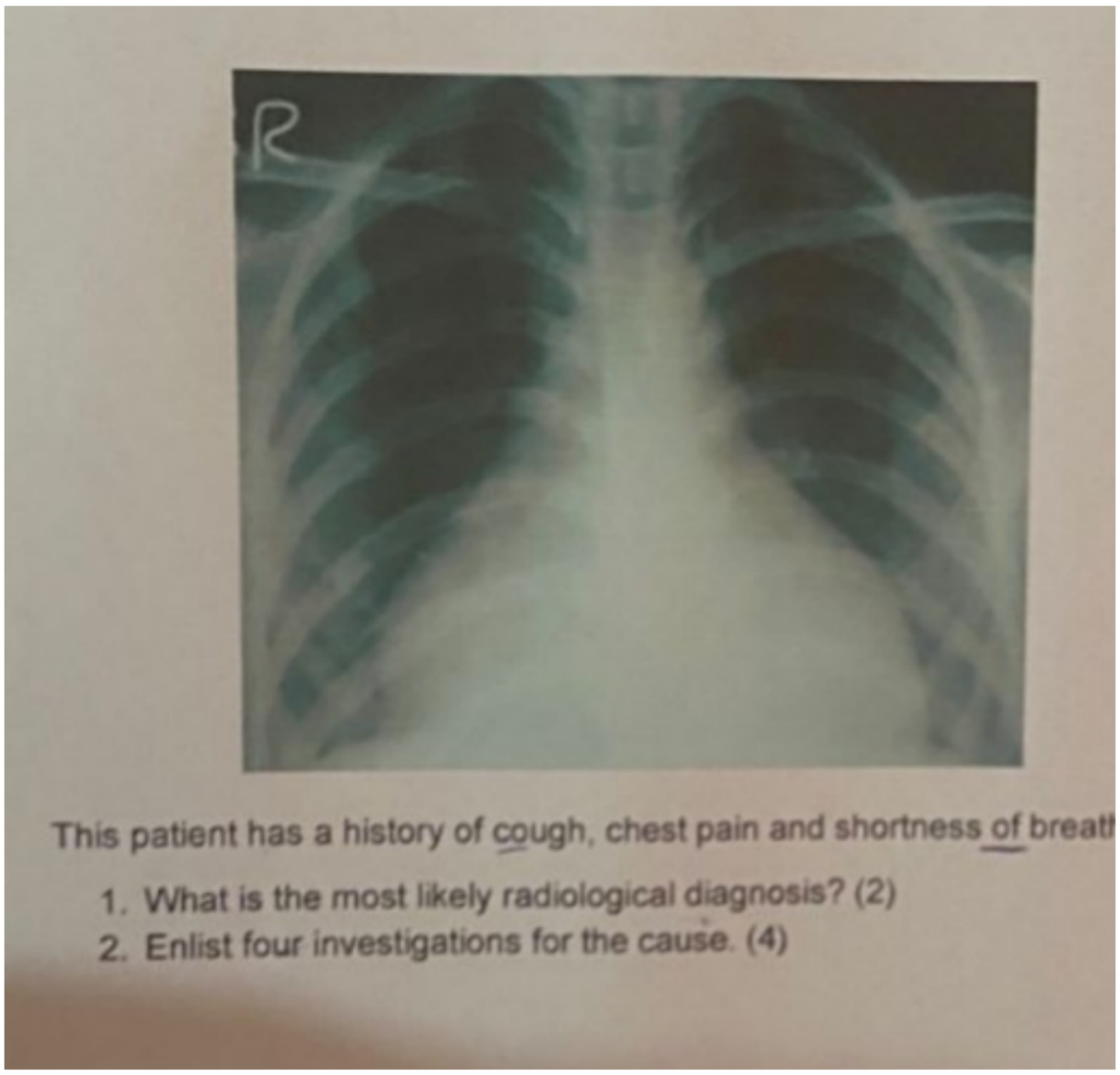
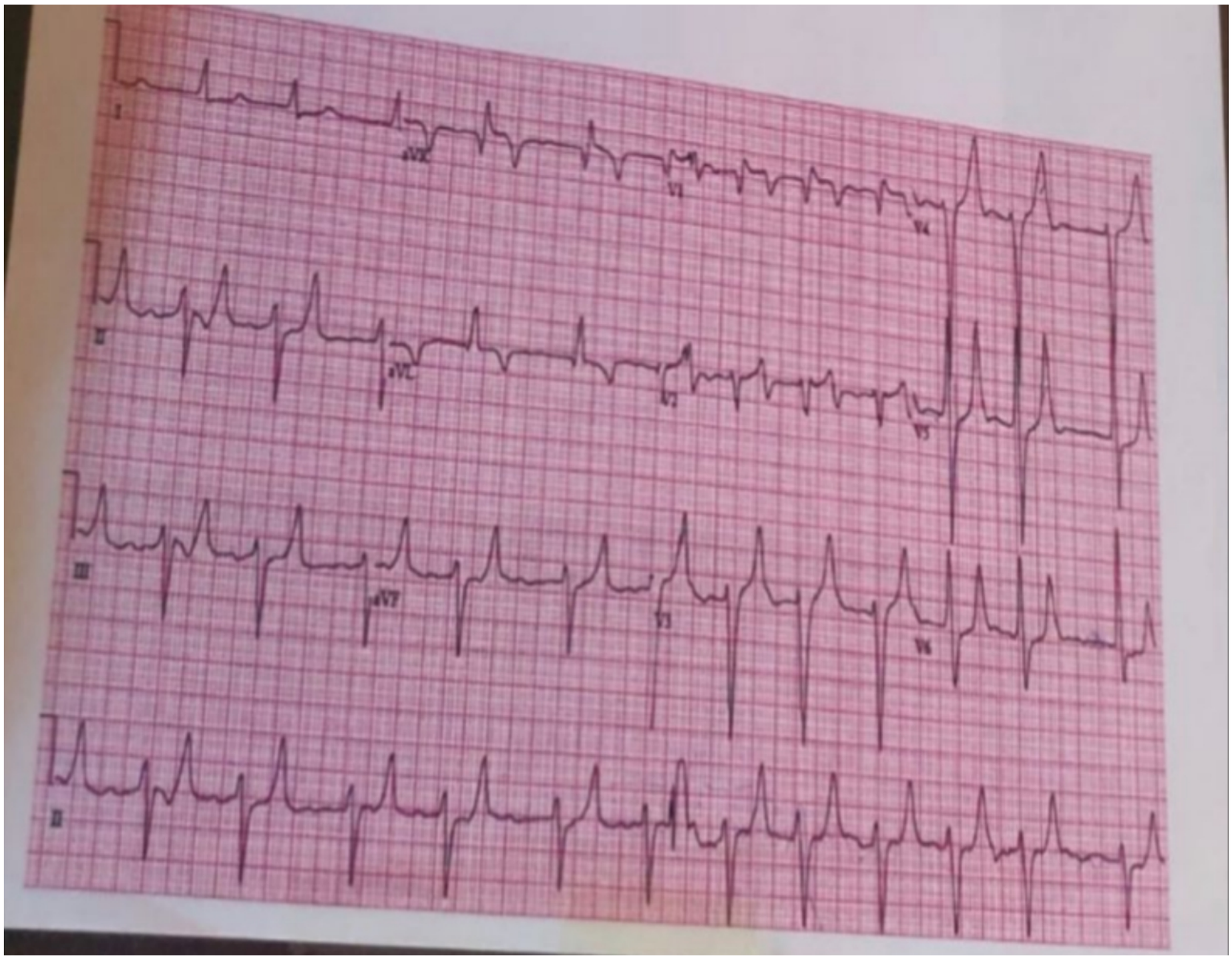
Table 7: OSCE station distribution of different subjects

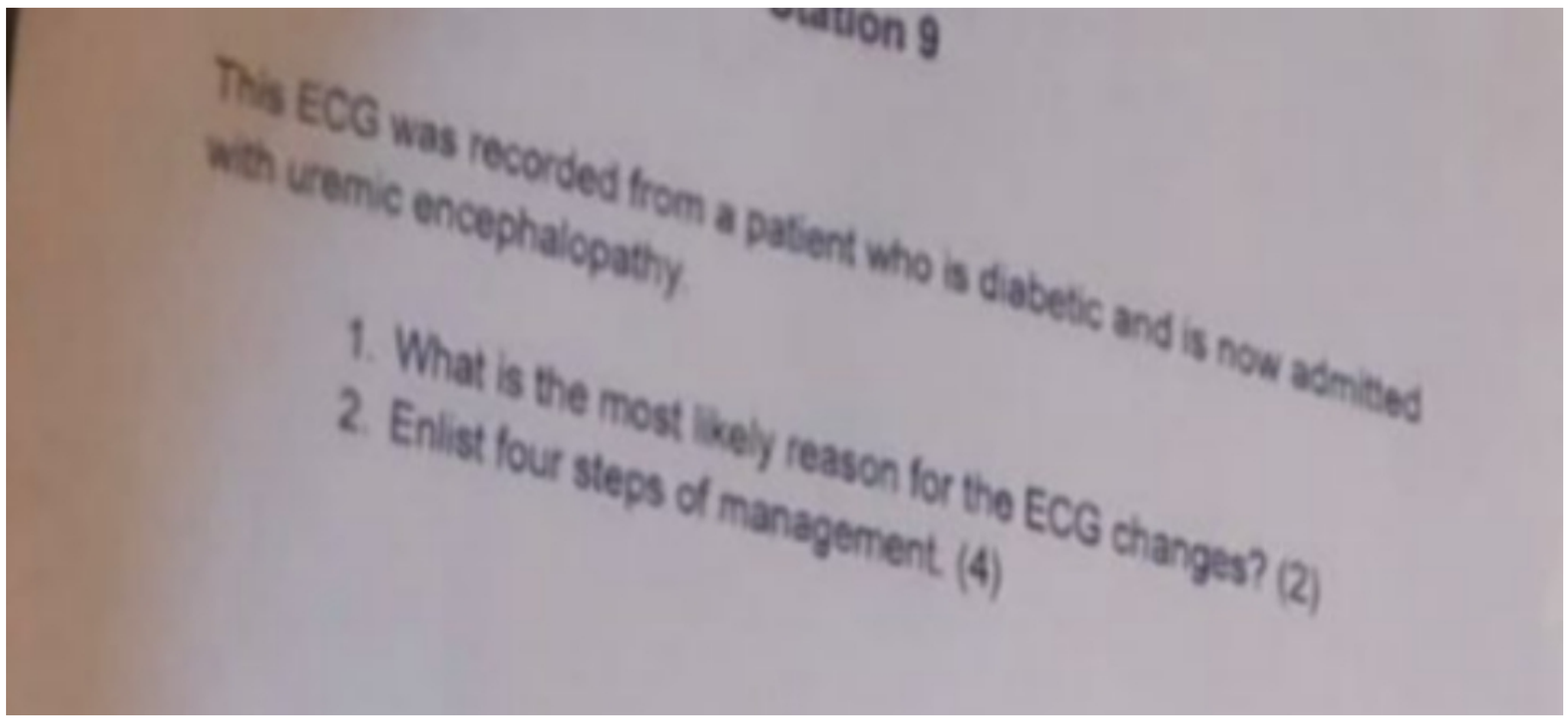
BLOCK-O (TOTAL STATIONS=20 and 6 marks/station)					
Subjects	OSCE stations		Viva stations	Logbook and history books (1-station)	Structured Long case =30 marks)
	Static/ interactive	Short cases			
Medicine	2	1	1	General medicine and allied	General Medicine
Cardiology	3	1	1		
Paediatrics	2	2	1		
Pulmonology	3	1	1		
Total	10	5	4	1	1



Most likely radiological diagnosis
Pericardial effusion (water-bottle / globular cardiomegaly) - enlarged, smooth cardiac silhouette with relatively clear lung fields.

2) Enlist 4 investigations for the cause
1. Echocardiography (confirm effusion + size + tamponade features)
2. ECG (low voltage +t electrical alternans)
3. Pericardiocentesis + pericardial fluid analysis (cell count, protein/LDH Gram stain & culture, AFB/ADA/GeneXpert, cytology for malignancy)
4. Etiology screen (any ONE set as your 4th):
TB workup: sputum AFB/GeneXpert + Mantoux/IGRA t CT chest
or
Renal function tests (urea/creatinine) for uremic pericarditis
or
Thyroid function tests (TSH) for hypothyroidism
or
ANA for connective tissue disear





Most likely reason for ECG changes (2 marks)
Severe hyperkalemia secondary to renal failure (uremia).

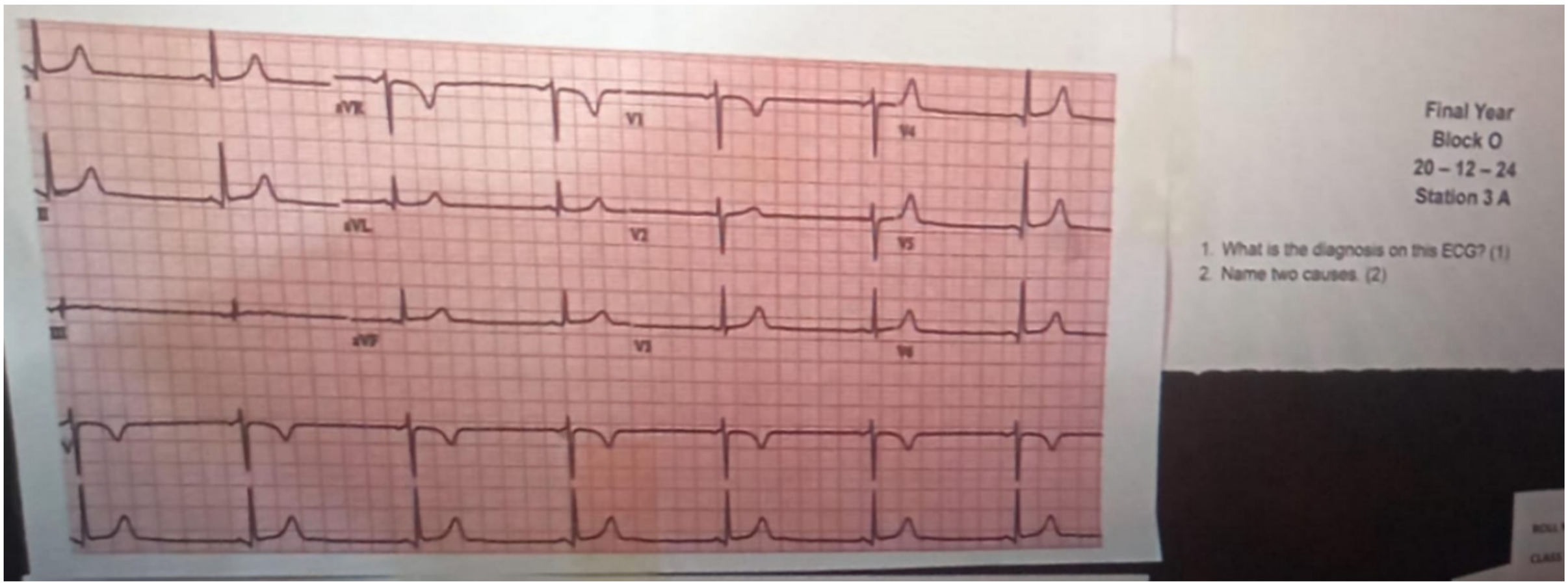
Four steps of management (4 marks)

Stabilize myocardium
• IV Calcium gluconate 10% (10 ml over 2-5 min)

Shift potassium into cells
IV Insulin (10 units regular) + 25 g glucose
+ Nebulized salbutamol
t IV sodium bicarbonate (if metabolic acidosis)

Remove potassium from body
Hemodialvsis (best in uremic patient)
+ Loop diuretics (if urine output present)
+ Potassium binders

Continuous cardiac monitoring



Hyperkalemia

Rate = 300/7 = 43
↳ Bradycardia

Tall T waves esp in v2 to v6

📈 ECG Changes in Hyperkalemia (Progression)

1️⃣ Tall peaked T waves

2️⃣ Flattened / absent P waves

3️⃣ Prolonged PR interval

4️⃣ Widened QRS

5️⃣ Sine wave pattern → Ventricular fibrillation → Asystole

🩸 Two Causes (OSPE Answer)

- Acute or chronic renal failure
- Potassium-sparing drugs (e.g., spironolactone, ACE inhibitors)

Other causes:

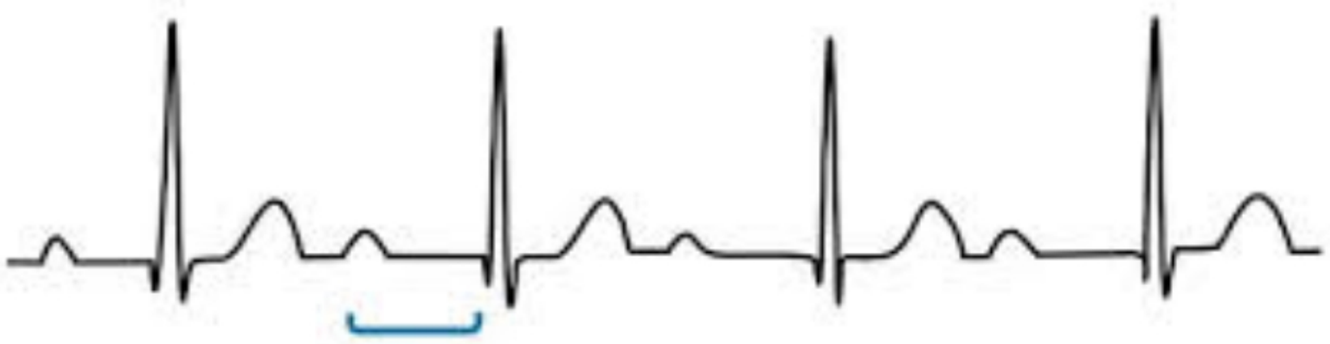
- DKA
- Massive hemolysis
- Rhabdomyolysis
- Adrenal insufficiency

🚑 Management (If They Push Further)

1. IV Calcium gluconate (stabilizes myocardium)
2. Insulin + glucose
3. Nebulized salbutamol
4. Sodium bicarbonate (if acidotic)
5. Dialysis (definitive)

HEART BLOCKS

First Degree



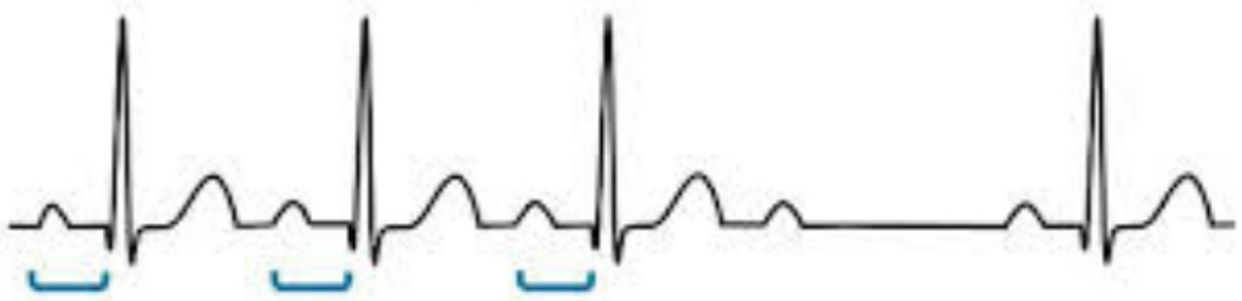
How to Identify

- Prolonged PR interval (>200ms)

Second Degree, Mobitz Type 1

- PR interval increases until a QRS is dropped

Second Degree, Mobitz Type 2

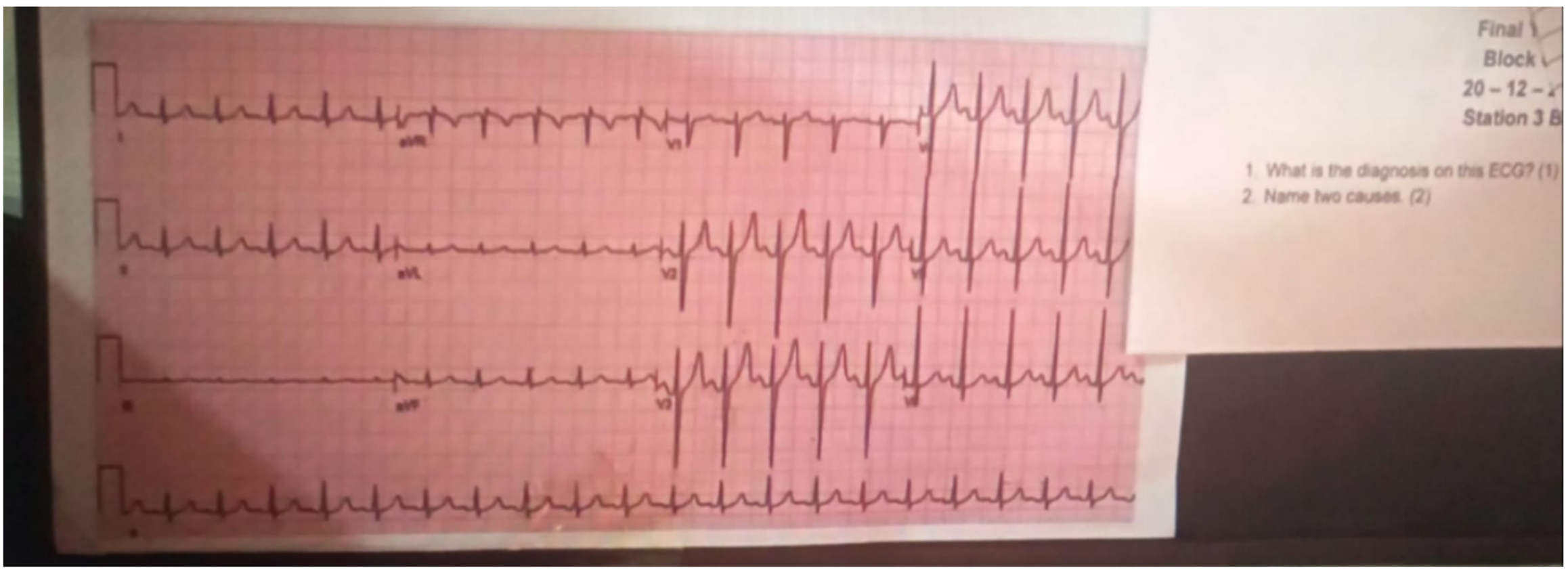


- Constant PR interval before a QRS is dropped

Third Degree/Complete Heart Block

- No relation between P waves and QRS

MV



Atrial flutter

Causes
Ischemic heart disease
Rheumatic heart disease(mitral valve)

1. Diagnosis

The diagnosis is **Atrial Flutter with 2:1 conduction**.

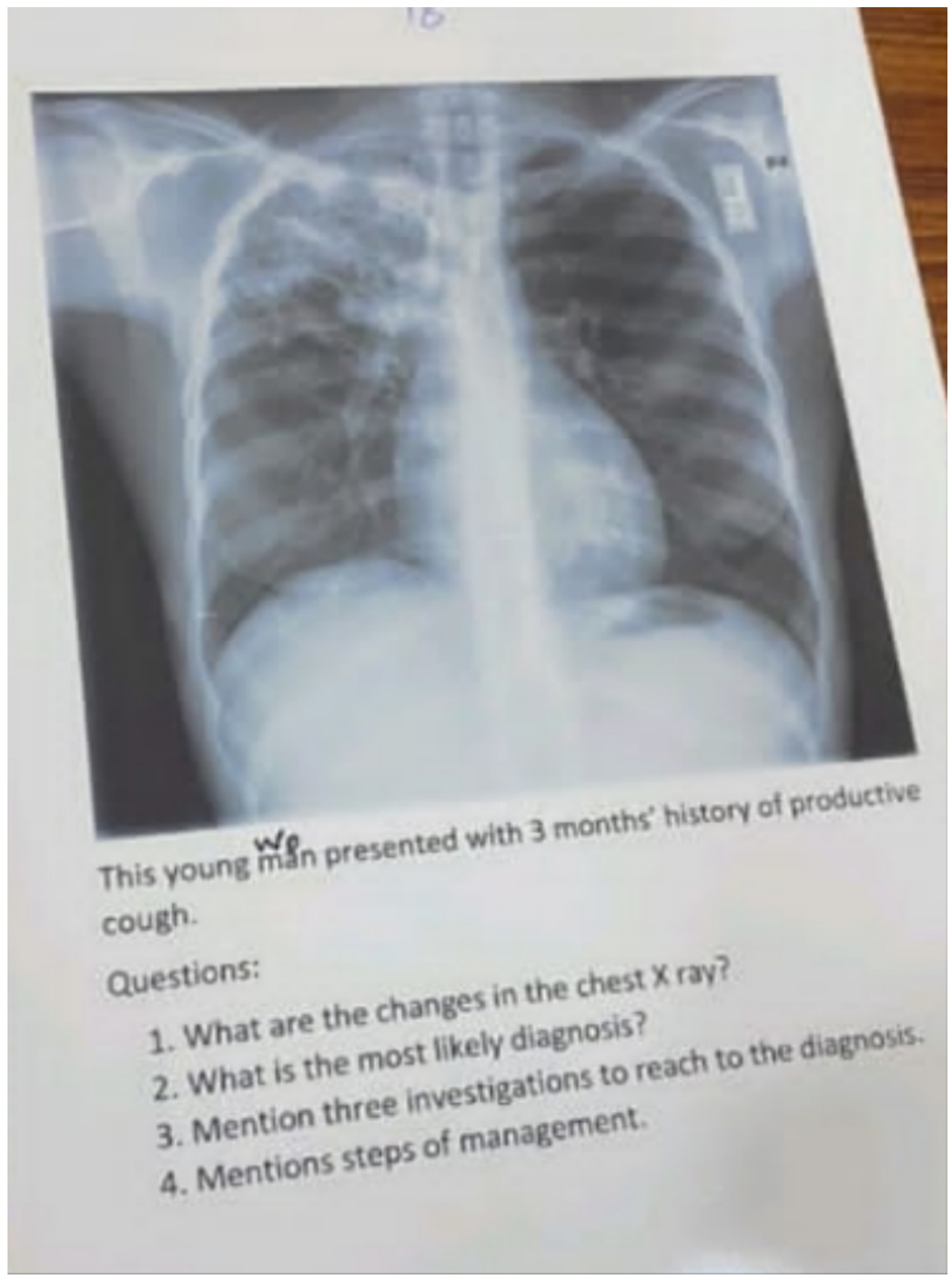
Key features visible in the image:

- **Sawtooth Pattern:** Look closely at the baseline in the inferior leads (II, III, and aVF). You can see the characteristic "sawtooth" flutter waves (F-waves).
- **Rate:** The atrial rate is typically around 300 bpm. Because of the 2:1 conduction (the AV node blocks every second impulse), the ventricular (pulse) rate is approximately **150 bpm**.
- **Regularity:** The rhythm is regular, which helps distinguish it from Atrial Fibrillation.

2. Common Causes

While several conditions can trigger this, the two most common categories are:

- **Structural Heart Disease:** Such as Ischemic Heart Disease (post-MI), Heart Failure, or Valvular Heart Disease (especially mitral valve issues).
- **Pulmonary Disease:** Chronic Obstructive Pulmonary Disease (COPD) or an acute Pulmonary Embolism.



