Valvular heart disease

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Introduction

the heart has four valves that prevent backflow of blood.

Types of heart valves

1- Atrioventricular valves

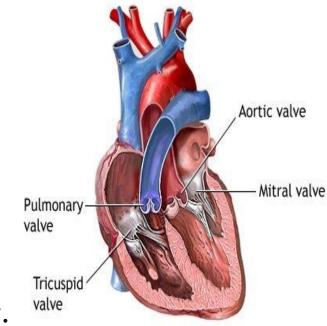
mitral valve: between left atrium and left ventricle.

tricuspid valve: between right atrium and right ventricle.

2-Semilunar valves

aortic: between left ventricle and aorta.

pulmonary: between right ventricle and pulmonary artery.



Valvular Lesions

1- stenosis

Is the failure of a valve to open completely, obstructing forward flow.

2-Insufficiency

Is failure of the valve to close completely, allowing regurgitation of blood (backflow).

Valve lesions - systole

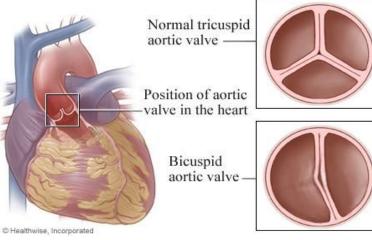
- -Aortic stenosis
- -Mitral regurgitation
- -Pulmonic stenosis
- -Tricuspid regurgitation

Valve lesions - diastole

- -Aortic regurgitation
- -Mitral stenosis
- -Pulmonic regurgitation
- -Tricuspid stenosis

Aortic valve diseases(tricuspid)

The aortic valve normally has three cusps or leaflets, although in 1-2% of the population it is found to <u>congenitally have two leaflets</u>. The aortic valve is the last structure in the heart the blood travels through before stopping the flow through the systemic circulation.



AORTIC STENOSIS (AS)

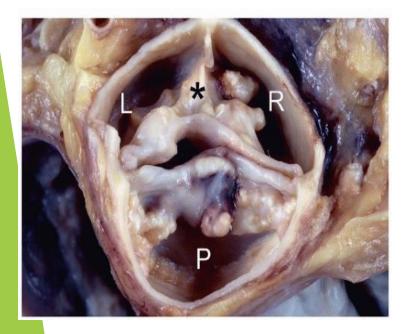
1-Pathophysiology

- -AS causes obstruction to LV outflow, which results in LVH to maintain COP without dilation of the ventricular cavity. therefore, the stroke volume is normal until late stages of the disease.
- -When aortic valve area falls below 1cm2, cardiac output fail to increase.
- -LVH and high intramyocardial wall tension account for the increased oxygen demands and along with decreased diastolic coronary blood flow ,account for the occurrence of angina pectoris .
- -With long-standing AS, LV dilate causing LV dysfunction.
- -With severe AS, LV dilation pulls the mitral valve annulus apart, causing MR.

Aortic stenosis

2-causes

- **a.** Calcification of a congenitally abnormal bicuspid aortic valve ,tend to present at earlier age.
- b. Calcification of tricuspid aortic valve in elderly. this is the most common cause.
- c. Rheumatic fever, aortic valve is affected in about 30-40% of cases .







Aortic stenosis

3-clinical manifestations

Symptoms

- **a.** Angina
- **b.** Syncope—usually exertional
- C. Heart failure symptoms, such as dyspnea on exertion, orthopnea, or PND.

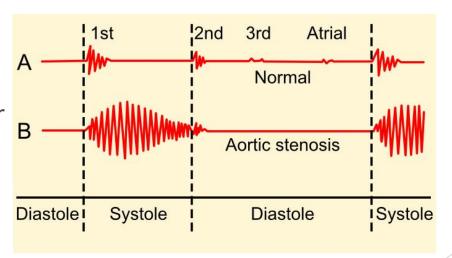
<u>Signs</u>

a. Murmur

Harsh crescendo-decrescendo systolic murmur (diamond- shaped)

