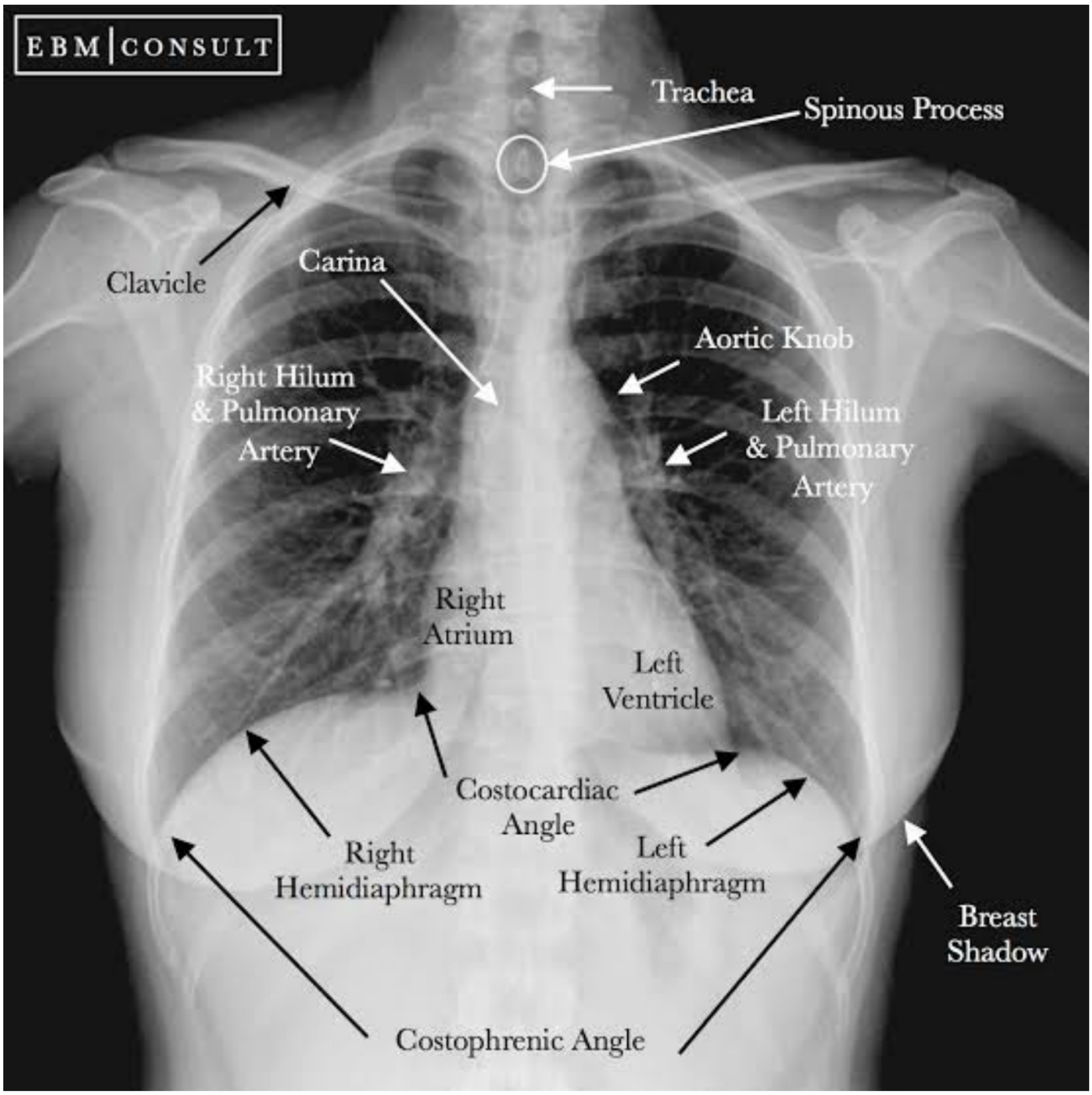
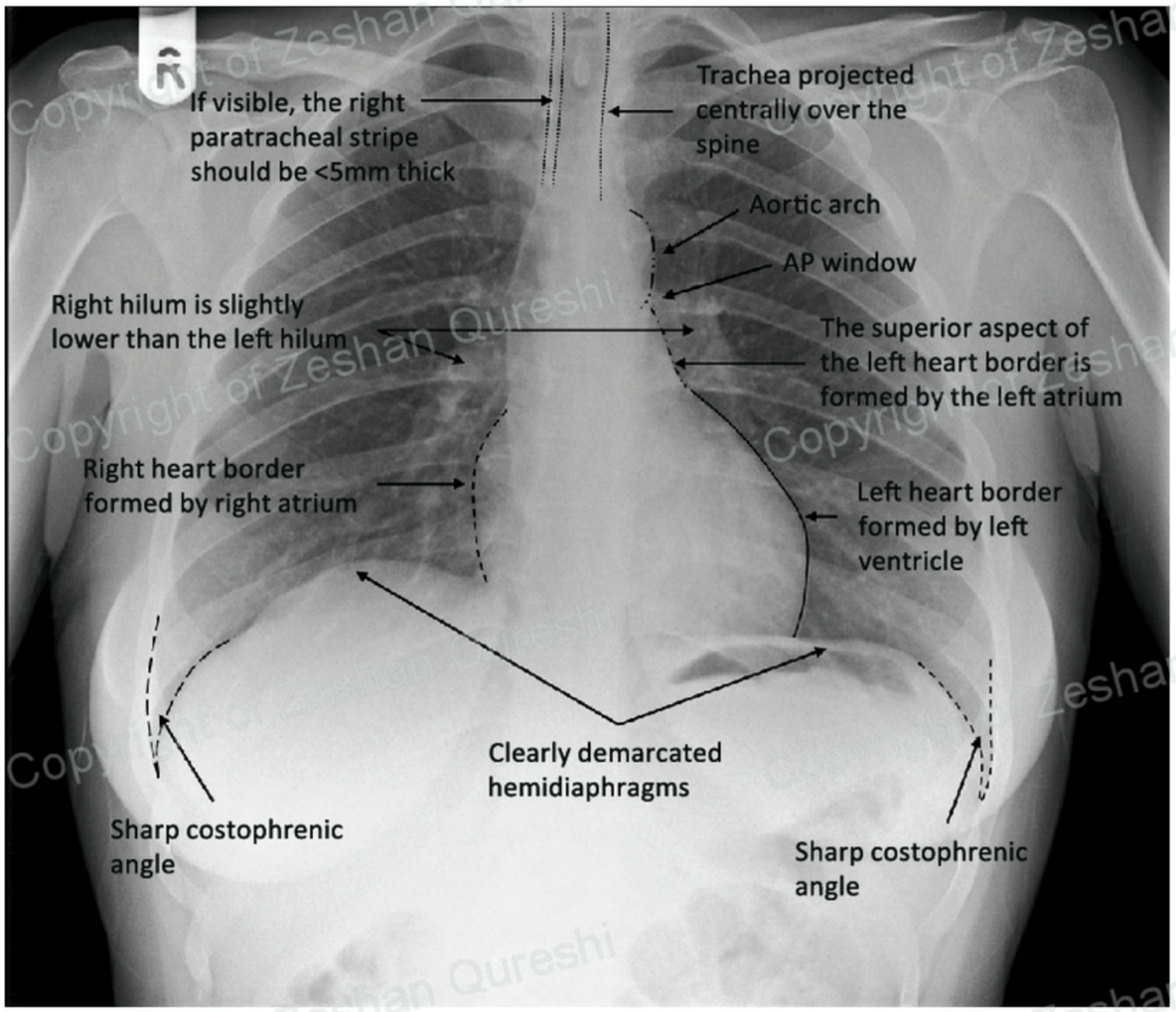
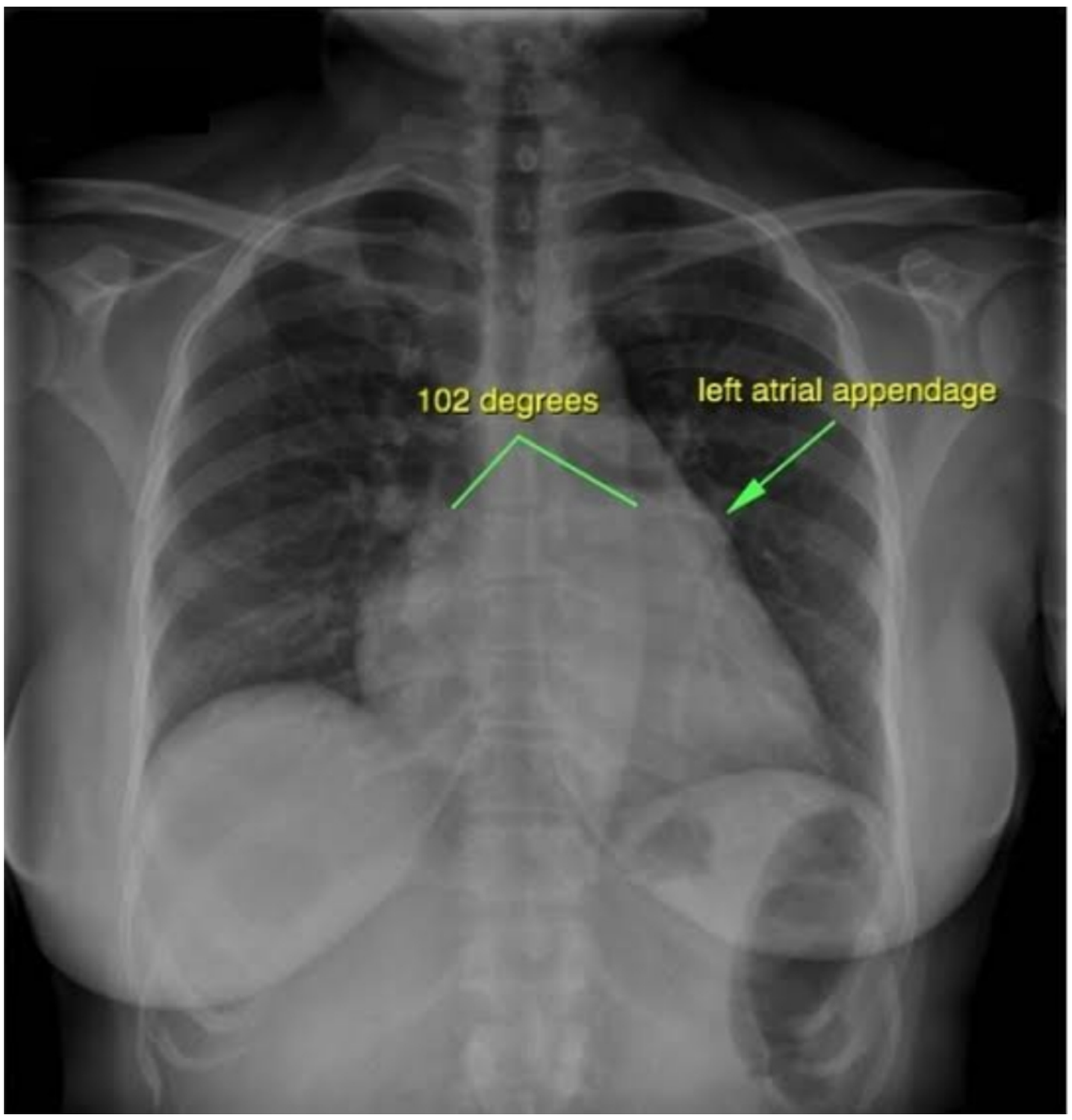
Normal X-Ray


EBM | CONSULT
Trachea
Spinous Process
Clavicle
Carina
Aortic Knob
Right Hilum & Pulmonary Artery
Left Hilum & Pulmonary Artery
Right Atrium
Left Ventricle
Costocardiac Angle
Right Hemidiaphragm
Left Hemidiaphragm
Breast Shadow
Costophrenic Angle

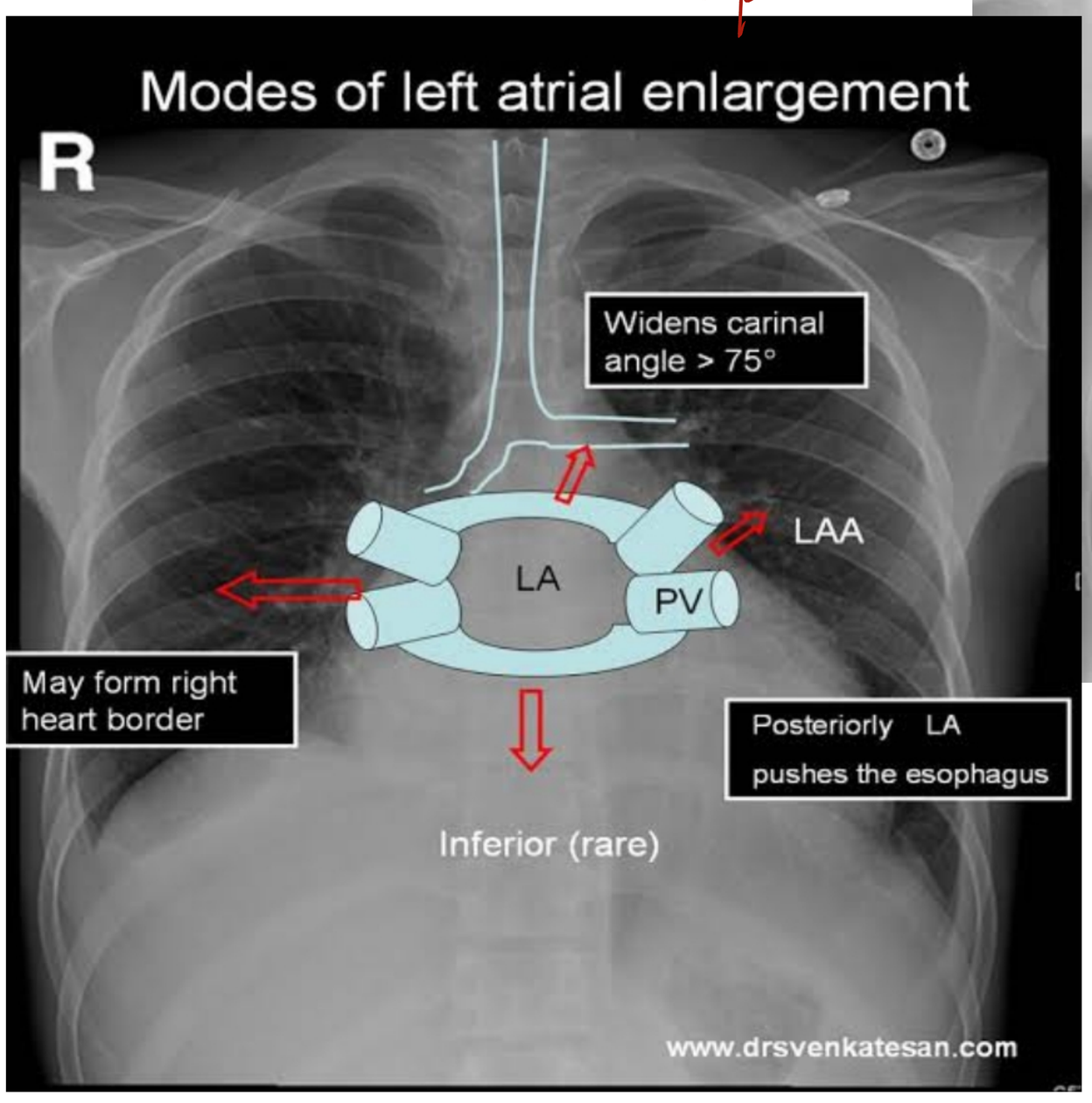
Normal PA Chest X-Ray

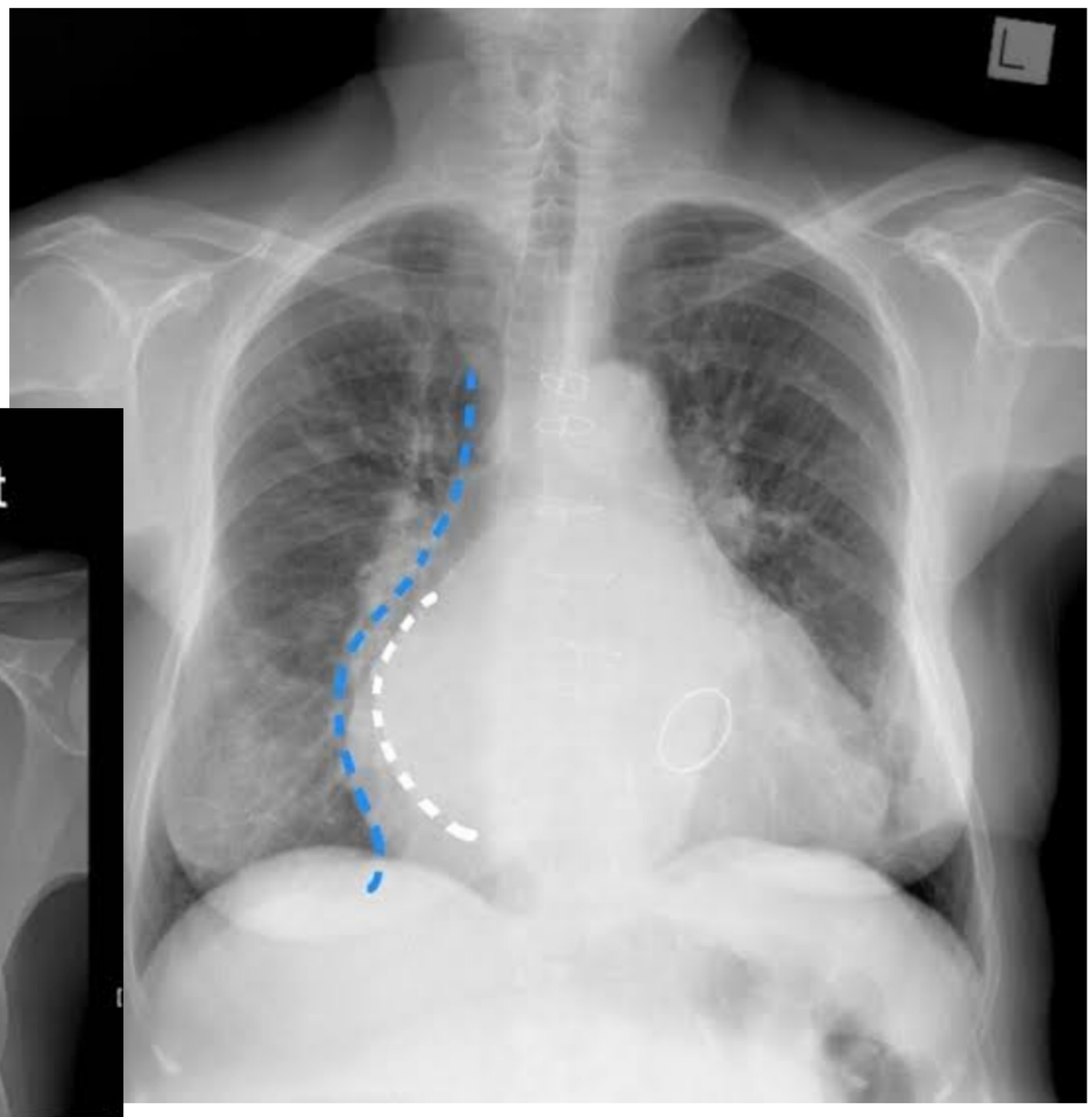


Left Atrial Dilatation (X-Ray)

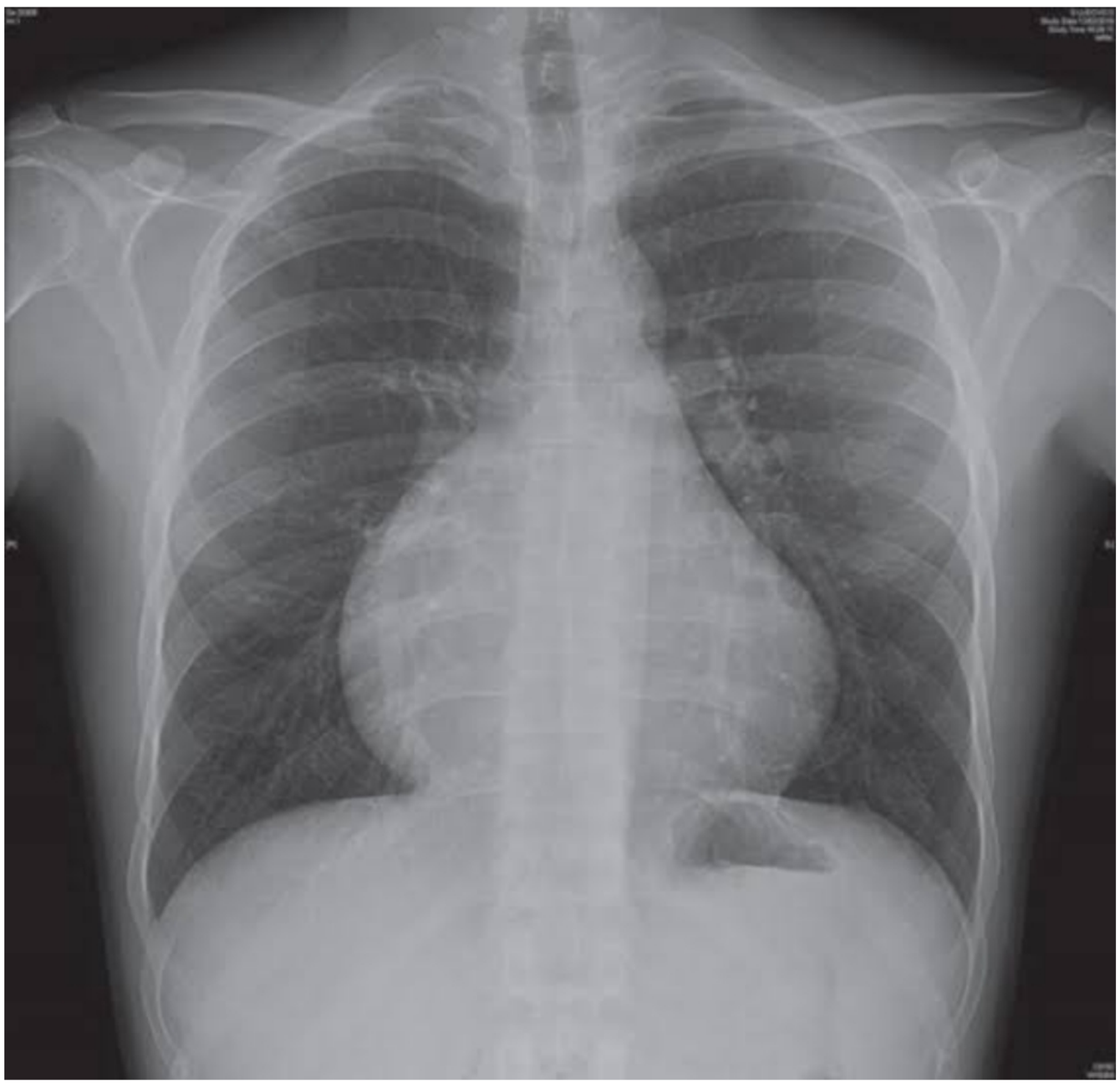


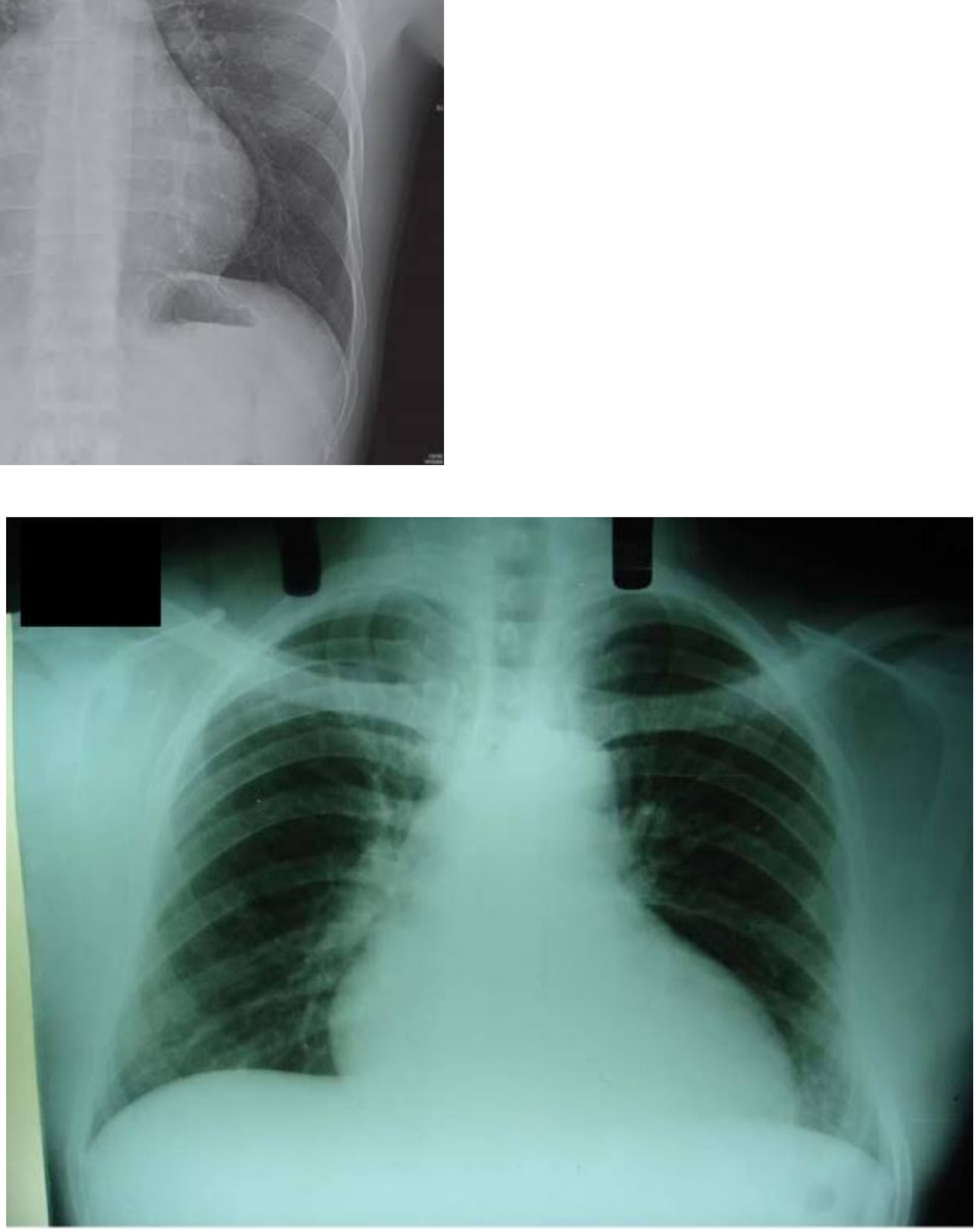
- Prominence of left atrial appendage
- Appearance of straight left heart border
- Double cardiac shadow to right of sternum
- Widening of angle of carina (bifurcation of trachea) as left main bronchus is pushed upwards



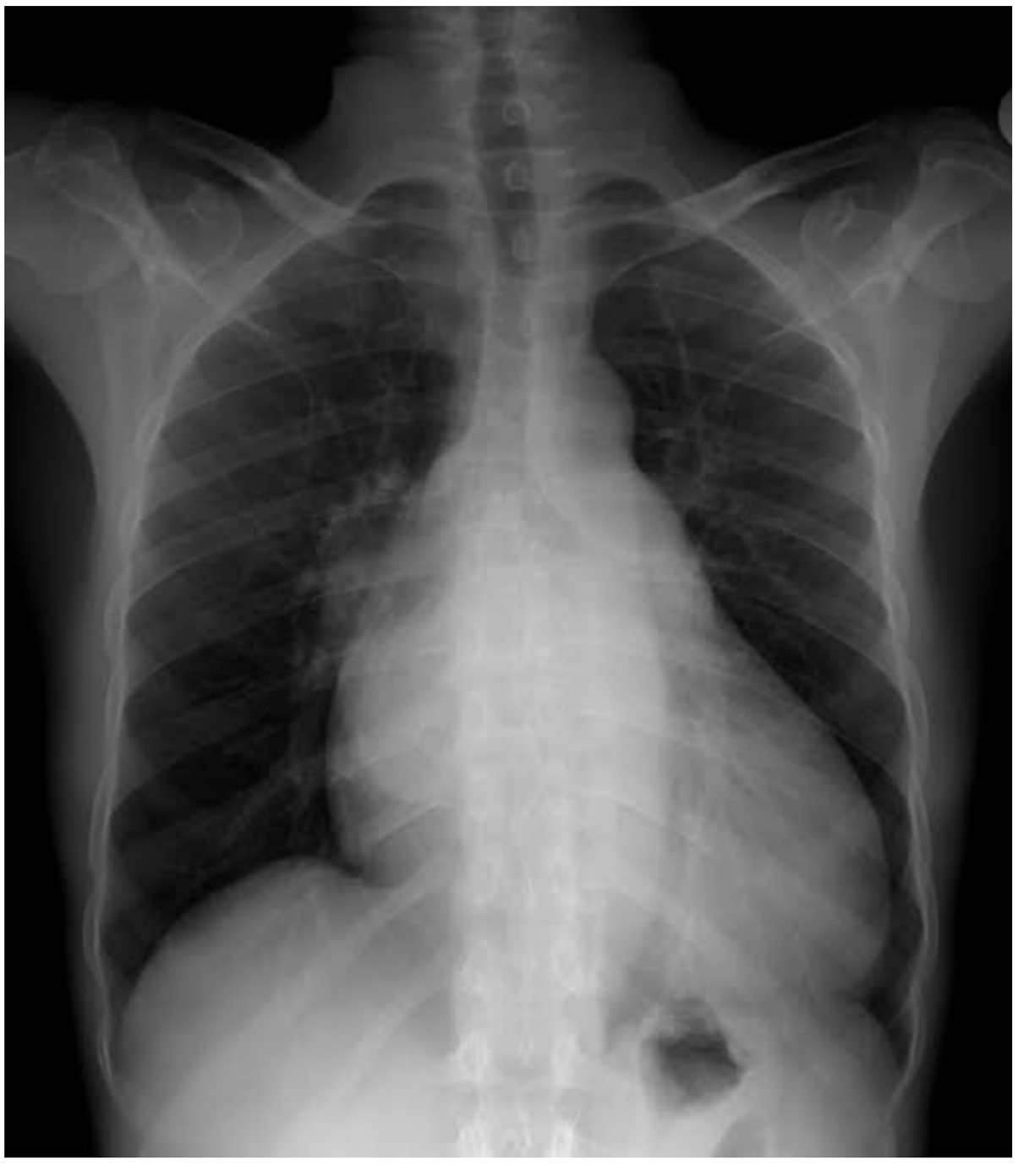


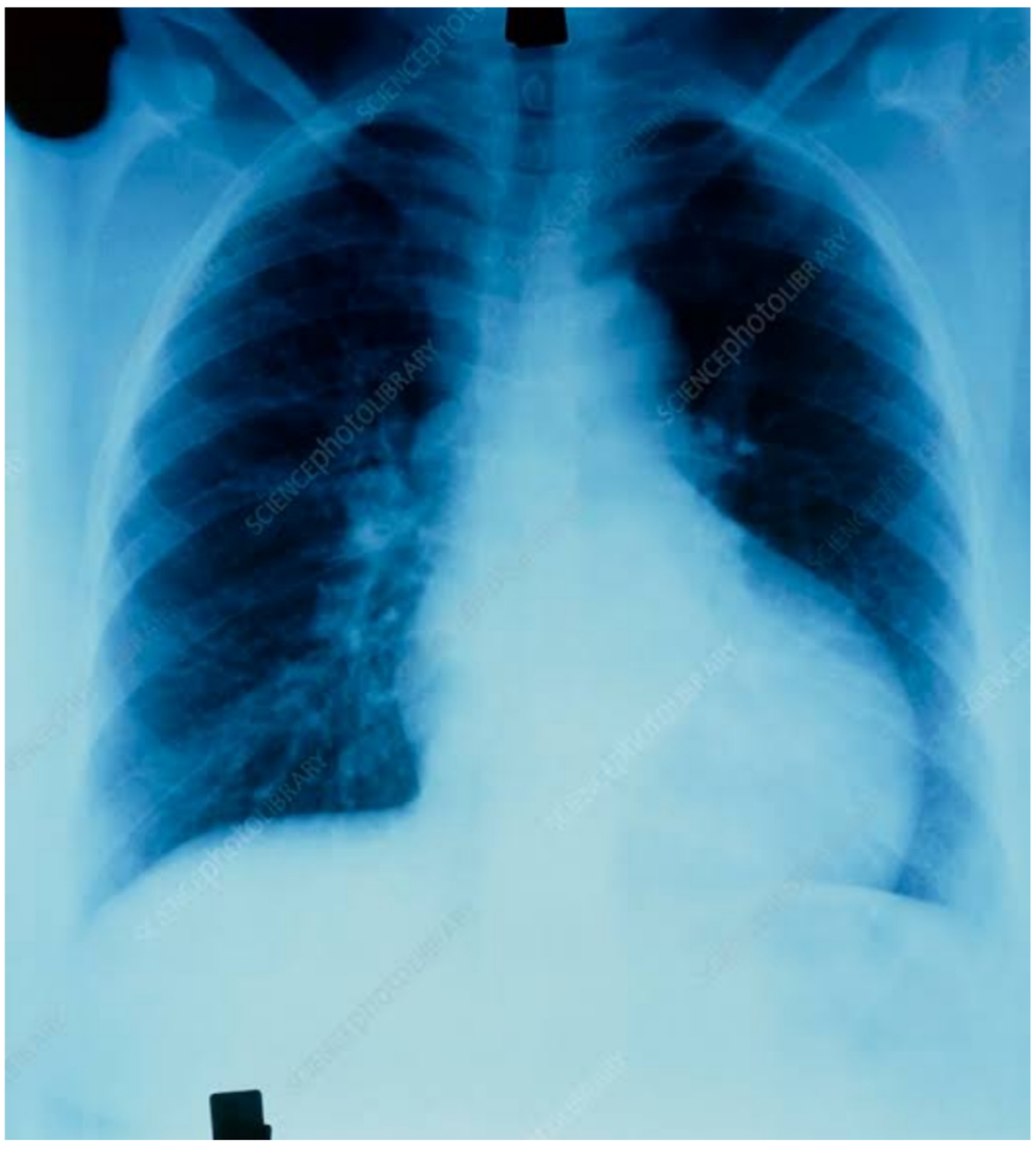
Right Atrial Enlargement (X-Ray)

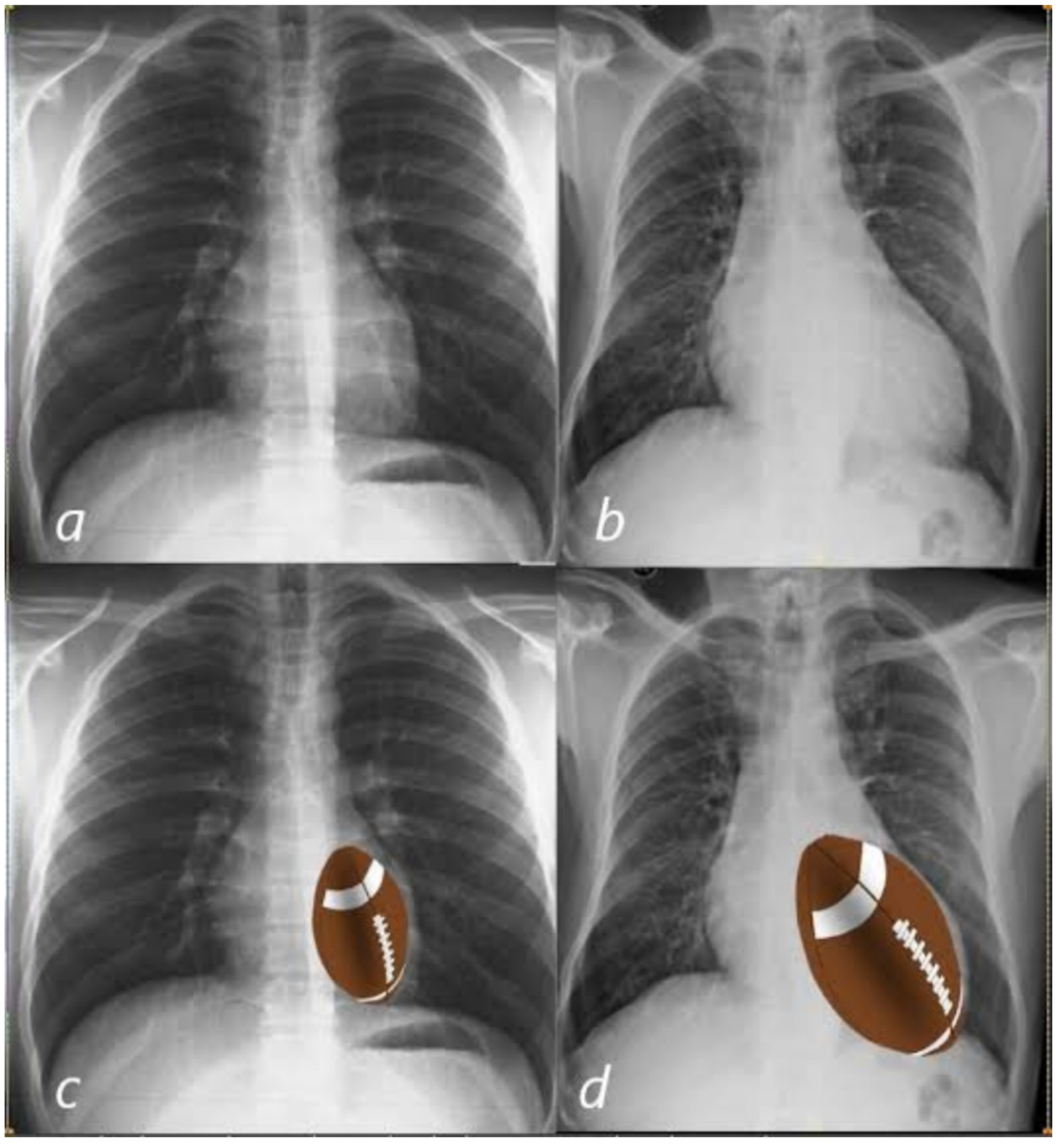


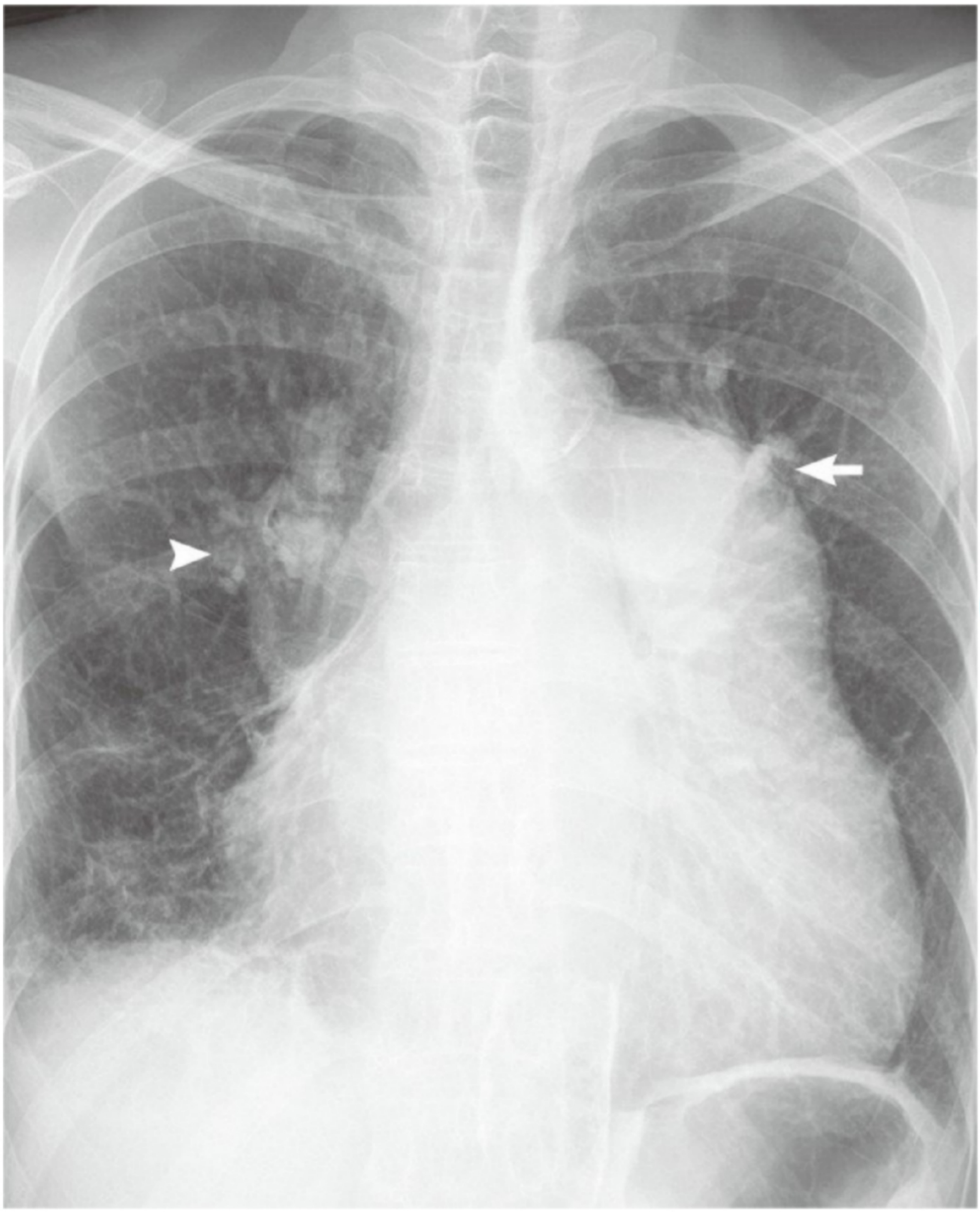


Left Ventricular Enlargement X-Ray

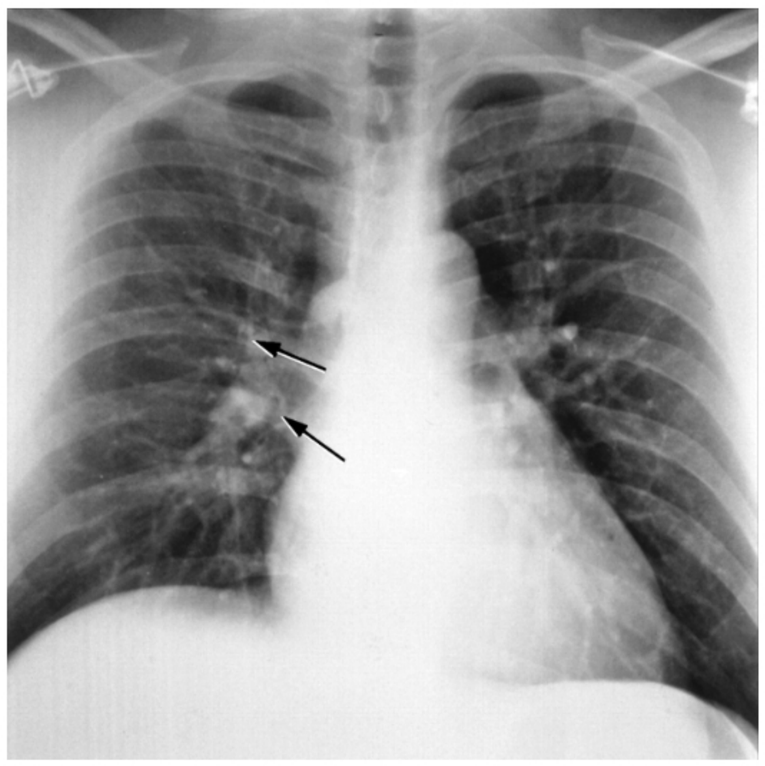


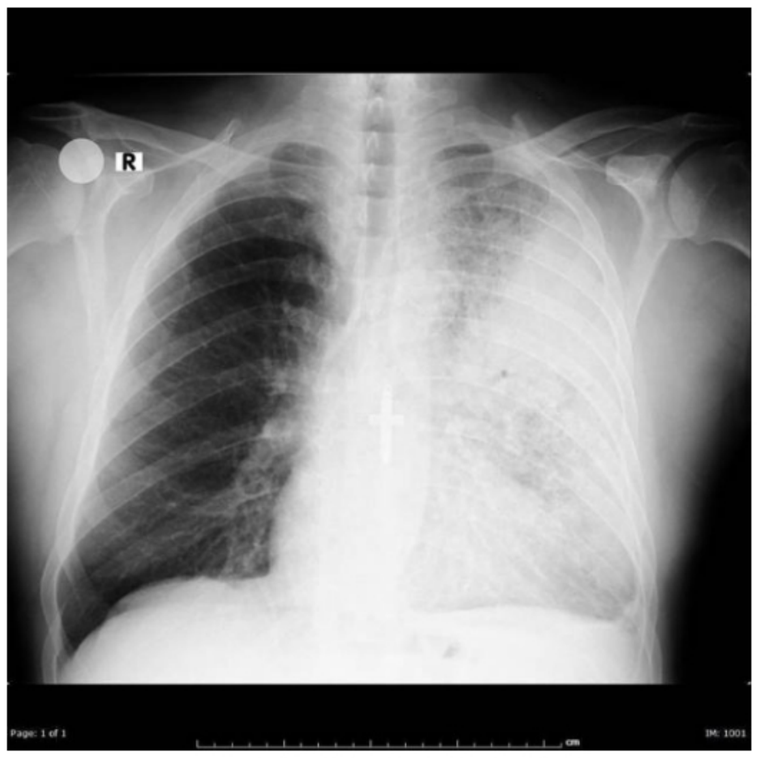






Chest X-ray. Cardiomegaly involving right chambers, enlarged pulmonary trunk (arrow), and right main pulmonary artery (head arrow).





