FINAL MBBS · GENERAL MEDICINE · CARDIOLOGY CASES

A E T C M

Valvular Heart Disease Case Sheets

Mitral Stenosis · Mitral Regurgitation · Aortic Stenosis · Aortic Regurgitation

Four fully worked cardiac cases written the way a final-year MBBS student should record and present them in the university examination — with the reasoning behind every sign, the signs of severity, echo criteria, current management and the viva questions examiners ask.

Approach to a cardiac case › | Pulse, JVP & precordium › | Describing a murmur › | Case 1 · Mitral stenosis ›
MS — PTMC & anticoagulation › | Case 2 · Mitral regurgitation › | MR — surgery timing › | Case 3 · Aortic stenosis ›
AS — TAVI vs SAVR › | Case 4 · Aortic regurgitation › | Peripheral signs of AR › | Four valves compared ›
Multivalvular disease › | Rheumatic fever & prophylaxis › | Viva questions ›

TAP A TOPIC TO JUMP TO IT

MS loud S1 · OS · MDM › **MR** soft S1 · PSM · S3 › **AS** slow pulse · ESM · soft A2 › **AR** collapsing pulse · EDM

HEAR THE MURMURS · SEE THE SIGNS — TAP TO OPEN AUDIO / X-RAY / ECG

MS murmur ♪ | MR murmur ♪ | AS murmur ♪ | AR murmur ♪ | Malar flush ↗ | Collapsing pulse ↗
AR peripheral signs ↗ | MS chest X-ray ↗ | P mitrale ECG ↗ | LVH ECG ↗ | All links — atlas ›

AETCM
Academy for Emergency, Trauma & Critical care Medicine

By

Dr. Gireesh Kumar K.P.

WATCH THE VIDEOS · TAP TO PLAY ON YOUTUBE

▶ Cardiac examination 1 | ▶ Cardiac examination 2 | ▶ Mitral regurgitation | ▶ Mitral stenosis

For final-year MBBS students, interns and teachers of clinical medicine

Valvular Heart Disease Case Sheets

Mitral stenosis · Mitral regurgitation · Aortic stenosis · Aortic regurgitation

Dr. Gireesh Kumar K.P.

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How to use these case sheets

A valvular heart disease case is the commonest cardiovascular long or short case in the final MBBS examination — and in India most of them are **rheumatic**. The examiner judges three things: can you **elicit and describe the signs** (pulse, apex, heart sounds, murmur); can you say **how severe** the lesion is and whether there are **complications** (pulmonary hypertension, heart failure, AF, endocarditis); and can you **plan management** in the light of current guidelines.

The book starts with a common **approach to every cardiac case**, followed by four complete cases. Each case has the **case record** (what you write), **green boxes** (what you conclude), **blue boxes** (examiner's tips), a **60-second presentation**, differential diagnosis, investigations with echo severity criteria, and management. A comparison of all four lesions, multivalvular disease, rheumatic fever prophylaxis and **45 viva questions** complete the book.

Tap to hear and see

Buttons beside each finding open free online material: ▶ Listen murmur recordings, ▶ Photo physical signs, ▶ X-ray radiographs and ▶ ECG tracings. A one-page **audio & visual atlas** with QR codes is included for the printed copy.

Patients described are composite teaching cases. Severity criteria follow the ACC/AHA valvular heart disease guideline; rheumatic fever guidance follows Indian (ICMR) and WHO practice. Always examine your own patient and record only what you find.

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SECTION A · APPROACH

Approach to a Cardiac Valve Case

The common framework — history, general examination, CVS examination and the diagnosis format

1. History — what to ask in every valve case

Symptom / item	What to record	Why it matters
Breathlessness	Onset, progression, **NYHA class**; orthopnoea (number of pillows), PND	Pulmonary venous congestion — MS, MR, LV failure
Palpitations	Regular / irregular, sudden onset, duration	AF (MS, MR), ectopics
Chest pain	Exertional, relieved by rest	Angina — AS, AR, coexisting CAD; pulmonary hypertension
Syncope / dizziness	On exertion?	Severe AS (fixed output), arrhythmia
Cough, haemoptysis	Frothy pink sputum, frank blood	MS — bronchial vein rupture, pulmonary oedema, infarction
Right heart failure	Leg swelling, abdominal distension, right hypochondrial pain, oliguria	PAH with RV failure, TR
Embolism	Sudden weakness, speech loss, limb pain, loss of vision	LA thrombus in MS/AF; endocarditis
Infective endocarditis	Fever, weight loss, joint pain, recent dental work	Fever in a valve patient is IE until proven otherwise
Hoarseness, dysphagia	—	Ortner's syndrome / LA pressing on oesophagus (MS)
Past history	**Joint pains with fever in childhood, chorea, sore throats**; penicillin injections (how often, regular?); previous PTMC, surgery, prosthetic valve	Rheumatic aetiology; secondary prophylaxis
Women	Pregnancies — breathlessness worsening in pregnancy; contraception	MS decompensates in pregnancy
Drugs & family	Diuretics, digoxin, warfarin (INR monitoring), β-blockers; family history of sudden death, Marfan, bicuspid valve	Treatment adherence; congenital causes

NYHA class	Definition
I	No limitation — ordinary activity causes no symptoms
II	Slight limitation — comfortable at rest, ordinary activity causes symptoms
III	Marked limitation — less than ordinary activity causes symptoms
IV	Symptoms at rest; any activity increases discomfort

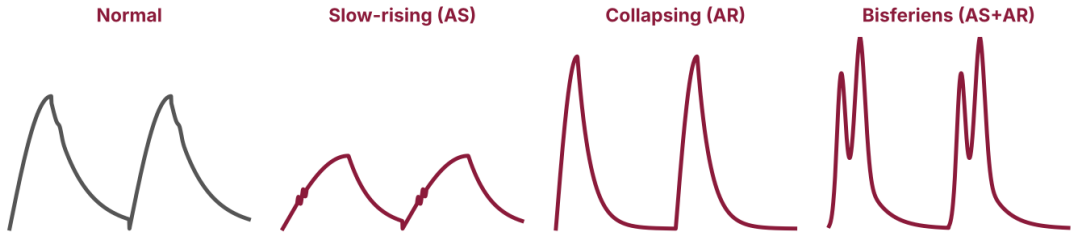
2. General examination — the cardiac checklist

- Build, **Marfanoid habitus**, high-arched palate, ankylosing spondylitis posture (AR)
- **Malar flush** (MS), cyanosis — central / peripheral
- **Clubbing**, splinter haemorrhages, Osler's nodes, Janeway lesions (IE)
- Pallor (IE, anaemia worsening failure), icterus (hepatic congestion)
- Pedal oedema, sacral oedema, ascites
- Rheumatic nodules, erythema marginatum (active carditis)
- Dental hygiene (IE source)
- Signs of thromboembolism — hemiparesis, cold limb

3. Pulse, blood pressure and JVP

Feature	What to look for
Rate	Count for a full minute in AF; **pulse deficit** (apex − radial >10 in AF)
Rhythm	Regular / regularly irregular / **irregularly irregular** (AF)
Volume	Low (AS, severe MS), high (AR, MR, high-output states)

Feature	What to look for
Character	Feel at the **carotid**: **slow-rising / anacrotic** (AS), **collapsing / water-hammer** (AR — raise the arm), **bisferiens** (AS + AR, HOCM), pulsus alternans (LV failure)
Radio-radial, radio-femoral	Delay — coarctation (with bicuspid AS/AR)
Vessel wall, all peripheral pulses	Thickening; absent pulses — embolism, Takayasu
Blood pressure	Both arms, lower limbs in AR; **pulse pressure** — narrow (AS), wide (AR); Hill's sign
JVP	Height (cm above sternal angle); waveform — absent a wave (AF), giant a wave (PAH, TS), **prominent v wave** (TR); hepatojugular reflux



Arterial pulse waveforms. Feel the character at the carotid, not the radial. ▶ Collapsing pulse ▶ AS pulse

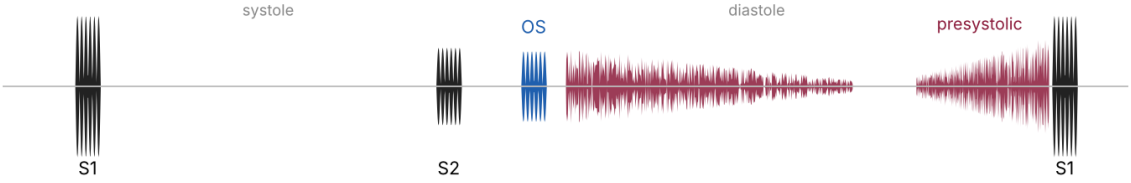
4. Precordium — inspection, palpation, percussion, auscultation

Step	What to record
Inspection	Precordial bulge (cardiomegaly since childhood — RHD), scars (sternotomy — valve replacement; left lateral thoracotomy — closed mitral valvotomy), visible apex, pulsations (left parasternal, epigastric, suprasternal), dilated veins
Apex beat	**Site** (intercostal space and relation to mid-clavicular line) and **character**: **tapping** (MS — palpable S1), **heaving / sustained** (pressure overload — AS, hypertension), **hyperdynamic / thrusting** (volume overload — MR, AR)
Parasternal heave	Grade I–III — RV hypertrophy (PAH)
Thrills	Diastolic thrill at apex (MS), systolic thrill at aortic area / carotids (AS), apex (MR)
Palpable sounds	Palpable S1 (MS), palpable P2 (PAH)
Percussion	Cardiac borders (right border, left border, 2nd left ICS dullness — pulmonary artery), liver span
Auscultation	All areas with diaphragm and bell; **S1, S2 (A2, P2, split)**, S3, S4, opening snap, ejection click; **murmurs**; manoeuvres; then the lung bases and back

