Lecture 4: VHD (Valvular Heart Disease)

Intro Section

- "Extra General Info" These are key foundational points about murmurs and types of cardiac overload.
- 1. "Volume problem (eccentric hypertrophy)" → causes Systolic Heart Failure- s3 sound regurge
- Volume overload leads to eccentric hypertrophy, where the ventricle dilates (walls become thin and stretched).
- Because the heart can't eject the extra volume, this causes systolic failure (\downarrow EF, weak pump).
- 2. "Pressure problem (concentric hypertrophy)" → causes Diastolic Heart Failure- s4 sound stenosis
- In pressure overload, the ventricle thickens (concentric hypertrophy), which makes it stiff and unable to relax.
- This causes diastolic failure: preserved EF but impaired filling.
- 3. "Systolic murmurs can be physiological but diastolic murmurs are always pathological"
- 🛛 💡 Key clinical pearl:
- Systolic murmurs may be innocent (e.g., in athletes, pregnancy).
- Diastolic murmurs are **always abnormal** and suggest structural valve disease.

Mitral Regurgitation (MR)

"Regurgitating diseases" -> MR (Mitral valve incompetence)

- 1. Acute vs. Chronic MR
- Acute: more severe, often due to sudden valve rupture or ischemia, and may not have murmurs.
- Chronic: allows time for LA adaptation, often has murmurs. Cloud)

Primary MR:

- "The problem is in the valve itself"
- Due to:
- 1. *Mitral Valve Prolapse* \rightarrow most common cause.
- 2. *Rheumatic Fever* \rightarrow damages chordae/papillary muscles.
- 3. **A Extra note**: caused by Streptococcus pharyngitis, Group A.

Secondary MR:

- Valve is structurally normal, but dilated ventricles (from heart failure or ischemia) pull valve leaflets apart.
- 1. Dilated ventricles (Functional MR)
- 2. Ischemic post-MI MR papillary muscle dysfunction.

Hemodynamics of MR

Two diagrams are shown for chronic vs acute MR:

- On the left: Chronic MR
- On the right: Acute MR
- Chronic MR:
- LA volume is large, so pressure doesn't rise quickly \rightarrow murmur is long and loud.
- Quote: "Pressure gradient is high → Loud murmur"
- Acute MR:
 - LA has no time to adapt, so pressure rises fast → absent or soft murmur
- Quote: the body is surprised by the amount of blood so pressure rises quickly, so the gradient difference is small which means absent or soft murmur

Clinical Features & Physical Signs

Clinical Features

- ◆ "Symptoms → mainly due to pulmonary congestion (dyspnea, orthopnea, PND, fatigue)"
- These symptoms reflect left-sided heart failure because of blood backing up into the lungs.
- Let's break it down:
- Dyspnea = shortness of breath (first on exertion, then at rest).
- Orthopnea = difficulty breathing when lying flat (needs multiple pillows).
- PND (Paroxysmal Nocturnal Dyspnea) = sudden breathlessness at night that wakes the patient up.
- Fatigue = from reduced cardiac output.

 \ll All of this comes from elevated **left atrial pressure** \rightarrow leads to **pulmonary venous hypertension**.

Signs

- "Apical displaced apex"
- In chronic MR, the left ventricle enlarges \rightarrow this pushes the apex beat downward and laterally.
- Palpable on physical exam.

"S3 gallop"

- Caused by **volume overload** → rapid ventricular filling during early diastole.
- Very characteristic of MR or HF.





'Pansystolic murmur loudest at apex, radiates to axilla"

- "Pansystolic" = murmur is heard throughout systole, from S1 to S2.
- Location: Loudest at apex.
- **Radiation:** Spreads to **left axilla** \rightarrow classic clue for MR.

unclear S2 unclear TUP.

Aifib is commen

Let's Organize These Signs in a Quick Table

Feature	Description
Apical Displacement	Due to LV dilation from volume overload
S3 Gallop	Early diastolic sound, indicates increased volume
Pansystolic Murmur	Begins at S1, ends at S2; classic of MR
Murmur Radiation	Radiates to axilla due to direction of regurgitant jet

Investigations for MR

CXR (Chest X-ray)

"Shows pulmonary congestion, LA enlargement"

- **Pulmonary congestion** = hazy lung fields, Kerley B lines \rightarrow due to backflow of blood into lungs.
- LA enlargement = might appear as a straightening of the left heart border or double right heart border.

