

CHEST PAIN

Presented By:

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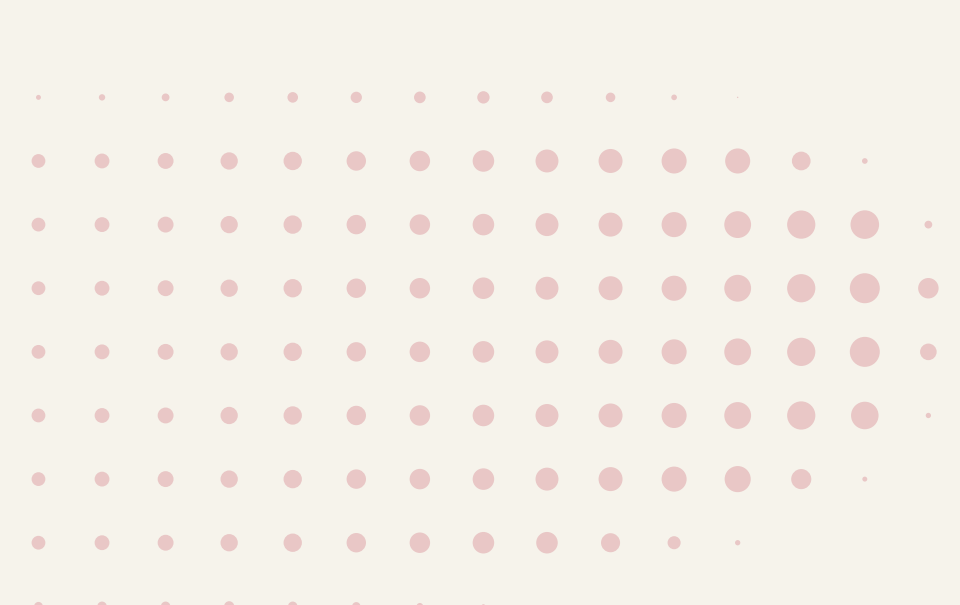
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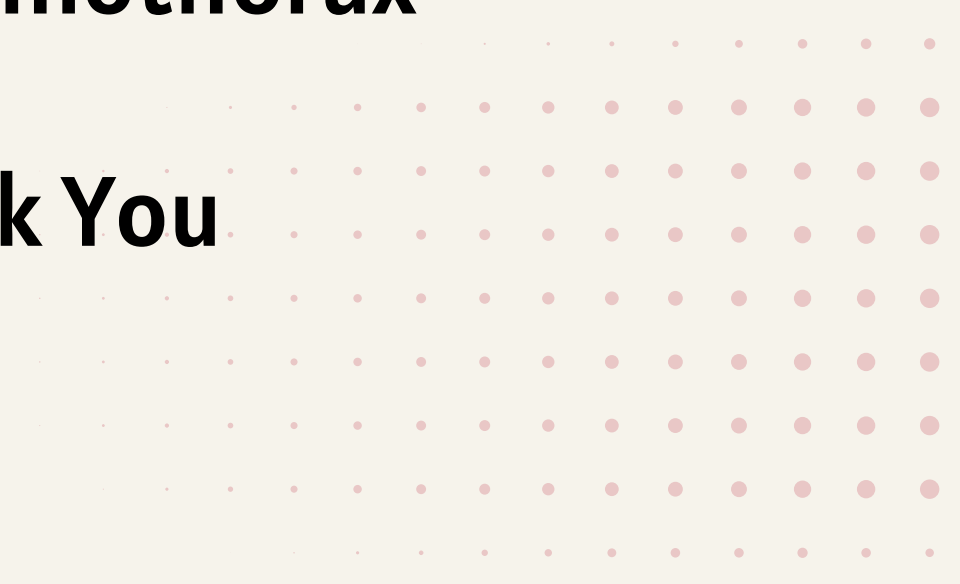
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OVERVIEW

- **Approach in ED**
 - **ACS**
 - **Aortic dissection**
 - **DVT**
 - **PE**
 - **Pneumothorax**
 - **Thank You**
- 

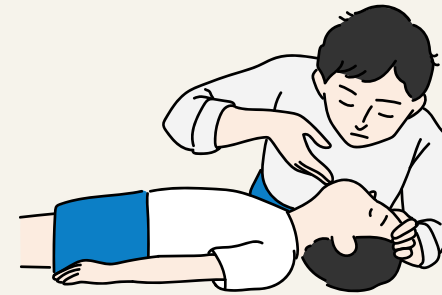
CHEST PAIN

Chest pain is one of the most common presenting symptoms in the emergency department, posing a challenge due to the wide range of possible causes.



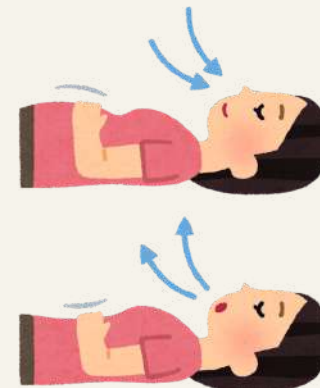
APPROACH IN THE ED

A Airway



Stable

B Breathing



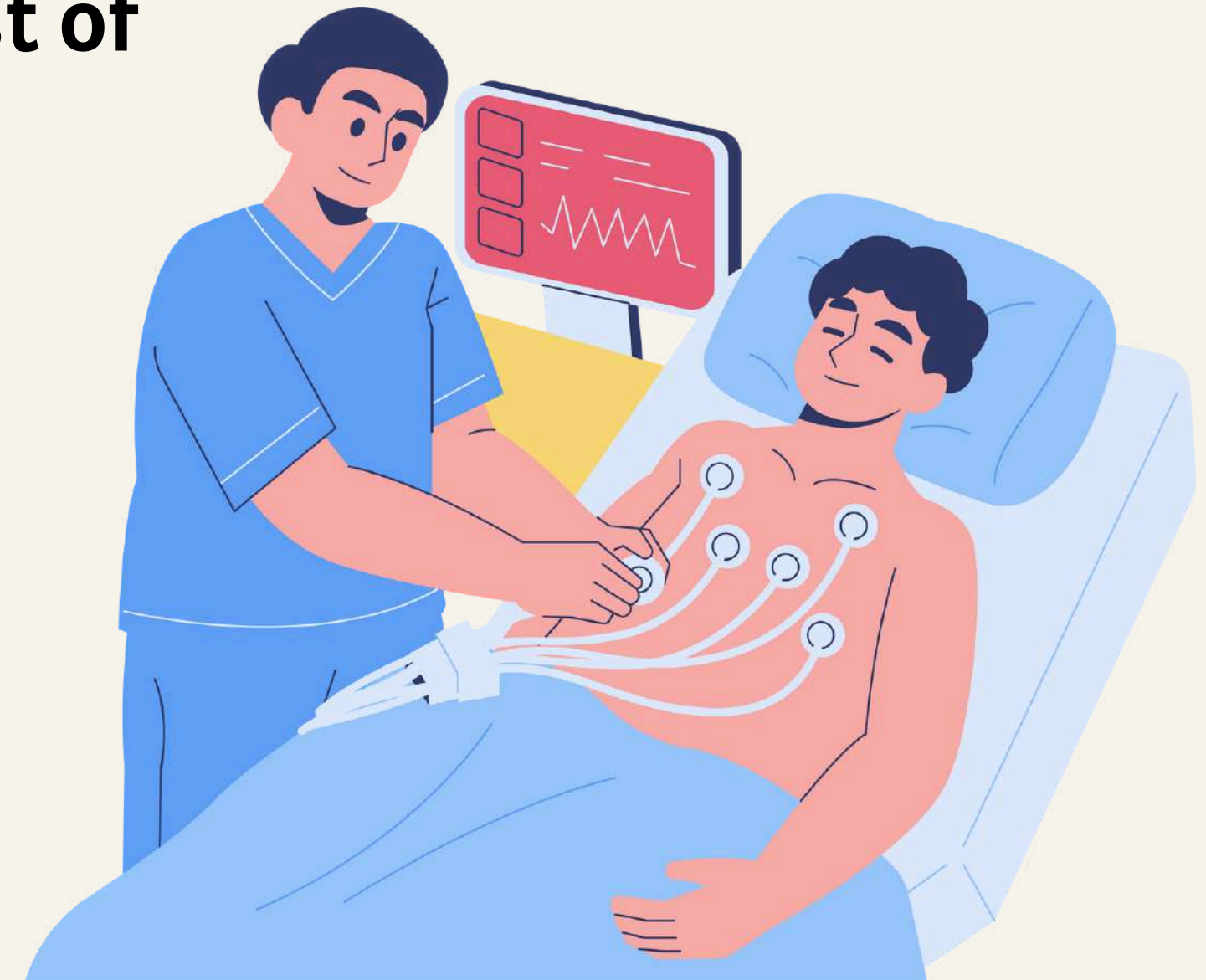
Unstable

C Circulation



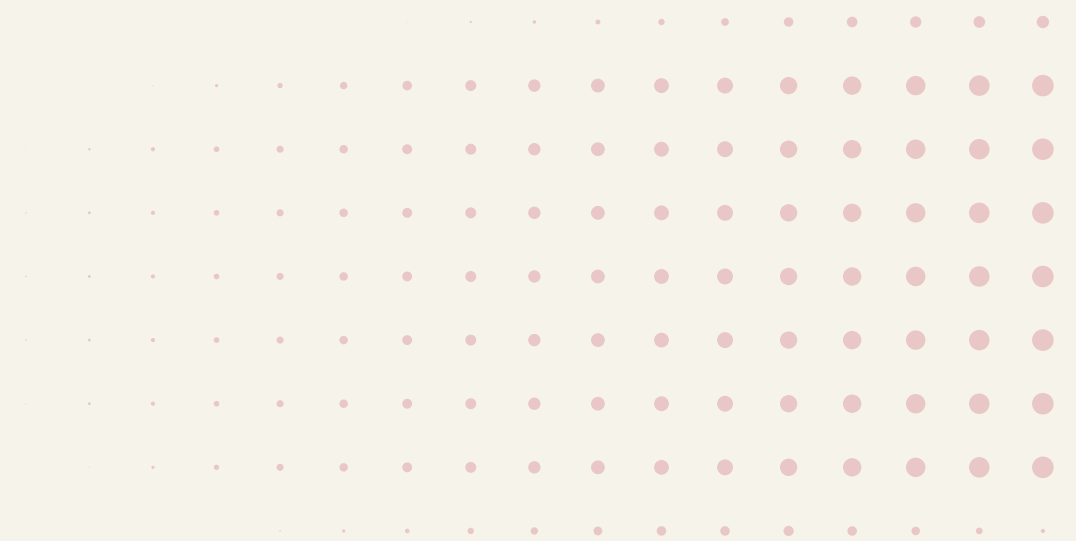
APPROACH IN THE ED

- In stable patients Initial Management consist of
 - Pulse oximeter
 - Supplemental O₂
 - IV Access
 - Cardiac monitoring
 - ECG
 - Hx and PE





DIFFERENTIALS

- **Assess for potentially life threatening causes of chest pain (PET-MAC)**
 - **Then consider the less emergent causes**
- 

Mnemonic : PET-MAC

P - Pulmonary Embolism

E - Esophageal Rupture

T - Tension Pneumothorax

M - Myocardial Infarction

A - Aortic Dissection

C - Cardiac Tamponade



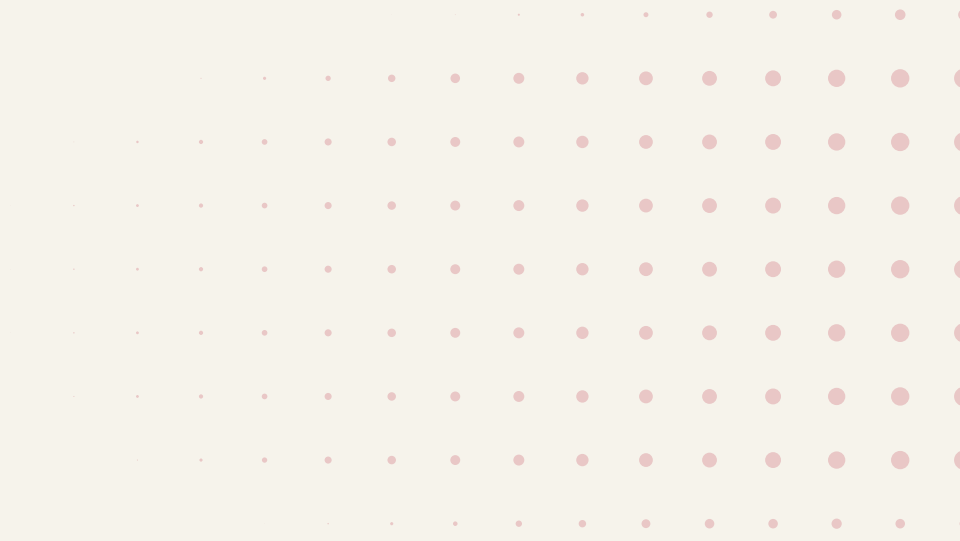


FOCUSED HISTORY

- **characteristics of the pain:**
 - **Site**
 - **Onset (sudden vs. gradual) and duration**
 - **Character of the pain**
 - **Radiation (arm, back, jaw, abdomen)**
 - **Associated symptoms (dyspnea, palpitations, nausea/vomiting, cough, syncope, dizziness, weakness, focal neurological symptoms)**
 - **Constant vs. intermittent**

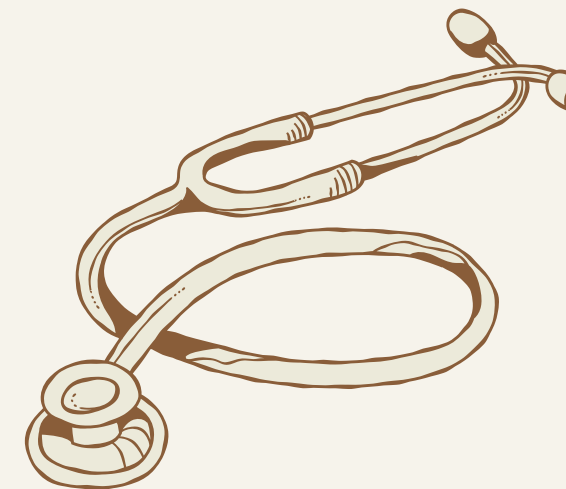


FOCUSED HISTORY

- **characteristics of the pain:**
 - **Aggravating factors (position, respiration, exertion, eating)**
 - **Relieving factors (position, rest, medications)**
 - **Any precipitating events and ask about history of recent trauma**
 - **Focused review of systems (CVS, RS, & GI)**
 - **Medical history**
 - **Previous episodes**
 - **Connective tissue disorders**
 - **Evaluate cardiac risk factors**
 - **PE risk factors**
- 

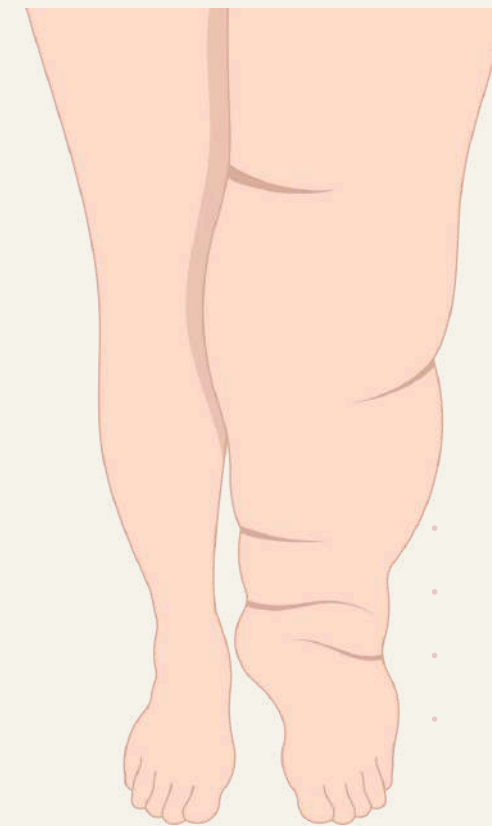
PHYSICAL EXAMINATION

- **Always start with the vitals**
 - BP in both arms
 - pulse (pulse deficit)
- **Neck**
 - Elevated jugular venous pressure
- **Heart**
 - Auscultation
- **lungs**
 - Palpation
 - Auscultation



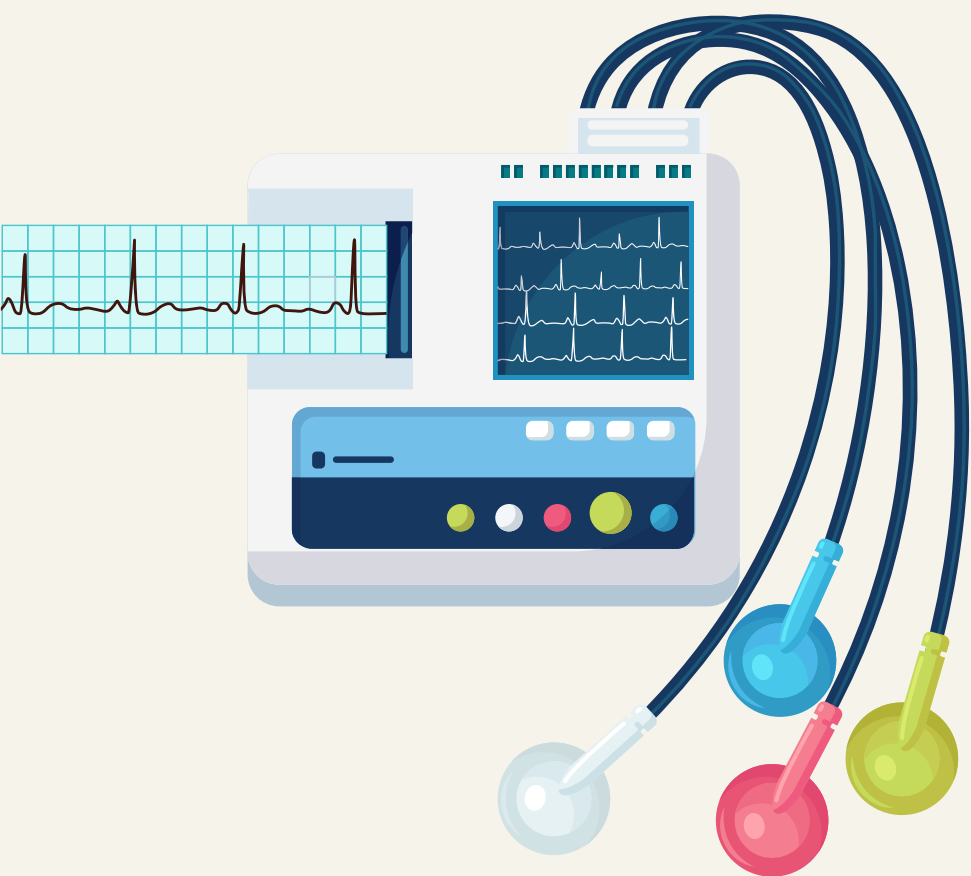
PHYSICAL EXAMINATION

- **Abdomen**
 - **Inspection, palpation and auscultation**
- **Extremities**
 - **Unilateral extremity enlargement, pain, and erythema**
 - **Limb pulse asymmetry**



INVESTIGATIONS

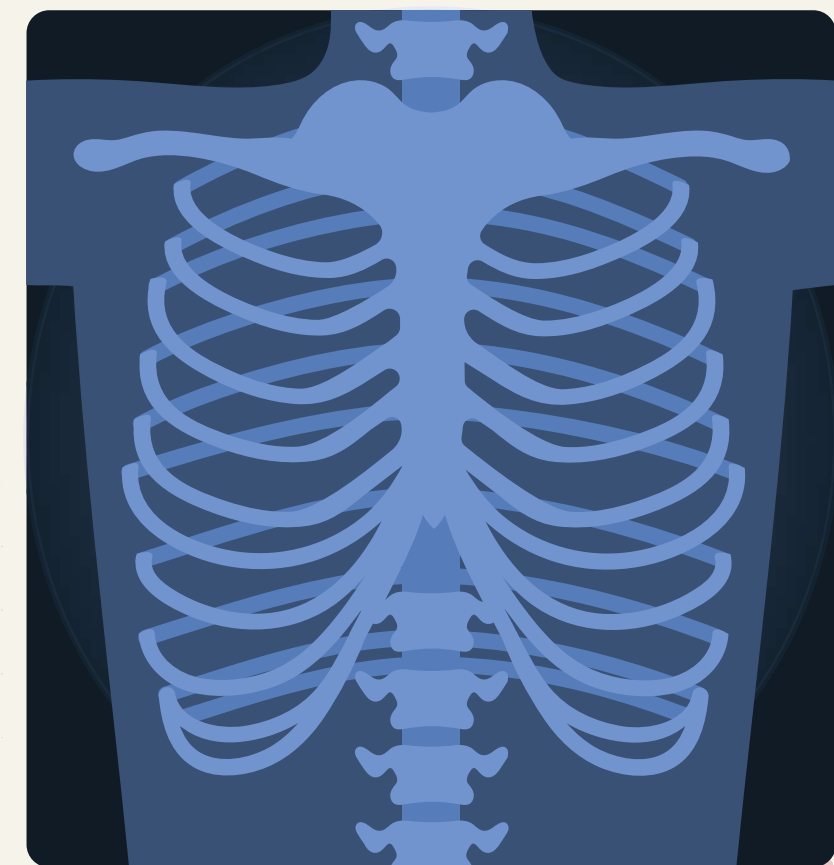
- ECG



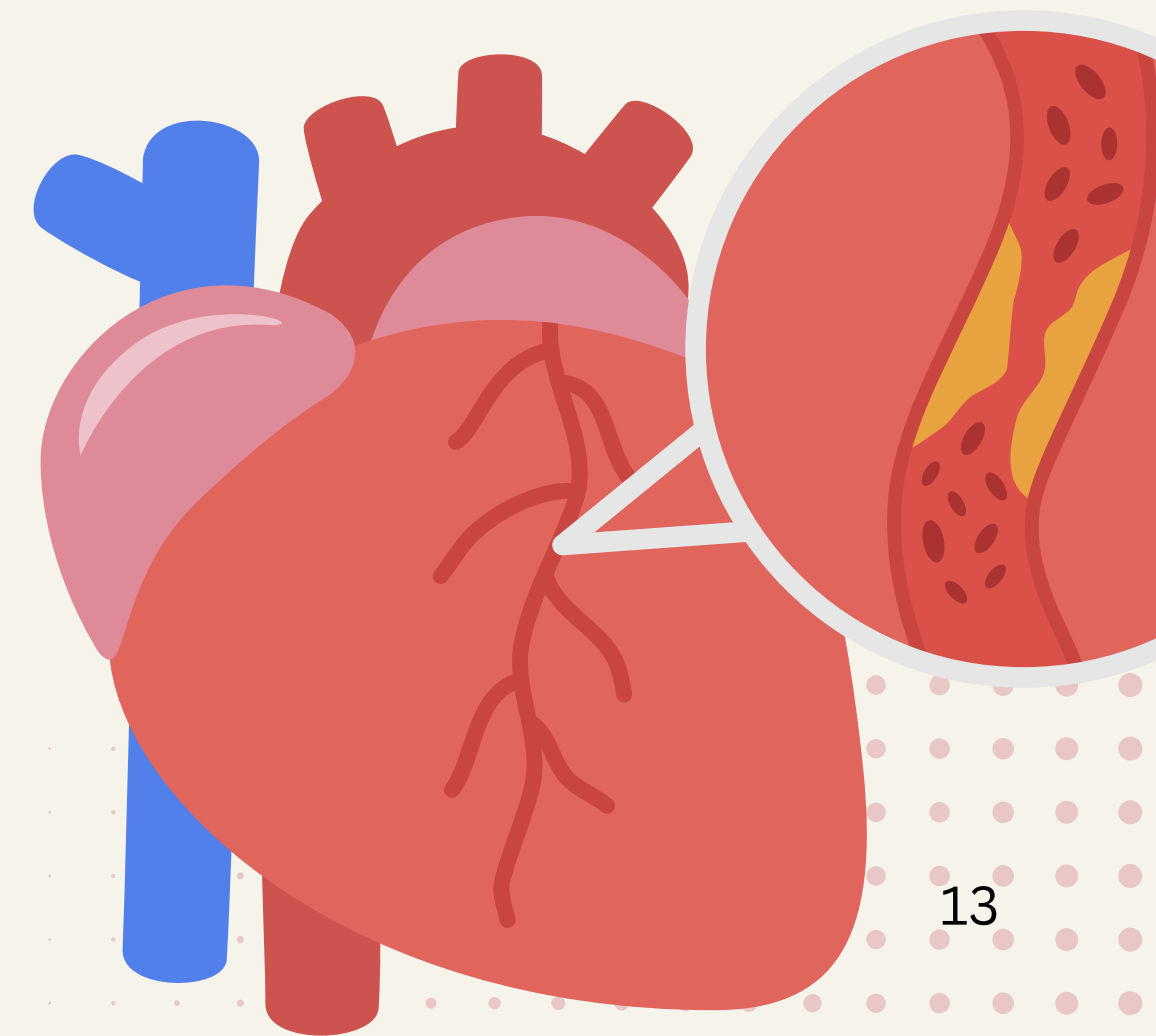
- LABS



- CXR



ACUTE CORONARY SYNDROM



INTRODUCTION TO ACUTE CORONARY SYNDROMES (ACS)

Definition: ACS encompasses a spectrum of conditions caused by reduced blood flow to the heart.