Heard in second right intercostal space

Radiates to carotid arteries



- b. Soft S2 when the aortic valve becomes immobile, S2 may also be single since the aortic component may be delayed and merge into P2. The softer the murmur, the more severe the AS
- c. S4 with progressive disease (S4 gallop)
- d. Parvus et tardus—diminished and delayed carotid upstrokes
- e. Sustained PMI
- f. Precordial thrill
- **g.**Aortic ejection click
- h. Slow- rising pulse

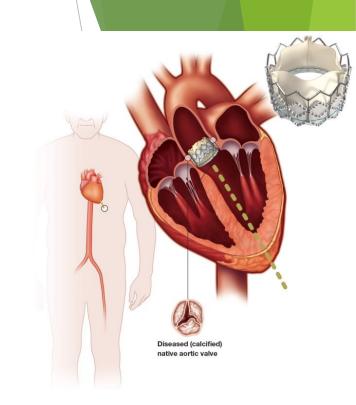
Aortic stenosis

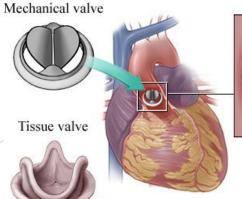
4-diagnosis

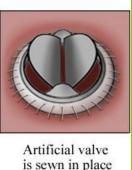
- **-ECG** will show LVH.
- -chest x-ray may present with calcification, cardiomegaly, pulmonary congestion.
- -echocardiography shows thickened calcified aortic valve leaflets .

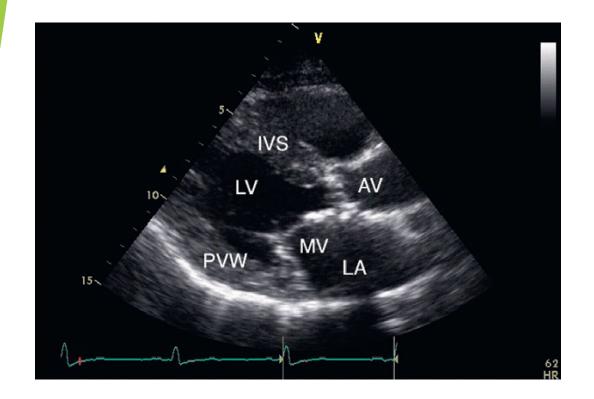
5-treatment

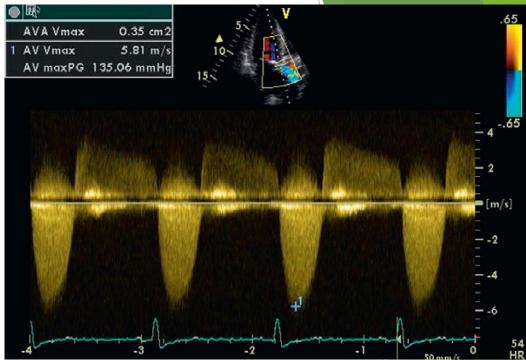
- -Medical therapy has a limited role
- -Asymptomatic patients should
- be under regular review for assessment of symptoms and
- Echocardiography.
- -surgical therapy (aortic valve replacement) is the treatment of choice , it is indicated in $_{
 m Me}$
- -balloon valvuloplasty maybe useful in those too ill to tolerate surgery.











Echocardiogram (long-axis view) in a patient with calcific aortic stenosis. The calcium in the valve generates abnormally intense echoes. There is some evidence of the associated left ventricular hypertrophy

AORTIC REGURGITATION

1-Pathophysiology

- -inadequate closure of the aortic valve leaflets, regurgitated blood flow increases LV end-diastolic volume.
- LV dilation and hypertrophy occur in response in order to maintain stroke volume and prevent diastolic pressure from increasing excessively.
- LV dilation is thought to overstretch the myofibrils, leading to less actin-myosin interaction and decreased contractility.
- Over time, these compensatory mechanisms fail, leading to increased left-sided and pulmonary pressures.
- The resting left ventricular EF is usually normal until advanced disease.

