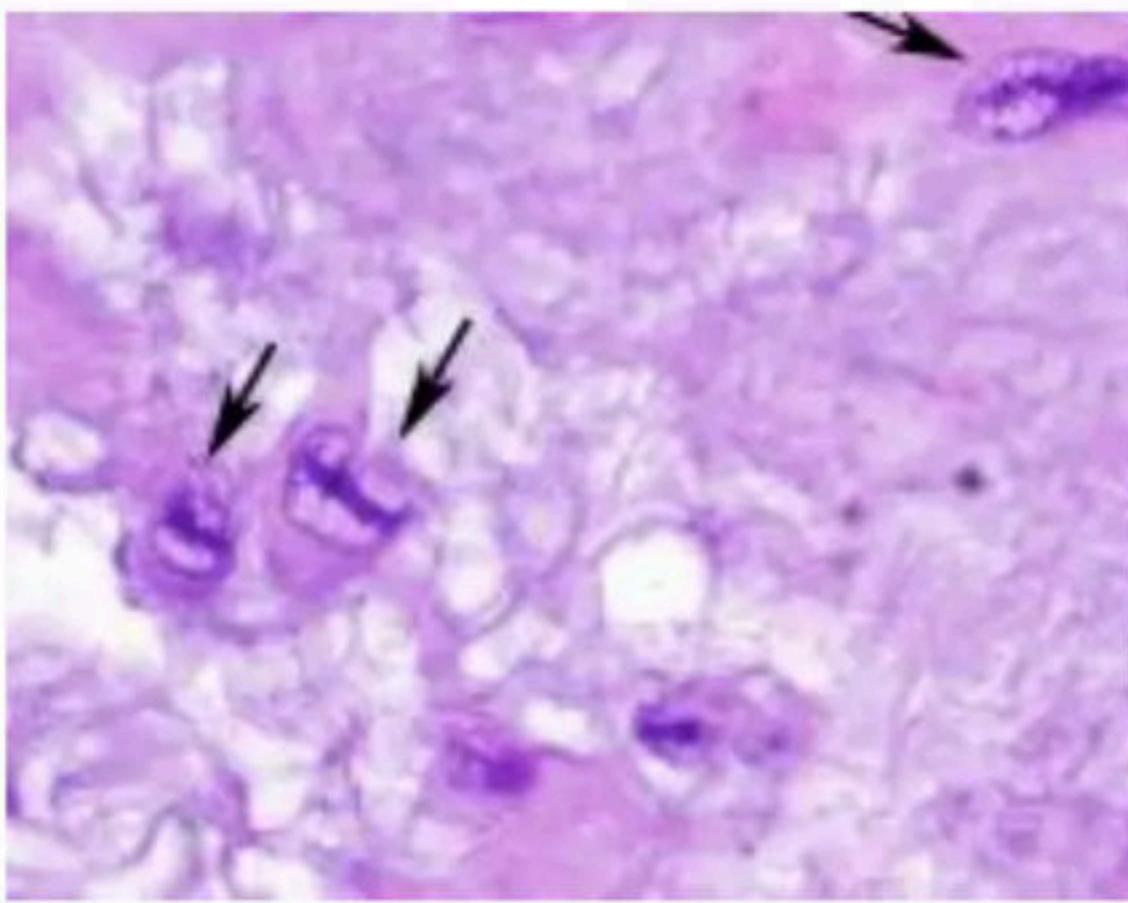
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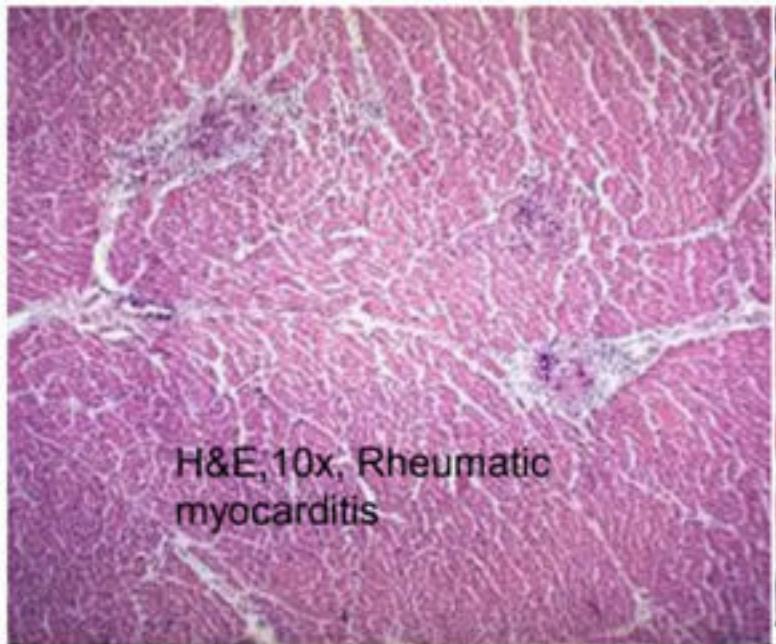


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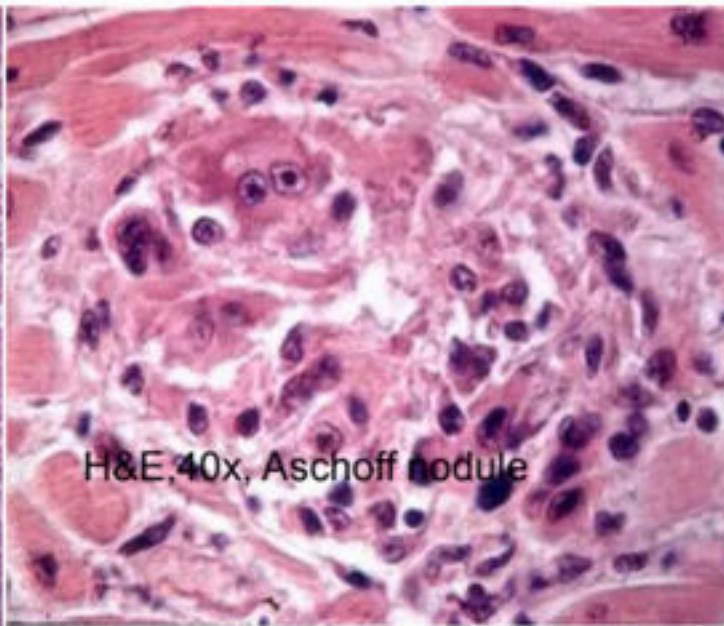




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H&E, 10x, Rheumatic myocarditis