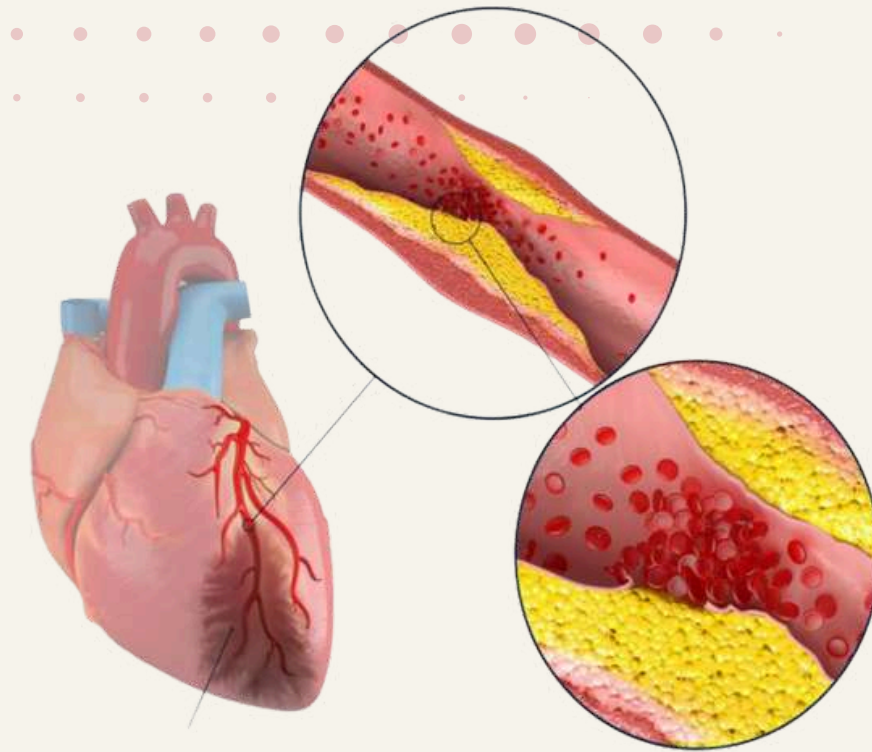
Clinical Spectrum:

- ST-Elevation Myocardial Infarction (***STEMI***)
- Non-ST-Elevation Myocardial Infarction (***NSTEMI***)
- Unstable Angina

ACS is a leading cause of morbidity and mortality worldwide

CAUSES OF ACS

- **Atherosclerosis:** thickening of intima due to plaque
- **Plaque Rupture or Erosion:** Triggers clot formation, obstructing blood flow.
- **Other Causes:** coronary artery dissection, and cocaine use



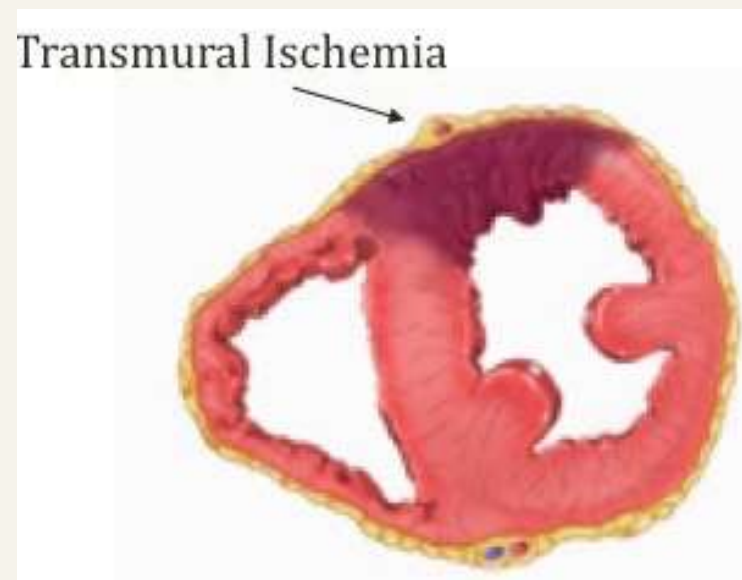
RISK FACTORS

- Prior coronary diseases
- DM
- peripheral artery disease
- chronic kidney disease
- hyperlipidemia
- HTN
- family history (M<50, F<60)
- obesity
- smoking

TYPES OF ACS

1. *ST-Elevation Myocardial Infarction (STEMI)*

Characterized by **complete** occlusion of a coronary artery.
Diagnosed via ECG showing ST-segment elevation.
Transmural ischemia.



Subtotal Occlusion



Total Occlusion

TYPES OF ACS

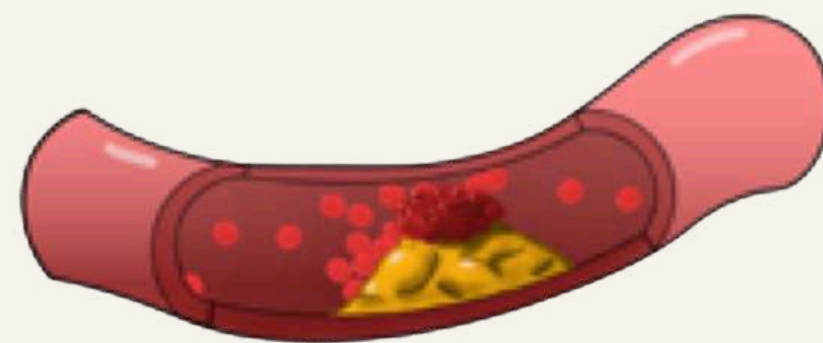
2. *Non-ST-Elevation Myocardial Infarction (NSTEMI)*

Partial occlusion or **reduced** blood flow.

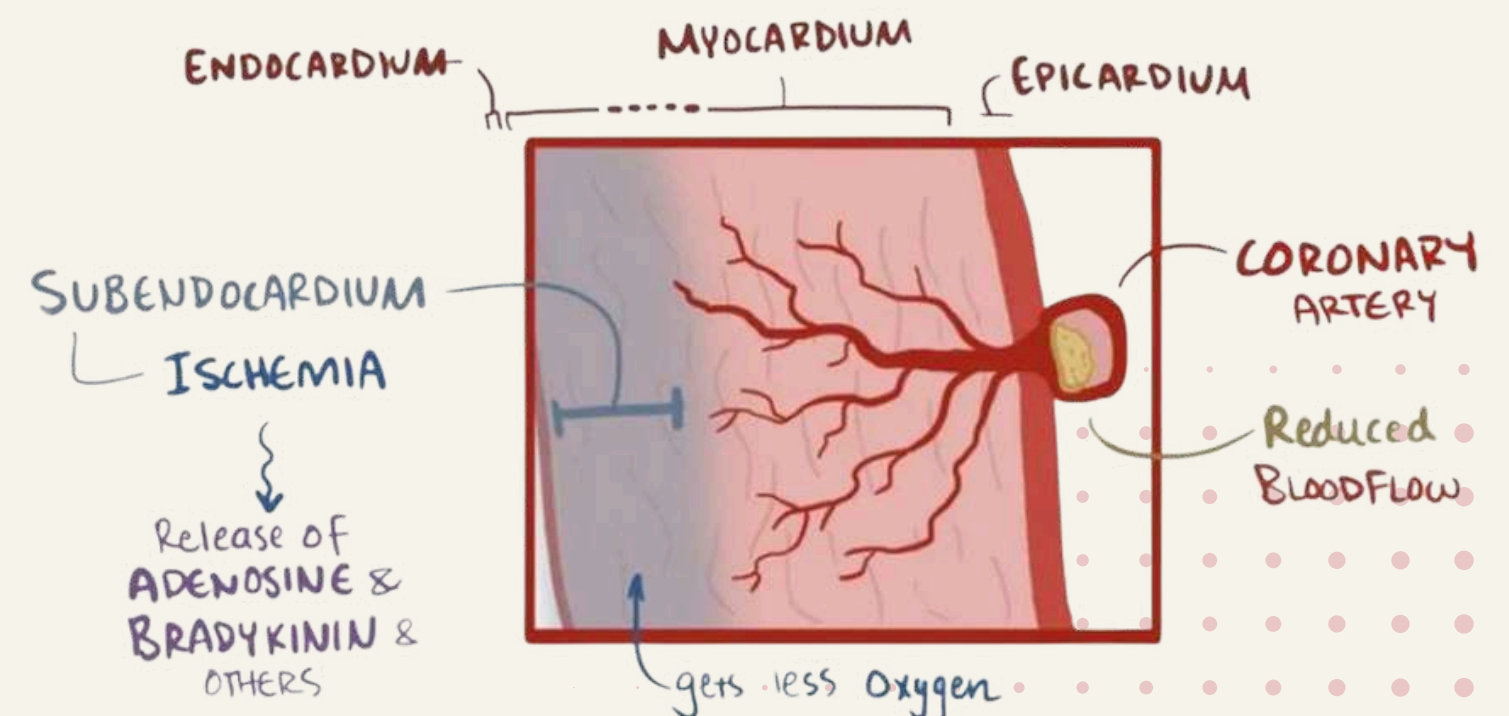
ECG may show ST depression or T wave inversion.

Elevated biomarkers (troponin /CK-MB)

Subendocardial ischemia



Subtotal Occlusion



TYPES OF ACS

3. Unstable Angina

Angina at rest or with minimal exertion.

Subtotal occlusion .

Normal biomarkers .

CLINICAL PRESENTATION

Typical Symptoms:

Chest pain or discomfort, radiating to left arm, back, neck, or jaw.
Shortness of breath, nausea, sweating.

Atypical Symptoms:

Especially in elderly, **diabetics**, and women: fatigue, abdominal pain, dizziness

PHYSICAL EXAMINATION

****Cardiogenic shock**

Hypotension

tachycardia

cool extremities

pale skin

**** Jugular venous distension**

****Crackles on Auscultation / murmur**

****Edema**

ELECTROCARDIOGRAPHY



INTRODUCTION

- **What is an ECG?**

Measures heart's electrical activity, using leads placed on the chest and limbs.

- **Purpose :**

To detect ischemia, infarction, arrhythmia , electrolytes abnormalities, pericardial diseases , valvular diseases and other cardiac abnormalities

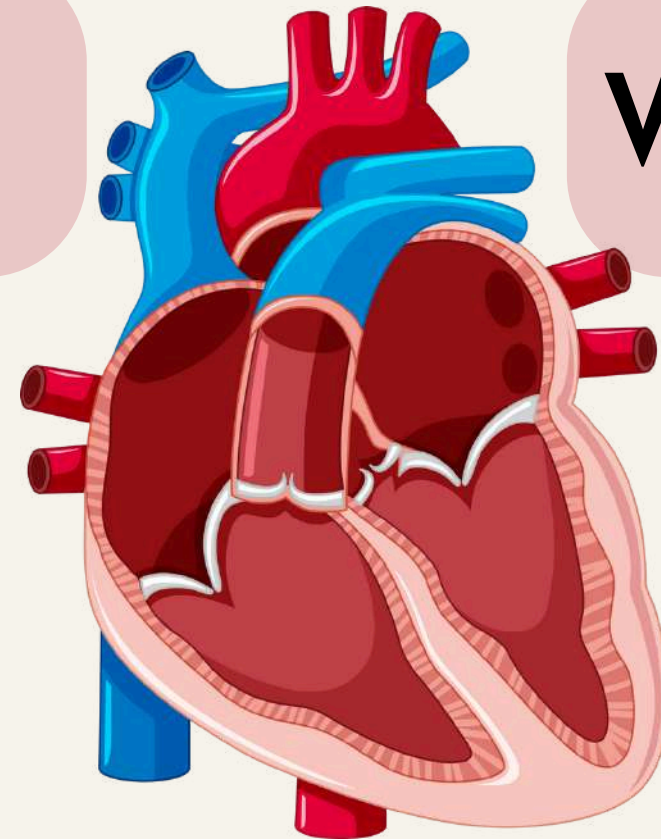


INTRODUCTION

Contraction = Depolarization

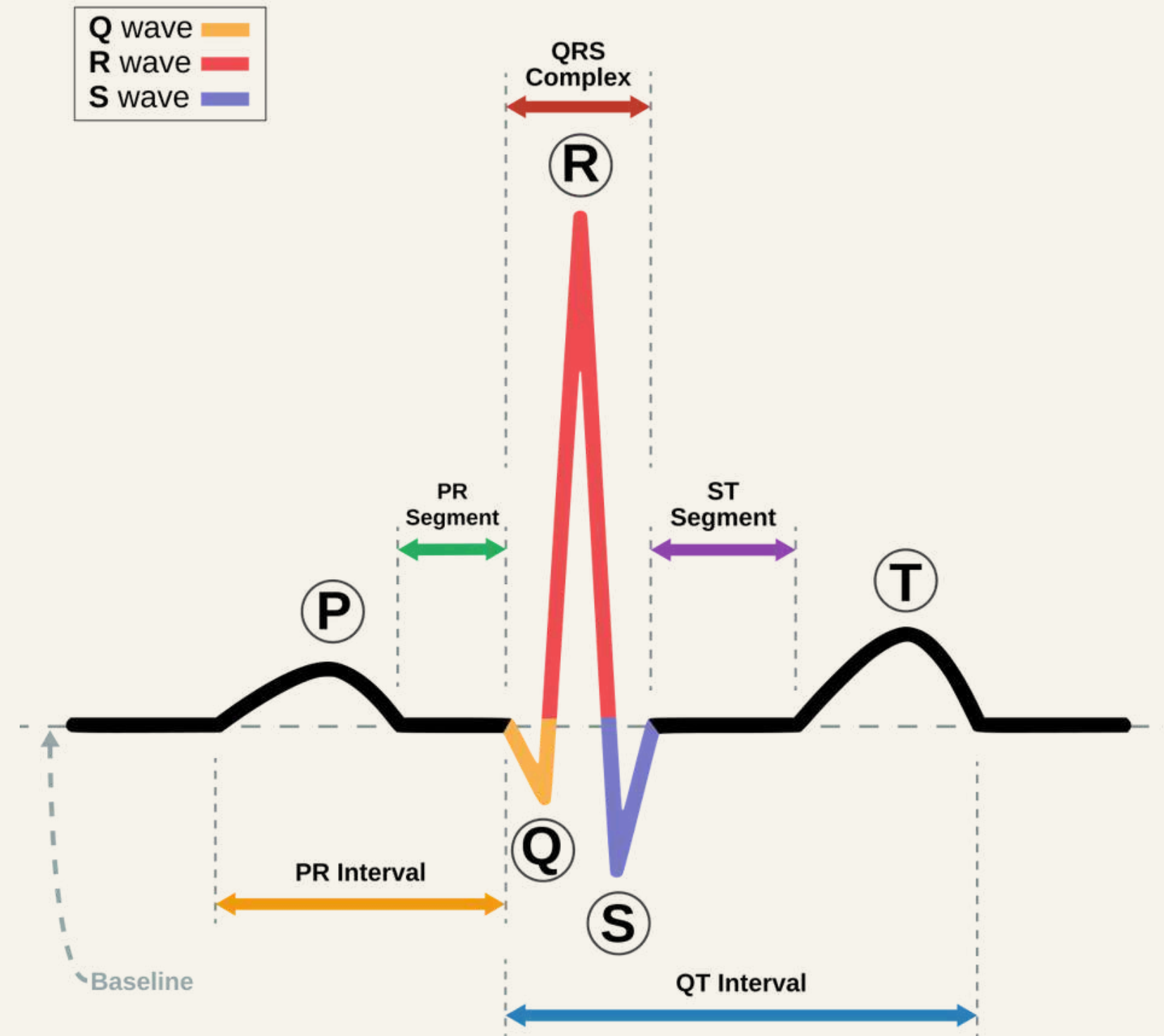
Relaxation = Repolarization

Atrial Contraction = ??



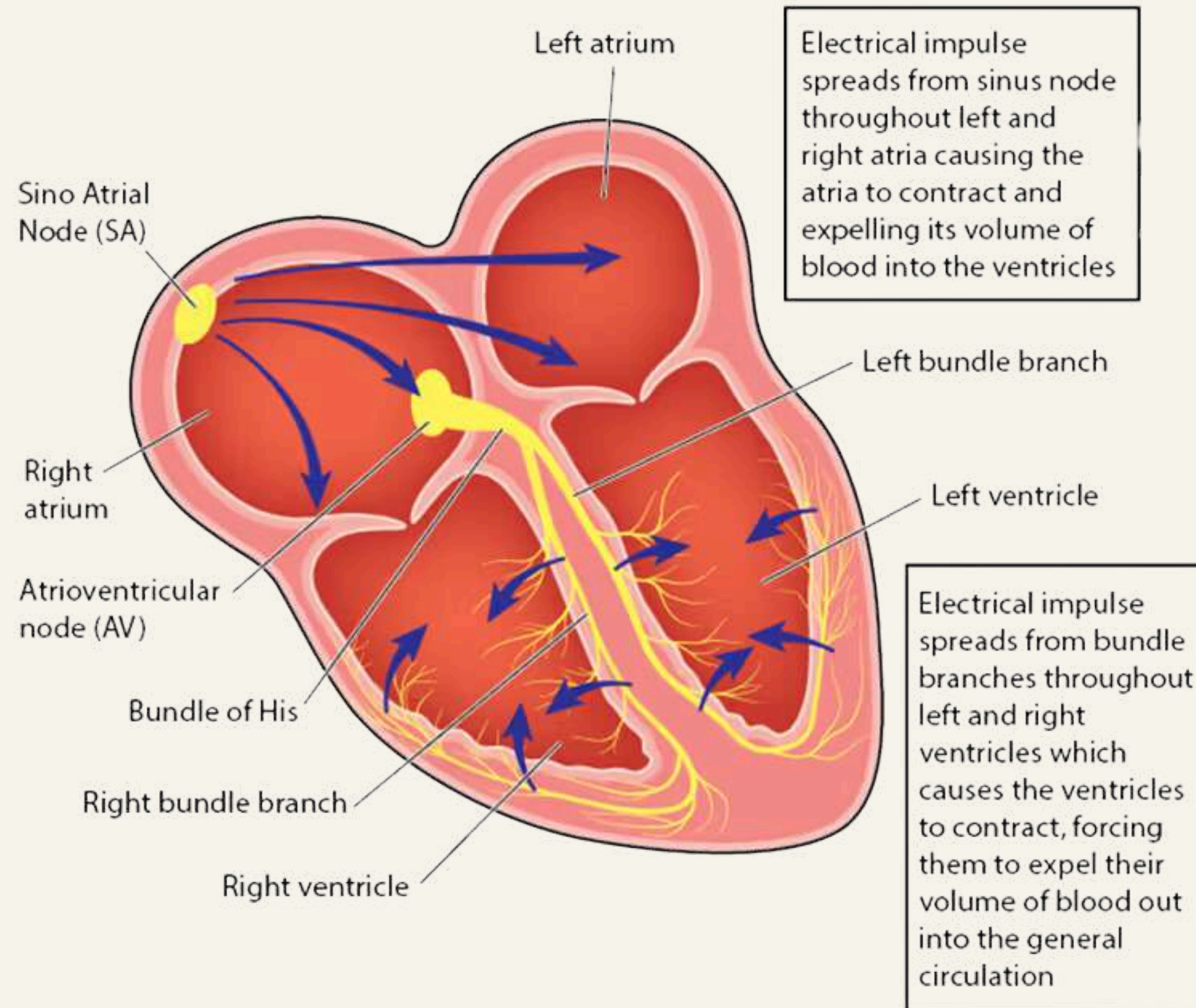
Ventricular Relaxation = ??

NORMAL WAVES

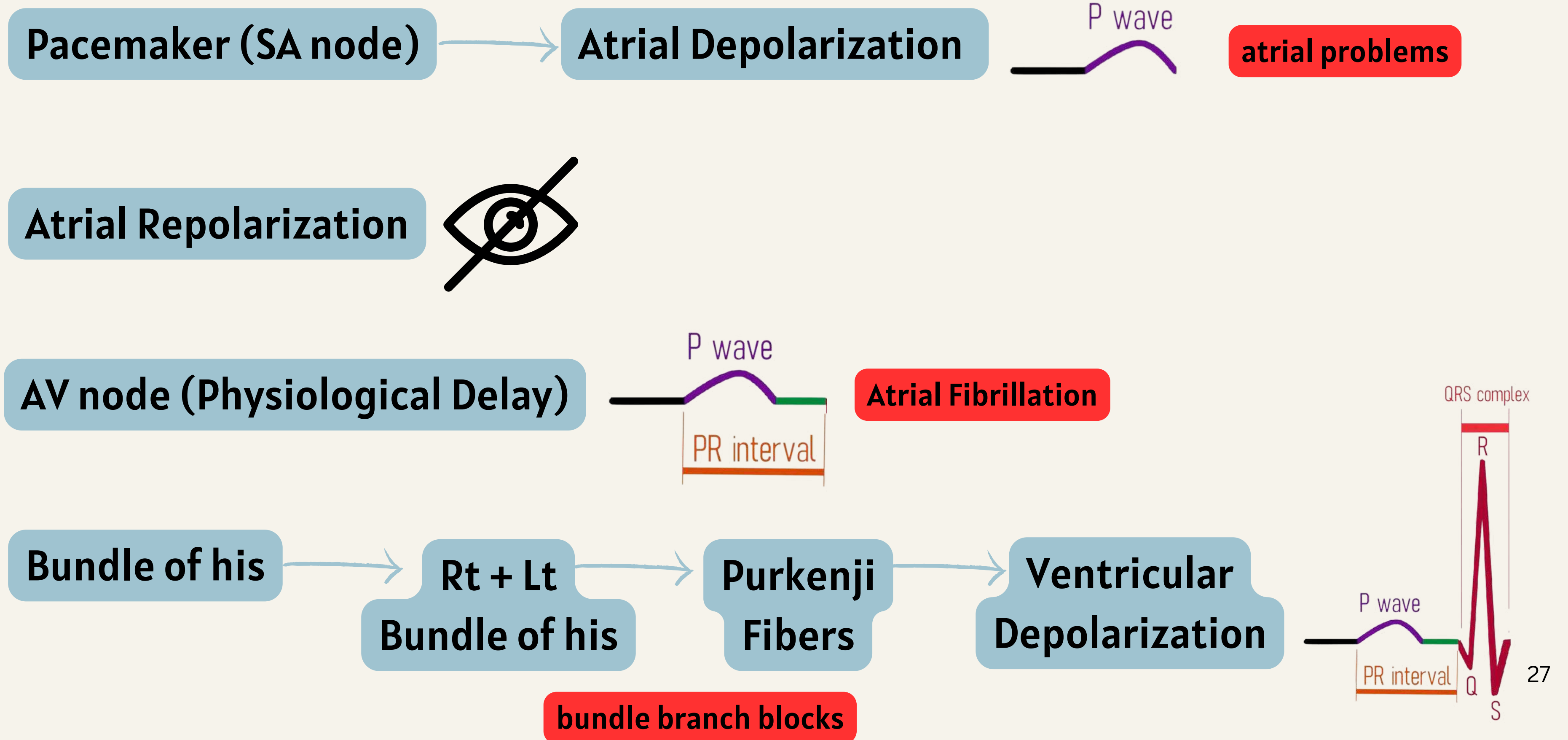


Prolonged in risk for arrhythmias

CONDUCTIVE SYSTEM OF HEART

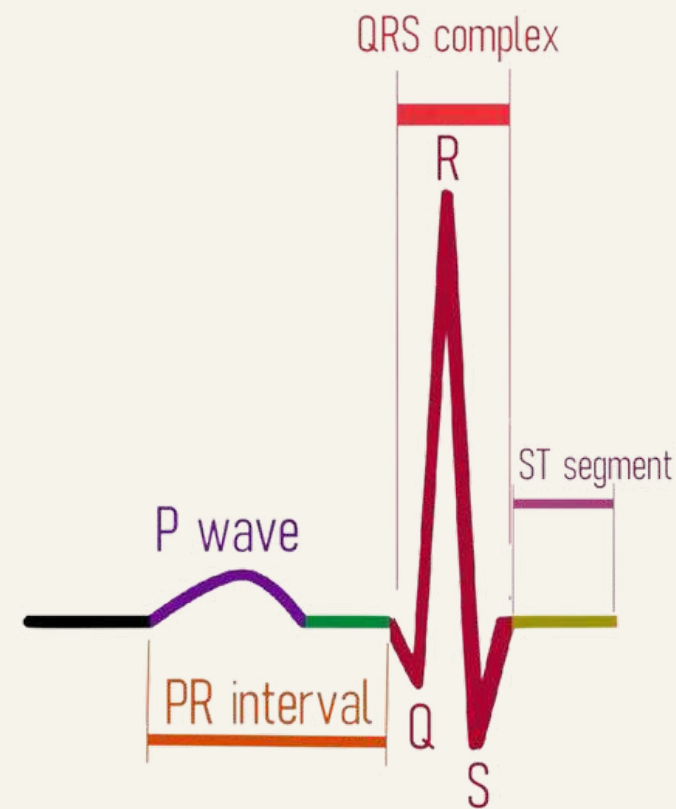


HOW IS EACH WAVE GENERATED IN THE ECG?



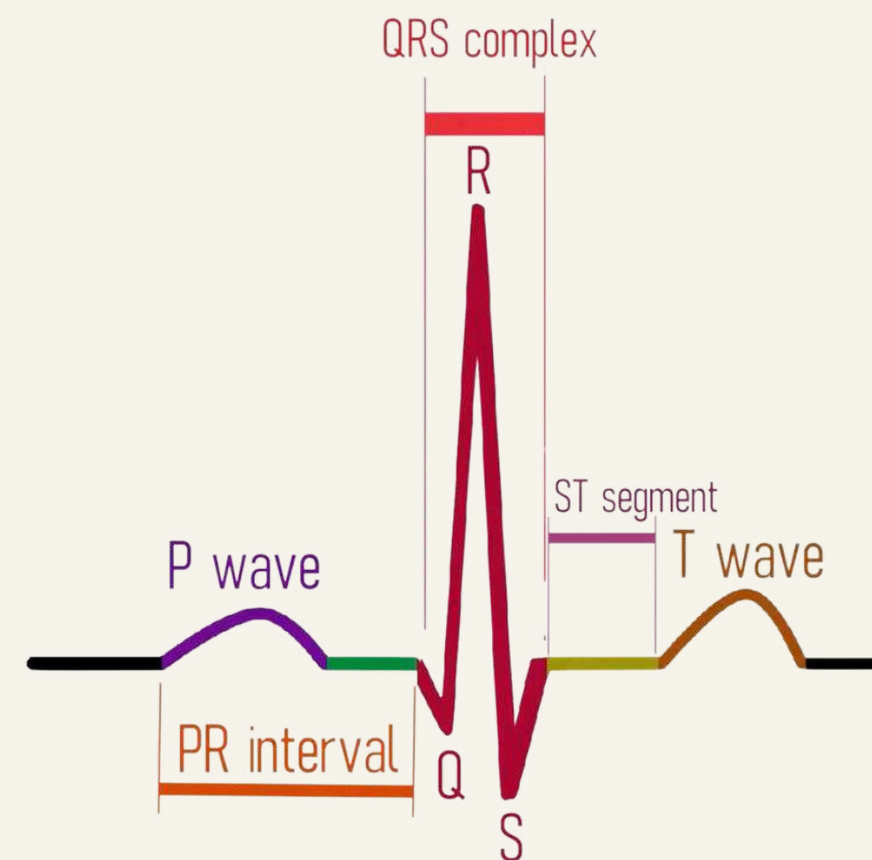
HOW IS EACH WAVE GENERATED IN THE ECG?

