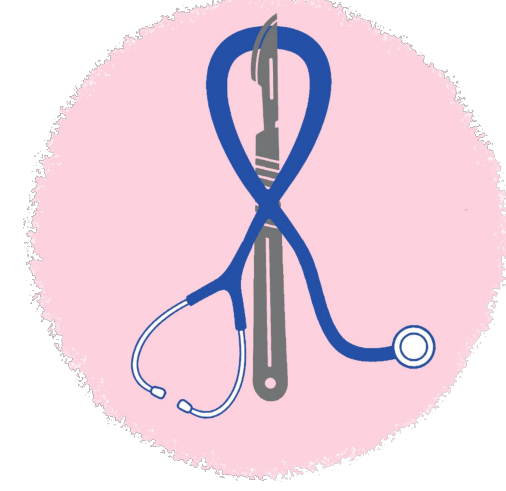




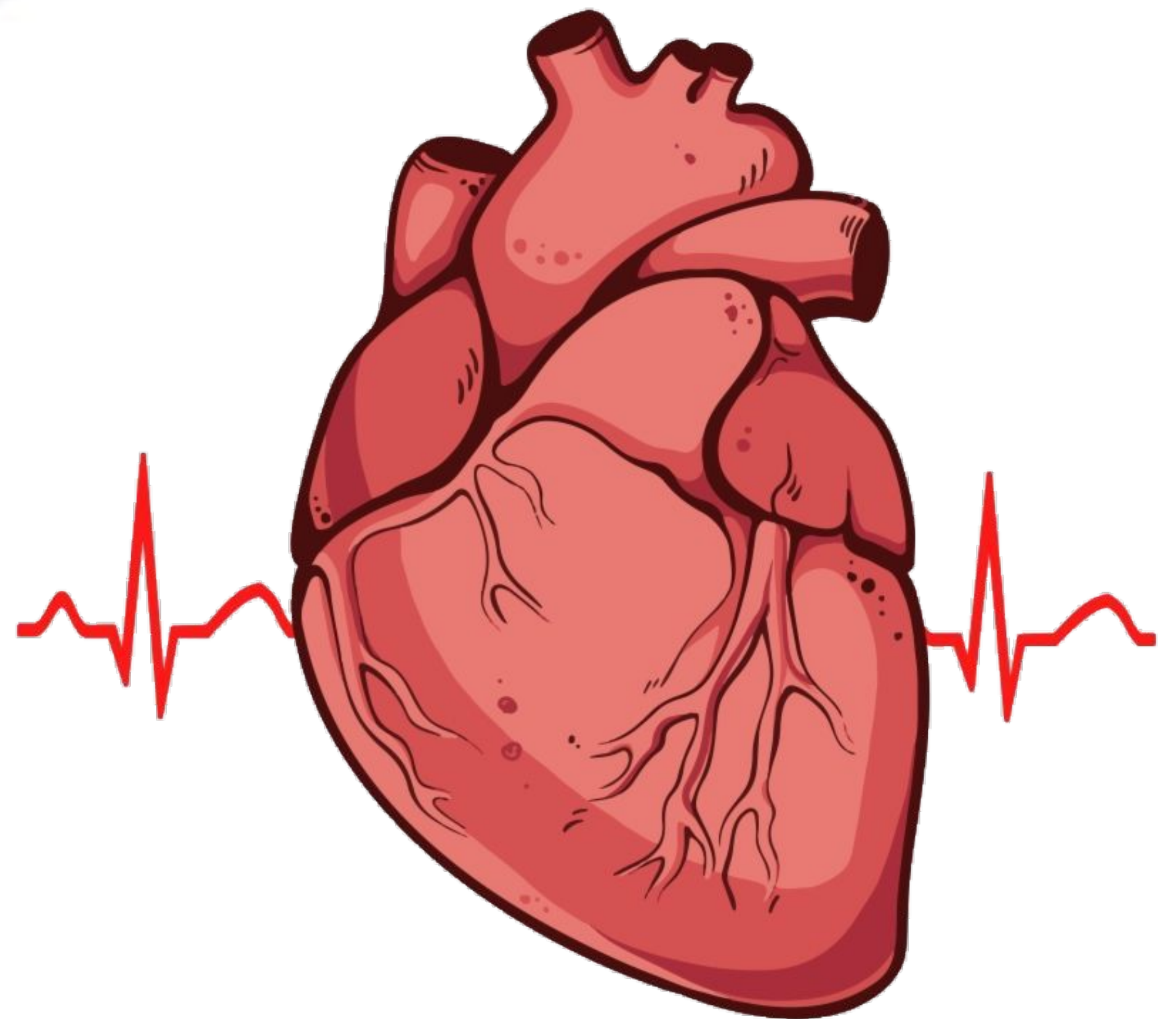
Pathology team 441

Revised & Reviewed  
by:  
Abdulaziz & Bahammam  
Faye Wael Sondi



**MED441**  
KING SAUD UNIVERSITY

# Rheumatic fever, endocarditis & heart valves



## Editing File

### Color Index:

- Main text
- **Important**
- Boys notes
- Girls notes
- Extra

[Click here for Prof  
notes](#)



# Objectives

1


Understands the clinicopathological features of rheumatic heart disease which is a major cause of acquired mitral and aortic valve diseases in the Kingdom of Saudi Arabia.

2

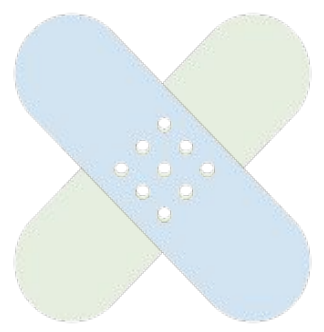
Know the pathological causes and pathophysiological consequences of stenosis and incompetence of all the cardiac valves but particularly the mitral and aortic valves.

3

Understands the pathology of infective endocarditis so as to be able to identify patients at risk and when appropriate ensure prophylactic treatment is given.

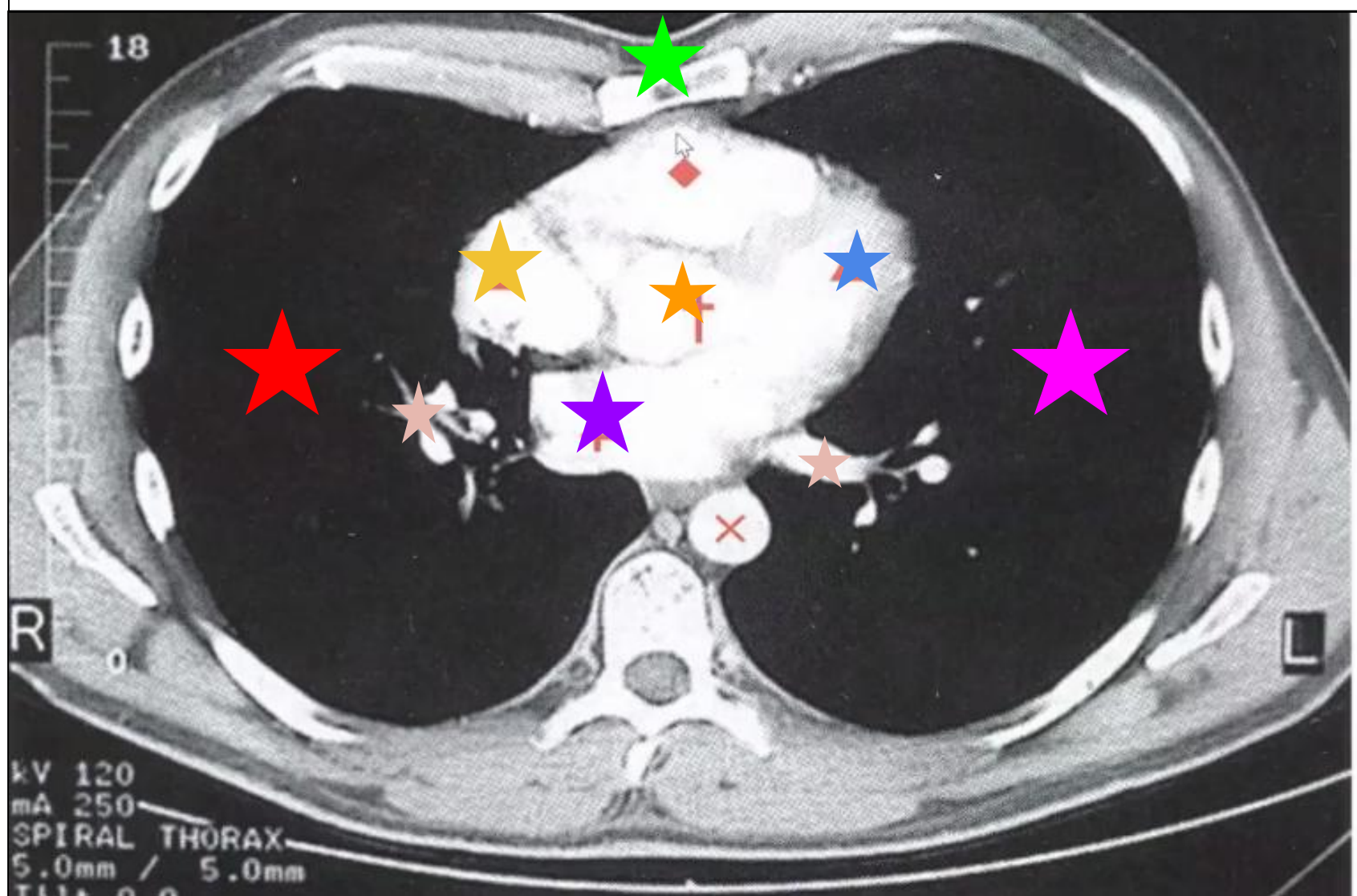
A decorative graphic in the bottom right corner consisting of several overlapping, curved, semi-transparent bands in shades of light blue, teal, and green, creating a sense of movement and depth.





