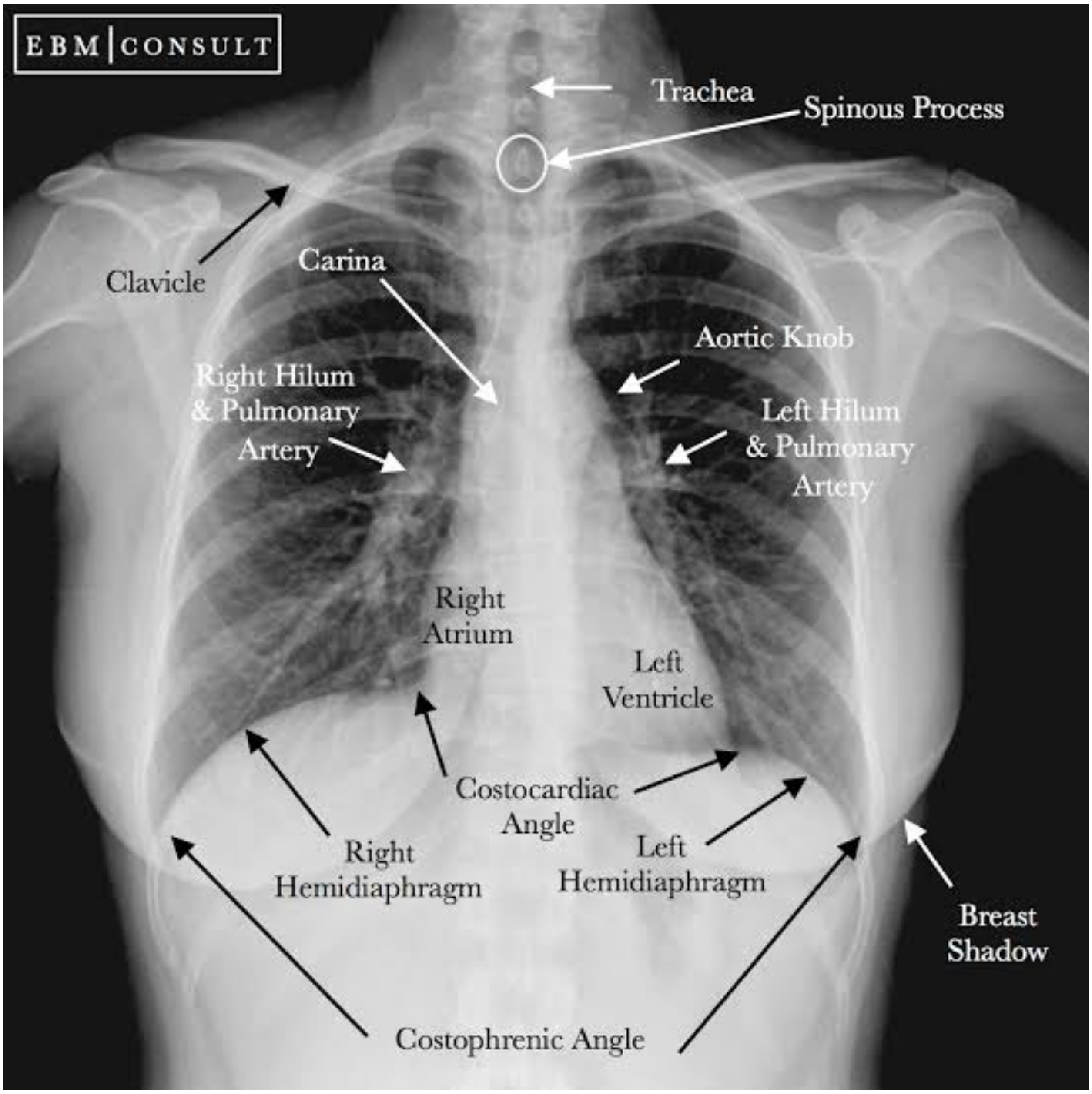
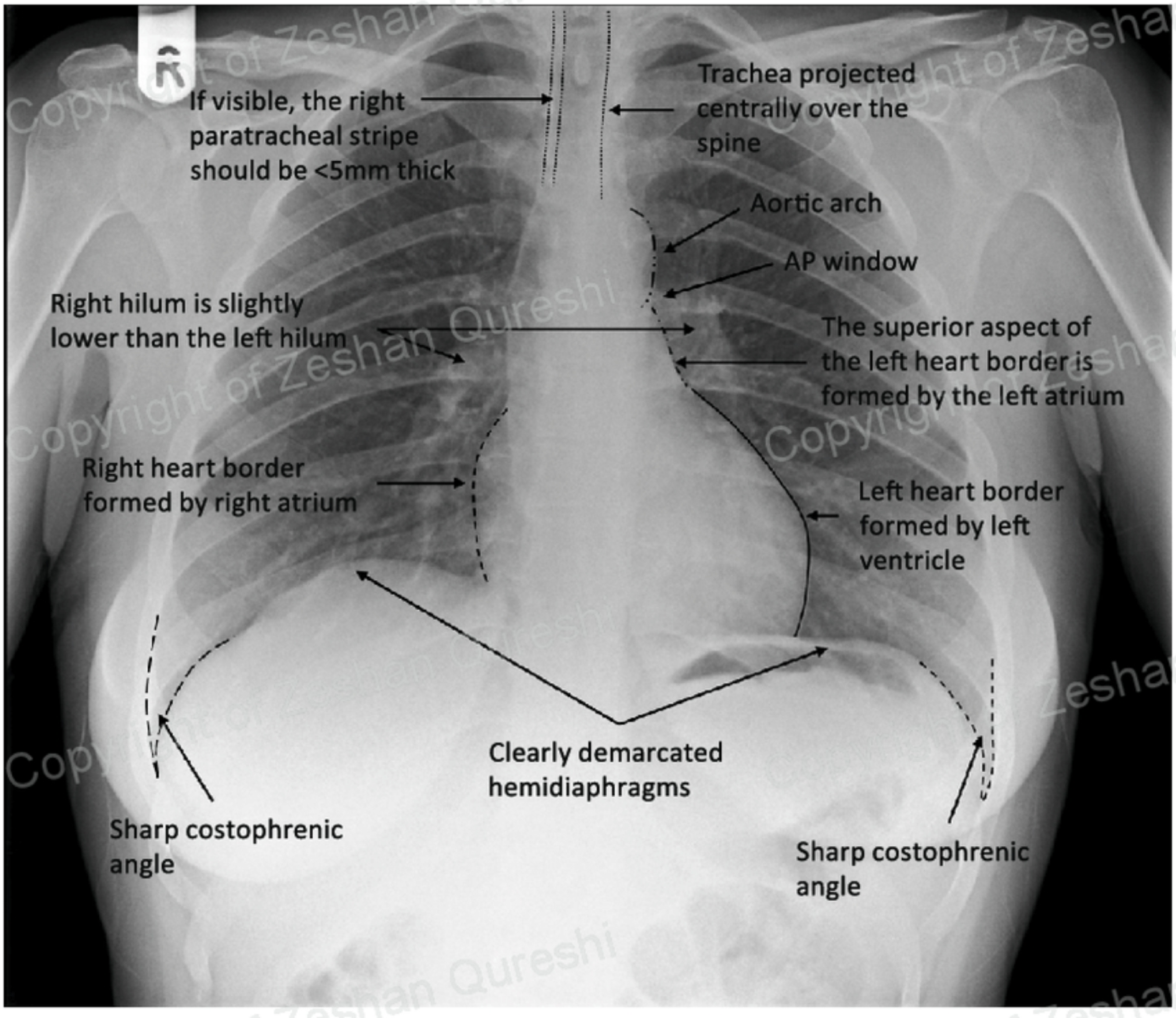
Normal X-Ray

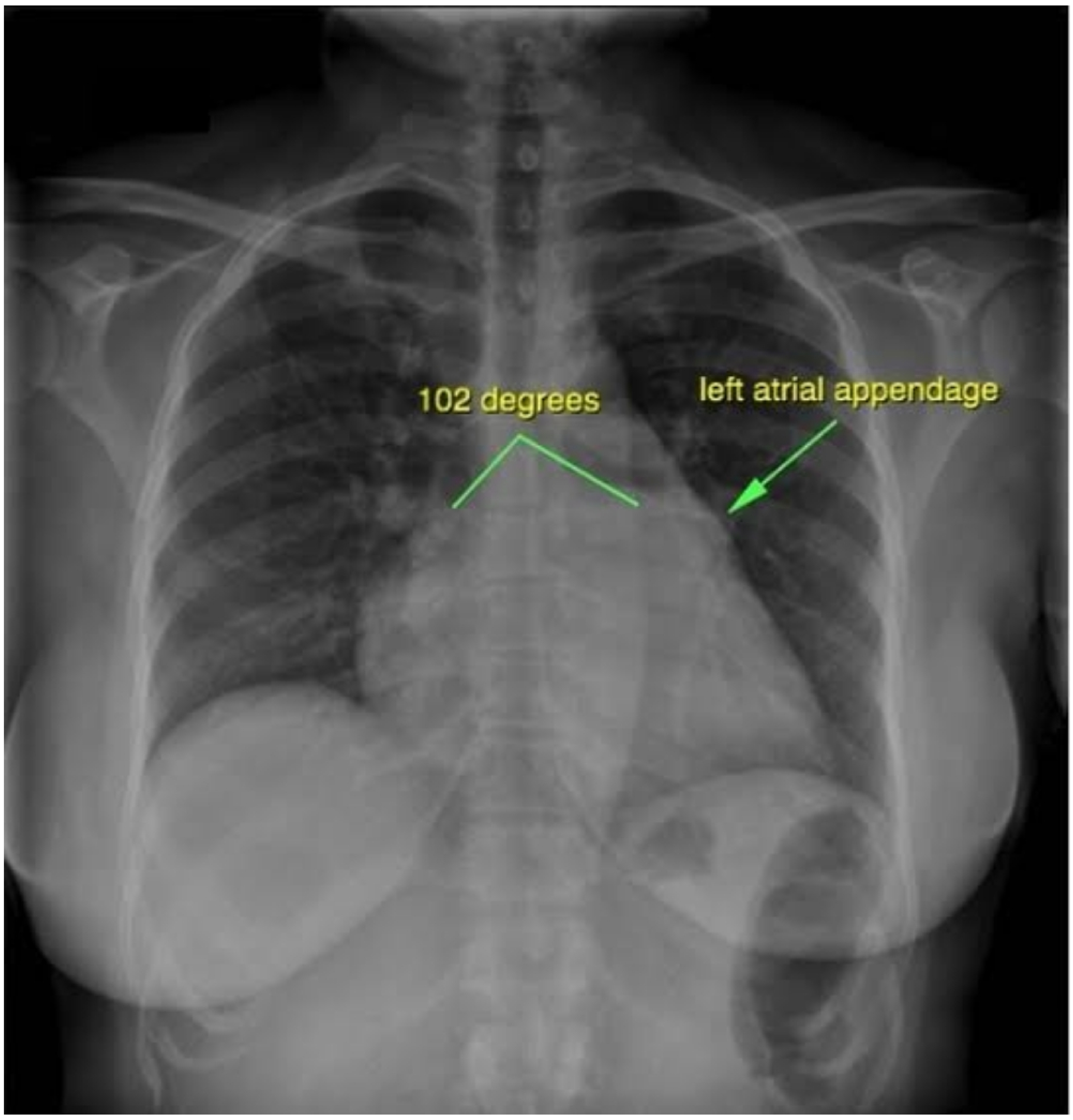


EBM CONSULT
Trachea
Spinous Process
Clavicle
Carina
Aortic Knob
Right Hilum & Pulmonary Artery
Left Hilum & Pulmonary Artery
Right Atrium
Left Ventricle
Costocardiac Angle
Right Hemidiaphragm
Left Hemidiaphragm
Breast Shadow
Costophrenic Angle

Normal PA Chest X-Ray

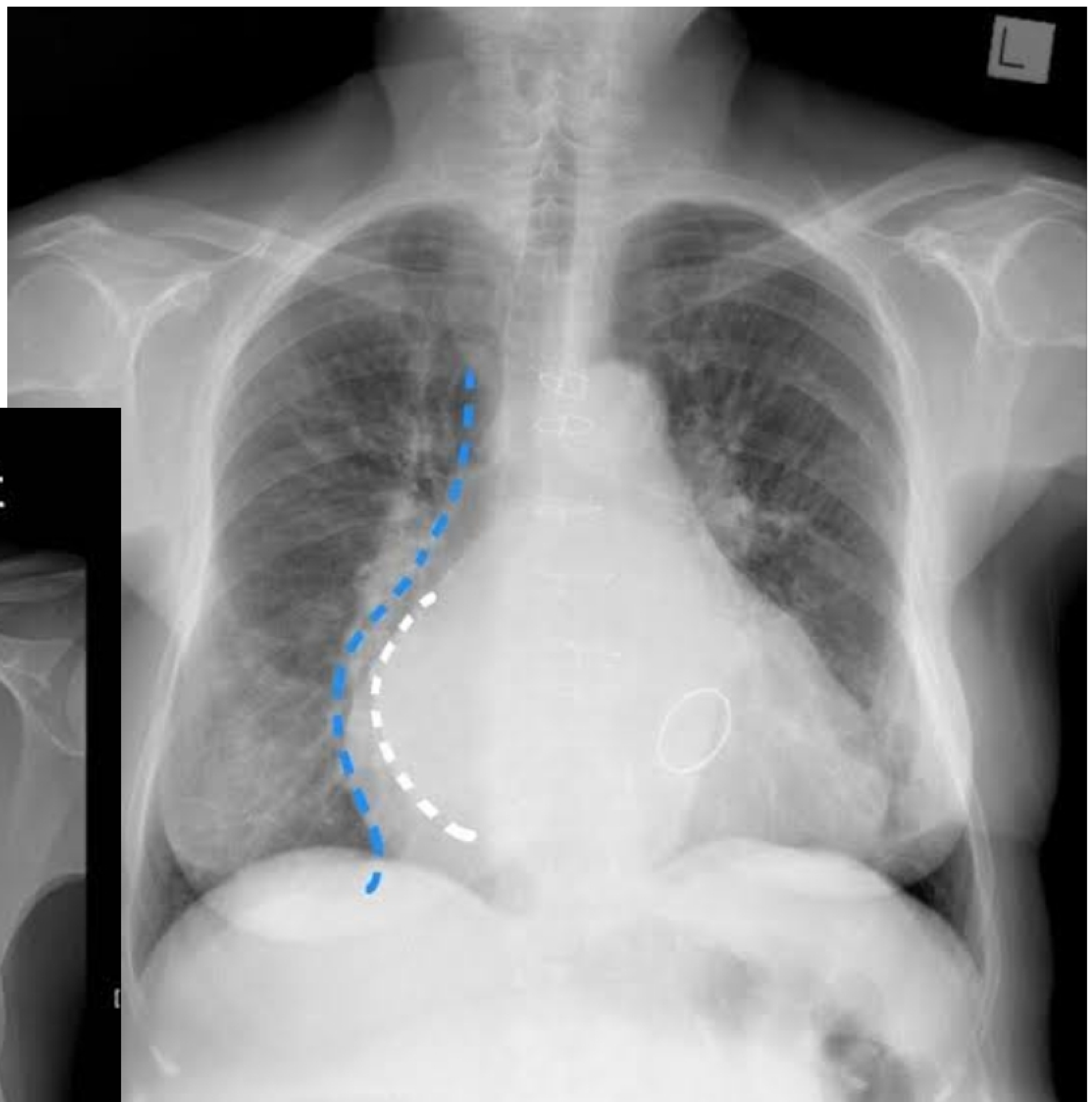


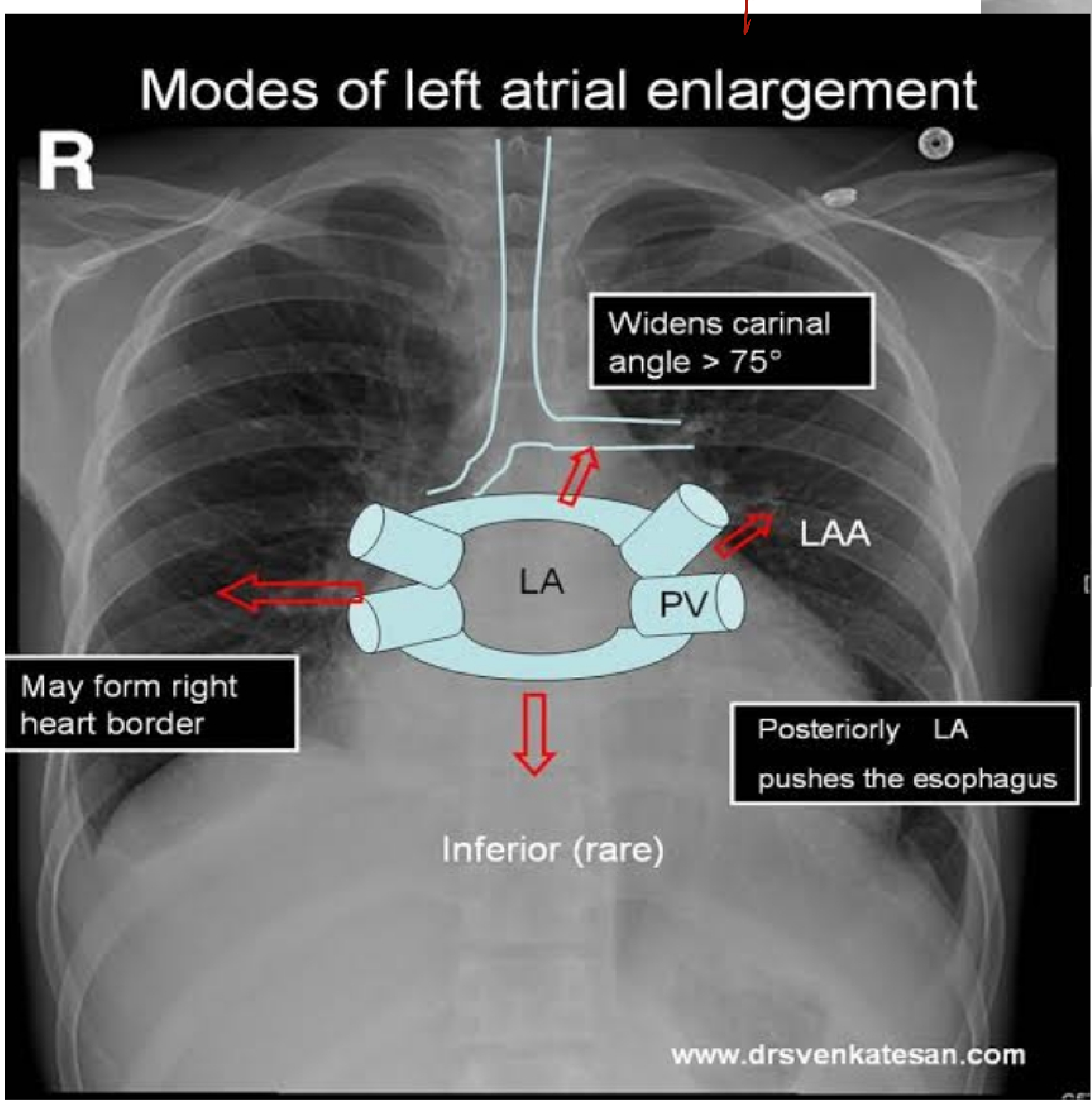
Left Atrial Dilatation (X-Ray)



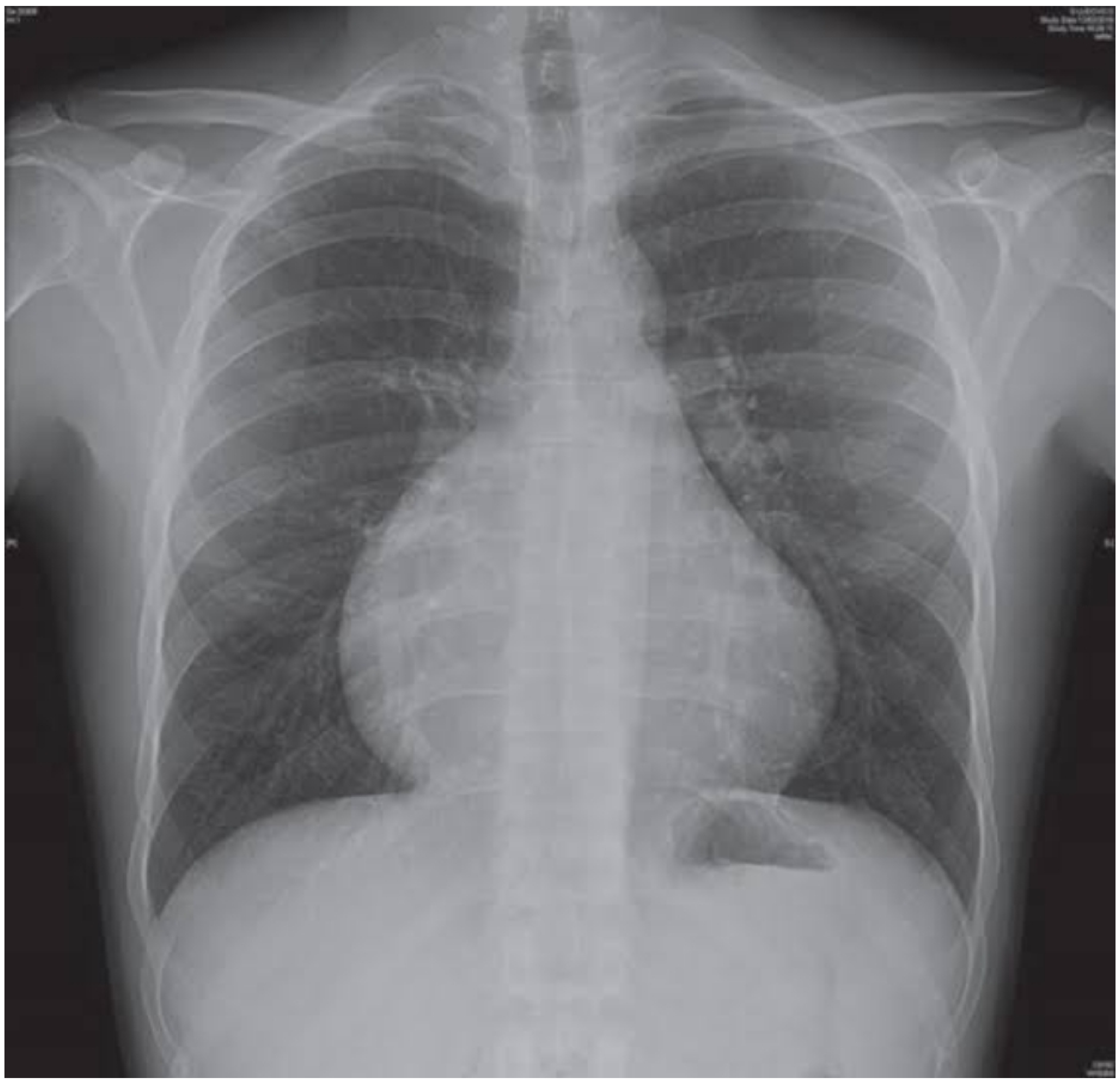
- Prominence of left atrial appendage
- Appearance of straight left heart border
- Double cardiac shadow to right of sternum

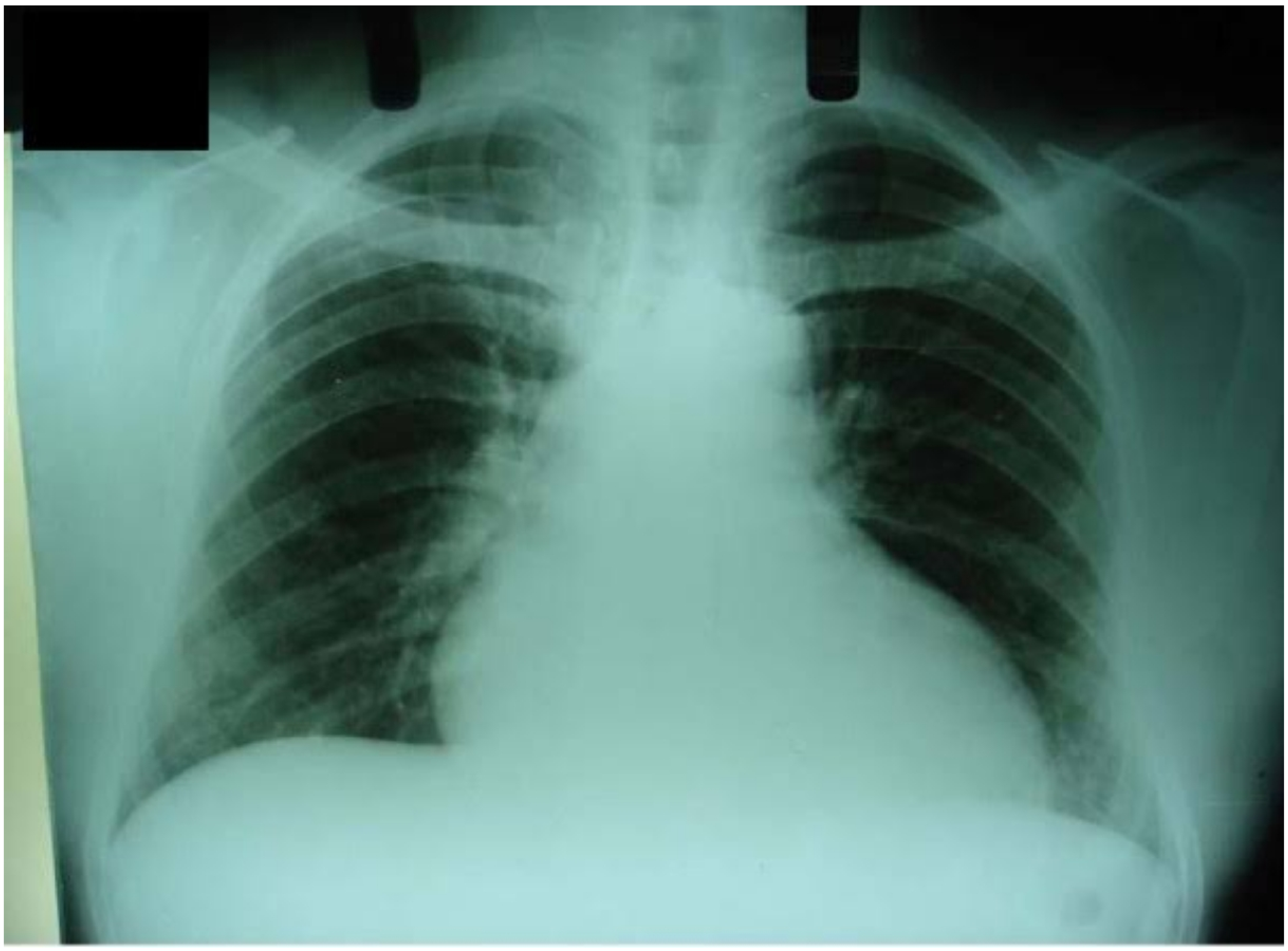
- Widening of angle of carina (bifurcation of trachea) as left main bronchus is pushed upwards



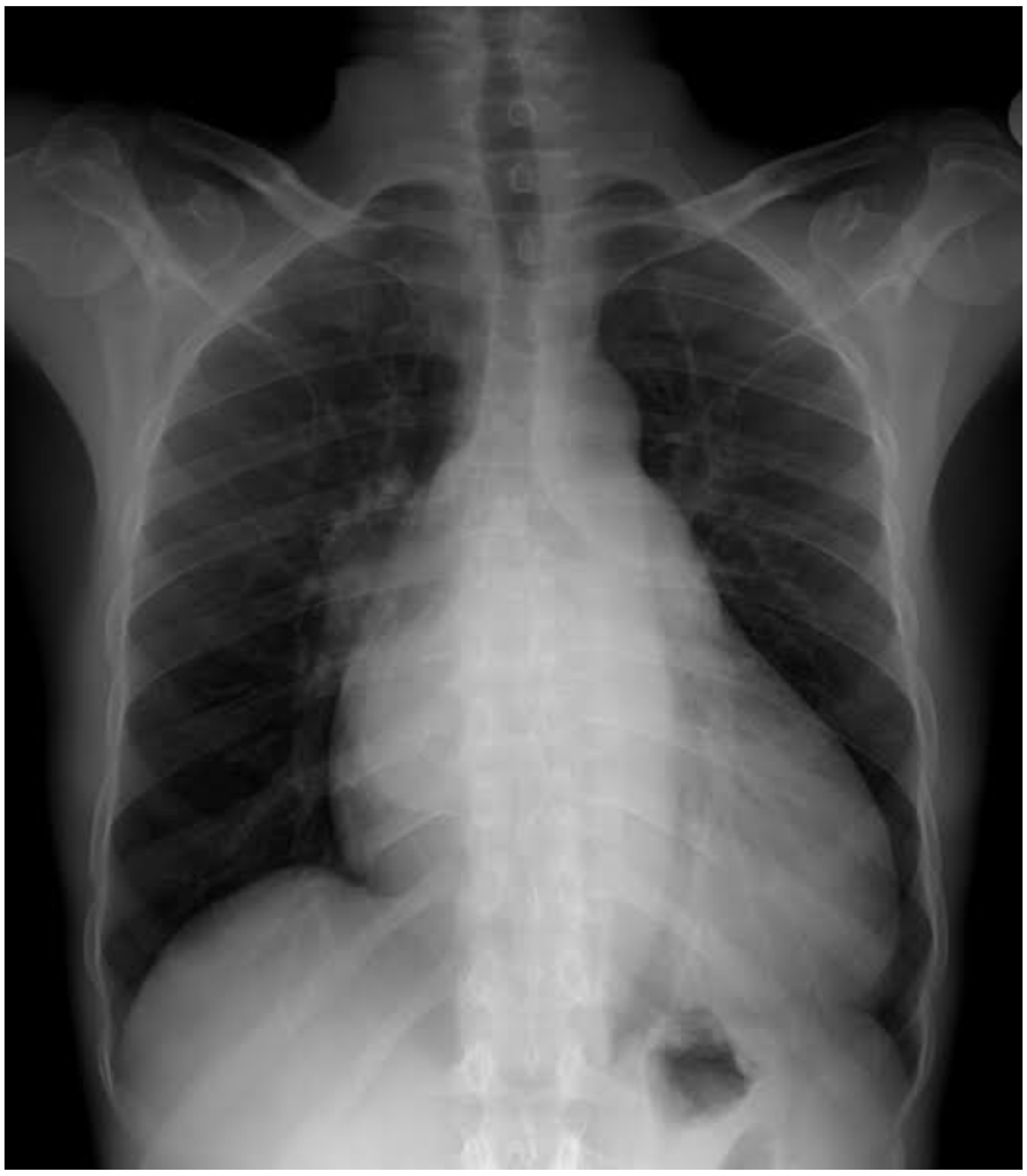


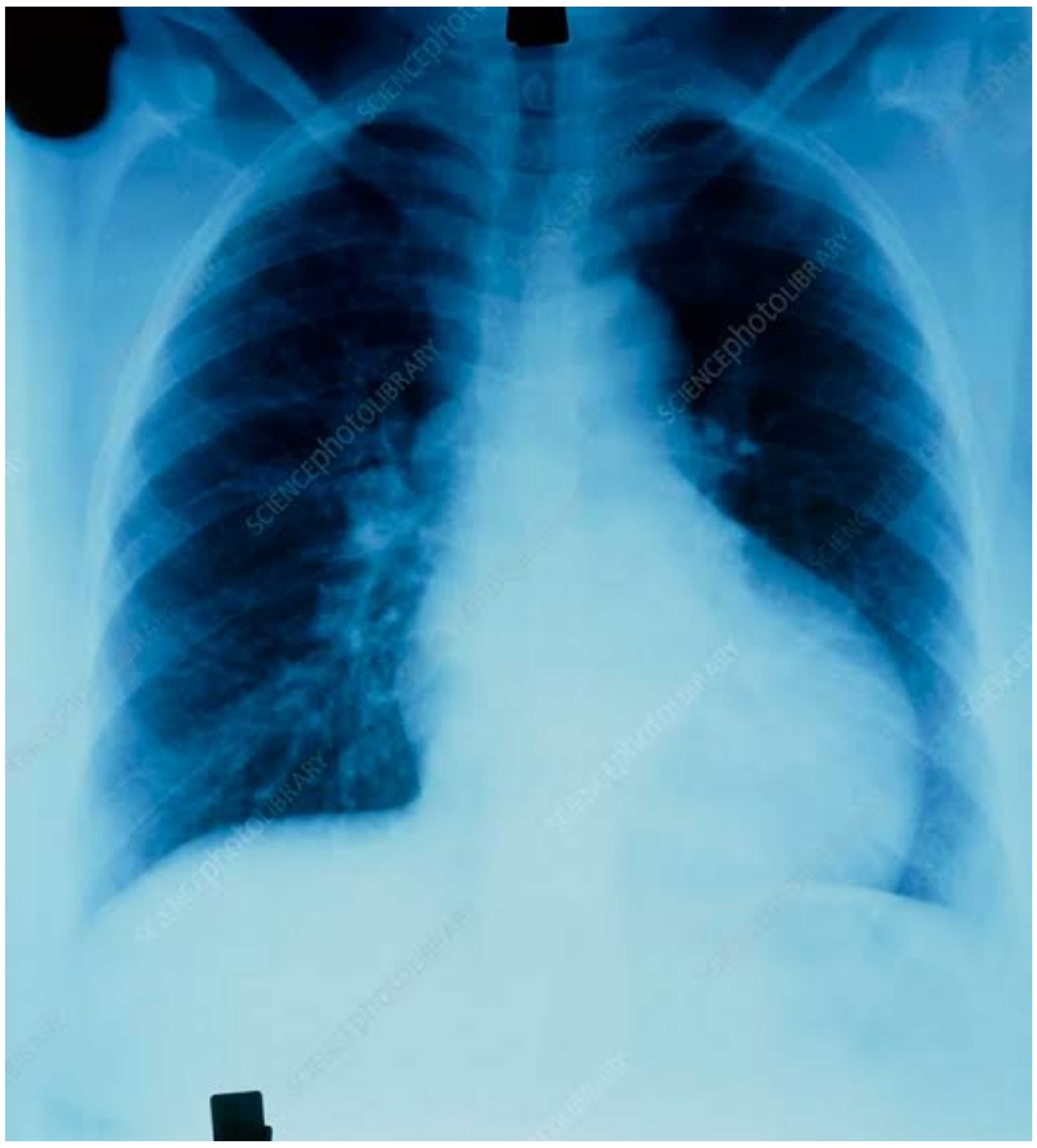
Right Atrial Enlargement (X-Ray)

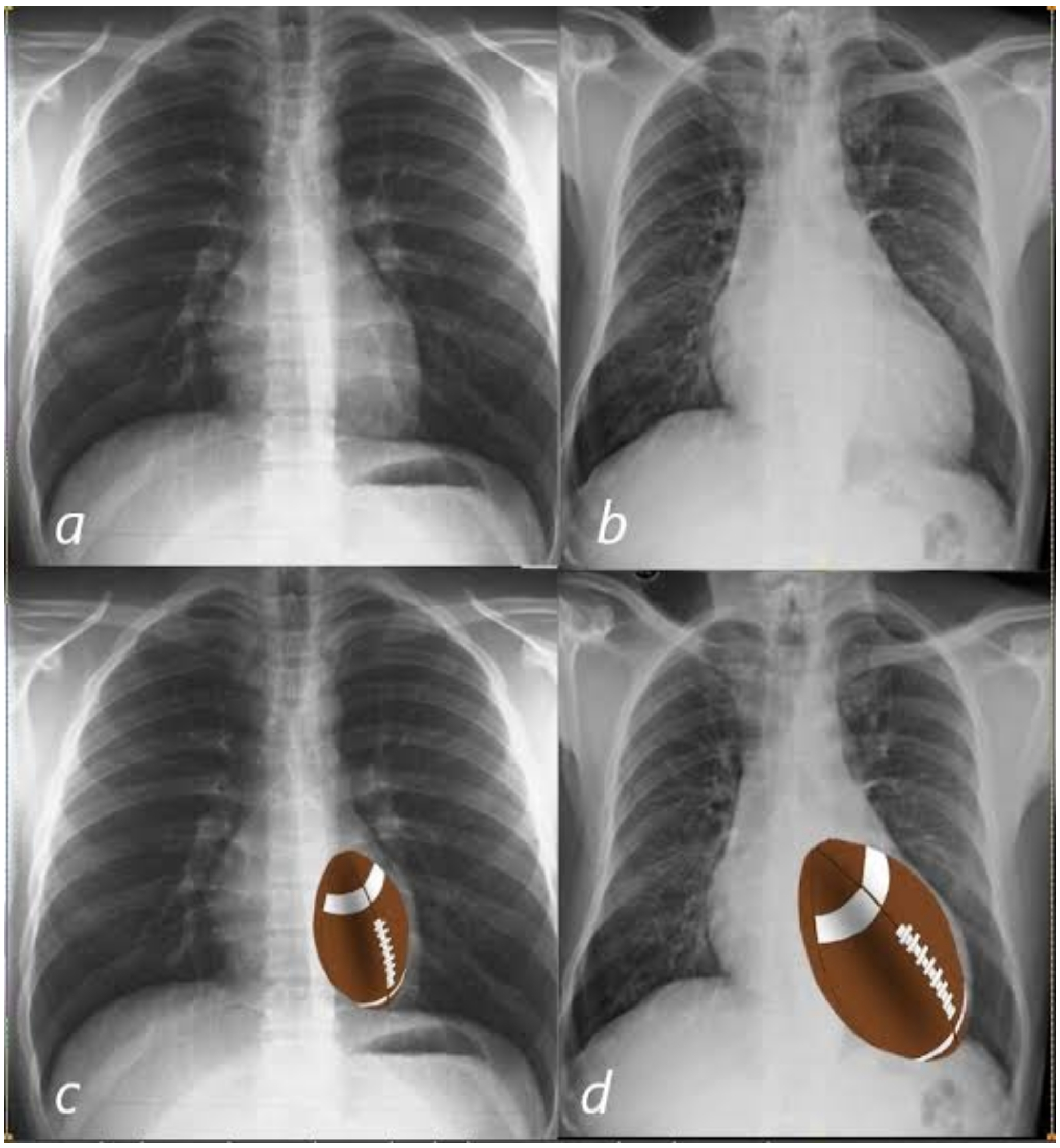


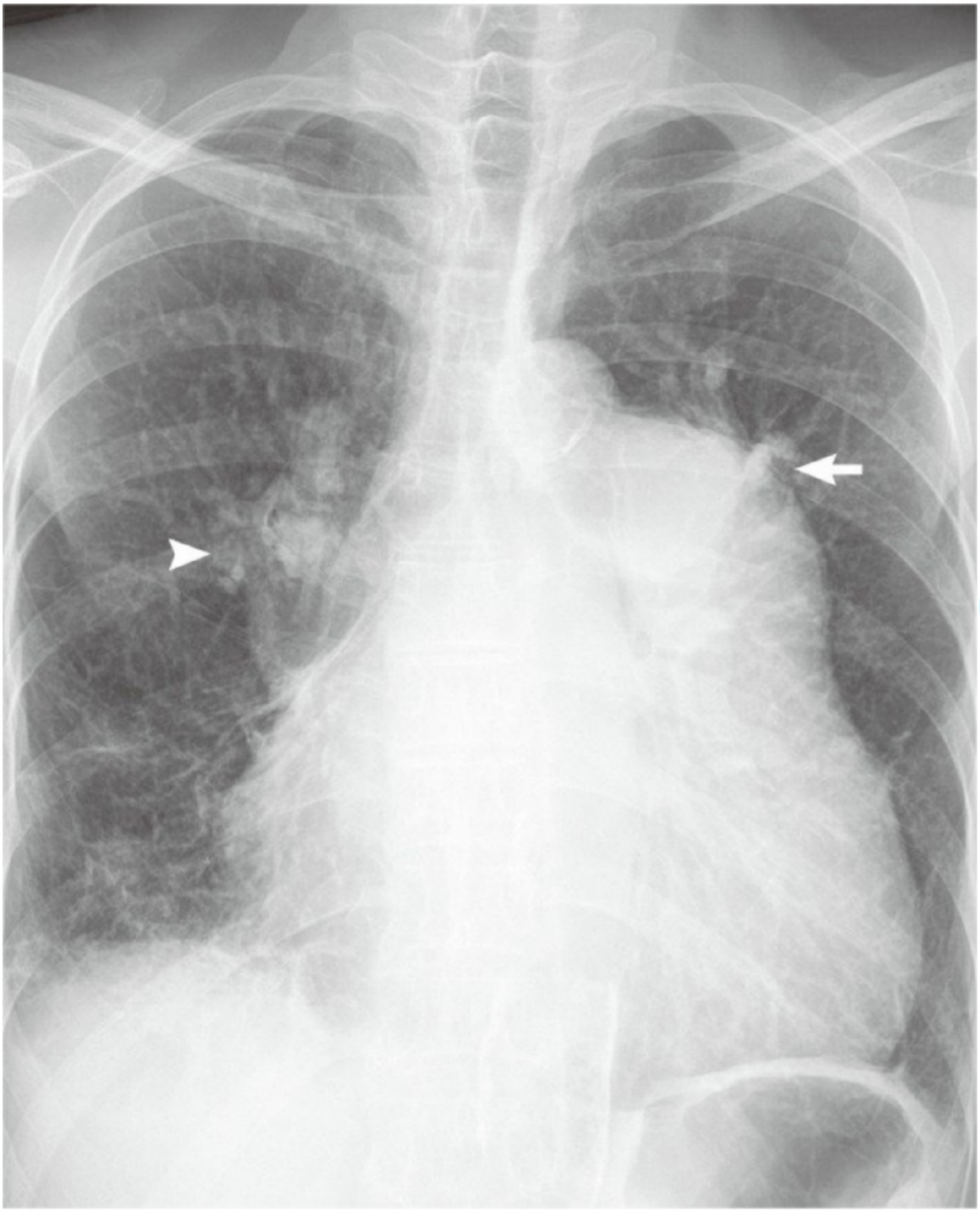


Left Ventricular Enlargement X-Ray









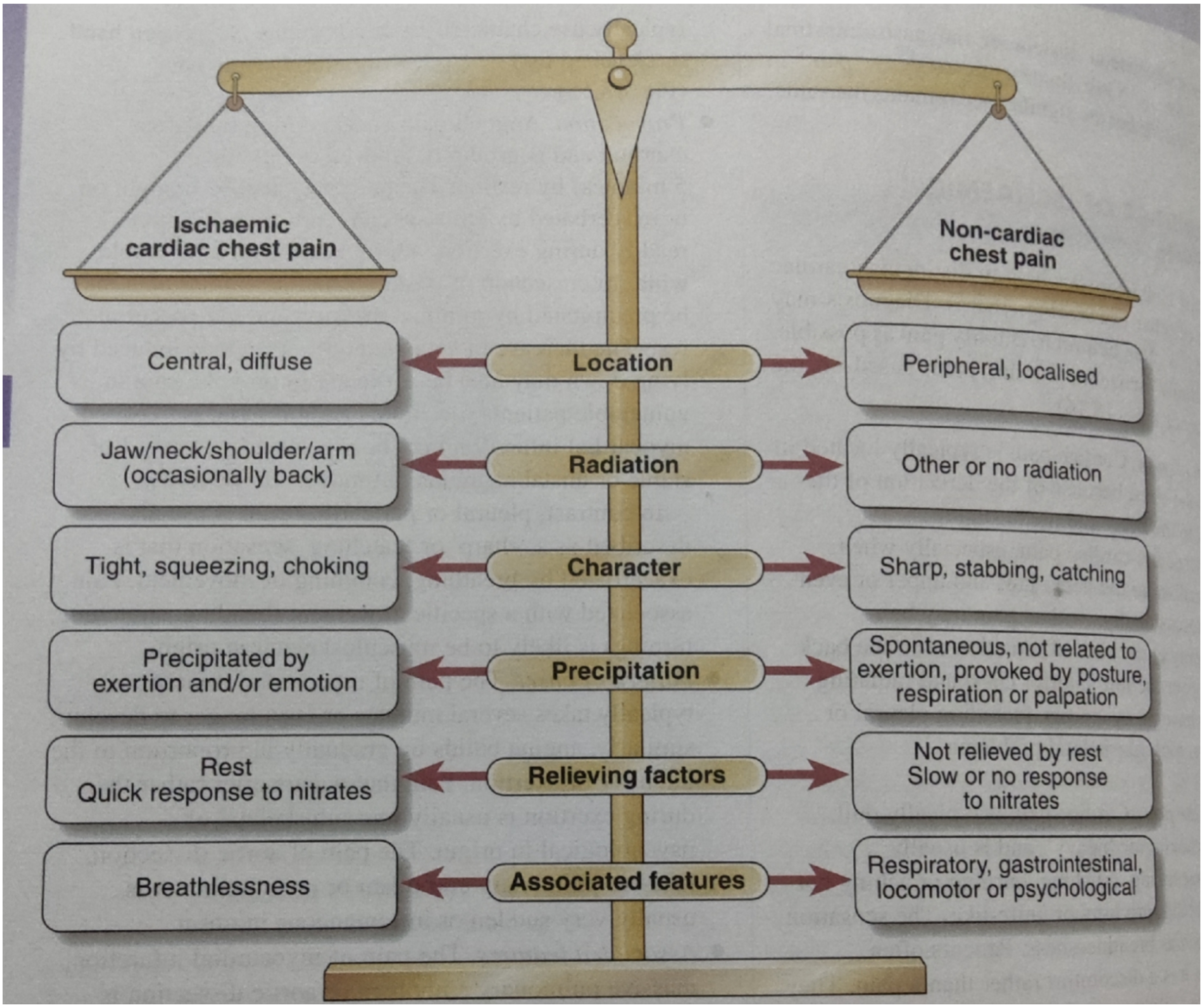
Chest X-ray. Cardiomegaly involving right chambers, enlarged pulmonary trunk (arrow), and right main pulmonary artery (head arrow).

Presenting problems in CVS

- Chest pain / Chest Discomfort
- Breathlessness (Dyspnea)
- Palpitation
- Pre Syncope and Syncope
- Peripheral edema
- Hypertension

Characteristics of Ischemic Cardiac Pain

- Location → centre of chest, diffuse (not localized)
- Radiate to neck, jaw, upper or even lower arms.
- Pain is usually dull, constricting, choking or heavy and usually described as squeezing, crushing, burning or aching (but not sharp, stabbing, pricking or knife-like)
- Anginal pain occurs during (not after) exertion



Ischaemic cardiac chest pain
Non-cardiac chest pain
Central, diffuse
Location
Peripheral, localised
Jaw/neck/shoulder/arm (occasionally back)
Radiation
Other or no radiation
Tight, squeezing, choking
Character
Sharp, stabbing, catching
Precipitated by exertion and/or emotion
Precipitation
Spontaneous, not related to exertion, provoked by posture, respiration or palpation
Rest
Quick response to nitrates
Relieving factors
Not relieved by rest
Slow or no response to nitrates
Breathlessness
Associated features
Respiratory, gastrointestinal, locomotor or psychological

18.8 COMMON CAUSES OF CHEST PAIN

Anxiety/emotion

Cardiac

- Myocardial ischaemia (angina)
- Myocardial infarction
- Myocarditis varies ē phase, movement of respiration
- Pericarditis
- Mitral valve prolapse

Aortic

- Aortic dissection abrupt to back
- Aortic aneurysm

Oesophageal — eating + posture related

- Oesophagitis
- Oesophageal spasm
- Mallory–Weiss syndrome

Lungs/pleura

- Bronchospasm
- Pulmonary infarct
- Pneumonia
- Tracheitis
- Pneumothorax
- Pulmonary embolism
- Malignancy
- Tuberculosis
- Connective tissue disorders (rare)

Musculoskeletal

- Osteoarthritis
- Rib fracture/injury
- Intercostal muscle injury
- Costochondritis (Tietze's syndrome)
- Epidemic myalgia (Bornholm disease)

Neurological

- Prolapsed intervertebral disc
- Herpes zoster
- Thoracic outlet syndrome

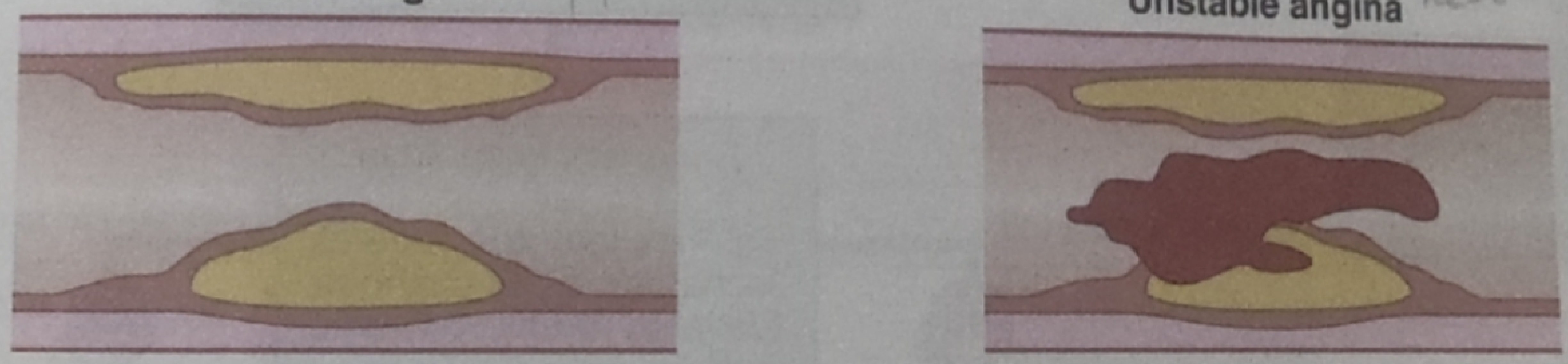
	Stable angina effort	Unstable angina rest
		
Pathophysiology	• Fixed stenosis	• Dynamic stenosis
Clinical features	• Demand-led ischaemia • Related to effort • Predictable • Symptoms over long term	• Supply-led ischaemia • Symptoms at rest • Unpredictable • Symptoms over short term
Risk assessment	• 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

Unstabe Angina
- Prolonged and severe cardiac chest pain

Acute pulmonary edema

- patient fighting for breath
- sitting upright or standing may provide some relief
- Patient unable to speak
- pt typically distressed, agitated, cyanosed, sweaty, pale
- Rapid respiration
- Extensive crepitations and rhonchi are usually audible in chest
- there may be signs of right heart failure

18.9 SOME CAUSES OF DYSPNOEA		
System	**Acute dyspnoea at rest**	**Chronic exertional dyspnoea**
Cardiovascular system	*Acute pulmonary oedema	*Chronic congestive cardiac failure Myocardial ischaemia
Respiratory system	*Acute severe asthma *Acute exacerbation of chronic obstructive pulmonary disease *Pneumothorax *Pneumonia *Pulmonary embolus Acute respiratory distress syndrome Inhaled foreign body (especially in the child) Lobar collapse Laryngeal oedema (e.g. anaphylaxis)	*Chronic obstructive pulmonary disease *Chronic asthma Chronic pulmonary thromboembolism Bronchial carcinoma Interstitial lung diseases: sarcoidosis, fibrosing alveolitis, extrinsic allergic alveolitis, pneumoconiosis Lymphatic carcinomatosis (may cause intolerable dyspnoea) Large pleural effusion(s)
Others	Metabolic acidosis (e.g. diabetic ketoacidosis, lactic acidosis, uraemia, overdose of salicylates, ethylene glycol poisoning) Hyperventilation	Severe anaemia Obesity

* Denotes a common cause.

