



Medicine

430

**Valvular Heart Disease**

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**This lecture has not been edited**

# VALVULAR HEART DISEASES

In order to understand "valvular heart disease" you need to revise the cardiac cycle (watch the videos first)

## Cardiac cycle:

-Ventricular contraction causes the A.V valves ( Tricuspid & Mitral) to close

Which signals the beginning of **Ventricular Systole**.

A- AV valves CLOSE.. Why?

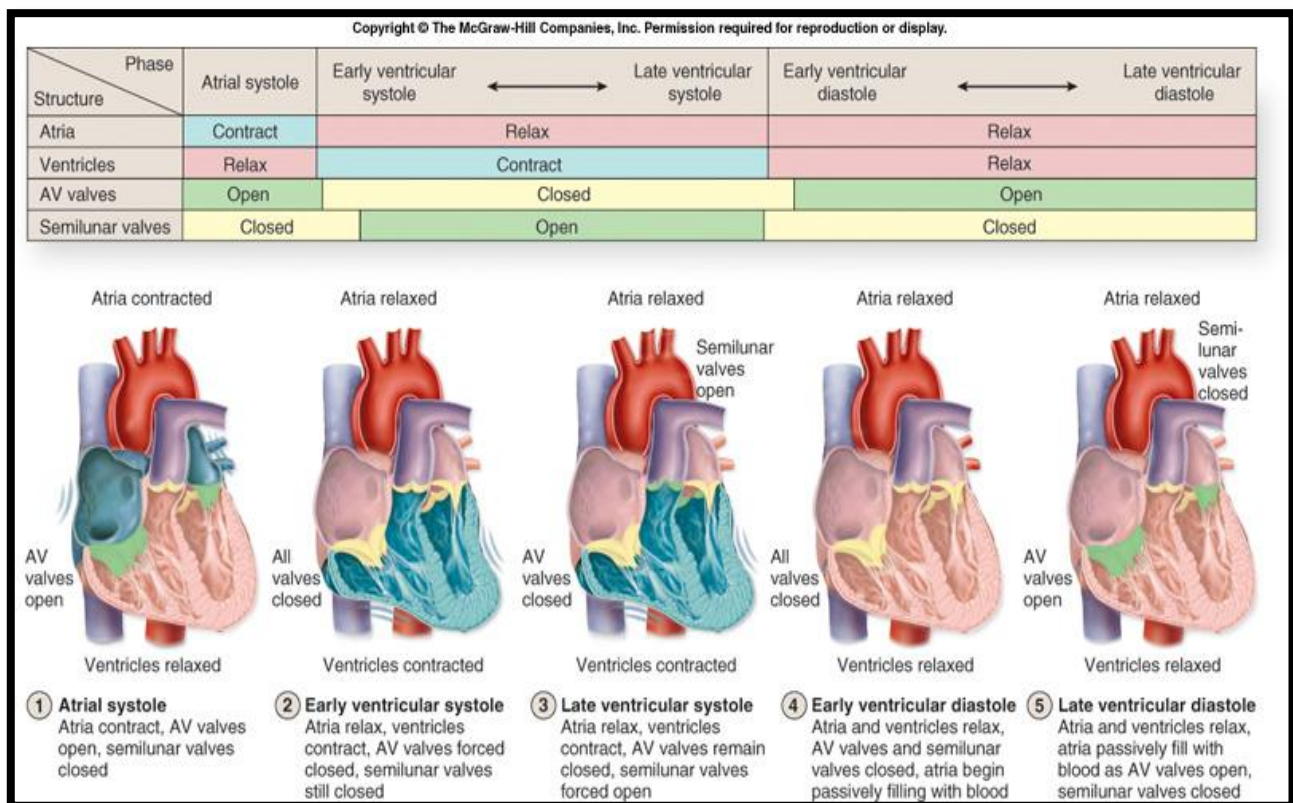
Because when the ventricles contract, the ventricular pressure rise above the atrial pressure.

- The semilunar valves ( Aortic& Pulmonary) were closed during the previous Diastole and remain closed during this period .

- Continued ventricular contractions increase the pressure in the ventricles above the pressure in the Aorta & Pulmonary trunk → causing the semilunar valves to open.

- When the ventricles relax, the ventricular pressure drop then blood flowing back toward the relaxed ventricle causing the semilunar valves to close which is the beginning of **Ventricular Diastole**.

- Note that the A.V valves open and the blood flows into the relaxed ventricles , this account for the most of the ventricular filling .

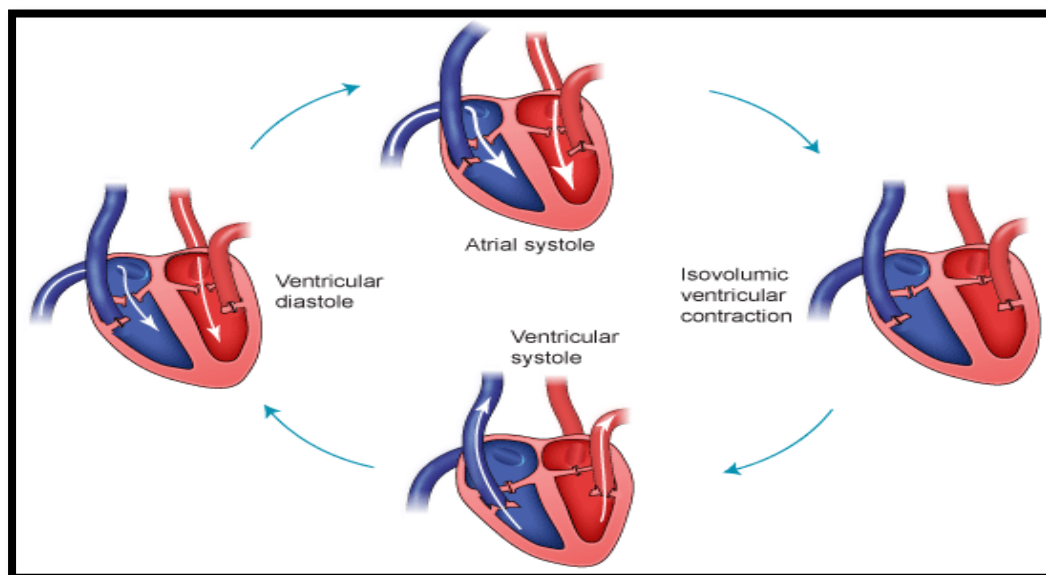


## Another explanation of the Cardiac Cycle:

The heart is a pump which must circulate blood through 2 different interconnected vascular systems, the smaller of these systems (Pulmonary system): blood returning from the upper part of the body is delivered to the RT Atrium of the heart by the "Superior Vena Cava "SVC"

(one of the body's two largest veins), while blood returning from the lower part of the body is delivered to the RT Atrium by the other major vein " Inferior Vena Cava IVC".