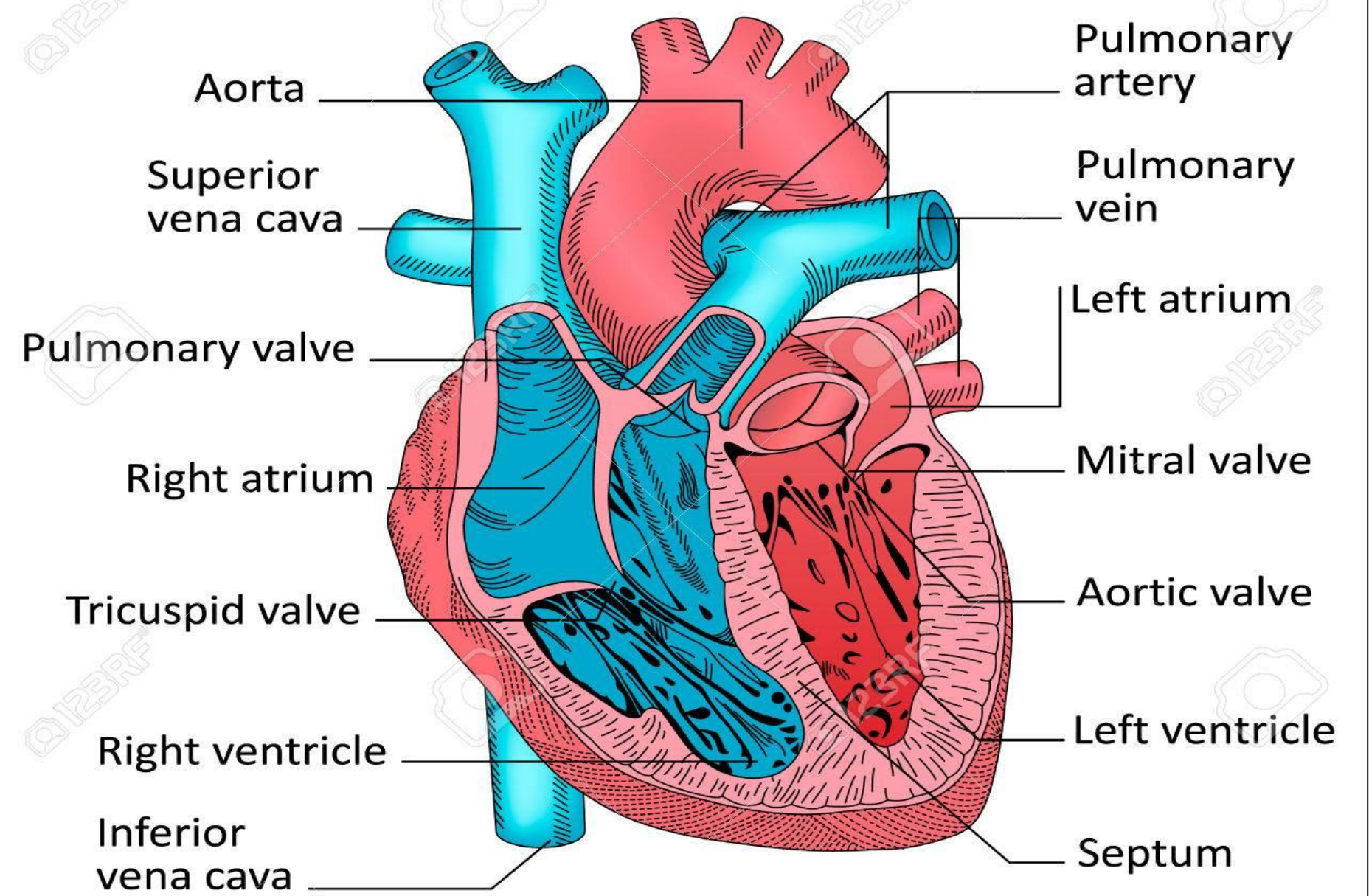
# Introduction

CT scan of the mediastinum



- Left lung
- Right lung
- Left ventricle
- Right ventricle
- Left atrium
- Right atrium
- Thoracic arch
- Branches of Pulmonary artery

ANATOMY OF THE HEART

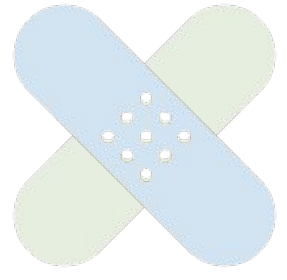


He explained the anatomy of the heart like this in the picture

The heart has 4 valves:

- aortic valve
- Tricuspid valve
- Mitral valve, which is the most important
- Pulmonary valve

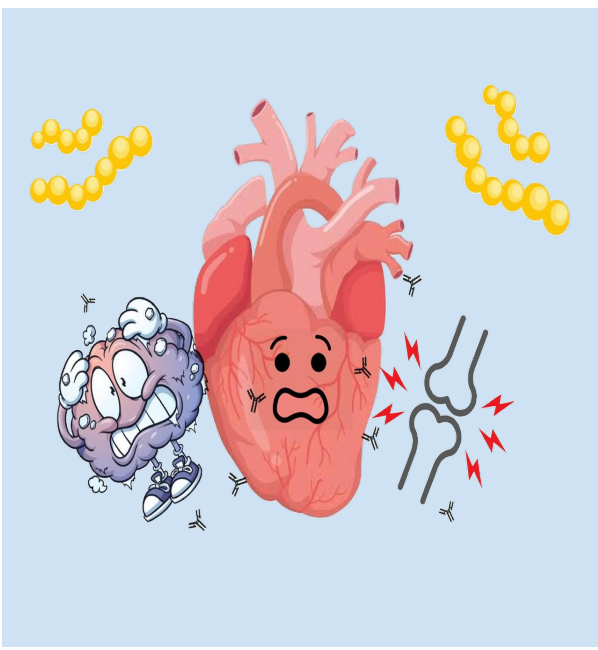




# Rheumatic heart disease & ACUTE RHEUMATIC FEVER

## Rheumatic heart disease

Rheumatic heart disease is a heart disease caused by **rheumatic fever**.  
It can be : **acute** or **chronic**



## Acute Rheumatic fever

- It is an acute, immune mediated, multi-system inflammatory disease that occurs a few weeks after, group A-beta hemolytic streptococcal infection.
- It is an acute post-streptococcal non-suppurative inflammatory disease with cardiac and extracardiac manifestations.

- The inflammation is mainly in the **heart, joints, central nervous system and skin.**
- Occurs in only 3% of patients with group A streptococcal pharyngitis.
- It is seen mainly in children, 5 to 15 years of age.
- Rheumatic fever is a major health problem in 3rd world countries and in crowded, low socioeconomic urban areas.
- The incidence and mortality of rheumatic fever has declined over the past 30 years (due to improved socioeconomic condition and rapid diagnosis and treatment of strep. pharyngitis).

## Etiopathogenesis:

(it is not yet clear and not completely understood).

1

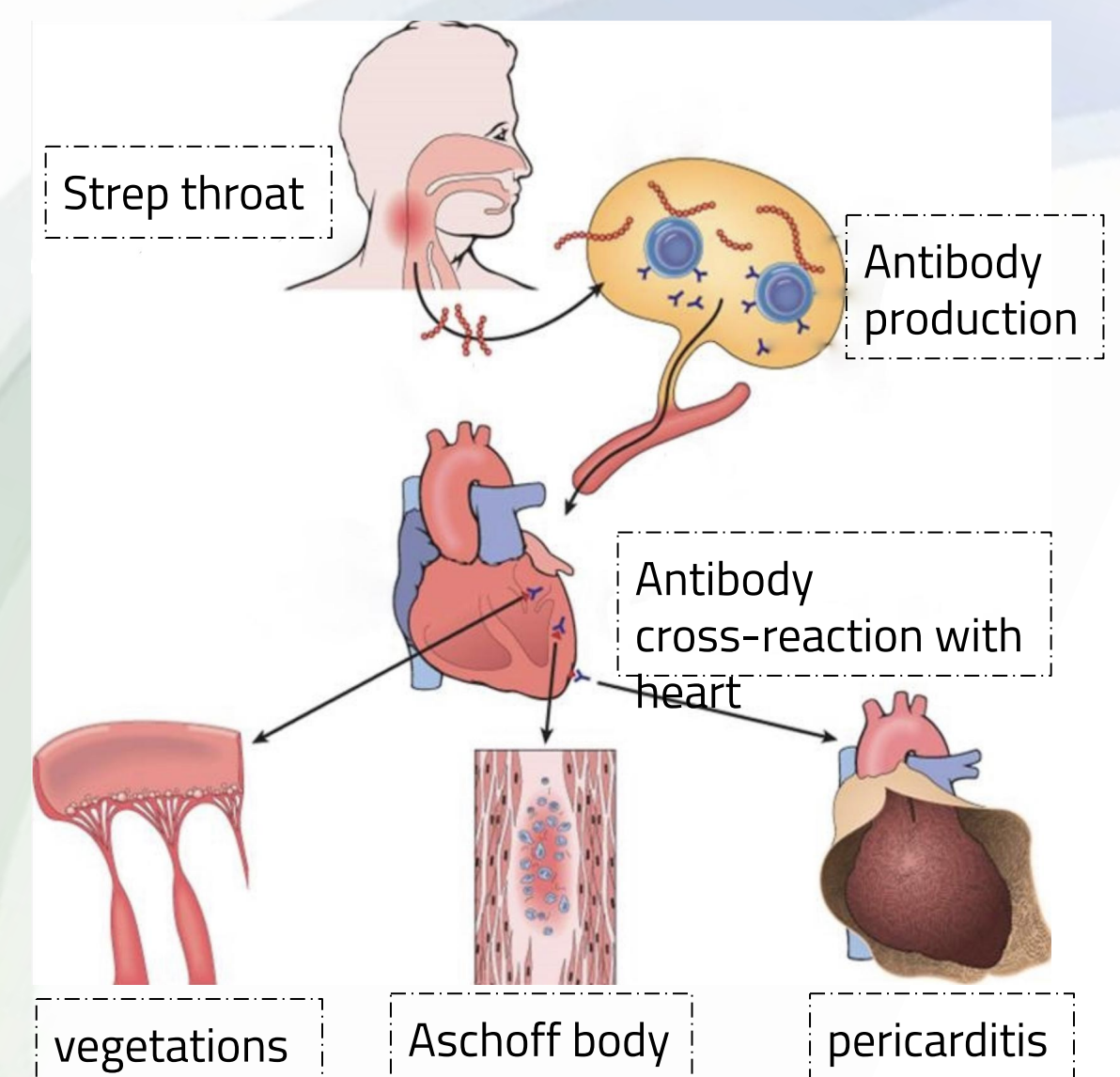
It is linked to streptococcal infection. Disease occurs 1 to 5 weeks after pharyngeal infection by Group A,  $\beta$ -Hemolytic Streptococcus.

