Valvular Heart Disease

Objectives:

- Etiology of valve diseases
- Pathogenesis
- Clinical presentation
- Clinical findings
- Investigation
- Management

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Resources: 435 team + Davidson + kumar + Recall questions step up to medicine.

- Editing file
- Feedback

Overview

Watch Video!

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.)

★ 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.

★ 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 .

★ **TYPES of Presentations** **Valvular heart disease can either be:**

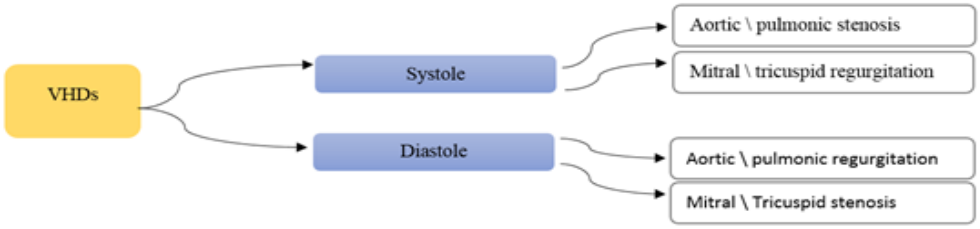
1- **Acute:**

acute mitral regurgitation is usually due to 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..

2- **Chronic:**

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



extra pics

★ 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)

★ Symptoms:

- Dyspnea Commonest (present for all valves), paroxysmal nocturnal dyspnea
- orthopnea .
- Palpitation .
- Chest pain. is typical of Aortic stenosis
- Dizziness , prefainting ,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)**
- **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 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 .

Mitral Stenosis

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:

1. **Rheumatic Fever;** (>90% cases):

related to streptococcus infections, causing damage to the mitral valve and leading to mitral stenosis later in life. (the most common)

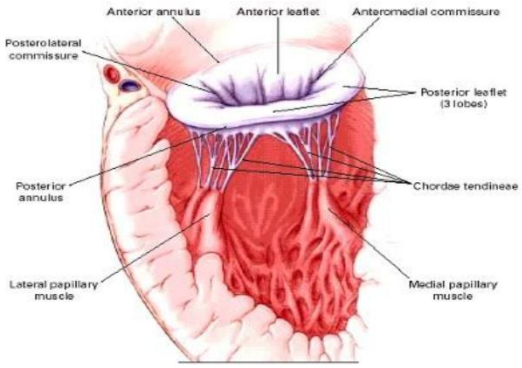
50% patients will have known history of RF, Average 20 years prior to clinical symptoms.

2. **Other less common causes:**

Congenital Mitral Stenosis (Shone syndrome) , Systemic Lupus Erythematosus, extensive calcification, Endocarditis – scarring & fusion of valve. Rheumatoid Arthritis, Atrial Myxoma (tumor), Malignant Carcinoid,, valvular dilation.

★ Mitral valve apparatus:

- Mitral valve leaflets (AML and PML)
- Mitral valve annulus
- Chordae tendinaea
- Papillary muscles
- Left ventricular myocardium



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

★ mitral stenosis results in several changes to the integrity of the valves :

- cusps thicken
- commissures fused together
- chordae tendineae becomes thickened & shortened
- calcium deposits form

★ 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 up normal 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.

★ Symptoms:

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 > 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.
  - 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):

- 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)
- 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.
- 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)

★ Diagnosis:

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)

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): shows pulmonary congestion and left 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, 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
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).

Mitral Regurgitation

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
   - Left Ventricular enlargement
   - Cardiomyopathies (dilated, hypertrophic)
   - 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.