- Contraction of the RT Atrium in each cardiac cycle forces blood into the Rt ventricle which pumps blood into the pulmonary artery (non oxygenated blood)
- As the RT ventricle contracts & pressure within the RT ventricle rises → the Tricuspid valve " situated between RT ventricle & RT Atrium" shuts preventing any back flow.
- The pressure generated by the contraction of the RT ventricle soon opens the pulmonary valve and blood enters the circulation of the lungs.
- After passing through the circulation of the lungs, the blood having been recharged with Oxygen & having rid of CO<sub>2</sub> is returned through the pulmonary veins into the Left Atrium.
- The Left Atrium too contracts forwarding blood into the Left Ventricle in order to fill it before it contracts.
- The Aortic valve opens and blood is forced into the Aorta which distribute it into the rest of the body apart from the lungs, as the contraction comes to an end and the pressure in the aorta falls → the aortic valve snap shut to prevent any back flow into the Left Ventricle.



## **Heart Sounds :**

( by knowing the causes of heart sounds, you can detect if there is any abnormality and what causes it )

### **S1 :**

- soft, low pitched and prolonged sound
- At the beginning of systole .
- Caused by closure of AV valves ( tricuspid- mitral).
- Heard clearly at the apex of the heart .

### **S2 :**

- Short, sharp and high-pitched sound.
- At the very end of systole. ( beginning of diastole )
- Caused by closure of Aortic and pulmonary valves .

### **S3 :**

- Due to intruding of the blood from the atria to the ventricles in the middle 1/3 of diastole. ( after S2 )
- Lower in pitch than S1 or S2 as it is not of valvular origin
- Best heard with the bell-side of the stethoscope

### **S4 :**

- Due to contraction in the last 1/3 of the diastole ( before S1 )
- (it is the sound of blood being forced into a stiff/hypertrophic ventricle )
- Rare , It is a sign of a pathologic state
- Called a presystolic gallop or atrial gallop

## **What do we mean by Valvular heart disease?**

The main function of the valves is to keep blood flowing in one direction through the heart's chambers. **What can happen when heart valves are not working properly?**

A-Blood can leak back through the valve in the wrong direction; this “backflow” is called **regurgitation**.

# when we have **regurgitation** there will be **blood volume overload** causing **dilatation** of the chambers which are connected to the affected valve

**B -Stenosis** (or a narrowing of a valve) occurs when the flaps of a valve thicken, stiffen, or fuse together. This prevents the valve from fully opening, making it harder for blood to be pumped through the valve.

# while in valvular **stenosis** conditions there will be **pressure overload** causing **Hypertrophy** of the chambers

- Any VHD will produce turbulent blood flow , which is heard as a murmur on auscultation.

- the most common problems are acquired left-sided valvular lesions: Aortic stenosis, Mitral stenosis , Mitral regurgitation and Aortic regurgitation.

-Each valve has two pathology either regurge or stenosis (4 valves x 2 = 8 pathology)

-Tricuspid and pulmonary diseases are less frequently to happen in adult (more in pediatric)

The Dr emphasized on the chronic diseases of Aortic & Mitral valves

## Heart Murmurs

Heart murmurs can be either Systolic or Diastolic. On auscultation you'll hear the Systolic murmur between S1&S2 = after S1, while the Diastolic murmur will be heard after S2.

In order to differentiate between them you have to know which valve opens during systole and which is close .

**During systole** , while the L.V is contracting the aortic valve is open and the mitral valve is closed. Turbulent flow can occur either because of an incompetent mitral valve, leading to regurgitation of blood back into the atrium, or from a narrowed aortic.

**In Diastole**, the situation is reversed, with filling of the left ventricle through an open mitral valve while the aortic valve is closed. Turbulent flow occurs when there is narrowing of the mitral valve or incompetence of the aortic valve.

This table summarizes the previous explanation

-in systolic phase of the left side of the heart " so now we are focusing on the Aortic & Mitral valves"

Valve that should <b>CLOSE</b> during <b>SYSTOLE</b>	Valve that should <b>OPEN</b> during <b>SYSTOLE</b>
Mitral valve	Aortic Valve
If the valve failed to close due to <b>Mitral regurgitation</b> →so we will hear a <b>Systolic murmur = the abnormal sound will be between S1&amp;S2</b>	If the valve failed to open due to <b>Aortic Stenosis</b> → so we will hear a <b>Systolic Murmur= the abnormal sound will be between S1&amp;S2</b>

Valve that should <b>CLOSE</b> during <b>DIASTOLE= filling</b>	Valve that should <b>OPEN</b> during <b>DIASTOLE</b>
Aortic valve	Mitral valve
<b>If the valve failed to close due to regurgitation (Aortic Regurgitation)→ we will hear Dyastolic Murmur= after S2</b>	<b>If the valve failed to open due to Stenosis (Mitral Stenosis) → we will hear Dyastolic Murmur= after S2</b>

## Aortic Stenosis (AS)

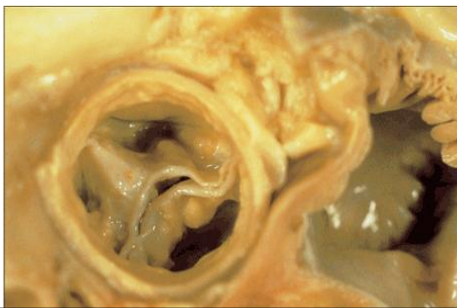
Causes: (3 causes): " according to the age":

1-Degenerative changes including fibrosis and calcification (>60 yrs )and if the pt is hypertensive and diabetic the severity of these changes will be more.

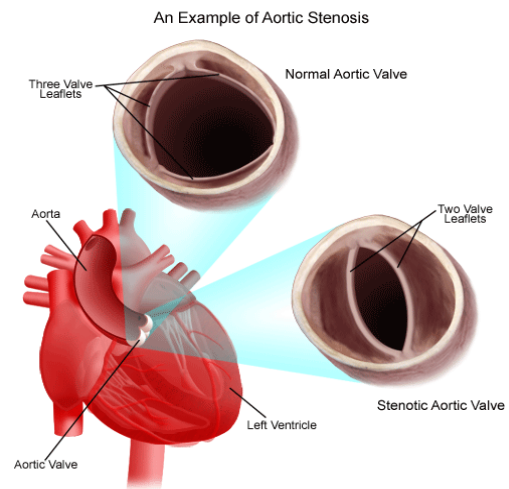
(Which is the most common cause of AS )

2- calcification of a Congenital (Bicuspid aortic valve) (30-50 yrs): it's a weak valve tha might be rupture with time

### Bicuspid Aortic Valve Stenosis



Bicuspid Aortic stenosis with clear nodes of calcification and fibrosed leaflets



3- Rheumatic heart disease (30-60 yrs)

### Pathophysiology of AS: imp

a-**pressure overload** causes obstruction to LV outflow, which results in left ventricular hypertrophy (LVH)

b-with long standing AS, the Left ventricle dilates, causing progressive LV dysfunction.