2

It is most likely an **immune mediated process** in which the causative organisms (streptococci) stimulates in the formation of antibodies these antibodies cross react with certain antigens present in the heart and joints, the antigen antibody reaction leads to **inflammation**.

3

Repeated attacks or a single severe attack can lead to chronic rheumatic heart disease leading to cardiac failure.





# Acute rheumatic heart disease (RHD)

## Cardiac manifestations of Rheumatic FEVER

- Also called acute rheumatic heart disease or acute rheumatic carditis/ pancarditis.
- Patients present with **pancarditis**.
- **Pancarditis** is inflammation in all 3 layers of the heart **endocardium, myocardium and pericardium**.

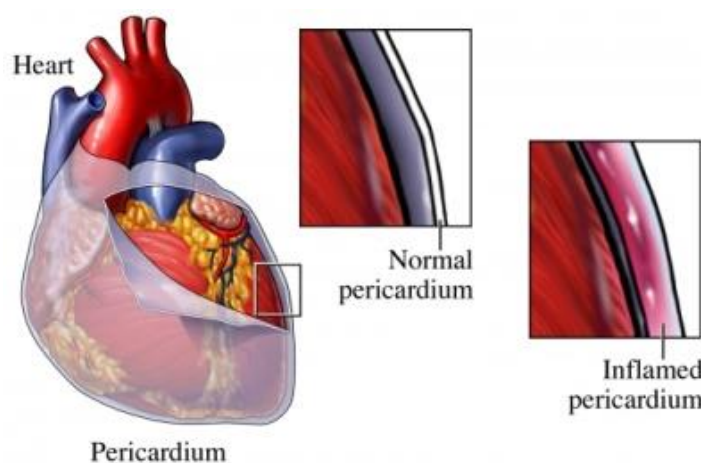
## Cardiac manifestations

### 1- Pericarditis

#### inflammation of Pericardium.

fibrinous or serofibrinous secretion in the pericardium between visceral and parietal layer.

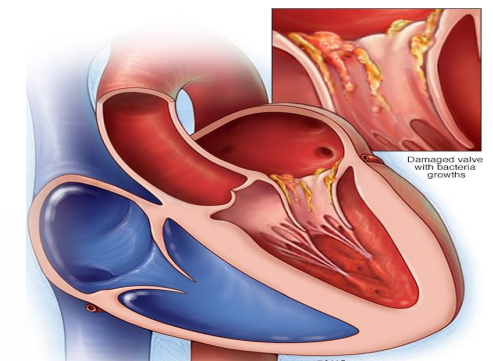
- **Bread and butter appearance**



### 2- Endocarditis

#### inflammation of Endocardium. (including heart valves and chordae tendineae)

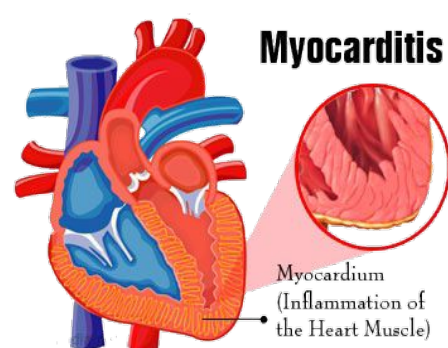
- results in fibrin deposition on valve leaflets forming tiny thrombi along lines of closure called **rheumatic vegetations**.
- **Mitral and aortic valve** are mainly involved.
- This acute inflammation may either resolve completely or progress to scarring with development of chronic fibrotic deformities of the heart valves and chordae tendineae **leading to chronic rheumatic heart disease many years later.**



### 3- Myocarditis

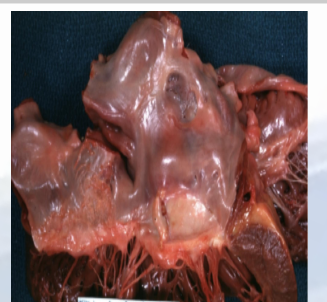
#### inflammation of Myocardium.

- many **Aschoff bodies**.
- **can cause sudden death.**



### 4- Subendocardial lesions

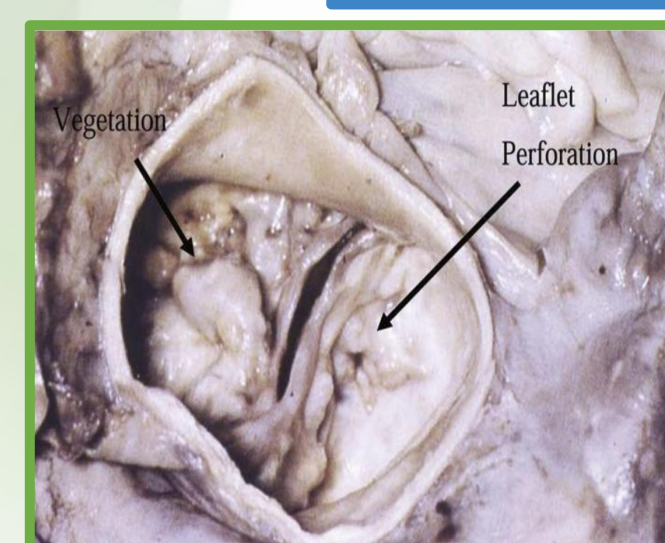
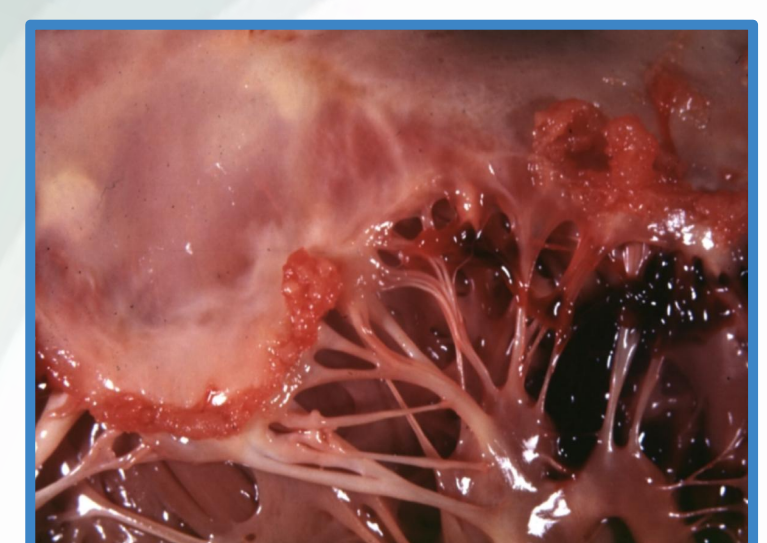
may be seen commonly in **left atrium**.  
Called **MacCallum plaques**.



MacCallum plaques

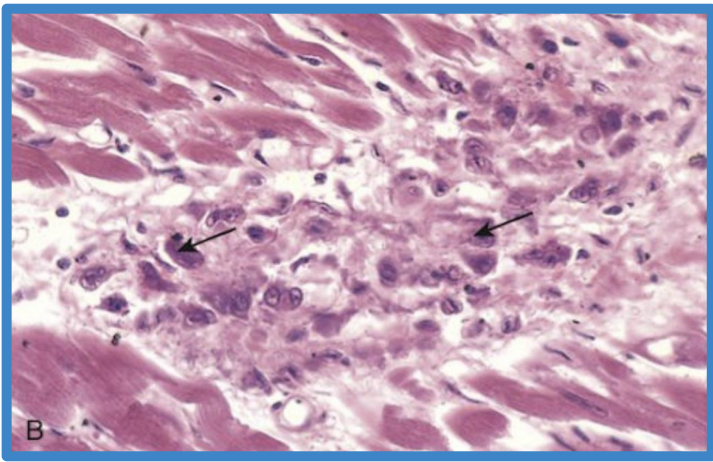
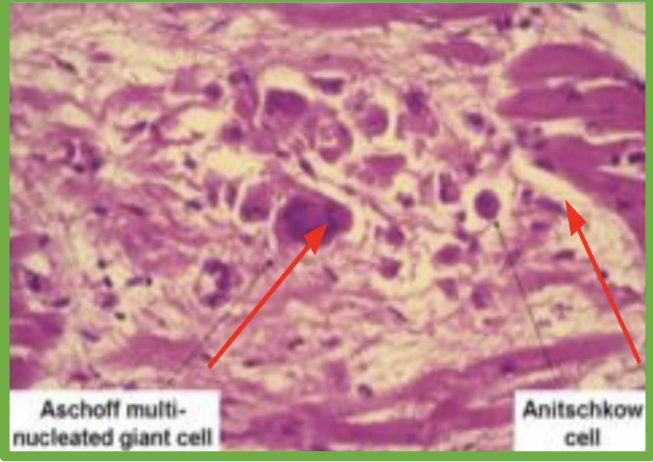
## What are Rheumatic vegetations?

- They are Tiny (size of pin's head), sessile arranged in a row and firmly with the underlying tissue.
- These are situated in the valve cusp, a few millimeters away from the free margin (this is the traumatized area)
- **From Robbins:** Valve involvement results in fibrinoid necrosis and fibrin deposition along lines of closure that cause disturbance in cardiac function.

