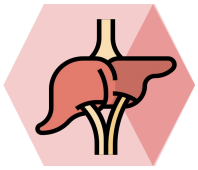
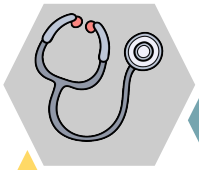
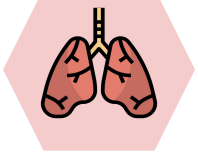
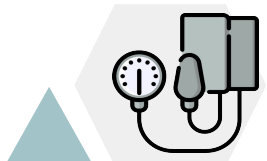
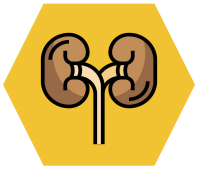
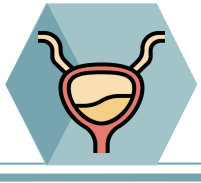
Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

Valvular Heart Diseases

Objectives :

1. Describe the etiology, pathology, and natural history of valvular heart disease.
2. Describe the clinical symptoms and signs of valvular heart disease.
3. Explain the clinical examination findings of particular valvular problems.
4. Determine the role of echocardiograms in valvular heart disease, both in diagnosis and prognosis.
5. Discuss the long-term systemic consequences of valvular heart disease.
6. Describe the management and identify the indications of surgical intervention for particular valvular heart diseases.

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Resources :

437 slides, 436 team, Davidson.

Mitral Stenosis

General Characteristics

1. Almost all cases are due to rheumatic heart disease. (Patient may not recall a history of rheumatic fever.)

Pathophysiology

a. Immune-mediated damage to the mitral valve (due to rheumatic fever) caused by cross-reactivity between the streptococcal antigen and the valve tissue 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.

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 due to increased left atrial pressure and size.

f. Patients are usually asymptomatic until the mitral valve area is reduced to approximately 1.5 cm 2 (normal valve area is 4 to 5 cm 2).

Mitral Regurgitation

General Characteristics

Pathophysiology

a. Acute Abrupt elevation of left atrial pressure in the setting of normal LA size and compliance, causing backflow into pulmonary circulation with resultant pulmonary edema Cardiac output decreases because of decreased forward flow, so hypotension and shock can occur

b. Chronic Gradual elevation of left atrial pressure in the setting of dilated LA and LV (with increased left atrial compliance) LV dysfunction occurs due to dilation Pulmonary HTN can result from chronic backflow into pulmonary vasculature

Extra slide from Step-up

Aortic Stenosis

General Characteristics

Pathophysiology

a. Causes obstruction to LV outflow, which results in LVH.

b. When the aortic valve area falls below 1 cm 2 , cardiac output fails to increase with exertion, causing angina (but may be normal at rest).

c. With long-standing AS, the LV dilates, causing progressive LV dysfunction.

d. With severe AS, LV dilation pulls the mitral valve annulus apart, causing MR.

2. Causes

a. Calcification of a congenitally abnormal bicuspid aortic valve.

b. Calcification of tricuspid aortic valve in elderly.

c. Rheumatic fever.

Course

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.

Survival is similar to that of the normal population before the development of these three classic symptoms. Without surgical intervention, the survival is poor:

Angina (35%)—average survival, 3 years Syncope (15%)—average survival, 2 years Heart failure (50%)—average survival, 1.5 years

Aortic Regurgitation

General Characteristics

Pathophysiology

a. Also called aortic insufficiency; this condition is due to inadequate closure of the aortic valve leaflets. Regurgitant blood flow increases left ventricular end-diastolic volume.

b. LV dilation and hypertrophy occur in response in order to maintain stroke volume and prevent diastolic pressure from increasing excessively.

c. Over time, these compensatory mechanisms fail, leading to increased left-sided and pulmonary pressures.

d. The resting left ventricular EF is usually normal until advanced disease.

Course

a. For chronic aortic regurgitation, survival is 75% at 5 years.

After the development of angina, death usually occurs within 4 years. After the development of heart failure, death usually occurs within 2 years.

b. For acute aortic regurgitation, mortality is particularly high without surgical repair.

Tricuspid Regurgitation

General Characteristics

1. Tricuspid regurgitation (TR) results from a failure of the tricuspid valve to close completely during systole, causing regurgitation of blood into the RA. It is estimated that up to 70% of normal adults have mild, physiologic TR as seen on high-resolution echocardiography. A much smaller percentage of people are actually symptomatic.

Mitral Valve Prolapse

General Characteristics

1. 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. The redundant leaflet(s) prolapse toward the LA in systole, which results in the auscultated click and murmur.

2. MVP is common in patients with genetic connective tissue disorders, such as Marfan syndrome, osteogenesis imperfecta, and Ehlers–Danlos syndrome.

3. MVP is a common cause of MR in developed countries.

4. Arrhythmias and sudden death are very rare.

Overview

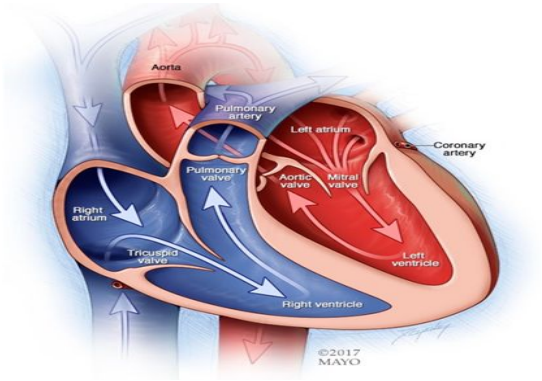
Cardiac cycle + Heart sounds 14 min start this lecture by watching this video, by one of our colleagues: (it includes the introductory part of female slides.)

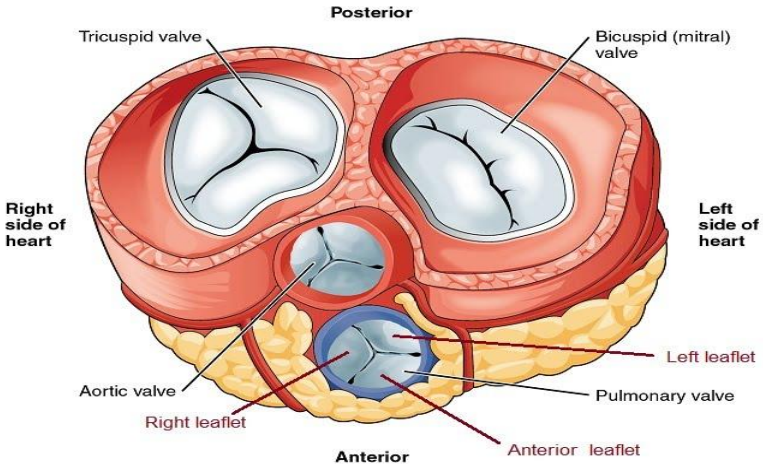
Valvular Heart Disease Explanation By OnlineMedEd

Notes from Osmosis	Mitral Valve	Pulmonary Valve
	Aortic Valve	Tricuspid Valve

Characteristics of heart valves:

Valve	Structure	Site of auscultation	Phase when valve open	Sound
Mitral	Bicuspid	Left 5th IS(intercostal space) at the midclavicular line	Diastole	S1 (LUB)
Tricuspid	Tricuspid	Left 5th IS at the SB(sternal border)		
Aortic	Semilunar (3 cusps)	Right 2nd IS at SB	Systole	S2 (DUB)
Pulmic	Semilunar (3 cusps)	Left 2nd IS at SB		



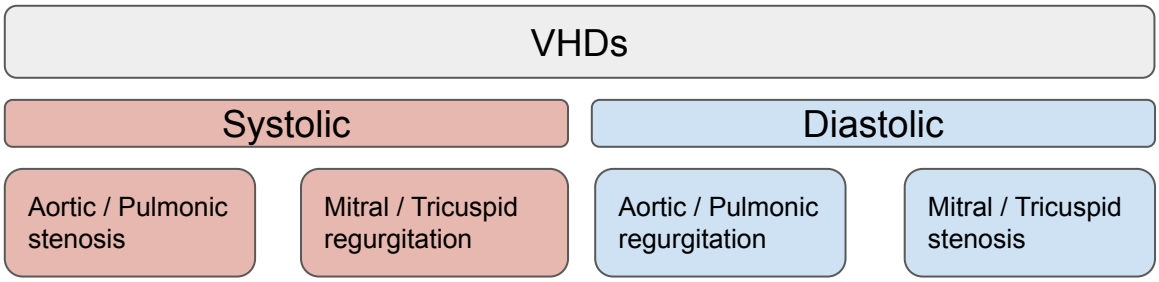


Heart sounds and significance

Sound	SIGNIFICANCE
S1	MV & TV closure; the MV closes before the tricuspid valve, so S1 may be split.
S2	AV & PV closure; the AV closes before the PV; inspiration causes increased splitting of S2.
S3	During rapid ventricular filling (early diastole) normal in children; in adults, associated with dilated ventricle (ie, dilated CHF) & increased filling pressure.
S4	Late diastole; not audible in normal adults; its presence suggest high atrial pressure or stiff ventricle (ie. Ventricular hypertrophy). The left atrium must push against a stiff LV wall ("atrial kick")

- Regurg/ Insuff: leaking (backflow; against its direction) of blood across a closed valve.
- Stenosis: Obstruction of (forward) flow across an opened valve.

Overview



Etiology

Congenital	Acquired
-Bicuspid or unicuspid . (most common congenital anomaly is bicuspid aortic valve: the valves become fused together into 2 valves and later result in aortic regurgitation and stenosis (occur together or separately) -Subvalvular or supravalvular .	-Rheumatic . -Degeneration due to: -myxomatous (abnormal collagen) -calcification (with old age) -Ischaemic . -Infective Endocarditis (who is most prone to get it? IV drug addicts). -Valve ring dilatation .