H&E, 40x, Aschoff nodule

OVERVIEW

- **Introduction**

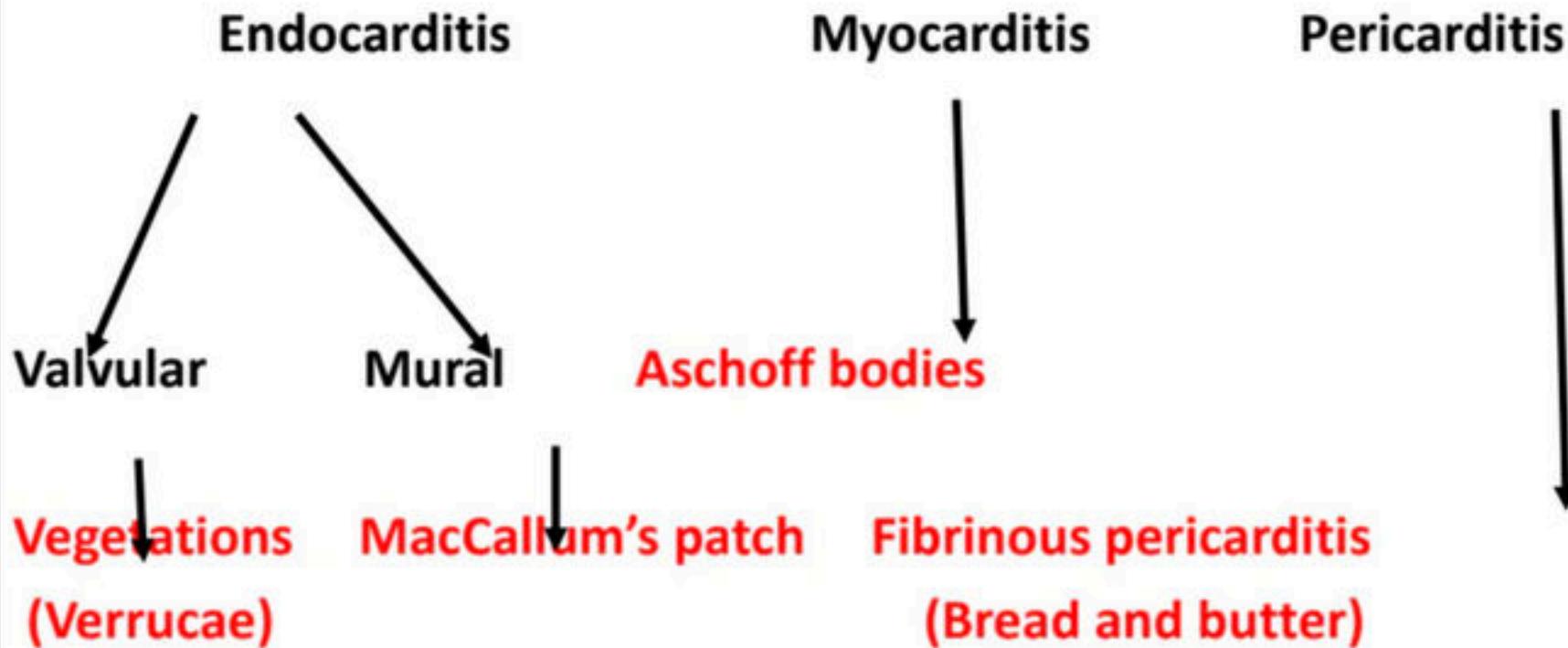
- 1. Rheumatic endocarditis**

- a) Rheumatic valvulitis**
- b) Rheumatic mural endocarditis**

- 2. Rheumatic myocarditis**

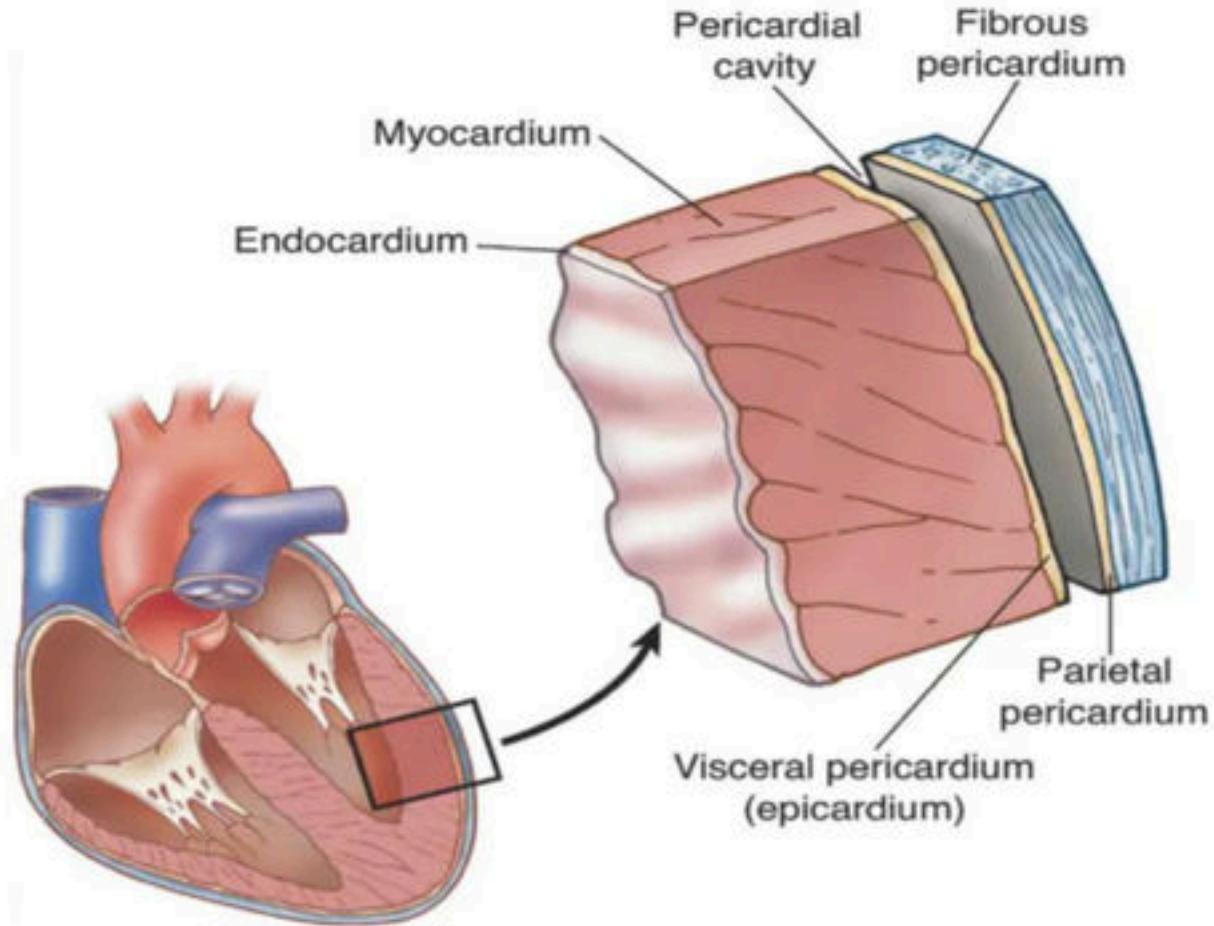
- 3. Rheumatic pericarditis**

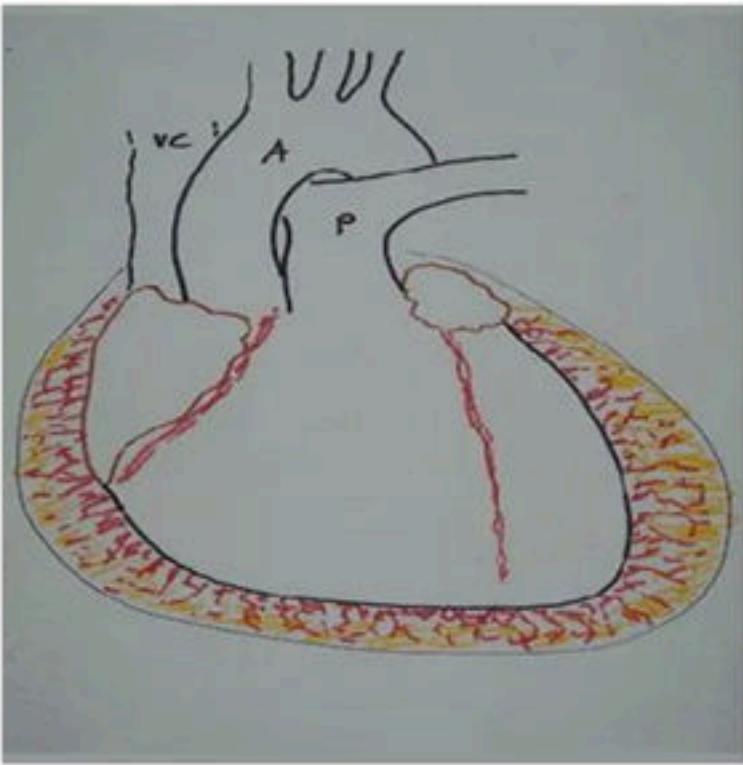
PANCARDITIS



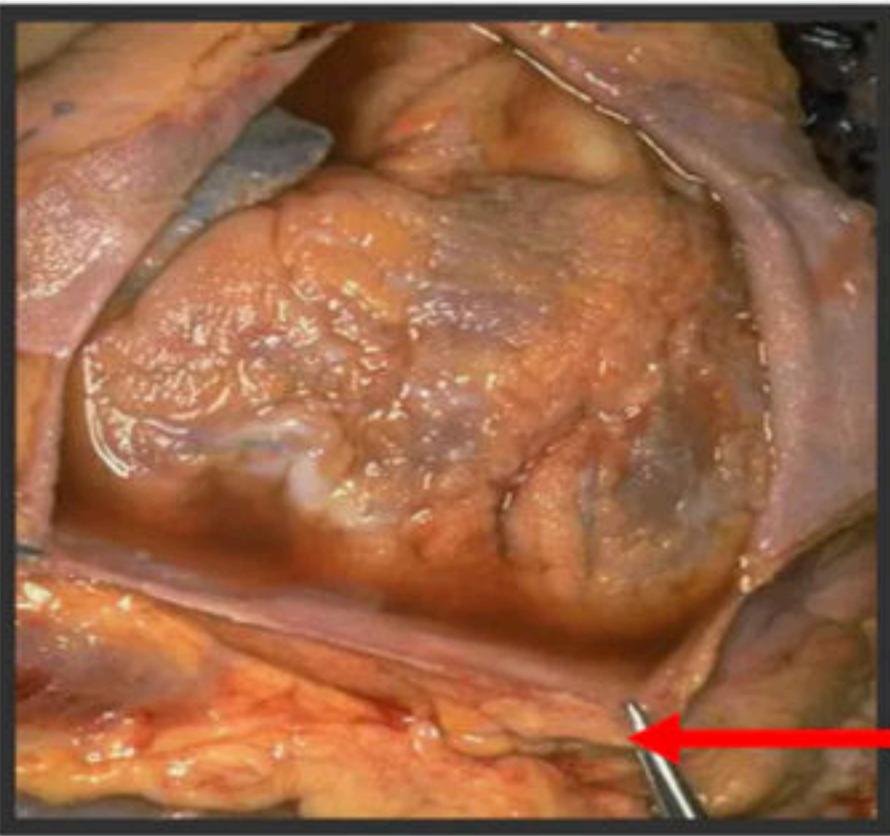
RHEUMATIC PERICARDITIS

- The usual finding is **fibrinous pericarditis**
- Two separated surfaces are shaggy due likened to '**bread and butter appearance**'





Fibrinous pericarditis



Can lead to heart sound far,
around the heart boundary
expanding, serious cardiac X-ray
showed a flask

pericardial
effusion



Adhesive pericarditis is in cardiac surface of patients. From the epicardial surface to the pericardial sac visible fibrinous exudate, which is typical for a fibrinous pericarditis.

FINALLY...

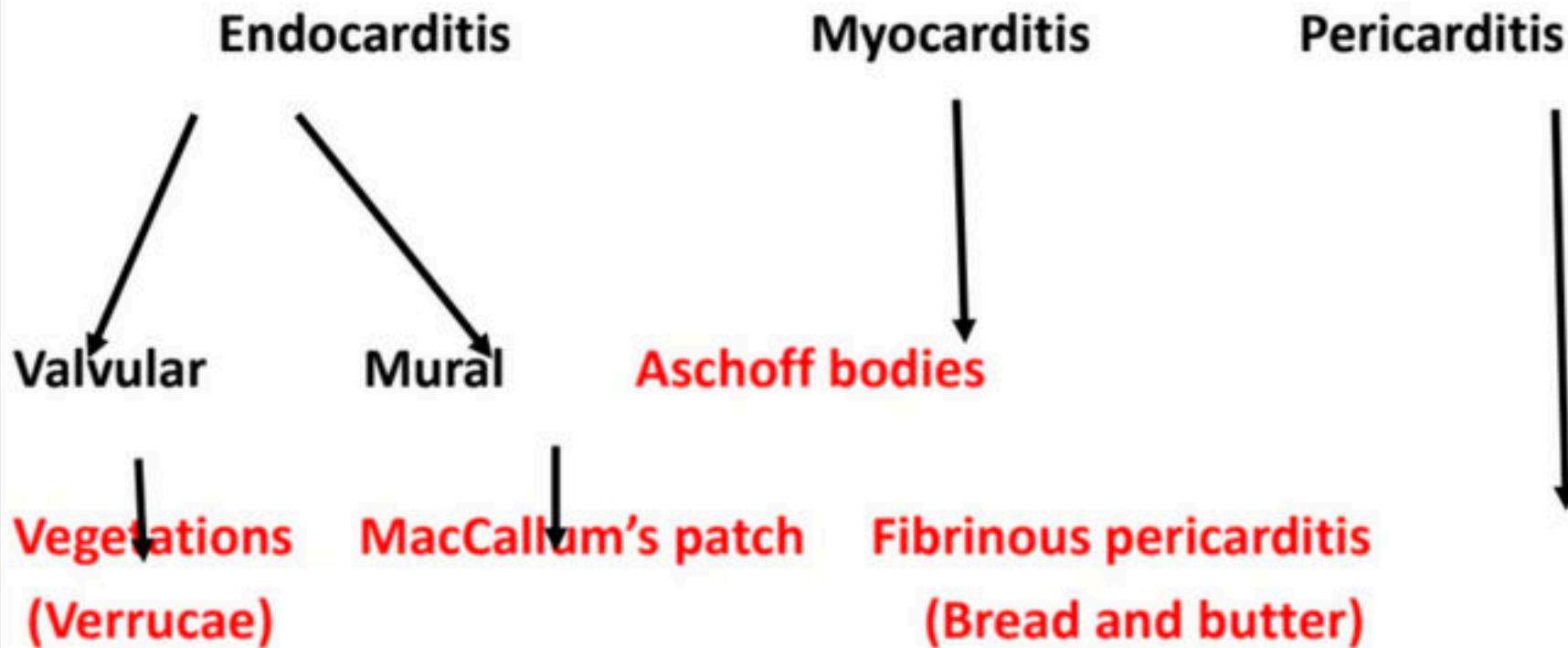
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*Click or Scan QR code to join
Telegram group discussion*



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PANCARDITIS



REMEMBER

- **Echo with Doppler** must be done in all cases of confirmed and suspected acute Rheumatic fever as auscultatory findings take time to develop.
- **Subclinical carditis seen on echo** is now considered a Major diagnostic criteria.