🫁 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

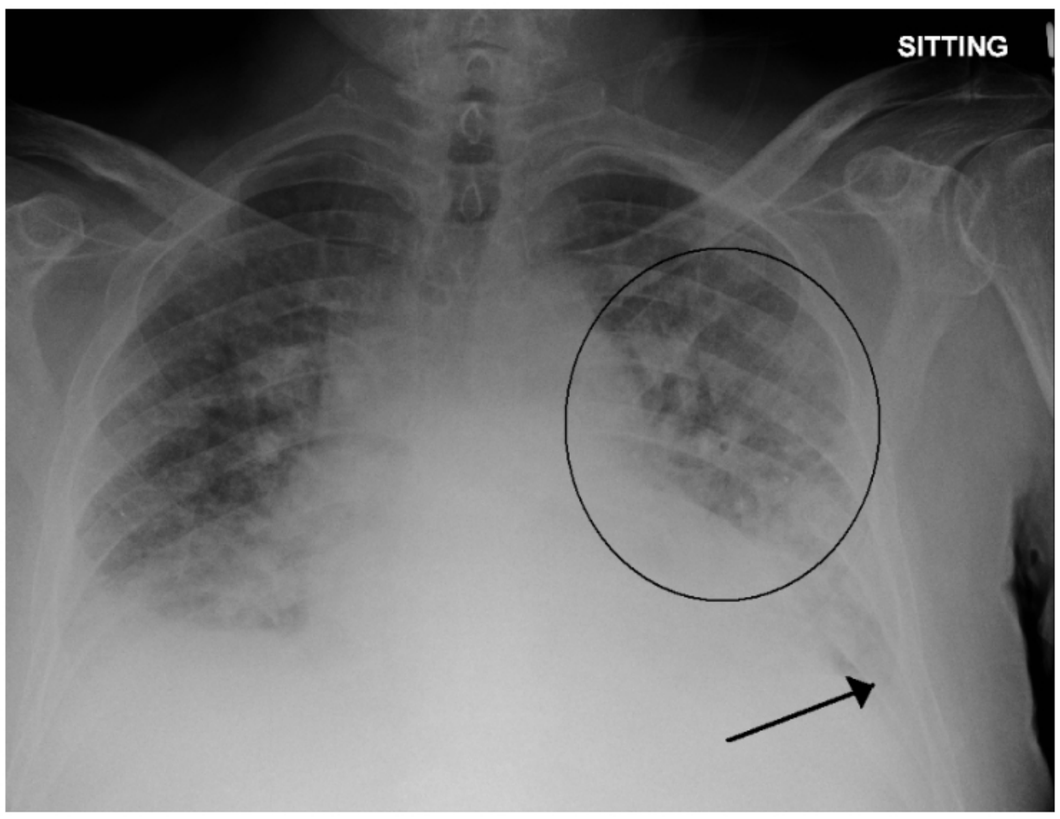
2️⃣ Air bronchogram strongly suggests pneumonia

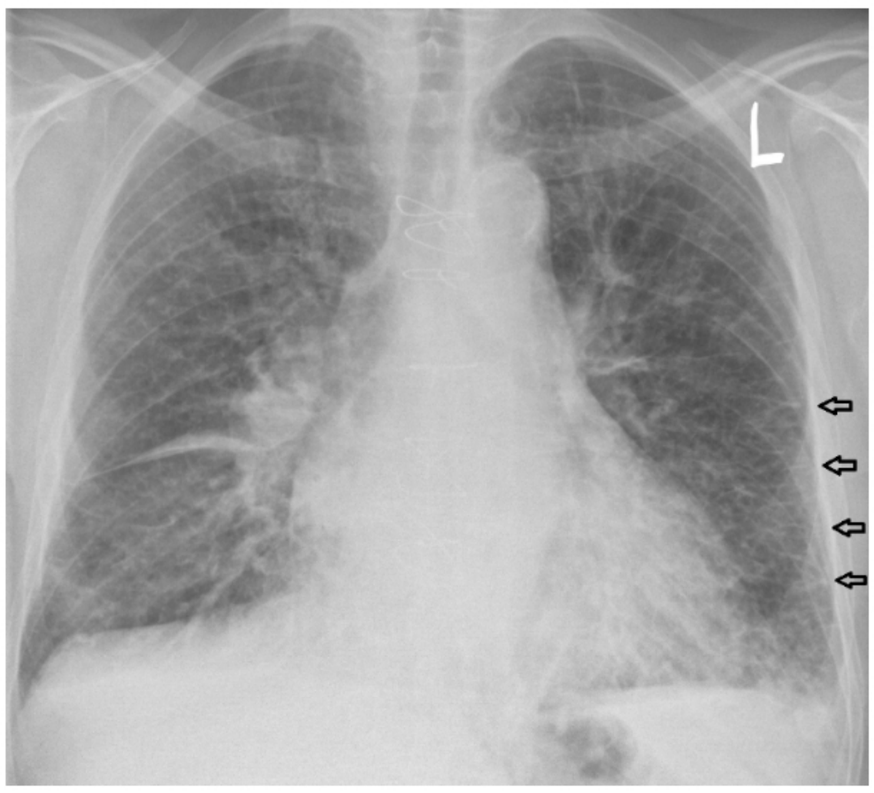
3️⃣ Rapid clearing after IV furosemide = pulmonary edema

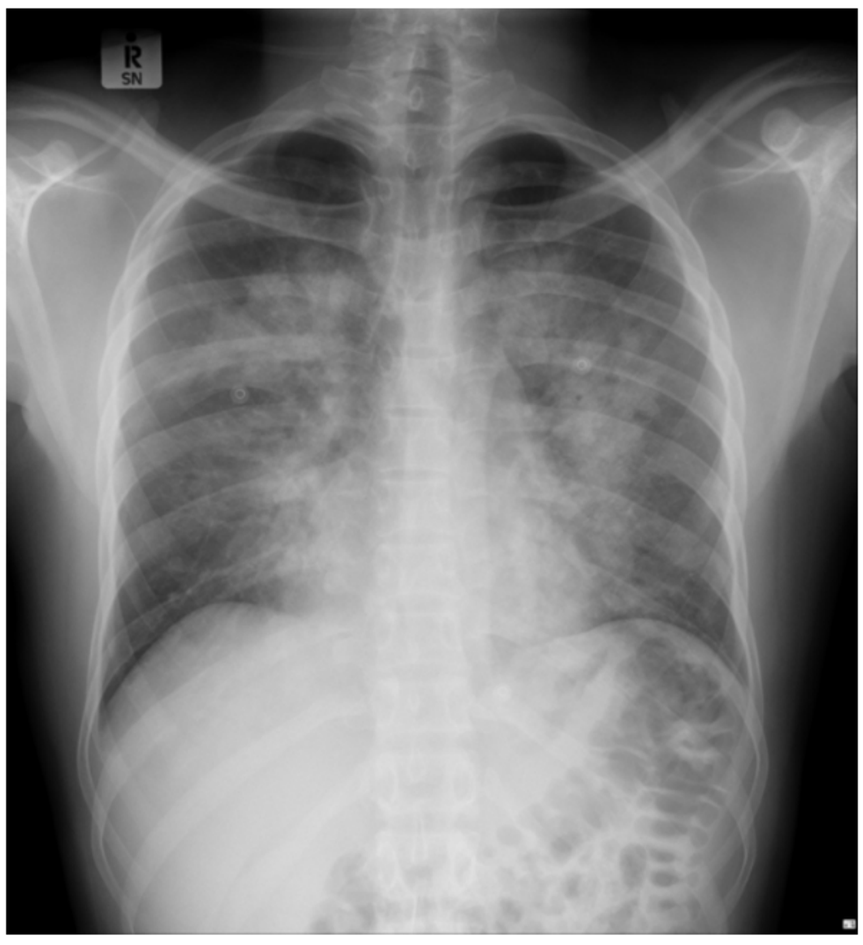
4️⃣ Cardiomegaly favors cardiogenic edema

5️⃣ BNP high → edema; Procalcitonin high → pneumonia

Pulmonary edema XRay and management







💊 Management

1️⃣ Immediate / Emergency

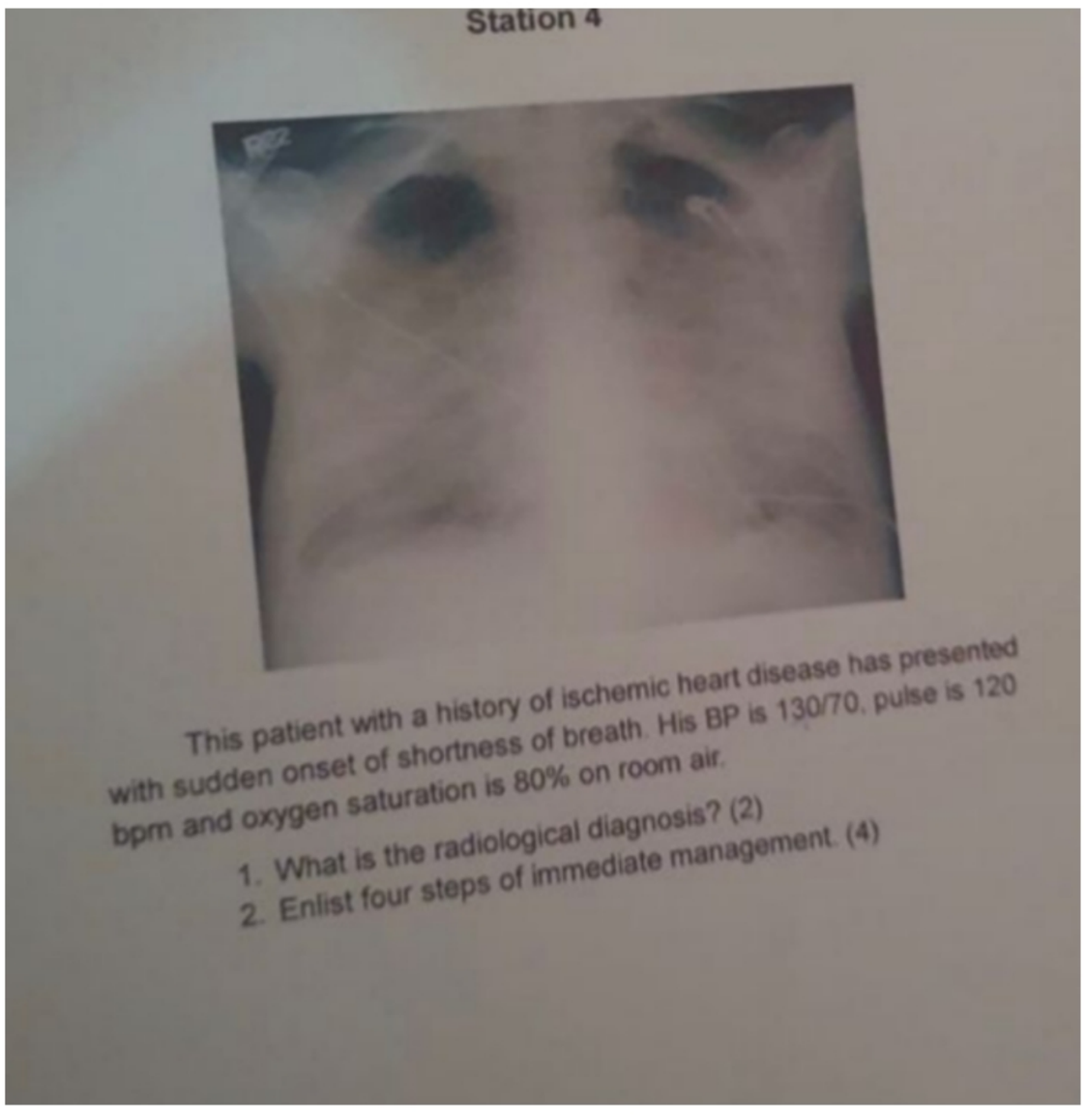
- **Oxygen therapy** – maintain SpO_2 > 90%
- **Sit upright** – reduces venous return
- **Diuretics (IV furosemide)** – reduce pulmonary congestion
- **Morphine** (optional) – for severe dyspnea and anxiety
- **Nitroglycerin** – if hypertension or acute left heart failure

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

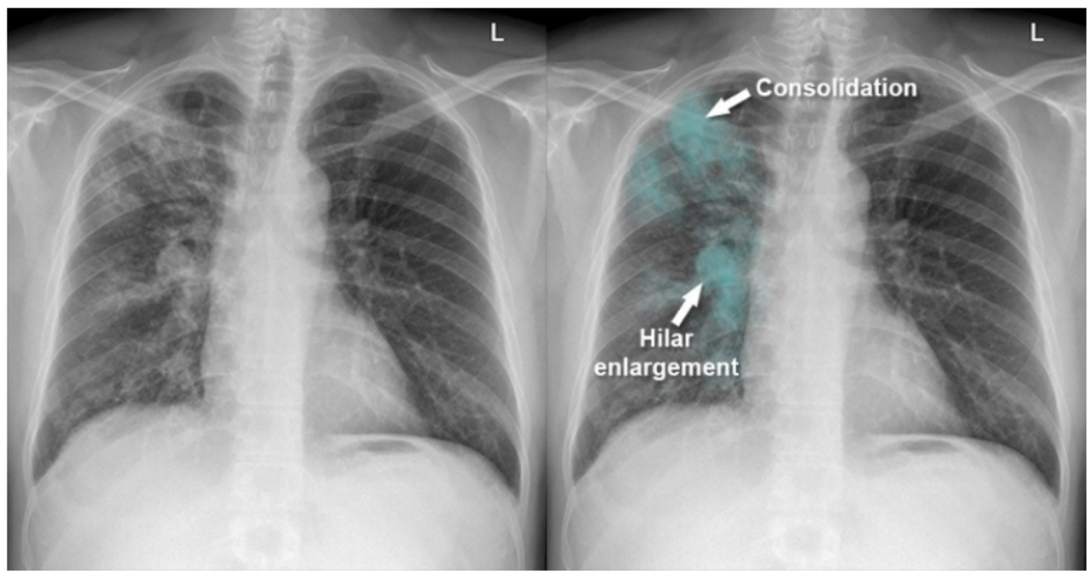
- **Non-invasive ventilation (CPAP / BiPAP)** for hypoxia
- **Intubation and mechanical ventilation** if severe
- **Inotropes / vasopressors** if cardiogenic shock



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)



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

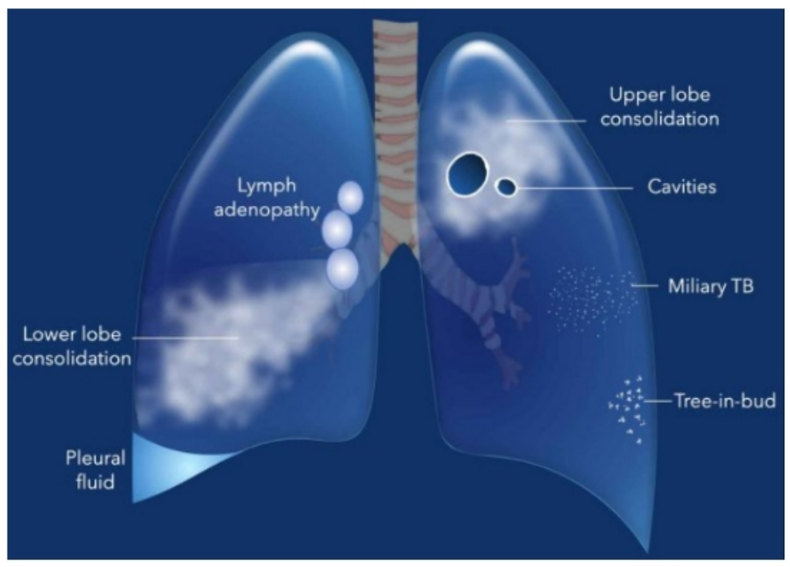
Caused by Mycobacterium tuberculosis.

🔹 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)

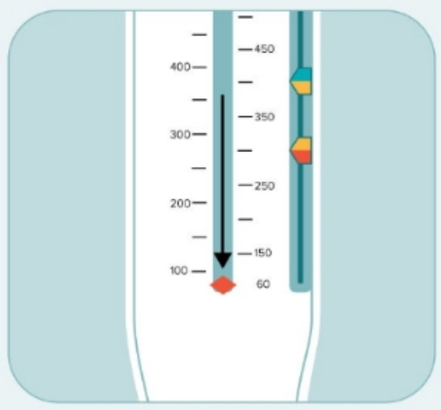


Peak flow meter

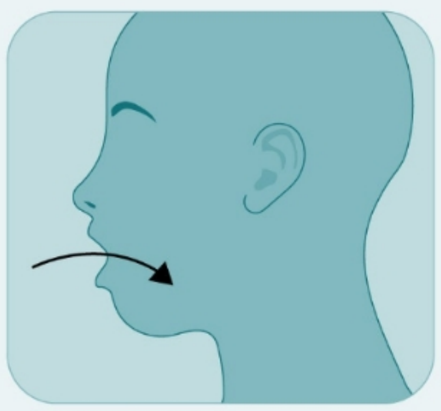
Peak expiratory flow measurement

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

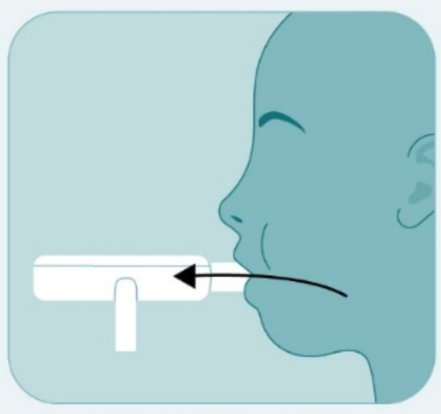
HOW TO USE A PEAK FLOW METER



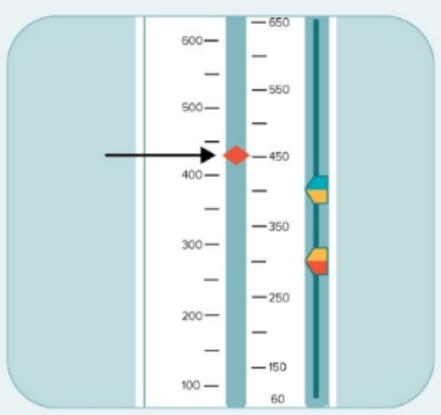
1. Set marker to lowest number



2. Take deep inhale



3. Blow hard and fast in a single blow



4. Read meter

5. Record results, repeat twice

healthline

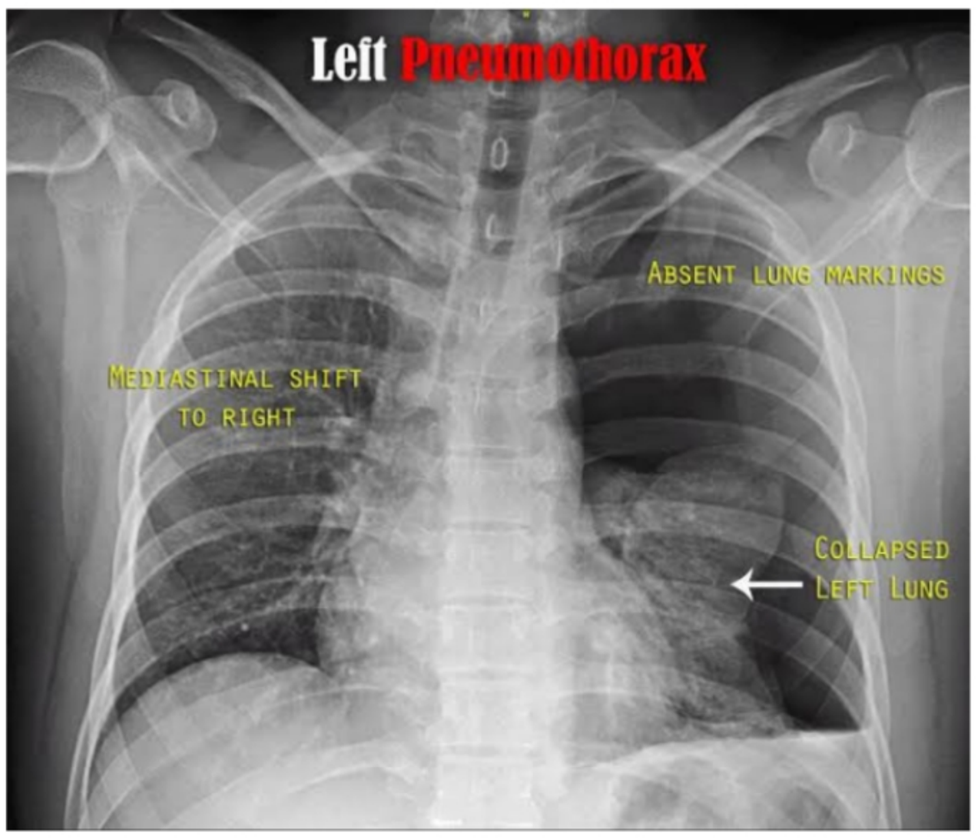
Station 15💌:observed

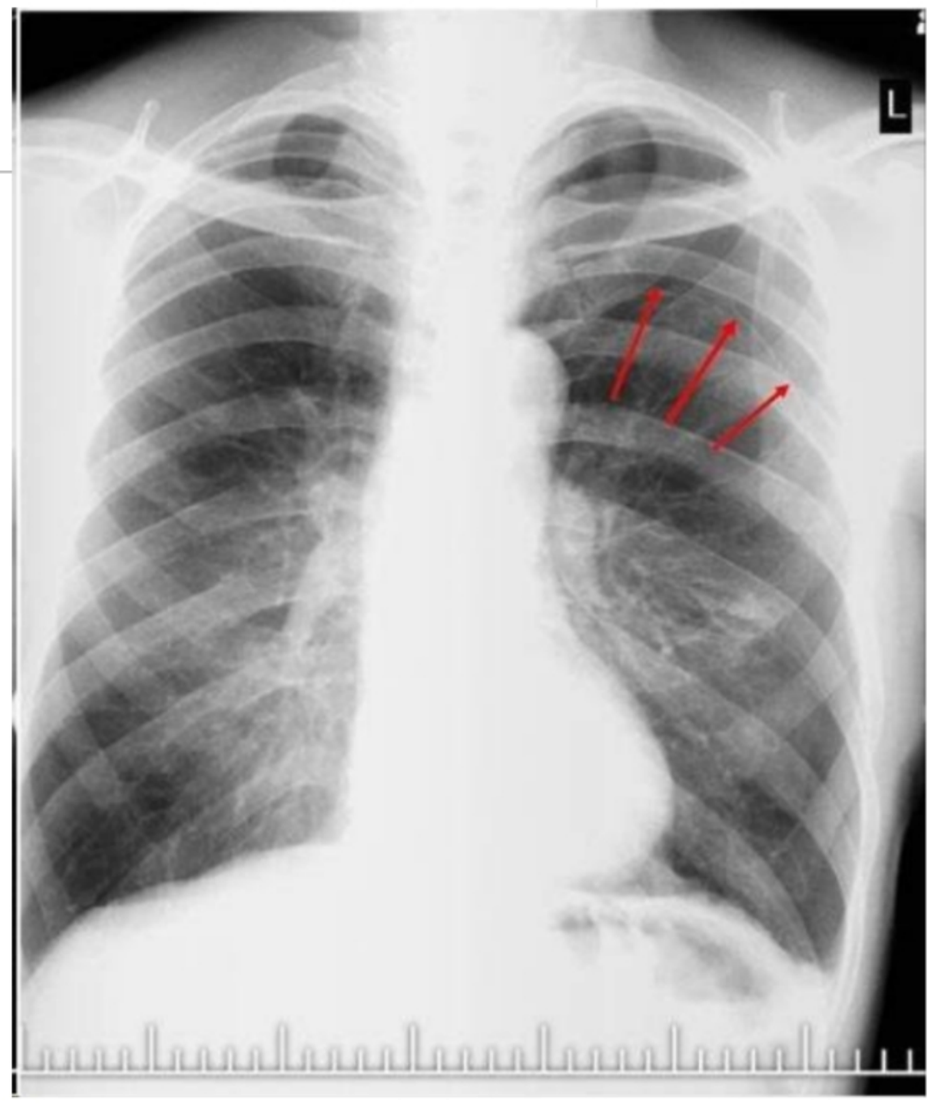
A chest X ray of Spontaneous Primary Pneumothorax (left sided):

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.**

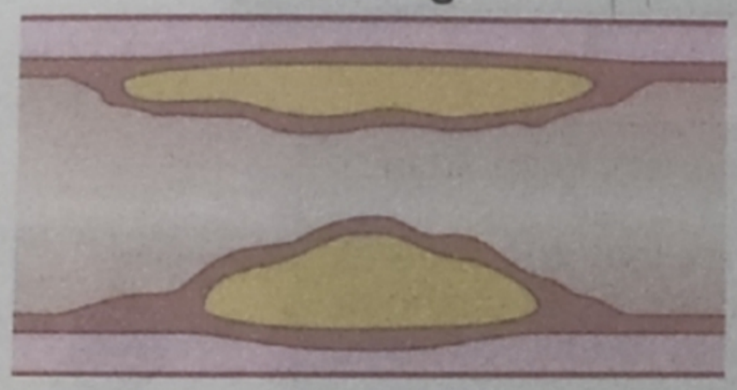
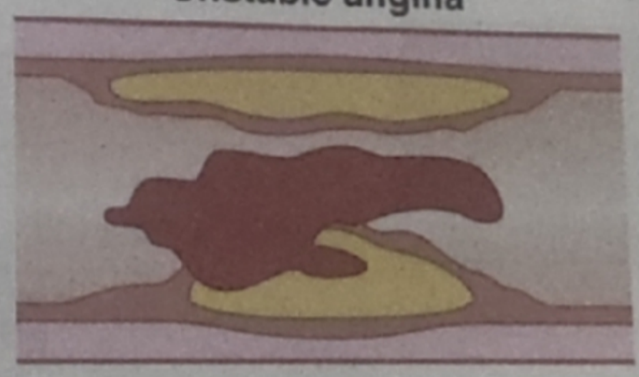
	Stable angina	Unstable angina
		
Pathophysiology	• Fixed stenosis	• Dynamic stenosis
Clinical features	• Demand-led ischaemia • Related to effort • Predictable • Symptoms over long term	• Supply-led ischaemia • Symptoms at rest • Unpredictable • Symptoms over short term
Risk assessment	• Symptoms on minimal exertion • Exercise testing Duration of exercise Degree of ECG changes Abnormal BP response	• Frequent or nocturnal symptoms • ECG changes at rest • ECG changes with symptoms • Elevation of troponin

Fig. 18.17 Pathophysiology, clinical features and risk assessment of patients with stable or unstable angina.

Stable Angina

- effort related chestpain

Unstable Angina

- prolonged and severe cardiac chest pain

Chronic Heart Failure

- Dyspnea (may first present on moderately severe exertion and later progress to dyspnea on lesser exertion)
- Orthopnea → on lying down
- Paroxysmal nocturnal dyspnea
- Cheyne Stokes Respiration → left ventricular failure

What is Cheyne-Stokes respiration?

Cheyne-Stokes respirtion is a cyclical pattern of breathing that has increases in rapid respiration followed by gradual decrease and then apnea from 5 to 50 seconds. It is also known as agonal respirations.

There are three phases, phase 1 is the cresendo phase. This is where tidal volume increases gradually with each breath from hypopnea to hyperpnea. Phase 2 is a decresendo phase from hyperpnea to hyponea. Phase 3 is a central apnea phase.

Cheyne Stokes Breathing

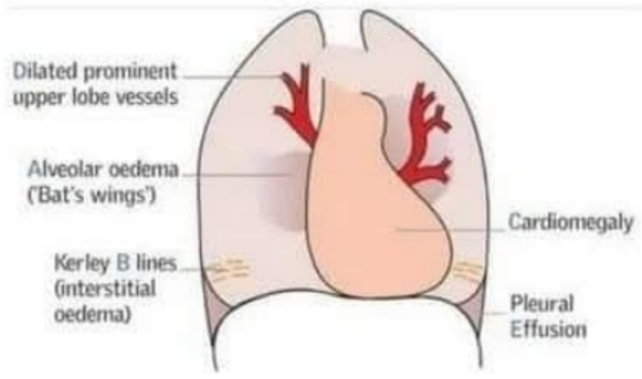
- The pattern of slowly diminishing respiration, leading to apnea, followed by progressively increasing respiration and hyperventilation, may be accompanied by a sensation of breathless and panic during the period of hyperventilation

- May be seen in
  - Left ventricular failure
  - Diffuse cerebral atherosclerosis
  - Stroke or head injury
  - may be exaggerated by sleep, barbiturates, opiates

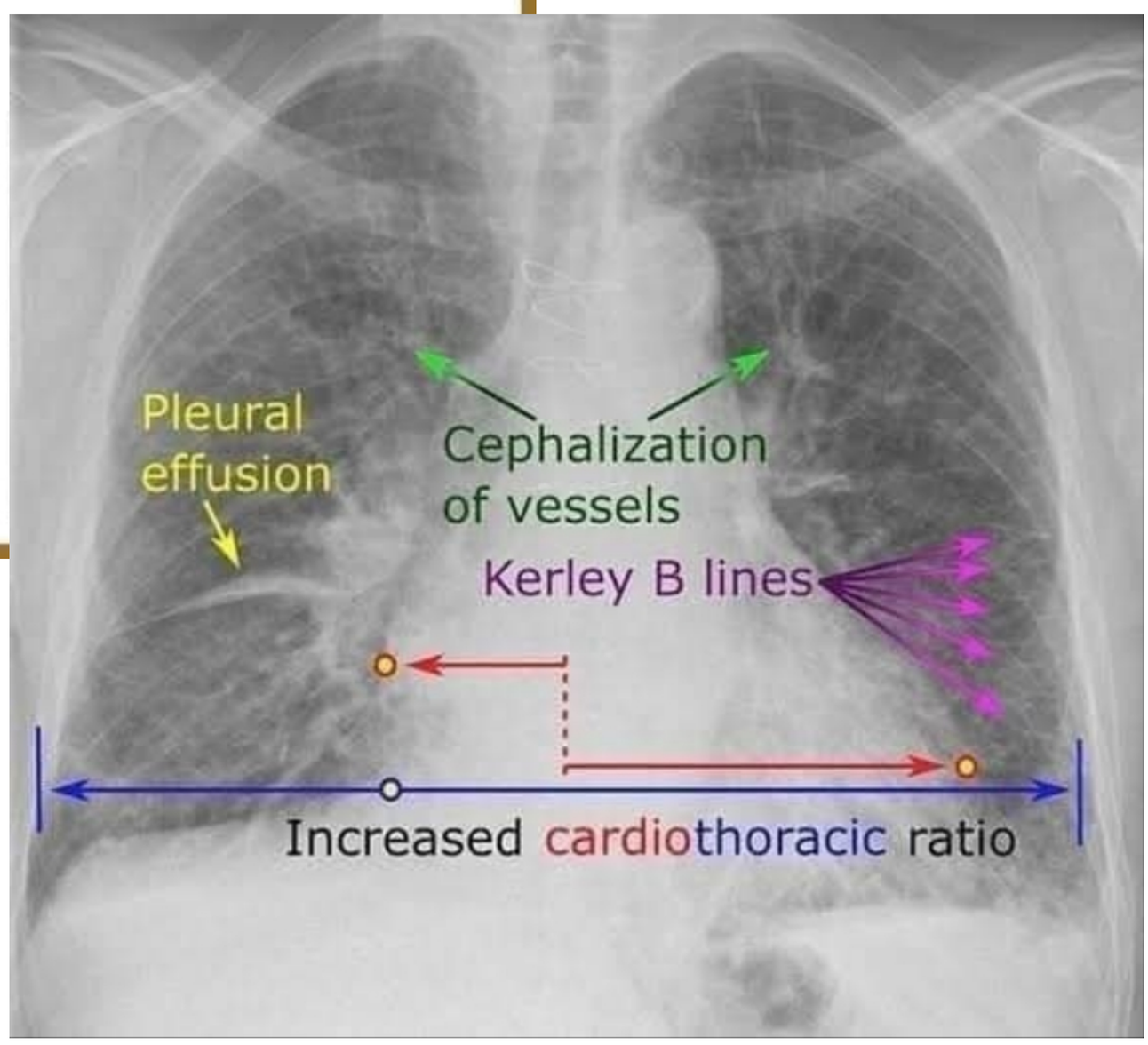
Chest X-ray in Left Ventricular Failure

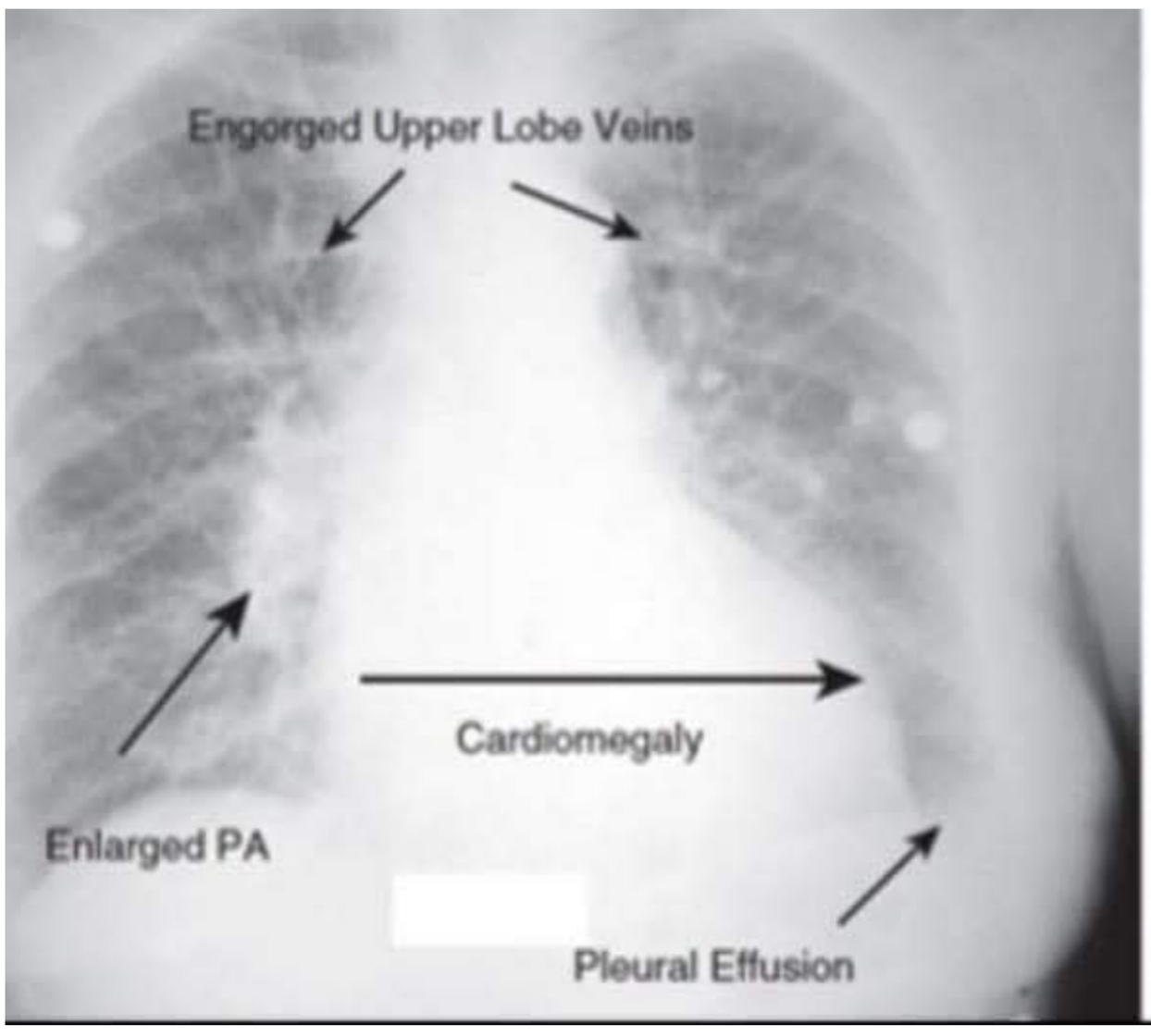
MEDREWIND

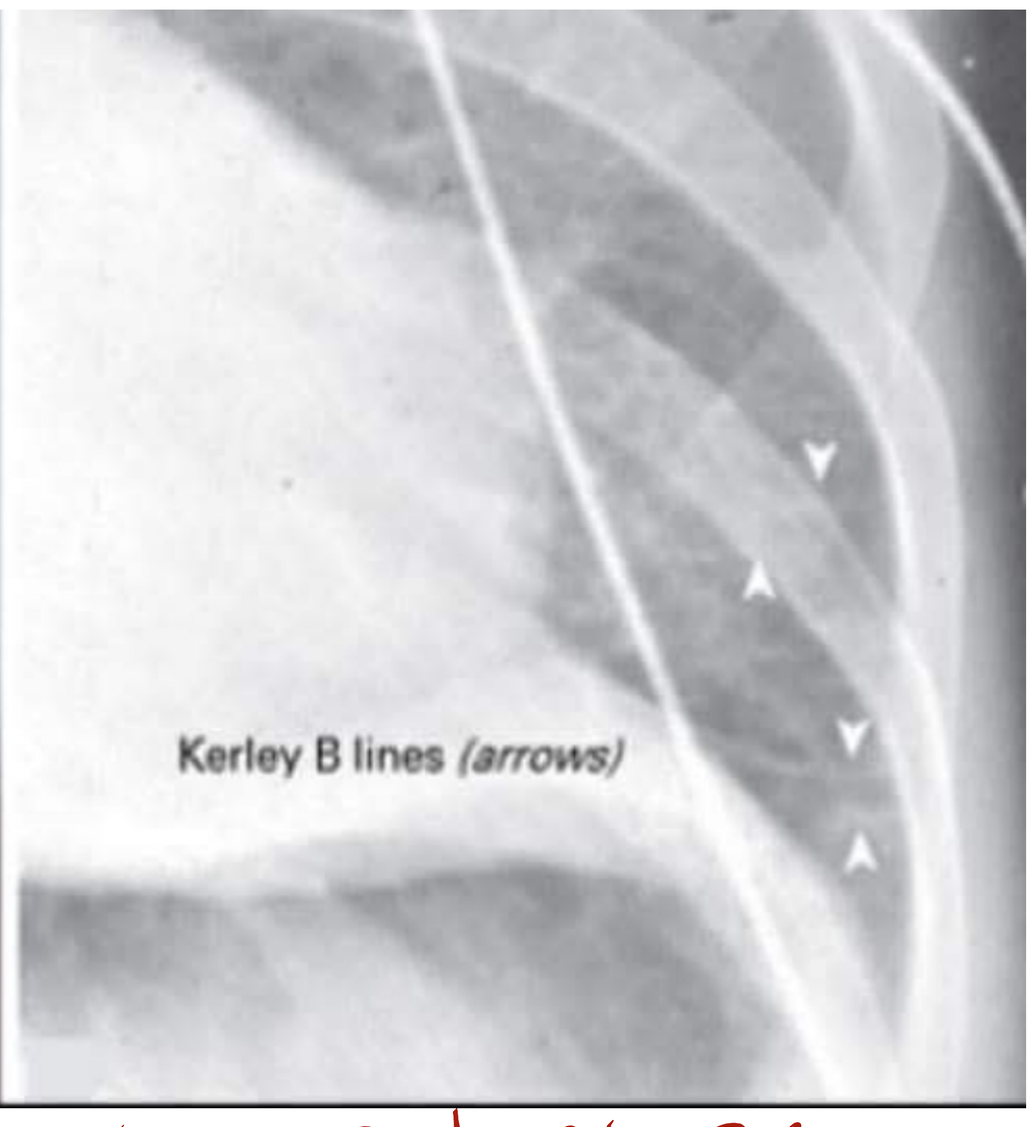
- Features can be remembered as: ABCDE
- **Alveolar Edema, kerley B lines, Cardiomegaly, Dilated prominent upper lobe vessels, pleural Effusion.**