Changes in chest X-ray
Upper lobe heterogeneous opacity
Areas of cavitation
Surrounding infiltrates/ patchy consolidation
Possible fibrosis in upper zone
This is classic upper lobe cavitary lesion

Most likely diagnosis
Pulmonary Tuberculosis (Post-primary TB)

Why?
Young patient
Chronic productive cough (>3 weeks)
Upper lobe cavitation

3 Three investigations
1. Sputum AFB smear (x3 samples)
2. GeneXpert (CBNAAT)
3. Sputum culture for Mycobacterium tuberculosis
(Alternative acceptable: CT chest, ESR_ Mantoux)

Steps of management
Standard Anti-TB therapy (Category I)

Intensive phase (2 months):
Isoniazid
Rifampicin
Pyrazinamide
Ethambutol

Continuation phase (4 months):
Isoniazid
Rifampicin
Total = 6 months
Plus:
Notify case
Screen contacts
Nutritional support

🫁 Difference in X-ray Findings: TB vs Pulmonary Edema

Feature	Pulmonary TB	Pulmonary Edema
Cause	Infection by Mycobacterium tuberculosis	Usually due to left heart failure (e.g., Congestive heart failure)
Distribution	Upper lobe predominance (apical/posterior segments)	Bilateral perihilar (“central”) distribution
Pattern	Patchy infiltrates, nodules	Fluffy, homogeneous opacities
Cavitation	Common (thick-walled cavity)	Absent
Lymph nodes	Hilar lymphadenopathy (esp. primary TB)	Not typical
Pleural effusion	May be unilateral	Often bilateral
Special sign	Miliary nodules (millet-seed pattern)	“Bat-wing” / “Butterfly” pattern
Heart size	Usually normal	Often cardiomegaly
Other signs	Fibrosis, volume loss	Kerley B lines, vascular redistribution

---

SVT and treatment
ECG

Tx of AVNRT

Acute Management

* Unstable Tachycardia with a pulse → Electrical cardioversion

* Stable with narrow QRS Complex
  - First Step: vagal maneuvers
  - If SVT persist → IV Medical Therapy
    - Adenosine (First line)
    - CCBs (verapamil, Diltiazim), β blockers (metoprolol)

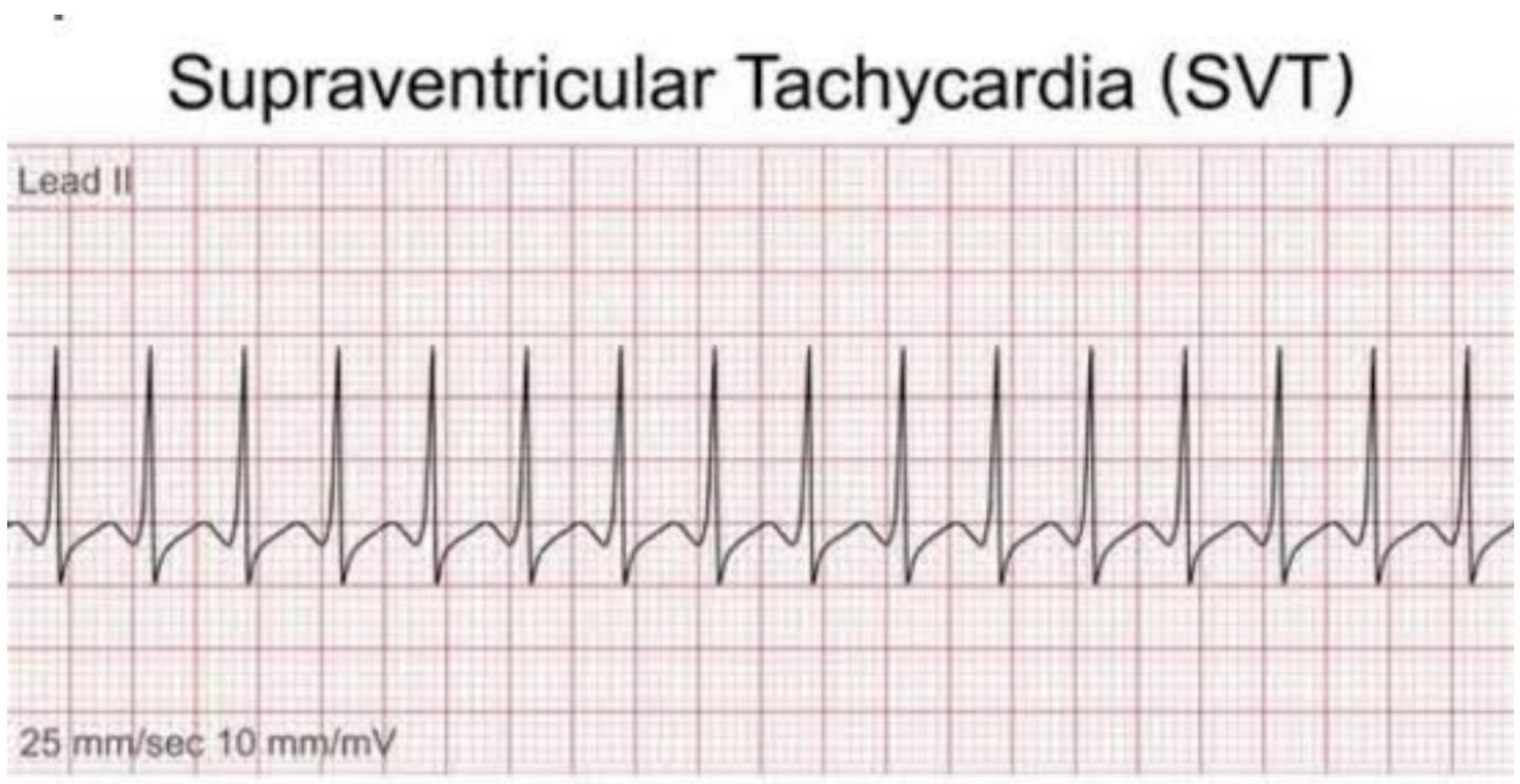
* Stable with wide QRS Complex (suspected aberrant conduction)
  - Established diagnosis of AVNRT → AV nodal blocking agents are safe
  - Uncertain Diagnosis → Avoid AV nodal blocking agents

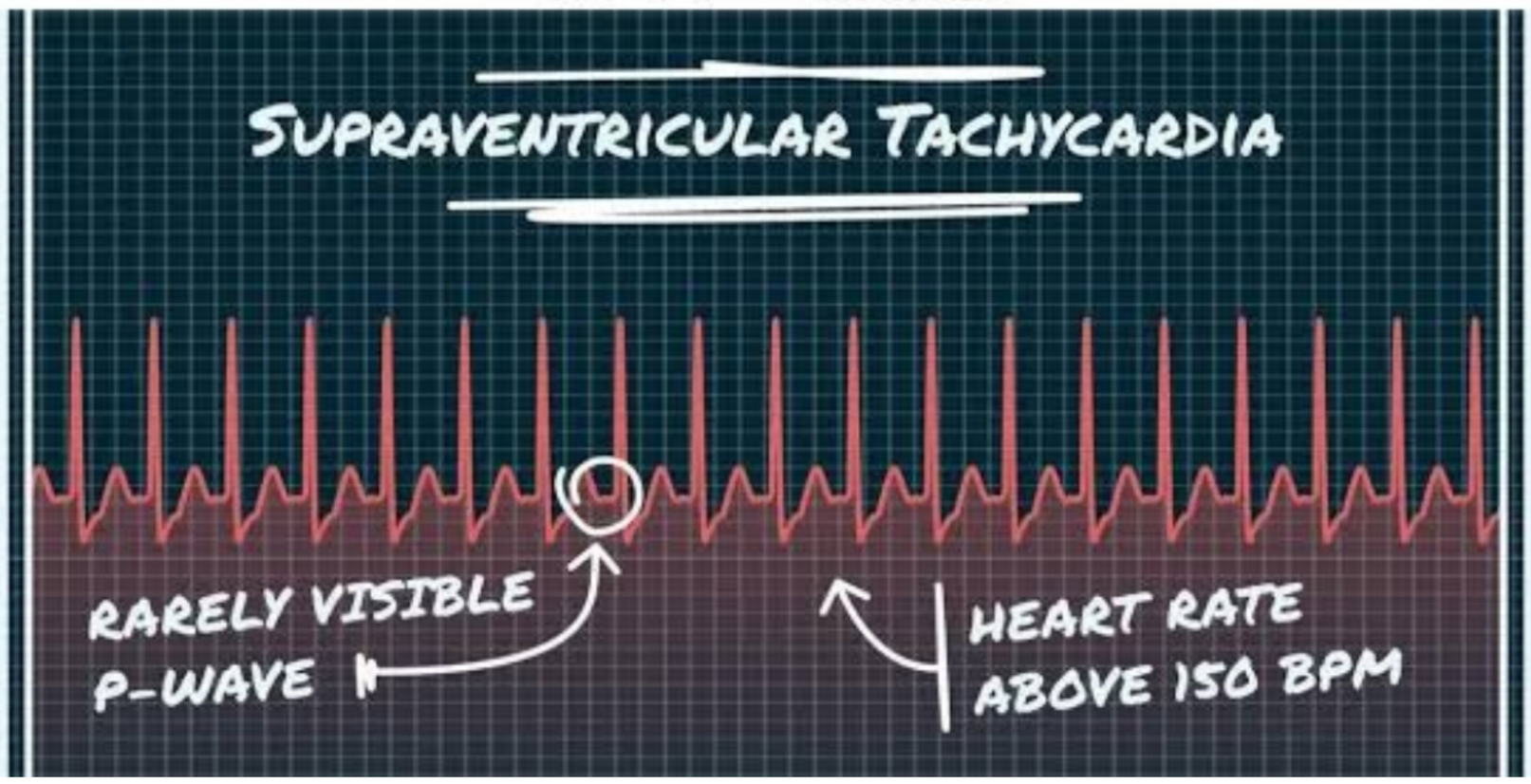
LONG TERM MANAGEMENT

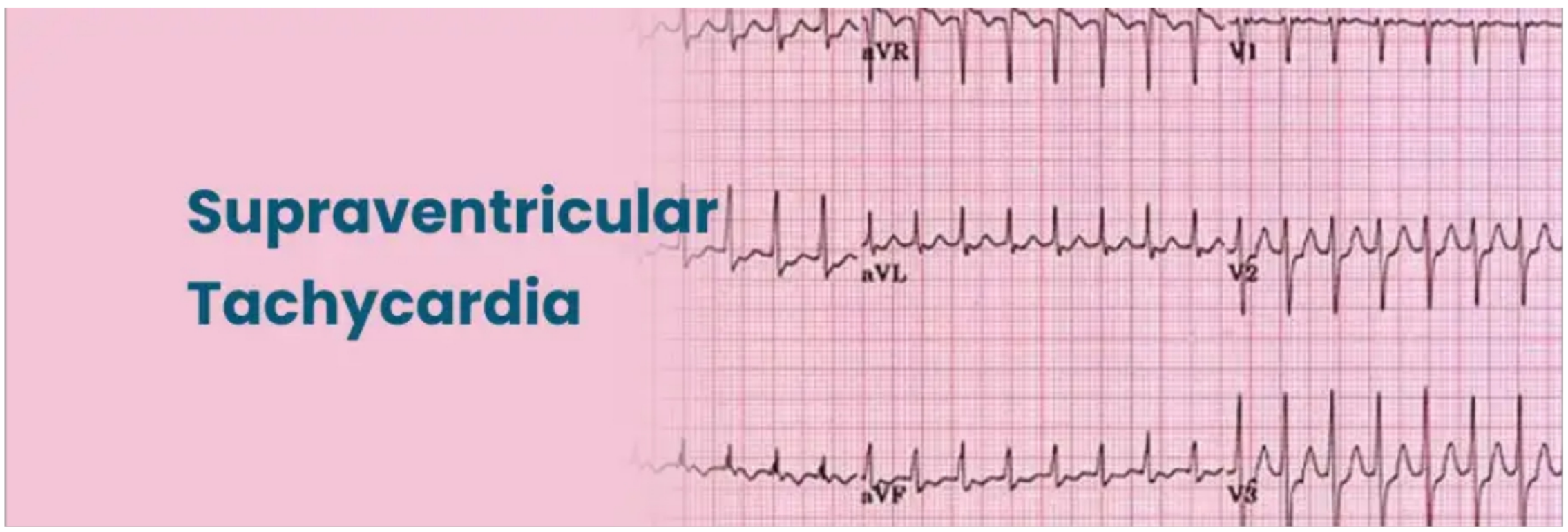
* Infrequent and mild episodes
  - managed with self-guided vagal maneuvers

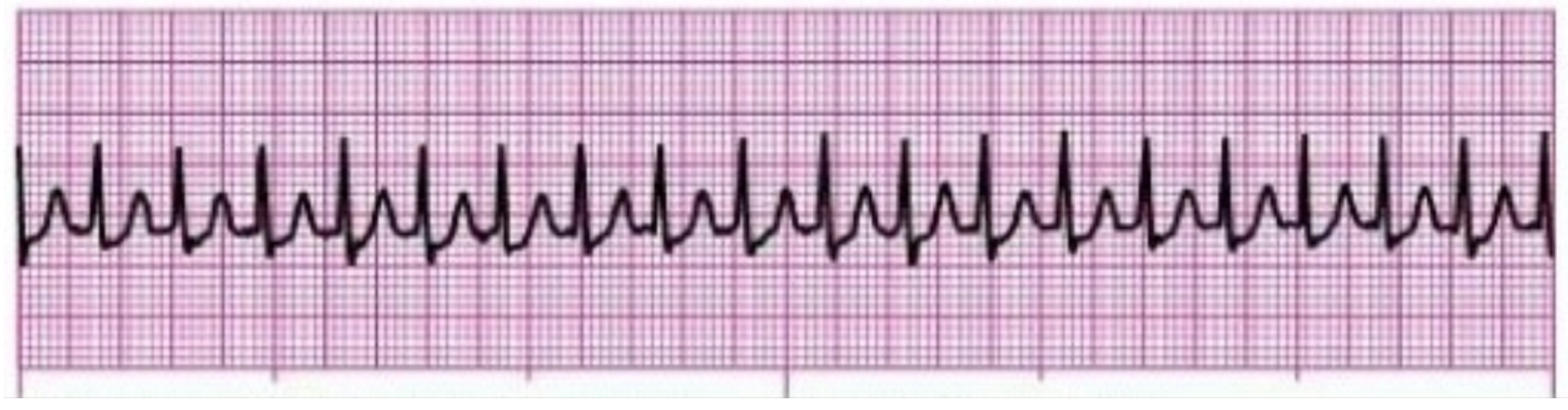
* First Line → percutaneous catheter ablation of the slow pathway

* Second Line: Pharmacological Therapy
  - β blockers eg propranolol
  - Verapamil
  - Diltiazem

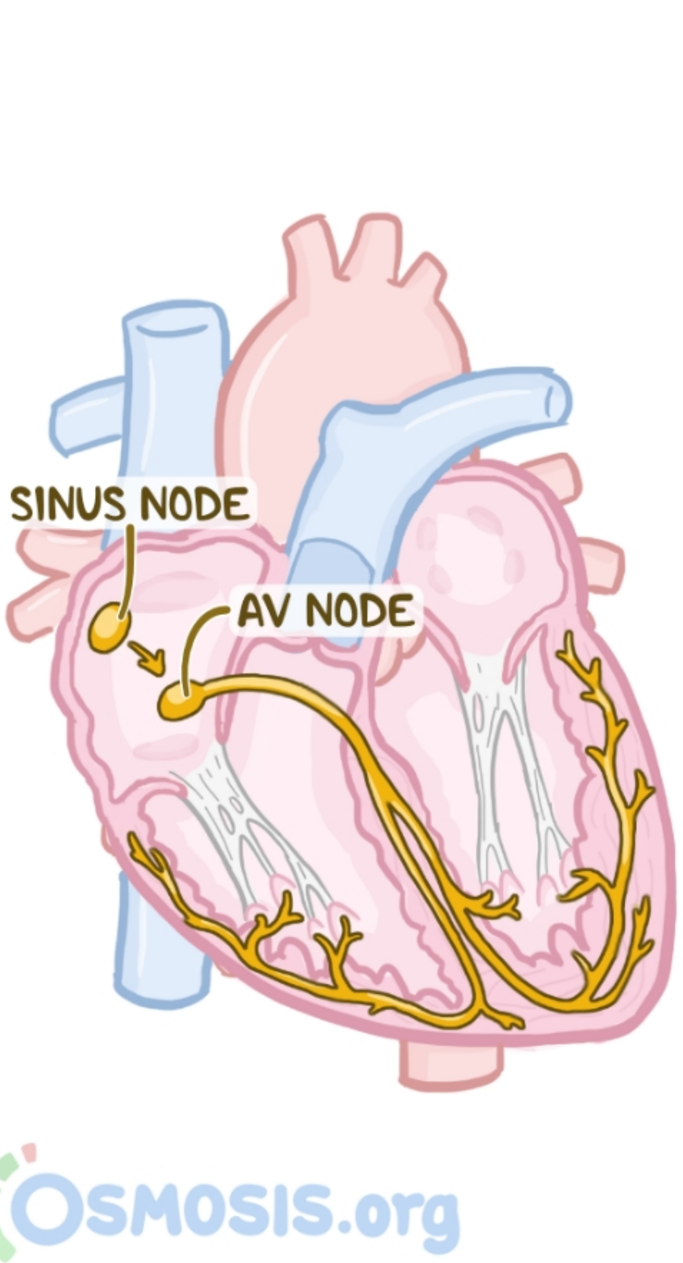

Supraventricular Tachycardia (SVT)
Lead II
25 mm/sec 10 mm/mV

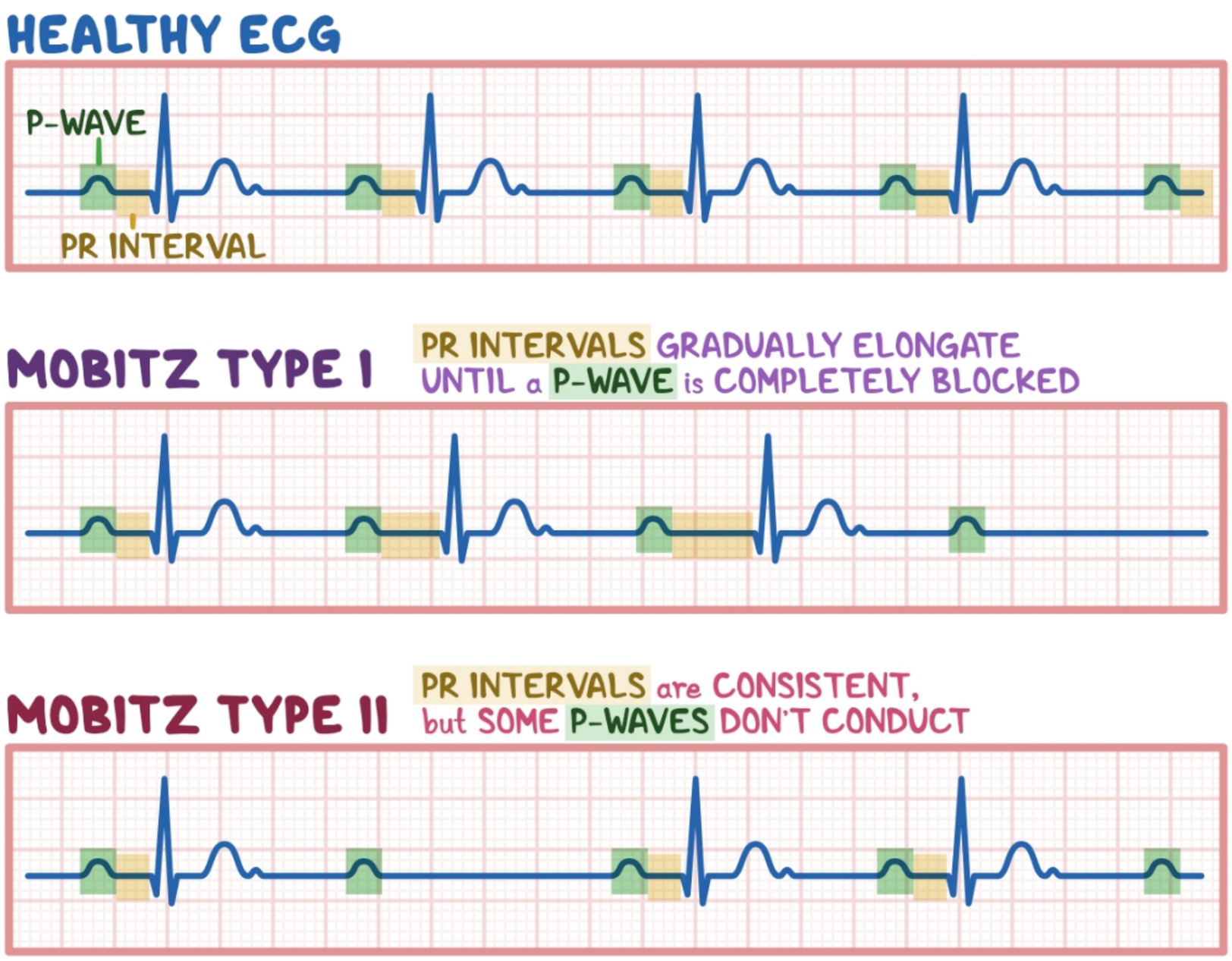

SUPRAVENTRICULAR TACHYCARDIA
RARELY VISIBLE P-WAVE
HEART RATE ABOVE 150 BPM


Supraventricular Tachycardia
aVR
V1
aVL
V2
aVF
V3



Mobitz type 2 ECG
4 conditions with this kind of ECG





Mobitz type II heart block (Second-degree AV block type II) is due to disease of the His-Purkinje system (below the AV node). It is more dangerous and often progresses to complete heart block.

Causes

Ischemic heart disease
Degenerative conduction system disease
Structural heart disease

3. Structural heart disease

- Cardiomyopathy
- Aortic stenosis
- Mitral valve disease
- Congenital heart disease

4. Inflammatory and infiltrative diseases

- Myocarditis
- Infective endocarditis
- Rheumatic carditis
- Sarcoidosis
- Amyloidosis

5. Post-cardiac procedures

- After cardiac surgery (especially valve surgery)
- After catheterization
- After septal defect repair

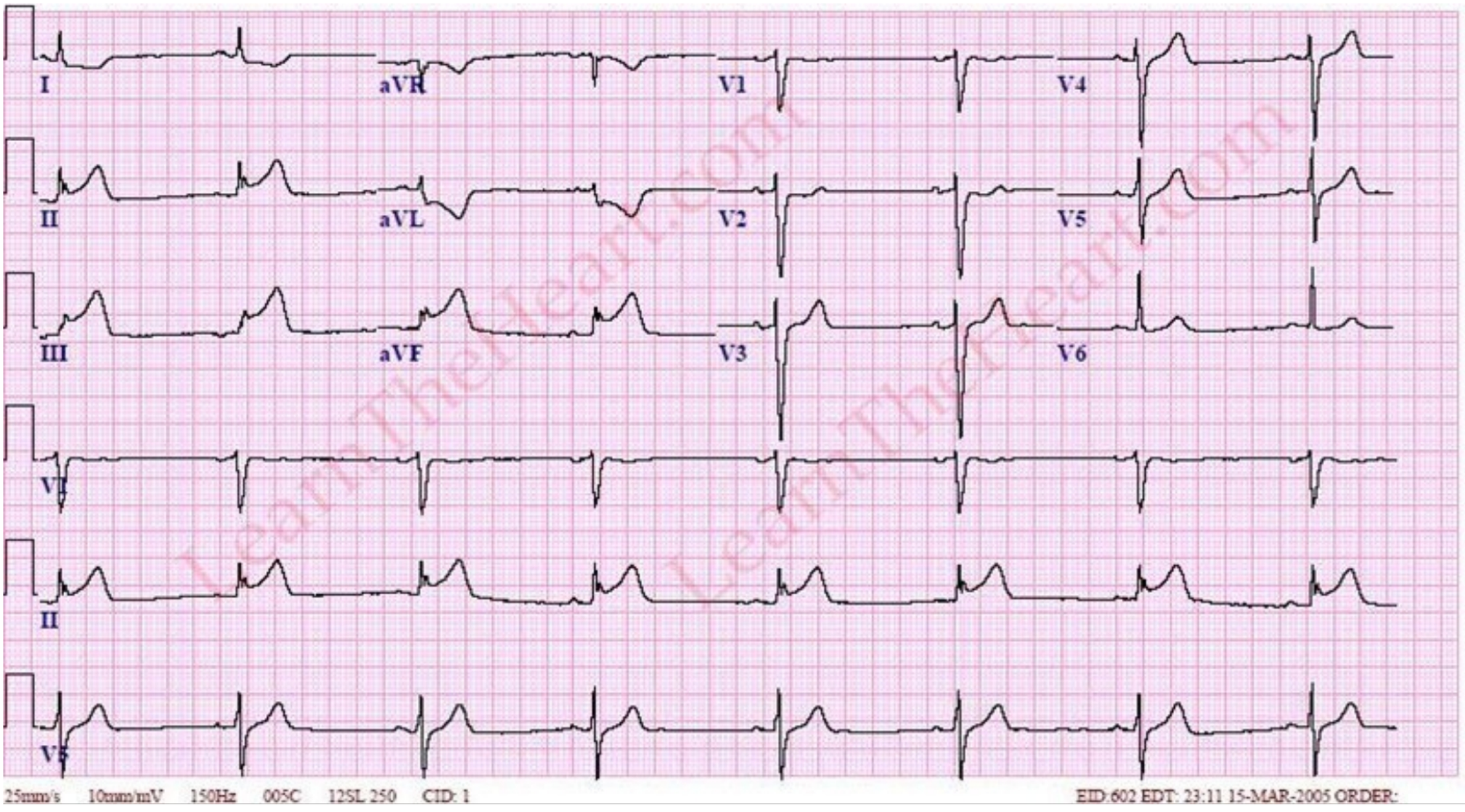
6. Drugs (less common cause compared to Mobitz I)

- Beta blockers
- Calcium channel blockers (verapamil, diltiazem)
- Digoxin
- Antiarrhythmics (amiodarone)