5. Describing a murmur — the seven points

Point	Example
1. Timing	Systolic (ejection / pansystolic / late) or diastolic (early / mid / presystolic)
2. Site of maximum intensity	Mitral area, tricuspid, aortic, second aortic (Erb's — left 3rd ICS), pulmonary
3. Character / pitch	Low-pitched rumble (bell), high-pitched blowing (diaphragm), harsh
4. Grade	Systolic 1–6 (Levine): 4 = with thrill. Diastolic 1–4
5. Radiation	To axilla (MR), carotids (AS), left sternal border towards apex (AR)
6. Relation to respiration	Right-sided murmurs ↑ on inspiration (Carvallo); left-sided ↑ on expiration
7. Position / manoeuvre	Left lateral (MS), sitting forward in held expiration (AR), Valsalva / standing (HOCM, MVP ↑), squatting / handgrip

Mitral stenosis — loud S1, opening snap, mid-diastolic rumble with presystolic accentuation



Mitral regurgitation — soft S1, pansystolic murmur, S3



Aortic stenosis — ejection click, crescendo–decrescendo ejection systolic murmur, soft A2



Aortic regurgitation — early diastolic decrescendo murmur after A2



Phonocardiogram sketches of the four lesions. ▶ MS ▶ MR ▶ AS ▶ AR

Levine grade	Systolic murmur
1	Very faint, heard only with concentration
2	Faint but heard immediately
3	Moderately loud, no thrill
4	Loud, **with a thrill**
5	Heard with the stethoscope edge just touching the chest
6	Heard with the stethoscope off the chest

The diagnosis format for every valve case — six parts

(1) Anatomical / lesion — e.g. mitral stenosis, **(2) severity** — severe, **(3) aetiology** — rheumatic, **(4) complications** — PAH, AF, CCF, embolism, IE, **(5) functional class** — NYHA III, **(6) rhythm and current status** — in AF with controlled rate, in compensated / decompensated failure. Example: “Rheumatic heart disease with severe mitral stenosis, in atrial fibrillation, with severe pulmonary arterial hypertension, NYHA class III, not in failure, no evidence of infective endocarditis.”

CASE 1 · MITRAL STENOSIS

Rheumatic Mitral Stenosis in Atrial Fibrillation

The commonest valve long case in India

History

Particulars	Mrs. S. R., 28 years, female, homemaker, Malappuram district; informant — patient, reliable
Chief complaints	**Breathlessness on exertion — 3 years**, worse for 2 months; **palpitations — 6 months**; **cough with blood-streaked sputum — 2 episodes** in 2 months

HOPI. Breathlessness began 3 years ago on climbing stairs and has progressed; for 2 months she is breathless on walking inside the house (**NYHA III**). She sleeps on 2 pillows (**orthopnoea**) and has woken up breathless twice (**PND**). Breathlessness first appeared during her **second pregnancy** (2 years ago). Palpitations started 6 months ago — **irregular**, persistent. She has had 2 episodes of **haemoptysis** (blood-streaked frothy sputum) on exertion. No chest pain, syncope, fever, joint pain, leg swelling or abdominal distension. No episodes of sudden weakness, speech difficulty or limb pain (**no embolic events**). No hoarseness, dysphagia.

Past history. At 11 years she had **fever with pain and swelling of the knees and ankles that "moved from joint to joint"** and was treated for 3 weeks. She received monthly "penicillin injections" for 2 years, then stopped. Recurrent sore throats in childhood. No previous valvotomy. **Obstetric:** G2P2L2; both normal deliveries; breathlessness worsened in the third trimester of the second pregnancy. No contraception at present.

Personal / family / drugs. Mixed diet; lives in a crowded two-room house in childhood. Currently on furosemide 40 mg OD, metoprolol 25 mg BD (irregular); **not on anticoagulation**; no penicillin prophylaxis now.

> **Examiner's tip — five things in every MS history**
>
> (1) **NYHA class** and when symptoms began — pregnancy, AF onset and fever often unmask MS. (2) **Rheumatic fever** in childhood and **secondary prophylaxis**. (3) **Haemoptysis**. (4) **Embolic events**. (5) **Pregnancy and contraception** plans — these decide timing of intervention.

General examination and vitals

Finding	In this patient	Significance
Facies	**Malar flush** — bluish-pink cheeks ▶ Photos	Low cardiac output, peripheral cyanosis (MS facies)
Cyanosis	Peripheral (lips, tip of nose); no clubbing	Low output; clubbing → think IE or cyanotic heart disease
Pallor, IE signs	No pallor, splinters, Osler's nodes	—
Oedema	Absent	No right heart failure now
Pulse	**96/min, irregularly irregular**, varying volume, low volume overall; **pulse deficit 14** (apex 110) ▶ ECG	**Atrial fibrillation** with fast ventricular rate
BP	104/70 mmHg, both arms similar	Low-normal; narrow-ish pulse pressure (low output)
JVP	Raised 4 cm; **no a wave** (AF); normal v wave	Mild elevation; no significant TR
RR, SpO_2, temperature	20/min, 96 % on room air, afebrile	—

Cardiovascular examination

Step	Finding	Interpretation
Inspection	No precordial bulge; apex not clearly visible; left parasternal pulsation; no scars	RV hypertrophy
Apex	Left 5th ICS, **within** the mid-clavicular line, **tapping**	Normal-sized LV (MS protects the LV); tapping = palpable loud S1
Thrill	Diastolic thrill at the apex in left lateral position	Significant MS
Parasternal heave	Grade II	RV hypertrophy — **pulmonary hypertension**
Palpable P2	Present, left 2nd ICS	Pulmonary hypertension

Step	Finding	Interpretation
S1	**Loud** at apex	Mitral leaflets still mobile; held wide open until late (high LA pressure) and close with force
S2	Normally split; **loud P2**	Pulmonary hypertension
Opening snap	Sharp high-pitched sound after S2 at apex/left sternal border; **S2–OS interval short**	Pliable valve; short interval = high LA pressure = **severe** MS
Murmur	**Low-pitched, rumbling mid-diastolic murmur** at the apex, best with the **bell** in the **left lateral position** after exercise, grade 3/4, localised, **long**; **no presystolic accentuation** (AF) ▶ Listen	Mitral stenosis; long murmur = severe
Other murmurs	No pansystolic murmur (no significant MR); no early diastolic murmur (Graham Steell or AR)	Isolated MS
Lungs / abdomen	Fine basal crepitations bilateral; liver not palpable; no ascites	Pulmonary venous congestion

Signs of severe mitral stenosis

- **Short S2–OS interval** (<0.08 s)
- **Long murmur** (duration, not loudness, reflects severity)
- Signs of **pulmonary hypertension** — loud P2, RV heave, Graham Steell murmur, TR
- Symptoms at low exertion, haemoptysis, PND
- Low-volume pulse, malar flush
- **Soft S1 and absent OS** = calcified, immobile valve (severe, not suitable for balloon)

Examiner's tip — how to listen for MS

Left lateral position, **bell** lightly on the apex, after a few sit-ups to increase flow; time the murmur against the carotid. Most students miss MS by listening sitting up with the diaphragm.

60-second presentation

Present it like this

"Mrs S. R., a 28-year-old homemaker, with a history of migratory polyarthritis at 11 years and irregular penicillin prophylaxis, presents with progressive exertional breathlessness for 3 years, now NYHA class III, with orthopnoea, PND, irregular palpitations for 6 months and two episodes of haemoptysis. Symptoms began in her second pregnancy. On examination she has malar flush, an irregularly irregular pulse of 96/min with a pulse deficit of 14, BP 104/70 and JVP raised 4 cm without a waves. The apex is in the 5th space within the MCL and tapping, with a diastolic thrill, a grade II parasternal heave and palpable P2. S1 is loud, P2 is loud, there is an opening snap close to S2 and a long low-pitched rumbling mid-diastolic murmur at the apex in the left lateral position without presystolic accentuation. There are bilateral basal crepitations. **My diagnosis is rheumatic heart disease with isolated severe mitral stenosis, in atrial fibrillation with a fast ventricular rate, with pulmonary arterial hypertension and pulmonary venous congestion, NYHA class III, no evidence of infective endocarditis or thromboembolism; the valve is likely pliable.**"

Differential diagnosis of a mid-diastolic murmur at the apex

Condition	Distinguishing features
Mitral stenosis	Loud S1, opening snap, presystolic accentuation (in sinus rhythm), normal-sized LV, tapping apex
Austin Flint murmur	Severe AR — regurgitant jet impairs anterior mitral leaflet opening; **no** loud S1, **no** OS; peripheral signs of AR; displaced hyperdynamic apex
Carey Coombs murmur	Acute rheumatic carditis — valvulitis; soft, short; fever, other features of active RF
Flow murmur	Severe MR, VSD, PDA — increased flow across a normal mitral valve; short, follows S3
Left atrial myxoma	Varies with posture, "tumour plop", constitutional symptoms, embolism; echo diagnostic
Tricuspid stenosis	Left lower sternal border, increases with inspiration, giant a wave, often with MS

Investigations

Test	Findings expected / in this patient	Remarks
ECG	AF with fast ventricular rate; in sinus rhythm — **P mitrale** (broad bifid P >120 ms in II, biphasic P in V1 with deep negative terminal part); **right axis deviation, RVH** ▶ ECG ▶ ECG	LV voltage normal in pure MS
Chest X-ray (PA)	Straightening of the left heart border; **double atrial shadow**; **widened carina** (>90°); elevated left main bronchus; prominent pulmonary artery; upper-lobe diversion, Kerley B lines; normal-sized LV; mitral valve calcification ▶ X-ray	Lateral / barium swallow: posterior indentation by LA
Transthoracic echo	**Thickened leaflets, commissural fusion, "hockey-stick" anterior leaflet**, restricted posterior leaflet; **MVA 0.9 cm²** (planimetry), mean gradient 12 mmHg (at HR 96), PASP 58 mmHg; LA 52 mm; mild MR; normal LV	**Wilkins score** for balloon suitability (below)
TEE	Done before PTMC — **excludes LA / LA appendage thrombus**; grades MR	Mandatory before balloon valvotomy
Blood	Hb, RFT, electrolytes, TSH, INR; ASO if active RF; cultures if fever	—

MS severity (ACC/AHA)	Mitral valve area	Mean gradient*	PASP
Progressive (mild–moderate)	>1.5 cm²	<5 mmHg	<30
Severe	**≤1.5 cm²**	>5–10 mmHg	>50 mmHg
Very severe	**≤1.0 cm²**	—	—

*Gradient depends on heart rate and flow — valve area is the key criterion. **Wilkins score** (0–16): leaflet mobility, thickening, calcification and subvalvular thickening, each 1–4; **≤8** favours a good balloon result.