POLL 7

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A 10 year old boy, Pappu, died of rheumatic fever. All the following can be expected at autopsy except -

- a) Ashoff nodules
 - b) Rupture of chordae tendinae
 - c) Mc Callum patch
 - d) Fibrinous pericarditis
-
-

B



Antischkow cells are -

- a) Modified macrophages
- b) Modified neutrophils
- c) Modified B cells
- d) Modified RBCs



A



Which is not seen in Aschoff bodies -

- a) Giant cells
- b) Aschoff cells
- c) Fibroblasts
- d) Polymorphonuclear cells



D



MC valve involved in Rheumatic fever -

- a) Mitral
- b) Aortic
- c) Pulmonary
- d) Tricuspid



A



Valve usually not involved in rheumatic fever -

- a) Mitral
- b) Aortic
- c) Pumonary
- d) Tricuspid



C



med[LIVE]

Ascoff's bodies are seen in -

- a) Rheumatic myocarditis
- b) Rheumatic arthritis
- c) Bacterial endocarditis
- d) Marantic endocarditis



A



Mc Callum's patch is diagnostic of -

- a) Infective endocarditis
- b) Rheumatic endocarditis
- c) Myocardial infarction
- d) Tetralogy of Fallot (ToF)

B



Characteristic feature of Rheumatic carditis is -

- a) Pericarditis
- b) Endocarditis
- c) Myocarditis
- d) Pancarditis



D



The most characteristic histological finding of acute rheumatic carditis is -

- a) Fibrinous pericarditis
- b) Vegetations on mitral valve leaflets
- c) Aschoff bodies in myocardium
- d) Increased vascularity of the valves



C



med[LIVE]

--, - - - - -

Exudate in rheumatic fever is -

- a) Serous
- b) Purulent
- c) Fibrinous
- d) Myxomatous



C



med[LIVE]

Pathognomonic features of acute rheumatic fever is-

- a) Pericarditis
- b) Myocarditis
- c) Mitral stenosis
- d) Aschoffs nodules

D



True about vegetation in rheumatic fever is -

- a) Soft & friable
- b) Large & firm
- c) Soft & firm
- d) Large & friable



C



med[LIVE]

MacCallum plaques are usually seen in the

- a) right atrium
- b) left atrium
- c) left ventricle
- d) right ventricle

B

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In Rheumatic fever, fibrinoid necrosis occurs in

- a) Myocardium
- b) Collagen
- c) Pericardium
- d) Endocardium

Dr. PKV

A

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OVERVIEW

- Introduction
 - Etiology
 - Pathogenesis
 - Jones criteria
 - Revised Jones criteria
-
- 1. Rheumatic heart disease (Heart)
 - 2. Polyarthritis (Joints)
 - 3. Sydenham's chorea (Brain)
 - 4. Erythema marginatum (Skin)
 - 5. Subcutaneous nodules (Subcutaneous tissue)

Extracardiac Lesions



Migratory Polyarthritis

- Earliest manifestation within 2 weeks with H/O sore throat
- Most common Manifestation (75%)
- Involves **larger joints**: the knees, ankles, wrists & elbows
- Rheumatic joints: **hot, red, swollen & exquisitely tender**
- The **pain** can precede & can appear to be disproportionate to the other findings

- The joint involvement is characteristically **migratory** in nature
- **Several joints are involved in quick succession and each for a brief period of time.**

Ankle swelling

Knee Swelling

Elbow swelling

Polyarthritis

- Low endemic (USA) → **Migratory polyarthritis**
- High endemic (India) → If single joint involved
i.e. **Monoarthritis** - Sufficient for diagnosis of Arthritis

- When swelling subsides, no deformity: **Non-erosive arthritis**
- Sometime can cause **Erosive arthritis** called as **Jacoud's Arthritis**

- A dramatic response to even small doses of salicylates is another characteristic feature of the arthritis
- Rheumatic arthritis is typically not deforming

- Arthritis; **earliest manifestation** of acute rheumatic fever
- An inverse relationship between the severity of arthritis & the severity of cardiac involvement

Chorea

- **St. Vitus'dance**
- Sydenham chorea: 10-15% of patients with acute rheumatic fever
- **Damage to caudate nucleus**

Chorea



- The characteristic picture of chorea is that it often occur in **isolation**, either unaccompanied by other major manifestations of RF
- Or after a **latent period** of several months, at a time when all other manifestations of RF have subsided.

- Often in **prepubertal girls (8-12 yrs)**
- A long latency period (**1-6 months**) between streptococcal pharyngitis & the onset of chorea

Neuropsychiatric disorder→

- **Neurologic signs:** choreic jerky movement & hypotonia → **Movements disappear during sleep**
- **Psychiatric signs:** emotional lability, hyperactivity, separation anxiety, obsessions & compulsions



- Begins with emotional lability & personality changes (poor school performance)
- Replace in 1-4 weeks by **characteristic spontaneous, purposeless movement of chorea** (lasts 4-8 months) followed by motor weakness
- **Exacerbation by stress & disappearing with sleep are characteristic**
- Elevated titers of “**antineuronal antibodies**” against basal ganglion tissues have been found in over 90% of patients

- Sex hormones (estrogen) or pregnancy can cause recurrences

Clinical maneuvers to elicit features of chorea include

- (1) demonstration of **milkmaid's grip** (irregular contractions of the muscles of the hands while squeezing the examiner's fingers)
- (2) **spooning and pronation** of the hands when the patient's arms are extended
- (3) **wormian darting movements** of the tongue upon protrusion
- (4) examination of **handwriting** to evaluate fine motor movements



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Chorea



SACHDEV

REMEMBER

- Chorea and arthritis cannot co - exists in a single patient

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Treatment

- Self limiting condition: 6 months - 12- months
- Valproate, Haloperidol , Phenobarbitone
 - If Fails
- Steroids
 - If Fails
- IV Immunoglobulin
- Medically refractory chorea: IV Ig

Erythema Marginatum

A rare (<3% of patients with acute rheumatic fever) but characteristic rash of acute rheumatic fever

- It consists of **erythematous**,
- **serpiginous**,
- **macular lesions with pale centers**
- **Not pruritic**
- It occurs primarily on the **trunk & extremities, not on the face**
- It can be accentuated by warming
- **the skin- bathing suit distribution**



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- Rash is evanescent **migrating** from place to place.
- **No residual scarring** occurs.



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Varicella (chickenpox)

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Subcutaneous Nodules

- A rare (**≤1%** of patients with acute rheumatic fever) finding
- Consist of firm nodules approximately **1 cm in diameter along the extensor surfaces of tendons near bony prominences**
- It is often seen in **association with carditis**
- **Painless and Nontender nodules**

- They last for a week or two and
disappear spontaneously.



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Arthritis of RF—all are true except

- a. Non deforming
- b. Associated with raised ASO titre
- c. Lasts for 3-6 weeks in untreated cases
- d. Small joint involvement is usual

D

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True about s\c nodules except

- a. delayed manifestation
- b. painful nodules
- c. seen in 3-5% of patients
- d. commonly associated with severe carditis

B

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All are true about erythema marginatum except

- a. Bathing suit distribution
- b. Early manifestation
- c. Pruritic lesion
- d. seen rarely

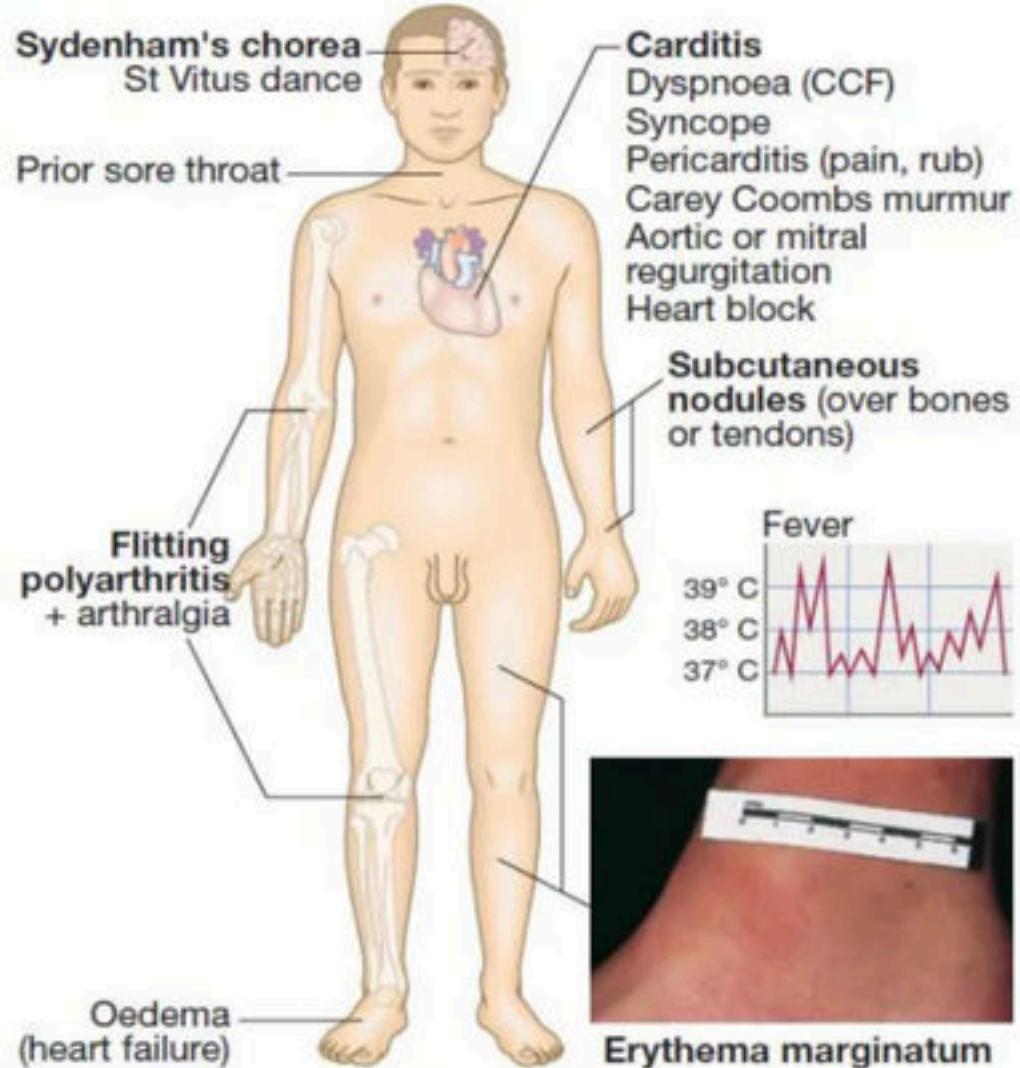
C

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Clinical features of rheumatic fever

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Investigations in acute rheumatic fever

Evidence of a systemic illness

- Leucocytosis, raised erythrocyte sedimentation rate and C-reactive protein

Evidence of preceding streptococcal infection

- Throat swab culture: group A β -haemolytic streptococci (also from family members and contacts)
- Antistreptolysin O antibodies (ASO titres): rising titres, or levels of >200 U (adults) or >300 U (children)

Evidence of carditis

- Chest X-ray: cardiomegaly; pulmonary congestion
- ECG: first- and, rarely, second-degree atrioventricular block; features of pericarditis; T-wave inversion; reduction in QRS voltages
- Echocardiography: cardiac dilatation and valve abnormalities

Management

- The aims of management are to **limit cardiac damage and relieve symptoms.**

Bed rest

- Bed rest is important, as it lessens **joint pain and reduces cardiac workload.**
- The duration should be guided by symptoms, along with temperature, leucocyte count and ESR, and should be continued until these have settled.
- Patients can then return to normal physical activity but strenuous exercise should be avoided in those who have had carditis.

Treatment of cardiac failure

- Cardiac failure should be treated as necessary.

LMNOP

1. LASIX
2. Morphine
3. NTG, Oz
4. Head high position

- Occasionally, AV block may occur but is seldom progressive and usually resolves spontaneously.
- Rarely, **pacemaker** insertion may be required.

Aspirin

- This usually relieves the symptoms of **arthritis** rapidly and a response within 24 hours helps confirm the diagnosis.
- A reasonable starting dose is **60 mg/kg body weight/day**, divided into six doses.
- In adults, **100 mg/kg per day** may be needed up to the limits of tolerance or a maximum of **8 g per day**.
- Mild toxicity includes nausea, tinnitus and deafness; vomiting, tachypnoea and acidosis are more serious.
- Aspirin should be continued **until the ESR has fallen** and then gradually tailed off.

Glucocorticoids

- These produce more rapid symptomatic relief than aspirin and are indicated in cases with **carditis or severe arthritis.**
- There is no evidence that long-term steroids are beneficial.
- **Prednisolone (1.0–2.0 mg/kg per day in divided doses)** should be continued until the ESR is normal and then tailed off.

Antibiotics

- A single dose of **benzathine benzylpenicillin (1.2 million U IM) or oral phenoxyethylpenicillin (250 mg 4 times daily for 10 days)** should be given on diagnosis to eliminate any residual streptococcal infection.
- If the patient is penicillin-allergic, **erythromycin or a cephalosporin** can be used.