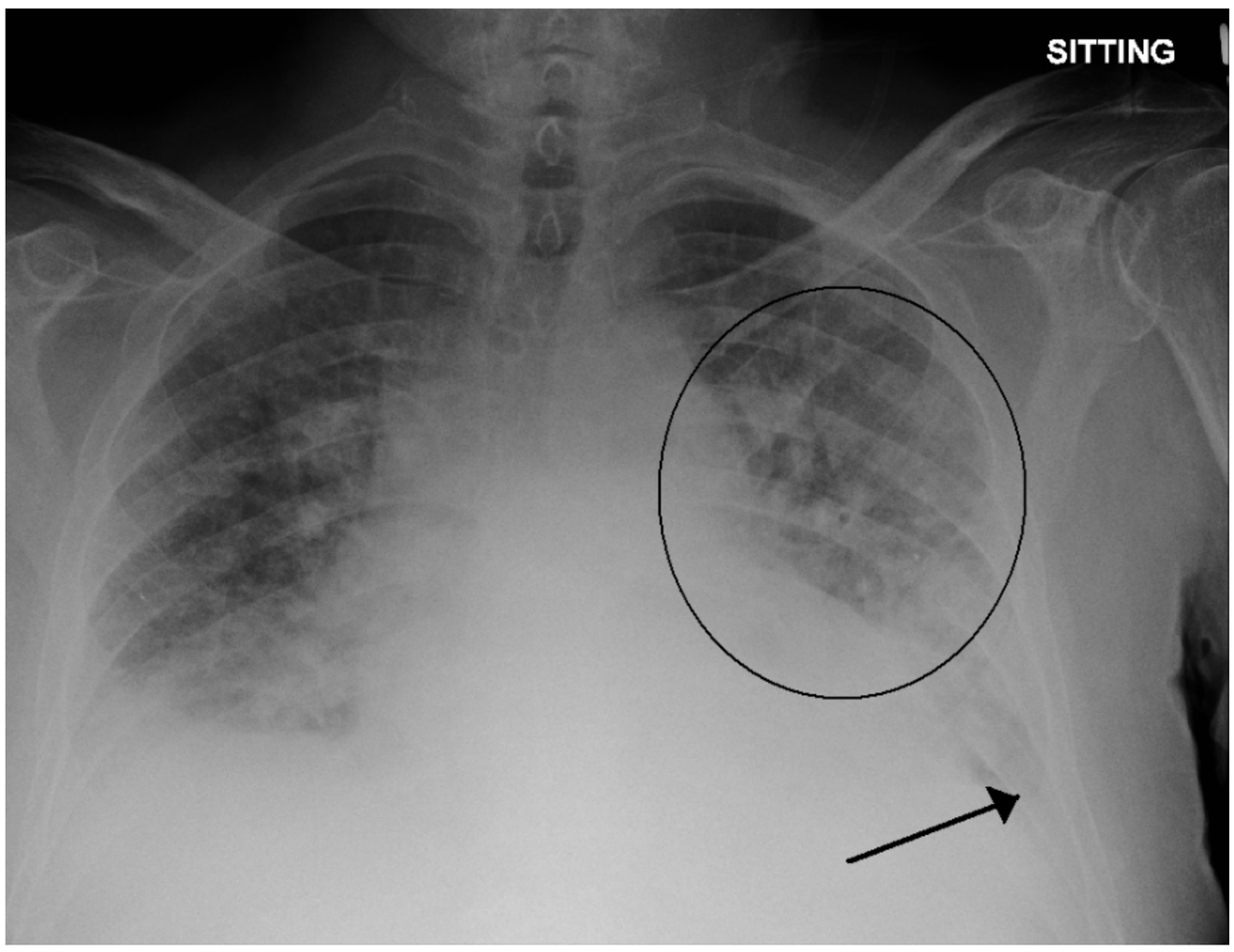
7. Electrolyte abnormalities

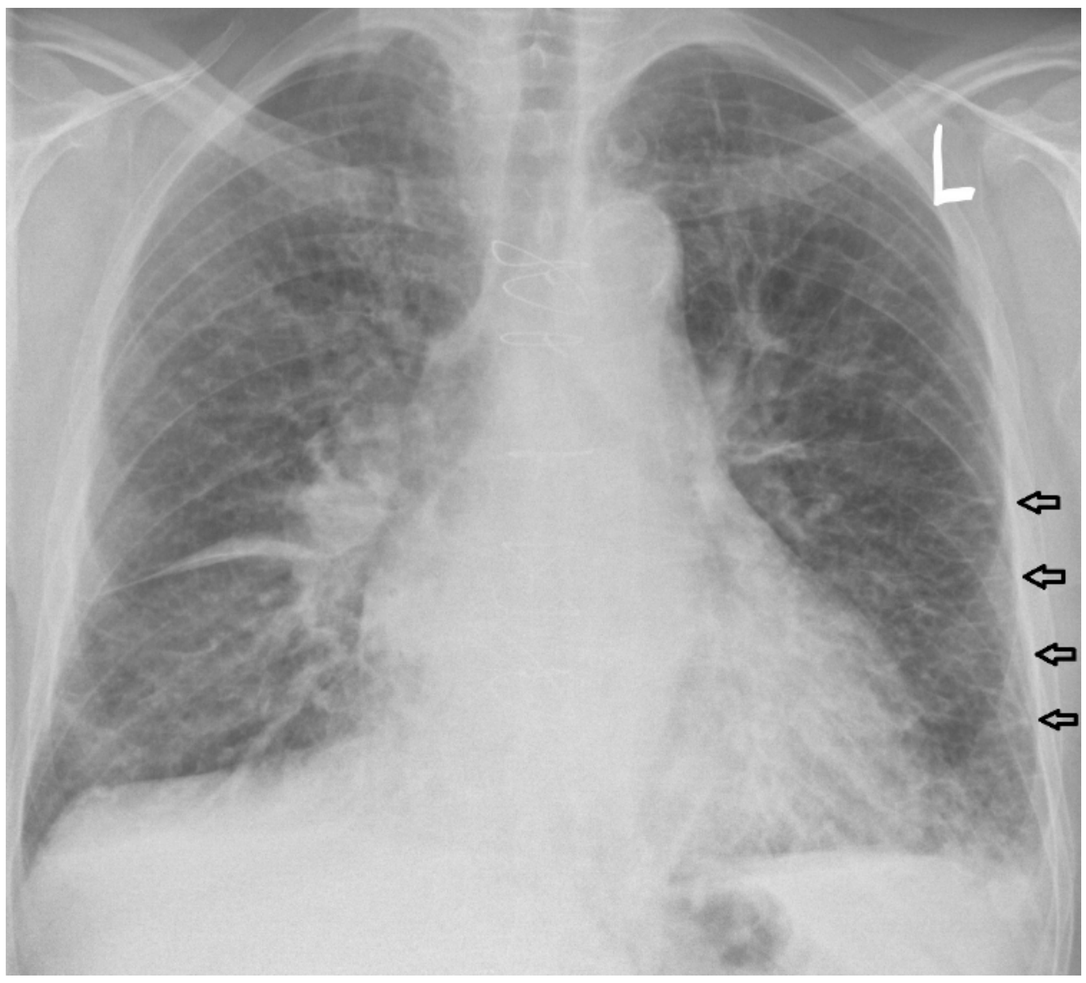
- Hyperkalemia

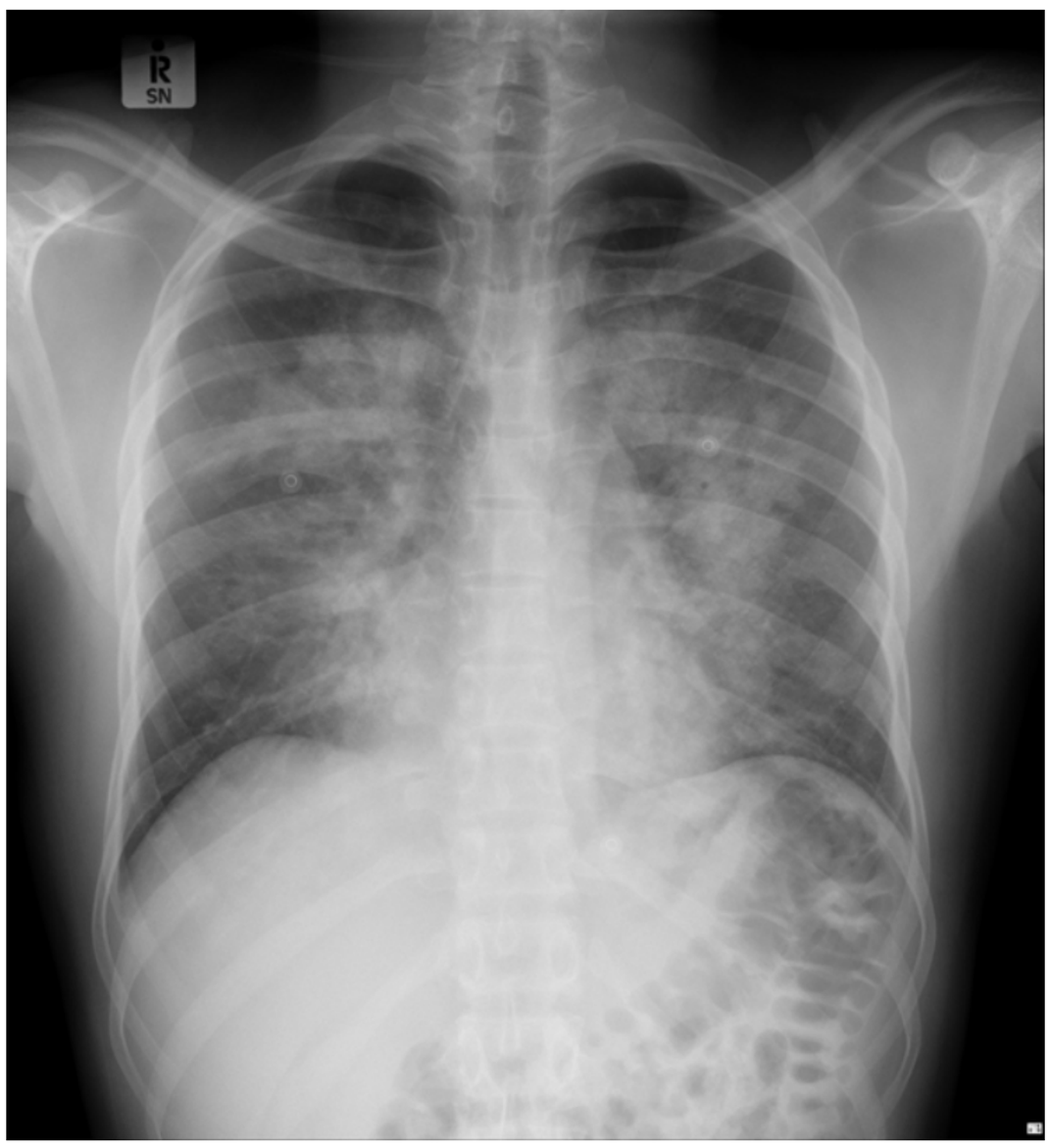
Inferior wall MI ECG and treatment



Pulmonary edema XRay and management







💊 Management

1️⃣ Immediate / Emergency

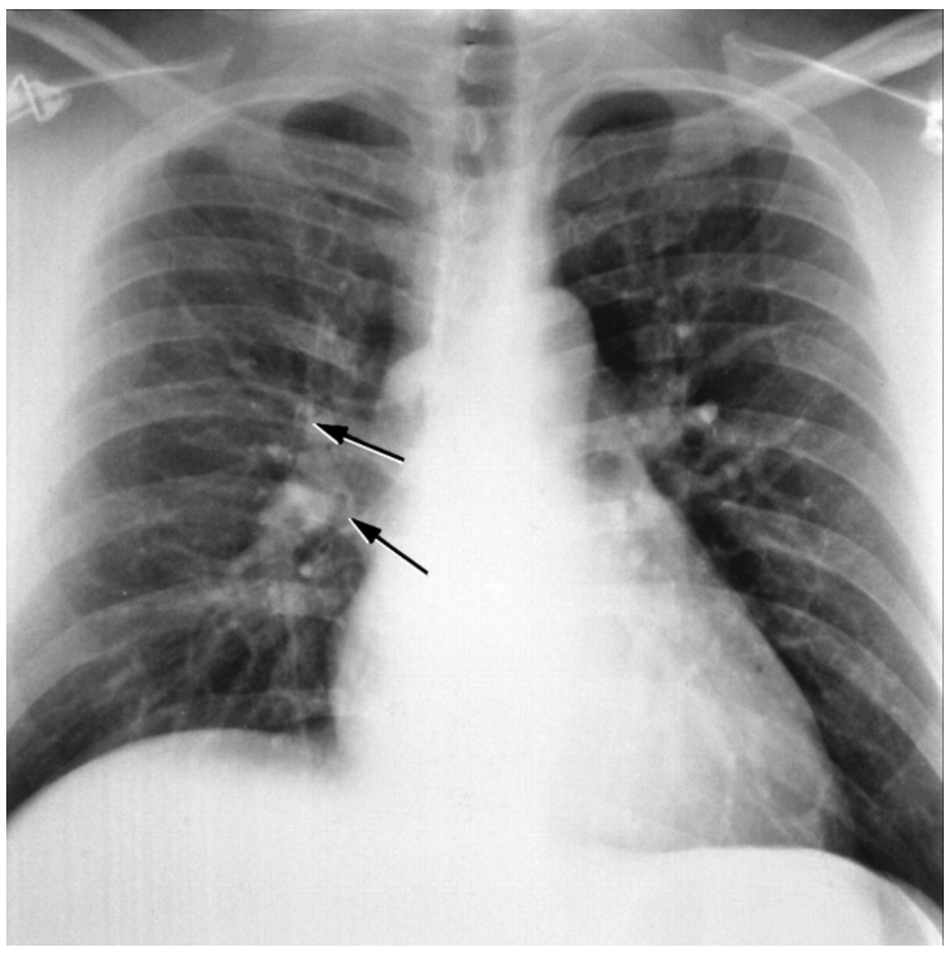
- **Oxygen therapy** – maintain SpO_2 > 90%
- **Sit upright** – reduces venous return
- **Diuretics (IV furosemide)** – reduce pulmonary congestion
- **Morphine** (optional) – for severe dyspnea and anxiety
- **Nitroglycerin** – if hypertension or acute left heart failure

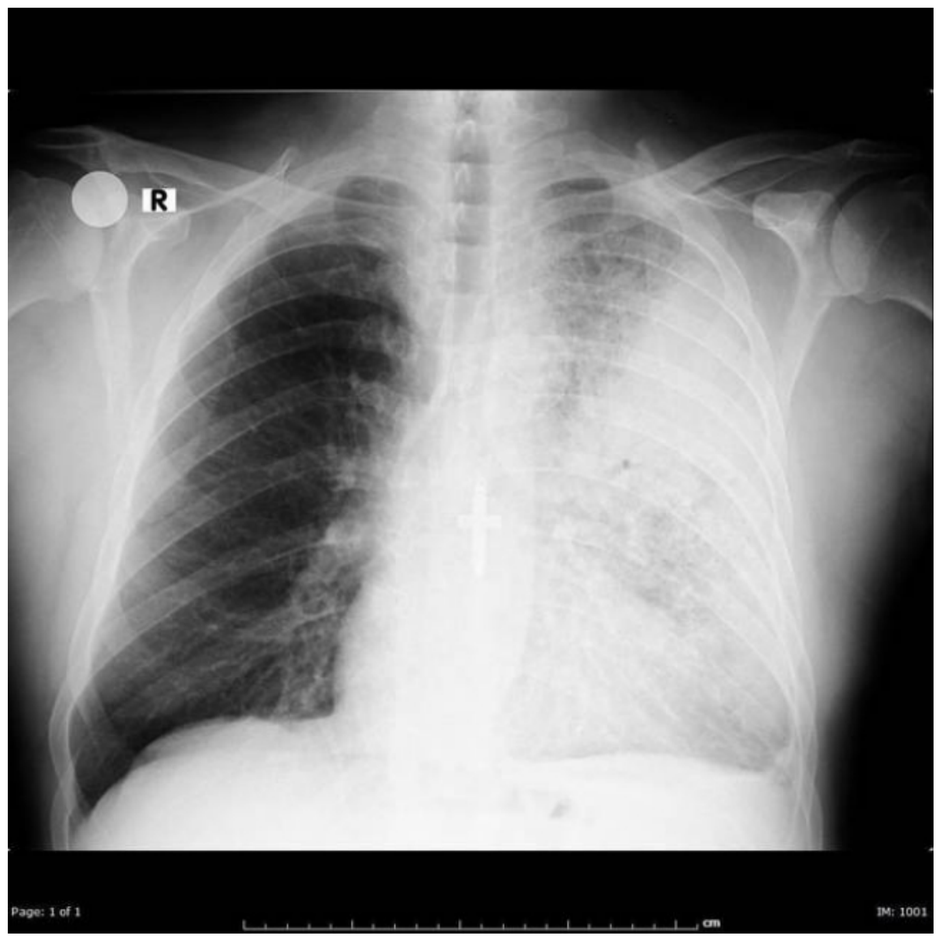
2️⃣ Treat Underlying Cause

- **Cardiogenic:** MI, heart failure → manage accordingly
- **Arrhythmias:** correct bradycardia or tachycardia
- **Non-cardiogenic:** treat sepsis, ARDS, fluid overload

3️⃣ Advanced / Critical Care

- **Non-invasive ventilation (CPAP / BiPAP)** for hypoxia
- **Intubation and mechanical ventilation** if severe
- **Inotropes / vasopressors** if cardiogenic shock





🫁 Pulmonary Edema vs Pneumonia on Chest X-ray

Feature	Pulmonary Edema	Pneumonia
Distribution	Bilateral, symmetrical	Usually unilateral or focal
Pattern	Perihilar “bat wing”	Lobar or segmental consolidation
Margins	Fluffy, ill-defined	More localized
Cardiomegaly	Usually present	Usually absent
Pleural Effusion	Common (bilateral)	May be unilateral
Kerley B lines	Present	Absent
Air bronchogram	Rare	Common
Rapid change	Improves in 24–48 hrs with diuretics	Slower resolution

🧠 High-Yield Viva Points

1️⃣ Bat wing pattern = think edema first

2️⃣ Air bronchogram strongly suggests pneumonia

3️⃣ Rapid clearing after IV furosemide = pulmonary edema

4️⃣ Cardiomegaly favors cardiogenic edema

5️⃣ BNP high → edema; Procalcitonin high → pneumonia

🔴 Pulmonary Edema (Classically Cardiogenic)

Typical CXR Findings:

- Bilateral perihilar opacities
- “Bat wing” appearance
- Enlarged cardiac shadow
- Upper lobe diversion
- Kerley B lines
- Bilateral pleural effusion

Common cause:

- **Acute decompensated heart failure**

Clinical Clues:

- Orthopnea
- PND
- Raised JVP
- Pedal edema
- S3 gallop

🔵 Pneumonia

Typical CXR Findings:

- Lobar consolidation
- Air bronchogram
- Unilateral involvement
- No cardiomegaly (usually)

Common cause:

- **Lower respiratory tract infection**

Clinical Clues:

- Fever
- Productive cough
- Pleuritic chest pain
- Localized crepitations
- Leukocytosis

STATION 7

A 23 year old girl with chronic productive cough her sister also had the same disease and died

1) Cystic fibrosis leading to bronchiectasis
2) Defect ?
3) First line investigation
4) Management

23-year-old girl with chronic productive cough
Positive family history (sister died of similar illness)
1️⃣ Most Probable Diagnosis
👉 Cystic Fibrosis (CF) leading to Bronchiectasis
Reasoning:
Young patient
Chronic productive cough
Recurrent chest infections
Positive family history (autosomal recessive disease)

2️⃣ What is the Defect?
🔬 Mutation in CFTR gene (Chromosome 7)
Defective CFTR chloride channel

3️⃣ First-Line Investigation
✅ Sweat Chloride Test (Gold Standard Screening Test)
Chloride > 60 mmol/L → diagnostic
Other investigations:
Genetic testing (CFTR mutation analysis)
HRCT chest → shows bronchiectasis
Sputum culture (often Pseudomonas)
4️⃣ Management
🫁 A. Respiratory Management
Chest physiotherapy (airway clearance)
Nebulized hypertonic saline
Bronchodilators
Inhaled antibiotics (e.g., Tobramycin for Pseudomonas)
Long-term antibiotics for recurrent infections
🍽️ B. Nutritional Support
High-calorie, high-protein diet
Pancreatic enzyme replacement
Fat-soluble vitamins (A, D, E, K)
💊 C. CFTR Modulator Therapy (if mutation eligible)
Ivacaftor
Lumacaftor/Ivacaftor combination
🏥 D. Advanced Disease
Oxygen therapy
Lung transplantation (end-stage)

STATION 12

50 YEAR OLD MAN HAVING PRODUCTIVE COUGH AND BILATERAL WHEEZE SMOKER FROM 15 YEARS SATURATION IS 93 BP AND PULSE NORMAL ,BY BRONCHODILATOR FEV1 IS IMPROVEMENT IS LESS THAN 15 %

1) DIAGNOSIS
2) HOW ASTHMA IS DIFFERENTIATED FROM COPD
3) INDICATIONS FOR INVASIVE MECHANICAL VENTILATION IN THIS PATIENT
4) MANAGEMENT

50-year-old man
Chronic productive cough
Bilateral wheeze
Smoker (15 years)
SpO_2 = 93%
FEV_1 improvement <15% after bronchodilator
1️⃣ Diagnosis
👉 Chronic Obstructive Pulmonary Disease (COPD)
Reasoning:
Age >40
Smoking history
Chronic productive cough
Bilateral wheeze
Poor reversibility (<12–15%) on bronchodilator
This suggests fixed airflow limitation, typical of COPD.

Indications for Invasive Mechanical Ventilation in This Patient
If COPD exacerbation worsens:
🚨 Indications:
Severe respiratory distress
RR > 35/min
Hypoxia not improving with oxygen (PaO_2 < 60 mmHg)
Hypercapnia with acidosis ($PaCO_2$ > 50 mmHg + pH < 7.25)
Altered consciousness
Hemodynamic instability
Failure of non-invasive ventilation (NIV)
🔑 First try NIV (BiPAP) before invasive ventilation.

Difference btw asthma and copd
Asthma usually occurs in young patients, while COPD occurs in older smokers.
Asthma symptoms are episodic and reversible, whereas COPD is progressive and persistent.
Asthma shows significant bronchodilator reversibility (≥12–15%), but COPD shows poor reversibility.
Asthma cough is usually dry, while COPD cough is chronic and productive.
Asthma is associated with allergy and eosinophilia, whereas COPD is mainly related to smoking and neutrophilic inflammation.

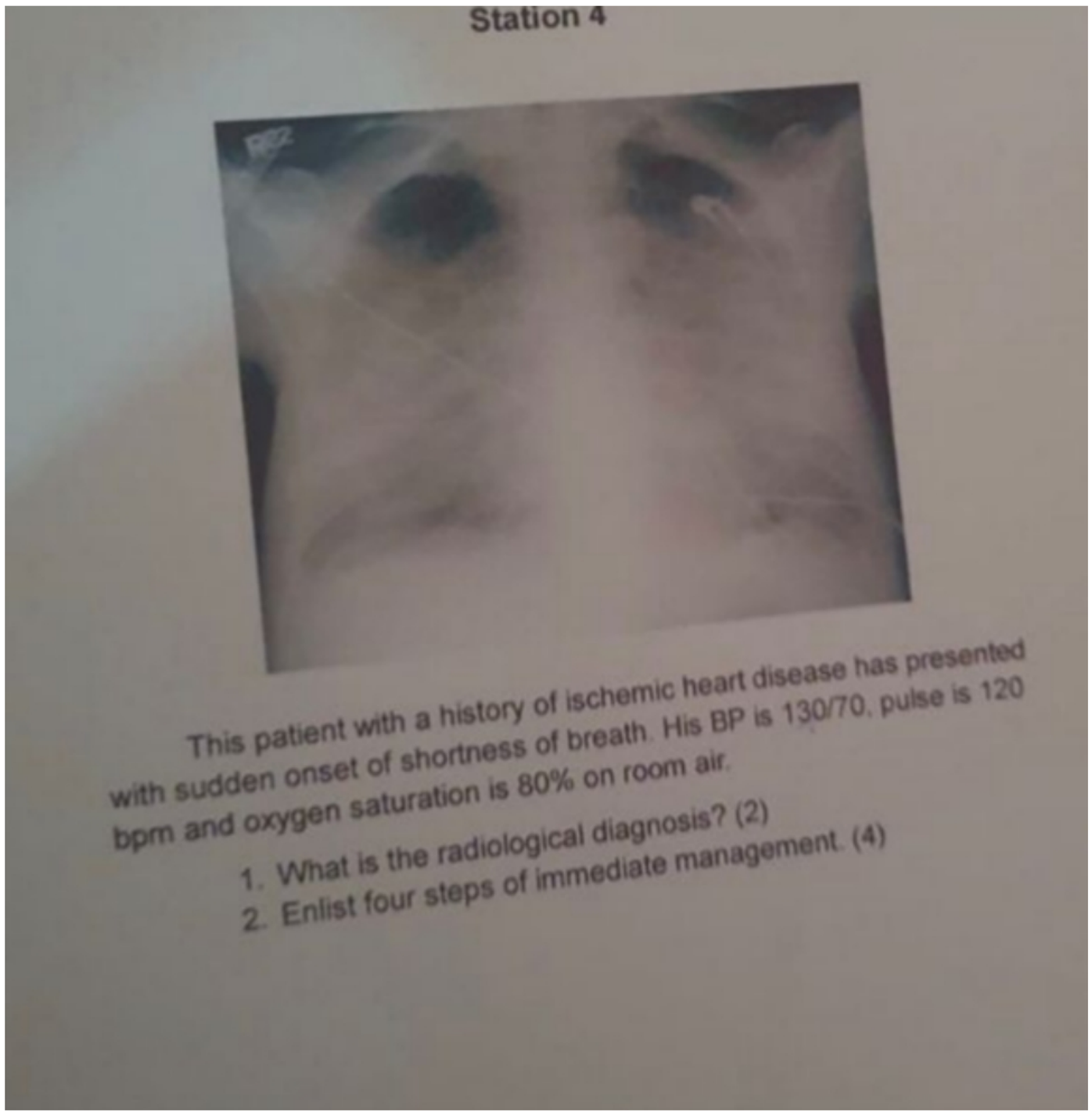
4️⃣ Management of COPD

🛑 A. Immediate / Acute Management

Controlled oxygen therapy (target SpO_2 88–92%)
Nebulized bronchodilators:
Salbutamol
Ipratropium
Systemic corticosteroids (Prednisolone)
Antibiotics (if infective exacerbation)
NIV if hypercapnic

🫁 B. Long-Term Management

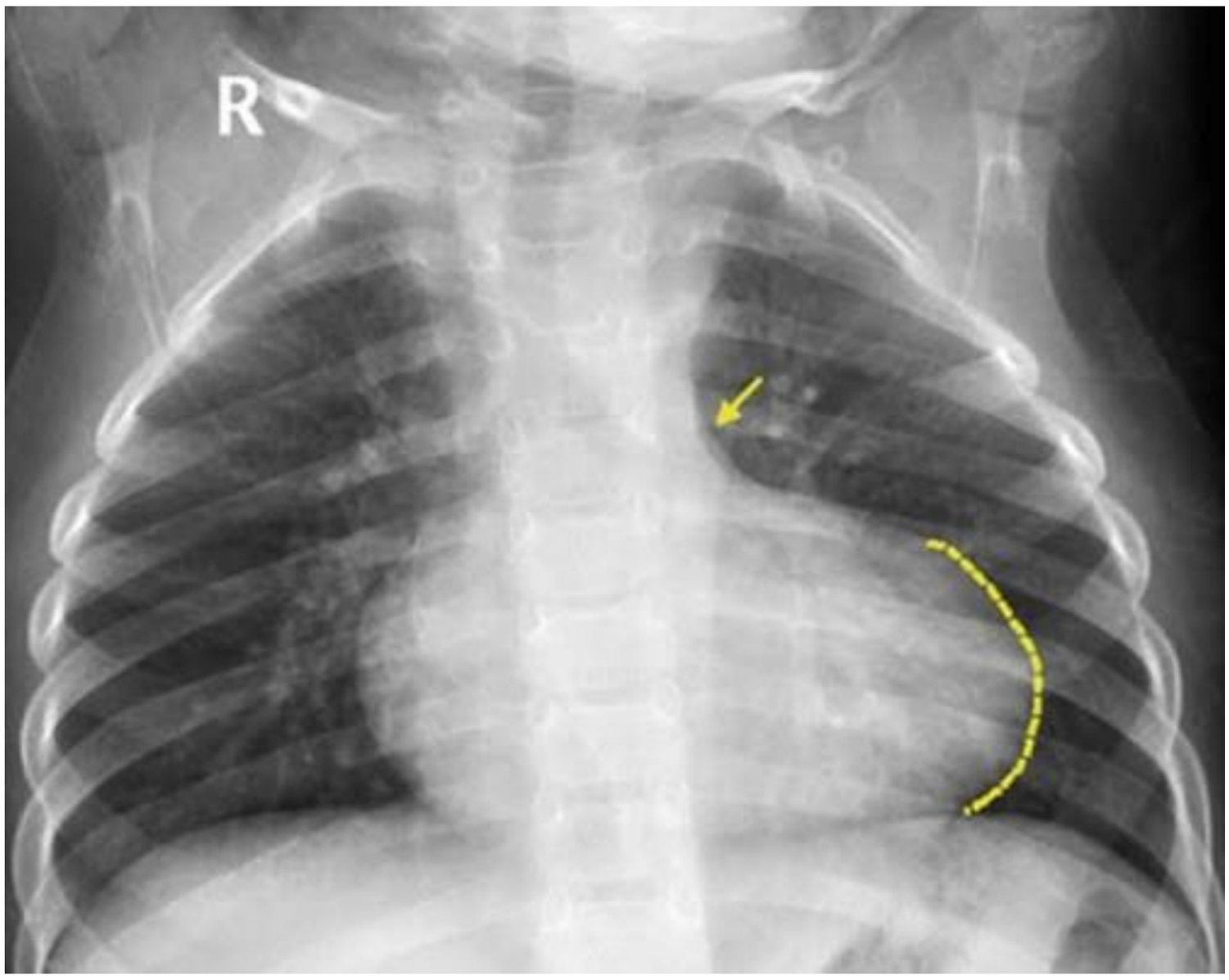
1️⃣ Smoking cessation (MOST IMPORTANT)
2️⃣ Inhaled Therapy
LABA (e.g., Salmeterol)
LAMA (e.g., Tiotropium)
ICS (if frequent exacerbations)
3️⃣ Pulmonary rehabilitation
4️⃣ Vaccination
Influenza
Pneumococcal
5️⃣ Long-term oxygen therapy
If:
$PaO_2 \leqslant 55$ mmHg
Or $SpO_2 \leqslant 88\%$



Acute pulmonary edema (cardiogenic)

Txt
Mnemonic: "LMNOP"
Loop diuretic (furosemide 50- 100 mg IV)
Morphine (decreases svmptoms, decreases afterload)
Nitrates (IV glyceryl trinitrate): 10 200 ug/min, titrated upwards every 10 minutes. Titrate until clinical improvement occurs or systolic BP falls to<110 mmHg.
Oxygen - High-flow, high-concentration pressure
Non-invasive positive pressure ventilation (continuous [CPAP] of 5- 10 mmHg) Aa facemask results in rapid improvement.
Position i.e. sit the patient up (decreases preload)

If no response, consider:
Inotropic agents especially in hypotensive patients e.g dobutamine)
Intra-aortic balloon pump (IABP)



5-year-old child with cyanosis and ejection systolic murmur heard over left sternal border.

1️⃣ Diagnosis

Tetralogy of Fallot (TOF) – the most common cyanotic congenital heart disease in children.

Reasoning: Cyanosis since early childhood + systolic murmur (pulmonary stenosis) + age 5 → “tet spells” possible.

2️⃣ Four Classical Components of TOF

Ventricular Septal Defect (VSD) – usually subaortic.

Pulmonary Stenosis – obstruction of right ventricular outflow tract.

Overriding Aorta – aorta sits above the VSD, receiving blood from both ventricles.

Right Ventricular Hypertrophy (RVH) – due to pressure overload from pulmonary stenosis.

3️⃣ Chest X-Ray Findings

“Boot-shaped heart” (coeur en sabot) due to RV hypertrophy

Concave main pulmonary artery segment (due to pulmonary stenosis)

Oligemic lung fields (reduced pulmonary vascular markings)

Sometimes right atrial enlargement

Laryngoscope

Parts of a Laryngoscope

Handle – holds the batteries and provides grip.
Blade – inserted into the mouth to visualize the vocal cords.

Types of blades:

Macintosh (curved) – fits into the vallecula.
Miller (straight) – lifts the epiglottis directly.
Light source – illuminates the larynx (often LED or fiber-optic).
Hinge/attachment point – connects blade to handle.

Uses

Endotracheal intubation.
Visualizing the larynx and vocal cords.
Removing foreign bodies from the airway.
Assessing airway anatomy in emergencies.

2 Nebulizer

Parts of a Nebulizer

Medication cup – holds the liquid drug.
Mouthpiece or mask – for the patient to inhale the mist.
Tubing – connects nebulizer to air/oxygen source.
Compressor (for jet nebulizer) – converts liquid medicine into aerosol.
Baffle (in some types) – ensures uniform particle size.

Uses

Deliver inhaled medications directly to lungs.
Treat asthma, COPD, cystic fibrosis, bronchiolitis.
Useful in acute exacerbations or for patients who cannot use inhalers.

Drugs Commonly Given via Nebulizer

Bronchodilators:
Salbutamol (albuterol)
Levosalbutamol
Ipratropium bromide
Steroids:
Budesonide
Fluticasone (less common in acute neb)
Mucolytics:
Hypertonic saline
Dornase alfa
Other:
Epinephrine (for croup)
Antibiotics (for inhalation therapy in some cases)
Antibiotics Administered by Inhalation
Tobramycin – commonly used for Pseudomonas infections in cystic fibrosis.
Others (less common): Colistin (polymyxin E).

Infective endocarditis

Investigations

A. Blood Culture - Most accurate investigation
B. Echo - TEE is more sensitive and specific than TTE
C. Other tests - CBC, ESR, urinalysis, urine cultures, ECG

Diagnostic Criteria

Duke criteria – combines major and minor criteria.
Major: Positive blood culture for typical organisms, evidence of endocardial involvement on echo.
Minor: Fever, vascular phenomena, immunologic phenomena, predisposing heart condition.

3️⃣ Treatment

A. Antibiotic Therapy (IV, long course)
Empirical therapy (before culture results, especially for native valve):
Vancomycin + Gentamicin
Targeted therapy (after culture & sensitivity):
Staphylococcus aureus: Nafcillin/Oxacillin (if MSSA) or Vancomycin (if MRSA)
Streptococci: Penicillin G or Ceftriaxone ± Gentamicin
Enterococci: Ampicillin + Gentamicin
Duration: Usually 4–6 weeks IV, depending on valve type & organism.