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 diminshing 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

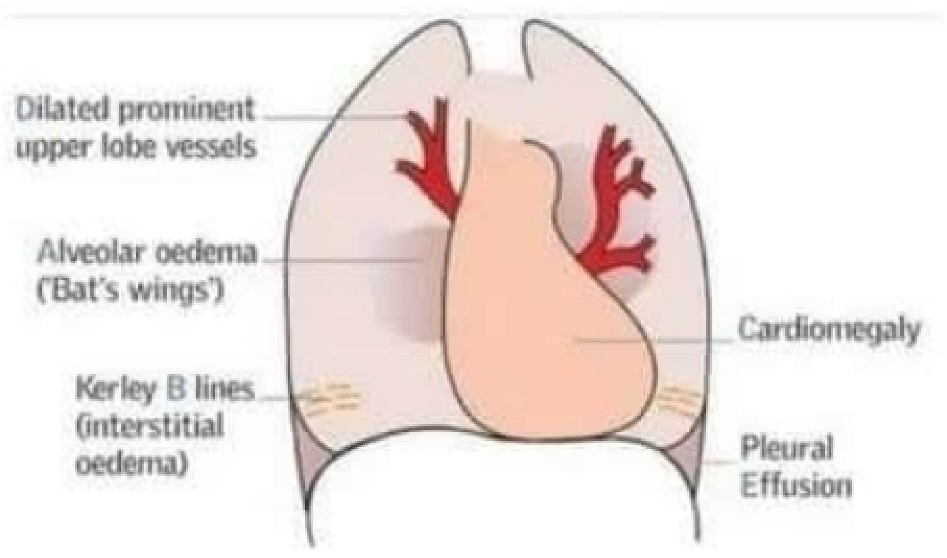
* Troponin I and T elevated in MI

* BNP elevated in heart failure

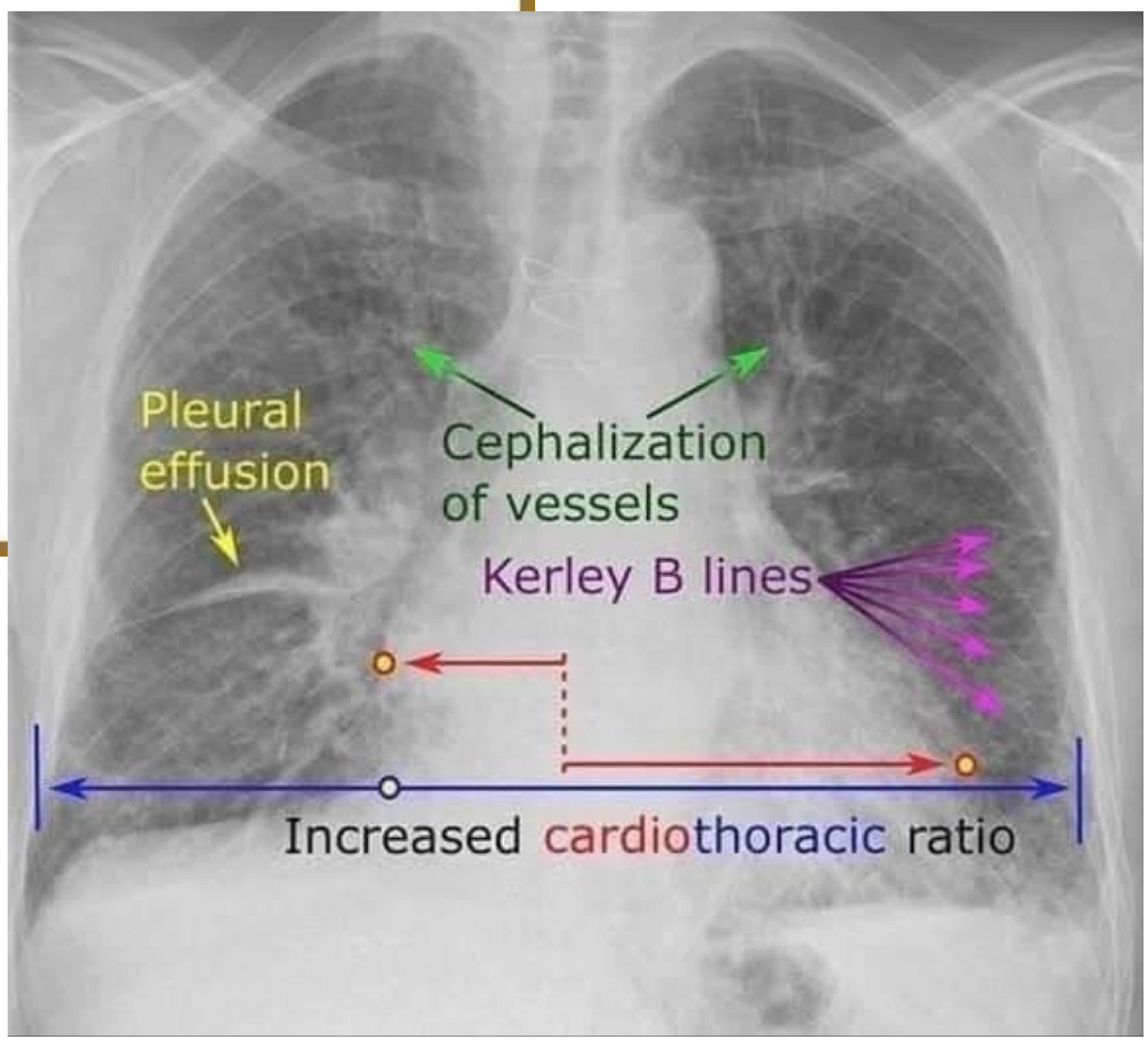
The combined presence of jugular venous distention, S3 gallop, and lung crackles/rales makes a diagnosis of acute heart failure highly likely. [9]

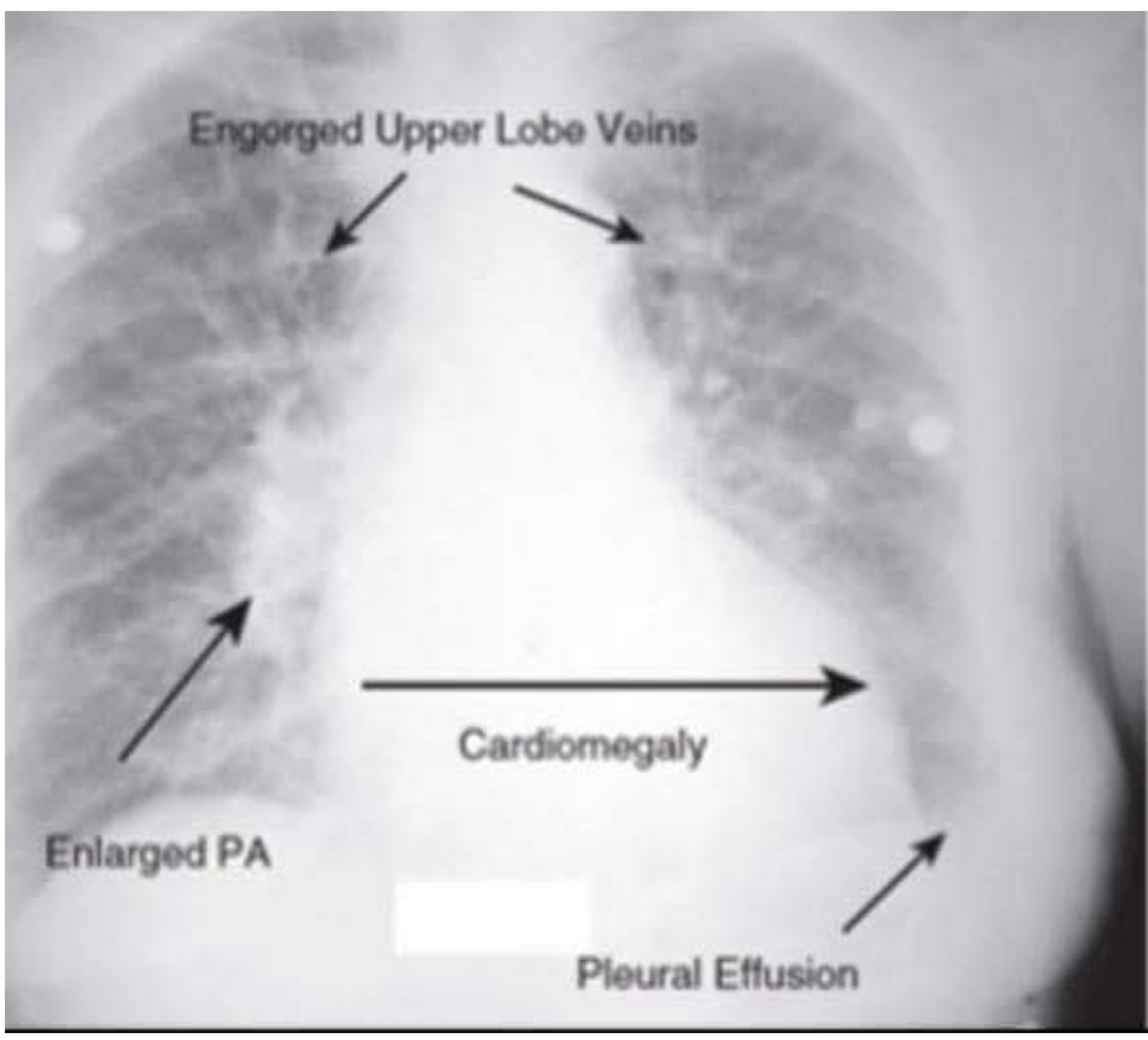
Chest X-ray in Left Ventricular Failure

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

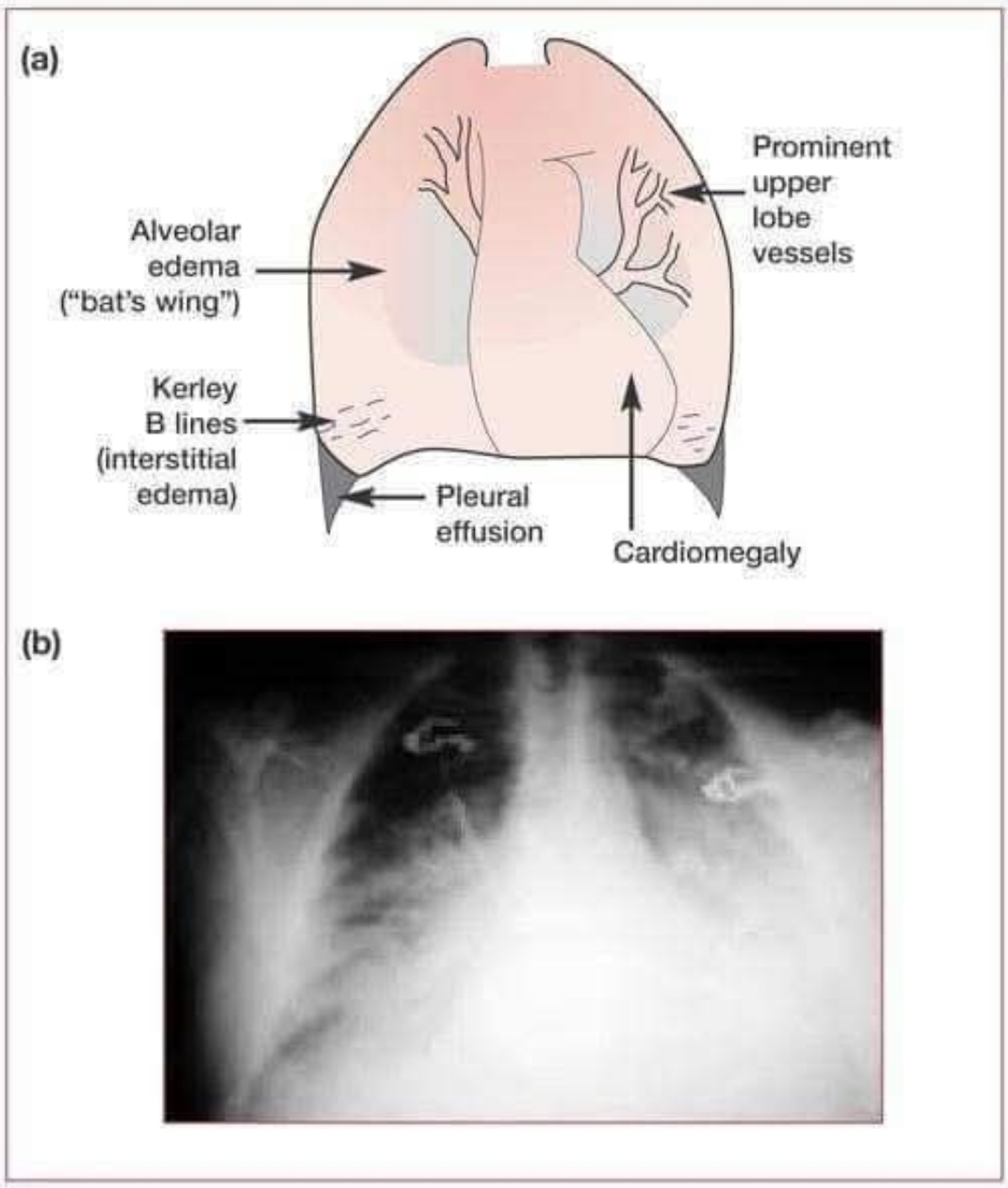


- Abnormal distension of upper lobe pulmonary veins
- vascularity of lung fields become more prominent
- Right and left pulmonary arteries dilate

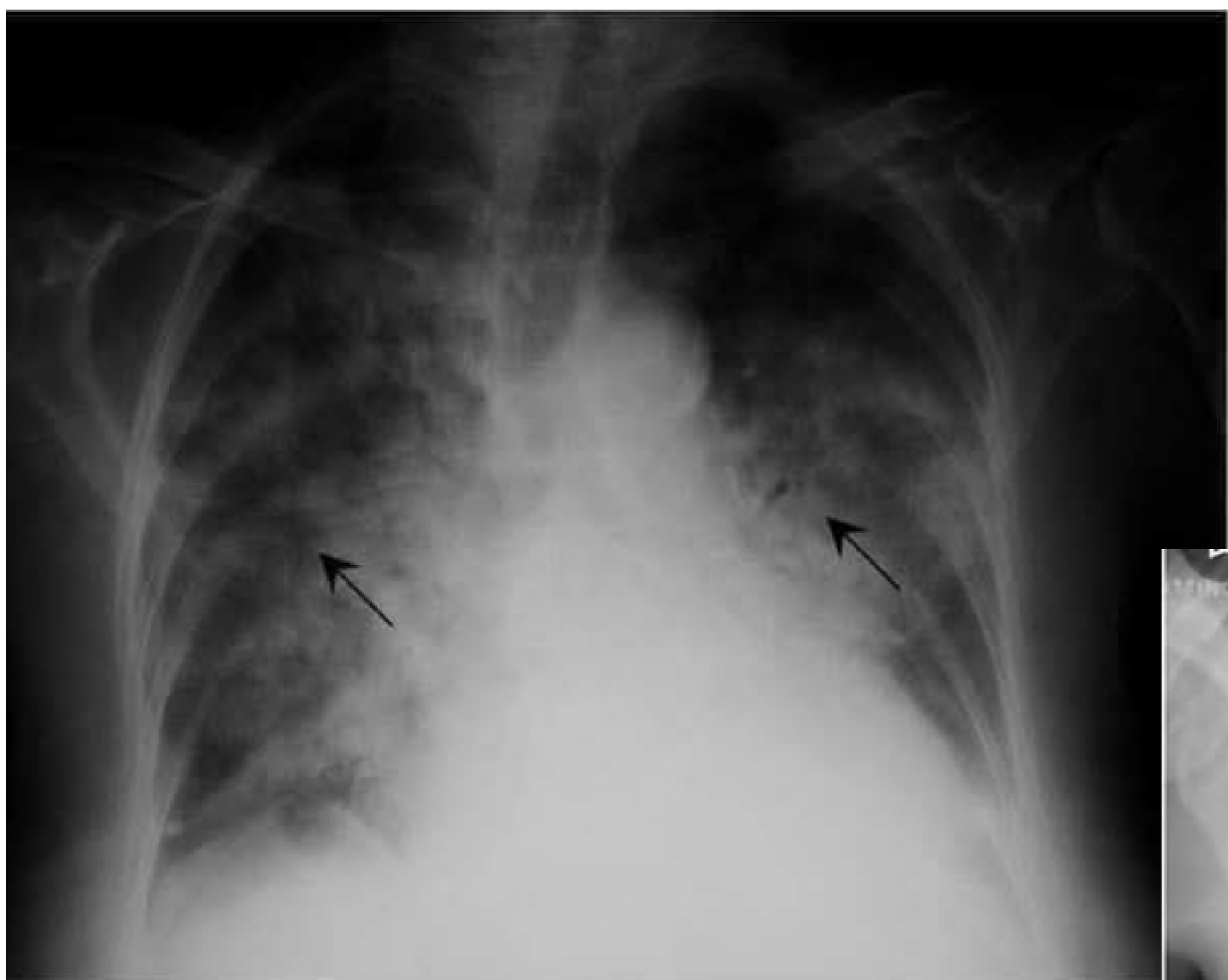




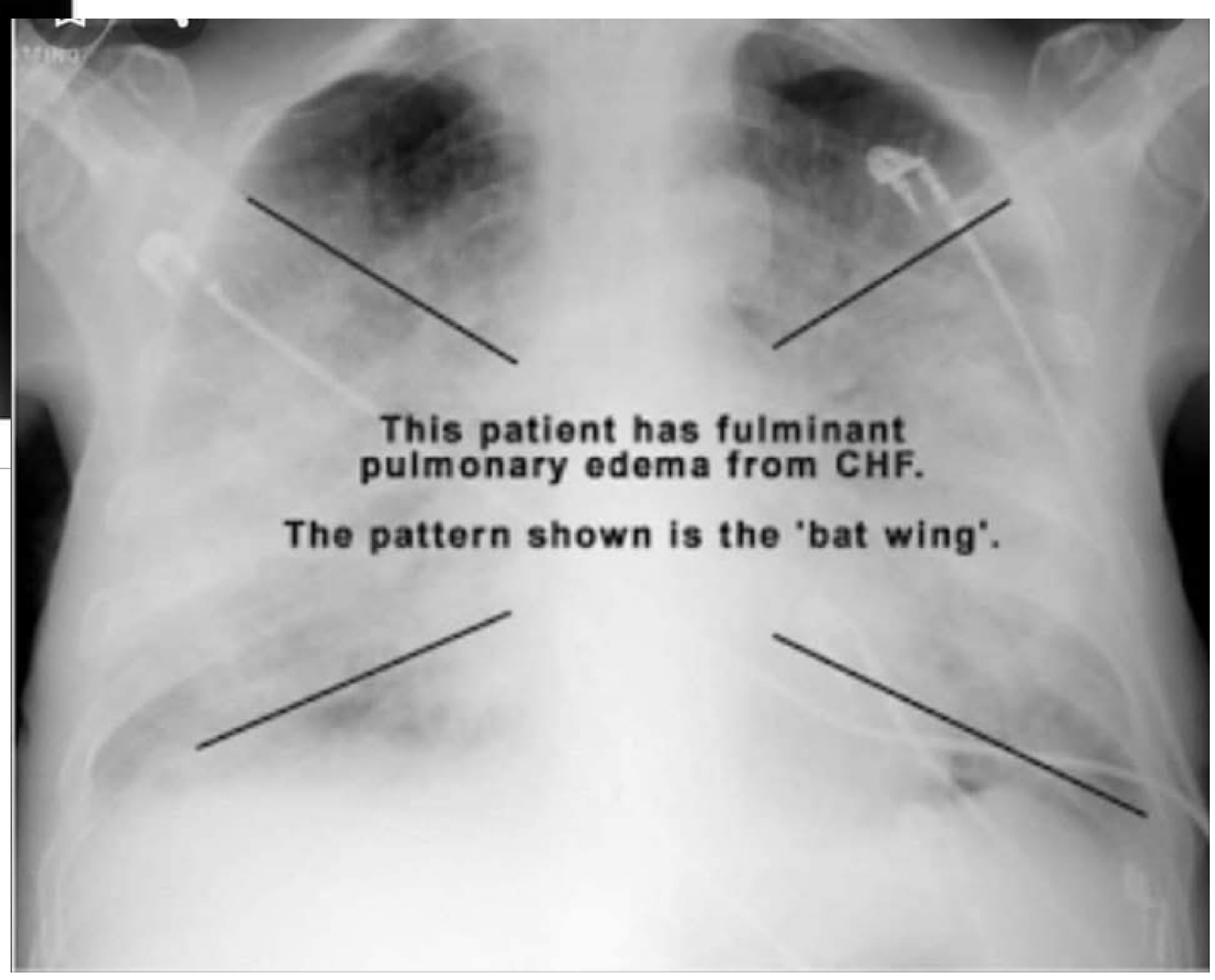
- Kerley B lines are evident horizontal lines in costophrenic angles



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



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



Management of Acute pulmonary edema

- Sit the patient
- Give oxygen
- Administer nitrates
- Administer a loop diuretic eg furosemide

Chronic Heart Failure

- Diuretics → First line
- Vasodilators eg nitrates reduce preload

  Arterial dilators eg hydralazine reduce afterload
- ACE inhibitors

  (If there is initial fall in BP, elevate foot of bed and administer IV saline)
- ARBs → Losartan, Candesartan, Valsartan
- β- blockers
- Digoxin → Heart failure and AFib
- Amiodarone → anti arrhythmic

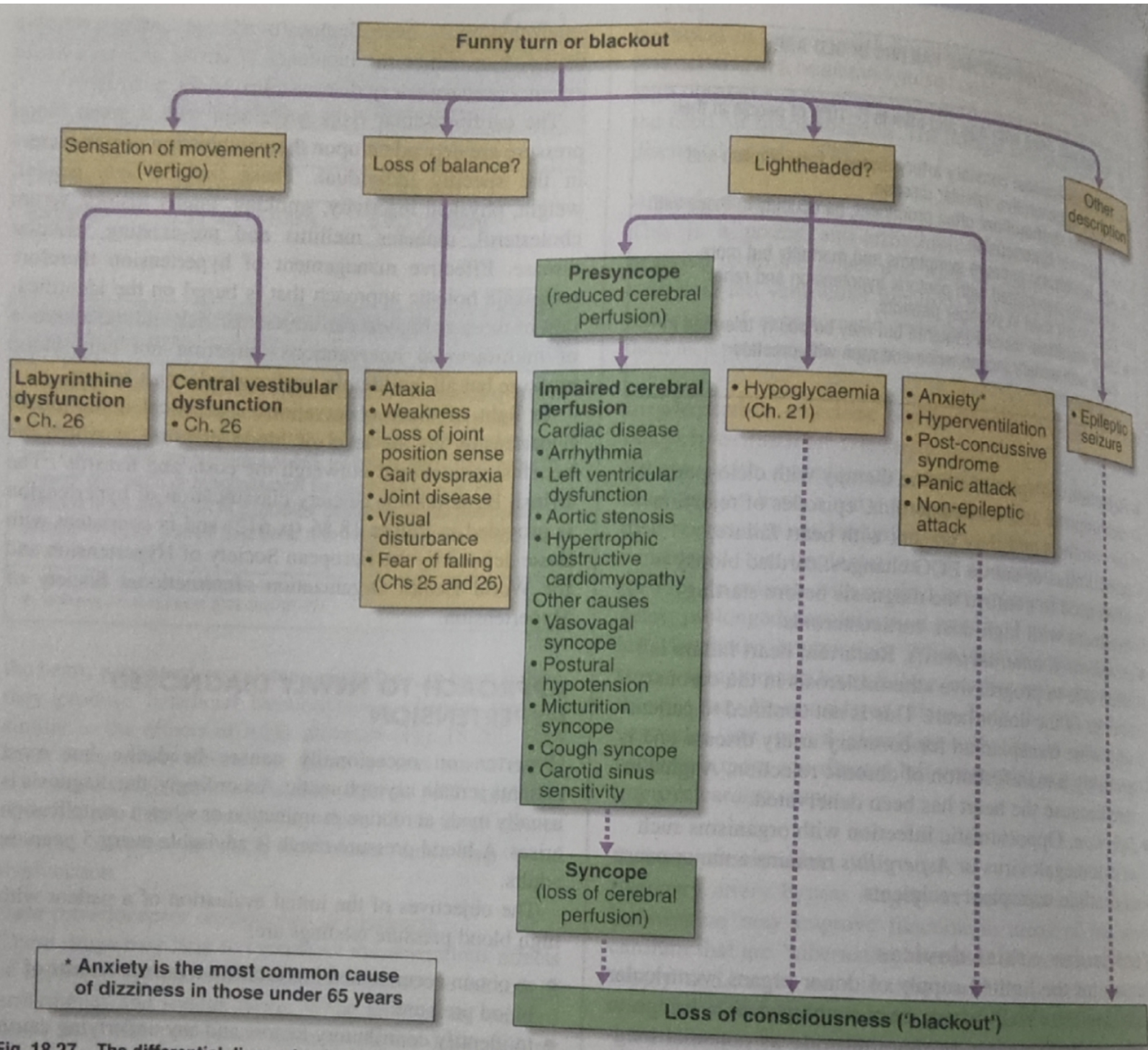


Fig. 18.27 The differential diagnosis of syncope and presyncope.

Palpitation

* 90 - 120/min heartbeat
    - Anxiety
    - Anemia
    - Pregnancy
    - Thyrotoxicosis
    - Some valve disease eg Aortic Regurgitation

* > 120/min
    - Paroxysmal Arrhythmia
    - Atrial, junctional, ventrical tachycardi
    - Atrial Fibrillation

*

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
  Valvular aortic stenosis,
  valvular pulmonary stenosis,
  Floppy mitral valve,
  prosthetic heart sounds

Normal heart sounds [1]

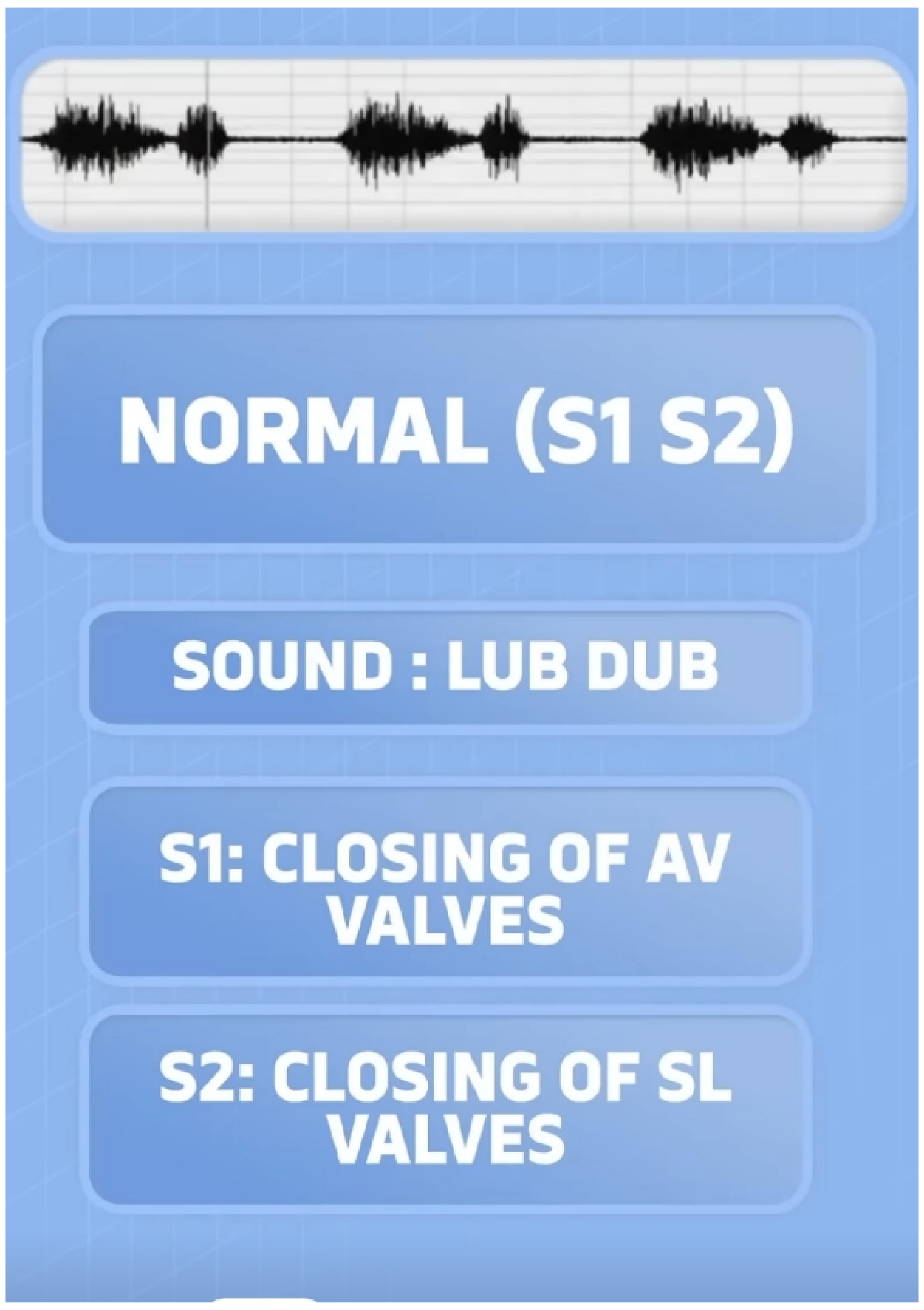
- The first (S1) and second (S2) heart sounds are physiological sounds heard in all healthy individuals.
- The third (S3) and fourth (S4) heart sounds may be physiological (particularly in young adults, pregnant women, and the elderly) or pathological.

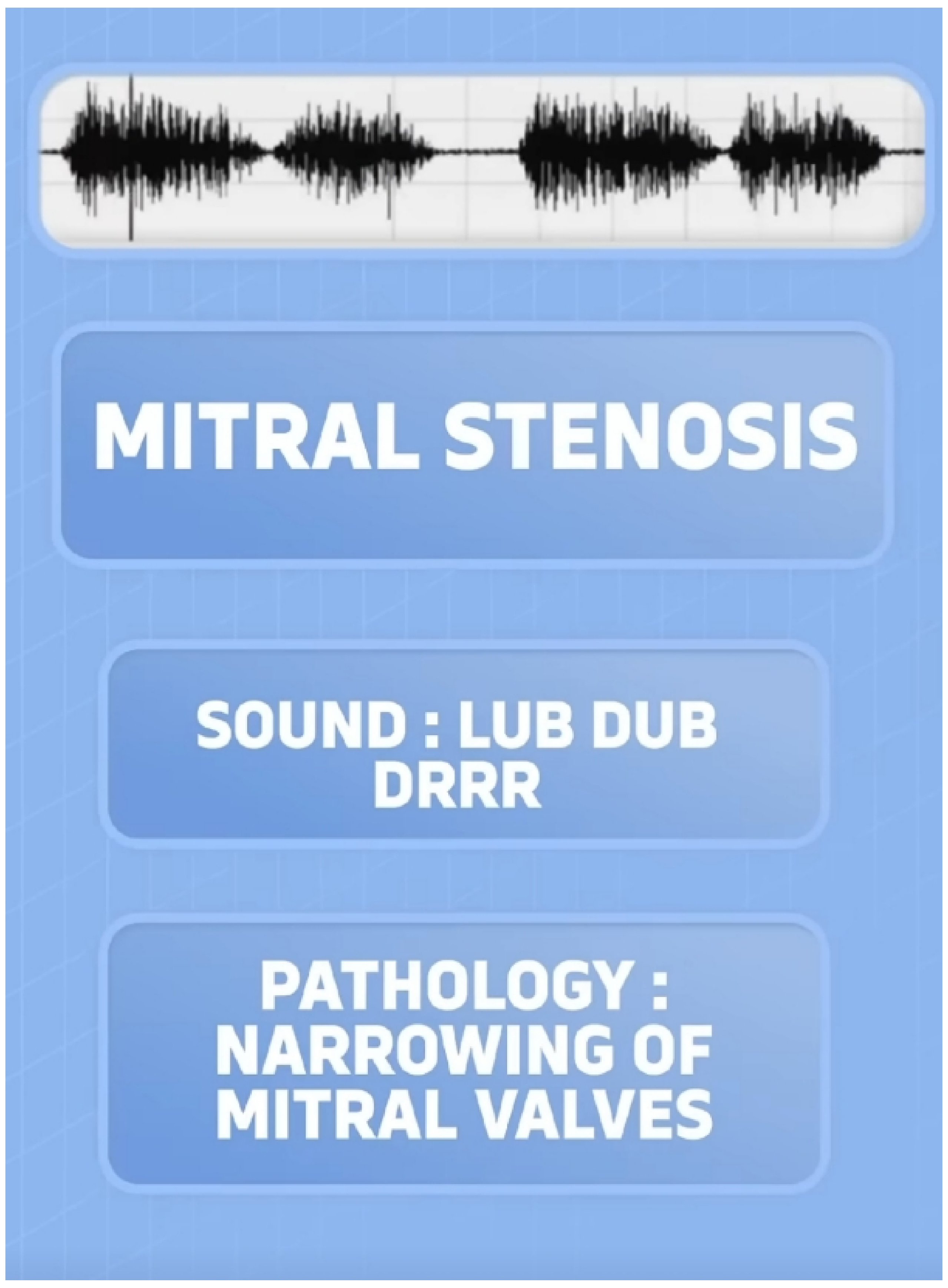
Normal heart sounds

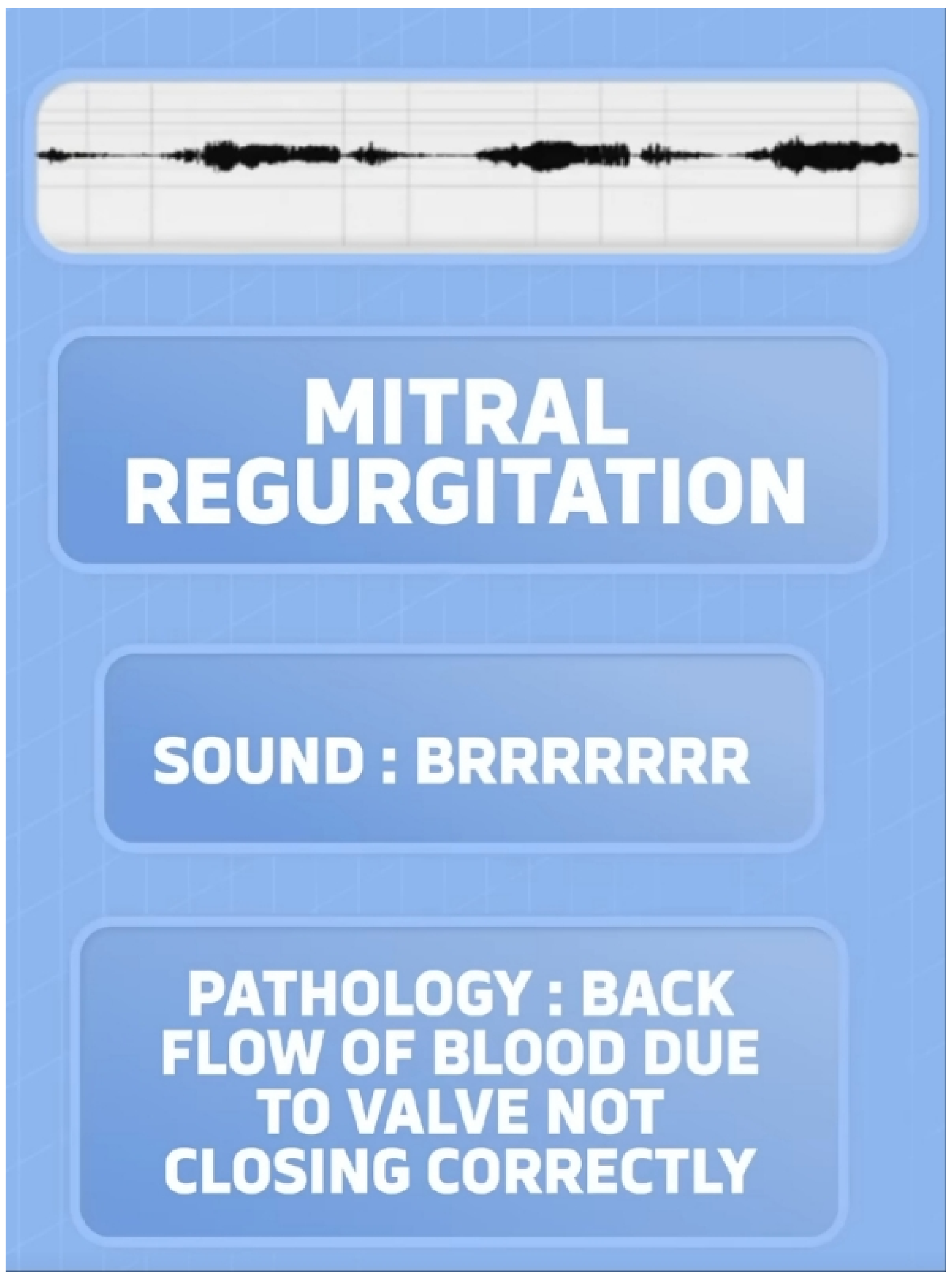
Sound		Origin	Location	Timing
First heart sound (S1)		• **Closure of the mitral valve and tricuspid valve**	• Heard best in the mitral area (cardiac apex)	• At the onset of systole • Heard just before the carotid pulsation is felt
Second heart sound (S2)	**Aortic component of the second heart sound**	• 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	

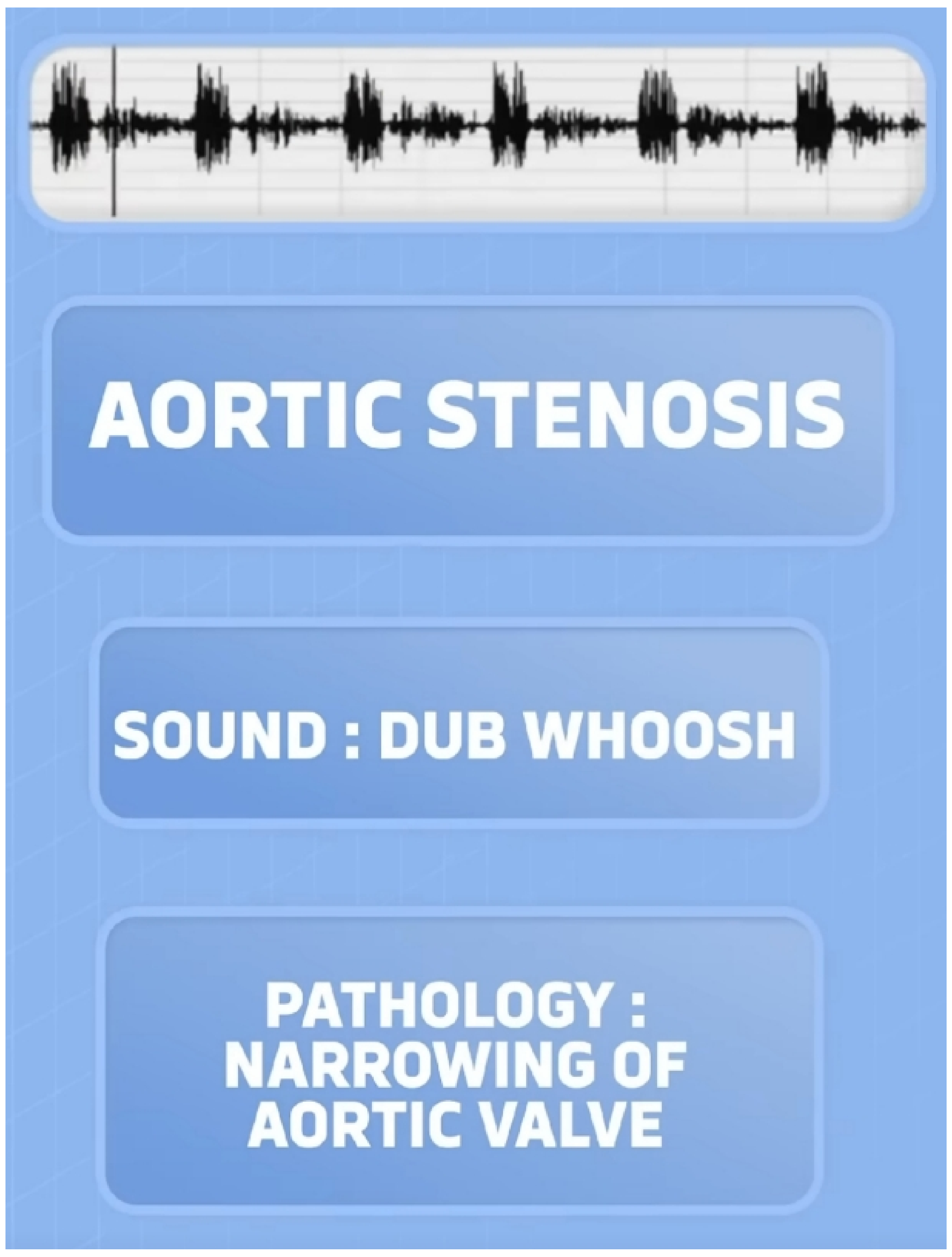
* Opening Snap → high pitch, brief duration, early in diastole

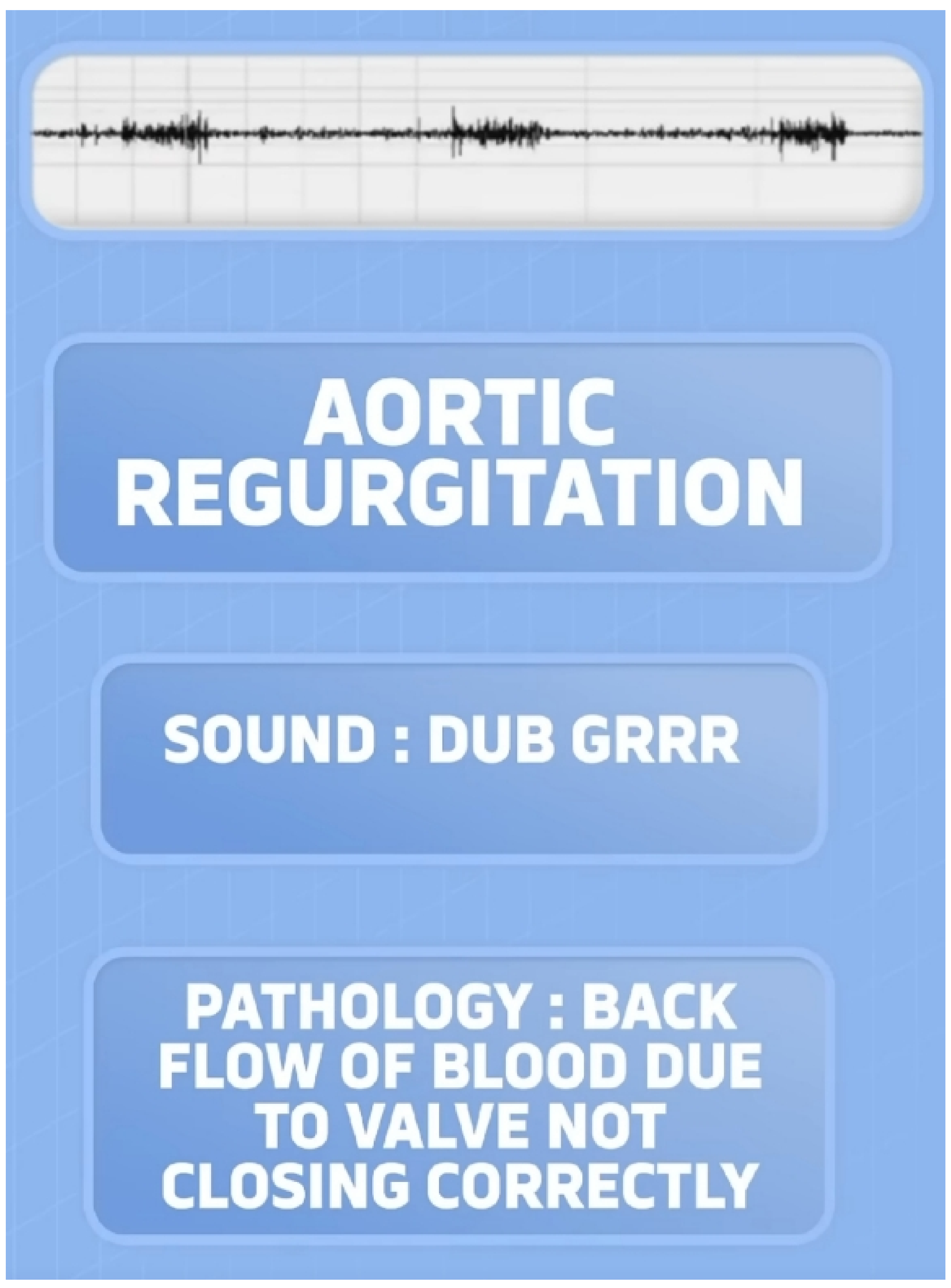
Opening of stenosed leaflets of mitral valve, prosthetic heart sounds











Video link:
https://youtube.com/shorts/e5oaouZ99dw?si=KI2mKV_AkzvP39Ih

- Mitral Regurgitation → blowing murmur
  ↳ Radiation from apex to axilla
- Aortic Stenosis → Rasping murmur
  ↳ Radiation from base to neck

1 Mid-Systolic Murmur

- crescendo decrescendo pattern

* Pan systolic Murmur
  - maintain a constant intensity
  - extend from S_1 throughout systole (upto and beyond S_2)
  - Seen in
    - Mitral Regurgitation
    - Tricuspid Regurgitation
    - Ventricular Septal Defect

* Late Systolic Murmur
  - Mitral Valve prolapse
  - Hypertrophic cardiomyopathy

- Uniform: unchanging intensity
- Crescendo: increasing intensity
- Decrescendo: decreasing intensity
- Crescendo-decrescendo: initial increase followed by a decrease in intensity

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

Neurofibromatosis is an autosomal dominant disorder characterized by neurofibromas, Cafe au lait spots, axillary freckles, there may be associated renal artery stenosis & pheochromocytoma leading to secondary hypertension

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.