Isoelectric Period

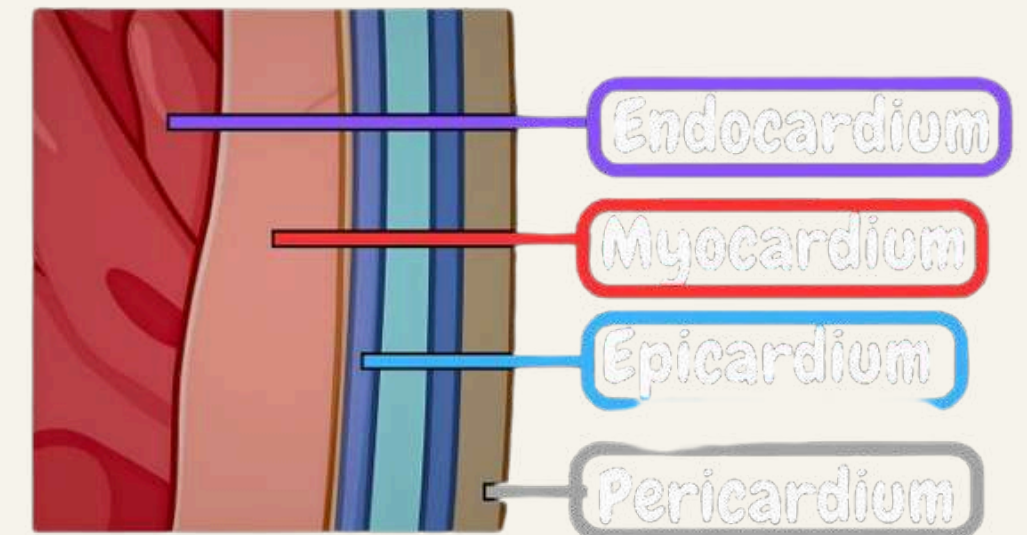


elevation or depression indicates ischemia

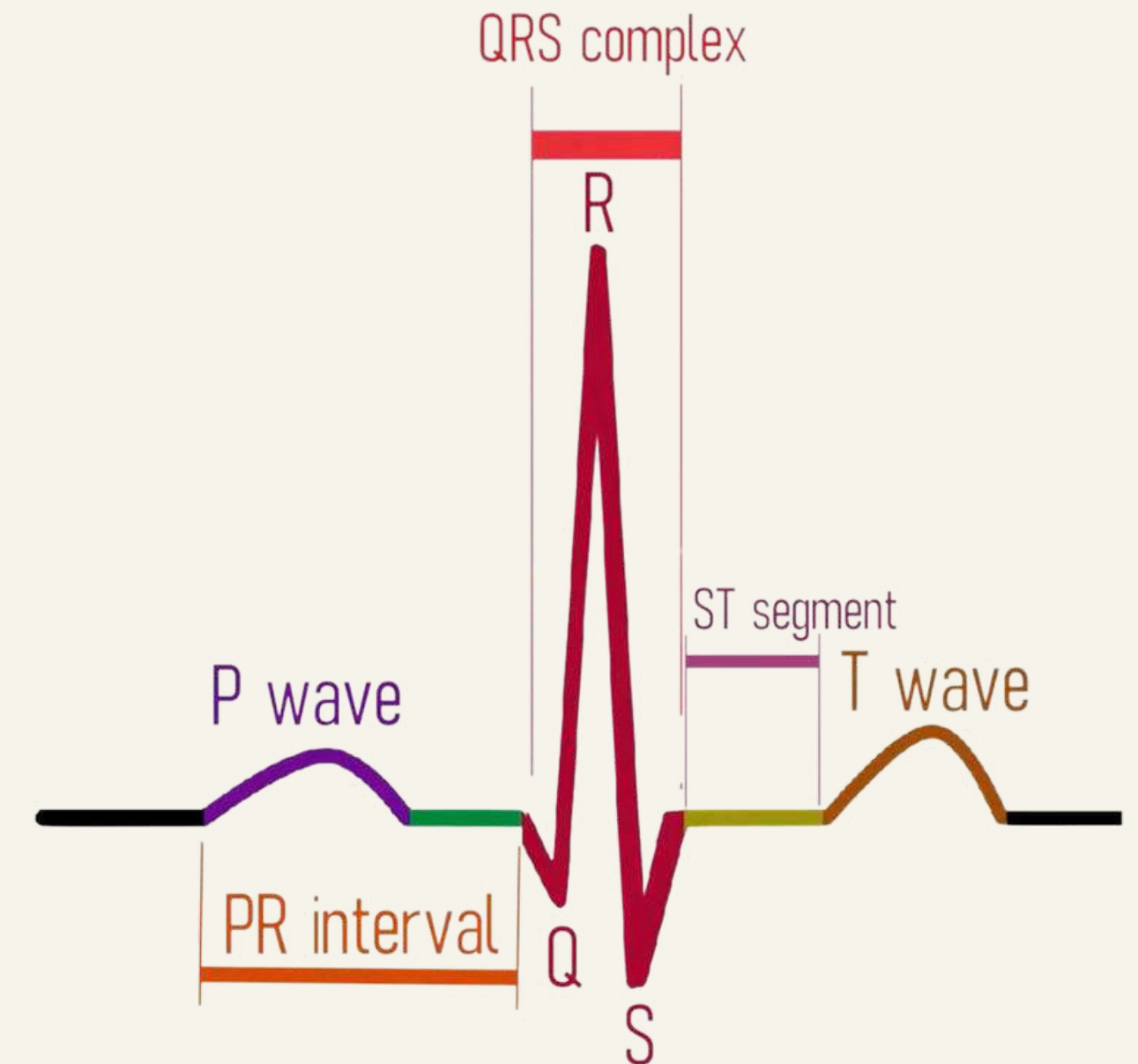
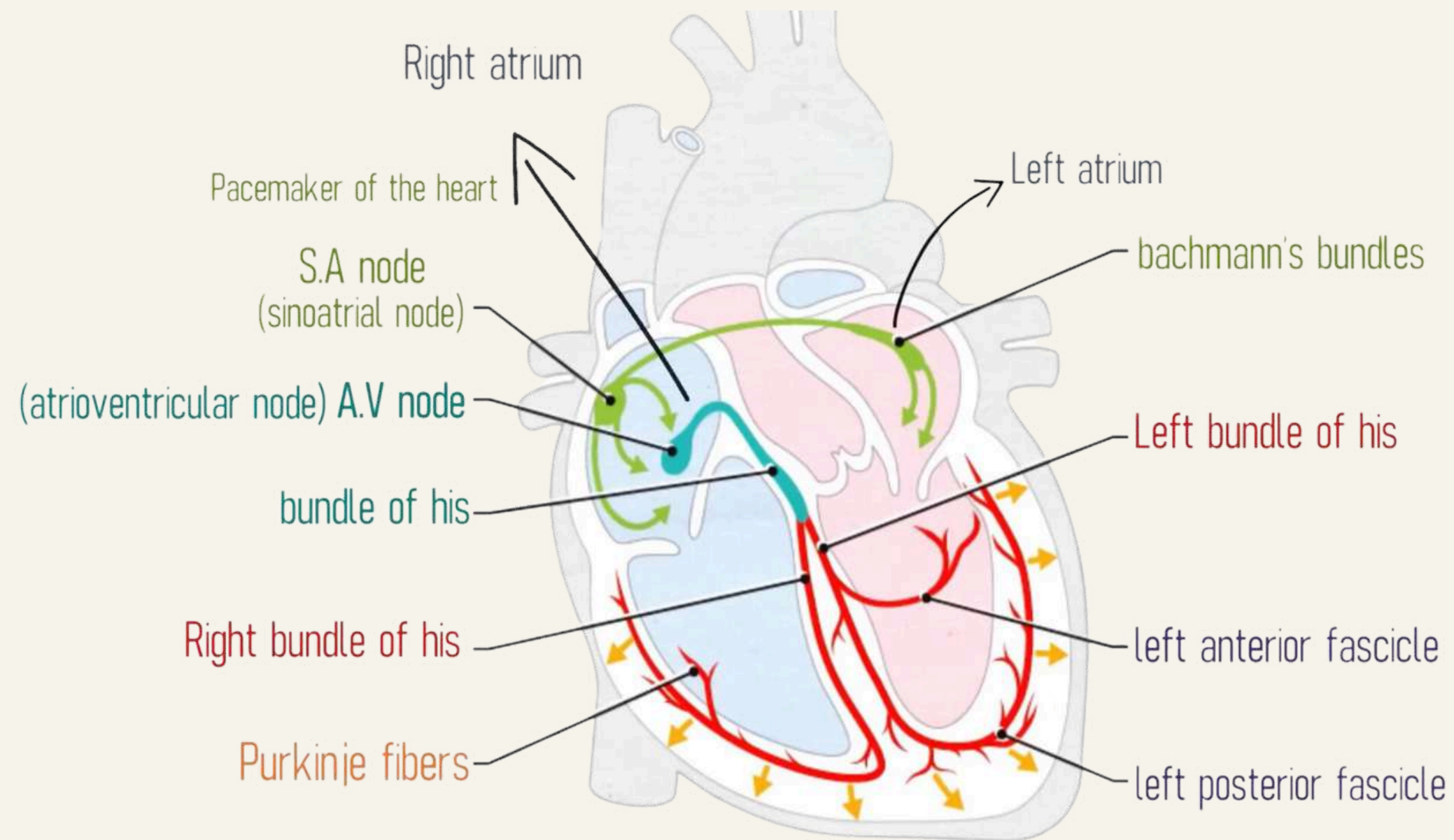
Ventricular Repolarization



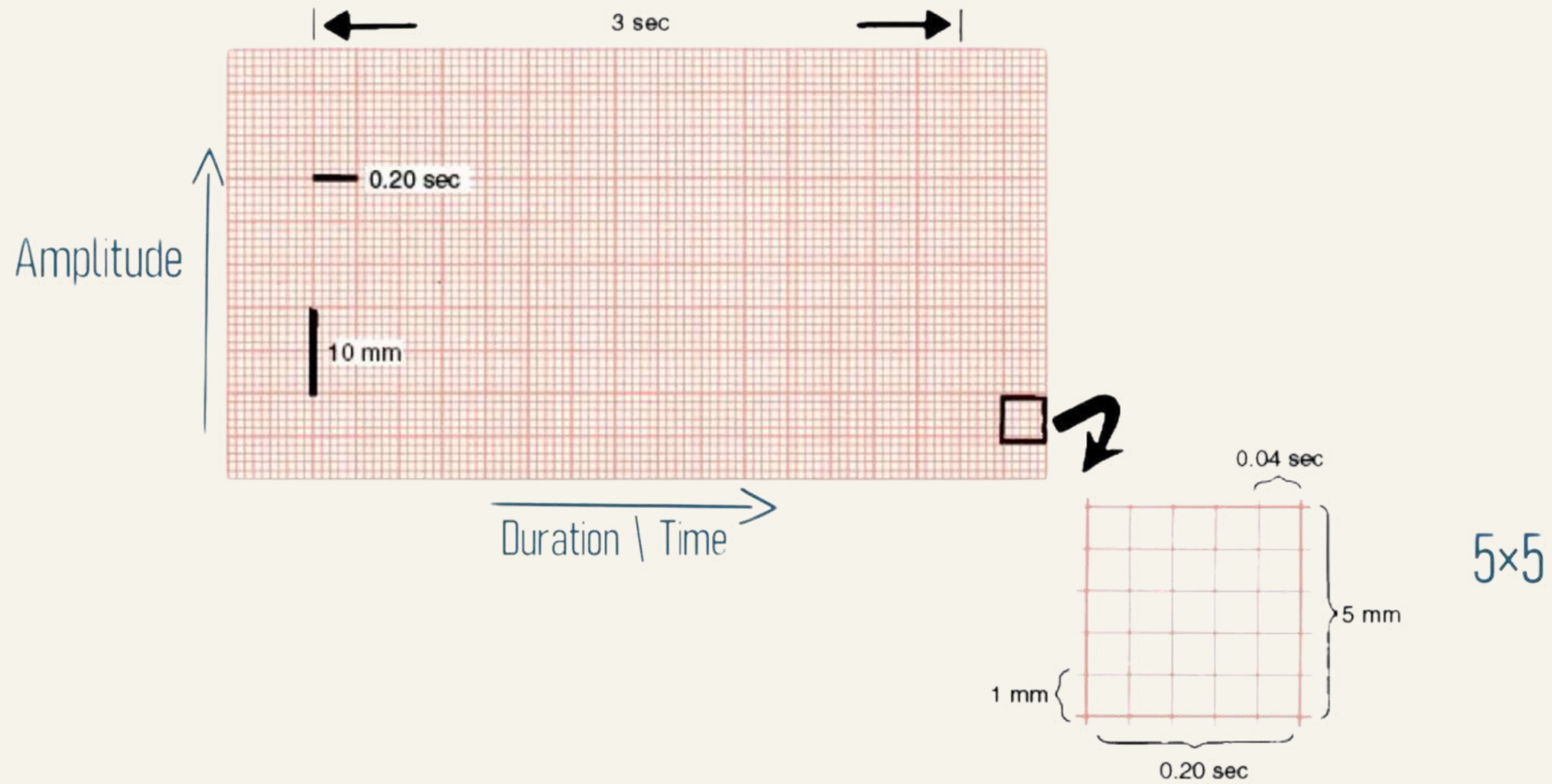
inversion suggests ischemia



HOW IS EACH WAVE GENERATED IN THE ECG?



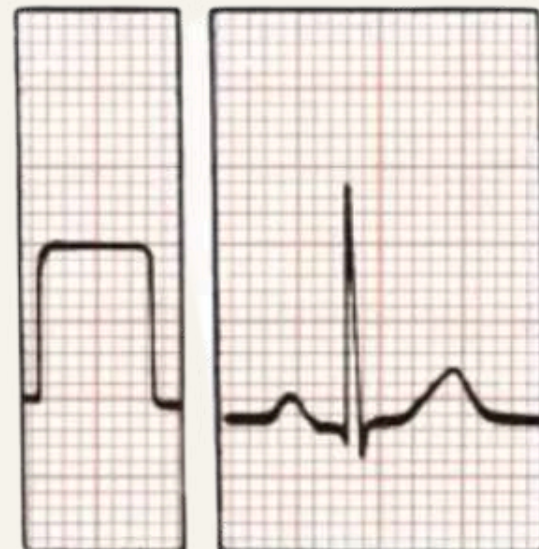
ECG PAPER



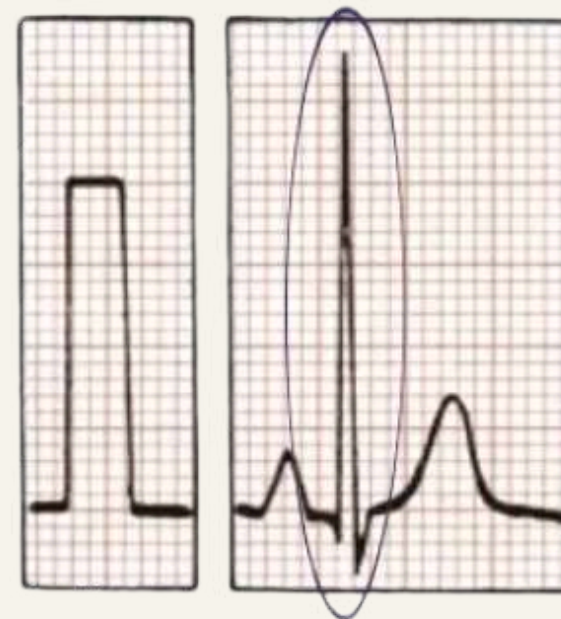
STANDARDIZATION MARK



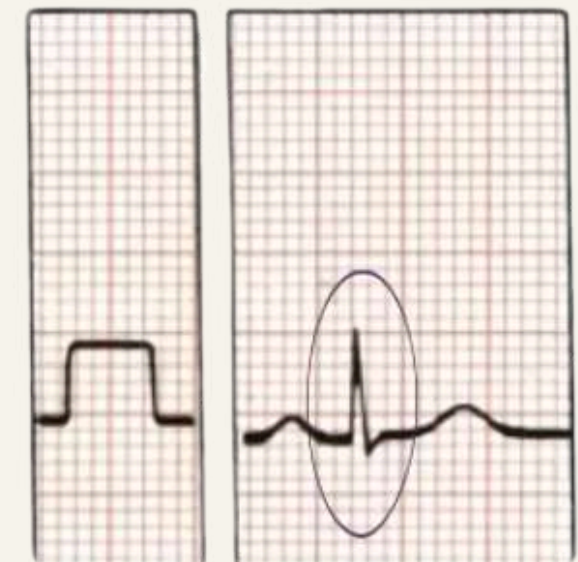
Normal



2 big squares tall
Indicates normal ventricular size and function.



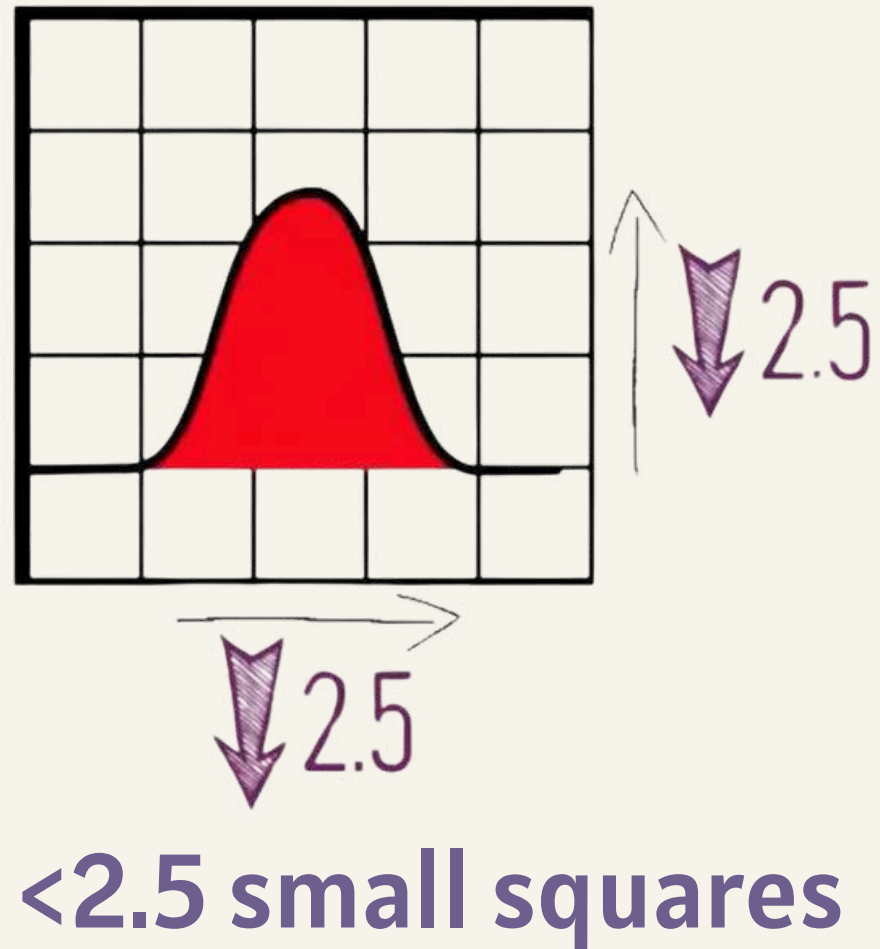
A smaller QRS complex -> obesity, pericardial effusion -> let make QRS taller to see it



A taller QRS complex -> ventricular hypertrophy -> let make QRS shorter to see it

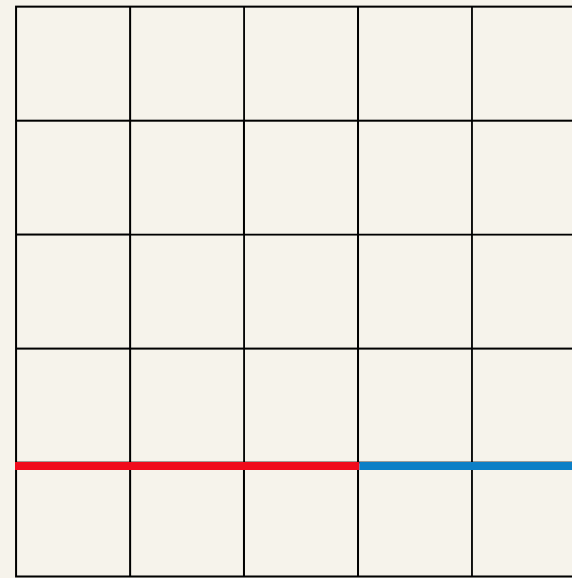
NORMAL WAVES

P wave



PR interval

P wave - QRS complex



3-5 small squares

QRS complex

Height : 1-5 big squares

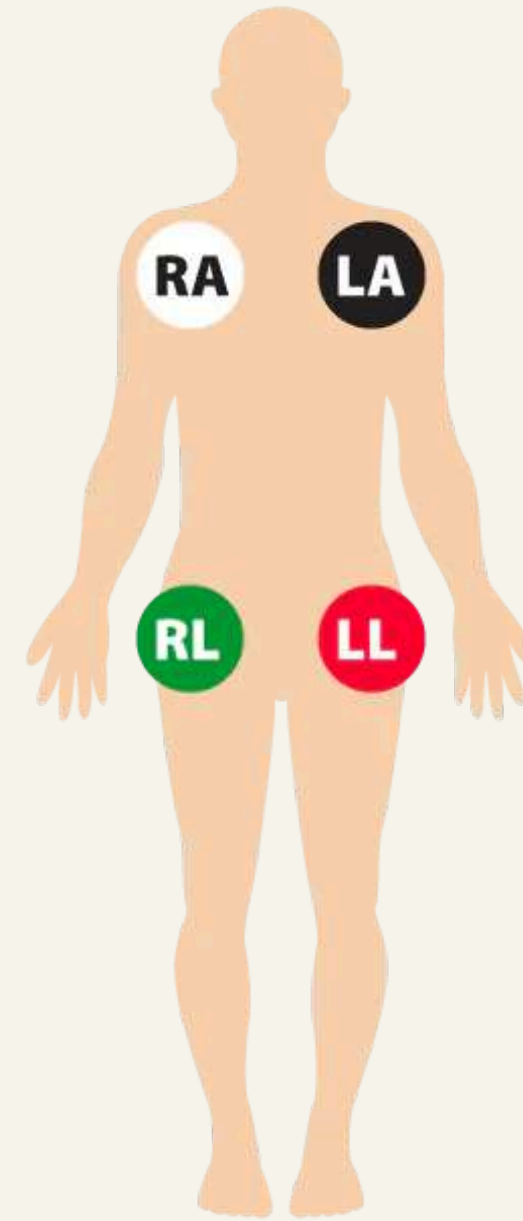
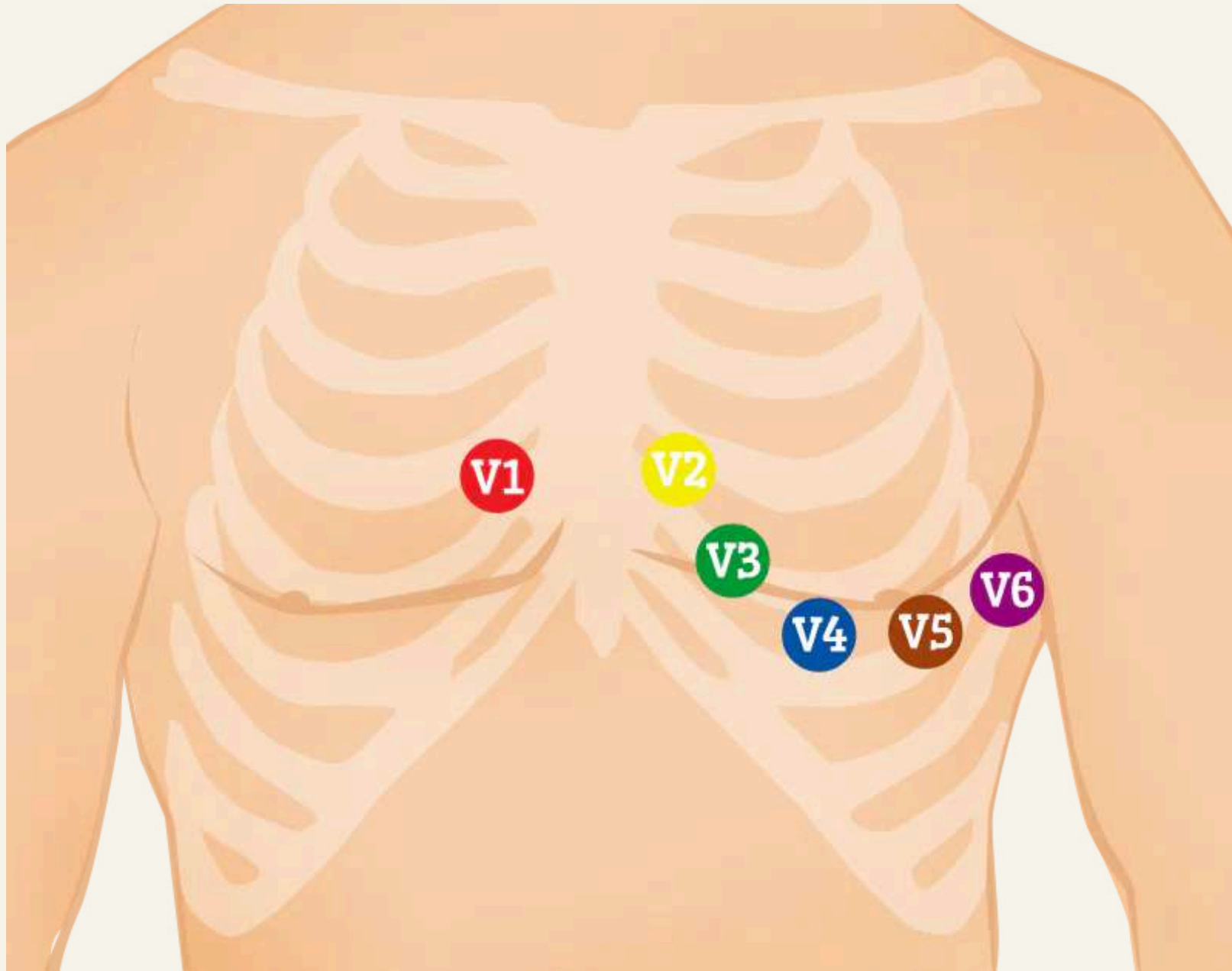
Width : <3 small squares

T wave

Height : <2 big squares

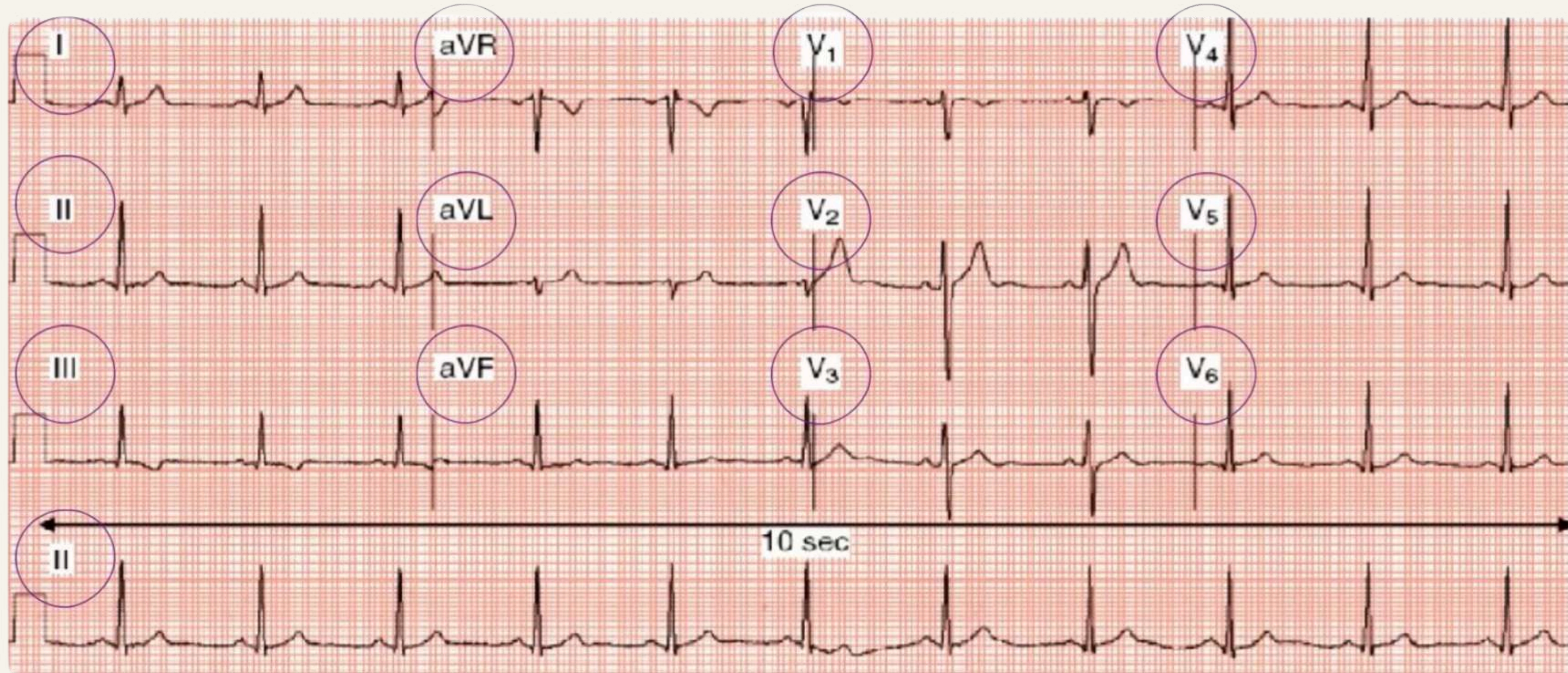
Width : <6 small squares

THE 12 LEAD ECG



- RA** Right Arm
- LA** Left Arm
- LL** Left Leg
- RL** Right Leg

THE 12 LEAD ECG



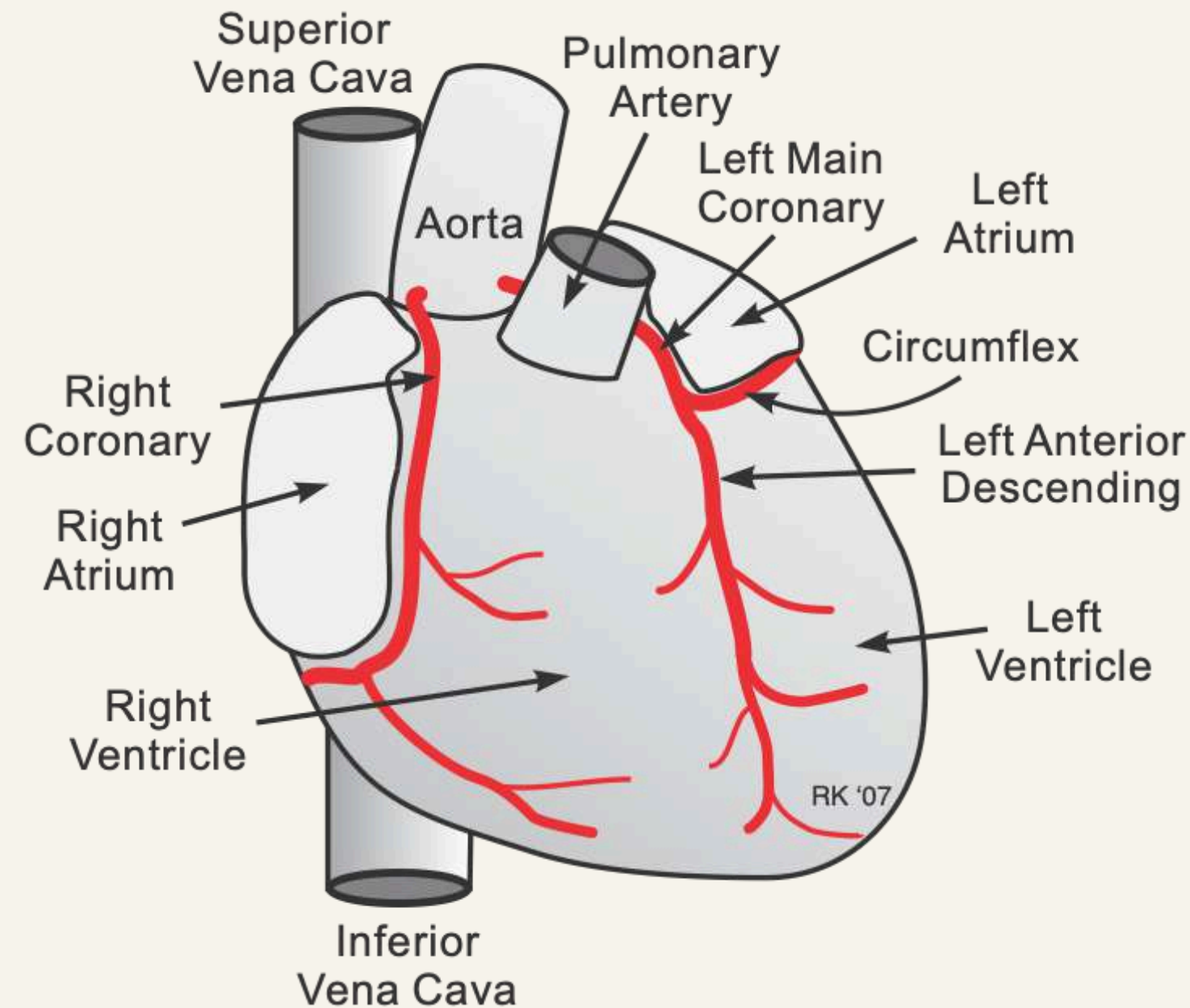
There are 12 ECG leads calculated using 10 electrodes.