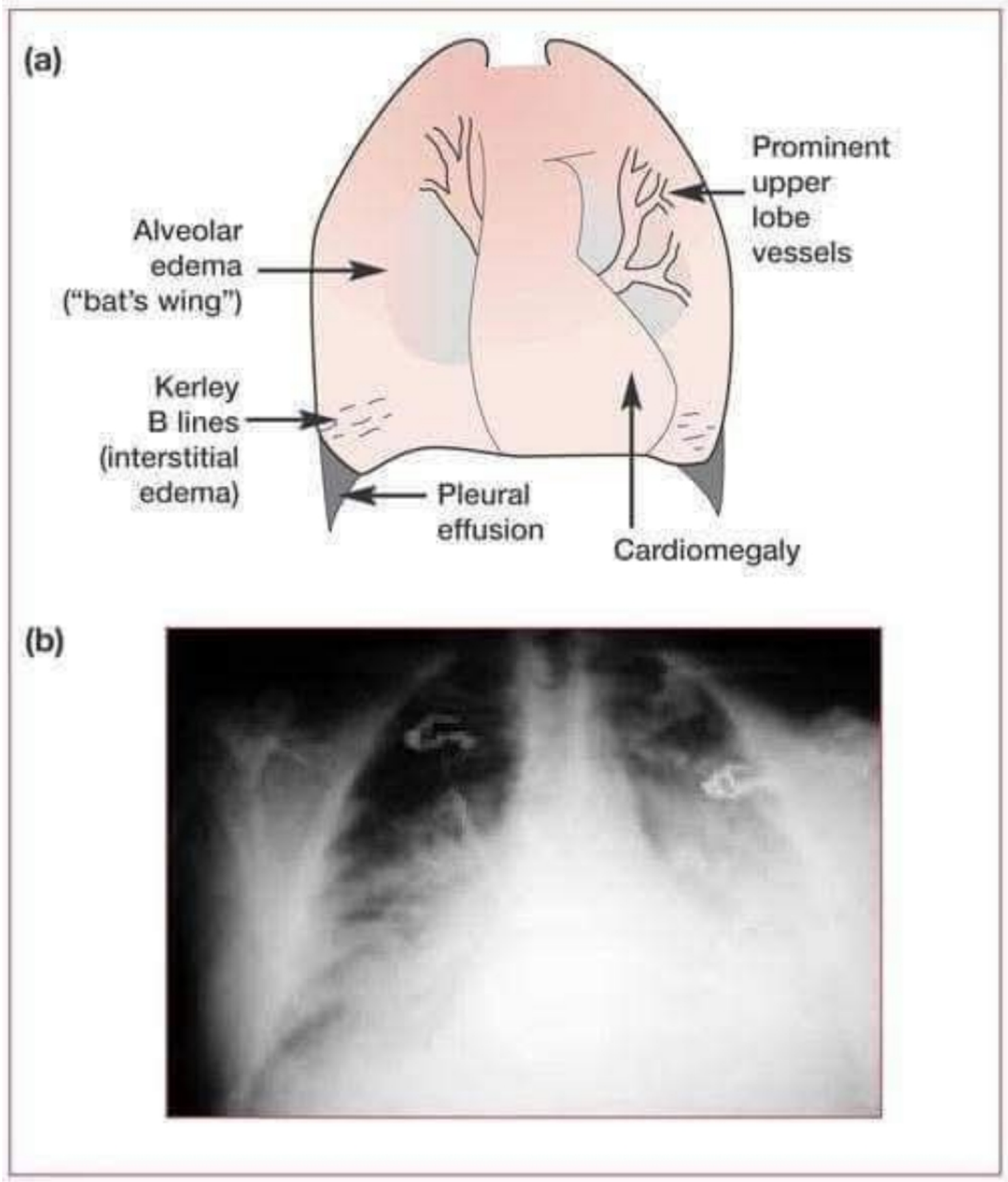
- Abnormal distension of upper lobe pulmonary veins
- vascularity of lung fields become more prominent
- Right and left pulmonary arteries dilate



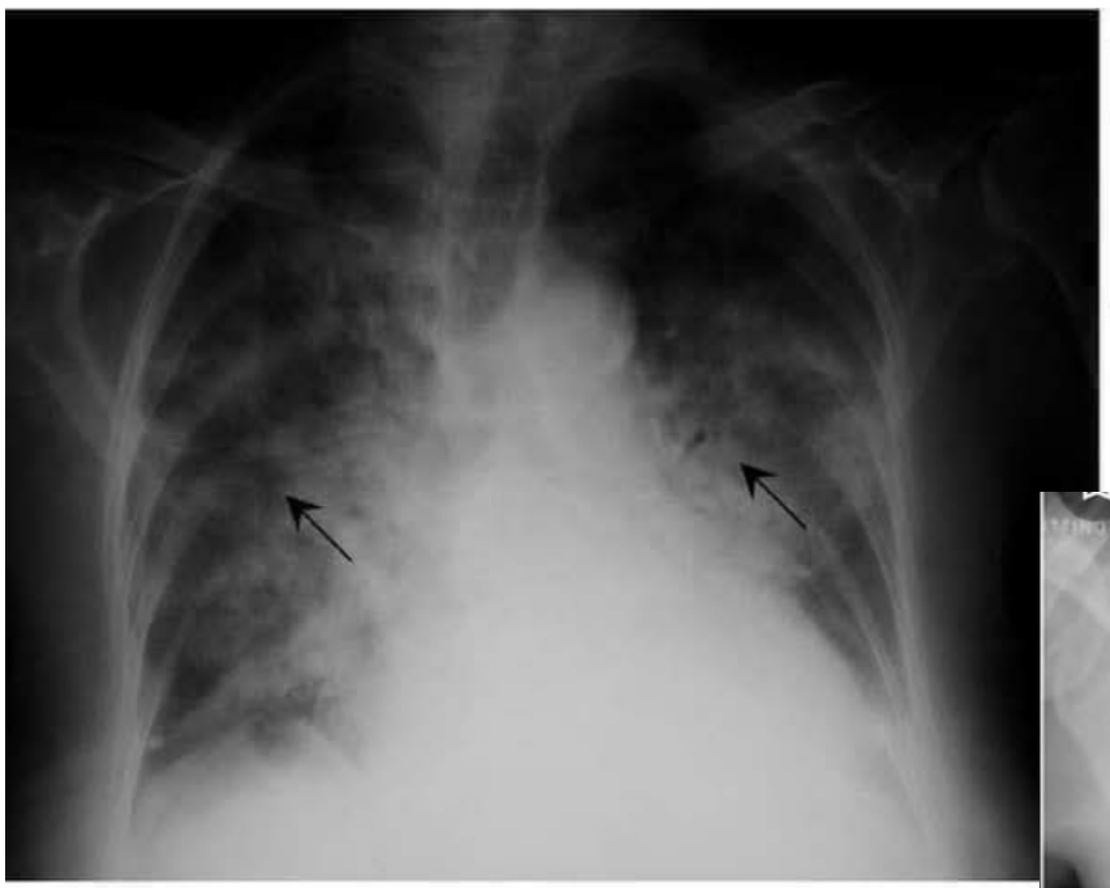




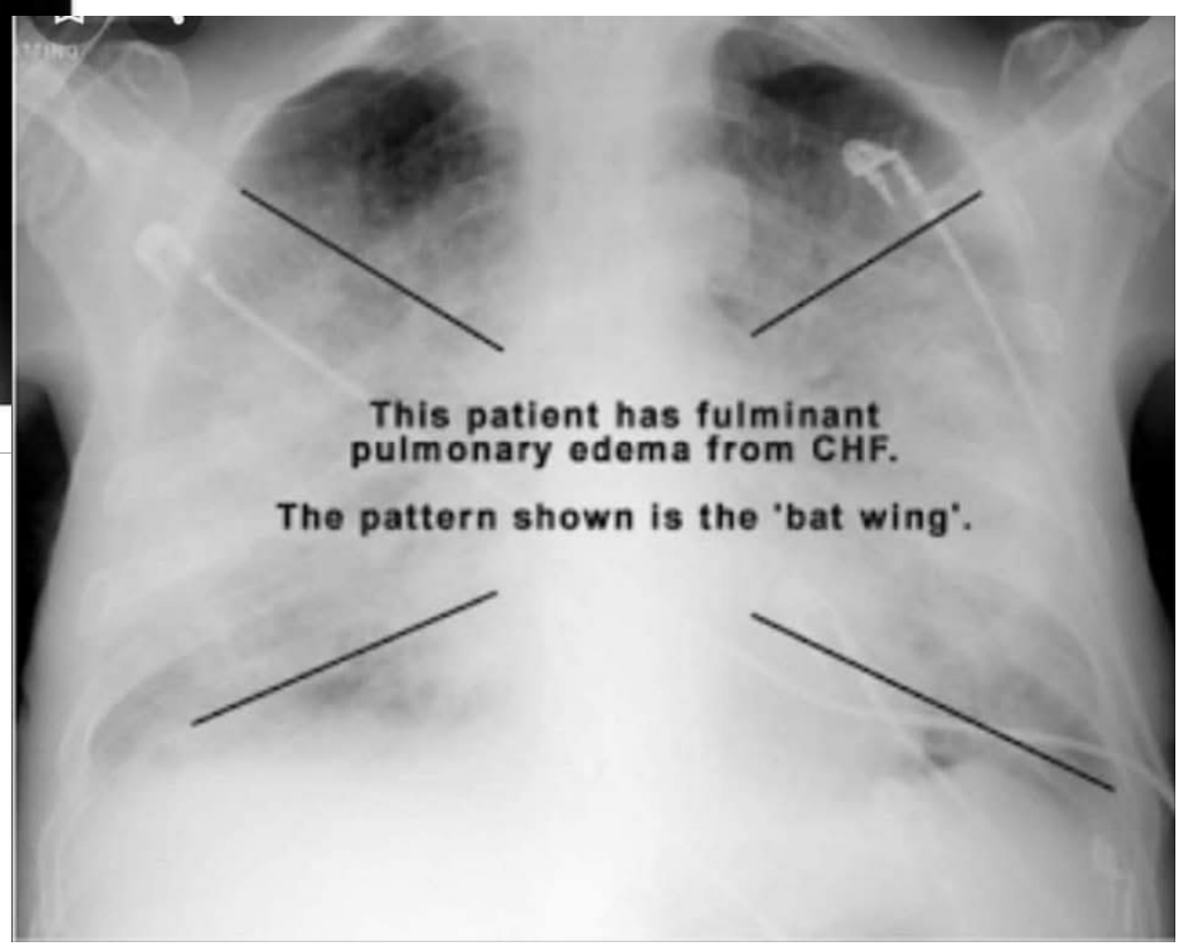
- Kerley B lines are evident horizontal lines in costophrenic angles



- More advanced changes due to alveolar edema cause hazy opacification spreading from hilar regions and pleural effusions



"bat's wing" shadowing
(alveolar pulmonary oedema)



Heart Sounds

- S_1 → closure of mitral and Tricuspid valves
- S_2 → Closure of aortic and pulmonary valves
  A_2 first and then P_2 (A_2P_2)
- S_3 → From ventricular wall due to abrupt cessation of rapid filling (low pitch, often heard as gallop)
  young ppl, pregnancy, heart failure, mitral Regurgitation
- S_4 → Ventricular origin (stiff ventricle and augmented atrial contraction) related to atrial filling
  . Low pitch
  AFib, left ventricular hypertrophy, hypertrophic cardiomyopathy
- Systolic Clicks → Brief high intensity sound
  Vulvular aortic stenosis, vulvular pulmonary stenosis, Floppy mitral valve, prosthetic heart sounds

Normal heart sounds [1]

- The first (S1) and second (S2) heart sounds are physiological sounds heard in all healthy individuals.
- The third (S3) and fourth (S4) heart sounds may be physiological (particularly in young adults, pregnant women, and the elderly) or pathological.

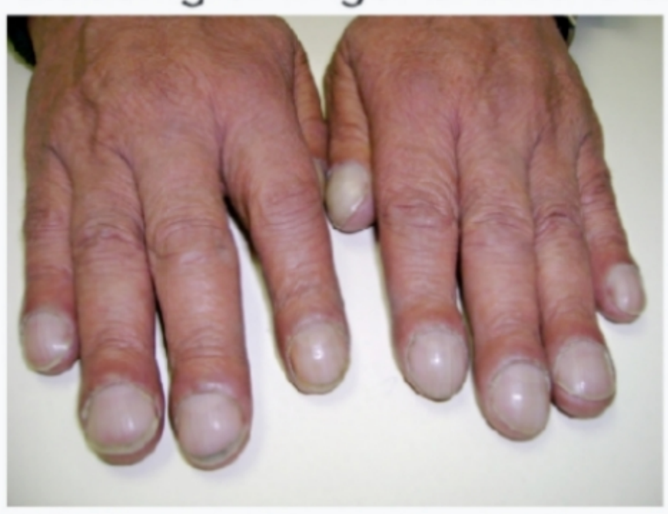
Normal heart sounds				
Sound		**Origin**	**Location**	**Timing**
First heart sound (S1)		• **Closure of the mitral valve and tricuspid valve**	• Heard best in the mitral area (cardiac apex)	• At the onset of systole • Heard just before the carotid pulsation is felt
Second heart sound (S2)	**Aortic component of the second heart sound**	• Closure of the aortic valve (louder)	• Heard best in the aortic area at the right upper sternal border	• During the transition from systole to diastole • Heard immediately after the carotid pulsation • See also "Splitting of S2."
	Pulmonary component of the second heart sound	• Closure of the pulmonary valve (softer)	• Heard best in the pulmonary region	

*Opening Snap → high pitch, brief duration, early in diastole

Opening of stenosed leaflets of mitral valve, prosthetic heart sounds

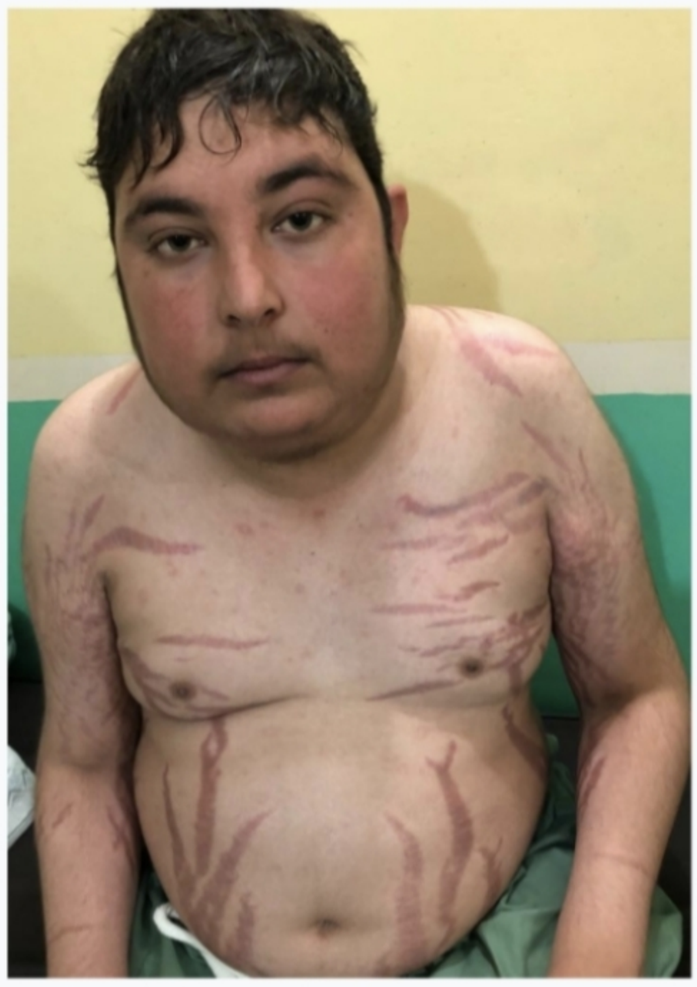
Clubbing

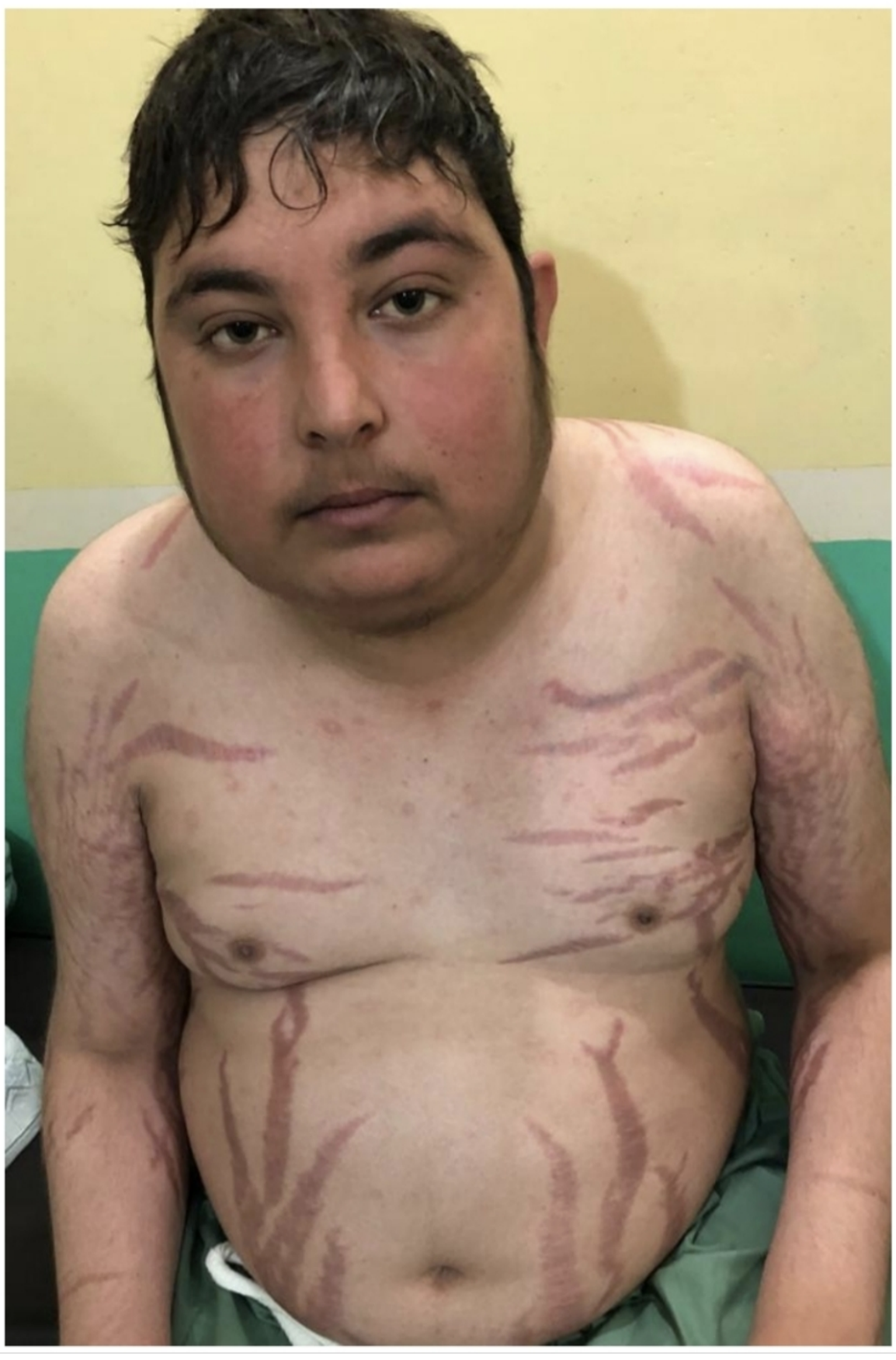
Clubbing of finger & toes is commonly seen in cyanotic heart diseases, IE, atrial myxoma in addition to pulmonary/GI diseases & primary biliary cirrhosis.

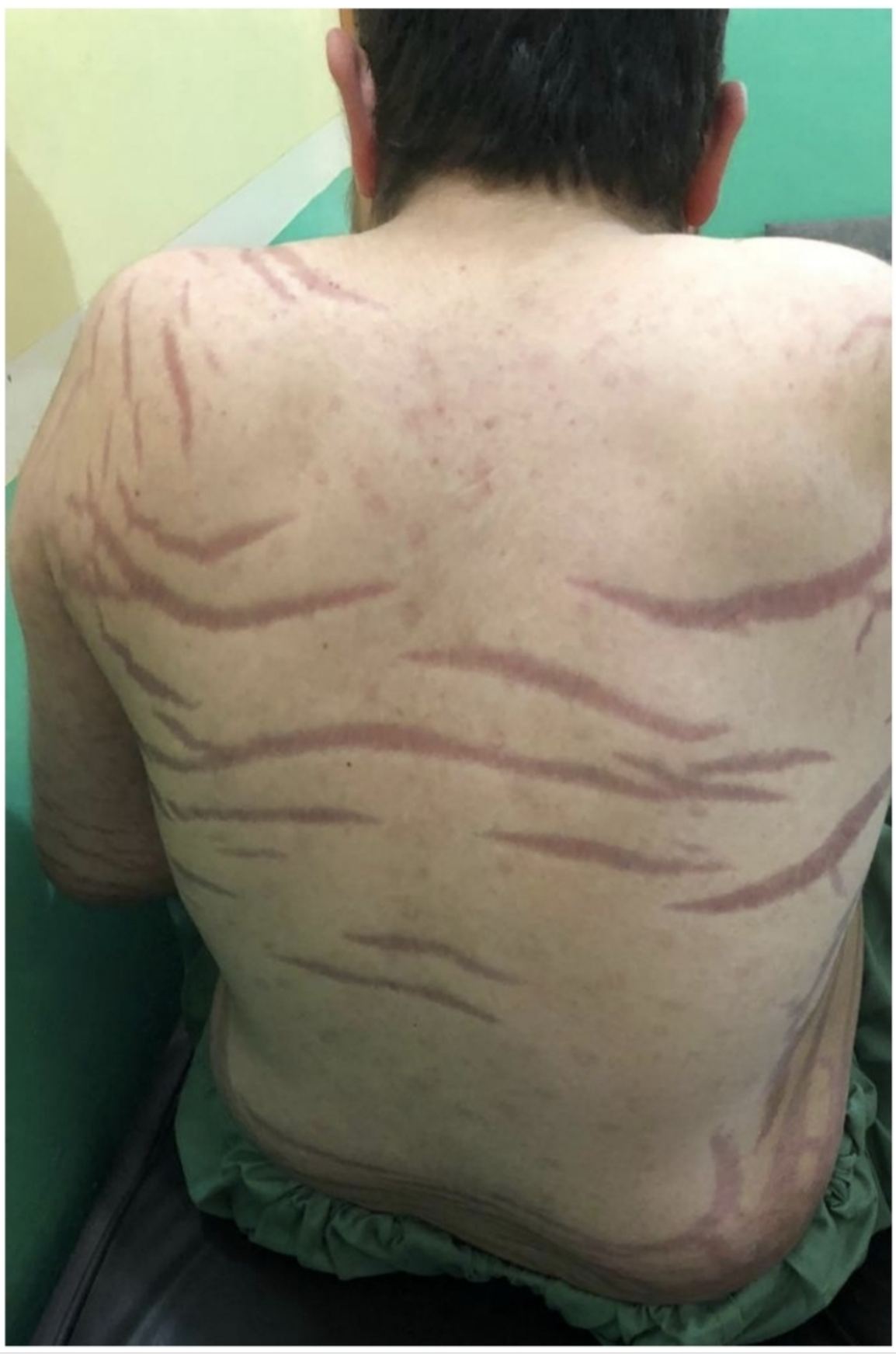


Summarise

Cushing syndrome there is excess of glucocorticoids, leading to central obesity, moon shaped greasy face with facial hairs, diabetes, proximal myopathy,, pink stria & secondary hypertension.



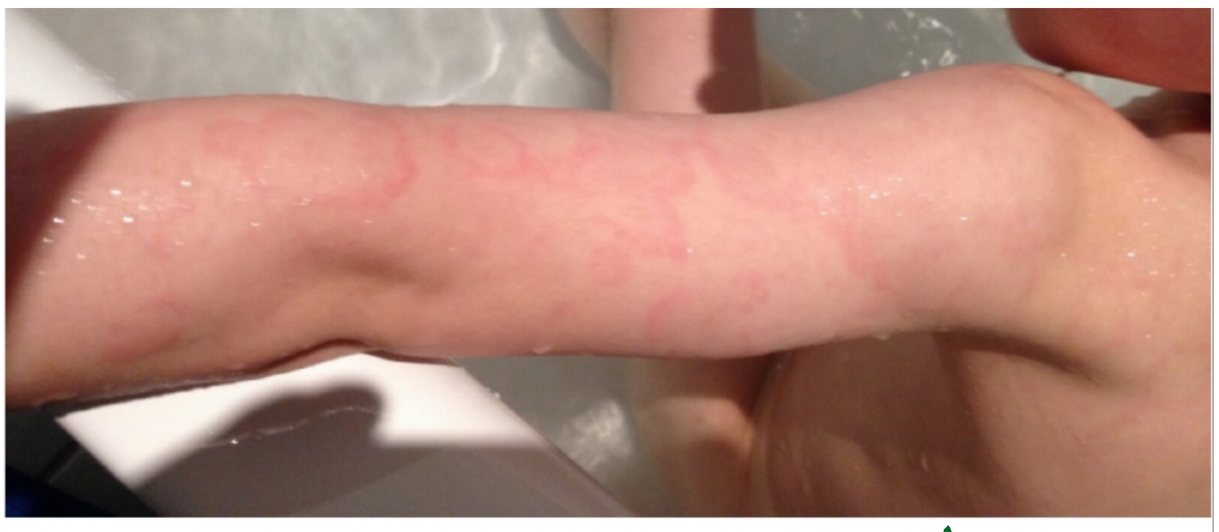




Clinical features of right-sided heart failure

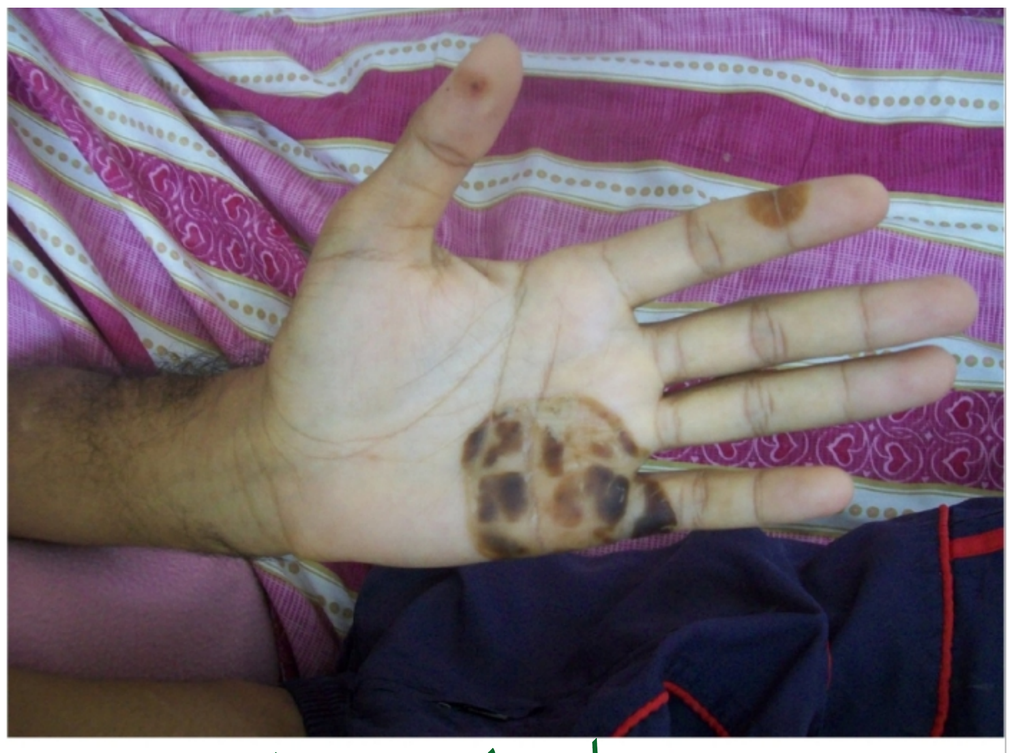
- **Symptoms of fluid retention and increased CVP**
  - **Peripheral** pitting edema: as a result of fluid transudation due to increased venous pressure
  - Hepatic venous congestion symptoms
    - Abdominal pain
    - Jaundice
  - Other symptoms of organ congestion (e.g., nausea, loss of appetite in congestive gastropathy)
- **Physical examination findings**
  - **Jugular venous distention**: visible swelling of the jugular veins due to an increase in CVP and venous congestion
  - Kussmaul sign
  - Hepatosplenomegaly: may result in cardiac cirrhosis and ascites
  - **Hepatojugular reflux**: jugular venous congestion induced by exerting manual pressure over the patient's liver → ↑ right heart volume overload → inability of the right heart to pump additional blood → visible jugular venous distention that persists for several seconds

Rheumatic Fever



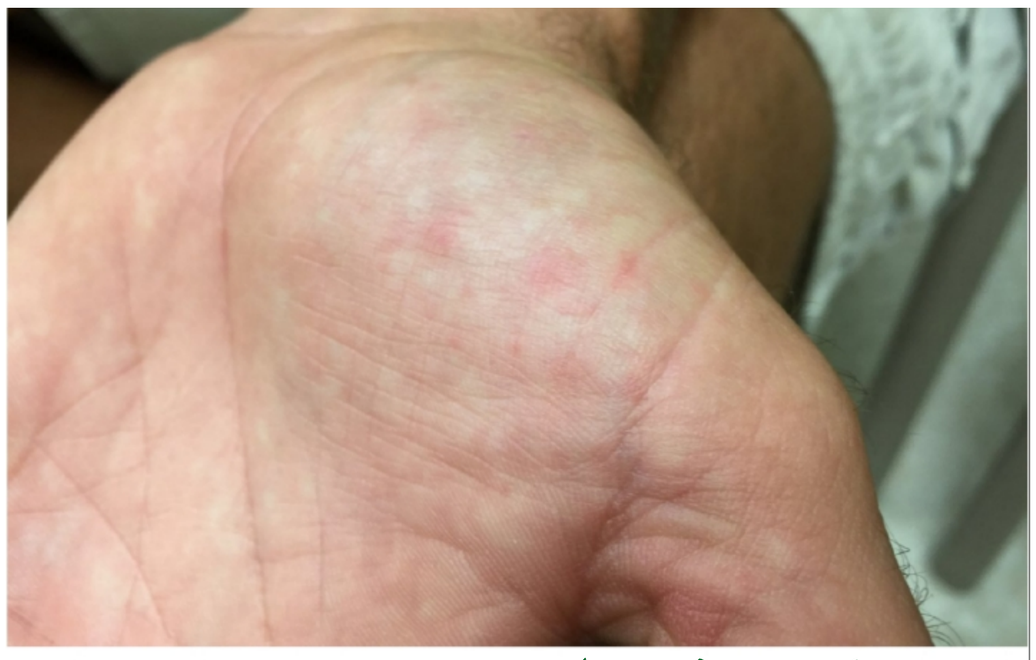
Erythema Marginatum

Erythema marginatum - Left upper arm of a child diagnosed with acute rheumatic fever (ARF) Erythematous plaques with areas of central clearing are visible on the ventral aspect of the forearm. This is characteristic of erythema marginatum, the characteristic annular erythema occuring in 10% of attacks of acute rheumatic fever in children, caused by group A streptococcus.



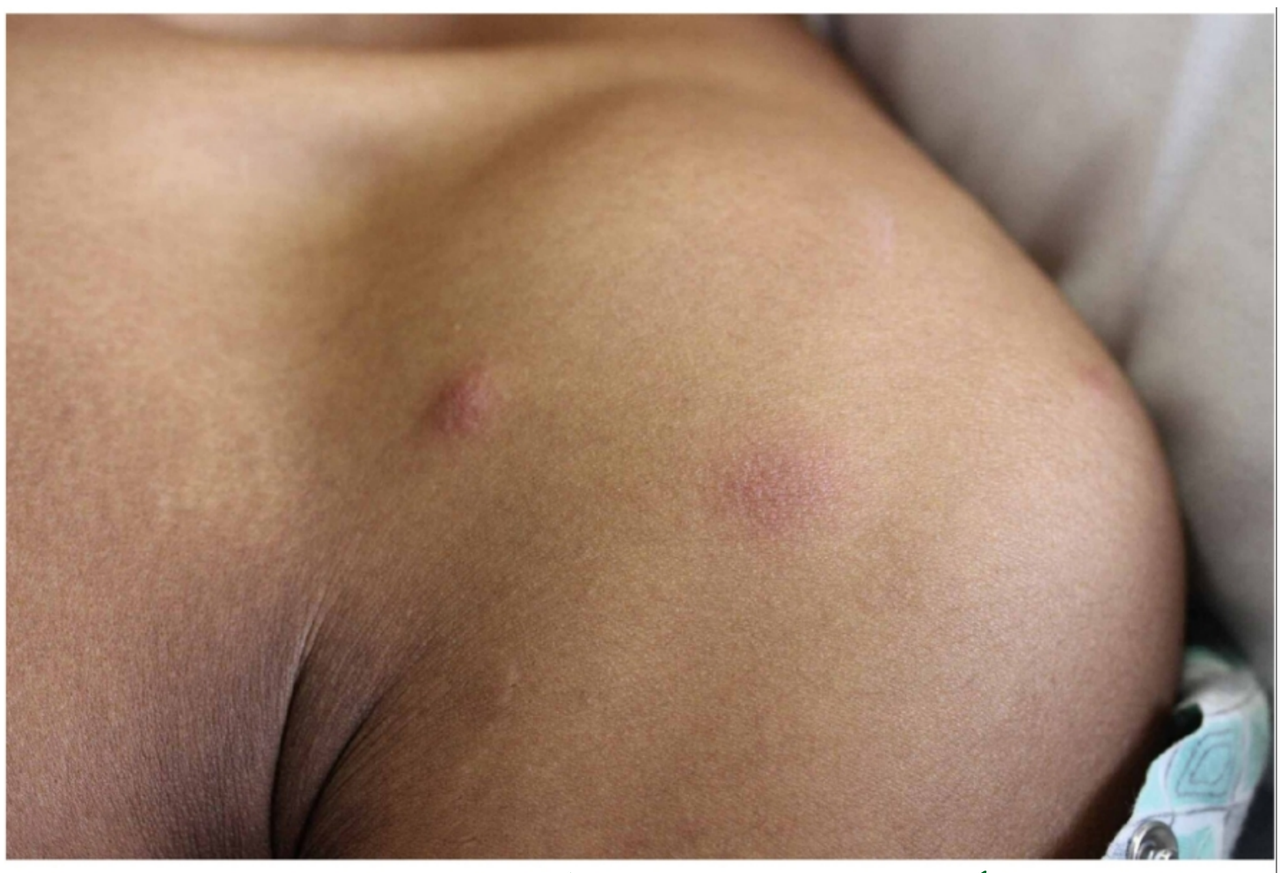
Osler Nodes

Osler nodes - There are multiple raised nodular lesions on the palm and palmar aspects of the first and second digits. The lesions appear dark brown, indicating subcutaneous hemorrhages. This is the typical appearance of Osler nodes.



Janeway lesions

Janeway lesions - Multiple erythematous macules are visible on the thenar eminence and the base of the thumb. These Janeway lesions are typically caused by septic microembolisms secondary to infective endocarditis.



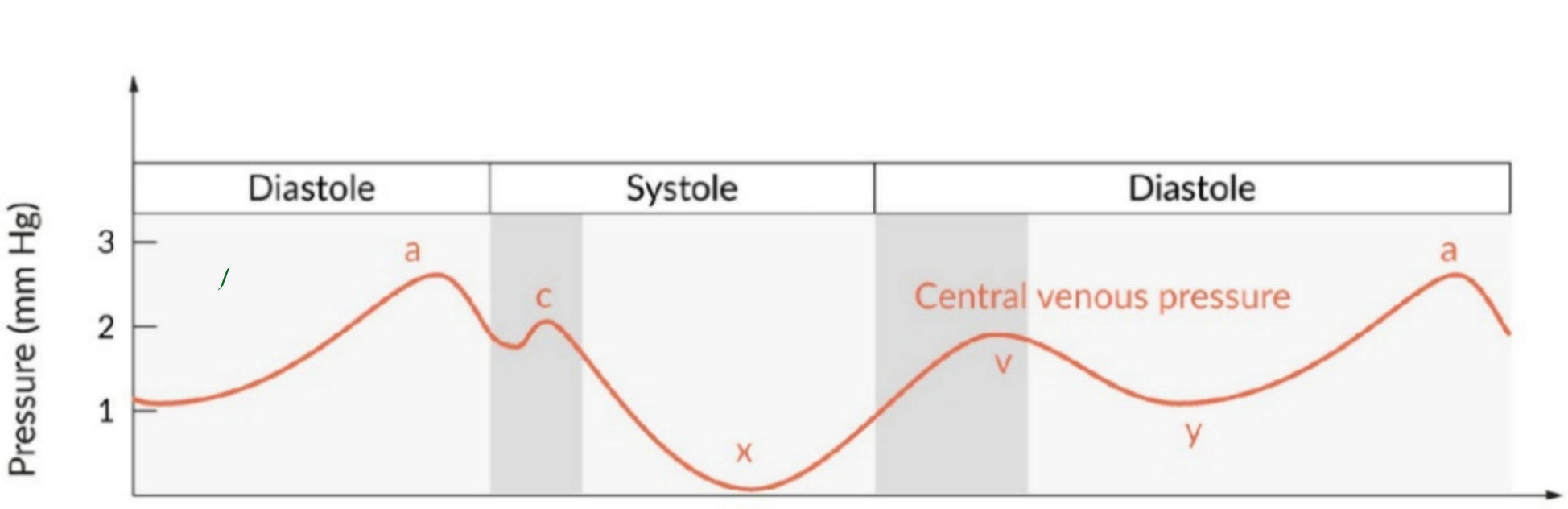
Subcutaneous Nodules

Subcutaneous nodules in rheumatic fever - Solitary subcutaneous, erythematous, warm and tender protuberances are visible on the left shoulder. In conjunction with flu-like symptoms, ankle swelling, and an elevated antistreptolysin-O (ASO) titer, these skin findings indicate rheumatic fever.

A normal JVP waveform consists of three waves (a, c, v) and two descents (x, y).