Prophylaxis

- Patients are susceptible to further attacks of rheumatic fever if another streptococcal infection occurs
- Long-term prophylaxis with penicillin should be given with **oral phenoxymethypenicillin (250 mg twice daily) or as benzathine benzylpenicillin (1.2 million U IM monthly)**

Duration of prophylaxis

whichever is longer

	Age	Years from date of diagnosis
RHP with valvular lesion	40 years	10 years
RHP without valve lesion	21 years	10 years
RF	21 years	5 years

- Best for prophylaxis of R.HD in penicillin allergic child:
Azithromycin > Sulphadiazine

ENDOCARDITIS

- Endocarditis is an **inflammation of the inner layer of the heart, i.e. endocardium**

TYPES

Table 14.6 Classification of endocarditis.

A. NON-INFECTIVE

1. Rheumatic endocarditis (page 420)
2. Atypical verrucous (Libman-Sacks) endocarditis
3. Non-bacterial thrombotic (cachectic, marantic) endocarditis

B. INFECTIVE

1. Bacterial endocarditis
2. Other infective types (tuberculous, syphilitic, fungal, viral, rickettsial)

1. Rheumatic fever and rheumatic heart disease (Endocarditis)

- ## **2. Non-infective**
- 2. Atypical verrucous (Libman-Sacks) endocarditis
 - 3. Non-bacterial thrombotic (cachectic, marantic) endocarditis

INFECTIVE

- 1. Bacterial endocarditis
- 2. Other infective types (tuberculous, syphilitic, fungal, viral, rickettsial)

Non-rheumatic endocarditis

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FEATURE	RHEUMATIC	LIBMAN-SACKS	NON-BACTERIAL THROMBOTIC	BACTERIAL (INFECTIVE)
1. Valves commonly affected	Mitral alone; mitral and aortic combined	Mitral, tricuspid	Mainly mitral; less often aortic and tricuspid	Mitral; aortic; combined mitral and aortic
2. Location on valve cusps or leaflets	Occur along the line of closure, atrial surface of atrioventricular valves and ventricular surface of semilunar valves	Occur on both surfaces of valve leaflets or cusps, in the valve pockets	Occur along the line of closure	SABE more often on diseased valves: ABE on previously normal valves; location same as in RHD
3. Macroscopy				
	Small, multiple, warty, grey brown, translucent, firmly attached, generally produce permanent valvular deformity	Medium-sized, multiple, generally do not produce significant valvular deformity	Small but larger than those of rheumatic, single or multiple, brownish, firm, but more friable than those of rheumatic	Often large, grey-tawny to greenish, irregular, single or multiple, typically friable
4. Microscopy	Composed of fibrin with superimposed platelet thrombi and no bacteria. Adjacent and underlying endocardium shows oedema, proliferation of capillaries, mononuclear inflammatory infiltrate and occasional Aschoff bodies.	Composed of fibrinoid material with superimposed fibrin and platelet thrombi and no bacteria. The underlying endocardium shows fibrinoid necrosis, proliferation of capillaries and acute and chronic inflammatory infiltrate including the haematoxylin bodies of Gross.	Composed of degenerated valvular tissue, fibrin-platelets thrombi and no bacteria. The underlying valve shows swelling of collagen, fibrinoid change, proliferation of capillaries but no significant inflammatory cell infiltrate.	Composed of outer eosinophilic zone of fibrin and platelets, covering colonies of bacteria and deeper zone of non-specific acute and chronic inflammatory cells. The underlying endocardium may show abscesses in ABE and inflammatory granulation tissue in the SABE.

ATYPICAL VERRUCOUS (LIBMAN-SACKS) ENDOCARDITIS

Dr. PRIYANKA SACHDEV

OVERVIEW

Introduction

Gross →

- a) Vegetations or Verrucae
- b) Valves
- c) Location
- d) Deformity

Microscopy

Introduction

- It is one of the manifestations of '**collagen diseases**'
- Characteristic lesions of Libman-Sacks endocarditis are seen in 50% cases of **systemic lupus erythematosus (SLE)**

OVERVIEW

Introduction

Gross →

- a) Vegetations or Verrucae
- b) Valves
- c) Location
- d) Deformity

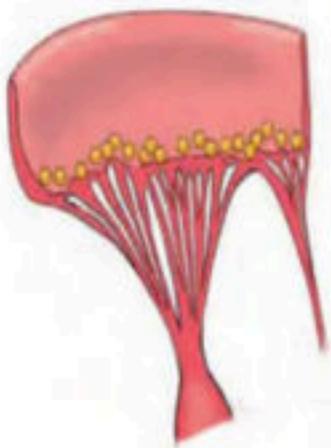
Microscopy

Grossly

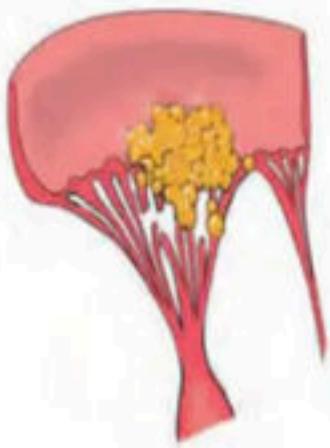
- Characteristic **vegetations**

Dr. PRIYANKA SACHDEV

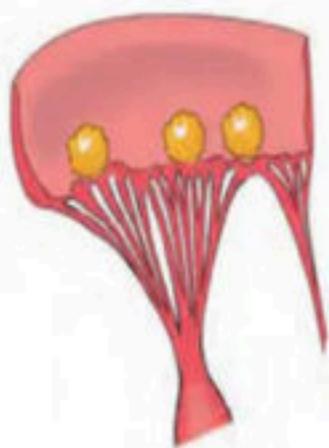
- The vegetations of atypical verrucous endocarditis are
 - Medium sized (1 to 4 mm in diameter)
 - Multiple
 - Granular
 - Sterile, bland



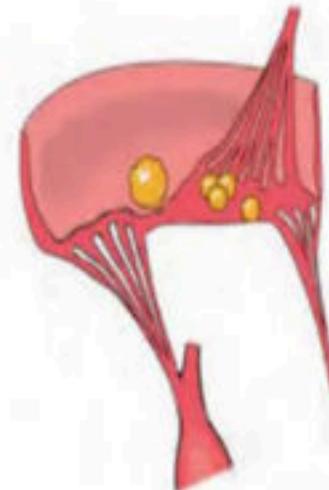
RHD



IE

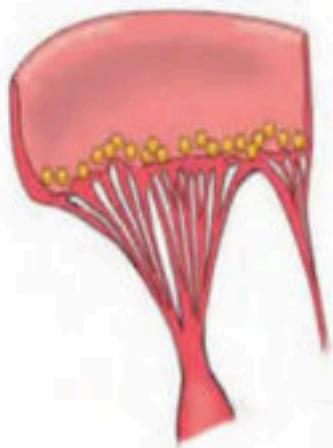


NBTE

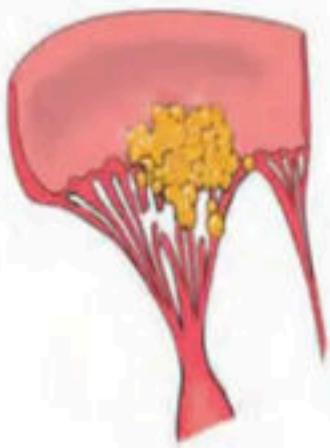


LSE

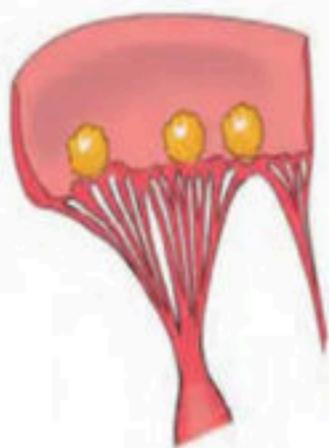
- **Valve** → occur most frequently on the **mitral and tricuspid valves**
- **Location** → occur on **both surfaces of affected valves, in the valve pockets and on the adjoining ventricular and atrial endocardium.**



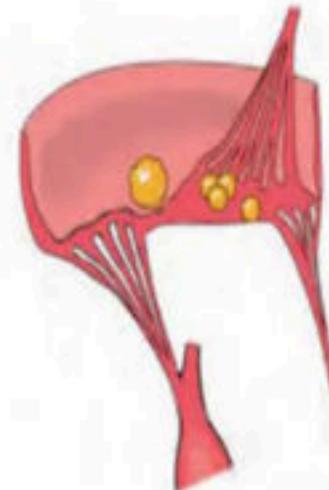
RHD



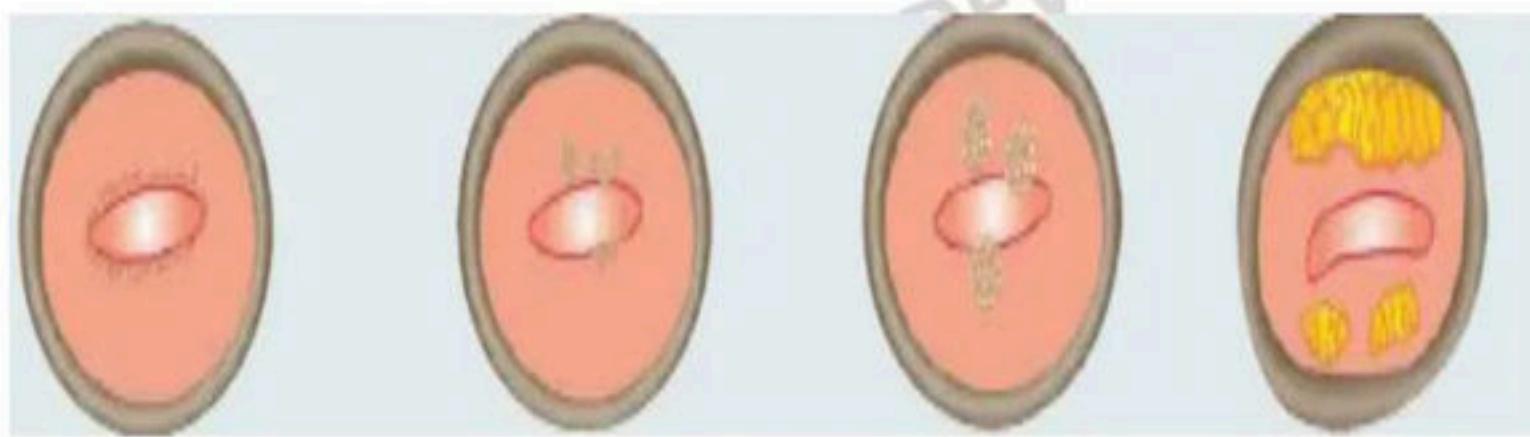
IE



NBTE



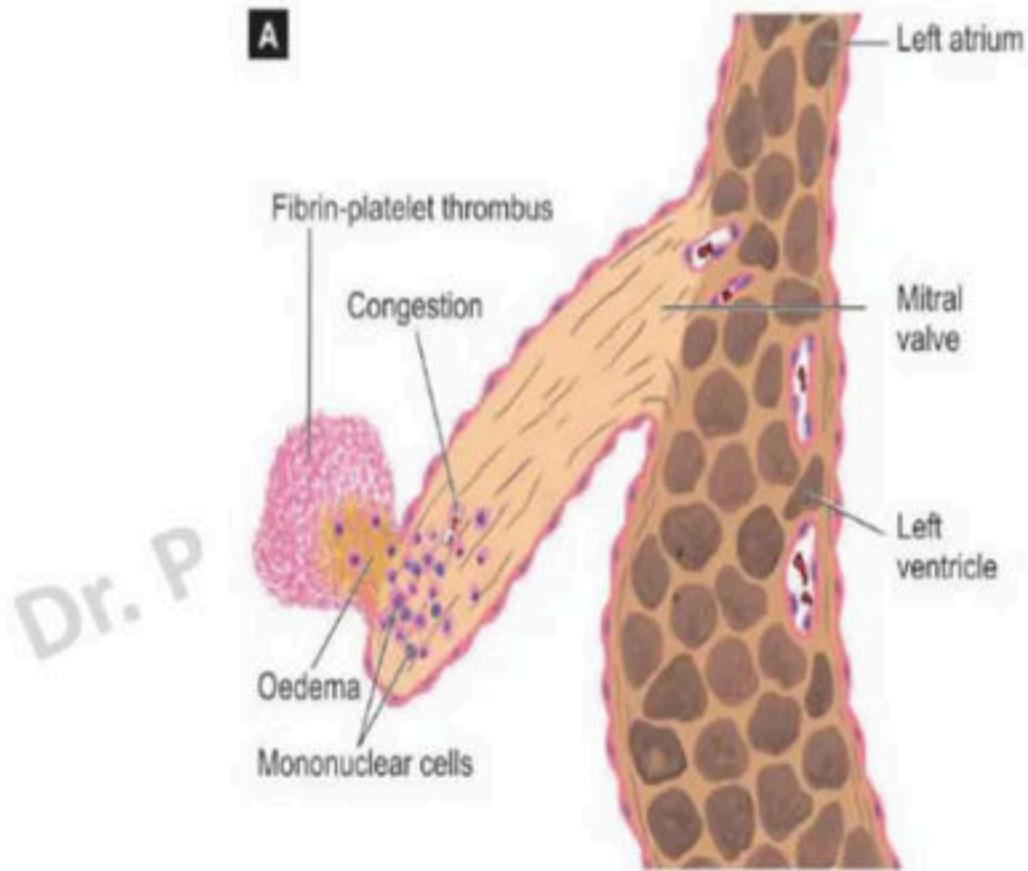
LSE



- **Deformity** → The vegetations are Unlike vegetations of RHD, the healed vegetations of Libman-Sacks endocarditis **do not produce any significant valvular deformity.**