ECG LEADS AND THEIR HEART VIEWS

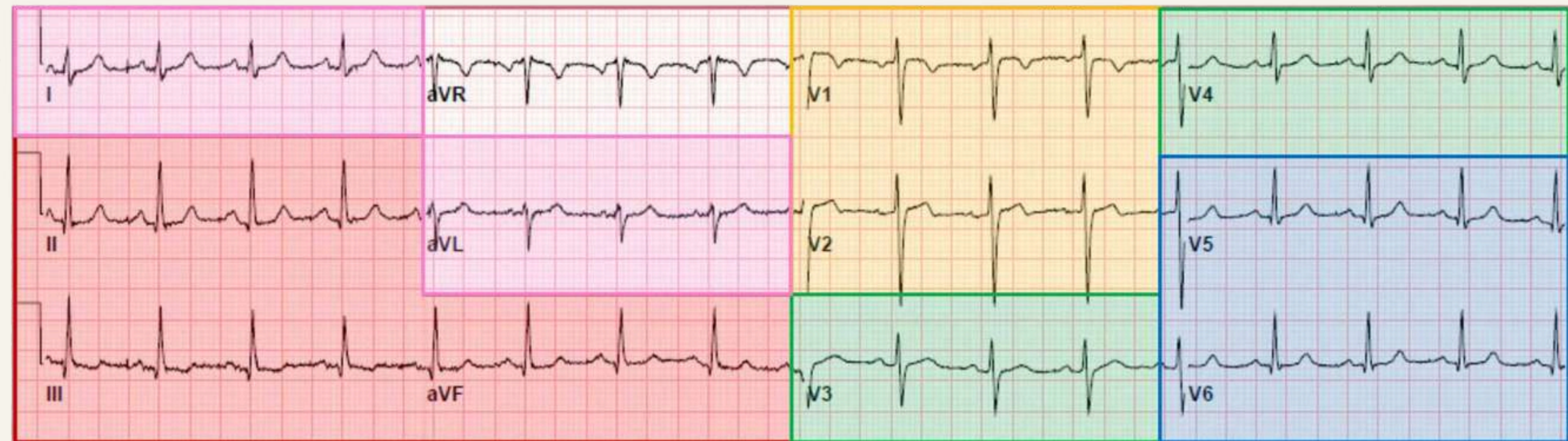
I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

- Limb Leads (I, II, III, aVR, aVL, aVF)
Lateral, inferior, and right-side views of the heart.
- Precordial Leads (V1-V6)
Septal, anterior, and lateral views

DETAILED ECG FINDINGS BY LEAD GROUP



INFERIOR II, III, aVF RCA	ANTEROSEPTAL V1 – V2 LAD	ANTEROAPICAL V3 – V4 LAD	ANTEROLATERAL V5 – V6 Cx	LATERAL I, aVL Cx
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- **Inferior Leads (II, III, aVF):** Right coronary artery; associated with inferior wall MI.
- **Lateral Leads (I, aVL, V5, V6):** Circumflex artery; lateral wall MI.
- **Anterior Leads (V3, V4):** Left anterior descending artery; anterior wall MI.
- **Septal Leads (V1, V2):** Left anterior descending artery; septal MI

HOW TO READ ECG??

Heart rate

What is a normal adult heart rate?

- **Normal:** 60-100 bpm
- **Tachycardia:** > 100 bpm
- **Bradycardia:** < 60 bpm

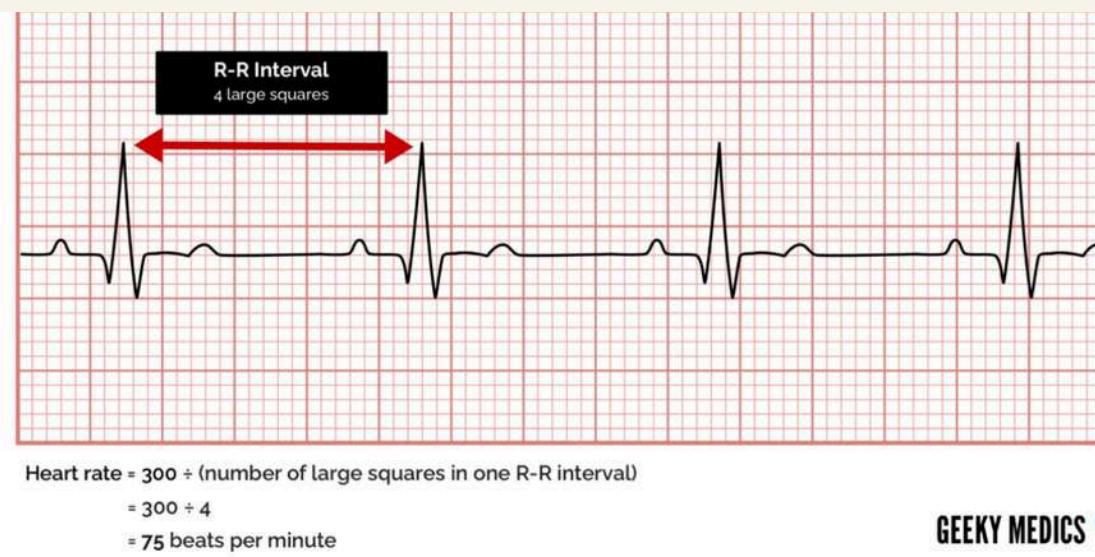
Regular heart rhythm

If a patient has a **regular heart rhythm**, their heart rate can be calculated using the following method:

- **Count** the number of **large squares** present within one **R-R interval**.
- **Divide 300** by **this number** to calculate **heart rate**.

Heart rate calculation example

- 4 large squares in an R-R interval
- $300/4 = 75$ beats per minute



Irregular heart rhythm

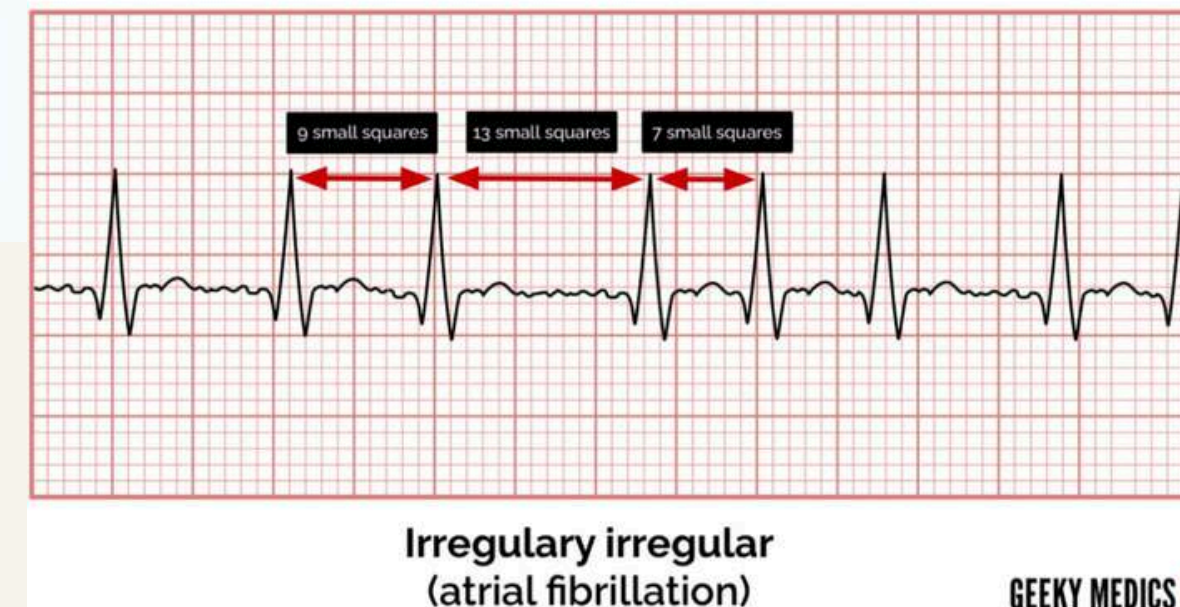
If a patient's heart rhythm is **irregular**, the first heart rate calculation method doesn't work (as the R-R interval differs significantly throughout the ECG). As a result, you need to apply a different method:

- Count the number of complexes on the rhythm strip (each rhythm strip is typically 10 seconds long).
- Multiply the number of complexes by 6 (giving you the average number of complexes in 1 minute).

Heart rate calculation example

- 10 complexes on a rhythm strip
- $10 \times 6 = 60$ beats per minute

Heart Rhythm



HOW TO READ ECG??

Cardiac axis

Cardiac axis describes the overall **direction of electrical spread** within the heart.

In a healthy individual, the axis should spread from **11 o'clock to 5 o'clock**.

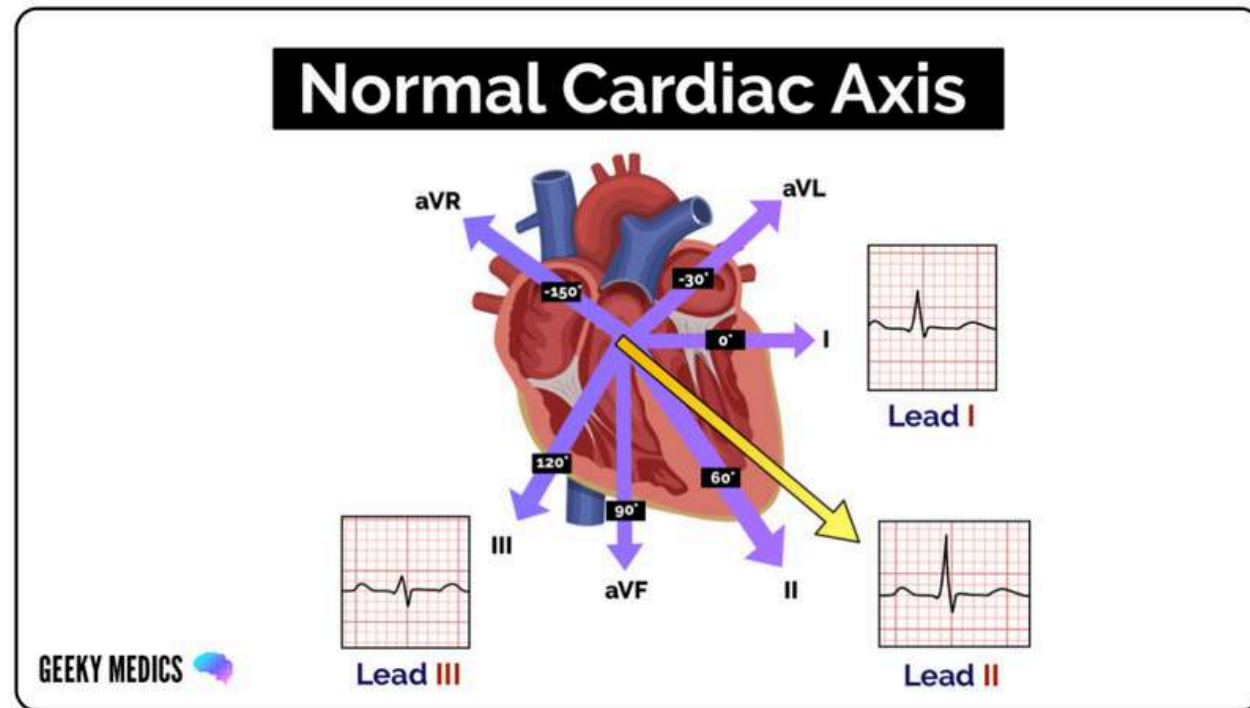
To determine the cardiac axis, you must look at **leads I, II and III**.

Read our [cardiac axis guide](#) to learn more.

Normal cardiac axis

Typical ECG findings for **normal cardiac axis**:

- **Lead II** has the **most positive deflection** compared to leads I and III.

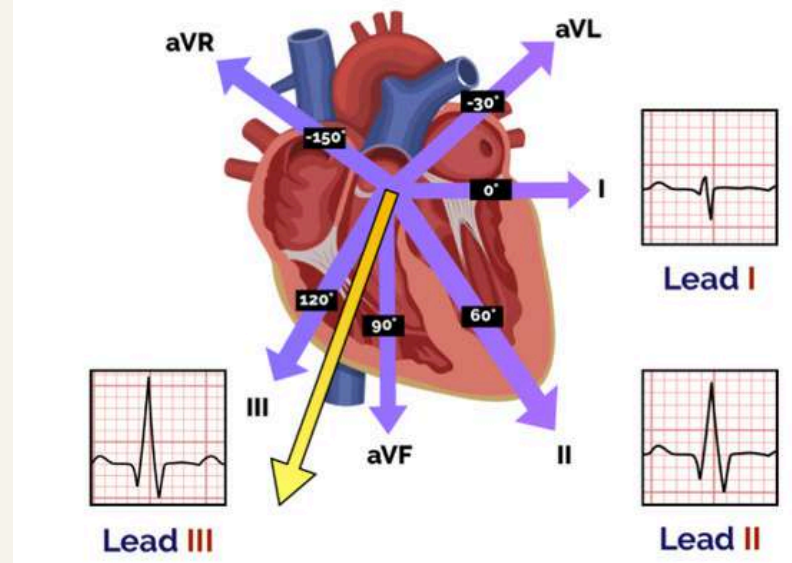


Right axis deviation

Typical ECG findings for **right axis deviation**:

- **Lead III** has the **most positive deflection**, and **lead I** should be **negative**.
- Right axis deviation is associated with right ventricular hypertrophy.

Right Axis Deviation

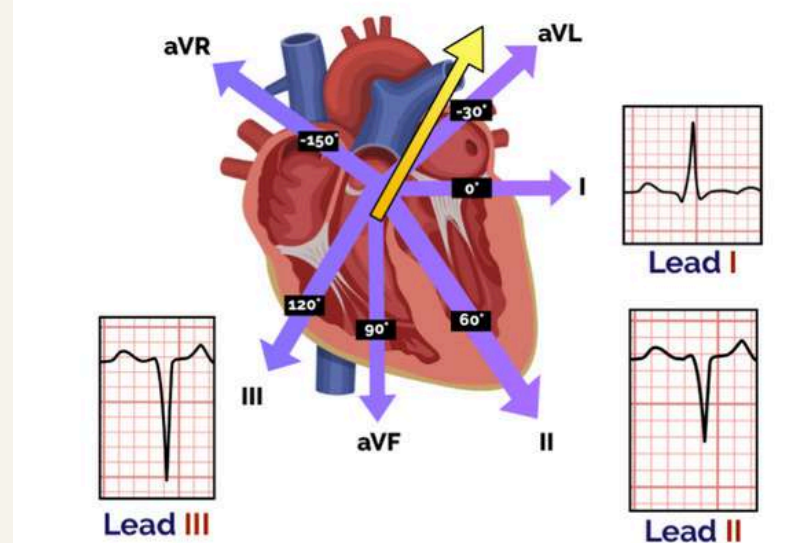


Left axis deviation

Typical ECG findings for **left axis deviation**:

- **Lead I** has the **most positive deflection**.
- **Leads II and III** are **negative**.
- Left axis deviation is associated with heart conduction abnormalities.

Left Axis Deviation



HOW TO READ ECG??

P waves

The next step is to **look at the P waves** and answer the following questions:

1. Are P waves present?
2. If so, is each P wave followed by a QRS complex?
3. Do the P waves look normal? – check duration, direction and shape
4. If P waves are absent, is there any atrial activity?
 - Sawtooth baseline → flutter waves
 - Chaotic baseline → fibrillation waves
 - Flat line → no atrial activity at all

Hint

If P waves are **absent** and there is an **irregular rhythm**, it may suggest a diagnosis of **atrial fibrillation**.

HOW TO READ ECG??

PR interval

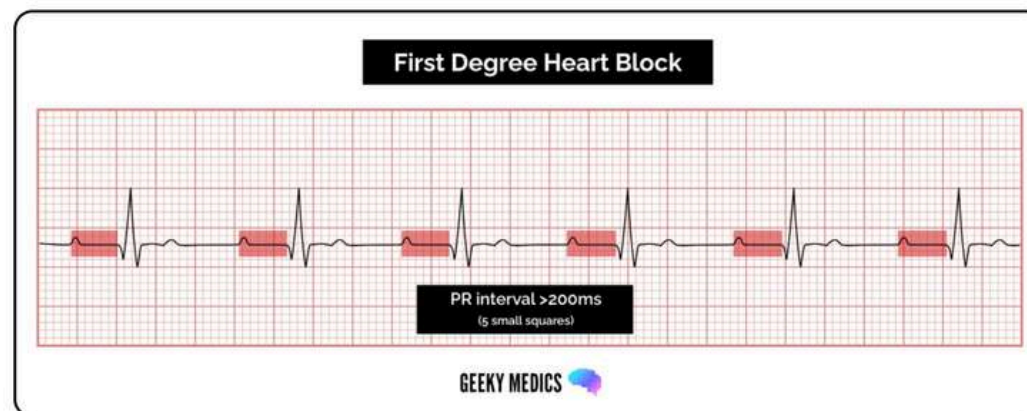
The **PR interval** should be between **120-200 ms** (3-5 small squares).

Prolonged PR interval (>0.2 seconds)

A prolonged PR interval suggests the presence of **atrioventricular delay (AV block)**.

First-degree heart block (AV block)

First-degree heart block involves a **fixed prolonged** PR interval (>200 ms).

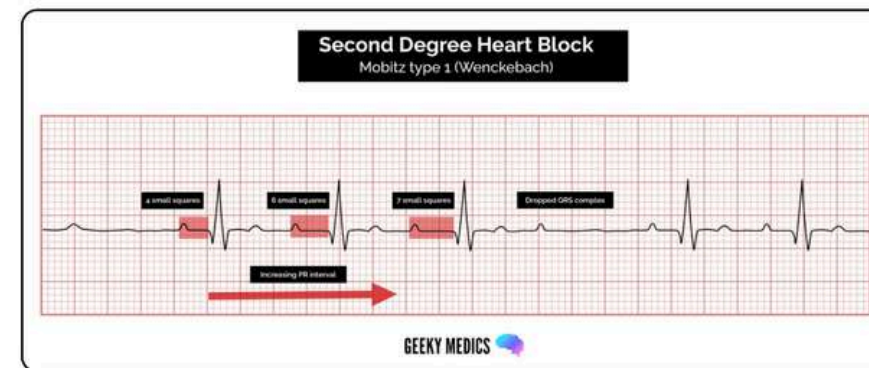


Second-degree heart block (type 1)

Second-degree AV block (type 1) is also known as **Mobitz type 1 AV block** or **Wenckebach phenomenon**.

Typical ECG findings in Mobitz type 1 AV block include **progressive prolongation of the PR interval** until eventually the atrial impulse is not conducted and the **QRS complex is dropped**.

AV nodal conduction **resumes** with the next beat, and the sequence of progressive PR interval prolongation and the eventual dropping of a QRS complex **repeats** itself.



Second-degree AV block (Mobitz Type 1 - Wenckebach)

Third-degree heart block (complete heart block)

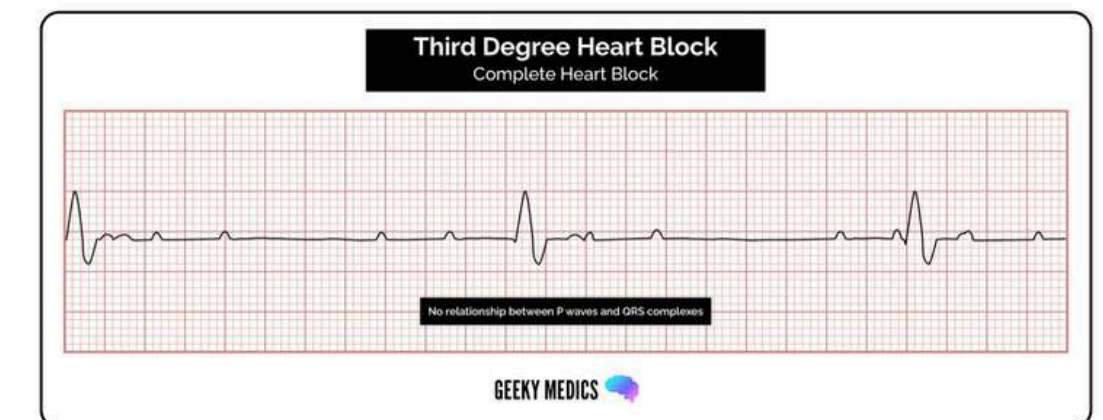
Third-degree (complete) AV block occurs when there is **no electrical communication between the atria and ventricles** due to a complete failure of conduction.

Typical ECG findings include the **presence of P waves** and **QRS complexes** that have **no association with each other**, due to the atria and ventricles functioning independently.

Cardiac function is maintained by a **junctional** or **ventricular pacemaker**.

Narrow-complex escape rhythms (QRS complexes of <0.12 seconds duration) originate **above the bifurcation of the bundle of His**.

Broad-complex escape rhythms (QRS complexes >0.12 seconds duration) originate from **below the bifurcation of the bundle of His**.



Complete heart block (3rd degree)

HOW TO READ ECG??

Tips for remembering types of heart block

To help remember the various types of AV block, it is useful to know the anatomical location of the block within the conducting system.

First-degree AV block:

- Occurs between the SA node and the AV node (i.e. within the atrium).

Second-degree AV block:

- Mobitz I AV block (Wenckebach) occurs IN the AV node (this is the only piece of conductive tissue in the heart which exhibits the ability to conduct at different speeds).
- Mobitz II AV block occurs AFTER the AV node in the bundle of His or Purkinje fibres.

Third-degree AV block:

- Occurs at or after the AV node resulting in a complete blockade of distal conduction.

Shortened PR interval

If the **PR interval** is **shortened**, this can mean one of two things:

- Simply, the P wave originates from somewhere closer to the AV node, so the conduction takes less time (the SA node is not in a fixed place, and some people's atria are smaller than others).
- The atrial impulse is getting to the ventricle by a **faster shortcut** instead of conducting slowly across the atrial wall. This **accessory pathway** can be associated with a **delta wave** (see below).

Delta Wave

Associated with Wolff-Parkinson-White Syndrome



HOW TO READ ECG??

Bundle branch block

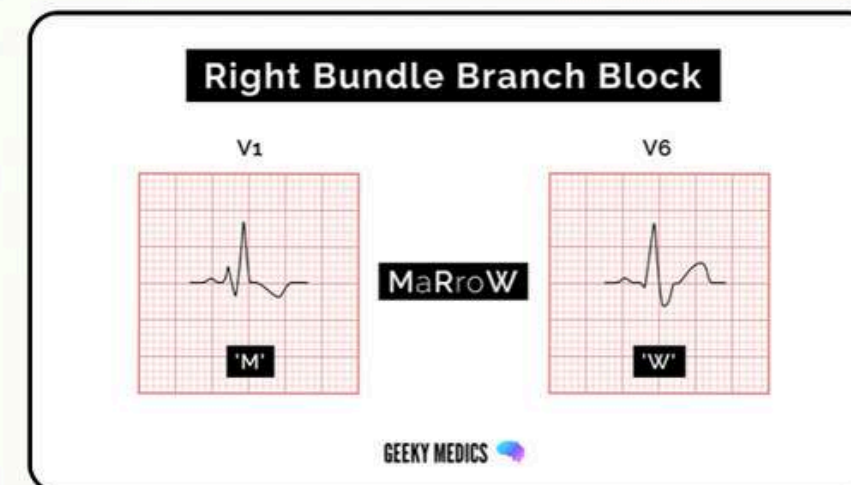
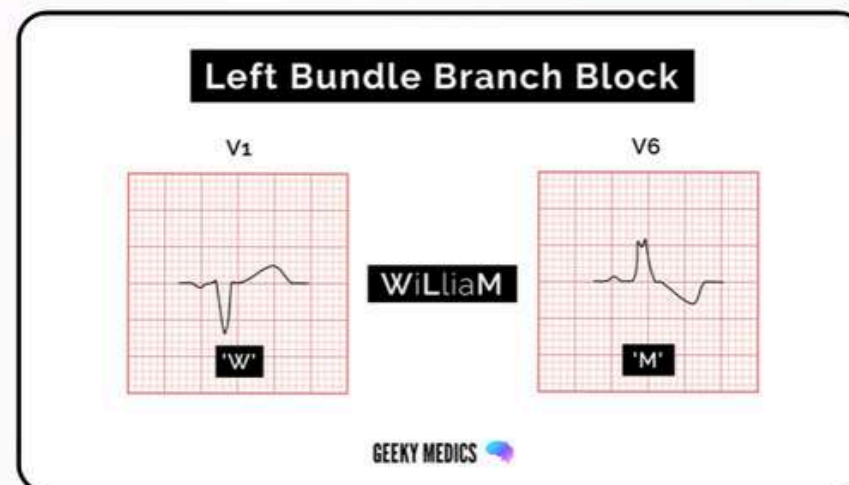
Broad QRS complexes are a hallmark feature of **bundle branch block**, which includes **left bundle branch block** (LBBB) and **right bundle branch block** (RBBB).