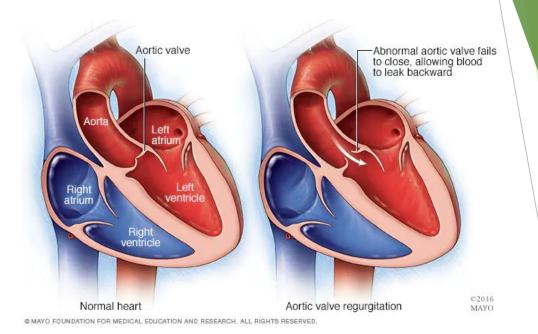
2-causes

Acute:

- 1. aortic dissection or aneurysm
- 2. trauma
- 3. infective endocarditic

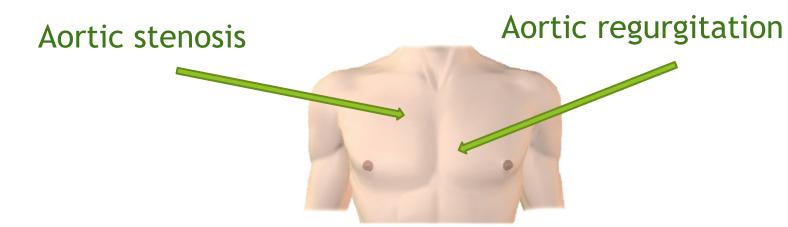
Chronic:

- 1. Bicuspid aortic valve
- 2. Rheumatic fever
- 3. Marfan Syndrome (dilation in aortic root)
- 4. SLE ,ankylosing spondylitis, Ehlers Danlos syndrome
- 5. Aortic root disease:syphilitic aoritis, osteogenesis imperfect, Reiter syndrome, systemic HTN.



- 2-clinical manifestations
- symptoms
- -dyspnea on exertion ,PND, Orthopnea.
- -palpitation
- -angina
- -cyanosis and shock in acute aortic regurgitation (emergency)
- Physical examination
- Early Diastolic decrescendo murmur best heared at the left sternal edge the fourth intercostal space.
- Corrigan ,collapsing pulse (water hammer pulse)
- Widened pulse pressure, increase SBP and decrease DBP
- Austin flint murmur (mid-diastolic murmur)
- Displaced apex beat (down and to the left) and is forceful in quality.





- Pulse: Sinus rhythm, low volume, slow rising
- Aortic area: Systolic thrill
- Apex: Not displaced, sustained
- Sounds: Ejection click, soft A2, S4
- Murmurs: Systolic, low pitched, ejection,
- radiating to carotids

- Pulse: Sinus rhythm, large volume, collapsing
- Blood pressure: Wide pulse pressure
- Apex: Displaced, diffuse, forceful
- Murmurs: (1) High pitched, early diastolic at LSE
- (2) Ejection systolic at base and into neck (high flow)
- (3) Mid-diastolic rumble at apex
- (Austin Flint)

3-diagnosis

-chest x-ray :dilated aorta, enlarged cardiac silhouette

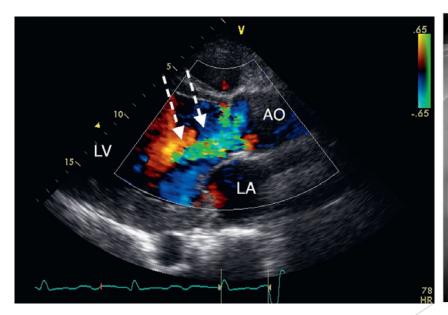
-ECG: LVH

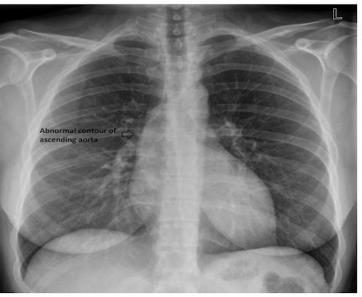
-Echocardiogram:

LVH

Dilated aorta

Early closure of mitral valve





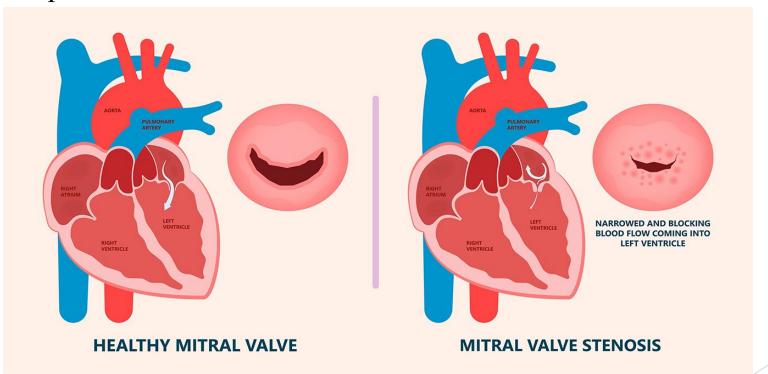
- 4- treatment
- If asymptomatic with normal LV function : no therapy is indicated
- If asymptomatic with LV dysfunction : ACE inhibitors or ARBs for afterload reduction.
- If symptomatic :salt restriction, diuretics, afterload reduction (ACE inhibitors or ARBs)
- Surgery is the definitive ttt in symptomatic patients or if asymptomatic with EF<55% or asymptomatic with left ventricular ejection fraction >50% but with a dilated left ventricle (end-diastolic dimension >70 mm or systolic dimension >50 mm)

Mitral valve stenosis

Dema Salem Fayez obeidat 1833392

Mitral valve stenosis

- ☐ Narrowing of the mitral valve orifice that blocks (obstructs) blood flow from the left atrium to the left ventricle. Usually due to chronic rheumatic valve disease
- ☐ Continuous, progressive, lifelong disease and about of 2/3 of patients are women.



Pathophysiology:

- Mitral stenosis results in elevated left atrial and pulmonary venous pressure leading to pulmonary congestion.
- Anything that increases flow across the mitral valve (exercise, tachycardia, and
 - so on) exacerbates the pulmonary venous HTN and associated symptoms.
- Long-standing mitral stenosis can result in pulmonary HTN and ultimately can result in right ventricular failure (RVF).
- Long-standing mitral stenosis can also lead to AFib due to increased left atrial size pressure and size.
- Patients are usually asymptomatic until the mitral valve area is reduced to approximately 1.5 cm2 (normal valve area is 4 to 5 cm2).

Mitral valve stenosis

Etiology:

- Rheumatic heart disease: the most common cause, it causes thickened mitral valve leaflets, fused commissures, and chordae tendineae.
- Congenital heart disease
- Infective endocarditis

Mitral valve stenosis

Etiology:

- Radiation treatment
- Carcinoid syndrome
- SLE
- Senile
- Tumers

Symptoms of MVS:

- The symptoms often appears when the caliber of the valve orifice is less than 1.5 cm²
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Fatigue

Symptoms of MVS:

- Cough with pink frothy sputum
- Hemoptysis (due to rupture of pulmonary vessels)
- Hoarseness (due to impingement of an enlarged left atrium on the recurrent laryngeal nerve)
- Systemic embolism
- If RVF occurs, ascites and edema may develop
- Dysphagia (esophagus)

Sign of MVS:

- Atrial fibrillation (irregular cardiac rhythm) due to the stretching and enlargement of your heart's left atrium.
- Pulmonary rales
- Decreased pulse pressure
- Malar flush (dusky-pink discoloration of upper cheeks) due to CO2 retention



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On auscultation:

First heart sound (S1) is loud

Opening snap following S2 after aortic valve closure.

- distance between s2 and opening snap indicates severity

The murmur is best heard at the cardiac apex with bell of steths, in left lateral position, at the height of expiration and after doing mild exercises.