Marfan syndrome Myxomatous, Infective Endocarditis Shone syndrome, Syphilis Arthritides and more
If you are interested to know how are these related to Valvular Heart disease Click Here

TYPES of Presentations Valvular heart disease can either be:

Acute	Chronic
Acute mitral regurgitation is usually due to acute Myocardial infarction or due to acute chordae tendinae rupture. Comes suddenly with severe dyspnea (due to Pulmonary edema and heart failure) and severe chest pain..	- Mitral valve prolapse - chronic mitral regurgitation usually due to rheumatic fever. A 40-year-old patient having rheumatic fever since he was 10 and for the last 25 years he has mitral regurgitation. - chronic aortic regurgitation may be due to Bicuspid aortic valve. History is essential in valvular disease: you have to know onset, duration and severity of symptoms

Hemodynamic consequences of Valvular heart disease:

Pressure overload:

1- Aortic stenosis
(which leads to Left ventricular hypertrophy)
2- Mitral stenosis
(which leads to Left atrial hypertrophy & dilation)

Volume overload:

1- Chronic mitral regurgitation:
(leads to dilation of Left atria and Left ventricle)
2- Chronic tricuspid regurgitation:
(leads to dilation of right atria and right ventricle)

Overview

Symptoms of Valvular heart disease:

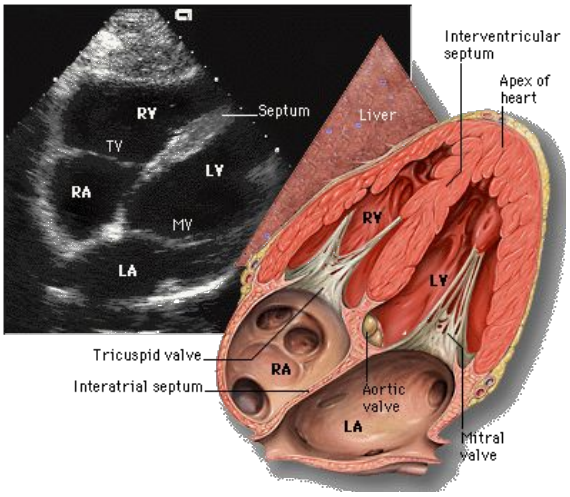
- Dyspnea Commonest (present for all valves), paroxysmal nocturnal dyspnea
- orthopnea .
- Palpitation .
- Chest pain. is typical of Aortic stenosis
- Dizziness , pre fainting ,syncope present in aortic stenosis.
- Oedema , Ascites typical of tricuspid regurgitation
- Cough. due to the increase in pulmonary artery pressure from mitral stenosis
- Fatigue due to low cardiac output
- Hemoptysis due to mitral stenosis (precisely from the high pulmonary artery pressure)
- Symptoms of thromboembolic complication (clot formation due to atrial fibrillation)

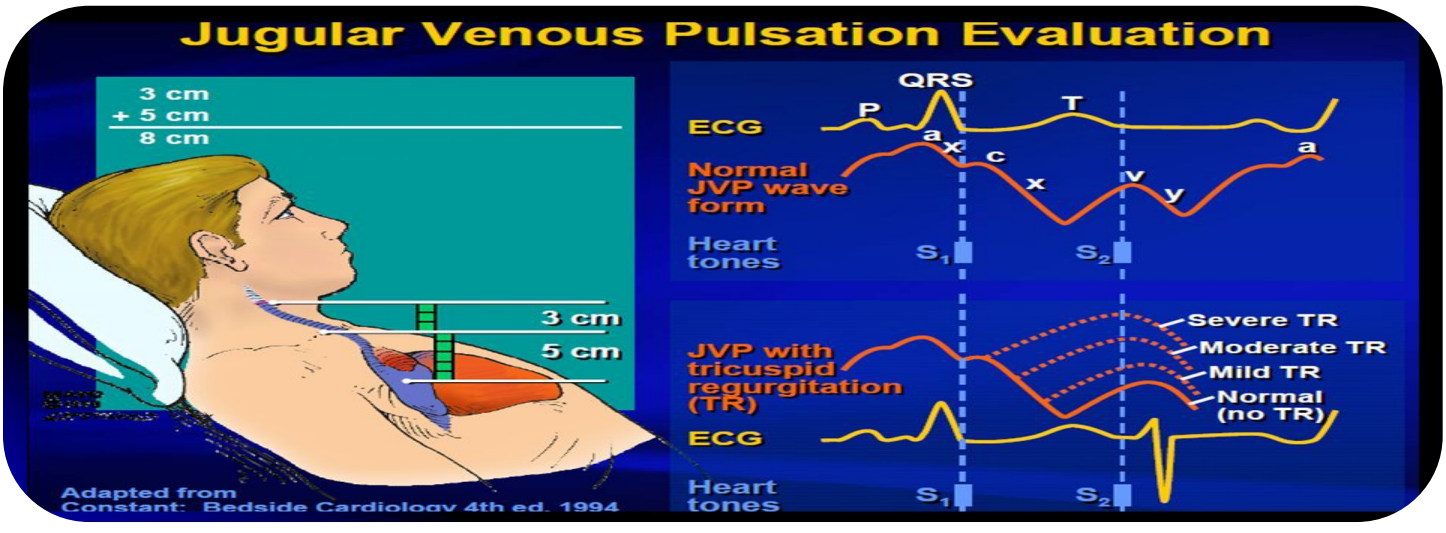
Signs of Valvular heart disease:

- **Abnormal look (mitral facies)** red patches similar to butterfly malar rash that comes with SLE, but the difference is that in SLE it extends to the nose bridge.
- **Abnormal pulse (Atrial fibrillation** = irregularly irregular pulse**)**
- **Abnormal JVP** due to right sided heart failure
- **Apex beat abnormality** if shifted from 5th intercostal space it means the heart is enlarged (but you should also check the trachea because the whole hilum or mediastinum may be shifted)
- **Sternal or parasternal heave** in pulmonary hypertension and right ventricular hypertrophy
- **Thrill (Palpable murmur)** at the apex, base of the heart and aortic area
- **Abnormal heart sound**
- **Murmurs (systolic or diastolic)**

Investigations:

- ECG .
- CXR .
- **Echo cardiology.** Gold standard test for the heart chambers (viewing valves) M mode, 2D ,3D, 4D,TEE, Doppler. Best initial Test
- 24 hours monitor for heart rhythm .
- MRI .
- Cardiac catheterization .
- Exercise test.





Overview

Stages of VHD

It is important to know the general stages of VHD

Stage	Definition	Description
A	At risk	Patients with risk factors for development of VHD
B	Progressive	Patients with progressive VHD (Mild-Moderate) (Asymptomatic) No hemodynamic consequences and the patient is tolerating well.
C	Asymptomatic Severe	Asymptomatic but reached the criteria of severe VHD C1: Asymptomatic with compensated cardiac function. C2: Asymptomatic but decompensated cardiac function.
D	Symptomatic Severe	Developed symptoms secondary to VHD

Frequency of Echocardiograms in Asymptomatic Patients With VHD and Normal Left Ventricular Function

Stage	Valve Lesion			
Stage	**Aortic Stenosis***	**Aortic Regurgitation**	**Mitral Stenosis**	**Mitral Regurgitation**
Progressive (stage B)	Every 3-5 y (mild severity V_{max} 2.0-2.9 m/s) Every 1-2 y (moderate severity V_{max} 3.0-3.9 m/s)	Every 3-5 y (mild severity) Every 1-2 y (moderate severity)	Every 3-5 y (MVA >1.5 cm^2)	Every 3-5 y (mild severity) Every 1-2 y (moderate severity)
Severe (stage C)	Every 6-12 mo (V_{max} ≥4 m/s)	Every 6-12 mo Dilating LV: more frequently	Every 1-2 y (MVA 1.0-1.5 cm^2) Once every year (MVA <1.0 cm^2)	Every 6-12 mo Dilating LV: more frequently

Mitral Stenosis

Definition:

It is the restriction and narrowing of the Mitral valve + impairment of left ventricular filling

Most common lesion caused by rheumatic fever consisting of thickened mitral valve leaflets, fused commissures, and chordae tendineae. May result in right ventricular failure.

(Commonly found in pregnant women because they have a 50% increase in plasma during pregnancy which makes the symptoms more prominent)

Etiology:

the left ventricle is spared.

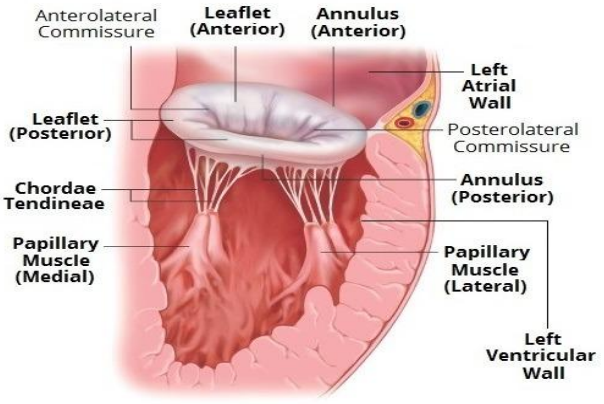
1. **Rheumatic Fever**: related to streptococcus infections, causing damage to the mitral valve and leading to mitral stenosis later in life. (the most common)
2. **Other less common causes:**

-Bacterial Endocarditis, Congenital mitral stenosis, Systemic Lupus Erythematosus, Rheumatoid Arthritis, Atrial Myxoma, Malignant Carcinoid. scarring & fusion of valve, extensive calcification,Rheumatoid Arthritis, Atrial Myxoma (tumor), Malignant Carcinoid, valvular dilation.

Mitral valve apparatus:

important to remember the anatomy

- Mitral valve leaflets (AML and PML) the area between 2 leaflets is called commissure.
- Mitral valve annulus
- Chordae tendinaea
- Papillary muscles
- Left ventricular myocardium



Mitral stenosis results in several changes to the integrity of the valves :

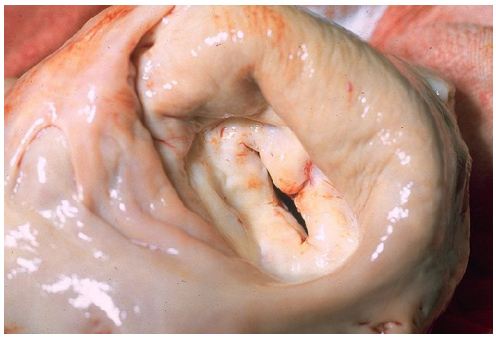
1- cusps thicken

2- calcium deposits form

3- commissures fused together

4- chordae tendineae becomes thickened & shortened

Fish mouth appearance (very imp)



A three minute video explaining the signs and symptoms of mitral stenosis

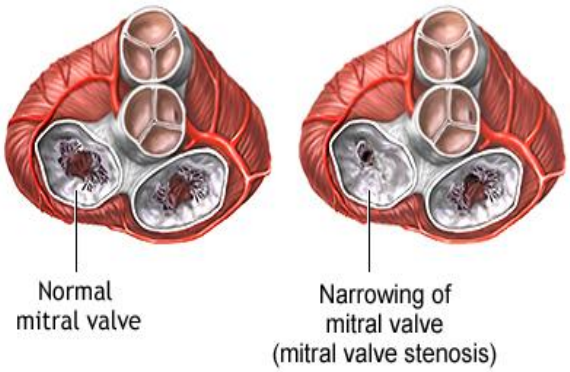
Mitral Stenosis

Pathophysiology:

The mitral valve is stenosed so we are not allowing blood to come down to LV. The atrium will become dilated and will have abnormal filling thus the ventricle will receive limited blood flow.

Pathogenesis:

Cusps thicken > commissures (the area between 2 leaflets) fused together > chordae tendineae becomes thickened and shortened > calcium deposits form.