The **WiLLiaM MaRRoW** mnemonic can be used to quickly recognise left and right bundle branch blocks by looking at **V1** and **V6**.

The **middle letters** of the names help you remember which bundle branch block each name is referring to (two Ls in Wi**LL**iaM = left bundle branch block, two Rs in Ma**RR**oW = right bundle branch block).

Each name's **first** and **last letter** helps you recognise the ECG features of the associated bundle branch block.

- **LBBB**: deep S wave in V1 which may be notched ("W") and broad "M" shaped R wave in V6
- **RBBB**: RSR' pattern in V1 ("M") and broad S wave in V6 ("W")



HOW TO READ ECG??

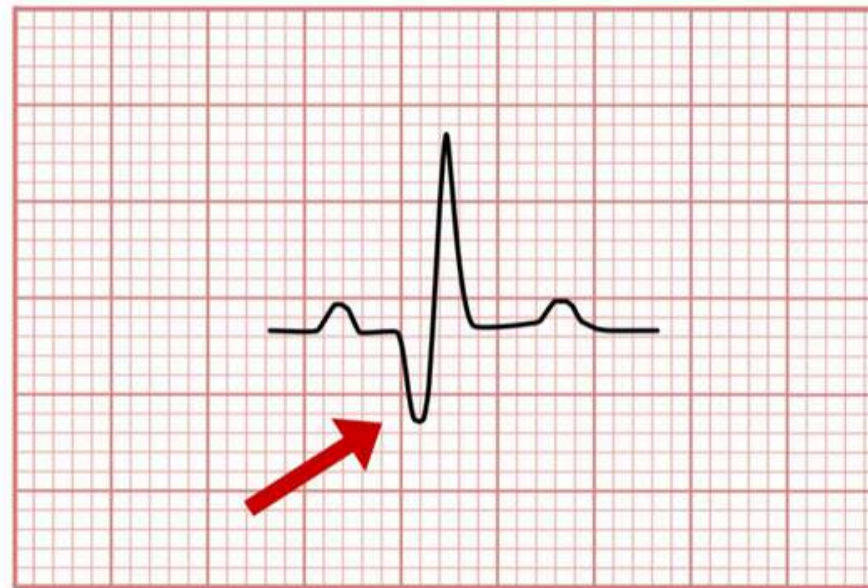
Q-waves

Isolated Q waves can be **normal**.

A **pathological Q wave** is **> 25% the size of the R wave that follows it** or **> 2mm in height and > 40ms in width**.

A single Q wave is not a cause for concern – look for Q waves in an entire territory (e.g. anterior/inferior) for evidence of previous myocardial infarction.

Pathological Q Wave



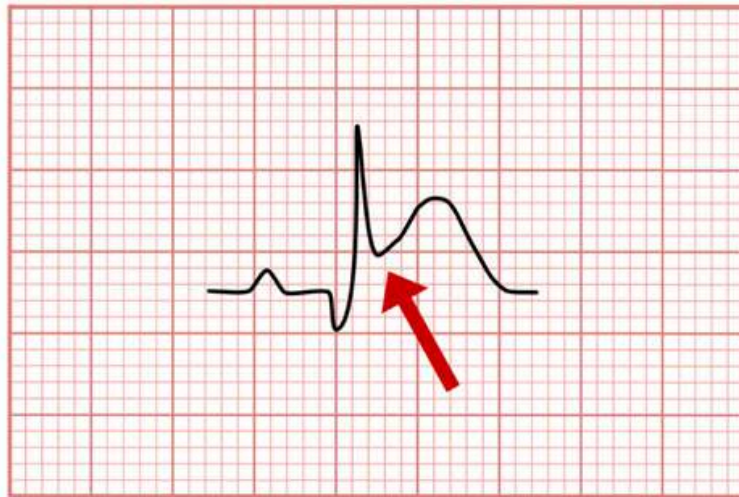
HOW TO READ ECG??

ST-elevation

ST-elevation is significant when it is **greater than 1 mm** (1 small square) in **2 or more contiguous limb leads** or **>2mm** in **2 or more chest leads**.

It is most commonly caused by **acute full-thickness myocardial infarction**.

ST Elevation



ST depression

ST depression ≥ 0.5 mm in **≥ 2 contiguous leads** indicates **myocardial ischaemia**.

ST Depression



GEEKY MEDICS 

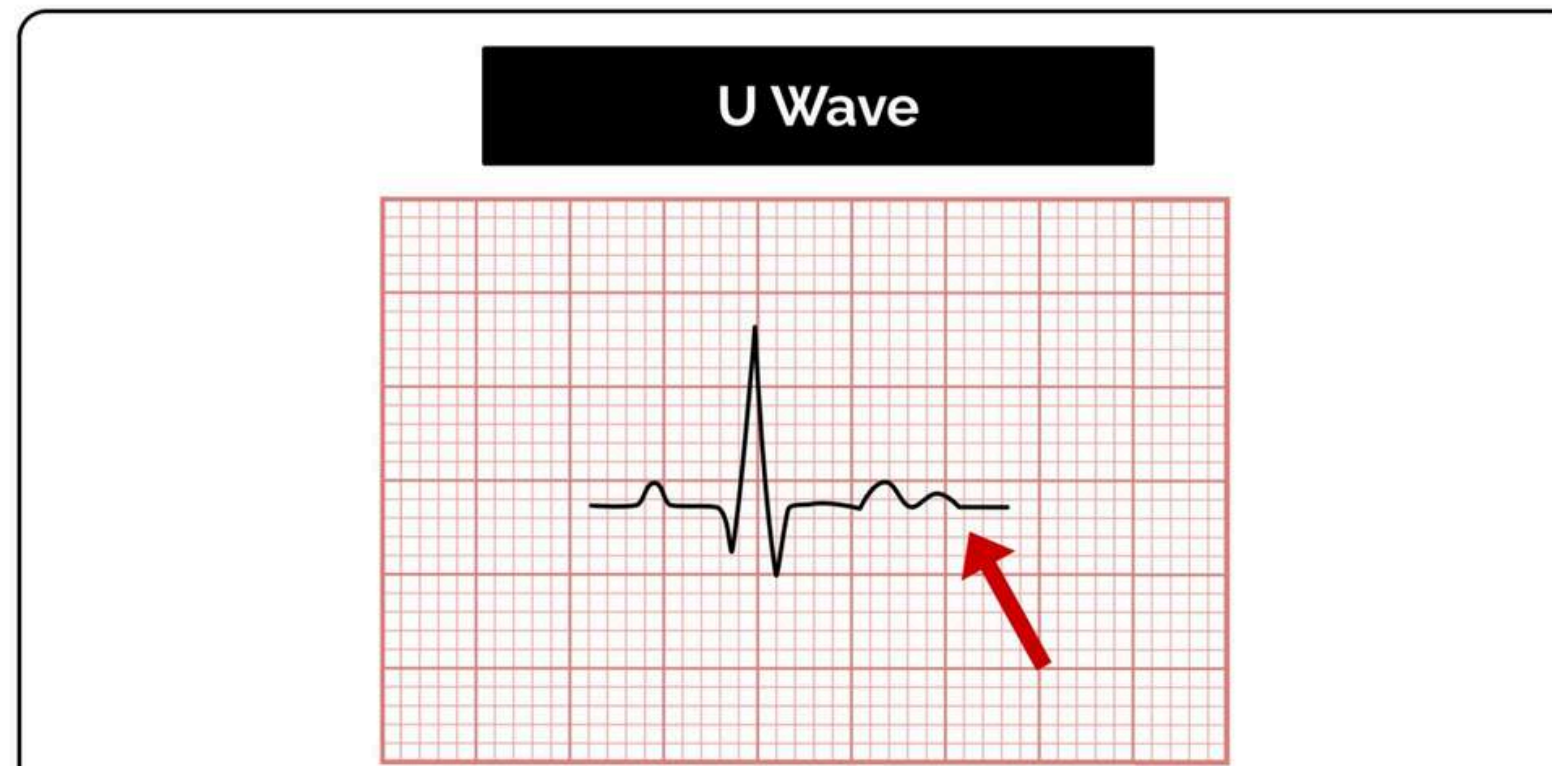
HOW TO READ ECG??

U waves

U waves are **not** a **common** finding.

The U wave is a **> 0.5mm deflection after the T wave** best seen in **V2** or **V3**.

These become larger the slower the bradycardia – classically U waves are seen in various **electrolyte imbalances**, **hypothermia** and secondary to **antiarrhythmic therapy** (such as digoxin, procainamide or amiodarone).

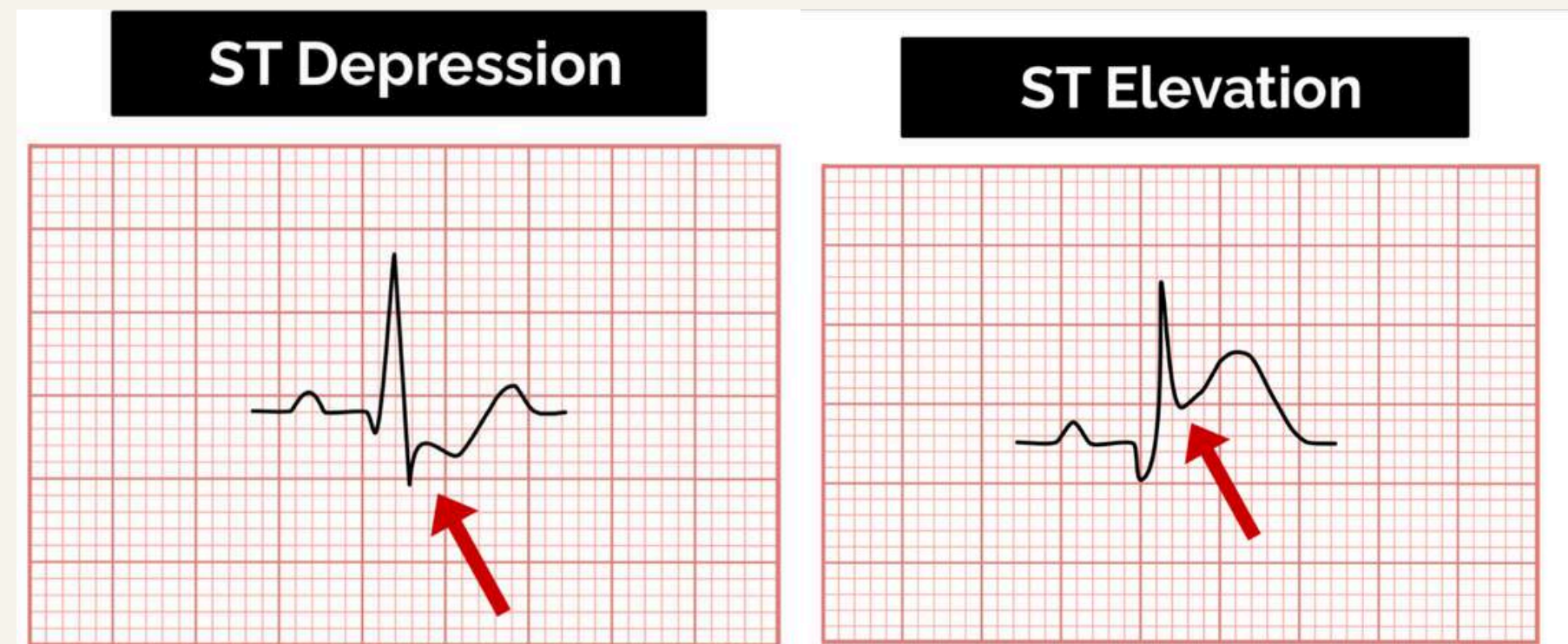


ASSESSMENT OF ACUTE CORONARY SYNDROME

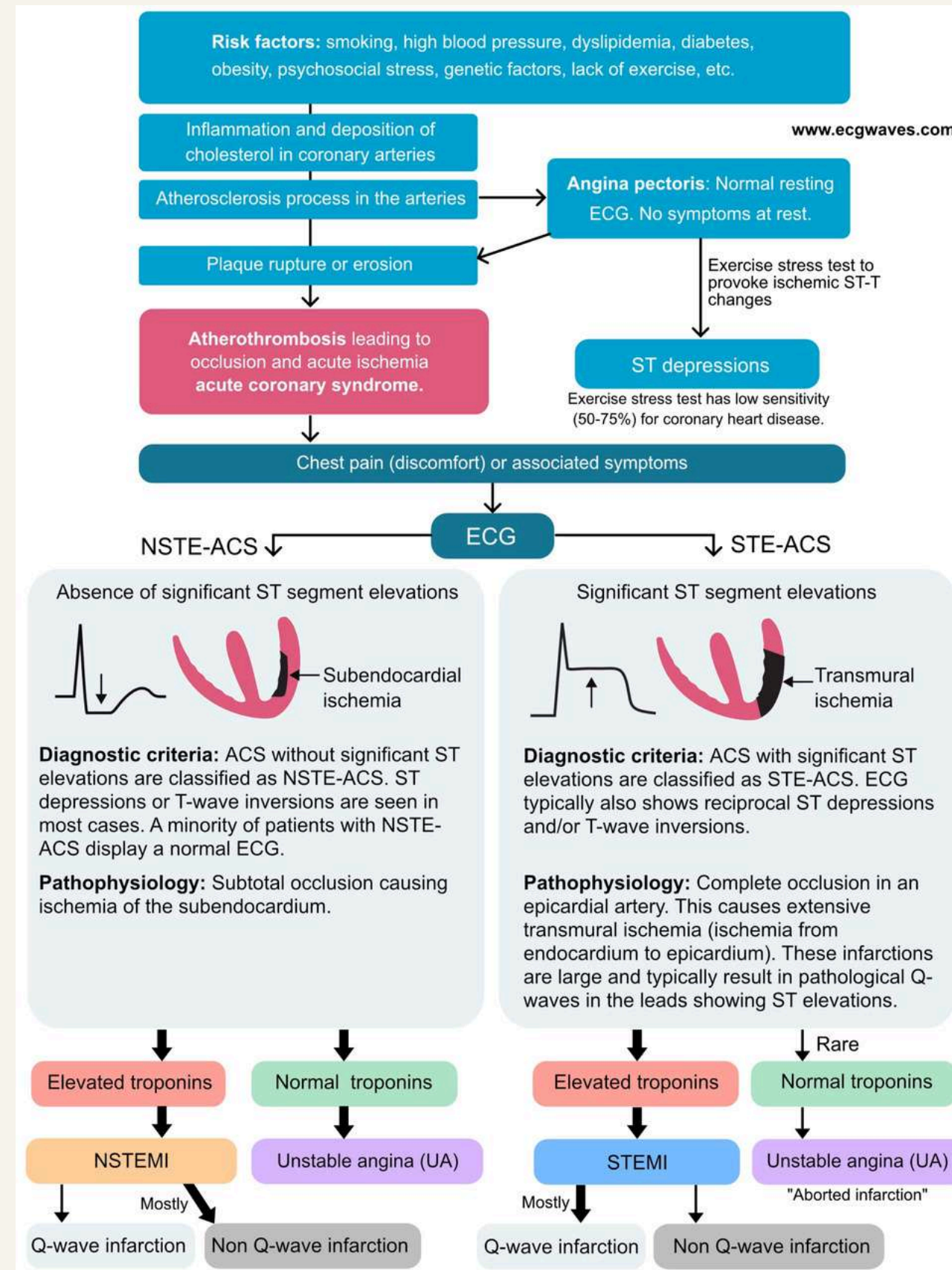
- **Patient History**
- **Physical Examination: Look for signs of heart failure, shock.**
- **ECG and Biomarkers:**
 - **12-lead ECG: Should be done within 10 minutes of presentation.**
 - **Troponin and CK-MB are Elevated in both STEMI and NSTEMI.**
 - **Negative troponin and CK -MB in unstable angina .**

ECG PATTERNS IN ACS

- **STEMI:** ST Elevation in specific leads. Q Waves: Develop over time, indicating full-thickness infarction.
- **NSTEMI:** ST Depression or T Wave Inversion; no ST elevation.
- **Stable and Unstable Angina:** May show normal ECG at rest; stress may reveal ST depression



ASSESSMENT OF ACUTE CORONARY SYNDROME



INITIAL ACS MANAGEMENT (MONA-B)

MONA-B Components:

- Morphine: For severe pain.
- Oxygen: If saturation <90%.
- Nitroglycerin: Unless contraindicated.
- Aspirin: Antiplatelet effect.
- Beta-blockers: To reduce heart workload



NSTEMI MANAGEMENT

Medical therapy:

1. DAPT (Oral antiplatelet). 2. Anticoagulan

- Assess for ongoing ischemia (Refractory chest pain, hemodynamic instability, dynamic ECG changes, and ventricular arrhythmia**
- If there is no evidence continued medical therapy. • If they do show evidence get immediate PCI or coronary artery bypass surgery**

STEMI MANAGEMENT

- **GOAL: QUICK REPERFUSION THERAPY.**

MEDICAL THERAPY :

1. DAPT (ORAL ANTIPLATELET) 2. ANTICOAGULAN

> REPERFUSION THERAPY: • 1) WITHIN LESS THAN 90 MIN OF ADMISSION: FIRST CORONARY ANGIOGRAPHY,

THEN PCI

- **(PERCUTANEOUS CORONARY INTERVENTION).**

• 2) FIBRINOLYTIC: MORE THAN 90 MIN. • 3) MORE THAN 12 HOUR: ASSESS FOR ONGOING ISCHEMIA (REFRACTORY CHEST PAIN, HEMODYNAMIC INSTABILITY, DYNAMIC ECG CHANGES, AND

VENTRICULAR ARRHYTHMIA.

- **IF THERE IS NO EVIDENCE CONTINUED MEDICAL THERAPY IF THEY DO SHOW EVIDENCE GET IMMEDIATE PCI.**

LONG-TERM MANAGEMENT FOR STABLE ANGINA

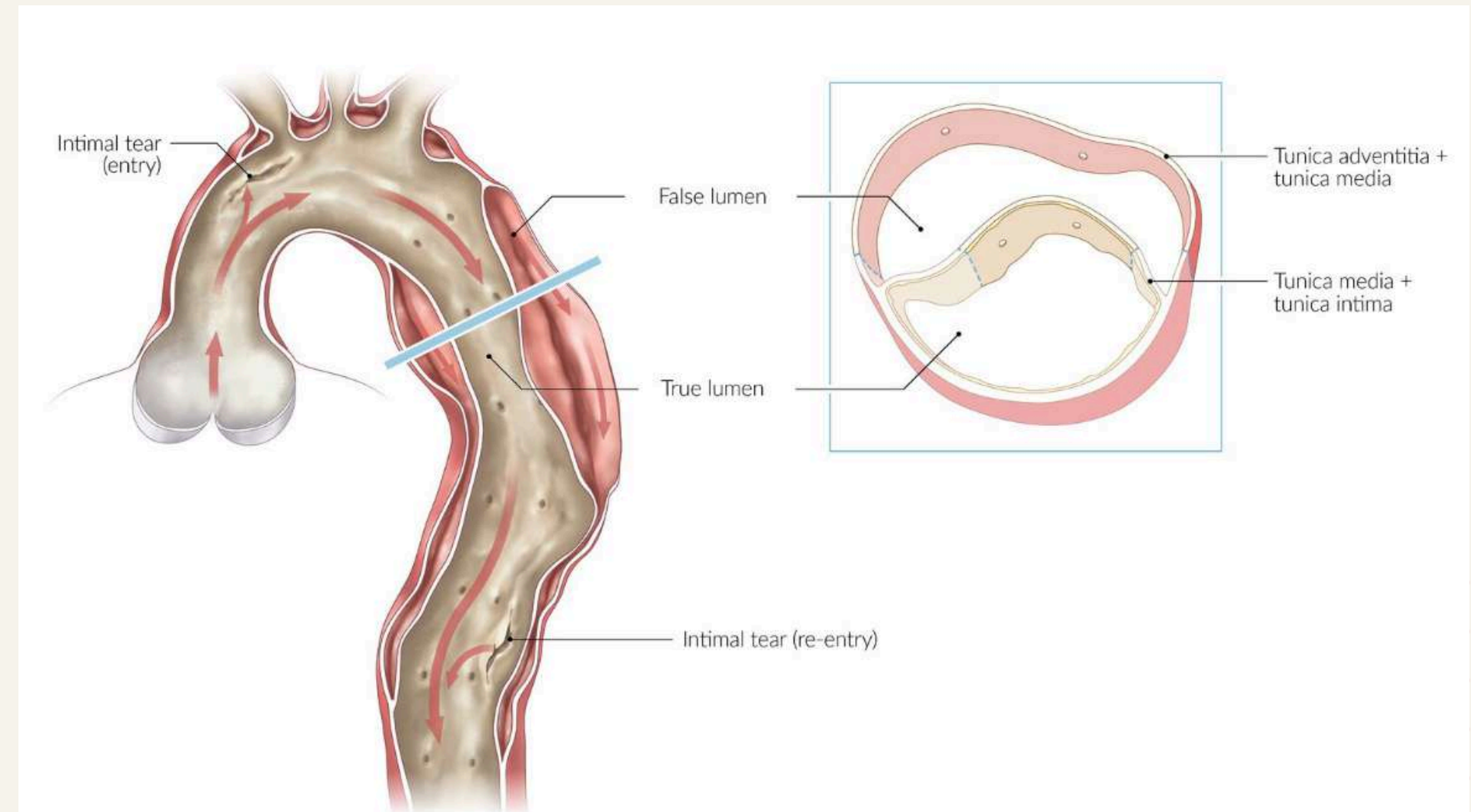
- **Lifestyle Modifications: Smoking cessation, diet, and exercise.**
- **Medications: Beta-blockers, calcium channel blockers, nitrates, and aspirin.**
- **Elective PCI or CABG: Considered if symptoms persist.**

AORTIC DISSECTION

Definition:

is a life-threatening emergency that requires prompt recognition in the Emergency Department (ED).

An aortic dissection is a tear in the inner layer of the aorta that leads to a progressively growing hematoma in the intima-media space.



ETIOLOGY

● Acquired

- Hypertension
- Trauma
- Vasculitis with aortic involvement (e.g., syphilis, Takayasu arteritis)
- Use of amphetamines and cocaine
- pregnancy
- Atherosclerosis

● Congenital

- Connective tissue disease (Marfan syndrome, Ehlers-Danlos syndrome)
- Bicuspid aortic valve (e.g., in Turner syndrome)
- Coarctation of the aorta

ACQUIRED CAUSES

HYPERTENSION

(most common risk factor)

Approx. 70% of patients with aortic dissection have elevated blood pressure, which can lead to propagation of the dissection and increases the risk of rupture.

Exception: In patients < 40 years of age, less than 40% of cases are due to hypertension.

ACQUIRED CAUSES

TRAUMA

- e.g. deceleration injury in a motor vehicle collision,
- iatrogenic injury during valve replacements , graft surgery (traumatic aortic dissection)

PREGNANCY

- Third-trimester pregnancy (or early postpartum period)



CLASSIFICATION

There are two classifications of aortic dissection to help direct management.

Stanford classification

groups dissections by whether the ascending or descending aorta is involved.

DeBakey classification

categorizes dissections according to their origin and extent.