Investigation:

- Chest X-RAY :
 - calcified valve
 - left atrial enlargement
 - pulmonary edema
- May show signs of pulmonary hypertension, including Kerley B lines and increased vascular markings



Investigation:

ECG

May show signs of right ventricular hypertrophy
May show left and right atrial abnormalities
Atrial fibrillation often occurs

- Echocardiography : most important test for confirming diagnosis
- "Hockey-stick" appearance of AML in diastole
- Immobility of PML
- "Fish-mouth" orifice in short-axis view
- Increased LA size



Left atrial enlagement on chest X-Ray



"Fish-mouth" orifice

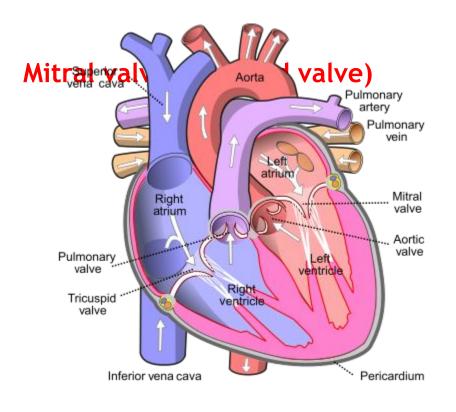
Treatment

- Medical
 - a. Diuretics—for pulmonary congestion and edema.
 - b. β-Blockers—to decrease heart rate and cardiac output.

- Surgical (for severe disease)
- a. Percutaneous <u>balloon valvuloplasty</u> usually produces excellent results.
- b. Open commissurotomy and mitral valve replacement are other options if
 - valvotomy is contraindicated.

Mitral valve regurgitation

Seham raed abdullah abu zeiter



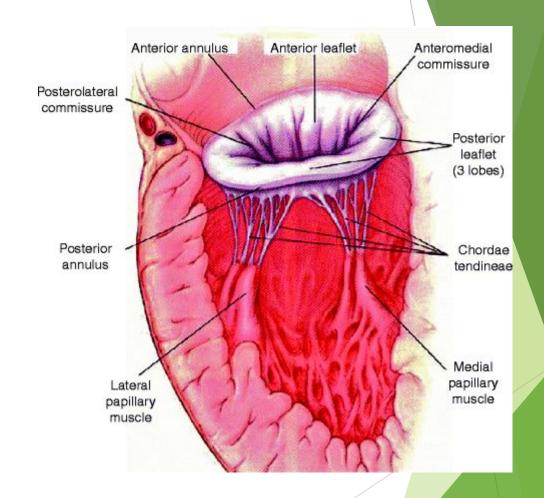
The mitral valve lies <u>between the left atrium</u> and the left ventricle.

It has two leaflets: the anterior leaflet and the posterior leaflet.out of all the valves the mitral is normally the only valve with two leaflets and that's why its called "bicuspid"

As the blood flow, the two leaflets separate creating a gap.

The atrioventricular valves are attached to the ventricular walls via the chordae tendineae, which in turn attached to the papillary muscles.

The small tendons prevent the backflow by stopping the valve leaflets from inverting.



<u>During systole the valve closes</u>, which means blood cannot do anything but be ejected out of the aortic valve and into circulation.

If the mitral valve doesn't completely shut, blood can leak back into the left atrium; this is called mitral valve regurgitation.

so "Mitral valve regurgitation means Reflux of blood from the left ventricle into the left atrium during systole".

Usually arises as a complication of mitral valve prolapse, also known as click murmur syndrome, balloon mitral valve or floppy valve syndrome (bulging of one or both of the mitral valve leaflets into the left atrium during contraction)

Causes:

a. Acute

- •Endocarditis (most often Staphylococcus aureus, it causes damage to the cardiac valves)
- •Papillary muscle rupture (from infarction) or dysfunction (from ischemia) If the papillary muscles die, they can't anchor the chordae tendineae, which then cause the mitral valve to flop back and allow blood to go from the left ventricle to the left atrium.
- •Chordae tendineae rupture(Myxomatious degeneration, mitral valve prolapse, bacterial endocarditis, and rheumatic heart disease are the leading underlying causes of chordae tendineae rupture)

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► b. Chronic

- Mitral valve prolapse (MVP): Ballooning of mitral valve into left atrium during systole
- Rheumatic fever
- Marfan syndrome: myxomatous degeneration of the leaflets and chordae results in leaflet thickening and redundancy, causing the leaflets to prolapse, or flop backwards, into the left atrium (detail). This sometimes allows leakage of blood through the mitral valve
- Cardiomyopathy

Left-sided heart failure that leads to ventricular dilation can lead to mitral regurgitation, because as the left ventricle dilates, it stretches the mitral valve annulus, or ring open, and lets blood leak into the left atrium.