Left ventricle has to work hard because of the stenoid valve

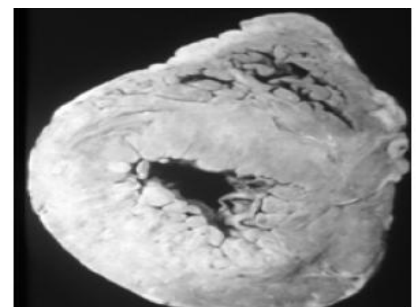
→within time that will result in **concentric hypertrophy** which means that the whole walls are hypertrophied

→ this hypertrophy will result in a very narrow ventricular cavity→  
**Left ventricular end diastolic pressure will be high** → also the **atrial pressure will increase**

Those patients are at higher risk than others to develop heart failure (why)?

Because of back pressure on the left atrium →pulmonary veins→ lungs→ will cause HF

When the patients reach this level → they need surgical replacement of the valve.



### Coarse:

a-patients are often asymptomatic for years (until middle or old age) despite severe obstruction.

b-Development of( Angina, Syncope or heart failure) is **a sign of poor prognosis**, when the aortic orifice reduced to a third of its normal size.

**Symptoms( 3 symptoms) :** Dr said that you have to know why each symptom occurs

**1-Angina:** imbalance between supply and demand

-Elevated LVED pressure decreases perfusion pressure.

-Myocardial hypertrophy increases the demand.

Angina arises because of the increased demands of the hypertrophied LV working against the high-pressure outflow tract obstruction, leading to a mismatch between oxygen demand and supply, but may also be due to coexisting coronary artery disease, especially in old age when it affects over 50% of patients

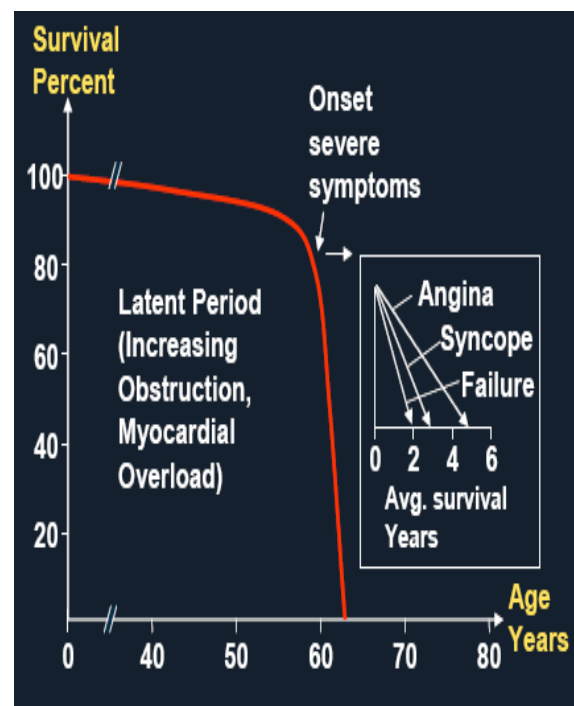
**2- syncope with exertion :** usually occurs on exertion when cardiac output fails to rise to meet demand, leading to a fall in BP.

**3- Congestive heart failure symptoms:**

↑ LVED p " left ventricular end diastolic pressure = ↑ Left Atrial pressure = Pulmonary venous congestion.

### Prognosis with Symptomatic Aortic Stenosis

Clinical Symptoms	Median Survival
Angina	5 years
Syncope	3 years
CHF	2 years





### Signs of AS:

#### 1-Harsh systolic ejection murmur:

- a- Heard in second right intercostal space (best heard in the aortic area).
- b-Radiates to carotid arteries.

Longer murmur indicates more stenosed valve

#### 2-S4 gallop from LV hypertrophy: (why?)

(Mechanism of S4= Atrial systole) but normally shouldn't be heard in adults because it's a low-pitched sound, so once we hear S4 that indicates a pathology → the atrium is contracting more → LA pressure will be high because of the back pressure when there is any abnormality in the left side of the heart such as( hypertension, MI , AS, hypertrophy) any of these will be reflected to the LA so the LA p will be increased.

- S4 can be heard in (a. Infants , b.Elderly-why?- because most of the elderly are hypertensive and as you get older you'll have a degree of hypertrophy)

#### 3-Pulsus Parvus et Tardus (Carotid Impulse)-One of the most imp signs of AS

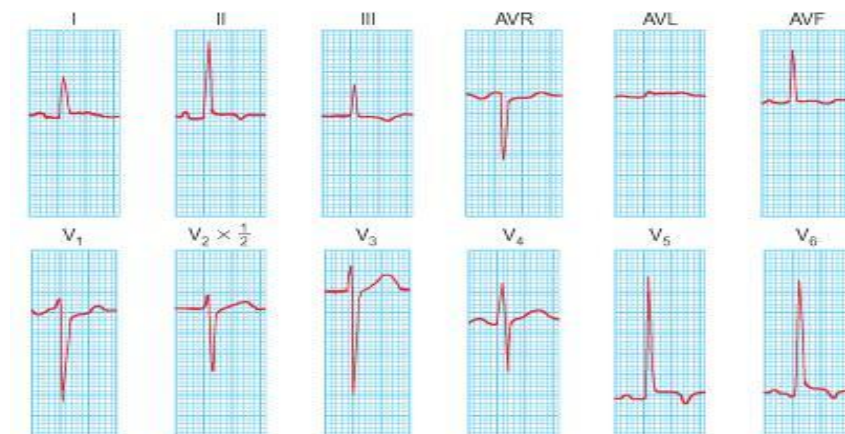
Parvus = low blood volume is coming out because of the stenosis ,

Tardus = Late , So the carotid pulses will be diminished and delayed, **On examination It'll take time until it hits ur hand.**

4-Apex beat (sustained Bifid): Sustained and not displaced because of theLVH.( it hits ur hand and stays)

### Investigations of AS:

1- ECG(to confirm the diagnosis) :ECG will show Left atrial enlargement (LAE) – Left ventricular hypertrophy (LVH)



Large R : hypertrophy

2- Echocardiogram, Doppler ( The test of choice ) diagnostic in most cases, Findings include Calcified , thickened aortic valve with restricted opening , Hypertrophied LV .