ECG

"May show LA enlargement ± AF"

- Chronic MR \rightarrow LA enlargement \rightarrow stretching of conduction fibers \rightarrow Atrial Fibrillation (AF)
- ECG signs of LA enlargement:
- Biphasic P wave in V1
- Wide, notched P waves in lead II ("P mitrale")

◆ ECHO (TTE → then TEE if unclear)

"ECHO confirms diagnosis and severity"

- TTE (Transthoracic Echo) = First-line, non-invasive.
- TEE (Transesophageal Echo) = If TTE is unclear or for surgical planning.
- It evaluates:
- Size and function of LV and LA
- Valve leaflet motion
- **Regurgitant volume**
- Pulmonary pressure

ECHO = gold standard for evaluating valve pathology and severity.

Management of MR

Now for treatment. This part is split into Medical and Surgical:

Medical Treatment (when mild/moderate or not surgical candidate):

"Control symptoms: diuretics, vasodilators, treat AF if present"

- **Diuretics** \rightarrow relieve pulmonary congestion.
- $\textbf{Vasodilators} \rightarrow \text{reduce afterload, reduce regurgitant volume (e.g. ACE inhibitors)}.$
- **Rate/rhythm control** if **AF is present** \rightarrow e.g., β -blockers, digoxin, anticoagulation if AF.

LABP — D Bridge for Surgery a

Surgical Treatment:

"Valve repair or replacement if symptomatic or LV EF \downarrow or LV dilated"

Indications for surgery:

- Symptomatic severe MR
- Asymptomatic with EF ≤60%
- Asymptomatic with LV dilation (LVESD ≥ 40 mm)
- New-onset AF or pulmonary hypertension

🧠 Valve repair is preferred over replacement if feasible — better survival, no need for lifelong anticoagulation.

(Note: Clip is prefered over surgery (for HF Dabients)

Summary Table: MR Investigation & Management

Investigation	Purpose
CXR	Pulmonary congestion, LA size
ECG	LA enlargement, AF
ECHO (TTE/TEE)	Confirm MR, assess severity
Treatment	Indications
Medical	Symptom control (diuretics, ACEI, AF mgmt)
Surgical Repair	Severe MR, symptoms, LV dysfunction/dilation







Pathophysiology

Let's decode this line by line.

"Blood leaks back from aorta \rightarrow LV during diastole"

- Normally, the aortic valve closes during diastole to prevent blood from returning to the LV.
- In AR, the valve is **incompetent**, so blood **leaks backward** \rightarrow from **aorta** \rightarrow **LV** \rightarrow during **diastole**.

"Causes volume overload → eccentric hypertrophy → systolic HF"

- The LV now receives:
- Blood from left atrium
- + Regurgitant blood from the aorta
- So total volume in LV = ↑↑
- Result: LV dilates \rightarrow eccentric hypertrophy $\rightarrow \downarrow$ ejection efficiency \rightarrow systolic heart failure
- Same as MR it's a volume overload lesion.

🔍 Etiologies of AR

Let's break your handwritten list into primary valve causes vs aortic root causes:

- 1. Valve pathology:
- Rheumatic fever
- Infective endocarditis
- Congenital bicuspid valve
- 2. Aortic root pathology:
- Marfan syndrome
- Aortic dissection
- Syphilitic aortitis (rare but classic)
- Aortic dissection causes sudden severe AR, especially type A.

Let's build a quick classification table:

Category	Examples
Valve causes	Rheumatic fever, endocarditis, bicuspid valve
Aortic root causes	Marfan syndrome, aortic dissection, syphilis

Hemodynamics

"Increased LVEDV, \uparrow SBP, \downarrow DBP \rightarrow widened pulse pressure"

Let's explain:

- LVEDV (LV end-diastolic volume) ↑ due to regurgitation
- Systolic BP (SBP) ↑ → strong contraction with high volume
- **Diastolic BP (DBP)** $\downarrow \rightarrow$ blood leaks back to LV \rightarrow pressure drops quickly
- Result = Widened pulse pressure
- Example: 160/50

% This gives **bounding pulses**, which you'll see in signs below.