JVP waves and abnormalities [1]

Wave	Description	Abnormalities
a wave	• The first peak caused by atrial contraction	• Absent in atrial fibrillation
c wave	• The second peak caused by tricuspid valve closure, contraction of the right ventricle, and bulging of the tricuspid valve into the right atrium	• cv wave : severe tricuspid valve regurgitation
x descent	• A drop in JVP caused by atrial relaxation	• Absent in: ◦ Tricuspid valve regurgitation ◦ Right heart failure
v wave	• The third peak caused by venous refilling of the right atrium against the closed tricuspid valve	• Prominent in: ◦ Tricuspid valve regurgitation ◦ Right heart failure
y descent	• A drop in JVP caused by decreased right atrial pressure as blood flows into the right ventricle after opening of the tricuspid valve	• Prominent in: [7] ◦ Tricuspid valve regurgitation ◦ Constrictive pericarditis • Absent in: ◦ Cardiac tamponade ◦ Tricuspid valve stenosis

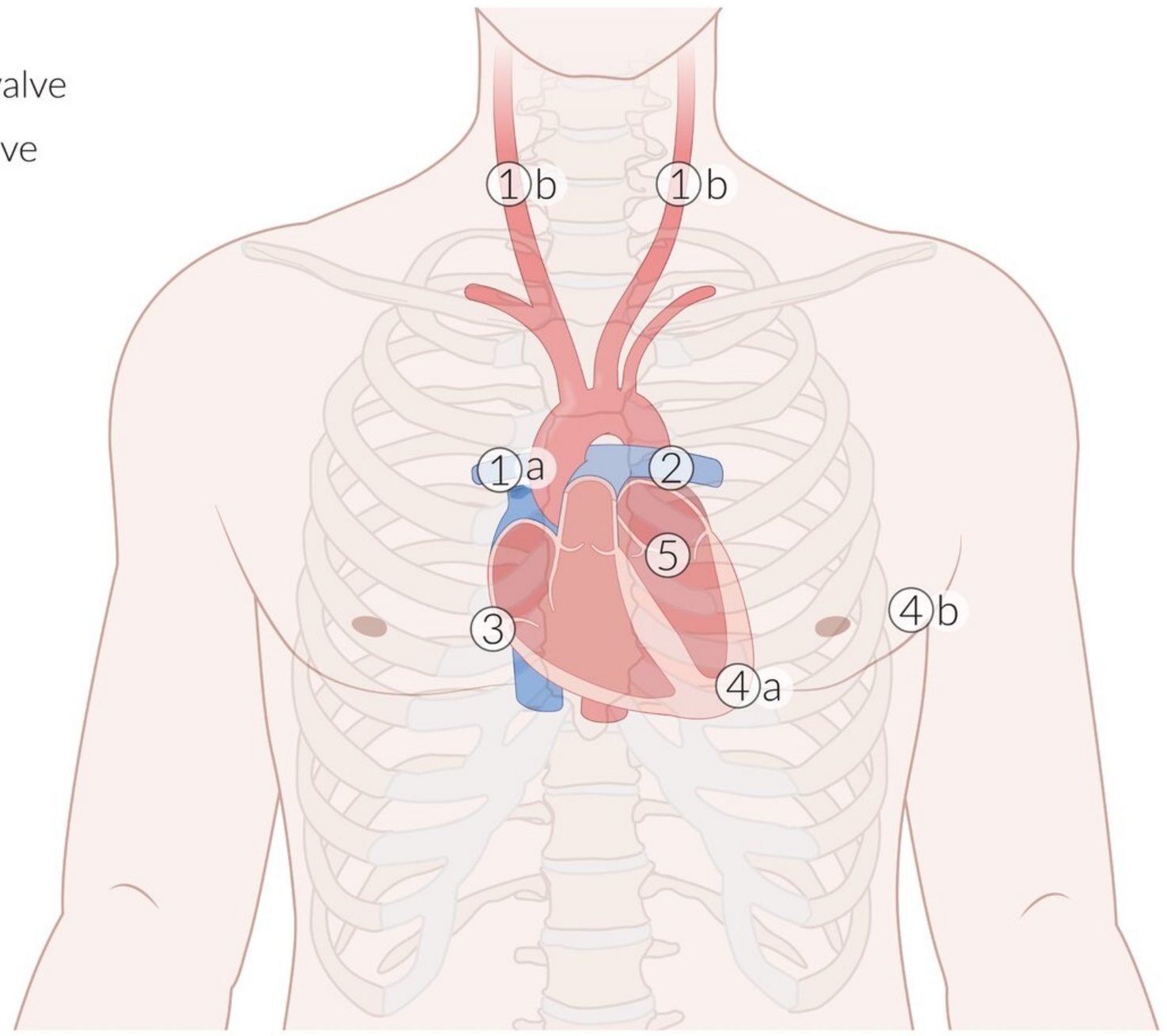


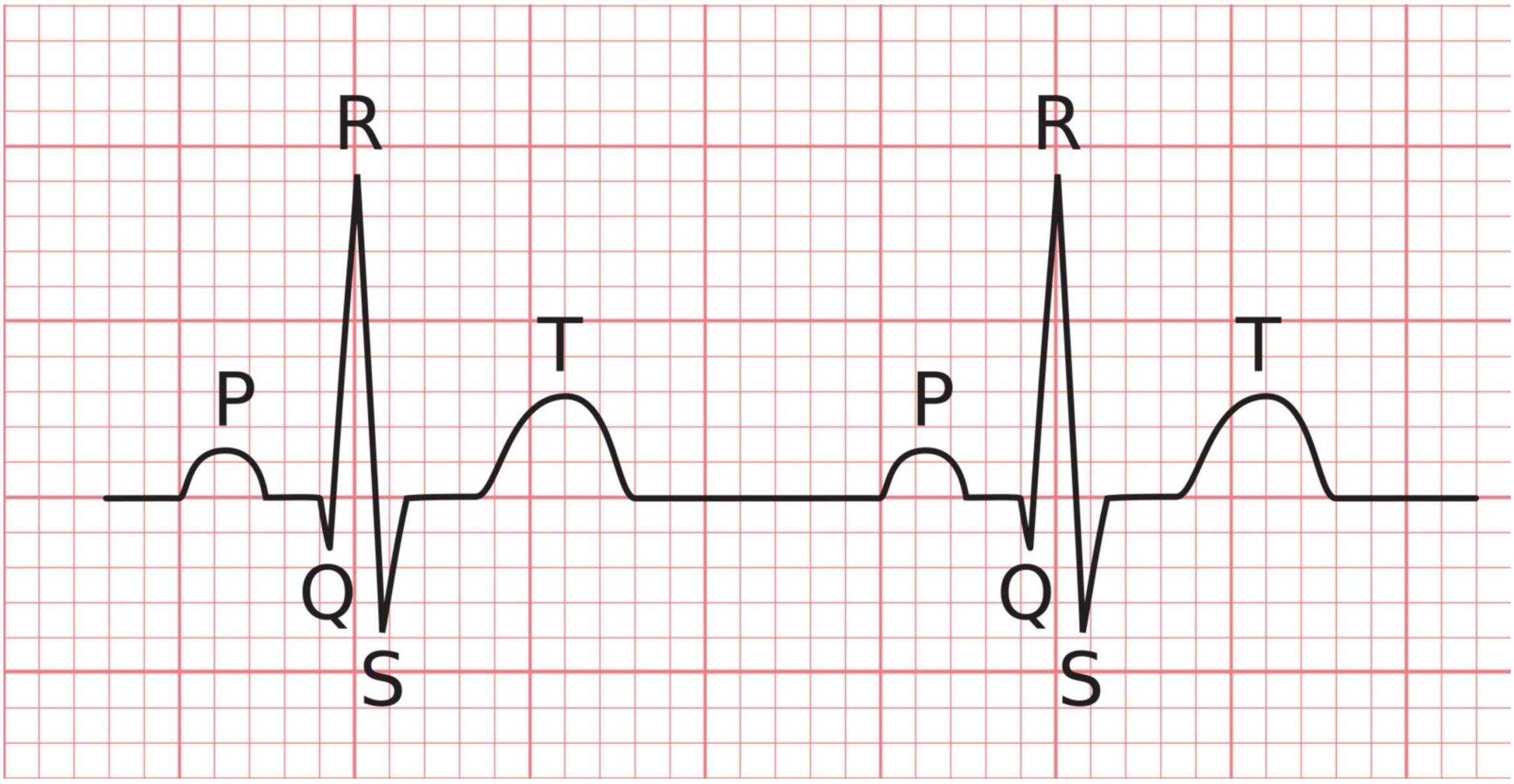
Auscultatory locations [1]

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




R
T
P
Q
S
R
T
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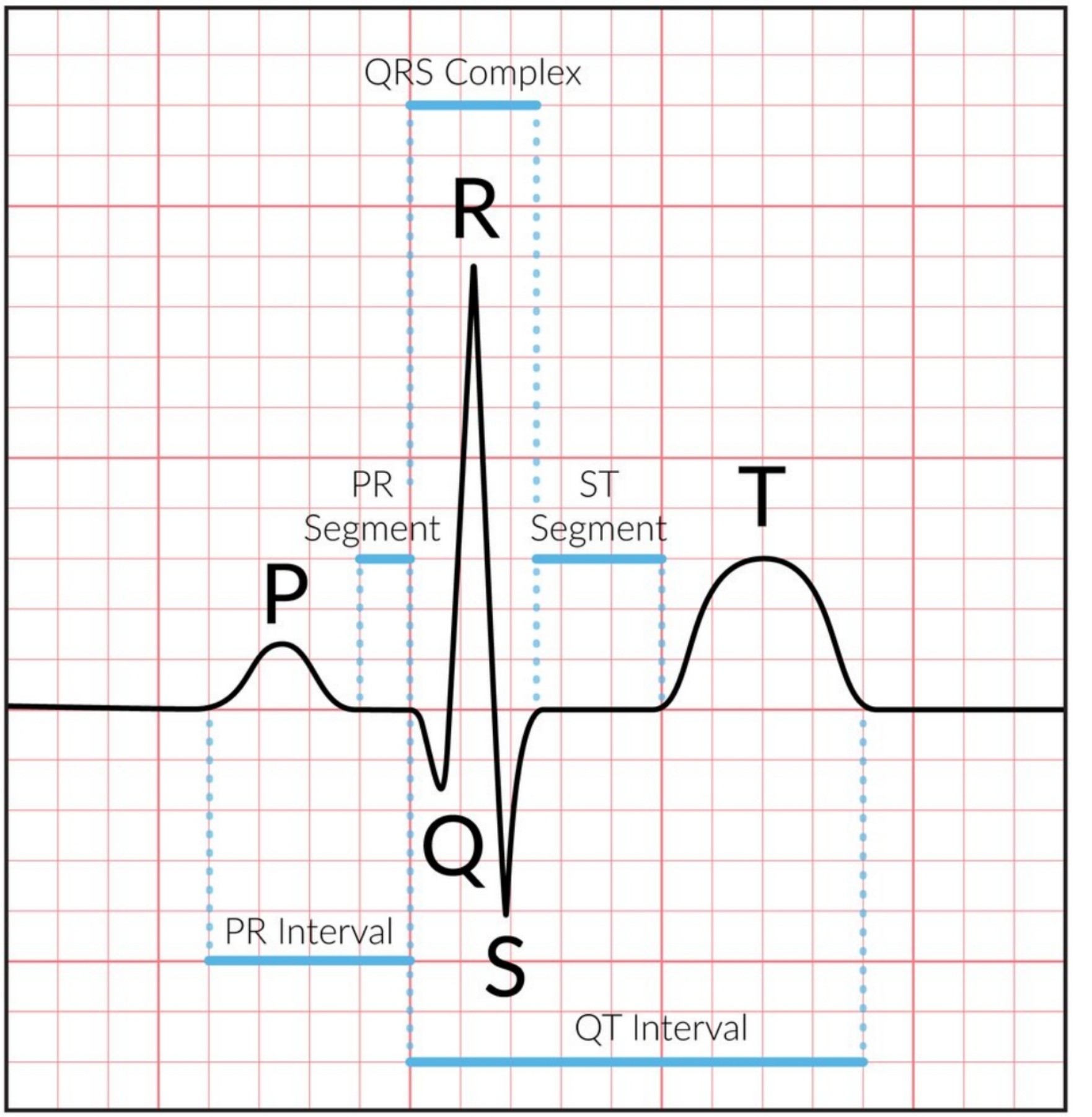
S


QRS Complex
R
PR
Segment
ST
Segment
T
P
Q
S
PR Interval
QT Interval

Localization of myocardial infarct on ECG [9][13][14]

ECG leads affected	Infarct location	Vessel involved [12][14]
V_1–V_6	• Extensive anterior MI (leads aVL and I may also be affected.)	• Proximal left anterior descending artery (LAD)
V_1–V_2	• (Antero)septal MI	• LAD
V_3–V_4	• (Antero)apical MI	• Distal LAD
V_5–V_6	• (Antero)lateral MI	• Diagonal branch of LAD • Distal LAD • Left circumflex artery (LCX) • In rare cases, can also be caused by right coronary artery (RCA) infarct
I, aVL	• Lateral MI	• Proximal LCX
II, III, aVF V_3R–V_6R	• Inferior MI	• RCA (more common) • Distal LCX (less common)
V_7-V_9	• Posterior/posterolateral MI	• Posterior descending artery (from RCA or LCX) • Reciprocal ST depressions in V_{1-4} may also be seen [12]

* Most important biological marker of myocardial necrosis is Cardiac Troponin (cTn)

* Cardiac Biomarkers
  - Troponin T
  - Troponin I
  - CK-MB
  - Myoglobin

* MI Diagnosis
  - ECG
  - Cardiac Biomarkers
  - CBC → for anemia, thrombocytopenia, elevated inflammatory markers
  - Coronary Angiography
  - Transthoracic Echo

ECG lead	V2-V5	V1-V3	V3-V4	V4-V6, aVL, I	I,aVL	II,III, aVF
Infarct location	Extensive anterior	(Antero) septal	(Antero) apical	(Antero) lateral	Lateral	Inferior
Vessel involved	Proximal LAD	LAD	Distal LAD	Distal & Diagonal branch of LAD or LCX	Proximal LCX	RCA (PDA)

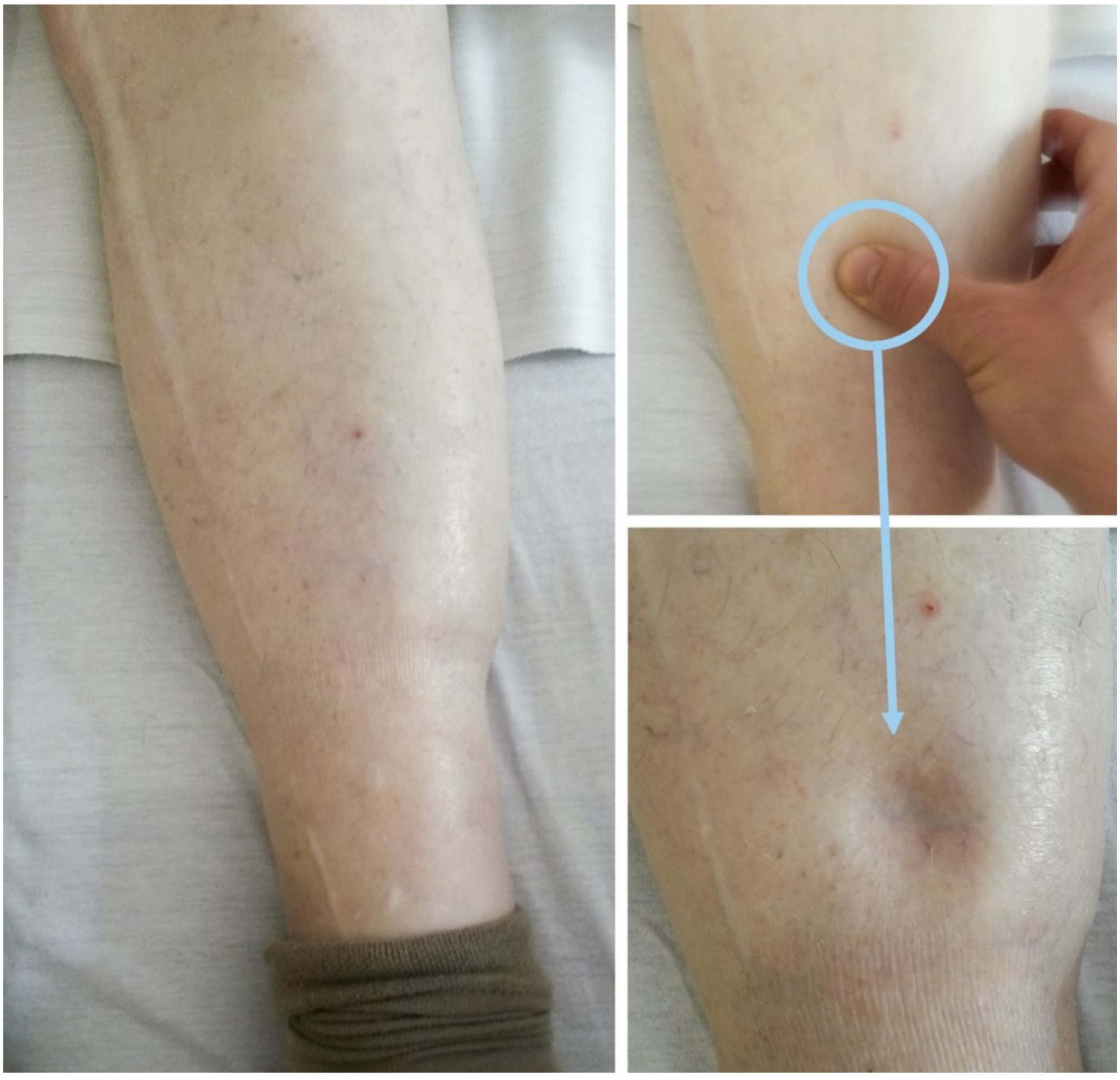
Left Sided Heart Failure

- Physical examination **findings** [17]
  - Bilateral basilar rales may be audible on auscultation.
  - Laterally displaced apical heart beat (precordial palpation beyond the midclavicular line)
  - Coolness and pallor of lower extremities

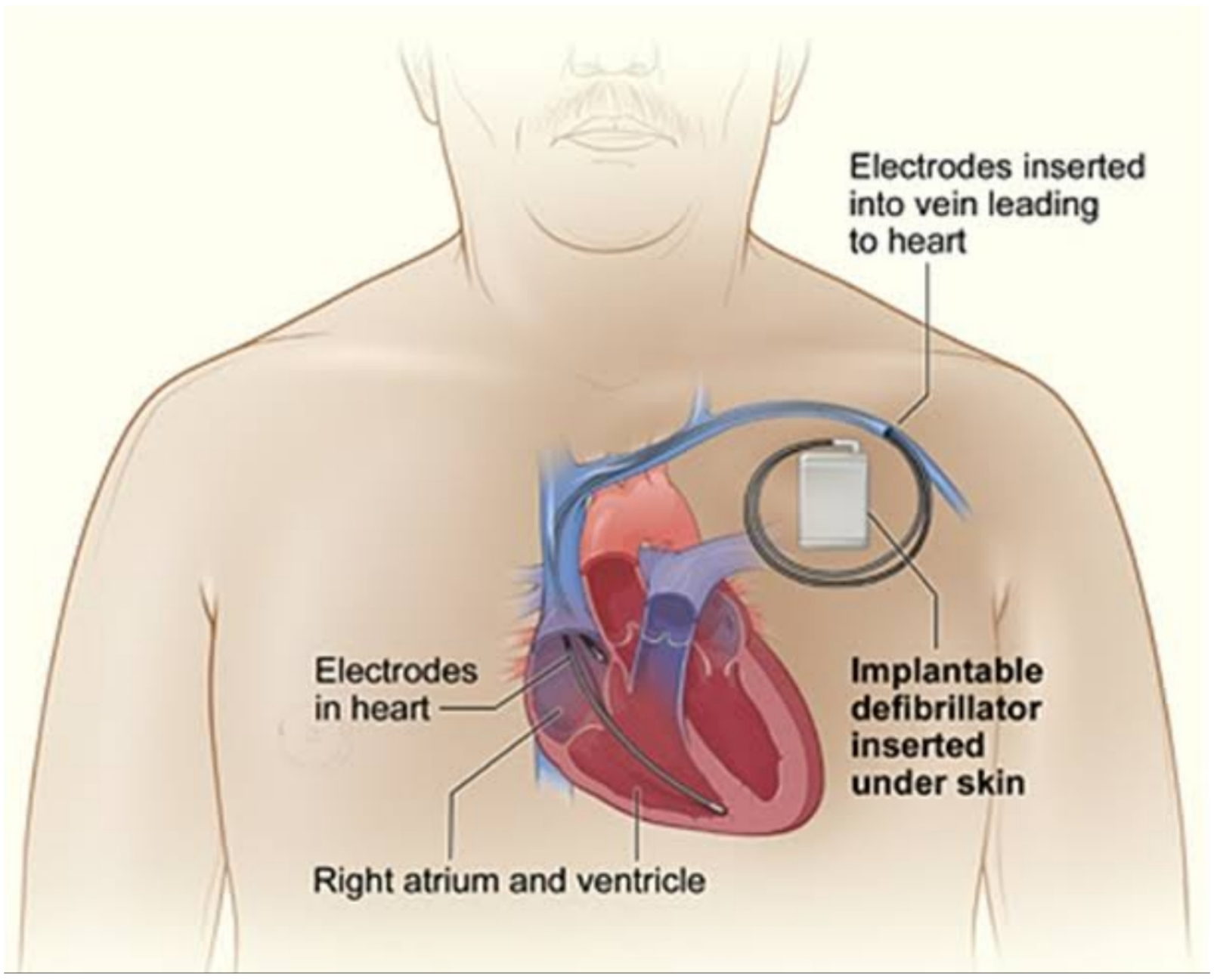
Right Sided Heart Failure

- Physical examination **findings**
  - **Jugular venous distention**: visible swelling of the jugular veins due to an increase in CVP and venous congestion
  - Kussmaul sign
  - Hepatosplenomegaly: may result in cardiac cirrhosis and ascites
  - **Hepatojugular reflux**: jugular venous congestion induced by exerting manual pressure over the patient's liver → ↑ right heart volume overload → inability of the right heart to pump additional blood → visible jugular venous distention that persists for several seconds

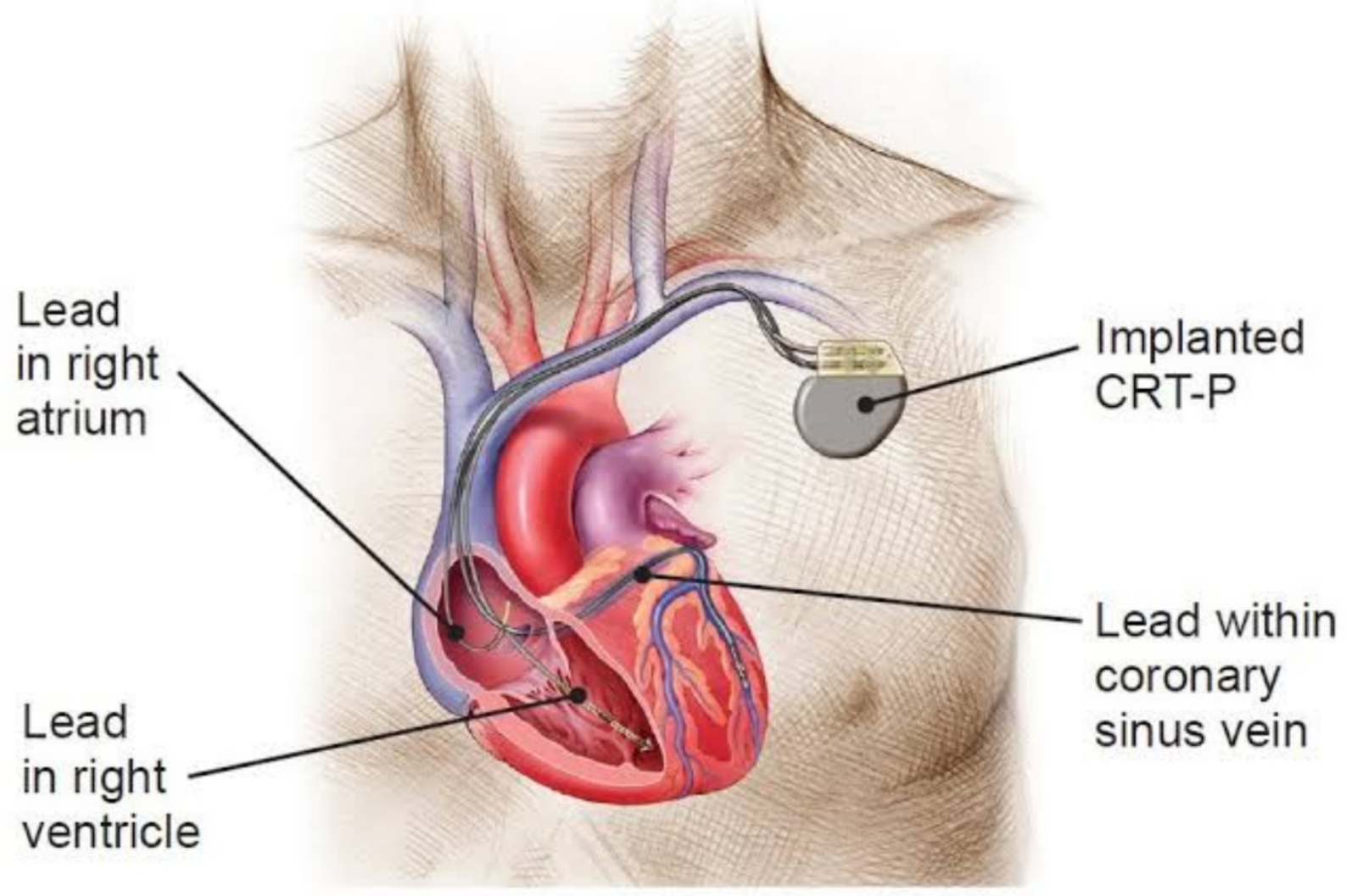
- **Kussmaul sign**: distention of the jugular veins during inspiration due to the negative intrathoracic pressure that attempts to pull blood into the right heart, which is restricted by noncompliant pericardium or myocardium (e.g., constrictive pericarditis, restrictive cardiomyopathy, right atrial tumors, ventricular tumors, right HF, massive PE) [6]



Pitting edema of lower leg



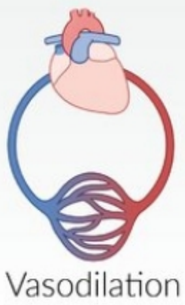
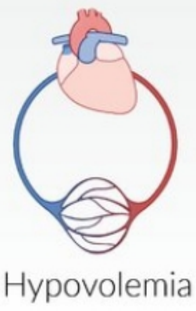
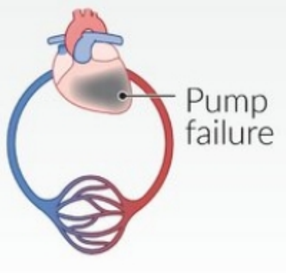
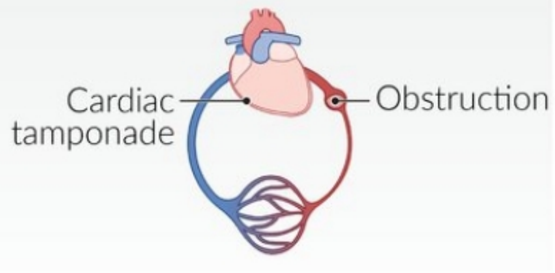
Automated implantable cardioverter defibrillators (AICDs)

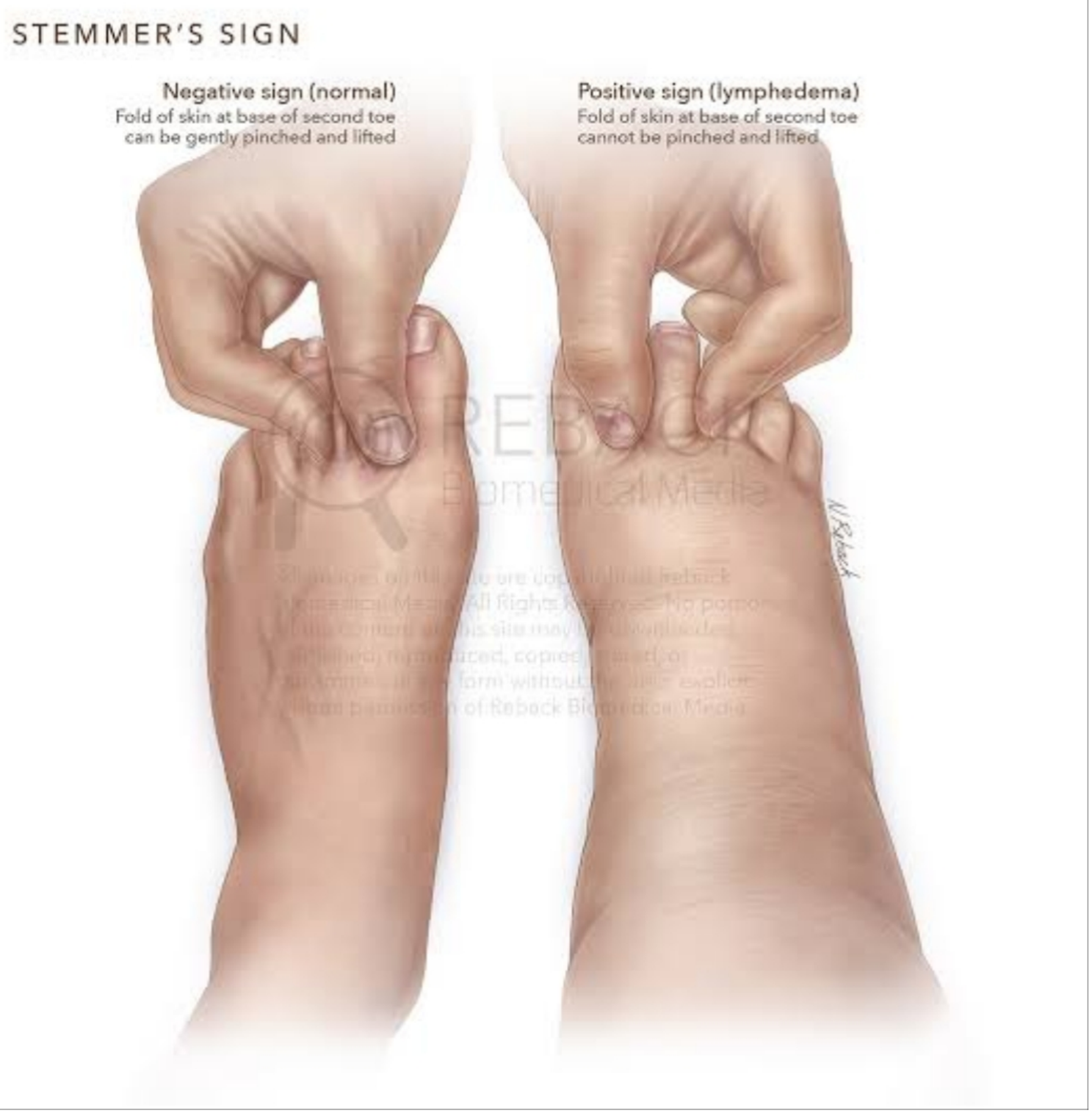


cardiac resynchronization therapy devices (CRTs)

In a patient with tachycardia and cold extremities,
Assume Shock

Classification of Shock

Volume				Output			
Shift Distributive shock		**Loss** Hypovolemic shock		**Cardiac** Cardiogenic shock		**Extracardiac** Obstructive shock	
Septic	Capillary leakage	Hemorrhagic (traumatic or nontraumatic)	Blood (whole)	Myocardial causes	Myocardium	Impaired diastolic filling	E.g., cardiac tamponade
Anaphylactic Anaphylactoid Neurogenic	Vascular tone dysregulation	Nonhemorrhagic (nontraumatic)	Body fluids (e.g., GI loss)	Arrhythmias	Conduction system	↑ Ventricular afterload	E.g., massive PE
		Nonhemorrhagic (traumatic)	Plasma (e.g., from burns)	Valvular heart disease		Obstruction of venous return	E.g., tension pneumothorax
 Vasodilation		 Hypovolemia					



Stemmer's Sign seen in lymphedema

Wheezing can be heard in both acute heart failure and obstructive lung disease (e.g., asthma exacerbation, AECOPD). [35]

Management of Acute Decompensated Heart Failure

(Mnemonic → LMNOP)

* Loop Diuretics (Furosemide)
* Modify medications
* Nitrates
* Oxygen (if hypoxic)
* Position (with elevated upper body)

Acute Rheumatic fever

Acute rheumatic fever (ARF) is an inflammatory sequela involving the heart, joints, skin, and central nervous system (CNS) that occurs two to four weeks after an untreated infection with group A Streptococcus

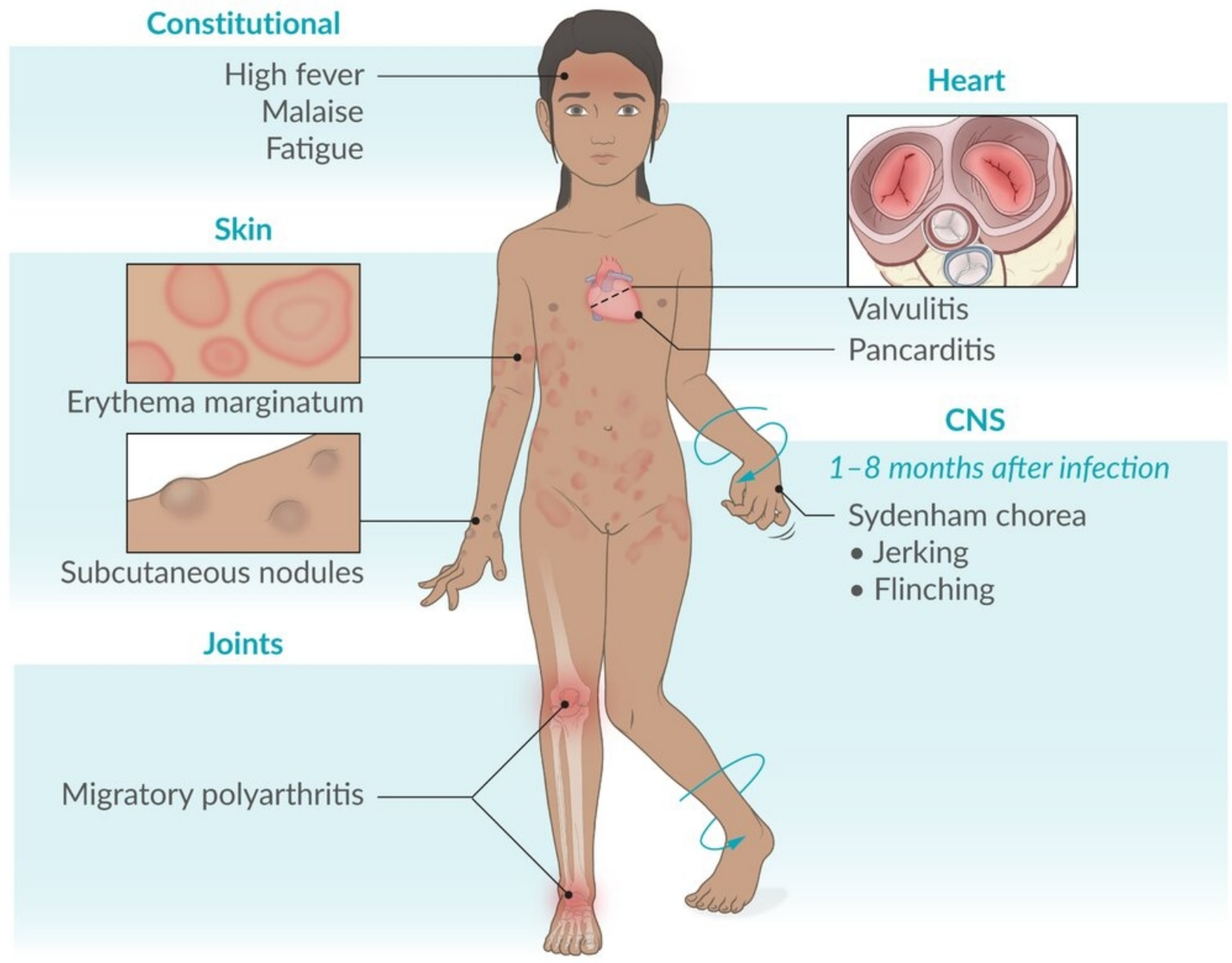
Acute rheumatic fever

Etiology
Previous GAS pharyngitis/tonsillitis without antibiotic treatment

Peak incidence
5–15 years of age

Diagnosis
Evidence of a previous GAS infection (e.g., throat culture, rapid antigen detection test, antistreptolysin O, antistreptococcal DNase B test)
Revised Jones criteria

Treatment
Antibiotic treatment of underlying GAS infection
Symptomatic treatment of arthritis and fever



Pathophysiology and disease course

Untreated GAS pharyngitis/tonsillitis	Development of antibodies	Molecular mimicry	Type II hypersensitivity reaction	Inflammation and cell damage
Acute infection		Latent period (2–4 weeks)		Rheumatic fever onset

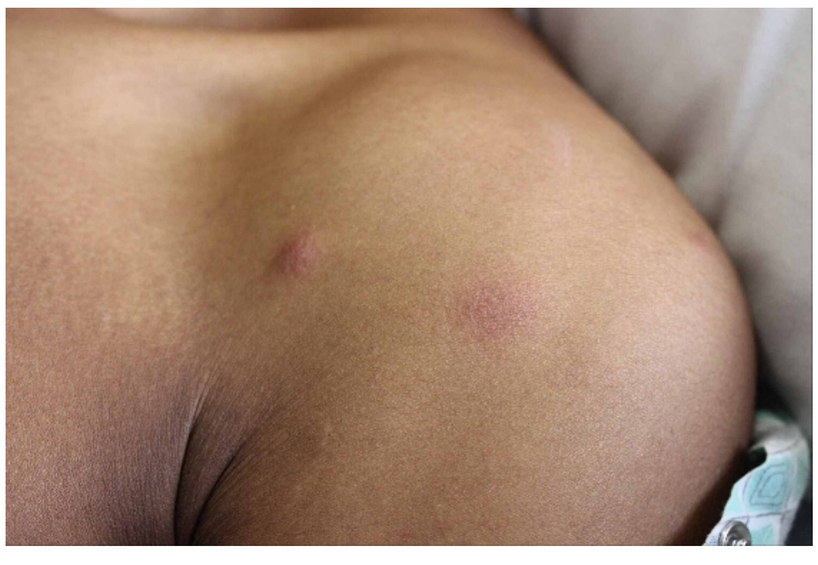
J → Joints (Migratory polyarthritis)

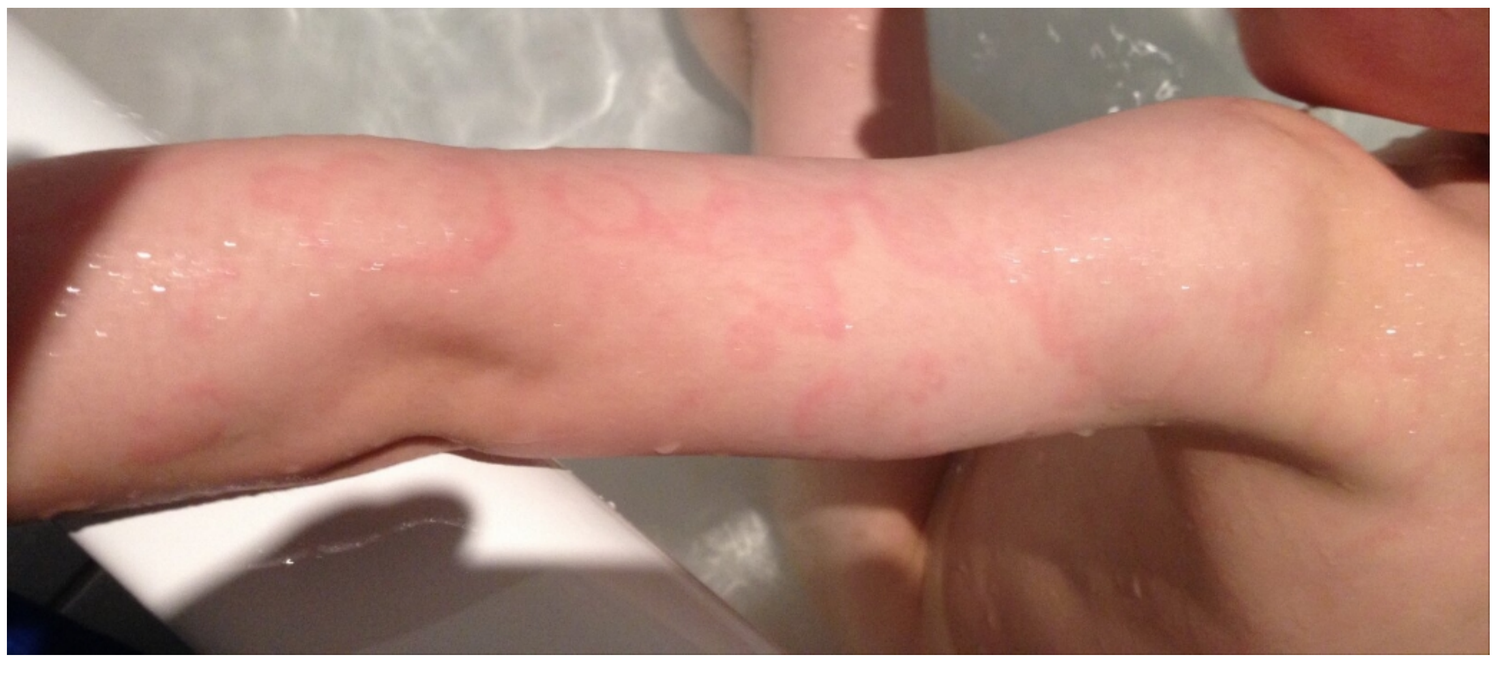
P → Pancarditis

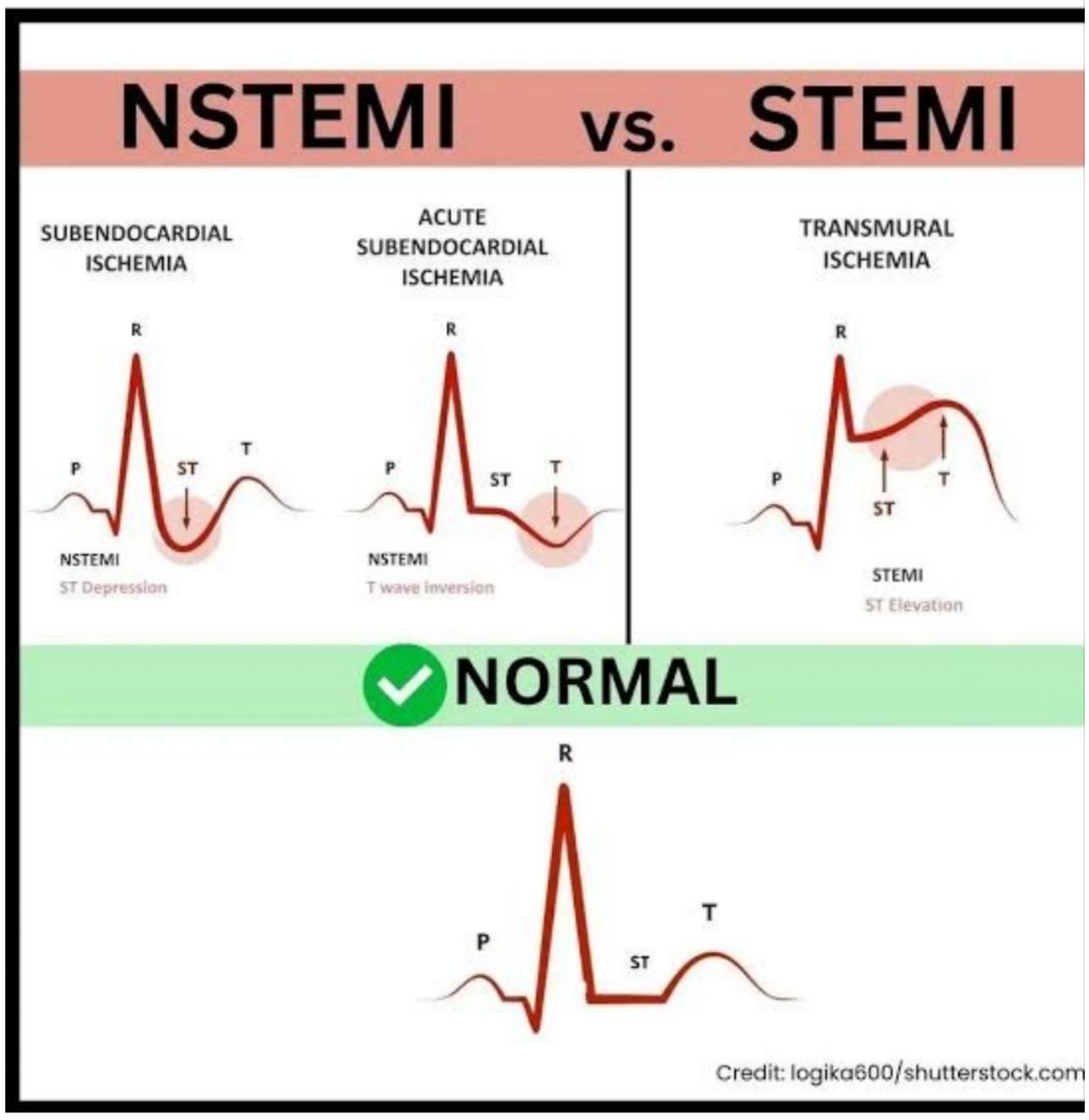
N → Nodules (Skin nodules)

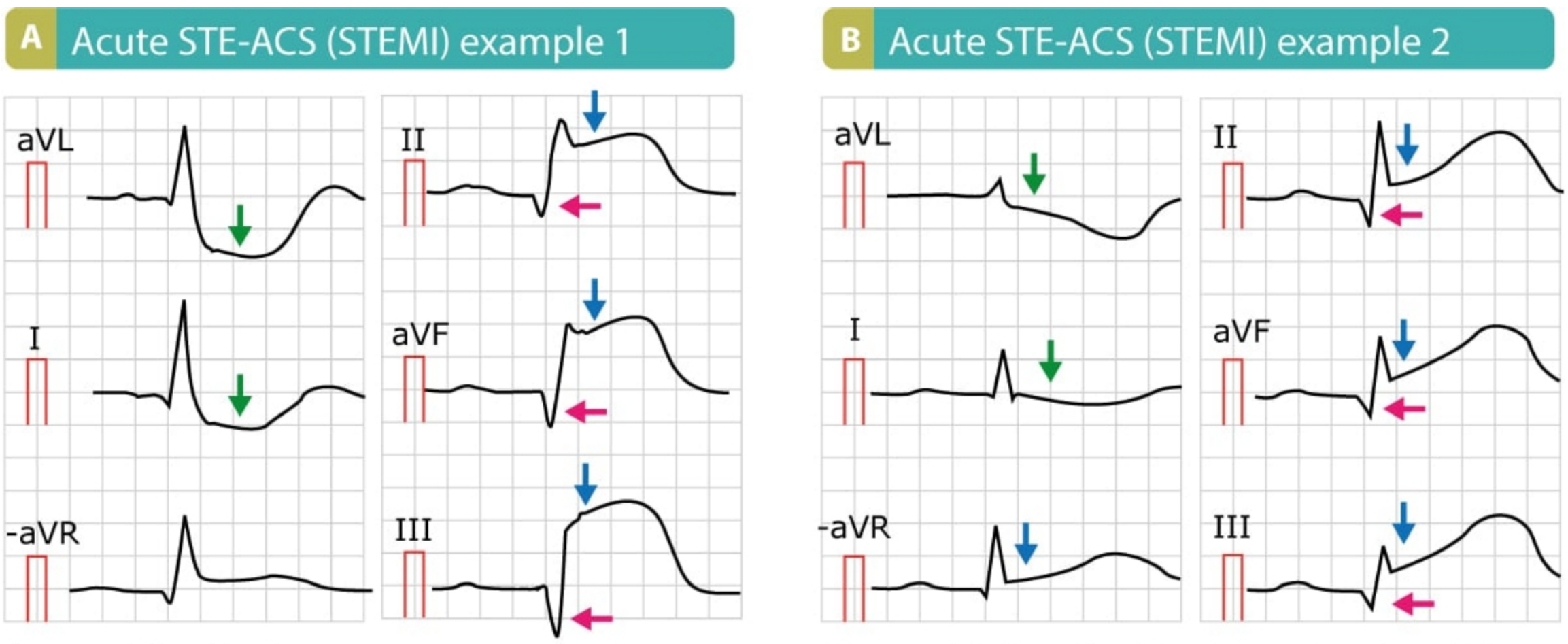
E → Erythema Marginatum

S → Sydenham chorea









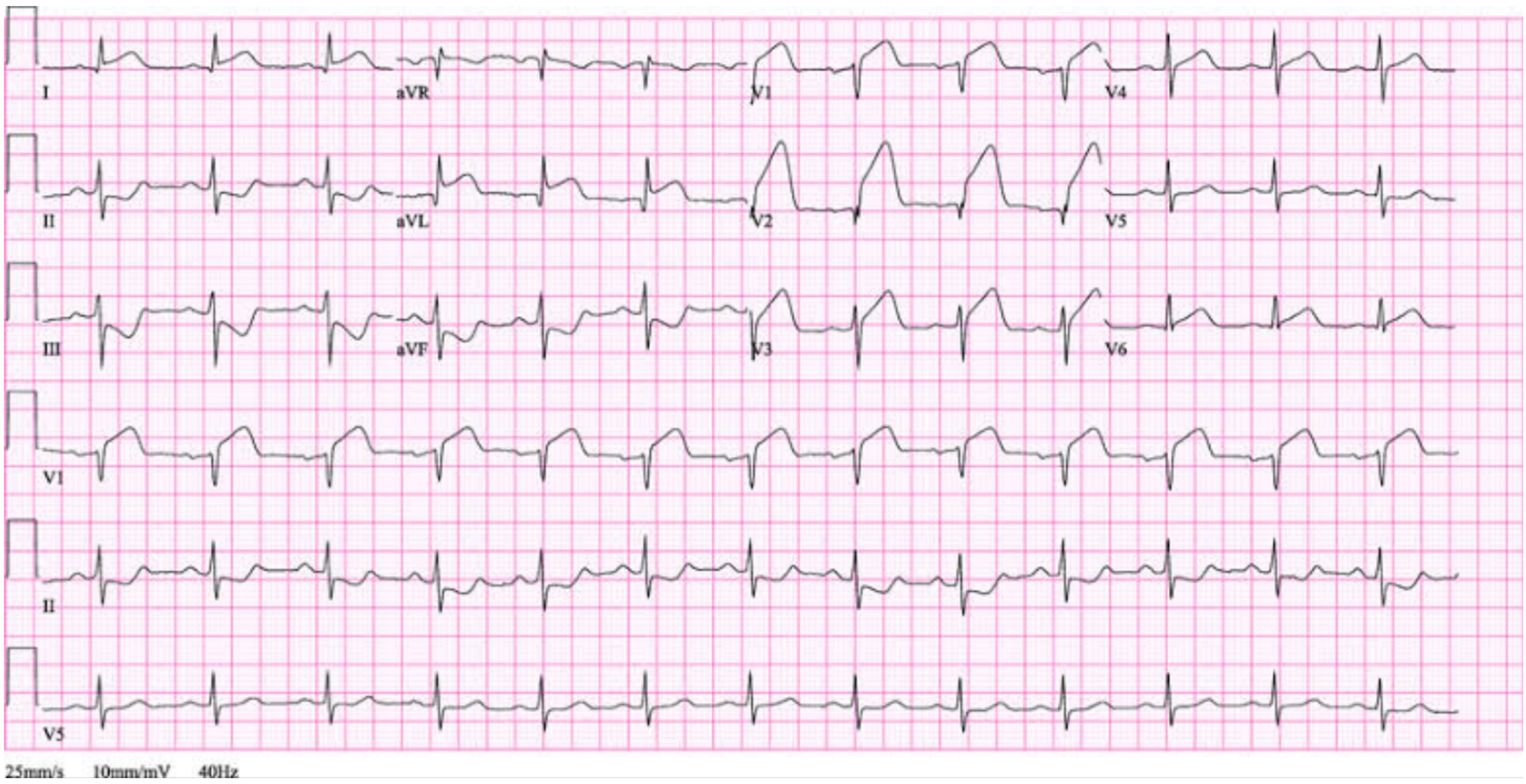
60 year old male with retrosternal chest pain. ECG shows ST segment elevations in inferior leads (II, aVF and III). There are reciprocal ST segment depressions in aVL and I. There are also pathological Q-waves in the inferior leads.