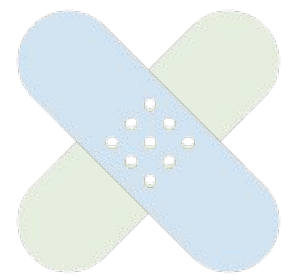




# Aschoff bodies & Extra cardiac

<b>Aschoff bodies</b> (characteristic lesion of acute rheumatic fever)	
<b>Definition</b>	They are multiple tiny granulomatous lesions of the heart. They are situated next to small arteries and are characteristically seen in the myocardium (rheumatic myocarditis).
<b>Components</b>	<p style="text-align: center;"><b>An Aschoff body, consists of:</b></p> <ul style="list-style-type: none"> <li>● a focus of eosinophilic <b>collagen necrosis</b> (representing the site of antibody-antigen reaction),</li> <li>● plump activated macrophages/ histiocytes called <b>Anitschkow/ caterpillar cells</b>.</li> <li>● Some of the macrophages become multinucleated to form <b>Aschoff giant cells</b>.</li> <li>● chronic inflammation.</li> </ul> <div style="display: flex; justify-content: space-around; align-items: center;">   </div>
<b>Site</b>	They are found mainly in the <b>myocardium</b> and <b>pericardium</b> . Uncommon in the <b>endocardium</b> and <b>heart valves</b> .
<b>They ultimately <u>heal</u> by fibrosis resulting in a <b>nodule of scar tissue</b>.</b>	

<b>Extra cardiac manifestations of Rheumatic FEVER</b> Involvement of other organs:	
<b>Joints: Arthralgia</b>	<b>Joints: Migratory polyarthriti</b> s which is fleeting arthritis in the large joints e.g. knee, ankle, elbow wrist etc. It is self limiting with no chronic deformities. Aschoff bodies may be present in the synovial membrane, joint capsule, ligament etc. with joint effusion.
<b>Skin:</b> skin nodules and erythema marginatum.	<b>Subcutaneous tissue:</b> Rheumatic nodules mainly seen over the bony prominences e.g. knuckle, elbow, patella etc.
<b>Lung:</b> uncommon, chronic interstitial inflammation and fibrinous pleuritis.	<b>Neurological disorder:</b> Sydenhem's chorea (St. Vitus' dance). <ul style="list-style-type: none"> <li>● characterized by series of rapid involuntary purposeless movements of the face and arms. This occurs late in the disease.</li> </ul>



# clinical features of acute Rheumatic fever

- 1 Peak incidence: 5-15 years.
- 2 **History of sore throat:** symptoms start 10 days to 6 weeks after by group A Streptococcal pharyngitis
- 3 By that time the symptoms start the throat/pharyngeal cultures are usually negative.
- 4 **Serum antistreptolysin O** (ASO titer/ antibodies to group A streptococcal antigens), **anti-DNAase B** and **antihyaluronidase are raised** & provide evidence of an infection with group A Streptococcus.
- 5 Acute symptoms usually subside within 3 months.
- 6 The mortality from acute rheumatic carditis is low.

There is **no** specific test for rheumatic fever.  
 The diagnosis is made based on the clinical findings when either:  
**1. two major or**  
**2. one major and two minor**  
 clinical features / criteria are met. This is called as **the Jones criteria.**

## Jones criteria

Major criteria	Minor criteria
<b>Carditis:</b> Murmurs, pericardial friction rubs, weak heart sounds, tachycardia and arrhythmias cardiomegaly, pericarditis, and congestive heart failure.	<b>Elevated acute phase reactants:</b> <ul style="list-style-type: none"> <li>● Elevated ESR (erythrocyte sedimentation rate).</li> <li>● Increased CRP (C-Reactive protein).</li> <li>● leukocytosis</li> </ul>
<b>Migratory polyarthritits of the large joints</b>	<b>Arthralgia</b>
<b>Erythema marginatum of the skin</b>	<b>Fever</b>
<b>Subcutaneous nodules</b>	<b>ECG changes</b> <b>Prolonged PR interval</b>
<b>Sydenhem's chorea (St. Vitus' dance)</b>	<b>Previous rheumatic fever</b>

Mnemonic: "JONES CAFE PAL"

Major Criteria	Minor Criteria
J Joint Involvement	C CRP Increased
O O looks like a heart = myocarditis	A Arthralgia
N Nodules, subcutaneous	F Fever
E Erythema marginatum	E Elevated ESR
S Sydenham chorea	P Prolonged PR Interval
	A Anamnesis of Rheumatism
	L Leukocytosis

Thanks to 438



# CHRONIC RHEUMATIC HEART DISEASE

## Components of rheumatic fever

Myocarditis and Pericarditis

Typically resolve without permanent sequelae.

Acute valvulitis or Chordae tendinitis

-Heals by **fibrosis** (scarring). -Irreversible deformity.  
 -Severe valvular scarring develops months or years **after** acute RF.  
 -Most harmful effect of rheumatic disease is due to involvement of cardiac valves → The valve leaflets develop diffuse fibrosis, become thickened, shrunken and less movable.

**This can lead to:**

- Cardiac failure
- Thromboembolism
- Infective endocarditis

## Valves affected in chronic rheumatic heart disease:

**Mitral valve alone**  
Most common

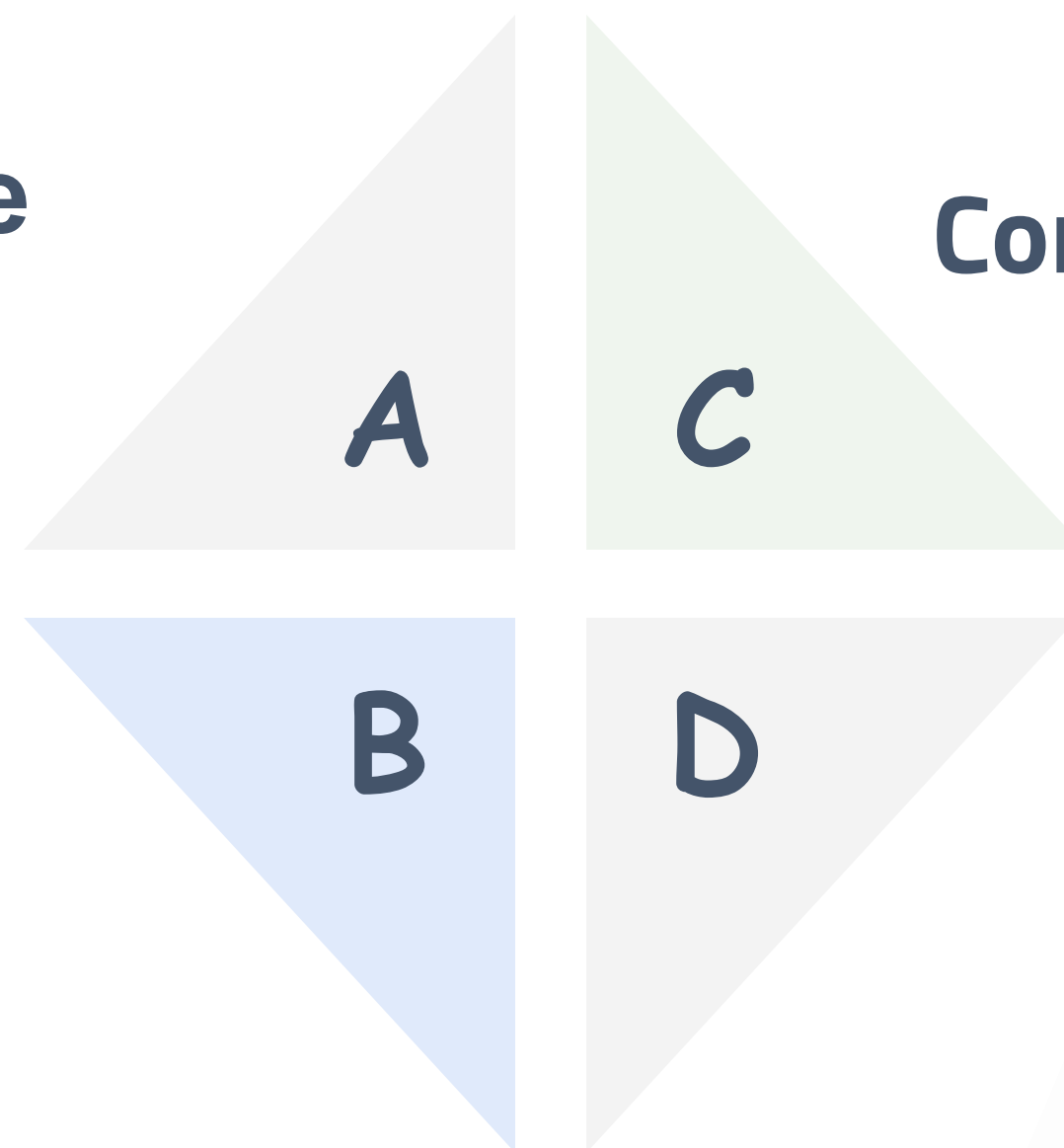
**Combined mitral/aortic valve**

Usually after Mitral valve damage

**Pulmonary valve**  
practically never affected

**Tricuspid valve**

Rarely affected



## Types of damage to the valves:

1

### Stenosis

fibrosis of valve leaflets (reduction of diameter)

2

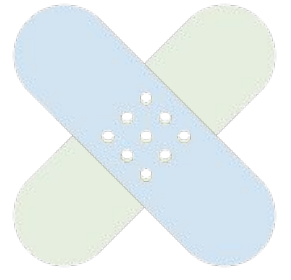
### Regurgitation

Fibrosis of chordae tendineae (improper closure)