Clinical features

1. Symptoms

- a. Dyspnea on exertion(shortness of breath during exercise), PND(its caused by the failure of left ventricle), orthopnea
- b. Palpitations(people with mitral valve prolapse may have imbalances in their autonomic nervous system)
- c. Pulmonary edema(increased intravascular hydrostatic pressure)

2. Signs

- a. Holosystolic murmur (starts with S1 and continues on through S2/meaning it lasts for the duration of systole) at the apex, which radiates to the back or clavicular area, depending on which leaflet is involved
- b. AFib is a common finding
- c. Other findings: Diminished S1, widening of S2, S3 gallop; laterally displaced PMI(left ventricle dilation)(point of maximal impulse); loud, palpable P2

Diagnosis

- 1. CXR: Cardiomegaly, dilated LV, pulmonary edema
- 2. Echocardiogram: MR; dilated LA and LV; decreased LV function





Treatment

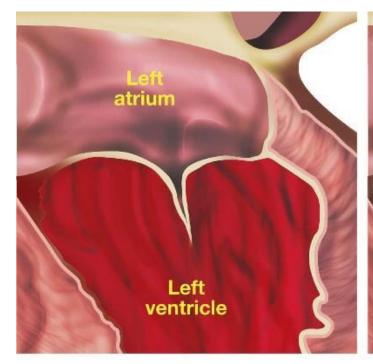
- 1. Medical
- a. Afterload reduction with <u>vasodilators</u> is recommended for <u>symptomatic</u> <u>patients only</u>; they are not recommended in most asymptomatic patients as they may mask progression of the disease.
- b. Chronic anticoagulation if patient has AFib
- c. IABP (intra-aortic ballon pump) as bridge to surgery for acute MR
- 2. Surgical
- a. Mitral valve repair or replacement
- b. Must be performed before left ventricular function is too severely

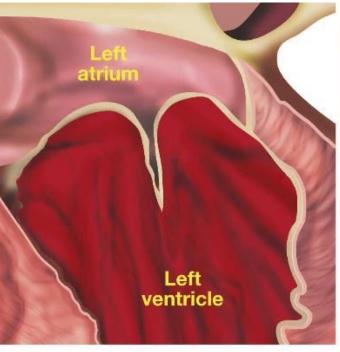
Mitral valve prolapse (MVP)

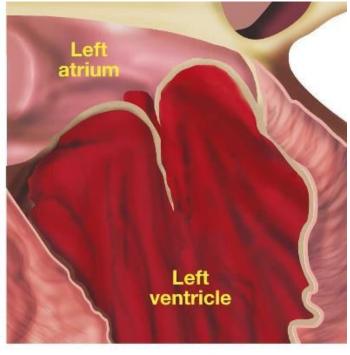
Nasser Alballam C2

Back ground

- Mitral valve prolapse (MVP) is generally benign condition, yet it is also an important contributing risk factor for arrhythmia, endocarditis, stroke, mitral regurgitation, mitral valve replacement surgery, and sudden death. but in now days the arrhythmia and sudden death are very rare.
- MVP is the most common valvular problems seen in practice, and is more common in women's.







NORMAL

Valve leaflets close and prevent backflow into the atrium.

PROLAPSE

Valve leaflets balloon upward as the ventricle contracts.

REGURGITATION

Valve leaflets do not properly close, forcing blood back into the atrium.

General characteristics

- MVP is common in pation with genetic connective tissue disorders like (Marfan syndrome, osteogenesis imperfect, and Ehlers danlos).
- MVP is a common cause of mitral regurgitation (MR) in developed countries.
- MVP is defined as the presence of excessive or redundant mitral leaflet tissue due to Myxomatous degeneration of mitral valve leaflets and/or chordae tendineae.
- In the MPV the redundant leaflets prolapse toward the LA in systole, which results in the auscultated Click and Murmur.