1. Increase in left atrial pressure

- Pulmonary interstitial edema (congestion).
- Pulmonary hypertension:
  - ➔ Passive > obligatory to preserve forward flow
  - ➔ Reactive > vascular changes in 40%
    - Protects interstitium from edema
    - Leads to right heart failure (pts w\ MS come w\ right side HF first)
- Left atrial stretch & enlargement which lead to atrial fibrillation:
  - ➔ ↑ HR ↓ Left ventricular filling.
  - ➔ ↓ atrial "kick" ↓ Left ventricular filling
  - ➔ Atrial thrombus formation & embolus.

2. Limited Left ventricular filling & cardiac output:

~ When the orifice is reduced to approximately 2cm², which is considered mild mitral stenosis, blood can flow from the left atrium to the left ventricle only if propelled by an abnormal pressure gradient – the hemodynamic hallmark of Mitral Stenosis.

3. RV failure, RVH, RV pressure overload
4. Hepatic congestion, JVD, Right Heart Failure, Tricuspid Regurgitation, RA enlargement.

Stages: (Very Important)

Know what is in red box; severe pathology

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of MS	• Mild valve doming during diastole	• Normal transmitral flow velocity	• None	• None
B	Progressive MS	• Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets • Planimetered MVA >1.5 cm^2	• Increased transmitral flow velocities • MVA >1.5 cm^2 • Diastolic pressure half-time <150 ms	• Mild-to-moderate LA enlargement • Normal pulmonary pressure at rest	• None
C	Asymptomatic severe MS	• Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets • Planimetered MVA ≤1.5 cm^2 • (MVA ≤1.0 cm^2 with very severe MS)	• MVA ≤1.5 cm^2 • (MVA ≤1.0 cm^2 with very severe MS) • Diastolic pressure half-time ≥150 ms • (Diastolic pressure half-time ≥220 ms with very severe MS)	• Severe LA enlargement • Elevated PASP >30 mm Hg	• None
D	Symptomatic severe MS	• Rheumatic valve changes with commissural fusion and diastolic doming of the mitral valve leaflets • Planimetered MVA ≤1.5 cm^2	• MVA ≤1.5 cm^2 • (MVA ≤1.0 cm^2 with very severe MS) • Diastolic pressure half-time ≥150 ms • (Diastolic pressure half-time ≥220 ms with very severe MS)	• Severe LA enlargement • Elevated PASP >30 mm Hg	• Decreased exercise tolerance • Exertional dyspnea

Mitral Stenosis

Symptoms:

Increased left atrial pressure and decreased cardiac output produce the symptoms of mitral stenosis.

The mitral valve orifice is normally about 5 cm in diastole and may be reduced to 1 cm in severe mitral stenosis. Patients usually remain asymptomatic until the stenosis is less than 2 cm.

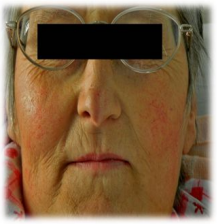
- Dyspnea on exertion (First bouts of dyspnea in patients with MS are usually precipitated by exercise, emotional stress, infection, or Atrial fibrillation all of which increase the rate of blood flow across the mitral orifice and result in further elevation of left atrial pressure) > pulmonary venous congestion.
- Fatigue > ↓ cardiac output.
- Inability to tolerate the increased volume,
- Inability to tolerate the increased HR: ↓ filling ↑ LA pressure , ↑ pulmonary hypertension pulmonary vein congestion & AF. (Hallmark of MS.)

➔ The increased HR ↓ flow rate in the valve, bc the valve is narrow, the diastole filling time is ↓ more tension & pressure

- Hemoptysis due to rupture of thin dilated bronchial veins.
- Orthopnea, paroxysmal nocturnal dyspnea (PND), pulmonary edema (developed when there is a sudden flow rate across a markedly narrowed mitral orifice), palpitation (arrhythmias), Chest pain, Peripheral edema.
- PND
- PHT symptoms: RHF, hemoptysis
- Palpitation
- Peripheral embolism secondary to AF
- Ortner's syndrome recurrent laryngeal nerve compression by dilated LA (cause Hoarseness)
- Hoarseness (due to impingement of an enlarged left atrium on the recurrent laryngeal nerve).
- Systemic embolism (due to stagnation of blood in an enlarged left atrium).

Signs (on examination):

A lot of the signs will be lost when the valve is calcified.



- Face: Mitral faces pink purple plaques on cheeks (due to systemic VC) ⟶
- Mitral stenosis murmur:
  - The opening snap is followed by a low-pitched diastolic rumble (due to turbulent blood flow across the stenotic valve) and **presystolic accentuation**. (squatting & leg raising increase the intensity) Diastolic murmurs need maneuvers because it is low pitched,
- Loud S1, due to abrupt leaflet closure > won't occur if the valve is calcified. may be the most prominent physical finding.
- Loud P2 (due to pulmonary hypertension) which is followed by an opening snap The develop heaves as a result of pulmonary hypertension leading to load S2,S1.
- Apex: Tapping beat (palpable S1)
- JVP: Prominent a wave in sinus rhythm
- Diastolic rumble
- S3 CANNOT BE HEARD IN MITRAL STENOSIS. (which makes complete sense when you actually think about it, S3 is caused by an increase in blood flow through the mitral valve but in MS flow is reduced)
- parasternal Heaves due to pulmonary hypertension
- May be associated with:
  - Mitral regurgitation or aortic stenosis.
  - Right sided murmurs:
    1. Pulmonary insufficiency > Graham Steel Murmur (early diastolic murmur)
    2. Tricuspid regurgitation
- Atrial fibrillation (irregular cardiac rhythm)
- Sternal lift (due to right ventricular enlargement)

Mitral Stenosis

Diagnosis:

Imp there will be a question about specifically ECG

1.Clinical evaluation of Mitral Stenosis begins with an in-depth history and physical exam

2.ECG: (ECG is helpful but not diagnostic!) may show

- ➔ Atrial fibrillation (AFib), right atrial enlargement (eventually because of Tricuspid regurgitation), right ventricular hypertrophy.
- ➔ Broad notched p waves. left atrial enlargement (biphasic P wave in leads V1 & V2)

the P wave is absent, or the P wave is present but the left atrium is enlarged so

I will see M shaped P wave (P mitrale)

Mitral stenosis shows left atrial enlargement is seen mainly. right atrial enlargement occurs in advanced stages.

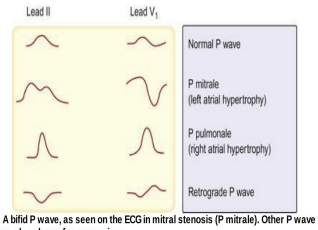


FIGURE 23.75 A bifid P wave, as seen on the ECG in mitral stenosis (P mitrale). Other P wave abnormalities are also shown for comparison.

3.Echocardiography (Echo 2D/color doppler) >TEST OF CHOICE.

- ➔ TransEsophageal Echocardiography.
- ➔ Asses mitral valve mobility, gradient and mitral valve area, thickened MV & LAE

4.Cardiac Cath >helpful, confirmatory. Needed if the pt is older (look at the coronaries). MOST ACCURATE TEST

5.Chest radiology (CXR): LAE: straightening of the left heart border(Because of left atrial enlargement), Pulmonary congestion and prominent PA, Calcified MV, Bulging on the right (because of the right atrial enlargement)

If you have a case with loud S1, Mid-diastolic murmur with AF >> Indicates MS

Complications:

- Atrial fibrillation
- Lung congestion.
- Blood clots with systemic embolization (due to stagnation of blood in an enlarged left atrium)
- Pulmonary hypertension
- Congestive heart failure (CHF)

Treatment:

Treatment of symptomatic mitral stenosis:

1. Medical therapy (treat the symptoms not the cause).

- **Diuretics** for pulmonary congestion. (most important group of drugs for MS)
- Digoxin (Digitalis), Beta & Ca channel blockers for AFib rate control. (Digoxin here is not used to increase contractility, it is used to block conduction through the AV node)
- Antiarrhythmic
- Anticoagulation for AFib & LA clots (warfarin is the only effective anticoagulant)
- Antibiotics, SBE prophylaxis prevent endocarditis
- Antiplatelets (Aspirin and clopidogrel) are NOT helpful


Mechanical Valve


Tissue Valve

2. Surgical therapy (treat the cause): you choose according to the severity of the condition

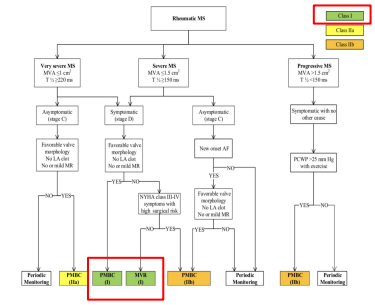
- - Percutaneous Balloon Valvuloplasty for Non-calcified, pliable valve (in pregnant women it is better to do PBV and to avoid Diuretics because they can cause intrauterine growth retardation) Also called Percutaneous Transvenous Mitral Commissurotomy (PTMC)
- Open Commissurotomy – valve repair
- Mitral Valve Replacement:

There are two types of valves For any valve replacement:

A- mechanic valve: recommended for young to mid aged patients, functional up to 20-30 years (long duration). anticoagulants are taken for life.

B- tissue valve: recommended for elderly and pregnant woman. No need for anticoagulants (if they have sinus rhythm).

Indications for Intervention for Rheumatic MS



Just know Class 1 is indicated for surgery

Mitral Regurgitation

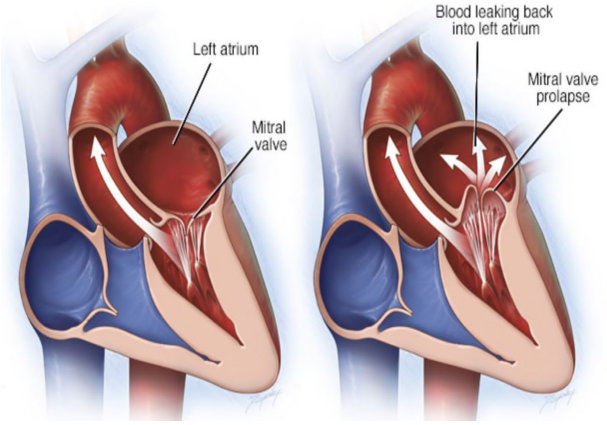
A three minute animation explaining mitral regurgitation and prolapse with a sound clip of the murmurs

Definition:

Backflow of blood from the left ventricle into the left atrium, due to inadequate functioning (insufficiency) of the mitral valve. Most commonly from ischemia. Nowadays, it is more common than MS.