65 year old diabetic with 3 hours duration of chest pain. ECG shows ST-segment elevations, reciprocal depressions and pathological Q-waves.

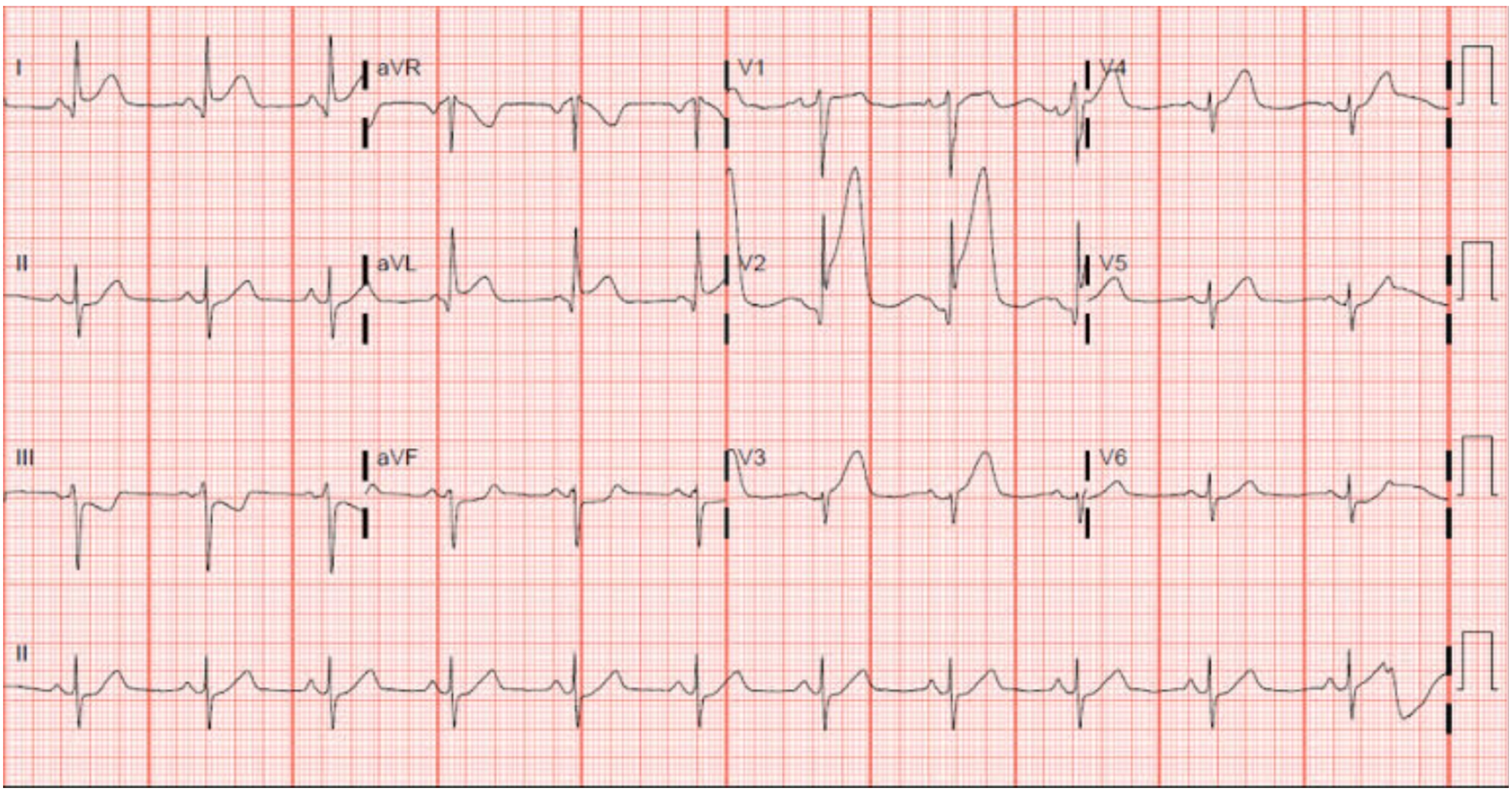
→ ST segment elevation

→ Pathological Q-waves

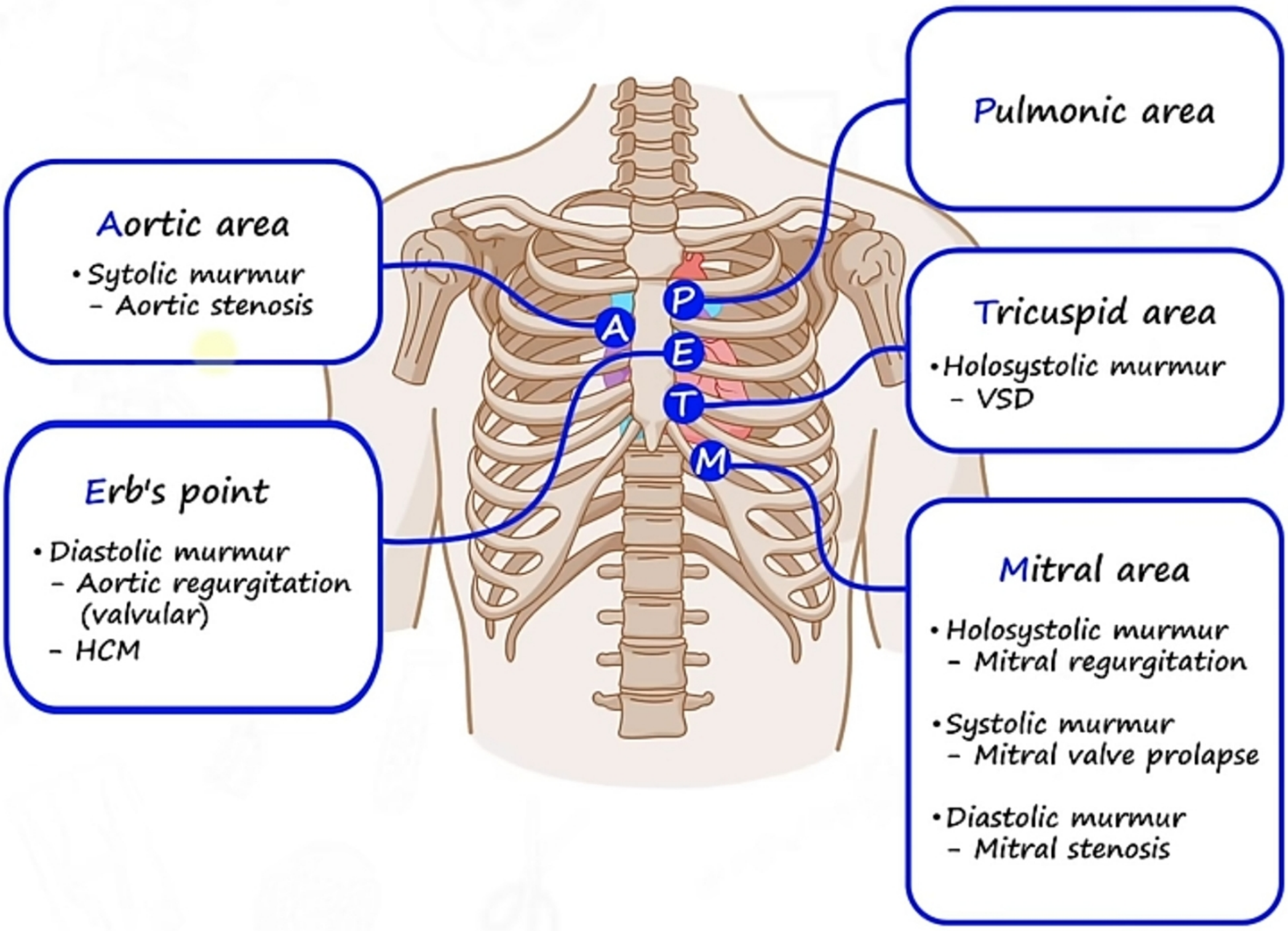
→ Reciprocal ST-segment depression

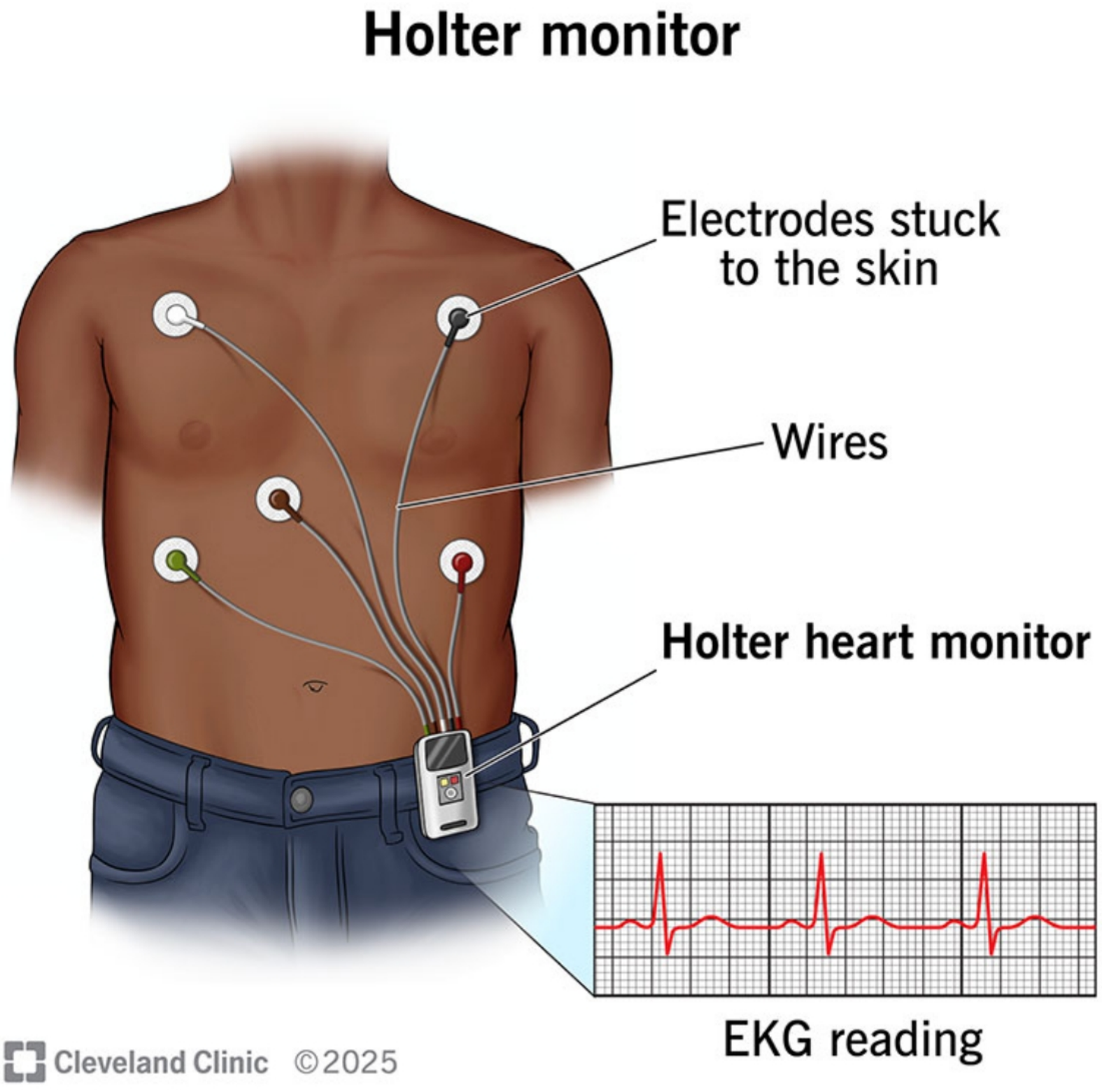

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz

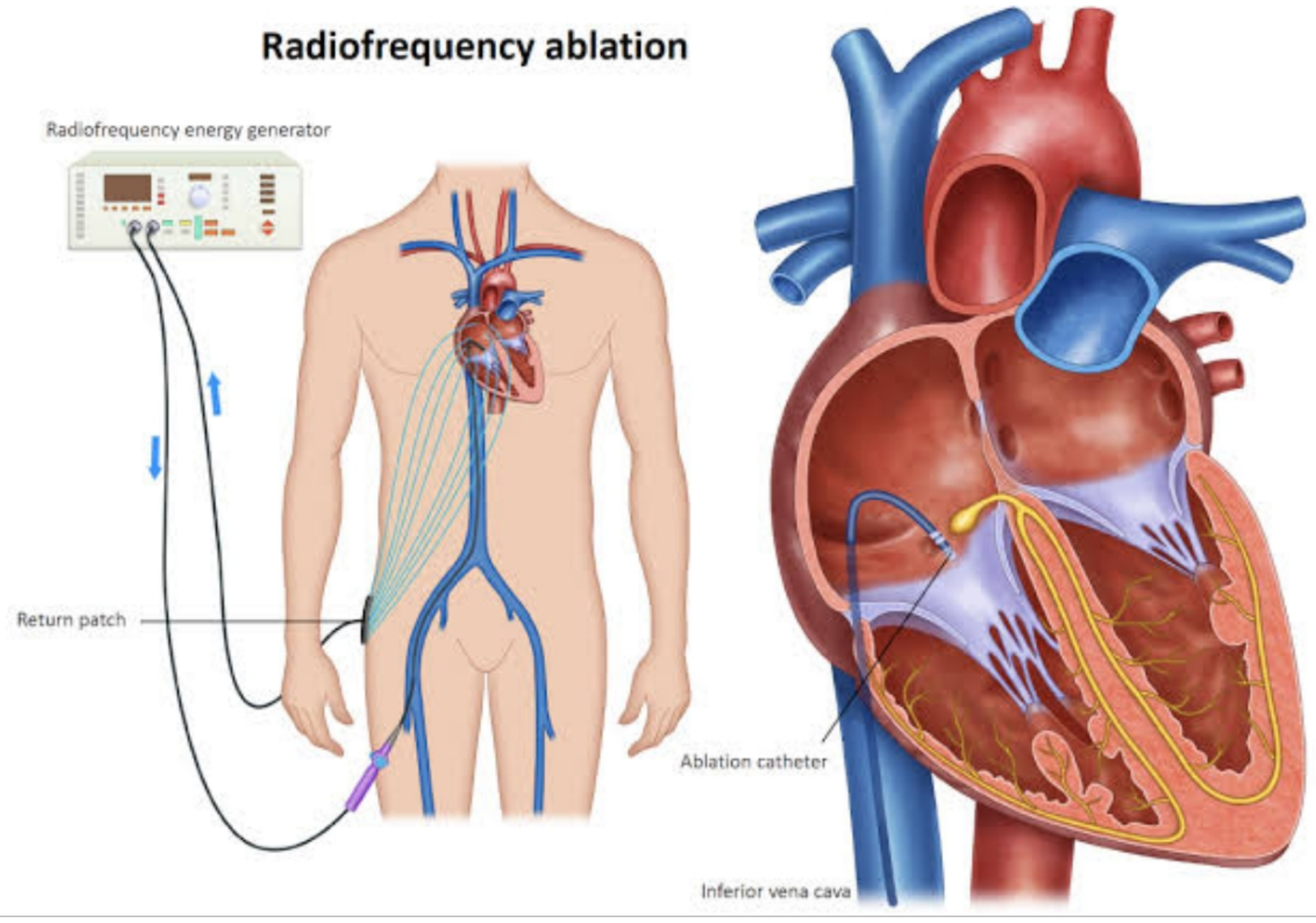
STEMI


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

1. Location of Murmur




Holter monitor
Electrodes stuck to the skin
Wires
Holter heart monitor
EKG reading
Cleveland Clinic ©2025



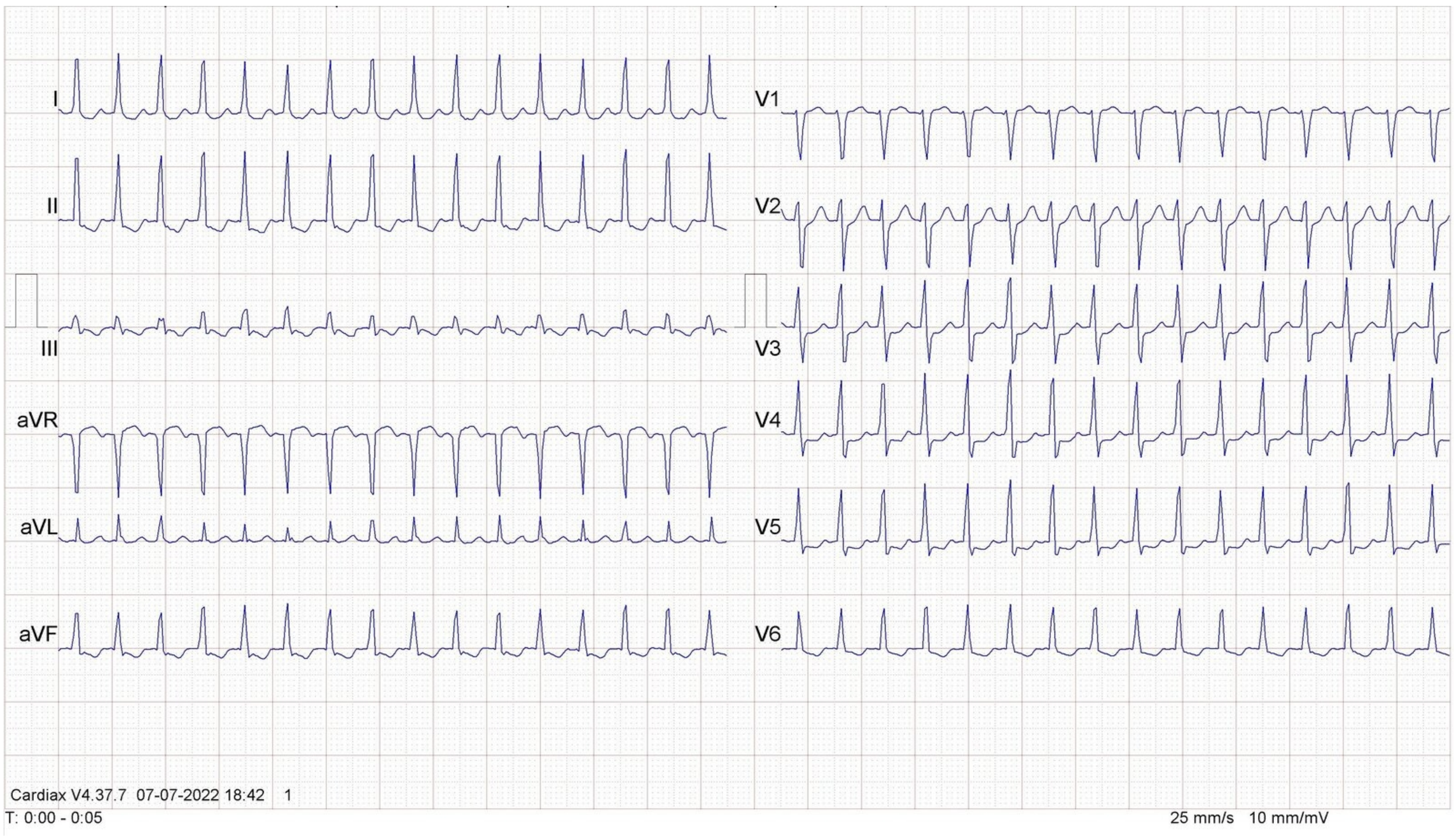
Catheter ablation

- **Description**
  - Radiofrequency or cryothermal energy is applied via a cardiac catheter to eliminate aberrant pathways or impulses from arrhythmogenic foci.
  - Usually performed in conjunction with an EP study
- **Indications** [2]
  - **Curative therapy** in AVNRT, AVRT with concealed pathway, or drug-refractory AT
  - Symptomatic patients who want to avoid long-term drug therapy (especially younger patients)
  - Asymptomatic patients with special lifestyle considerations (e.g., pilots)

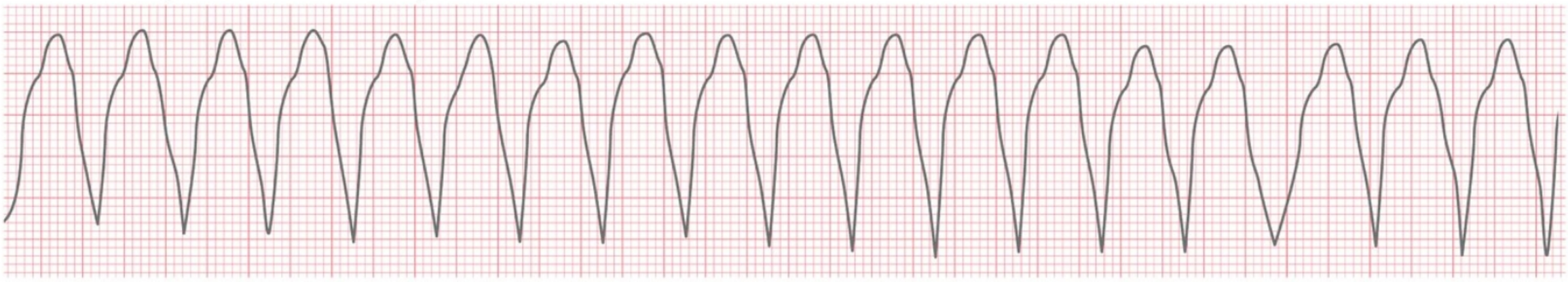
ECG findings in AVNRT [24][25]

ECG may be normal between episodes of tachycardia. Findings may be indistinguishable from those of orthodromic AVRT and include:

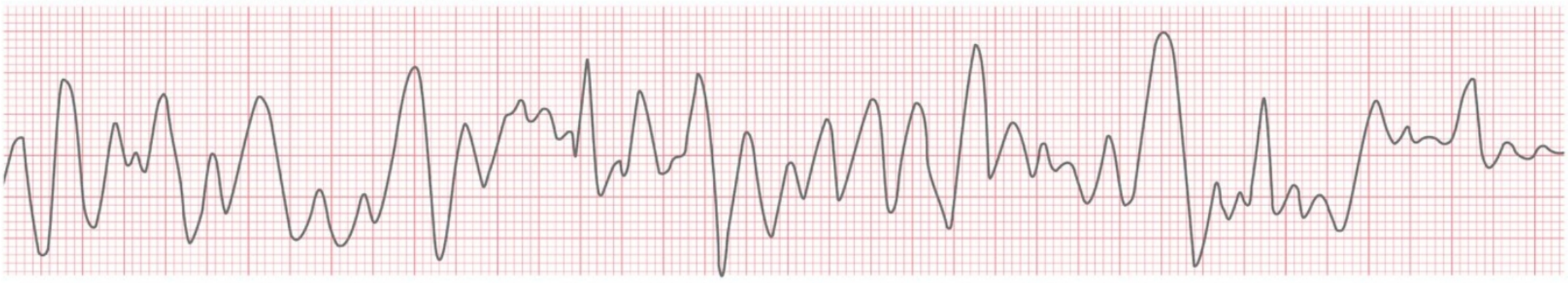
- Heart rate typically 150–220/minute
- Typically narrow QRS complexes (a wide QRS complex may be seen if there is aberrant conduction)
- Regular rhythm
- **P wave is typically not visible** (it falls in or is "buried" in the QRS complex)



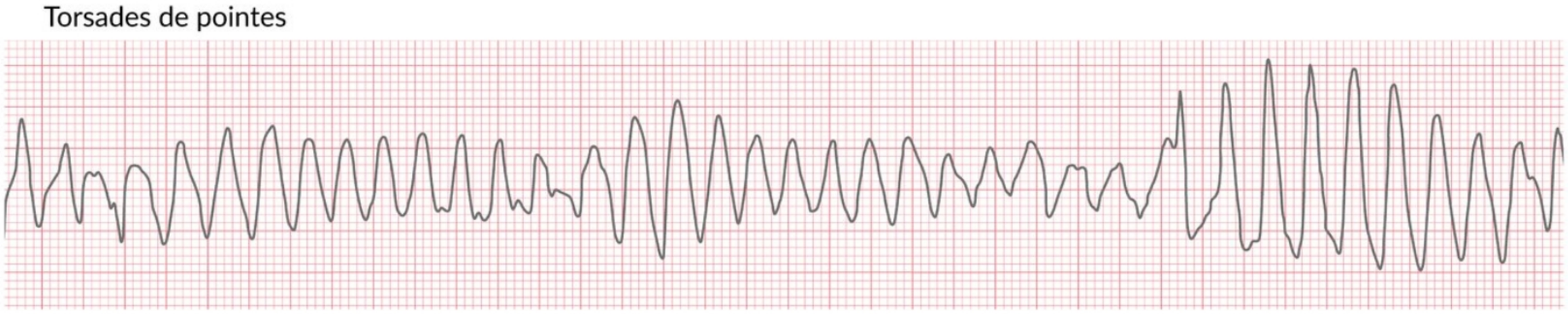
Monomorphic ventricular tachycardia

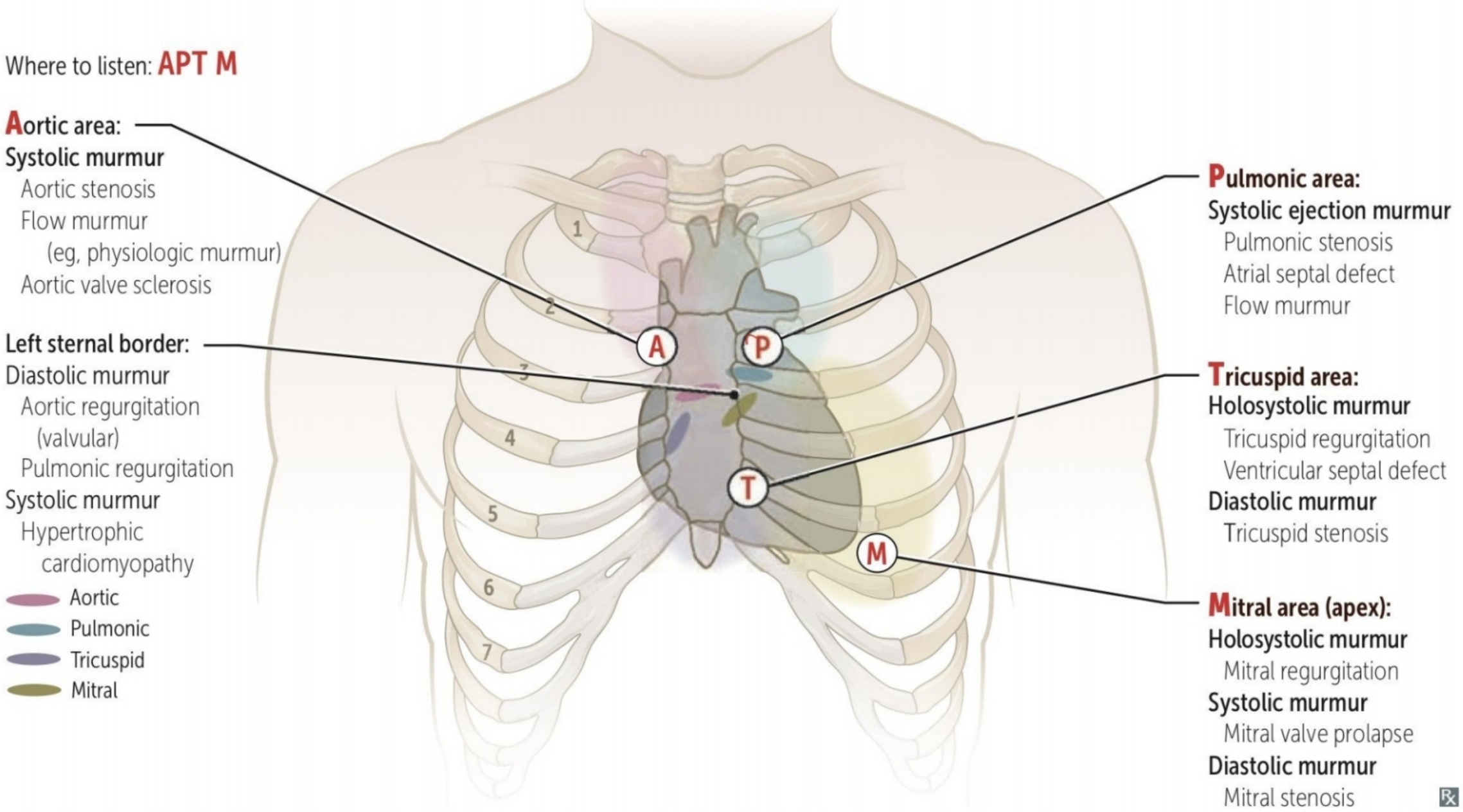


Polymorphic ventricular tachycardia



Torsades de pointes





◀ Characteristics of heart valves

Valve	Structure	Site of auscultation	Open in	Sound
Mitral valve	Bicuspid	Left 5th intercostal space at the midclavicular line (Apex)	Diastole	S1 (LUB)
Tricuspid valve	Tricuspid	Left 5th intercostal space at the sternal border		
Aortic valve	Semilunar	Right 2nd intercostal space at the sternal border	Systole	S2 (DUB)
Pulmonary valve	Semilunar	Left 2nd intercostal space at the sternal border		

◀ Heart sounds

i **16.8 Normal and abnormal heart sounds**

Sound	Timing	Characteristics	Mechanisms	Variable features
First heart sound (S1)	Onset of systole	Usually single or narrowly split	Closure of mitral and tricuspid valves	Loud: hyperdynamic circulation (anaemia, pregnancy, thyrotoxicosis); mitral stenosis Soft: heart failure; mitral regurgitation
Second heart sound (S2)	End of systole	Split on inspiration Single on expiration (p. 447)	Closure of aortic and pulmonary valve A_2 first P_2 second	Fixed wide splitting with atrial septal defect Wide but variable splitting with delayed right heart emptying (right bundle branch block) Reversed splitting due to delayed left heart emptying (left bundle branch block)
Third heart sound (S3)	Early in diastole, just after S2	Low pitch, often heard as 'gallop'	From ventricular wall due to abrupt cessation of rapid filling	Physiological: young people, pregnancy Pathological: heart failure, mitral regurgitation
Fourth heart sound (S4)	End of diastole, just before S1	Low pitch	Ventricular origin (stiff ventricle and augmented atrial contraction) related to atrial filling	Absent in atrial fibrillation A feature of severe left ventricular hypertrophy
Systolic clicks	Early or mid-systole	Brief, high-intensity sound	Valvular aortic stenosis Valvular pulmonary stenosis Floppy mitral valve Prosthetic heart sounds from opening and closing of normally functioning mechanical valves	Click may be lost when stenotic valve becomes thickened or calcified Prosthetic clicks lost when valve obstructed by thrombus or vegetations
Opening snap (OS)	Early in diastole	High pitch, brief duration	Opening of stenosed leaflets of mitral valve Prosthetic heart sounds	Moves closer to S2 as mitral stenosis becomes more severe. May be absent in calcific mitral stenosis

Acute Rheumatic Fever

◄ Diagnostic criteria

- **A firm diagnosis requires:** 2 Major manifestations **OR** 1 Major and 2 Minor manifestations along with Evidence of a recent streptococcal infection (Elevated ASO titer or Anti-Dnase B titer and a positive throat culture)

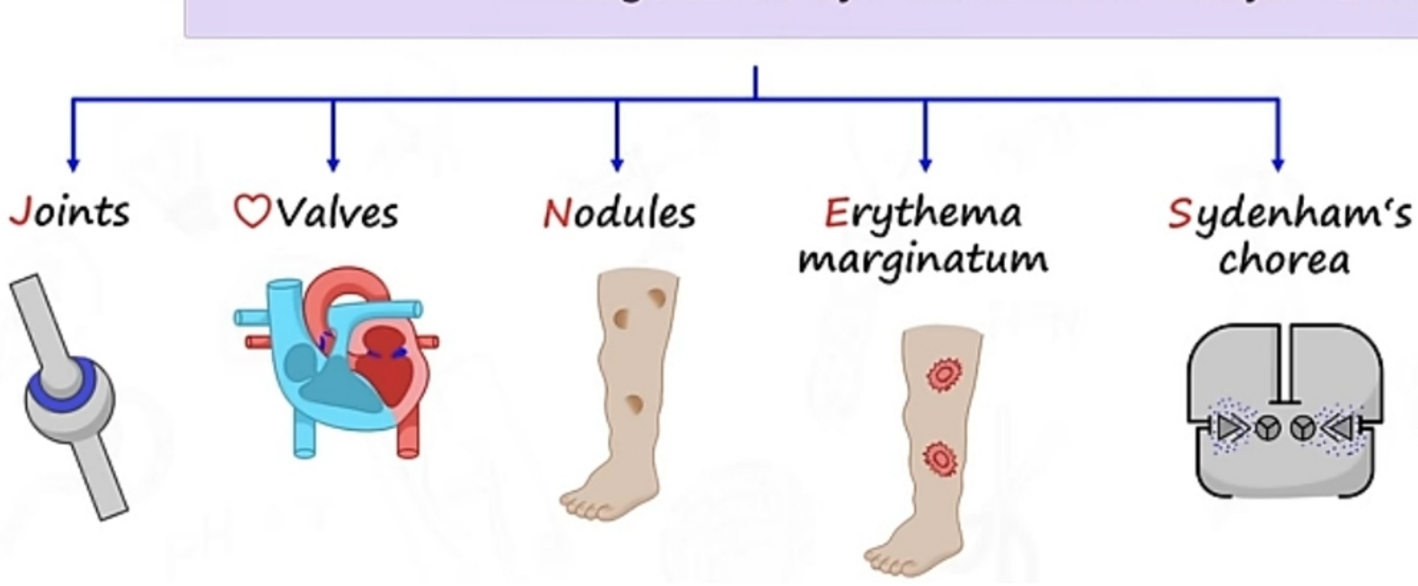
◆ 1992 Modified Jones criteria

J♡NES

Major criteria[1]	Minor criteria
1) **Migratory polyarthritis (Joints)** 2) ♡ **Carditis** 3) **Subcutaneous Nodules.** 4) **Erythema Marginatum** 5) **Sydenham chorea**	1) **Arthralgia** 2) **First degree heart block** (Prolonged PR interval) 3) **Fever** 4) **Elevated inflammatory markers (ESR, CRP)**

Diagnostic Approach to
Rheumatic Heart Disease

Major Criteria	Minor Criteria	Lab evidence of GAS infection
Joint pain ❤️ → **Cardiac valve damage** (mitral > aortic > tricuspid) **Nodules** **Erhythema Marginatum** **Sydenham's Chorea**	▪ Fever ▪ ↑ESR ▪ ↑PR-I ▪ Polyarthritis	**(+) strep test** **(+) Anti-ASO antibodies**
Diagnosis: **2 Major** criteria met or **1 major + 2 minor** criteria met		



Diagnostic criteria of IE

◄ Duke criteria

BE_FEVER I

★Major criteria

1) **Positive blood culture:**
   a) **Typical organism from two separate cultures e.g. Strep, Staph, HACEK**
   b) **Persistent positive blood cultures**
      i) ≥ 2 positive blood cultures of blood samples drawn >12 hrs apart
      ii) All of 3 or a majority of ≥ 4 separate cultures of blood (with 1st and last samples drawn ≥ 1h apart)
   c) **Single positive culture for coxiella burnetii or phase I IgG antibody titre > 1:800**
2) **Endocardial involvement:** (Check definitions next page)
   a) **Positive echocardiographic findings of vegetations,** Abscess , pseudoaneurysm ,intracardiac fistula , valvular perforation, aneurysm or **New partial dehiscence of prosthetic valve**
   b) **Abnormal activity around the site of prosthetic valve implantation** detected by F-FDG PET/CT (only if the prosthesis was implanted for >3 months) or radiolabeled leukocytes SPECT/CT.
   c) **Definite paravalvular lesion by cardiac CT**
   d) **New valvular regurgitation**

Minor criteria

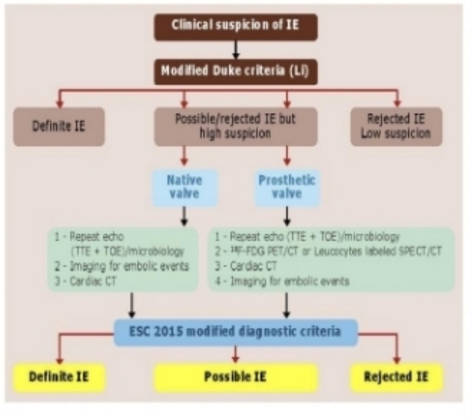
1) **Fever**: > 38°C
2) **Echo findings**: Any finding not involved in the major criteria e.g. calcification
3) **Vascular phenomena (Including these detected only by imaging):**
   a) Major arterial emboli , septic pulmonary infarcts , infectious (mycotic) aneurysm , intracranial hemorrhage , conjunctival hemorrhage and janeway's lesions.
4) **Evidence from microbiology:**
   a) Positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE.
5) **Risk factors and predisposition:**
   a) Such as heart conditions (e.g. VHD, prosthetic valve, previous IE) or IV drug users
6) **Immunological phenomena:**
   a) Glomerulonephritis , osler's nodes , Roth's spots and Rheumatoid factor

◄ Duke criteria

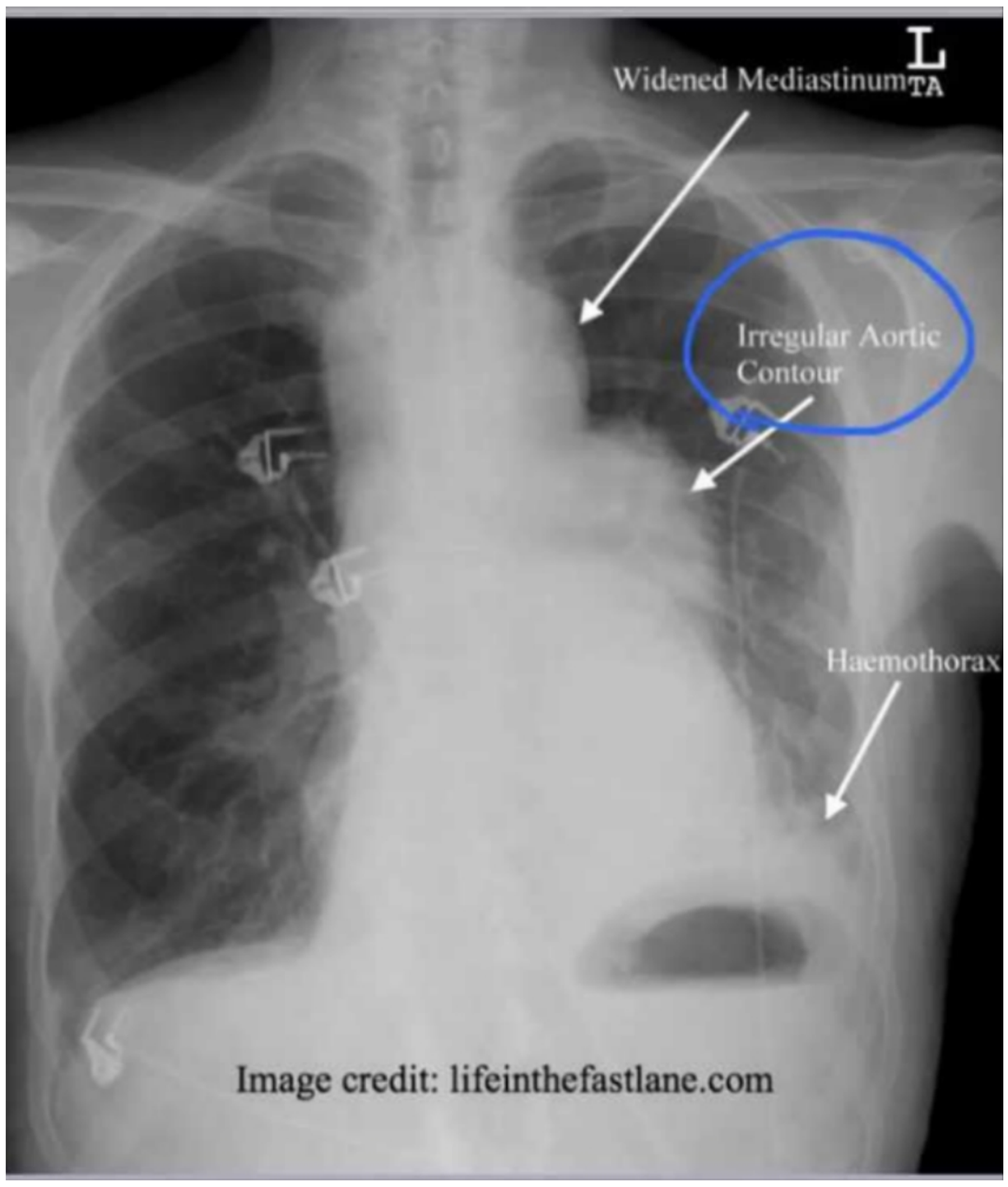
★ **Definitive IE:** (Begin treatment right away)
- **Clinical criteria:** Patients with 2 major, **OR** 1 major and 3 minor, **OR** 5 minor.
- **Pathologic criteria:** Microorganisms or pathologic lesions: demonstrated by culture or histology in a vegetation, or in a vegetation that has embolized, or in an intracardiac abscess

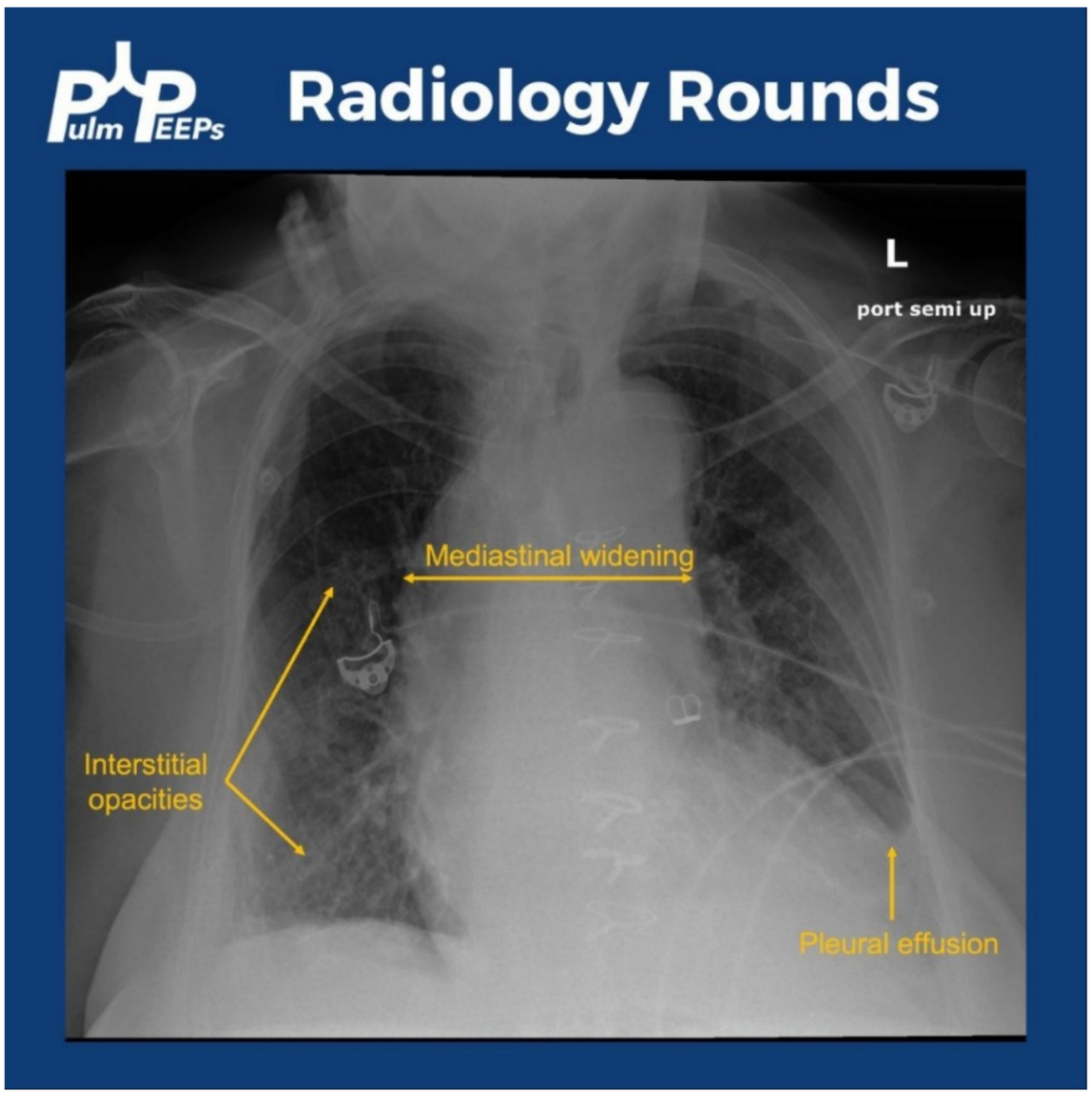
● **Possible IE:** (Requires further tests)
- **Clinical criteria:** Patients with 1 major and 1 minor, **OR** 3 minor.