B. Supportive & Adjunctive
Treat heart failure if present.
Surgical intervention if:
Valve destruction with heart failure
Large vegetations (>10 mm)
Recurrent emboli
Uncontrolled infection

Rheumatic Fever

1️⃣ Diagnosis

✅ Clinical Diagnosis — Based on Modified Jones Criteria

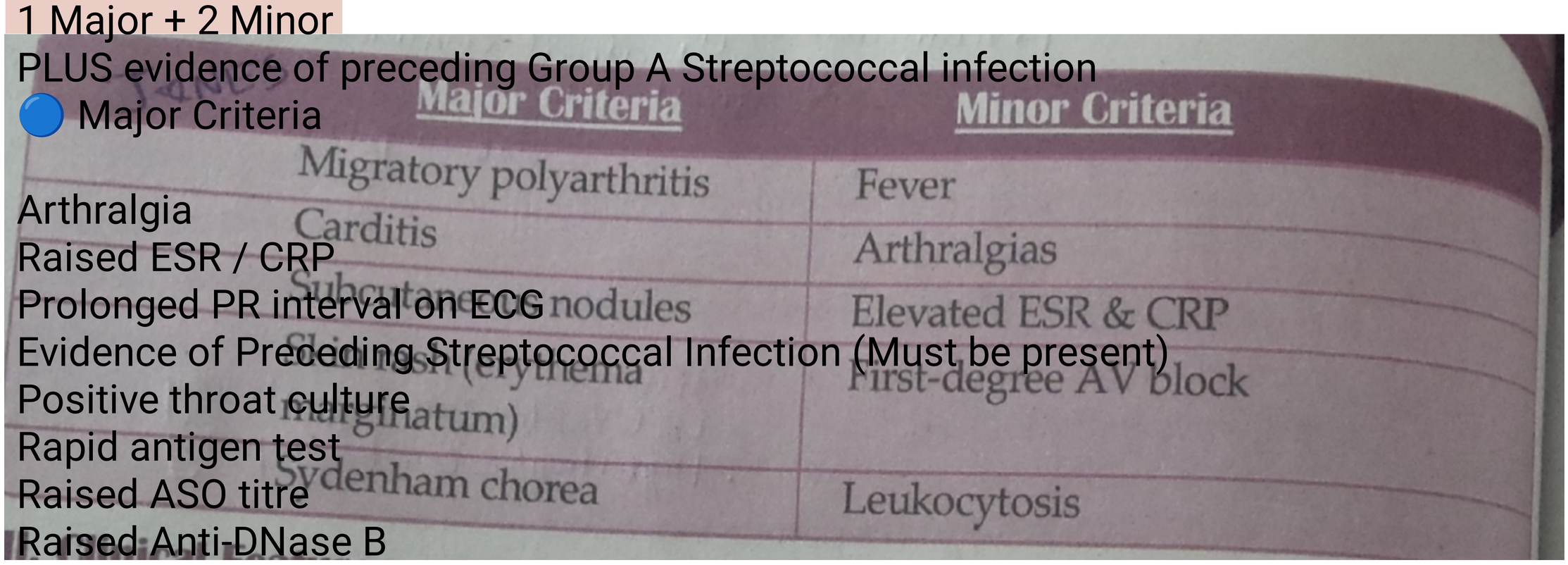
Diagnosis requires:

2 Major, OR

1 Major + 2 Minor

PLUS evidence of preceding Group A Streptococcal infection

🔵 Major Criteria



Major Criteria	Minor Criteria
Migratory polyarthritis	Fever
Carditis	Arthralgias
Subcutaneous nodules	Elevated ESR & CRP
Skin rash (erythema marginatum)	First-degree AV block
Sydenham chorea	Leukocytosis

Arthralgia

Raised ESR / CRP

Prolonged PR interval on ECG

Evidence of Preceding Streptococcal Infection (Must be present)

Positive throat culture

Rapid antigen test

Raised ASO titre

Raised Anti-DNase B

2️⃣ Specific Investigations

🧪 Blood Tests

ASO titre (most commonly asked)

Anti-DNase B

ESR ↑

CRP ↑

CBC → mild leukocytosis

❤️ Cardiac Investigations

Echocardiography → detects valvulitis (especially mitral regurgitation)

ECG → prolonged PR interval

3️⃣ Management

A. Eradication of Streptococcus

Benzathine Penicillin G (IM single dose)

OR

Oral Penicillin V for 10 days

(Erythromycin if penicillin allergy)

B. Anti-inflammatory Treatment

Aspirin (for arthritis)

Prednisolone (if severe carditis)

C. Management of Complications

Heart failure → Diuretics + ACE inhibitors

Chorea → Haloperidol / Valproate

D. Secondary Prevention (VERY IMPORTANT in viva)

Benzathine penicillin IM every 3–4 weeks Duration depends on cardiac involvement:

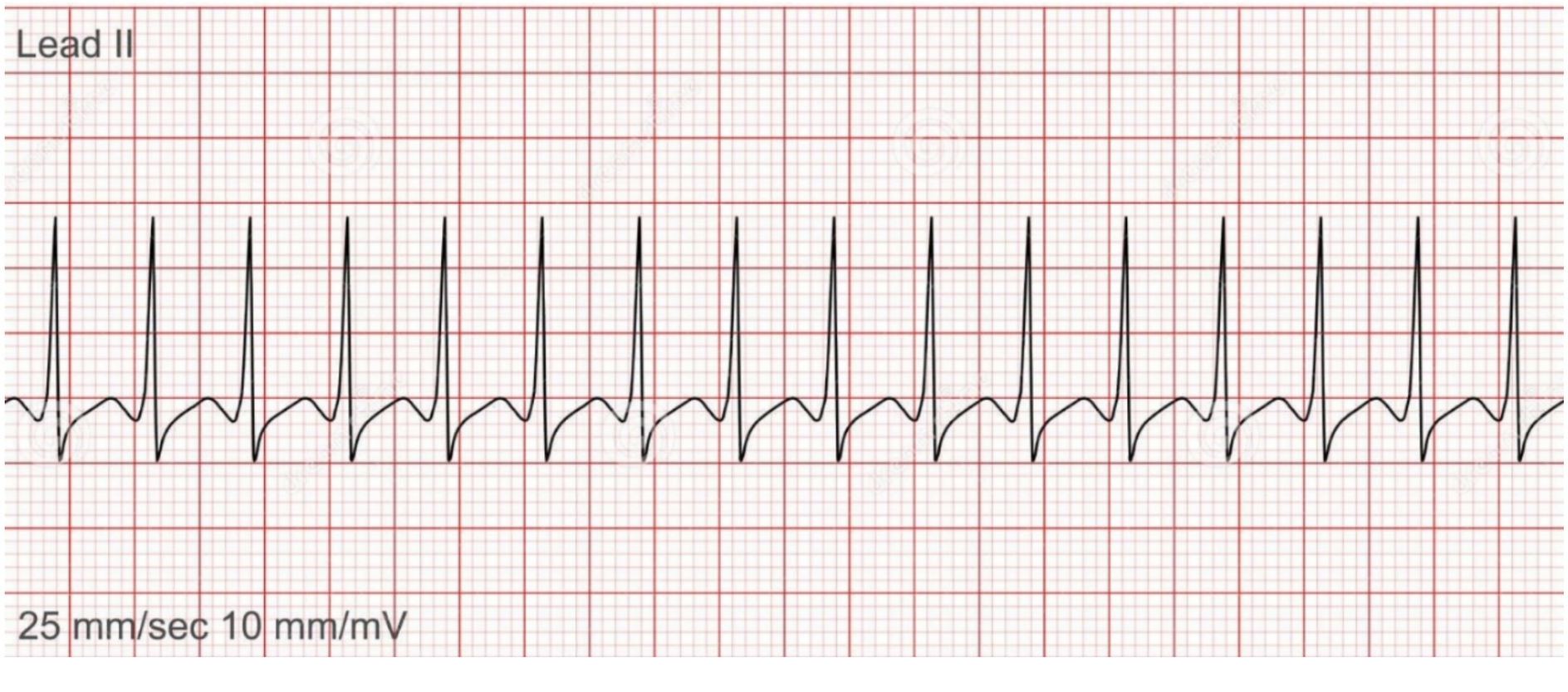
No carditis → 5 years or until age 21

Carditis without residual disease → 10 years or until 21

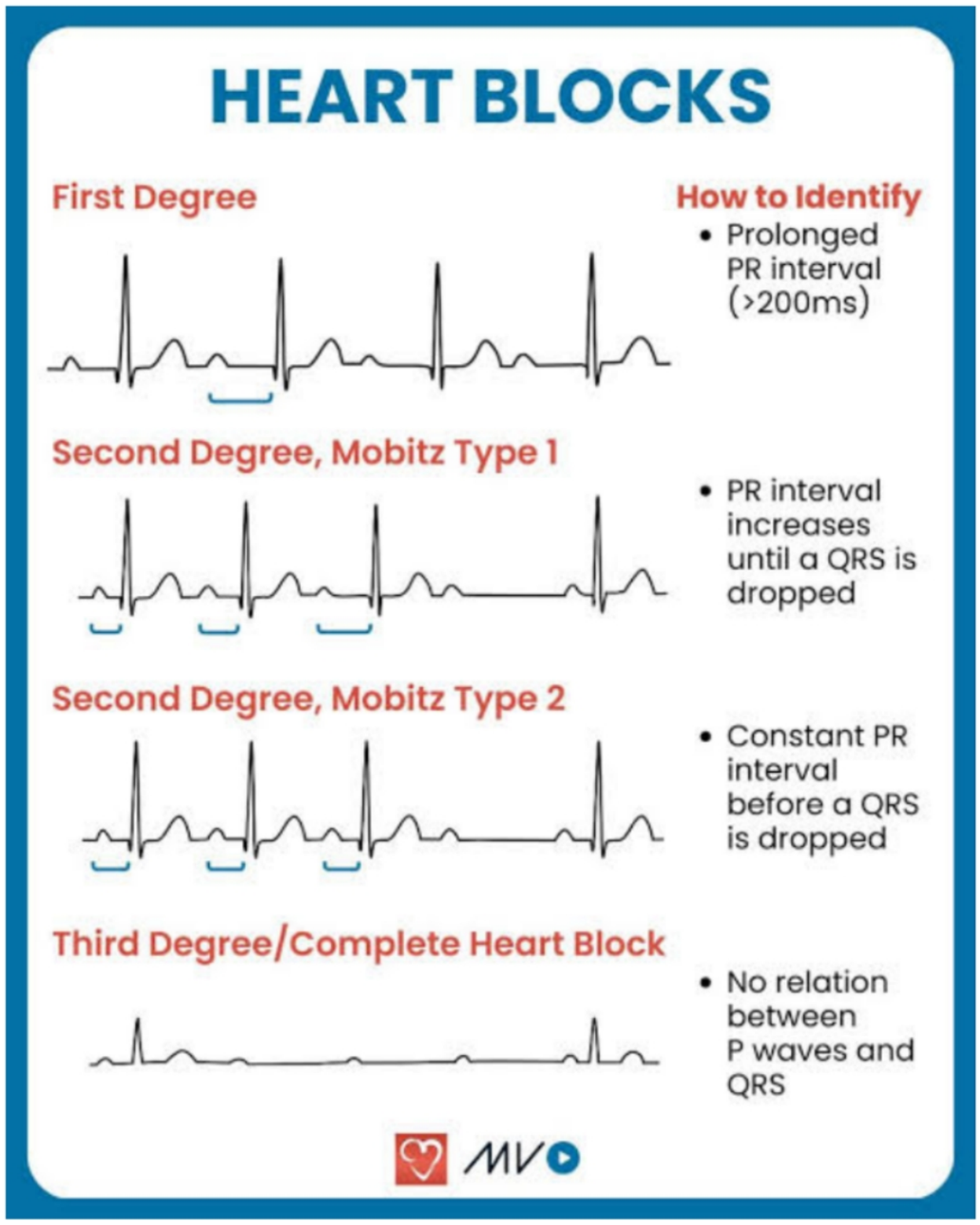
Carditis with residual valvular disease → 10 years or until 40 (sometimes lifelong)

NSTEMI (Non–ST Elevation Myocardial Infarction)
1️⃣ Diagnosis
✅ Clinical Features
Chest pain (retrosternal, crushing, radiating to left arm/jaw)
Sweating, nausea, dyspnea
Pain >20 minutes, not relieved by rest
✅ ECG Findings
ST depression
T-wave inversion
No persistent ST elevation
✅ Cardiac Biomarkers (Key Point)
Troponin I or T elevated
CK-MB may also be raised
👉 Difference from Unstable Angina:
Troponin is elevated in NSTEMI, normal in unstable angina.
2️⃣ Risk Factors
Non-modifiable
Increasing age
Male gender
Family history of IHD
Modifiable
Hypertension
Diabetes mellitus
Smoking
Hyperlipidemia
Obesity
Sedentary lifestyle
3️⃣ Management
🚨 Immediate (Emergency Management)
MONA-BASH approach
Morphine (for severe pain)
Oxygen (if saturation <90%)
Nitrates (sublingual GTN)
Aspirin (300 mg loading dose)
Beta blockers
Anticoagulation (LMWH e.g., enoxaparin)
Statins (high-intensity e.g., atorvastatin)
Heparin (if not already covered)
🩺 Dual Antiplatelet Therapy (DAPT)
Aspirin + Clopidogrel / Ticagrelor
🔎 Risk Stratification
TIMI or GRACE score
High-risk patients → Early coronary angiography ± PCI
⏳ Long-Term Management
Lifestyle modification
Control BP, sugar, lipids
Continue DAPT for 12 months
Beta blocker, ACE inhibitor, statin

Supraventricular Tachycardia (SVT)

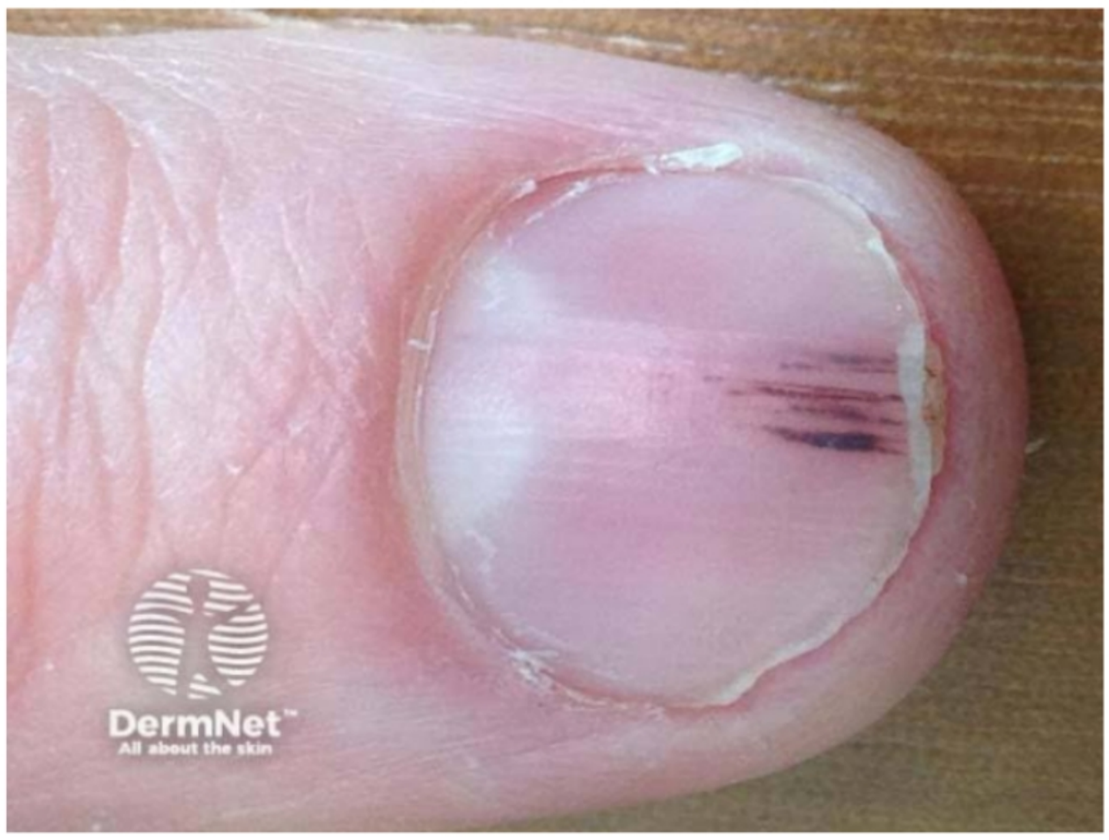


SVT (Supraventricular Tachycardia)
1️⃣ Definition
SVT is a rapid heart rhythm originating above the ventricles (atria or AV node), usually due to a re-entry mechanism.
Common type: AVNRT (AV nodal re-entrant tachycardia).
2️⃣ ECG Findings
Classic ECG features:
Heart rate: 150–250 bpm
Regular rhythm
Narrow QRS complex (<120 ms)
P waves:
Often absent, hidden in QRS
Or retrograde P waves (may appear after QRS)
Sudden onset and sudden termination
👉 If QRS is wide → consider SVT with aberrancy or VT.
3️⃣ Clinical Features
Sudden palpitations
Dizziness
Chest discomfort
Shortness of breath
Sometimes syncope
May have hypotension if unstable
4️⃣ Treatment
Management depends on hemodynamic stability
🚨 A. If Patient is UNSTABLE
(Shock, hypotension, altered consciousness, chest pain)
👉 Immediate synchronized DC cardioversion
✅ B. If Patient is STABLE
Step 1: Vagal Maneuvers
Valsalva maneuver
Carotid sinus massage (avoid in elderly with carotid disease)
Step 2: IV Adenosine (Drug of Choice)
6 mg rapid IV bolus
If no response → 12



Mobitz Type II (Second-Degree AV Block Type II)
📉 ECG Features
Regular P waves
Sudden dropped QRS complexes (some P waves not followed by QRS)
Constant PR interval in conducted beats (no progressive prolongation)
May show fixed ratios like 2:1 or 3:1 block
QRS often wide (because block is usually infranodal — His–Purkinje system)
👉 Important: It is more serious than Mobitz I and can progress to complete heart block.
⚠️ 4 Conditions Associated with Mobitz Type II
1️⃣ Anterior wall myocardial infarction
(especially LAD involvement affecting septum & conduction system)
2️⃣ Degenerative conduction system disease
(e.g., Lev's or Lenègre's disease in elderly)
3️⃣ Myocarditis
(viral or inflammatory damage to conduction pathways)
4️⃣ Post–cardiac surgery / Structural heart disease
(e.g., after valve surgery or congenital defect repair)
👉 Other possible causes (if examiner pushes):
Infiltrative diseases (sarcoidosis)
Electrolyte disturbances
Drug toxicity (less common than Mobitz I)

Inferior Wall Myocardial Infarction (IWMI)
1️⃣ ECG Findings
✅ ST Elevation in:
Leads II, III, aVF
✅ Reciprocal ST Depression in:
Lead I and aVL
✅ Artery Involved:
Most commonly Right Coronary Artery (RCA)
Sometimes Left Circumflex (LCX)
⚠️ Important ECG Points
ST elevation in Lead III > Lead II → suggests RCA involvement
May be associated with:
Bradycardia
AV block (RCA supplies AV node)
Always do Right-sided ECG (V4R) to rule out Right Ventricular MI
2️⃣ Clinical Features
Chest pain
Hypotension (especially if RV infarct)
Nausea, vomiting (vagal stimulation)
Bradycardia
3️⃣ Treatment
Management follows STEMI protocol
🚨 Immediate Management
MONA-B
Morphine (if severe pain)
Oxygen (if $SpO_2 < 90\%$)
Nitrates (⚠️ Avoid in RV MI or hypotension)
Aspirin 300 mg (loading)
Beta-blocker (if no contraindications)
🔬 Reperfusion Therapy (Most Important)
Preferred:
Primary PCI (within 90 minutes)
If PCI not available:
Thrombolysis (e.g., Streptokinase, Tenecteplase)
💊 Additional Treatment
Dual antiplatelet therapy (Aspirin + Clopidogrel/Ticagrelor)
Anticoagulation (Heparin/LMWH)
High-intensity statin (Atorvastatin 80 mg)
ACE inhibitor (if stable)
⚠️ Special Consideration: RV Infarction
If hypotension + clear lungs + ST elevation in V4R:
Give IV fluids
Avoid nitrates
Avoid diuretics



Splinter hemorrhages — classically associated with Infective Endocarditis, but can occur in other conditions.

🔎 4 Causes of Splinter Hemorrhages

1️⃣ Infective Endocarditis

– Due to septic emboli damaging nail bed capillaries

– Often accompanied by fever, murmur, Janeway lesions

2️⃣ Trauma (Most Common Cause Overall)

– Minor nail injury

– Usually isolated and distal

3️⃣ Vasculitis

– e.g., small vessel vasculitis

4️⃣ Systemic diseases

Systemic Lupus Erythematosus (SLE)

Rheumatoid arthritis

👉 Other possible causes (if examiner pushes):

Antiphospholipid syndrome

Psoriasis

Hematological disorders

Treatment: IV Antibiotics (4–6 weeks)

Example: Ceftriaxone + Vancomycin

EXAMINATIONS

Satation 1:Chest examination on dummy (medicine) mitral regurgitation

Station 2 :chest examination on dummy peasds

Station 3: respiratory examination on dummy paeds

Station4: respiratory examination on dummy medicine...crackles

Station 5: JVP examination

-Causes of increase in JVP

-how to differentiate between Jugular and Carotids

Stataion 9:💌
Precordium examination of a child

JVP EXAMINATION

- What are the causes of elevated JVP?
- How do you differentiate JVP from carotid pulse?
- What does a positive hepatojugular reflux indicate?

Causes of Elevated JVP (Raised Jugular Venous Pressure)

Elevated JVP reflects **raised right atrial pressure**. Common causes include:

1️⃣ Right-Sided Heart Failure

- Most common cause
- Secondary to left heart failure or pulmonary disease

2️⃣ Constrictive / Restrictive Conditions

- Constrictive pericarditis
- Cardiac tamponade
- Restrictive cardiomyopathy

3️⃣ Pulmonary Causes (↑ RV afterload)

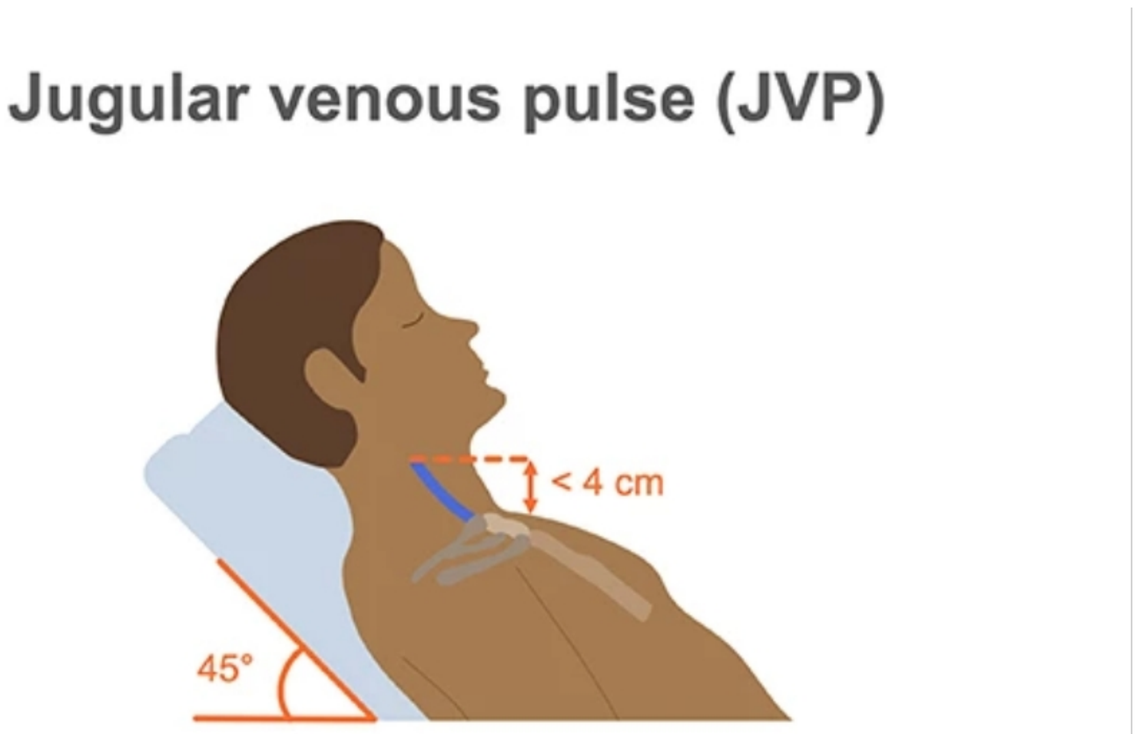
- Pulmonary hypertension
- Pulmonary embolism
- Cor pulmonale (chronic lung disease)

4️⃣ Tricuspid Valve Disease

- Tricuspid regurgitation
- Tricuspid stenosis

5️⃣ Volume Overload

- Renal failure
- Excess IV fluids



Normal = 4-10 cm of water

Differentiating JVP from Carotid Pulse

Feature	JVP	Carotid Pulse
Palpable?	❌ Not palpable	✅ Palpable
Waveform	Biphasic (a and v waves)	Single upstroke
Effect of position	Falls when sitting upright	No change
Effect of pressure at base of neck	Disappears	Persists
Respiration	Falls on inspiration	No change
Hepatojugular reflux	Increases	No change

💡 Clinical tip: If you can **feel it, it's carotid. If you can only see it, it's JVP.**

Positive Hepatojugular Reflux (HJR)

Method:

Firm pressure over right upper abdomen (liver area) for ~10–15 seconds.

Positive HJR:

- Sustained rise in JVP (>3–4 cm)
- Persists while pressure is applied

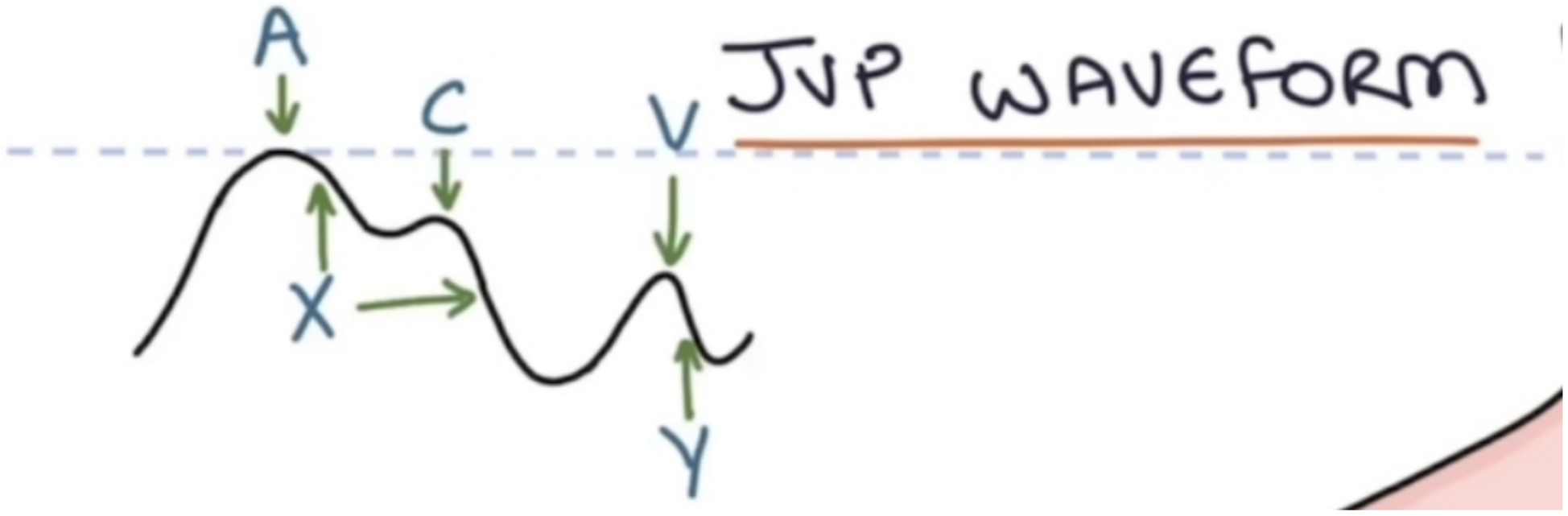
What It Indicates:

👉 **Right ventricular failure**

👉 Inability of the right heart to handle increased venous return

Commonly seen in:

- Right heart failure
- Constrictive pericarditis
- Severe tricuspid regurgitation



A : Atrial contraction

X : relaX

C : Closure

V : Volume

Y : the blood goes awaY (emptY)

A wave - Atrial Contraction - Atrial pressure rises

X descent - RelaXation of RA, ventricles shrink and allows the atrium to fill in!

C - Closure of Tricuspid valve - at the start of systolic contraction

V - Villing(filling) of Ra as atria relax - slight rise in JVP

Y - Triscuspid opens - EmptYing RA

Hepatojugular reflux test

The **hepatojugular reflux test** involves the **application of pressure to the liver** whilst observing for a **sustained rise in JVP**.

Eliciting hepatojugular reflux

To be able to perform the test, there should be at least a **3cm distance** from the **upper margin** of the **baseline JVP** to the **angle of the mandible**:

- Position the patient in a **semi-recumbent position** (45°).
- Apply direct pressure to the **liver**.
- Closely **observe the IJV** for a rise.
- In healthy individuals, this rise should last **no longer than 1-2 cardiac cycles** (it should then fall).
- If the rise in JVP is **sustained** and **equal to or greater than 4cm** this is deemed a **positive resul**
- This assessment can be uncomfortable for the patient and therefore it should only be performed when felt necessary (an examiner will often prevent you from performing it in an OSCE but you should mention it).