**Left side** of heart is **more commonly** involved than the right

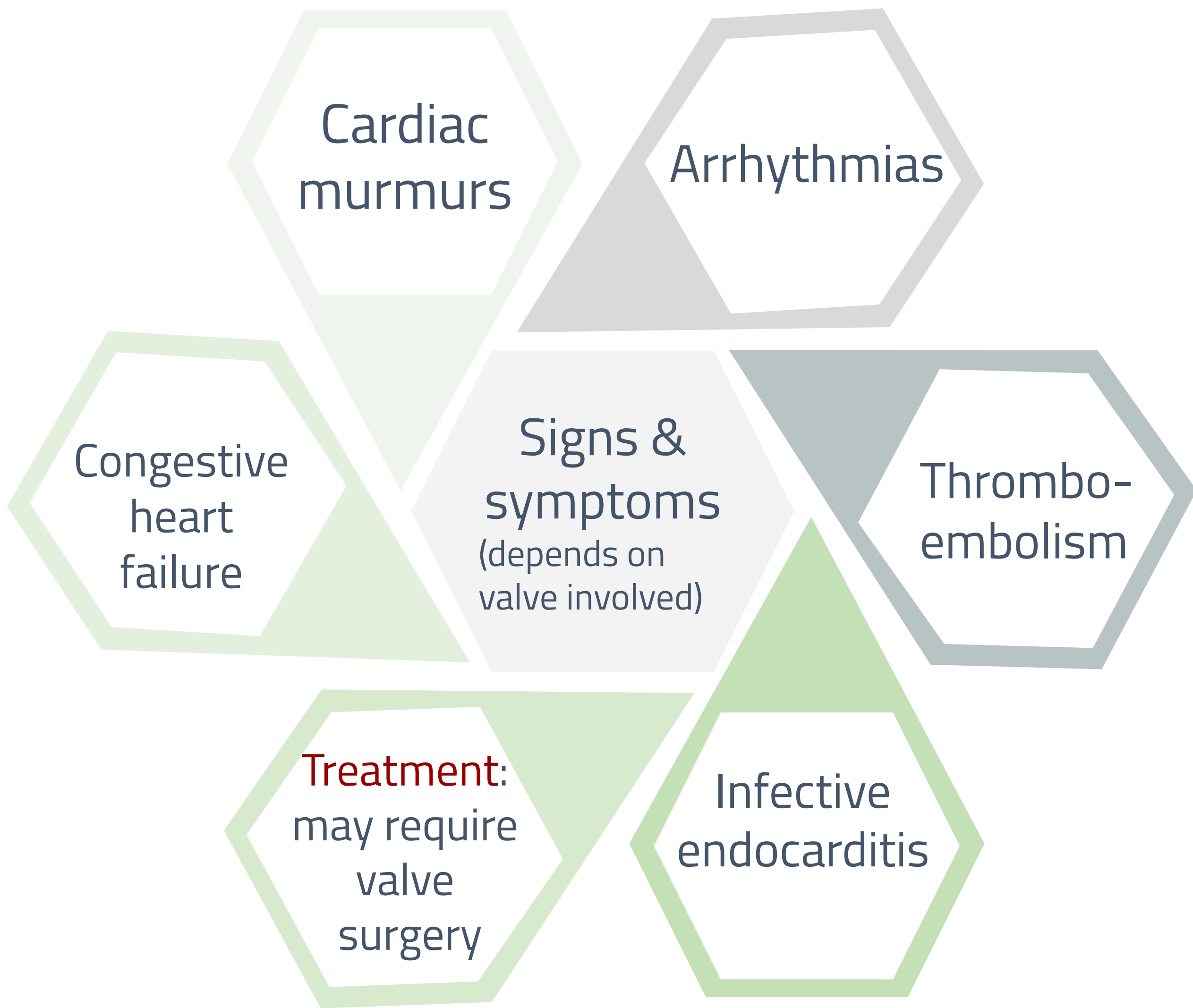
Therefore patient can have **mitral** stenosis (most common), **mitral** regurgitation, **aortic** stenosis and **aortic** regurgitation. (all are structures found on left side of heart).





# Cont. Chronic rheumatic heart disease

Manifestations occurs many years after the initial episode of rheumatic fever



## Mural thrombi

form in cardiac chambers. They give rise to **thromboemboli**, which can produce infarcts in various organs

## Bacterial infective endocarditis

the scarred valves of rheumatic heart disease provide an attractive environment for bacteria to grow.



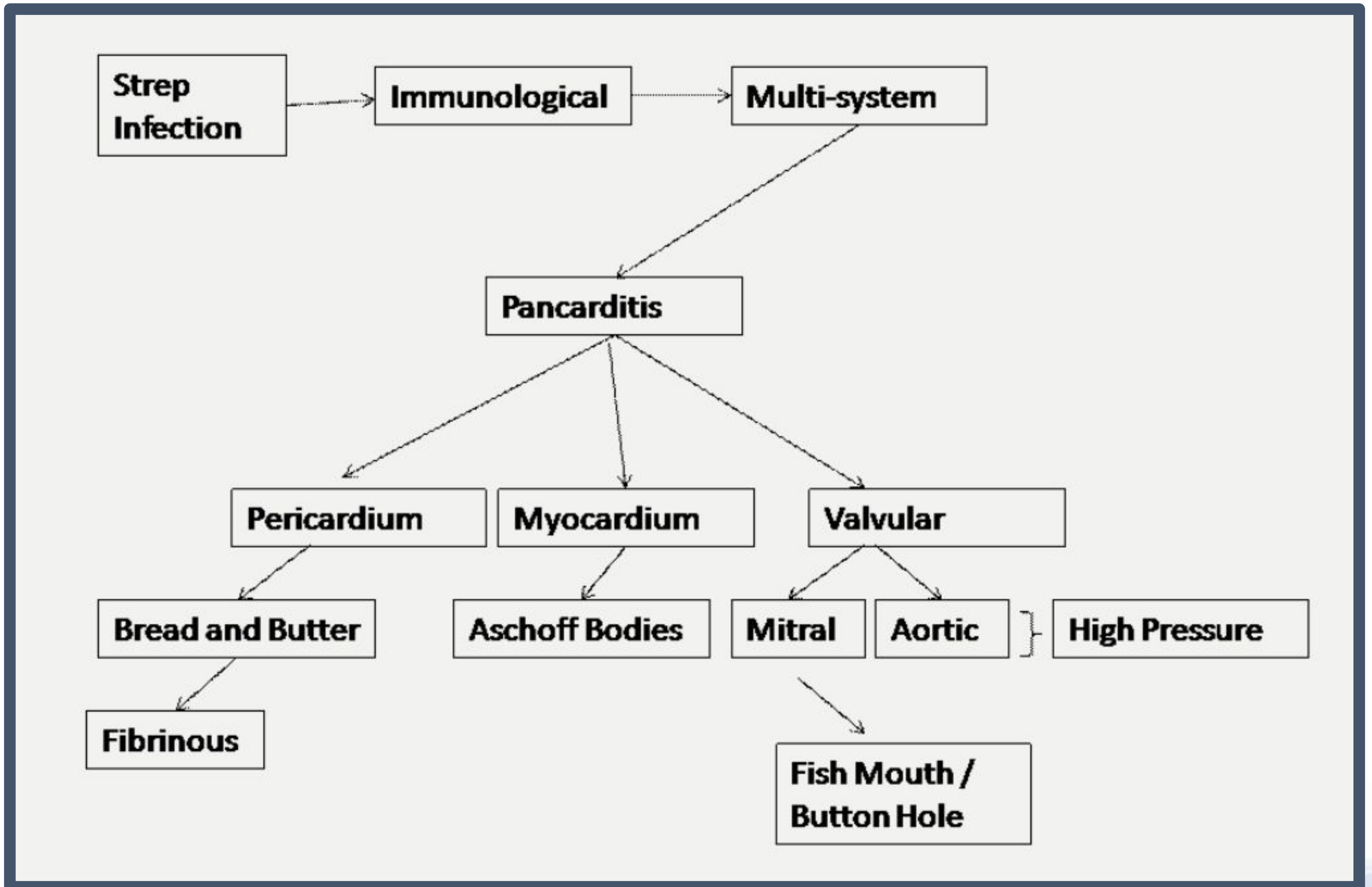
## Atrial fibrillation

## Congestive heart failure

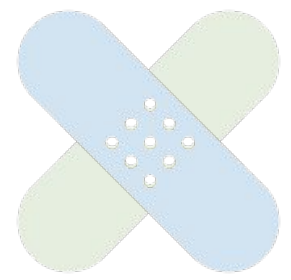
## Adhesive pericarditis



# Summary:







# INFECTIVE ENDOCARDITIS

## Definition:

- infection of the cardiac valves surface of the endocardium, forms an adherent mass of thrombotic debris that contains microorganisms.
- **Mitral valves** are the most common sites of IE followed by aortic valve
- Single or multiple vegetations, involve one or valve(s), differ in appearance according to the causative agent.

	Acute IE	Subacute IE
Causative organism	<b>staphylococcus aureus</b> (highly virulent)	<b>alpha-hemolytic streptococci viridans</b> (low virulence)
Infected valves	infects even healthy valves	previously abnormal/ damaged valves
Host reaction	has little local host reaction	induces a local inflammatory reaction.
Progress	progresses rapidly	progress slowly