Microscopically

- The **verrucae of Libman-Sacks endocarditis** are composed of **fibrinoid material with superimposed fibrin and platelet thrombi**, no bacteria (sterile)
- The **endocardium underlying the verrucae** shows oedema, proliferation of capillaries and infiltration by histiocytes, plasma cells, lymphocytes, neutrophils (acute and chronic inflammatory infiltrate) and the **pathognomonic haematoxylin bodies of Gross**
- The Aschoff bodies are never found in the endocardium or myocardium



FEATURE	RHEUMATIC	LIBMAN-SACKS	NON-BACTERIAL THROMBOTIC	BACTERIAL (INFECTIVE)
1. Valves commonly affected	Mitral alone; mitral and aortic combined	Mitral, tricuspid	Mainly mitral; less often aortic and tricuspid	Mitral; aortic; combined mitral and aortic
2. Location on valve cusps or leaflets	Occur along the line of closure, atrial surface of atrioventricular valves and ventricular surface of semilunar valves	Occur on both surfaces of valve leaflets or cusps, in the valve pockets	Occur along the line of closure	SABE more often on diseased valves: ABE on previously normal valves; location same as in RHD
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TYPES

A. NON-INFECTIVE

1. Rheumatic endocarditis (page 420)
2. Atypical verrucous (Libman-Sacks) endocarditis
3. Non-bacterial thrombotic (cachectic, marantic) endocarditis

B. INFECTIVE

1. Bacterial endocarditis
2. Other infective types (tuberculous, syphilitic, fungal, viral, rickettsial)

NON-BACTERIAL THROMBOTIC (CACHECTIC, MARANTIC) ENDOCARDITIS (NBTE)

Dr. PRIYANKA SACHDEV

OVERVIEW

Introduction

Gross →

- a) Vegetations or Verrucae
- b) Valves
- c) Location
- d) Deformity

Microscopy

Introduction

- Non-bacterial thrombotic endocarditis is an involvement of the heart valves by **sterile thrombotic vegetation**
- Seen in patients having **hypercoagulable state** from various etiologies e.g. advanced cancer (in 50% case of NBTE) especially mucinous adenocarcinomas, chronic tuberculosis, renal failure and chronic sepsis

HEADINGS

Introduction

Gross →

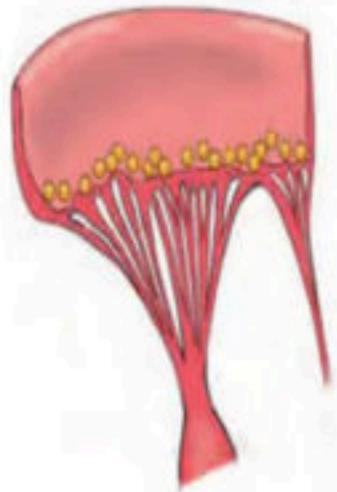
- a) Vegetations or Verrucae
- b) Valves
- c) Location
- d) Deformity

Microscopy

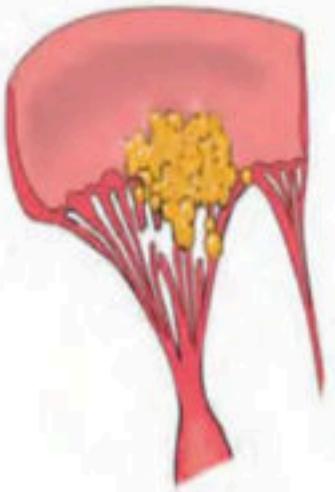
Grossly

- Vegetations/ verrucae of NBTE →
- Small (1 to 5 mm in diameter),
- Single or multiple,
- Brownish
- More friable than the vegetations of RHD.
- Sterile, bland

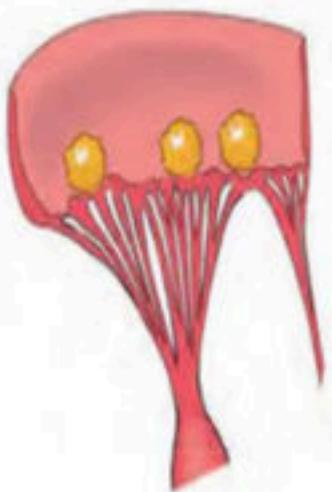
- **Valves** → chiefly mitral, and less often aortic and tricuspid valve.
- **Location** → Occur along the line of closure of the leaflets



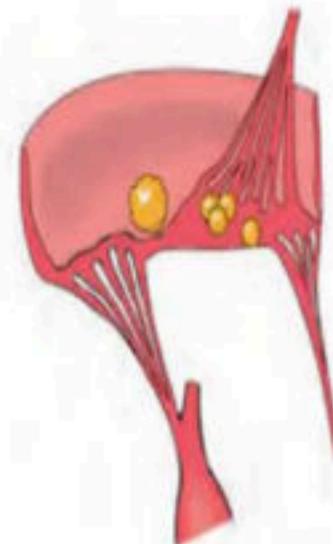
RHD



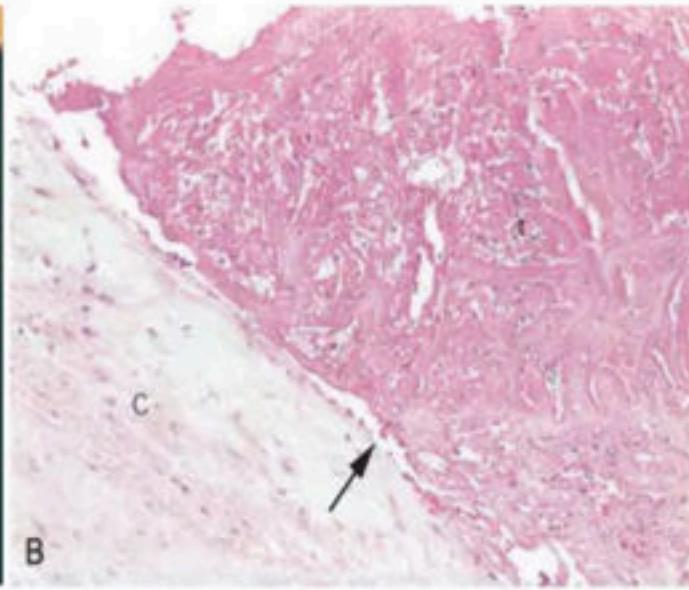
IE



NBTE



LSE



- **Deformity** → Organised and healed vegetations appear as **fibrous nodules**

Microscopically

- **The vegetations** in NBTE are composed of fibrin along with entangled RBCs, WBCs and platelets.
- **The underlying valve** shows swollen collagen, fibrinoid change and capillary proliferation but **does not show any inflammatory infiltrate**

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INFECTIVE (BACTERIAL) ENDOCARDITIS

Dr. PRIYANKA SACHDEV

OVERVIEW

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Types

Etiology

Predisposing factors

Pathogenesis

Gross→

- a) Vegetations or Verrucae
- b) Valves
- c) Location
- d) Deformity

Microscopy

Complications

Modified Duke criteria

INFECTIVE (BACTERIAL) ENDOCARDITIS

- Infective or bacterial endocarditis (IE or BE) is serious infection of the valvular and mural endocardium caused by different forms of **microorganisms**
- Characterised by **typical infected and friable vegetations**

Types

- 1. Acute bacterial endocarditis (ABE)**
- 2. Subacute bacterial endocarditis (SABE) or endocarditis lenta (lenta = slow)**

1. Acute bacterial endocarditis (ABE)

- Caused by **highly virulent bacteria (staph aureus)**
- In a **previously normal heart**
- Runs a rapidly fatal course in a period of **2-6 weeks**

2. Subacute bacterial endocarditis (SABE) or endocarditis lenta (lenta = slow)

- Caused by **less virulent bacteria (strep viridians)**
- In a **previously diseased heart**
- Has a gradual downhill course in a period of **6 weeks to a few months** and sometimes years.

FEATURE	ACUTE	SUBACUTE
1. <i>Duration</i>	<6 weeks	>6 weeks
2. <i>Most common organisms</i>	<i>Staph. aureus</i> , β-streptococci	<i>Streptococcus viridans</i>
3. <i>Virulence of organisms</i>	Highly virulent	Less virulent
4. <i>Previous condition of valves</i>	Usually previously normal	Usually previously damaged
5. <i>Lesion on valves</i>	Invasive, destructive, suppurative	Usually not invasive or suppurative
6. <i>Clinical features</i>	Features of acute systemic infection	Splenomegaly, clubbing of fingers, petechiae

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ETIOLOGY

- **Infective agents** About 90% cases of BE are caused by **streptococci and staphylococci.**

- In ABE → the most common causative organisms are virulent strains of staphylococci, chiefly **Staphylococcus aureus**
- In SABE → the commonest causative organisms are the streptococci with low virulence, predominantly **Streptococcus viridans**

REMEMBER

- **Staphylococcus aureus** is the most common **overall** cause of infective endocarditis
- **Staphylococcus aureus** is also the most common cause in **native valve and intravenous drug users**

- **Staphylococcus aureus** is the most common cause in **early prosthetic valve endocarditis**, i.e. upto 12 months after valve surgery) and in patients with **intravenous catheter**.
- **Streptococcus viridans** is the most common cause in **late prosthetic valve endocarditis** (> 12 months after valve surgery).
- **Streptococcus mutans** is the most common cause of endocarditis after **dental procedure** (tooth extraction)

Predisposing conditions

- i) High risk lesions : VSD, MR, prosthetic valves, TOF , PDA , AS, MR
- ii) Moderate risk lesions : TS, TR, PS, and MS
- iii) Low risk : ASD

REMEMBER

- **VSD** is the most common congenital heart disease involved in infective endocarditis.
- Infective endocarditis is rare in **ASD**