Management of mitral stenosis

Area	Management
Secondary prophylaxis of rheumatic fever	**Benzathine penicillin G 1.2 million units deep IM every 3 weeks** (India — every 21 days). Duration: with established valve disease — at least **10 years after the last attack or until 40 years**, whichever is longer; severe disease / after intervention — often lifelong. Penicillin allergy: erythromycin 250 mg BD
Rate control in AF	**β-blocker** (metoprolol) or diltiazem — slows the rate and **prolongs diastolic filling time**, the single most important haemodynamic measure in MS; digoxin as add-on. Rhythm control / cardioversion in selected patients after anticoagulation and relief of stenosis
Anticoagulation	**Warfarin, target INR 2.0–3.0** — in MS with AF, prior embolism or LA thrombus. **DOACs are not used in rheumatic MS with AF** (INVICTUS trial — warfarin superior). Rheumatic MS with AF does not need a CHA_2DS_2-VASc score — all are anticoagulated
Congestion	Loop diuretic, salt restriction; avoid exertion until intervention
Intervention — PTMC / PBMV	**Percutaneous transmitral commissurotomy** (balloon, Inoue): **symptomatic severe MS (MVA ≤1.5 cm²) with favourable valve morphology** (Wilkins ≤8), **no LA thrombus** on TEE, no more than mild-to-moderate MR. Also asymptomatic very severe MS with favourable valve, and before pregnancy. Can be done in pregnancy if refractory
Surgery	**Mitral valve replacement** (or open commissurotomy) — Wilkins >8 / heavy calcification, persistent LA thrombus, moderate–severe MR, other valve surgery needed. Mechanical valve — lifelong warfarin (INR 2.5–3.5)
Pregnancy	MS is the most dangerous common valve lesion in pregnancy (rising HR and volume). β-blocker, diuretic, PTMC if NYHA III–IV despite medical therapy; plan pregnancy after PTMC; counsel about warfarin embryopathy (switch to heparin in the first trimester if needed)
IE prophylaxis & vaccines	Dental hygiene; IE prophylaxis (see Section B); influenza and pneumococcal vaccines

In this patient: metoprolol up-titrated to a resting rate <80/min, furosemide, warfarin (INR 2–3), benzathine penicillin restarted every 3 weeks; TEE showed no LA appendage clot, Wilkins score 7 → **PTMC** planned; contraception (copper IUD) counselled.

Complications of MS — recite in order

Atrial fibrillation · systemic embolism (stroke) · pulmonary hypertension → RV failure, TR · pulmonary oedema, haemoptysis · recurrent chest infections · infective endocarditis (less common in pure MS) · Ortner's syndrome (hoarseness — LA/pulmonary artery compressing the left recurrent laryngeal nerve) · dysphagia · pulmonary haemosiderosis.

CASE 2 · MITRAL REGURGITATION

Chronic Rheumatic Mitral Regurgitation

Volume overload of the left atrium and left ventricle

History

Particulars	Mr. A. K., 34 years, male, autorickshaw driver, Thrissur; informant — patient, reliable
Chief complaints	**Breathlessness on exertion — 2 years**; **easy fatigability — 1 year**; **palpitations on exertion — 6 months**

HOPI. Insidious exertional breathlessness for 2 years, now on walking 200 m on level ground (**NYHA II–III**); fatigue out of proportion to breathlessness (low forward output). Palpitations — regular, on exertion. Orthopnoea for 2 months (one extra pillow); no PND. No haemoptysis, chest pain, syncope. No fever, weight loss or joint pain now. No embolic events. No leg swelling.

Past history. Joint pains and fever at 13 years; told he had a "heart murmur" at 16 during a school check-up; took penicillin injections irregularly for 1 year. No history of chest trauma, previous MI, hypertension or diabetes. **Drugs:** none regularly. **Personal:** non-smoker, occasional alcohol. **Family:** no sudden death, no Marfan features.

> **Examiner's tip — in MR always ask for the cause**
>
> Rheumatic (young, past RF) · **mitral valve prolapse** (palpitations, atypical chest pain, mid-systolic click) · **ischaemic / functional** (previous MI, angina, dilated cardiomyopathy) · **infective endocarditis** (fever, sudden worsening) · acute MR from chordal or papillary muscle rupture (sudden pulmonary oedema after MI) · connective tissue disease (Marfan, Ehlers–Danlos).

General examination and vitals

Finding	In this patient	Significance
General	Thin build; no Marfanoid features; no pallor, cyanosis, clubbing, oedema; no IE stigmata	—
Pulse	92/min, regular, **normal-to-high volume, short sharp upstroke** (brisk), all peripheral pulses felt	Rapid LV emptying into the low-pressure LA
BP	118/70 mmHg	Normal
JVP	Not raised	No right heart failure
RR, SpO_2	18/min, 97 %	—

Cardiovascular examination

Step	Finding	Interpretation
Inspection	Apex visible in left 6th ICS; no scars	LV enlargement
Apex	**Left 6th ICS, 2 cm lateral to the MCL, hyperdynamic (thrusting), diffuse**	LV volume overload — dilated LV
Thrill	Systolic thrill at apex	Murmur grade ≥4
Parasternal heave	Grade I (late systolic lift — from expanding LA in systole, or RV)	Large LA / mild PAH
S1	**Soft / buried in the murmur**	Incomplete leaflet coaptation
S2	**Widely split** (early A2 — LV empties early); P2 normal	Wide physiological split
S3	**Present at apex**	Rapid filling of the LV with the large regurgitant volume — sign of **severe** MR (not necessarily failure)
Murmur	**High-pitched, blowing pansystolic murmur** at the apex, grade 4/6 with thrill, **radiating to the axilla** and inferior angle of scapula, louder in expiration and with **handgrip**; short mid-diastolic flow murmur after S3 ▶ Listen	MR; diastolic flow murmur = large regurgitant volume = severe
Lungs	Few basal crepitations	Pulmonary venous congestion

Signs of severe chronic MR

- Displaced, hyperdynamic apex (LV dilatation)
- **Soft S1**, wide split S2
- **S3** and a **mid-diastolic flow murmur**
- Signs of pulmonary hypertension, AF
- Symptoms; LV dysfunction on echo
- Note: the **loudness of the murmur does not reflect severity**

60-second presentation

Present it like this

"Mr A. K., a 34-year-old autorickshaw driver with a past history of rheumatic polyarthritis at 13 and irregular prophylaxis, presents with exertional breathlessness for 2 years, now NYHA II–III, fatigue and exertional palpitations, with recent orthopnoea. Pulse 92/min regular with a brisk upstroke, BP 118/70, JVP not raised. The apex is in the left 6th space 2 cm outside the MCL, hyperdynamic, with an apical systolic thrill. S1 is soft, S2 widely split, with an S3 and a grade 4/6 high-pitched pansystolic murmur at the apex radiating to the axilla, with a short mid-diastolic flow murmur. There are a few basal crepitations. **My diagnosis is chronic rheumatic heart disease with severe mitral regurgitation, with LV dilatation, in sinus rhythm, NYHA class II–III, with mild pulmonary venous congestion, no evidence of pulmonary hypertension or infective endocarditis.**"

Differential diagnosis of an apical pansystolic murmur

Condition	Distinguishing features
Mitral regurgitation	Apex, radiates to axilla, ↑ expiration, ↑ handgrip, soft S1, S3
Tricuspid regurgitation	Left lower sternal border, ↑ **inspiration** (Carvallo), large v waves, pulsatile liver
Ventricular septal defect	Left 3rd–4th ICS, radiates across the precordium, harsh, thrill at left sternal border
Mitral valve prolapse	**Mid-systolic click + late systolic murmur**; murmur longer and click earlier on standing / Valsalva
HOCM	Ejection murmur at left sternal border, ↑ Valsalva / standing, jerky pulse, double apical impulse
Aortic stenosis (Gallavardin)	Ejection murmur may radiate to the apex — slow-rising pulse, soft A2

Acute versus chronic MR

Feature	Acute MR	Chronic MR
Causes	Papillary muscle rupture (MI), chordal rupture, IE	Rheumatic, MVP, functional (DCM), ischaemic
LA / LV	Normal size, non-compliant LA → very high LA pressure	Dilated, compliant LA and LV
Presentation	Sudden pulmonary oedema, cardiogenic shock	Slowly progressive dyspnoea, fatigue
Murmur	Short, early systolic, may be soft or absent	Pansystolic
Treatment	Emergency — vasodilators, IABP, urgent surgery	Timing of surgery by symptoms, LV size and function