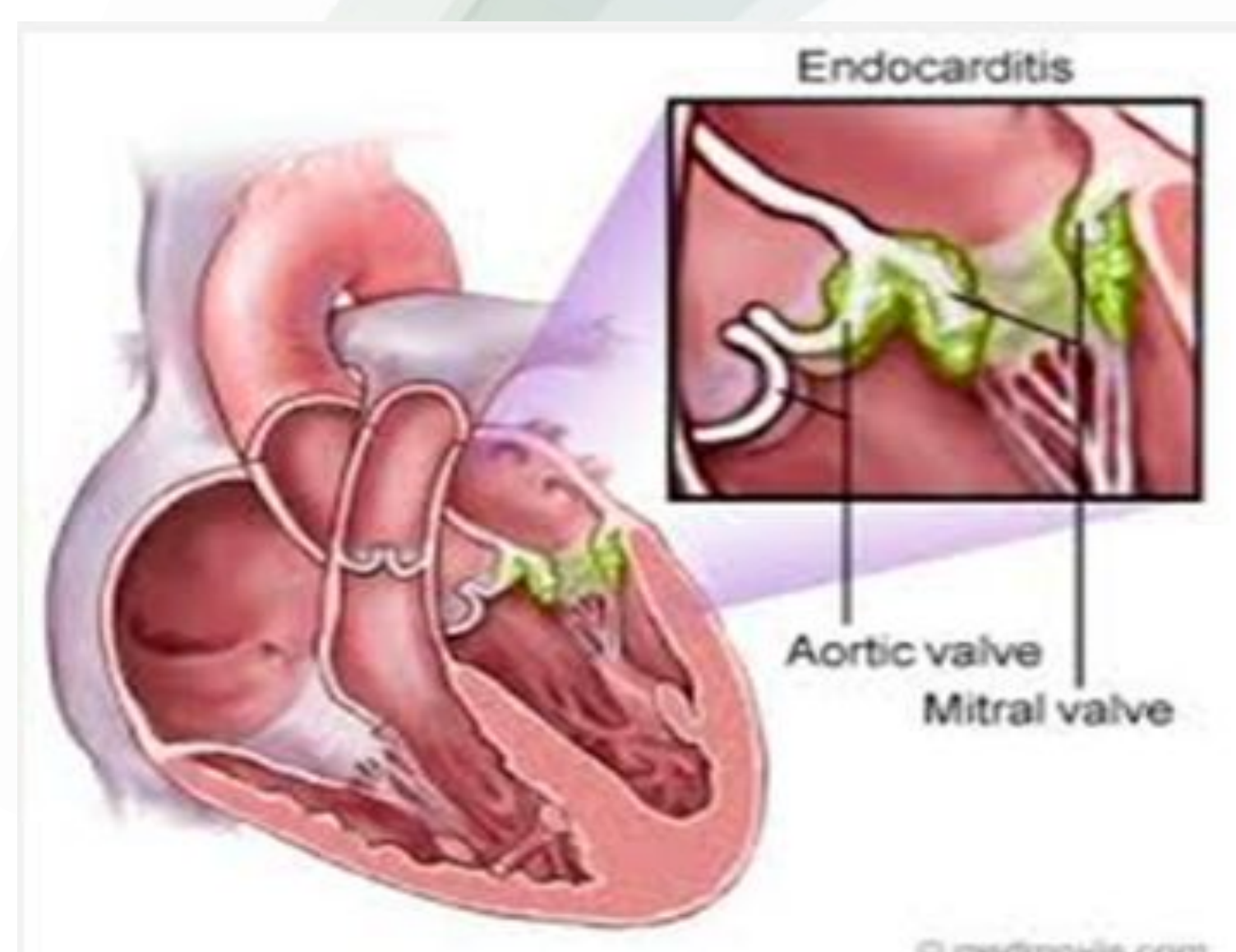
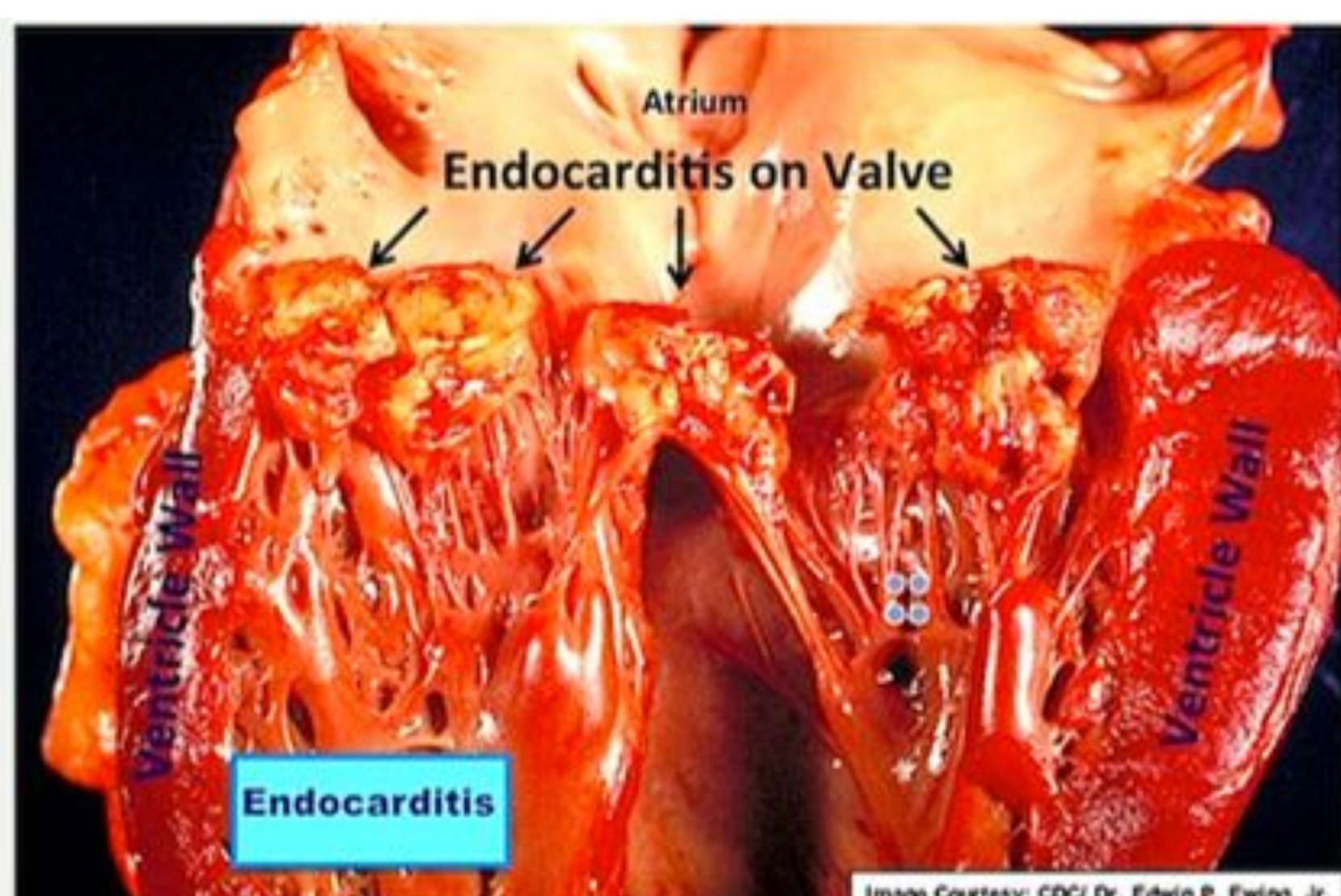
## Prognosis:

- difficult to eradicate because of the avascular nature of the heart valves. (no blood supply=no regeneration)
- depends on the infecting organism and the stage at which treated.
- 1/3 Staph. aureus endocarditis of cases are still fatal.

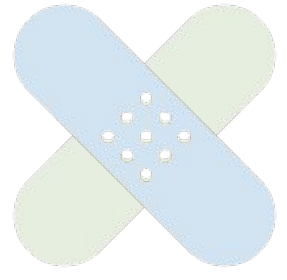


# Risk factors of IE

<b>In children</b>	an underlying cardiac lesion (congenital heart disease is most common)
<b>In Adults</b>	<ul style="list-style-type: none"> <li>• More than 50% of adults with bacterial endocarditis <b>don't have</b> predisposing cardiac lesion             <ul style="list-style-type: none"> <li>• Mitral valve prolapse and congenital heart disease (most common risk factor for bacterial endocarditis)</li> </ul> </li> </ul>
<b>In IV drug abusers</b>	<ul style="list-style-type: none"> <li>• Micro-organisms are injected intravenously when taking IV drugs leading to IE</li> <li>• Tricuspid valve is most commonly infected.             <ul style="list-style-type: none"> <li>• 50% of IE in IV drug abusers are by S.aureus</li> </ul> </li> </ul>
<b>Prosthetic valves users</b>	commonly by coagulase -ve staph.cocci (e.g. S. epidermidis).
<b>Transient bacteremia from any procedure</b>	E.g.: <ul style="list-style-type: none"> <li>• dental procedures,</li> <li>• urinary catheterization,</li> <li>• infected indwelling vascular catheters gastrointestinal endoscopy</li> <li>• obstetric procedures.</li> </ul>
<b>Other risk factors include</b>	<ul style="list-style-type: none"> <li>• Rheumatic heart disease</li> <li>• Elderly (degenerated heart valves e.g. calcific aortic stenosis)</li> <li>• Diabetics</li> <li>• pregnant women</li> </ul>







## Common clinical features

Cardiac **murmurs**

**Positive blood culture** for the organisms (only minority of cases remain negative)

**Splenomegaly, petechiae, and clubbing** of the fingers

**Fever**, fatigue, weight loss and chills

## Complications

**Septicemia** or **Septic systemic embolization** of infected vegetations which travel to multiple sites, causing infarcts or abscesses in many organs

Ulceration and perforation of valves

Rupture of chordae tendineae

Mycotic/ infected aneurysms of vessels

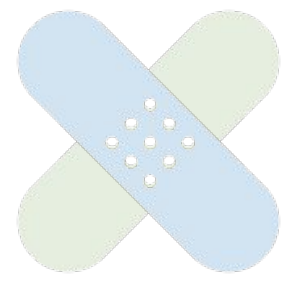
Arrhythmias, valvular regurgitation and congestive heart failure (due to destruction of a valve)

Renal failure

Pulmonary emboli in IV drug addicts (tricuspid valve/right sided endocarditis)







# Other types of endocarditis

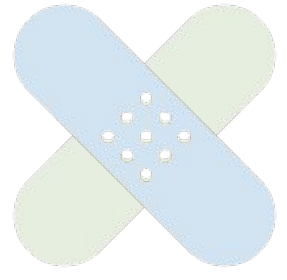
Libman sacks endocarditis	Endocarditis of carcinoid syndrome	Nonbacterial thrombotic endocarditis ( marantic endocarditis)
<ul style="list-style-type: none"> <li>● Less common</li> <li>● non infective</li> <li>● verrucous endocarditis with elevated level of circulating immune complexes .</li> <li>● <b>Seen in patients with systemic lupus erythematosus</b></li> </ul>	<ul style="list-style-type: none"> <li>● Secretory products of carcinoid syndrome, especially 5 hydroxytryptamine can cause endocarditis.</li> <li>● The endocardial plaques are seen in the right side of heart</li> </ul>	<p>Characterized by <b>sterile</b> ( no infection) <b>vegetation</b> ( small masses of fibrin , platelets, and other blood components) on the leaflets of the cardiac valves . There is no infective organism . It is aseptic.</p> <ul style="list-style-type: none"> <li>● <b>Pathogenesis/ association:</b> <ul style="list-style-type: none"> <li>-Subtle endothelial abnormalities</li> <li>-Hypercoagulability</li> </ul> </li> <li>-Association with <b>malignancy</b> (50%) and other debilitating diseases</li> <li>● Aortic valve most common site . The fibrin deposits are randomly arranged.</li> <li>● May embolize to different parts of the body including brain , but the emboli are sterile .</li> </ul>



Sterile = no infection  
 Vegetation = small mass of fibrin, platelets & other blood components.