-Doppler (to assess the severity)

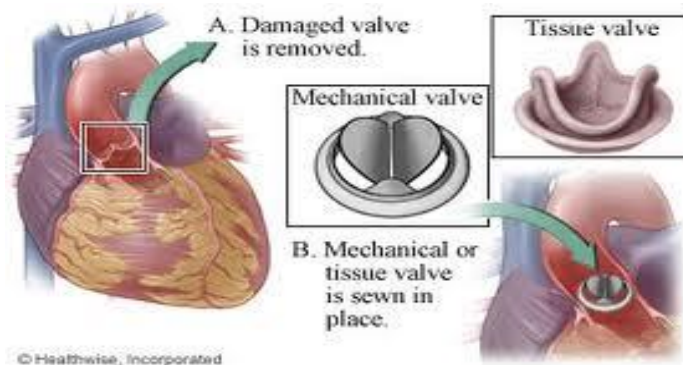
- Measurement of severity of stenosis
- Detection of associated aortic regurgitation

3- Cardiac catheterization (helpful & confirmatory, needed if the patient is old suspecting Ischemic heart disease-look at The coronaries)



**Treatment of AS:** (Follow up and clinical assessment if asymptomatic - Valve replacement if symptomatic)

- **Medical Therapy:** has a limited role (treats the symptoms).
- **Surgical Therapy:** Aortic valve replacement (Bioprosthetic or Mechanical)
- In patients with aortic stenosis, symptoms are a good index of severity and all symptomatic patients should have aortic valve replacement.
- Asymptomatic patients should be under regular review for assessment of symptoms and echocardiography



## **Aortic Regurgitation (AR)**

Regurgitation  
=Incompetence =  
Prolapse

This condition is due to disease of the aortic valve cusps or dilatation of the aortic root and valve ring .The LV dilates to compensate for the regurgitation. The stroke volume of the LV may eventually be doubled or trebled, and the major arteries are then conspicuously pulsatile. As the disease progresses, left ventricular diastolic pressure rises and breathlessness develops.

### **Causes of AR:**

-(Valvular defects )Abnormalities of the Leaflets

- 1-Rheumatic heart disease
- 2- Bicuspid (Congenital)
- 2-Degenerative=(Calcification or fibrosis with age)
- 3- Endocarditis.

-(Aortic defects )Dilation of the Aortic Annulus

**1-Aortic Aneurysm / Dissection ( aortic wall very weak )**

- 2- Inflammatory (Syphillis, Giant Cell Arteritis, Coll Vasc, Dis-Ankylosis spondylitis, Reiters)
- 3- Inheritable (Marfans, Osteogenesis Imperfecta)

### .111 Causes of aortic regurgitation

#### Congenital

- Bicuspid valve or disproportionate cusps

#### Acquired

- Rheumatic disease
- Infective endocarditis
- **Aortic dilatation** (Marfan's syndrome, **aneurysm**, **dissection**, syphilis, ankylosing spondylitis)

### Pathophysiology of AR: Imp

**Regurgitant** blood flow increases left ventricular and diastolic volume causing **LV dilation** and hypertrophy occur in response in order to maintain stroke volume and prevent diastolic pressure from increasing excessively→Over time, these compensatory mechanisms fail, leading to increased left-sided and pulmonary pressures

when the blood is leaking back from the aortic valve to the LV →volume overload will cause **LV dilatation** and within time the LV will be hypertrophied also(in sever AR) , the stroke volume will be high= ↑ Systolic pressure because with each cardiac cycle more & more blood will accumulate in the LV . On the other hand the Diastolic pressure will be decreased (why?) the perfusion of the coronaries occur during diastole so in this case there'll be less blood coming out to the coronaries →↓ perfusion pressure to the myocardium→ imbalance between myocardial supply and demand (that's why some pt may have chest pain )

-Increased LV size ( and thus wall stress)= increased demand .

### -Symptoms of AR:

-Pulmonary venous congestion (pulmonary veins get congested because of the back pressure on LA) : Dyspnea on exertion, PND and Orthopnea  
Inadequate cardiac output: Fatigue, Diminished exercise tolerance.( because most of the blood is going back to the ventricles instead of going to the rest of the body )

### -Signs of AR on physical examination:

a. **Widened pulse pressure**—markedly increased systolic BP, with decreased diastolic BP

b. **Early Diastolic decrescendo murmur** best heard at left sternal border

c. **Corrigan's pulse**(water-hammer pulse)—rapidly increasing pulse that collapses suddenly as arterial pressure decreases rapidly in late systole and diastole; can be palpated at wrist or femoral arteries.

d. **Mid Diastolic apical Rumble =Austin-Flint murmur**—low-pitched diastolic rumble due to narrowing of mitral valve orifice by aortic regurgitation, resulting in relative mitral stenosis

- e- Hyperdynamic LV apical impulse (LV works harder because of the backflow → everything will be hyperdynamic – radial – femoral pulses)
- f- S4, S3 Gallop- in advanced AR = low pitched sound – مثل صوت جري الحصان -

### **Investigations of AR :**

1-ECG will show: Left atrial enlargement (LAE) – Left ventricular hypertrophy (LVH)

2- Echocardiogram, Doppler ( The test of choice )

3- Cardiac catheterization (helpful & confirmatory, needed if the patient is old suspecting Ischemic heart disease)

### **-Treatment of AR:**

#### **-Surgical Therapy (if symptomatic): Aortic Valve Replacement.**

Because symptoms do not develop until the myocardium fails and because the myocardium does not recover fully after surgery, operation is performed before significant symptoms occur.

The timing of the operation is best determined according to hemodynamic echocardiographic or angiographic criteria.

#### **-Medical Therapy (if there is NO significant symptoms):**

1- Serial Checkups with Echos (evaluate EF, Severity AR).

2- Diuretics – for congestion.

3- Vasodilators (Nifedipine – ACE inhibitors) – to decrease afterload.

4- Subacute Bacterial Endocarditis (SBE) Prophylaxis – prevent endocarditis.

### **Summary of AR**

#### **Symptoms**

##### **Mild to moderate AR**

- Often asymptomatic
- Awareness of heart beat, 'palpitations'

##### **Severe AR**

- Breathlessness
- Angina

#### **Signs**

##### **Pulses**

- Large-volume or 'collapsing' pulse
- Low diastolic and increased pulse pressure
- Bounding peripheral pulses
- Capillary pulsation in nail beds: Quincke's sign
- Femoral bruit ('pistol shot'): Duroziez's sign
- Head nodding with pulse: de Musset's sign
- 