Clinical features 1- Symptoms

- Most patient are asymptomatic for their entire lives.
- Palpitations and atypical chest pain may occur also dyspnea and panic attacks.
- Transient ischemic attack (TIAs) due to emboli from mitral valve have been reported, but are very rare.

2-signs

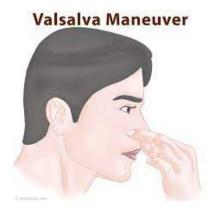
- In physical examination we will heard Midsystolic Click followed by a mid to late systolic murmur.
- In normal situation, the LV start to contracts during systole. So systolic pressure start to rise to eject the blood into aorta, the papillary muscles contract to tether their chordae tendineae and prevent mitral valve leaflets from prolapsing into left atrium.
- In MVP, because some of the chordae tendineae are elongated, they allow the mitral valve leaflet (usually posterior leaflet) to prolapse in the left atrium at midsystole. So, MR due to MVP usually starts in midsystole rether then of begening of systole (like in most of chronic MR), immediately before the MR stars at midsystole, there is a (midsystolic click) it's sound of overstrech and tension of elongated chordaea tendineae.

Cont.

- If there is also MR, the clicks followed by a mid to late systolic murmur (click murmur syndrome).
- The midsystolic click is present in MPV, but not other causes of chronic MR.
- some patients may present with sever MR after rupture of the chordae tendineae or with endocarditis.

Cont.

- Standing and valsalva maneuver increase murmur and click, because it reduce LV chamber size (by decrease preload), allowing the click and murmur to occure earlier in systole.
- Squatting and spine position decreases murmur and click, because it increases LV chamber size (by increase preload), thus delaying the onset of the click and murmur.





Diagnosis

- The Echocardiogram (echo)is the most useful diagnosis.
- Most of the patients are asymptotic, so diagnosis is typically made on the basis of the murmur and echocardiogram alone.

Treatment

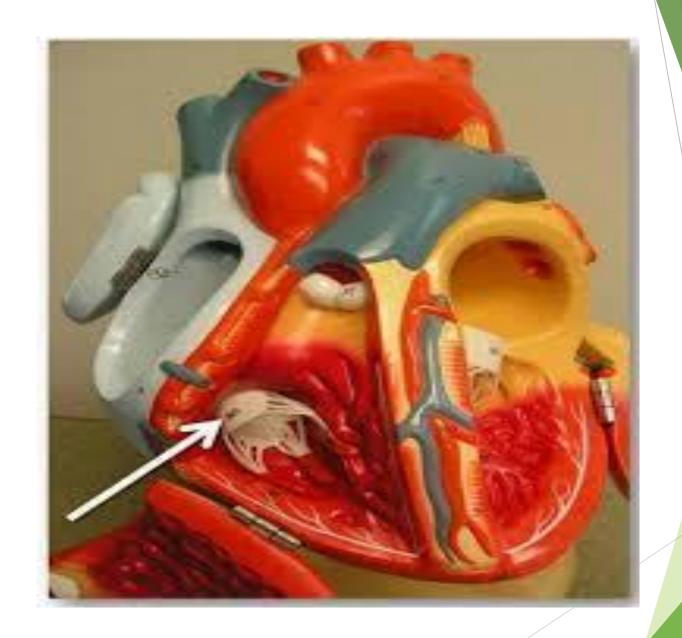
- If patient is asymptomatic, reassurance. There is some association between MVP and anxiety, so all patients should be reassured about the benign nature of this condition.
- For chest pain, B-blockers have been useful, but they are unlikely to be required.
- Surgery is rarely required. The condition is generally benign.

Thank you



Tricuspid disorder

- 1. Tricuspid regurgitation
- 2. Tricuspid stenosis



tricuspid regurgitation

Failure of tricuspid valve to close completely during systole, causing the blood to flow back to the RA.