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Microscopy

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Modified Duke criteria

PATHOGENESIS

**Conditions producing haemodynamic stress
on the valves**



cause damage to the endothelium



formation of platelet-fibrin thrombi



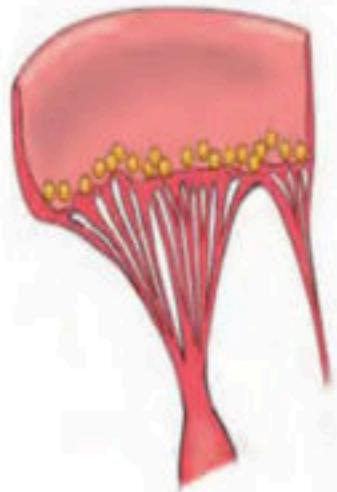
Get infected from circulating bacteria

Grossly

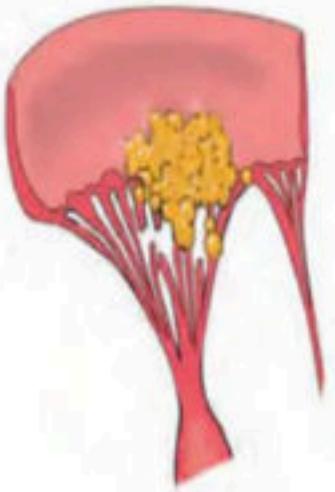
- The vegetations in **SABE** are more often seen on **previously diseased valves**
- The vegetations of **ABE** are often found on **previously normal valves**

The vegetations are→

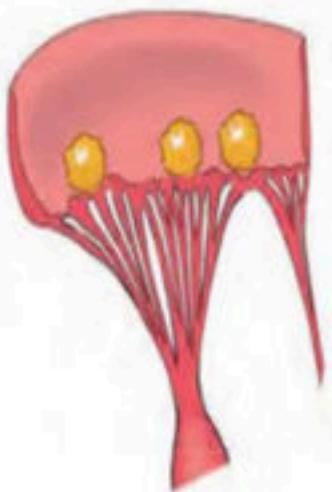
- Large
- Grey-tawny to greenish,
- Irregular,
- Single or multiple
- Typically friable
- The vegetations in ABE tend to be **bulkier** and globular than those of SABE



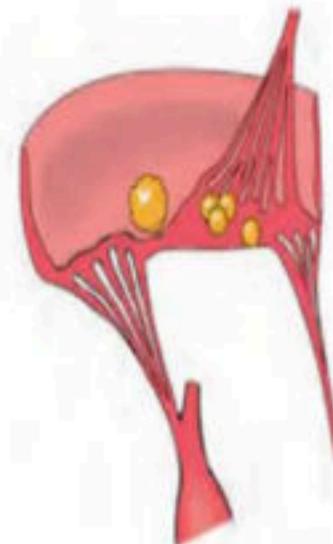
RHD



IE



NBTE



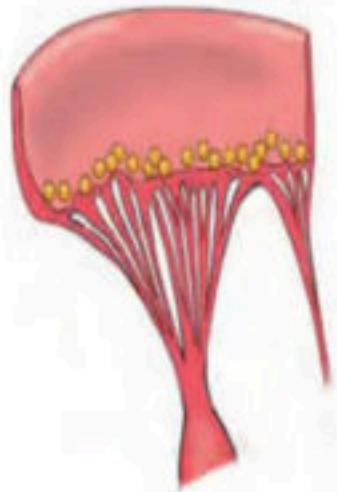
LSE

- **Valve** → The lesions are found most frequently on the **mitral > aortic > both mitral and aortic valves > rarely on the valves of the right heart.**

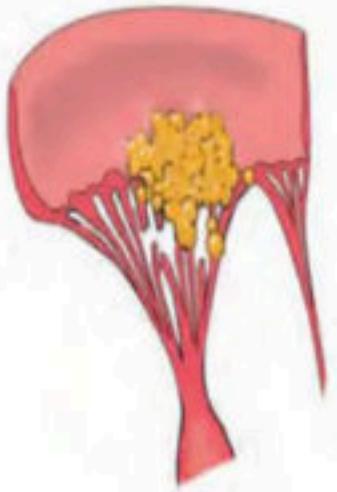
REMEMBER

- **Most commonly** involved valve in endocarditis is **mitral valve**.
- **In intravenous drug users** → **Tricuspid valve** is most commonly involved valve
- **In prosthetic valve endocarditis** → **Aortic valve** is the most commonly affected valve

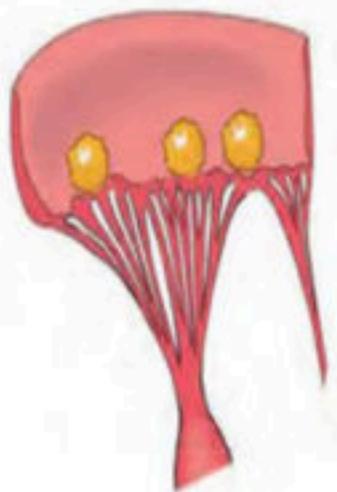
- Location → atrial surface of atrioventricular valves and ventricular surface of the semilunar valves
- They begin from the contact areas of the valve and extend along the surface of the valves and on to the adjacent endocardium



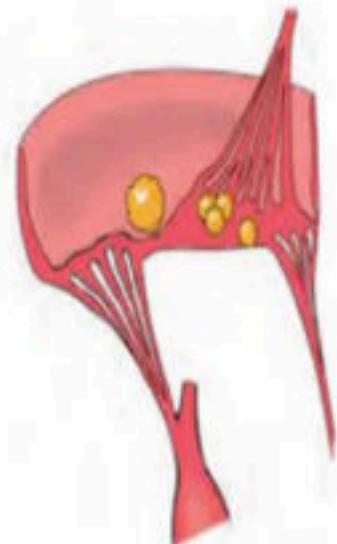
RHD



IE

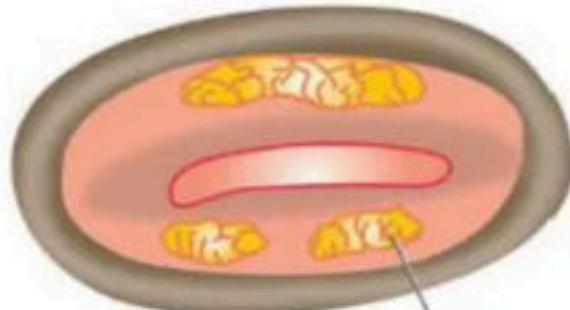


NBTE



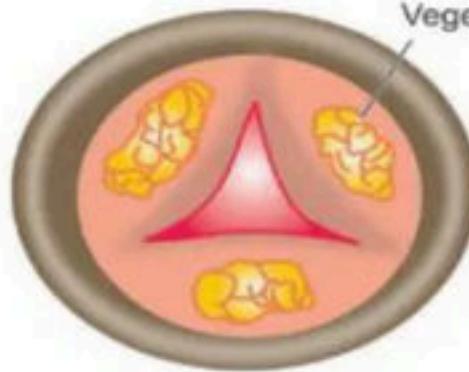
LSE

MITRAL VALVE (atrial surface)



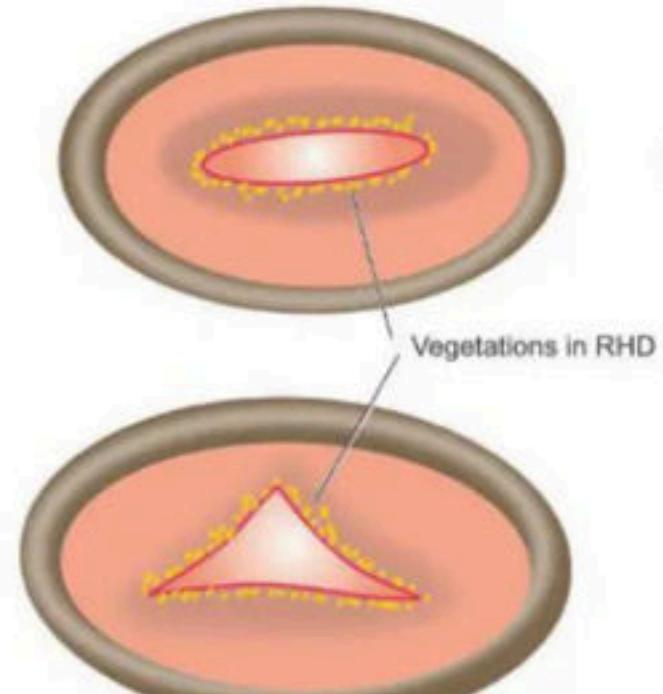
A

Vegetations in BE

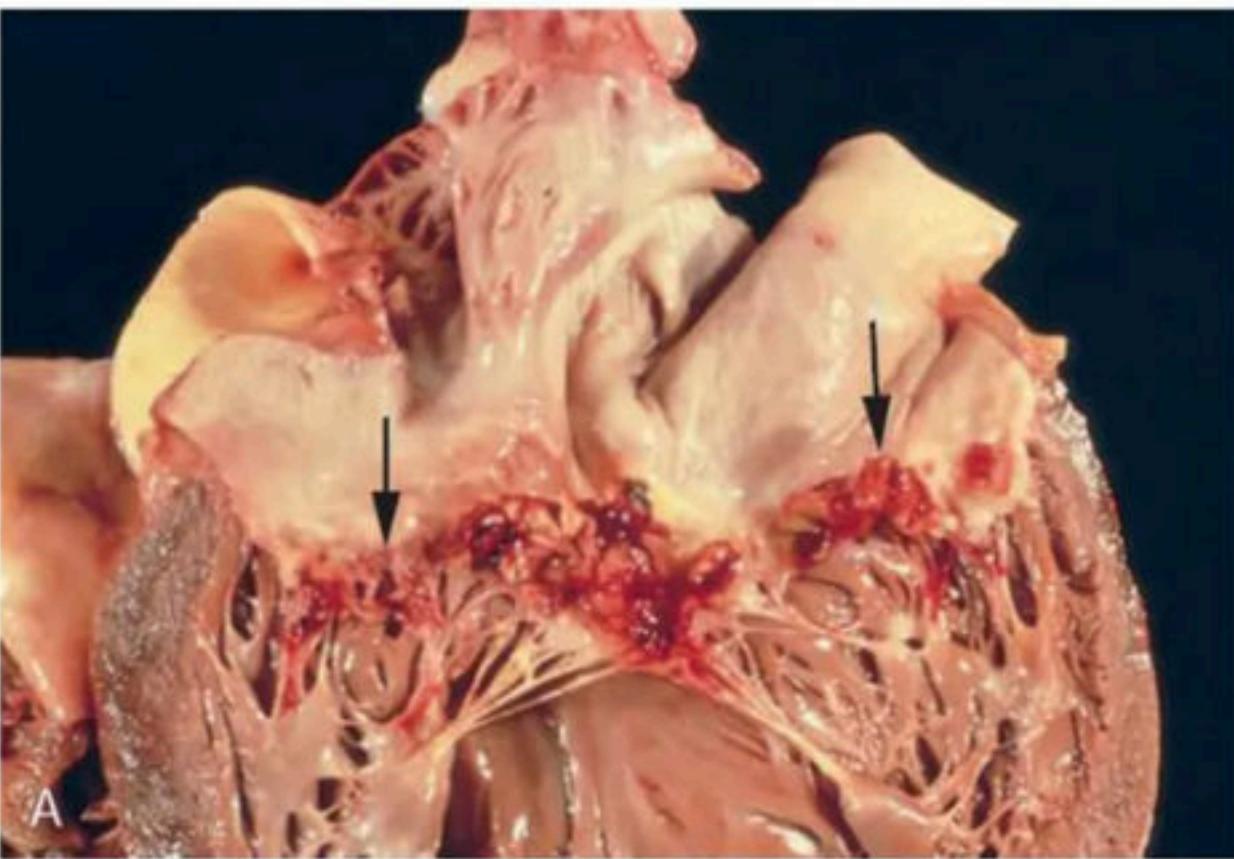


AORTIC VALVE (VENTRICULAR SURFACE)

MITRAL VALVE (ATRIAL SURFACE)



AORTIC VALVE (VENTRICULAR SURFACE)



Dr. PRIYANKA SACHDEV

Deformity →

- **Ulceration or perforation** of the underlying valve leaflet
- **Myocardial abscesses**