Clinical Features & Physical Signs of Aortic Regurgitation (AR)

"Symptoms: mostly due to LV failure"

- AR causes volume overload → LV dilation → systolic failure
- Symptoms = same as left-sided heart failure:
- Fatigue
- Dyspnea on exertion
- Orthopnea
- Paroxysmal nocturnal dyspnea (PND)
- Palpitations (from large stroke volume)

"Signs" – this is the GOLD section

Let's go one by one:

🛛 1. Early Diastolic Murmur (EDM) (Decresendo murmur) (the Shorber the murmur = More Severe)

"High-pitched, blowing, best heard at LSB, leaning forward, end expiration" • "Early diastolic" = after A2 (aortic valve closure)

- "Blowing" = due to blood flowing backward into LV
- Best heard at: Left sternal border (3rd–4th ICS)
- Leaning forward, full expiration = brings aorta closer to chest wall → improves sound

This is the hallmark murmur of AR

2. Widened Pulse Pressure





- Example: 160/50 mmHg
- High SBP (↑ stroke volume) + Low DBP (aortic leak)
- Causes bounding pulses

3. Corrigan's Pulse

- "Water-hammer pulse"
- Rapid upstroke, then quick collapse
- Can be seen in radial or carotid pulse

🗹 4. Quincke's Sign

"Capillary pulsation in nails"

Blanching and flushing when light pressure applied to fingernail

5. Musset's Sign

"Head bobbing with each heartbeat"

• Seen in severe AR → transmitted bounding pulse to neck muscles

🗹 6. Duroziez's Sign

"Systolic & diastolic bruit over femoral artery when compressed"

- Place stethoscope over femoral artery while applying pressure
- You'll hear both systolic & diastolic flow sounds

Why all these weird signs?

They all come from one thing: a huge stroke volume + rapid diastolic runoff = hyperdynamic circulation

Summary Table: Classic AR Physical Findings

Sign	Description
EDM	High-pitched diastolic murmur at LSB
Widened PP	↑ SBP, ↓ DBP (e.g., 160/50)
Corrigan's pulse	Water-hammer pulse
Quincke's sign	Nailbed capillary pulsation
Musset's sign	Head bobbing
Duroziez's sign	Dual femoral artery murmur with compression

Q Investigations and Treatment of Aortic Regurgitation (AR)

🔬 Investigations

Chest X-ray (CXR)

"Shows LV enlargement ± aortic root dilation"

- In chronic AR, the left ventricle dilates due to volume overload.
- You may also see:
- Enlarged cardiac silhouette
- Prominent ascending aorta (if root is involved)

ECG

"Shows LVH"

- In chronic AR, the LV muscle thickens as it adapts.
- ECG signs of LVH include:
- Tall R waves in V5–V6
- Deep S waves in V1–V2
- LV strain pattern (ST-T changes)

Echo (TTE, TEE if needed) GS

"Confirm severity, LV size/function"

- **TTE** is the first-line test.
- It shows:
- Valve morphology
- Regurgitant jet (via Doppler)
- LV size and function
- Aortic root dimensions
- TEE is used:
- If TTE image quality is poor
- Or if endocarditis, aortic dissection, or pre-op assessment is needed

Nedical Treatment

"Control symptoms: Diuretics, vasodilators, ACEI"

Used in:

- Chronic mild/moderate AR
- Inoperable patients
- Bridge to surgery
- Diuretics: ↓ pulmonary congestion
- Vasodilators (e.g. ACEI, nifedipine): \downarrow afterload \rightarrow \downarrow regurgitation
 - **Beta blockers**: Avoid in AR they prolong diastole \rightarrow more regurg!