Investigations

Test	Findings	Remarks
ECG	Sinus rhythm; **P mitrale**; **LVH** (volume overload); AF later ▶ ECG ▶ ECG	Q waves → ischaemic MR
Chest X-ray	**Cardiomegaly** (LV + LA), double atrial shadow, pulmonary venous hypertension; giant LA in long-standing MR ▶ X-ray	Cardiothoracic ratio >0.5
Echo (TTE)	Thickened, restricted posterior leaflet (rheumatic), central / eccentric jet; **vena contracta 0.8 cm, EROA 0.45 cm², regurgitant volume 65 mL**; LVEF 62 %, **LVESD 38 mm**; LA 54 mm; PASP 36 mmHg	Severe primary MR, preserved LV
TEE	Mechanism, repairability, vegetations	Before surgery
Others	Coronary angiography before surgery (age >40 / risk factors); NT-proBNP; blood cultures if fever	—

Severe primary MR (ACC/AHA)	Criterion
Vena contracta	≥0.7 cm
Effective regurgitant orifice area (EROA)	≥0.40 cm²
Regurgitant volume / fraction	≥60 mL / ≥50 %
Supporting	Central jet >40 % of LA; LA and LV enlargement; pulmonary vein systolic flow reversal

Management of MR

Area	Management
Rheumatic prophylaxis	Benzathine penicillin 1.2 MU every 3 weeks (as in MS)
Medical	Diuretics for congestion. **No drug alters the natural history of primary MR.** If LV dysfunction or heart failure — guideline-directed therapy (ACEi/ARNI, β-blocker, MRA, SGLT2 inhibitor); vasodilators reduce regurgitation only in acute MR or hypertension
AF	Rate control; anticoagulation — DOAC or warfarin (DOACs are acceptable in MR without significant MS or a mechanical valve)
Surgery — primary severe MR	**Symptomatic** (any LVEF >30 %) → surgery. **Asymptomatic** with **LVEF ≤60 %** or **LVESD ≥40 mm** → surgery. Asymptomatic with normal LV — consider repair if success likely (>95 %) and new AF or PASP >50 mmHg
Type of surgery	**Repair preferred over replacement** (degenerative MR); rheumatic valves are often less repairable → replacement
Secondary (functional) MR	Optimise heart failure therapy and CRT first; **transcatheter edge-to-edge repair (TEER, MitraClip)** in selected patients who remain symptomatic

In this patient: severe symptomatic rheumatic MR with LVESD 38 mm → referred for **mitral valve surgery** (repair if feasible, otherwise replacement); diuretic; penicillin prophylaxis; dental review.

Mitral valve prolapse — a favourite short case

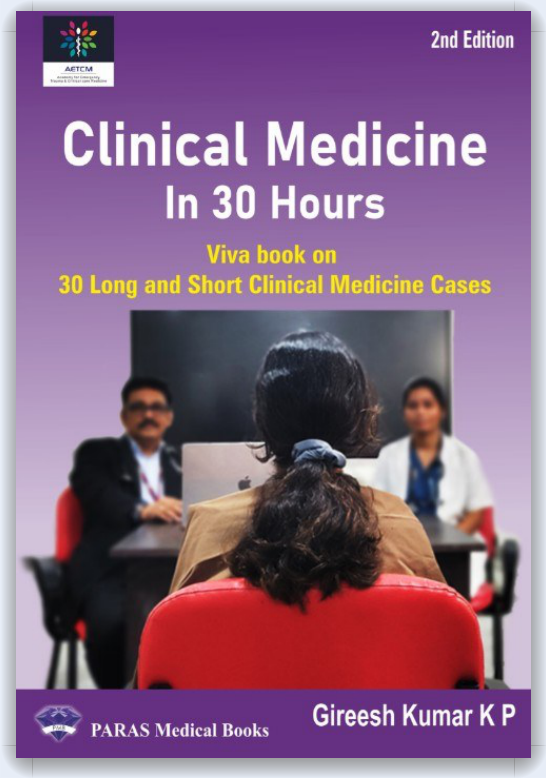
Young woman, palpitations, atypical chest pain; **mid-systolic click followed by a late systolic murmur**; on **standing or Valsalva** the click moves earlier and the murmur lengthens; squatting delays it. Echo: leaflet displacement >2 mm above the annulus. Usually benign; β-blocker for palpitations; surgery if severe MR.

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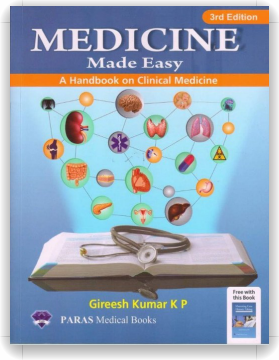
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CASE 3 · AORTIC STENOSIS

Severe Calcific Aortic Stenosis

Pressure overload of the left ventricle — angina, syncope, breathlessness

History

Particulars	Mr. P. G., 66 years, male, retired schoolteacher, Kottayam; informant — patient, reliable
Chief complaints	**Chest pain on exertion — 6 months**; **breathlessness on exertion — 4 months**; **one episode of fainting while walking — 2 weeks ago**

HOPI. Central chest tightness on walking briskly or climbing stairs for 6 months, lasting 5 minutes, relieved by rest (**exertional angina**). Breathlessness on moderate exertion for 4 months (**NYHA II**). Two weeks ago, while walking uphill, he felt dizzy and **lost consciousness for about 30 seconds**, recovering fully without confusion, tongue bite or incontinence (**exertional syncope**). No palpitations, orthopnoea, PND, leg swelling. No fever. No history of rheumatic fever or a murmur in childhood. No GI bleeding (Heyde's syndrome).

Past history. Hypertension for 10 years on amlodipine; dyslipidaemia on atorvastatin; no diabetes. **Personal:** ex-smoker (15 pack-years). **Family:** no sudden death. **Drugs:** he had been given sublingual nitrate for the chest pain by a local clinic (dangerous in severe AS).

> **Examiner's tip — the classic triad and prognosis**
>
> Once symptoms appear in severe AS, average survival without valve replacement is short: about **5 years after angina, 3 years after syncope and 2 years after heart failure**. That is why the history of these three symptoms is the most important part of the AS case.

General examination and vitals

Finding	In this patient	Significance
General	Comfortable at rest; xanthelasma absent; arcus present; no pallor, cyanosis, clubbing, oedema	Atherosclerotic risk
Pulse	68/min, regular, **low volume, slow-rising (anacrotic) at the carotid**; carotid shudder ▶ Article	**Pulsus parvus et tardus** — fixed obstruction
BP	124/90 mmHg — **pulse pressure 34 mmHg (narrow)**	Severe AS (may be masked by hypertension)
JVP	Not raised; prominent a wave (Bernheim effect)	—
RR, SpO_2	16/min, 98 %	—

Cardiovascular examination

Step	Finding	Interpretation
Apex	**Left 5th ICS just outside MCL, heaving (sustained)**, not much displaced; palpable S4 (double apical impulse)	Concentric LVH — pressure overload; LV dilates only late
Thrill	**Systolic thrill** at the aortic area and over the carotids	Significant AS
S1	Normal	—
S2	**Soft / absent A2**; **reversed (paradoxical) split**	Calcified immobile valve; delayed LV emptying — severe
S4	Present at apex	Stiff hypertrophied LV
Ejection click	Absent	Calcified valve (click present in mobile bicuspid valve)
Murmur	**Harsh, crescendo–decrescendo ejection systolic murmur** at the aortic area (right 2nd ICS), grade 4/6, **radiating to both carotids**, louder in expiration and sitting forward; **late peaking**; musical high-frequency component at the apex (**Gallavardin**) ▶ Listen	AS; late peak and soft A2 = severe
Lungs	Clear	—

Signs of severe aortic stenosis

- Slow-rising, low-volume pulse; narrow pulse pressure
- Heaving apex, palpable S4
- Thrill at aortic area / carotids
- **Soft or absent A2**, reversed split S2
- **Late-peaking** murmur (loudness falls when LV fails)
- Signs of LV failure

60-second presentation

Present it like this

"Mr P. G., a 66-year-old retired teacher, hypertensive and ex-smoker, presents with exertional angina for 6 months, exertional breathlessness for 4 months (NYHA II) and one episode of exertional syncope 2 weeks ago. He has a regular, low-volume, slow-rising pulse of 68/min, BP 124/90 with a narrow pulse pressure, and a non-raised JVP. The apex is in the 5th space just outside the MCL and heaving, with a palpable S4. There is a systolic thrill at the aortic area and carotids. A2 is soft with reversed splitting; there is an S4 and a harsh grade 4/6 late-peaking ejection systolic murmur at the aortic area radiating to both carotids. **My diagnosis is severe calcific (degenerative) aortic stenosis with concentric LV hypertrophy, symptomatic with angina, syncope and NYHA class II dyspnoea, in sinus rhythm, not in failure, with no evidence of infective endocarditis; coexisting coronary artery disease must be excluded.**"

Causes of aortic stenosis

Cause	Features
Calcific / degenerative	Commonest worldwide; >65 years; shares risk factors with atherosclerosis
Bicuspid aortic valve	Presents at 40–60 years; ejection click; associated **coarctation** and **aortic dilatation** (check radio-femoral delay); screen first-degree relatives
Rheumatic	Younger; almost always with mitral valve disease (MS); commissural fusion
Others	Congenital (unicuspid), supravalvular (Williams syndrome), subvalvular membrane