Auscultatory Evaluation of a heart murmur

- Timing
- Intensity
- Location
- Radiation
- Quality

Mid Diastolic Murmur

- Mitral Stenosis → located at apex and axilla

- Tricuspid Stenosis → located at left sternal edge

- Increased flow across mitral valve

- ↑ flow across tricuspid valve

Sinus Rhythms

- Symptomatic sinus bradycardia usually responds to IV atropine 0.6–1.2 mg

18.30 SOME PATHOLOGICAL CAUSES OF SINUS BRADYCARDIA AND TACHYCARDIA

Sinus bradycardia <60/min	
• Myocardial infarction • Sinus node disease (sick sinus syndrome) • Hypothermia • Hypothyroidism	• Cholestatic jaundice • Raised intracranial pressure • Drugs, e.g. β-blocker, digoxin, verapamil
Sinus tachycardia >100/min	
• Anxiety • Fever • Anaemia • Heart failure	• Thyrotoxicosis • Phaeochromocytoma • Drugs, e.g. β-adrenoceptor agonists (bronchodilators)

- Atrial Flutter → Digoxin, β blockers or verapamil can be used to control ventricular rate

• Atrial Fibrillation → Irregularly irregular pulse
↳ ECG: normal but irregular QRS complexes, no P waves
↳ palpitation, breathlessness, fatigue

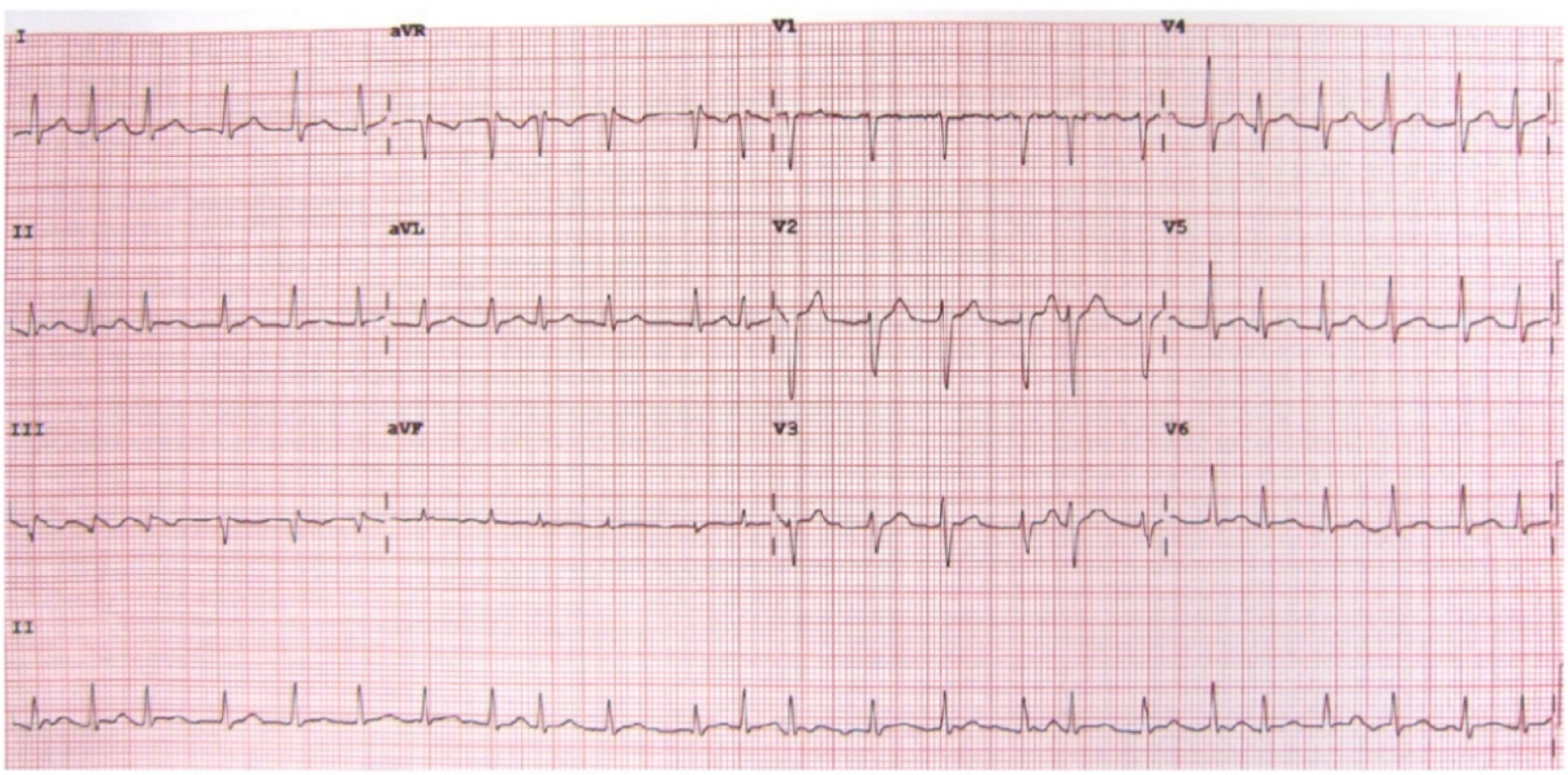
ECG [20]

- Initial investigative study to confirm Afib

Characteristic ECG findings in atrial fibrillation [20]

	Appearance
Rhythm	• Irregularly irregular RR intervals • Rarely may be regular if there is complete AV dissociation
Rate	• Variable; tachycardia is common. • Atrial rate > ventricular rate
P waves	• P waves are indiscernible. • Fibrillatory waves (f waves) are seen instead at a frequency of 300–600/minute • PR intervals: not distinguishable

QRS complex	• Typically narrow QRS complex (< 0.12 seconds)
	• Wide QRS complex may be seen in some situations:
	  ◦ Aberrant conduction, e.g., bundle branch block or preexcitation (as seen in Afib with WPW)
	  ◦ Complete AV block with a ventricular escape rhythm



Major Risk Factors for Acute AFib

Mnemonic : PARASITE

Pulmonary

Anemia

Rheumatic Heart Disease

Atrial Myxoma

Sepsis

Ischemia

Thyroid Disease

Ethanol

Clinical features of left-sided heart failure

- **Symptoms of pulmonary congestion**
  - Dyspnea , orthopnea (a sensation of shortness of breath that occurs upon lying down and is relieved by sitting up)
  - Pulmonary edema
  - **Paroxysmal nocturnal dyspnea**
    - Nocturnal bouts of coughing and acute shortness of breath
    - Caused by reabsorption of peripheral edema at night → increased venous return
  - **Cardiac asthma**
    - Increased pressure in the bronchial arteries → airway compression and bronchospasm
    - Symptoms mimic asthma, with shortness of breath, wheezing, and coughing. [16]
- 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

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

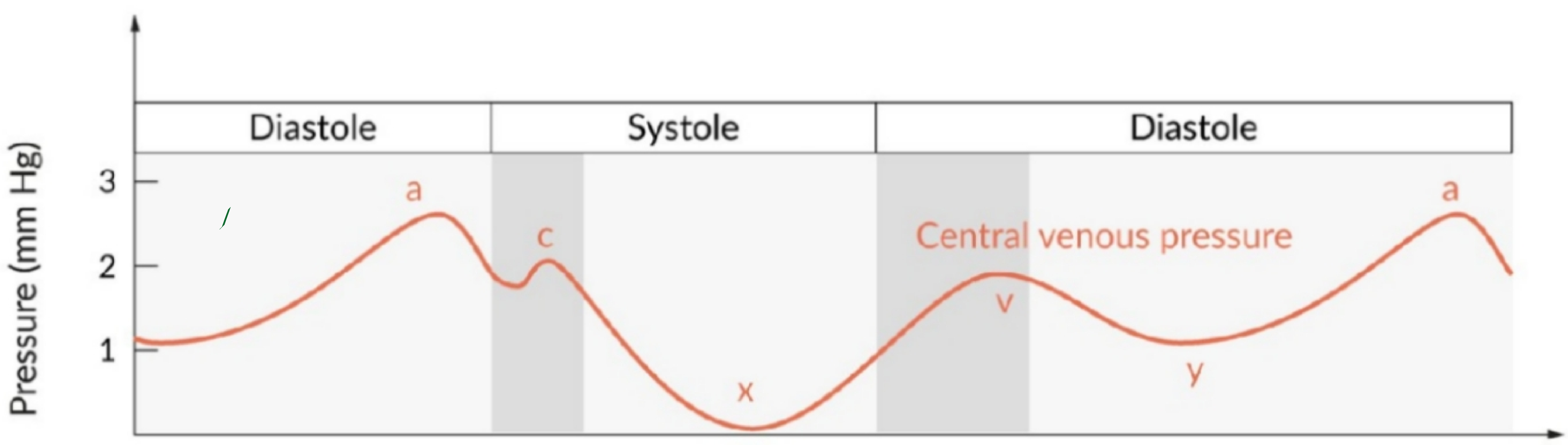
Janeway lesions - Multiple erythematous macules are visible on the thenar eminence and the base of the thumb. These Janeway lesions are typically caused by septic microembolisms secondary to infective endocarditis.

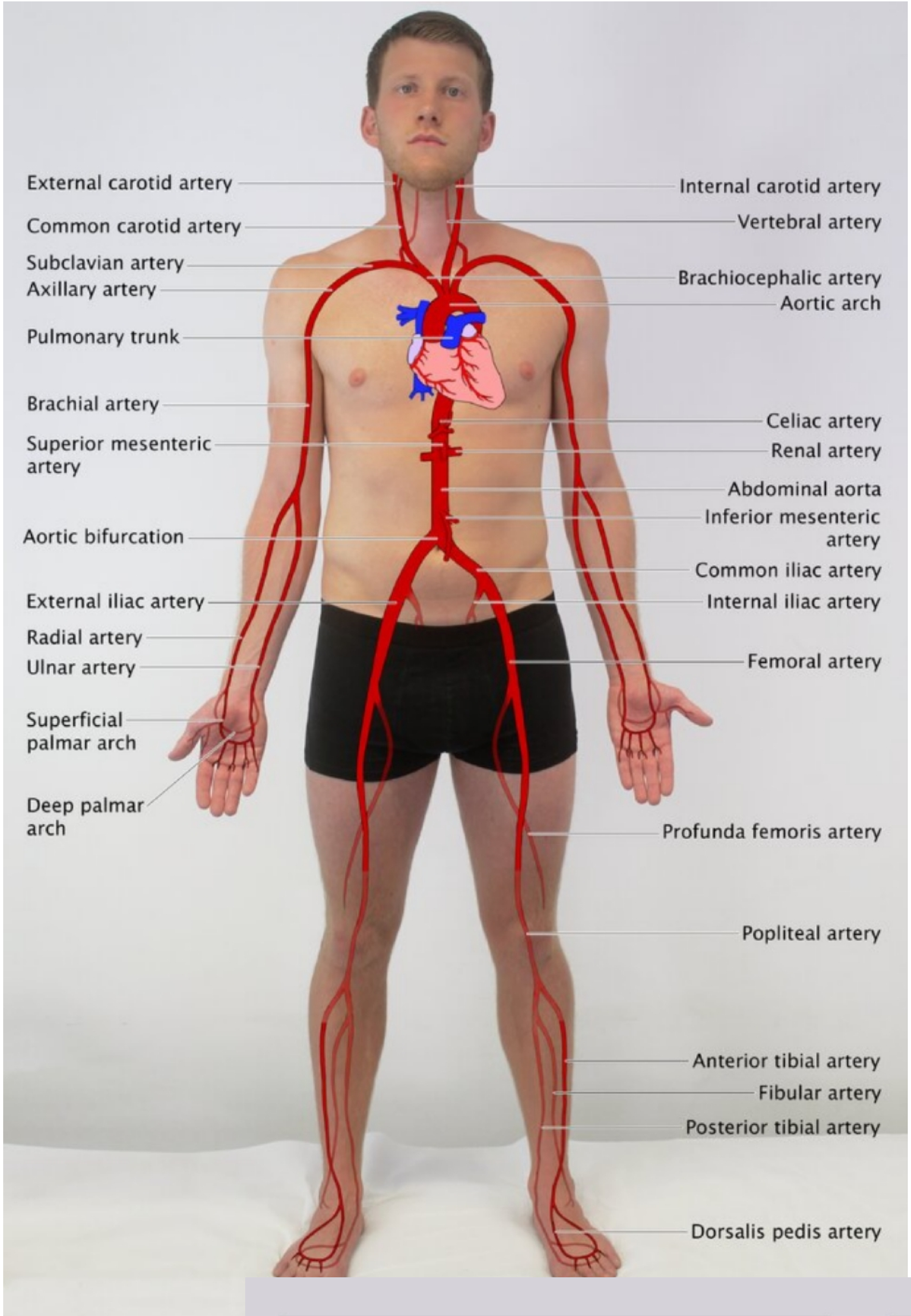
Subcutaneous Nodules

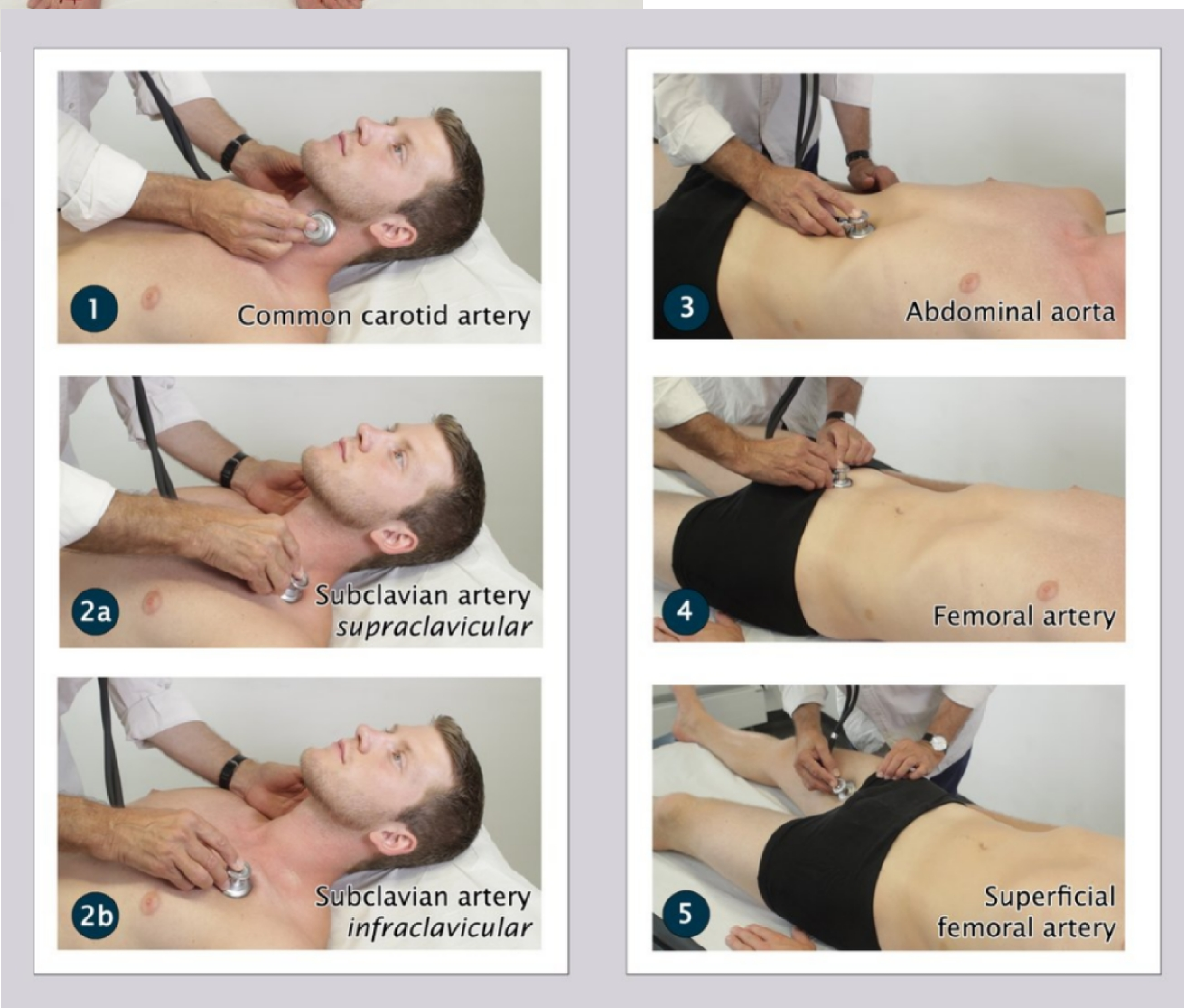
Subcutaneous nodules in rheumatic fever - Solitary subcutaneous, erythematous, warm and tender protuberances are visible on the left shoulder. In conjunction with flu-like symptoms, ankle swelling, and an elevated antistreptolysin-O (ASO) titer, these skin findings indicate rheumatic fever.

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



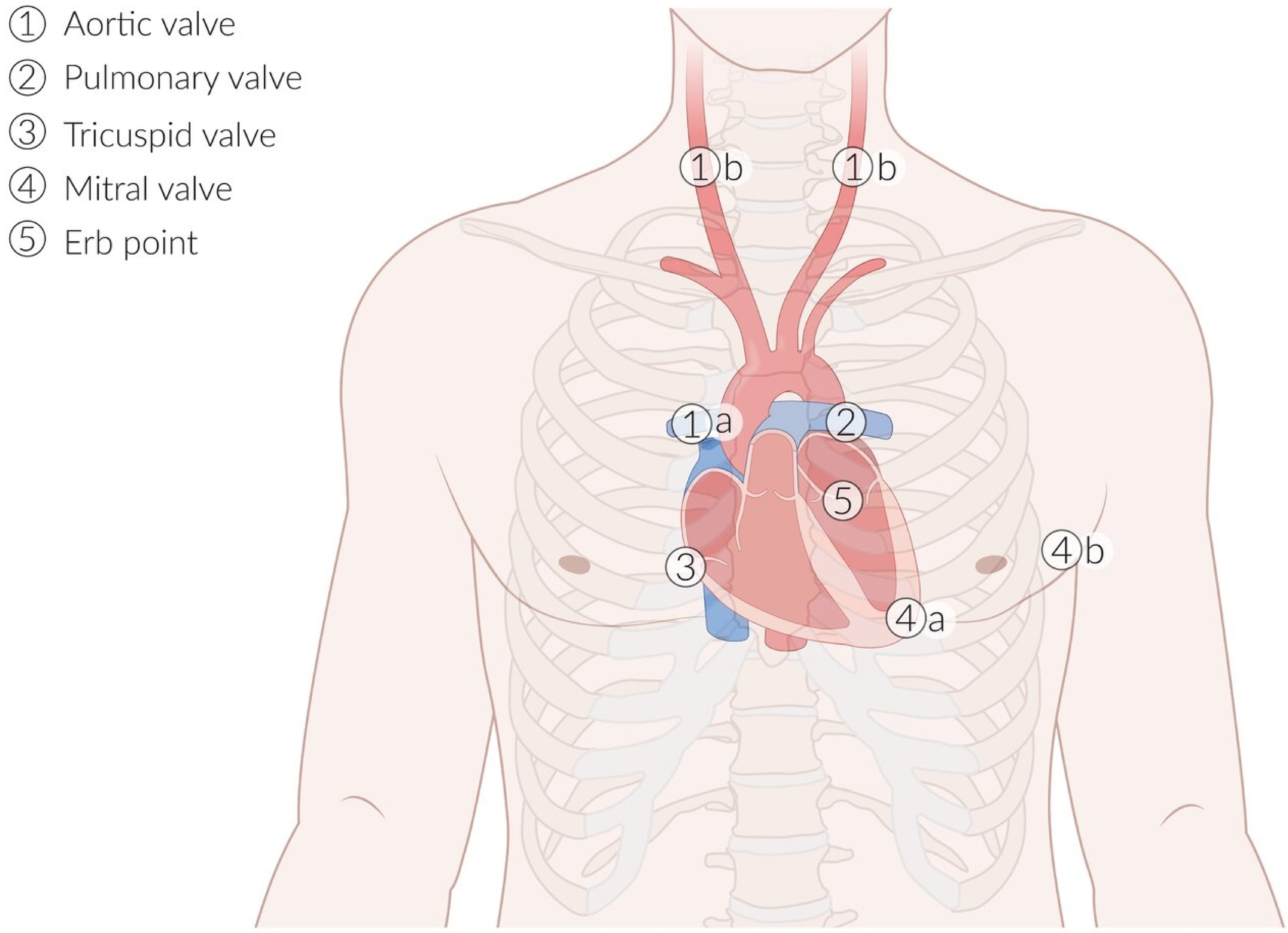

External carotid artery
Common carotid artery
Subclavian artery
Axillary artery
Pulmonary trunk
Brachial artery
Superior mesenteric artery
Aortic bifurcation
External iliac artery
Radial artery
Ulnar artery
Superficial palmar arch
Deep palmar arch
Internal carotid artery
Vertebral artery
Brachiocephalic artery
Aortic arch
Celiac artery
Renal artery
Abdominal aorta
Inferior mesenteric artery
Common iliac artery
Internal iliac artery
Femoral artery
Profunda femoris artery
Popliteal artery
Anterior tibial artery
Fibular artery
Posterior tibial artery
Dorsalis pedis artery


1 Common carotid artery
2a Subclavian artery supraclavicular
2b Subclavian artery infraclavicular
3 Abdominal aorta
4 Femoral artery
5 Superficial femoral artery

Auscultatory locations [1]

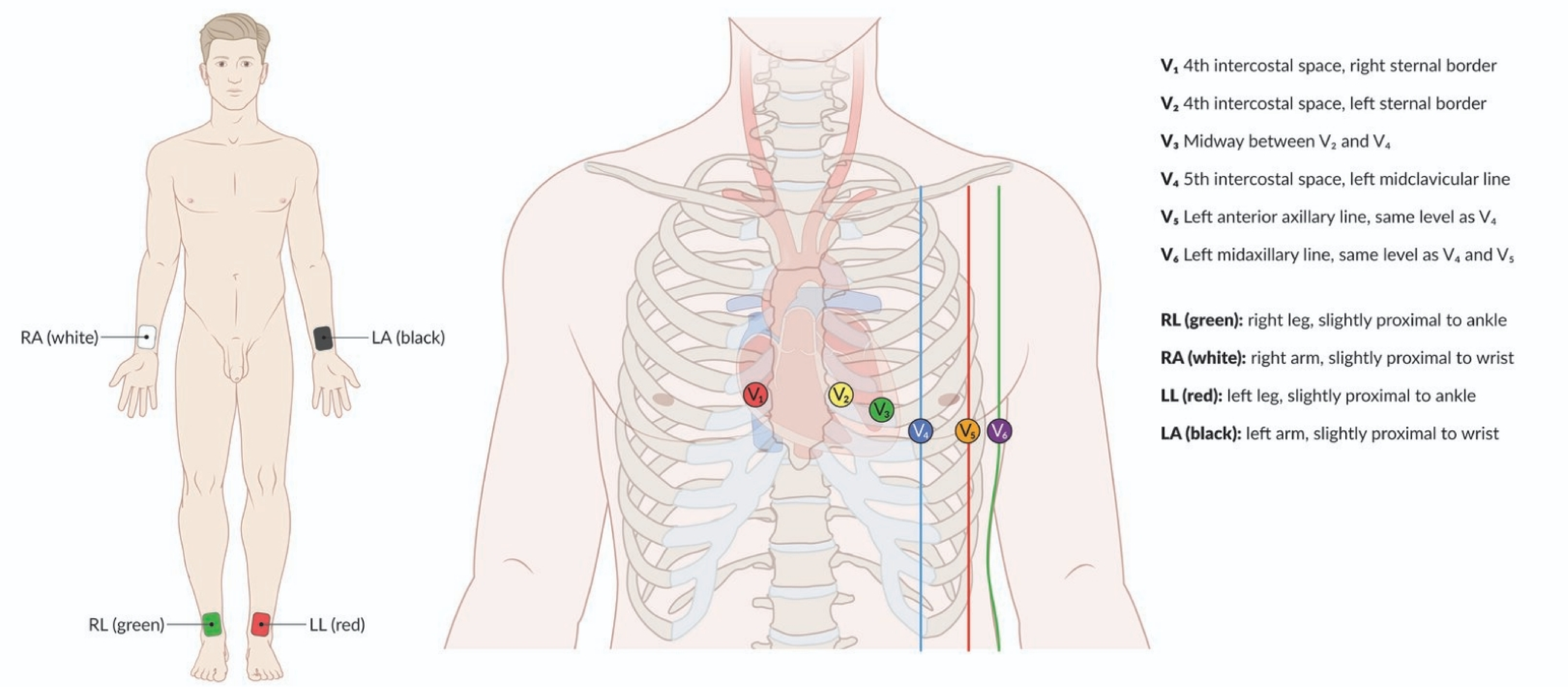
Heart sound auscultation sites		
Name of area	**Location**	**Pathology**
Erb point (cardiology)	• 3rd left parasternal intercostal space	• Diastolic murmurs: aortic regurgitation, pulmonic regurgitation • Systolic murmurs: HOCM
Aortic area	• 2nd right parasternal intercostal space	• Aortic stenosis • 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)

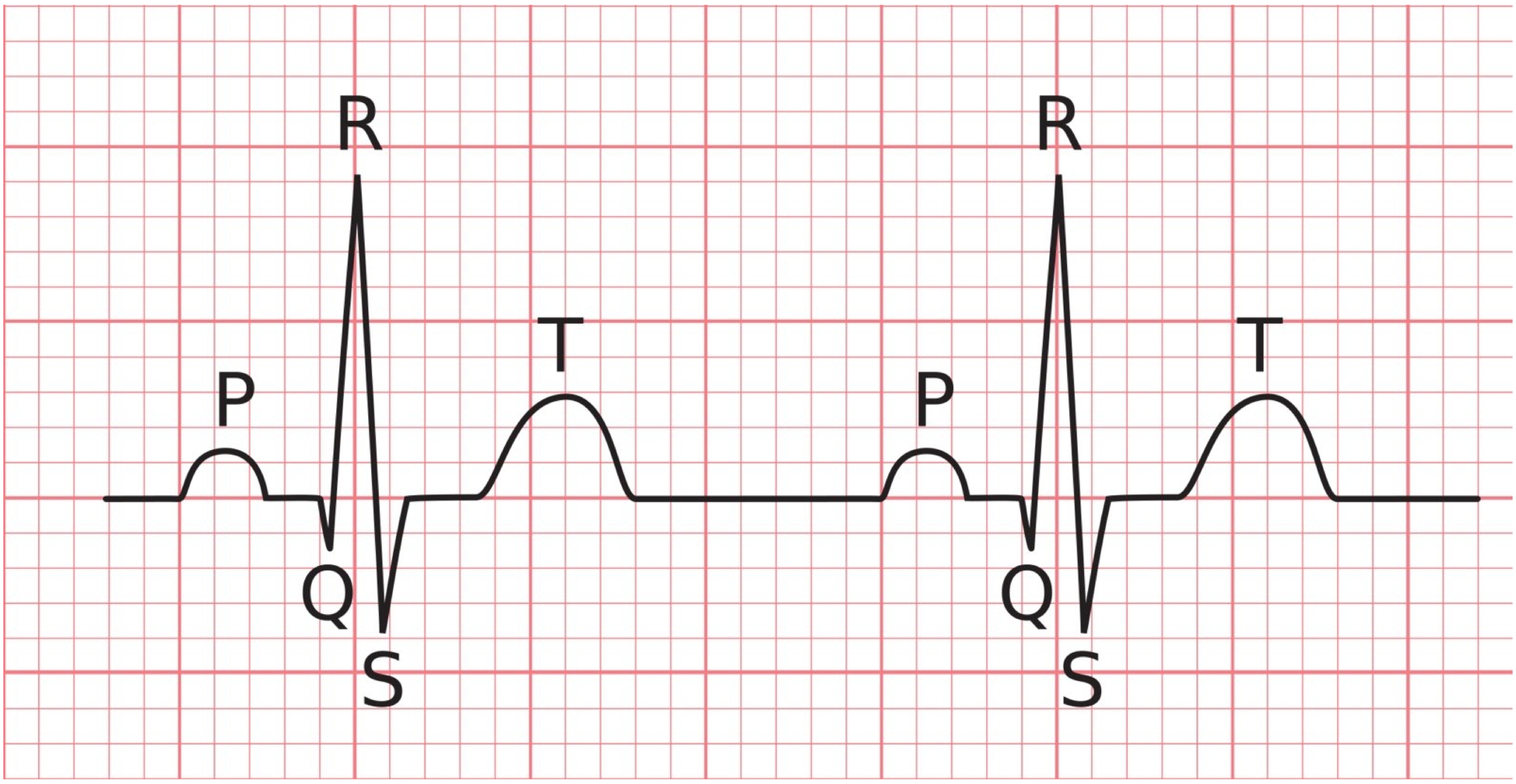


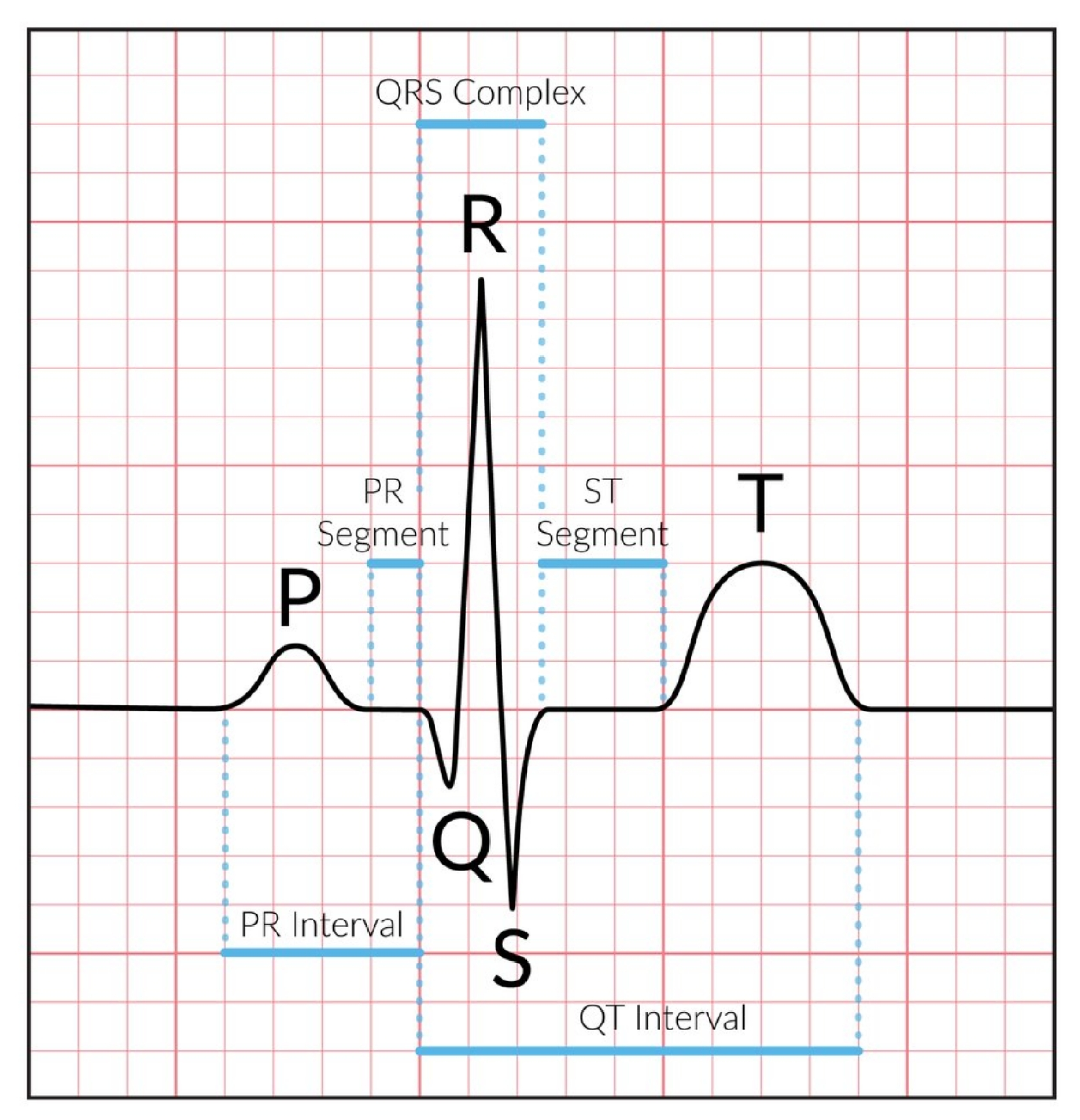
ECG electrode placement [1][2]

- **Four limb electrodes**
  - Left arm
  - Right arm
  - Left leg
  - Right leg (neutral electrode)
- **Six chest electrodes**
  - V_1: fourth intercostal space, right parasternal region
  - V_2: fourth intercostal space, left parasternal region
  - V_3: midway between V_2 and V_4
  - V_4: fifth intercostal space, left midclavicular line
  - V_5: fifth intercostal space, left anterior axillary line
  - V_6: fifth intercostal space, left midaxillary line

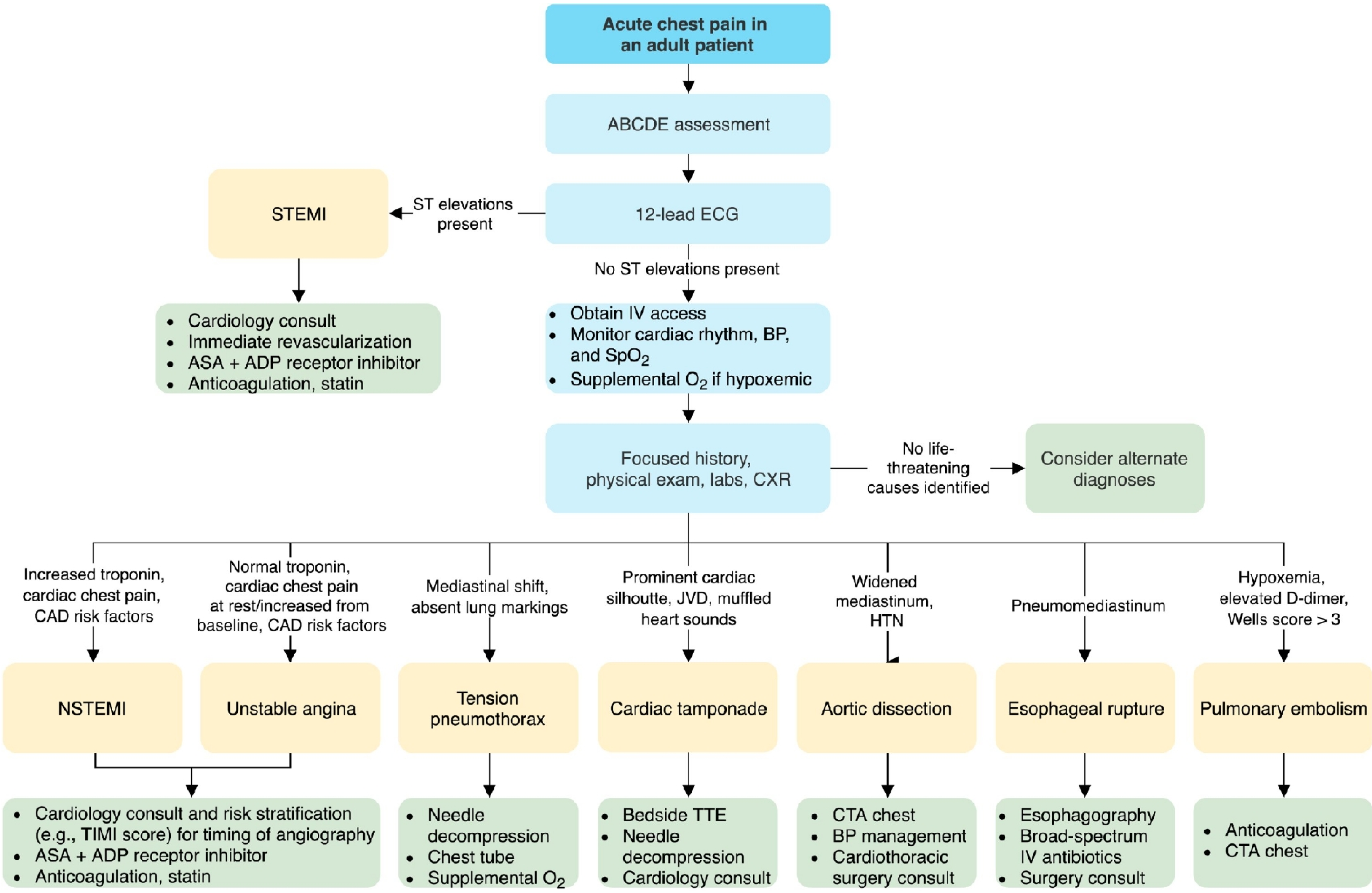


Anatomical relationships of ECG leads			
	Limb leads	**Precordial leads**	**Corresponding heart structure**
Inferior leads	• II • III • aVF	• N/A	• Inferior surface of the heart
Lateral leads	• I • aVL	• V_5 • V_6	• Left ventricle, lateral wall
Anteroseptal leads	• N/A	• V_1–V_4	• Anterior wall of both 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


R
P
T
Q
S
R
P
T
Q
S


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

Approach to Chest Pain



Routine investigations

- Troponin [6]
- CBC
- BMP

Additional investigations

- BNP or NT-proBNP
- D-dimer
- Lactate
- Preoperative studies: PT, PTT, type and screen
- Abdominal studies: LFTs , lipase, amylase
- Acute phase reactants: ESR, CRP , procalcitonin
- Cultures: blood cultures, sputum cultures
- Acute viral hepatitis panel
- Respiratory viral panel
- Urine toxicology screen

Causes	Characteristic clinical features	Diagnostic findings
STEMI [7]	• Heavy, dull, pressure/squeezing sensation • Substernal pain with radiation to left shoulder • Nausea, vomiting • Diaphoresis, anxiety • Dizziness, lightheadedness, syncope • Pain may improve with nitroglycerin.	• Labs: ↑ Troponin • ECG: **ST-segment elevation/depression**, T-wave inversions, Q waves • TTE: hypokinesis, regional wall motion abnormalities
NSTEMI/UA [8]		• Labs: Increased or normal troponin • ECG: nonspecific changes, including T-wave inversions, ST-depressions • TTE: Regional wall motion abnormalities may be present.
Aortic dissection [9][10][11]	• Sudden onset of severe, sharp tearing chest or abdominal pain that radiates to the back • Hypertension or hypotension • Asymmetrical blood pressure, pulse deficit • New diastolic murmur • Symptoms of myocardial ischemia • Syncope, neurological symptoms	• Labs: Elevated D-dimer • ECG: nonspecific ST-segment changes • CXR: widening of the aorta • CTA chest, abdomen, and pelvis: intimal flap with false lumen • TEE: proximal aortic dissection, tamponade, aortic regurgitation

Monckeberg
Arteriosclerosis

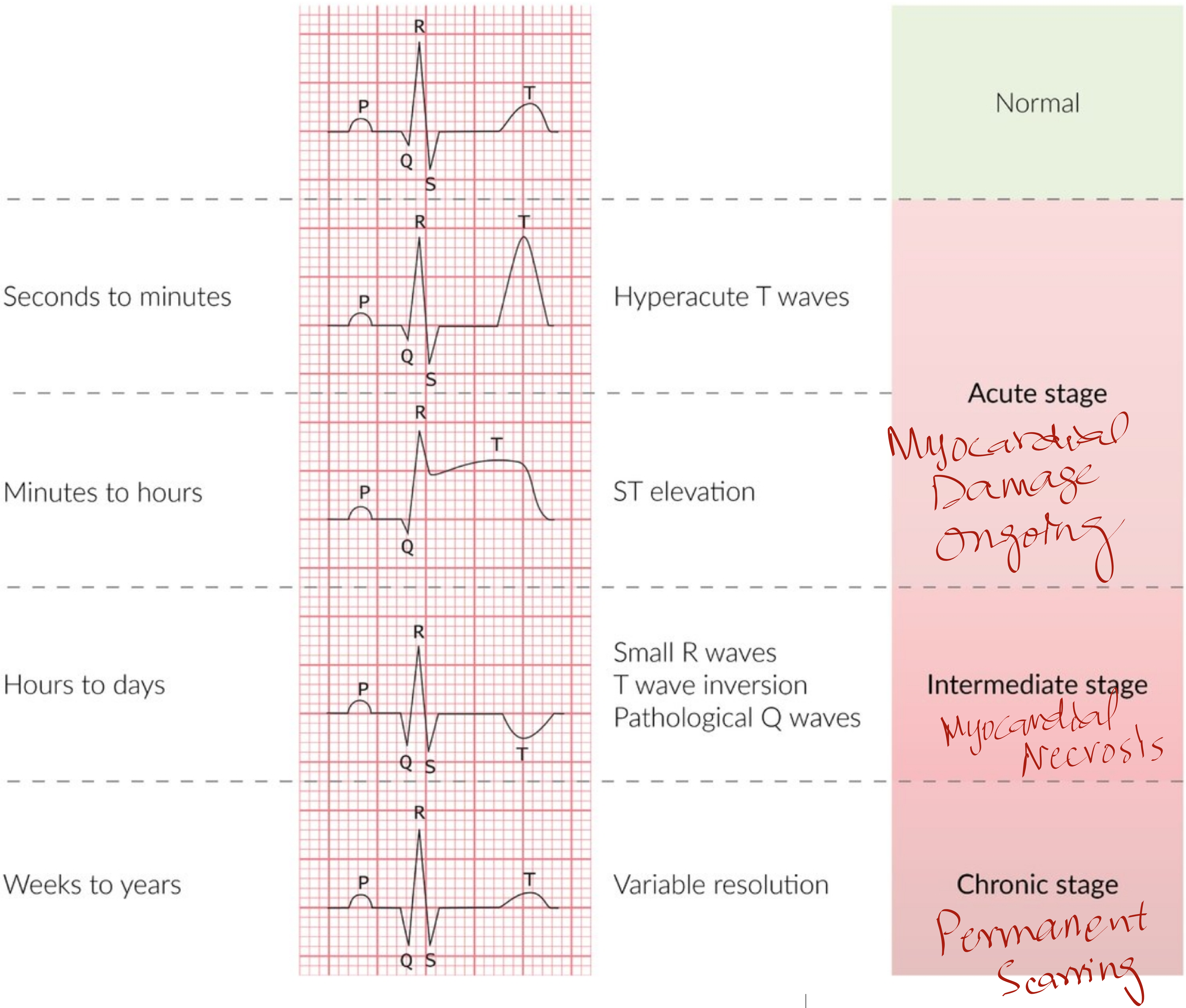
→ Pipe stem appearance
on X-Ray

- **Monckeberg arteriosclerosis**
  - A form of arteriosclerosis characterized by **dystrophic calcification** of the **tunica media** and internal elastic lamina
  - There is no blood flow obstruction.
  - Mainly affects medium-sized arteries
  - Causes: diabetes mellitus and/or progressive kidney disease
  - X-ray: pipestem appearance

Atherosclerotic Cardiovascular Disease (ASCVD)

Remember the ABCDS of ASCVD primary prevention**: A**spirin (if there are indications), **B**lood pressure control, **C**holesterol management, **D**iabetes management, **S**moking cessation. [16]

STEMI



Classical timeline of ECG changes in STEMI

- **Acute stage**: myocardial damage ongoing
  - Hyperacute T waves (**peaked T wave**)
  - **ST elevations** in two contiguous leads with reciprocal ST depressions
- **Intermediate stage:** myocardial necrosis present
  - Absence of R wave
  - T-wave inversions
  - Pathological Q waves
    - Duration ≥ 0.04 seconds
    - Amplitude ≥ ¼ of the R wave or ≥ 0.1 mV
    - Any Q wave in leads V1–3
- **Chronic stage**: permanent scarring
  - Persistent, broad, and deep **Q waves**
  - Often incomplete recovery of R waves
  - Permanent T-wave inversion is possible.

The sequence of ECG changes over several hours to days: hyperacute T wave → ST elevation → pathological Q wave → T-wave inversion → ST normalization → T-wave normalization

'Time is Muscle'

Revascularisation should occur as soon as possible in STEMI

Options for initial MI treatment include “**MONA-BASH**”: **M**orphine, **O**xygen, **N**itroglycerin, **A**ntiplatelet drugs (aspirin + ADP receptor inhibitor), **B**eta blockers, **A**CE inhibitors, **S**tatins, and **H**eparin. The scope of interventions depends on the patient's risk profile (see “Indications”).