REMEMBER

- Most destructive vegetations are of
infective endocarditis

Microscopically

- The vegetations of BE consist of **3 zones** →
 - i) **The outer layer or cap** consists of eosinophilic material composed of **fibrin and platelets**.
 - ii) **Basophilic zone** containing **colonies of bacteria**.
 - iii) **The deeper zone** consists of **non-specific inflammatory reaction in the cusp itself**

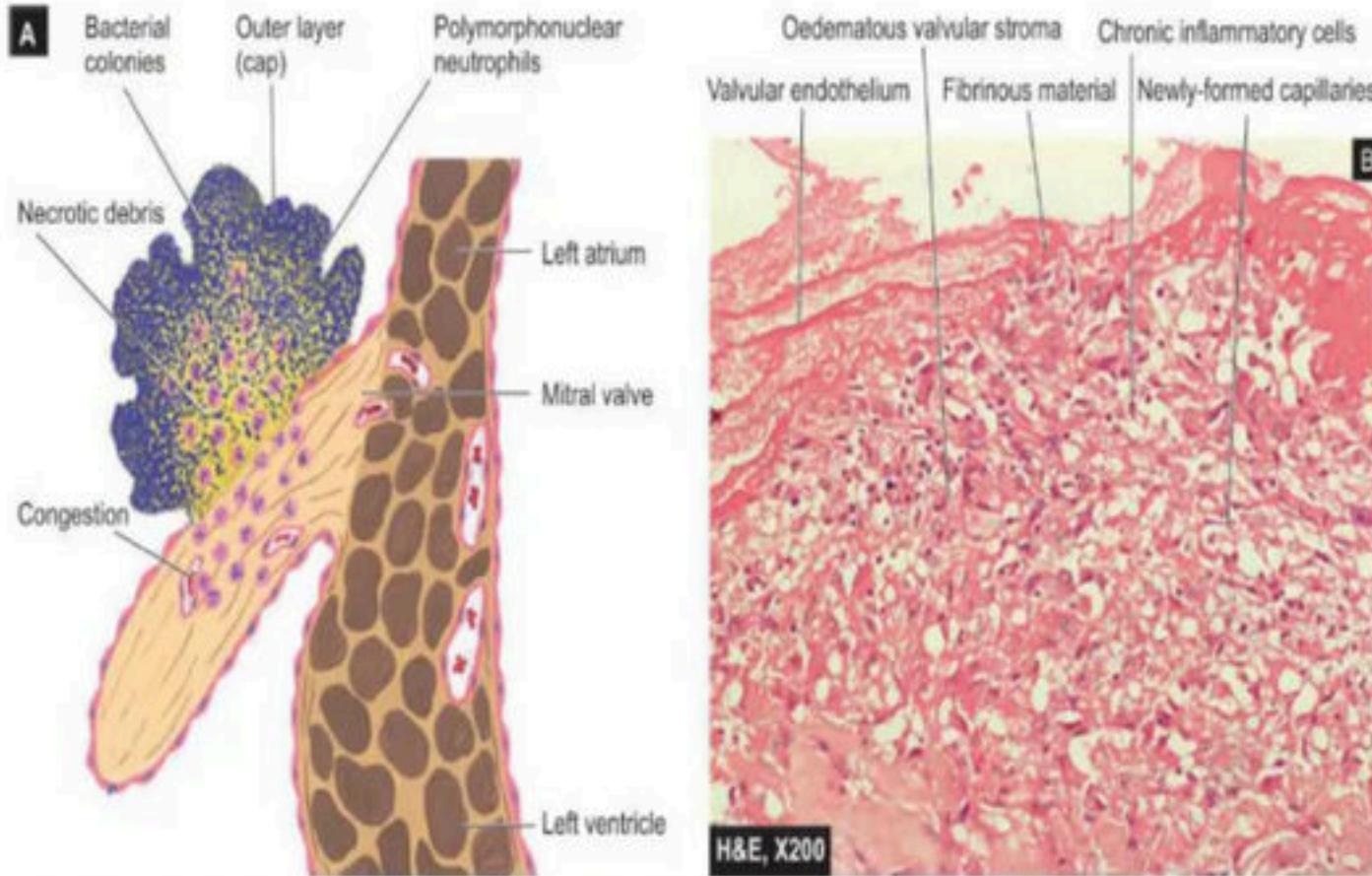


Figure 14.30 Infective endocarditis. A, Microscopic structure of a vegetation of BE on the surface of mitral valve in sagittal section. B, Section of the mitral valve shows fibrin cap on luminal surface, layer of bacteria, and deeper zone of inflammatory cells, with prominence of neutrophils.

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Rheumatic Fever	Non bacterial Thrombotic	Libman Sack's Endocarditis (SLE)	Infective Endocarditis (Marantic)
<ul style="list-style-type: none"> • Small, warty^(AI 00) • Firm^(AI 00) • Friable (but less than those of NBTE) • Along lines of closure^(AI 00, PGI 98) • Sterile (no organisms) • No destruction of under-lying valves or myocardium • Seen in Rheumatic fever 	<p>Small (1-5 mm)^(AI 13)</p> <ul style="list-style-type: none"> • Friable (<i>produce emboli</i>)^(AI 13) • Along lines of closure • Sterile • No destruction^(AI 13) • No inflammation^(AI 13) • Seen in hypercoagulable states eg cancer, promyelo cytic leukemia increased estrogenic 	<ul style="list-style-type: none"> • Medium sized (small)^(AI 00) • Flat^(AI 00), Verrucous • Irregular • On surface of cusps both surfaces^(NEET) may be involved most common being the undersurface, less often on mural endocardium^(AI 00) • In pockets of valves^(AI 00) • Sterile • Seen in SLE^(AI 13) 	<ul style="list-style-type: none"> • Large • Friable^(AI 10) (easily detachable) • bulky • Irregular • On upper surface of cusps • Less often on mural endocardium • Non-Sterile^(PGI 99) (bacteria) • Ulcerates or perforates underlying valve (or myocardium) • Seen in I.E.

OVERVIEW

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Pathogenesis

Gross→

- a) Vegetations or Verrucae
- b) Valves
- c) Location
- d) Deformity

Microscopy

Complications

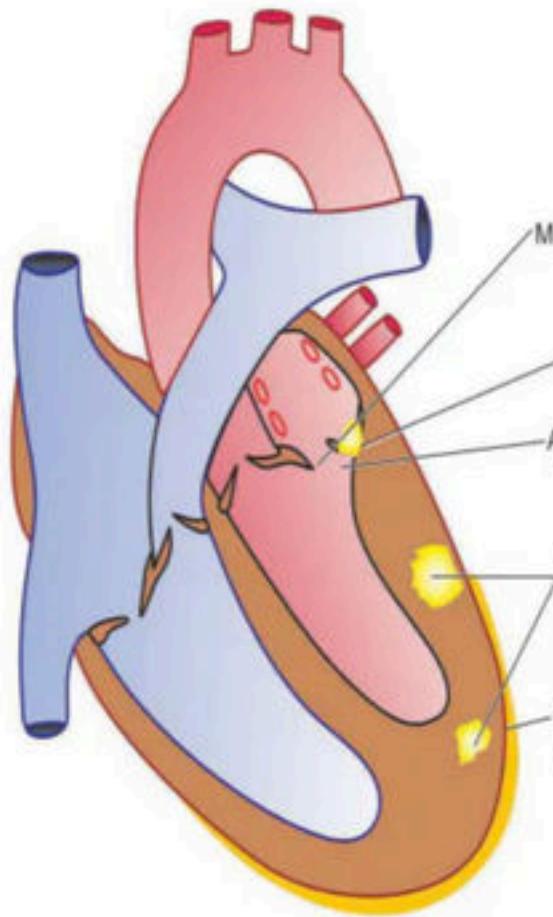
Modified Duke criteria

COMPLICATIONS AND SEQUELAE

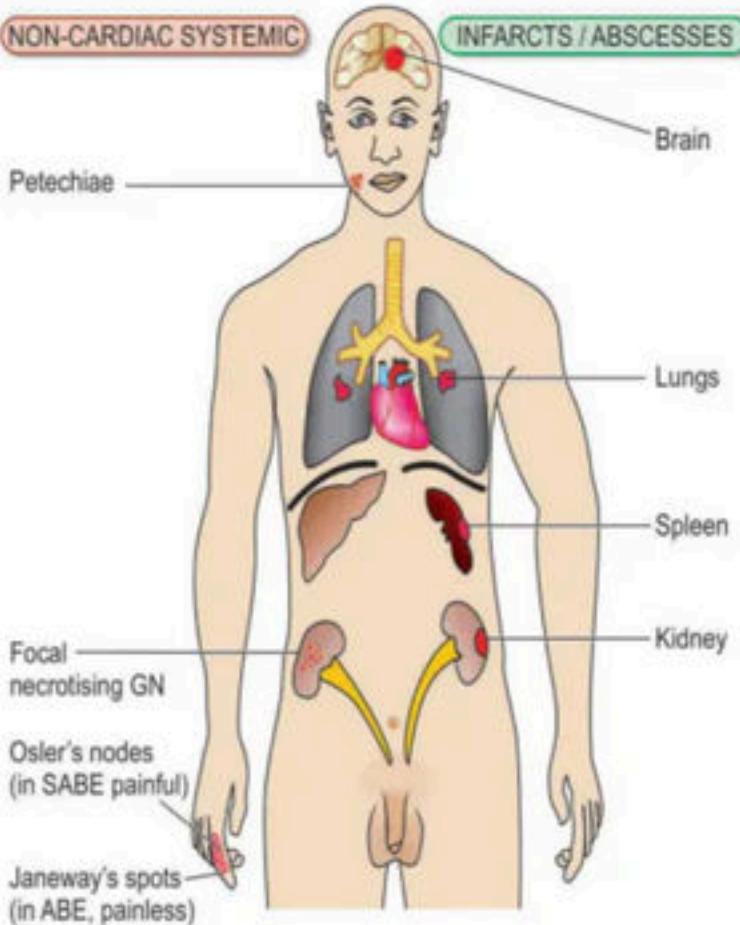
- Cardiac
- Extracardiac

Cardiac complications

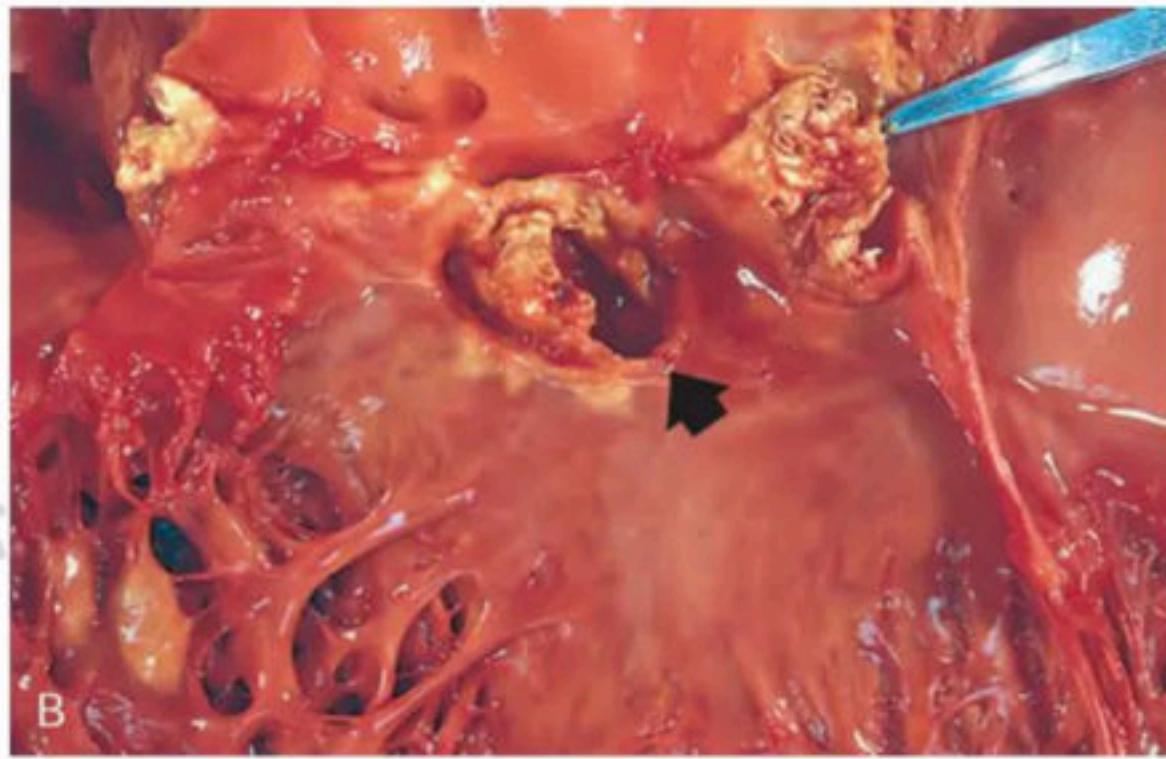
- i) Valvular stenosis or insufficiency
- ii) Perforation, rupture, and aneurysm of valve leaflets
- iii) Abscesses in the valve ring
- iv) **Myocardial abscesses (most common)**
- v) Suppurative pericarditis
- vi) Cardiac failure