Differential diagnosis of an ejection systolic murmur at the aortic area

Condition	Distinguishing features
Aortic sclerosis	Elderly; normal pulse and A2; no thrill; short early-peaking murmur; no LVH
HOCM	Jerky (brisk) pulse; murmur at left sternal border, not to carotids; ↑ **on Valsalva / standing,** ↓ **on squatting / handgrip** (opposite of AS)
Flow murmur	Anaemia, pregnancy, fever, thyrotoxicosis, AR (high stroke volume) — soft, early
Pulmonary stenosis	Pulmonary area, ↑ inspiration, soft P2, RVH
MR / VSD	Pansystolic, different site and radiation
Coarctation	Radio-femoral delay, murmur at the back, upper-limb hypertension

Investigations

Test	Findings	Remarks
ECG	**LVH with strain** (ST depression, T inversion in I, aVL, V5–6); LA enlargement; LBBB or AV block (calcium into conduction system) ▶ ECG	—
Chest X-ray	Often normal heart size (concentric hypertrophy); **post-stenotic dilatation of the ascending aorta**; valve calcification on lateral view ▶ X-ray	Cardiomegaly = late, LV dilatation
Echo	Heavily calcified tricuspid valve, reduced opening; **AVA 0.7 cm², peak velocity 4.6 m/s**, **mean gradient 52 mmHg**; concentric LVH; LVEF 58 %	Severe AS
Coronary angiography / CT angiography	Before valve replacement — CAD coexists in about half of elderly AS patients	Do not do exercise stress testing in **symptomatic** severe AS
CT aorta / TAVI planning CT	Annulus size, access vessels, calcium score (low-flow AS)	—
Low-flow, low-gradient AS	AVA <1.0 but gradient <40 with low EF → **dobutamine stress echo** to separate true severe AS from pseudo-severe	—

AS severity (ACC/AHA)	Peak velocity	Mean gradient	Valve area
Mild	2.0–2.9 m/s	<20 mmHg	>1.5 cm²
Moderate	3.0–3.9 m/s	20–39 mmHg	1.0–1.5 cm²
Severe	**≥4.0 m/s**	**≥40 mmHg**	**≤1.0 cm²**
Very severe	≥5.0 m/s	≥60 mmHg	—

Management of AS

Area	Management
Indications for valve replacement	**Severe AS with symptoms** (angina, syncope, dyspnoea) — this patient. Asymptomatic severe AS with **LVEF <50 %**, abnormal exercise test, very severe AS (Vmax ≥5 m/s), rapid progression, or when undergoing other cardiac surgery
SAVR vs TAVI	**Surgical AVR** preferred in younger patients (<65 years) and low surgical risk; **TAVI (transcatheter AVR)** in older patients (>80 years) or high surgical risk; 65–80 years — shared decision by the Heart Team. Balloon aortic valvuloplasty only as a bridge
Medical (no drug relieves obstruction)	Treat hypertension cautiously (ACEi / ARB, start low); statins do not slow AS; diuretics cautiously (preload-dependent)
Drugs to avoid / use with care	**Nitrates and potent vasodilators** (can cause severe hypotension and syncope with a fixed outflow), excessive diuresis, strenuous exertion
Follow-up of asymptomatic AS	Echo every 6–12 months (severe), 1–2 years (moderate), 3–5 years (mild); educate to report symptoms immediately
IE prophylaxis & dental care	Good oral hygiene; prophylaxis after prosthetic valve

In this patient: symptomatic severe AS, 66 years → coronary angiography, Heart Team discussion; **SAVR or TAVI**; sublingual nitrate stopped; avoid strenuous exertion until replacement.

Examiner's tip — complications of AS

Sudden death (arrhythmia), LV failure, syncope, angina, **infective endocarditis**, heart block (calcium extending into conduction tissue), embolisation of calcium (stroke, retinal emboli), and **Heyde's syndrome** — GI bleeding from angiodysplasia with acquired von Willebrand deficiency.

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CASE 4 · AORTIC REGURGITATION

Chronic Rheumatic Aortic Regurgitation

Volume and pressure overload — a large heart, a bounding pulse and an early diastolic murmur

History

Particulars	Mr. J. T., 26 years, male, construction worker, Idukki; informant — patient, reliable
Chief complaints	**Breathlessness on exertion — 1 year**; **awareness of heartbeat (pounding) when lying down — 8 months**; **chest pain on exertion — 3 months**

HOPI. Gradually progressive exertional breathlessness for 1 year, now on carrying loads at work (**NYHA II**). Uncomfortable **awareness of a forceful heartbeat** and pulsation in the neck and head, especially on lying on the left side (large stroke volume). Exertional chest pain for 3 months (reduced diastolic coronary perfusion). No orthopnoea, PND, syncope, haemoptysis, fever, leg swelling. No back pain or chest trauma.

Past history. Fever with knee and elbow pain at 12 years; treated at a local hospital; **no penicillin prophylaxis**. No history of syphilis, back stiffness (ankylosing spondylitis), joint hypermobility or eye problems (Marfan). **Family:** no sudden death, no tall relatives with aortic disease. **Personal:** non-smoker; no IV drug use.

Examiner's tip — two groups of causes of AR

Valve disease: rheumatic (commonest in India; usually with mitral disease), bicuspid valve, infective endocarditis, rheumatoid / SLE. **Aortic root disease:** hypertension, Marfan syndrome, aortic dissection, syphilitic aortitis, ankylosing spondylitis, Takayasu arteritis, Ehlers–Danlos, osteogenesis imperfecta. Always look for Marfanoid habitus, high-arched palate and ask about back pain.

General examination and vitals

Finding	In this patient	Significance
General	Average build; no Marfanoid habitus, high-arched palate or spinal deformity; no pallor, clubbing, IE stigmata; no oedema	Rheumatic aetiology likely
Pulse	**88/min, regular, high volume, collapsing (water-hammer)** — felt best with the arm raised above the head ▶ Video	Large stroke volume + rapid diastolic run-off
BP	**150/40 mmHg — pulse pressure 110 mmHg**; lower limb (popliteal) systolic 220 mmHg	Wide pulse pressure; **Hill's sign** (difference >60 mmHg = severe AR)
JVP	Not raised	—
Neck	Vigorous carotid pulsations (**Corrigan's sign**) ▶ Article	—

Peripheral signs of aortic regurgitation — learn them all

Sign	Description	This patient
Collapsing (water-hammer) pulse	Rapid upstroke and sudden collapse; feel with arm elevated	Present
Corrigan's sign	Visible forceful carotid pulsation (“dancing carotids”)	Present
de Musset's sign	Head nodding with each heartbeat	Present
Quincke's sign	Capillary pulsation in the nail bed on gentle pressure on the nail tip ▶ Article	Present
Traube's sign (pistol shot)	Sharp sound over the femoral artery with the stethoscope placed lightly	Present
Duroziez's sign	Systolic and diastolic bruit over the femoral artery on compression with the stethoscope	Present
Others	Müller's (uvula), Becker's (retinal arteries), Landolfi's (pupil), Mayne's (diastolic BP falls >15 on raising the arm), Rosenbach's / Gerhardt's (pulsatile liver / spleen), lighthouse sign (forehead flushing)	—

Sign	Description	This patient
Hill's sign	Popliteal systolic BP > brachial by >20 mmHg (>60 = severe)	70 mmHg

These signs indicate a **large stroke volume with wide pulse pressure** — they reflect chronic severe AR but are also seen in other high-output states (thyrotoxicosis, anaemia, PDA, AV fistula, pregnancy). They are **absent in acute AR**. ▶ All signs

Cardiovascular examination

Step	Finding	Interpretation
Inspection	Visible apical impulse in left 6th ICS; suprasternal pulsation	LV dilatation
Apex	**Left 6th ICS, 2.5 cm outside the MCL, hyperdynamic, diffuse**	Volume-overloaded dilated LV
Thrill	Systolic thrill at the aortic area (flow)	Large stroke volume
S1, S2	S1 normal; **A2 soft**	Valve fails to coapt
Murmur 1	**High-pitched, blowing, early diastolic decrescendo murmur** beginning with A2, best at the **left 3rd ICS (Erb's / 2nd aortic area)**, with the **diaphragm**, patient **sitting up and leaning forward in held expiration**; grade 3/4; **long** (pandiastolic) ▶ Listen	AR (left sternal border = valvular; right sternal border = aortic root dilatation)
Murmur 2	Ejection systolic murmur at the aortic area (flow across the valve)	Large stroke volume — not necessarily AS
Murmur 3	Low-pitched mid-diastolic rumble at the apex **without** loud S1 or opening snap	**Austin Flint murmur** — sign of severe AR
S3	Present	LV volume overload
Lungs, abdomen	Clear; liver not palpable	—

Signs of severe chronic AR

- Wide pulse pressure, **diastolic BP <50 mmHg**, Hill's sign >60
- Prominent peripheral signs
- Displaced hyperdynamic apex
- **Long** early diastolic murmur (in acute or very severe AR the murmur **shortens** as LV diastolic pressure rises)
- **Austin Flint** murmur, S3
- Signs of LV failure

60-second presentation

Present it like this

"Mr J. T., a 26-year-old construction worker with rheumatic fever at 12 and no prophylaxis, presents with exertional breathlessness for 1 year (NYHA II), awareness of a pounding heartbeat for 8 months and exertional chest pain for 3 months. He has a regular collapsing pulse of 88/min, BP 150/40 with a pulse pressure of 110 and Hill's sign of 70 mmHg, with Corrigan's, de Musset's, Quincke's, Traube's and Duroziez's signs. The apex is in the 6th space 2.5 cm outside the MCL, hyperdynamic. A2 is soft; there is an S3, a long high-pitched early diastolic decrescendo murmur at the left 3rd space heard best sitting forward in expiration, a flow ejection systolic murmur and an Austin Flint murmur at the apex. **My diagnosis is chronic rheumatic heart disease with severe aortic regurgitation, with LV dilatation, symptomatic — NYHA class II with angina — in sinus rhythm, not in failure, no evidence of infective endocarditis; no clinical mitral valve involvement.**"