Etiology:

1. Alterations of the Leaflets, Commissures, Annulus:
   - Rheumatic heart disease
   - Mitral valve prolapse
   - Infective Endocarditis
2. Alteration of Left ventricular or Left atrial size and function:
   - Papillary muscle (Ischemia, MI, Myocarditis)
   - Hypertensive heart disease
   - Cardiomyopathies (dilated, hypertrophic)
   - Left Ventricular enlargement
   - LA enlargement from Mitral Regurgitation (MR begets MR)
3. Others:
   - Connective tissue disorders (SLE)
   - Collagen abnormality (Marfan's syndrome)



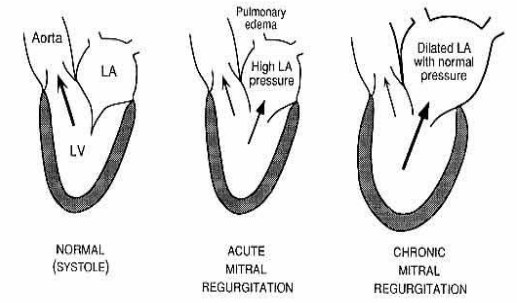
Pathophysiology:

1. A portion of the left ventricular stroke volume is pumped backward into the left atrium instead of forward into the aorta, resulting in increased left atrial pressure and decreased forward cardiac output.(LAv is increased bc of increased LVEDv)
2. Volume overload occurs, increasing preload.
3. Afterload is decreased as the left ventricle empties part of its contents into the relatively low- pressure left atrium.
4. This helps to compensate for the regurgitation by augmenting ejection fraction.
5. Left ventricular dysfunction occurs after prolonged compensation.

Mitral Regurgitation

Symptoms:

Acute MR	Chronic MR
• Decompensated HF symptoms: Dyspnea, orthopnea, PND) • Low cardiac output state • Cardiogenic shock	• Initially asymptomatic • HF symptoms (Dyspnea, orthopnea, PND, LL edema) • Decreased exercise tolerance • Palpitation with AF if present • PHTN symptoms if present



Signs:

- Pulse: Large Volume Collapsing
- JVP: prominent V wave
- Apex: Diffuse (you can't pinpoint it) with Lateral displacement +/- palpable thrill
- HS: Normal or Soft S1, S2 physiological split or wide split due to premature AV closure.
- Laterally displaced (forceful) diffuse apex beat and a systolic thrill. Due to large heart
- Soft first heart sound. (owing to the incomplete apposition of the valve cusps and their partial closure by the time ventricular systole begins.)
- Prominent **S3** Gallop (increased volume during diastole). owing to the sudden rush of blood back into the dilated left ventricle in early diastole (sometimes a short mid-diastolic flow murmur may follow the third heart sound). Gallop= S3 + tachycardia
- Split S2 (but is obscured by the murmur).
- Pansystolic murmur (due to the occurrence of regurgitation throughout the whole of systole, being loudest at the apex but radiating widely over the precordium and into the axilla.) **radiating to the axilla** and often accompanied by a thrill. Best heard on the lateral side when lying on the side.
- Significant crackles are heard due to pulmonary edema (acute)

example of questions:
- Criteria of severe
- signs of MR
- Physical signs of severe MR

Stages of Chronic MR

Know what is in red box; severe pathology

Grade	Definition	Valve Anatomy	Valve Hemodynamics*	Hemodynamic Consequences	Symptoms
A	At risk of MR	• Mild mitral valve prolapse with normal coaptation • Mild valve thickening and leaflet restriction	• No MR jet or small central jet area <20% LA on Doppler • Small vena contracta <0.3 cm	• None	• None
B	Progressive MR	• Severe mitral valve prolapse with normal coaptation • Rheumatic valve changes with leaflet restriction and loss of central coaptation • Prior IE	• Central jet MR 20%–40% LA or late systolic eccentric jet MR • Vena contracta <0.7 cm • Regurgitant volume <60 mL • Regurgitant fraction <50% • ERO <0.40 cm^2 • Angiographic grade 1–2+	• Mild LA enlargement • No LV enlargement • Normal pulmonary pressure	• None
C	Asymptomatic severe MR	• Severe mitral valve prolapse with loss of coaptation or flail leaflet • Rheumatic valve changes with leaflet restriction and loss of central coaptation • Prior IE • Thickening of leaflets with radiation heart disease	• Central jet MR >40% LA or holosystolic eccentric jet MR • Vena contracta ≥0.7 cm • Regurgitant volume ≥60 mL • Regurgitant fraction ≥50% • ERO ≥0.40 cm^2 • Angiographic grade 3–4+	• Moderate or severe LA enlargement • LV enlargement • Pulmonary hypertension may be present at rest or with exercise • **C1:** LVEF >60% and LVESD <40 mm • **C2:** LVEF ≤60% and LVESD ≥40 mm	• None
D	Symptomatic severe MR	• Severe mitral valve prolapse with loss of coaptation or flail leaflet • Rheumatic valve changes with leaflet restriction and loss of central coaptation • Prior IE • Thickening of leaflets with radiation heart disease	• Central jet MR >40% LA or holosystolic eccentric jet MR • Vena contracta ≥0.7 cm • Regurgitant volume ≥60 mL • Regurgitant fraction ≥50% • ERO ≥0.40 cm^2 • Angiographic grade 3–4+	• Moderate or severe LA enlargement • LV enlargement • Pulmonary hypertension present	• Decreased exercise tolerance • Exertional dyspnea

Mitral Regurgitation

Examination:

Can be either acute or chronic:

1 **Acute:** sitting upright, you can hear rales (wet lungs) and the murmur can be subtle.

2 **Chronic:** Apical impulse, diffuse, tapping. May have pulmonary findings. S3 +/- palpable

Diagnosis:

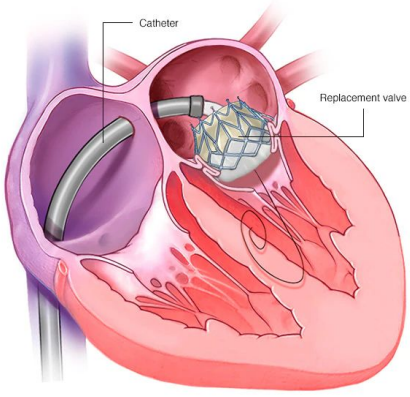
- ECG:
  - ➔ Left Atrial Enlargement & LVH.
- Echocardiography (Echo 2D/color doppler)test of choice.
- Cardiac Cath – helpful, confirmatory, needed if the pt is older – look at the coronaries

Treatment:

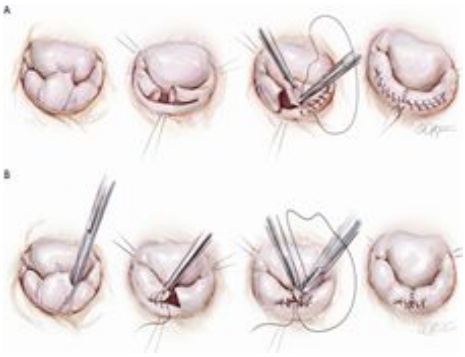
- Evidence of progressive cardiac enlargement warrants early surgical intervention by either mitral valve repair or replacement (an increase in left ventricular end diastolic diameter by 45 mm and an a 60% decrease in Ejection fraction)
- Treatment of symptomatic mitral regurgitation:

1. Medical therapy Diuretics, Vasodilators (ACE inhibitors, they are the most important group of drugs in MR because they decrease Afterload which enhances cardiac output and they also reduce pulmonary congestion) & SBE prophylaxis. (also possibly anticoagulant)
2. Surgical therapy:
   - Mitral valve repair or replacement Must be performed before left ventricular function is too severely compromised
   - Mitraclip

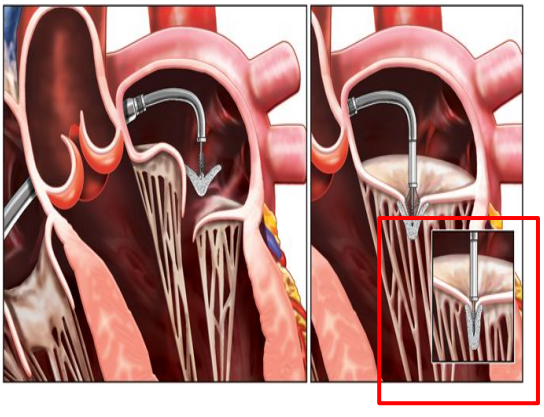
Transcatheter Mitral valve replacement



Mitral valve repair



Mitraclip



Mitral Regurgitation

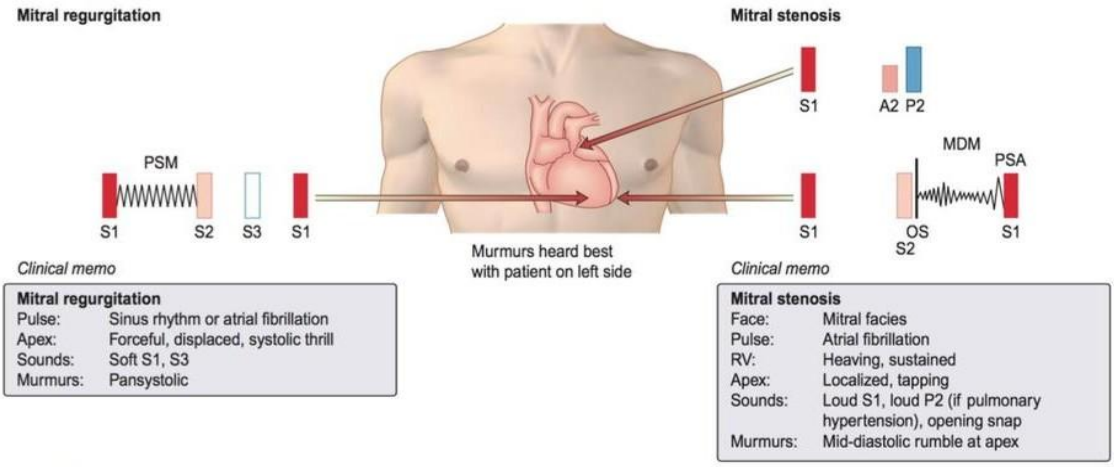


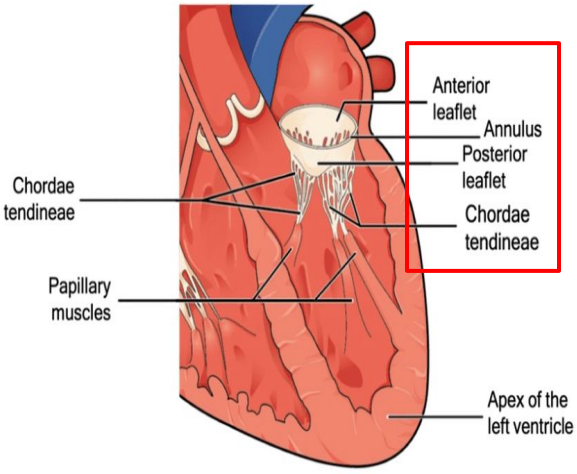
Figure 14.71 Features associated with mitral regurgitation and mitral stenosis. A2, aortic component of the second heart sound; MDM, mid-diastolic murmur; OS, opening snap; P2, pulmonary component of the second heart sound (loud with pulmonary hypertension); PSA, presystolic accentuation; PSM, pansystolic murmur; S1, first heart sound; S2, second heart sound; S3, third heart sound.