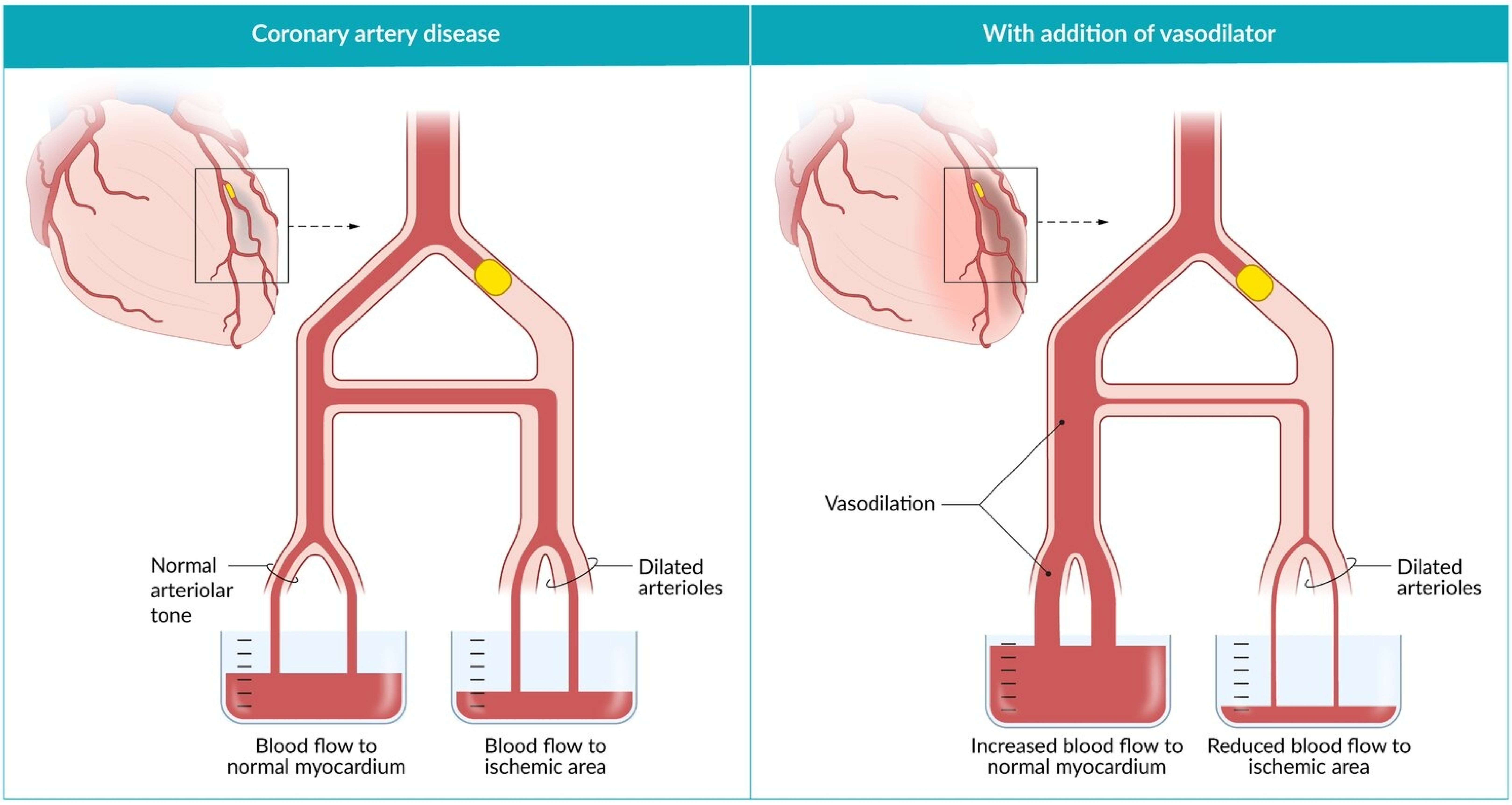
Differential diagnoses of increased troponin

- **Cardiovascular causes**
  - Myocarditis
  - Decompensated congestive heart failure
  - Pulmonary embolism
  - Cardiac arrhythmia, tachycardia
  - Aortic dissection
  - Hypertensive emergencies
  - Structural heart disease
  - Myocardial drug toxicity (e.g., doxorubicin)
  - Cardiac trauma (including iatrogenic/periprocedural)
  - Takotsubo cardiomyopathy
  - Stroke
- **Noncardiovascular causes**
  - Renal failure
  - Critical illness (e.g., sepsis)
  - Hypothyroidism or hyperthyroidism

- **Typical angina** fulfills all of the following criteria:
  - Retrosternal chest pain of characteristic nature and duration (e.g., transient retrosternal pressure)
  - Provoked by exertion or emotional stress
  - Relieved by rest and/or nitroglycerin
- **Atypical angina**: fulfills only two of the aforementioned criteria
- **Nonanginal chest pain**: fulfills one or none of the aforementioned criteria

Coronary steal syndrome

- **Definition:** a phenomenon of vasodilator-induced alteration of coronary blood flow in patients with coronary atherosclerosis resulting in myocardial ischemia and symptoms of angina



  - **Administration of vasodilators (e.g., dipyridamole)** → coronary vasodilation → decreased hydrostatic pressure in the normal coronary arteries → **blood shunting back to well-perfused myocardium** → decreased flow to the ischemic myocardium → myocardial ischemia downstream to the pathologically dilated vessels → **angina pectoris** and/or ECG changes

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

◀ Differential diagnosis of chest pain in ER

★Life threatening causes of CP:

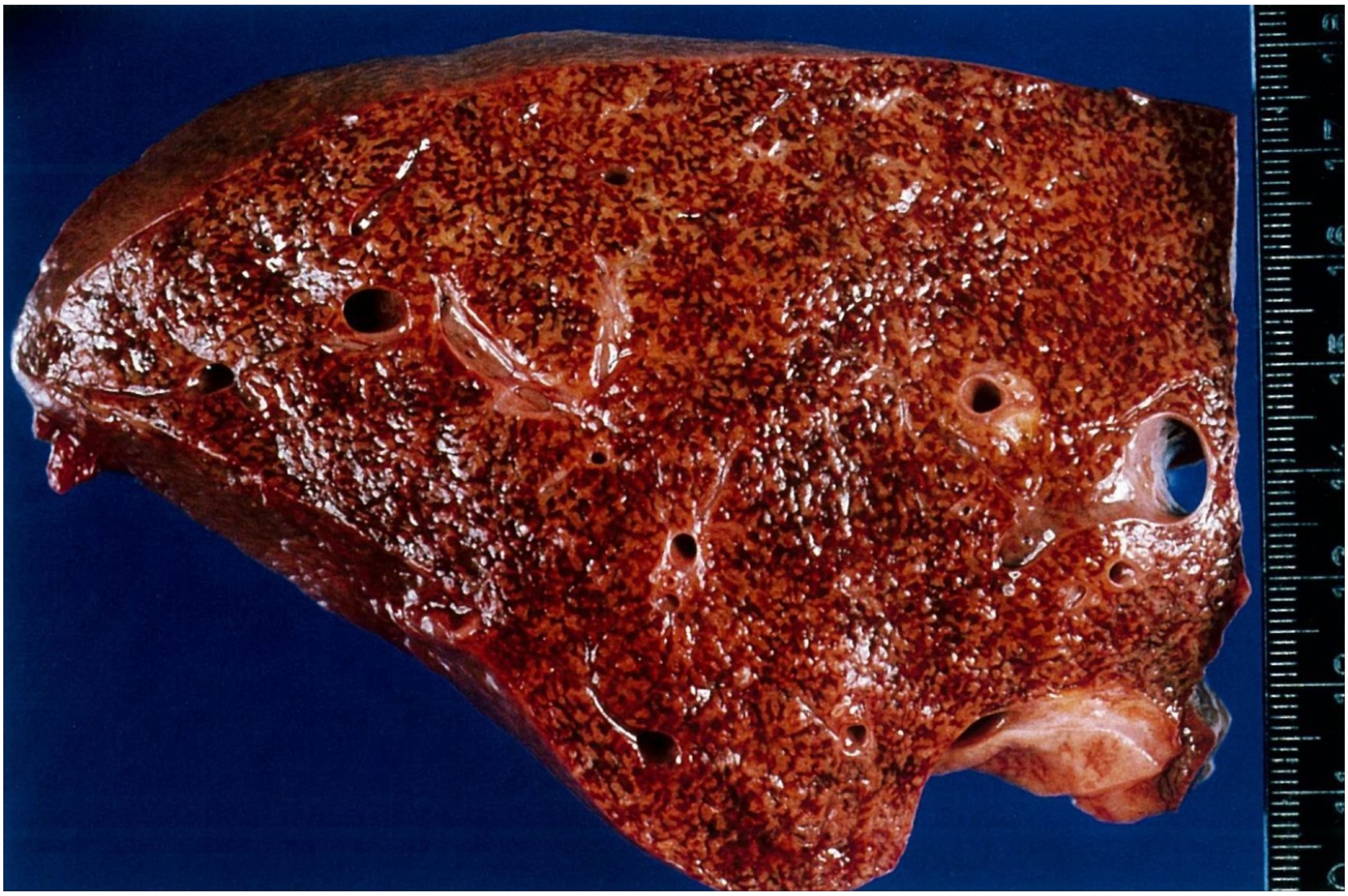
You have to know how to differentiate between them
Click here for a quick summary of these conditions

PPPPAA

Cardiac	Non-Cardiac
1- Acute coronary syndrome	**4- Acute pulmonary embolism**
Substernal, radiating to arm, dyspnea on exertion, diaphoresis, worse with exertion.	Sudden onset, pleuritic, dyspnea, tachycardia, tachypnea, hypoxia, evidence of lower extremity deep venous thrombosis
★2- Aortic dissection[1]	**5- Tension pneumothorax**
Sudden onset, severe, tearing, radiating to the back (associated with neurologic deficits, AR), unequal arm BP >20 mmHg, wide mediastinum	Sudden onset, sharp, pleuritic, decreased breath sounds and chest excursion, hyperresonant percussion, hypoxia
★ 3- Acute pericarditis & tamponade	**6- Esophageal rupture/perforation**
Sudden onset, pleuritic, better with sitting forward, radiating to the back, pericardial rub, ± tamponade (distant heart sounds, hypotension, JVD)	Severe, increase with swallowing, fever, abdominal pain, history of endoscopy, foreigns body ingestion, trauma, vomiting

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)

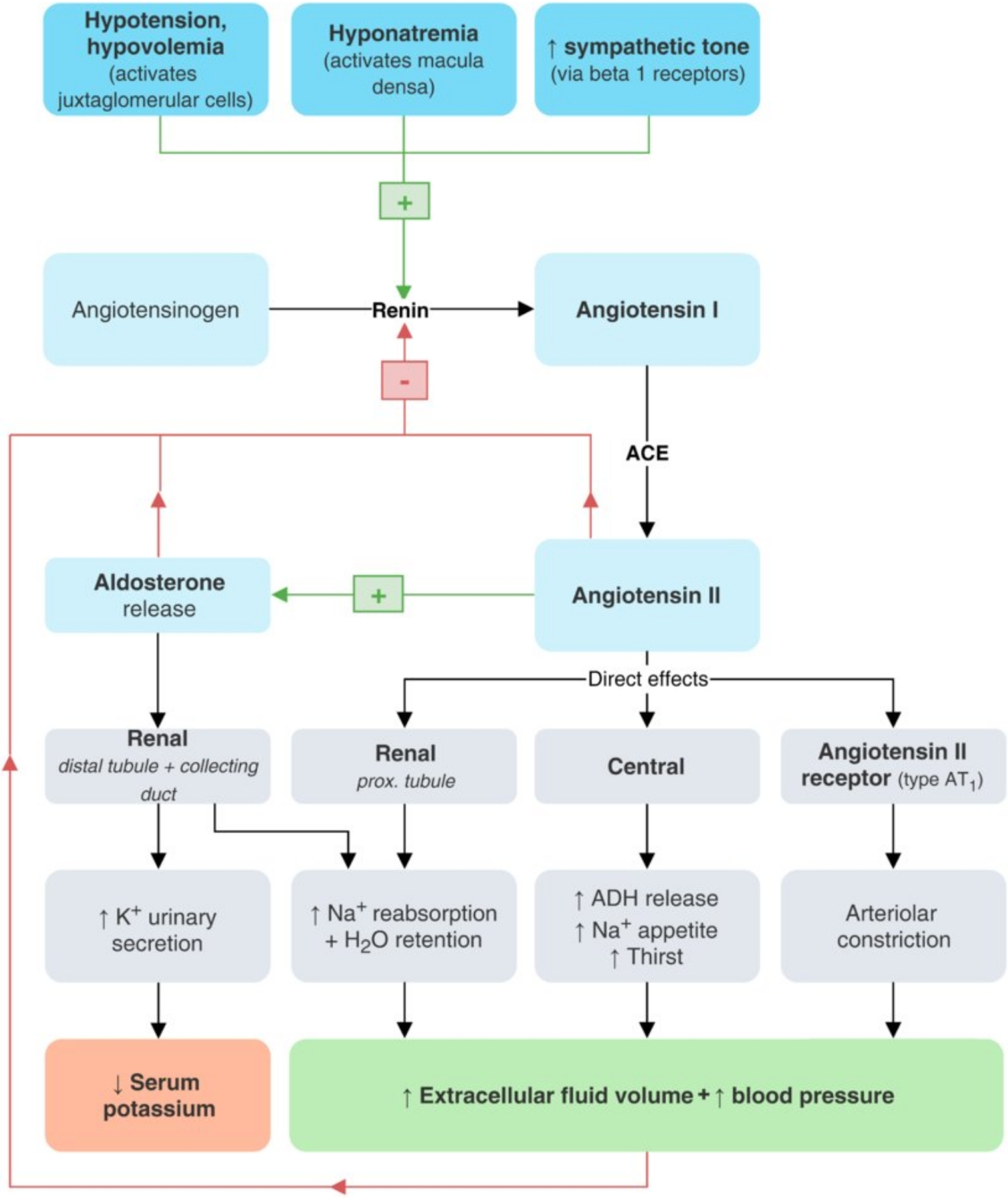
- **Heart failure (HF)**: a complex clinical syndrome in which there is structural or functional impairment of ventricular filling and/or ejection of blood [1]
- **Congestive heart failure (CHF)**: HF with signs and/or symptoms of fluid overload, e.g., peripheral edema, jugular venous distention
- **Left heart failure (LHF)**: HF caused by structural or functional impairment of the left heart circulatory system that results in tissue hypoperfusion and/or increased pulmonary capillary pressure
- **Right heart failure (RHF)**: HF caused by structural or functional impairment of the right heart circulatory system that results in impaired blood flow to the pulmonary circulation and/or elevated venous pressures [4]
- **Biventricular (global) heart failure**: HF in which both the left and right ventricles are affected, resulting in the development of **both RHF and LHF** symptoms
- **Chronic compensated heart failure**: HF with stable symptoms
- **Acute decompensated heart failure (ADHF)**: AHF due to decompensation of preexisting disease and/or cardiomyopathy (most common) [5]
- **Systolic dysfunction**: reduced ventricular contractility resulting in ventricular enlargement and reduced ejection fraction [6]
- **Diastolic dysfunction**: reduced ventricular compliance characterized by elevated filling pressures, abnormal relaxation, and increased ventricular stiffness [7]



Nutmeg Liver

the macroscopic appearance of the liver which resembles a nutmeg seed due to ischemia and fatty degeneration from hepatic venous congestion

Renin-Angiotensin-Aldosterone System (RAAS)



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

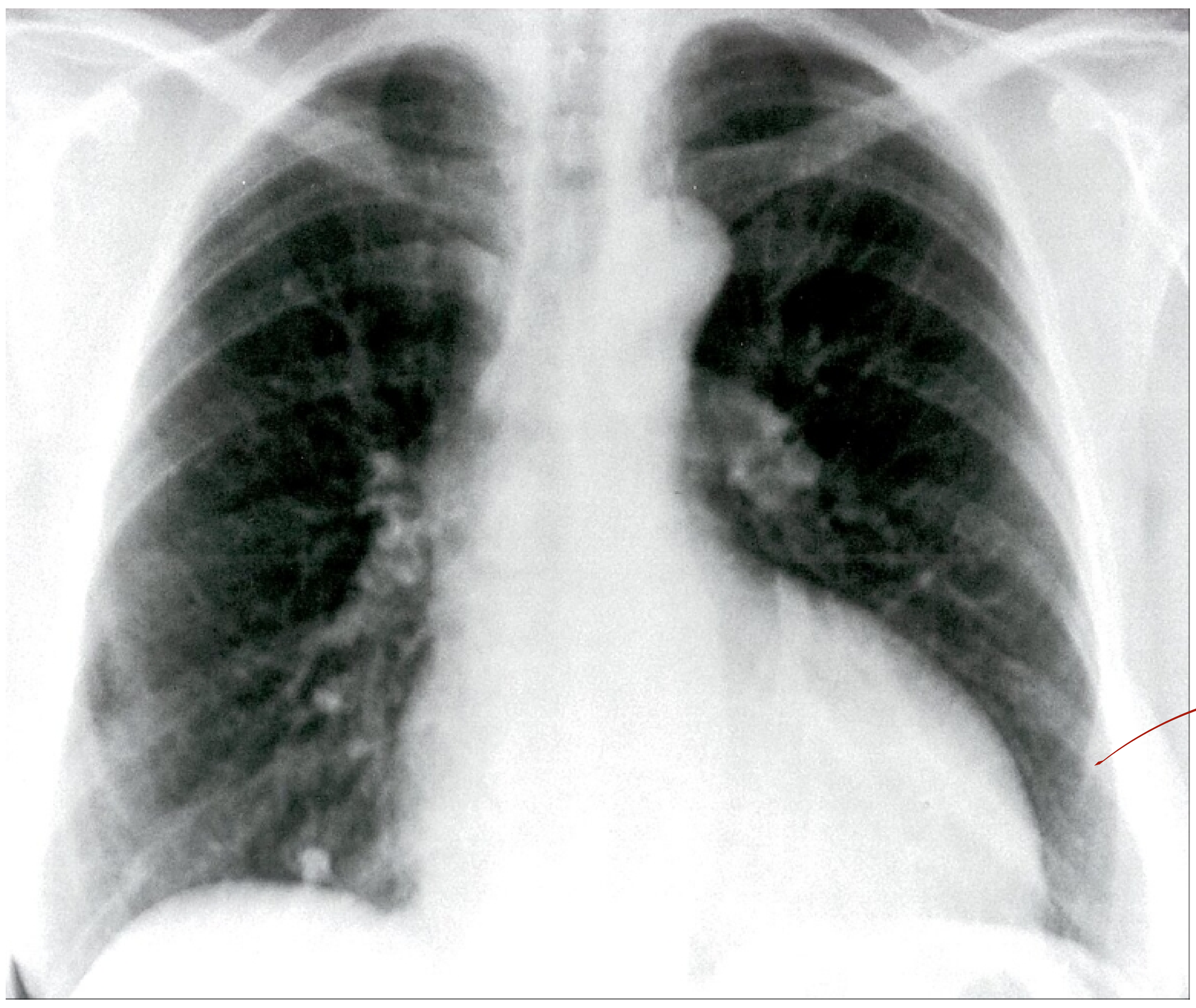
- Symptoms specific to high-output HF
  - Midsystolic murmur, S_3 **gallop**
  - Jugular distention with an audible hum over the internal jugular vein
  - Pulsatile tinnitus
  - Bounding peripheral pulses
  - Laterally displaced apex beat
- **Diagnostics**
  - Primarily a clinical diagnosis
  - X-ray and echocardiography: cardiomegaly

- HF is unlikely if:
  - BNP is < 35 pg/mL or NT-proBNP is < 125 pg/mL in chronic HF [
  - BNP is < 100 pg/mL or NT-proBNP is < 300 pg/mL in AHF [21]

* Preferred initial imaging modality for suspected HF is TTE (transthoracic echo)

Chest x-ray [1]

- **Indication**: all patients with suspected HF; especially useful in AHF
- **Findings**
  - Changes to the cardiac silhouette
    - Cardiomegaly, i.e., cardiothoracic ratio > 0.5 [29]
    - **Boot-shaped heart** on PA view: RV enlargement
    - Signs of pericardial effusion (e.g., water bottle heart)
  - X-ray findings of pulmonary congestion
  - Signs of concomitant heart conditions, including:
    - Valvular calcifications in valvular disease
    - Pericardial calcification in constrictive pericarditis



→ LV enlargement

Pharmacotherapy for HFrEF [1][3][34]

- **ACC/AHA stage B**
  - Beta blocker
  - PLUS either an ACEI or ARB
- **ACC/AHA stages C and D**
  - One agent from each of the following drug classes, unless contraindicated (e.g., prior hypersensitivity reaction):
    - Diuretics → Loop Diuretics, Thiazides
    - RAAS inhibitors
    - Beta blockers
    - SGLT2 inhibitors → glifozins
    - Mineralocorticoid receptor antagonists

Initial pharmacotherapy for HFrEF [1][3][34]			
Class		**Indications**	**Recommended agents**
Diuretics	**Loop diuretics**	• Preferred option for all patients with **congestion**	• Bumetanide • Furosemide • Torsemide
	Thiazide diuretics	• May be added if no response to moderate or high-dose loop diuretics	• Chlorthalidone • Hydrochlorothiazide • Metolazone
RAAS inhibitors	**Angiotensin receptor-neprilysin inhibitors (ARNIs)**	• Preferred initial agent for RAAS inhibition for patients with ACC/AHA stage C HFrEF and/or NYHA class II-IV HF [34] • Stop ACEIs 36 hours before starting an ARNI to avoid an elevated risk of angioedema.	• **Sacubitril/valsartan**
	ACE inhibitors (ACEIs)	• All patients with ACC/AHA stage B HFrEF • Patients with ACC/AHA stages C and D HFrEF if ARNI is not tolerated or affordable	• Enalapril • Ramipril • Lisinopril
	Angiotensin receptor blockers (ARBs)	• Patients with ACC/AHA stages B, C, and D HFrEF if ARNI and ACEIs are not tolerated (e.g., because of a dry cough or history of angioedema) or affordable	• Candesartan • Losartan • Valsartan
Beta blockers		• All patients with ACC/AHA stages B, C, and D HFrEF	• Bisoprolol • Carvedilol • Metoprolol succinate

SGLT2 inhibitors (SGLT2is)		• Patients with any of the following: [34]
		  ◦ ACC/AHA stage C
		  ◦ NYHA class II-IV HFrEF
		  ◦ LVEF ≤ 40%
			• Dapagliflozin
			• Empagliflozin

Mineralocorticoid receptor antagonists (MRAs)		• All patients with ACC/AHA stage C HFrEF without contraindications i.e., eGFR < 30 mL/min/1.73 m^2 and serum K^+ > 5.0 mEq/L	• Spironolactone • Eplerenone

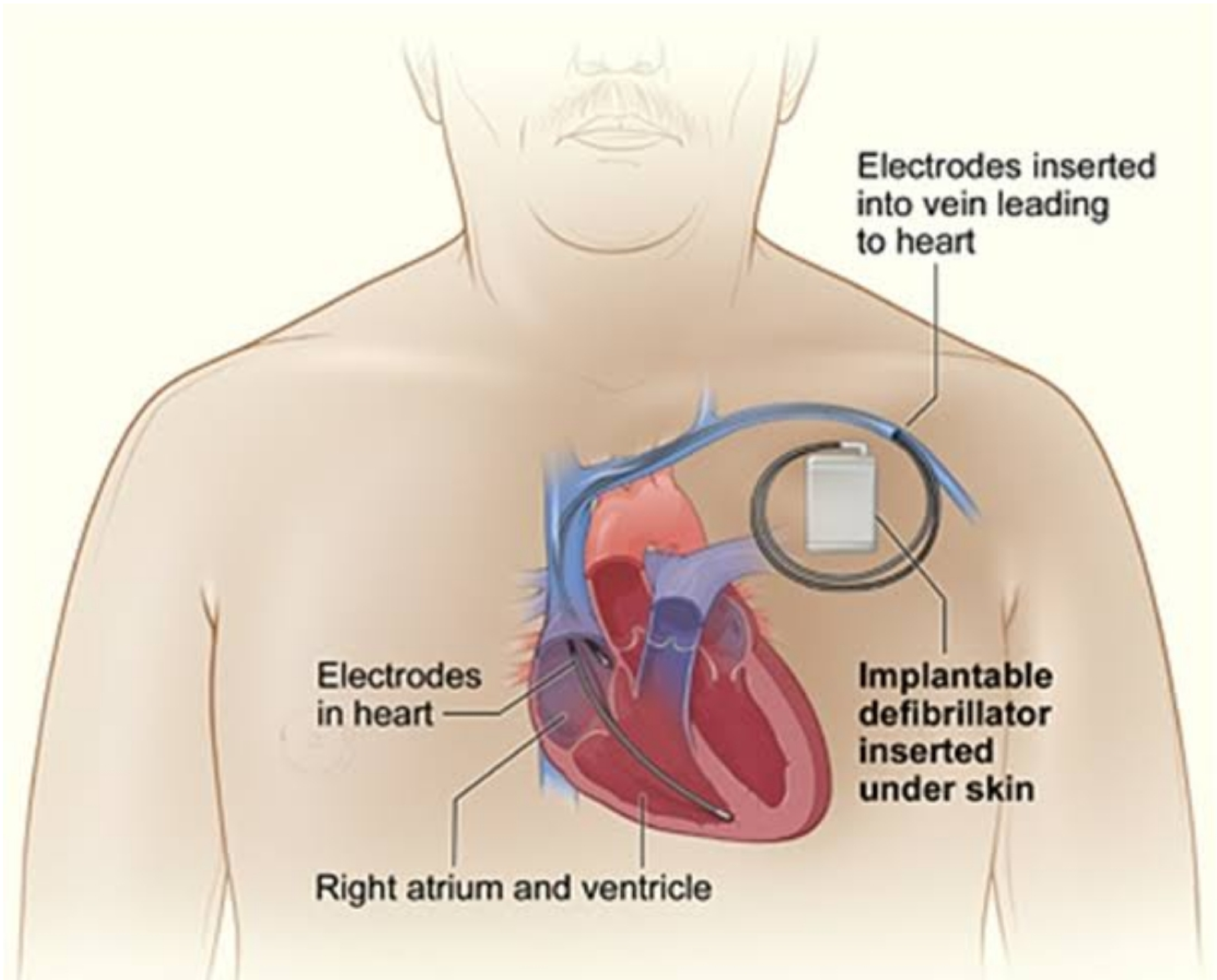
Pharmacotherapy for HFpEF [2][3][20]

- First-line agents
  - SGLT2i for all patients: e.g., dapagliflozin or empagliflozin
  - Loop diuretic for patients with congestion: e.g., furosemide or torsemide
- Other agents may be considered, e.g.:
  - MRA
  - ARNI or ARB

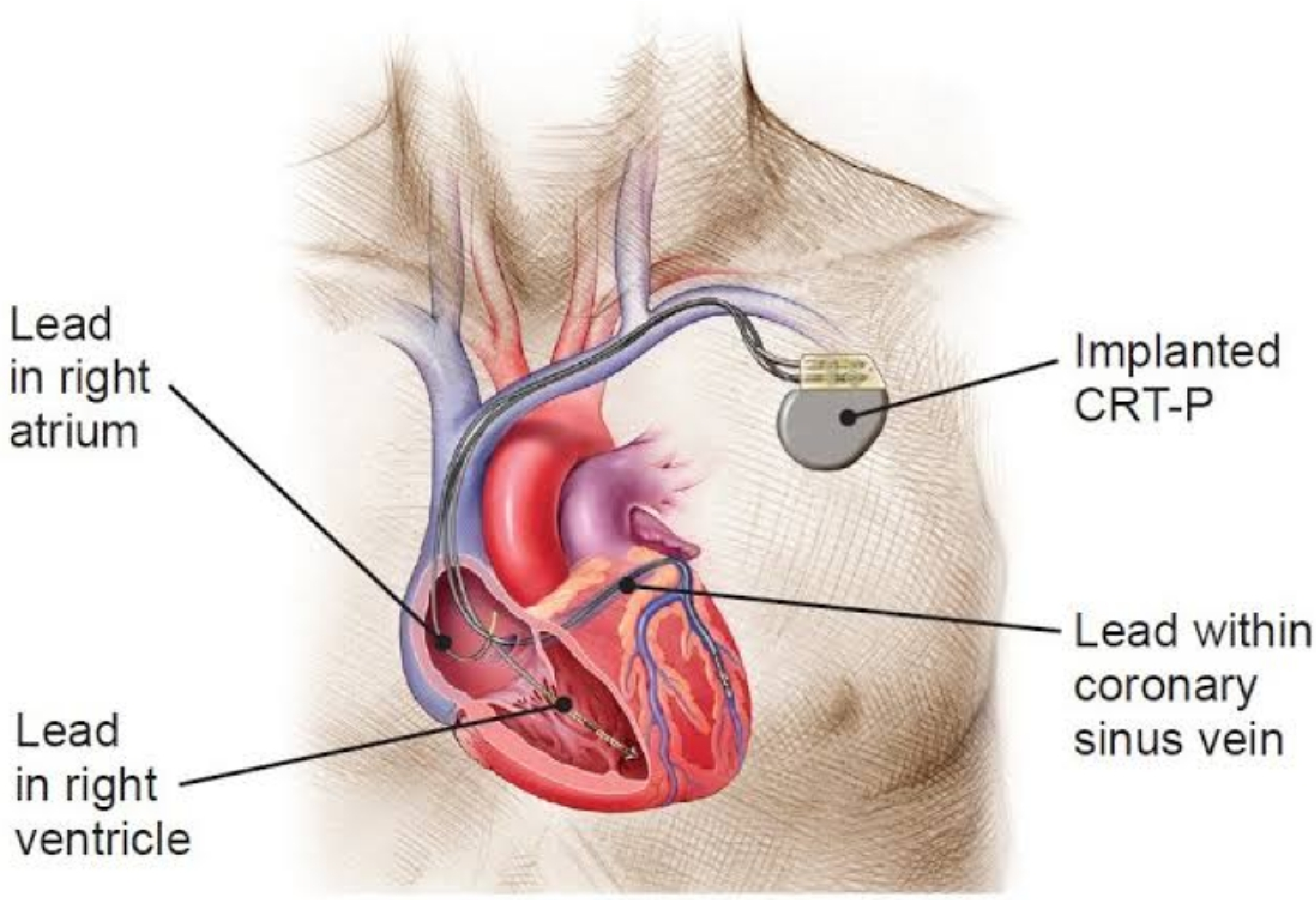
Drugs that may worsen HF [1]

The following drugs should be avoided or used with caution in patients with HF.

- Nondihydropyridine calcium channel blockers: associated with higher HF rate of recurrence
- NSAIDs: can worsen HF symptoms
- Thiazolidinediones (e.g., pioglitazone): increased risk of congestion and hospitalization
- Antidepressants (e.g., tricyclic antidepressants): Consider interactions with HF pharmacotherapy. [40]
- Inhalation anesthetics: may induce myocardial depression and peripheral vasodilation, and decrease sympathetic activity
- Class IC and class III antiarrhythmic drugs: increased mortality
- DPP-4 inhibitors (e.g., saxagliptin, alogliptin): increased risk of hospitalization for HF



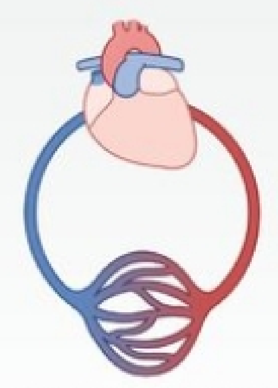
Automated implantable cardioverter defibrillators (AICDs)

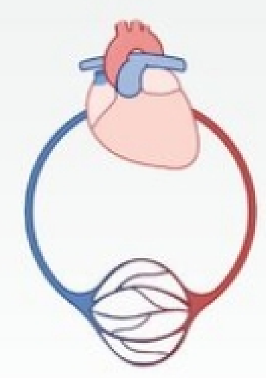


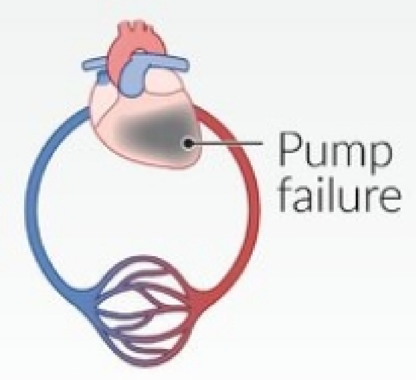
cardiac resynchronization therapy devices (CRTs)

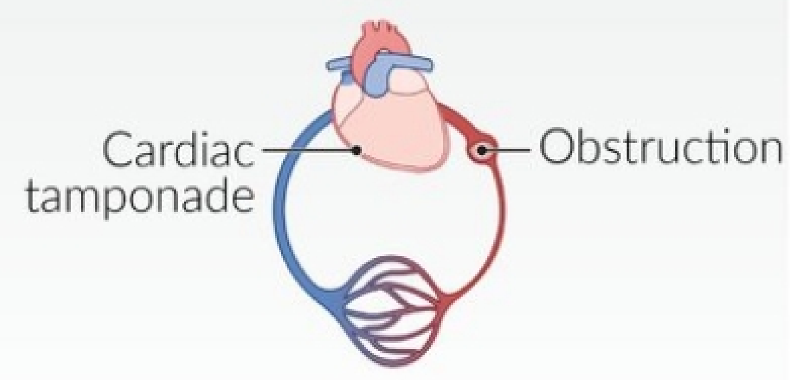
In a patient with tachycardia and cold extremities, Assume Shock

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]

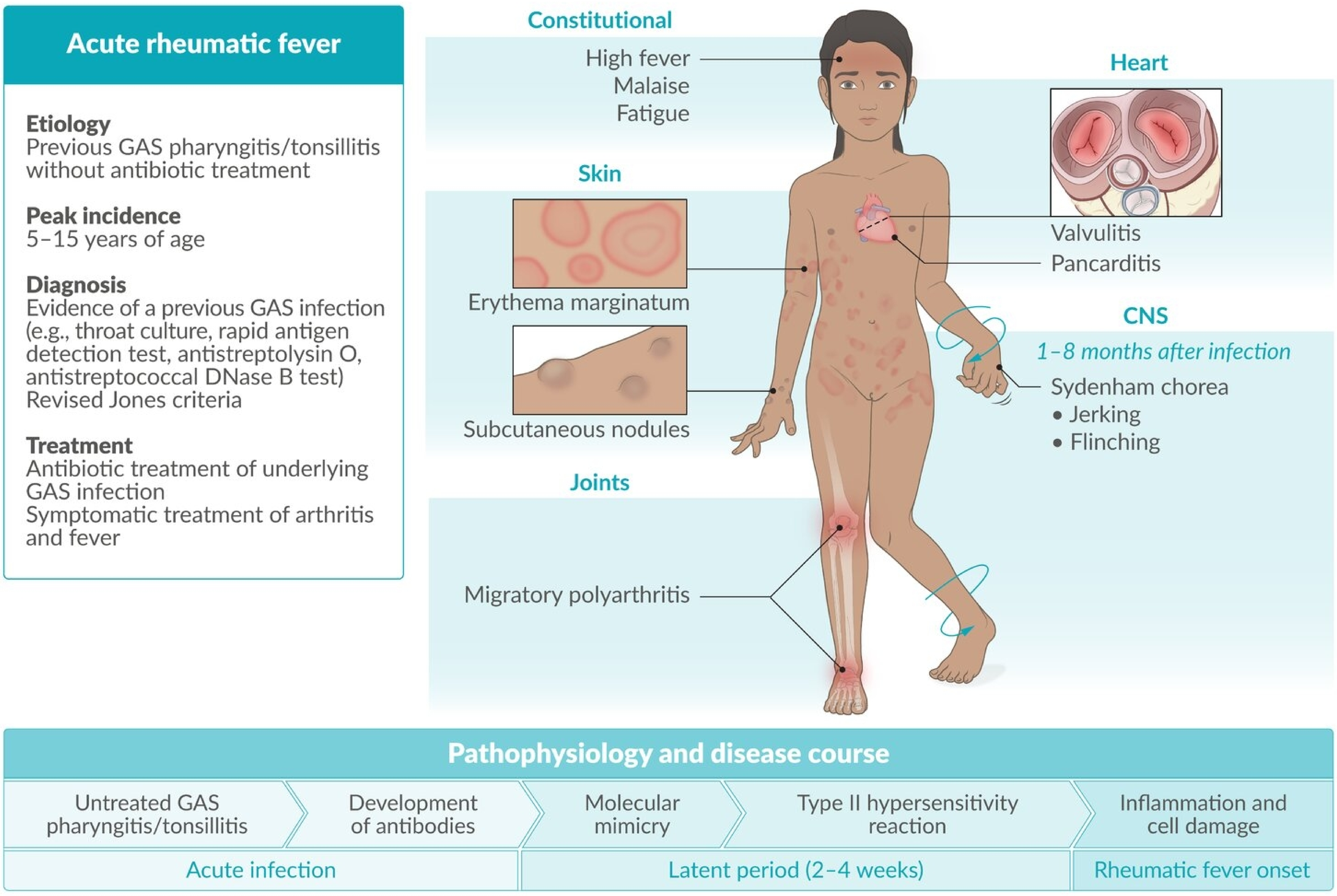
Management of Acute Decompensated Heart Failure

(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



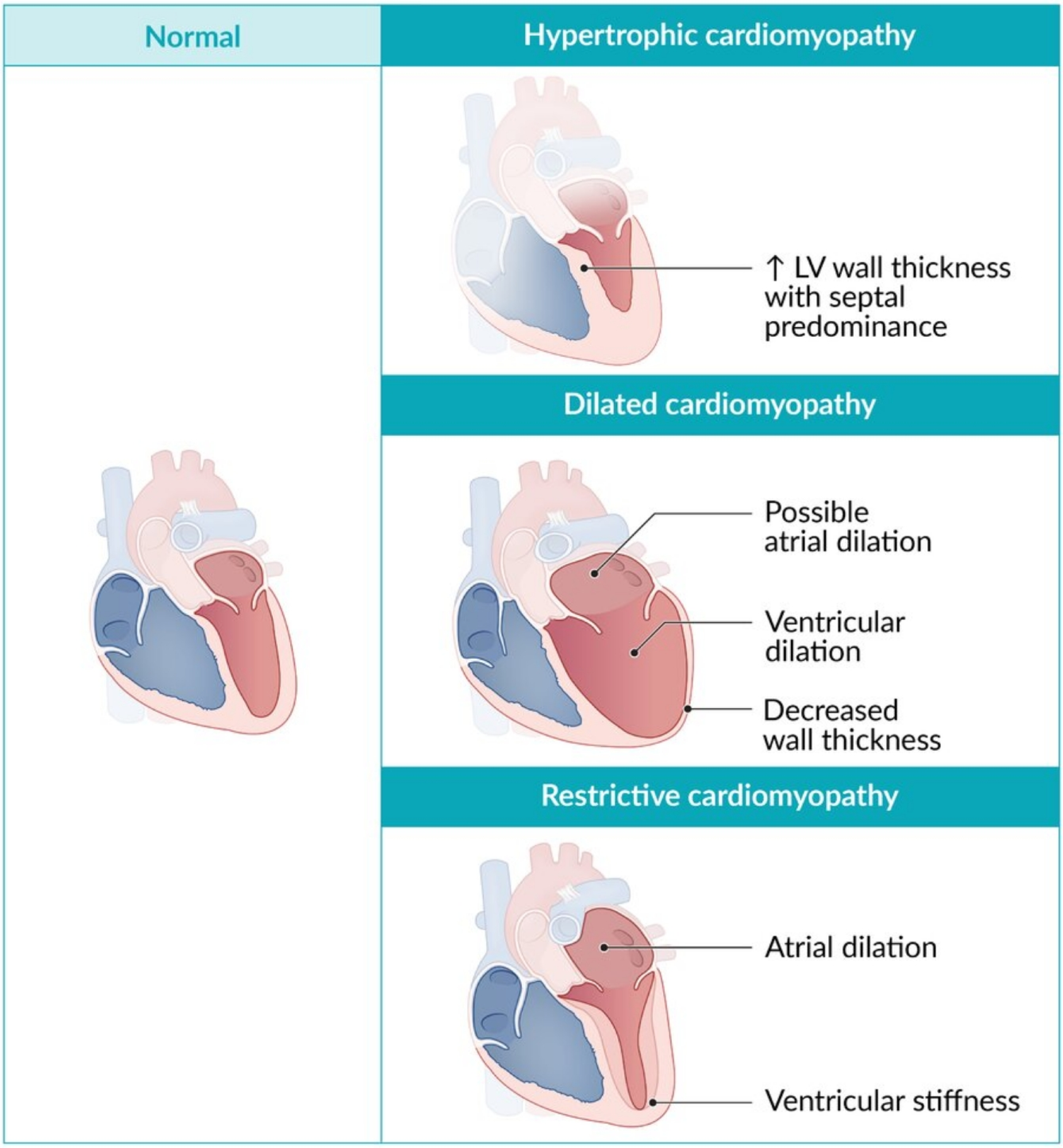
J → Joints (Migratory polyarthritis)

♡ → Pancarditis

N → Nodules (Skin nodules)

E → Erythema Marginatum

S → Sydenham chorea


Normal
Hypertrophic cardiomyopathy
↑ LV wall thickness with septal predominance
Dilated cardiomyopathy
Possible atrial dilation
Ventricular dilation
Decreased wall thickness
Restrictive cardiomyopathy
Atrial dilation
Ventricular stiffness

Secondary Causes of Dilated cardiomyopathy

ABCCCDD

A → Alcohol

B → Beriberi

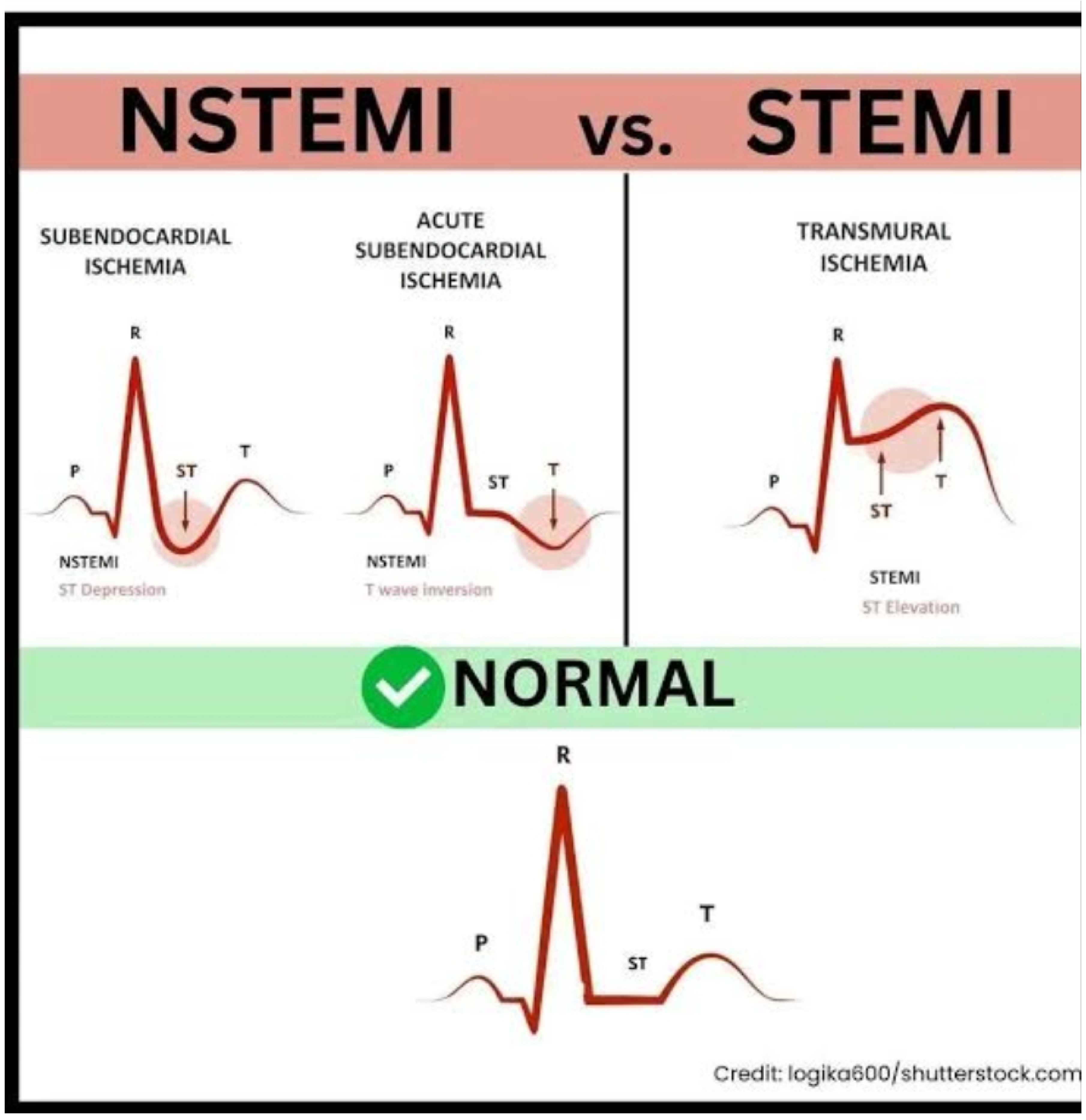
C → Cocaine

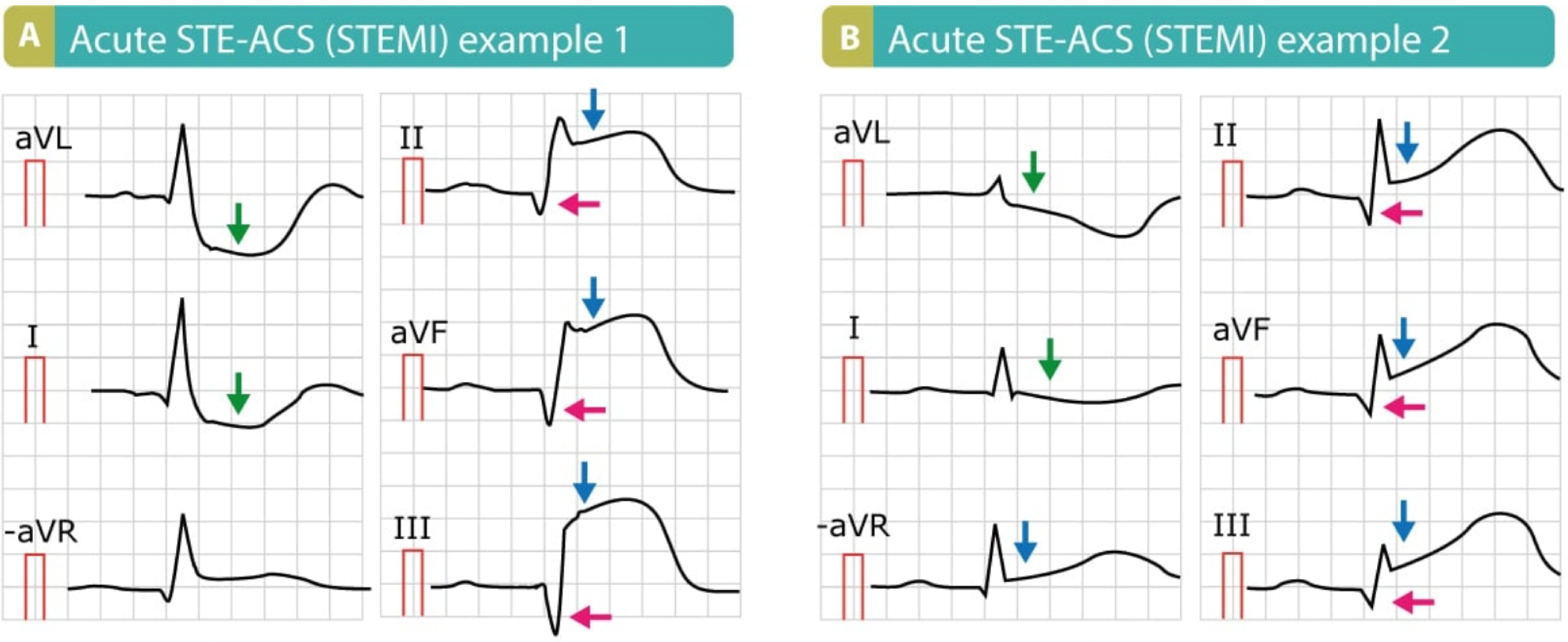
C → Chagas Disease

C → Coxsackie B virus

D → Doxorubicin

D → Daunorubicin





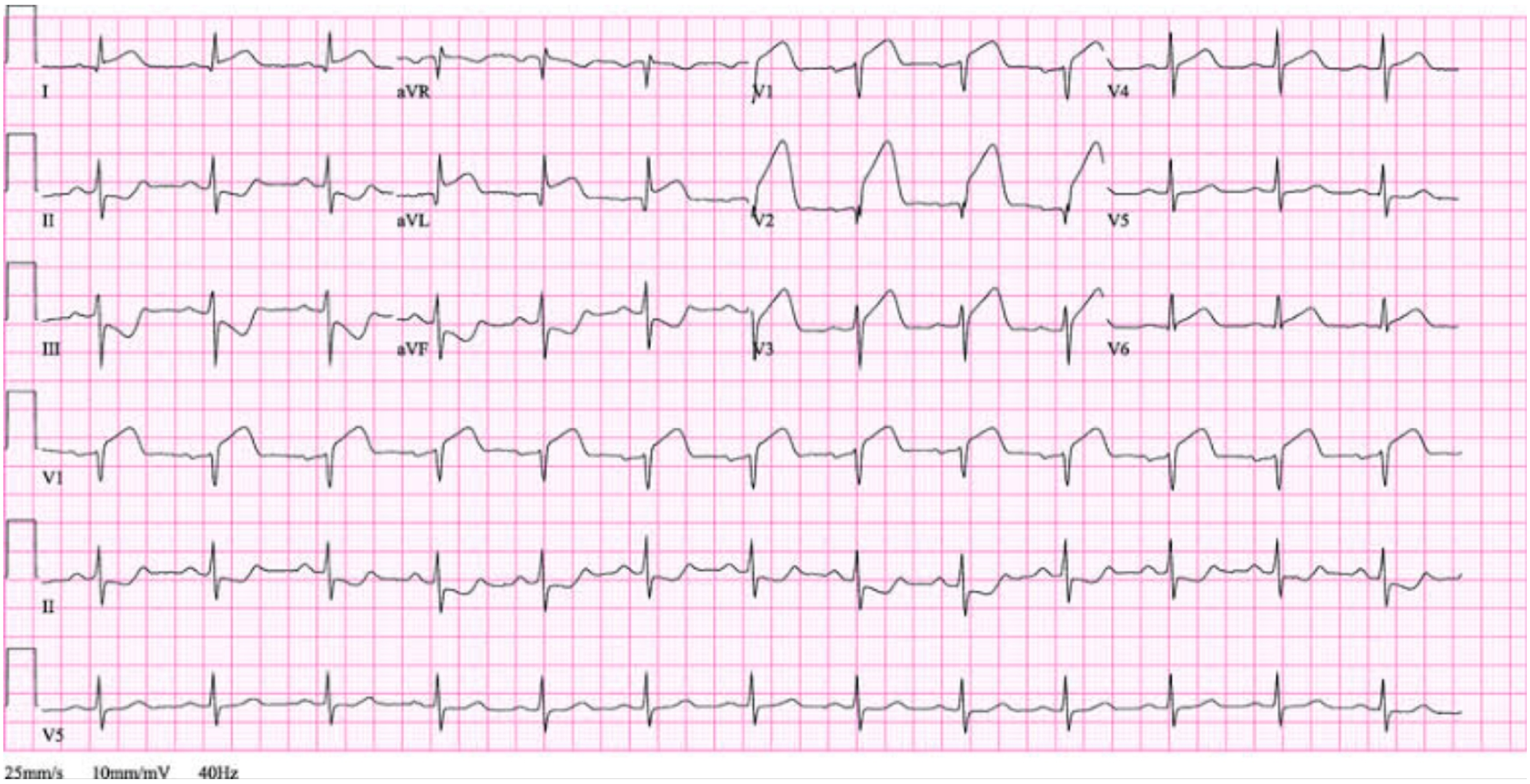
60 year old male with retrosternal chest pain. ECG shows ST segment elevations in inferior leads (II, aVF and III). There are reciprocal ST segment depressions in aVL and I. There are also pathological Q-waves in the inferior leads.