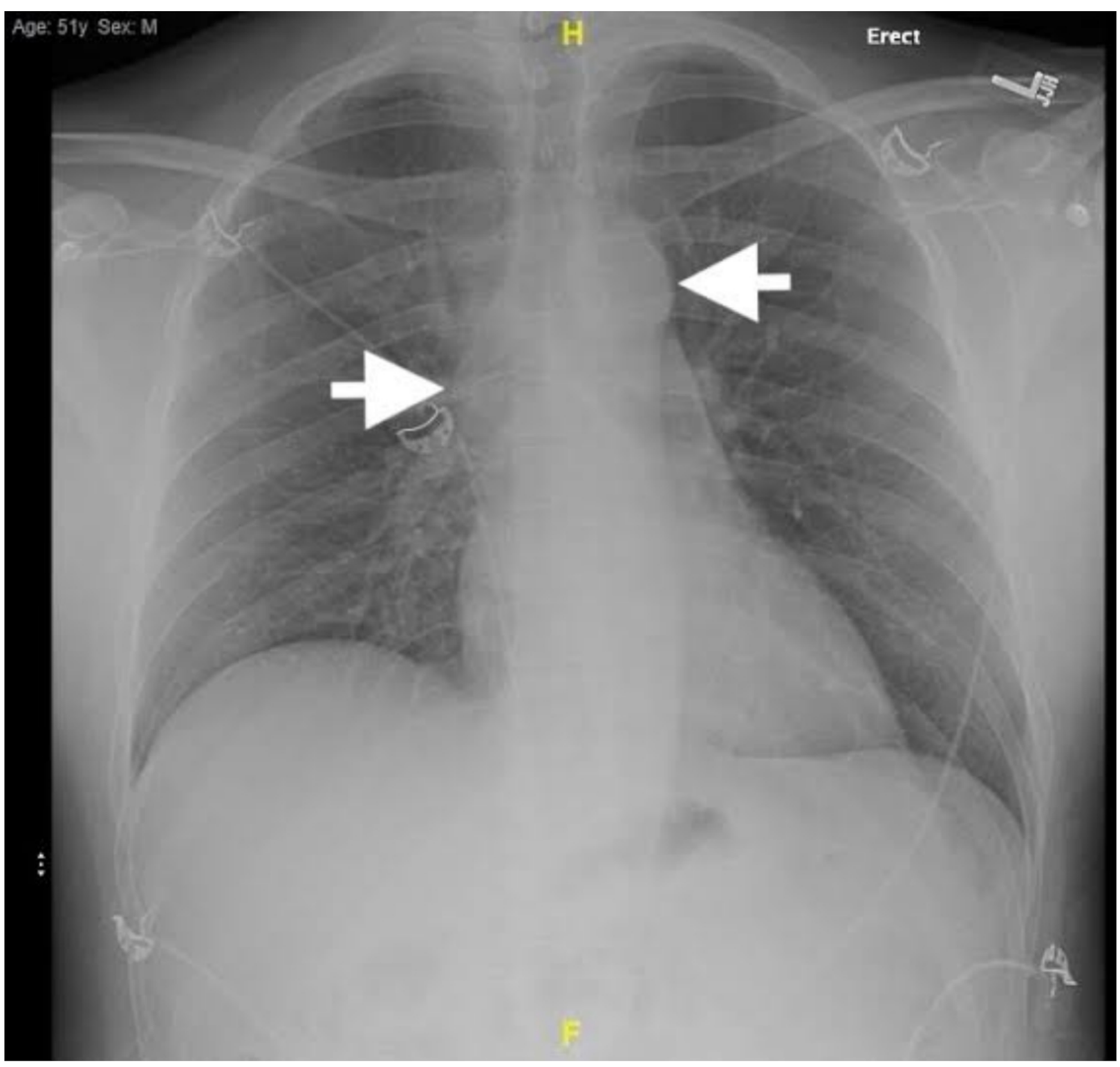
● **Rejected IE:**
- Firm alternate Diagnosis for manifestation of IE
- Resolution of manifestations of IE, with antibiotic therapy for ≤ 4 days
- No pathologic evidence of IE at surgery or autopsy, after antibiotic therapy for ≤ 4 days)



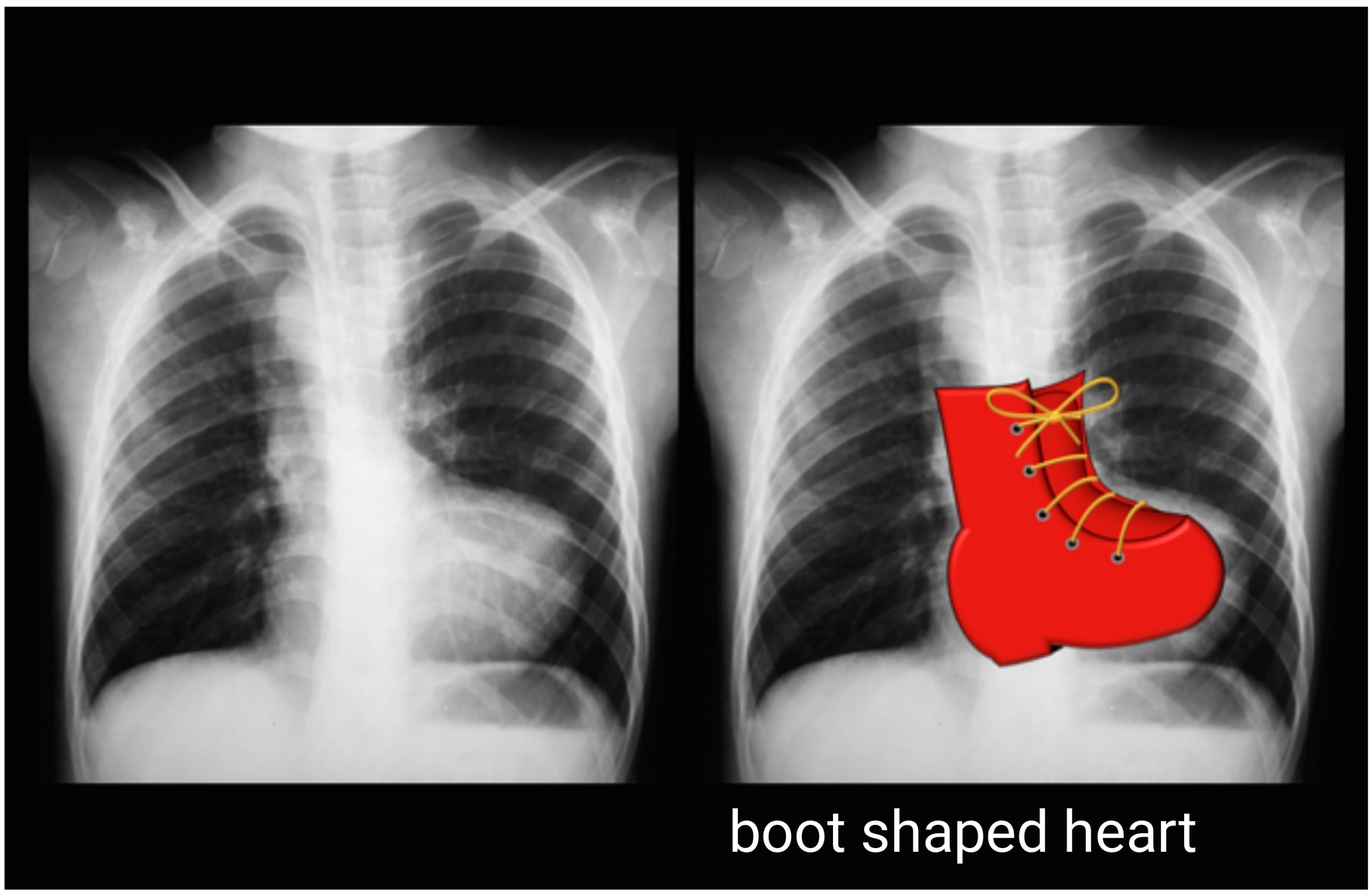
Widened mediastinum in Aortic dissection on chest X Ray