Mitral Valve Prolapse

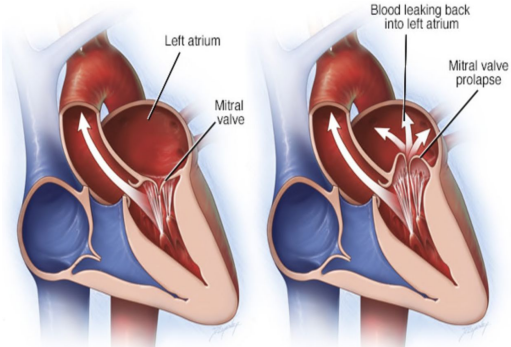
Pathophysiology:

Prolapsing (billowing) mitral valve This is also known as Barlow syndrome or floppy mitral valve. It is due to excessively large mitral valve leaflets, an enlarged mitral annulus, abnormally long chordae or disordered papillary muscle contraction. Histology may demonstrate myxomatous degeneration of the mitral valve leaflets. Prolapsing mitral valve is more commonly seen in young women than in men or older women, and has a familial incidence. Its cause is unknown but it is associated with connective tissue disorders (Marfan syndrome, Ehlers–Danlos syndrome and pseudoxanthoma elasticum). It also occurs in association with an atrial septal defect and Ebstein's anomaly.

- Large mitral valve leaflets, an enlarged mitral annulus, abnormally long chordae or disordered papillary muscle contraction. The valve moves toward the atria in systole
- Demonstrate myxomatous degeneration due to volume overload of the mitral valve leaflets.
- Associated with Marfan's syndrome, thyrotoxicosis, rheumatic or ischaemic heart disease.
- Myxomatous Heart Disease is the most common cause of chronic Mitral Regurgitatic in middle aged asymptomatic men (marfan's syndrome)



Abnormal mitral apparatus
↓
mitral leaflet prolapse
↓
papillary muscle traction, activation of stretch receptors
↓
papillary muscle & subendocardial ischemia
↓
pain, ventricular arrhythmias.
↓



Mitral Valve Prolapse

Signs:

- 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.
- Late systolic murmur (if associated with MR). > Mid-or-late systolic click

★ **Prognosis:**

- Often benign, rare complications:
  - ➔ Endocarditis
  - ➔ Cardiac failure
  - ➔ Progressive Mitral regurgitation: (Acute or chronic).
  - ➔ Thromboembolism.
  - ➔ Atrial & ventricular arrhythmias.

Symptoms:

- Atypical chest pain is the most common symptom.
- Palpitations may be experienced because of the abnormal ventricular contraction or because of the atrial and ventricular arrhythmias.
- Sudden cardiac death due to fatal ventricular arrhythmias is a very rare but recognized complication.

Treatment:

- B-Blockers (for hyperadrenergic symptoms, atypical chest pain & Palpitations).
- Mitral valve prolapse associated with significant mitral regurgitation and atrial fibrillation, anticoagulation is advised to prevent thromboembolism. Mitral valve prolapse associated with severe mitral regurgitation has a risk of sudden cardiac death.
- SBE Prophylaxis (only if associated with MR).
- Severe Symptomatic MR – same as chronic MR.

Aortic Stenosis

A three minute animation explaining both aortic stenosis and regurgitation with a sound clip of the murmurs

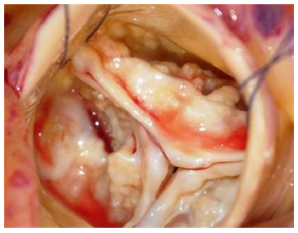
Definition:

It is a chronic progressive disease that produces obstruction to the left ventricular stroke volume leading to symptoms of chest pain, breathlessness, syncope and presyncope and fatigue. Most common VHD.
It is usually a disease of the young (bicuspid) or the elderly (degenerative)

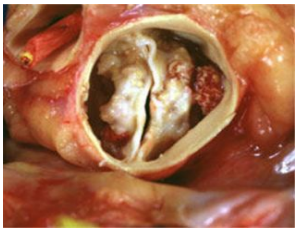
Etiology:

- Calcification and degeneration of a normal valve; more common in the elderly population.
  - (the most common cause) > 70 yrs.
- Calcification and fibrosis of a congenitally bicuspid aortic valve.
  <= 60 years (1.8% of population)
- Rheumatic valvular disease (3rd most common cause) 30-60 yrs
  - If the aortic valve is affected by the rheumatic fever, the mitral valve is also invariably affected.

Degenerative

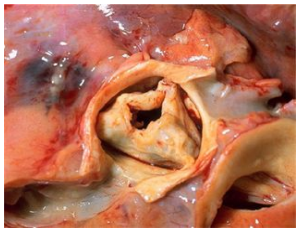


Bicuspid



Rheumatic

Rheumatic fever affect the commissures. inflammation of the endocardial lining leads to carditis which cause almost complete attachment of the leaflets (commissures).



Aortic Stenosis

Stages:

Doctor said : no need to know the measurements, just know the general concept and what each stage mean .
*Focus on 4m/s and 40 mm hg

Stage	Definition	Valve Anatomy	Valve Hemodynamics	Hemodynamic Consequences	Symptoms
A	At risk of AS	• Bicuspid aortic valve (or other congenital valve anomaly) • Aortic valve sclerosis	• Aortic V_{max} <2 m/s	• None	• None
B	Progressive AS	• Mild-to-moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion or • Rheumatic valve changes with commissural fusion	• Mild AS: Aortic V_{max} 2.0–2.9 m/s or mean ΔP <20 mm Hg • Moderate AS: Aortic V_{max} 3.0–3.9 m/s or mean ΔP 20–39 mm Hg	• Early LV diastolic dysfunction may be present • Normal LVEF	• None
C: Asymptomatic severe AS					
C1	Asymptomatic severe AS	• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	• Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg • AVA typically is ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2) • Very severe AS is an aortic V_{max} ≥5 m/s or mean ΔP ≥60 mm Hg	• LV diastolic dysfunction • Mild LV hypertrophy • Normal LVEF	• None: Exercise testing is reasonable to confirm symptom status
C2	Asymptomatic severe AS with LV dysfunction	• Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	• Aortic V_{max} ≥4 m/s or mean ΔP ≥40 mm Hg • AVA typically ≤1.0 cm^2 (or AVAi ≤0.6 cm^2/m^2)	• LVEF <50%	• None

Grades of AS

what you have to know
(Severe AS)

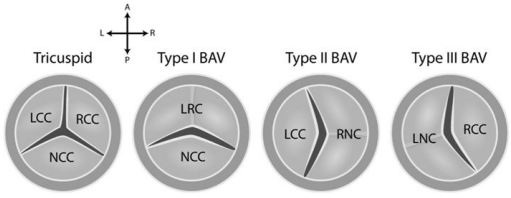
	Aortic sclerosis	Mild AS	Moderate AS	Severe AS
Peak Velocity (m/s)	≤ 2.5 m/s	2.6- 2.9	3.0 - 4.0	≥ 4.0
Mean gradient (mmHg)	-	< 20	20 -40	≥ 40
AVA (cm2)	-	>1.5	1.0 - 1.5	< 1
Indexed AVA (cm2/m2)	-	> 0.85	0.60 - 0.85	< 0.6

Bicuspid Aortic Valve:

Not only affect the valve but it also may lead to aortic dilatation.
When I screen the patient, I should screen the whole family too.

- 1-2% of the population
- 70-80% fusion of the right & left coronary and non-coronary leaflets
- 20-30% fusion of the right & non-coronary leaflets
- Fusion of the non coronary & left coronary leaflets is rare
- One commissure
- Associated aortopathy (Medial degeneration) : aneurysm, dissection.
- Requires annual imaging if aorta > 4.5 cm should do imaging not just echo.
- Beta blockers in absence of significant AI

Types of BAV! This is very important.



Asc A replacement if:

Aorta > 5.5 cm

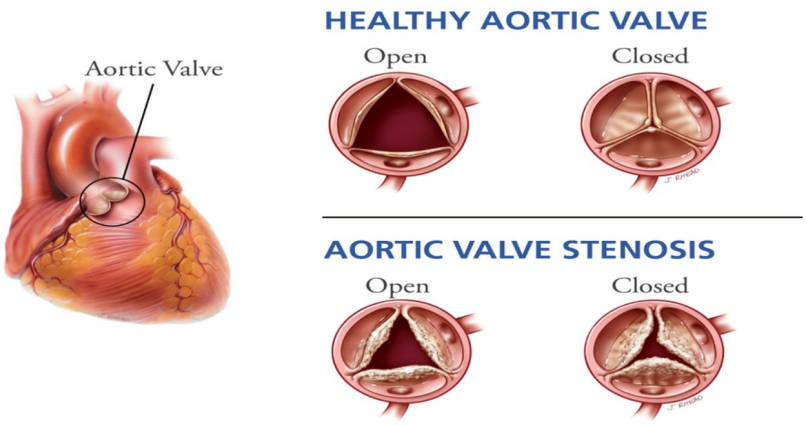
Aorta > 4.5 cm if AVR indicated

Aorta >5 cm with risk factors for dissection e.g FHX or progression of >0.5 cm/y

Aortic Stenosis

Pathophysiology:

- Obstructed left ventricular emptying > ↑ left ventricular pressure (pressure overload) > compensatory left ventricular hypertrophy (reduce wall stress, reduce vent. Compliance, ↑ LVEDp & ↑ LAp, Cardiac output is reduced) Forceful atrial contraction augments filling at the thick, non compliant ventricle and generates a prominent S4 gallop that elevates the left ventricular end-diastolic pressure.
- Left ventricular hypertrophy 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. soon, the actin and myosin fibers give up and they seperate leading to dilation
- As the myocardium fails, mean left ventricular diastolic pressure increases, and symptoms of pulmonary congestion ensue.



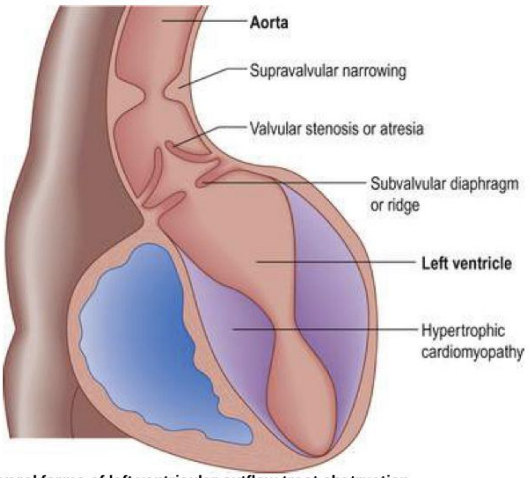


FIGURE 23.82 Several forms of left ventricular outflow tract obstruction.