So if : 1- asymptomatic, no LV dysfunction, no systemic HTN = no tx

- 2- asymptomatic , severe AR and LV dysfunction = vasodilators to relieve afterload
- 3- if symptomatic, severe AR = surgery
- 4- indications for surgery = surgery

🛠 Surgical Treatment

"Valve replacement if symptomatic or LV EF <55%, or LVESD >50mm"

- Indications for surgery:
- Symptomatic severe AR
- Asymptomatic with ↓ LV EF (<55%)
- Asymptomatic with 个 LV end-systolic dimension (LVESD >50 mm)
- Significant aortic root dilation (>50 mm)

Surgical options:

- Aortic valve replacement (AVR)
- May be combined with root replacement (e.g. Bentall procedure) if needed

Summary Table – AR: Investigations & Management

Mitral Stenosis (MS)

As always, I'll explain every line of your handwritten notes clearly, with logic and extra tools to help you truly master it.

Pathophysiology

"Mitral valve narrowing \rightarrow obstructs LV filling $\rightarrow \uparrow$ LA pressure \rightarrow pulmonary HTN \rightarrow RHF"

Let's break this sequence down step by step:

- 1. Mitral valve becomes stenotic \rightarrow The opening between LA and LV is narrowed.
- 2. LV can't fill easily during diastole \rightarrow Blood backs up in the LA.
- 3. LA pressure increases \rightarrow Left atrium dilates and pressure pushes backward.
- 4. Pulmonary venous congestion \rightarrow Leads to pulmonary hypertension.
- 5. Chronic pressure in lungs \rightarrow right heart overload \rightarrow Eventually causes right heart failure (RHF)

The LV is usually normal size and function in MS — the problem is before it.

Causes

"Rheumatic fever is #1 (esp in developing countries)"

- Rheumatic fever → fibrosis + fusion of mitral valve leaflets
- This is the most common cause worldwide.
- Others are rare (congenital MS, radiation, etc.)

Hemodynamics of MS

" \uparrow LA pressure \rightarrow pulmonary HTN \rightarrow RVH, TR"

Let's chart this sequence:

Valve Event	Effect on Chambers
Mitral stenosis	↑ LA pressure
	→ Pulmonary congestion
	\rightarrow Pulmonary HTN
	→ Right ventricular hypertrophy (RVH)
	→ Functional tricuspid regurg (TR)

Bonus Concept:

- MS = pressure overload of LA → LA dilation
- If untreated → pulmonary vasculature remodeling → RV failure



Explanation:

- Mitral valve narrowing = less space for blood to flow from LA to LV.
- This slows filling of LV in diastole, so LA pressure increases (↑ LAP).
- The LA backs up into **pulmonary veins** → **pulmonary hypertension (PHTN)**.
- Chronic PHTN stresses the right side of the heart, eventually causing right heart failure (RHF).

UV is usually unaffected in MS — it's a pre-LV problem!

Your Note:

"Cause: RF >> fibrosis of mitral valve"

Explanation:

- **RF = Rheumatic fever**, the most common cause worldwide.
- It leads to thickening and fusion of leaflets, especially at the commissures.
- Over time → fixed, narrow valve opening

Your Note:

" \uparrow LAP \rightarrow \uparrow pulm v. pressure \rightarrow pulm HTN \rightarrow RVH + TR"

C Explanation:

- Blood backs up from LA \rightarrow pulmonary veins \rightarrow pulmonary arteries \rightarrow right ventricle.
- RV faces more pressure → hypertrophies (RVH).
- RV dilation may stretch the tricuspid value \rightarrow functional tricuspid regurgitation (TR).

Let's Summarize This Cascade in a Flow Table:

Pathological Step	Result
Mitral valve narrowing (MS)	\downarrow LV filling (diastolic flow obstruction)
↑ LA pressure	LA dilation
↑ Pulmonary venous pressure	Pulmonary congestion
↑ Pulmonary artery pressure	Pulmonary hypertension
↑ RV pressure load	RV hypertrophy → Tricuspid Regurgitation

Mitral Stenosis – Symptoms & Signs

Your Note:

"Symptoms due to pulmonary congestion + \downarrow CO"

Explanation:

- Blood can't flow easily from LA to LV \rightarrow LA pressure rises \rightarrow lungs get congested \rightarrow You get:
- Dyspnea (most common)
- Orthopnea
- PND
- ↓ LV filling → ↓ cardiac output (CO) → Leads to:
- Fatigue
- Cold extremities
- Exercise intolerance

Your Note:

"Signs → Malar flush, loud S1, opening snap, diastolic rumble" Let's go one by one and explain them like a master:

Malar Flush

- Reddish-purple cheeks
- Due to low cardiac output → vasodilation + CO₂ buildup
- Classic in young females with chronic MS

Loud S1

- Mitral valve slams shut forcefully (because it's stiff but still mobile)
- Earlier in disease when valve is still pliable
- As disease progresses → S1 becomes **soft**

Opening Snap (OS)

- High-frequency sound after S2
- Comes from forced opening of stiff mitral valve during diastole
- More severe MS = earlier OS

(+) hemopbysis.