STANFORD CLASSIFICATION

- Stanford A = Affects ascending aorta
- Stanford B = Begins beyond brachiocephalic vessels

Stanford type A

any dissection involving the ascending aorta (defined as proximal to the brachiocephalic artery), regardless of origin

- Can extend proximally to the aortic valve and distally to the descending aorta

Stanford type B

any dissection not involving the ascending aorta

- Descending aorta; originating distal to the left subclavian artery



DEBAKEY CLASSIFICATION

Type I

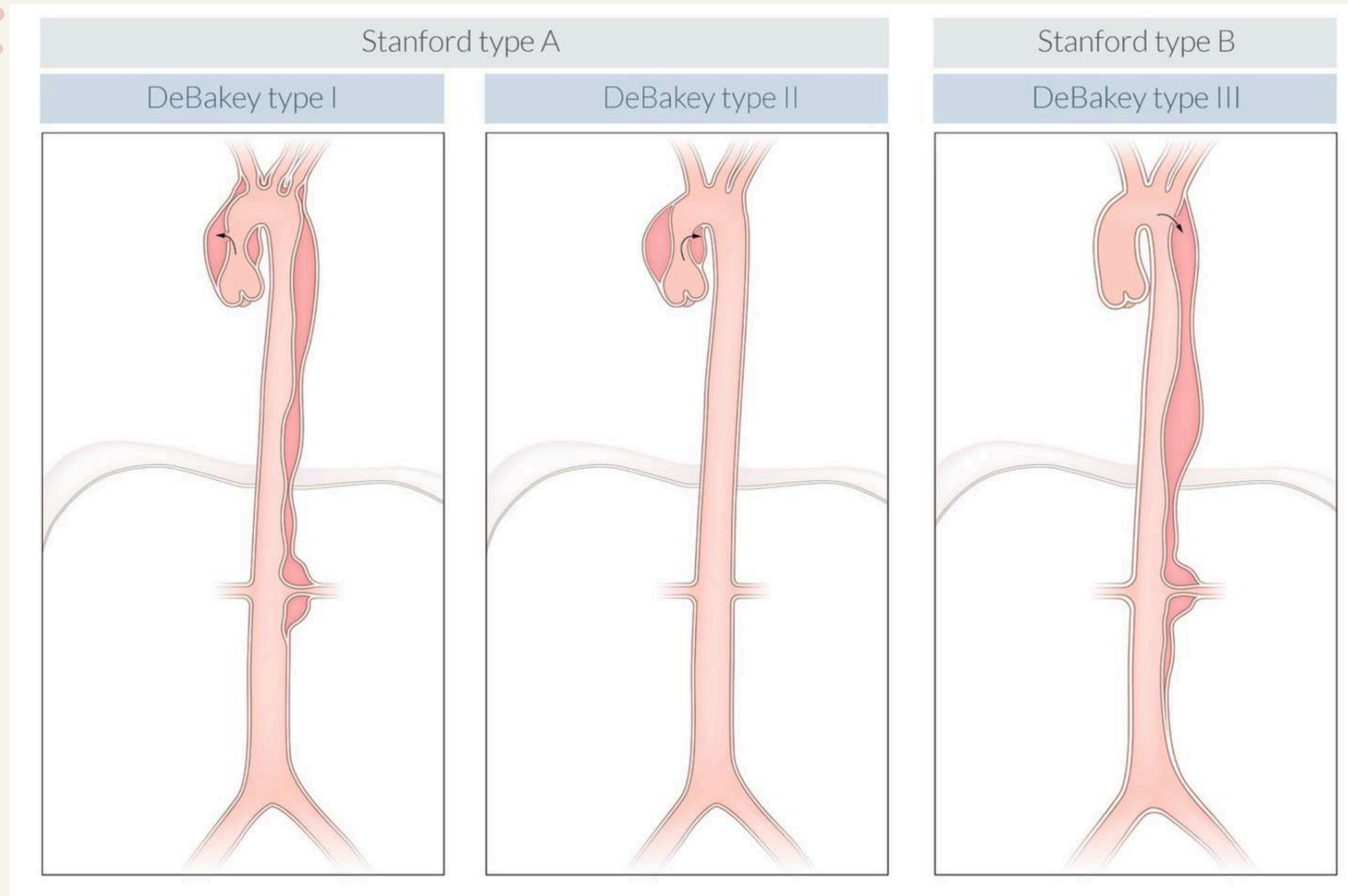
- Dissections originate in the ascending aorta and continue to at least the aortic arch but typically as far as the descending aorta.
- Generally requires surgery

Type II

- Dissections originate in, and are restricted to, the ascending aorta.
- Generally requires surgery

Type III

- Dissections originate in the descending aorta and most often extend distally.
- Most cases can be managed by medical therapy.
- Can be further subdivided into:
 - Type IIIa: limited to the descending thoracic aorta above the level of the diaphragm
 - Type IIIb: extends below the diaphragm



CLINICAL FEATURES

1

Sudden and severe tearing/ripping pain

- Location
 - Anterior chest (ascending) or back (descending)
 - Interscapular or retrosternal pain
 - Neck and jaw
 - Abdomen or periumbilical, colicky pain
- Character: migrates as the dissected wall propagates caudally

2

Hypertension or hypotension

3

Asymmetrical blood pressure and pulse readings between limbs

4

Wide pulse pressure

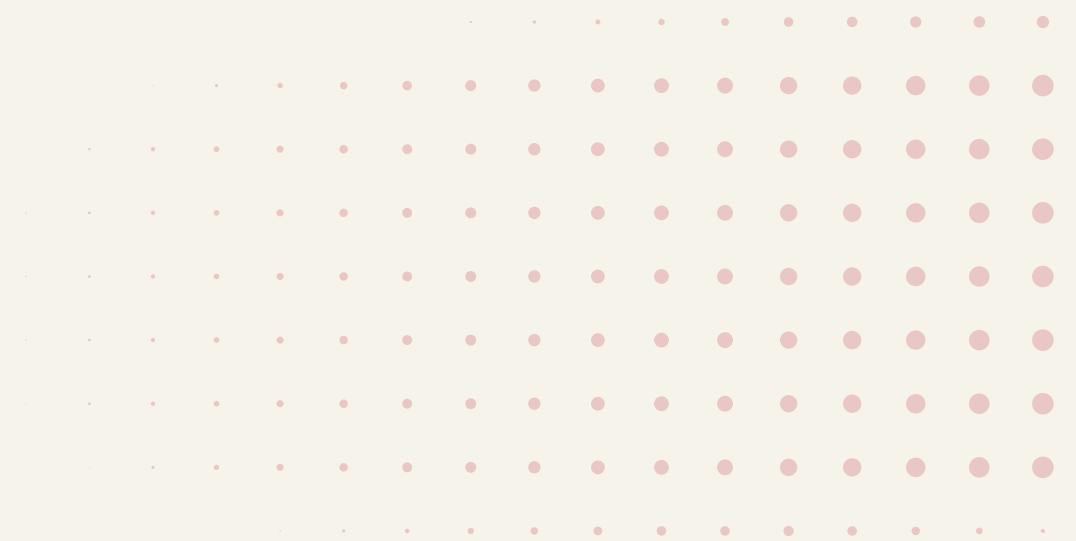
5

Syncope, diaphoresis, confusion, or agitation



CASE STUDY

A 65-year-old man with hypertension and type 2 diabetes mellitus is brought to the emergency department 20 minutes after the onset of severe anterior chest pain and shortness of breath. He has smoked one pack of cigarettes daily for 30 years. He appears distressed. His pulse is 116/min, respirations are 22/min, and blood pressure is 156/88 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 98%. A grade 3/6, high-pitched, blowing, diastolic murmur is heard over the right upper sternal border. Which of the following is the most likely cause of this patient's symptoms?

1. Tear in the tunica intima
 2. fibrofatty plaque in the aortic wall
 3. obstruction of the pulmonary arteries
 4. rupture of bulla in the lung
 5. perforation of the esophageal wall
 6. infarction of the ventricular wall
- 

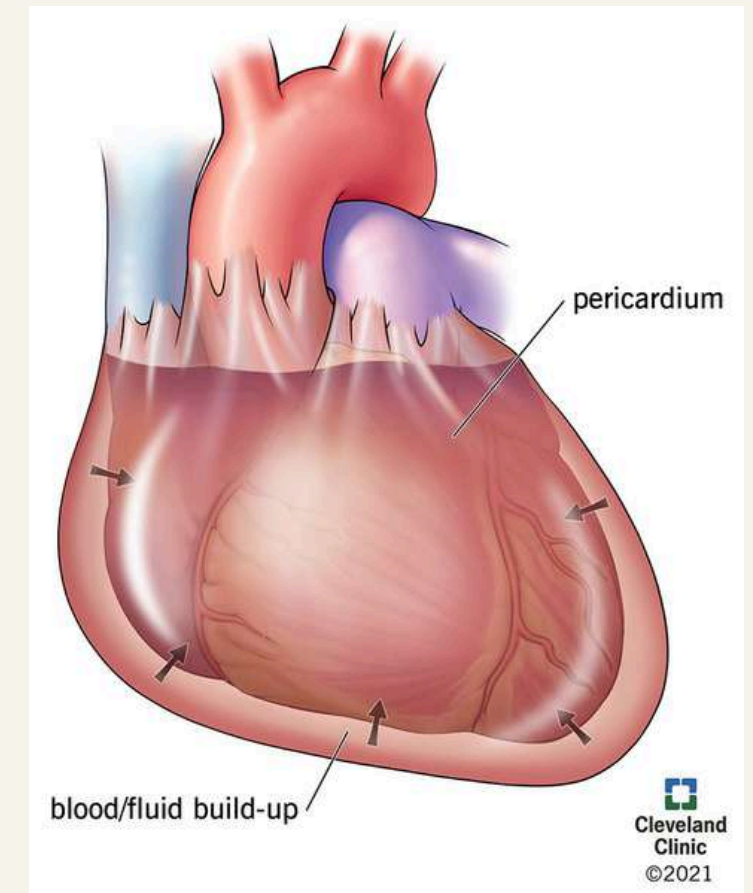
COMPLICATIONS

1 Malperfusion syndrome

2 Aortic rupture and acute blood loss (shock & death)

3 Complications of Stanford A

- MI
- Aortic regurgitation
- Cardiac tamponade
- Stroke

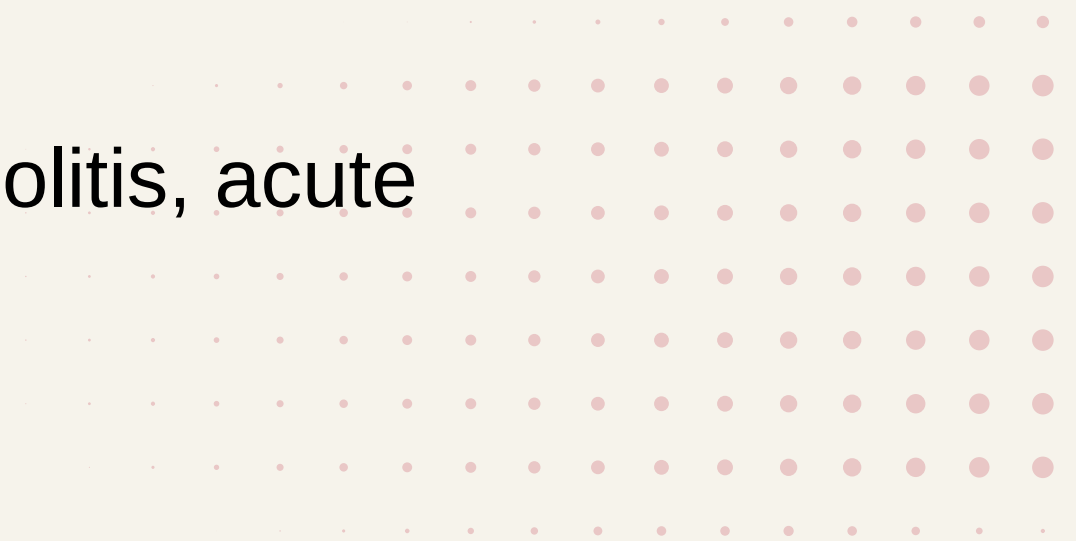




COMPLICATIONS

Malperfusion syndrome :

A compromise of blood flow to major vascular beds resulting in ischemia and end organ damage

- Renal arteries : acute renal failure
 - Celiac trunk , superior & inferior mesenteric : ischemic colitis, acute abdomen
 - Spinal arteries: acute paraplegia
- 



DIAGNOSIS

- **Clinical assessment**
- **Physical Examination**
- **Laboratory Assessment**
- **Imaging**



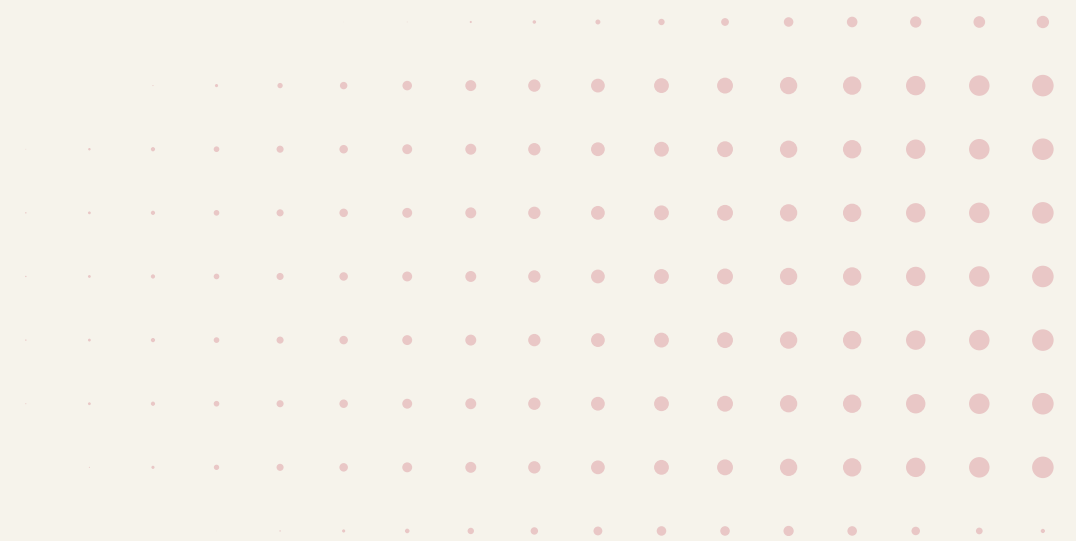
DIAGNOSIS

Clinical assessment: evaluation of symptoms and risk factors

PE findings: (depend on location and the progression of dissection)

- diastolic murmur if aortic insufficiency develops
- hypertension and tachycardia, hypotension may also be present
- decreased or asymmetrical pulsation in the peripheral arteries

Lab: D-dimer



DIAGNOSIS

Imaging modalities :

- CT angiography : the modality of choice, careful in renal insufficiency
- TEE and MRA : initial study for high risk patients, confirmatory if uncertain
- CXR : abnormal in 80% , may show : abnormal aortic contour, widening of mediastinum , deviation of trachea or esophagus , left pleural effusion, aortic calcification, blurring of aortic knuckle .
- ECG should be ordered for all patients to exclude other causes like MI

Note: Myocardial infarction can occur in combination with aortic dissection, and treatment of the myocardial infarction (e.g., thrombolysis) can cause fatal progression of the aortic dissection.



CT chest (with contrast; oblique sagittal reconstruction)

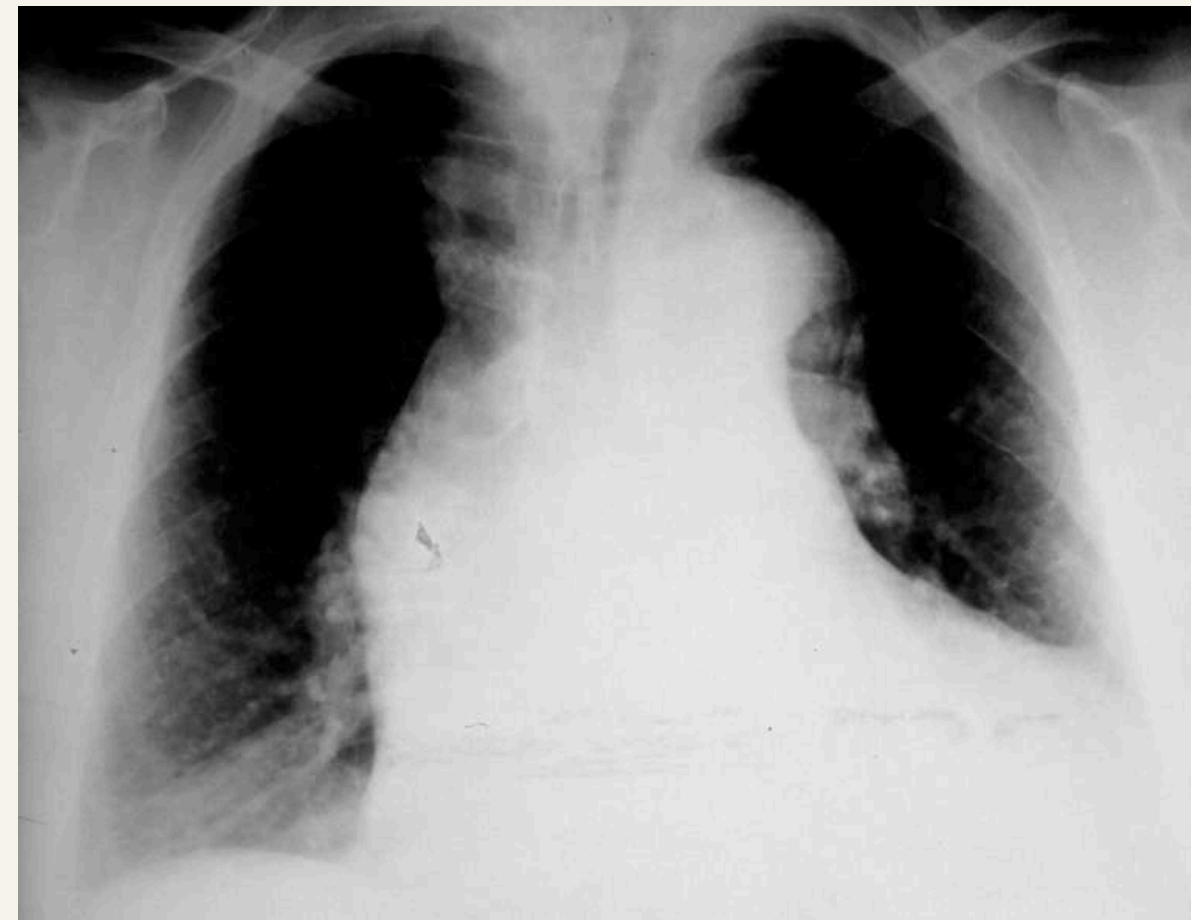
A complex Stanford type A dissection is seen, which involves the ascending aorta, arch, and descending aorta.

1: descending aorta; 2: left subclavian artery; 3: ascending aorta; red arrowheads: dissection flap

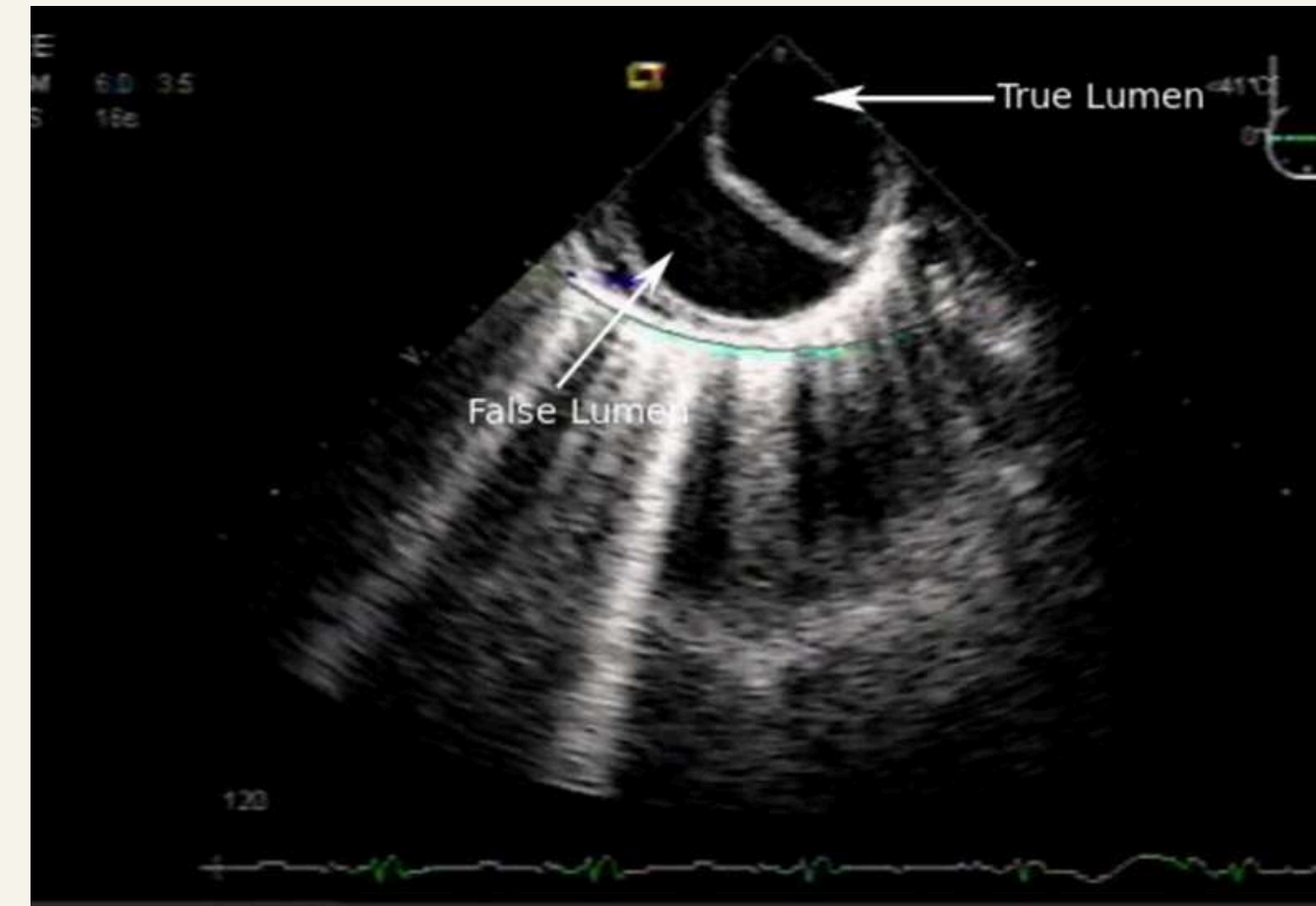
IMAGING



CT thorax (with contrast; axial view)
A mucosal flap (green line) can be seen dividing the lumen of the ascending (A) and descending (D) aorta.
This radiological appearance confirms a Stanford type A aortic dissection.



X-ray chest
Widening of the mediastinum is accompanied by a prominent aortic knob on the left side



Transesophageal echocardiography of aortic dissection
Echocardiography (transesophageal; transverse view)
The descending thoracic aorta shows a crescentic false lumen and rounded true lumen.



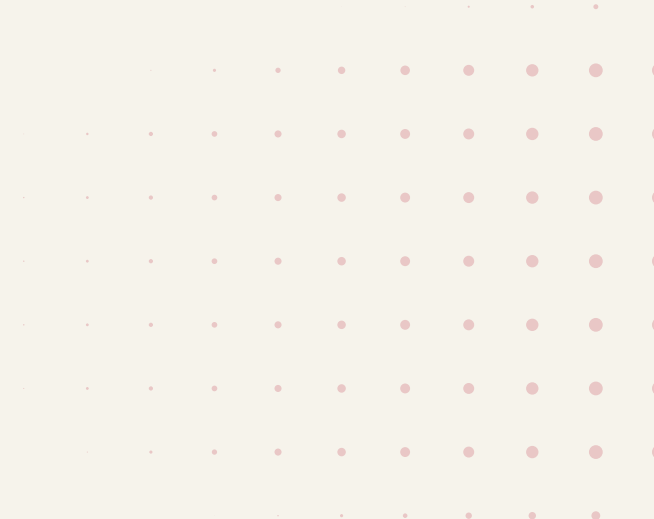
MANEAGEMENT

- Hemodynamically unstable
- surgical intervention
- medical intervention
- supportive care



MANEAGEMENT

Hemodynamically unstable :

1. Resuscitate with ACLS (ABCDE approach)
 2. Perform simultaneous bedside investigation
 3. treat life threatening complications (hemodynamic support)
 4. Expedite surgical management
- 

MANEAGEMENT

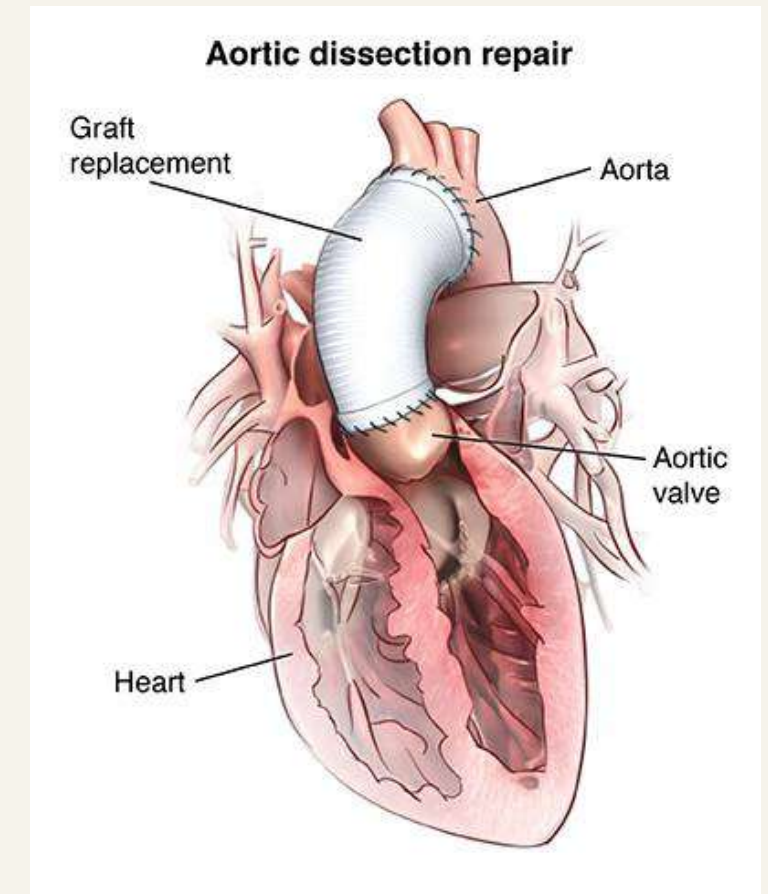
Surgical

- **All Stanford A**
- **Stanford B with complications**

Open surgery (midline sternotomy) to remove the section of the aorta with the defect in the wall and replace it with a synthetic graft. The aortic valve may need to be replaced during the procedure.

- **Stanford B**

Endovascular treatment with aortic stent implantation



MANEGEMENT

Medical

- **Hypotensive pt**

IV fluids

Vasopressor : norepinephrine, phynylephrine

- **Hypertensive pt**

IV beta blocker: esmolol , labetalol or CCB if

contraindicated : verapamil or diltiazim

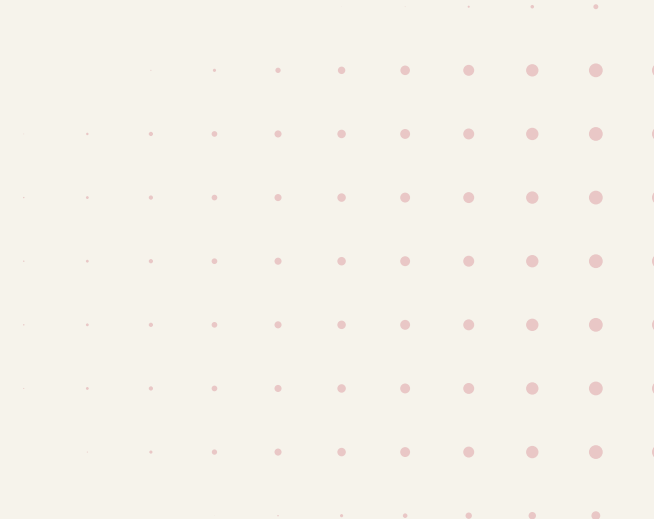
IV vasodilator : sodium nitroprusside

Patients who remain stable on IV treatment can be transitioned to oral medications and discharged with outpatient imaging surveillance (Stanford B)



MANEAGEMENT

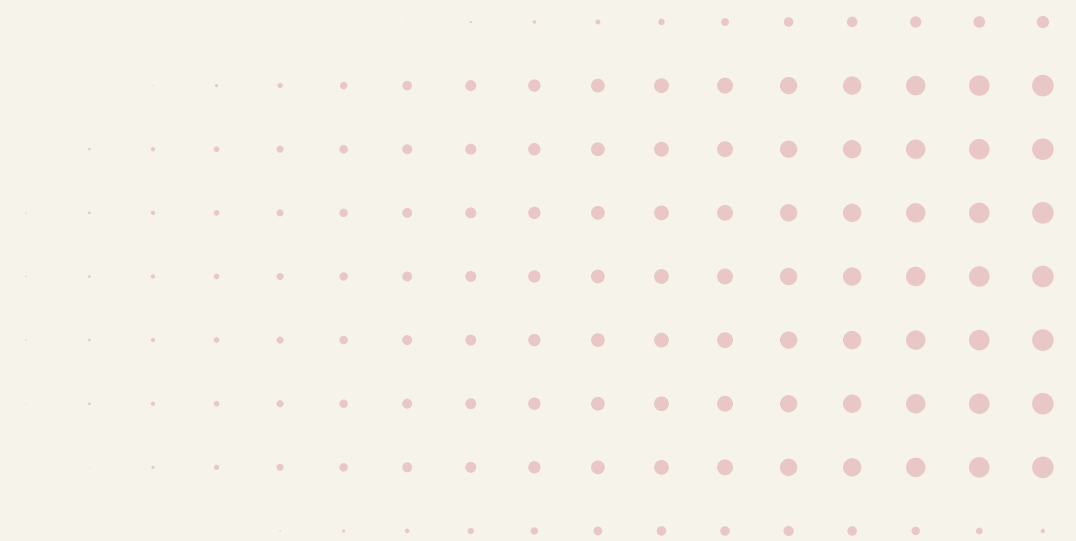
Supportive care: (Management of pain and anxiety which lowers sympathetic tone)

1. Pain management (morphine)
 2. Procedural sedation
 3. Anticoagulant reversal
- 



CASE STUDY

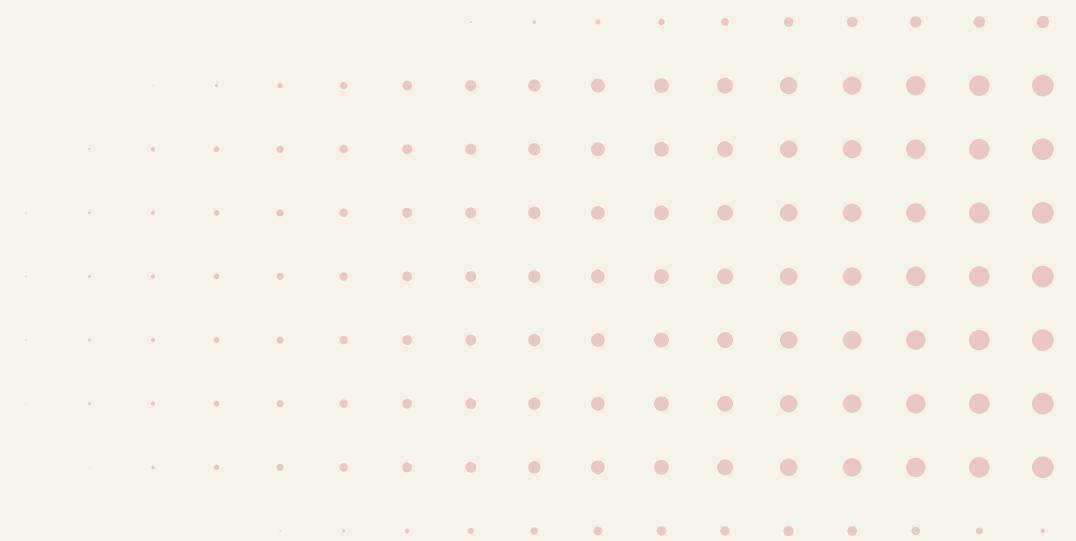
A 31-year-old man comes to the emergency department for acute tearing chest pain that radiates to the back. Despite appropriate therapy, the patient dies. Autopsy shows an increase in mucoid extracellular matrix and loss of smooth muscle cell nuclei in the media of large arteries. Which of the following additional findings is most likely in this patient?