Symptoms:

severe Aortic stenosis usually presents with acute symptoms but moderate and mild Aortic stenosis can be Asymptomatic.

- Angina (most common) > imbalance between supply & demand > 5 yrs survival.
  1. ↑ LVEDp > leads to ↓ perfusion pressure.
  2. Myocardial hypertrophy ↑ demand.
- Syncope with exertion > 3 yr survival
  - Syncope usually occurs on exertion when cardiac output fails to rise to meet demand, leading to a fall in BP.
- Congestive heart failure (CHF) > 2 yrs survival (the **worst**). (such as dyspnea on exertion, orthopnea, or PND) we have to intervene right away.
  - ↑ LVEDp > ↑ LAp > pulmonary venous congestion.

Differential diagnosis:

↳Supravalvular - murmur R carotid, ↑ A2

↳Subvalvular - often leads to AR Hypertrophic CardioMyopathy

* Stenosis doesn't have to happen at the valve level, it can happen above or below the valve.
In hypertrophic cardiomyopathy(HCM) all ventricular wall are thickened, so when the ventricles contract during systole they obstruct the blood flow.
It gives something similar to Aortic stenosis, but in HCM the obstruction is transient, it is a dynamic obstruction not a fixed one.

Aortic Stenosis

Signs:

- In severely AS > low BP, bc of low blood ejected from the ventricle to aorta.
- Moderate AS usually has normal BP
- Pulses:

- Pulsus Parvus et Tardus (slow rising , delayed peaking) (narrow pulse pressure) (Carotid Impulse) > The carotid pulse is of small volume & slow-rising or plateau in nature. (Slow & late impulses)
- Sustained Bifid LV impulse (from LVH). - Brachioradial delay.
- Presence of a thrill at the base of the heart

- Auscultation:

- Harsh Systolic Ejection Murmur – late peaking (diamond-shaped, Crescendo- decrescendo), usually with thrill, radiates to carotids.
- Prominent S4 gallop (from LVH) > it is heard unless co-existing mitral stenosis prevent this.
  In case there is left atrial fib > no S4 is heard bc there is no atrial contraction.

Diagnosis:

- ECG: (helpful but not diagnostic)
  - Left ventricular hypertrophy (deep S-waves in V1 and V2, tall R-waves in V5 and V6) & strain pattern
  - left atrial enlargement (Bifid & wide p wave)
  - Absent LVH, doesn't rule out aortic stenosis
- Echocardiography (Echo 2D/color doppler) > test of choice.
- CXR
  - Typically demonstrates a small heart; cardiomegaly occurs if heart failure develops.
  - Dilated ascending aorta.
- Cardiac Cath > helpful, confirmatory. Needed if the pt is older (look at the coronaries)

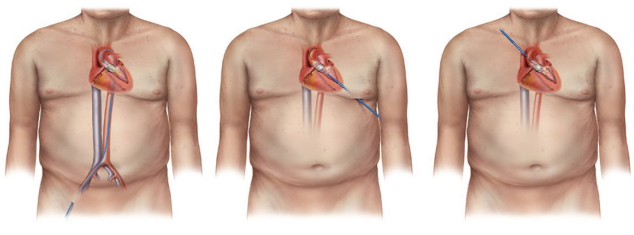
Treatment:

- Symptoms are a good index of severity:
- Treatment of symptomatic aortic stenosis or ↓ LV function:

1. Medical therapy > treat the symptoms not the cause.
   Anticoagulants are only required in patients who have atrial fibrillation or those who have had a valve replacement with a mechanical prosthesis.
2. Aortic valve replacement (the only truly effective therapy for AS)
   - Bioprosthetic vs Mechanical AVR.

- Asymptomatic: under regular review for assessment of symptoms and echocardiography.

Surgical intervention for asymptomatic people with severe aortic stenosis is recommended in those with:
• symptoms during an exercise test or with a drop in blood pressure
• an LVEF of <50%
• moderate to severe stenosis undergoing CABG, surgery of the ascending aorta or other cardiac valve.



Aortic Regurgitation

A three minute animation explaining both aortic stenosis and regurgitation with a sound clip of the murmurs

Aortic insufficiency

Etiology:

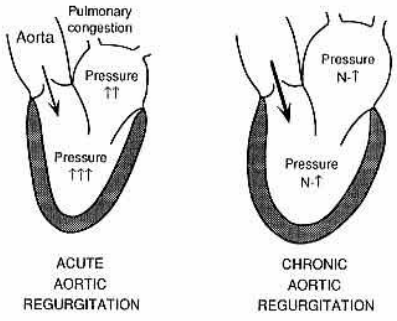
* AR results from either problems with aortic valve or aortic root

	Valvular abnormality	Aortic root abnormality
Chronic	Rheumatic, bicuspid.	HTN, Marfan, Aortitis
Acute	Endocarditis	Dissection

Acute	Chronic
• Acute rheumatic fever • Infective endocarditis • Dissection of the aorta • Ruptured sinus of Valsalva aneurysm • Failure of prosthetic heart valve Acute AR is very bad news! Pt will have acute pulmonary edema immediately!!	• Rheumatic heart disease • Syphilis Arthritides: - Reiter's syndrome, Ankylosing spondylitis, Rheumatoid arthritis. • Hypertension (severe) • Bicuspid aortic valve • Aortic endocarditis • Marfan's syndrome • Osteogenesis imperfecta

Pathophysiology:

- Widened pulse pressure ↑ systolic BP and ↓Diastolic BP (collapsing pulse) Seen in hyperdynamic circulation (pregnancy, anemia, infection, thyrotoxicosis)
  - Stroke volume increased > (high Systolic BP)
  - Regurgitant volume increased > (Low Diastolic BP)
- Imbalance between myocardial supply and demand:
- ↓ Diastolic BP > ↓ perfusion pressure > ↓ supply.
- ↑ LV size (thus ↑ wall stress) > ↑ demand > Those pts may get angina



1. Aortic regurgitation results in a volume overload of the left ventricle.
2. The ventricle compensates by increasing its end-diastolic volume according to the Frank-Starling mechanism.
3. The left ventricular dilation is thought to overstretch the myofibrils, leading to less actin–myosin interaction and decreased contractility.
4. In acute severe aortic regurgitation, the left ventricle has not had the opportunity to dilate, its compliance is relatively high, and the aortic regurgitation therefore leads to very high left ventricular end-diastolic pressure.

If mitral regurgitation ensues, the elevated left ventricular diastolic pressure is reflected back to the pulmonary vasculature, and acute pulmonary edema may occur.

Aortic Regurgitation

Acute AR

Volume suddenly increase, causing increase in pressure > reflecting on the left atrium > reflecting on the lungs > pulmonary edema

- A medical emergency

- The compensatory changes. seen in chronic disease do not have time to develop
- Presents with pulmonary oedema & cardiogenic shock.

- 2 Main consequences:
  1- Reduced coronary flow - the coronaries fill predominantly during diastole, regurgitant flow at this time reduces filling. Results in angina or in severe cases myocardial ischaemia.
  2- Increased EDP- causes increased pulmonary pressures with resulting pulmonary oedema and dyspnoea. In severe cases, cardiogenic shock may occur.
- Causes:

1. Acute rheumatic fever
2. Infective endocarditis
3. Dissection of the aorta
4. Ruptured sinus of Valsalva aneurysm
5. Failure of prosthetic heart valve

Symptoms	Signs
Dyspnea	Cardiogenic shock & Heart failure: Hypotension, tachycardia, elevated JVP…etc
Chest pain	Peripheral signs of chronic AI are usually absent
Symptoms of low cardiac output & HF	S3+ Murmur is early, short, faint and may be absent

Chronic AR

- Patients may remain asymptomatic for many decades.

- Develops slowly with compensatory changes:
  - Increase in the left ventricular end-diastolic volume (essentially the preload).
  - Increased stroke volume compensating for regurgitant flow supported by the ventricular hypertrophy to maintain ejection fraction, with a greater preload leading to greater contractility (frank- starling law)
  - Eventually further increases in preload cannot be met by greater contractility and heart failure develops.
- Causes:

1. Rheumatic heart disease
2. Syphilis Arthritides: Reiter's syndrome, Ankylosing spondylitis and Rheumatoid arthritis
3. Hypertension (severe)
4. Bicuspid aortic valve
5. aortic endocarditis
6. marfan syndrome
7. osteogenesis imperfecta

Symptoms	Signs
Dyspnea	**Pulse:** Pulsus Bisferience/ collapsing/ water hammer
Chest pain	**BP:** Elevated with wide pulse pressure
	Apex: Diffuse & Displaced
	HS: Soft S1 & S2
	Murmurs: Decrescendo early Diastolic & Austin flint murmur
	Pulsus bisferiens, also known as biphasic pulse, is an aortic waveform with two peaks per cardiac cycle, a small one followed by a strong and broad one.
	Diffuse hyperdynamic LV.

Aortic Regurgitation

Symptoms:

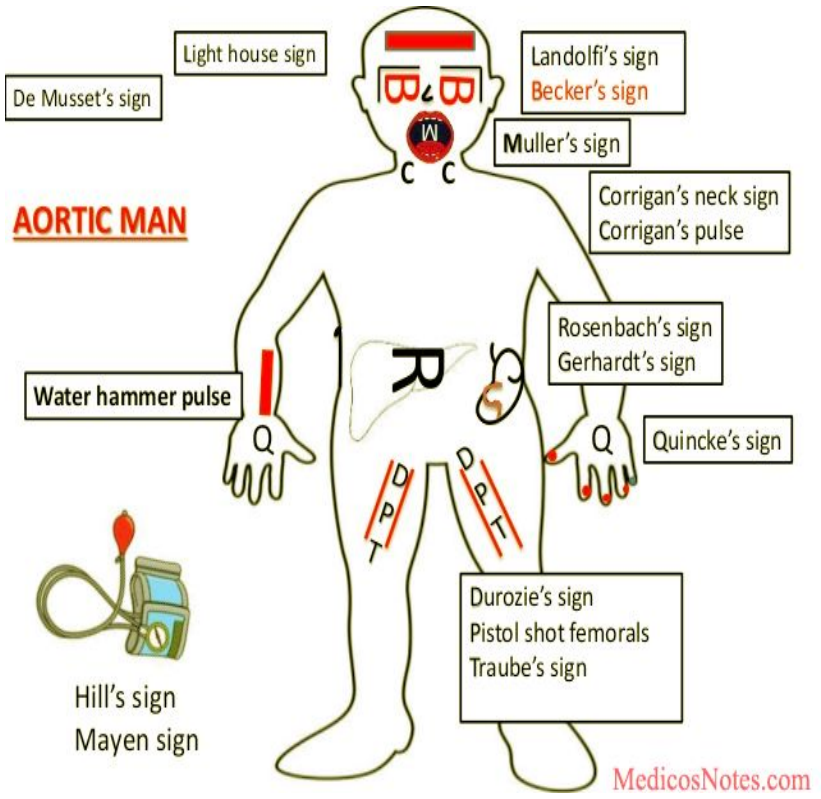
- Pulmonary venous congestion
  - Dyspnea on exertion (most common complaint)
- Inadequate cardiac output
  - Fatigue
  - Diminished exercise tolerance & angina pectoris

Signs:

They might ask you: "there was an early diastolic murmur and then a mid diastolic rumble"they are giving you a hint that this is AR.