##### **Murmurs**

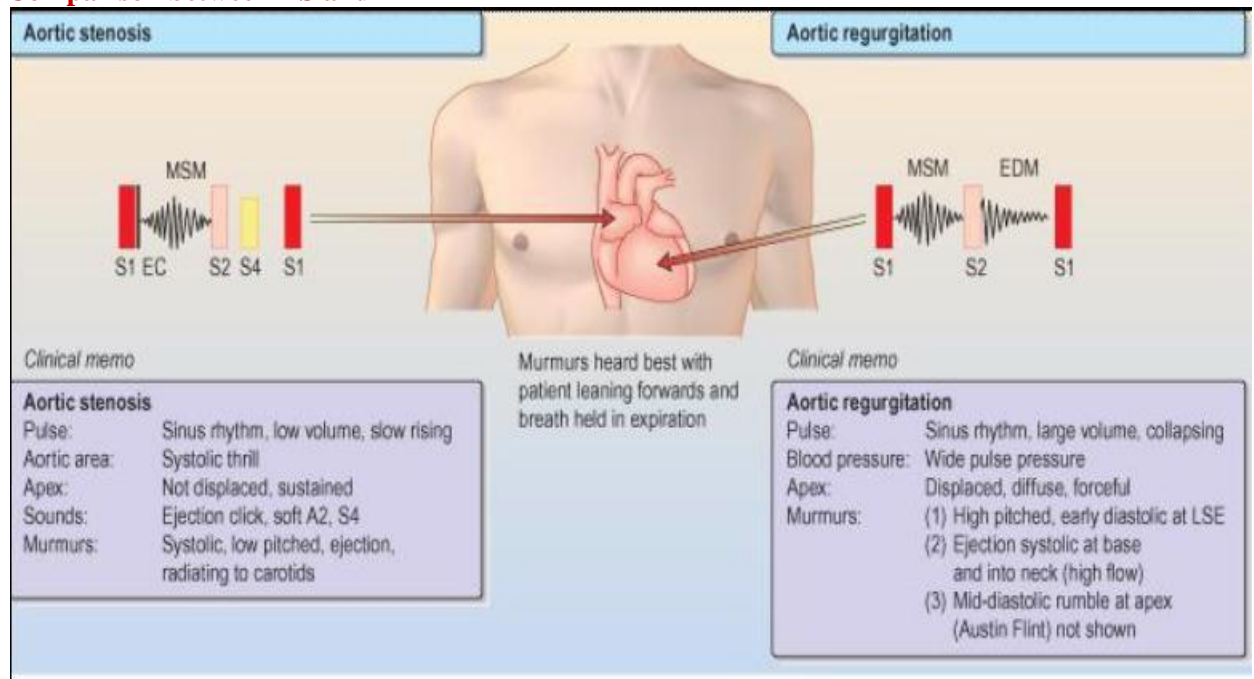
- Early diastolic murmur
- Systolic murmur (increased stroke volume)

- Austin Flint murmur (soft mid-diastolic)

#### Other signs

- Displaced, heaving apex beat (volume overload)
- Presystolic impulse
- Fourth heart sound
- Crepitations (pulmonary venous congestion)

### Comparison between AS and AR



### Mitral Stenosis (MS)

#### Causes of MS:

1-Rheumatic heart disease : Mitral stenosis is almost always rheumatic in origin

2-Mitral Annular calcification – Rare - (MAC) chronic degenerative process of the mitral valve ring ( old age group)

3-Congenital – rare.

#### Pathophysiology of MS: (Pressure overload )

a.Immune-mediated damage to the mitral valve (due to rheumatic fever) leads to scarring and narrowing of the mitral valve orifice.

- b. Mitral stenosis results in elevated left atrial and pulmonary venous pressure leading to pulmonary congestion.
- c. Anything that increases flow across the mitral valve (exercise, tachycardia, and so on) exacerbates the pulmonary venous HTN and associated symptoms. (Any increase in heart rate shortens diastole when the mitral valve is open and produces a further rise in left atrial pressure. Situations that demand an increase in cardiac output also increase left atrial pressure, so exercise and pregnancy are poorly tolerated)
- d. Long-standing mitral stenosis can result in pulmonary HTN and ultimately can result in right ventricular failure (RVF).
- e. Long-standing mitral stenosis can also lead to AFib. (Atrial fibrillation due to progressive dilatation of the LA is very common. Its onset often precipitates pulmonary oedema because the accompanying tachycardia and loss of atrial contraction lead to marked haemodynamic deterioration with a rapid rise in left atrial pressure. In contrast, a more gradual rise in left atrial pressure tends to cause an increase in pulmonary vascular resistance, which leads to pulmonary hypertension that may protect the patient from pulmonary oedema. Pulmonary hypertension leads to right ventricular hypertrophy and dilatation, tricuspid regurgitation and right heart failure)
- f. Patients are usually asymptomatic until the mitral valve area is reduced to approximately 1.5 cm<sup>2</sup> (normal valve area is 4 to 5 cm<sup>2</sup>).

## Pathophysiology

- **LA hypertension**
  - **Pulmonary interstitial edema**
  - **Pulmonary hypertension**
    - **Passive** = obligatory to preserve forward flow
    - **Reactive** = vascular changes in 40%
      - Protects interstitium from edema
      - Leads to right heart failure
  - **LA stretch and atrial fibrillation**
    - Increased heart rate thus decreased LV filling
    - Decreased atrial “kick” thus decreased LV filling
    - Atrial thrombus formation and embolus
- **Limited LV filling and cardiac output**

## Symptoms of MS:

### 1. Dyspnea on exertion (**hallmark signs of the beginning of MS**)

- The first bouts of dyspnea in patients with mitral stenosis are usually precipitated by exercise, emotional stress, infection. ( flow of blood from LA to LV is restricted by mitral stenosis so the pressure in the LA increases causing pulmonary congestion which present as dyspnea)

### 2. Paroxysmal nocturnal dyspnea (PND)

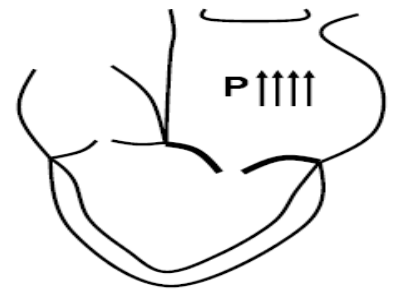
### 3. Orthopnea

### 4. Fatigue ( low cardiac output).

5. **Palpitations (due to atrial fibrillation)** All patients with mitral stenosis, and particularly those with atrial fibrillation, are at risk from left atrial thrombosis and systemic thromboembolism. Prior to the advent of anticoagulant therapy, emboli caused one-quarter of all deaths.

### 6. Hemoptysis (as the elevated LA pressure ruptures anastomoses of small bronchial veins)

### 7. Peripheral edema & **symptoms of Right heart failure.**



Normal MV area =  $4-6\text{cm}^2$   
Symptoms begin =  $< 2\text{cm}^2$   
Critical MS =  $< 1\text{cm}^2$

## Signs of MS:

**a-Mitral facies= Malar flush** (dusky-pink discoloration on the upper cheeks)

**b- Tapping apex beat** : as a result of a combination of a palpable first heart sound and left ventricular backward displacement produced by an enlarging right ventricle.

**c-Opening snap** (sound in early diastole after second heart sound due to sudden opening of the valve), The distance between S2 and the opening snap can give an indication as to the severity of the stenosis. **The closer the opening snap follows S2, the worse is the stenosis**

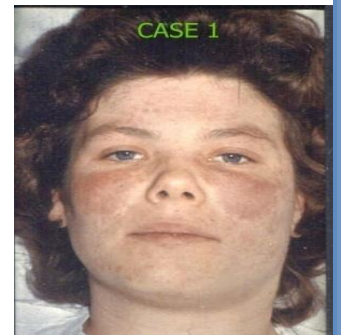
**d- Mitral stenosis Murmur: Diastolic Apical Rumble- low pitched diastolic rumble** (due to turbulent blood flow across the stenotic valve) : The opening snap is followed by a low-pitched diastolic rumble and presystolic accentuation. **This murmur increases in length as the disease worsens**

.•Heard best with bell of stethoscope) in left lateral decubitus position

**e-Heart sounds:** Murmur is followed by **Loud S1** (y be the most prominent physical finding.) The forces that open and close the mitral valve increase as left atrial pressure rises→ (S1) is therefore loud and can be palpable (tapping apex beat) , Loud P2 (due to pulmonary hypertension , **No Third heart sound**

**d.**With longstanding disease, will find signs of RVF (e.g., right ventricular heave,JVD, hepatomegaly, ascites) and/or pulmonary HTN (loud P2)

-All signs and symptoms will increase with exercise and during pregnancy.