-* it will cause HF even though HV function is preserved.

Mid-diastolic murmur (rumble)

- Low-pitched, best heard at apex, with bell of stethoscope
- Occurs when blood tries to flow from LA \rightarrow LV through narrow valve

\bigcirc Quick Table of Auscultation in MS:

Sound	What it means
Loud S1	Stiff valve slamming shut (early MS)
Opening Snap	Valve forced open (after S2)
Diastolic Rumble	Turbulent flow during diastole
Malar Flush	Vasodilation from low output (facial sign)

Mitral Stenosis – Investigations

Your Note:

"ECG \rightarrow LAE ± AF"

Explanation:

- Chronic MS causes left atrial enlargement (LAE):
- Because of constant pressure overload
- This stretched atrium may lead to:
- Atrial fibrillation (AF) very common in MS
- On ECG:
- LAE → "P mitrale" (broad, notched P waves in lead II)
- AF → no clear P waves, irregularly irregular rhythm

Your Note:

"CXR → LAE, pulm congestion, straight left heart border"

Explanation:

- LAE → causes visible bulge in left atrial appendage = straightening of left heart border
- Pulmonary congestion:
- Hazy vascular markings
- Kerley B lines
- Upper lobe diversion ("cephalization")
- Classic exam image = straight left border + pulmonary edema

Your Note:

"Echo \rightarrow confirm diagnosis and assess severity"

Explanation:

- Echo is the gold standard for diagnosing and grading MS.
- What we see:
- Mitral valve thickening, calcification, fused commissures
- Valve area calculation (normal = 4–6 cm²)
- Severe MS = <1.5 cm²
- Pressure gradient between LA and LV
- < Echo can also assess:
- LA size
- Pulmonary pressures
- RV function
- Presence of thrombus (via TEE)

🍤 Mitral Stenosis – Treatment

Your Note:

"Treat AF: rate control, anticoagulation"

Explanation:

- AF is common in MS → may cause palpitations, syncope, emboli
- Rate control:
- β-blockers or digoxin (esp if heart failure)
- Anticoagulation:
- Needed if AF present to prevent stroke

Always use warfarin (DOACs not reliable in MS with AF)

🌛 Your Note:

"Symptom control \rightarrow diuretics"

Explanation:

- Diuretics like furosemide help reduce:
- Pulmonary congestion
- Dyspnea
- They **do not treat the valve** just manage volume overload

Your Note:

"Definitive → balloon valvotomy or valve replacement"

Explanation:

- Percutaneous balloon mitral valvotomy (PBMV):
- Best for young patients, non-calcified valves
- Opens valve by stretching fused leaflets
- Valve replacement:
- If PBMV is contraindicated (calcified, thrombus, MR present)
- Requires lifelong anticoagulation (if mechanical valve)

Summary Table – MS Investigation & Management

* Surgery: 7 1) Severe symptoms at rest or exercise 2) if Mild Symptoms - D consider a balloon altheough it's not curative.