- Diastolic decrescendo blowing murmur is the most typical. (main murmur in AR , early diastolic)
- Bounding Pulses (widened pulse pressure) "water hammer" pulse, (collapsing pulse).
- Apical Rumble – "Austin Flint Murmur" (low-pitched diastolic rumble due to competing flow anterograde from the LA and retrograde from the aorta) (its sounds is like mitral stenosis) (mid diastolic rumble, caused by venturi effect)
- Peripheral signs of AR
  a. Quincke's - capillary pulsation of nail beds.
  b. De Musset's - systolic nodding of the head.
  c. Corrigan's sign – abrupt distension with prominent pulse then rapid collapse. (you see the carotid pulsation really quickly).
  d. Traube's (pistol shot femoral) – systolic & diastolic bruit in the femoral artery
  e. Duroziez's - systolic bruit in the FA with proximal compression and diastolic sound with distal compression using the stethoscope.
  f. Müller's - systolic pulsation of uvula.
  g. Hill's sign – SBP in legs > 20 mmHg higher than SBA in arms.

- Video of Quincke pulse
- Video of Corrigan sign
- Video of De Musset Sign



Aortic Regurgitation

Diagnosis:

Echo is the main thing.

♦CXR: LVH, dilated aorta

♦ECG: LVH, T-wave inversion

- Left ventricular hypertrophy (deep S-waves in V1 and V2, tall R-waves in V5 and V6).
- Left ventricular strain may be seen in severe disease.

♦Echocardiogram—perform serially in chronic, stable patients to assess need for **Surgery**:

a. Assess LV size and function

b. Look for dilated aortic root and reversal of blood flow in aorta

c. In acute aortic regurgitation, look for early closure of mitral valve

♦ Cardiac catheterization: To assess severity of aortic regurgitation and degree of LV Dysfunction

Treatment:

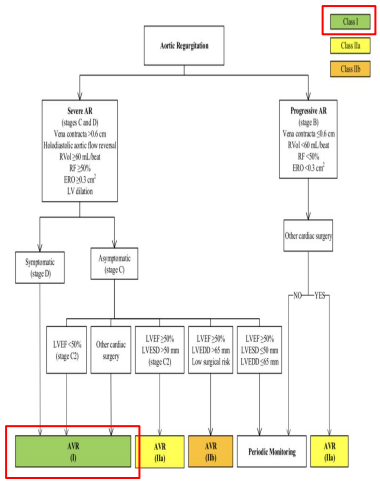
depends on symptoms and size of the heart

Asymptomatic AR	Symptomatic AR
• Medical Therapy, treats the symptoms not the cause. - Serial Check ups with Echos (eval EF, Severity AR) - SBE (Subacute bacterial endocarditis) Prophylaxis - Vasodilators (Nifedipine, ACE-I) - Diuretics	• Aortic Valve Replacement: - Bioprosthetic vs Mechanical AVR

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 haemodynamic,echocardiographic or angiographic criteria

Tissue valves are preferred in the elderly and when anticoagulants must be avoided, but are contraindicated in children and young adults because of the rapid calcification and degeneration of the valves.

Acute	Chronic
Emergency AVR	A. Medical treatment: • Treat HTN (most chronic AR patients are hypertensive). • ACE inhibitors/ARBs are reasonable in patients with severe symptomatic AR and/or LV dysfunction when surgery is not performed because of comorbidities.
	B. Surgical treatment: • AVR



Just know Class 1 is indicated for surgery

Aortic Regurgitation

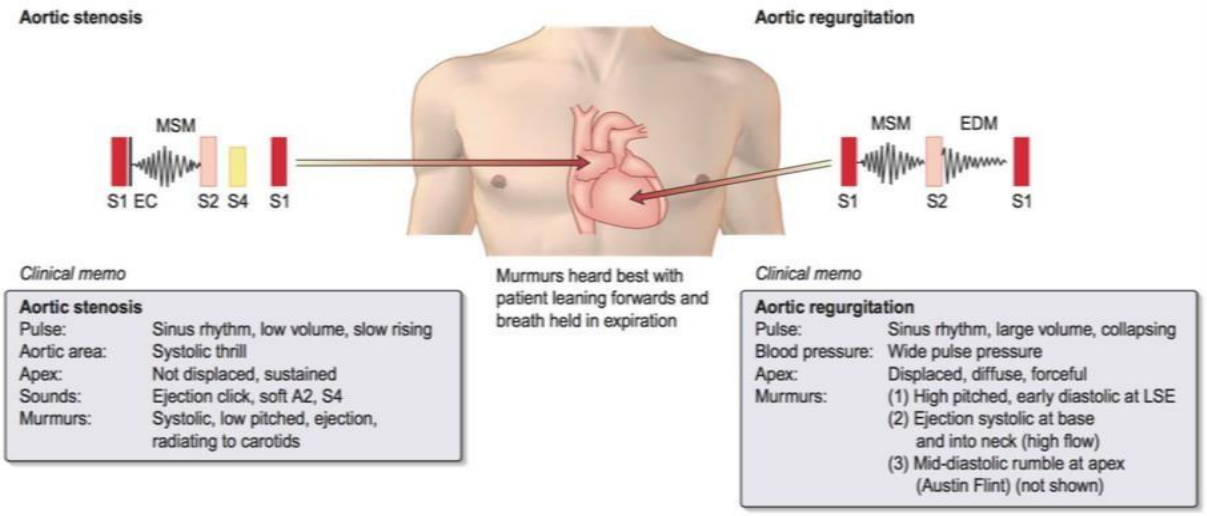


Figure 14.80 Features of aortic stenosis and aortic regurgitation. EC, ejection click; EDM, early diastolic murmur; MSM, mid-systolic murmur; S1, first heart sound. LSE, left sternal edge.

Right sided VHDs:

Tricuspid	Pulmonic
• Endocarditis: - IV drug abusers, or inpatient with IVs Because the needle is inserted into the vein and circulates to the right side of the heart first , and lead to tricuspid stenosis or regurg.	• Pediatrics: - Pulmonic stenosis very rare and comes with rheumatic. we see it in childhood and usually pure or combined with complex heart disease. They present with some fatigue and palpitations, and on echo we find they have pulmonary stenosis. Intervention (balloon dilatation) is done early in life to relieve stenosis
• Carcinoid heart disease : - Tricuspid stenosis (less common) due to rheumatic fever	• Rheumatic heart disease: - Pulmonary regurg. Very common due to the number of HF. it is well tolerated by the heart because it is not a high pressure volume area and it leads to some dilation, we do not need to do much just reduce the volume a little bit. - - Graham steell murmur When Pulmonic regurg is caused by RHD , most likely there are other valves that are affected.
• Tricuspid regurgitation: - Common benign, maybe secondary to pulmonary HTN - may be 1ry in patients who use IV drugs or on dialysis - With signs of heart failure eg. ascites Most of TR cases are 2ry to something else. It is usually a result of some Right side cardiac diseases, rarely seen as a major problem.	

- Right-sided valvular lesions change in intensity with inspiration

TABLE 2.1-18. Types of Valvular Heart Disease

Type	Etiology	History	Exam/Diagnosis	Treatment
Aortic stenosis	Most often seen in the elderly (senile calcific aortic stenosis) Unicuspid in childhood and adolescence. Rheumatic heart disease can predispose to AS	May be asymptomatic for years despite significant stenosis Once symptomatic, usually progresses from angina to syncope to CHF to death within 2 years Sx (also indications for valve replacements): **ACS**—**A**ngina, **C**HF, **S**yncope	PE: Pulsus parvus et tardus (weak, delayed carotid upstroke) and a single or paradoxically split S2 sound; systolic crescendo-decrescendo murmur at the right second intercostal space radiating to the carotids Severe AS characterized by soft and single S2 Dx: Echocardiography	Aortic valve replacement (surgical or transcatheter methods)
Aortic regurgitation	Acute: Infective endocarditis, aortic dissection, chest trauma, MI Chronic: Valve malformations, rheumatic fever, connective tissue disorders (ie, Marfan syndrome), syphilis, inflammatory disorders	Acute: Rapid onset of pulmonary congestion, cardiogenic shock, and severe dyspnea Chronic: Slowly progressive onset of dyspnea on exertion, orthopnea, and PND. Uncomfortable heart pounding when lying on left side	PE: Early blowing diastolic murmur at the left sternal border, mid-diastolic rumble (Austin Flint murmur), and midsystolic apical murmur Widened pulse pressure causes de Musset sign (head bob with heartbeat), Corrigan sign (water-hammer pulse; wide and bounding), and Duroziez sign (femoral bruit) Dx: Echocardiography	Vasodilator therapy (dihydropyridines or ACEIs) for isolated aortic regurgitation until symptoms become severe enough to warrant valve replacement. Digoxin and diuretics have little benefit Monitor LV function and size
Mitral valve stenosis	The most common etiology continues to be rheumatic fever Uncommon in the US	Sx: Include dyspnea, orthopnea, PND, and hemoptysis. Unique features secondary to LAE include AF, dysphagia, and hoarseness	PE: Opening snap and mid-diastolic murmur at the apex; pulmonary edema Dx: Echocardiography	Antiarrhythmics (β-blockers, digoxin, or CCBs) and warfarin for AF. Mitral balloon valvotomy and valve replacement are effective for severe cases
Mitral valve regurgitation	Primarily 2° to rheumatic fever or chordae tendineae rupture after MI Myxomatous degeneration due to mitral valve prolapse Infective endocarditis	Patients present with dyspnea, orthopnea, PND, and fatigue	PE: Holosystolic/pansystolic murmur radiating to the axilla Dx: Echocardiography will demonstrate regurgitant flow; angiography can assess the severity of disease	ACEIs or ARBs to vasodilate and ↓ rate of progression. Antiarrhythmics if necessary (AF is common with LAE). Digoxin and diuretics may be needed in CHF Valve repair or replacement for severe cases

Summary (Most Common)