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

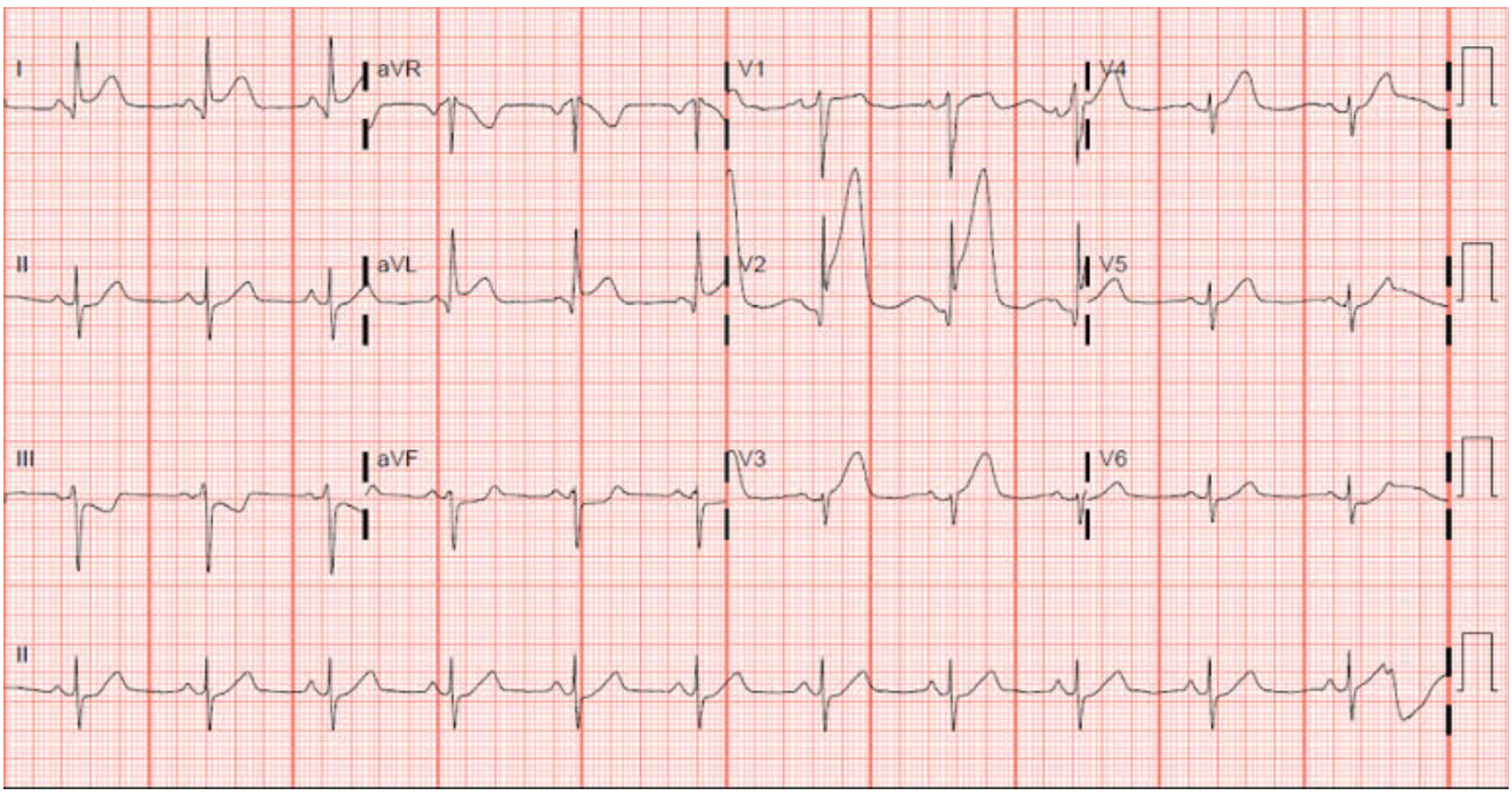
→ ST segment elevation

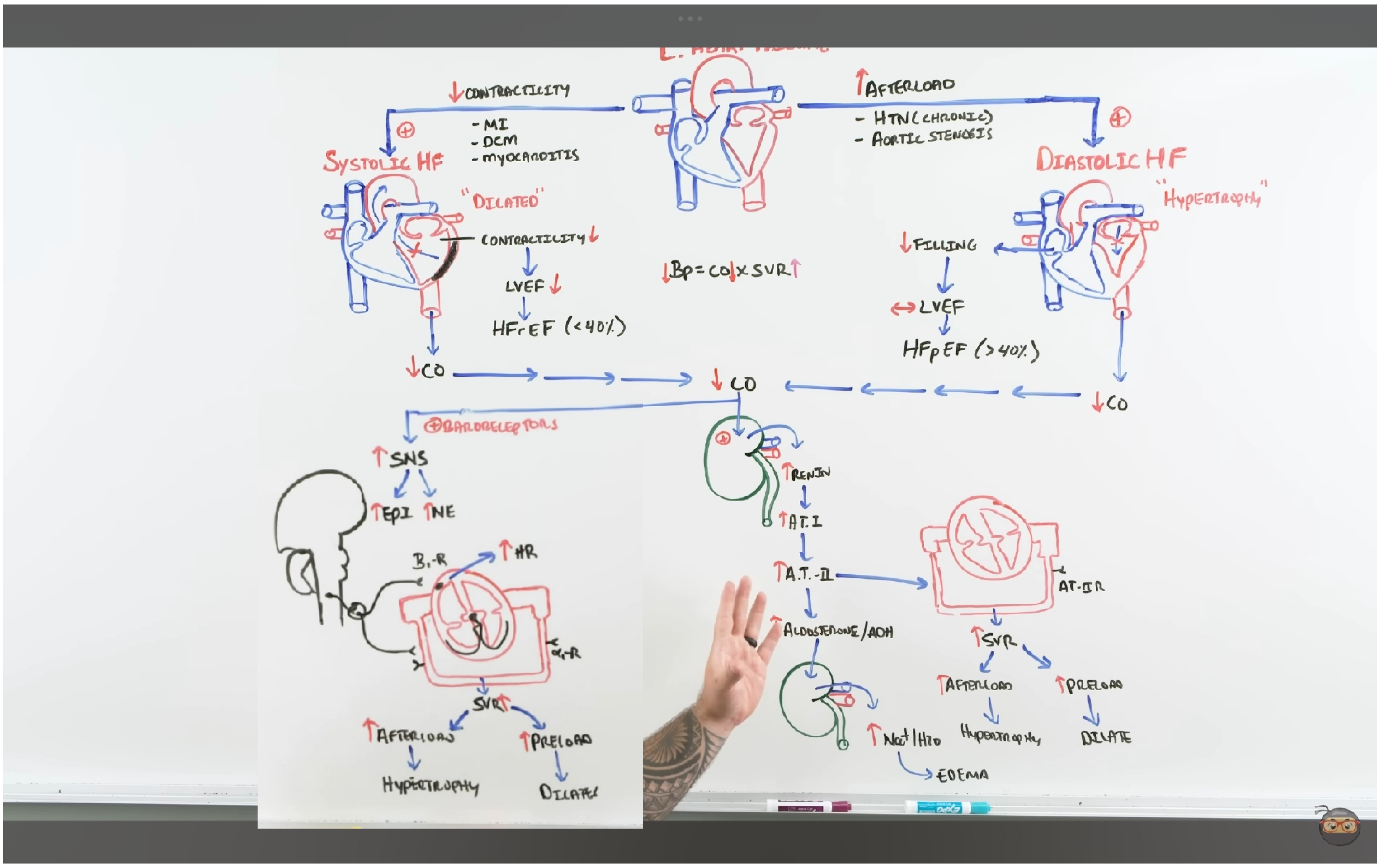
→ Pathological Q-waves

→ Reciprocal ST-segment depression

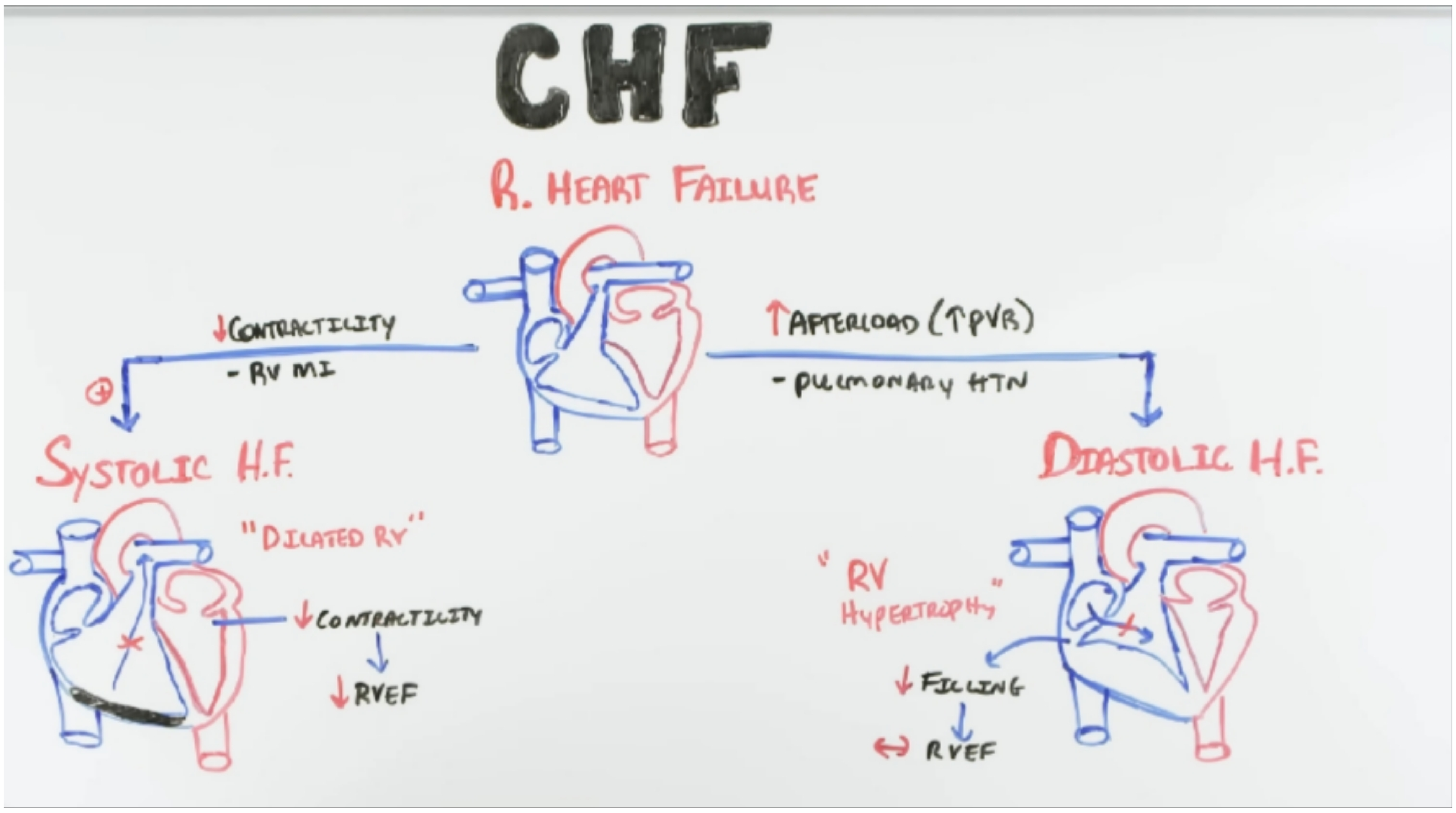

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz

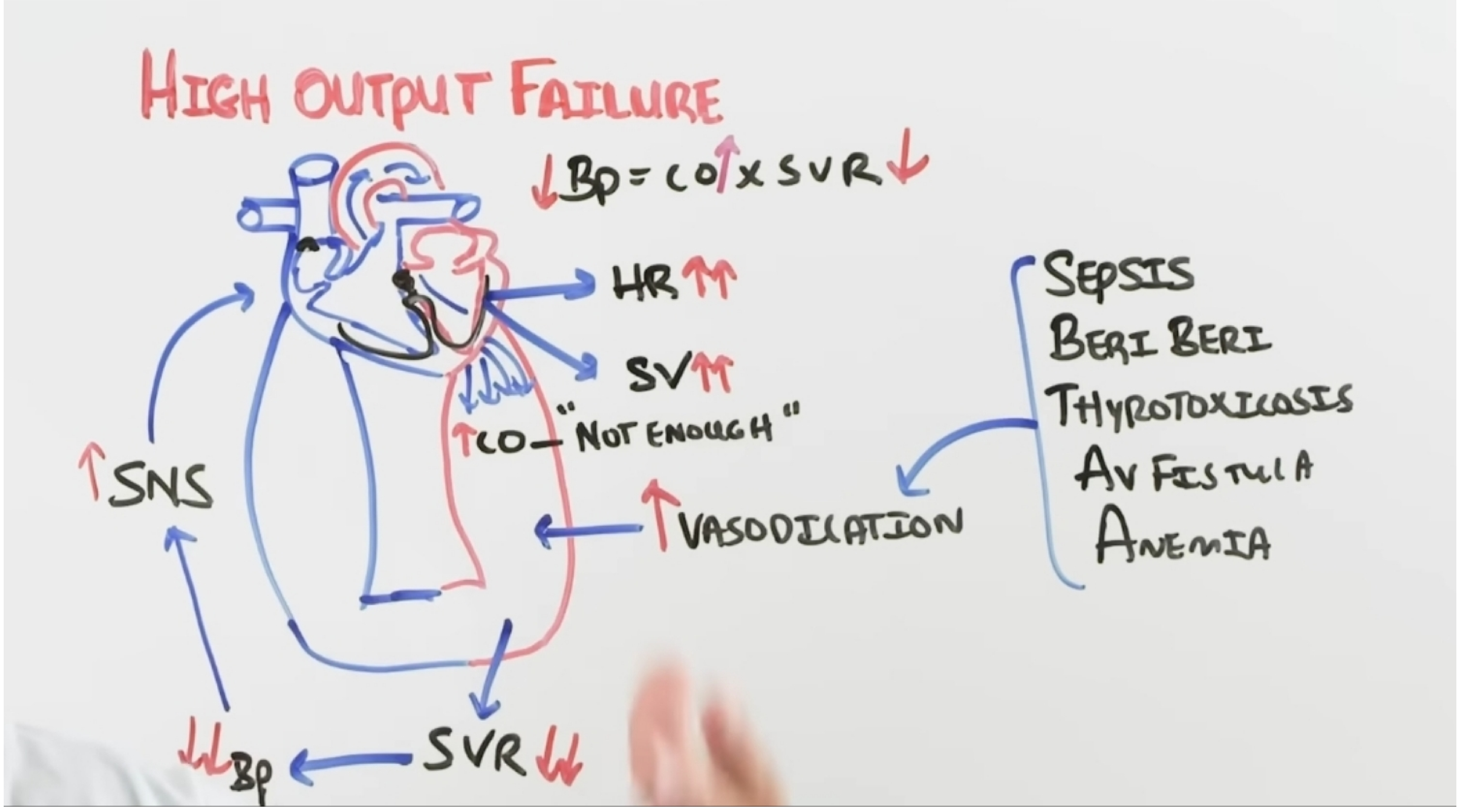
STEMI


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



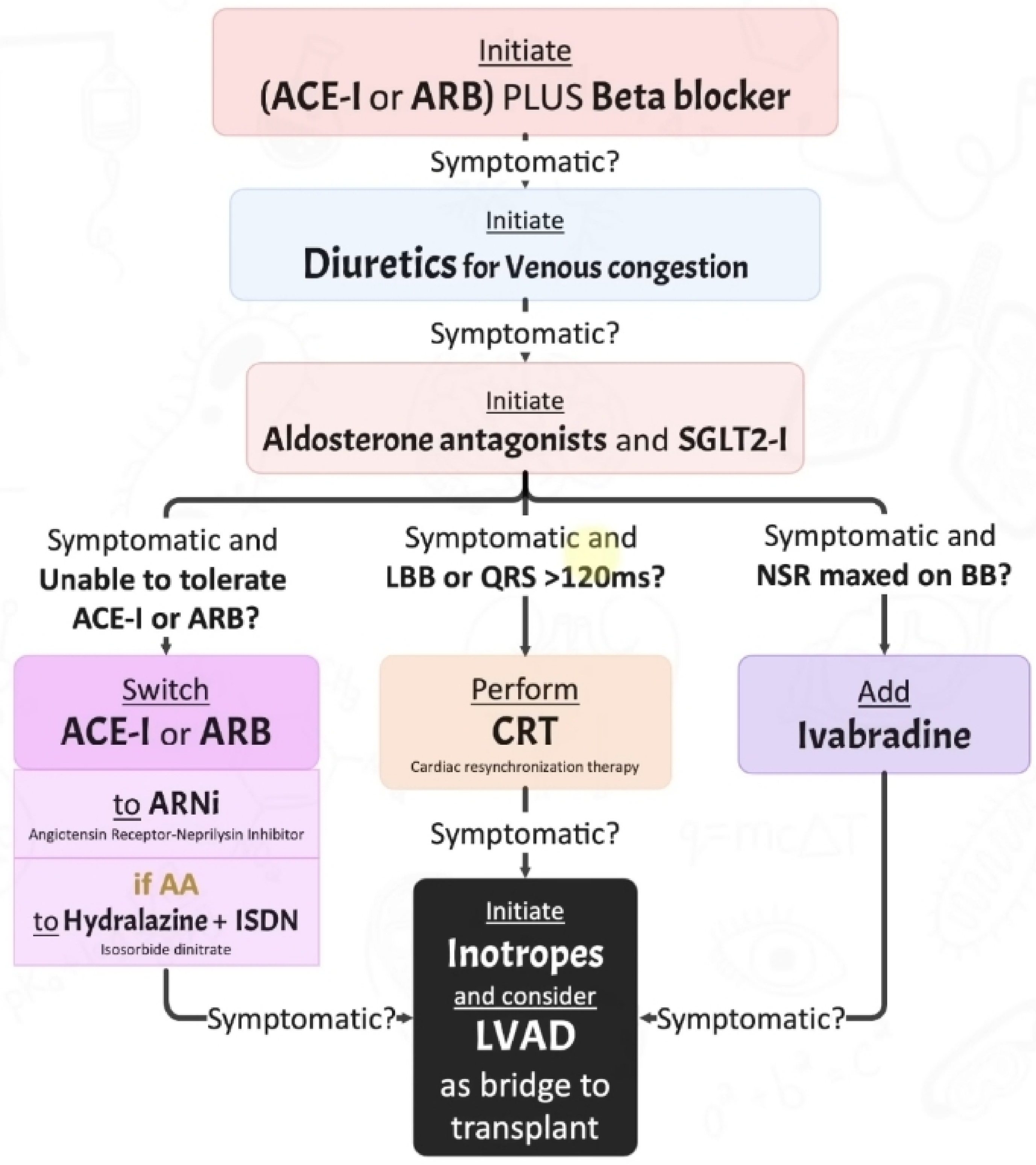
↓CONTRACTILITY
- MI
- DCM
- myocarditis
Systolic HF
"Dilated"
Contractility ↓
LVEF ↓
HFrEF (<40%)
↑Afterload
- HTN (chronic)
- Aortic stenosis
Diastolic HF
"Hypertrophy"
↓Filling
↔LVEF
HFpEF (>40%)
↓Bp = CO↓ x SVR↑
↓CO
↓CO
↓CO
⊕ Baroreceptors
↑SNS
↑Epi ↑NE
B1-R
↑HR
α1-R
SVR↑
↑Afterload
Hypertrophy
↑Preload
Dilate
↑Renin
↑A.T. I
↑A.T.-II
AT-II R
↑Aldosterone/ADH
↑Na+/H2O
Edema
↑SVR
↑Afterload
Hypertrophy
↑Preload
Dilate


CHF
R. HEART FAILURE
↓CONTRACTILITY
- RV MI
↑AFTERLOAD (↑PVR)
- PULMONARY HTN
SYSTOLIC H.F.
DIASTOLIC H.F.
"DILATED RV"
↓CONTRACTILITY
↓RVEF
"RV HYPERTROPHY"
↓FILLING
↔ RVEF

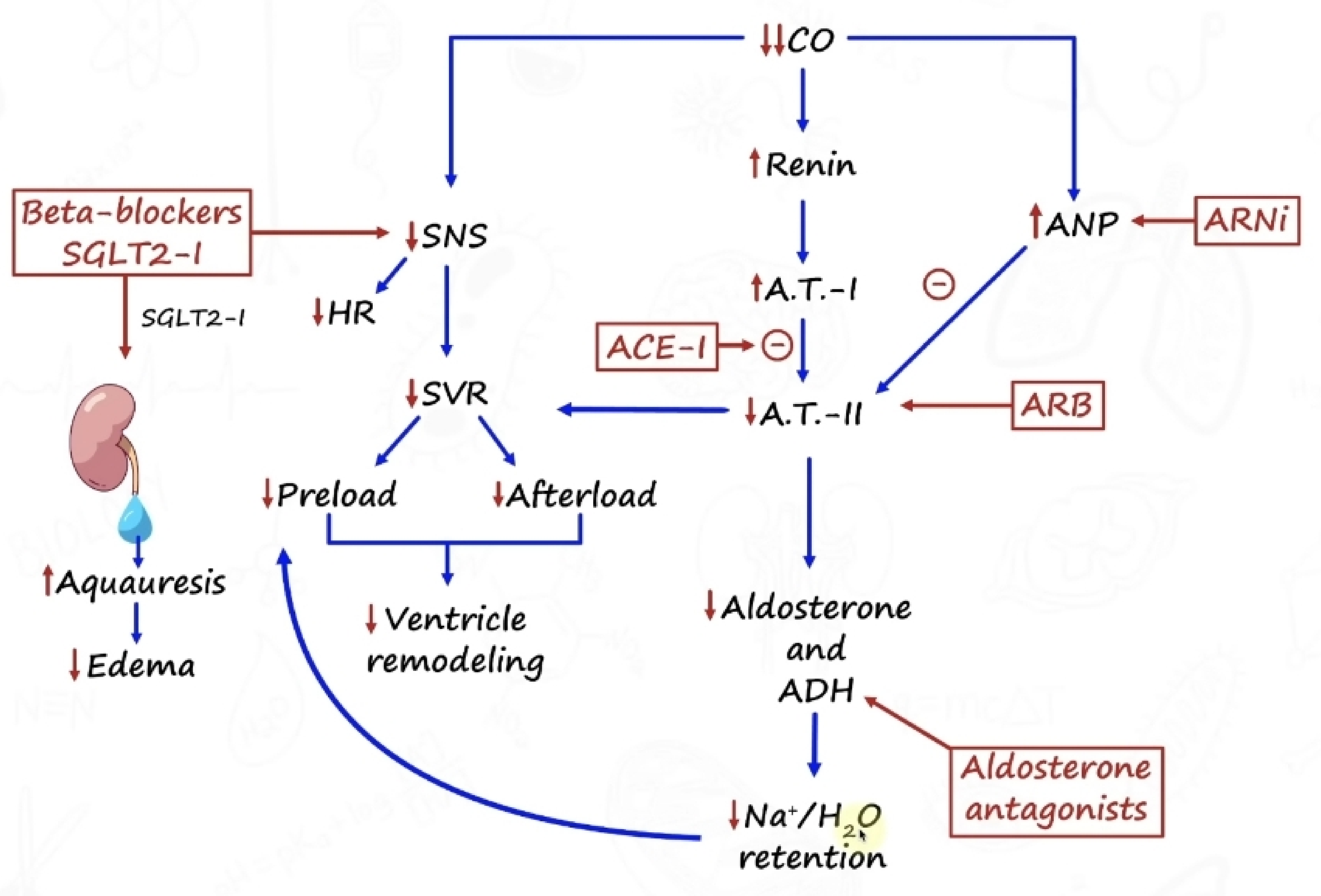

HIGH OUTPUT FAILURE
↓BP = CO↑ x SVR↓
HR↑↑
SV↑↑
↑CO – "NOT ENOUGH"
↑SNS
↑VASODILATION
↓↓BP
SVR↓↓
SEPSIS
BERI BERI
THYROTOXICOSIS
AV FISTULA
ANEMIA

Treatment of
Heart Failure

Type of Therapy	Purpose of Treatment	Treatment
Guideline Directed Medical Therapy (↓ Ventricular Dysfunction)	↓ SNS Activity	- Beta Blockers - SGLT2-I
	↓ RAAS Activity	- ACE-I or ARBs or ARNi - Aldosterone Antagonists
	Alternatives to ACE-I / ARBs **in AA**	- Hydralazine + ISDN
	Alternative to Beta Blocker **if NSR** (normal sinus rhythm)	- Ivabradine
↓ Venous Congestion	↑ Natriuresis	- Loop Diuretics - Thiazide Diuretics
Device Therapy (LVEF < 35%)	↑ Ventricular Synchrony (e.g. LBBB)	- CRT Cardiac resynchronization therapy
	↓ Ventricular Arrhythmias (e.g. VT/VF)	- AICD
	Bridge to Transplant	- LVAD
Cardiogenic Shock	↑ Systemic Perfusion	- Inotropes - IABP or VA-ECMO
	↓ Pulmonary Edema	- Diuretics and BIPAP



Treatment of
Heart Failure



◀ Management of Chronic HF cont.

Angiotensin-converting enzyme inhibitors (ACEI)			
Drug name	**Perindopril**	**Lisinopril**	**Quinapril**
M.O.A	• Block the R-A-A system by inhibiting the conversion of angiotensin I to angiotensin II → vasodilation and ↓ Na and water retention.(decrease Afterload and preload) • ↓ Bradykinin degradation ↑ its level → ↑ PG secretion & nitric oxide → Vasodilation • They also ultimately inhibit cardiac remodelling		

Uses	★ They improve symptoms, limit the development of progressive heart failure and **prolong survival**, and **should be given to all patients with heart failure.**
	• ACE Inhibitors were found to improve survival in CHF patients:
	  ○ Delay onset & progression of HF in pts with asymptomatic LV dysfunction
	  ○ ↓ cardiac remodeling		

Side effects	• Angioedema • Renal insufficiency, Hepatic dysfunction • Rash, Hyperkalemia[1] ★ Cough (If patient develops cough switch to ARBs) • **The major side-effect is first-dose hypotension.** ○ ACEI treatment should be introduced gradually with a **low initial dose and gradual titration** every 2 days to full dose with regular blood pressure monitoring and a check on serum potassium and renal function 1-2 weeks after starting therapy; **creatinine levels normally rise by about 10–15% during ACEI therapy.** • If patient developed hyperkalemia or renal impairment or is pregnant→ **switch from ACEI to Hydralazine with isosorbide dinitrate.**		
Contraindications	• Renal failure • Bilateral renal artery stenosis (RAS) • Aortic stenosis		

Trials for ACEI:
- CONSENSUS 1987 – enalapril vs. placebo – 31% reduction mortality in enalapril group. Confirmed by SOLVD, AIRE, SAVE, TRACE.
- 1995 meta-analysis showed 23% reduction total mortality, 35% in combined mortality/hosp admission

Angiotensin II type 1 receptor blockers (ARBs)			
Drug name	**losartan**	**irbesartan**	**valsartan**
Uses	• **Second-line therapy** in certain conditions when ACE I are contraindicated **(angioneurotic edema, cough)** • Has comparable effect to ACEI. Has mortality benefit.		
ADRs	• Renal dysfunction, hyperkalemia		

Heart failure

Classification

Systolic dysfunction (HFrEF)

- Impaired contractility, EF is reduced.
- Causes: IHD, HTN, VHD etc

Diastolic dysfunction (HFpEF)

- Impaired ventricular filling, EF is preserved.
- Causes: HTN leading to myocardial hypertrophy.

High Output HF

Conditions that increase demand on CO, causing a clinical picture of heart failure due to an excessively high CO e.g. Severe anemia, thyrotoxicosis, pregnancy, A/V fistula, Beriberi and Paget's disease

Low Output HF

Cardiac output is inadequate to perfuse the body (i.e. EF <40%), or can only be adequate with high filling pressures.

Acute HF

- Acute left heart failure presents with a sudden onset of dyspnoea at rest that rapidly progresses to **acute respiratory distress**, orthopnoea and prostration.
- Often there is a clear precipitating factor (e.g. large MI, aortic valve dysfunction, myocarditis, and cardiogenic shock) which may be apparent from the history.

Chronic HF

- Patients with chronic heart failure commonly follow a **relapsing and remitting course**, with periods of stability and episodes of decompensation*, leading to worsening symptoms that may necessitate hospitalisation

Left sided HF

- **Reduction in left ventricular output** and an **increase in left atrial and pulmonary venous pressure. T**his increases pulmonary vascular resistance and causes pulmonary hypertension, which in turn impairs right ventricular function.
- **Hallmark: Increased LVEDP**
- **Symptoms:** Dyspnea, Orthopnea, PND
- **Signs:** Displaced PMI, Cardiomegaly, S3, S4 and crackles at lung bases.

Right sided HF

- **Reduction in right ventricular output** and an **increase in right atrial and systemic venous pressure.**
- The most common cause of right Hf is left HF other causes include: Pulmonary HTN and chronic lung disease **(cor pulmonale)**
- **Symptoms:** Peripheral edema, Nocturia, Abdominal symptoms
- **Signs:** JVD, Hepatomegaly and Ascites

Treatment

Acute HF

1) **Oxygen**
2) **Loop diuretics** (Furosemide)
3) **Nitrate** (IV and Buccal Glyceryl trinitrate)
4) **Morphine:** Can be of value in distressed patients but must be used sparingly, as they may cause respiratory depression and exacerbation of hypoxaemia and hypercapnia.

Note: If these measures **prove ineffective use dobutamine.**

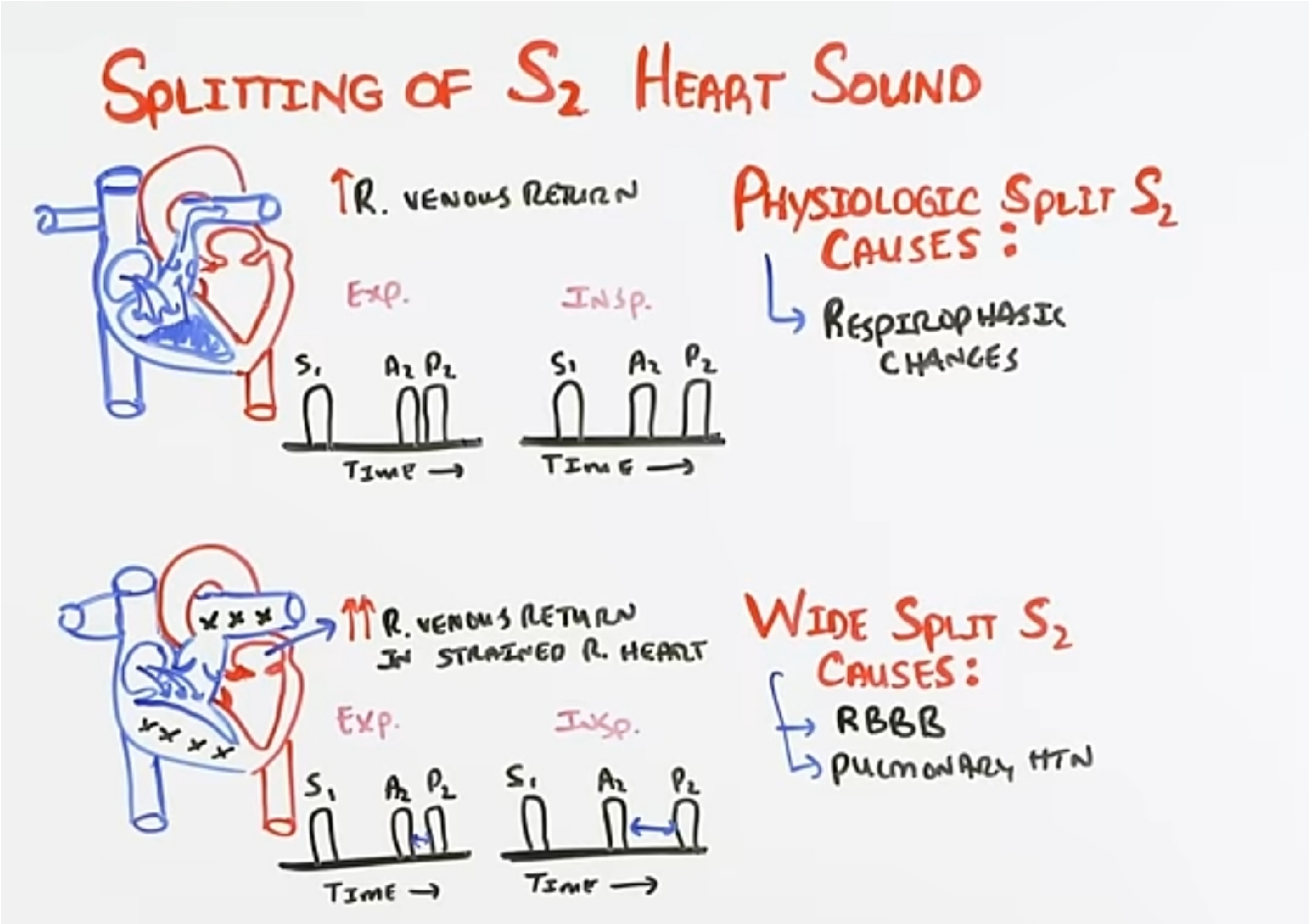
Chronic HF

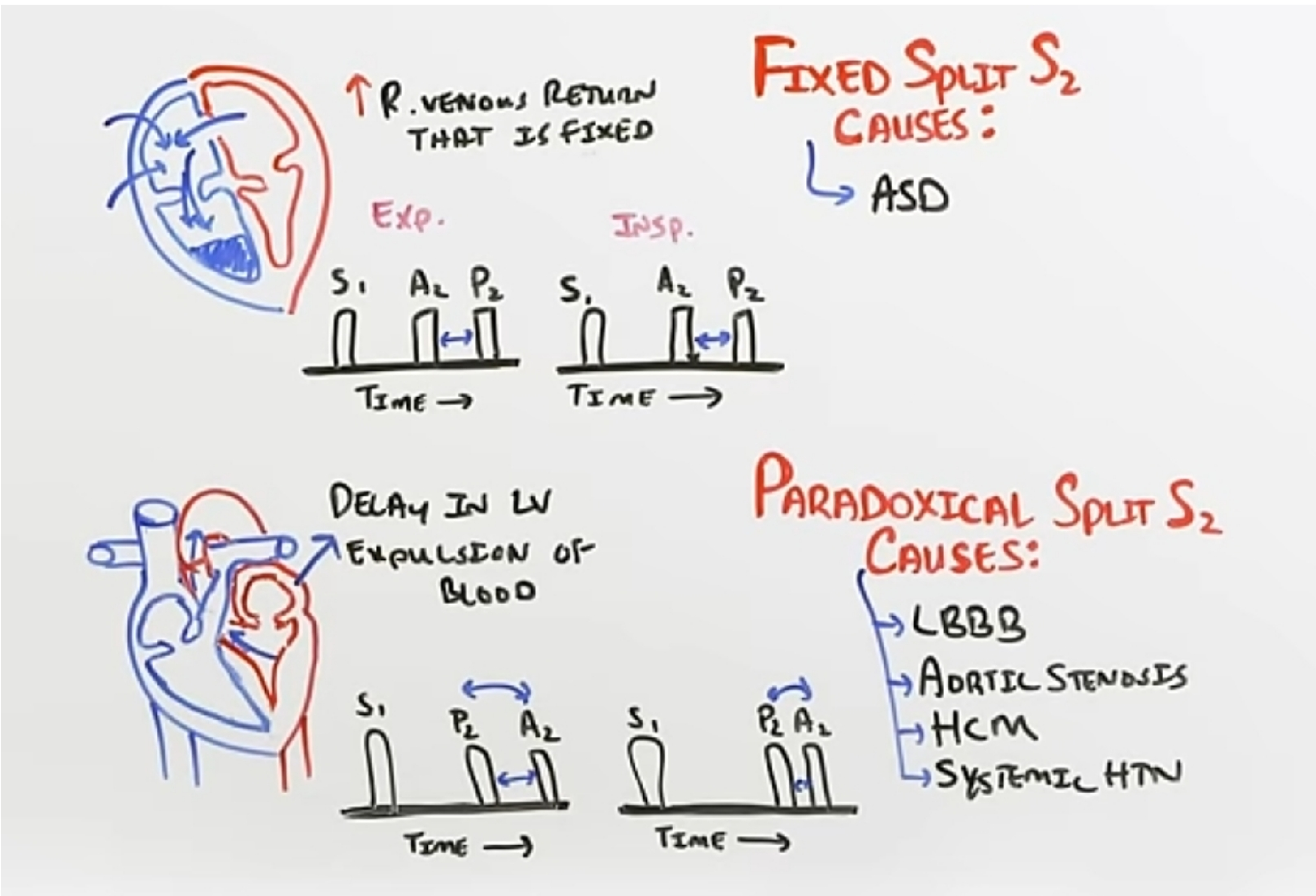
HFrEF

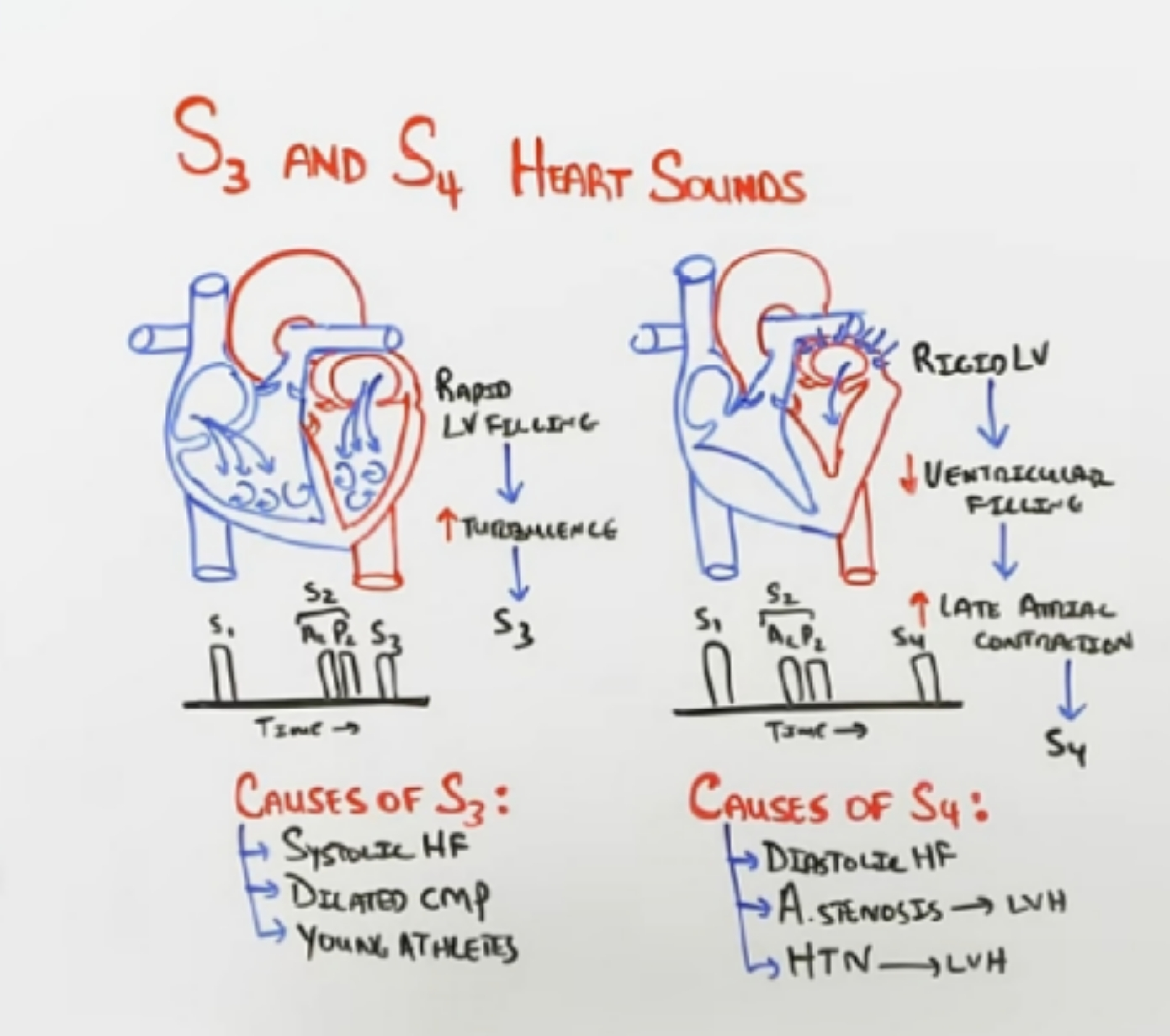
- ACEI or ARB
- BB
- Spironolactone
- Diuretics
- Digoxin

HFpEF

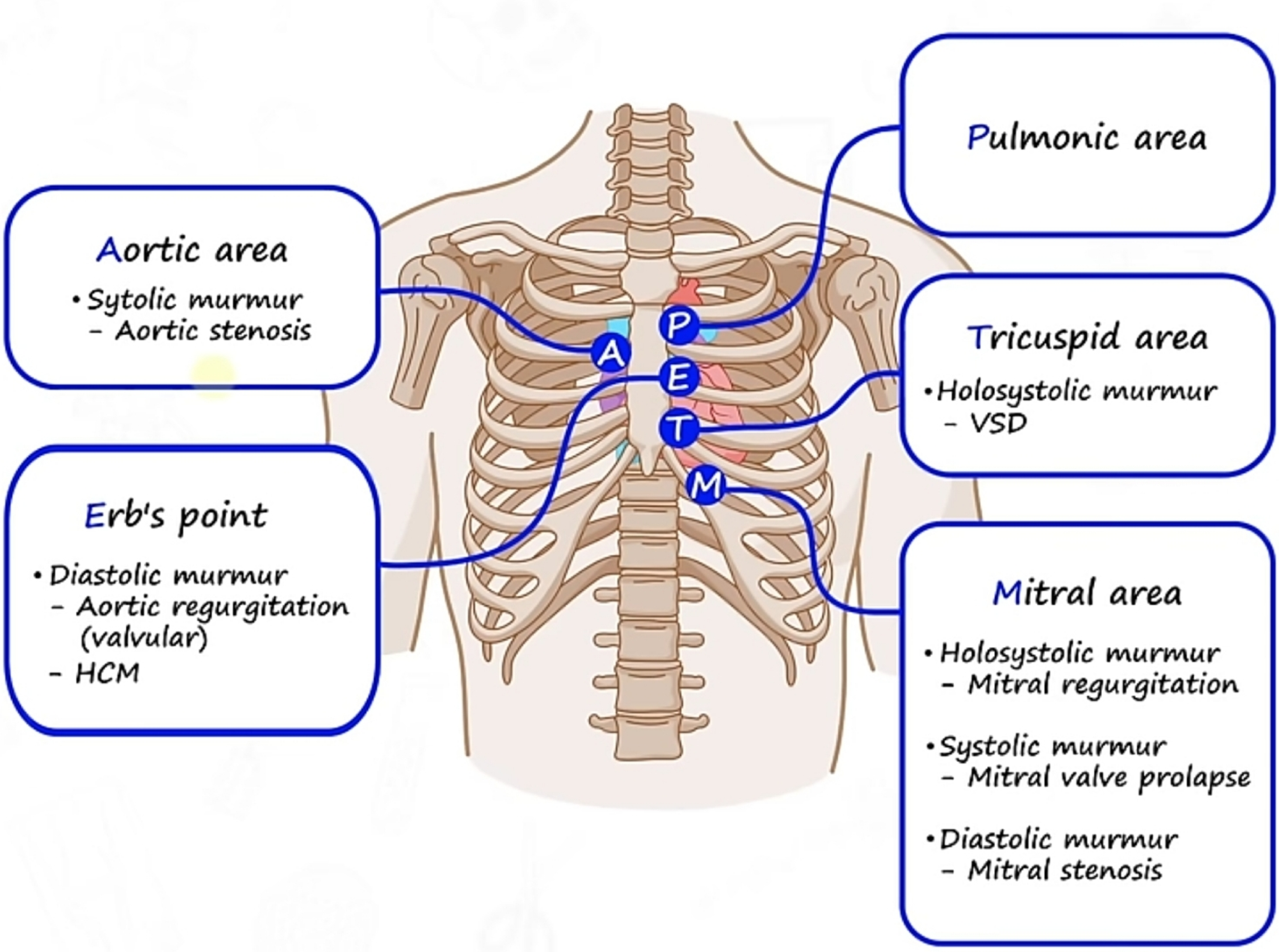
- Spironolactone
- Diuretics
- Uncertain: ACEI, ARBs
- **NOT used:** BB, Digoxin


SPLITTING OF S2 HEART SOUND
↑R. VENOUS RETURN
EXP.
INSP.
S1 A2 P2
S1 A2 P2
TIME →
TIME →
PHYSIOLOGIC SPLIT S2
CAUSES:
RESPIROPHASIC CHANGES
↑↑R. VENOUS RETURN IN STRAINED R. HEART
EXP.
INSP.
S1 A2 P2
S1 A2 P2
TIME →
TIME →
WIDE SPLIT S2
CAUSES:
RBBB
PULMONARY HTN


↑R. VENOUS RETURN THAT IS FIXED
EXP.
INSP.
S1 A2 P2
S1 A2 P2
TIME →
TIME →
FIXED SPLIT S2
CAUSES:
ASD
DELAY IN LV EXPULSION OF BLOOD
S1 P2 A2
S1 P2 A2
TIME →
TIME →
PARADOXICAL SPLIT S2
CAUSES:
LBBB
AORTIC STENOSIS
HCM
SYSTEMIC HTN


S3 AND S4 HEART SOUNDS
RAPID LV FILLING
↑ TURBULENCE
S3
S1
S2
A2 P2
S3
TIME →
RIGID LV
↓ VENTRICULAR FILLING
↑ LATE ATRIAL CONTRACTION
S4
S1
S2
A2 P2
S4
TIME →
CAUSES OF S3:
SYSTOLIC HF
DILATED CMP
YOUNG ATHLETES
CAUSES OF S4:
DIASTOLIC HF
A. STENOSIS → LVH
HTN → LVH