Diagrammatic comparison of the lesions in the four major forms of vegetative endocarditis. The rheumatic fever phase of RHD (rheumatic heart disease) is marked by a row of warty, small vegetations along the lines of closure of the valve leaflets. IE (infective endocarditis) is characterized by large, irregular masses on the valve cusps that can extend onto the cords. NBTE (nonbacterial thrombotic endocarditis) typically exhibits small, bland vegetations, usually attached at the line of closure. One or many may be present. LSE (Libman Sacks endocarditis) has small or medium sized vegetations on either or both sides of the valve leaflets.





Both cause murmur

## Types of valvular heart disease

1- Stenosis :  
Failure to open

2- Regurgitation:  
Insufficiency or  
failure to close

## Causes

Acquired

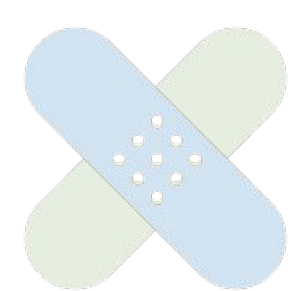
Congenital

Post inflammatory scarring  
e.g. as a late complication of  
**rheumatic fever ( most  
common )** or secondary to  
various other inflammatory  
processes

Can occur even  
with **prosthetic  
cardiac valves**

Can be secondary to  
thrombus formation  
**infectious  
endocarditis**





# Mitral valve prolapse

## Prolapse(MVP)

### Definition

A condition in which the two valve flaps of the mitral valve do not close smoothly or evenly, but instead bulge ( prolapse) upward into the left atrium .

### Epidemiology

- Most frequent valvular lesion in **developed countries**.
  - Seen in **young women**

### Pathogenesis

Unknown

There is mucous/ mucoid degeneration of the valve which causes **ballooning** of mitral valves( **floppy cusp**) results in stretching of the mitral valve , producing a **parachute deformity** of the cusp with prolapse of the cusp into the atrium during systole. These changes produce characteristic **systolic murmur with a click**.

### Clinical features

Most patients asymptomatic but can occasionally lead to mitral insufficiency and arrhythmias.

### Complications

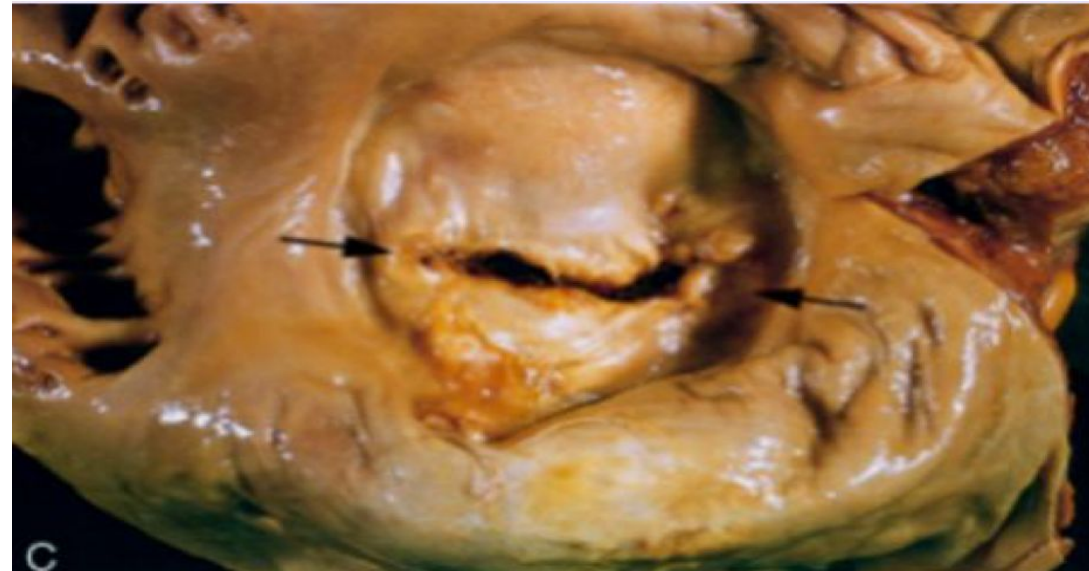
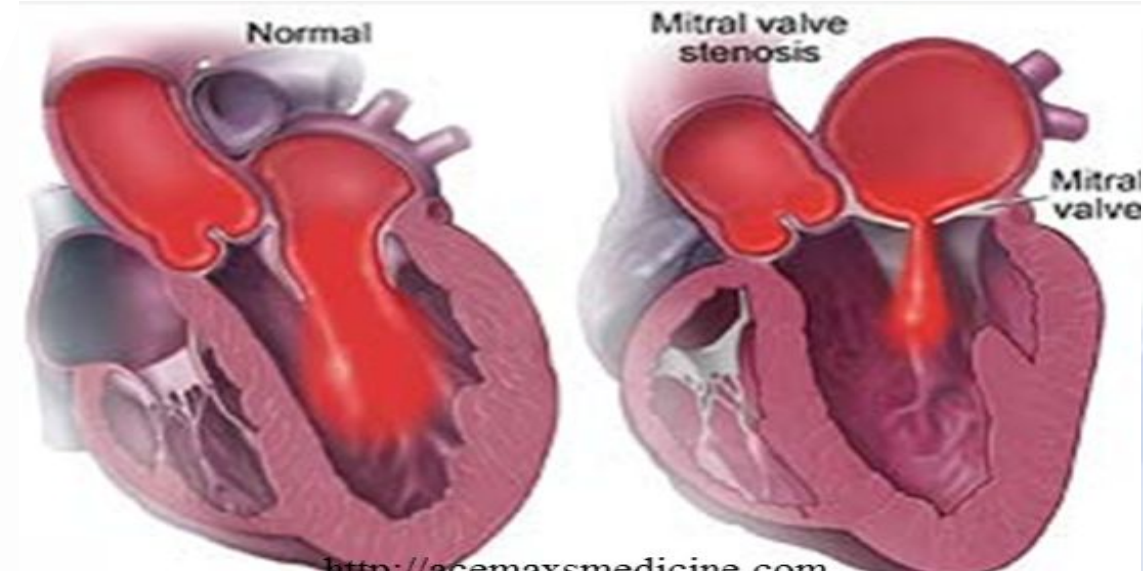
- Patients are predisposed to infective endocarditis ( **subacute**) .
- Can be associated with Marfan syndrome

### Morphology





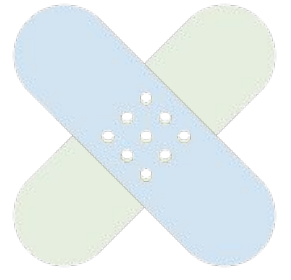
# Mitral valve stenosis

<b>Caused by</b>	<b>Rheumatic heart disease.</b>
<b>Epidemiology</b>	Mitral stenosis is more common than mitral regurgitation.
<b>Pathogenesis</b>	Valve closed —> <b>blood can't flow</b> to left ventricle which will <b>increase the pressure in the left atrium leading to hypertrophy and dilatation</b> —> due to high pressure in left atrium the blood coming from the pulmonary veins won't be able to fill in the left atrium—> the blood will return to the lungs which will lead to pulmonary hypertension and lung are firm and heavy ( chronic passive congestion). —> Right side of the heart may be affected later ( right ventricular hypertrophy).
<b>Morphology</b>	<ul style="list-style-type: none"> <li>• Leaflets are thickened, fibrotic and fused leading to fish mouth / buttonhole deformity(<b>stenosed valve looks like fish's mouth or buttonhole</b>).</li> <li>• Secondary deposition of <math>Ca^{++}</math>(any deformity will show deposition of calcium leading to heart failure).</li> </ul> <div style="display: flex; justify-content: space-around; align-items: center;">   </div>

# Mitral valve Regurgitation

<b>Caused by</b>	<b>Rheumatic heart disease</b> , mitral valve prolapse, infective endocarditis, papillary muscle injury in myocardial infarction etc.
<b>Complications</b>	Left ventricular hypertrophy and dilatation





# Aortic valve

Stenosis	
<b>Epidemiology</b>	Usually seen in old people over 60 years old
<b>Caused by</b>	Calcification and is called as calcific aortic stenosis. Also caused by Rheumatic heart disease
<b>Affects</b>	<ul style="list-style-type: none"> <li>• Normal aortic valve as part of the aging degenerative process in &gt; 60 years old.</li> <li>• Congenital bicuspid aortic valve.</li> <li>• Valves scarred by rheumatic heart disease.</li> </ul>