Tool	Finding
ECG	LAE, ± AF
CXR	LA enlargement, straight LHB, pulmonary edema
Echo	Valve area <1.5 cm ² , LA pressure gradient
Treatment	When to Use
Diuretics	Symptom relief only
Rate control + warfarin	If AF present
Balloon valvotomy (PBMV)	Young, non-calcified valves
Valve replacement	Severe, calcified, or thrombus

1. Aortic Stenosis (AS): General Overview

It has 3 locations: ISupravalvular ISubvalvular IValvular (Most Common)

Location	Description
Supravalvular	Above the valve (in the ascending aorta) 🛲
Subvalvular	Below the valve (in the outflow tract) $ ightarrow$
Valvular	At the level of the valve itself (most common) $\Im_{\!$

A Supravalvular Aortic Stenosis

Definition: Stenosis at the level of the aorta (not the valve!) alt is a single discrete narrowing with long tubular hyperplasia of the ascending aorta. Clinical Examination:

- On physical exam (PF):
- Thrills felt at:
- Suprasternal notch
- Or Right carotid artery 🇳
- Auscultation findings:
- Loud S2 🔉
- Systolic murmur best heard over right 2nd intercostal space (ICS)
- Special feature: ¹/₇
- Ejection click early in systole (♣) → due to sudden valve opening
- % Management:
- Surgical, and may need a conduit (prosthetic graft tube) if severe %

B Subvalvular Aortic Stenosis

Seen in ~10% of AS cases

- 📌 Etiology:
- A discrete ridge or tunnel-like stenosis below the aortic valve.
- Often associated with Aortic Regurgitation (AR) due to jet impact on valve.

🖊 Diagnosis:

- Echocardiography (ECHO):
- TTE (Transthoracic) 🂐 first-line



• If unclear, use TEE (Transesophageal) 📏

[™] Treatment:

- Surgical resection, especially if:
- Severe
- Or AR is progressing X

Valvular Aortic Stenosis (Most Common Type)

PEtiologies (Causes):

Туре	Notes
Degenerative	Due to aging (senile calcific AS) \overline{a}
2Rheumatic	Seen in 40–60% of cases 🍾
Calcified bicuspid	~40–60% – congenital anomaly 🖋
4 Congenital	<30%, either bicuspid or unicuspid 📴 😔

📕 Note: Always screen 1st degree relatives for bicuspid valve 離 You must scan the whole aorta if bicuspid valve is present!

Hemodynamics:

 \sim Wall tension = (Radius × Pressure) / (2 × wall thickness) \leq At early stages: LV hypertrophies \rightarrow normalizes tension $\frac{1}{2}$ Late stage: \downarrow LVEF (left ventricular ejection fraction)

A Clinical Features:

Feature	Description
1	Pulmonary edema (due to 个 LV pressure) 🍐
2	Shortness of breath (SOB) 😵
3	Angina due to ↑ oxygen demand to normalize LV pressure ♥ ⊇4
4	Syncope (especially during exertion) 😵

\bigcirc Auscultation Signs:

Sign	Meaning		
1Single or soft S2	Softer = more severe AS (valve can't close well)		
亞 rescendo-decrescendo murmur	Classic systolic murmur \swarrow radiating to carotid arteries		
£]s4 gallop	Stiff LV, late diastolic sound 🔈		
4 Pulsus parvus et tardus	Weak + delayed pulse 🖖 🕒		

Investigations:

- Echocardiography (ECHO): First-line for diagnosis
- CT Scan: To assess valve calcium load or anatomy for surgery

X Treatment:

Condition	Management	
Asymptomatic + normal LV	No treatment required 🗙	
Symptomatic	Surgical aortic valve replacement (SAVR) X or TAVR	
	(Transcatheter Aortic Valve Replacement) for elderly	
· · · · · · · · · · · · · · · · · · ·		

🔺 In young patients: use mechanical valves 🧇 In elderly: use bioprosthetic valve (TAVR)

Signs of Severe Aortic Stenosis:

Clinical Sign	Meaning		
flate systolic murmur	Indicates higher severity 🛑		
∑Single S2 sound	Suggests poor valve closure 🗳 soft sound 🌂		

Tricuspid Regurgitation (TR)

Definition:

TR = Backward leakage of blood from the right ventricle (RV) into the right atrium (RA) during systole.

Why? Because the tricuspid value doesn't close properly — so blood regurgitates (leaks) back when the RV contracts.