★ Symptoms:

- Dyspnoea (pulmonary venous congestion)
- Fatigue (low cardiac output)
- Palpitation (atrial fibrillation, increased stroke volume)
- Oedema, ascites (right heart failure)

★ Signs:

- Laterally displaced (forceful) diffuse apex beat and a systolic thrill. Due to large heart
- Soft first heart sound.
- Prominent **S3** Gallop (increased volume during diastole). Gallop= S3 + tachycardia
  Bc pressure in the LA is high which goes to LV and blood falls quickly making noise
- Split S2 (but is obscured by the murmur).
- Pansystolic murmur 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)

★ 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: diffuse apical impulse + there may be some pulmonary findings

★ 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 cardiac enlargement warrants early surgical intervention (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 replacementMust be performed before left ventricular function is too severely compromised

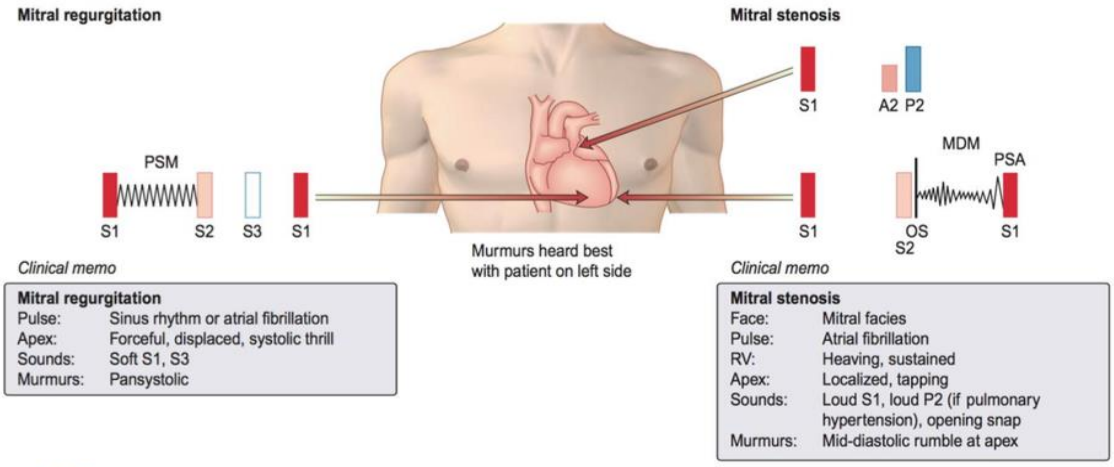


Figure 14.71 Features associated with mitral regurgitation and mitral stenosis. A2, aortic component of the second heart sound; MDM, mid-diastolic murmur; OS, opening snap; P2, pulmonary component of the second heart sound (loud with pulmonary hypertension); PSA, presystolic accentuation; PSM, pansystolic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

Mitral Valve Prolapse

★ Pathophysiology:

- 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.
- Most common in Women
- Myxomatous Heart Disease is the most common cause of chronic Mitral Regurgitation in middle aged asymptomatic men (marfan's syndrome)

★ Pathophysiology:

- Abnormal mitral apparatus > mitral leaflet prolapse > papillary muscle traction, activation of stretch receptors > papillary muscle & subendocardial ischemia > pain, 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.

★ 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.
-

★ 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

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. 30-50 yrs. (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.

- Differential diagnosis:
  supravalvular - murmur R carotid, ↑ A2
  subvalvular - often leads to AR Hypertrophic CardioMyopathy

★ 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.

★ 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.

★ 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 (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).
- 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 & left atrial enlargement (Bifid & wide p wave)
  - Absent LVH, doesn't rule out aortic stenosis
- Echocardiography (Echo 2D/color doppler) > test of choice.
- 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. Bioprosthetic given to pt 10-15 yrs, while Mechanical to >30 yr
- Asymptomatic: under regular review for assessment of symptoms and echocardiography.

Aortic Regurgitation

★ Etiology:

❖ 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

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

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

-

★ Pathophysiology:

- Widened pulse pressure (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.