	Definition	Etiology	Symptoms	Signs	Diagnosis	Treatment
Mitral Stenosis	Restriction & narrowing of the Mitral valve Common in pregnant women	Rheumatic Fever	- Dyspnea on exertion - AF	-Loud S1 - Loud P2 followed by an opening snap - S3 **cannot** be heard in mitral stenosis	-Test of choice: Echocardiography - Most accurate test: Cardiac Cath	Diuretics **Complications of MS:** Atrial fibrillation
Mitral Regurgitation	Backflow of blood from the left ventricle into the left atrium	- Rheumatic heart disease - Mitral valve prolapse - Cardiomy-opathies	Dyspnea	-Prominent **S3** Gallop - Pansystolic murmur: Best heard on the L side when lying on the side.	- ECG - Echocardiography	- When cardiac enlargement: surgical intervention - Medical therapy: Vasodilators, ACE inhibitors
Mitral Valve Prolapse	It is due to excessively large mitral valve leaflets	In chronic MVP: Myxomatous Heart Disease is the most common cause in middle aged asymptomatic men (marfan's syndrome)	Atypical chest pain	Mid-systolic click		B-Blockers
Aortic Stenosis	chronic progressive disease that produces obstruction to the left ventricular stroke volume	Calcification and degeneration of a normal valve more common in the elderly.	Angina	-Pulsus Parvus et Tardus :carotid pulse is of small volume & slow-rising or plateau in nature - thrill at the base of the heart -Harsh Systolic Ejection Murmur -Prominent S4 gallop	- ECG - Echocardiography	Aortic valve replacement
Aortic Regurgitation	is the diastolic flow of blood from the aorta into the left ventricle	If chronic: Rheumatic heart disease If acute: Acute rheumatic fever	Dyspnea	-Diastolic decrescendo blowing murmur -Apical Rumble "Austin Flint Murmur"	CXR ECG	-If Asymptomatic: Vasodilators (Nifedipine, ACE-I) Diuretics -If Symptomatic:Aortic Valve Replacement

Explanation

Extra definitions and how are they connected to Valvular heart disease

Marfan syndrome Amboss	An autosomal dominant connective tissue disorder that affects microfibrils and elastin in connective tissue throughout the body. Caused by fibrillin 1 deficiency due to FBN1 gene mutation. Can result in pathological manifestations in the cardiovascular system (e.g., mitral valve prolapse), the musculoskeletal system (e.g., joint hypermobility), and the eyes (e.g., lens subluxation).
Myxomatous Amboss	A pathologic increase in the deposition of glycosaminoglycans in tissue. Myxomatous degeneration of the mitral valve can weaken the valve and result in mitral valve prolapse.
Infective Endocarditis	Infective endocarditis is an endovascular infection of cardiovascular structures, including cardiac valves, atrial and ventricular endocardium, large intrathoracic vessels and intracardiac foreign bodies, such as prosthetic valves, pacemaker leads and surgical conduits.
Shone syndrome Wikipedia	Shone's syndrome is a rare congenital heart disease. In the complete form, four left-sided defects are present: -Supravalvular mitral membrane (SVMM) -Parachute mitral valve -Subaortic stenosis (membranous or muscular) -Coarctation of the aorta
Dissection of the aorta Amboss	A tear in the intima of the aortic wall that leads to blood entering between the intima and media or adventitia at high pressure and separating these layers, which creates a false lumen. Propagation of the tear can lead to obstruction of aortic branches and subsequent ischemia (e.g., neurological deficits, renal infarction), aortic regurgitation, and/or cardiac tamponade.
Syphilis Arthritides:	In the late stages of the disease, there may be syphilitic involvement of the heart, confined almost purely to the aorta and aortic valve. A particularly severe form of aortic insufficiency may develop, with subsequent dilation and enlargement of the heart and, eventually, heart failure.
Ankylosing spondylitis	Ankylosing spondylitis, is a form of arthritis that primarily affects the spine, although other joints can become involved. It causes inflammation of the spinal joints (vertebrae) that can lead to severe, chronic pain and discomfort. Also cause inflammation, pain, and stiffness in other areas of the body such as the shoulders, hips, ribs, heels, and small joints of the hands and feet. Sometimes the eyes can become involved (known as iritis or uveitis), and -- rarely -- the lungs and heart can be affected.
Reiter's syndrome	**Reactive arthritis, also called Reiter's syndrome, is the most common type of inflammatory polyarthritis in young men.**
Rheumatoid arthritis.	In people with rheumatoid arthritis, the immune system attacks the synovium—the lining of the membranes around the joints. This causes the synovium to thicken, eventually damaging the cartilage and bone. But the process doesn't stop at the joints. The inflammation can damage systems throughout the body, including the skin, eyes, lungs, and heart. Inflammation narrows the arteries, raising blood pressure and reducing blood flow to the heart, for instance.
Osteogenesis imperfecta Amboss	A genetic disorder characterized by defective synthesis of type 1 collagen, which is important in bone formation. Patients present with signs that are sometimes mistaken for child abuse (e.g., easy bruising, predisposition to bony fractures). Additional clinical features include blue sclerae, joint laxity, hearing loss, and brittle, opalescent teeth.
Graham steell murmur Wikipedia	A Graham Steell murmur is a heart murmur typically associated with pulmonary regurgitation.It is a high pitched early diastolic murmur heard best at the left sternal edge in the second intercostal space with the patient in full inspiration
Mitral facies	Severe mitral stenosis with pulmonary hypertension is associated with the so-called mitral facies or malar flush. This is a bilateral, cyanotic or dusky pink discoloration over the upper cheeks, which is due to arteriovenous anastomoses and vascular stasis.

Questions

1. A 40-year-old female presented with dyspnea on exertion, fatigue and palpitations. Auscultation revealed an ejection diastolic murmur in the mitral valve area. What is the best investigation to confirm our diagnosis?

A. Echocardiography
B. ECG
C. Chest x-ray
D. CBC

2. A 70-year-old male presented with chest pain and syncope. On examination his pulse was 60 bpm and his blood pressure 100/70 mmHg. On auscultation there was a harsh systolic ejection murmur in the aortic valve area with prominent S4. What is the most likely underlying pathology that is causing his symptoms?

A. Rheumatic valvular disease
B. Congenital anomaly
C. Calcification and degeneration of the valve
D. Endocarditis

3. Which valvular disease is associated with S3 gallop and radiates to the axilla?

A. Mitral stenosis
B. Mitral regurgitation
C. Aortic stenosis
D. Aortic regurgitation

4. A known case of rheumatic heart disease developed mitral stenosis. Which of the following is a known complication?

A. Ventricular fibrillation
B. Atrial fibrillation
C. Sick sinus syndrome
D. AV block

5. What is the most effective treatment for aortic stenosis?

A. Diuretics Beta blockers
B. Percutaneous
C. Ballon Valvulaoplasty
D. Valve replacement

6. Narrow pulse pressure is a sign of which valvular disease?

A. Mitral stenosis
B. Mitral regurgitation
C. Aortic stenosis
D. Aortic regurgitation

Questions

7-A patient is admitted with pneumonia. A murmur is heard on examination. What finding points to mitral regurgitation?

A. Murmur louder on inspiration
B. Murmur louder with patient in left lateral position
C. Murmur louder over the right 2nd intercostal space midclavicular line
D. Corrigan's sign
E. Narrow pulse pressure

8-You see a 57-year-old woman who presents with worsening shortness of breath coupled with decreased exercise tolerance. She had rheumatic fever in her adolescence and suffers from essential hypertension. On examination she has signs which point to a diagnosis of mitral stenosis. Which of the following is not a clinical sign associated with mitral stenosis?

A. Malar flush
B. Atrial fibrillation
C. Pan-systolic murmur which radiates to axilla
D. Tapping, undisplaced apex beat
E. Right ventricular heave

9- A 76-year-old male is brought to accident and emergency after collapsing at home. He has recovered within minutes and is fully alert and orientated. He says this is the first such episode that he has experienced, but describes some increasing shortness of breath in the previous six months and brief periods of central chest pain, often at the same time. On examination, blood pressure is 115/88mmHg and there are a few rales at both bases. On ECG there are borderline criteria for left ventricular hypertrophy. Which of the following might you expect to find on auscultation?

A. Mid-diastolic murmur best heard at the apex
B. Crescendo systolic murmur best heard at the right sternal edge
C. Diastolic murmur best heard at the left sternal edge
D. Pan-systolic murmur best heard at the apex
E. Pan-systolic murmur best heard at the left sternal edge

10- A 32-year-old woman attends her GP for a routine medical examination and is noted to have a mid-diastolic murmur with an opening snap. Her blood pressure is 118/71mmHg and the pulse is regular at 66 beats per minute. She is entirely asymptomatic and chest x-ray and ECG is normal. What would be the most appropriate investigation at this point?

A. Echocardiography
B. Anti-streptolysin O titre
C. Cardiac catheterization
D. Thallium radionuclide scanning
E. Color Doppler scanning

Questions

11- A 49-year-old woman presents with increasing shortness of breath on exertion developing over the past three months. She has no chest pain or cough, and has noticed no ankle swelling. On examination, blood pressure is 158/61mmHg, pulse is regular at 88 beats per minute and there are crackles at both lung bases. There is a decrescendo diastolic murmur at the right sternal edge. What is the most likely diagnosis?

A. Aortic regurgitation
B. Aortic stenosis
C. Mitral regurgitation
D. Mitral stenosis
E. Tricuspid regurgitation

12- a 75-year-old patient presents to the ER after a syncopal episode. He is again alert and retrospectively describes occasional substernal chest pressure and shortness of breath on exertion. His BP is 110/80 and lungs have a few bibasilar rales. Which auscultatory finding would best explain?

A. A harsh systolic crescendo-decrescendo murmur heard best at the upper right sternal border
B. A diastolic decrescendo murmur heard at the mid left sternal border
C. A holosystolic murmur heard best at the apex
D. A mid-systolic click
E. A pericardial rub

13-a 68-year-old man was intubated in the ER because of pulmonary edema. ECG reveals an ejection fraction of 45% and severe mitral regurgitation. In Spite of aggressive diuresis with furosemide, the patient continues to require mechanical ventilation secondary to pulmonary edema. What is the next best step in treating this patient?

A. Arrange for mitral valve replacement surgery
B. Begin IV milrinone
C. Begin metoprolol
D. Begin a second loop diuretic
E. Begin IV enalapril

14-a tall, thin 19-year-old woman with little previous health care complains primarily of decreased vision. You note a strong pulse, BP of 180/70 and a high-pitched, blowing diastolic decrescendo murmur. What is the associated valvular heart disease?

A. Aortic stenosis
B. Aortic regurgitation
C. Mitral regurgitation
D. Mitral stenosis
E. Tricuspid regurgitation

Questions

15- a 23-year-old graduate student complains of extreme fatigue and a vague sense of feeling ill the past few weeks. He has been under much stress recently and slightly agitated. On examination BP is 110/70 and his temperature is 38c. the neck veins are distended with prominent v waves. A holosystolic murmur is heard at the left sternal border, the murmur intensifies on inspiration.

What is the associated valvular heart disease?

A. Aortic stenosis
B. Aortic regurgitation
C. Mitral regurgitation
D. Mitral stenosis
E. Tricuspid regurgitation

Answers:

1. A 2. C 3. B 4. B 5. D 6. C 7. B 8. C 9. B 10. A
11. A 12. A 13. E 14. B 15. E