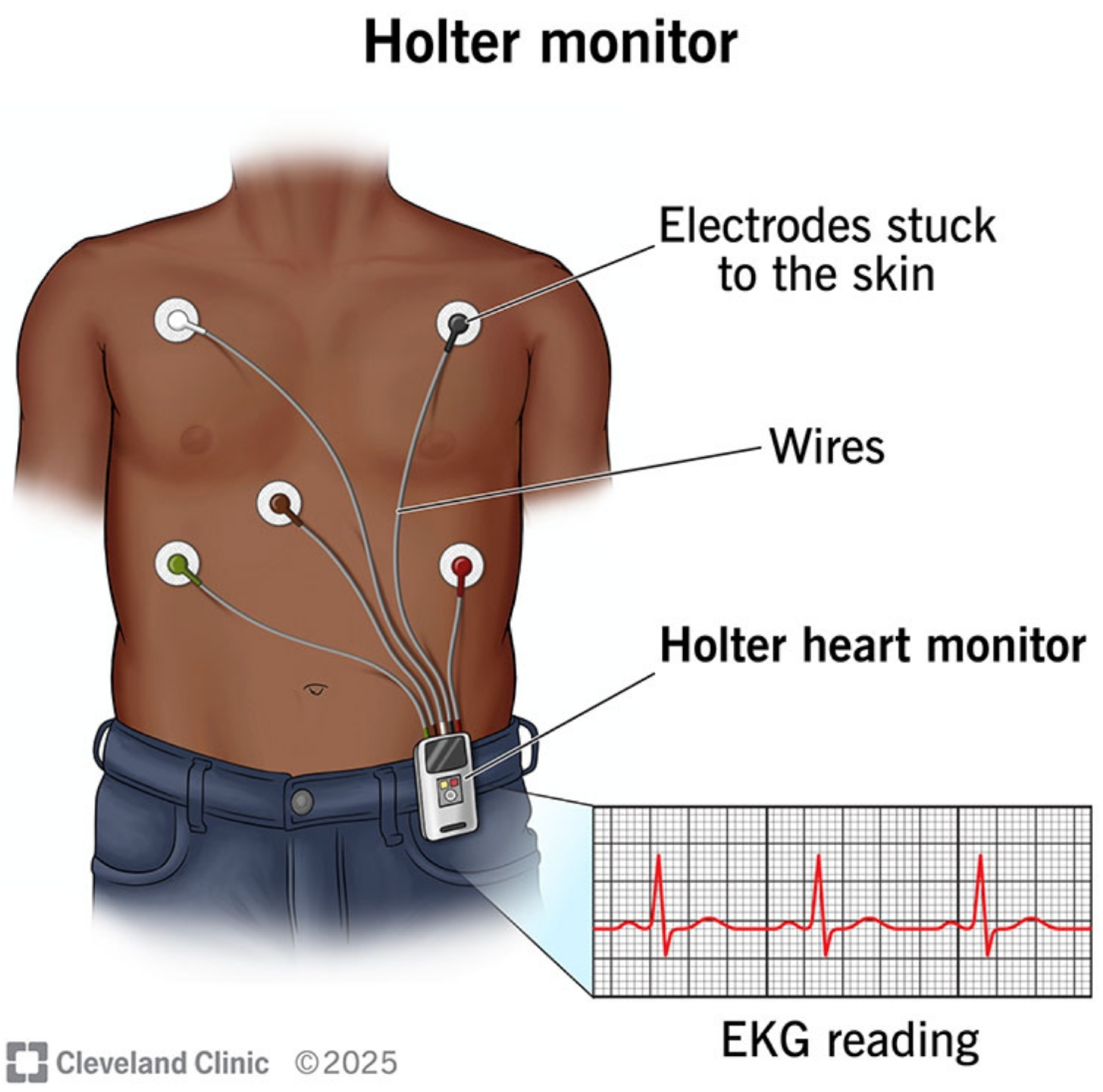
1. Location of Murmur

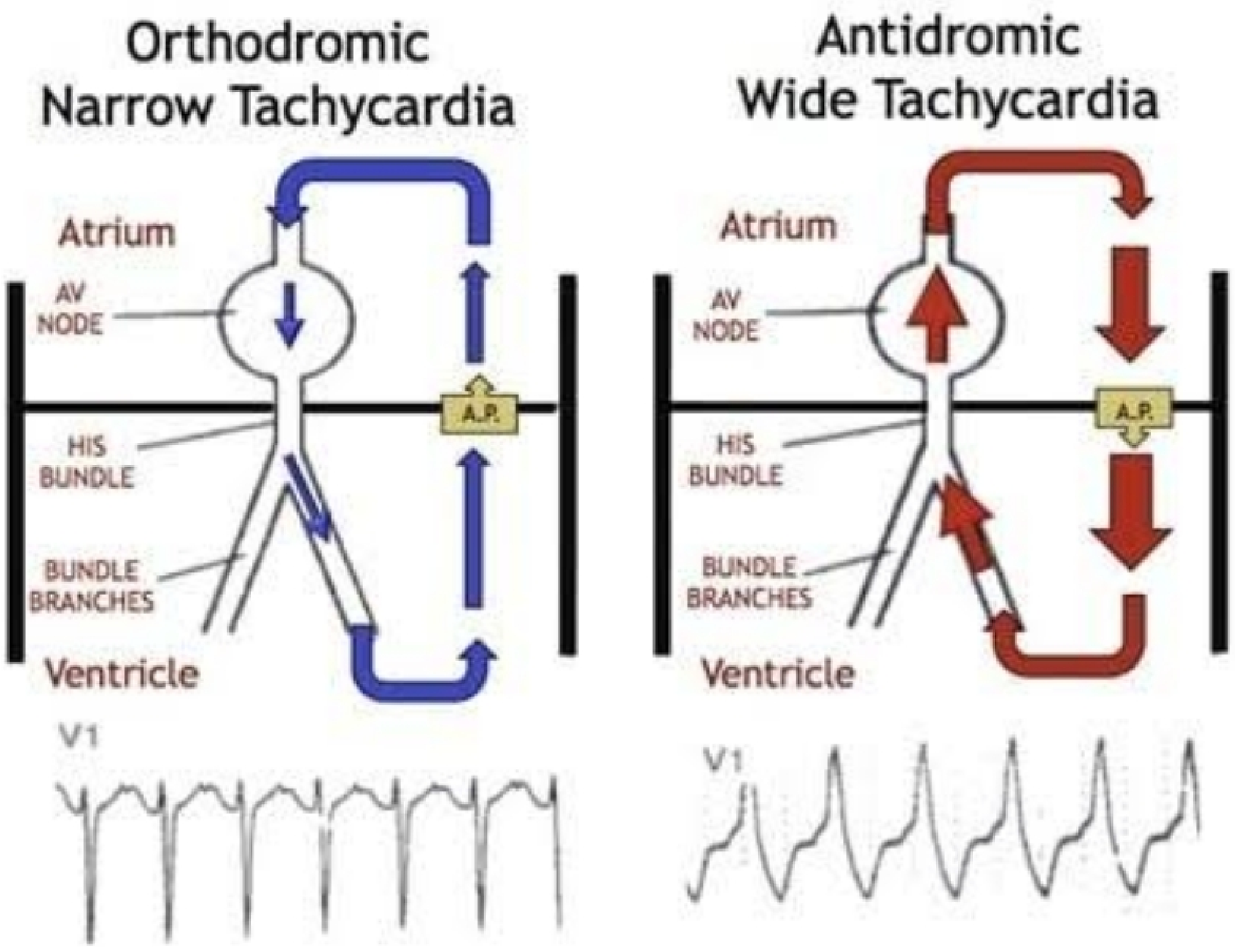


3. Murmur Maneuvers

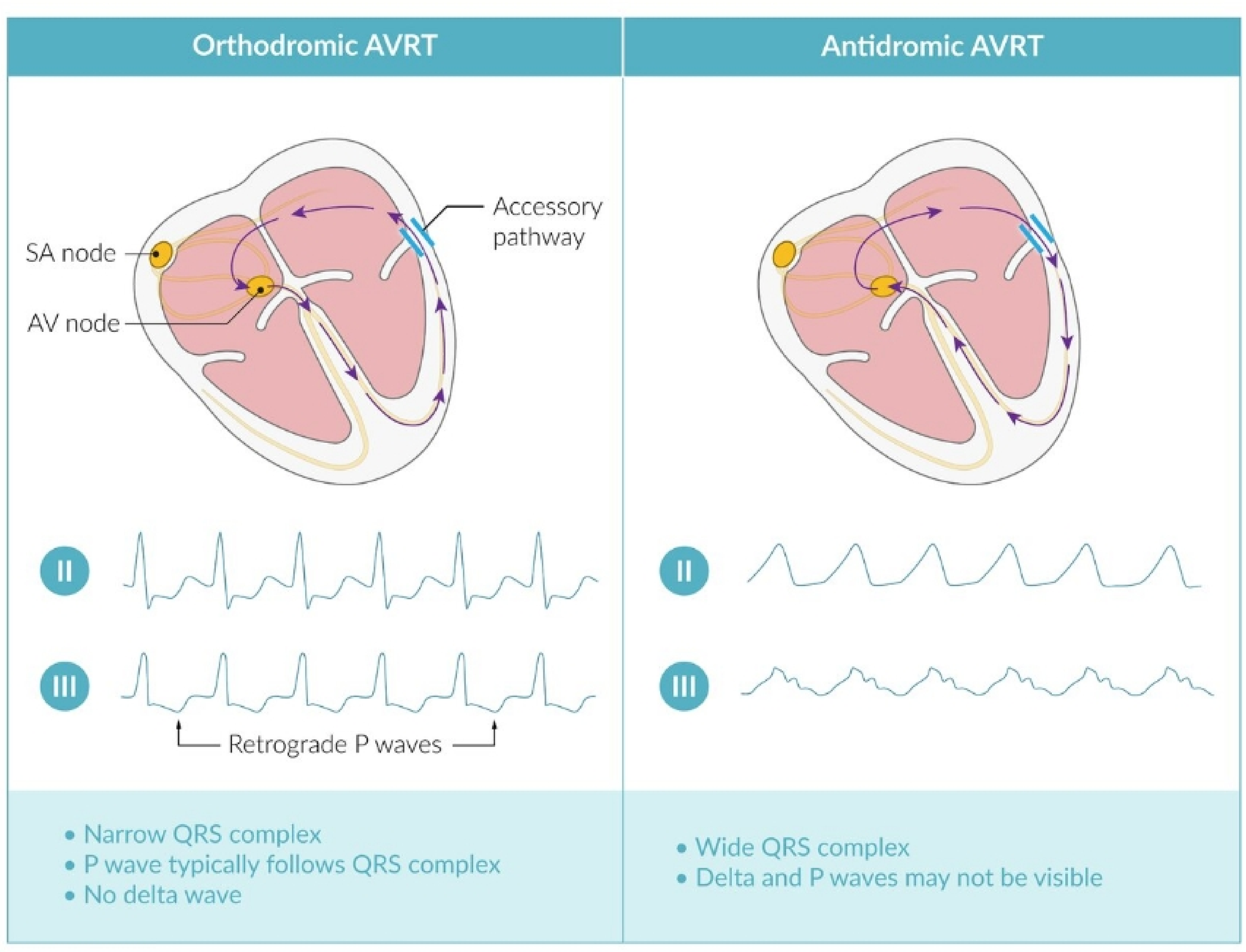
Maneuver	Effects on Murmur
Inspiration (↑ RA venous return)	↑ R side murmurs ↓ L side murmurs
Leaning Forward (bring AV close to chest wall)	↑ Aortic Murmurs (ex: AS/AR)
↑ Venous Return (Squat/Leg raise)	↑ All Murmurs Exception: MVP, HCM (↓ Murmur)
↑ Afterload (handgrips)	↑ AR/MR Murmur ↓ AS, MVP, HCM Murmur

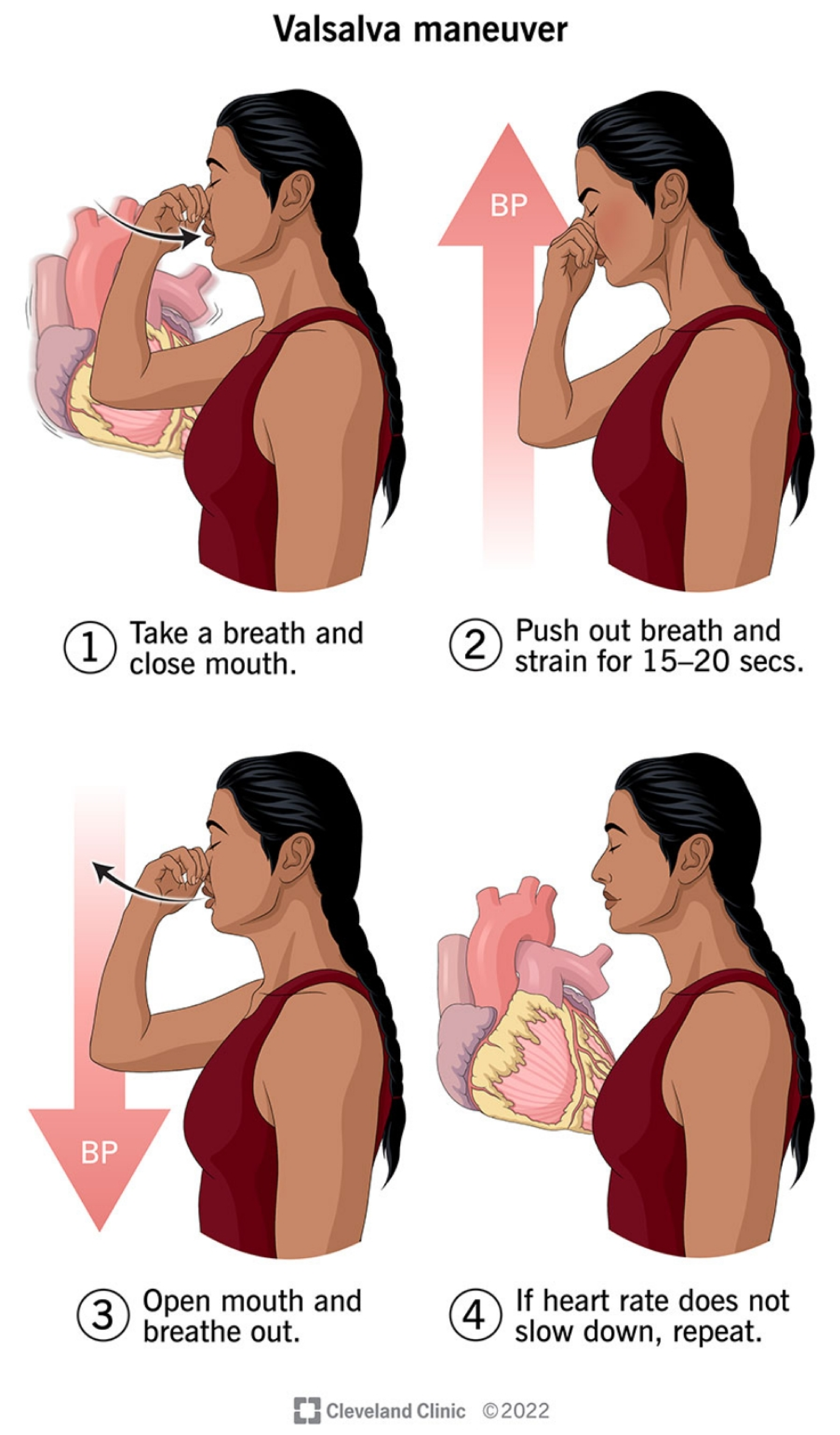
Maneuver	Effects on Murmur
Expiration (↑ LA venous return)	↑ L side murmurs ↓ R side murmurs
Lateral decubitus (bring MV close chest wall)	↑ Mitral Murmurs (ex: MS/MR)
↓ Venous Return (Valsalva/Stand)	↓ All Murmurs Exception: MVP, HCM (↑ Murmur)
↓ Afterload (amyl nitrate)	↓ AR/MR Murmur ↑ AS, MVP, HCM Murmur


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

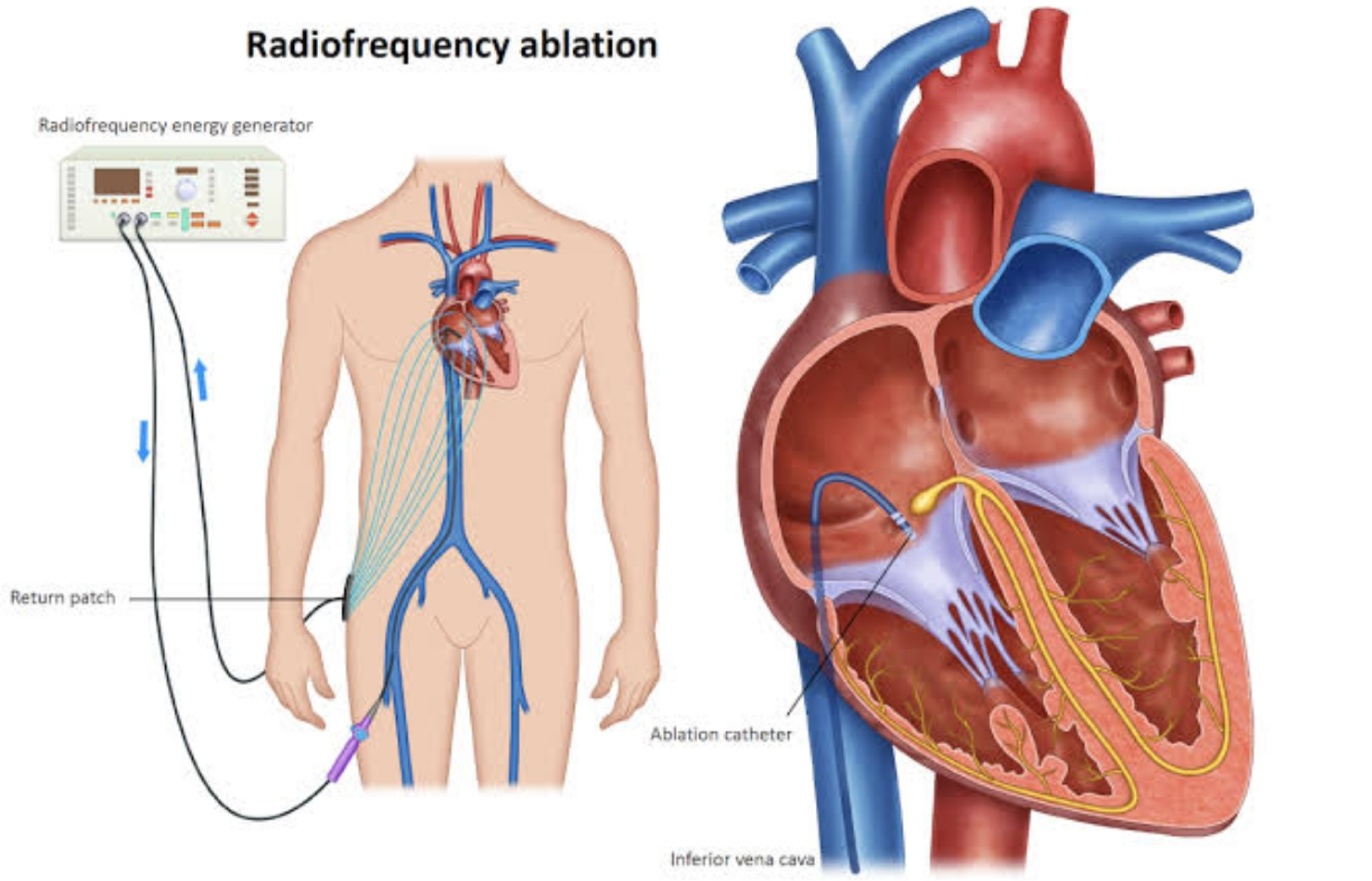


Orthodromic AVRT	• Caused by a reentry circuit that conducts antegrade through the AV node and retrograde through an accessory pathway	• HR: 120–250/minute • Rhythm: regular • Narrow QRS complex • P wave follows QRS complex
Antidromic AVRT	• Caused by a reentry circuit that conducts antegrade through an accessory pathway and retrograde through the AV node	• HR: 120–250/minute • Rhythm: regular • Wide QRS complex • Shortened PR interval





- **Applications**
  - Treatment of supraventricular tachycardia (e.g., AVNRT)
  - Evaluate conditions of the heart
    - Augments heart sounds on physical exam (e.g., earlier click in mitral valve prolapse and louder murmur in hypertrophic obstructive cardiomyopathy)
    - Decreases heart sounds intensity (most valve stenosis or regurgitation murmurs)
  - Test for hernia or varicocele (increased intraabdominal pressure → bulging)
  - Diagnosis of urinary stress incontinence (urine leakage during the maneuver)
  - Measure to normalize middle-ear pressure (e.g., in diving)
- **Contraindications**: include recent myocardial infarction, aortic stenosis, and glaucoma. [18]



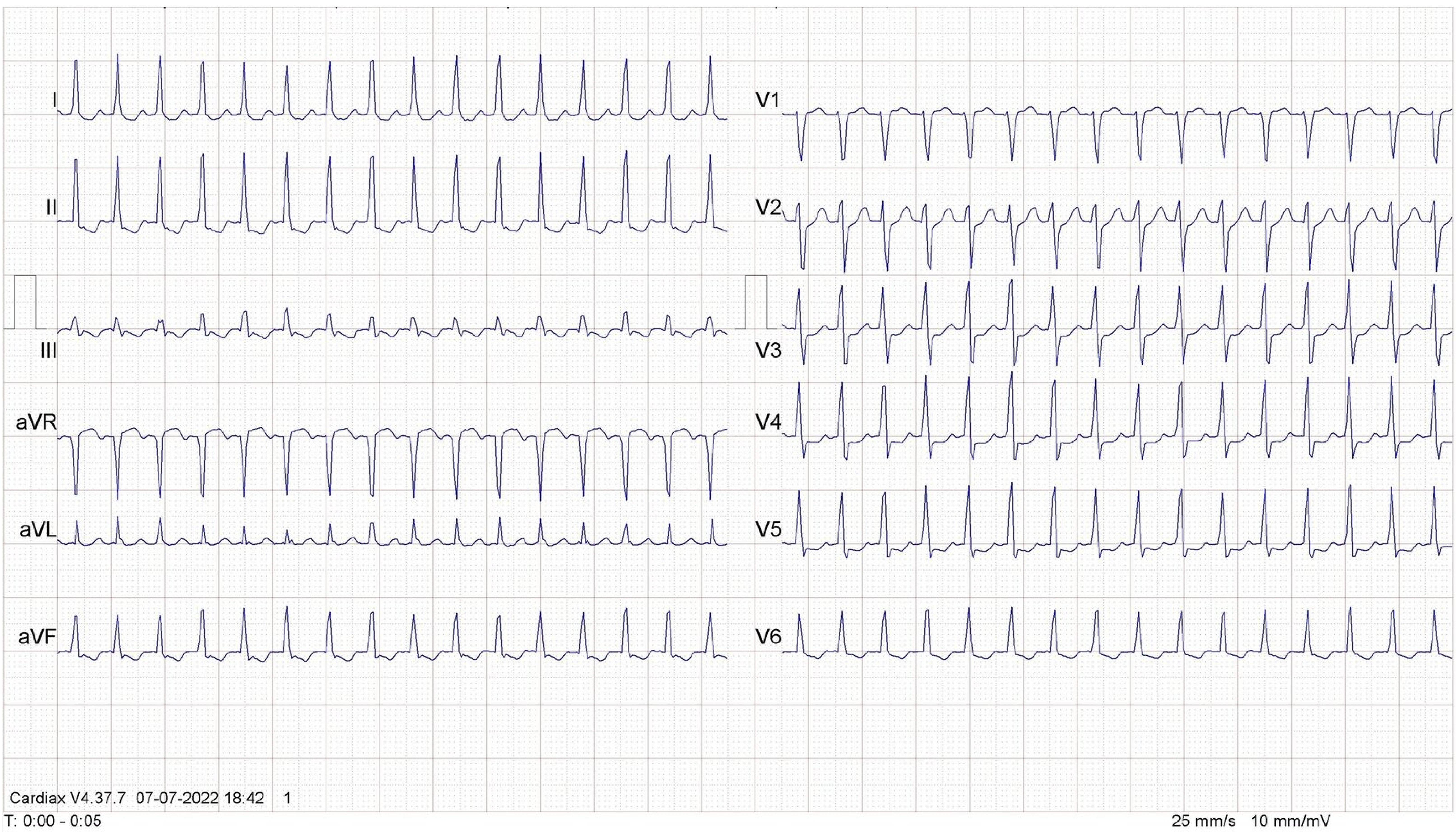
Catheter ablation

- **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)

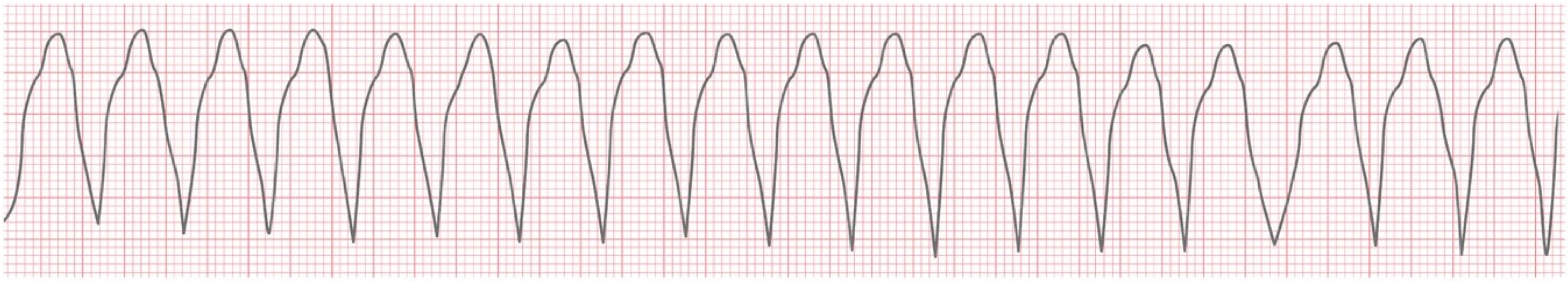
ECG findings in AVNRT [24][25]

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

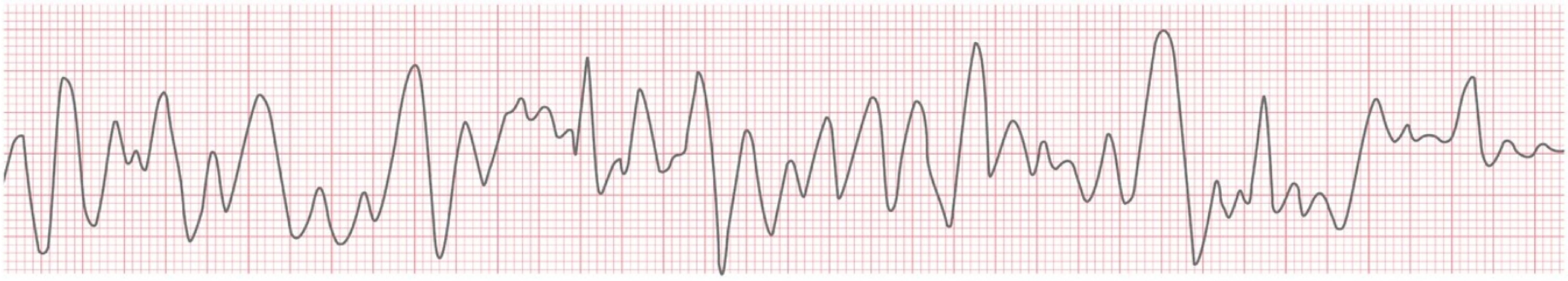
- Heart rate typically 150–220/minute
- Typically narrow QRS complexes (a wide QRS complex may be seen if there is aberrant conduction)
- Regular rhythm
- P wave **is typically not visible** (it falls in or is "buried" in the QRS complex)



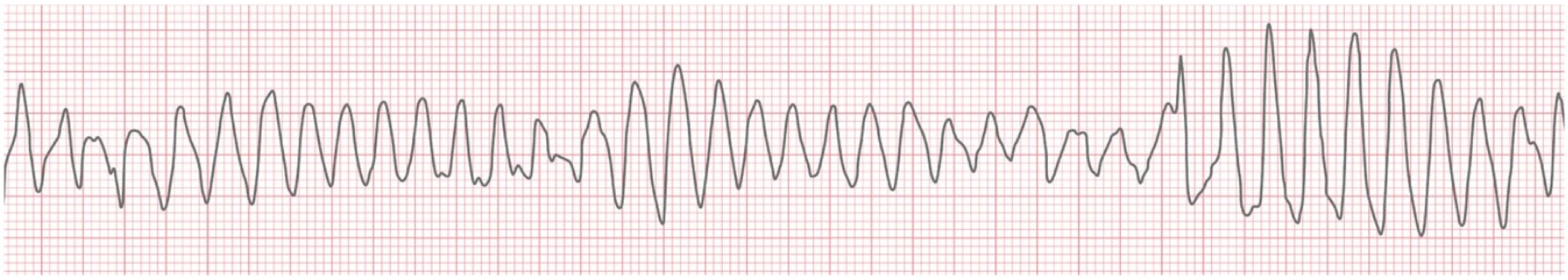
Monomorphic ventricular tachycardia



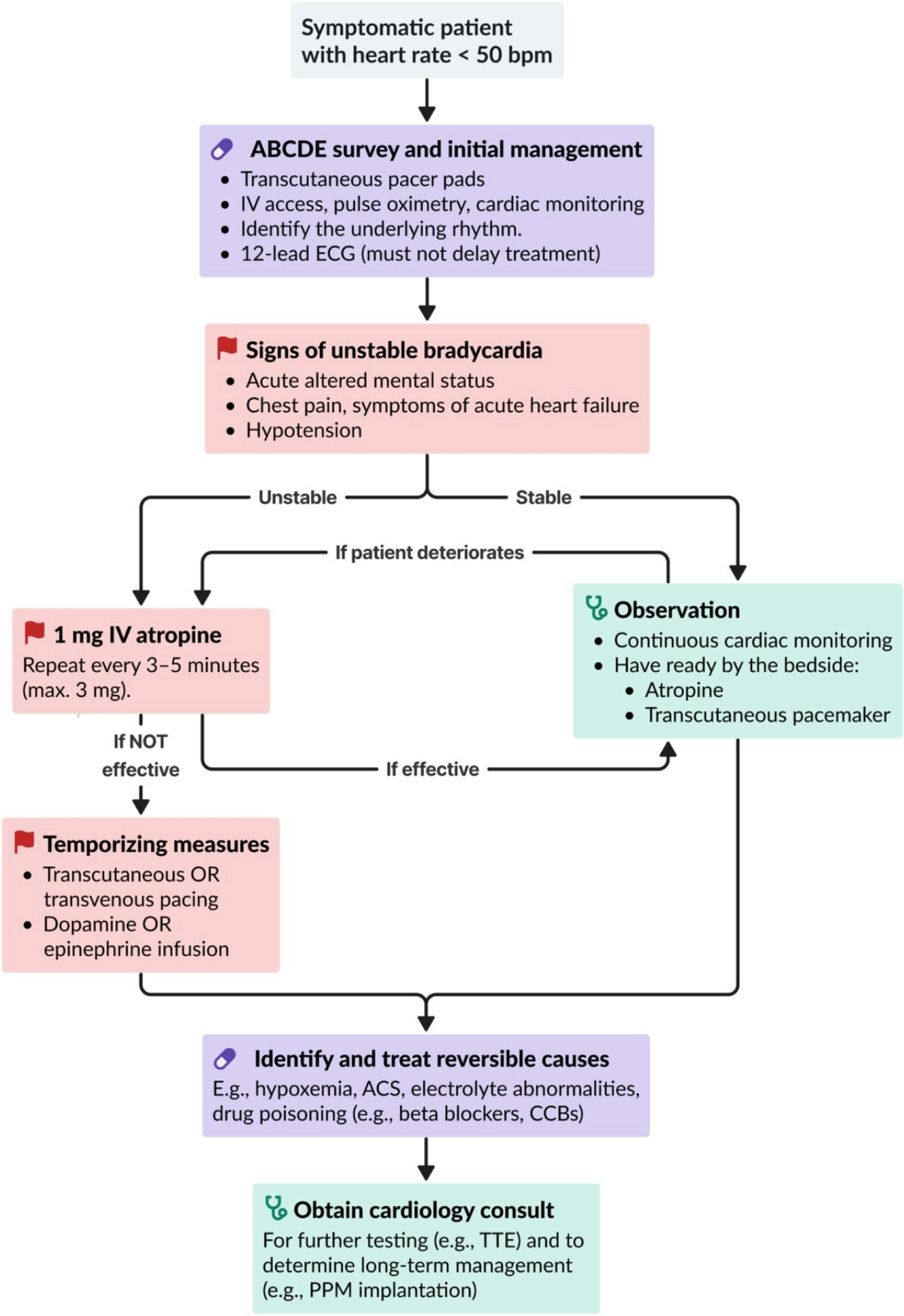
Polymorphic ventricular tachycardia



Torsades de pointes



Management of bradycardia



Overview of bradyarrhythmias

Overview of common bradyarrhythmias by ECG findings		
	Narrow QRS complex	**Wide QRS complex**
Regular rhythm	• Sinus bradycardia • First-degree AV block • Second-degree AV block, Mobitz II, with fixed conduction ratio	• Third-degree AV block • Second-degree AV block, Mobitz II, with fixed conduction ratio

Irregular rhythm	• Atrial fibrillation
	• Atrial flutter with variable AV block
	• Second-degree AV block
	  ◦ Mobitz I
	  ◦ Mobitz II
		• Second-degree AV block, Mobitz II
		• Combination of atrial fibrillation and any degree of AV block

Diagnostic Approach to
Pericardial Diseases

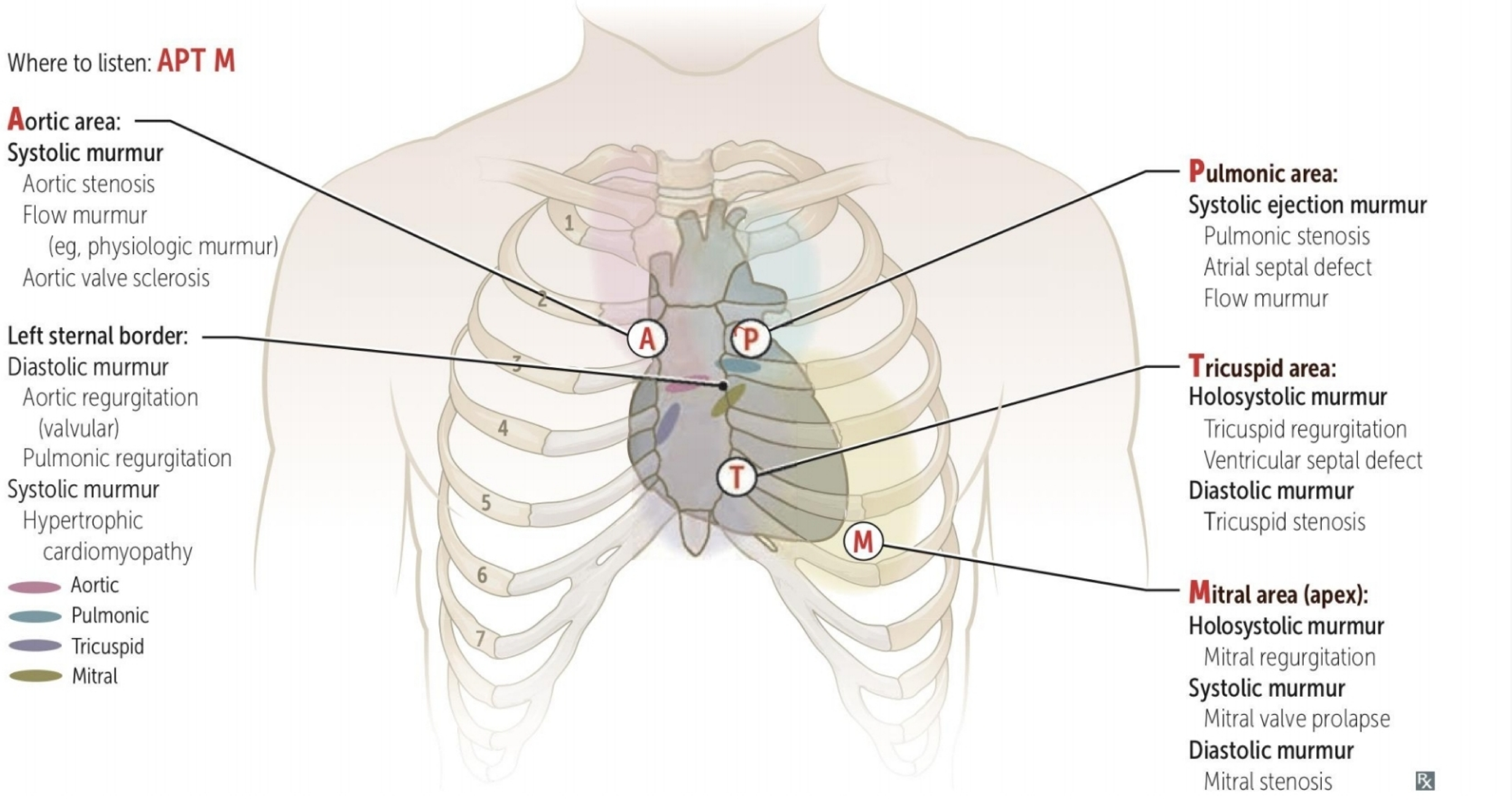
Pericardial Disease	Clinical Presentation	ECG	Echocardiogram	Additional Studies/comments
Acute Pericarditis	▪ Pleuritic Chest pain ▪ Friction rub	▪ Diffuse ST elevation and ▪ PR-segment Depression	▪ Possible Pericardial Effusion	▪ ≥ 2/4 findings suggests pericarditis
Constrictive Pericarditis	▪ Right Heart Failure ▪ Kussmaul's Sign ▪ Pericardial Knock	▪ Low voltage QRS	▪ Thick pericardium ▪ Septal bounce ▪ Abrupt ↓ in Ventricular filling	▪ Cardiac CT/MRI and Cath (differentiates between RCM)
Pericardial Effusion	▪ Muffled Heart sounds ▪ No Becks Triad or Pulsus Paradoxus	▪ Low voltage QRS ▪ Electrical alternans	▪ Pericardial Effusion	
Cardiac Tamponade	▪ Becks Triad ▪ Pulsus Paradoxus	▪ Low voltage QRS ▪ Electrical alternans	▪ Pericardial Effusion	

Treatment of
Pericardial Diseases

Pericardial Disease	Treatment
Acute Pericarditis	▪ NSAIDS + Colchicine (Viral) ▪ ASA + Colchicine (Post MI) ▪ Dialysis (Uremia)
Constrictive Pericarditis	▪ Pericardiectomy
Pericardial Effusion	▪ Observation (serial TTE) ▪ Pericardial window (if recurrent effusions)
Cardiac Tamponade	▪ Stabilize Hemodynamics (IVF and Pressors) ▪ Pericardiocentesis (non-hemopericardium) ▪ Surgical drain + repair (hemopericardium)

Differentiating
Constrictive vs **Restrictive**
Cardiomyopathy

	Constrictive Pericarditis	Restrictive Cardiomyopathy
Physical Exam	▪ Kussmaul's Sign ▪ Pericardial Knock	▪ Kussmaul's sign
Echocardiogram	▪ Thick pericardium ▪ Septal Bounce ▪ Abrupt ↓ in Ventricular filling	▪ Biatrial enlargement ▪ Diastolic dysfunction
Cardiac CT/MRI	▪ Thick pericardium	▪ Normal pericardium
Cardiac Catheterization	▪ Discordance of EDP	▪ Concordance of EDP



◀ 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

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

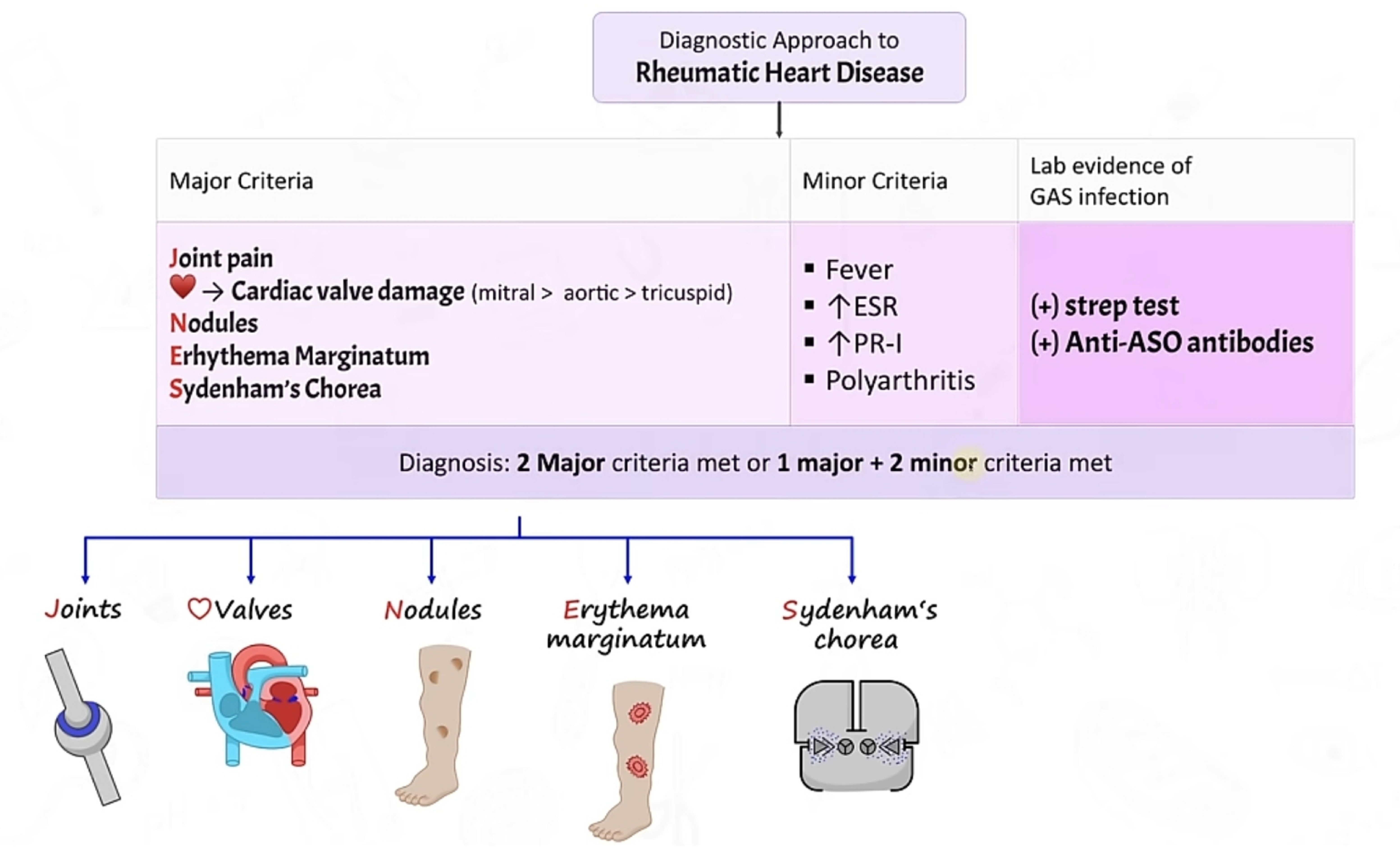
◀ Diagnostic criteria

- **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

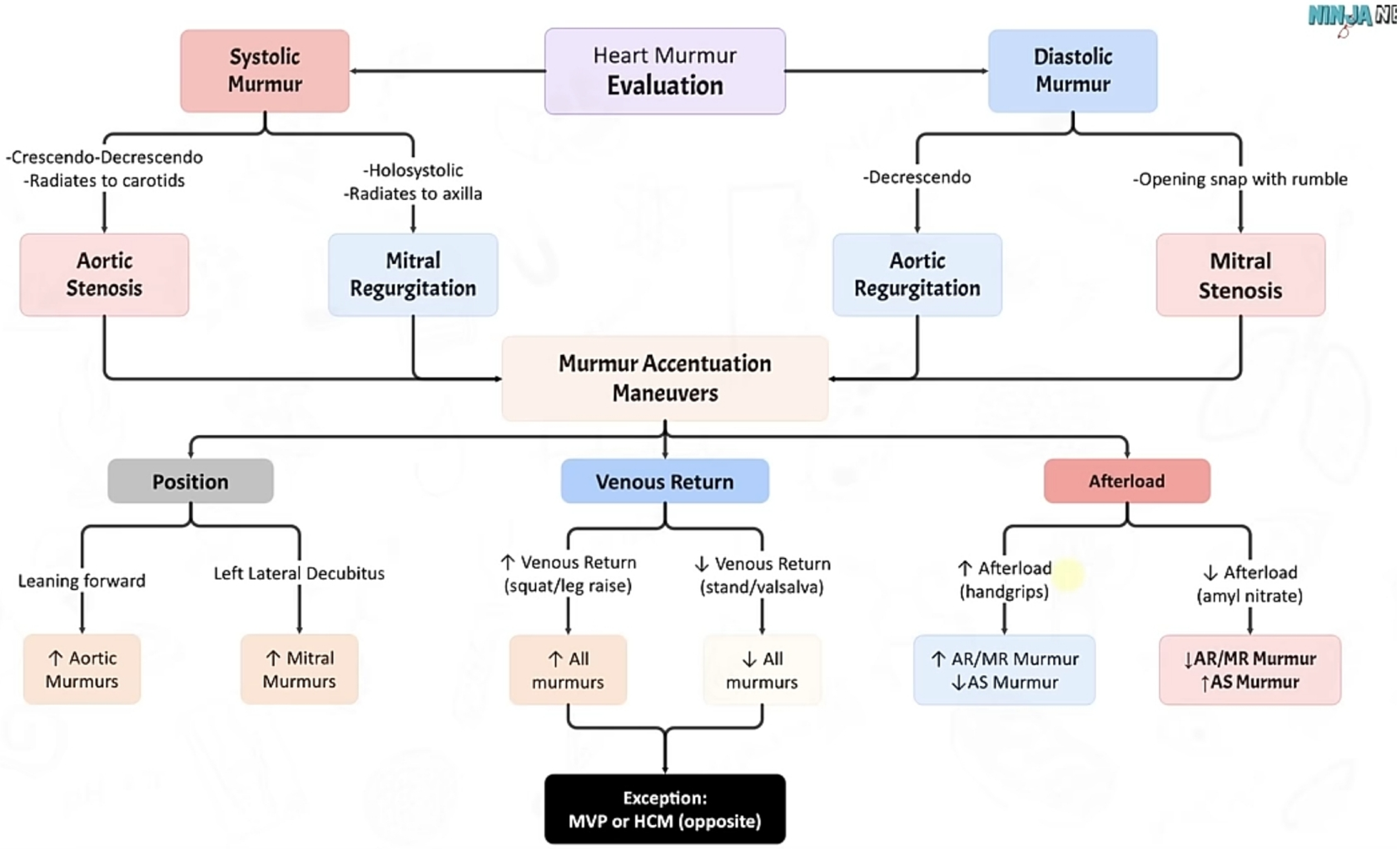
J♡NES

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 Approach to
Valvular Heart Diseases

↓

Murmur Auscultated

↓

Obtain
Echocardiogram and Cardiac Cathterization

↓

Type of VHD	Echocardiogram	Cardiac Catheterization
Aortic Stenosis	▪ ↓ blood flow across AV ▪ LV Hypertrophy	Narrow Pulse pressure
Aortic Regurgitation	▪ ↑ regurgitation jet across AV ▪ LV Dilation	Wide Pulse pressure
Mitral Stenosis	▪ ↓ blood flow across MV ▪ LA Dilation	LAP > LVP during diastole
Mitral Regurgitation	▪ ↑ regurgitation jet across MV ▪ LA and LV Dilation	↑ LAP during systole

Treatment of
Valvular Heart Diseases

↓

Manage Heart Failure Symptoms
Medically

↓

Type of VHD	Surgical Intervention	Indication for Surgical Intervention
Aortic Stenosis	TAVR or SAVR	Severe AS + ↑ Symptoms + LVEF < 50%
Aortic Regurgitation	SAVR	Acute HF or Severe AR refractory to medical therapy
Mitral Stenosis	Balloon Valvuloplasty	Severe mitral stenosis
Mitral Regurgitation	MV repair or replacement	Acute HF or Severe MR refractory to medical therapy

Mitral Stenosis
→ ↑ Afib → ↑Emboli
(Anticoagulate with Warfarin)

Rheumatic fever → Penicillin G

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**

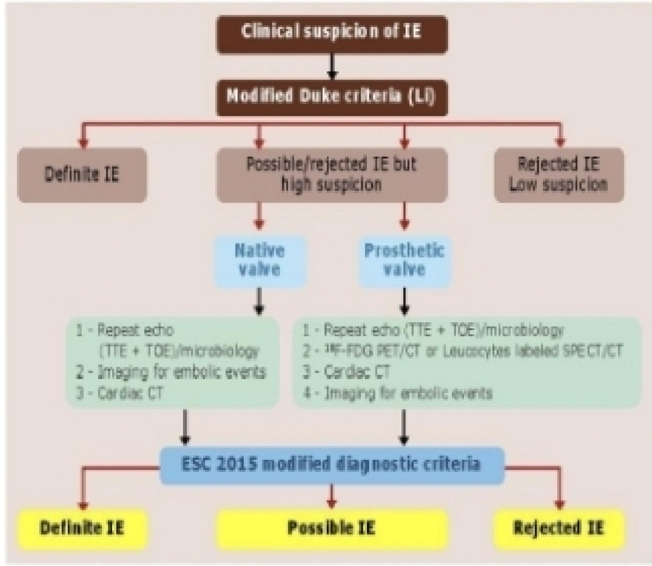
Minor criteria

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

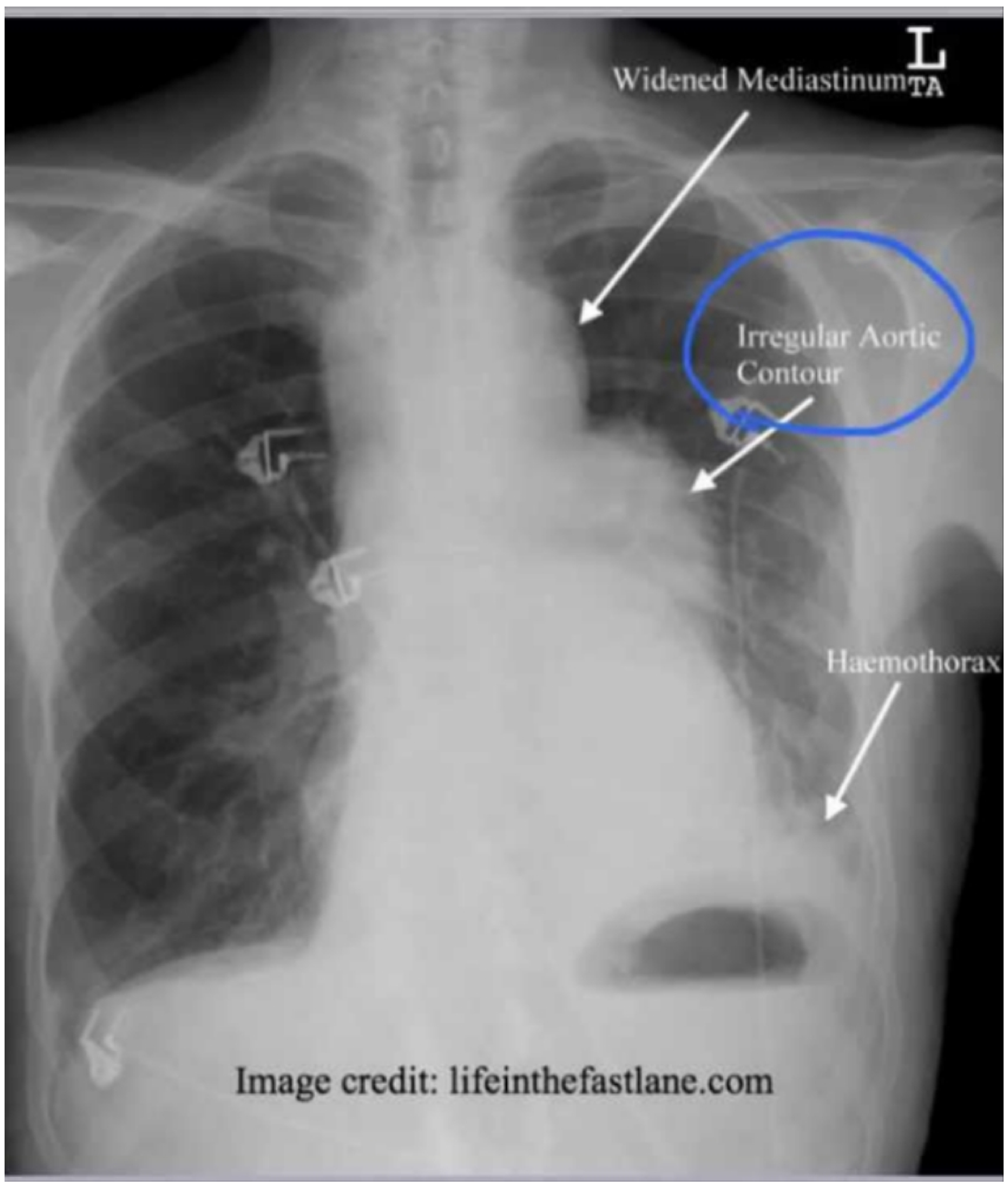
◀ Duke criteria

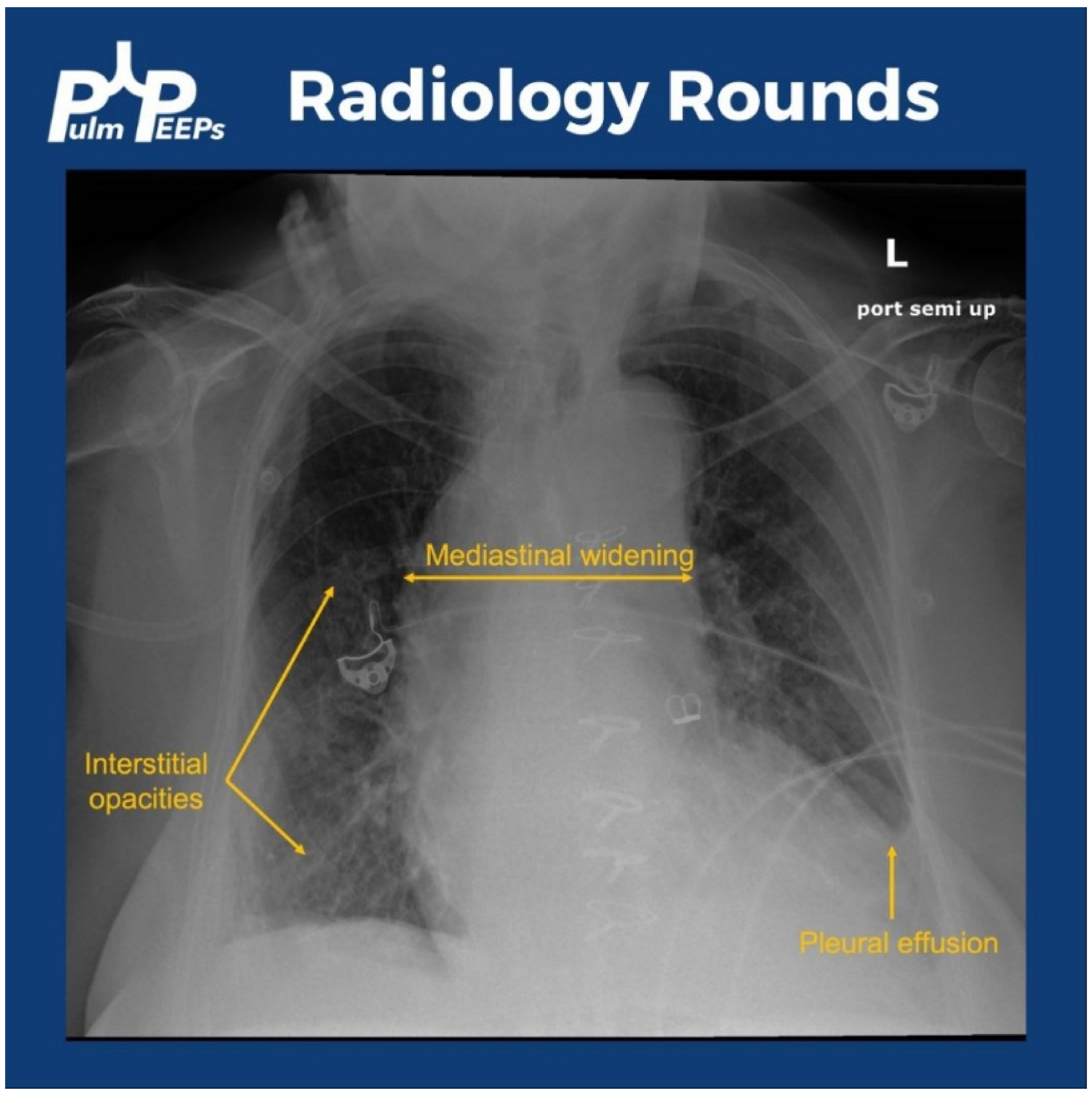
★ **Definitive IE:** (Begin treatment right away)
- **Clinical criteria:** Patients with 2 major, **OR** 1 major and 3 minor, **OR** 5 minor.
- **Pathologic criteria:** Microorganisms or pathologic lesions: demonstrated by culture or histology in a vegetation, or in a vegetation that has embolized, or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