1. nasal septum perforation
 2. inferonasal lense dislocation
 3. polycystic kidneys
 4. pectus carniatum
 5. condylomata lata
- 



CASE STUDY

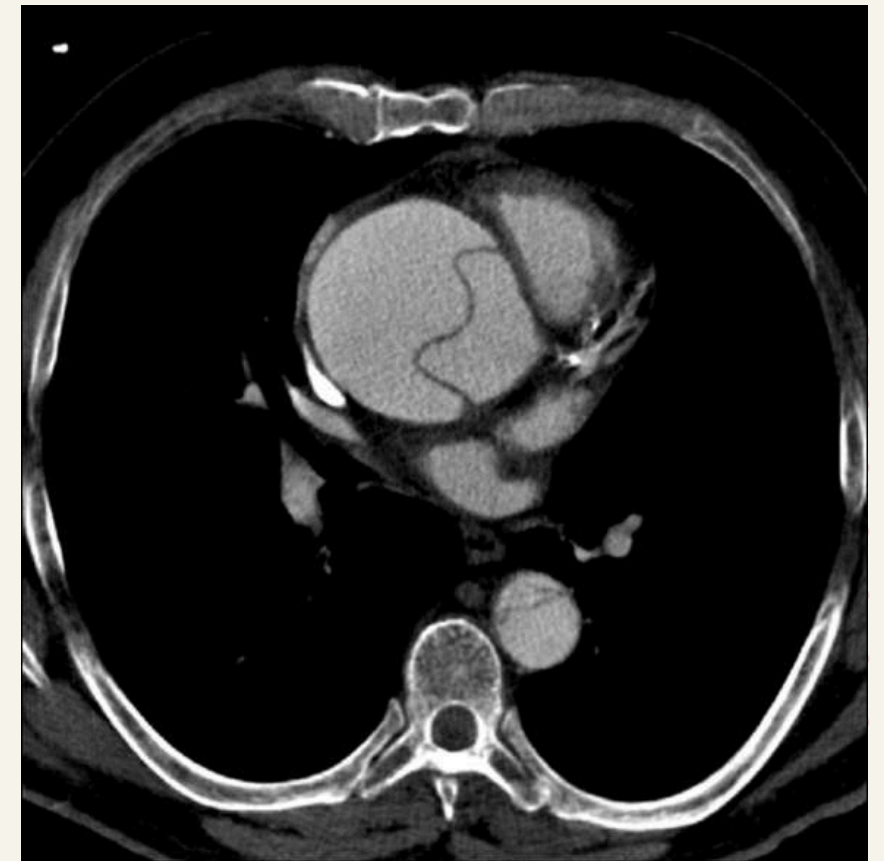
A 55-year-old man is brought to the emergency department 30 minutes after the sudden onset of severe, migrating anterior chest pain, shortness of breath, and sweating at rest. He has hypertension, hypercholesterolemia, and type 2 diabetes mellitus. Medications include atorvastatin, hydrochlorothiazide, lisinopril, and metformin. He has smoked one pack of cigarettes daily for 25 years. He is in acute distress. His pulse is 110/min, respirations are 20/min, and blood pressure is 150/85 mm Hg. Pulse oximetry on room air shows an oxygen saturation of 98%. Cardiac examination shows a grade 3/6, high-pitched, blowing, diastolic murmur heard best over the right sternal border. The lungs are clear to auscultation. Femoral pulses are decreased bilaterally. An ECG shows sinus tachycardia and left ventricular hypertrophy. Which of the following is the most likely diagnosis?



CASE STUDY

A 58-year-old man is brought to the emergency department by his wife 30 minutes after the sudden onset of severe retrosternal chest pain radiating to his back. He has a history of hyperlipidemia, hypertension, and type 2 diabetes mellitus. He has smoked one-half pack of cigarettes daily for 20 years. Medications include aspirin, captopril, atorvastatin, and metformin. His pulse is 80/min and blood pressure is 160/60 mm Hg. A CT scan of the chest is shown. Which of the following is the strongest predisposing factor for this patient's current condition?

1. Hyperlipidemia
2. DM
3. age
4. genetic collagen disorder
5. hypertension
6. history of smoking



Deep Vein Thrombosis (DVT)

Introduction

DVT is a blood clot that develops in the deep veins most commonly forming in the lower limbs, and less commonly DVT can form in the deep veins of the arms (upper limbs).

Arterial clots are usually due to artery wall damage, while venous clots don't require vein damage.



Deep Vein Thrombosis (DVT)

Causes and Risk Factors

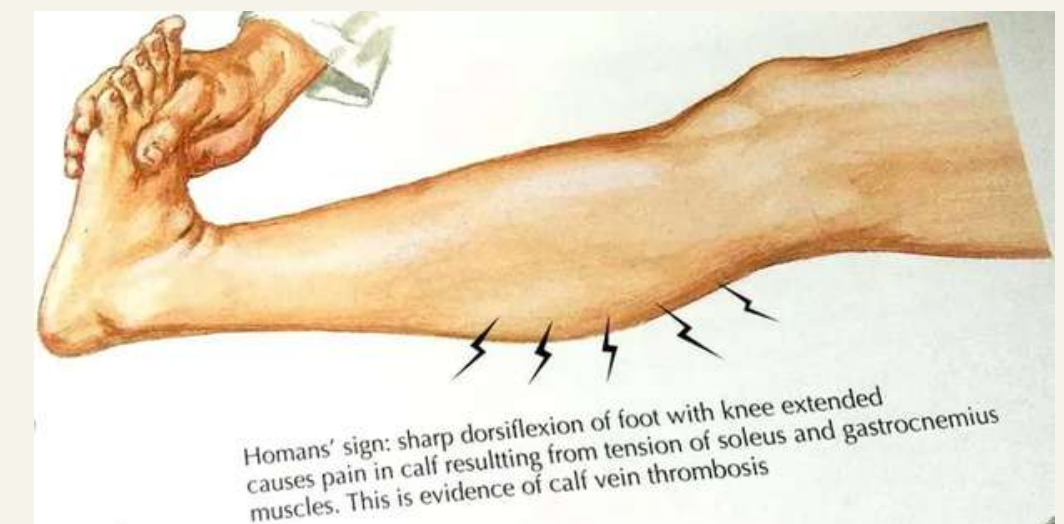
1. Immobility
2. Injury
3. Surgery
4. Medical Conditions
5. Age
6. Obesity
7. Smoking
8. Previous DVT or a family history of DVT



Deep Vein Thrombosis (DVT)

Signs and Symptoms

1. **Swelling**: Unilateral, may occur in the foot or ankle
2. **Pain**: Described as cramping or soreness, typically starting in the calf or posterior thigh
3. **Redness and Warmth**
4. **Enlarged Veins**: Surface veins may appear more prominent
5. **Palpable Cord**: Firm, palpable cord along the course of the deep vein
6. **Homan's Sign**: Calf pain with dorsiflexion of the foot (not a highly reliable sign)



Deep Vein Thrombosis (DVT)

Diagnosis: Wells Criteria

TABLE 25-3 Wells Score for Deep Vein Thrombosis (DVT)	
Clinical Feature	Points*
Active cancer (treatment within 6 months, or palliation)	1
Paralysis, paresis, or immobilization of lower extremity	1
Bedridden for >3 days because of surgery (within 4 weeks)	1
Localized tenderness along distribution of deep veins	1
Entire leg swollen	1
Unilateral calf swelling of >3 cm (below tibial tuberosity)	1
Unilateral pitting edema (greater in symptomatic leg)	1
Collateral superficial veins (nonvaricose)	1
Alternative diagnosis as likely as or more likely than DVT	-2

Risk Score interpretation:

- **≥ 3 points: high risk (75%)**
- **1 or 2 points: moderate risk (17%)**
- **<1 point: low risk (3%)**

Deep Vein Thrombosis (DVT)

Diagnosis: D-Dimer

- Blood test measuring the presence of a substance released when a blood clot breaks down.
- Elevated levels may suggest a thrombus but it's not specific to location or type, and it's not a definitive indicator.
- D-dimer test is mostly useful when negative as its negative predictive value is >99%.

Deep Vein Thrombosis (DVT)

Imaging Modalities

Doppler Ultrasound:

- assess blood flow through vessels
 - abnormal blood flow is seen when a clot is present
 - reduced flow in the clot
 - absent flow past the clot in some cases (severe)
 - turbulent flow around the clot
- modified vein diameter
 - acute thrombus = increased vein diameter (in comparison to adjacent artery)
 - chronic thrombus = decreased vein diameter (vein becomes more rigid with irregular surface)

Compression Ultrasound:

- mainstay for DVT diagnosis
- assess vein compressibility (non-compressibility indicates a clot)
- often used in conjunction with Doppler ultrasound

Deep Vein Thrombosis (DVT)

Treatment of Suspected/Confirmed DVT in ER

Anticoagulation

- Low Molecular Weight Heparin (LMWH) or Fondaparinux = indicated for immediate anticoagulation
- Warfarin or Direct Oral Anticoagulants (DOC) = for long term anticoagulation

Thrombolytics

- Role: for massive/high-risk PE with hemodynamic instability
- Indication: for patients with significant compromise
- Risk: increased risk for bleeding, so careful consideration is needed

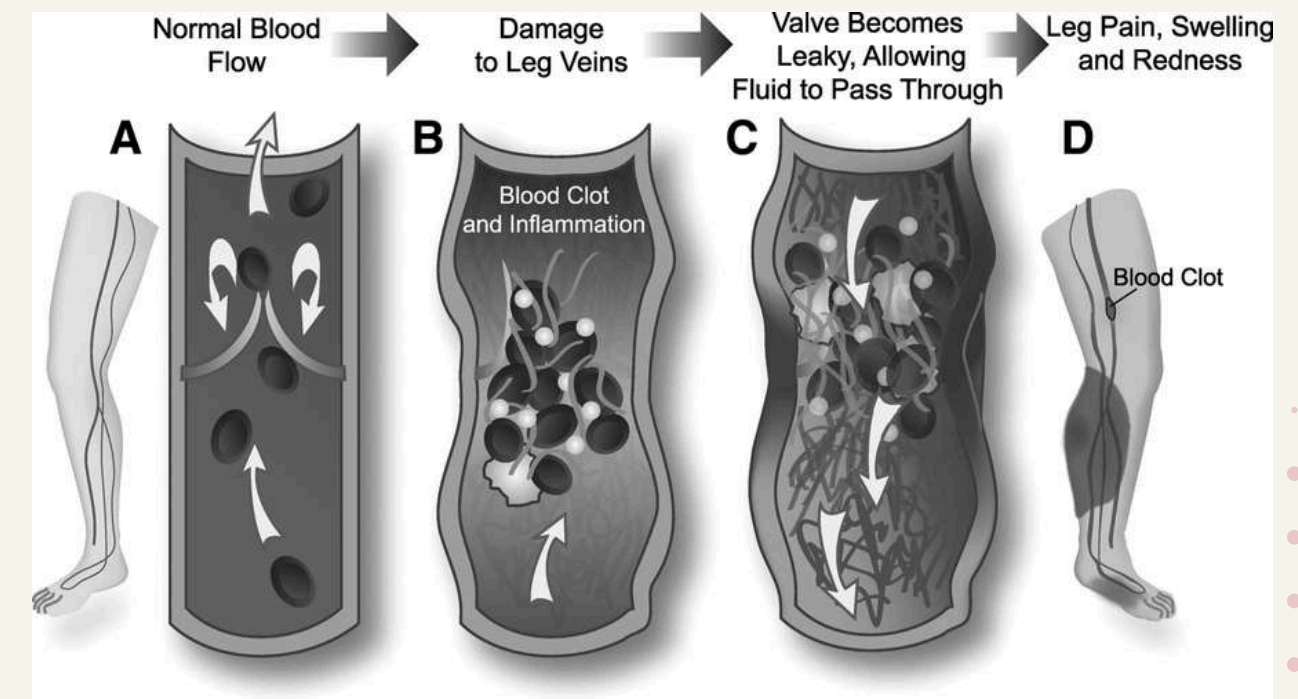
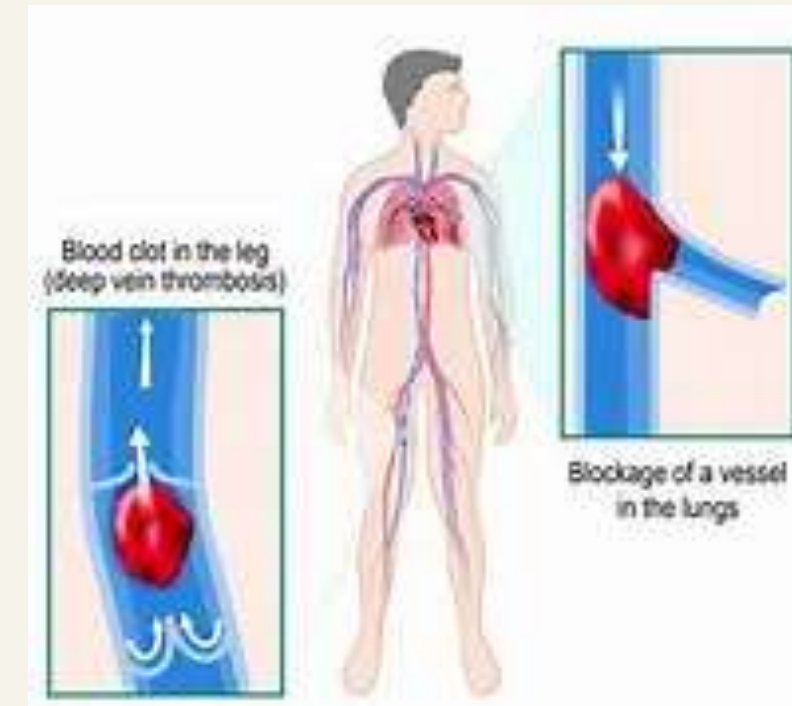
It's important to tailor the management to the individual patient, considering their presentation, comorbidities, and risk factors.

Consultation with specialists and follow-up for ongoing anticoagulation is typically warranted.

Deep Vein Thrombosis (DVT)

Complications

- **Pulmonary embolism (PE):** clot travels to lungs, blocking the flow
- **Post-Thrombotic Syndrome (PTS):** long-term pain, swelling, and ulcers in the affected limb due to vein damage
- **Chronic venous Insufficiency:** due to degeneration of valves



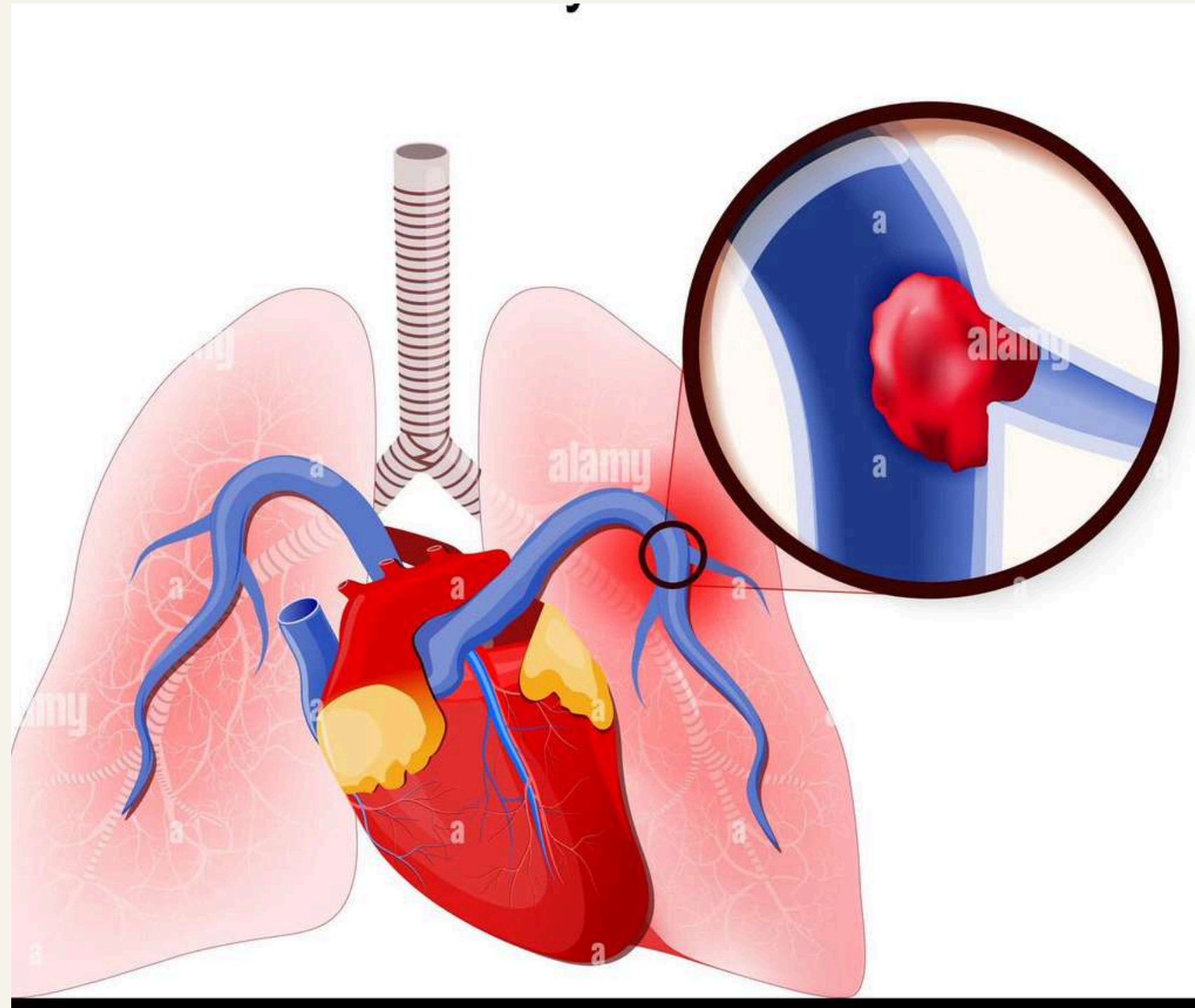
Deep Vein Thrombosis (DVT)

Sample Question

Three days after undergoing laparoscopic colectomy, a 67-year-old man reports swelling and pain in his right leg. He was diagnosed with colon cancer one month ago. His temperature is 38.5°C. Physical examination shows swelling of the right leg from the ankle to the thigh. There is no erythema or rash. Which of the following is likely to be most helpful in establishing the diagnosis?

1. D-dimer
2. Transthoracic Echocardiography
3. CT Pulmonary Angiography
4. Blood Cultures
5. Compression Ultrasonography

PULMONARY EMBOLISM



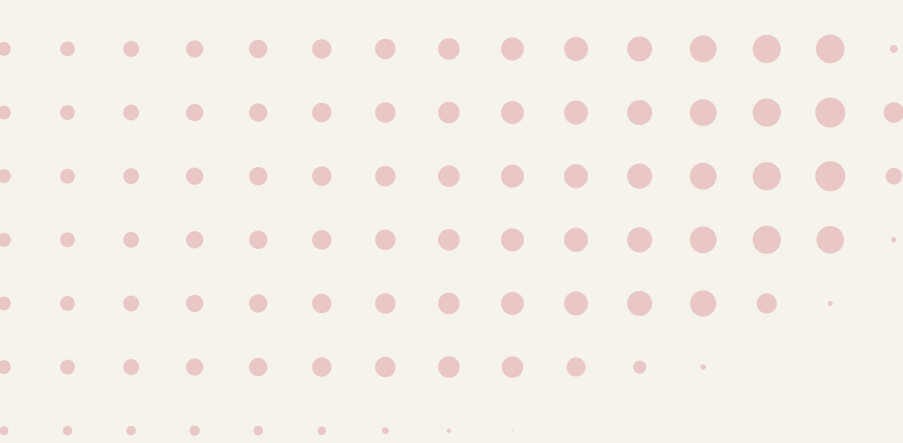


PULMONARY EMBOLISM

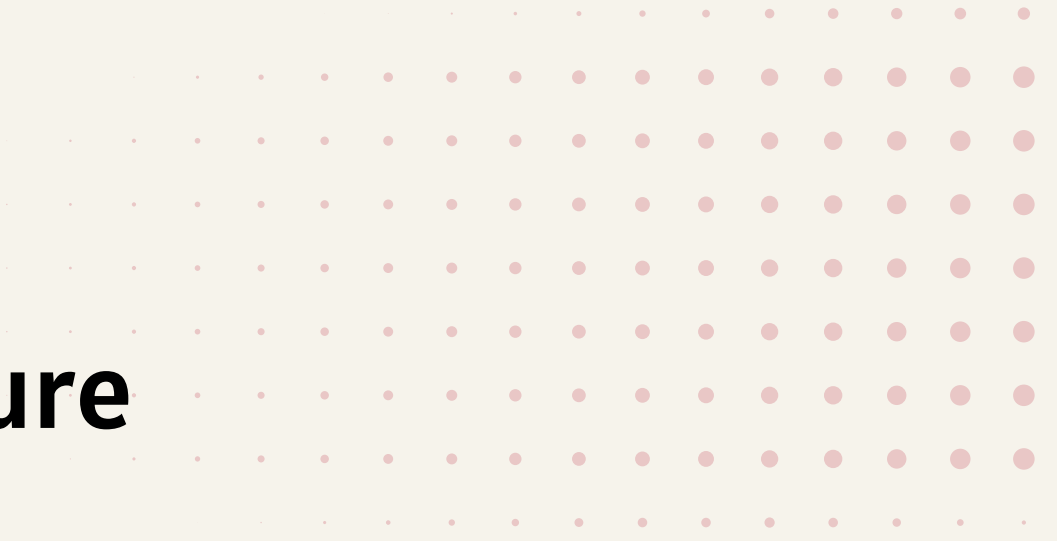
PE occurs when a portion of a clot breaks off and travels to a pulmonary artery, blocking it, decreasing the blood flow to the lungs.

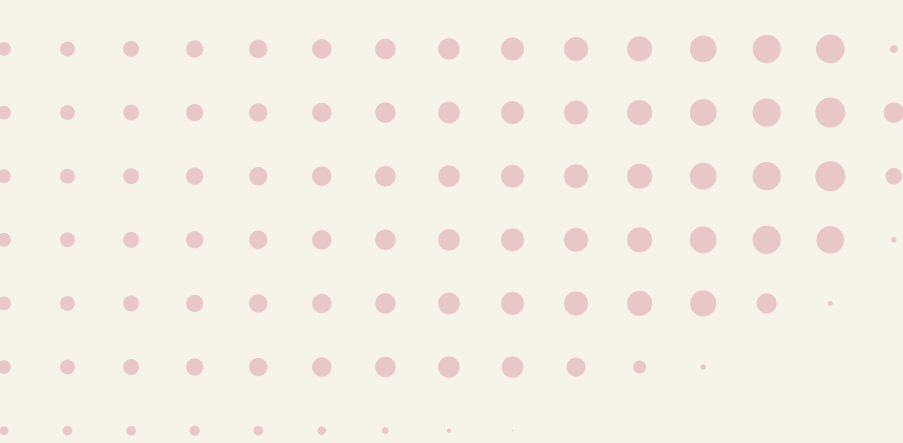
Risk Factors

- major surgery or trauma or prolonged hospitalization
- permanent immobilization and age
- malignancy, other hypercoagulable state
- prior venous thromboembolism

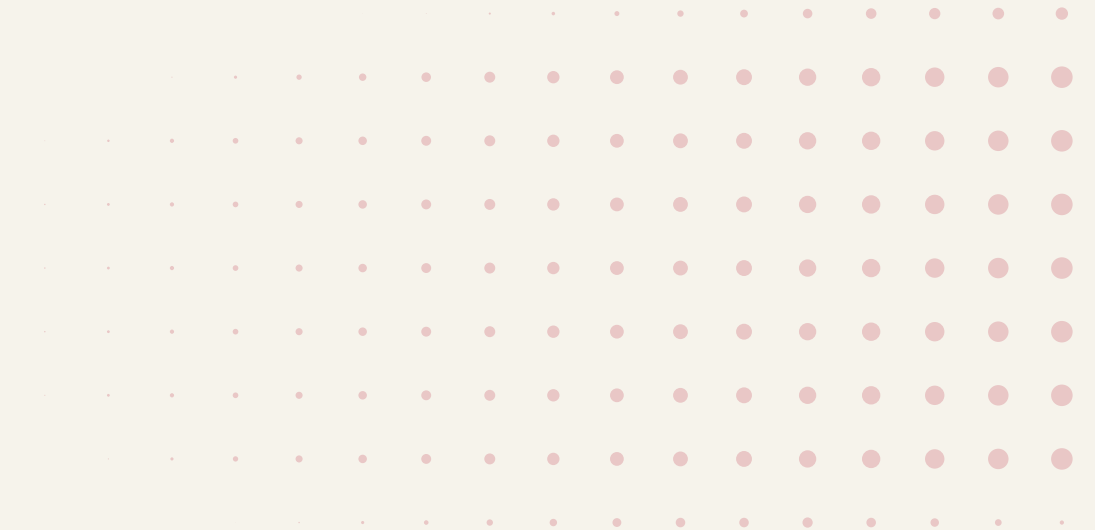


SYMPTOMS

- **Dyspnea**
 - **Pleuritic or non pleuritic chest pain**
 - **Hemoptysis**
 - **Excessive sweating**
 - **Cough**
 - **Syncope**
 - **Can present as confusion or even seizure**
- 



SIGNS

- **Hypoxemia**
 - **Cyanosis**
 - **Tachypnea**
 - **Tachycardia**
 - **Low-grade fever**
- 

WELLS SCORE FOR PULMONARY EMBOLISM

Objective Criteria	Points	Subjective Criteria	Points
Heart rate > 100 beats/min	1.5	Clinician considers alternative diagnoses to be less likely than PE	3
Hemoptysis	1		
History of venous thromboembolism	1.5		
Malignancy (active)	1		
Recent immobility or Surgery	1.5		
Leg swelling, pain with palpation of deep veins (clinical signs of DVT)	3		

Low probability = 0-2 ; Intermediate probability = 2-6 ; High probability = >6

D-DIMER

For patients with low or intermediate pretest probability , D-dimer testing is the recommended first test.