### 18.100 Clinical features (and their causes) in mitral stenosis

#### Symptoms

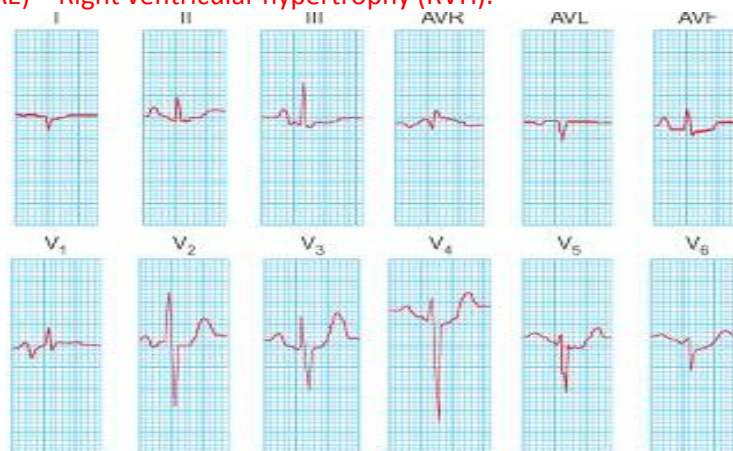
- Breathlessness (pulmonary congestion)
- Fatigue (low cardiac output)
- Oedema, ascites (right heart failure)
- Palpitation (atrial fibrillation)
- Haemoptysis (pulmonary congestion, pulmonary embolism)
- Cough (pulmonary congestion)
- Chest pain (pulmonary hypertension)
- Thromboembolic complications (e.g. stroke, ischaemic limb)

#### Signs

- Atrial fibrillation
- Mitral facies
- Auscultation
  - Loud first heart sound, opening snap
  - Mid-diastolic murmur
- Crepitations, pulmonary oedema, effusions (raised pulmonary capillary pressure)
- RV heave, loud P<sub>2</sub> (pulmonary hypertension)

#### Investigation of MS:

1- ECG will show: atrial fibrillation – Left atrial enlargement (LAE) – Right atrial enlargement (RAE) – Right ventricular hypertrophy (RVH).



2- Echo will show: (most important test in confirming diagnosis)

- a. Left atrial enlargement
- b. Thick, calcified mitral valve
- c. Narrow, "fish-mouth"-shaped orificed. Signs of RVF if advanced disease

3CXR: left atrial enlargement (early)

### 18.101 Investigations in mitral stenosis

#### ECG

- P mitrale or atrial fibrillation
- Right ventricular hypertrophy: tall R waves in V<sub>1</sub>-V<sub>3</sub>

#### Chest X-ray

- Enlarged LA and appendage
- Signs of pulmonary venous congestion

#### Echo

- Thickened immobile cusps
- Reduced valve area
- Reduced rate of diastolic filling of LV
- Enlarged LA

#### Doppler

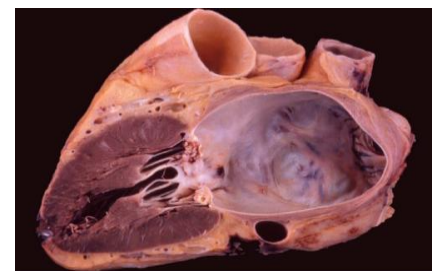
- Pressure gradient across mitral valve
- Pulmonary artery pressure
- Left ventricular function

#### Cardiac catheterisation

- Coronary artery disease
- Mitral stenosis and regurgitation
- Pulmonary artery pressure

### Complications of MS:

1. Atrial fibrillation (due to atrial dilatation)
2. Pulmonary edema
3. Blood clots with systemic embolization:
4. Left atrial dilatation → loss sinus rhythm → atrial fibrillation  
→ thrombus formation (can reach brain)
5. Pulmonary hypertension may lead to Right heart failure



Left Atrial Dilatation

### Treatment of symptomatic MS:

#### **-Medical Therapy (treats the symptoms not the cause):**

- 1- Diuretics: for congestion.
- 2- Digoxin (digitalis): to treat the fibrillation by blocking the conduction through AV node (doesn't work as inotropes)
- 3- Beta and Ca Channel Blockers (anti-arrhythmics) – for atrial fibrillation rate control.
- 4- Anticoagulation (to prevent thrombus formation)
- 5- Antibiotics: In case of rheumatic fever and before procedures that may develop infection (E.g. endocarditis)

**-Surgical Therapy (treats the cause):**

**1) Percutaneous transvenous mitral commissurotomy (PTMC)**

**"PercutaneoValvuloplasty"**

- for a Non-calcified, pliable valve.



**2) Surgical commissurotomy=Valve repair**

**3) Mitral valve replacement:**

-Mechanical valve : for young patients – stay for years but need anticoagulation through life

-Tissue valve : work 10 to 15 years ( given to old patients )

## **Mitral Regurgitation (MR)**

### **Causes of MR:**

**1- Alterations of the Leaflets, Commissures, Annulus of the valve:**

a-Rheumatic heart disease

b-Mitral valve prolapse (MVP): also known as 'floppy' mitral valve and is one of the more common causes of mild mitral regurgitation . It is caused by congenital anomalies or degenerative myxomatous changes, and is sometimes a feature of connective tissue disorders such as Marfan's syndrome .

c-Infective endocarditis

(Common causes a,b)

**2- Alterations of LV or LA size and Function (functional MR):**

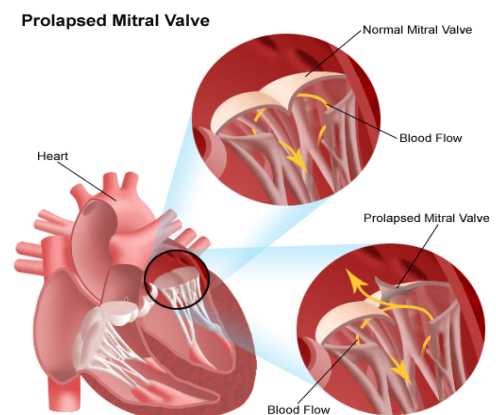
a- IHD

b- Hypertensive heart disease

c- Myocarditis

d- Cardiomyopathy: Dilated cardiomyopathy and heart failure from coronary artery disease are common causes of so-called 'functional' mitral regurgitation.

f- Papillary muscle ( ischemia), Mitral valve function depends on the chordae tendineae and their papillary muscles.