Differential diagnosis of an early diastolic murmur

Condition	Distinguishing features
Aortic regurgitation	Left 3rd ICS, ↑ sitting forward in expiration, peripheral signs, wide pulse pressure, LV enlargement
Graham Steell murmur (pulmonary regurgitation)	Severe pulmonary hypertension (e.g. MS); left 2nd ICS; ↑ inspiration; loud P2, RV heave; **no** peripheral signs, normal pulse pressure
Aortic root dilatation	Murmur loudest at the right sternal border (Harvey's sign); Marfan, hypertension, dissection

Acute versus chronic AR

Feature	Acute AR	Chronic AR
Causes	**Infective endocarditis, aortic dissection**, trauma, prosthetic valve failure	Rheumatic, bicuspid, root dilatation
LV	Normal size, cannot accommodate volume — LV diastolic pressure rises steeply	Dilated, compliant
Presentation	Sudden pulmonary oedema, cardiogenic shock	Slowly progressive dyspnoea
Pulse pressure / peripheral signs	Normal or narrow / **absent**	Wide / present
Murmur	Short, soft early diastolic; soft S1 (premature mitral closure)	Long
Treatment	**Emergency surgery**; vasodilators, inotropes; **no IABP** (worsens AR)	Timed surgery

Investigations

Test	Findings	Remarks
ECG	**LVH** (volume overload — tall R, deep S, often with tall T waves; strain later); left axis deviation ▶ ECG	—
Chest X-ray	**Cardiomegaly with LV configuration** ("boot-shaped" — apex down and out); dilated ascending aorta / aortic knuckle ▶ X-ray ▶ Article	Wide mediastinum → dissection
Echo	Thickened aortic cusps with central jet; **vena contracta 0.7 cm**, jet width 70 % of LVOT, **holodiastolic flow reversal** in the descending aorta; **LVEF 58 %**, **LVESD 46 mm**, LVEDD 68 mm; mild MR	Severe AR, preserved EF
Others	VDRL, blood cultures (fever), CT/MRI aorta (root), HLA-B27 if spondylitis suspected	—

Severe AR (ACC/AHA)	Criterion
Jet width / LVOT width	≥65 %
Vena contracta	>0.6 cm
Regurgitant volume / fraction · EROA	≥60 mL / ≥50 % · ≥0.3 cm²

Management of AR

Area	Management
Indications for aortic valve replacement (chronic severe AR)	**Symptomatic** — regardless of LV function (this patient). **Asymptomatic** with **LVEF ≤55 %**, or **LVESD >50 mm** (indexed >25 mm/m²); consider with progressive LV dilatation (LVEDD >65 mm). Also when undergoing other cardiac surgery. Aortic root replacement if root ≥5.0–5.5 cm (lower thresholds in Marfan)
Medical	**Vasodilators** (ACEi / ARB, dihydropyridine CCB) for hypertension and in those unfit for surgery; GDMT if LV dysfunction; avoid bradycardia (β-blockers lengthen diastole and increase regurgitation — except Marfan)
Rheumatic prophylaxis	Benzathine penicillin every 3 weeks
IE & follow-up	Dental hygiene; fever → blood cultures before antibiotics. Asymptomatic severe AR — echo every 6–12 months
Valve choice	Mechanical (young; warfarin INR 2–3) vs bioprosthetic / Ross — consider adherence and pregnancy plans

In this patient: symptomatic severe rheumatic AR with LVESD 46 mm → referred for **aortic valve replacement**; penicillin prophylaxis started; amlodipine for systolic hypertension until surgery; dental check-up.

> **Examiner's tip — AR sequence in 90 seconds**
>
> Pulse with arm raised → BP arm and leg (Hill) → head (de Musset), neck (Corrigan), nails (Quincke) → apex → left 3rd ICS leaning forward in held expiration → apex (Austin Flint) → femorals (Traube, Duroziez).

SECTION B · REVISION

The Four Valve Lesions Compared

One table to revise the night before the examination

Feature	Mitral stenosis	Mitral regurgitation	Aortic stenosis	Aortic regurgitation
Common cause (India)	Rheumatic	Rheumatic, MVP, functional	Calcific, bicuspid, rheumatic	Rheumatic, root disease
Key symptoms	Dyspnoea, haemoptysis, palpitations (AF), embolism	Fatigue, dyspnoea	Angina, syncope, dyspnoea	Palpitations (pounding), dyspnoea, angina
Pulse	Low volume; AF	Brisk, normal / high volume	**Slow-rising, low volume**	**Collapsing**
Pulse pressure	Normal / narrow	Normal	**Narrow**	**Wide**
Apex	Normal site, **tapping**	Displaced, **hyperdynamic**	Slightly displaced, **heaving**	Displaced down and out, **hyperdynamic**
Thrill	Diastolic at apex	Systolic at apex	Systolic at aortic area, carotids	Systolic (flow) at aortic area
S1	**Loud**	**Soft**	Normal	Normal / soft
S2	Loud P2 (PAH)	Wide split	**Soft A2, reversed split**	Soft A2
Added sounds	**Opening snap**	**S3**	S4, ejection click (bicuspid)	S3, ejection click
Murmur	Mid-diastolic rumble with presystolic accentuation	Pansystolic, blowing	Ejection systolic, harsh, crescendo–decrescendo	Early diastolic, blowing, decrescendo
Site / radiation	Apex, localised	Apex → axilla	Aortic area → carotids	Left 3rd ICS → apex
Best heard	Bell, left lateral, after exercise	Diaphragm, expiration, handgrip	Diaphragm, sitting forward, expiration	Diaphragm, leaning forward, held expiration
Severity sign	Short S2–OS, long murmur	S3, MDM flow murmur, soft S1	Soft A2, late peak, slow pulse	Wide PP, Austin Flint, long murmur
ECG	P mitrale, AF, RVH	P mitrale, LVH, AF	LVH with strain	LVH (volume)
Chest X-ray	LA enlargement, normal LV, PVH	LA + LV enlargement	Normal size, post-stenotic dilatation	LV enlargement, dilated aorta
Echo — severe	MVA ≤1.5 cm²	EROA ≥0.4 cm², RV ≥60 mL	Vmax ≥4 m/s, MG ≥40, AVA ≤1.0	VC >0.6 cm, RF ≥50 %
Definitive treatment	**PTMC** / MVR	Repair / MVR, TEER	**SAVR / TAVI**	AVR
Timing (asymptomatic)	Very severe + favourable valve	EF ≤60 %, LVESD ≥40 mm	EF <50 %, Vmax ≥5 m/s	EF ≤55 %, LVESD >50 mm

Dynamic auscultation — effect of manoeuvres

Manoeuvre	Effect
Inspiration	↑ right-sided murmurs (TR, TS, PS, PR) — Carvallo's sign
Expiration	↑ left-sided murmurs (MS, MR, AS, AR)
Valsalva (strain) / standing	↓ most murmurs; ↑ **HOCM**; MVP click earlier, murmur longer
Squatting / passive leg raise	↑ most murmurs (venous return); ↓ HOCM; MVP murmur shorter
Isometric handgrip (↑ afterload)	↑ MR, AR, VSD; ↓ AS, HOCM
Amyl nitrite (↓ afterload)	↑ AS, HOCM; ↓ MR, AR
Exercise / left lateral position	↑ MS murmur

Multivalvular rheumatic heart disease — which lesion is dominant?

Combination	Clues to dominant lesion	Pearls
MS + MR	**MS dominant:** loud S1, OS, tapping apex, normal-sized LV. **MR dominant:** soft S1, S3, displaced hyperdynamic apex, LV enlargement	Commonest combination; significant MR contraindicates PTMC

Combination	Clues to dominant lesion	Pearls
MS + AR	AR features (wide PP, collapsing pulse) may be **blunted by MS** (low stroke volume); MS murmur may be confused with Austin Flint	Austin Flint: no loud S1, no OS
AS + AR	**Bisferiens pulse** when both significant; pulse pressure, apex character and murmur length decide dominance	Rheumatic or bicuspid
MS + AS	MS reduces LV filling → AS murmur and gradient **underestimated**; angina/syncope less prominent	Echo essential
MS + TR (functional)	PAH, RV dilatation; TR murmur ↑ inspiration, large v waves, pulsatile liver	TR may regress after PTMC

Order of frequency of valve involvement in RHD: **mitral > aortic > tricuspid > pulmonary**. Isolated aortic valve disease without mitral involvement is unusual in rheumatic disease — think of bicuspid valve or root disease.