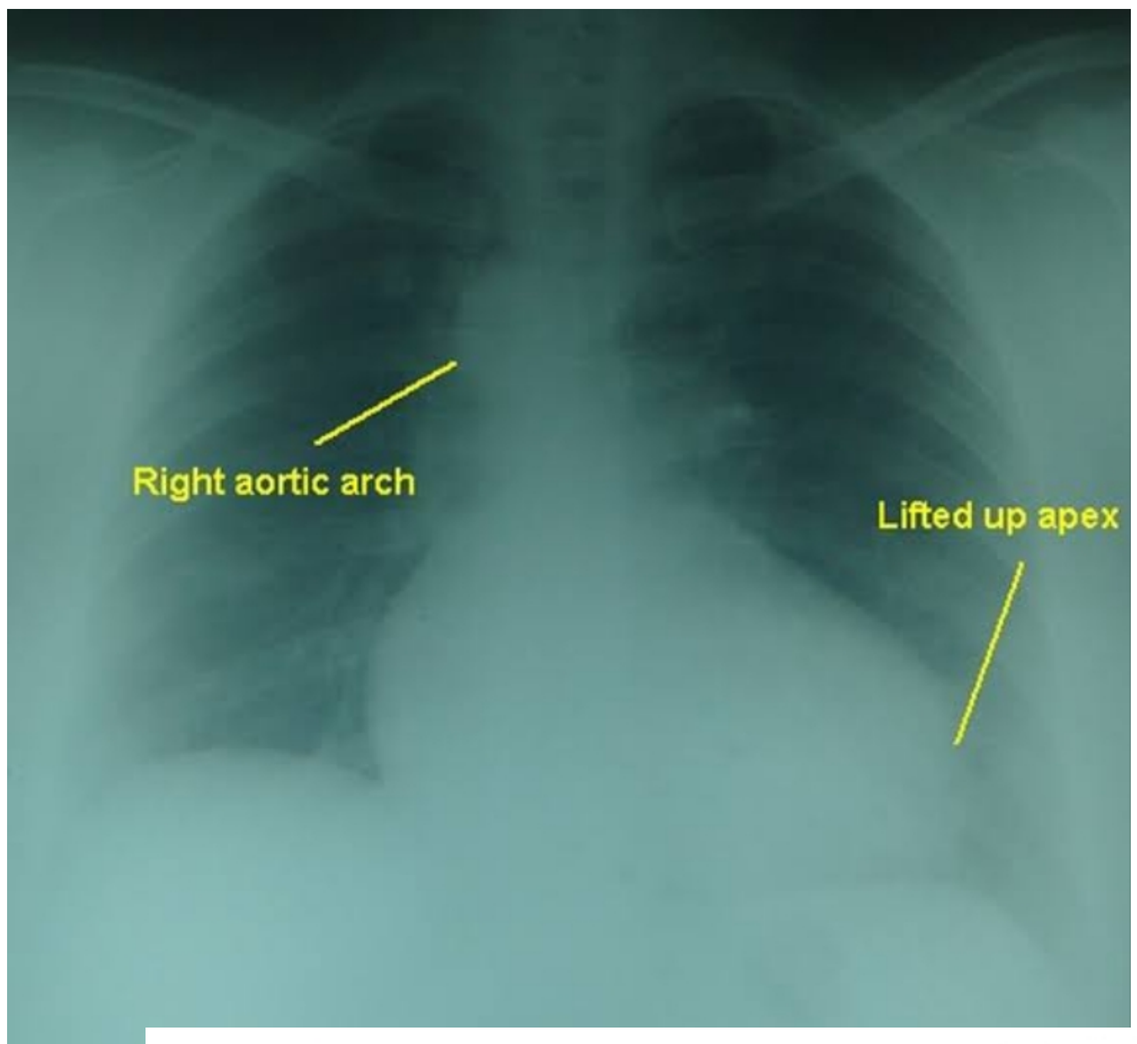


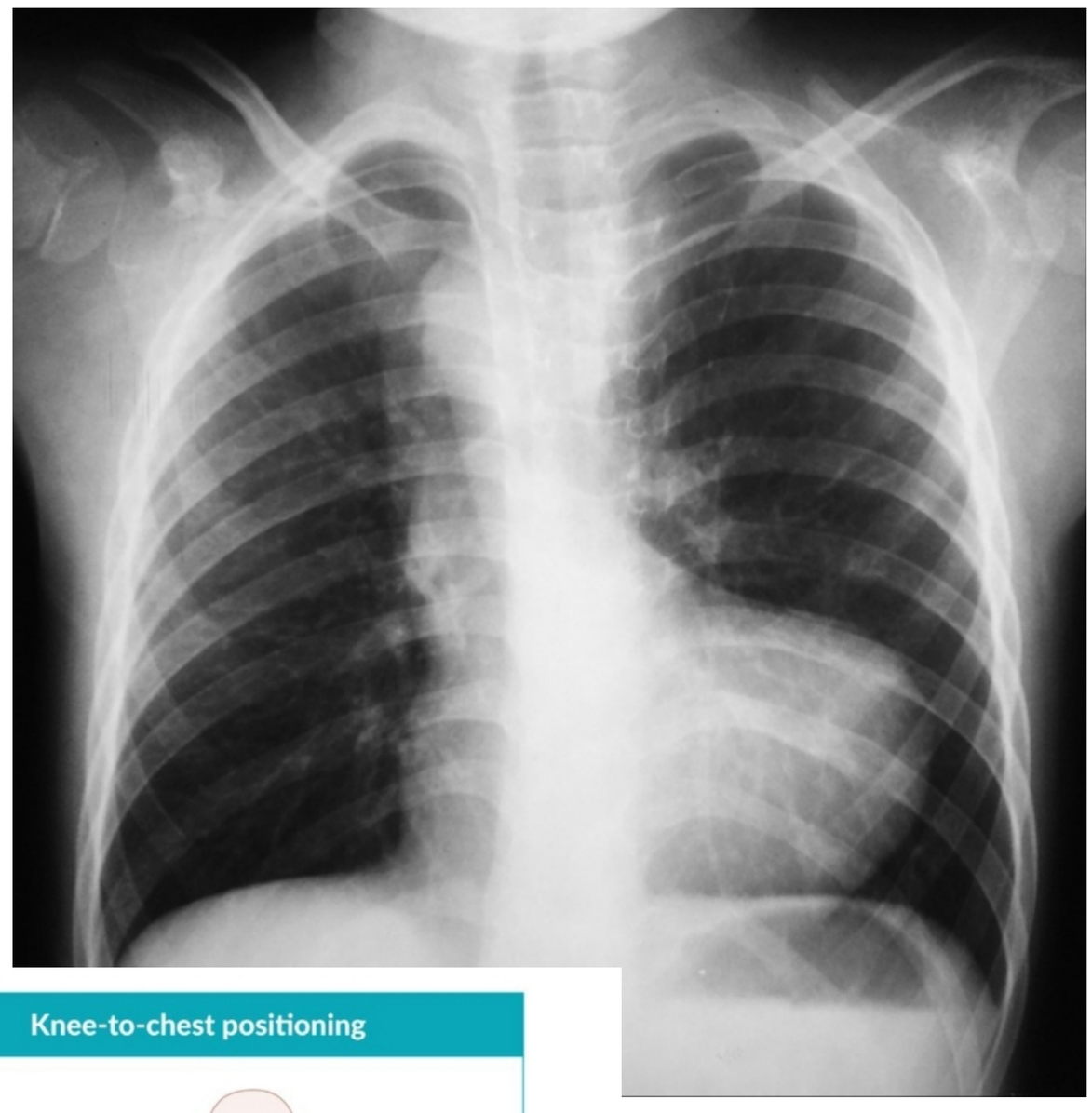


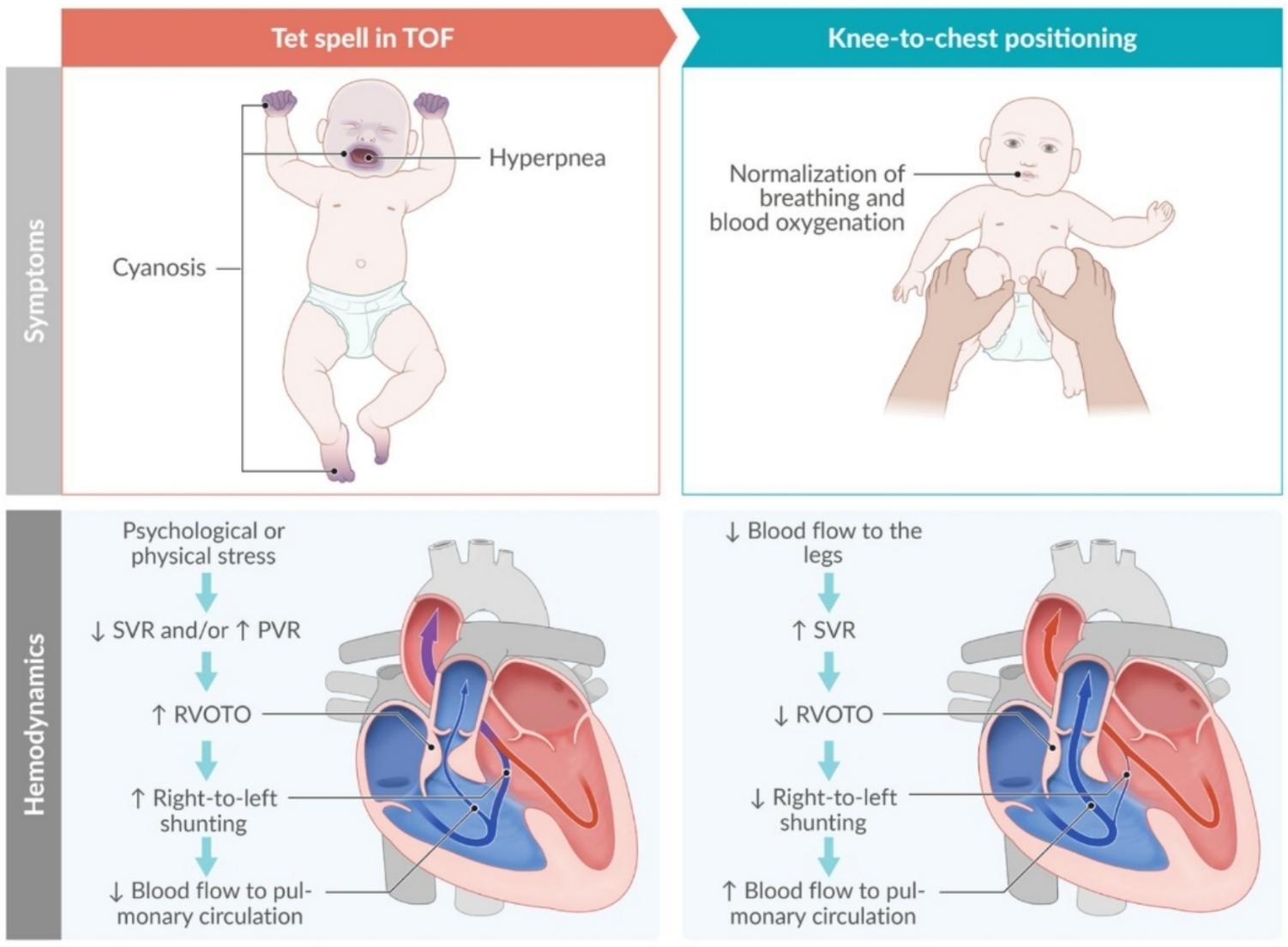


Tetralogy of fallot

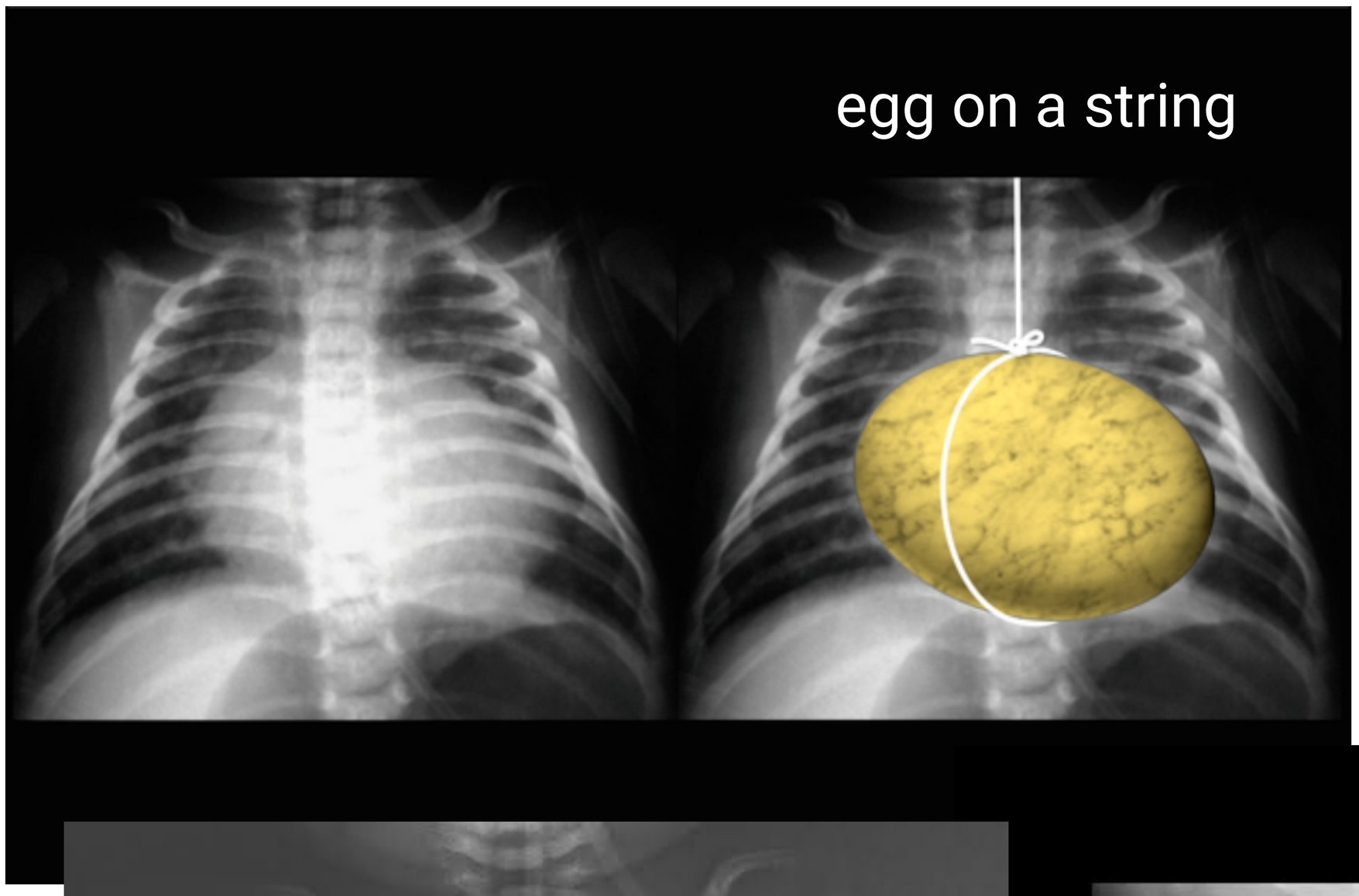


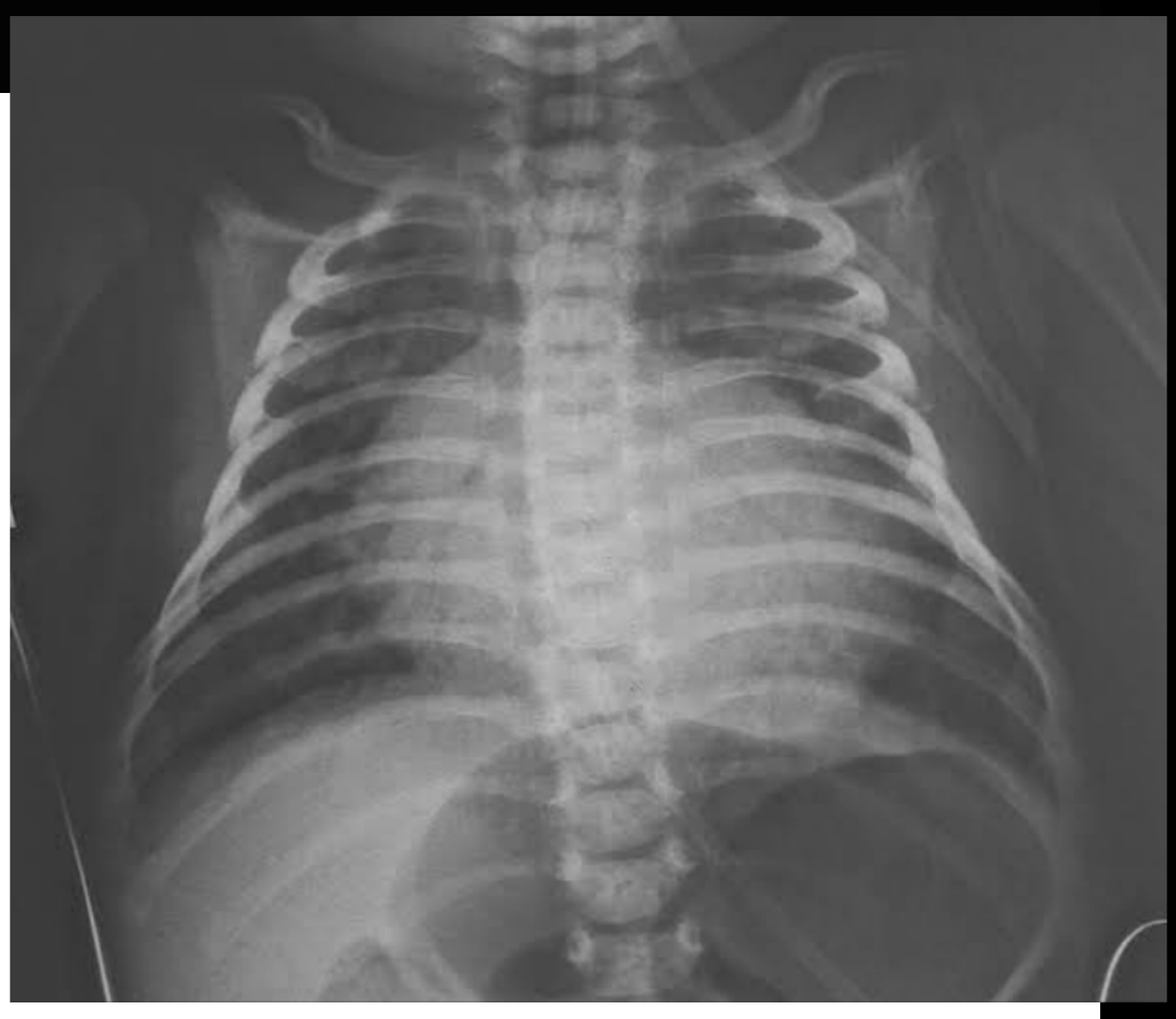


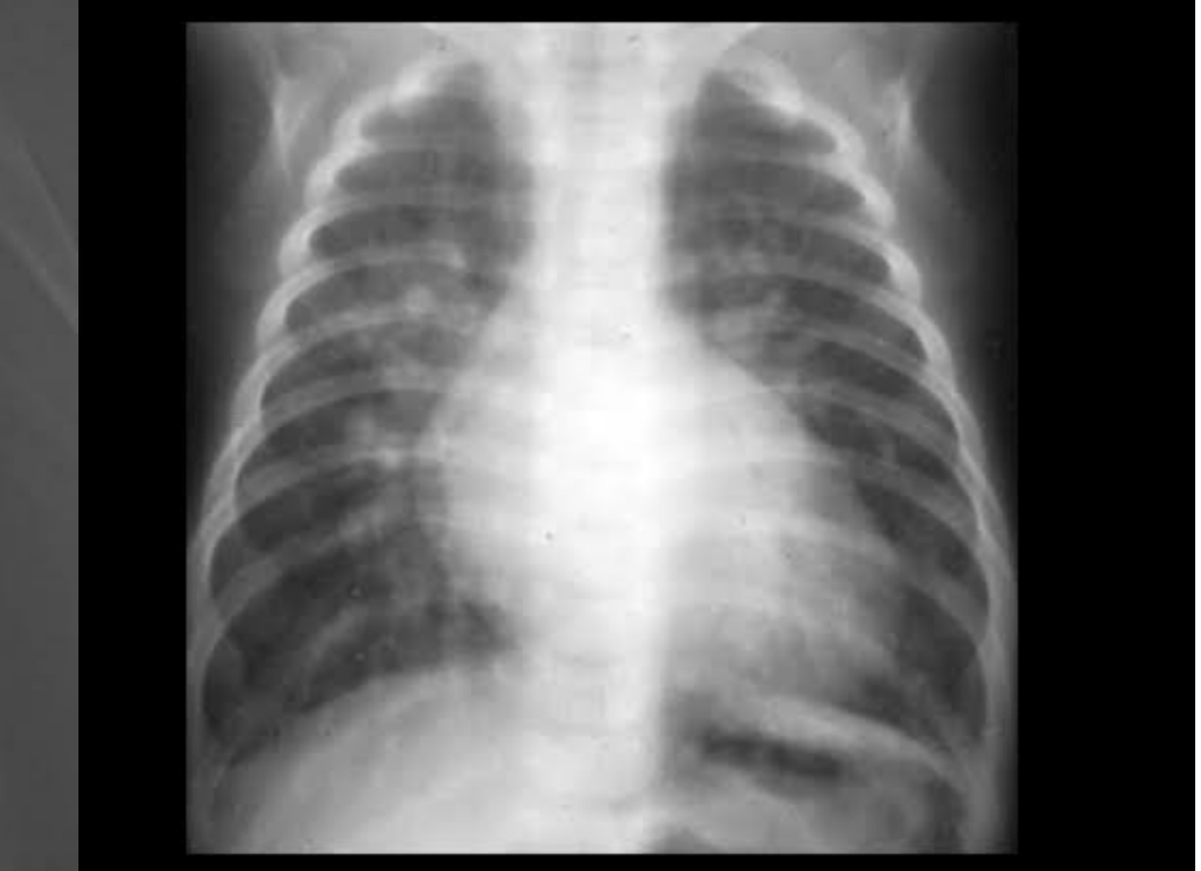


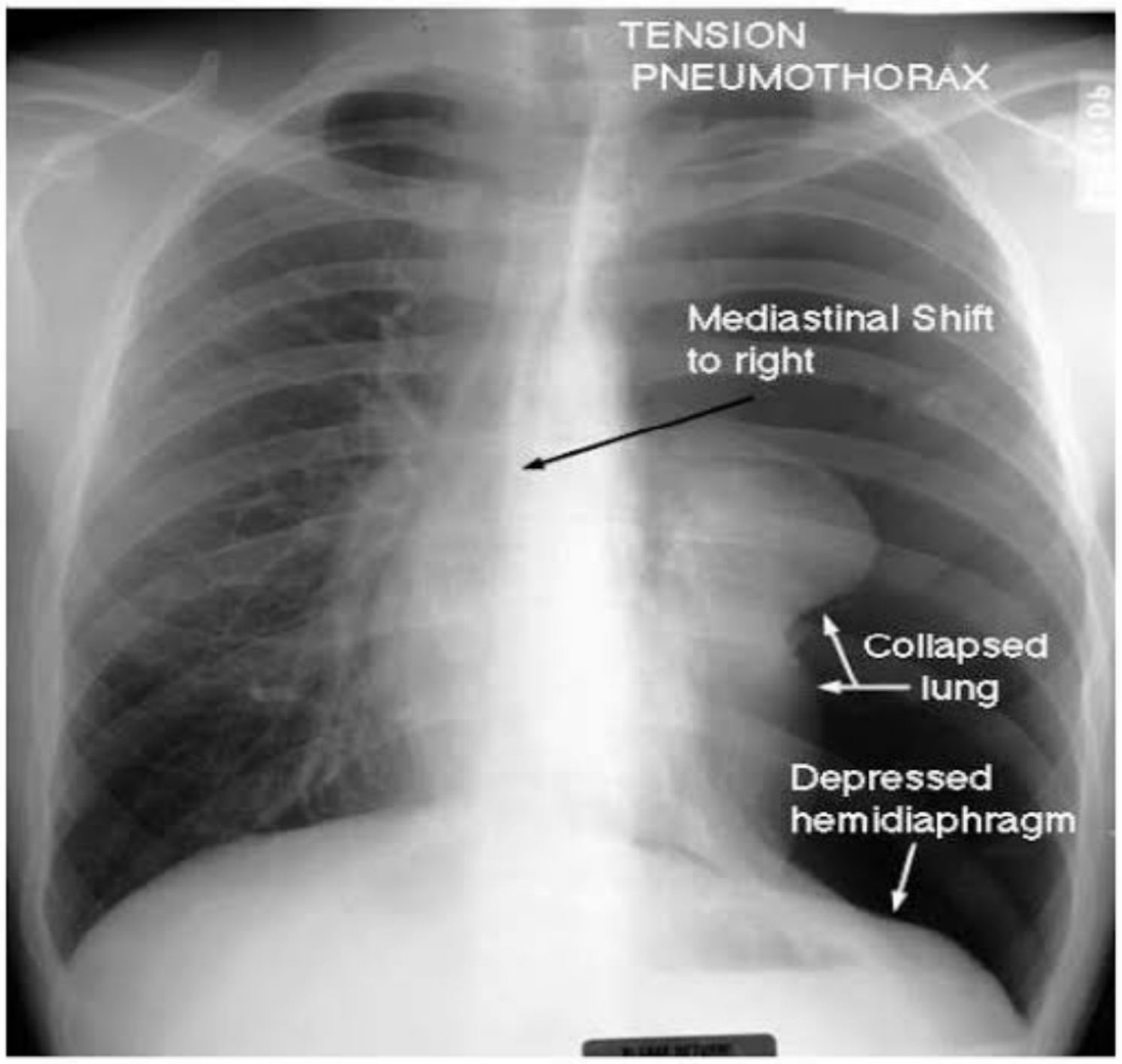


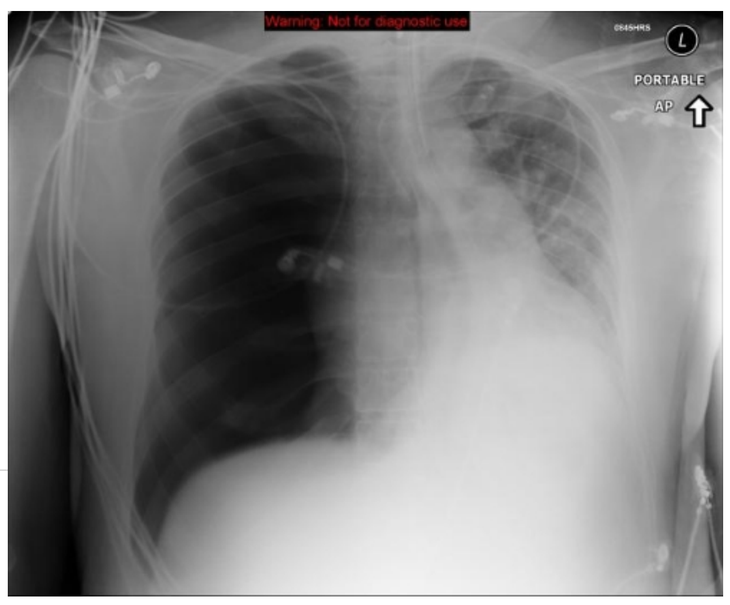
Transposition of great arteries

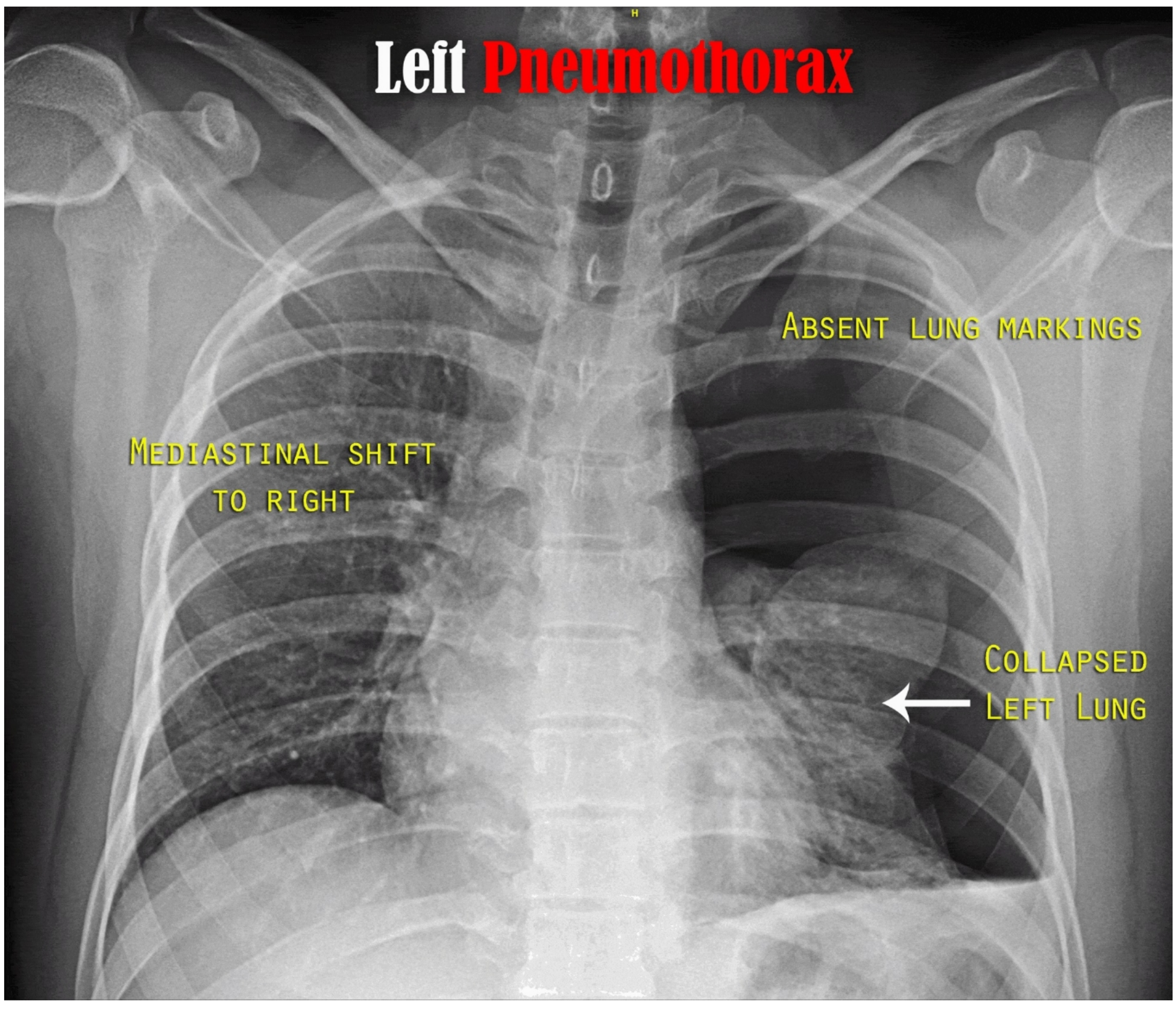




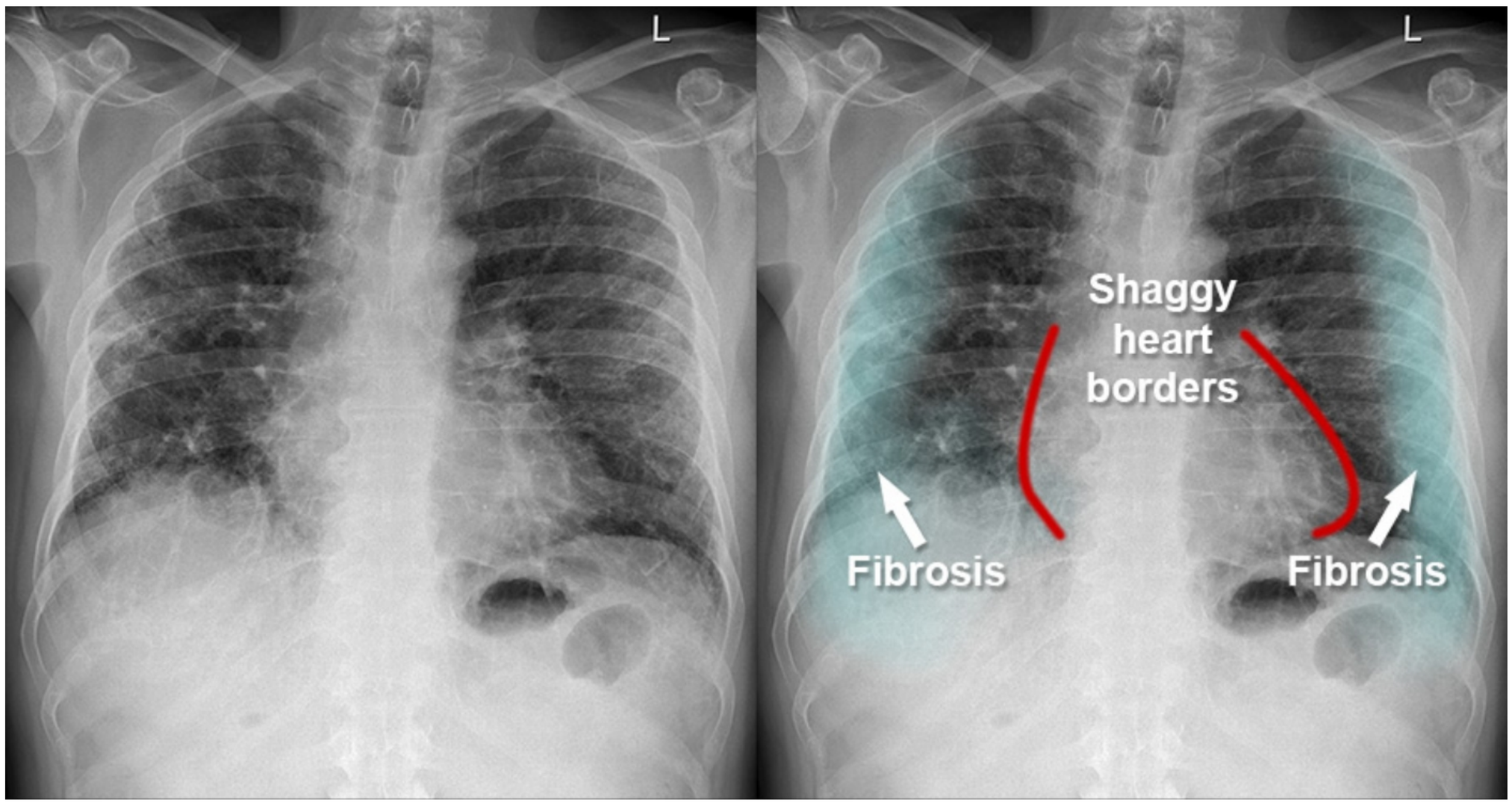


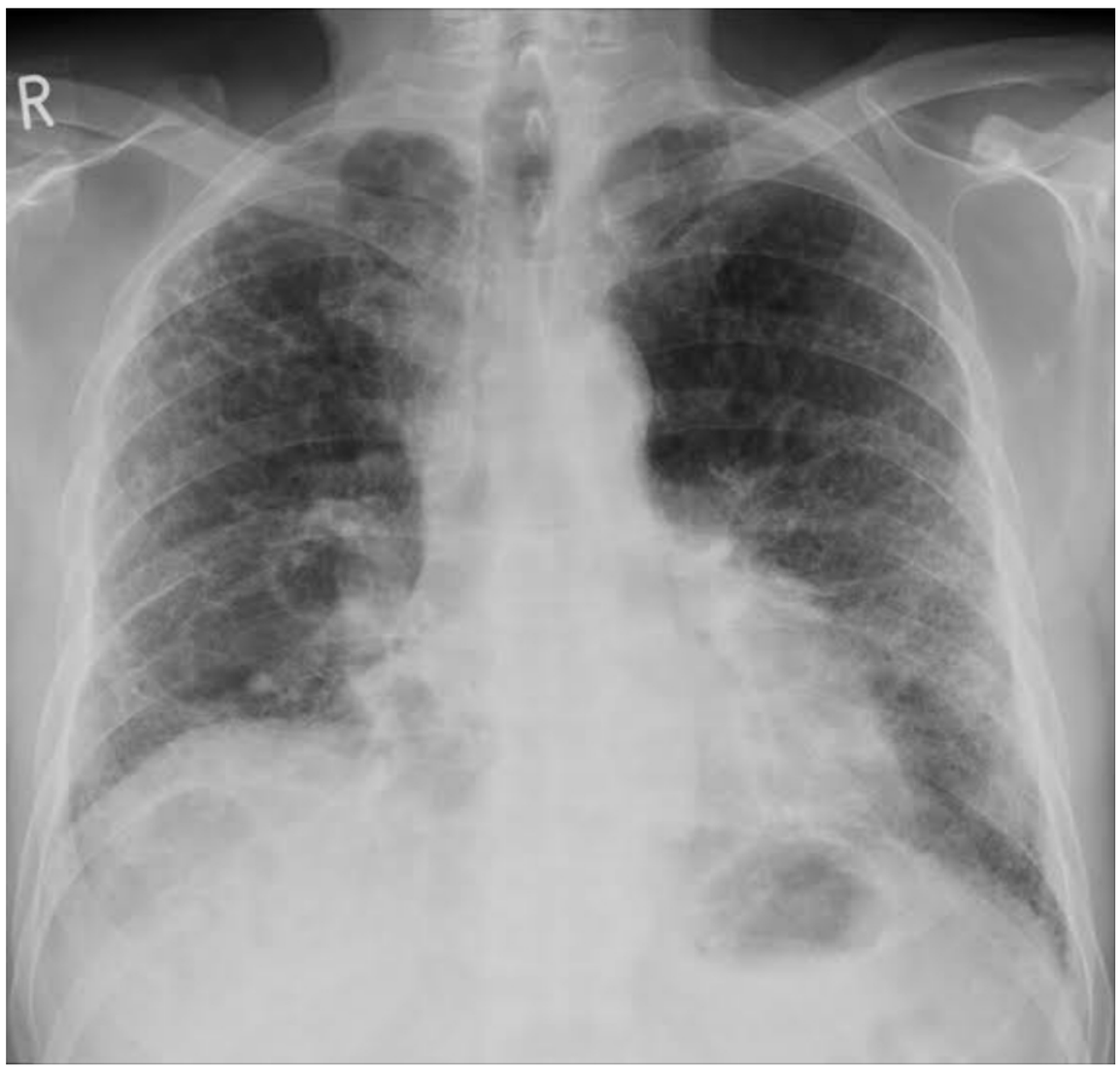

TENSION
PNEUMOTHORAX
Mediastinal Shift
to right
Collapsed
lung
Depressed
hemidiaphragm

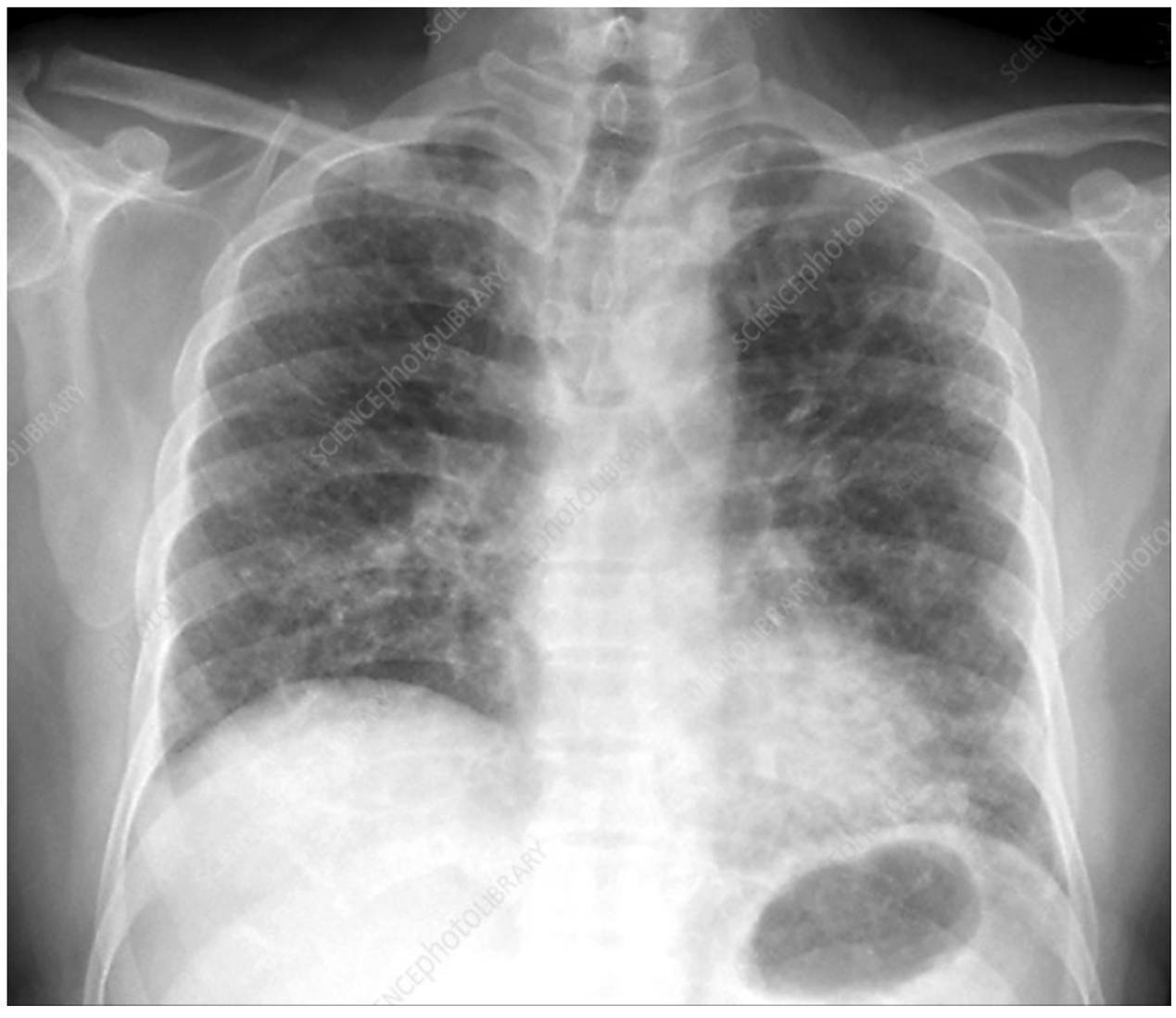

Warning: Not for diagnostic use
PORTABLE
AP


Left Pneumothorax
Absent lung markings
Mediastinal shift
to right
Collapsed
Left Lung

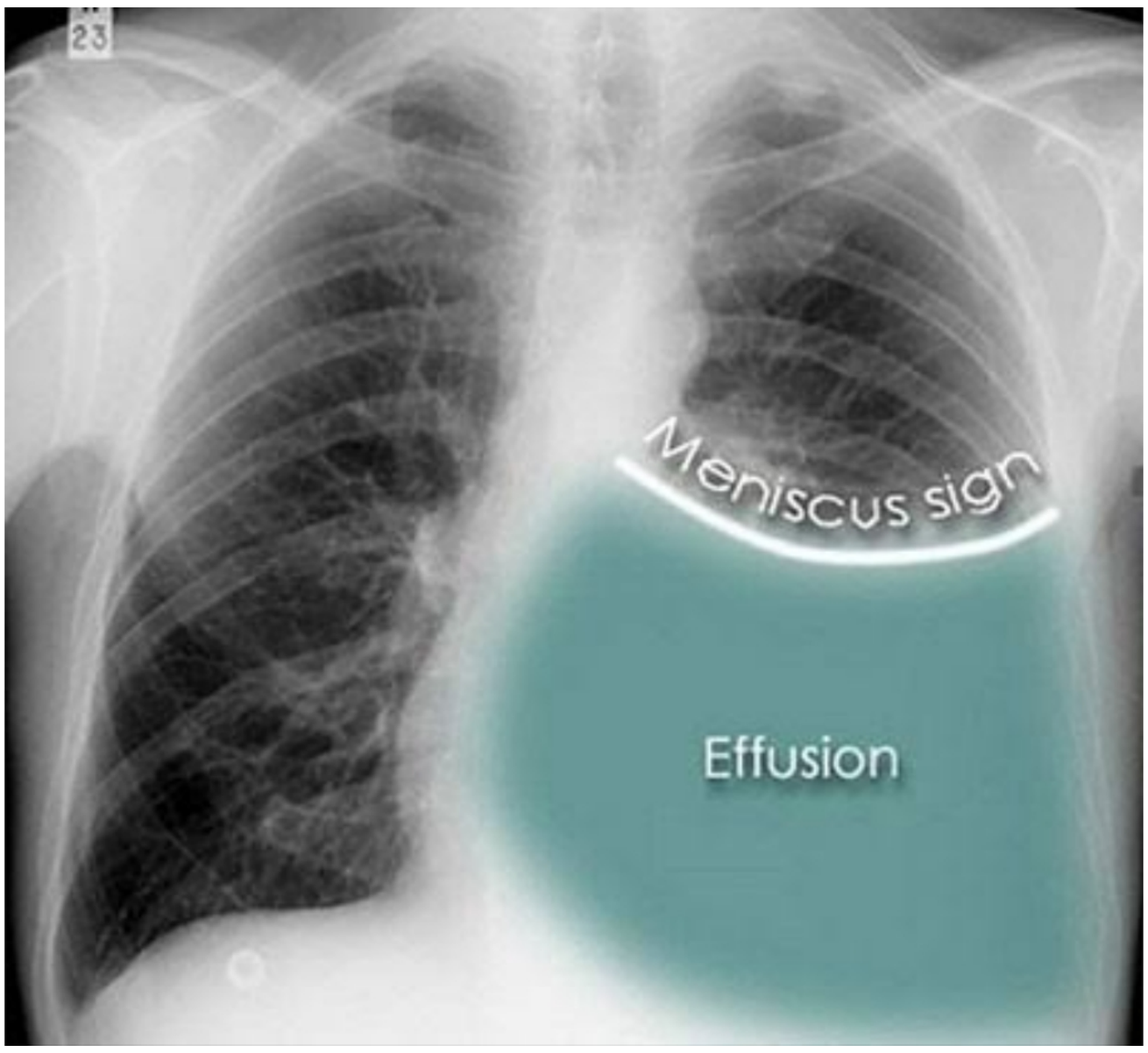
Pulmonary Fibrosis

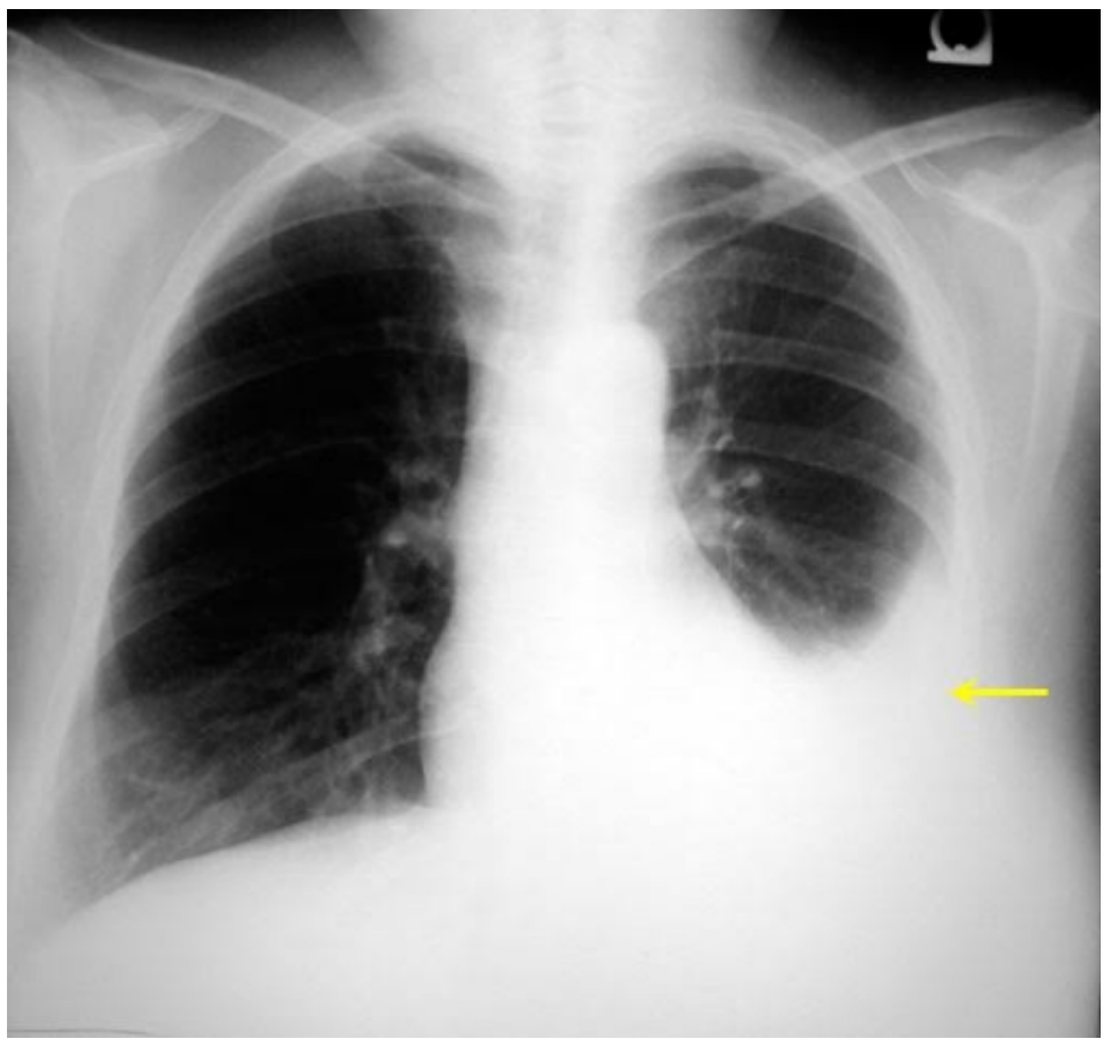


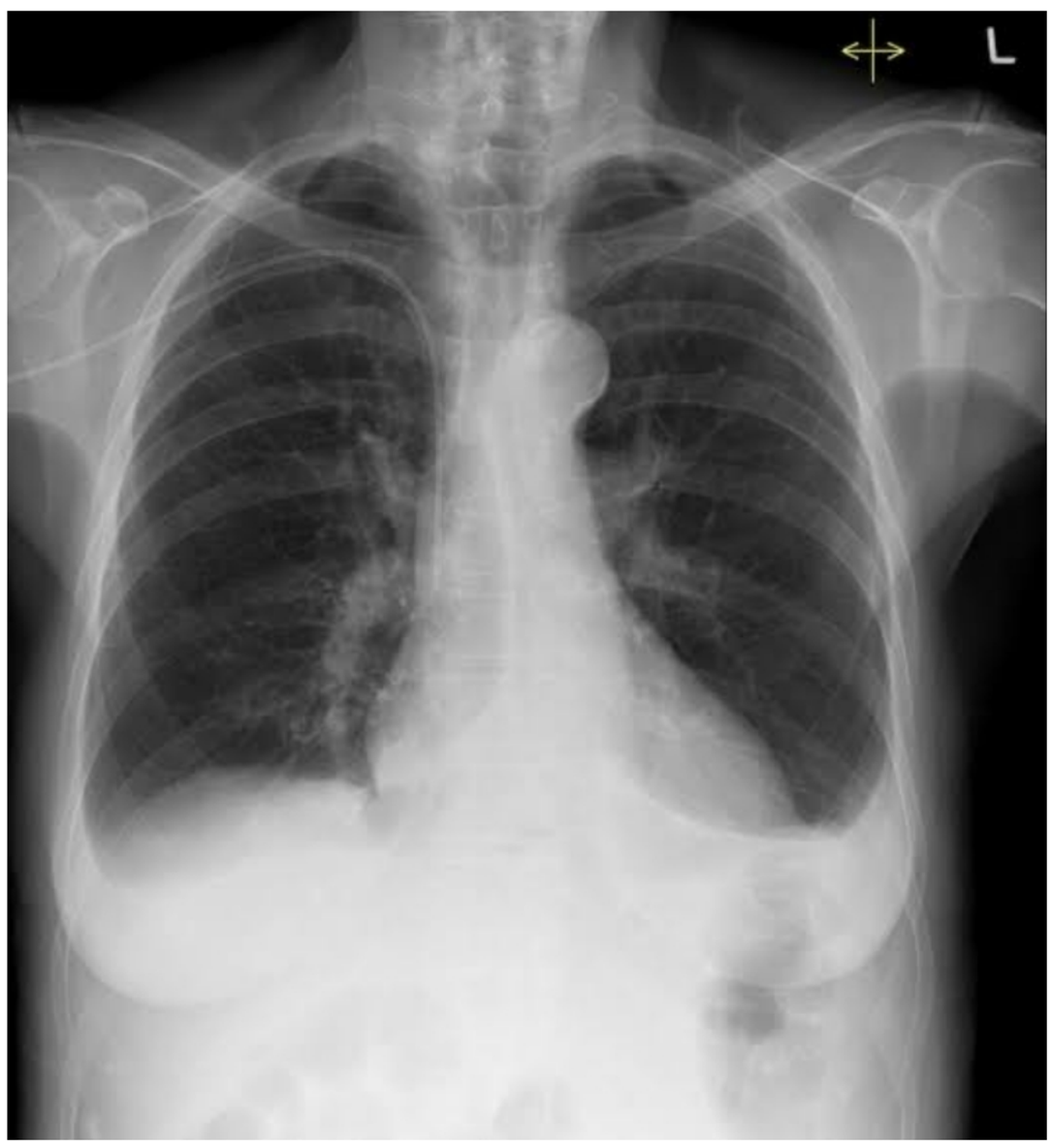


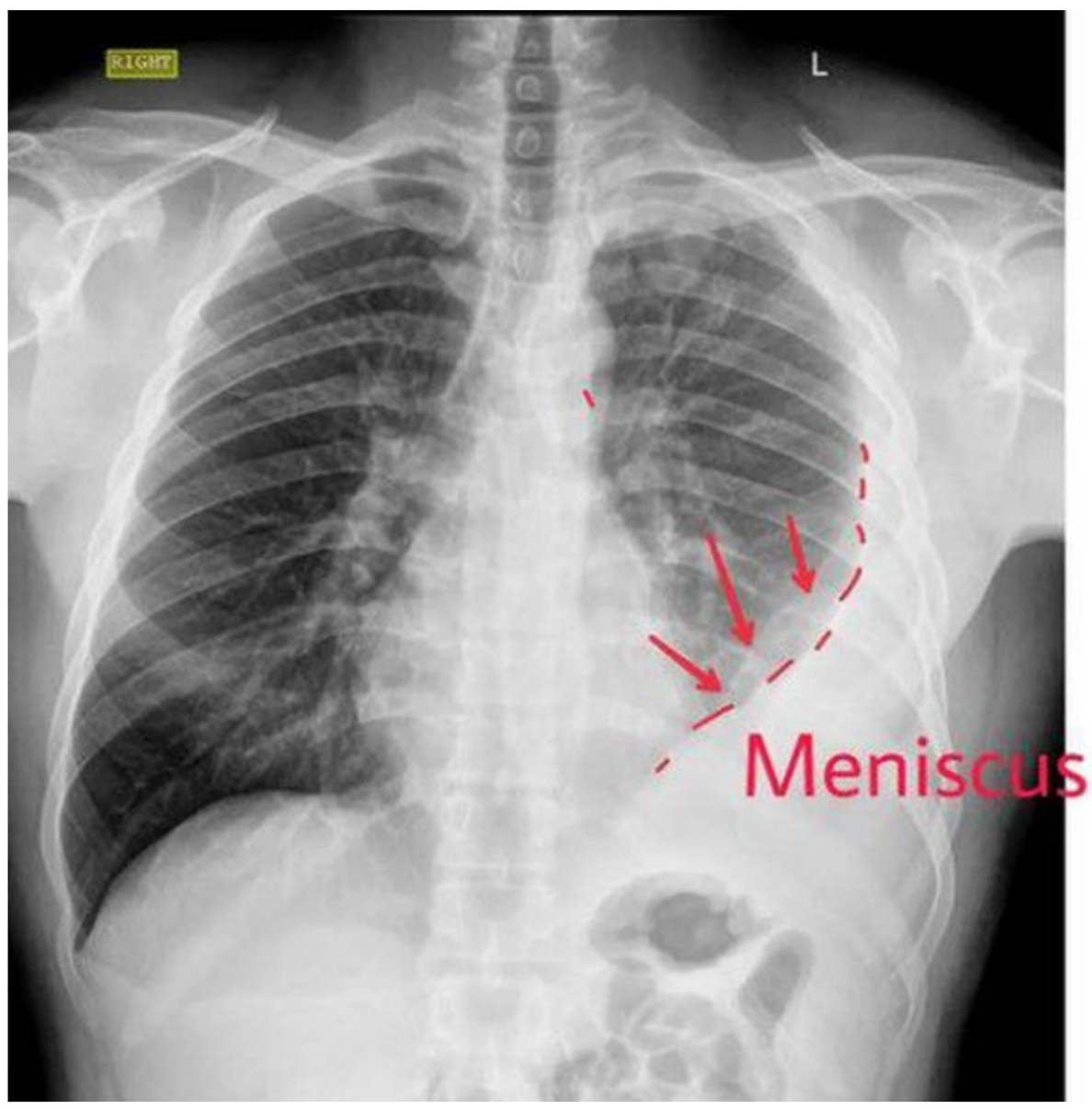


Meniscus sign in Pleural effusion





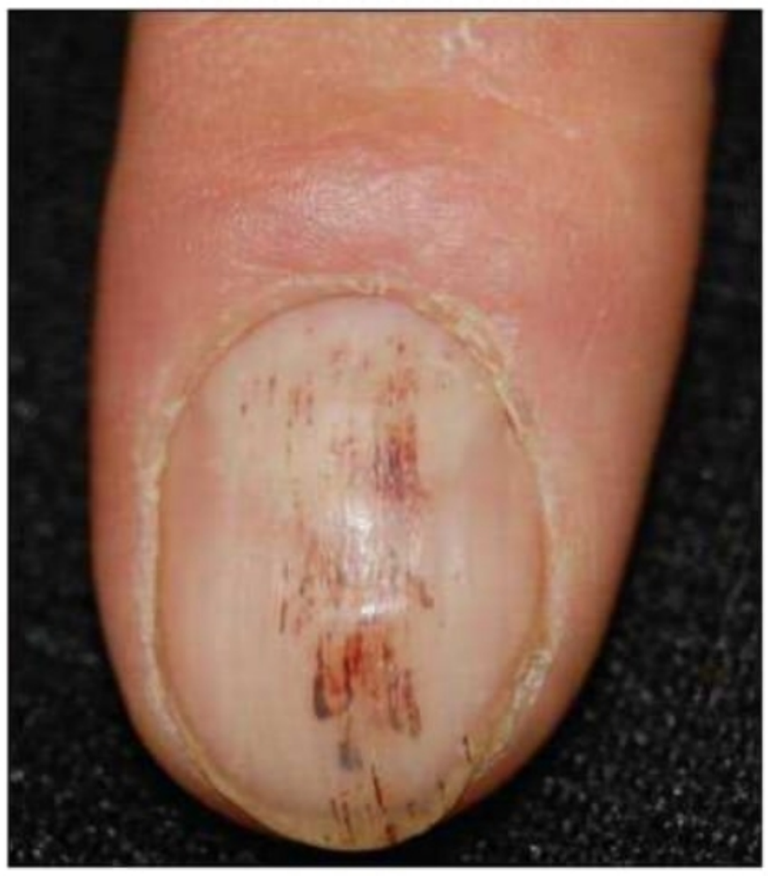




Leuconychia

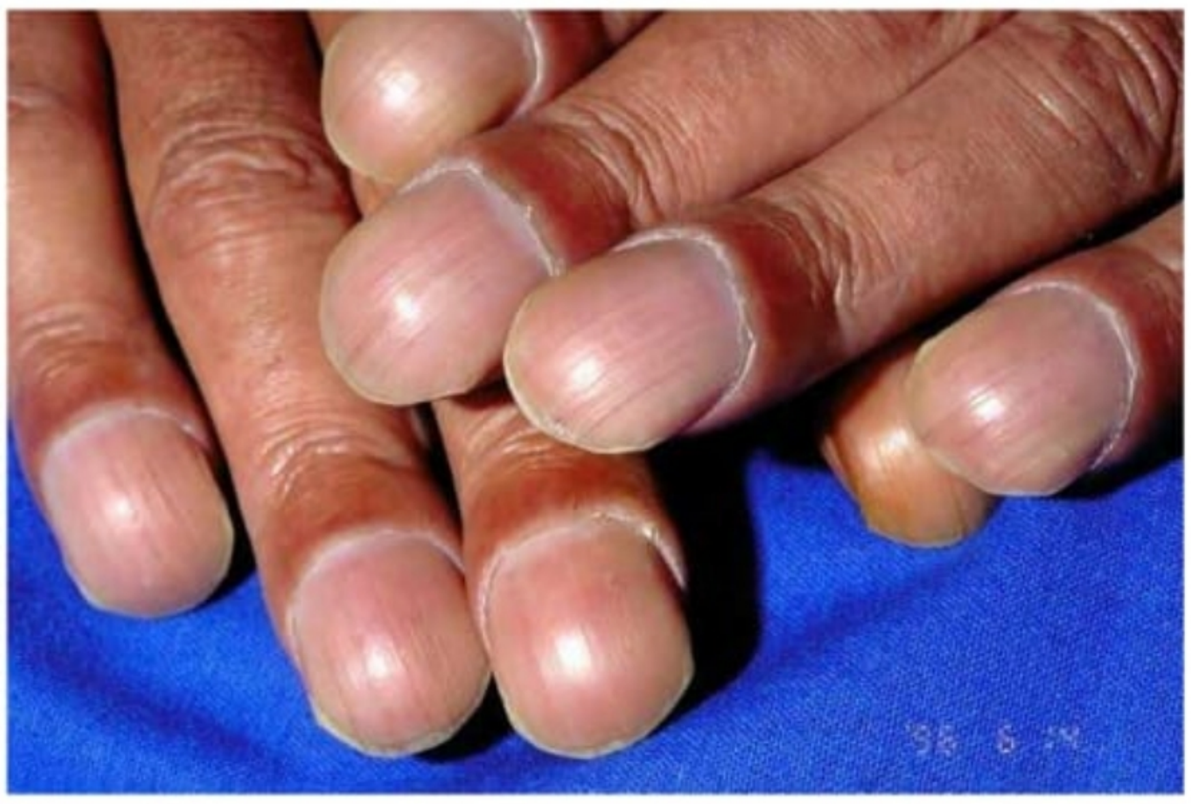
pallor and opacification of nail bed.

- Chronic Liver Disease
- Hypo albuminemia



Splinter Hemorrhages

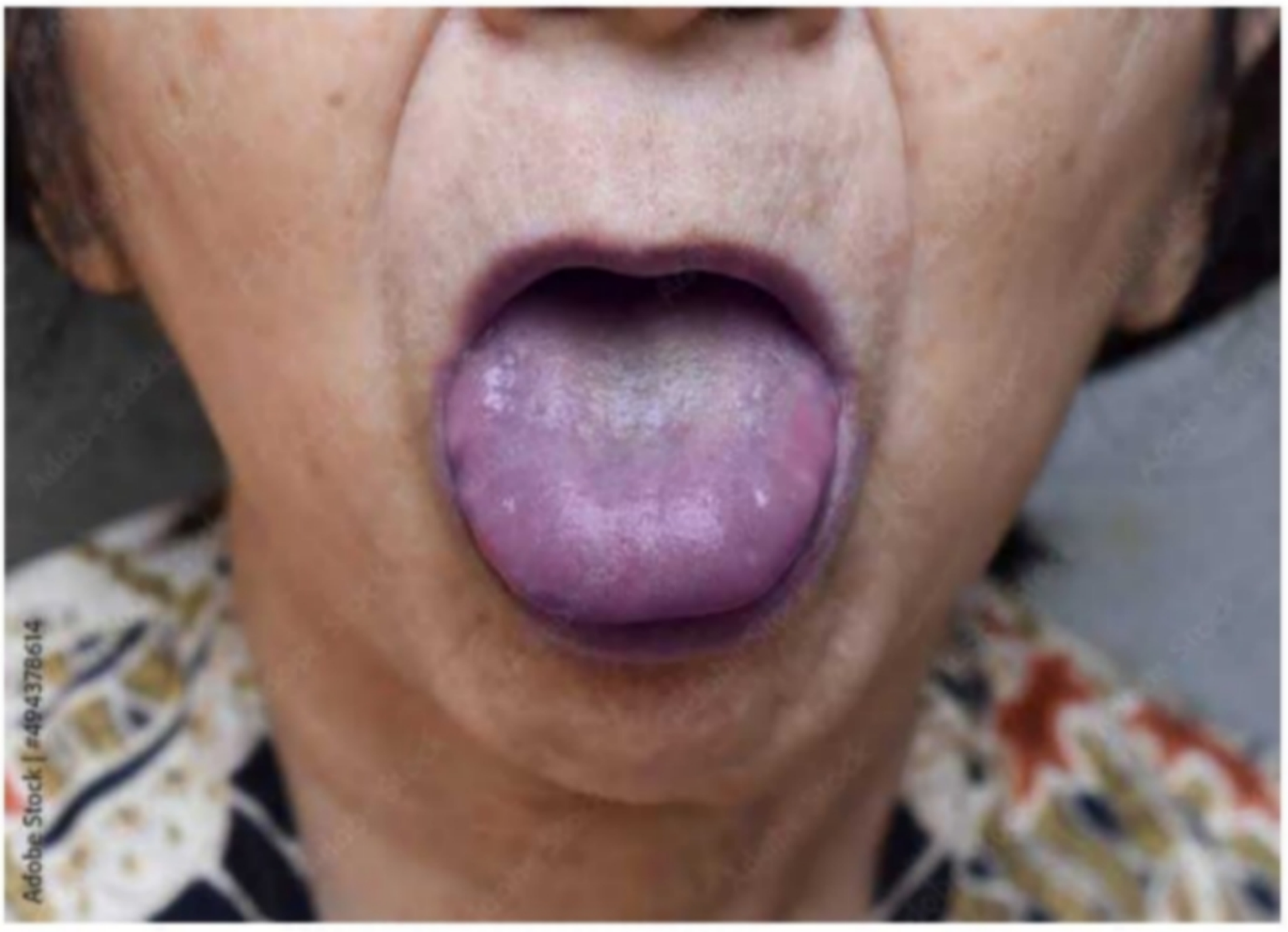
- Vasculitis !
- EndoCarditis



Clubbing

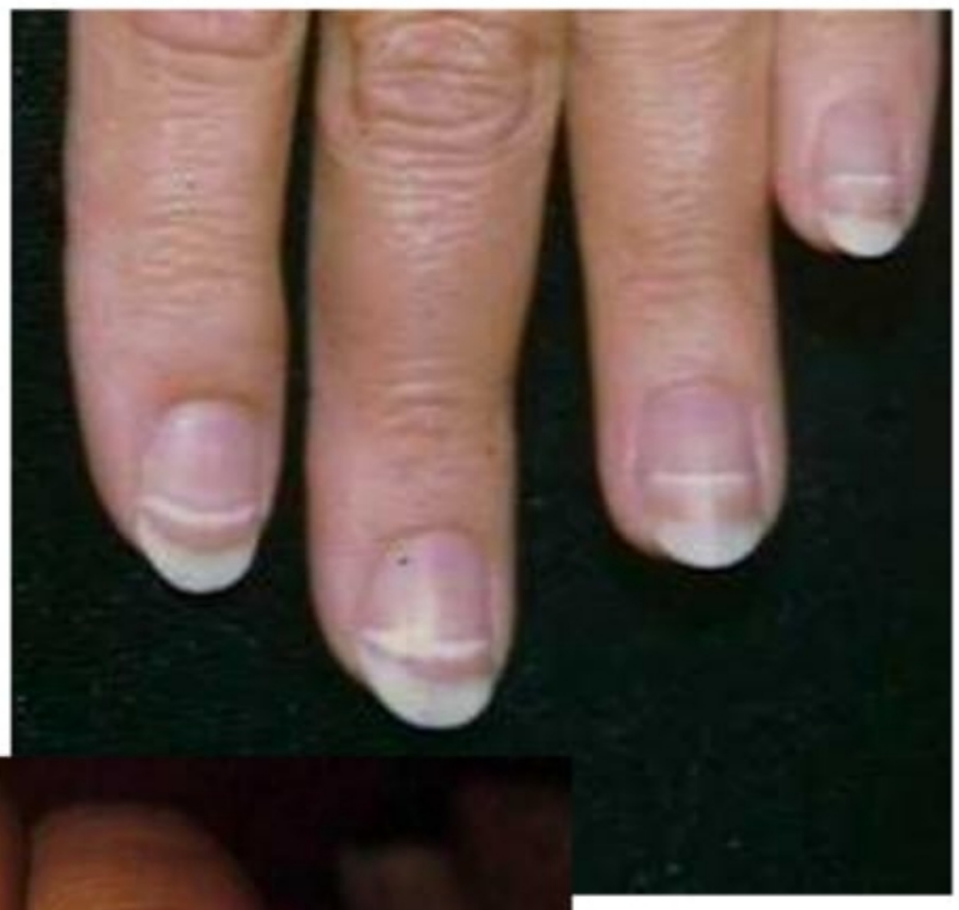
Table 7.5 Causes of clubbing

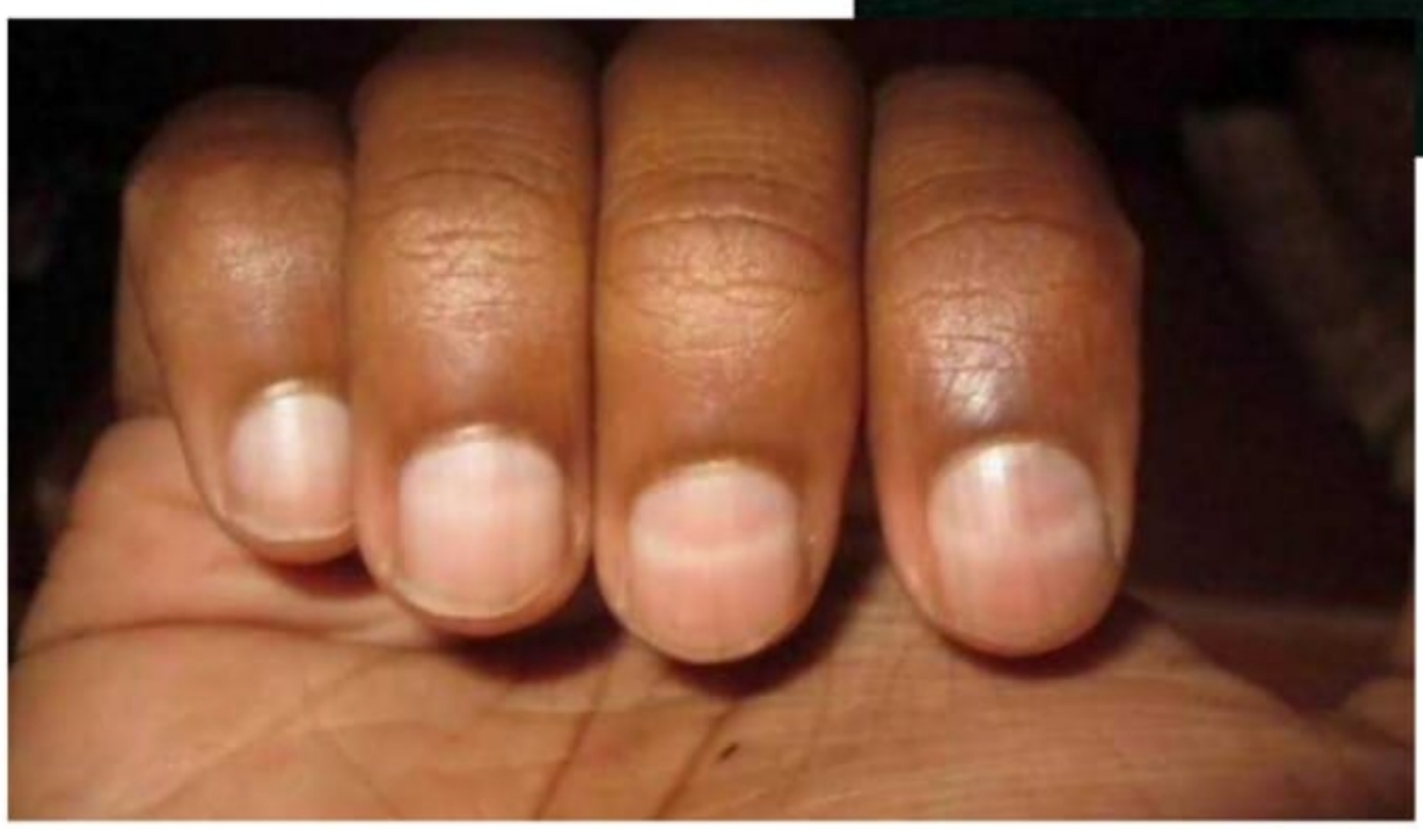
Cardiovascular	Cyanotic congenital heart disease Infective endocarditis Atrial myxoma Axillary artery aneurysm (unilateral clubbing)
Respiratory	Lung cancer Mesothelioma Fibrosing alveolitis Bronchiectasis Cystic fibrosis Empyema
Gastrointestinal	Malabsorption (e.g. coeliac disease) Crohn's disease Ulcerative colitis Cirrhosis
Endocrine	Thyroid acropachy
Familial	'Pseudoclubbing'