Con

Conditions associated with hepatojugular reflux

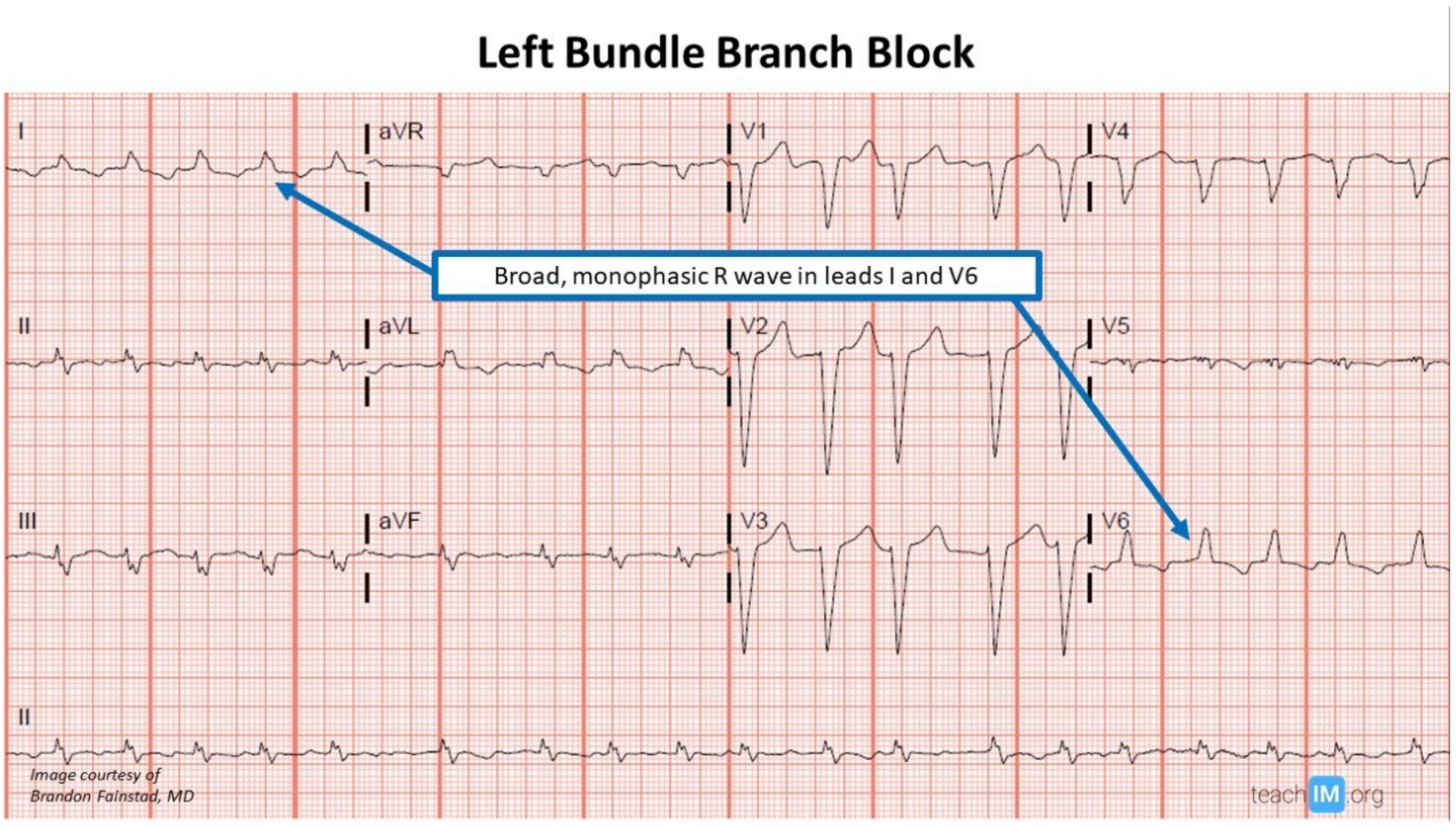
A **positive hepatojugular reflux result** suggests the **right ventricle is unable to accommodate an increased venous return**, but it is **not diagnostic** of any specific condition. The following conditions frequently produce a positive hepatojugular reflux test:

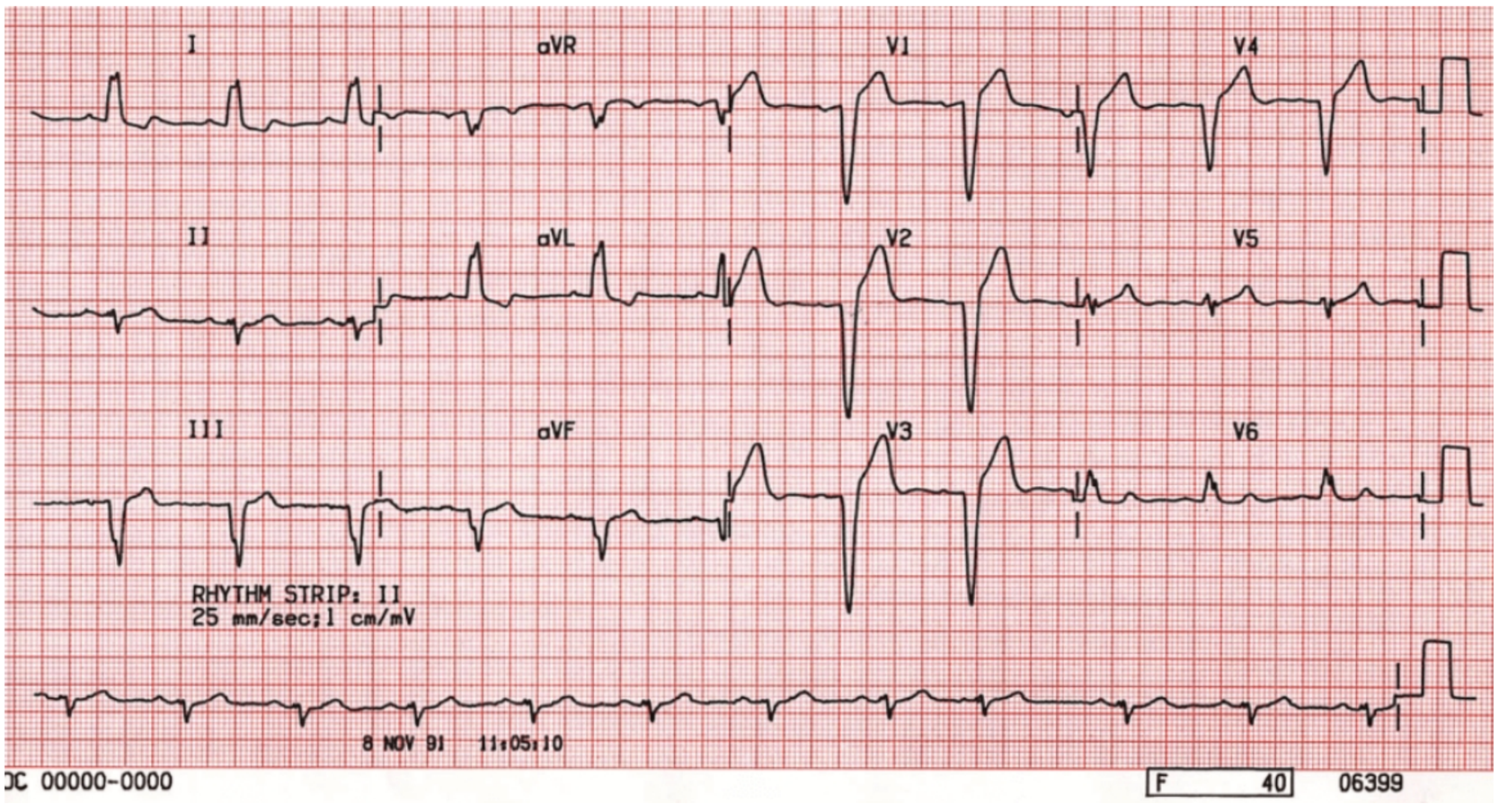
- Constrictive pericarditis
- Right ventricular failure
- Left ventricular failure
- Restrictive cardiomyopathy

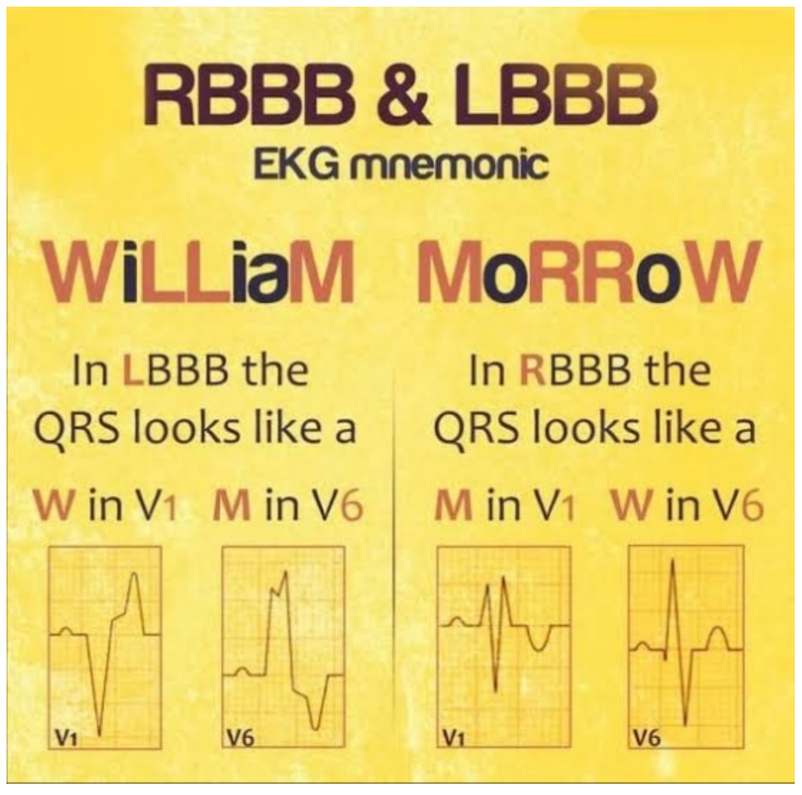
Station 6:Left bundle branch block

Ecg

Causes







Major causes include ischemic heart disease, long-term hypertension, cardiomyopathy, and structural issues like aortic stenosis

SPIROMETRY

- FVC → Forced Vital Capacity – total air forcibly exhaled
- FEV_1 → Forced expiratory volume in 1 sec
- FEV_1/FVC → Ratio used to classify disease
- PEFR → Peak Expiratory Flow Rate

Obstructive Disease
(Asthma, COPD)

- ↓ FEV_1
- Normal or ↓ FVC
- ↓ FEV_1/FVC (<70%)

Restrictive Disease
(ILD, Fibrosis)

- ↓ FEV_1
- ↓ FVC
- Normal or ↑ FEV_1/FVC

Station7: Asthma

Diagnosis

Managment

Asthma is a chronic inflammatory disease of the airways characterized by reversible airflow obstruction and bronchial hyper-responsiveness.

🧠 Clinical Features (Any 4)

- Episodic **shortness of breath**
- **Wheezing**
- **Chest tightness**
- **Cough** (worse at night/early morning)
- Symptoms vary and are reversible

🔬 Investigations

1️⃣ Spirometry (Gold standard)

- ↓ FEV1
- ↓ FEV1/FVC (< 0.75–0.8)
- **Reversibility test positive**

  → Increase in FEV1 ≥ 12% AND ≥ 200 mL after bronchodilator

2️⃣ Peak Expiratory Flow Rate (PEFR)

- Diurnal variation > 10%

3️⃣ Other supportive tests

- Eosinophilia
- ↑ IgE
- CXR (usually normal; done to exclude other causes)

🚑 Management

📌 Acute Attack (Emergency)

Mild–Moderate

- **Nebulized Salbutamol**
- Oxygen (target SpO_2 94–98%)
- Oral/IV steroids (Prednisolone or Hydrocortisone)

Severe/Life-Threatening

- High-flow Oxygen
- Nebulized Salbutamol + Ipratropium
- IV Hydrocortisone
- IV Magnesium sulfate (if not responding)
- ICU if deteriorating

Station 8: Interstitial lung disease /COPD

Diagnosis management

And fine crackles reason

🔹 1️⃣ Interstitial Lung Disease (ILD)

✅ Diagnosis (clinical + investigations)

Clinical features:

- Progressive dyspnea
- Dry cough
- Fine end-inspiratory crackles ("Velcro crackles")
- Clubbing (sometimes)

Examination:

- Reduced chest expansion
- Fine basal crackles

Investigations:

- CXR: Reticulonodular pattern
- HRCT: Honeycombing, ground glass
- PFTs: Restrictive pattern (↓ TLC, ↓ FVC, normal/high FEV1/FVC)
- ABG: Hypoxemia

✅ Management

- Treat underlying cause (e.g., autoimmune, occupational)
- Corticosteroids
- Antifibrotics (pirfenidone, nintedanib)
- Oxygen therapy
- Pulmonary rehab
- Lung transplant (advanced cases)

🔹 Why Fine Crackles in ILD?

- Due to **sudden opening of collapsed small airways and alveoli** during inspiration
- Fibrosis makes lung stiff → airways close during expiration → pop open in inspiration
- Produces "Velcro-like" fine crackles

👉 They are **late inspiratory** and do NOT disappear on coughing.

🔹 2️⃣ COPD (Chronic Obstructive Pulmonary Disease)

✅ Diagnosis

Risk factors:

- Smoking (most common)
- Biomass fuel exposure

Clinical features:

- Chronic productive cough
- Dyspnea
- Wheeze

Examination:

- Barrel chest
- Hyperresonant percussion
- Wheeze
- Decreased breath sounds

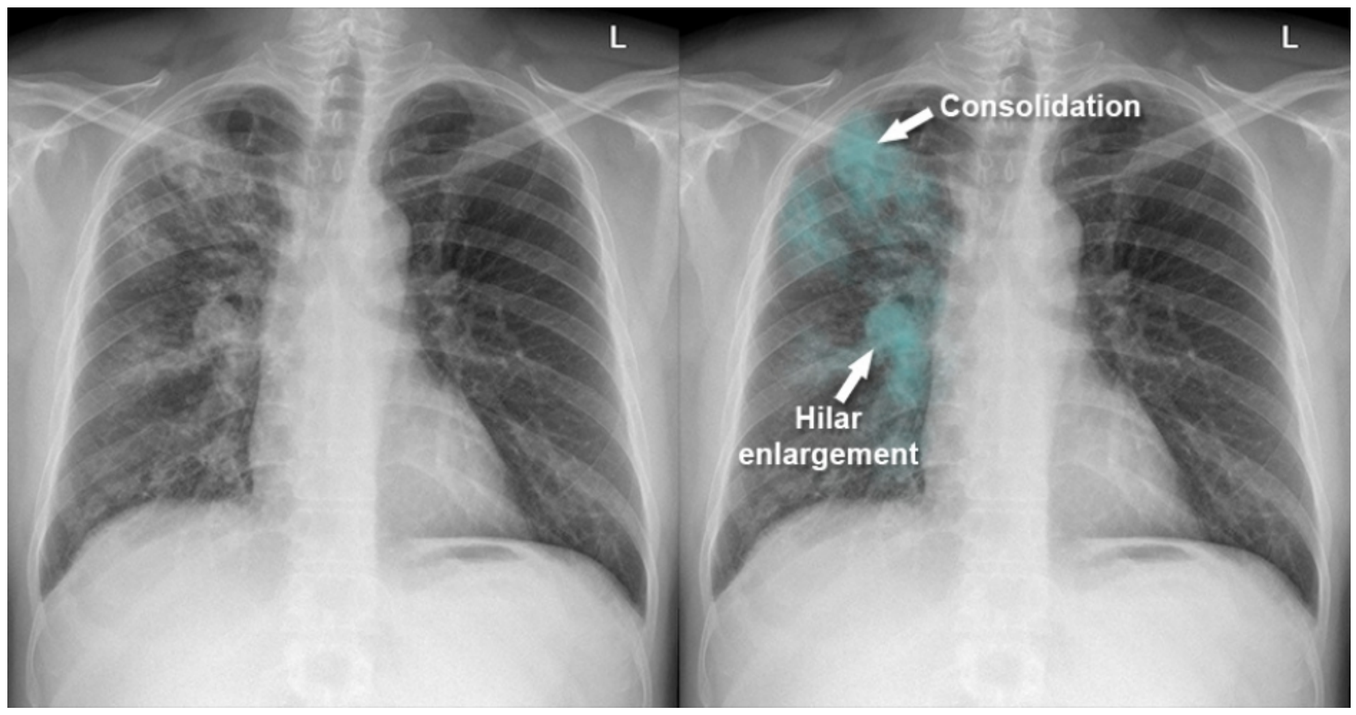
Investigations:

- Spirometry: Obstructive pattern
  ↓FEV1, ↓FEV1/FVC (<70%)
- CXR: Hyperinflation, flattened diaphragm

✅ Management

- Smoking cessation (most important)
- SABA (salbutamol)
- LABA / LAMA
- Inhaled corticosteroids
- Oxygen (if hypoxic)
- Vaccination (influenza, pneumococcal)

Feature	ILD	COPD
Pattern	Restrictive	Obstructive
Crackles	Fine (Velcro)	Wheeze ± coarse
CXR	Fibrosis	Hyperinflation
Clubbing	Common	Rare



Station9: TB scenario

Dx

TB drugs

Investigations

Chest x ray finding

Multi drug resistant tb

Extrapulmonary TB names

: How to treat multi drug resistant

Pulmonary Tuberculosis

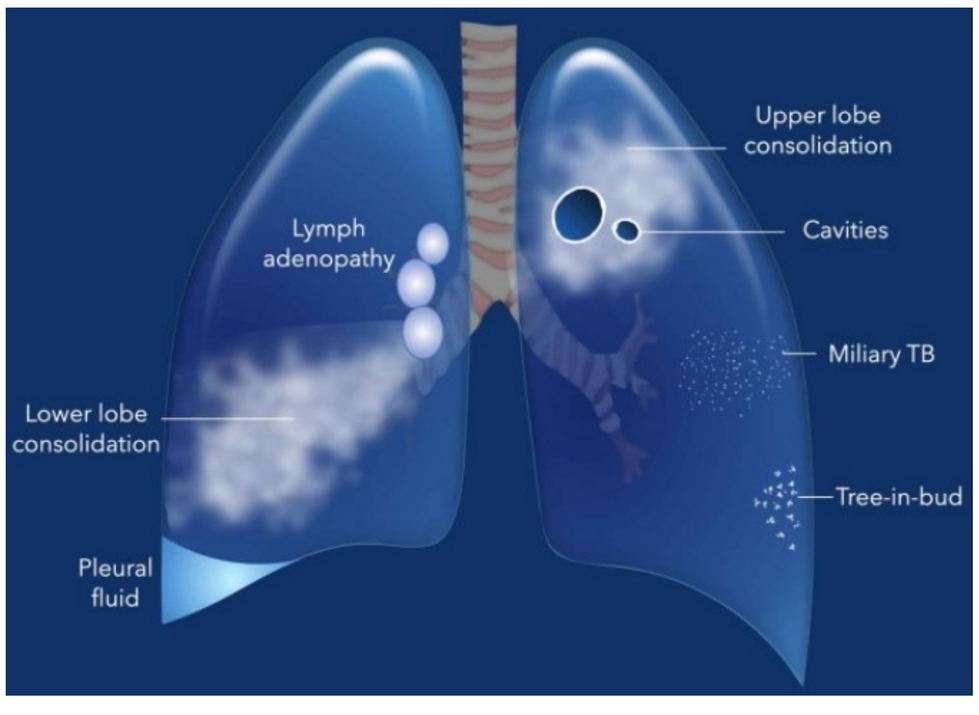
Caused by Mycobacterium tuberculosis.

🔹 Investigations

1. **Sputum AFB smear microscopy**
2. **GeneXpert (CBNAAT)** – detects TB + rifampicin resistance
3. Sputum culture (gold standard)
4. Chest X-ray
5. ESR (raised)
6. Mantoux test (supportive, not confirmatory)

🔹 Chest X-ray Findings in Pulmonary TB

- Upper lobe infiltrates
- Cavitation (classically apical)
- Fibrosis
- Hilar lymphadenopathy (more common in children)
- Miliary mottling (in miliary TB)



🔹 First-Line Anti-TB Drugs (HRZE)

Mnemonic: **HRZE**

1. Isoniazid (H)
2. Rifampicin (R)
3. Pyrazinamide (Z)
4. Ethambutol (E)

Standard regimen:

- 2 months: HRZE (intensive phase)
- 4 months: HR (continuation phase)

🔹 What is MDR-TB?

Multidrug-Resistant TB = Resistance to at least:

- Isoniazid AND
- Rifampicin

🔹 Treatment of MDR-TB

- Use second-line drugs
- Longer duration (18–24 months traditionally; now shorter regimens possible)

Common drugs:

- Fluoroquinolones (e.g., levofloxacin, moxifloxacin)
- Bedaquiline
- Linezolid
- Clofazimine
- Cycloserine

Treatment is based on:

- Drug susceptibility testing (DST)

🔹 Extrapulmonary TB (Common Types)

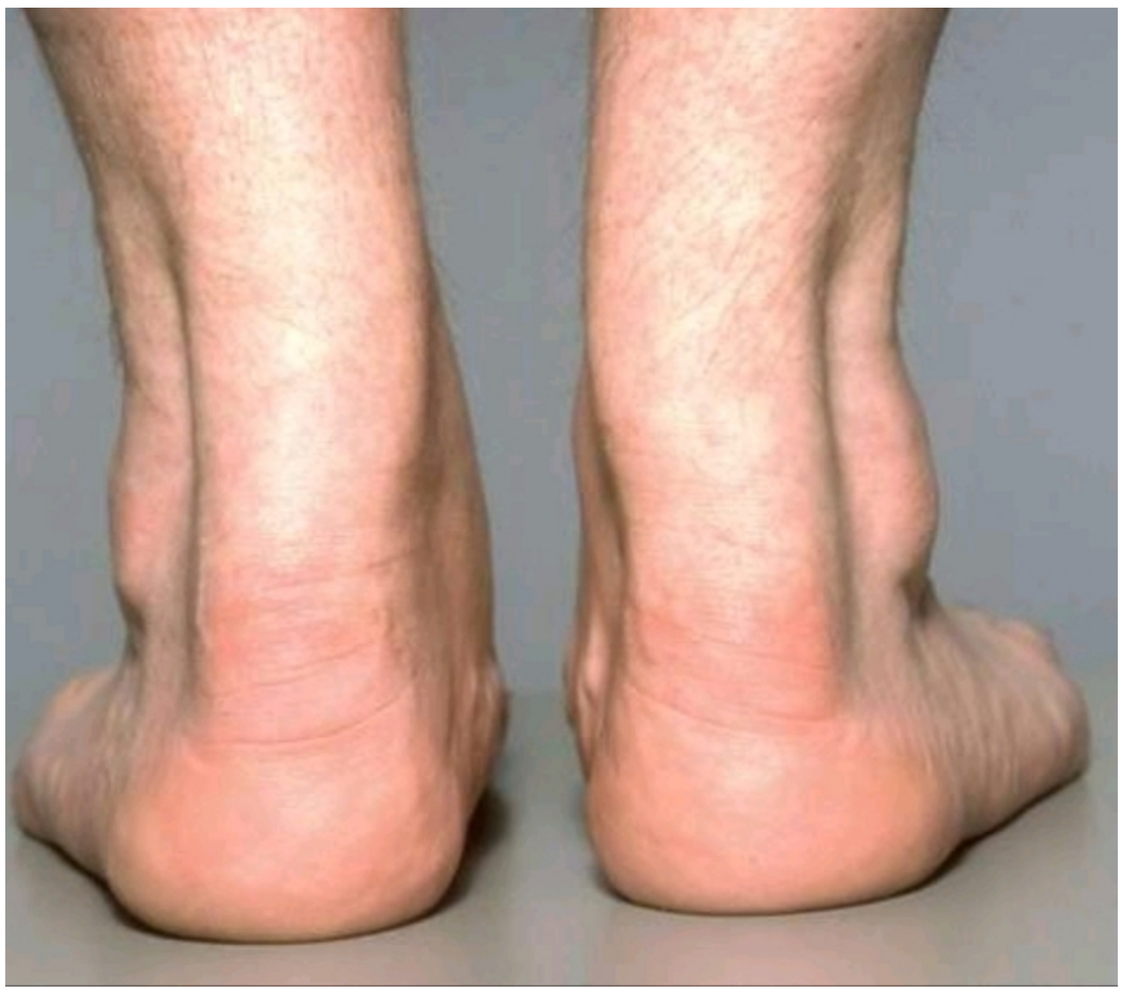
1. TB lymphadenitis (most common)
2. TB meningitis
3. Pleural TB
4. Abdominal TB
5. Spinal TB (Pott's disease)
6. Genitourinary TB
7. Miliary TB

Station 12:Hyperlipidemia in pt

How we managed it

Management of hyperlipidemia:

1. Lifestyle modification
2. Statins (first line for high LDL)
3. Fibrates if high triglycerides
4. Regular monitoring of lipid profile



👣 Bilateral heel valgus with flattened medial arch

👉 The heels are everted outward

👉 The medial longitudinal arch appears collapsed

✅ Diagnosis:

Pes planus (Flat foot)

OSPE Answer Format:

1️⃣ What do you see?

- Loss of medial longitudinal arch
- Heel valgus deformity
- Medial deviation of Achilles tendon

2️⃣ Diagnosis:

Flat foot (Pes planus)

3️⃣ Causes:

- Congenital ligament laxity
- Obesity
- Posterior tibial tendon dysfunction
- Rheumatoid arthritis
- Trauma

4️⃣ Management:

- Usually conservative
  - Arch support insoles
  - Proper footwear
  - Physiotherapy
- Surgery if severe and symptomatic

Station 13: Mitral stenosis scenario

🔎 Examination Findings:

🫀 General:

- Malar flush
- Irregularly irregular pulse (if AF present)

🩺 Pulse:

- Atrial fibrillation common

👀 JVP:

- May be elevated if pulmonary hypertension develops

🫁 Chest:

- Basal crepitations (if pulmonary edema)

❤️ CVS Examination (MOST IMPORTANT IN OSPE):

1. **Loud S1**
2. **Opening snap after S2**
3. **Mid-diastolic rumbling murmur**
4. Best heard at:
    - **Apex**
    - With **bell**
    - In **left lateral position**
5. Murmur increases after mild exercise

📊 ECG Findings:

- P mitrale (broad notched P wave)
- Atrial fibrillation
- Right ventricular hypertrophy (late stage)

🖥️ Chest X-ray:

- Straight left heart border
- Double density sign
- Pulmonary congestion

🧪 Investigation of Choice:

👉 **Echocardiography** (confirms diagnosis, valve area measurement)

⚠️ Causes:

- Most common: **Rheumatic fever**
- Congenital (rare)

Common Scenario Given:
Young female with:
Dyspnea on exertion
Orthopnea / PND
Palpitations
Hemoptysis
History of rheumatic fever

💊 Treatment:

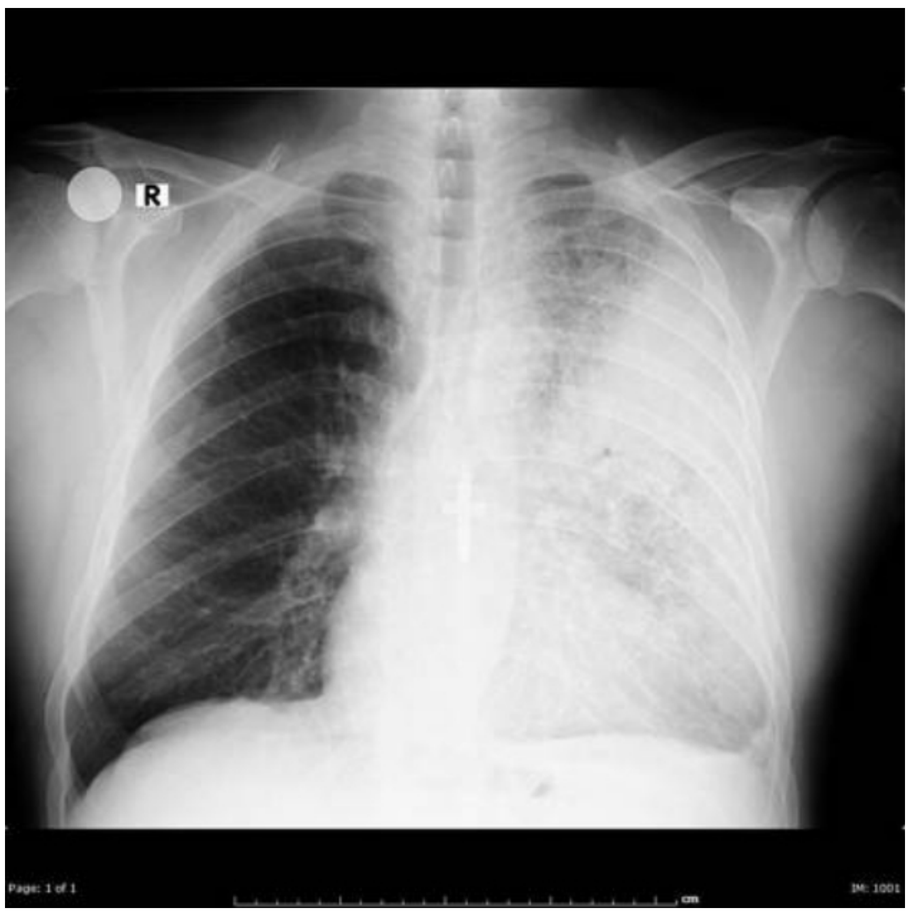
Medical:

- Diuretics
- Beta blockers
- Anticoagulation (if AF)
- Penicillin prophylaxis

Definitive:

- Balloon mitral valvotomy
- Valve replacement (severe cases)

Station 17: lobar pneumonia scenario dx and mx



Lobar Pneumonia

Classic scenario:

- Sudden onset fever, chills
- Pleuritic chest pain
- Productive cough with **rust-colored sputum**
- Dyspnea

🔎 Examination Findings

General:

- Fever, tachypnea, tachycardia

Respiratory:

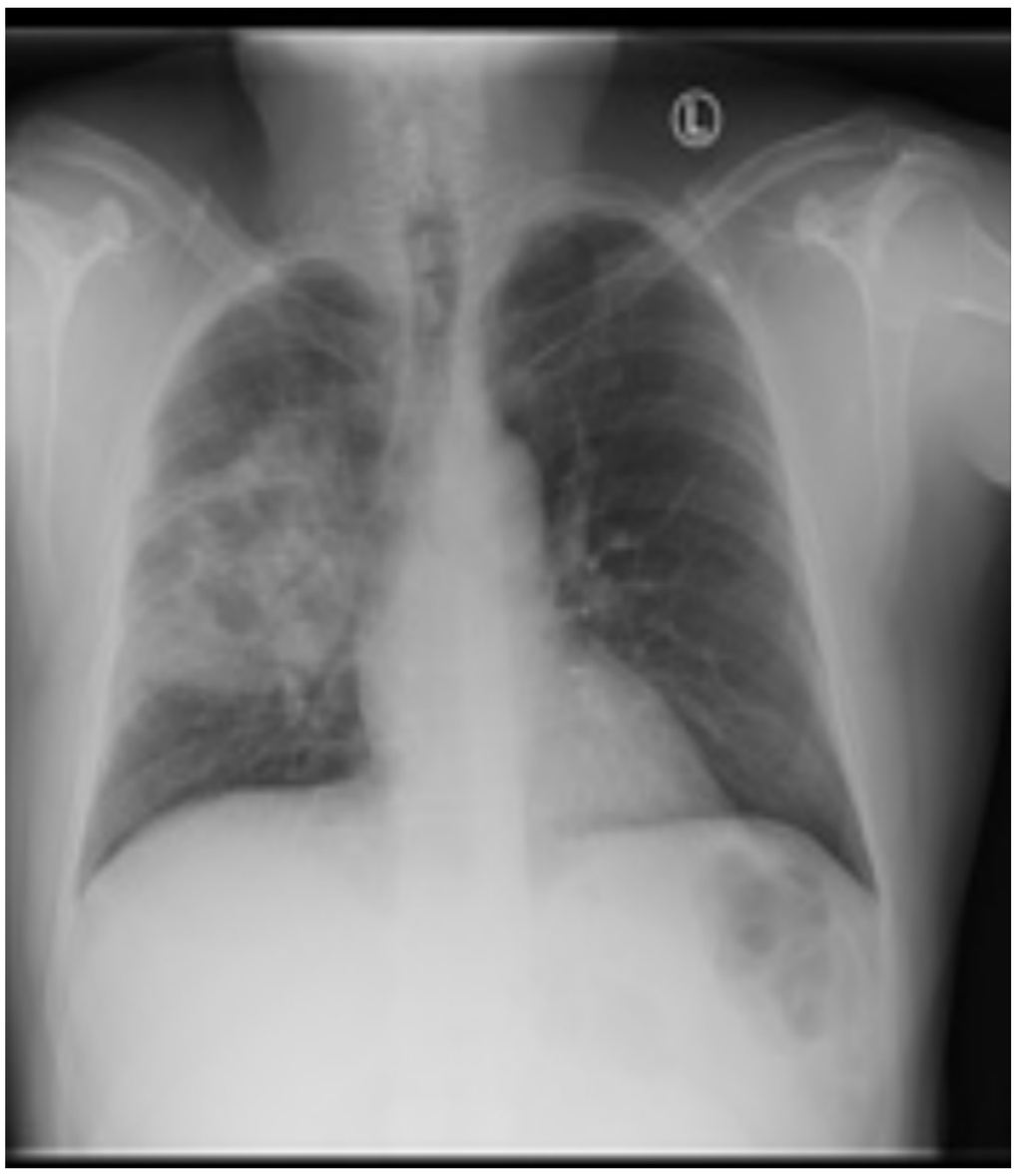
- **Bronchial breath sounds** over affected lobe
- **Dullness on percussion**
- **Increased tactile fremitus**
- **Egophony (“ee” → “ay”)**
- Crackles at the lobe edges

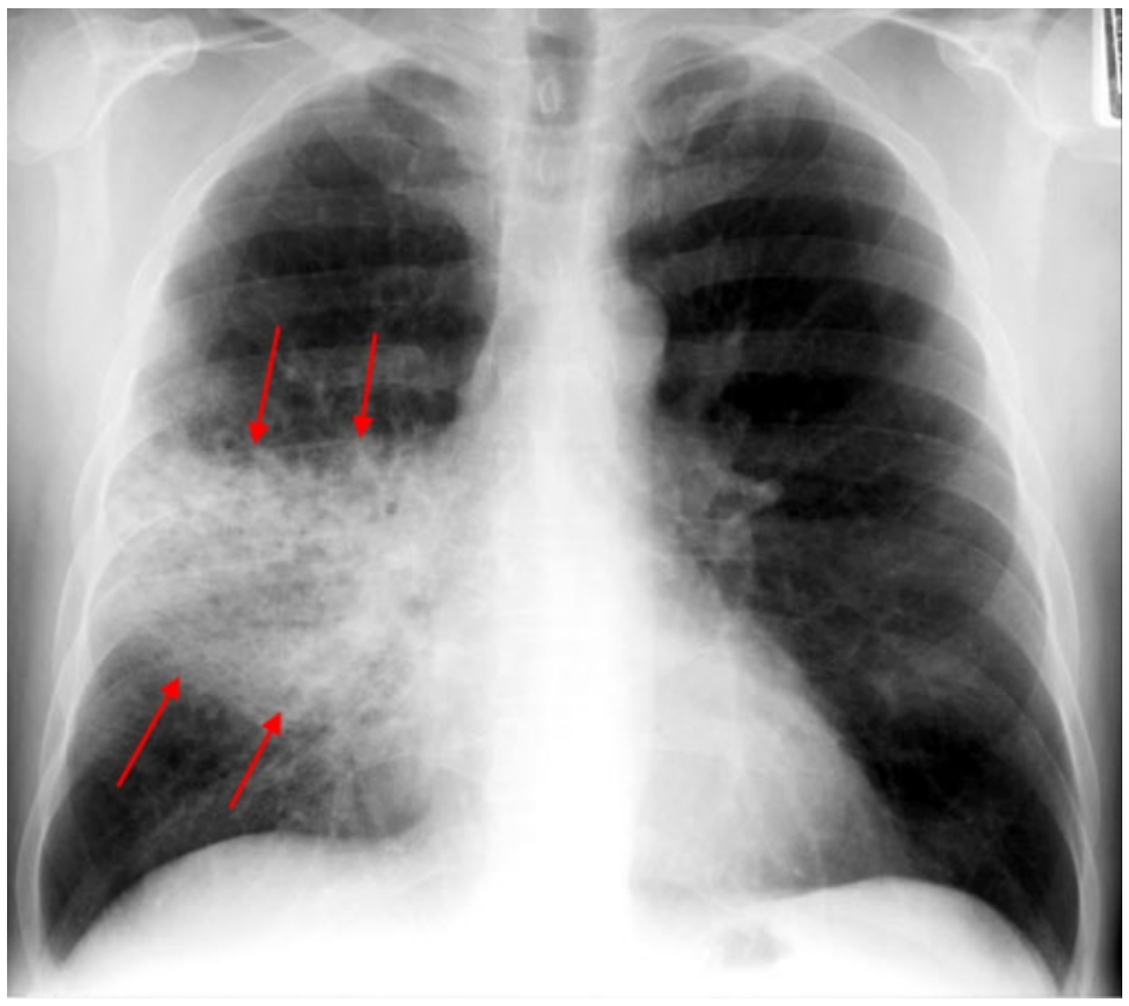
Vital signs:

- May show hypoxia in severe cases

⚠️ Complications

- Pleural effusion / empyema
- Lung abscess
- Sepsis / septic shock
- Respiratory failure





LOBAR PNEUMONIA

🧪 Investigations

1. **CXR (investigation of choice)**
    - Homogenous consolidation of one lobe
    - Air bronchograms
2. **CBC**
    - Leukocytosis with neutrophilia
3. **Sputum culture & Gram stain**
    - Common pathogen: **Streptococcus pneumoniae**
4. **Pulse oximetry / ABG** if severe

💊 Management

Supportive:

- Oxygen if hypoxic
- Fluids, antipyretics

Antibiotics:

- **Community-acquired**:
    - Amoxicillin / Ampicillin
    - Macrolides (if penicillin allergy)
- **Hospitalized / severe:**
    - IV beta-lactam ± macrolide

Other:

- Chest physiotherapy for sputum clearance

Station 16 : continuous murmur scenarion dx and inv and complication

Patent ductus arteriosus (PDA)

🧪 Investigations

1️⃣ Investigation of Choice

👉 Echocardiography with Doppler

- Confirms PDA
- Shows left-to-right shunt
- Measures size

2️⃣ ECG

- Left ventricular hypertrophy
- ± Left atrial enlargement

3️⃣ Chest X-ray

- Cardiomegaly
- Pulmonary plethora

⚠️ Complications

1. **Heart failure**
2. Pulmonary hypertension
3. **Eisenmenger syndrome**
4. Infective endarteritis
5. Atrial fibrillation (late)

🔎 Clinical Findings

- Continuous murmur (systole + diastole)
- Hyperdynamic precordium
- Water hammer pulse
- Signs of heart failure (if large shunt)

💊 Management (if asked)

- Indomethacin (in preterm infants)
- Transcatheter device closure
- Surgical ligation

Station 18 : Bradycardia scenario Any question you wanna ask from patient

🩺 Questions to Ask the Patient

1️⃣ Symptoms Suggestive of Bradycardia

- **Syncope / Fainting**: "Have you ever felt faint or lost consciousness?"
- **Dizziness or lightheadedness**: "Do you feel dizzy or lightheaded, especially on standing?"
- **Fatigue / Weakness**: "Do you get unusually tired or weak?"
- **Palpitations**: "Do you notice your heartbeat slowing down or skipping?"
- **Exercise intolerance / shortness of breath**: "Do you get unusually short of breath on activity?"
- **Chest pain / angina**: "Do you ever feel chest pain or pressure?"

2️⃣ Past Medical History / Risk Factors

- **Heart disease**: "Any history of heart attack, heart failure, or valve disease?"
- **Medications**: "Do you take any heart medications, beta-blockers, or calcium channel blockers?"
- **Thyroid disease**: "Any history of thyroid problems?"
- **Electrolyte disorders**: "Do you have kidney problems or take diuretics?"
- **Recent infections**: "Have you been ill recently?"

3️⃣ Family / Social History

- **Sudden cardiac death in family**
- **Athletic lifestyle** (athletes can have benign bradycardia)

4️⃣ Red Flag Symptoms

- Syncope with **chest pain, shortness of breath, confusion** (possible heart block)
- Rapid worsening fatigue or confusion

Station 19 ecg of 2nd degree heart block dx and treatment

🧪 Investigations

1. **12-lead ECG** – confirms type
2. **Continuous Holter monitoring** – if intermittent
3. **Electrolytes, thyroid function** – to rule out reversible causes
4. **Echocardiography** – if structural heart disease suspected

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💊 Management / Treatment

Type I (Mobitz I)

- Usually **asymptomatic** → no treatment
- Symptomatic → **atropine** or temporary pacing

Type II (Mobitz II)

- Often **symptomatic / high risk → permanent pacemaker**
- Temporary pacing and supportive care if unstable
- Treat reversible causes if identified

⚠️ Complications

- Progression to **complete heart block**
- Syncope / sudden cardiac death
- Heart failure if prolonged bradycardia

Station 1:💌 examiner

Women presented with palpitations and exertional dyspnea ,water hammer pulse

Aortic regurgitation scenario interactive station

Causes treatment initial treatment

Especially drugs name don't forget to mention statins

Asked why beta blockers are preffered in AR

Aortic Regurgitation

⚠️ Causes of AR

1️⃣ Chronic / Most Common

- Rheumatic heart disease
- Bicuspid aortic valve

2️⃣ Acute

- Infective endocarditis
- Aortic dissection
- Trauma

3️⃣ Other / Secondary

- Ankylosing spondylitis
- Marfan syndrome / connective tissue disorders

🧪 Investigations

1. **Echocardiography** – confirms diagnosis, assesses severity
2. **CXR** – cardiomegaly (LV dilation)
3. **ECG** – LVH, conduction abnormalities
4. **BNP** – if heart failure suspected

💊 Medical Management (Initial / Chronic)

Goals: Reduce LV afterload, control heart rate, prevent complications

1️⃣ First-line / Preferred Drugs

- **β-blockers** (e.g., **Metoprolol**) → slow heart rate, improve diastolic filling, reduce regurgitant volume
  - **Exam question:** "Why β-blockers?" → They **prolong diastole**, reduce LV volume overload, and prevent tachycardia-induced worsening.
- **ACE inhibitors** (e.g., **Enalapril, Ramipril**) → afterload reduction
- **ARB** (e.g., **Losartan**) → if ACE not tolerated
- **Diuretics** → if symptomatic heart failure

2️⃣ Lipid management

- **Statins** (e.g., **Atorvastatin**) → for atherosclerotic prevention, especially in older patients or mixed valve disease

3️⃣ Other supportive meds

- **Digoxin** → if systolic dysfunction present
- **Anticoagulation** → if AF develops

⚡ Surgical Indications

- Symptomatic severe AR
- LV ejection fraction <50%
- LV end-diastolic dimension >65 mm or end-systolic >50 mm
- Acute severe AR

Surgery: Aortic valve replacement (mechanical or bioprosthetic)

Why β-blockers are used in Aortic Regurgitation

1. **Slow the heart rate** → Heart has **more time to fill and empty properly**.
   - In AR, some blood leaks back into the left ventricle during **diastole**.
   - If the heart beats too fast, there's **less time for proper filling**, so the leak worsens.
2. **Reduce stress on the heart** → Slower heart rate and lower pressure mean the left ventricle doesn't have to work as hard.
3. **Help with symptoms** → Less palpitations, less shortness of breath.

In short:

> β-blockers = "slow the heart, give the ventricle more time to handle the extra blood, and reduce strain."

Station 2:💌
After c section female develop sudden shortness of breath,chest auscultation _right lung middle lobe crepts
Diagnosis :pulmonary embolism
4 risk factors
Investigations to confirm the diagnosis

Pulmonary Embolism

🔑 Risk Factors (Postpartum / Post-Cesarean)

1. **Recent surgery / immobility** – cesarean section → venous stasis
2. **Pregnancy-related hypercoagulability**
3. **Obesity**
4. **Previous history of DVT/PE or thrombophilia**

🧪 Investigations to Confirm Diagnosis

1️⃣ Initial / Screening

- **D-dimer** – sensitive but not specific postpartum
- **ABG** – may show hypoxemia

2️⃣ Imaging (Gold Standard)

- **CT Pulmonary Angiography (CTPA) → investigation of choice**
- **Ventilation-Perfusion (V/Q) scan** – if CTPA contraindicated (e.g., contrast allergy)

3️⃣ Supportive Tests

- **ECG** – may show sinus tachycardia, S1Q3T3 pattern
- **Chest X-ray** – usually normal, may show atelectasis or infiltrates

Station 6 📩 peads Interactive
4 month old infant dry cough sob feeding difficulty...it was scenario about pertussis
Qns asked : Name of organism(bortadella pertussis)
Phases ; 3 ..catarhal , paroxsymal convalescent
Prevention ;Vaccines and its epi scheudle dpt week 6 10 14
Cbc findings(lymphocytosis)
Tx which drug is used (azithromycin doc; macrolides)
Drug used in adult (same)
Protective measures if other family member has the disease?

📌 Diagnosis

Pertussis

🦠 Organism

Bordetella pertussis

📖 Clinical Phases (3)

1️⃣ **Catarrhal phase** (1–2 weeks)

- Mild fever
- Rhinorrhea
- Dry cough
- Highly infectious

2️⃣ **Paroxysmal phase** (2–6 weeks)

- Severe coughing fits
- Inspiratory "whoop"
- Post-tussive vomiting
- Apnea in infants

3️⃣ **Convalescent phase**

- Gradual recovery
- Cough decreases

🧪 CBC Finding

👉 **Lymphocytosis** (very important exa

💊 Treatment

Drug of choice:

- **Azithromycin**

Class:

- **Macrolides**

Other options:

- Clarithromycin
- Erythromycin

In Adults:

👉 Same – **Azithromycin (macrolide)**

💉 Prevention

Vaccine:

- **DPT vaccine**

EPI Schedule (Pakistan):

- 6 weeks
- 10 weeks
- 14 weeks

🏠 Protective Measures for Family Members

If another family member has pertussis:

1️⃣ **Post-exposure prophylaxis:**
👉 **Azithromycin for close contacts** (even if asymptomatic)

2️⃣ Isolate infected person (droplet precautions)

3️⃣ Ensure vaccination status is up to date

4️⃣ High-risk contacts (infants, pregnant women) must receive prophylaxis immediately

Station 7💌 written
A child presented with shortness of breath continous murmur...severe condition thi according to scenerio
Diagnosis
Patent ductus arteriosus
Write management?

in **women**		
Persistence of ductus arteriosus after birth-**hole b/w pulm artery and aorta**). L→R, Common in **LBW, rubella infected babies, females**	Echocardiogram, CXR cardiomegaly, ↑pulm vascular markings	

Continuous machinery-like murmur, **bounding** with **wide pulse pressure**	Breathlessness and sweating while feeding,	In Preterm babies- **indomethacin** Surgery- **ligation and division of ductus**

Station 8:written 💌
Scenario
35 yr old man has cough and night sweats for past 6 weeks and symptoms worse at night
Bilateral wheezing
Family members have same symptoms

Which investigation would confirm the diagnosis?diagnosis was athma according to my brain 🧠
What is the treatment ?
Which device u give him home to monitor _peak flow meter

Bronchial Asthma

This scenario is pointing more toward **bronchial asthma**, not TB 👀

Clues:

- 35-year-old
- Cough worse at night
- Bilateral wheezing
- Family members with similar symptoms → suggests **atopy/genetic tendency**

✅ Investigation to Confirm Diagnosis

The **best confirmatory test:**

- **Spirometry with bronchodilator reversibility test**
- ↓ FEV1
- ↓ FEV1/FVC (<70%)
- After giving salbutamol → FEV1 improves by **≥12% and ≥200 mL**
  → This confirms asthma (reversible airway obstruction)

If spirometry not available:

- Peak expiratory flow variability (>20% variation)

✅ Treatment

- **Acute attack:**

(Short-Acting Beta-2 Agonist)

- Inhaled SABA (Salbutamol)

- **Long-term control:**

- Inhaled corticosteroids (ICS) (first-line controller
- LABA + ICS (if moderate–severe)

(Long-Acting Beta-2 Agonist)

- Leukotriene receptor antagonists (montelukast)

Also:

- Avoid triggers
- Smoking cessation
- Vaccination

✅ Device Given for Home Monitoring

- **Peak Flow Meter**

It measures:

- Peak Expiratory Flow Rate (PEFR)

Used to:

- Monitor asthma control
- Detect early exacerbations
- Assess variability

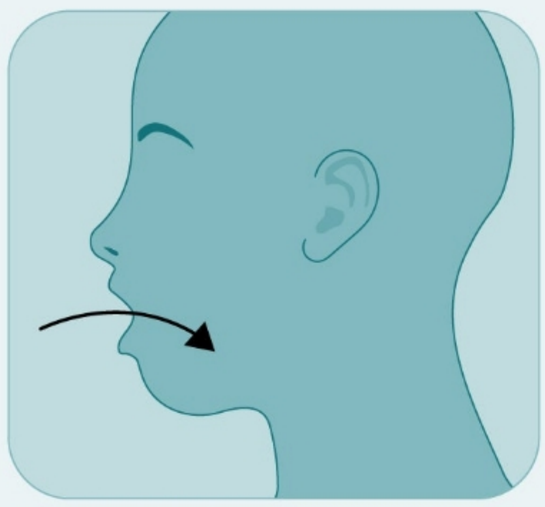
Peak flow meter

Peak expiratory flow measurement

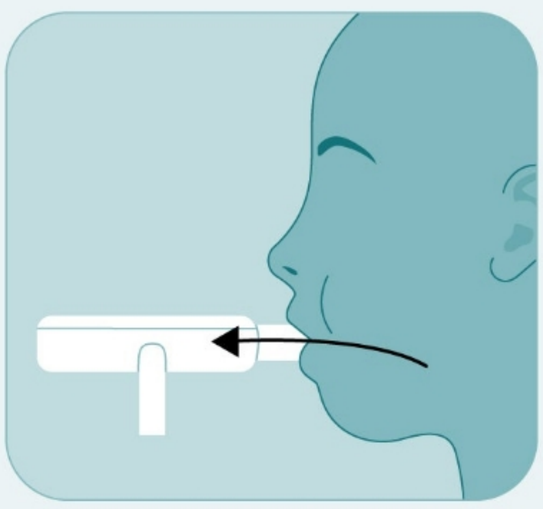
Zone	Meaning
Green	Asthma in good control
Yellow	Asthma not in good control / getting worse
Red	Asthma is severe

HOW TO USE A PEAK FLOW METER

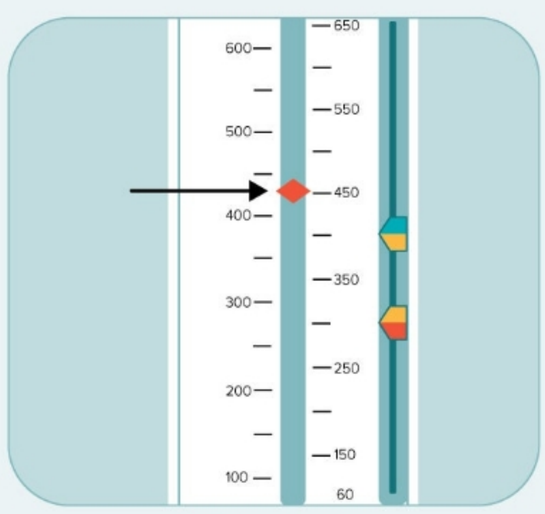
1. Set marker to lowest number



2. Take deep inhale



3. Blow hard and fast in a single blow



4. Read meter

5. Record results, repeat twice

healthline

Station 16 📩 examiner tha observed
:A patient presents with chest pain for the past one hour. His ECG shows ST-segment depression and T-wave inversion. Cardiac biomarkers (Troponin-I and CK-MB) are normal
a) What is the most likely diagnosis?unstable angina
b) How can unstable angina be differentiated from NSTEMI?
c) What is the appropriate treatment for unstable angina.

a) Most likely diagnosis

- **Presentation:** Chest pain for 1 hour.
- **ECG:** ST-segment depression and T-wave inversion.
- **Cardiac biomarkers:** Troponin-I and CK-MB **normal**.

✅ This is classic **Unstable Angina (UA)**.

- **Reasoning:**
  - NSTEMI would have the same ECG changes **plus elevated cardiac biomarkers**.
  - UA: ischemic symptoms + ECG changes **without biomarker elevation**.

b) Differentiation: Unstable Angina vs NSTEMI

Feature	Unstable Angina (UA)	NSTEMI
Chest pain	New, worsening, or at rest	Same as UA
ECG	ST depression / T-wave inversion	ST depression / T-wave inversion
Cardiac biomarkers	**Normal**	**Elevated** (Troponin ± CK-MB)
Myocardial necrosis	None	Present
Risk	Both high risk of MI	Higher risk, already some myocardial injury

Key point: The **biomarker status** is the main differentiator.

c) Treatment of Unstable Angina

1. Immediate management (acute phase)

- **MONA (modified for UA):**
  - **M** – Morphine for pain (if severe)
  - **O** – Oxygen (if SpO_2 < 90%)
  - **N** – Nitrates (sublingual or IV for chest pain)
  - **A** – Aspirin 162–325 mg (chewable, immediately)
- **Anti-platelet therapy:**
  - **Aspirin** + **P2Y12 inhibitor** (e.g., clopidogrel)
- **Anticoagulation:**
  - **LMWH** or **unfractionated heparin**
- **Beta-blockers:** Unless contraindicated
- **Statins:** High-intensity statin therapy
- **ACE inhibitors:** If hypertensive, diabetic, or LV dysfunction

2. Risk stratification for further therapy

- **High-risk features:** recurrent pain, dynamic ECG changes, diabetes, reduced EF, elevated BNP.
- **Coronary angiography ± PCI** may be indicated based on risk.

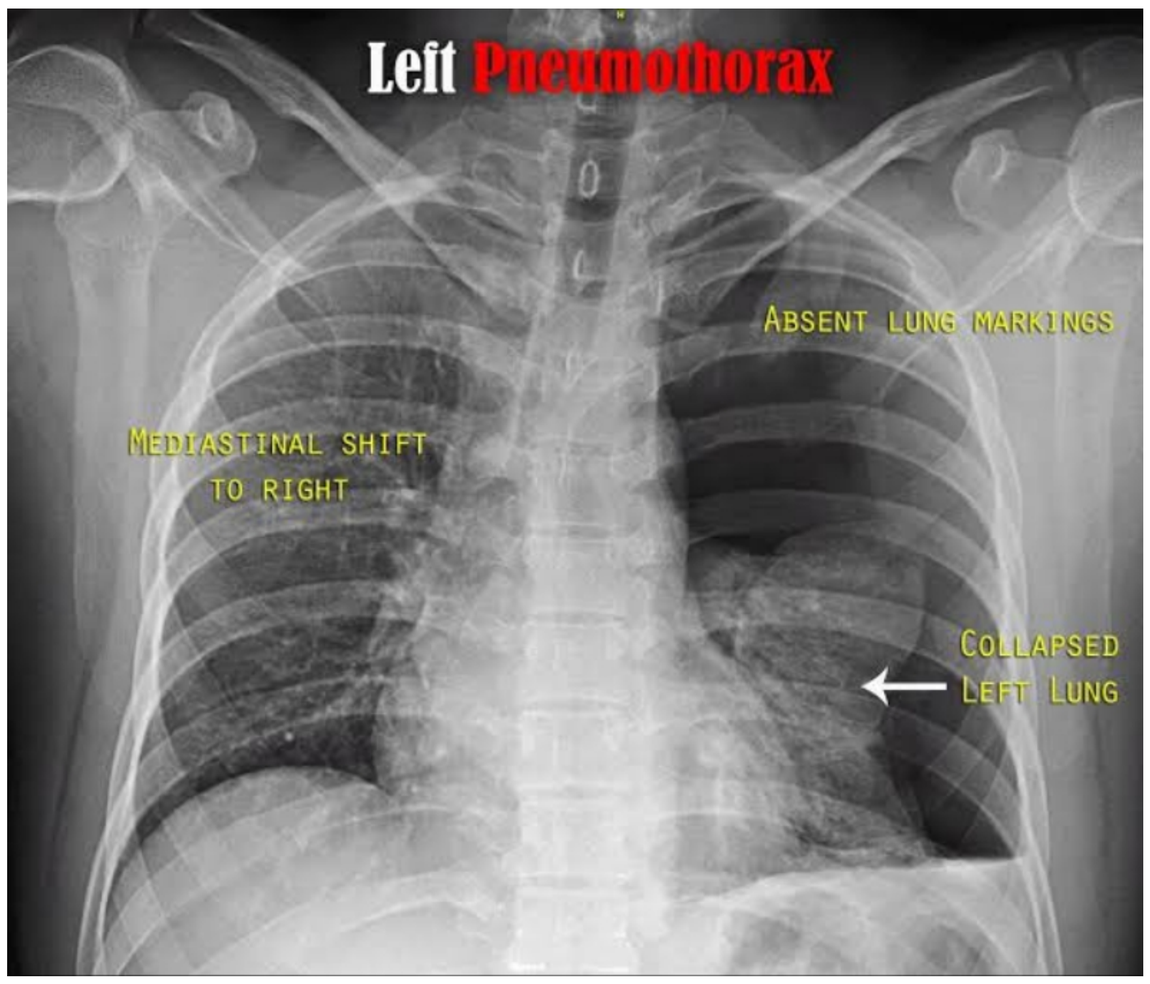
Station 15💌:observed

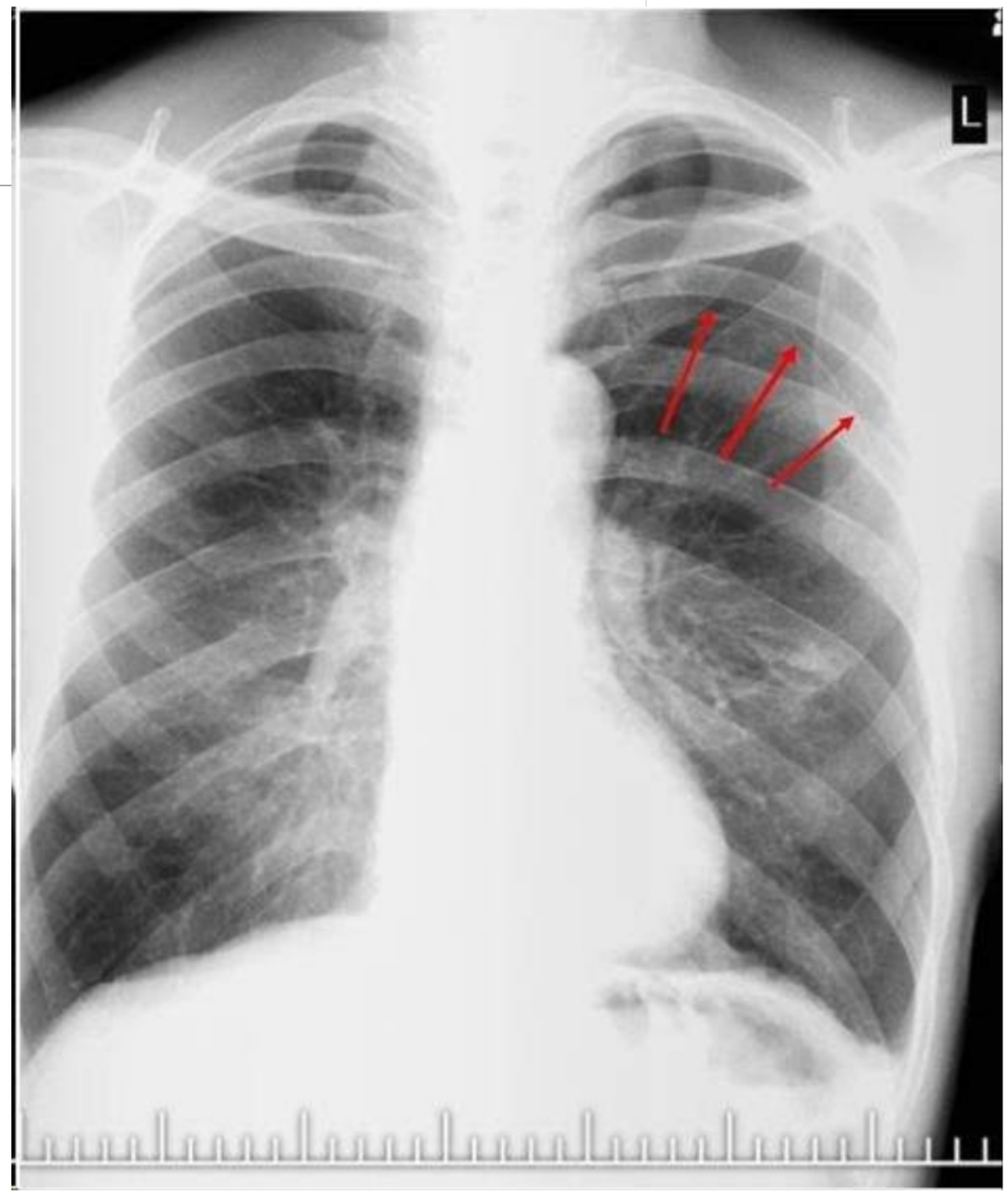
A chest X ray of Spontaneous Primary Pneumothorax (left sided):

Types

Diagnosis

Management





Spontaneous Primary Pneumothorax (SPP) – Left-sided

1. Types of Pneumothorax

1. **Primary Spontaneous Pneumothorax (PSP)**
   - Occurs **without underlying lung disease**.
   - Usually in **tall, thin young males** (age 10–30).
   - Often due to rupture of **subpleural blebs**.
2. **Secondary Spontaneous Pneumothorax (SSP)**
   - Occurs **with underlying lung disease** (COPD, TB, CF, pneumonia).
   - More severe, higher risk of complications.
3. **Traumatic Pneumothorax**
   - Due to **blunt or penetrating trauma** or iatrogenic (e.g., central line, mechanical ventilation).
4. **Tension Pneumothorax**
   - Life-threatening, mediastinal shift, hypotension.
   - Can arise from any of the above types if air accumulates under pressure.