Rheumatic fever — diagnosis and prophylaxis

Revised Jones criteria (2015) — low-risk vs moderate/high-risk populations (India is high-risk)

	Major criteria (high-risk population)	Minor criteria (high-risk population)
Criteria	Carditis (clinical or subclinical on echo); arthritis (**polyarthritis, monoarthritis or polyarthralgia**); chorea; erythema marginatum; subcutaneous nodules	Monoarthralgia; fever ≥38 °C; ESR ≥30 mm/h and/or CRP ≥3 mg/dL; prolonged PR interval (if carditis not a major criterion)
Diagnosis	Evidence of preceding group A streptococcal infection (ASO / anti-DNase B, throat culture, rapid antigen) **plus** 2 major, or 1 major + 2 minor (initial episode); recurrence — 2 major, 1 major + 2 minor, or 3 minor	

Prevention

Type	Regimen
Primary (treat sore throat)	Benzathine penicillin G 1.2 MU IM once (0.6 MU if <27 kg), or oral penicillin V / amoxicillin for 10 days
Secondary (prevent recurrence)	**Benzathine penicillin G 1.2 MU IM every 3 weeks** (every 21 days recommended in India); penicillin allergy — erythromycin 250 mg BD
Duration	RF without carditis: 5 years or until 18 years (whichever longer) · RF with carditis, no residual disease: 10 years or until 25 years · **Residual valve disease: 10 years after the last episode or until 40 years**, whichever is longer; sometimes lifelong (severe disease, after valve surgery)

Infective endocarditis — remember in every valve case

Look for: fever, new or changing murmur, splinter haemorrhages, Osler's nodes (painful, finger pulp), Janeway lesions (painless, palms), Roth spots, splenomegaly, clubbing, microscopic haematuria, embolic events. **Modified Duke criteria**: major — two positive blood cultures with typical organisms, echo evidence (vegetation, abscess, new dehiscence), new regurgitation. Draw **three sets of blood cultures** before antibiotics. Antibiotic prophylaxis before dental procedures (amoxicillin 2 g orally 30–60 min before) is recommended for high-risk patients — **prosthetic valves, previous IE**, certain congenital heart disease; most Indian cardiologists also advise it in rheumatic heart disease. Good oral hygiene for all.

Anticoagulation in valve disease — quick rules

Rheumatic MS with AF → **warfarin** (INR 2–3), not DOAC · AF with other native valve disease (MR, AS, AR) → DOAC or warfarin · **Mechanical valve → warfarin only** (mitral INR 2.5–3.5; aortic 2.0–3.0, higher with risk factors) — DOACs contraindicated · Bioprosthetic valve → warfarin or aspirin for 3–6 months, then by rhythm.

SECTION G · VIVA VOCE

Frequently Asked Viva Questions with Answers

Short, exam-ready answers

Q1 Why is S1 loud in mitral stenosis?

High LA pressure keeps the mitral leaflets widely open until the end of diastole; when the LV contracts they close from a wide excursion, rapidly and forcefully. S1 becomes soft when the leaflets are heavily calcified and immobile.

Q2 What is the opening snap and what does the S2–OS interval tell you?

A sharp, high-pitched sound produced when the domed, pliable stenotic mitral valve suddenly reaches its maximum opening in early diastole. The higher the LA pressure, the earlier the valve opens — a **short S2–OS interval (<0.08 s) means severe MS**. An OS indicates a pliable valve (favourable for balloon valvotomy).

Q3 Why is presystolic accentuation absent in AF?

Presystolic accentuation is produced by atrial contraction pushing blood across the stenotic valve at the end of diastole. In AF there is no effective atrial contraction. (Some authors attribute it partly to narrowing of the orifice as the valve begins to close.)

Q4 Why does the LV remain normal in size in pure MS?

The stenosis is proximal to the LV; the LV is under-filled, not overloaded. LV enlargement in a patient with MS suggests associated MR, AR, or another LV disease.

Q5 Describe the chest X-ray in mitral stenosis.

Straight left heart border (enlarged LA appendage, prominent pulmonary artery), double atrial shadow on the right, widened carina (>90°) with elevated left main bronchus, upper-lobe venous diversion, Kerley B lines, normal-sized LV, sometimes mitral calcification and haemosiderosis; lateral view / barium swallow — posterior displacement by LA.

Q6 What are the causes of haemoptysis in MS?

Rupture of dilated bronchial veins (pulmonary apoplexy), pink frothy sputum of pulmonary oedema, blood-streaked sputum with bronchitis, pulmonary infarction (embolism from RV / leg veins), and chronic pulmonary haemosiderosis.

Q7 What is the Wilkins score?

An echo score for suitability of balloon mitral valvotomy: leaflet mobility, leaflet thickening, calcification and subvalvular thickening, each graded 1–4 (total 4–16). A score of **≤8** predicts a good result.

Q8 What are the contraindications to PTMC?

LA or LA appendage thrombus, more than mild-to-moderate MR, heavy or bicommissural calcification, severe concomitant aortic or tricuspid disease needing surgery, coexisting CAD needing bypass, and unfavourable anatomy (high Wilkins score).

Q9 Why is warfarin, and not a DOAC, used in rheumatic MS with AF?

In the INVICTUS trial, warfarin was associated with fewer major cardiovascular events and deaths than rivaroxaban in rheumatic heart disease with AF. Guidelines therefore recommend vitamin K antagonists in moderate-to-severe rheumatic MS with AF, and in all mechanical valves.

Q10 Why is a β-blocker useful in MS even without AF?

It slows the heart rate and lengthens diastole, allowing more time for blood to cross the stenotic valve and lowering LA pressure. Tachycardia (exercise, fever, pregnancy, AF) is what decompensates MS.

Q11 What is Ortner's syndrome?

Hoarseness due to compression of the left recurrent laryngeal nerve between an enlarged left atrium / dilated pulmonary artery and the aorta, classically in MS.

Q12 What is Lutembacher syndrome?

Atrial septal defect with mitral stenosis (congenital or acquired). The ASD decompresses the LA, so pulmonary congestion is less, but right-sided volume overload is greater.

Q13 What are the causes of mitral regurgitation?

Primary (leaflet disease): rheumatic, myxomatous / MVP, infective endocarditis, connective tissue disease, congenital cleft. Secondary (functional): LV dilatation — dilated cardiomyopathy, ischaemic heart disease; papillary muscle dysfunction or rupture after MI; annular dilatation or calcification.

Q14 Why is S1 soft and S3 present in MR?

S1 is soft because the leaflets do not coapt properly. S3 is due to rapid filling of the LV in early diastole by the large volume returning from the LA — it indicates severe MR, not necessarily heart failure.

Q15 Why does the murmur of MR radiate to the axilla?

The regurgitant jet in rheumatic MR is commonly directed posterolaterally towards the left axilla. With anterior leaflet prolapse the jet is directed posteriorly and the murmur may radiate to the back; with posterior leaflet prolapse it may radiate to the base and mimic AS.

Q16 When is surgery indicated in chronic primary MR?

Severe MR with symptoms; asymptomatic severe MR with LVEF ≤60 % or LVESD ≥40 mm; repair may be considered earlier when the likelihood of successful durable repair is high, or with new AF or PASP >50 mmHg.

Q17 Why is the LVEF cut-off for surgery in MR 60 % and not 50 %?

In MR the LV ejects part of its volume into the low-pressure LA, so the ejection fraction overestimates true LV contractility. An EF of 60 % in severe MR already indicates LV dysfunction.

Q18 How do you differentiate MR from TR clinically?

MR — apex, radiates to the axilla, louder in expiration, no change in JVP. TR — left lower sternal border, louder on inspiration (Carvallo's sign), large cv waves in the JVP, pulsatile liver, often with signs of pulmonary hypertension.

Q19 What is mitral valve prolapse and how do manoeuvres affect it?

Systolic billowing of one or both leaflets into the LA. Mid-systolic click followed by a late systolic murmur. Standing or Valsalva reduces LV volume — the click comes earlier and the murmur lengthens; squatting increases LV volume — the click and murmur are delayed and shortened.

Q20 What is TEER (MitraClip)?

Transcatheter edge-to-edge repair — a clip grasps the anterior and posterior leaflets to create a double-orifice valve. Used in high-surgical-risk primary MR and in selected symptomatic secondary MR despite optimal heart-failure therapy.

Q21 What are the causes of aortic stenosis?

Valvular — calcific degenerative (elderly), congenital bicuspid (middle age), rheumatic (with mitral disease), unicuspid. Subvalvular — membrane, HOCM (dynamic). Supravalvular — Williams syndrome.

Q22 What is the classic triad of AS and its prognosis?

Angina, syncope and breathlessness (heart failure). After the onset of symptoms average survival without valve replacement is about 5 years with angina, 3 years with syncope and 2 years with heart failure.

Q23 Why does angina occur in AS with normal coronaries?

Increased oxygen demand of the hypertrophied LV, raised LV wall stress, compression of intramyocardial vessels by high LV pressure, reduced diastolic perfusion time and reduced coronary perfusion pressure; capillary density does not keep pace with hypertrophy.

Q24 Why does syncope occur in AS?

Exertion causes peripheral vasodilatation, but cardiac output cannot rise across the fixed obstruction, so BP falls; LV baroreceptor-mediated vasodepressor reflex; arrhythmias (VT, heart block).

Q25 Why is A2 soft and S2 reversed in severe AS?

A2 is soft because the calcified cusps barely move. Prolonged LV ejection delays A2 so that it follows P2 — the split widens on expiration and narrows on inspiration (paradoxical splitting).

Q26 What is the Gallavardin phenomenon?

In calcific AS, the high-frequency musical component of the ejection murmur is transmitted to the apex, where it may be mistaken for MR; the harsh component remains at the base.

Q27 How do you differentiate AS from HOCM?

AS: slow-rising pulse, murmur at aortic area radiating to carotids, soft A2, ejection click (bicuspid); ↓ with Valsalva. HOCM: brisk / jerky (bisferiens) pulse, double or triple apical impulse, murmur at left sternal border not radiating to carotids, ↑ with Valsalva and standing, ↓ with squatting and handgrip.

Q28 What are the echo criteria for severe AS?

Peak aortic jet velocity ≥4.0 m/s, mean gradient ≥40 mmHg, aortic valve area ≤1.0 cm^2 (indexed ≤0.6 cm^2/m^2). Very severe: velocity ≥5 m/s or mean gradient ≥60 mmHg.

Q29 What is low-flow, low-gradient AS?