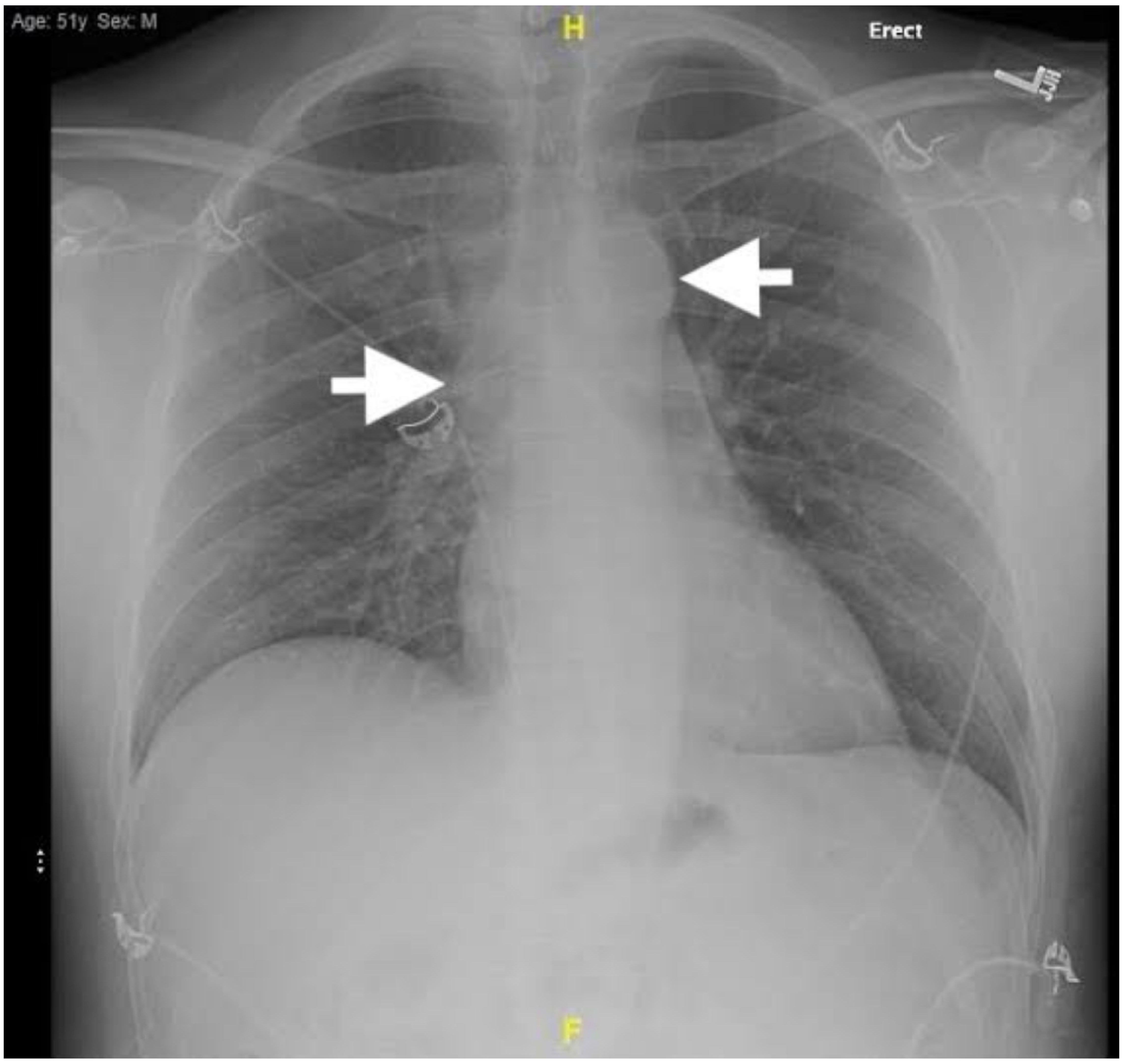
- **Possible IE:** (Requires further tests)
  - **Clinical criteria:** Patients with 1 major and 1 minor, **OR** 3 minor.
- **Rejected IE:**
  - Firm alternate Diagnosis for manifestation of IE
  - Resolution of manifestations of IE, with antibiotic therapy for ≤ 4 days
  - No pathologic evidence of IE at surgery or autopsy, after antibiotic therapy for ≤ 4 days)



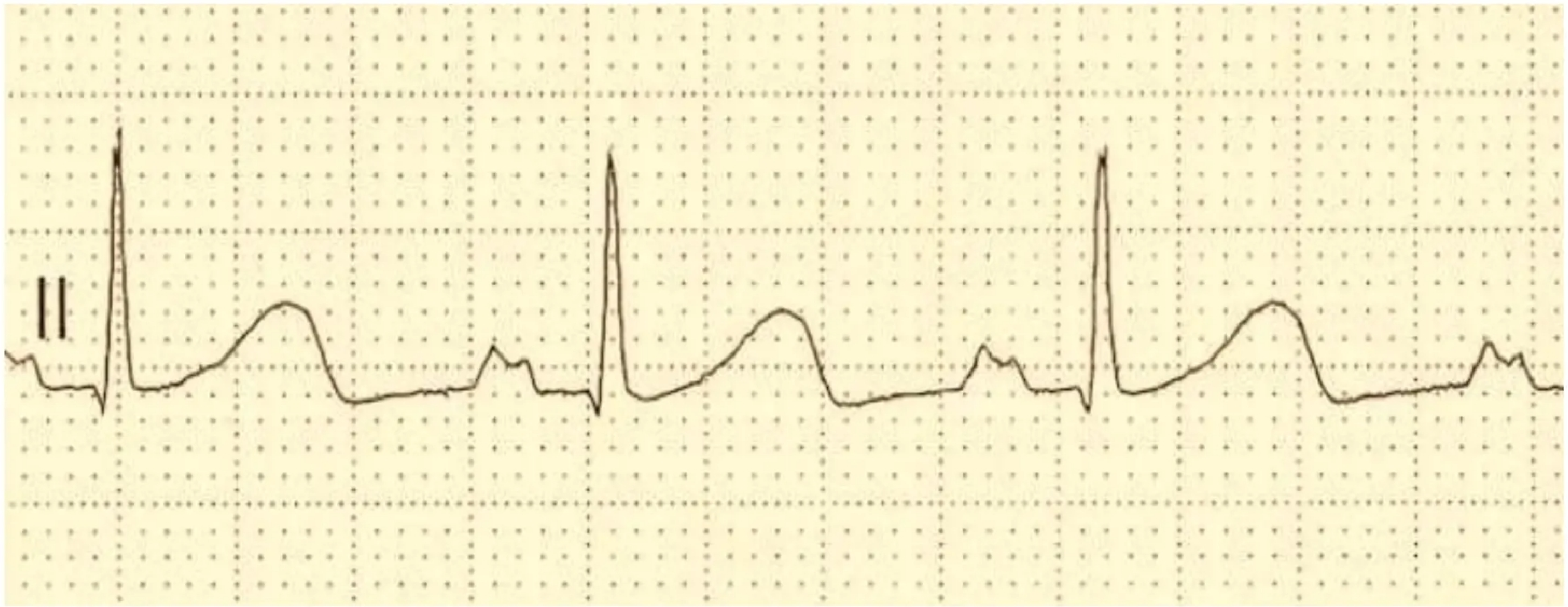
Widened mediastinum in Aortic dissection on chest X Ray

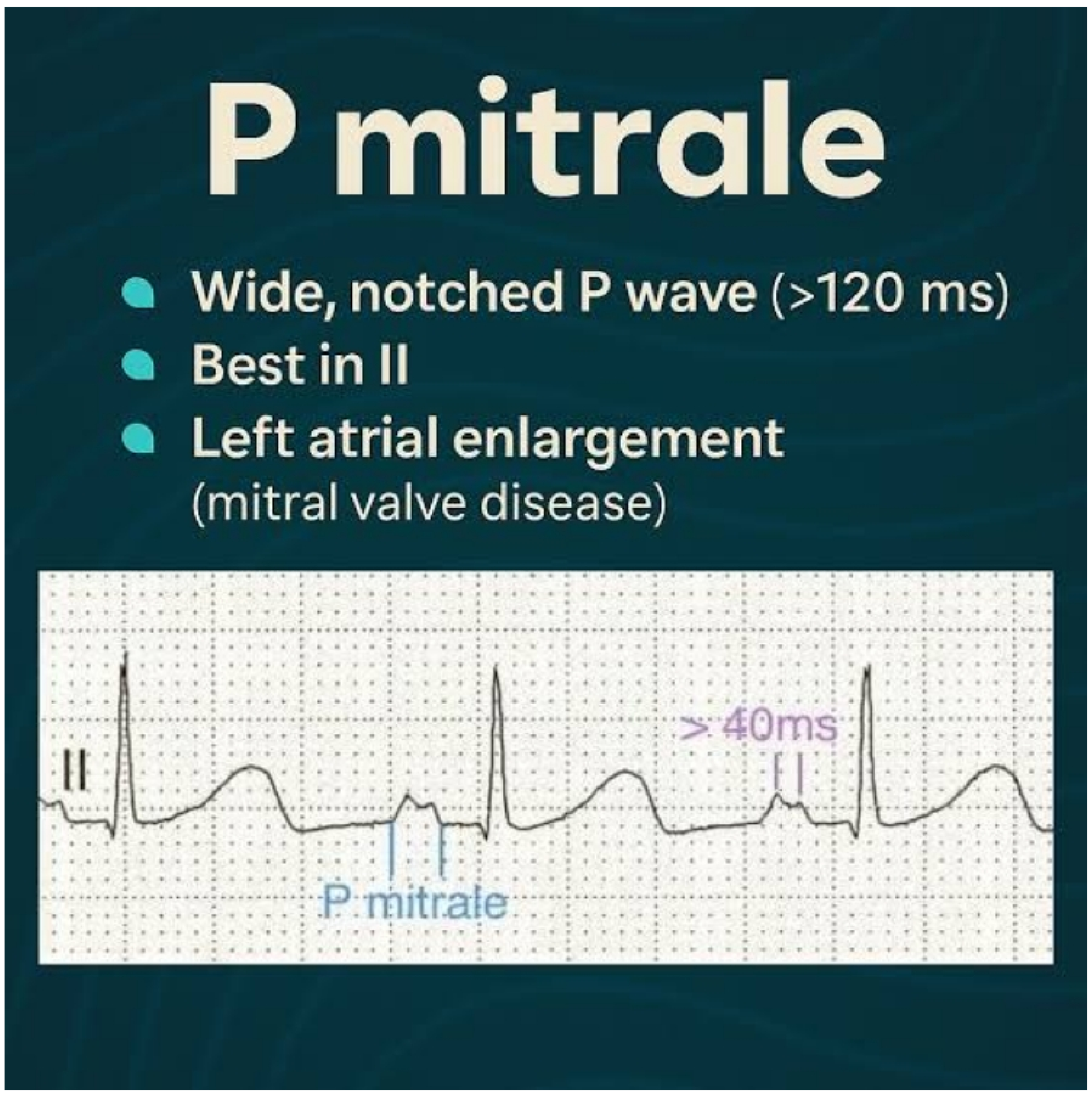




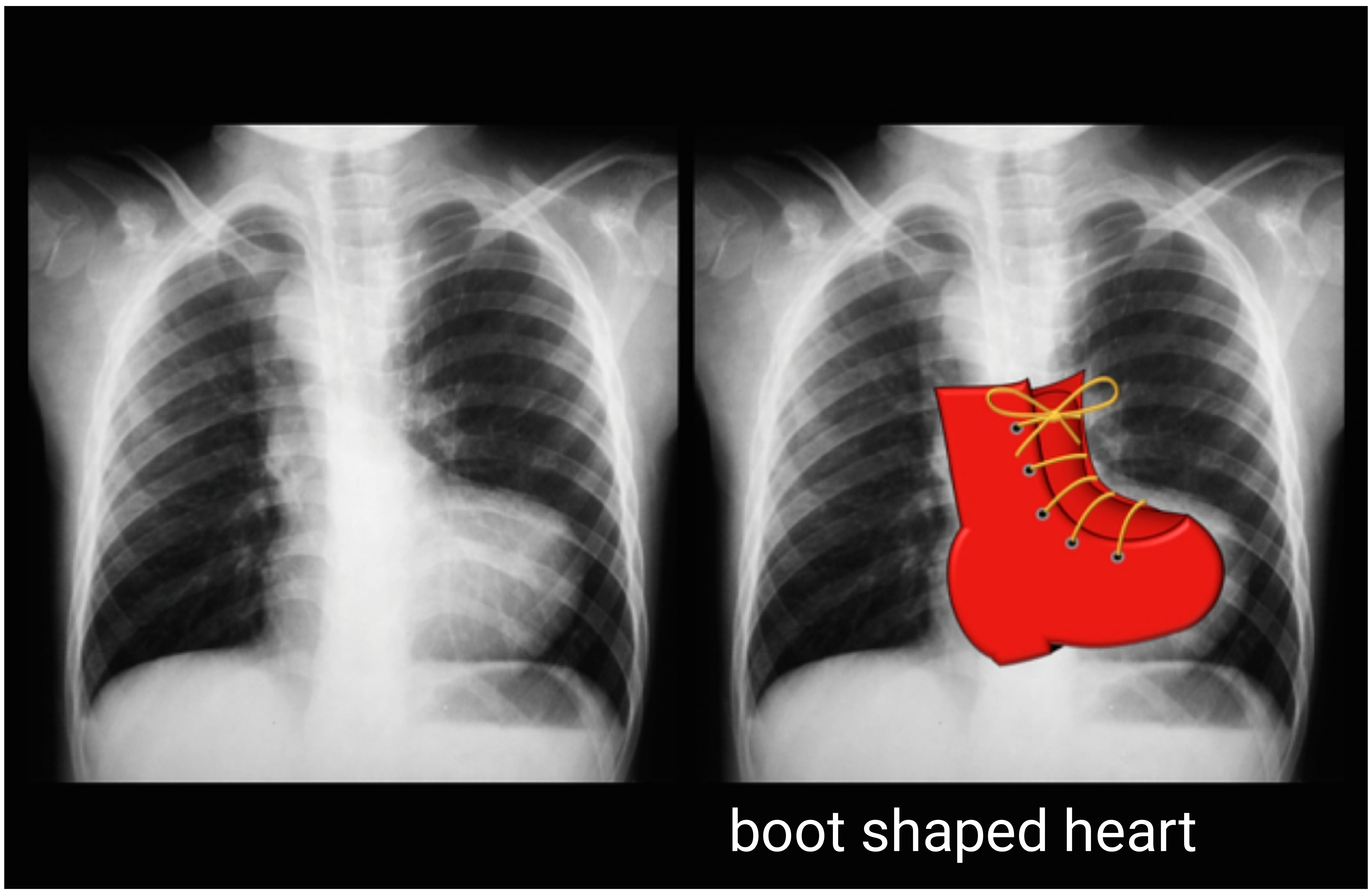


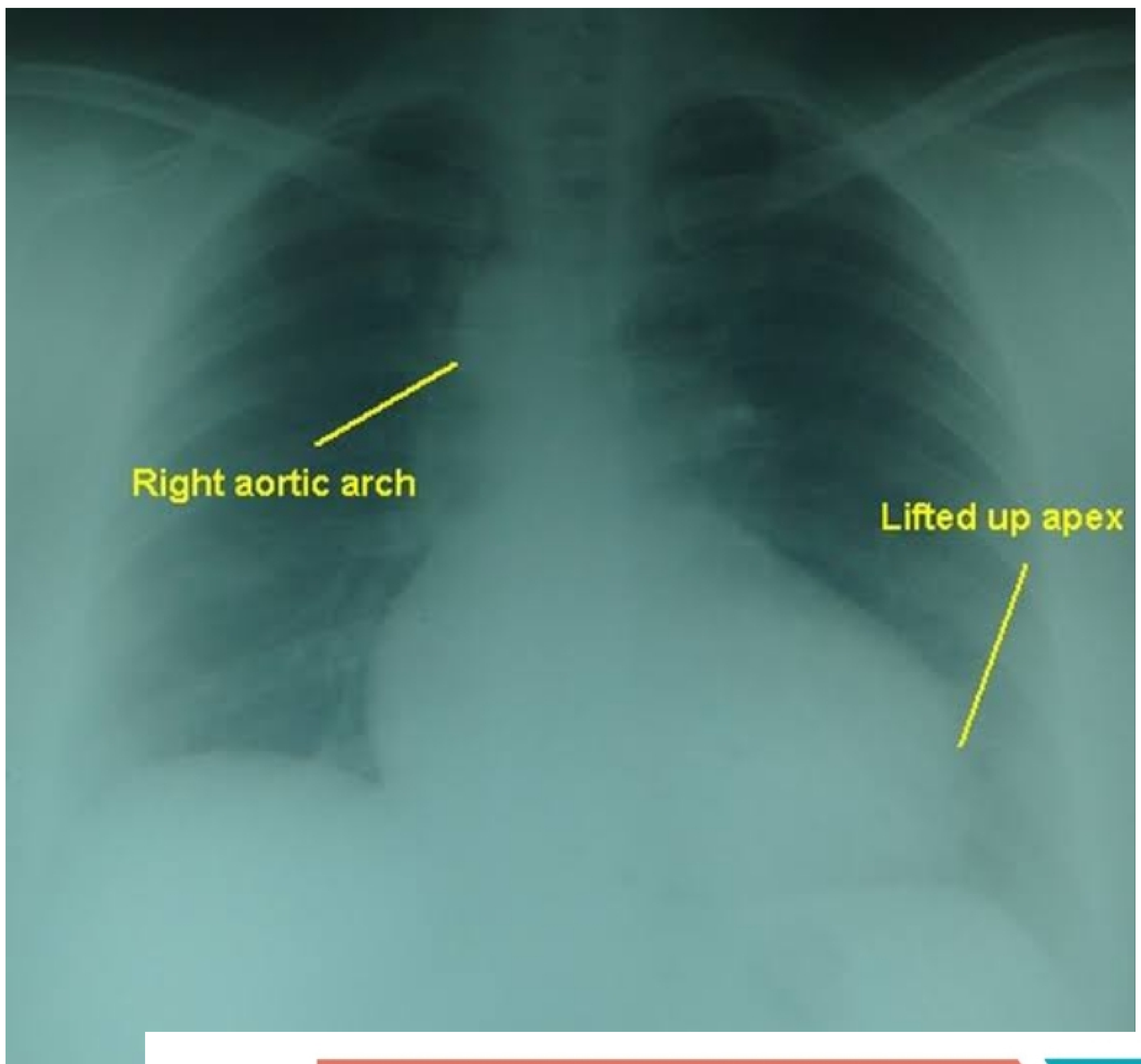
- **P Mitrale**
- The presence of broad, notched (bifid) P waves in lead II is a sign of **left atrial enlargement,** classically due to **mitral stenosis**

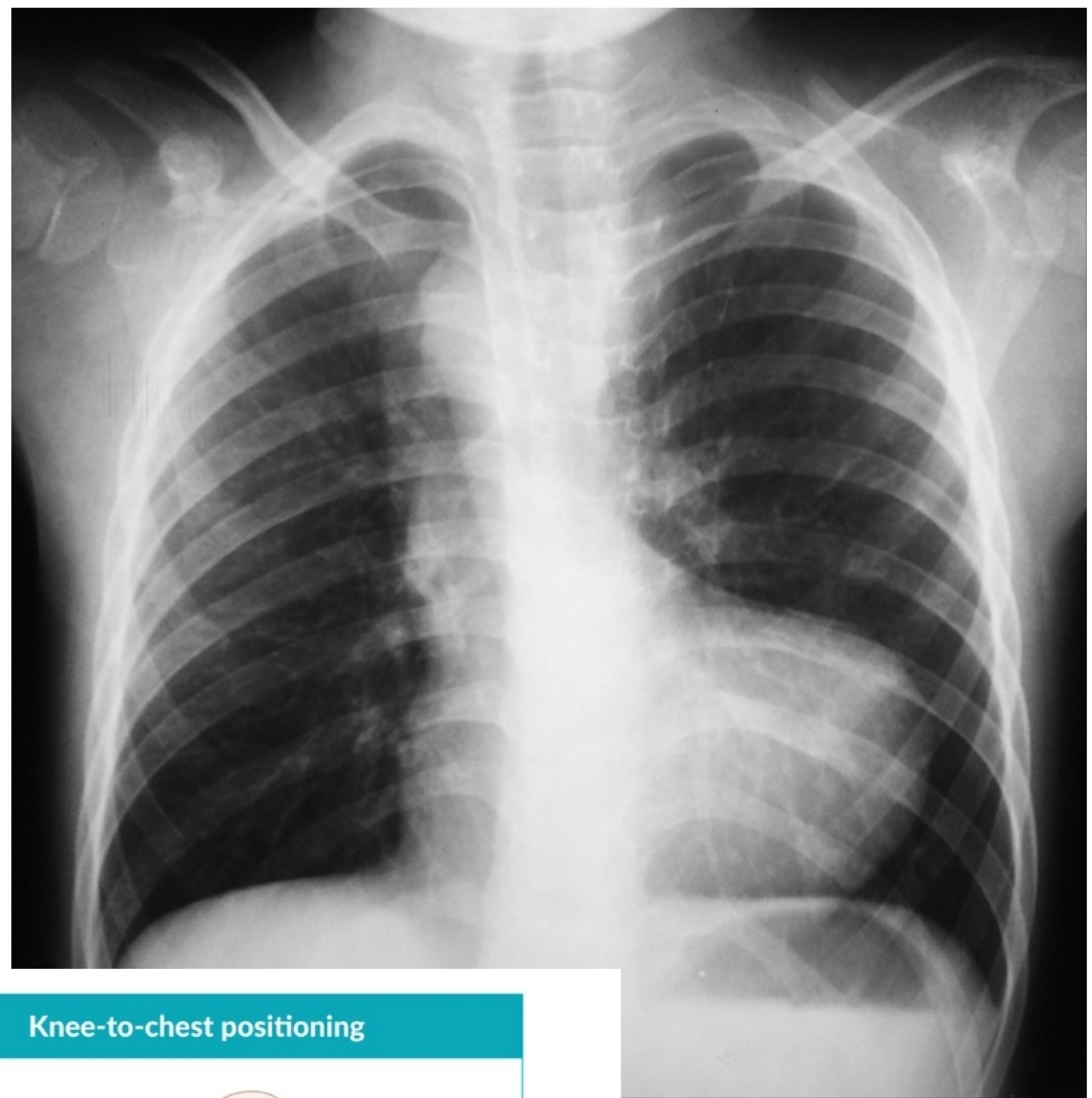


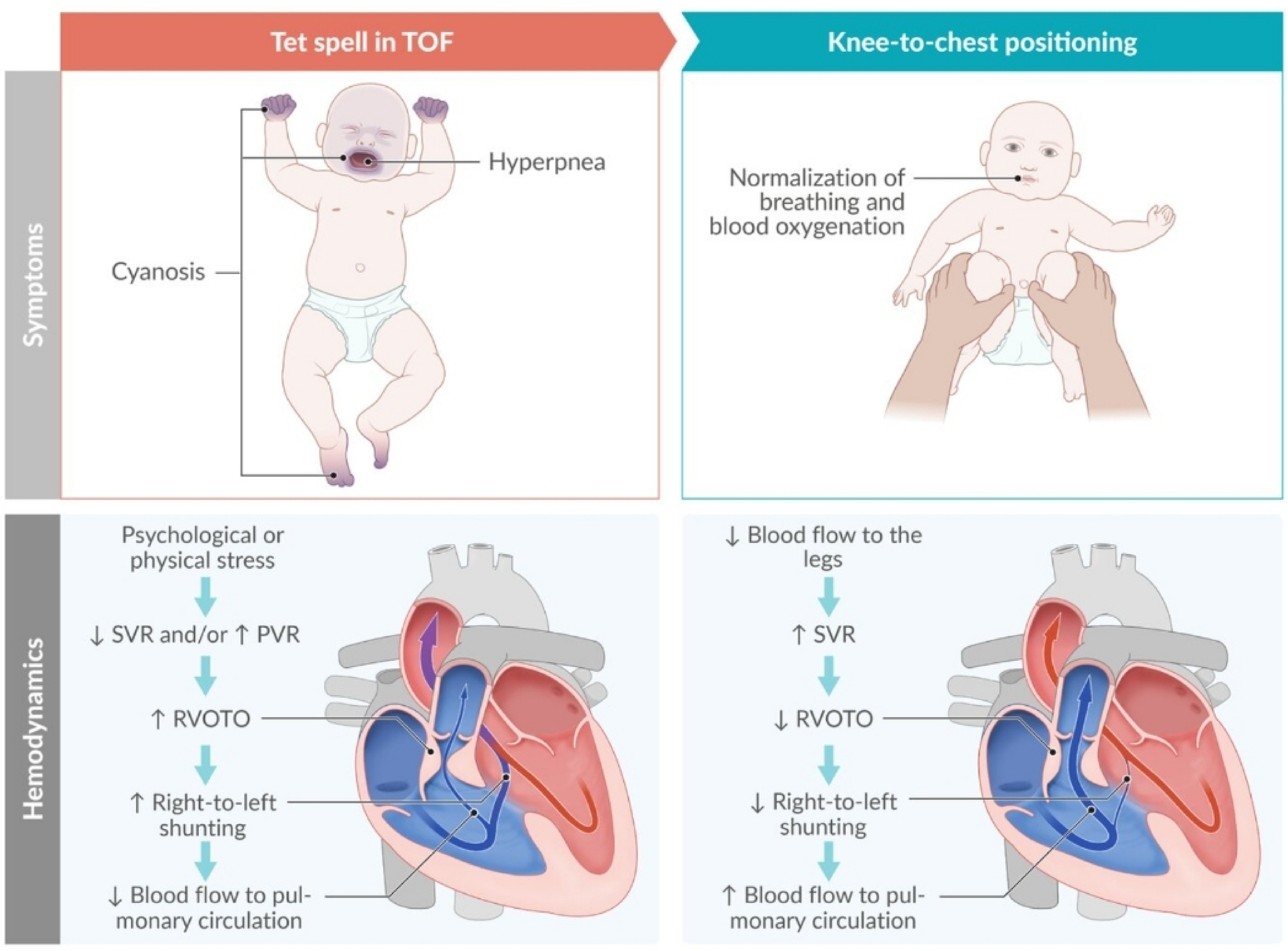


Tetralogy of fallot

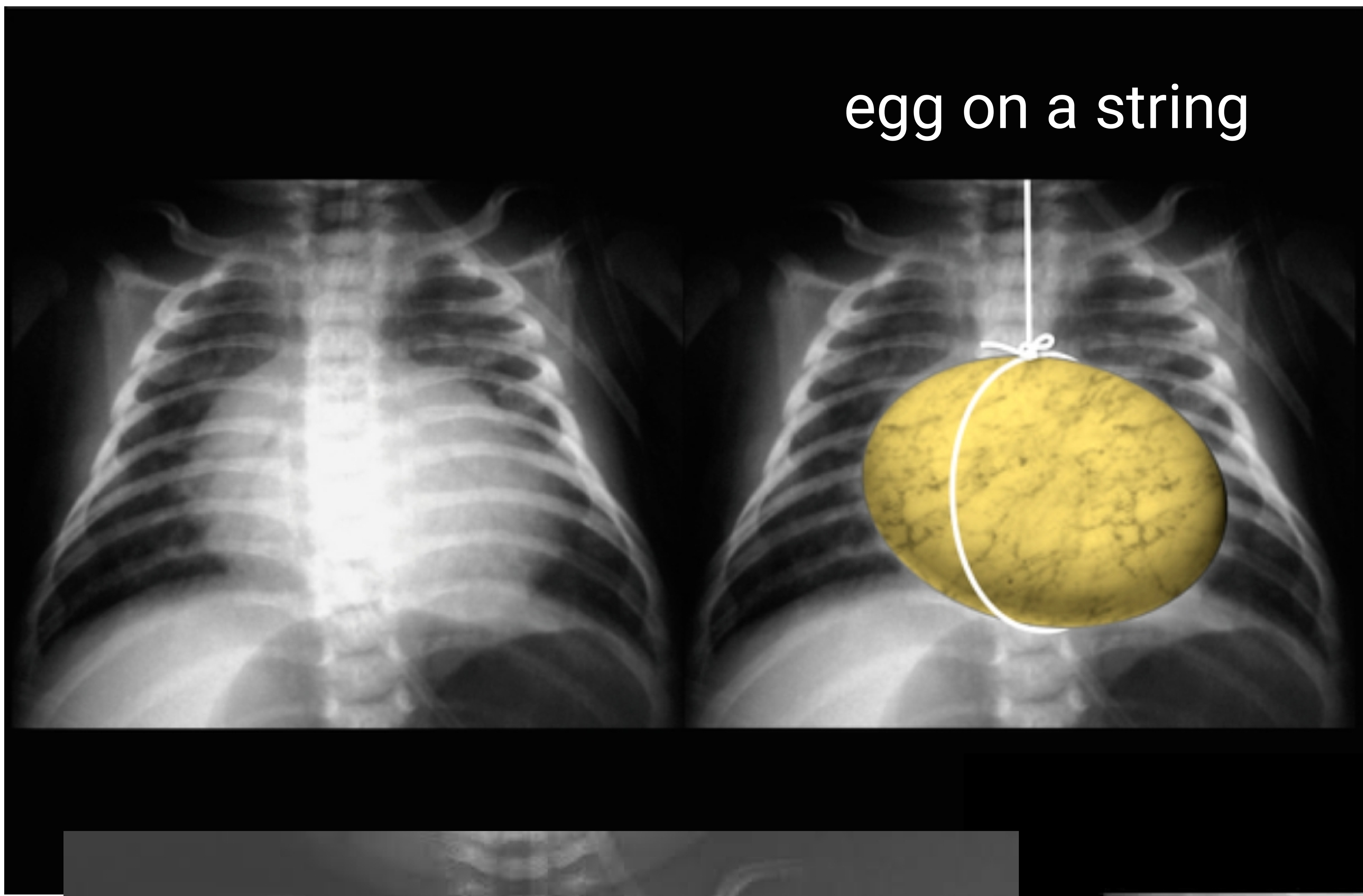








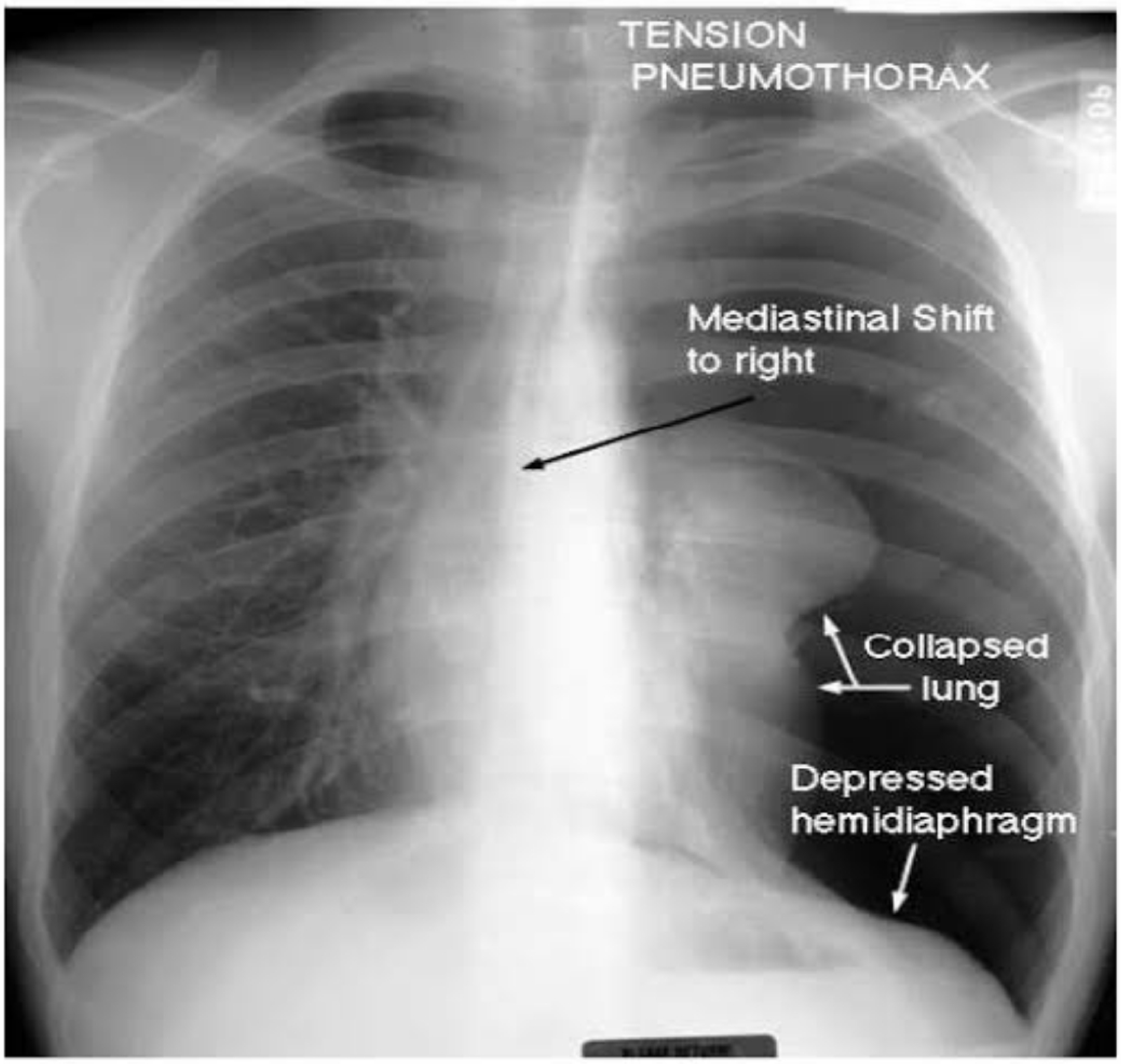
Transposition of great arteries

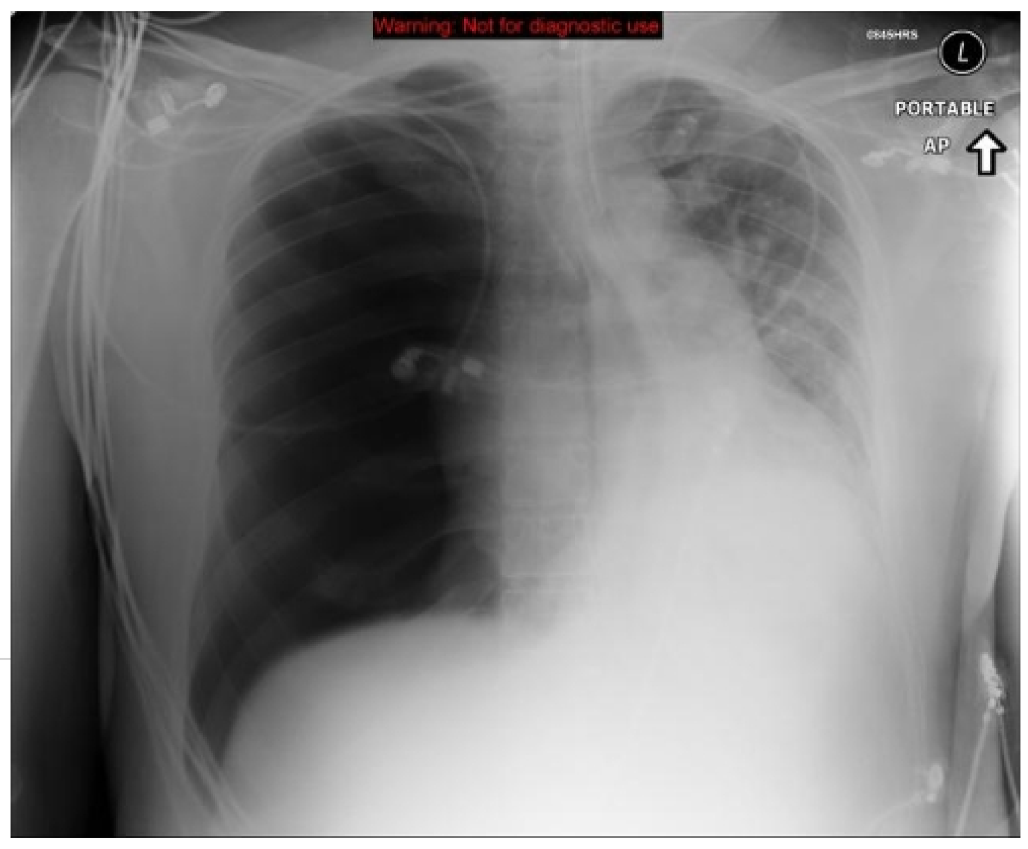


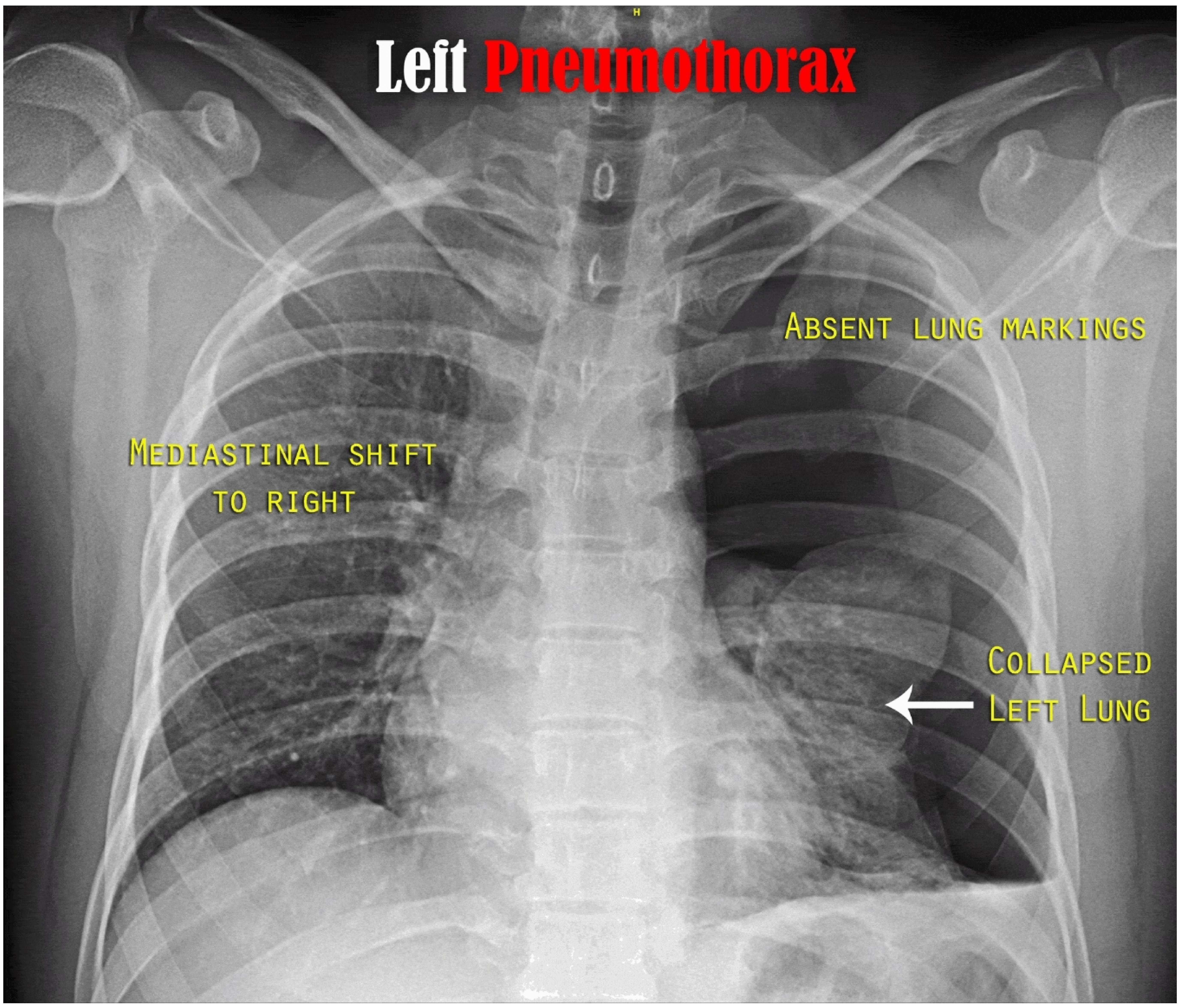
	Most common associated conditions and risk factors

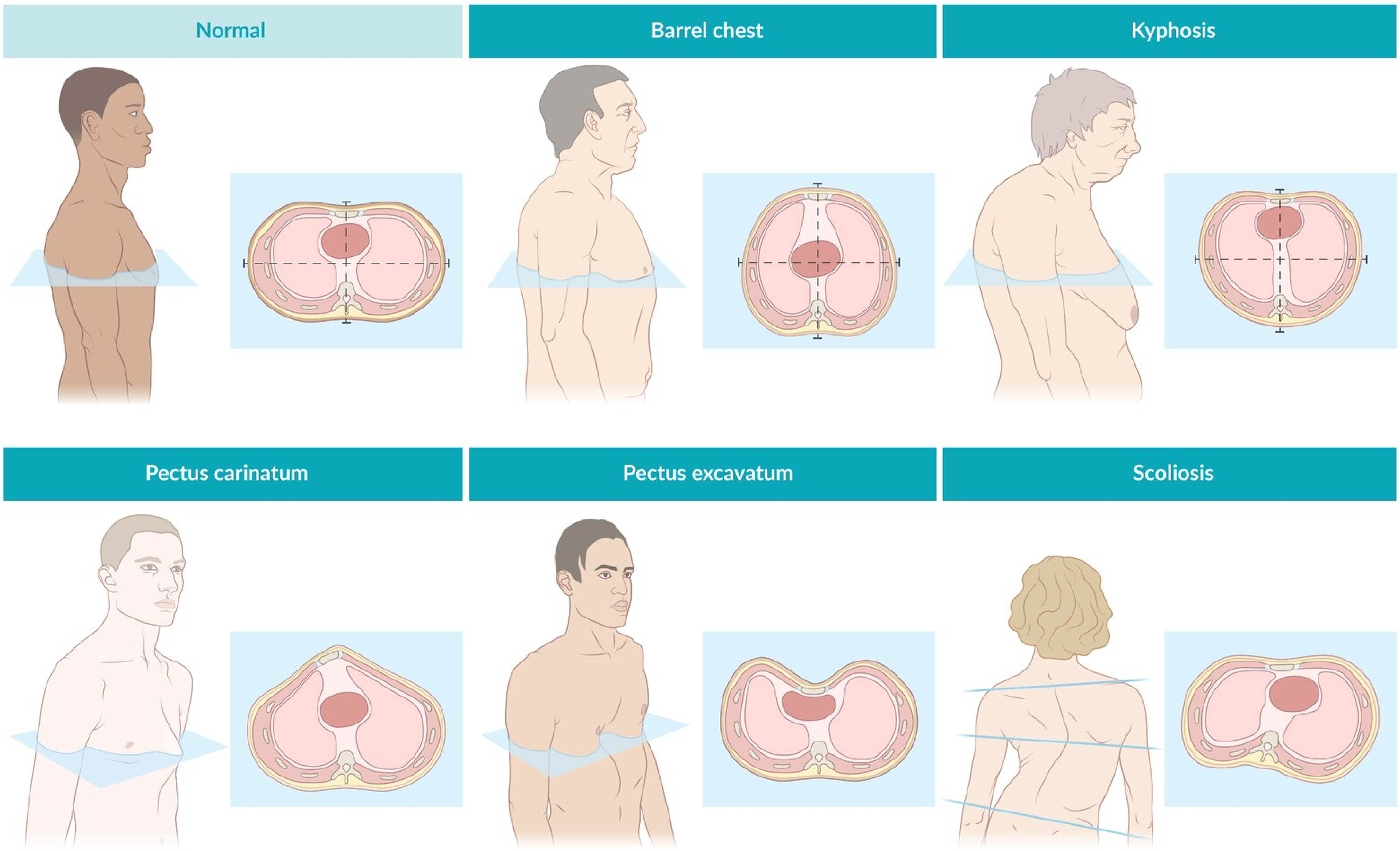
Tetralogy of Fallot	• DiGeorge syndrome
	• Down syndrome
	• Maternal risk factors
	  ◦ Alcohol consumption
	  ◦ Phenylketonuria
	  ◦ Diabetes

Transposition of great vessels	• Maternal risk factors: diabetes
Tricuspid valve atresia	• Down syndrome
Ebstein anomaly	• Prenatal lithium exposure
Total anomalous pulmonary venous return	• Heterotaxy syndromes
Persistent truncus arteriosus	• DiGeorge syndrome
Hypoplastic left heart syndrome	• Trisomy 13 • Trisomy 18 • Turner syndrome • Jacobsen syndrome


TENSION
PNEUMOTHORAX
Mediastinal Shift
to right
Collapsed
lung
Depressed
hemidiaphragm




Left Pneumothorax
ABSENT LUNG MARKINGS
MEDIASTINAL SHIFT
TO RIGHT
COLLAPSED
LEFT LUNG



Normal
Barrel chest
Kyphosis
Pectus carinatum
Pectus excavatum
Scoliosis

Increased effort of breathing

- Tachypnea
- Use of accessory muscles of respiration during inspiration
  - Sternomastoid muscles
  - Scalene muscles
  - Pectoralis major
- Trachea off midline
- Tripod position: patients with emphysema and respiratory distress will lean forward while sitting, resting with their hands on their knees.

Sputum production or secretions, if any

- **White and translucent**: viral infection (for example, bronchitis that presents with a typical early-morning cough)
- **White and foamy**: pulmonary edema
- **Yellow-green**: bacterial infection
- **Green**: an indication of a pseudomonal infection
- **Grayish**: pneumoconiosis, a waning bacterial infection
- **Blackish-brown**: possibly old blood; should be further investigated (can also be a harmless incidental finding)
- **Friable**: tuberculosis, actinomycosis
- **Hemoptysis**: See "Etiology of hemoptysis."