★ Symptoms:

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

★ Signs:

- Diastolic decrescendo blowing murmur is the most typical.
- Hyperdynamic LV apical impulse (bc of ↑ SV)
- Bounding Pulses (widened pulse pressure) "water hammer" pulse, (collapsing pulse).
- S4, S3 Gallop-advanced AI > any pt has ↑ LV pressure > will have ↑ LA pressure > S4.
- 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)
- Quincke pulse (pulsations in the nail bed)
- Hill sign (BP in legs as much as 40 mm Hg above arm BP)
- Head bobbing (de Musset sign)
- Pistol shot which is femoral bruit (Duroziez sign)

★ Diagnosis:

1. CXR: LVH, dilated aorta
2. ECG: LVH, T-wave inversion
3. 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

4. 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 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

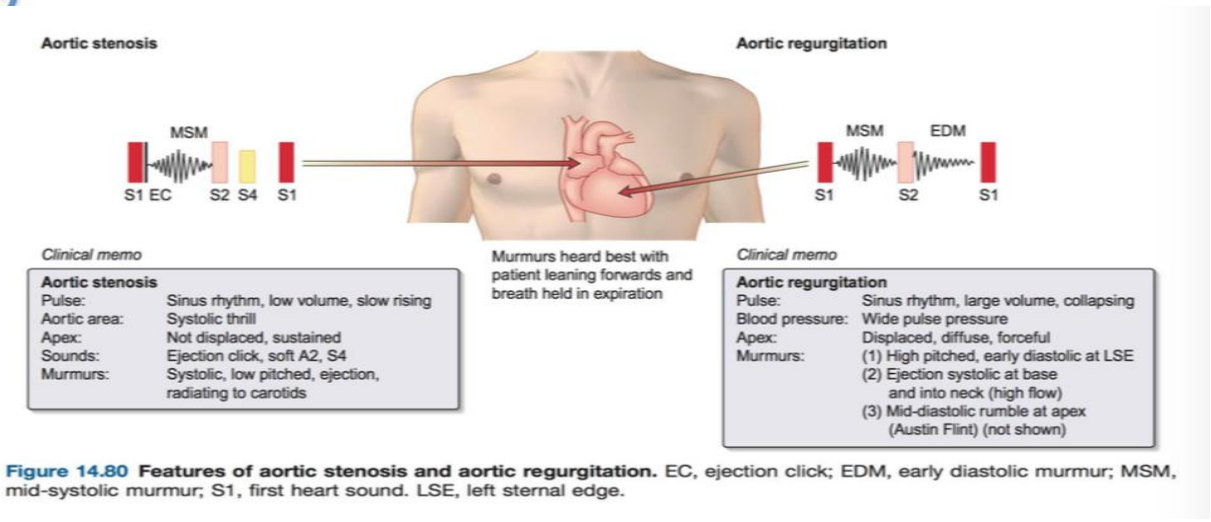


Figure 14.80 Features of aortic stenosis and aortic regurgitation. EC, ejection click; EDM, early diastolic murmur; MSM, mid-systolic murmur; S1, first heart sound. LSE, left sternal edge.

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

Summary

✯ Remember:

Mitral stenosis	- Almost all cases are due to rheumatic heart disease. - Murmurs: 1. Opening snap followed by low- pitched diastolic rumble. 2. Loud S1. - Causes pressure overload
Mitral regurgitation	- Causes volume overload. - Murmurs: 1. Holo\Pan- systolic murmur 2. S3 gallop. ● Mitral prolapse: - Large mitral valve leaflets. - Midsystolic or late systolic click. Mid-to-late systolic murmur
Aortic stenosis	- MCC: Calcification and degeneration of a normal valve in elderly. - Murmur: 1. Harsh crescendo– decrescendo systolic murmur. 2. S4 gallop. - Causes pressure overload
Aortic regurgitation	- Widened pulse pressure(markedly increased systolic BP, with decreased diastolic BP). - Diastolic decrescendo murmur. - Apical Rumble – "Austin Flint Murmur" - Causes volume overload

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
B. Beta blockers
C. Percutaneous 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

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

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 left 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. Inspite 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

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