### 18.103 Causes of mitral regurgitation

- Mitral valve prolapse
- Dilatation of the LV and mitral valve ring (e.g. coronary artery disease, cardiomyopathy)
- Damage to valve cusps and chordae (e.g. rheumatic heart disease, endocarditis)
- Ischaemia or infarction of the papillary muscle
- MI

### Pathophysiology of MR:

#### **Acute MR:**

- LA is not dilated, which result in high LA pressure, high pulmonary venous pressure, severe pulmonary edema and congestion

#### **Chronic MR:**

↓ Cardiac output

↑ Total stroke volume → volume overload → LV dilation.

- long standing MR causes gradual elevation of left atrial pressure in setting of dilated LA and LV (with increased left atrial compliance) → LV dysfunction occurs due to dilatation.
- .→ Pulmonary HTN can result from chronic backflow into pulmonary vasculature

#### Another explanation :

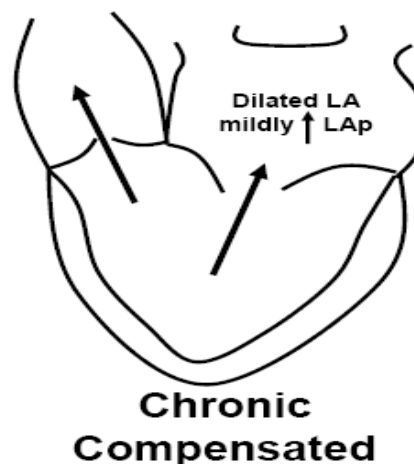
Chronic mitral regurgitation causes gradual dilatation of the LA with little increase in pressure and therefore relatively few symptoms.

Nevertheless, the LV dilates slowly and the left ventricular diastolic and left atrial pressures gradually increase as a result of chronic volume overload of the LV.

In contrast, acute mitral regurgitation causes a rapid rise in left atrial pressure (because left atrial compliance is normal) and marked symptomatic deterioration.

### **Pathophysiology**

- **Eccentric hypertrophy**
  - Increased preload
  - Increased afterload
  - Increased total stroke volume AND forward stroke volume AND LVESV returns to normal
- **Increased LA size**
  - Increased LA compliance
  - Larger volume at lower pressure



**Symptoms of MR:** Symptoms depend on how suddenly the regurgitation develops . chronic MR produces a symptom complex that is similar to that of mitral stenosis but sudden-onset mitral regurgitation usually presents with acute pulmonary oedema.

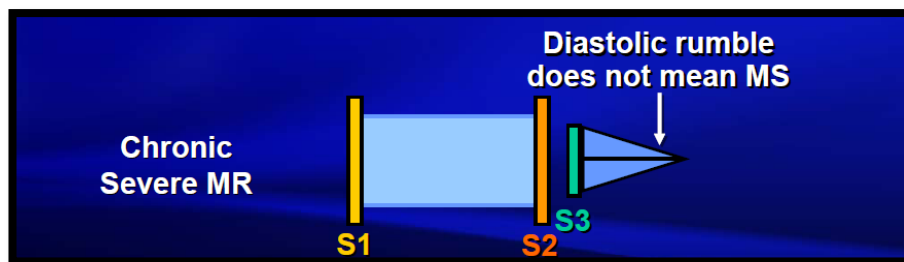
Chronic MR:

- 1-Exertional dyspnea and PND
- 2-Fatigue and lethargy (resulting from reduced cardiac output)
- 3-Thromboembolism is less common than with MS
- 4-Palpitations

Acute MR: presents as pulmonary oedema.

**Signs of MR on physical examination :**

- 1- **Apex beat: laterally displaced** & diffuse forceful - because the ventricle is dilated (in chronic cases )
- 2- **Systolic apical thrill (usually radiating to axilla)**
- 3- Heart sounds: Soft S1 (due to incomplete leaflet closure), Split S2 (but is obscured by the murmur)
- 4- Prominent third heart sound followed by diastolic rumble , caused by rapid filling of the dilated LV in early diastole.
- 5- Pansystolic murmur = Holosystolic murmur (starts with S1 and continues on through S2): palpable as a thrill, loudest at the apex and radiating widely to the back or clavicular area, depending on which leaflet is involved
- 6- AFib is common finding



**Investigations :**

1-ECG : shows LA dilatation & LVH

2-CXR(chest x-ray) : dilated LV, pulmonary edema

CXR and ECG changes are not sensitive or specific

3- **Echo(confirms the diagnosis):** Dilated LA&LV, structural abnormalities of mitral valve(eg. Prolapse)

-**Doppler( to assess the severity of the disease ):** detects and quantifies regurgitation

**4-Cardiac catheterization:** Dilated LA, dilated LV, Pulmonary hypertension , Coexisting coronary artery disease.

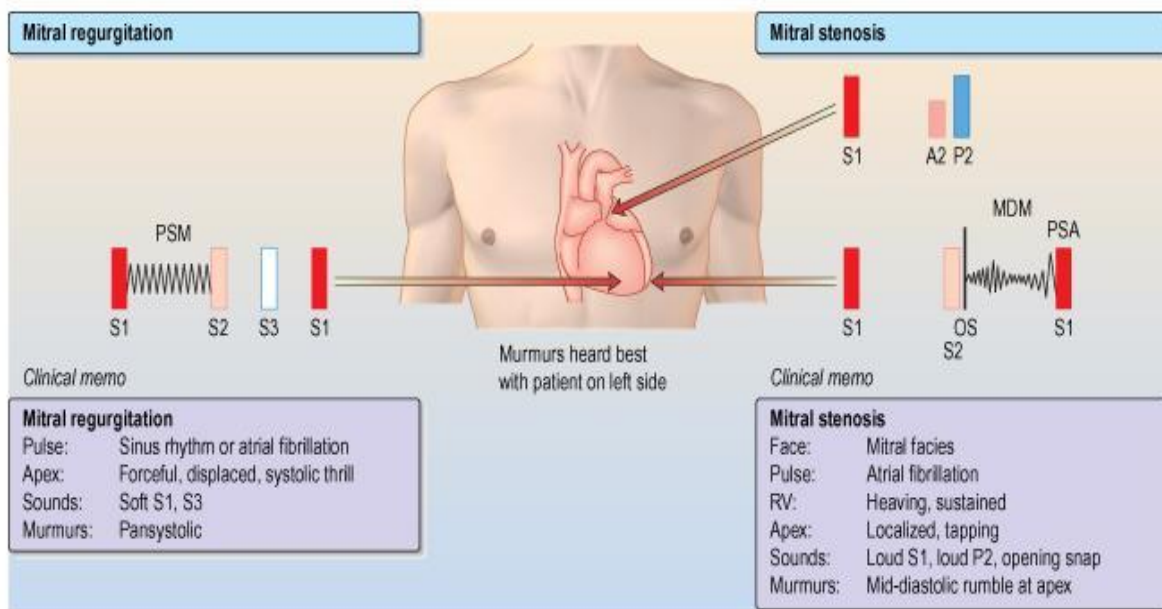
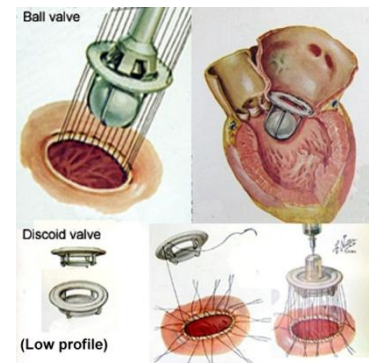
### Treatment of MR:

#### **Medical Therapy (treats the symptoms not the cause):**

1. Diuretics – for congestion
2. Vasodilators (ACE inhibitors) – to decrease afterload
3. Anticoagulation – for atrial fibrillation and LA clots
4. SBE Prophylaxis – prevent endocarditis

#### **Surgical Therapy (treats the cause):**

1. Mitral valve repair
2. Mitral valve replacement

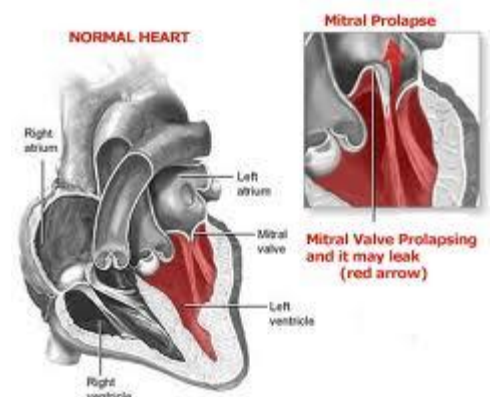


### Mitral valve prolapse (MVP)

This is a common condition occurring mainly in young women. One or more of the mitral valve leaflets prolapses back into the LA during ventricular systole, producing mitral regurgitation in few cases.

Causes:

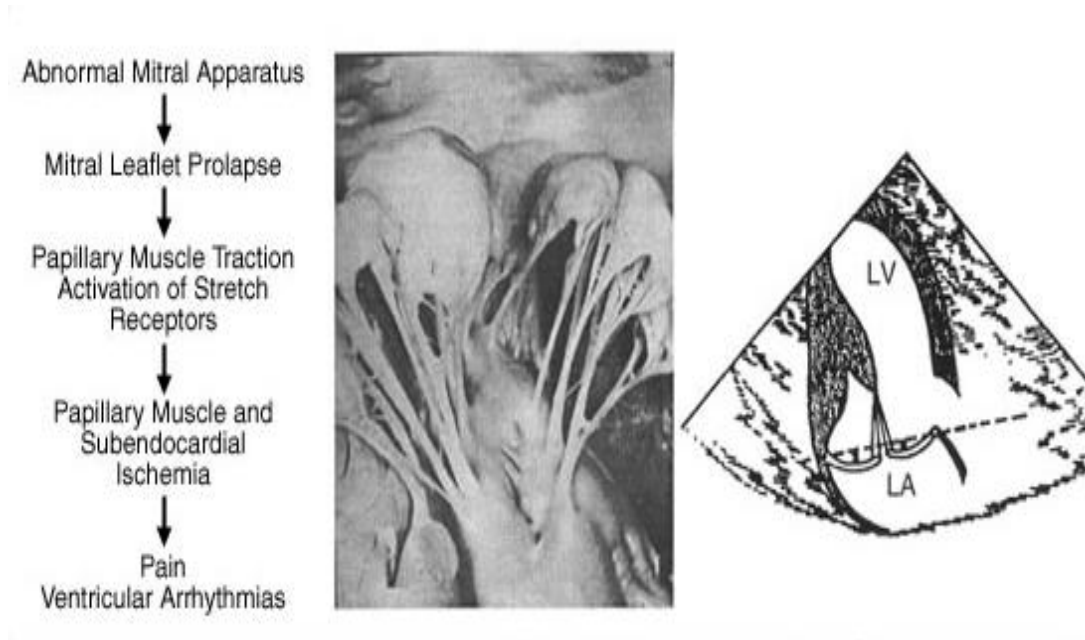
The cause is unknown but it may be associated with Marfan's syndrome, thyrotoxicosis and RF or ischaemic heart disease.





### Pathophysiology of MVP:

- Large mitral valve leaflets, an enlarged mitral annulus, abnormally long chordae, or disordered papillary muscle contraction
- Demonstrate myxomatous degeneration of the mitral valve leaflets



### Symptoms of MVP:

- 1- Atypical chest pain (**most common symptom**)
- 2- Palpitations (because of the abnormal ventricular contraction or because of the atrial and ventricular arrhythmias)
- 3- Sudden cardiac death (due to fatal ventricular arrhythmias) – very rare but recognized complication

### Signs of MVP:

- 1-Mid-systolic click (**most common sign**) – produced by the sudden prolapse of the valve and the tensing of the chordae tendineae that occurs during systole
- 2-A late systolic murmur (if associated with mitral regurgitation)

### Complications of MVP:

- Infective endocarditis
- Progressive MR (acute – chronic)
- Thromboembolism
- Atrial-ventricular arrhythmias

### Treatment of MVP:

- 1- Beta-blocker → effective for the treatment of the atypical chest pain and palpitations
- 2-In mitral valve prolapse associated with significant mitral regurgitation and atrial fibrillation, anticoagulation is advised to prevent thromboembolism
- 3- In mitral valve prolapse associated with severe mitral regurgitation which has a risk of sudden cardiac death → surgery is advised

## Right sided valvular heart diseases ( Tricuspid& Pulmonary)

Both tricuspid and pulmonary are rare in adults

**Remember that the Right sided valvular lesions change in intensity with inspiration and the right sided murmurs will be louder during inspiration (why?)**

Because with inspiration the venous return to the Rt side of the heart will be increased = large amount of blood going to the IVC → to the Right side of the heart so the murmurs will be heard louder with inspiration

### Tricuspid Valve: (stenosis, Regurgitation)

#### Causes

**1-Endocarditis** : the most common disease that can affect the Tricuspid in adults

**It seen mostly with IV drug abusers or inpt with IVs** ( they take the injections through the vein → IVC connected to the Right side of the heart)

**2-Carcinoid HD:** hormonal disease which can affect the intestine and the Rt side of the heart causing (classically TS) – but this disease is very rare every (one case in every 5 years)

**-TR – common, benign, may be secondary to pulmonary hypertension.**

**Pulmonary Valve:** (Usually a congenital problem seen in young patients)

#### Causes:

**- In Pediatrics – Pulmonary Stenosis**

- Rheumatic HD – Pulmonary Regurgitation (Graham Steel Murmur)

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### **Resources: -**

- Cardiac cycle videos
  - o <http://www.youtube.com/watch?v=jLTdgrhpDCg>
  - o <http://www.youtube.com/watch?v=rguztY8aqqk>
- Step-up to Medicine – second edition
- Davidson's principles and practice of Medicine – 21<sup>st</sup> edition-
- Clinical Medicine fourth edition by Kumar&Clark-
- Medicine 428 Team notes-