> In your case: **Primary spontaneous, left-sided.**

2. Diagnosis (Chest X-ray features)

CXR Findings (PA view preferred):

1. **Visible pleural line:**
   - A thin, sharp white line representing the **visceral pleura**, separated from the chest wall.
2. **Absent lung markings beyond pleural line:**
   - The area between pleural line and chest wall appears **radiolucent (dark)**.
3. **Deep sulcus sign** (on supine X-ray):
   - Costophrenic angle appears abnormally deep if patient supine.
4. **Lung collapse:**
   - Degree can be **small (<2 cm apex to cupula)** or **large (>2 cm)**.
5. **Mediastinum usually normal** in primary pneumothorax (no tension).

Optional:

- **CT scan** is not needed for routine diagnosis, but can identify **blebs or bullae** if recurrent.

3. Management

Depends on size, symptoms, and stability:

Type/Severity	Management
Small (<2–3 cm), asymptomatic or mild	- **Observation** with **O_2 supplementation** (high-flow O_2 helps reabsorption) - Serial CXRs to monitor resolution
Large (>2–3 cm) or symptomatic	- **Needle aspiration** (14–16G cannula, anterior 2nd ICS midclavicular line) - **Chest tube thoracostomy** (intercostal drain, usually 4–5th ICS midaxillary line)
Recurrent or persistent air leak	- **Surgical intervention** (VATS – video-assisted thoracoscopic surgery, pleurodesis)

Supportive Care:

- Analgesics for pain
- Oxygen therapy speeds resolution
- Avoid air travel until resolved
- Smoking cessation (prevents recurrence)

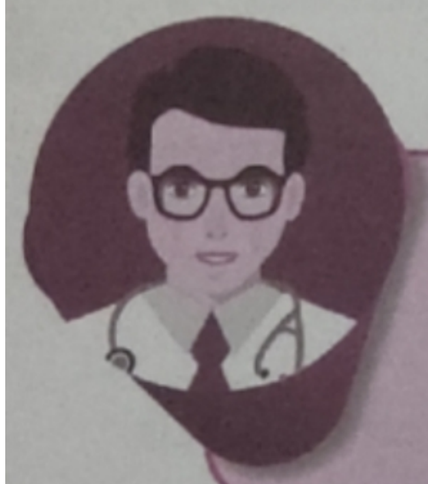
I. Tension Pneumothorax:

- **Introduction:**
  - It develops when a one-way valve air leak occurs from the lung or through the chest wall and air is forced into the pleural space without any means of escape, completely collapsing the affected lung.

- **Causes:**
  - Penetrating chest trauma
  - Blunt chest trauma with parenchymal lung injury
  - Iatrogenic lung punctures (e.g. central line placement)
  - Mechanical positive pressure ventilation

- **Clinical Features:**
  - Dyspnea, chest pain, and air hunger
  - Tachycardia and tachypnea,

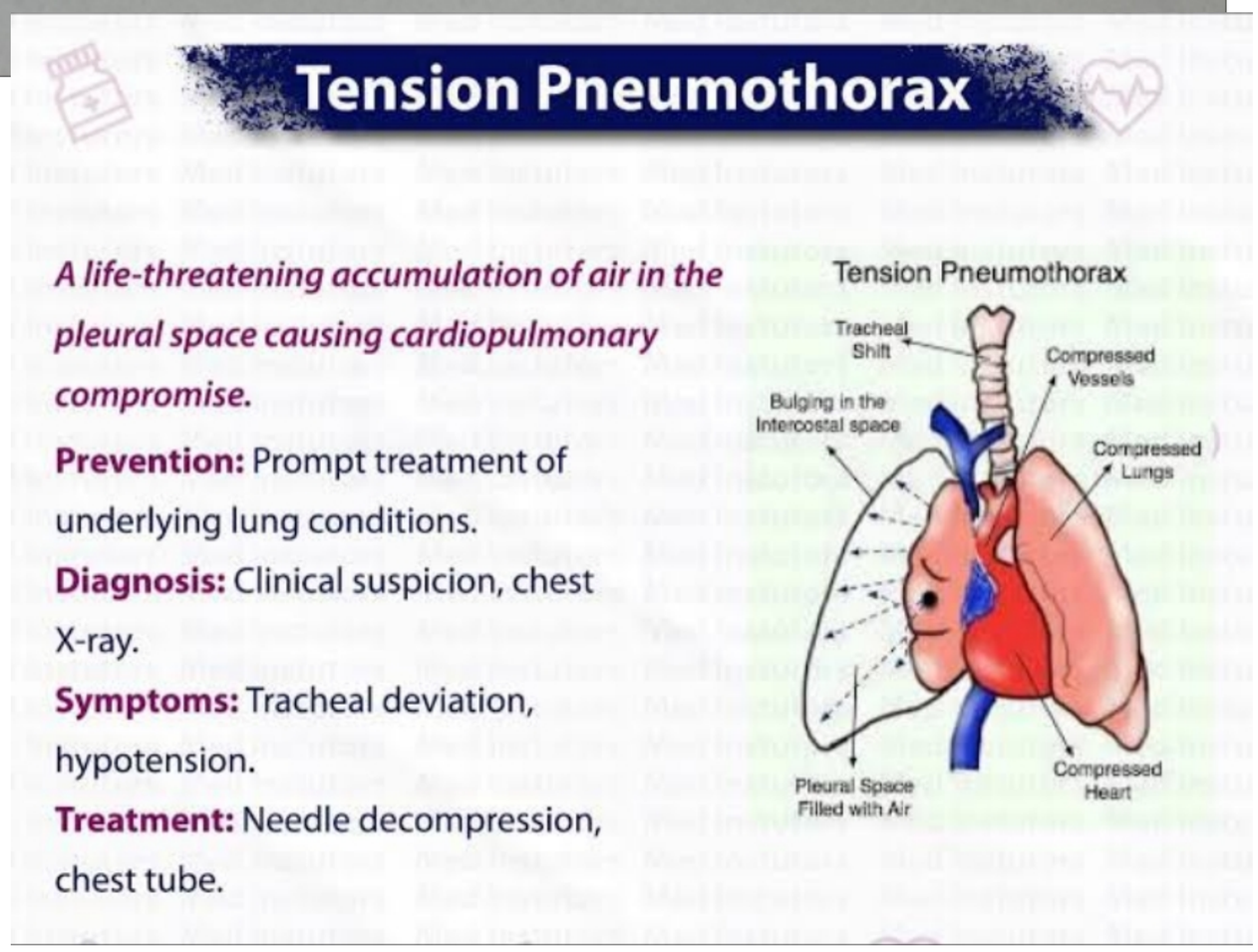
  - Hypotension
  - **Tracheal deviation AWAY from the side of injury**
  - **Absent breath sounds on the affected hemi-thorax.**
  - **Hyper-resonant percussion note on the affected hemi-thorax**
  - Raised jugular venous pressure (JVP)



Tension Pneumothorax:

- It is a Clinical Diagnosis and treatment should not be delayed by waiting for radiologic confirmation.

- **Treatment:**
  - Initial Management:
    - **"Needle decompression"** – i.e. insertion of a large-bore needle into the 2nd intercostal space in the mid-clavicular line of affected hemi-thorax.
  - Definite Management:
    - **"Chest tube insertion"** aka **"tube thoracostomy"** – needle decompression is followed by insertion of a chest tube through the 5th intercostal space in the anterior axillary line.



A life-threatening accumulation of air in the pleural space causing cardiopulmonary compromise.

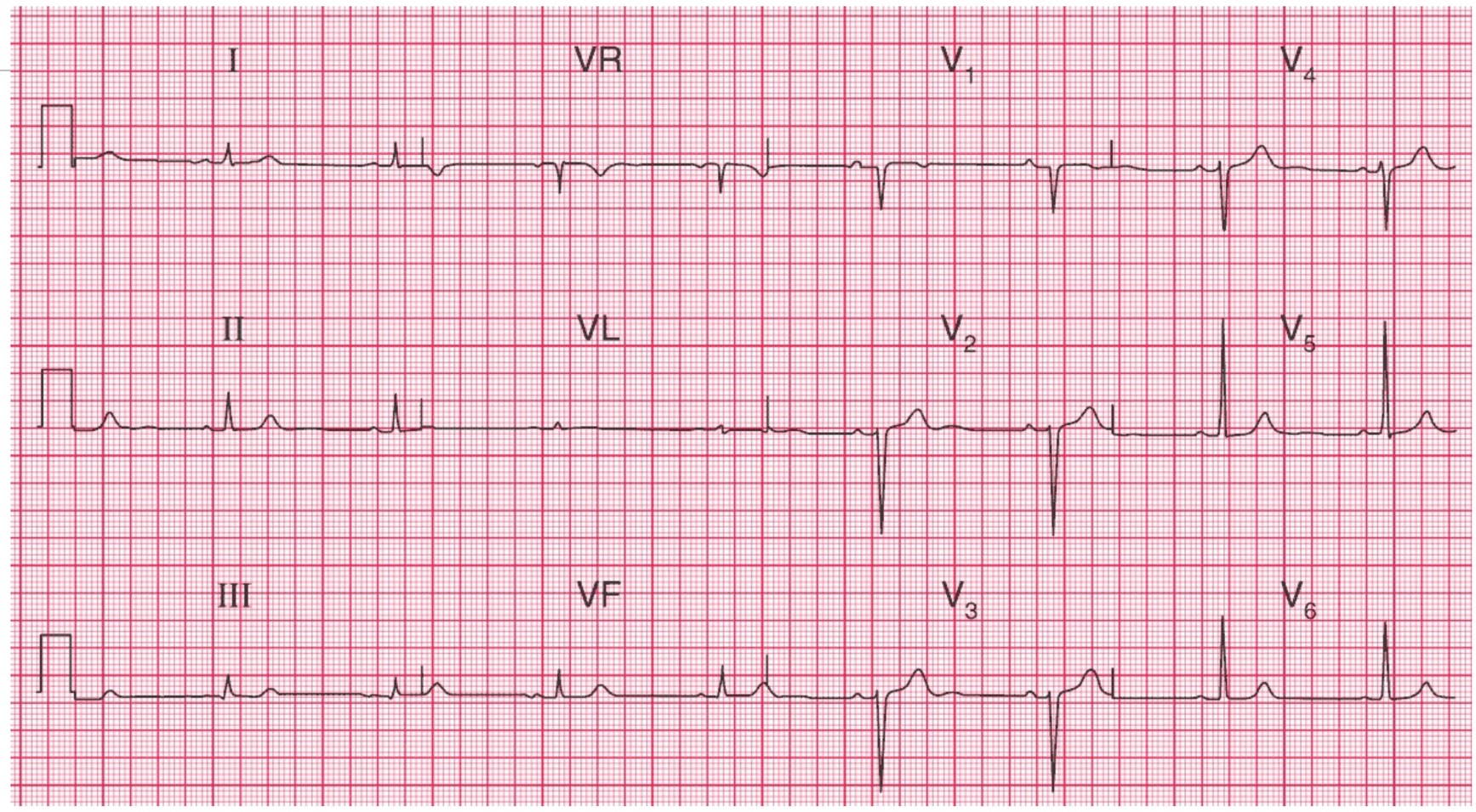
Prevention: Prompt treatment of underlying lung conditions.

Diagnosis: Clinical suspicion, chest X-ray.

Symptoms: Tracheal deviation, hypotension.

Treatment: Needle decompression, chest tube.

Station 14📩: Ecg was given and we identify .... Ant wall myocardial infarction. And write 4 investigation



ST-segment elevation in anterior leads:

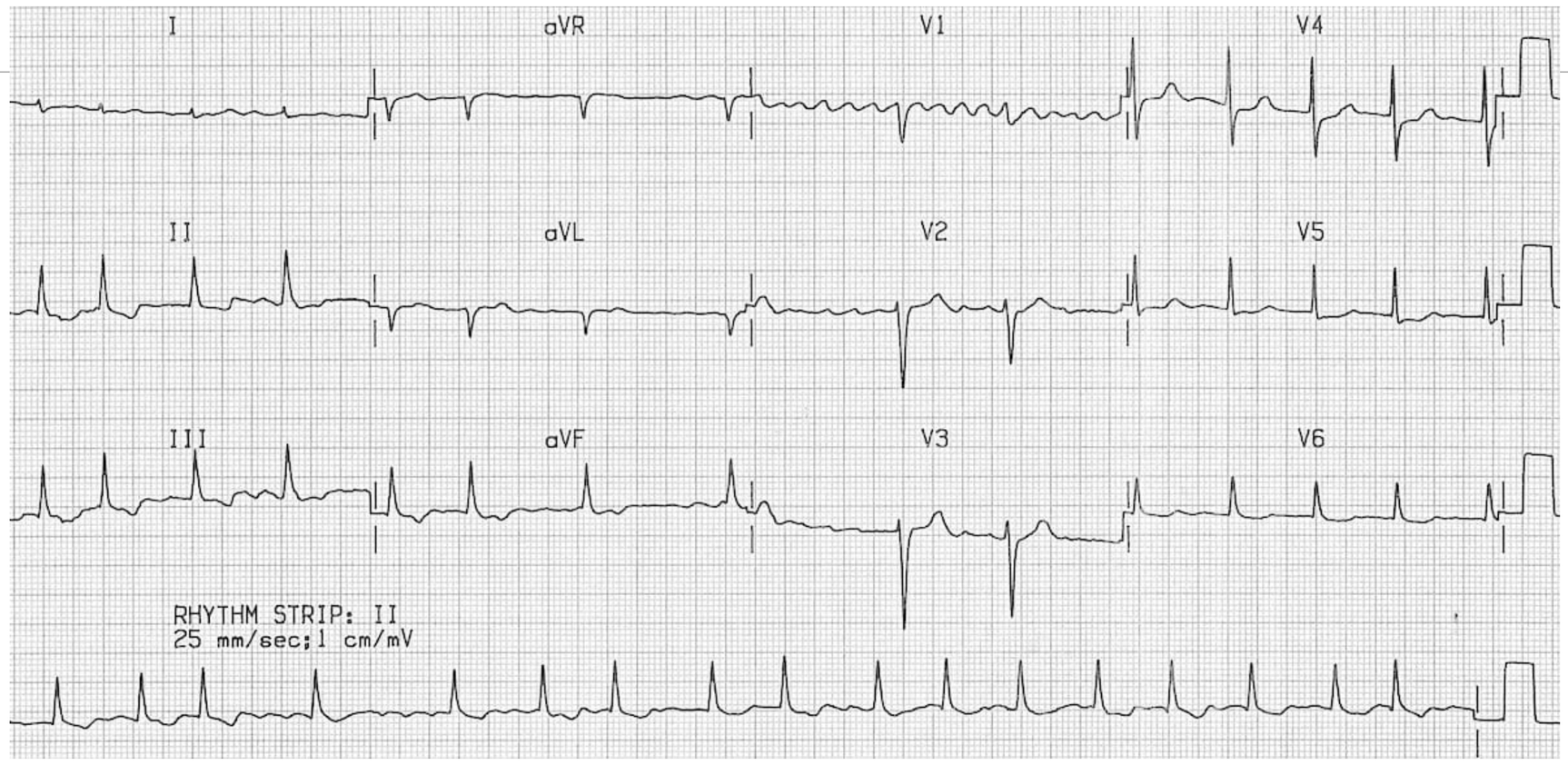
V1–V4 (sometimes V5–V6 if large)

Investigation	Purpose / Finding
ECG	ST elevation V1–V4, reciprocal depression II, III, aVF
Cardiac biomarkers	Troponin I/T ↑, CK-MB ↑
Echocardiography	Anterior wall hypokinesia, LV function
Coronary angiography	LAD occlusion, guide PCI/CABG
CXR	Rule out other causes, pulmonary edema
Labs (CBC, electrolytes, renal)	Baseline & risk stratification

Station 13:💌 ECG of a 70 year old man who had an ischaemic stroke in the past

diagnosis Atrial Fibrillation

Management

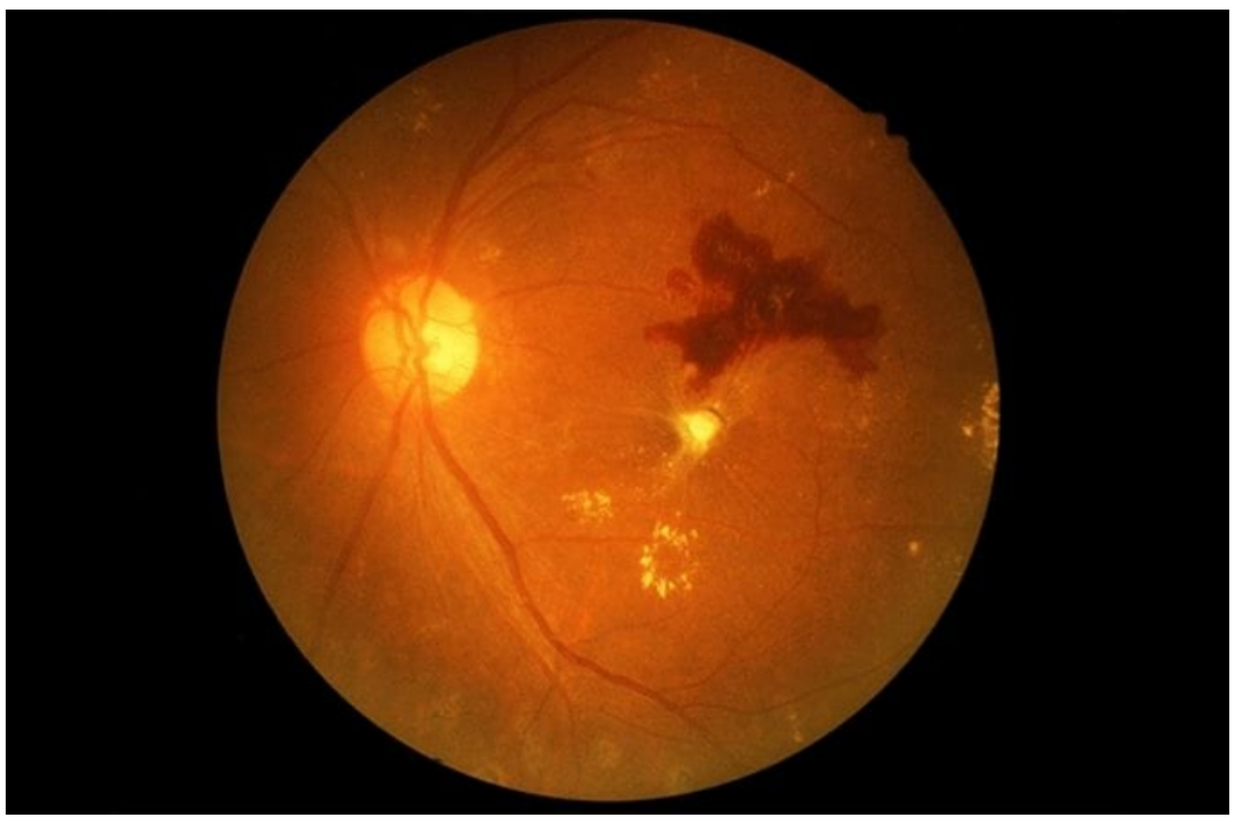


- Irregularly irregular rhythm
- No P waves
- Absence of an isoelectric baseline
- Variable ventricular rate
- QRS complexes usually < 120ms, unless pre-existing bundle branch block, accessory pathway, or rate-related aberrant conduction
- Fibrillatory waves may be present and can be either fine (amplitude < 0.5mm) or coarse (amplitude > 0.5mm)
- Fibrillatory waves may mimic P waves leading to misdiagnosis

Diagnosis	AF (irregularly irregular, absent P waves)
Stroke prevention	DOACs preferred, warfarin if needed
Rate control	Beta-blockers, CCBs, digoxin
Rhythm control	Only if symptomatic or heart failure
Risk factor management	HTN, diabetes, alcohol, lifestyle

Station 12💌

Retinal hemorhages image and 4 causes ?



1. Vascular / Hypertensive Causes

- **Hypertensive retinopathy** – sudden rise in BP can cause flame-shaped hemorrhages
- **Diabetic retinopathy** – dot and blot hemorrhages, often with microaneurysms
- **Retinal vein occlusion** – central or branch (blood and thunder appearance)

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2. Hematologic / Blood Disorders

- **Anemia / severe hypoxia** – often in sickle cell anemia
- **Thrombocytopenia / platelet disorders** – spontaneous retinal bleeding
- **Leukemia / blood dyscrasias** – widespread retinal hemorrhages

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3. Trauma

- **Direct ocular trauma** – commotio retinae, retinal hemorrhage
- **Shaken baby syndrome / non-accidental injury** – bilateral retinal hemorrhages

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4. Valsalva / Increased Venous Pressure

- **Valsalva retinopathy** – sudden rise in intrathoracic pressure (coughing, vomiting, heavy lifting)
- **Intrathoracic pressure spikes** – can rupture small retinal vessels

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5. Infections / Inflammatory Causes

- **Endocarditis** – Roth spots (hemorrhage with white center)
- **Sepsis / septic emboli** – can cause retinal hemorrhages
- **HIV retinopathy**

Station 11Examine the chest of patient and tell findings on Harvey
It was bilateral wheezing
Conditions in which u hear wheeze ?

Wheeze / Crackles difference.

Causes of Wheezing in Children and Infants

- **Common:**

Allergies
Asthma or reactive airway disease
Gastroesophageal reflux disease
Infections
Bronchiolitis
Bronchitis
Pneumonia
Upper respiratory infection
Obstructive sleep apnea

- **Uncommon:**

Bronchopulmonary dysplasia
Foreign body aspiration

- Rare:

Bronchiolitis obliterans
Congenital vascular abnormalities
Congestive heart failure
Cystic fibrosis
Immunodeficiency diseases
Mediastinal masses
Primary ciliary dyskinesia
Tracheobronchial anomalies
Tumor or malignancy
Vocal cord dysfunction

Farmer suddenly develops shortness of breath , chest pain, on examination there were basal inspiratory crakles and clubbing. What is ur diagnosis? (pulmonary fibrosis),
investigations that can be done for this (pfts, ct chest, chest x ray,)..
name any 2 conditions in which inspiratory crakles can be heard? (heart failure, pulmonary fibrosis , pneumonia).

Most Likely Diagnosis

✅ **Pulmonary Fibrosis** (likely **farmer's lung / hypersensitivity pneumonitis**)

Reasoning:

- Basal inspiratory crackles → "Velcro crackles" typical of interstitial lung disease
- Clubbing → chronic hypoxia
- Farmer → exposure to organic dust → hypersensitivity pneumonitis leading to pulmonary fibrosis

Investigations

Investigation	Findings / Purpose
Chest X-ray (CXR)	Reticular or reticulonodular pattern, basal predominance, volume loss
High-Resolution CT (HRCT) Chest	Ground-glass opacities, honeycombing, traction bronchiectasis (definitive for fibrosis)
Pulmonary Function Tests (PFTs)	Restrictive pattern: ↓ FVC, ↓ TLC, ↑ FEV1/FVC ratio
Oxygen saturation / ABG	Hypoxia in advanced disease

Optional: Lung biopsy if uncertain

Conditions in which inspiratory crackles can be heard

1. **Pulmonary Fibrosis** (interstitial lung disease) – basal, fine "Velcro-like"
2. **Heart Failure** – pulmonary edema → coarse basal crackles
3. **Pneumonia** – coarse crackles over affected lobe

Tip for Exams:

- **Fine basal crackles** → fibrosis
- **Coarse crackles** → heart failure or pneumonia

Status asthmaticus.

Danger signs.

Management.

Complications.

1. Definition

Status asthmaticus is a severe acute asthma attack that **fails to respond to standard inhaled β2-agonist therapy** and can lead to respiratory failure.

2. Danger Signs

Recognizing these early is critical. Danger signs indicate severe airflow obstruction or impending respiratory failure:

Clinical Danger Signs:

- Silent chest (no wheeze) – indicates severe obstruction
- Inability to speak full sentences
- Exhaustion or confusion
- Cyanosis (lips/fingernails)
- Use of accessory muscles, intercostal retractions
- Tachypnea >30–40 breaths/min
- Tachycardia >120–130 bpm (or bradycardia in impending arrest)
- Hypotension (late sign)
- Peak expiratory flow (PEF) <50% of predicted

Investigations danger signs:

- Hypoxemia (PaO_2 <60 mmHg)
- Hypercapnia ($PaCO_2$ >40 mmHg) — **normal or high CO_2 is ominous** in severe asthma
- Severe acidosis (pH <7.3)

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3. Management

Management is stepwise and often requires ICU care.