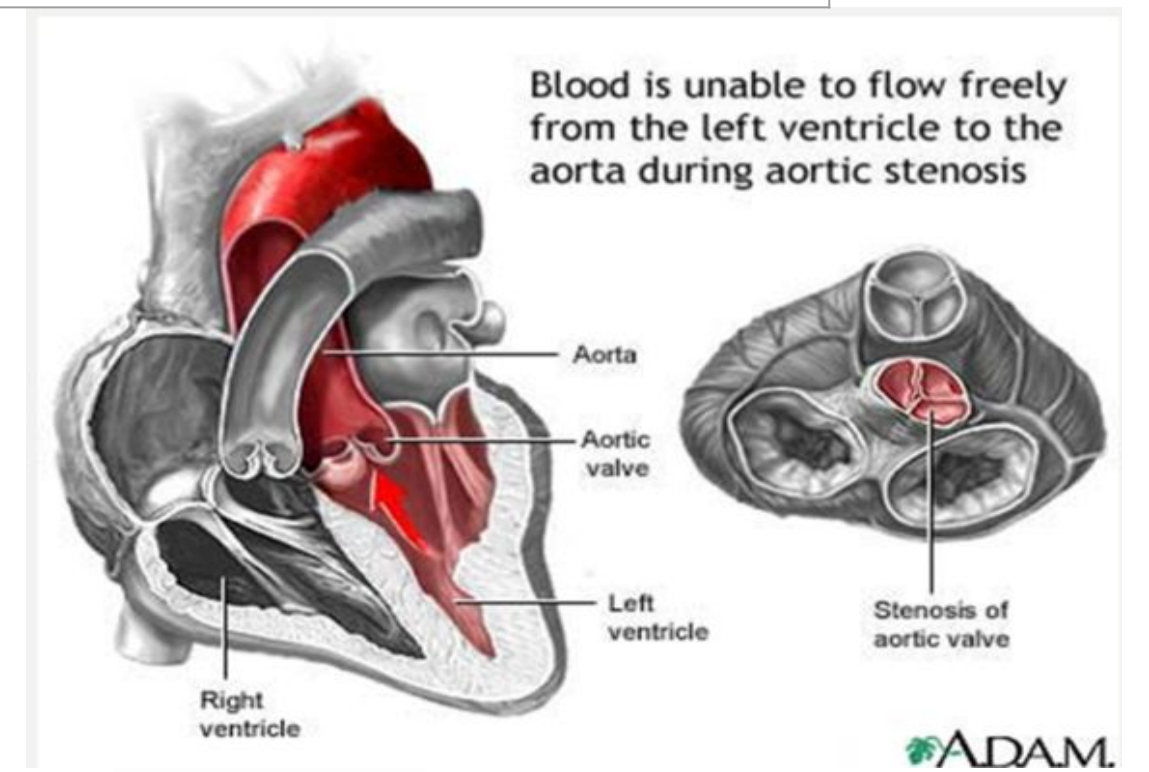
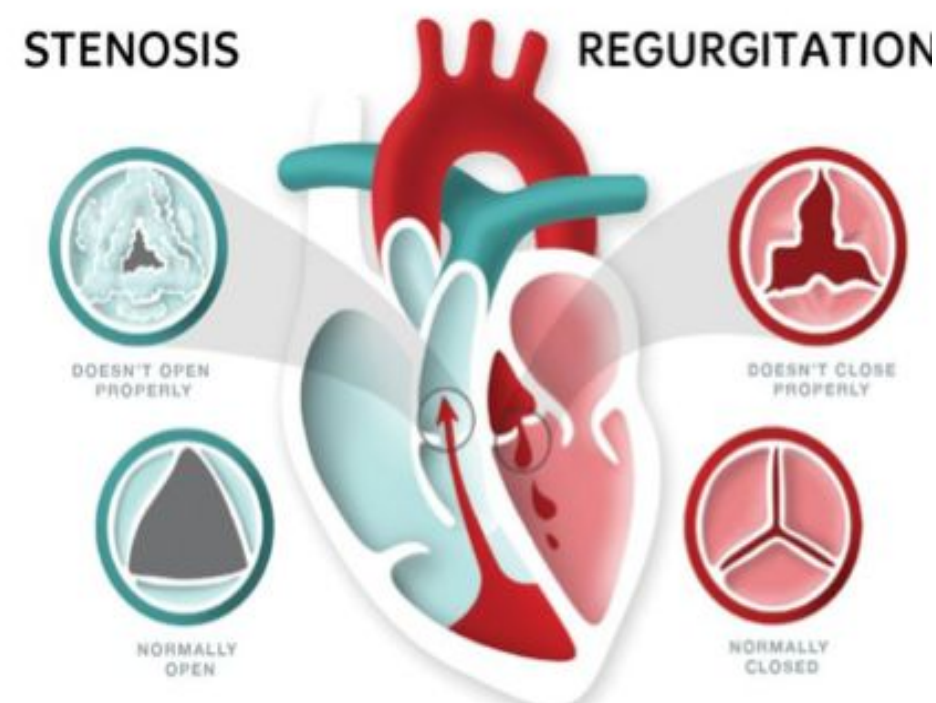
Normal Valve



Stenotic Valve



regurgitating valve



Regurgitation	
<b>Caused by</b>	<ol style="list-style-type: none"> <li>1- Non dissecting aortic aneurysm.</li> <li>2- Rheumatic heart disease.</li> <li>3- infective endocarditis.</li> <li>4- Syphilitic (luetic) aortitis(rare).</li> </ol>

## Right side of heart

Valvular heart disease of the right side of heart is very uncommon

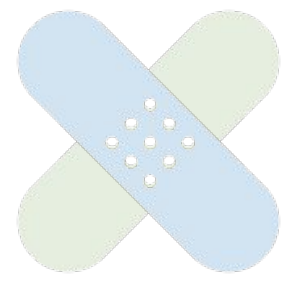


# Summary

Rheumatic fever		
<b>Type</b>	Acute	Chronic (chronic rheumatic heart disease)
<b>cause</b>	Post group A streptococcus infection	sever/ repeated attacks of rheumatic fever
<b>Characteristic</b>	Aschoff bodies	- Scarring - Thickened valvular cusps
<b>site</b>	<p>Pericarditis → fibrinous or serofibrinous secretion "Bread and butter"</p> <p>Myocarditis → Aschoff bodies</p> <p>Endocarditis → rheumatic vegetations</p> <p>Subendocardial lesions → MacCallum plaques.</p>	<ul style="list-style-type: none"> <li>● Left side of the heart</li> <li>● Mitral valve alone</li> <li>● Followed by Combination of mitral/ aortic valve</li> <li>● Tricuspid valve is rarely affected.</li> <li>● Pulmonary valve is practically never affected.</li> </ul>
<b>Clinical features</b>	<ol style="list-style-type: none"> <li>1. Elevated antistreptolysin O</li> <li>2. Jones criteria: <ul style="list-style-type: none"> <li>- Two major</li> <li>- One major &amp; two minor</li> </ul> </li> </ol>	<ol style="list-style-type: none"> <li>1. Cardiac murmurs</li> <li>2. Thromboembolism</li> <li>3. Infective endocarditis</li> </ol>
<b>complications</b>	-	<ul style="list-style-type: none"> <li>● Bacterial infective endocarditis</li> <li>● Mural thrombi</li> <li>● Congestive heart failure</li> <li>● Adhesive pericarditis</li> <li>● Atrial fibrillation.</li> </ul>

لا يَضَعُكَ اللهُ في مَوَاقِفٍ  
لا تَسْتَطِيعُ التَّغْلِبَ عَلَيْهَا،  
اللهُ يَعْلَمُ أَنَّكَ تَمْلِكُ  
الْقُدْرَةَ عَلَى تَجَاوُزِهَا وَتَحْمِلِهَا،  
اطْمَئِنِّ؛ لِأَنَّ اللهَ  
لَا يُكَلِّفُ نَفْسًا إِلَّا وُسْعَهَا.





# Cont. Summary

Infective Endocarditis		
<b>Site of infection</b>	Mitral valve followed by aortic valve, tricuspid valve is seen in IV drug users	
<b>types</b>	Acute	Subacute
<b>cause</b>	highly virulent organisms ( <b>staphylococcus aureus</b> )	low virulent organism ( <b>a-hemolytic streptococci viridans</b> )
<b>affect</b>	<b>normal/healthy valves</b>	<b>previously abnormal/damaged valves</b>
<b>progress</b>	progresses rapidly About 1/3rd of cases are fatal	progresses slowly
<b>Host reaction</b>	Has little local host reaction.	induces a local inflammatory reaction.
<b>Clinical features</b>	<ul style="list-style-type: none"> <li>• Fever</li> <li>• Cardiac murmur</li> <li>• petechiae</li> </ul>	<ul style="list-style-type: none"> <li>• clubbing of the fingers.</li> <li>• Splenomegaly</li> <li>• +ve blood culture for the organisms</li> </ul>
<b>complications</b>	<ul style="list-style-type: none"> <li>• Ulceration and perforation of valves</li> <li>• Rupture of chordae tendineae</li> <li>• Arrhythmias</li> <li>• valvular regurgitation</li> <li>• Renal failure</li> </ul>	<ul style="list-style-type: none"> <li>• Septicemia</li> <li>• congestive heart failure</li> <li>• Septic systemic embolization of infected vegetations</li> <li>• In IV drug addicts → pulmonary emboli.</li> <li>• Mycotic/ infected aneurysms of vessels.</li> </ul>

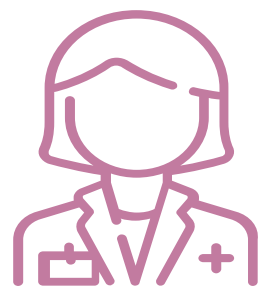
Valvular Heart Disease				
<b>Cause</b>	- Congenital - Acquired: post inflammatory scarring e.g. as a late complication of rheumatic fever			
<b>types</b>	Stenosis of valves: failure to open		Regurgitation of valves: Insufficiency or failure to close	
	Mitral	Aortic	Mitral	Aortic
<b>Cause</b>	<b>RHD</b>	<b>Calcification</b>	<ul style="list-style-type: none"> <li>• <b>RHD</b></li> <li>• mitral valve prolapse</li> <li>• IE</li> <li>• papillary muscle injury</li> </ul>	<ul style="list-style-type: none"> <li>• Non-dissecting aortic aneurysm.</li> <li>• <b>RHD</b></li> <li>• Infective endocarditis</li> <li>• Syphilitic (luetic) aortitis (rare)</li> </ul>



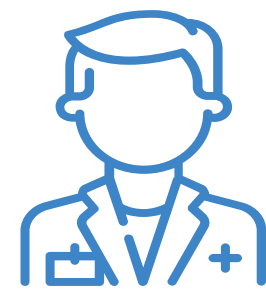


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