Etiologies (Causes)

Туре	Cause/Example		
Functional TR	Due to RV dilation, the valve annulus stretches.		
No structural defect in the valve leaflets themselves.			
Often seen with:			
Bulmonary hypertension			

Pulmonary hypertension

Left heart failure

- RV infarction
- COPD | | Primary (organic) TR | Structural problem in the valve leaflets. Examples:
- Rheumatic heart disease
- Endocarditis (especially in IV drug users)
- Ebstein anomaly (congenital)
- Carcinoid syndrome (serotonin damages right heart valves) |

Seven and the seven of the seve

- 1. **Annular dilation** \rightarrow valve can't close
- 2. Blood flows back into RA during systole
- 3. \rightarrow RA dilates
- 4. \rightarrow Leads to right-sided heart failure

Signs and Symptoms

System Affected	Clinical Features		
🏺 Cardiac	Fatigue, palpitations		
🛝 Pulmonary	May be clear or mild congestion (not dominant)		
> Systemic	JVD, hepatomegaly, peripheral edema, ascites		
9 Murmur	Holosystolic murmur best heard at left lower sternal border, with inspiration (Carvallo's sign)		

🖓 Carvallo's Sign = 🔈

- TR murmur gets louder with inspiration
- This helps distinguish it from mitral regurgitation, which does not increase with inspiration.

Investigations:

- Echocardiography: Main tool to evaluate valve structure and RV function
- ECG: May show signs of RA enlargement or RV strain
- CXR: Cardiomegaly, prominent RA/RV

🛠 Management:

Case	Management	
Mild/Moderate Functional TR	Treat the underlying cause (e.g., PH, LHF)	
Severe or Primary TR	Surgery if symptomatic or progressive	
Valve repair vs replacement	Depends on anatomy & severity	

MR	AR	AS	MS	TR	
Valve	Mitral	Aortic	Aortic	Mitral	Tricuspid
Phase affected	Systole	Diastole	Systole	Diastole	Systole
Mechanism	Blood leaks $LV \rightarrow LA$	Blood leaks Aorta \rightarrow LV	Obstruction $LV \rightarrow Aorta$	Obstruction $LA \rightarrow LV$	Blood leaks $RV \rightarrow RA$
Overload type	Volume	Volume	Pressure	Pressure	Volume
Hypertrophy	Eccentric	Eccentric	Concentric	Concentric	Eccentric (secondary)
Heart Failure	Systolic	Systolic	Diastolic	Diastolic	Right-sided
Most common cause	MVP, Rheumatic	Bicuspid, Endocarditis, Marfan	Degeneration (aging), Bicuspid	Rheumatic Fever	Pulm. HTN, RV dilation
Key symptoms	SOB, PND, palpitations	SOB, fatigue, palpitations	Angina, Syncope, Dyspnea	SOB, Hemoptysis, Palpitations	Fatigue, edema, hepatomegaly
Key signs	S3, AFib	Corrigan, Quincke, wide PP, S3	Parvus et Tardus, S4	Loud S1, Opening Snap	JVP v-wave, Carvallo's sign
Murmur type	Pansystolic	Early diastolic decrescendo	Crescendo-decrescendo	Mid-diastolic	Pansystolic
Murmur heard at	Apex \rightarrow axilla	LSB (best at 2nd-4th ICS)	2nd RICS \rightarrow carotids	Apex	LLSB (\uparrow with inspiration)
ECG findings	LAE, AFib	LVH	LVH	LAE, AFib	RA dilation
Echo role	Severity + repair plan	Severity + aortic root	Valve area + gradient	Valve area + gradient	Valve structure + severity
Best test (Dx)	TTE + Doppler	Echo	$Echo \pm CT$	Echo	Echo
Medical Tx	Vasodilators (if symptomatic)	Vasodilators (if \downarrow EF)	None if asymptomatic	Balloon if mild, anticoag if AF	Diuretics, treat underlying cause
Surgical	Severe + EF <60% or	Severe + EF $\leq 50\%$ or	Symptomatic or severe	Severe MS, symptoms	Severe symptoms, failed
indications	symptoms	symptoms			diuretics
Surgical options	Repair > Replacement	AVR	AVR or TAVR	Surgery or balloon	Repair / Replace if left-side op

Percutaneous	Mitral clip	TAVR (elderly/high-risk)	TAVR	Balloon commissurotomy	No device (remove lead if
option					cause)
EF cutoff for	<60%	<50%	Not fixed (symptoms-	Not EF-based	Not EF-based
surgery			based)		