A. Initial / Emergency Treatment

1. **Oxygen therapy:**
    - Maintain SpO_2 94–98%
    - High-flow oxygen if needed
2. **Bronchodilators:**
    - **Inhaled SABA (Salbutamol / Albuterol):** via nebulizer, high dose q20 min initially
    - Consider **Ipratropium bromide** nebulization for additive effect
3. **Systemic corticosteroids:**
    - Oral or IV prednisolone / methylprednisolone
    - Reduces inflammation; onset takes hours, but essential
4. **Adjuncts:**
    - Magnesium sulfate IV (for severe, refractory cases)
    - Consider aminophylline infusion (rare, only in ICU under monitoring)
5. **Monitoring:**
    - Continuous SpO_2, HR, RR, BP
    - Frequent PEF or clinical assessment
    - ABGs if patient deteriorates

B. Escalation / ICU Care

- **Intubation & mechanical ventilation** if:
    - Severe hypoxemia or hypercapnia
    - Exhaustion, altered mental status
    - Silent chest

4. Complications

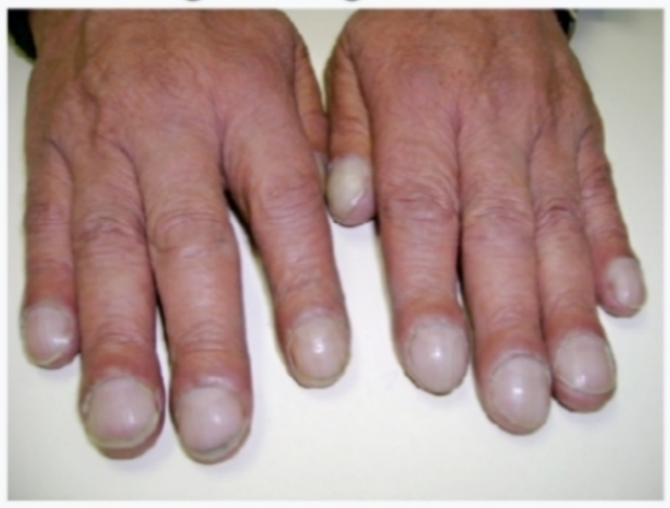
Status asthmaticus can lead to:

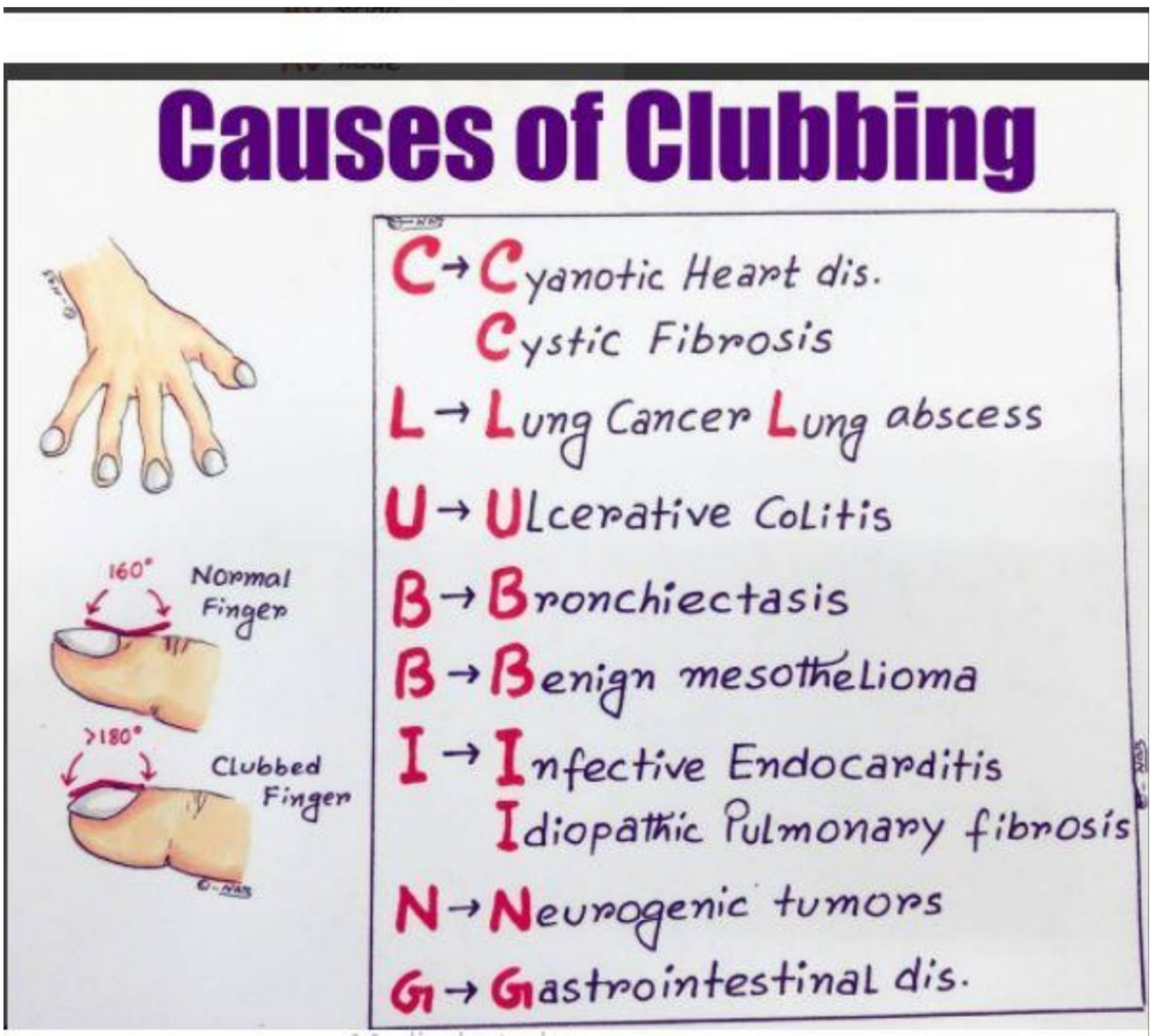
- **Respiratory failure**
- **Hypoxemia & hypercapnia**
- **Pneumothorax or pneumomediastinum** (due to air trapping)
- **Cardiac arrhythmias** (from hypoxia or β-agonist therapy)
- **Hypotension / shock**
- **Death** if untreated

Clubbing positive.

Question: 3 conditions in which clubbing happens.

Clubbing of finger & toes is commonly seen in cyanotic heart diseases, IE, atrial myxoma in addition to pulmonary/GI diseases & primary biliary cirrhosis.





Hypertension management.

What is refractory hypertension.

When will the patient come for follow-up.

1. Hypertension Management

Management is based on **blood pressure (BP) stage**, **comorbidities**, and **risk factors**.

A. Lifestyle Modifications (for all patients)

- **Weight reduction:** BMI 18.5–24.9 kg/m²
- **Diet:** DASH diet, reduce salt (<5g/day), increase fruits & vegetables
- **Exercise:** 30 min moderate intensity most days
- **Limit alcohol** and **stop smoking**
- **Stress management**

B. Pharmacologic Therapy

- First-line agents (choose based on comorbidities):
  1. **Thiazide diuretics** (e.g., hydrochlorothiazide)
  2. **ACE inhibitors** (e.g., enalapril, ramipril)
  3. **ARBs** (e.g., losartan)
  4. **Calcium channel blockers** (e.g., amlodipine)

Combination therapy is often needed if BP ⩾20/10 mmHg above target.

Special Considerations

- **Diabetes / CKD:** ACEi or ARB preferred
- **Heart failure / post-MI:** Beta-blocker + ACEi

C. BP Targets

- <130/80 mmHg (general population, high-risk patients may need stricter control)

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2. Refractory (Resistant) Hypertension

Definition: BP remains **above target despite adherence to 3 antihypertensives of different classes (including a diuretic)** at optimal doses.

Causes to consider:

- Poor adherence
- Secondary hypertension (renal artery stenosis, hyperaldosteronism, pheochromocytoma)
- Drug interactions (NSAIDs, steroids, OCPs, etc.)
- Obesity, high salt intake

Next steps:

- Confirm adherence
- Rule out secondary causes
- Consider adding **mineralocorticoid receptor antagonist (spironolactone)**
- Refer to specialist / hypertension clinic

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3. Follow-up Schedule

- **Initial phase (after starting or changing therapy):**
  - **2–4 weeks** to assess response and side effects
- **Once BP controlled:**
  - Every **3–6 months**
- **If unstable / uncontrolled / comorbidities:**
  - More frequent, e.g., **monthly**
- **Home monitoring:**
  - Encourage **home BP checks** to track trends

Station 15: Child diagnosed with VSD.

What findings do you expect?

Pathophysiology	Diagnosis (echo)
Hole in the ventricular septum (membranous or muscular part) **L→R shunt**, **biventricular hypertrophy→ pulm HTN**	Echocardiogram, Catheterization, CXR- **enlarged proximal pulmonary arteries**

Heart Murmurs	Signs and Symptoms
Pansystolic/Holosystolic murmur in lower left sternal border, **loud S2** (pulm HTN)	Fatigue Shunt reversal R→L /Eisenmenger effect- cyanosis **bulging sternum, systolic thrill**

Management

Small-Observation
CHF- digoxin + diuretics,
Pulm HTN/volume overload/supracristal VSD- surgery

Ventricular Septal Defect

3. Cardiovascular Examination

Feature	Small VSD	Large VSD
Precordial activity	Normal	Hyperdynamic apex
Heart sounds	Normal S1 & S2	S2 may be **accentuated**
Murmur	**Harsh holosystolic murmur** at **lower left sternal border (LLSB)**	Softer or less intense due to rapid equalization of pressures
Thrill	Possible in moderate/large VSD	Often palpable in large VSD
Pulmonary flow	Normal	Increased pulmonary flow → possible **pulmonary hypertension** signs
Other	Usually none	Possible **diastolic rumble at apex** (functional mitral flow murmur)

Classic auscultation point:

- **Holosystolic murmur at LLSB**, often radiates to the **right sternal border**.

4. Investigations

- **Chest X-ray:**
  - Small VSD → normal
  - Large VSD → cardiomegaly, **increased pulmonary vascular markings**
- **ECG:**
  - Small VSD → normal
  - Large VSD → **left atrial enlargement**, **left ventricular hypertrophy**, sometimes **right ventricular hypertrophy** if pulmonary hypertension develops
- **Echocardiography:**
  - Confirms **size, location, and flow direction**
  - Shows **shunt magnitude and pulmonary pressure**
- **Cardiac catheterization:**
  - Rarely needed, used if **pulmonary hypertension** or **complex defects**

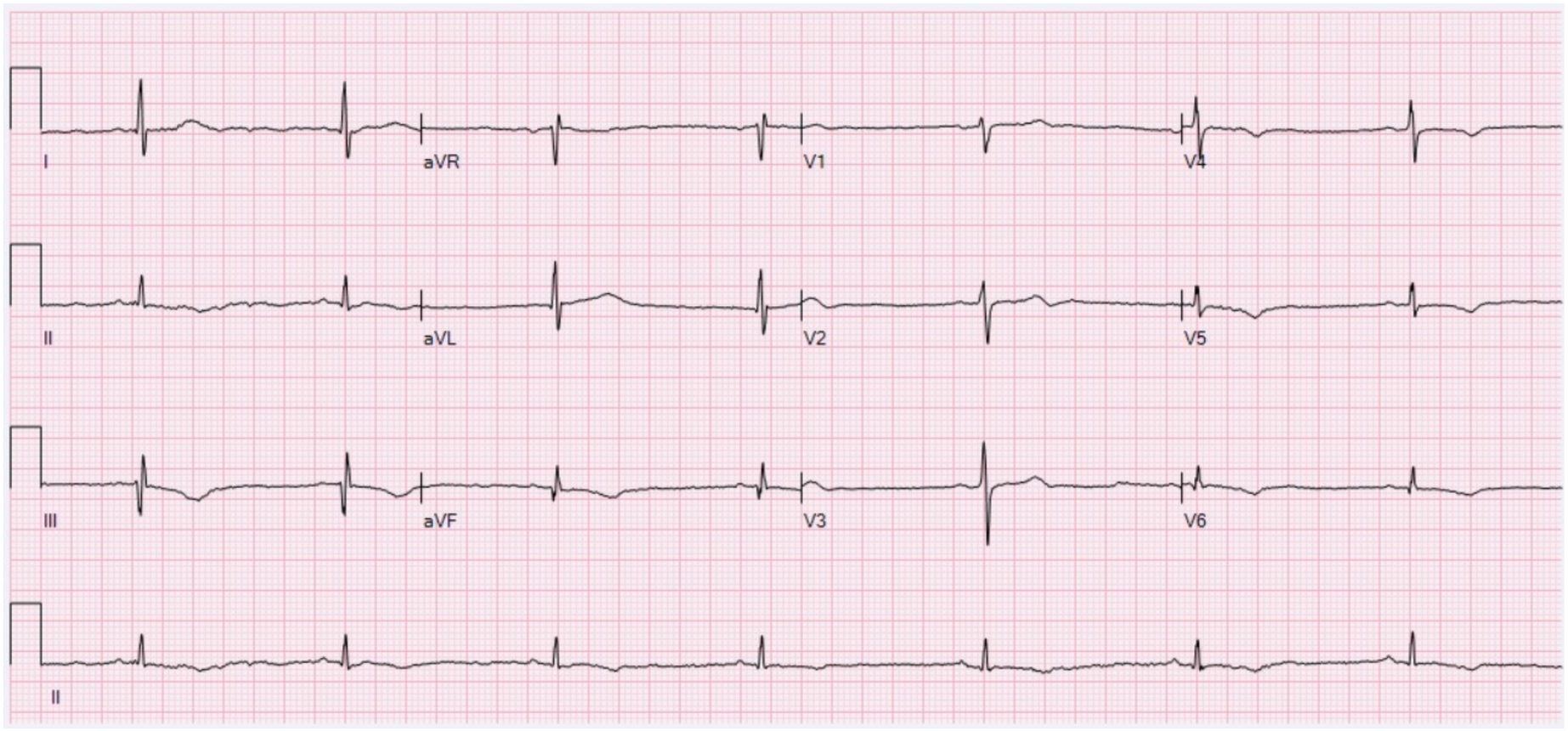
Acyanotic Congenital Heart Anomalies:

Congenital Heart Anomaly VCP3A3	**Pathophysiology**	**Diagnosis (echo)**	**Heart Murmurs**	**Signs and Symptoms**	**Management**
Ventricular Septal Defect (VSD)	Hole in the ventricular septum (membranous or muscular part) **L→R shunt**, **biventricular hypertrophy→ pulm HTN**	Echocardiogram, Catheterization, CXR- **enlarged proximal pulmonary arteries**	**Pansystolic/Holosystolic** murmur in lower left sternal border, **loud S2** (pulm HTN)	Fatigue Shunt reversal R→L /Eisenmenger effect- cyanosis **bulging sternum, systolic thrill**	Small-Observation CHF- digoxin + diuretics, Pulm HTN/volume overload/supracristal VSD- surgery
Atrial Septal Defect (ASD)	Hole in the atrial septum**. L→R** High in septum: sinus venosus Mid: ostium secundum **(m/c)** Low: ostium primium	Echocardiogram **septum moves in paradoxical way**, Catheterization	**split S2, systolic murmur**	Secundum-asympt, Primium- fatigue, recurrent PNA, MR	Bact **endocarditis prophylaxis**, definitive is Surgery
Coarctation of the Aorta	**Narrowing of the aorta** at origin of left subclavian artery near ligamentum arteriosum, associated with **turner** syndrome in **women**	BP, Echocardiogram, CXR **notching of ribs** & **figure 3 sign**	**Systolic murmur** may be heard over the back or abdomen	**HTN in arms but ↓BP & pulse in lower extremities, radio-femoral delay**	**Surgical** decompression, or percutaneous balloon arthroplasty **>2years**
Patent Ductus Arteriosus (PDA)	Persistence of ductus arteriosus after birth-**hole b/w pulm artery and aorta**). L→R, Common in **LBW, rubella infected babies, females**	Echocardiogram, CXR cardiomegaly, ↑pulm vascular markings	**Continuous machinery-like murmur**, **bounding** with **wide pulse pressure**	Breathlessness and sweating while feeding,	In Preterm babies- **indomethacin** Surgery- **ligation and division of ductus**
Patent foramen ovale	**Hole in atrial septum** that closes at birth normally	Bubble echo	murmur	**Closes spontaneously**	Observe, **surgery** closure if **symptoms** occur
Aortic Stenosis	Narrowing of the aortic valve or aorta. Most common form- valvular	Echocardiogram, Catheterization	**Mid systolic crescendo decrescendo murmur**, soft S2	Chest pain, Fainting, Fatigue	Medications, **Balloon valvuloplasty**, Surgical
Pulmonary Stenosis	Narrowing of the pulmonary valve or artery	Echocardiogram, Catheterization	**Mid systolic crescendo decrescendo murmur**	Cyanosis, Fatigue, Shortness of breath	**Balloon valvuloplasty**, Surgical
Atrioventricular Canal Defect	**Incomplete fusion of atrial and ventricular septa and malformation of AV valves**	Echocardiogram, Catheterization	**Holosystolic murmur**	Poor weight gain, Respiratory infections	Surgical repair

Cyanotic Congenital Heart Anomalies:

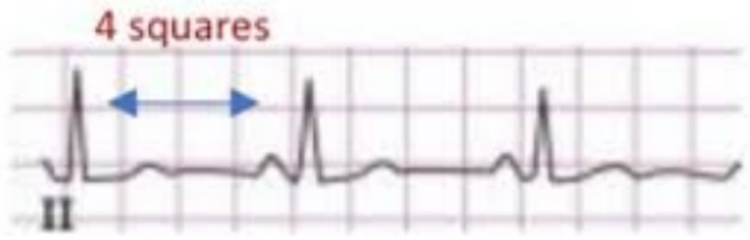
Congenital Heart Anomaly T5E	Pathophysiology	Diagnosis (echo)	Heart Murmurs	Signs and Symptoms (clubbing)	Management
Tetralogy of Fallot	Combination of **four** heart defects (**PVOR**)-pulmonary stenosis-VSD-over riding of aorta over ventricular septum-right ventricular hypertrophy	Echocardiogram, Chest X-ray: **boot shaped heart**+ diminished pulmonary vascular markings '**oligemic lung fields'**	**Systolic ejection** (pulmonic stenosis), **holosystolic murmur** (VSD)	Cyanotic attacks/**blue spells** usually in morning after crying, Dyspnea	**Squatting** (↓left-right shunt by ↑SVR), iron supplements, **endocarditis prophylaxis**, **Palliative** surgery- Blalock-taussig, waterston, potts shunt Surgical repair (total correction)
Transposition of the Great Arteries (TGA)	**Switched positions of the pulmonary artery and aorta**, two parallel circuits m/c infants of **diabetic mothers**	Echocardiogram, Arterial blood gases, CXR; **egg** placed on its side	**Single loud** second heart sound **(S2), systolic murmur**	Cyanosis tachypnea **within 1hr of birth**, large babies (TGA w/ VSD), Poor feeding	**PGE IV** after birth, digoxin, diuretics, Surgery after 2 wks age but in the meantime -**Rashkind/BAS, mustard procedure**
Truncus Arteriosus	**Single large vessel arises from the heart**, instead of separate aorta and pulmonary arteries	Echocardiogram, **Catheterization**, CXR; **waterfall/hilar comma sign**	**Single loud heart sound, Continuous murmur**	Cyanosis, Poor feeding, **diaphoresis**	Surgical repair
Total Anomalous Pulmonary Venous Drainage (TAPVD)	Pulmonary veins don't connect normally to the left atrium	Echocardiogram, Cardiac **catheterization**	**Loud S2**	Cyanosis, Rapid breathing, feeding intolerance, **pulmonary edema**	Surgical repair (reattaching pulmonary veins to the left atrium)
Tricuspid Atresia	Absence of tricuspid valve	Echocardiogram, **Catheterization**	**Single loud S2, maybe S3**	Cyanosis, trouble feeding	Symptomatic (**digitalis** diuresis for congestion) surgery **blalock-taussig shunt**
Ebstein Anomaly	Malformation of the tricuspid valve, displacement towards the apex, backflow into right atrium Also with ASD	Echocardiogram, right atrial enlargement (**tall broad p waves**)	**Systolic murmur** (Tricuspid regurgitation), **S3,S4**	Cyanosis, **Heart failure**	Medications, Surgery **Da silva's cone repair**

Station 13: ECG – Sinus bradycardia.



REGULAR rhythm

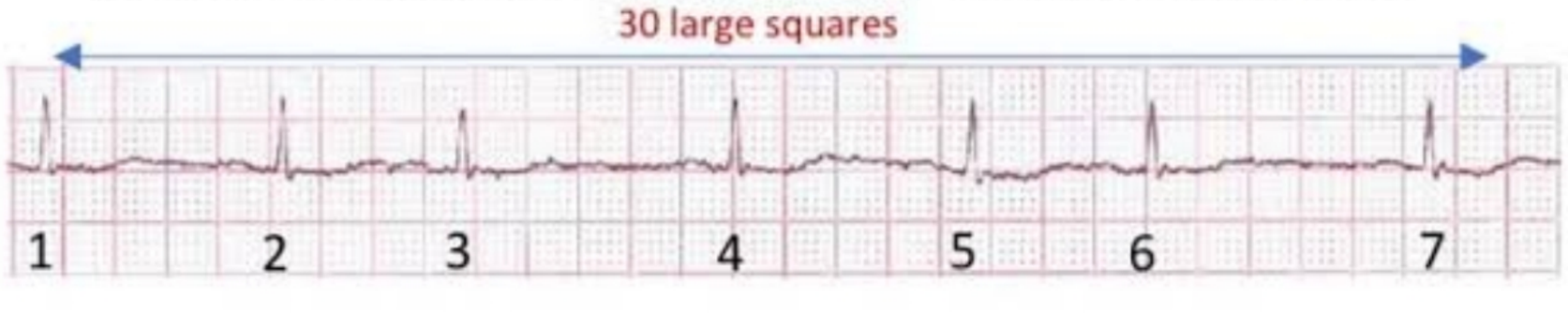
300 ÷ (*number of large squares between QRS complexes*)



300 **÷ 4 = 75bpm**

IRREGULAR rhythm

Total number of QRS complexes over 30 big squares (x10)

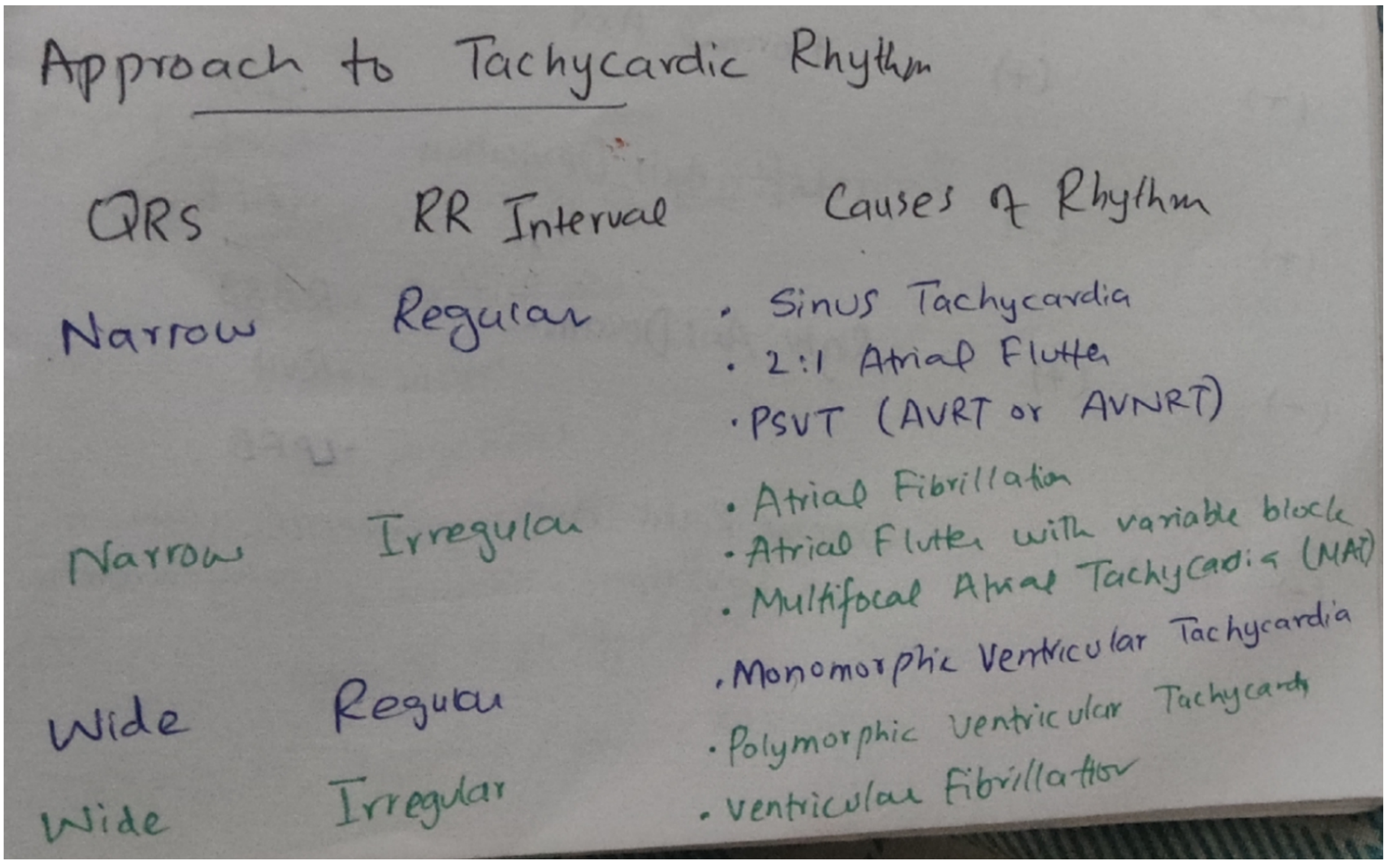


7 (QRS complexes) **x 10 = 70bpm**

STEP WISE APPROACH OF ECG

1. Rate
2. Rhythm
3. Axis
4. Intervals
5. P wave
6. QRS complex
7. ST Segment and T wave

Rate 300 ÷ No. of big boxes



Approach to Bradycardic Rhythm

PR Interval	QRS	Cause of Rhythm
Normal	No drop in QRS	• Sinus Bradycardia
Prolonged	"	• 1st Degree AV Block
Progressively Prolonged	Drops QRS	• 2nd Degree AV Block (Mobitz I)
Constant	"	• 2nd Degree AV Block (Mobitz II)
AV Dissociation		• 3rd Degree AV Block

Approach to Axis

Lead I	Lead aVF	Type of Axis	Cause of Axis Deviation
(+)	(+)	Normal Axis	
(+)	(−)	Left Axis Deviation	• LBB • LVH • LAFB
(−)	(+)	Right Axis Deviation	• RBBB • RVH • LPFB
(−)	(−)	Extreme Right Axis Deviation	• Ventricular Tachycardia

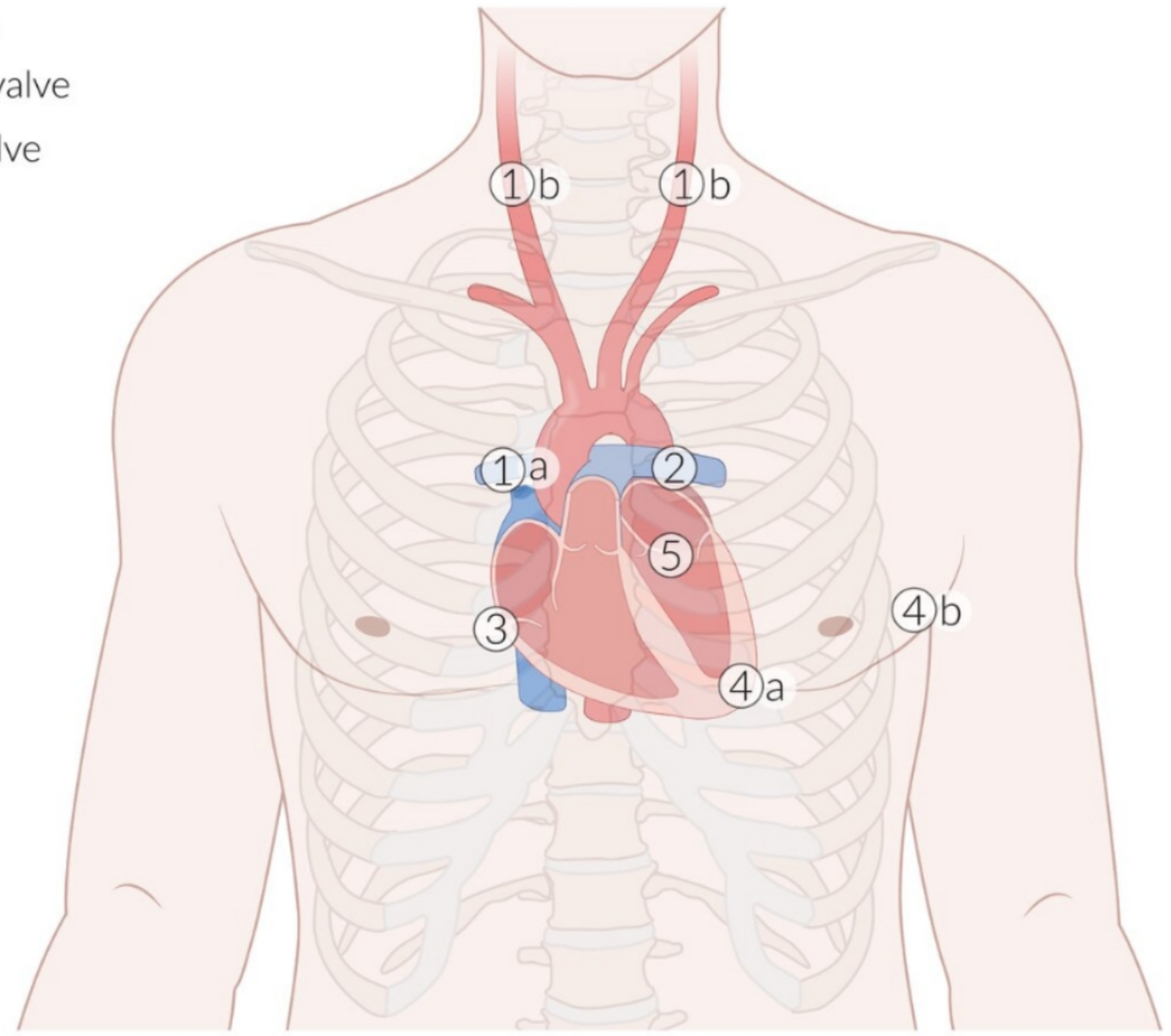
Station 11: ECG – Right bundle branch block.

Auscultatory locations [1]

Heart sound auscultation sites		
Name of area	**Location**	**Pathology**
Erb point (cardiology)	• 3rd left parasternal intercostal space	• Diastolic murmurs: aortic regurgitation, pulmonic regurgitation • Systolic murmurs: HOCM
Aortic area	• 2nd right parasternal intercostal space	• Aortic stenosis • Aortic regurgitation • Coarctation of the aorta
Pulmonic area	• 2nd left parasternal intercostal space	• Pulmonary stenosis • Pulmonary regurgitation • ASD
Mitral area	• 5th left intercostal space in the midclavicular line	• Mitral stenosis • Mitral regurgitation • Mitral valve prolapse (MVP)
Tricuspid area	• 4th left parasternal intercostal space	• Tricuspid stenosis • Tricuspid regurgitation • VSD

"**A**ll **P**hysicians **E**arn **T**oo **M**uch" (**A**ortic, **P**ulmonary, **E**rb point, **T**ricuspid, **M**itral)

① Aortic valve
② Pulmonary valve
③ Tricuspid valve
④ Mitral valve
⑤ Erb point



Anatomical relationships of ECG leads			
	Limb leads	**Precordial leads**	**Corresponding heart structure**
Inferior leads	• II • III • aVF	• N/A	• Inferior surface of the heart
Lateral leads	• I • aVL	• V_5 • V_6	• Left ventricle, lateral wall
Anteroseptal leads	• N/A	• V_1–V_4	• Anterior wall of both ventricles • Ventricular septum
Right-sided leads	• N/A	• V_3R–V_6R • OR V_1R–V_6R	• Right ventricle
Posterior leads	• N/A	• V_7–V_9	• Posterior wall of the left ventricle