Central Cyanosis

Beau's Lines
(Transverse lines)



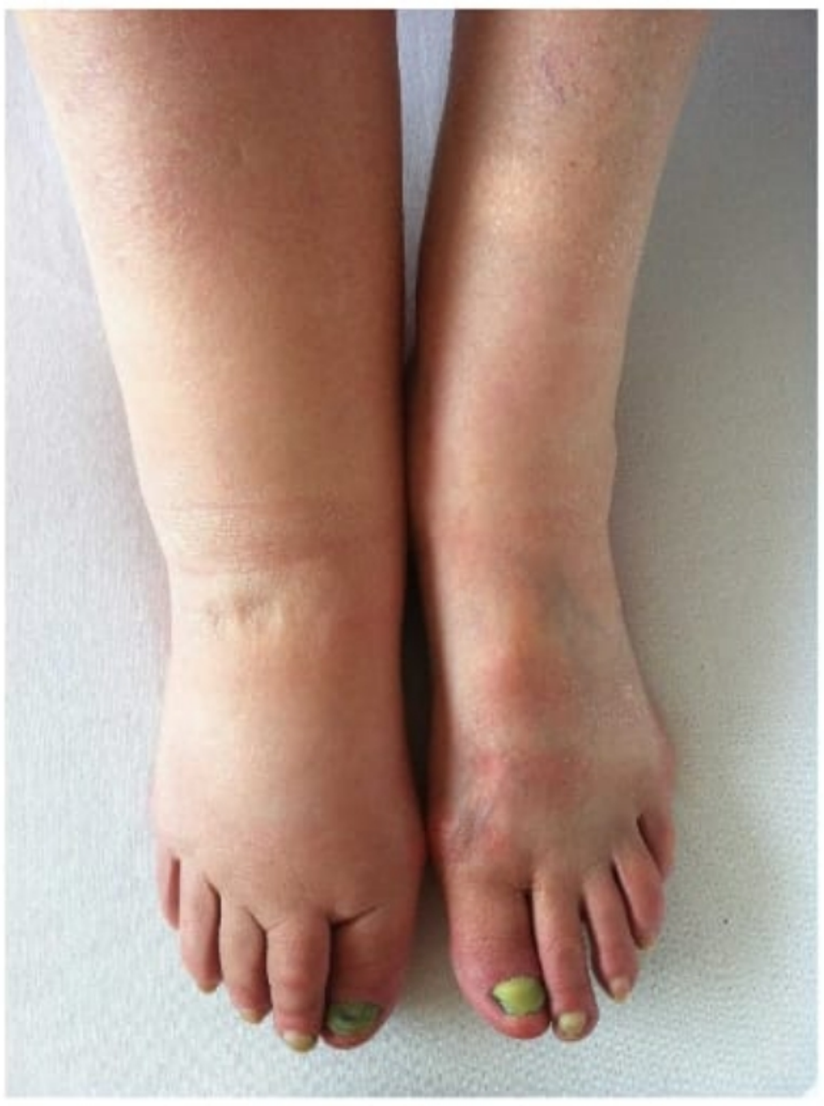


- Malnutrition
- Cachexia

Lung Sounds

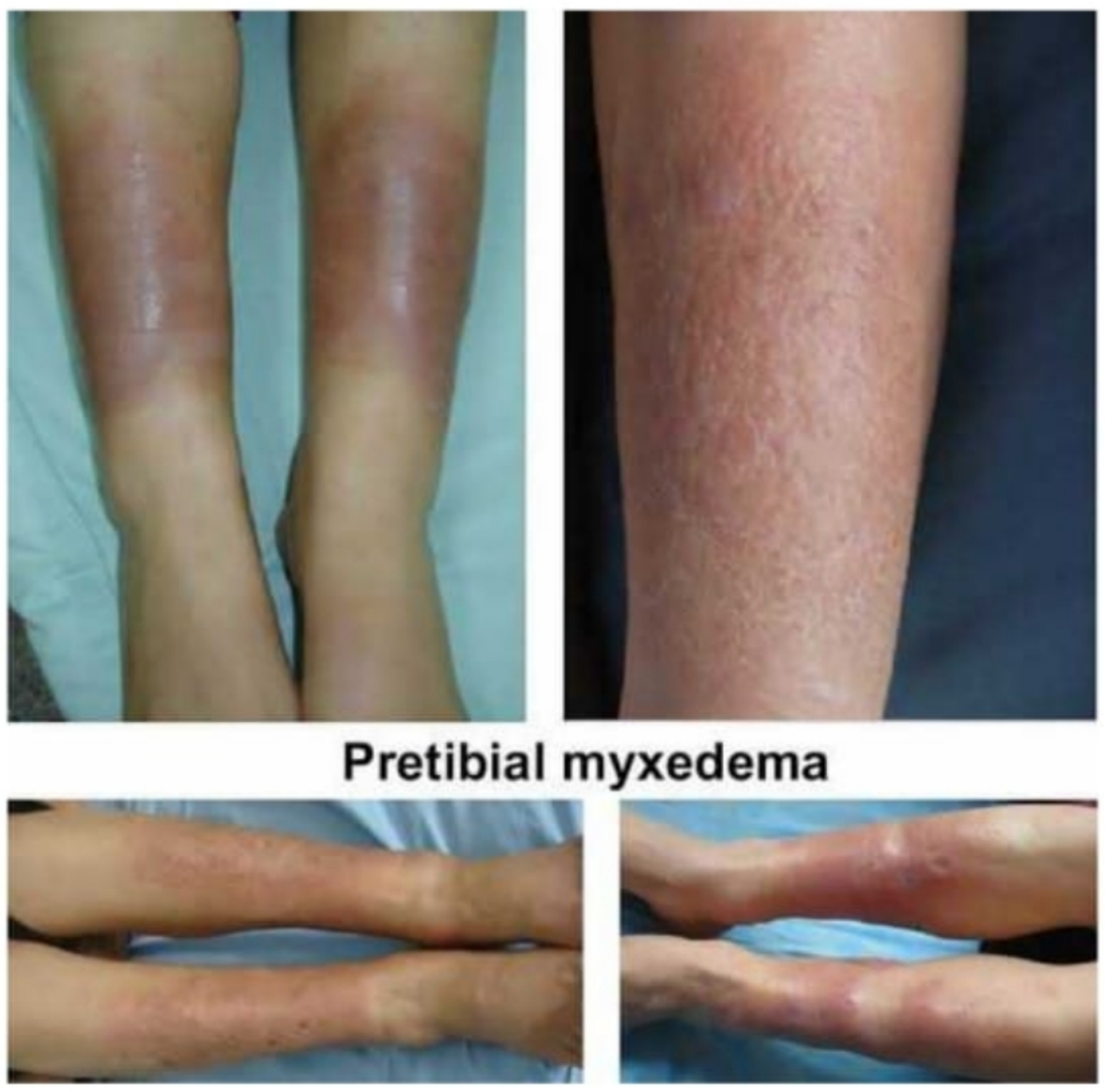
- Musical wheeze → Asthma
  Bronchitis

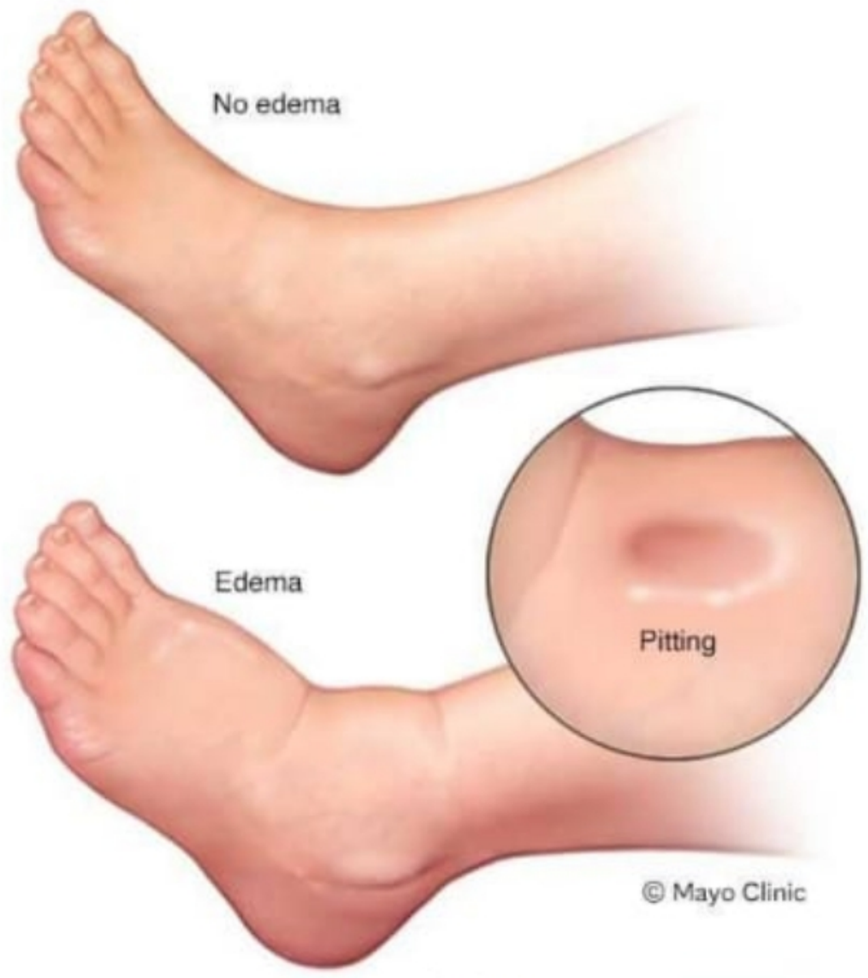
- Fine Crackles → Heart Failure
  Fibrosis



Non Pitting Edema

* Hypothyroidism (pretibial myxedema)
* Lymphoedema



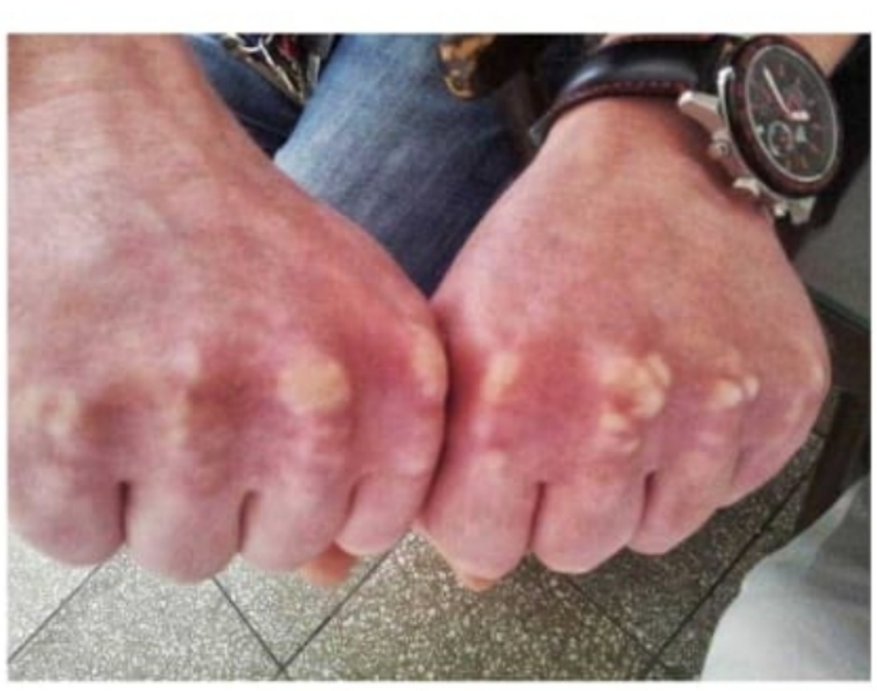


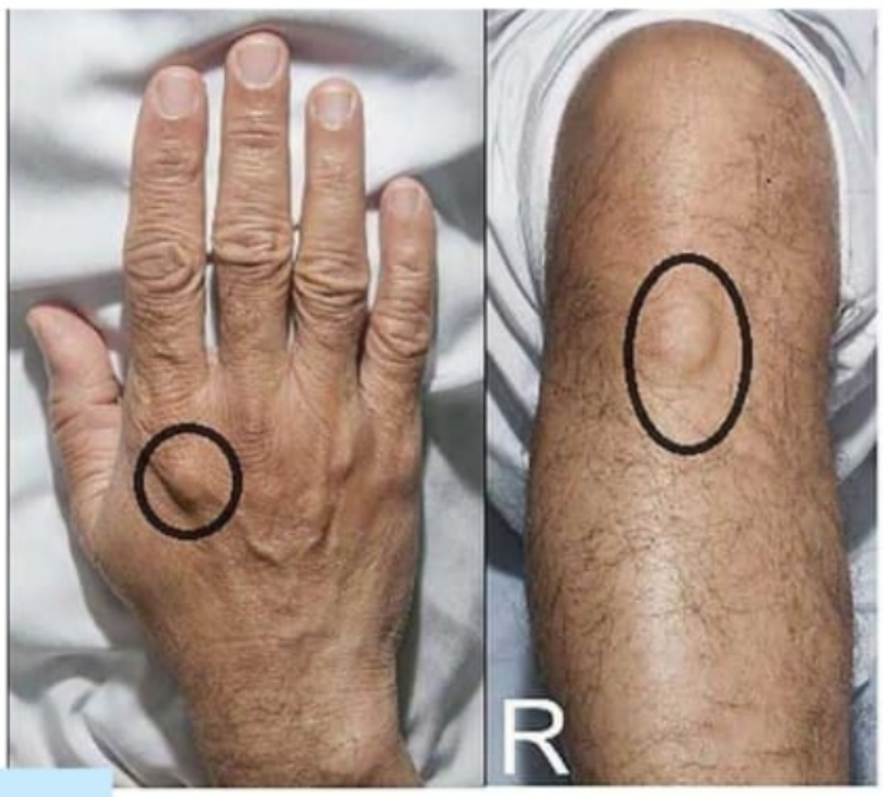
Pitting Edema

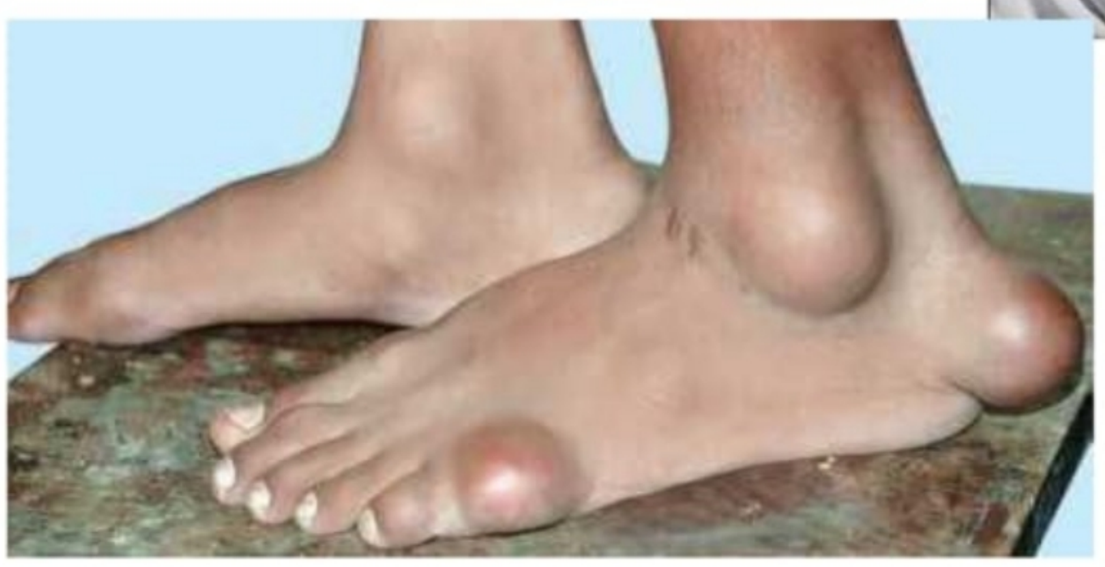
Table 7.4 Causes of pitting oedema

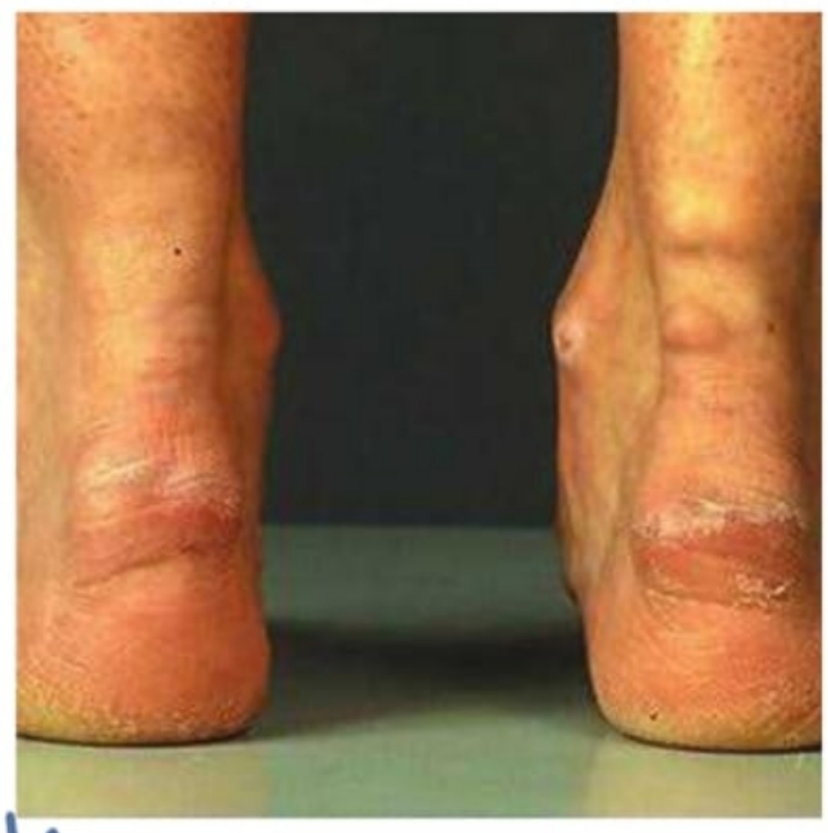
System	Causes
Cardiac	Congestive cardiac failure Constrictive pericarditis
Vascular	Deep vein thrombosis (usually unilateral) Chronic venous insufficiency (uni- or bilateral) Venous compression by pelvic or abdominal mass (uni- or bilateral) Inferior vena cava obstruction
Gastrointestinal	Hypoalbuminaemia secondary to malabsorption, protein-losing enteropathy, cirrhosis of the liver
Renal	Hypoalbuminaemia secondary to nephrotic syndrome
Pharmacological	Calcium channel blockers Fludrocortisone
Metabolic	Thiamine deficiency (wet beri-beri)
General	Immobility ('dependent oedema')

Tendon Xanthomas









- yellowish cholesterol deposits in the tendons, often in hands or at elbow/knee

- Seen in hyperlipidemia

JVP

Table 7.8 Distinguishing characteristics of venous and arterial neck pulsation

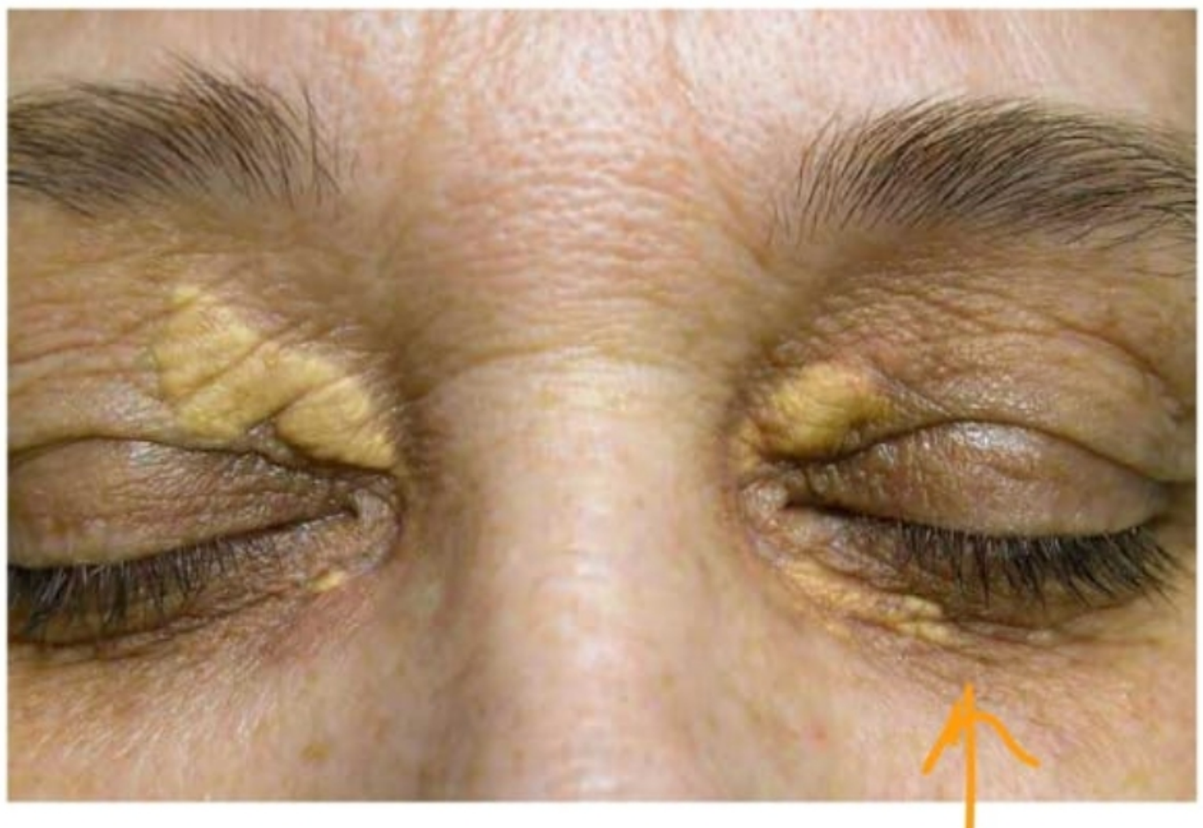
Internal jugular vein	Carotid artery
Two pulsations per cardiac cycle (if in sinus rhythm)	One pulsation per cardiac cycle
Prominent inward movement	Prominent outward movement
Varies with respiration	No variation with respiration
Varies with patient position	No variation with patient position
Palpable pulsation	Impalpable pulsation
Easily occluded by light pressure	Not easily occluded by light pressure

- Examination of JVP tells about
  1. pressure in Right Atrium → from height of JVP
  2. Right heart Function → from JVP waveform

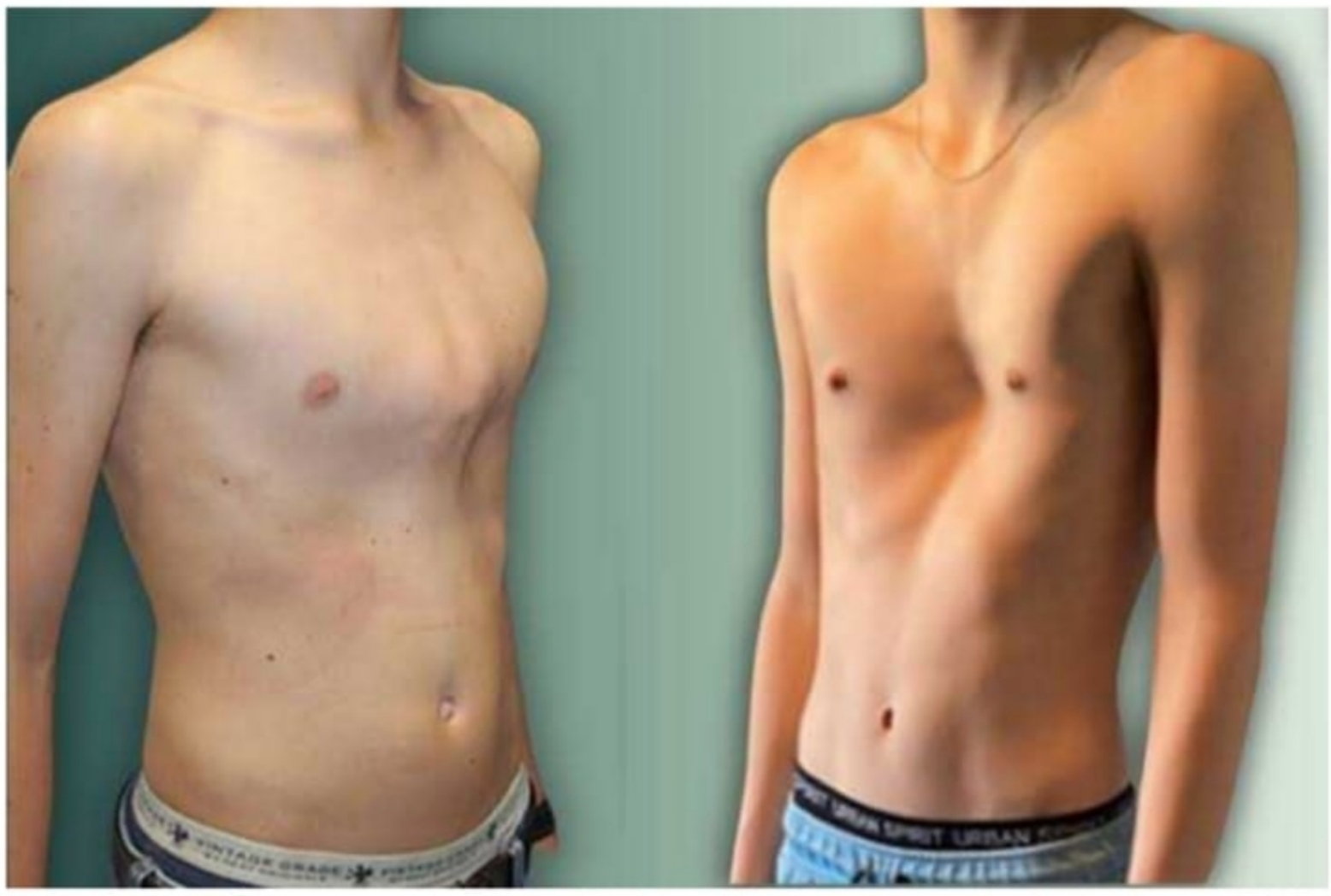
BOX 7.2 CAUSES OF ELEVATED JVP

- Heart failure
- Fluid overload
- Superior vena cava obstruction
- Pulmonary embolism
- Constrictive pericarditis
- Cardiac tamponade

Xanthelasma



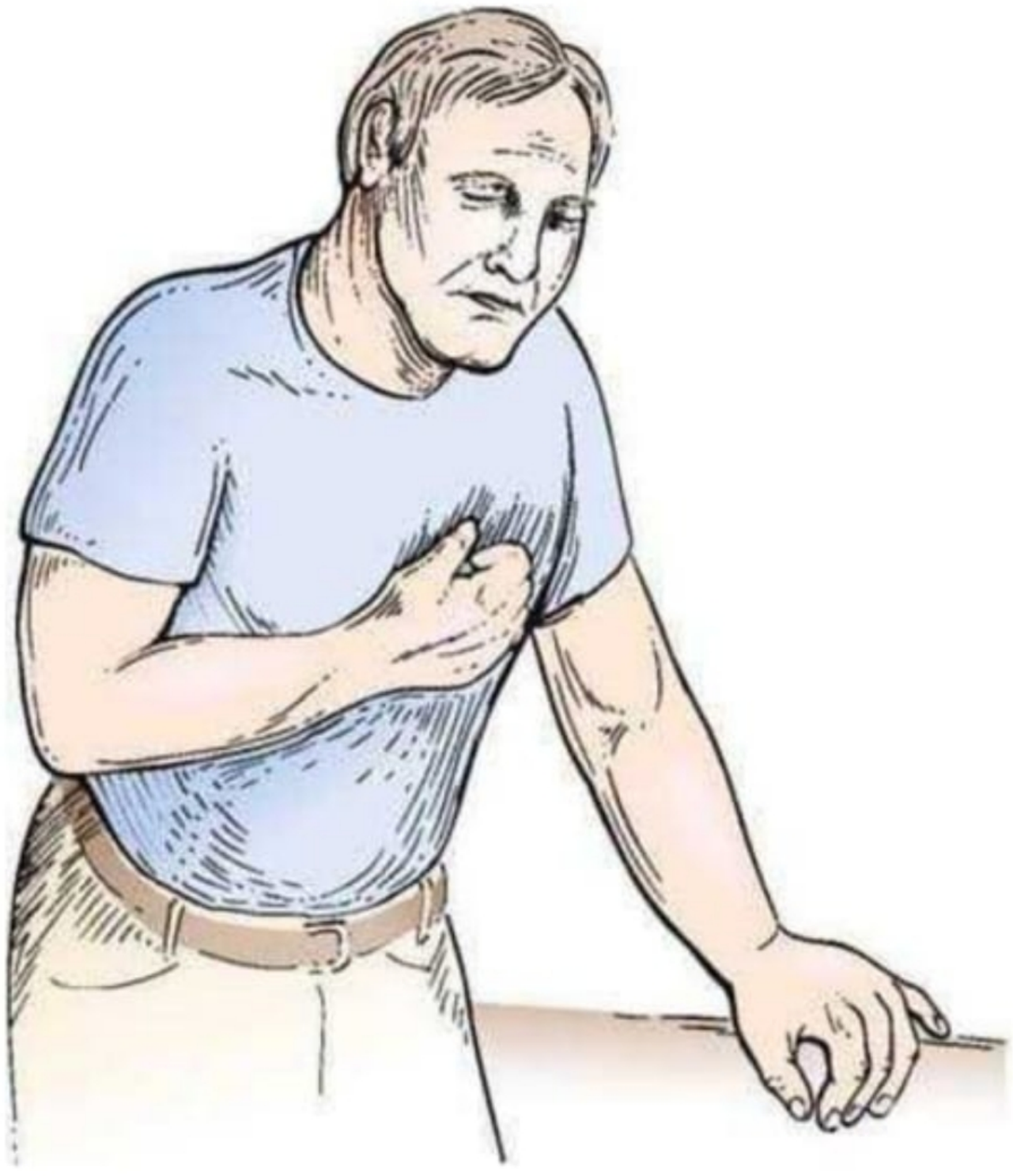
deposits of cholesterol



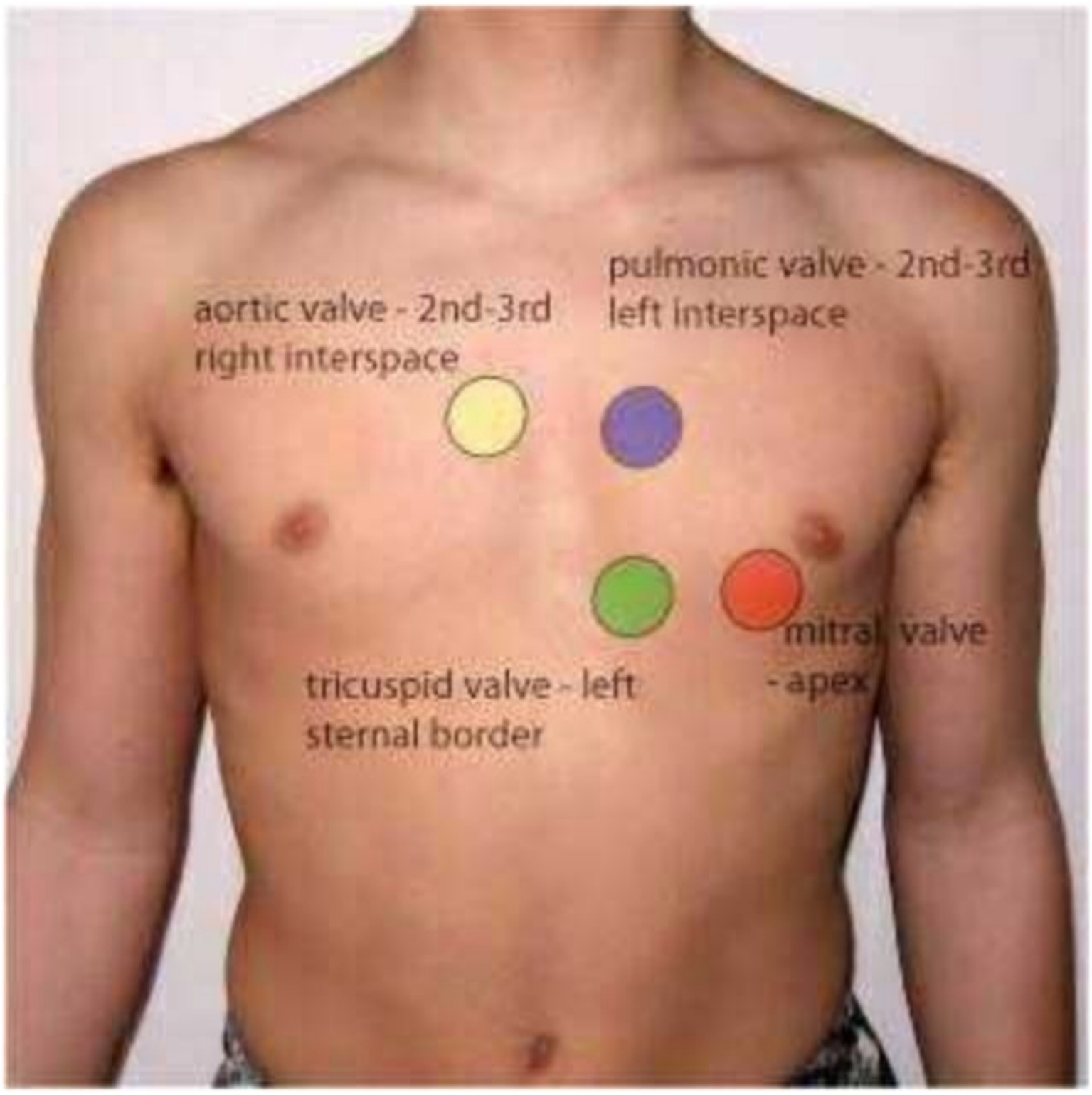
Pectus Carinatum
(Pigeon Chest Deformity)

Pectus Excavatum
(Funnel Chest
Deformity)

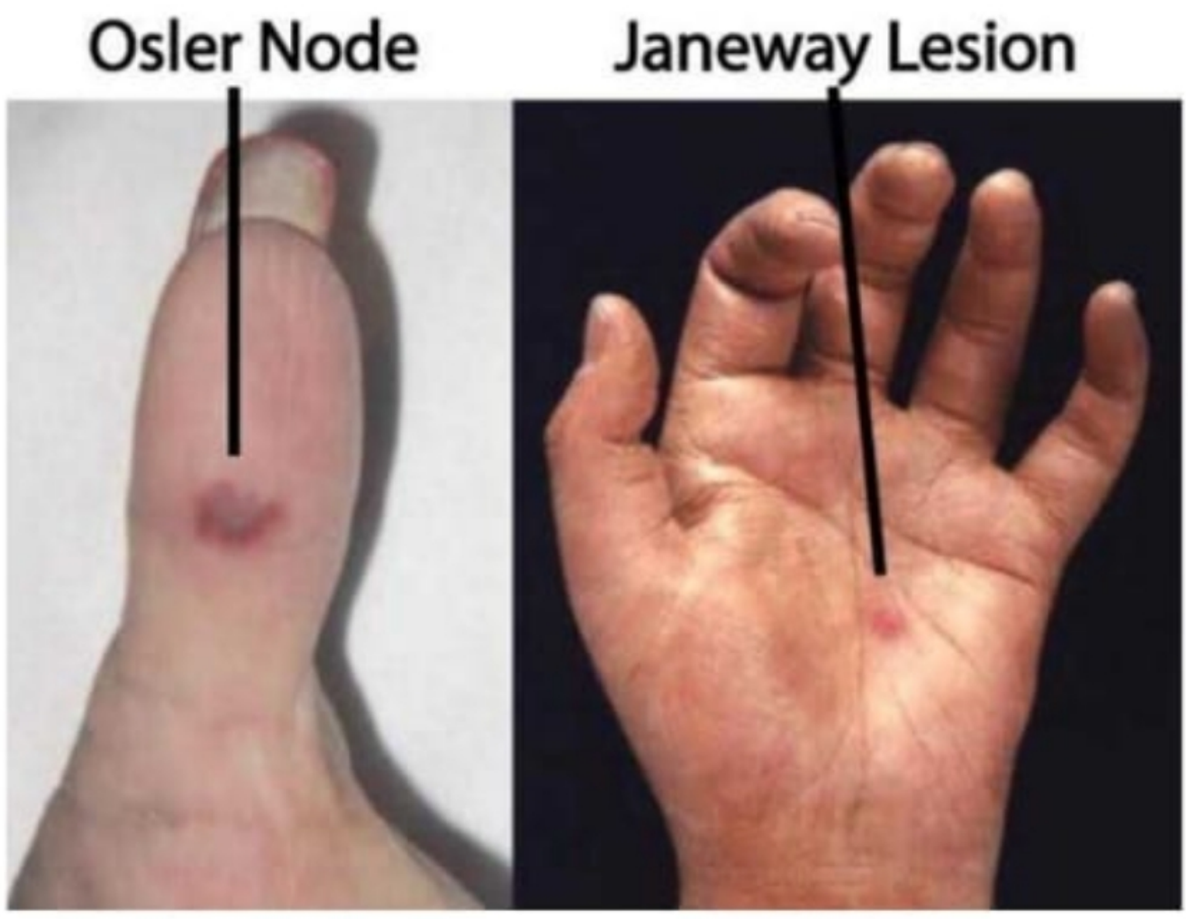
Levine's Sign

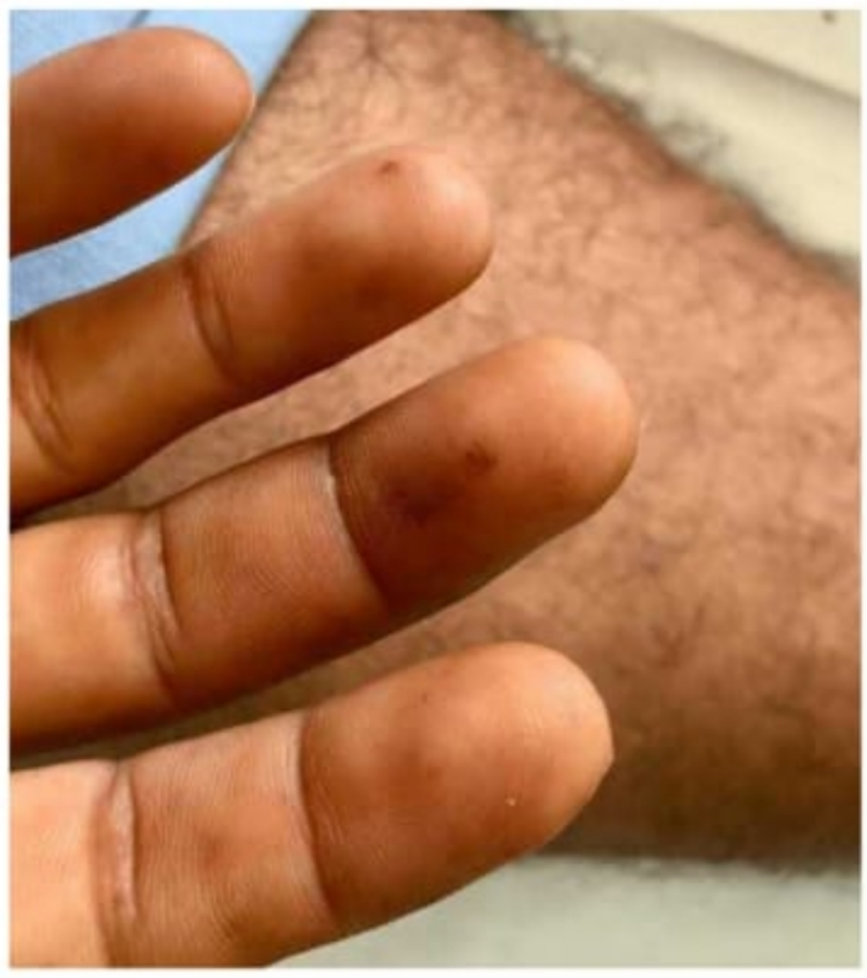


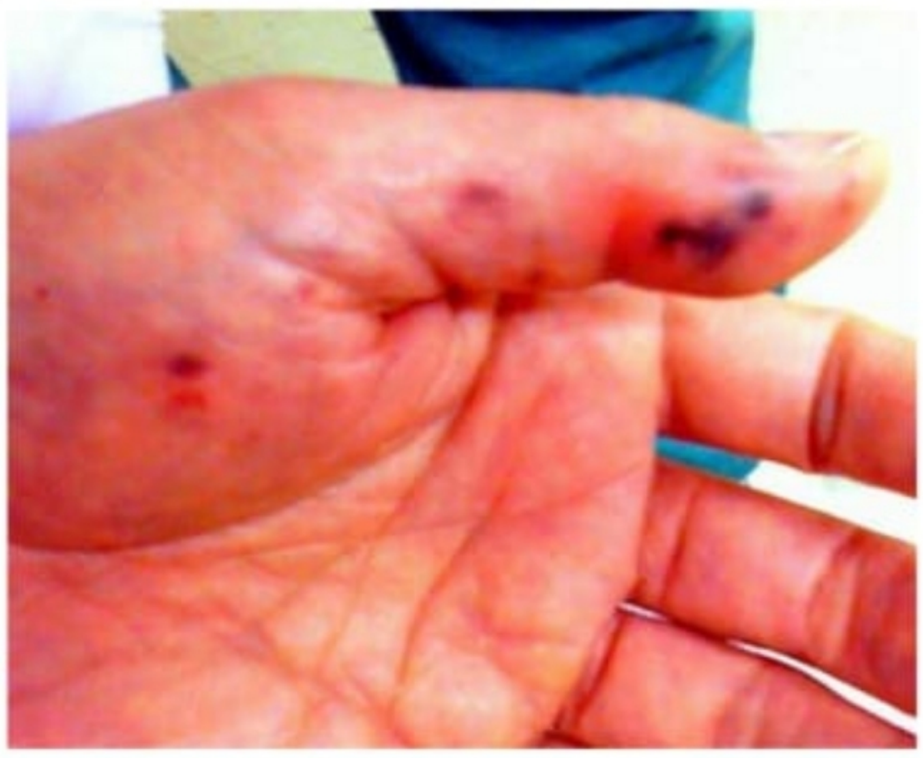
Levine's sign, also known as the "clenched fist sign," is a gesture where a person clenches their fist and places it over their chest, often while describing chest pain.


aortic valve - 2nd-3rd
right interspace
pulmonic valve - 2nd-3rd
left interspace
tricuspid valve - left
sternal border
mitral valve
- apex

OSLER'S NODES







- Tender, red lesions that occur on the fingertips, palms and soles

- uncommon manifestation of infective endocarditis

* Janeway lesions are non-tender!