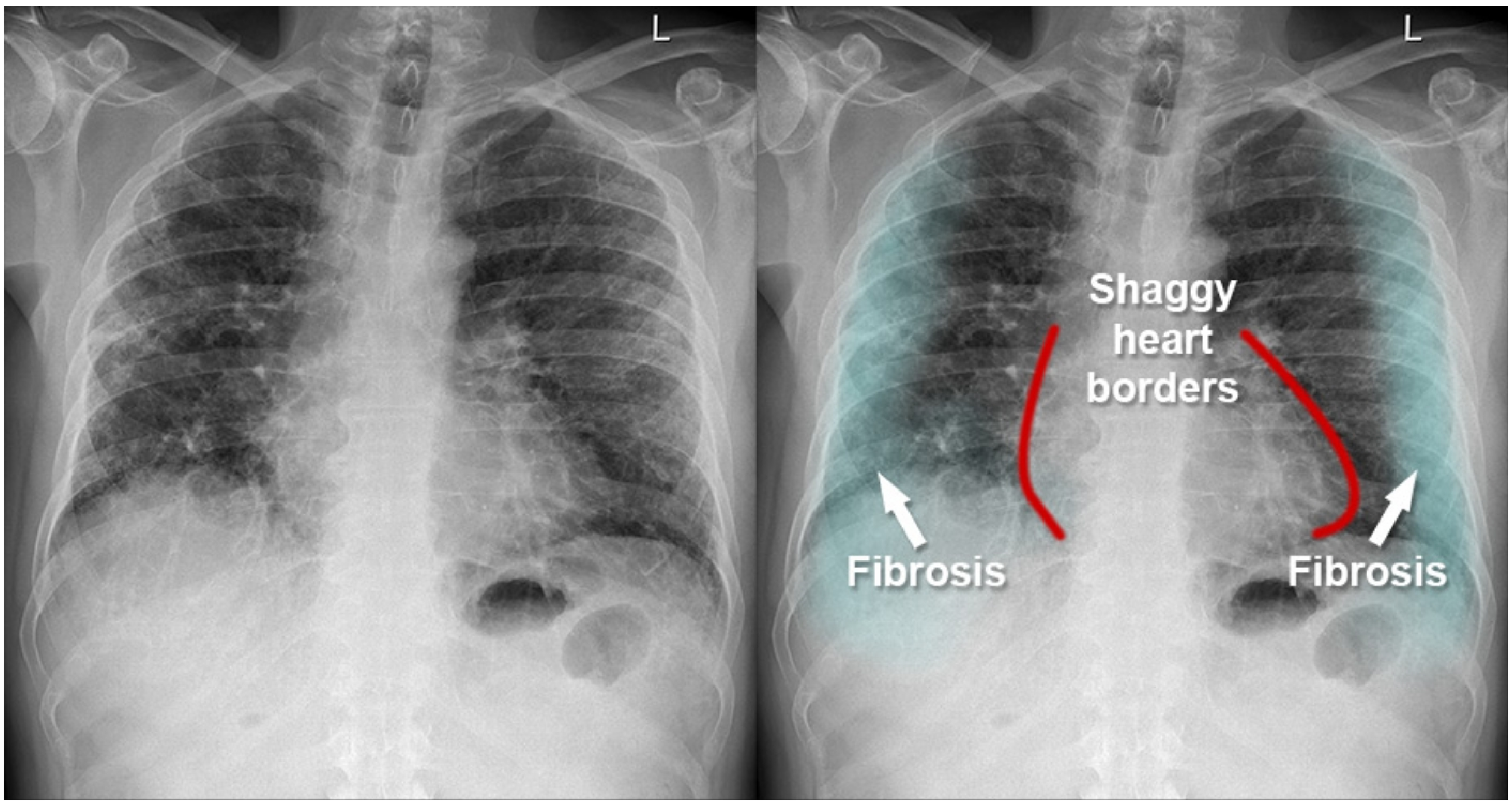
Physiological breath sounds

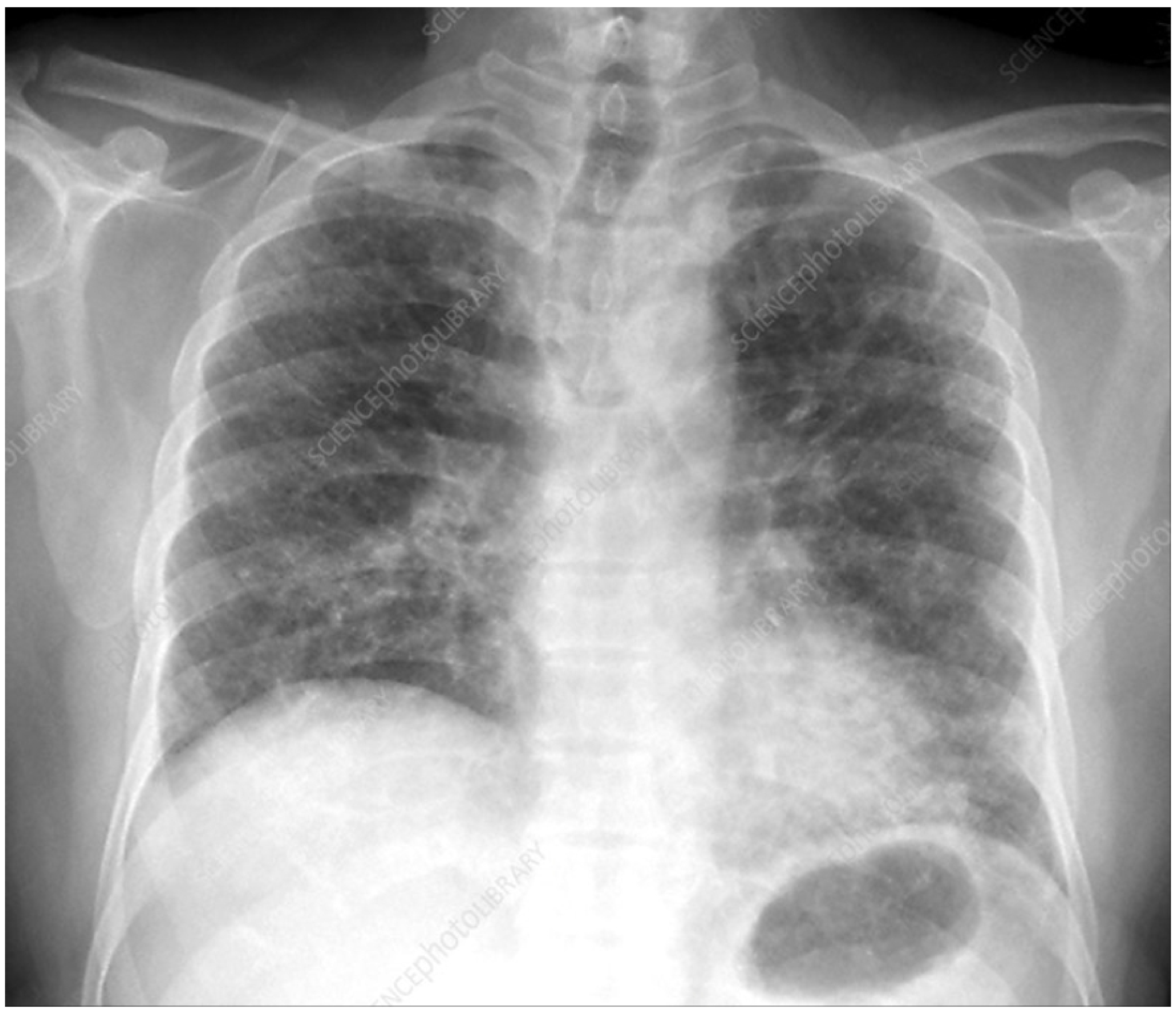
- **Vesicular breathing**
  - Soft and low pitched, through inspiration and part of expiration
  - Heard over both lungs
- **Bronchovesicular breathing**
  - Intermediate intensity and pitch, through both inspiration and expiration
  - Heard over 1st and 2nd intercostal spaces
- **Bronchial breathing**
  - Loud and high pitched, through part of inspiration and all of expiration
  - Heard over the sternum
- **Tracheal breathing**
  - Very loud and high pitched, through both inspiration and expiration
  - Heard over the neck
- **Cavernous breathing**
  - A harsh, hollow breath sound with high-pitched overtones heard on lung auscultation
  - It is produced by air passing through a large, superficial cavity that communicates with the bronchial system (e.g., a cyst or abscess)
  - Can not be heard if there is fluid or a fungal ball within the cavity

Types of pathological breath sounds

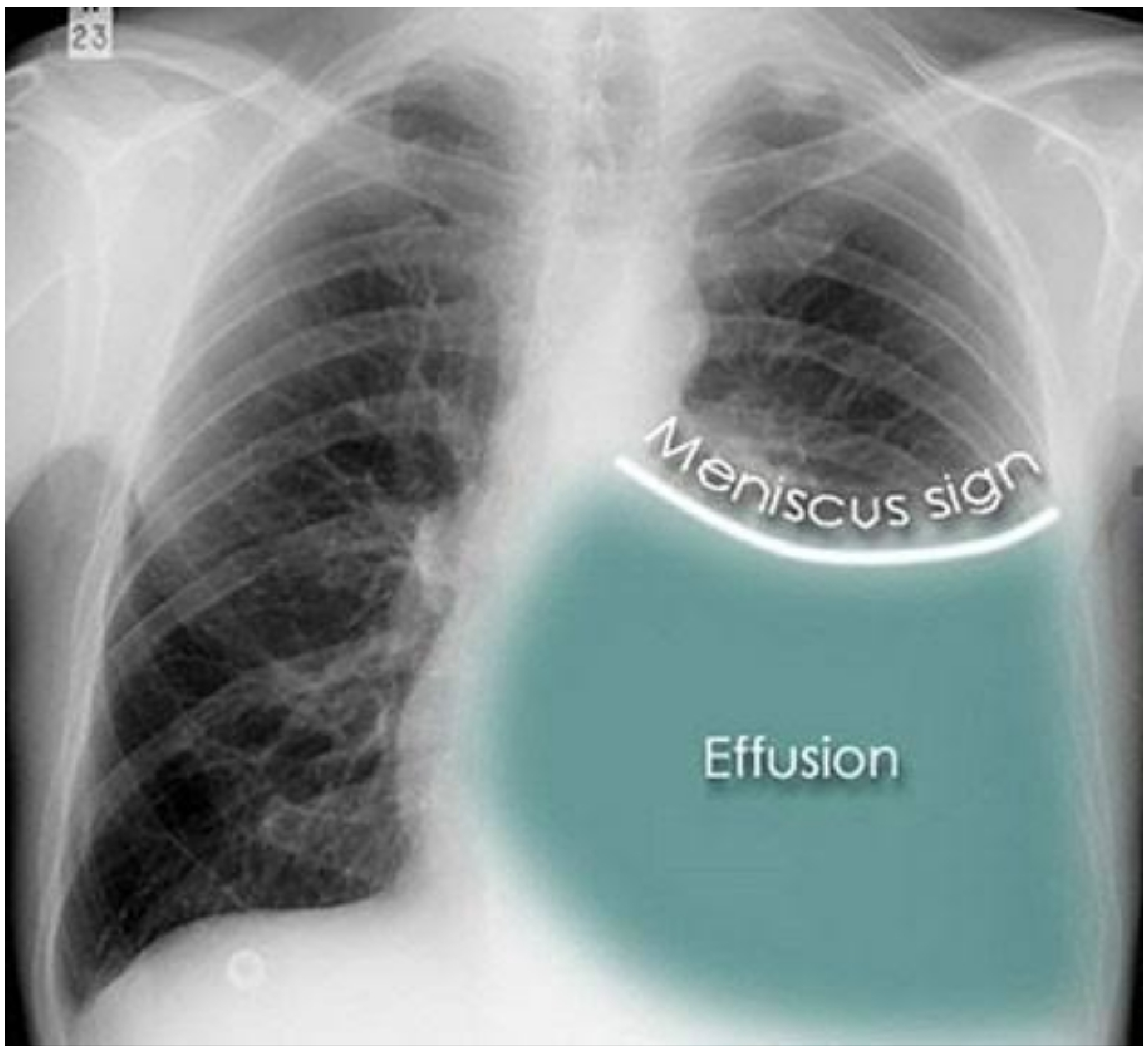
- **Crackles or rales**: discontinuous, intermittent
  - Fine: soft, high-pitched (e.g., normal, asbestosis, sarcoidosis)
  - Coarse: loud, low-pitched (e.g., COPD, pulmonary edema) [4]
- **Wheezes** (sibilant wheezing): musical, prolonged
- **Rhonchi** (sonor wheezing): low-pitched, snoring
- **Stridor**: high-pitched, over trachea which may occur on:
  - Inspiration (inspiratory stridor): narrowing of the extrathoracic airway; characteristic of epiglottitis, pseudocroup, foreign body aspiration, bilateral vocal cord palsy
  - Expiration (expiratory stridor): obstruction of the intrathoracic airways; characteristic of bronchial asthma, COPD
  - Inspiration and expiration (biphasic stridor): obstruction at the level of the glottis
- **Pleural friction rub**: scratchy, high-frequency sound
- **Muffled or absent breath sounds**: suggest presence of air or fluid between the lung and the chest wall (e.g., pleural effusion, pneumothorax)

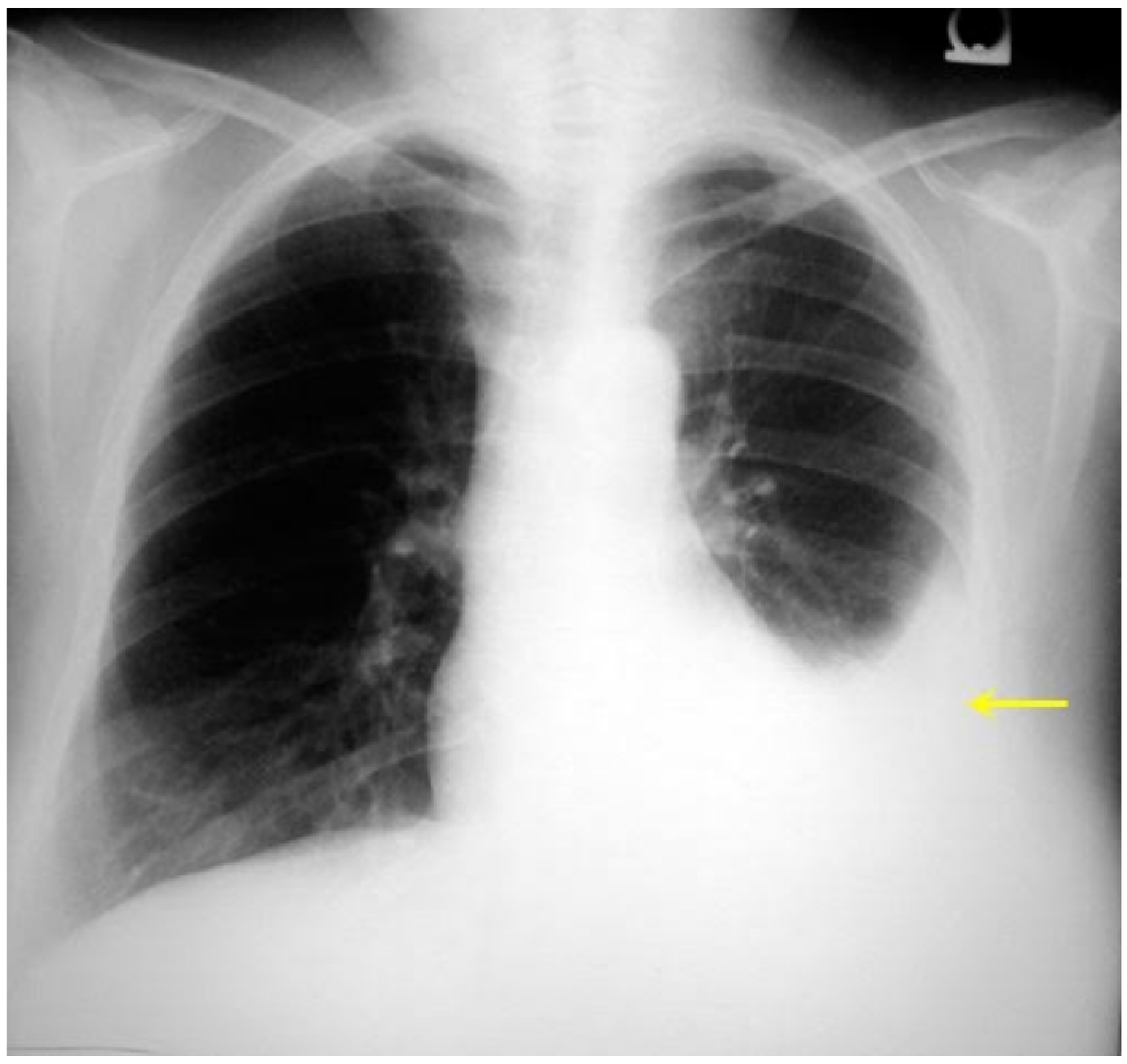
Pulmonary Fibrosis

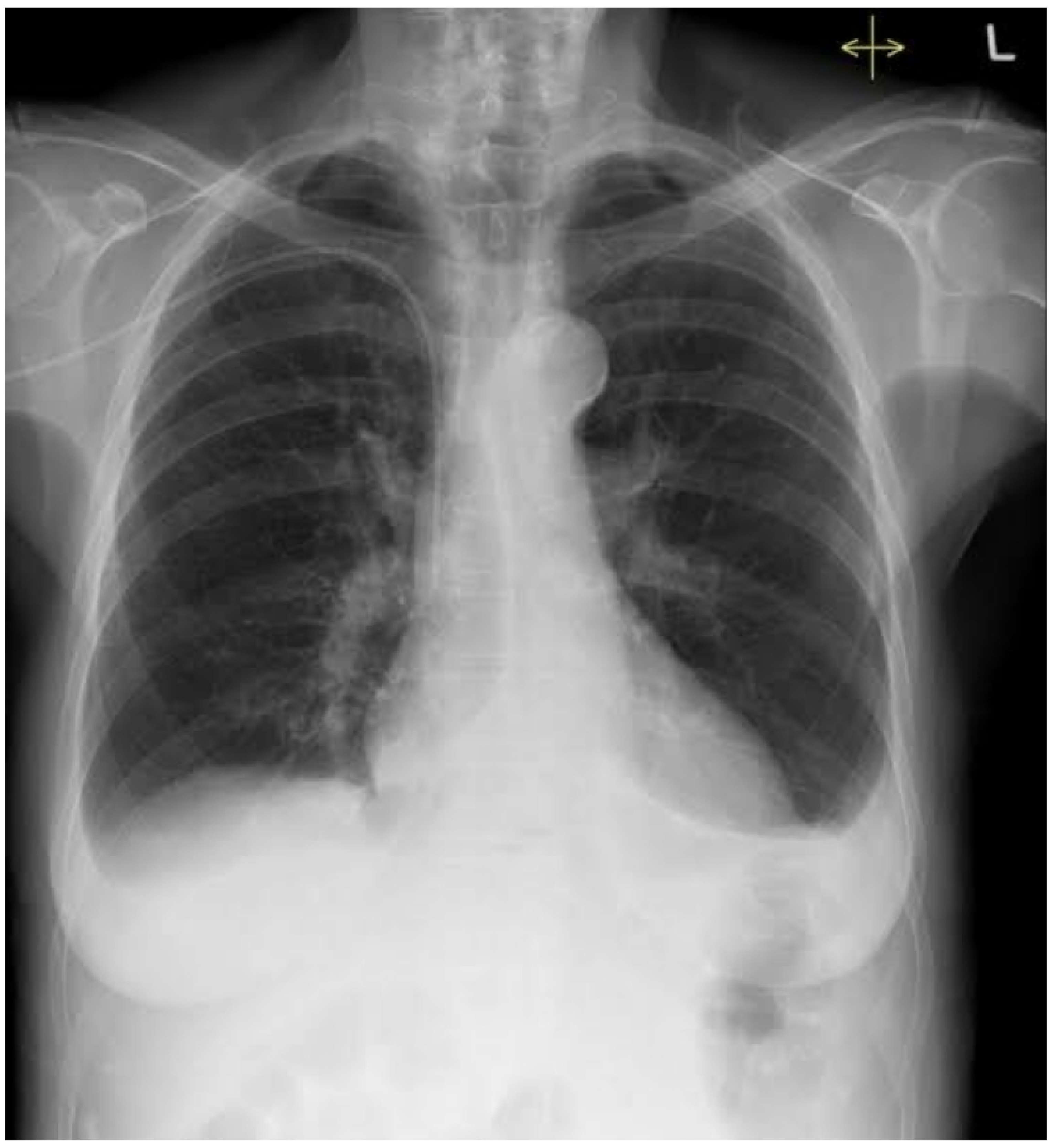


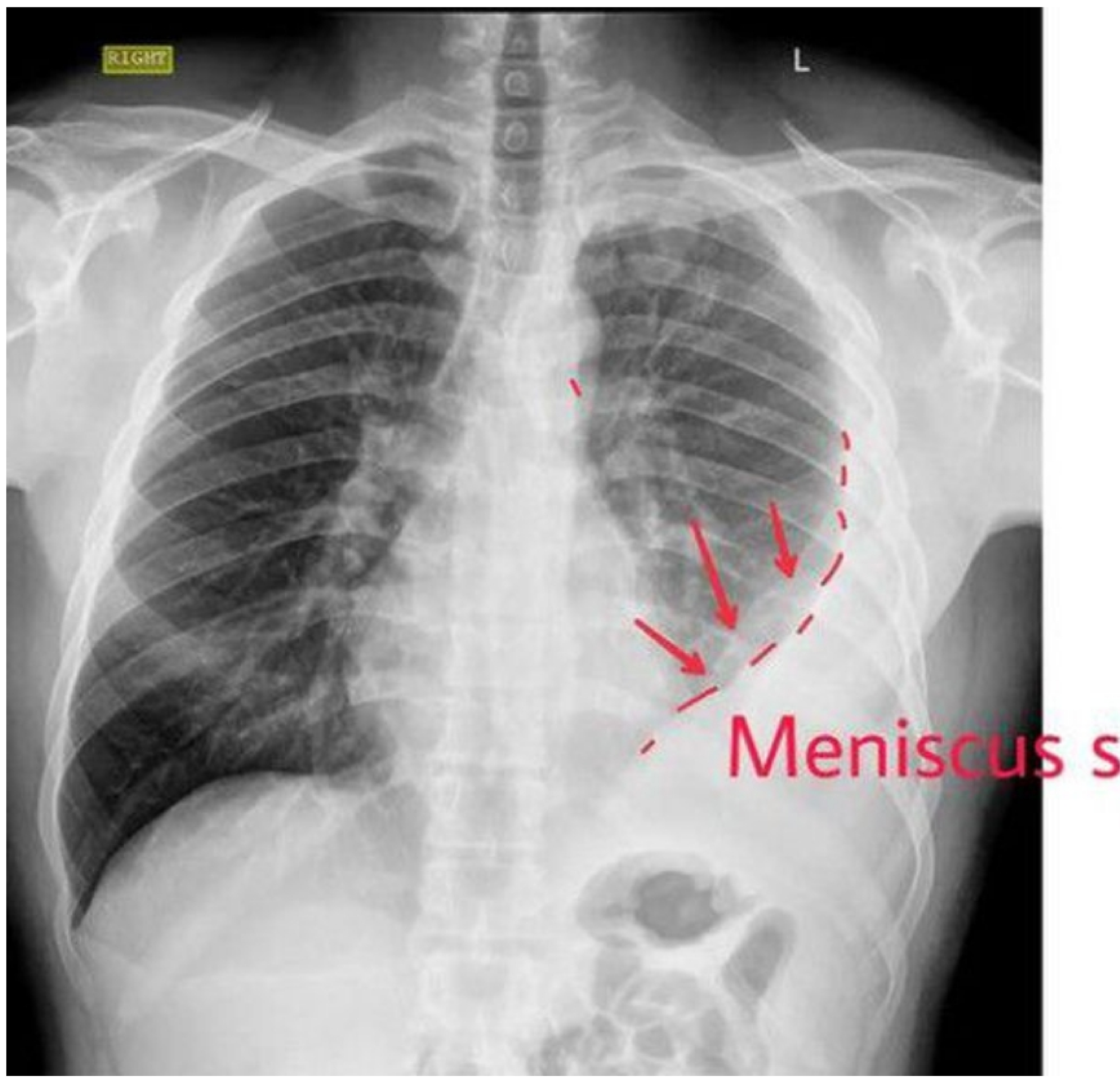


Meniscus sign in Pleural effusion









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'

- Auscultate the heart and simultaneously palpate the arterial pulse so that you know when systole occurs.

- A murmur coinciding with a palpable pulse means the murmur must be systolic. If murmur and pulse alternate, the murmur is diastolic

- Use the bell of stethoscope at the apex, the best place to hear mitral stenosis. If you hear a loud S_1, then listen carefully for a diastolic murmur characteristic of mitral stenosis

Central Cyanosis

Beau's Lines
(Transverse lines)

- Malnutrition
- Cachexia

Lung Sounds

- Musical wheeze → Asthma
  Broncnitis
- Fine Crackles → Heart Failure
  Fibrosis

CVS Symptoms

- Chest Discomfort
- Breathlessness
- Palpitation
- Dizziness
- Syncope
- Peripheral edema

- Orthopnea
  Sensation of breathlessness on lying flat

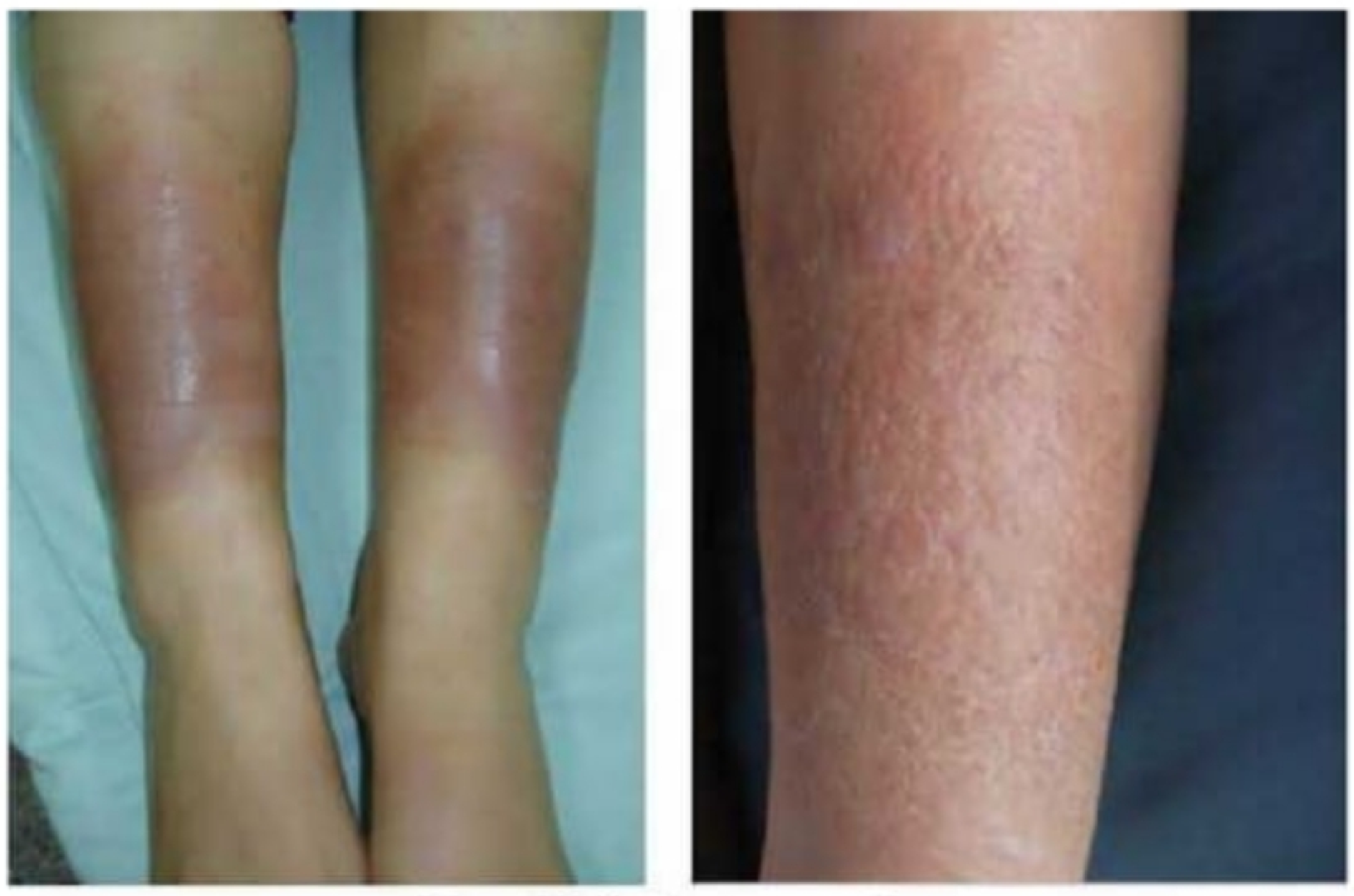
- Palpitation
  an awareness of heart beat

Syncope

Transient loss of consciousness resulting from transient global cerebral hypoperfusion

Non Pitting Edema

* Hypothyroidism (pretibial myxedema)
* Lymphoedema



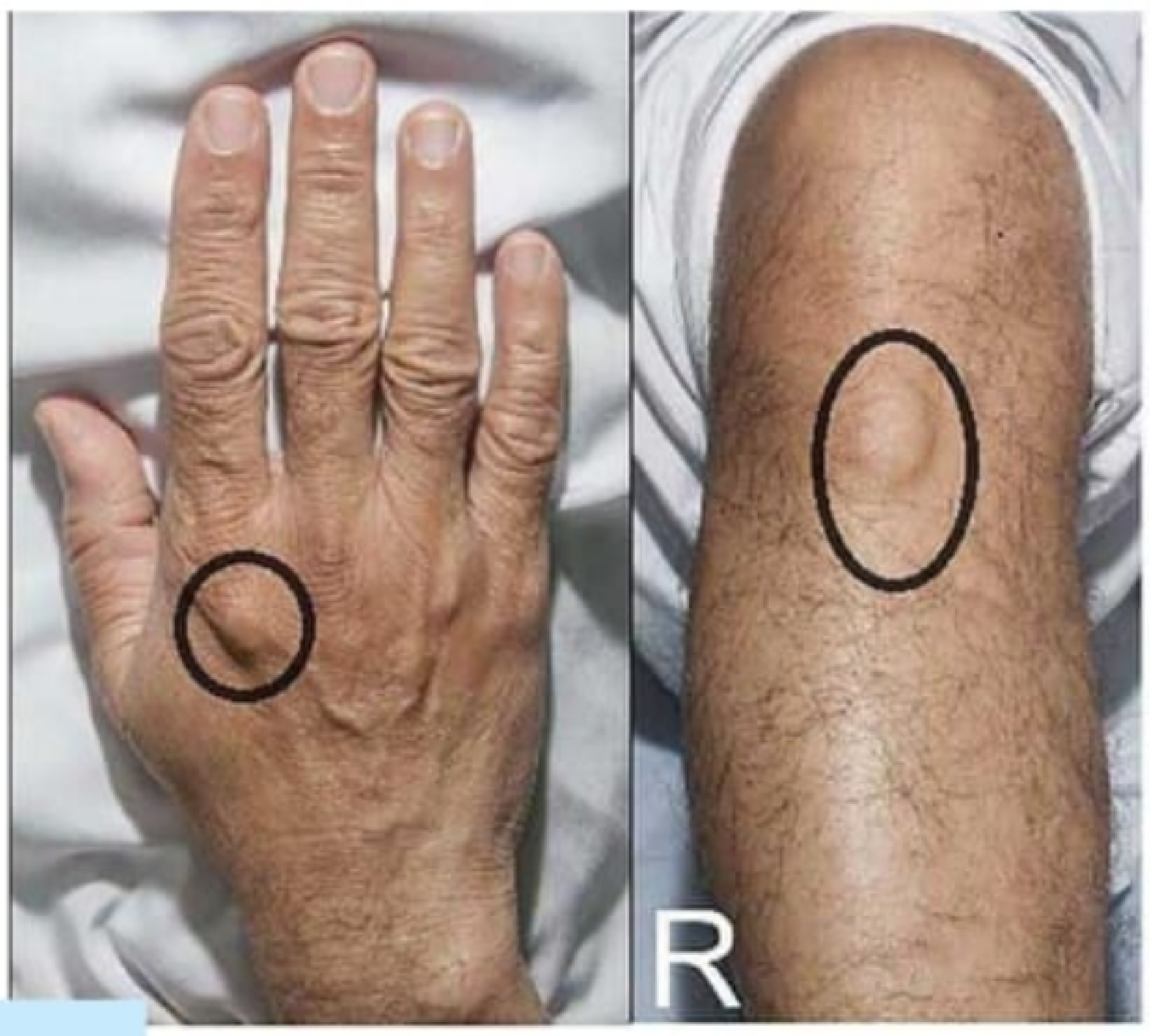
Pretibial myxedema

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

Bradycardia

<60 bpm

- Sinus bradycardia → normal physiological
  eg in athletes or during sleep
- Sick Sinus Syndrome
- Hypothyroidism
- Hypothermia
- Drugs → Verapamil, Diltiazem,
  Digoxin
- 2nd or 3rd Degree AV Block
- Apparent bradycardia → due to
  ventricular ectopic beats

Tachycardia

- pregnancy
- Anxiety
- Pain
- Inappropriate Sinus Tachycardia
- Thyrotoxicosis
- Blood loss
- Drugs → Atropine, Dobutamine, Salbutamol

- Abnormal Rhythms
  - L. Supraventricular Tachycardia
  - Ventricular Tachycardia
  - AFib
  - Atrial Flutter

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

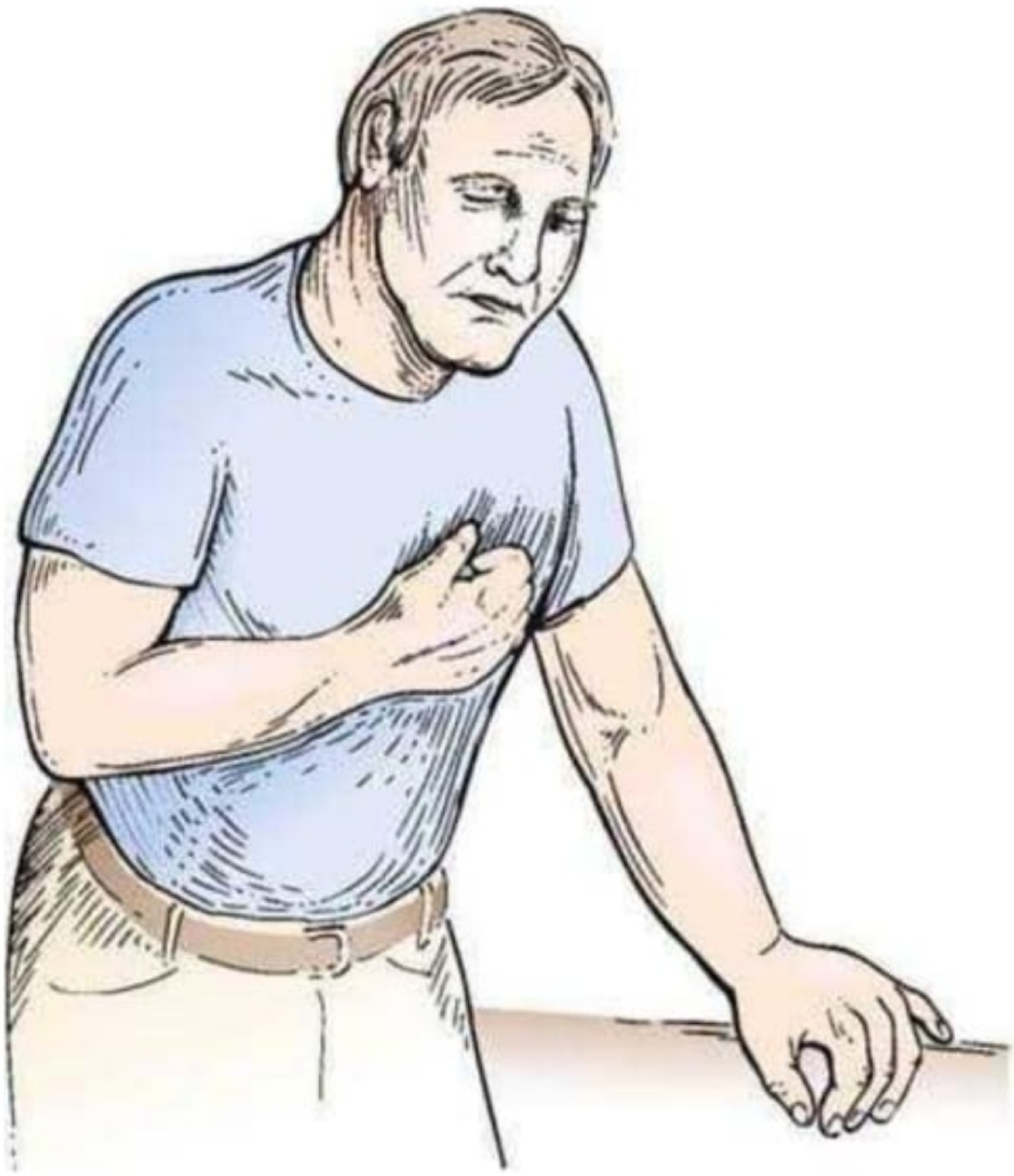
Xanthelasma

deposits of cholesterol

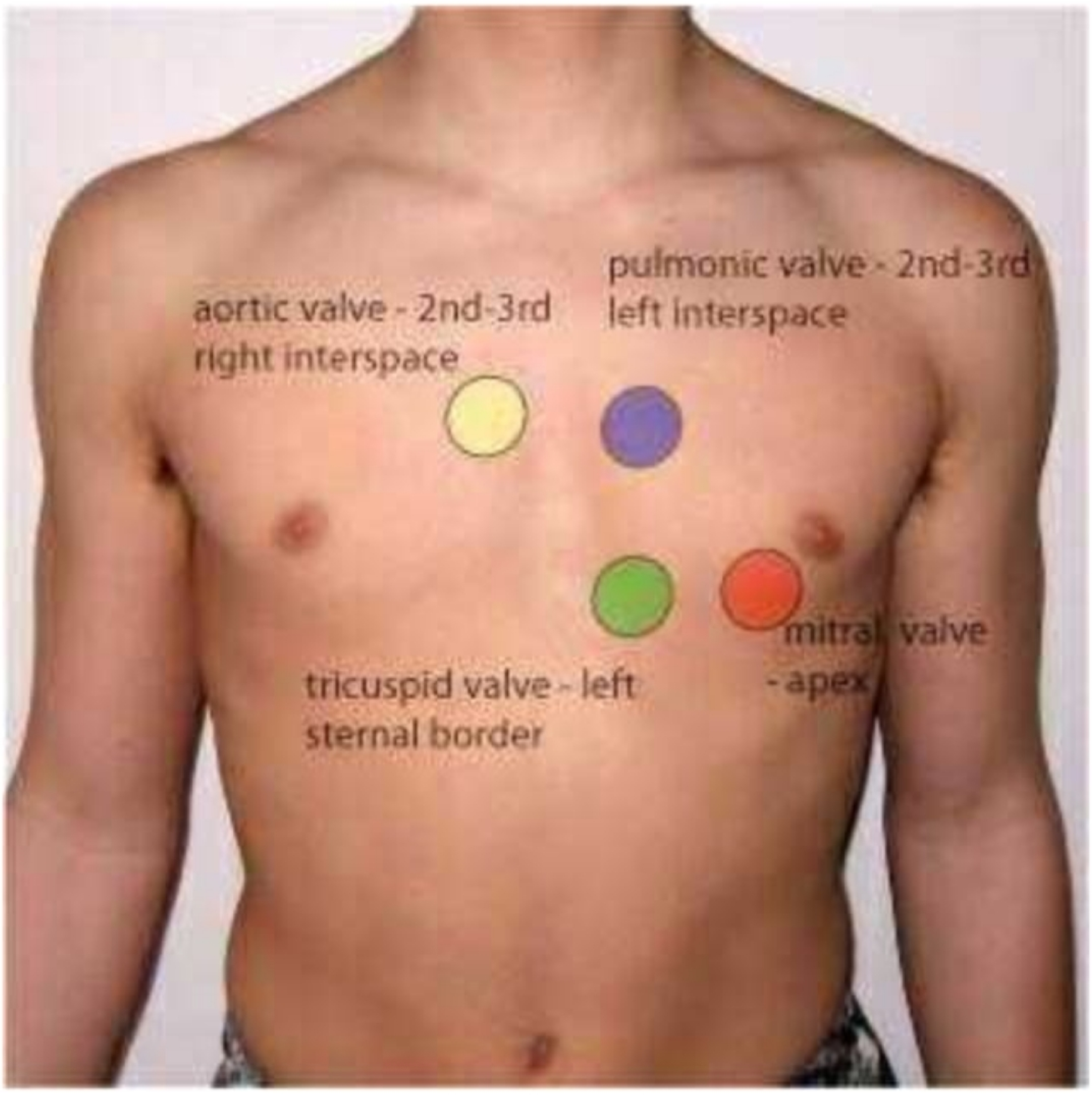
Pectus Carinatum
(Pigeon Chest Deformity)

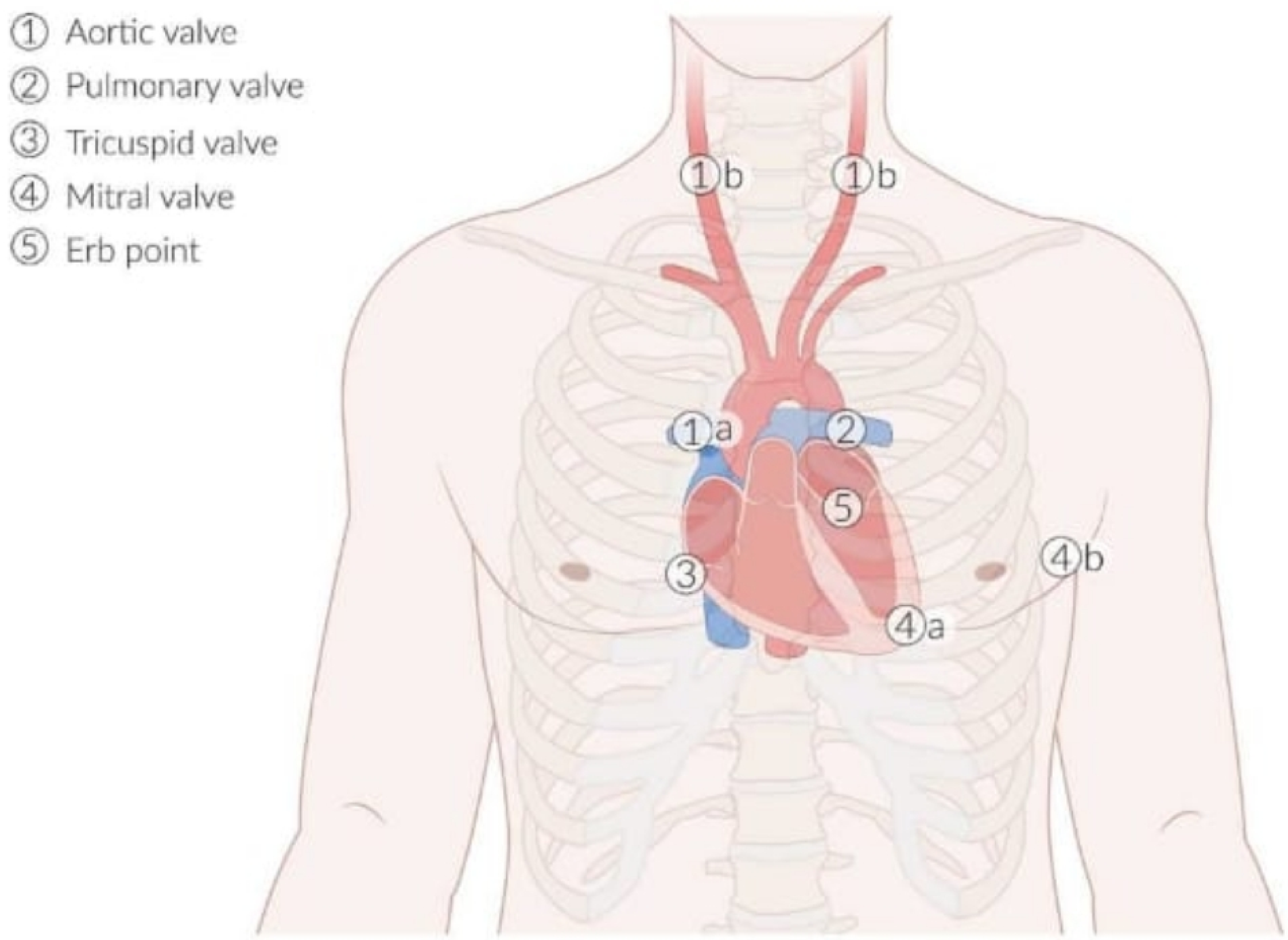
Pectus Excavatum
(Funnel Chest
Deformity)

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



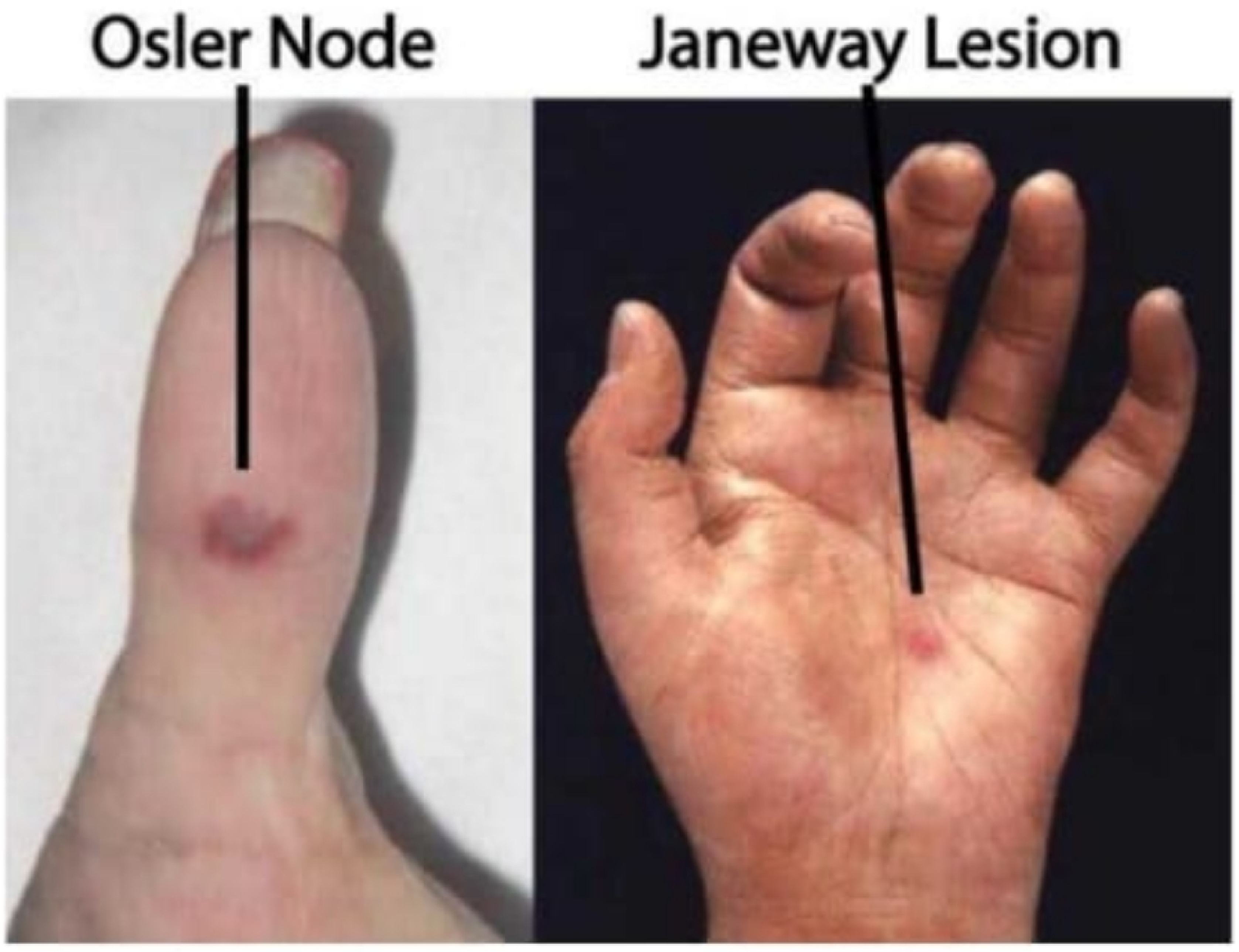
Auscultatory Sites for Heart Sounds

Auscultatory sites for heart sounds - 1. Aortic area: 2 nd right parasternal intercostal space (ICS) - Ideal site for auscultation of aortic valve murmurs (1a). The murmur of aortic stenosis also radiates to the carotid arteries (1b). 2. Pulmonary area: 2 nd left parasternal ICS - Ideal site for auscultation of pulmonary valve murmurs 3. Tricuspid area: 4 th left parasternal ICS - Ideal site for auscultation of tricuspid valve murmurs 4. Mitral area: 5 th left ICS in the midclavicular line (over the cardiac apex) - Ideal site for auscultation of mitral valve murmurs (4a). The murmur of mitral regurgitation also radiates to the left axilla (4b). 5. Erb point: 3 rd left parasternal ICS - Ideal site for auscultation of aortic regurgitation murmurs and for gaining an overview of heart sounds and murmurs from a single point

The LAD is the most commonly occluded coronary artery and is often referred to as the “widow maker” due to the high mortality rate associated with LAD infarction.

The RCA usually supplies the heart's conduction system (**sinus and AV node**) so that stenosis or occlusion of this vessel often leads to cardiac arrhythmias.

OSLER'S NODES



- Tender, red lesions that occur on the fingertips, palms and soles
- uncommon manifestation of infective endocarditis

* Janeway lesions are non-tender