Aetiology

- The most common cause due to dilatation of the tricuspid ring secondary to RV dilatation as M.S.
- Another Causes:
- pulmonary hypertension
- rheumatic heart disease, endocarditis
- Damage to papillary muscle due to heart attack
- Congenital: Epstein anomaly

Pathophysiology

- During systole, blood regurgitates from RV to RA causing ventricular overload
- 1. RA enlargement
- 2. RV enlargement
- 3. Right ventricular failure

Clinical features

- Symptoms:
- Systemic congestion
- General signs:
- 1. Congested pulsating neck veins
- 2. Enlarged tender pulsating liver
- 3. Ascites before oedema of LLs((Ascites precox)
- 4. Mild jaundice & peripheral cyanosis

cardiac signs:

- Precordial examination: RA & RV enlargement.
- Auscultation:"over tricuspid area"
- 1. Weak muffled \$1
- 2. S3
- 3. Pansystolic murmur

Investigations

| X- Ray | RA & RV enlargement | |
|----------|---|--|
| ECG | RA & RV enlargement | |
| Echo | Detects the severity of the lesion.Detects chamber enlargement | |
| Catheter | AS ECHO | |

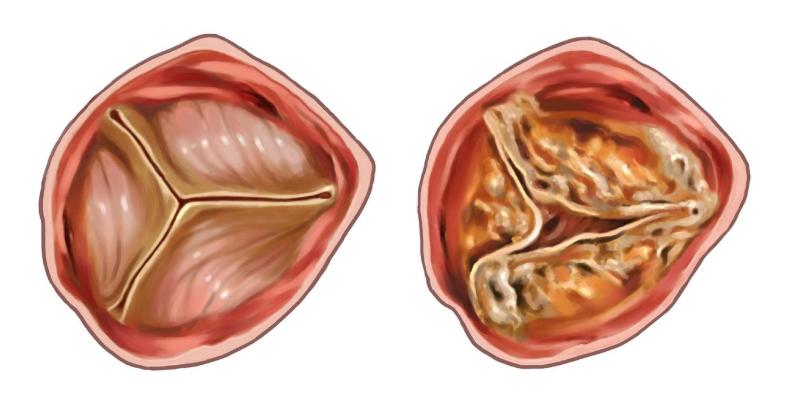
Treatment

- Treat any underlying etiology of symptomatic TR
- • 2. Diuretics for volume overload and venous congestion/edema
- • 3. Treat left-sided heart failure, endocarditis, or pulmonary HTN
- • 4. surgical treatment:
- a. Native valve repair surgery
- b. Valvuloplasty of tricuspid ring
- c. Valve replacement surgery: Rarely performed

TRICUSPID STENOSIS

 Failure of tricuspid valve to open completely during diastole, difficult to fill the RV

TRICUSPID STENOSIS



Aetiology

- Usually due to rheumatic heart disease
 Frequently associated with mitral and/or aortic valve disease(most common cause)
- TS is also seen in carcinoid syndrome

- Uncommon valve lesion
- More common in female than in males

Pathophysiology

- Reduced cardiac output
- Right atrial pressure increases
- Systemic venous congestion
- Produces hepatomegaly, ascites and dependent oedema

Clinical picture

- Symptoms:
- ► They complain the symptoms due to left sided rheumatic valve lesions
- Systemic venous congestion.

General sign:

Congested pulsating neck veins with giant A wave.

Enlarged tender pulsating liver. Ascites before oedema of lower limbs (ascites precox).

cardiac signs:

- Precordial examination:
- RA enlargement (dullness to the right border of the sternum).
- RV enlargement
- Auscultation: Over the tricuspid area:
- 1/Accentuated first heart sound.
- 2/Opening snap.
- Mid-diastolic murmur that increases by inspiration.

Investigations

- Chest X-ray:
- Prominent Right atrial bulge
- ECG:
- 1. Enlarged right atrium
- 2. Peaked, tall P waves (>3mm) in lead II
- Echocardiogram:
- May show thickened, immobile tricuspid valve

Treatment

- Diuretic therapy & salt restriction
- Tricuspid valvotomy
- Other valves usually also need replacement because tricuspid stenosis is rarely an isolated lesion

-Thanks

dania althawabieh