Valve area ≤1.0 cm^2 but mean gradient <40 mmHg because the LV generates low flow — either reduced EF (classical) or small hypertrophied LV with normal EF (paradoxical). Dobutamine stress echo or CT calcium scoring distinguishes true severe from pseudo-severe AS.

Q30 SAVR or TAVI?

Decided by the Heart Team: surgical AVR in younger (<65 years) low-risk patients; TAVI in patients >80 years or at high / prohibitive surgical risk; 65–80 years — either, considering anatomy, life expectancy and patient preference.

Q31 Why should nitrates and vasodilators be avoided in severe AS?

The cardiac output is fixed by the obstruction and the LV is preload-dependent; vasodilatation and reduced preload cause profound hypotension and syncope.

Q32 What is Heyde's syndrome?

GI bleeding from intestinal angiodysplasia in patients with AS, due to acquired type 2A von Willebrand deficiency (shear-induced cleavage of large vWF multimers). It improves after valve replacement.

Q33 What are the causes of aortic regurgitation?

Valve: rheumatic, bicuspid, infective endocarditis, rheumatoid arthritis, SLE. Aortic root: hypertension, Marfan syndrome, aortic dissection, syphilitic aortitis, ankylosing spondylitis and other spondyloarthritides, Takayasu, Ehlers–Danlos, osteogenesis imperfecta. Acute: IE, dissection, trauma.

Q34 Why is the pulse collapsing in AR?

A large stroke volume is ejected rapidly (sharp upstroke, high systolic pressure), and the aortic pressure then falls rapidly in diastole as blood leaks back into the LV and runs off to the periphery (low diastolic pressure).

Q35 Name the peripheral signs of AR.

Collapsing pulse, Corrigan's (carotid), de Musset's (head nodding), Quincke's (nail-bed capillary pulsation), Traube's (pistol shot femorals), Duroziez's (femoral to-and-fro murmur), Hill's (popliteal–brachial gradient), Müller's (uvula), Becker's (retinal arteries), Landolfi's (pupil), Mayne's, Rosenbach's (liver), Gerhardt's (spleen), lighthouse sign.

Q36 What is the Austin Flint murmur?

A low-pitched mid-diastolic / presystolic rumble at the apex in severe AR, due to the regurgitant jet striking the anterior mitral leaflet and preventing its full opening (functional MS). Differentiated from organic MS by the absence of a loud S1 and an opening snap.

Q37 What is Hill's sign?

Popliteal (lower limb) systolic pressure exceeding brachial systolic pressure by >20 mmHg in AR; a difference of >40 indicates moderate and >60 mmHg severe AR. It is due to summation of reflected pressure waves in a high-stroke-volume state.

Q38 Where is the AR murmur best heard and why does the site matter?

Left 3rd intercostal space (Erb's area) with the patient leaning forward in held expiration. A murmur louder at the right sternal border suggests aortic root dilatation (Marfan, dissection, syphilis) rather than primary valve disease.

Q39 When is surgery indicated in chronic AR?

Symptomatic severe AR regardless of LV function; asymptomatic severe AR with LVEF ≤55 % or LVESD >50 mm (or indexed >25 mm/m²); consider with progressive LV dilatation; also when other cardiac surgery is planned.

Q40 Why are the peripheral signs absent in acute AR?

The non-dilated, non-compliant LV cannot accommodate the sudden regurgitant volume; LV diastolic pressure rises rapidly to equal aortic diastolic pressure, so pulse pressure does not widen and the murmur is short. Patients present with pulmonary oedema and shock.

Q41 What is the Graham Steell murmur?

An early diastolic murmur of pulmonary regurgitation due to severe pulmonary hypertension (classically in MS), heard at the left 2nd ICS, louder on inspiration, with loud P2 and RV heave but without peripheral signs of AR.

Q42 What is a bisferiens pulse?

A pulse with two systolic peaks — seen in combined AS and AR (AR dominant) and in HOCM.

Q43 What are the revised Jones criteria for a high-risk population like India?

Major: carditis (clinical or subclinical), polyarthritis / monoarthritis / polyarthralgia, chorea, erythema marginatum, subcutaneous nodules. Minor: monoarthralgia, fever ≥38 °C, ESR ≥30 and/or CRP ≥3 mg/dL, prolonged PR interval. With evidence of preceding streptococcal infection — 2 major or 1 major + 2 minor.

Q44 What is the regimen and duration of secondary prophylaxis in RHD?

Benzathine penicillin G 1.2 million units deep IM every 3 weeks (21 days). With residual valve disease — at least 10 years after the last episode or until 40 years of age, whichever is longer; sometimes lifelong.

Q45 Which valve lesions are the most dangerous in pregnancy?

Severe mitral stenosis and severe aortic stenosis (fixed obstruction cannot accommodate the increased cardiac output and heart rate), along with pulmonary hypertension and mechanical valves (thrombosis risk). Regurgitant lesions are usually well tolerated because of reduced systemic vascular resistance.

Last-minute checklist before you go to the examiner

✔ NYHA class · ✔ rheumatic fever & penicillin prophylaxis · ✔ angina / syncope / haemoptysis · ✔ embolic events · ✔ pulse — rate, rhythm, volume, character, both radials, radio-femoral · ✔ BP both arms, pulse pressure · ✔ JVP · ✔ apex — site and character · ✔ thrill · ✔ S1, S2 (A2/P2), added sounds · ✔ murmur — timing, site, radiation, grade, manoeuvre · ✔ signs of severity · ✔ PAH and heart failure · ✔ IE signs · ✔ complete diagnosis in six parts.

VISUAL ATLAS

Hear the Murmurs, See the Signs

Murmur recordings, physical signs, X-rays and ECGs — from Easy Auscultation, Practical Clinical Skills, Radiopaedia, LITFL, Healio and Wikipedia

Mitral stenosis murmur
▶ Listen
Loud S1, opening snap, rumbling MDM — audio

Mitral regurgitation murmur
▶ Listen
Pansystolic murmur at apex — audio

Aortic stenosis murmur
▶ Listen
Crescendo–decrescendo ESM — audio

Aortic regurgitation murmur
▶ Listen
Early diastolic decrescendo — audio

Diastolic murmurs course
▶ Listen
Compare MS and AR murmurs — audio

Mitral facies
▶ Photos
Malar flush of MS

Collapsing pulse
▶ Video
Water-hammer pulse of AR

Corrigan's pulse
▶ Article
Visible carotid pulsation

Peripheral signs of AR
▶ Article
de Musset, Quincke, Duroziez, Hill…

AS — physical signs
▶ Article
Slow-rising pulse, soft A2, thrill

P mitrale (LA enlargement)
▶ ECG
Bifid P in II, biphasic P in V1

LVH with strain
▶ ECG
Voltage criteria, ST–T strain

Atrial fibrillation
▶ ECG
Irregularly irregular, no P waves

MS chest X-ray
▶ X-ray
Left atrial enlargement

MR imaging case
▶ X-ray
Radiopaedia case

AR imaging case
▶ X-ray
Radiopaedia case

AS chest X-ray
▶ X-ray
Post-stenotic dilatation, calcified valve

Aortic incompetence
▶ X-ray
Radiopaedia article

MR on colour Doppler echo
▶ Echo
Regurgitant jet into LA

AS on echocardiography
▶ Echo
Calcified valve, high gradient

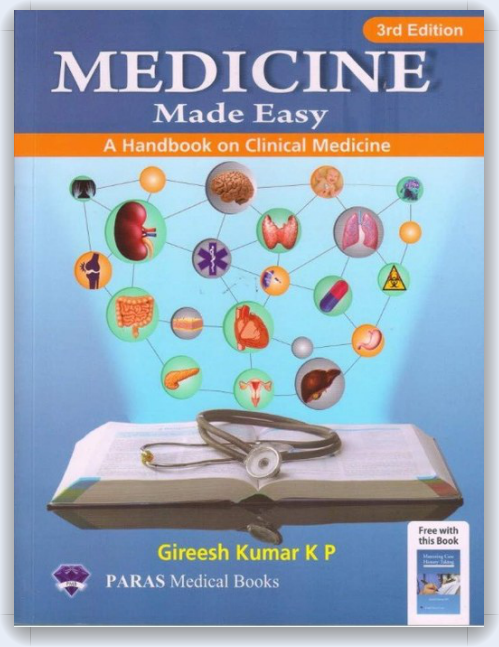
AR on colour Doppler echo
▶ Echo
Diastolic jet into LVOT

Quincke (eponym)
▶ Article
Capillary pulsation in nail bed

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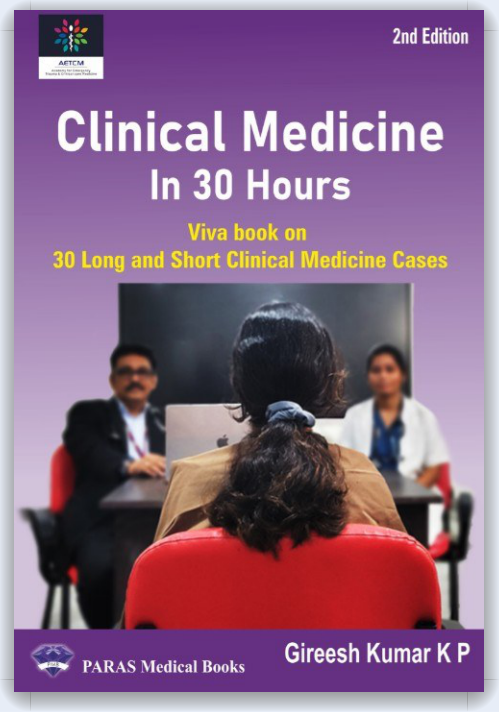
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