A. CARDIAC SEQUELAE OF INFECTIVE ENDOCARDITIS



B. EXTRA-CARDIAC COMPLICATIONS



B

Dr. PRIYANKA SACHDEV

B. Extracardiac complications

- i) Emboli originating from the **left side of the heart** and entering the systemic circulation affect organs **like the spleen, kidneys, and brain causing infarcts, abscesses.**
- ii) Emboli arising from **right side of the heart** enter the pulmonary circulation and produce **pulmonary abscesses.**

- In SABE, there are painful, tender nodules on the finger tips of hands and feet called Osler's nodes
- In ABE there is appearance of painless, non-tender subcutaneous maculopapular lesions on the pulp of the fingers called Janeway's spots.

Osler's nodes



Dr. PRIYANKA SACHDEV



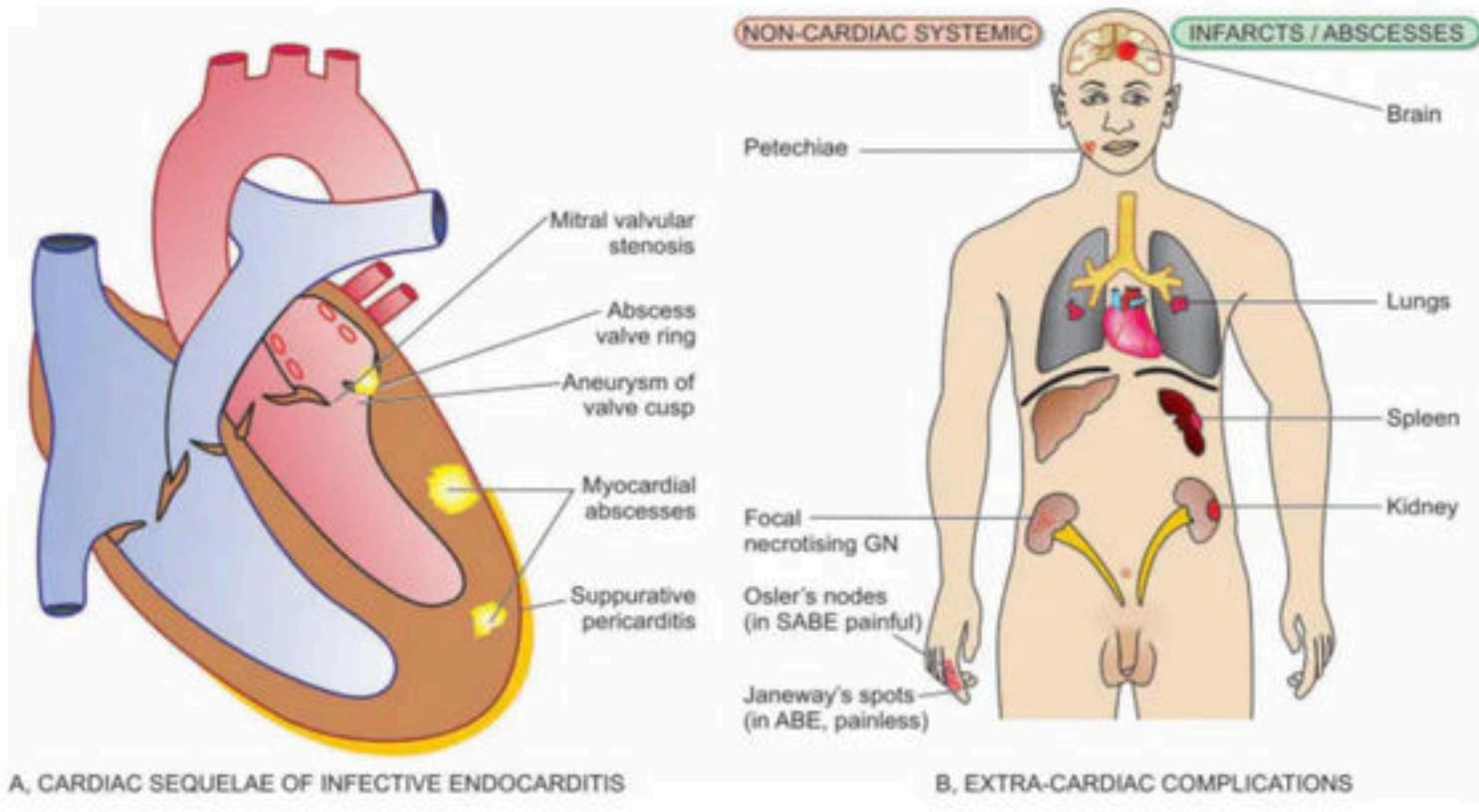
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Osler Node



Janeway Lesion





Modified Duke criteria

Major

Blood culture(s) positive for a characteristic organism or persistently positive for an unusual organism

Echocardiographic identification of a valve-related or implant-related mass or abscess, or partial separation of artificial valve

New valvular regurgitation

Minor

Predisposing heart lesion or intravenous drug use

Fever

Vascular lesions, including arterial petechiae, subungual/splinter hemorrhages, emboli, septic infarcts, mycotic aneurysm, intracranial hemorrhage, Janeway lesions[†]

Immunological phenomena, including glomerulonephritis, Osler nodes,[‡] Roth spots,[§] rheumatoid factor

Microbiologic evidence, including a single culture positive for an unusual organism

Echocardiographic findings consistent with but not diagnostic of endocarditis, including worsening or changing of a preexistent murmur

Pathologic Criteria

Microorganisms, demonstrated by culture or histologic examination, in a vegetation, embolus from a vegetation, or intracardiac abscess

Histologic confirmation of active endocarditis in vegetation or intracardiac abscess

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FEATURE	RHEUMATIC	LIBMAN-SACKS	NON-BACTERIAL THROMBOTIC	BACTERIAL (INFECTIVE)
1. Valves commonly affected	Mitral alone; mitral and aortic combined	Mitral, tricuspid	Mainly mitral; less often aortic and tricuspid	Mitral; aortic; combined mitral and aortic
2. Location on valve cusps or leaflets	Occur along the line of closure, atrial surface of atrioventricular valves and ventricular surface of semilunar valves	Occur on both surfaces of valve leaflets or cusps, in the valve pockets	Occur along the line of closure	SABE more often on diseased valves: ABE on previously normal valves; location same as in RHD
3. Macroscopy				
4. Microscopy	Composed of fibrin with superimposed platelet thrombi and no bacteria. Adjacent and underlying endocardium shows oedema, proliferation of capillaries, mononuclear inflammatory infiltrate and occasional Aschoff bodies.	Composed of fibrinoid material with superimposed fibrin and platelet thrombi and no bacteria. The underlying endocardium shows fibrinoid necrosis, proliferation of capillaries and acute and chronic inflammatory infiltrate including the haematoxylin bodies of Gross.	Composed of degenerated valvular tissue, fibrin-platelets thrombi and no bacteria. The underlying valve shows swelling of collagen, fibrinoid change, proliferation of capillaries but no significant inflammatory cell infiltrate.	Composed of outer eosinophilic zone of fibrin and platelets, covering colonies of bacteria and deeper zone of non-specific acute and chronic inflammatory cells. The underlying endocardium may show abscesses in ABE and inflammatory granulation tissue in the SABE.

Rheumatic Fever	Non bacterial Thrombotic	Libman Sack's Endocarditis (SLE)	Infective Endocarditis (Marantic)
<ul style="list-style-type: none"> • Small, warty^(AI 00) • Firm^(AI 00) • Friable (but less than those of NBTE) • Along lines of closure^(AI 00, PGI 98) • Sterile (no organisms) • No destruction of under-lying valves or myocardium • Seen in Rheumatic fever 	<p>Small (1-5 mm)^(AI 13)</p> <ul style="list-style-type: none"> • Friable (<i>produce emboli</i>)^(AI 13) • Along lines of closure • Sterile • No destruction^(AI 13) • No inflammation^(AI 13) • Seen in hypercoagulable states eg cancer, promyelo cytic leukemia increased estrogenic 	<ul style="list-style-type: none"> • Medium sized (small)^(AI 00) • Flat^(AI 00), Verrucous • Irregular • On surface of cusps both surfaces^(NEET) may be involved most common being the undersurface, less often on mural endocardium^(AI 00) • In pockets of valves^(AI 00) • Sterile • Seen in SLE^(AI 13) 	<ul style="list-style-type: none"> • Large • Friable^(AI 10) (easily detachable) • bulky • Irregular • On upper surface of cusps • Less often on mural endocardium • Non-Sterile^(PGI 99) (bacteria) • Ulcerates or perforates underlying valve (or myocardium) • Seen in I.E.

POLLS 9

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Bacterial endocarditis is most commonly caused by-

- a) α -Hemolytic streptococci
- b) β -Hemolytic streptococci
- c) Staphylococcus aureus
- d) Cardiobacterium
- e) Staph epidermidis

C

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The risk of developing infective endocarditis is the least in patient with -

- a) Small VSD
- b) Severe aortic regurgitation
- c) Severe mitral regurgitation
- d) Large ASD

D

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Non-sterile vegetations are seen in -

- a) Rheumatic fever
- b) Infective endocarditis
- c) Non bacterial thrombotic endocarditis
- d) Libman sack's endocarditis

B

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Flat vegetations in pockets of valves are due to -

- a) Rheumatic heart disease
- b) Libman sacks Endocarditis
- c) NBTE
- d) Infective endocarditis

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B

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Which type of endocarditis has vegetation on both sides of the valves -

- a) Infective endocarditis
- b) Libman Sack's endocarditis
- c) RF
- d) None

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B

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Which of these is TRUE regarding Libman sach's lesion -

- a) Causes perforation of valves
- b) Involves multiple valves
- c) Consists of large vegetation
- d) Vegetations on the surface of valve spreads to mural endocardium

D

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Vegetations in Libman sacendocarditis are -

- a) Large and fragile
- b) Small warty along the line of closure of valve
- c) Small or medium sized on either or both sides of valve
- d) Small bland vegetations

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C

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Vegetations below the AV valves are present in -

- a) Libman sach's endocarditis
- b) Chronic rheumatic carditis
- c) Acute rheumatic carditis
- d) Non thrombotic endocarditis

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A

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In which of the following vegetation are friable and easily detachable from the cardiac valves -

- a) Rheumatic fever b) Rheumatoid heart
- c) SLE d) Infective endocarditis

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D

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Firm warty vegetations along the line of apposition of heart valves is present in -

- a) NBTE
- b) Bacterial endocarditis
- c) Rheumatic heart disease
- d) Libman Sach's endocarditis

Dr. V.

C

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Thank you for being awake



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