D-dimer is only useful when negative

D-dimer assay = <500 ng/ml PE excluded

D-dimer assay = >500 ng/ml proceed to further investigation with CT pulmonary angiogram

IMAGING MODALITIES OF PE

- CT angiography has high sensitivity and specificity for PE.
- V/Q scan useful when CT angiography not available, or patient unable to tolerate IV contrast (eg, contrast allergy).
- Chest radiographs frequently demonstrate cardiomegaly or atelectasis, although are not specific for PE.
- Echocardiogram: Right heart strain, enlarged atrium/ventricle, abnormal septum movement.



THE EMERGENCY TREATMENT

Anticoagulation:

Heparin (UFH or LMWH) is used, with dosing adjusted by weight and patient characteristics. UFH is preferred for those with severe renal issues or bleeding risk, while LMWH is generally more effective and economical for PE/DVT.

For patients with heparin allergies, use alternatives like fondaparinux or lepirudin, contraindicated in severe renal insufficiency.

Warfarin can be initiated alongside heparin, targeting an INR of 2-3.

Thrombolytic Therapy (alteplase):

Used selectively, primarily for massive PE (e.g., hypotension and cardiac instability), with agents like tPA. Thrombolysis may improve outcomes in submassive PE cases with certain signs but is unnecessary for smaller PEs.

QUESTION

A 56-year-old woman with a history of ovarian cancer presents to the ED with acute onset of right-sided chest pain, shortness of breath, and dyspnea. Her BP is 131/75 mm Hg, HR is 101 beats per minute, respirations are 18 breaths per minute, and oxygen saturation is 97% on room air. You suspect this patient has a pulmonary embolism (PE). Which of the following tests is most likely to be abnormal?

- a. Arterial blood gas
- b. Oxygen saturation
- c. ECG
- d. Chest radiograph
- e. D-dimer

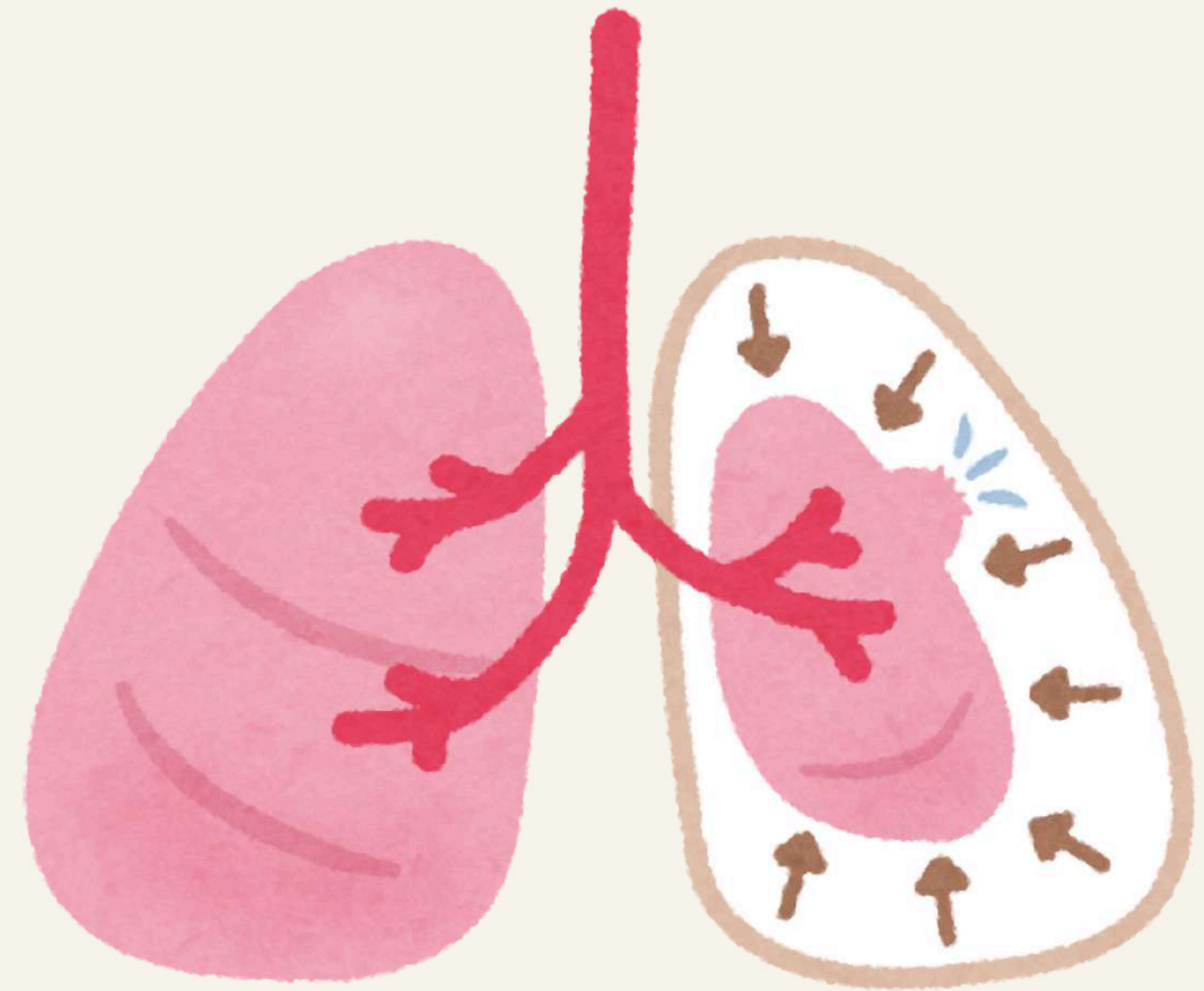
QUESTION

Which imaging study is commonly used to diagnose PE in patients who cannot undergo CT?

- A) Duplex ultrasonography
- B) Chest X-ray
- C) Ventilation-perfusion (V/Q) scan
- D) MRI

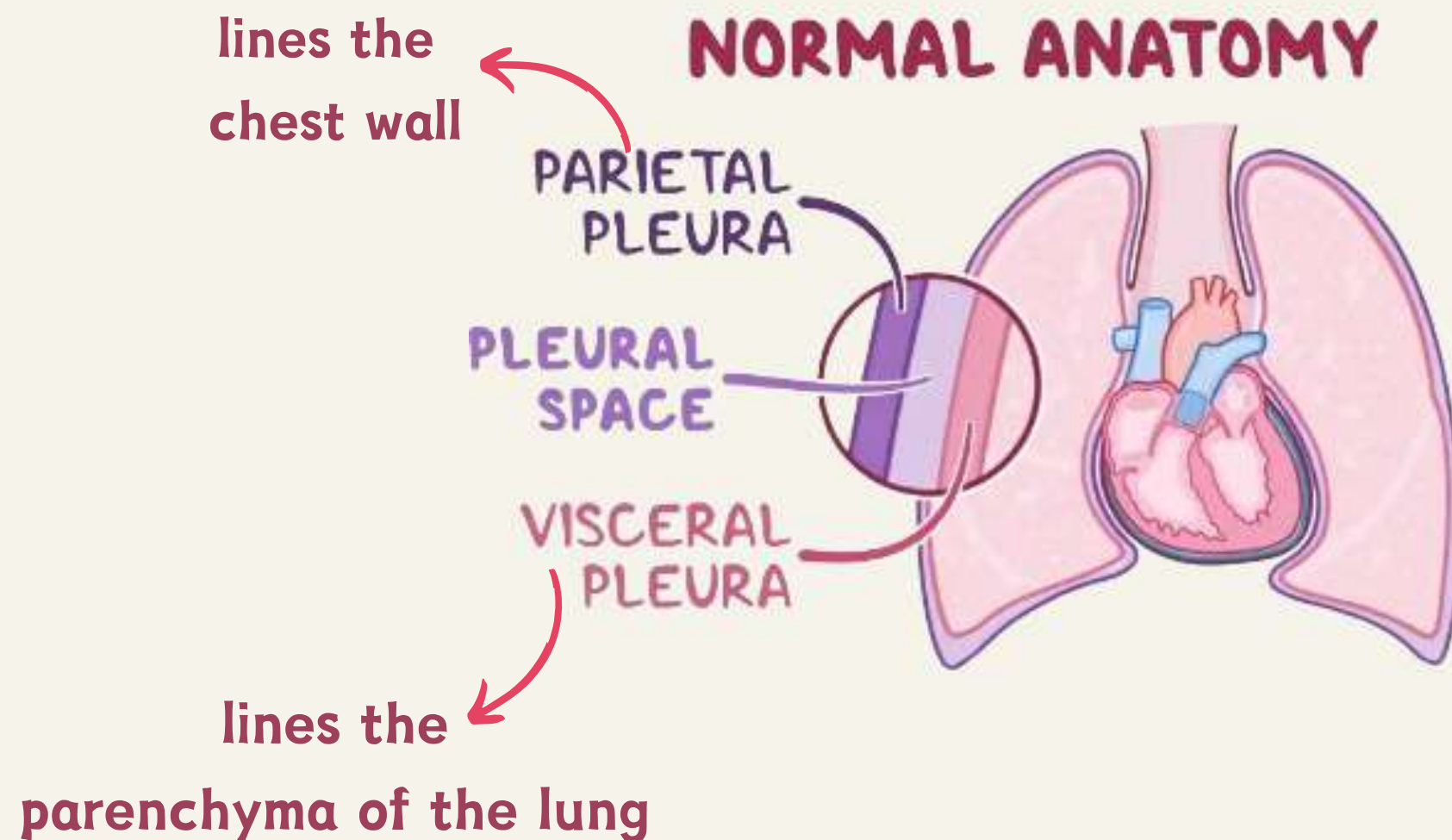
PNEUMOTHORAX

- Definition
- Types
- Tension Pneumothorax
- Radiographic Features

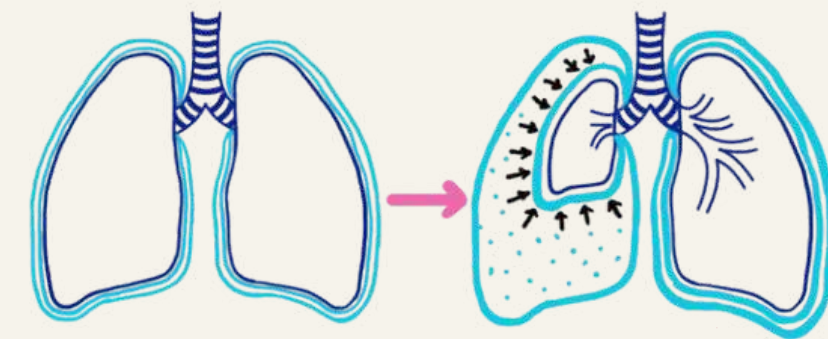
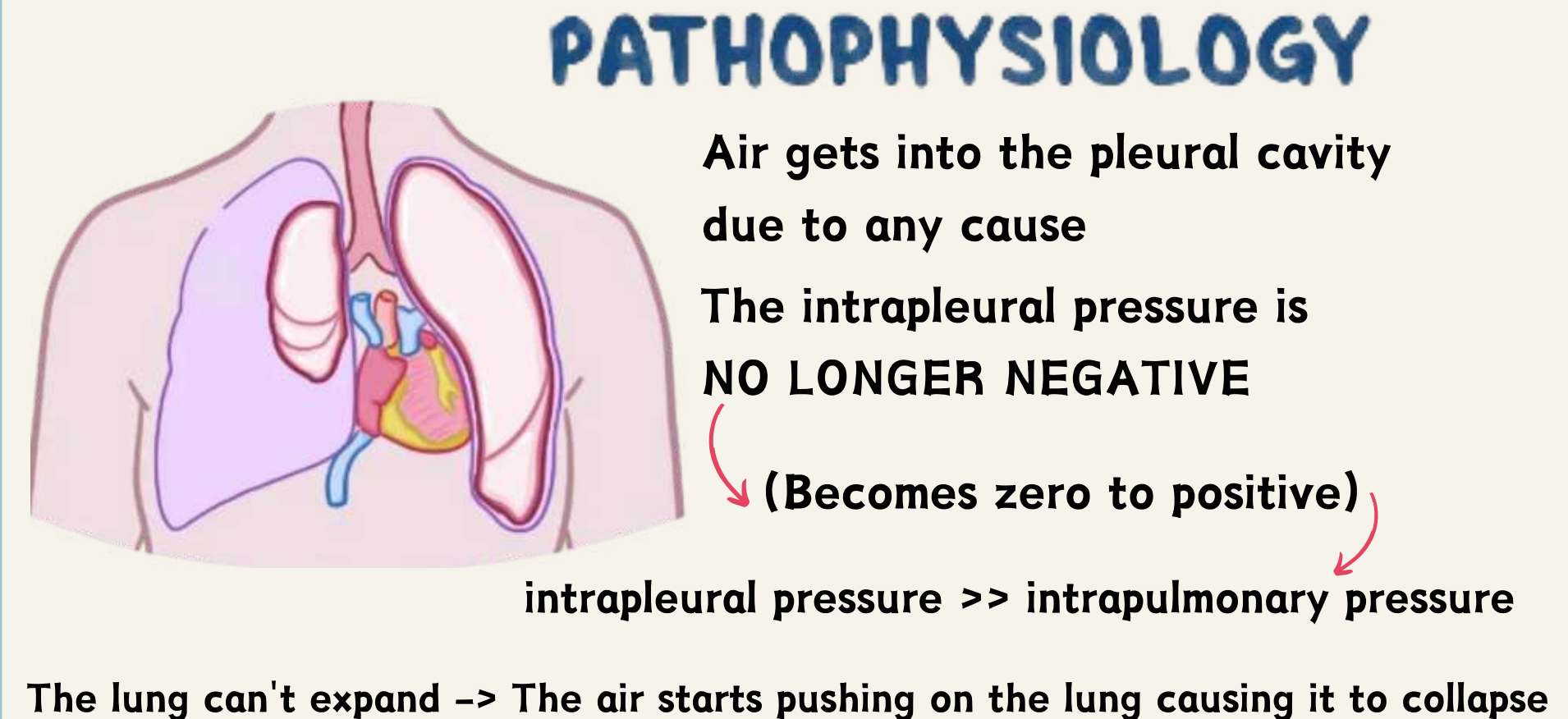


DEFINITION

An abnormal presence of air within the **pleural space** that can result in a deflated or collapsed lung



Disruption of either of the pleural layers can allow air to enter the pleural space



TYPES OF PNEUMOTHORAX

Pneumothorax

The air leaks through a defect
in the lung tissue

Spontaneous

Primary

Secondary

Non-Spontaneous

Blunt

Penetrating

Traumatic

Iatrogenic

TYPES OF PNEUMOTHORAX

Spontaneous Pneumothorax most likely results from subpleural bulla rupture

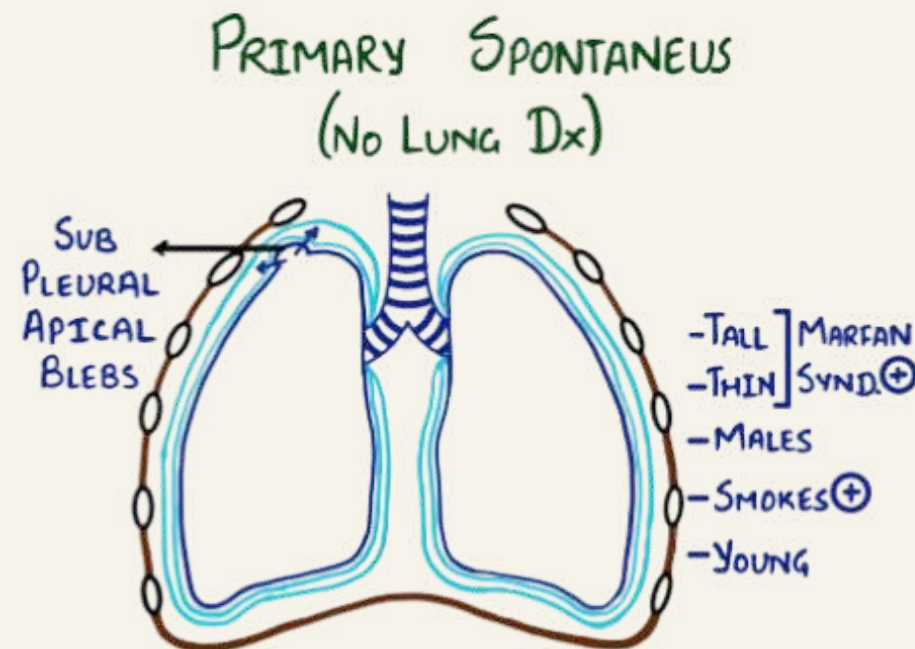
Smoking is the most common risk factor for spontaneous pneumothorax

Primary Spontaneous Pneumothorax

The patient **does not** have lung diseases

Factors related to the condition

- o Tall
 - o Thin
 - o Young
 - o Males
 - o Smokers
- Marfan Syndrome



Secondary Spontaneous Pneumothorax

The patient **has** an underlying lung disease

Causes hyperinflation or air trapping of the lung

Occurs **most often in patients with** chronic obstructive pulmonary disease (**COPD**), but other underlying diseases such as asthma, cystic fibrosis, interstitial lung disease, cancer, and Pneumocystis carinii pneumonia have been implicated

TYPES OF PNEUMOTHORAX

Iatrogenic Pneumothorax:

Occurs secondary to invasive procedures such as:

- Needle biopsy of the lung
 - Placement of a subclavian line
 - Nasogastric tube placement
 - Positive pressure ventilation
-
- Transthoracic needle procedures account for 50% of cases
 - Subclavian vein catheterization accounts for an additional 25%

A postprocedure chest x-ray is indicated

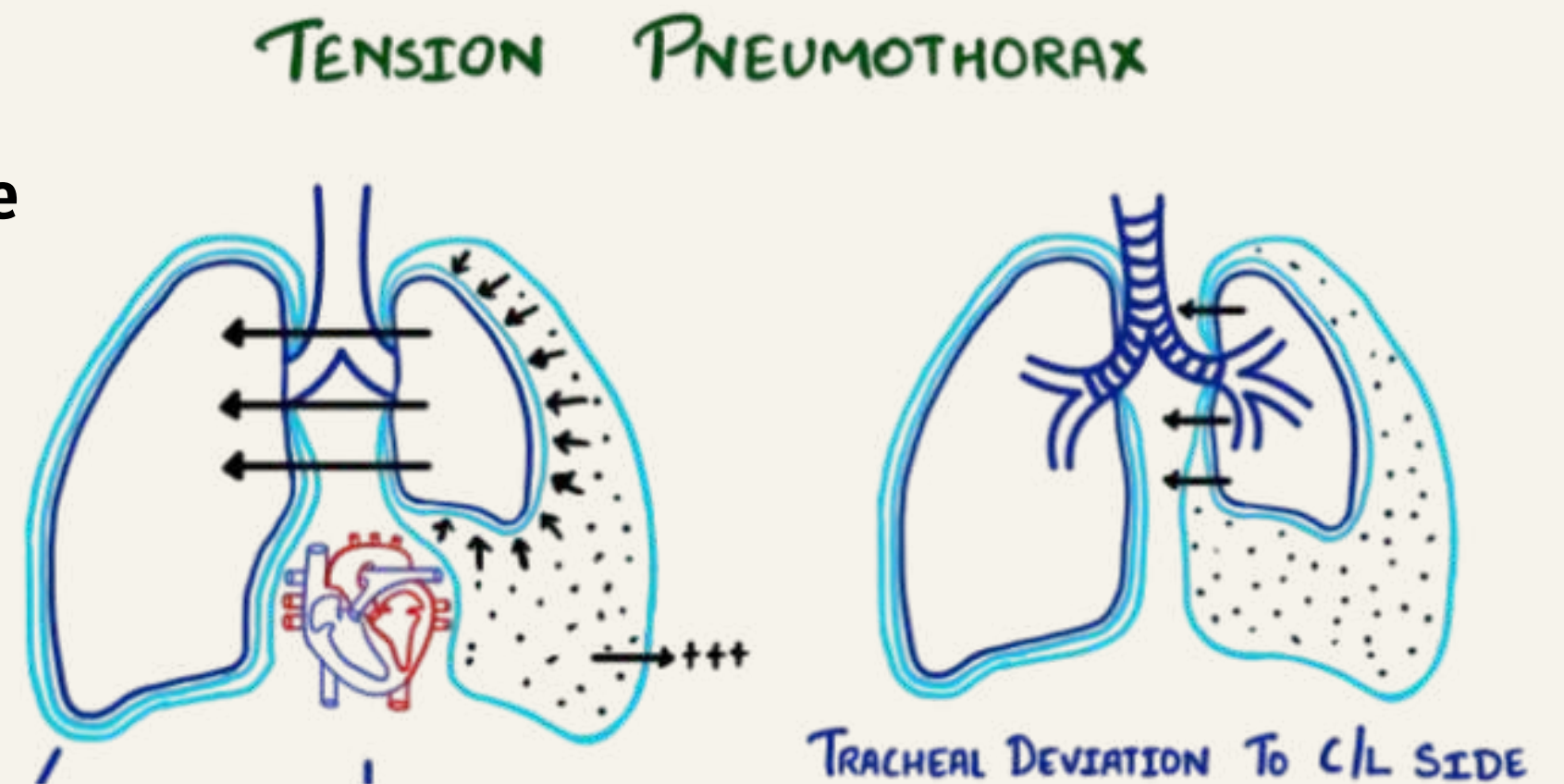
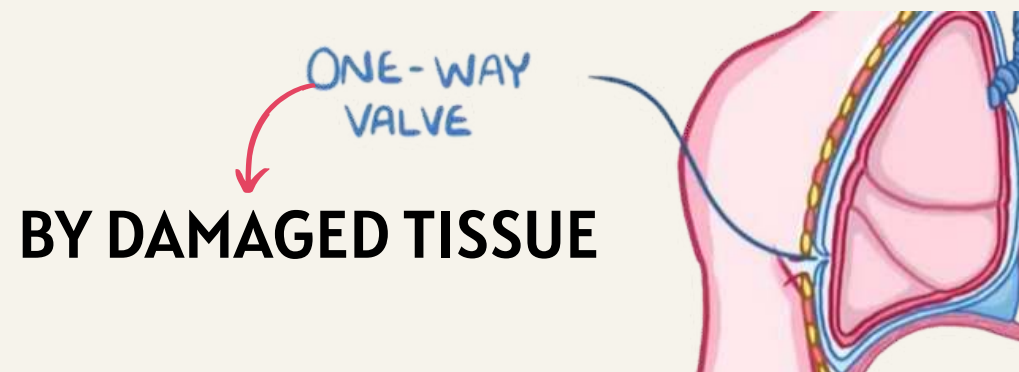


TENSION PNEUMOTHORAX

- A very serious medical complication (**Life Threatening**)
- Can develop from any other pneumothorax
- More commonly from traumatic origin
- Results from positive pressure in the pleural space

Air can enter but CAN'T Leave !

Creates a one-way valve



Through the valve air will accumulate → increased positive pressure → lung collapse on the affected side with shift of mediastinum to the other side → **impaired cardiac and respiratory function**

TENSION PNEUMOTHORAX

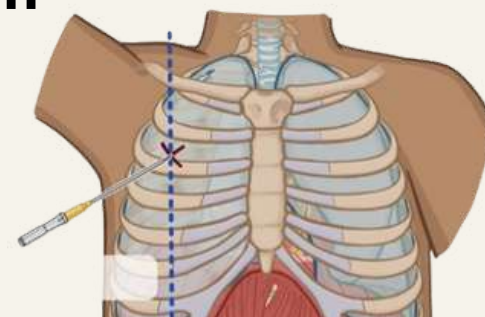
Symptoms:

- SOB
- Acute Chest Pain (Pleuritic)
- Hypotension
- Tachycardia
- Hypoxia

Management :

1) Needle Decompression (When unstable):

The pressure should be relieved immediately with needle decompression (2nd intercostal space midclavicular)



Diagnosis :

It is a clinical diagnosis

Can't wait for diagnostic imaging

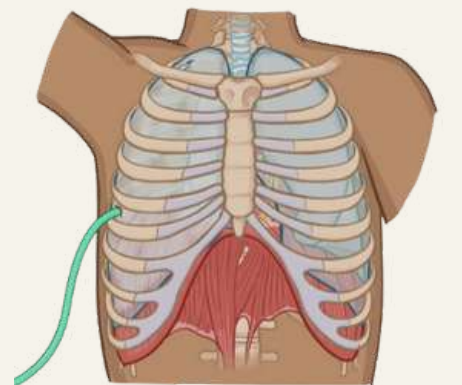
Hallmarks of tension pneumothorax

- Hypotension
- Tracheal deviation
- Hyperresonance of the affected side with absent breath sounds

The patient may develop jugular venous distension (JVD)

2) Chest Tube:

After using the Needle, a chest tube should be inserted (5th intercostal space anterior-axillary) later.

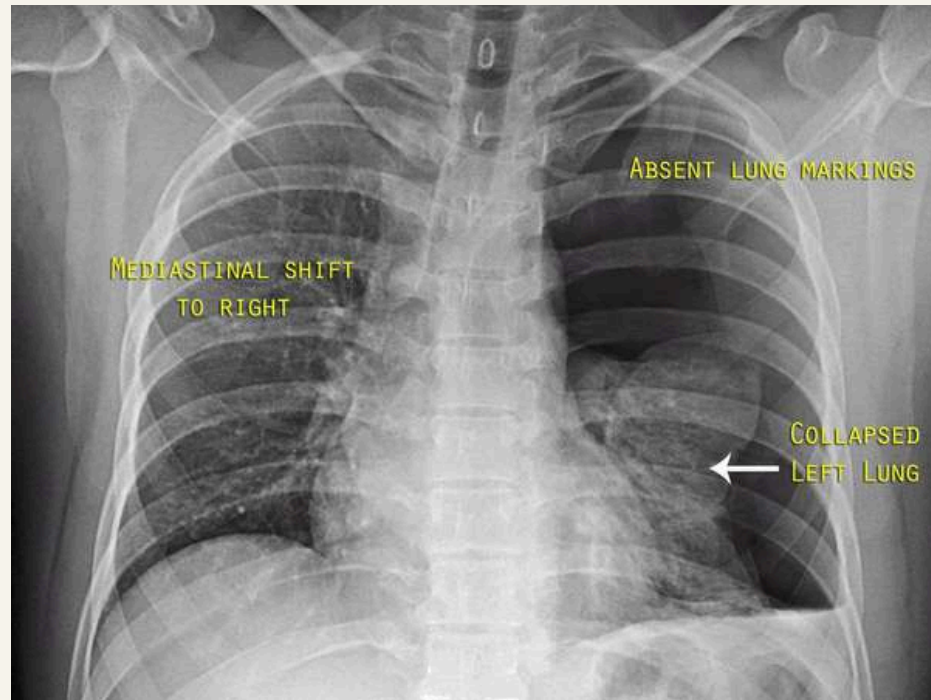


When UNSTABLE

X-rays should not be obtained before treatment.

RADIOGRAPHIC FEATURES

Plain radiograph

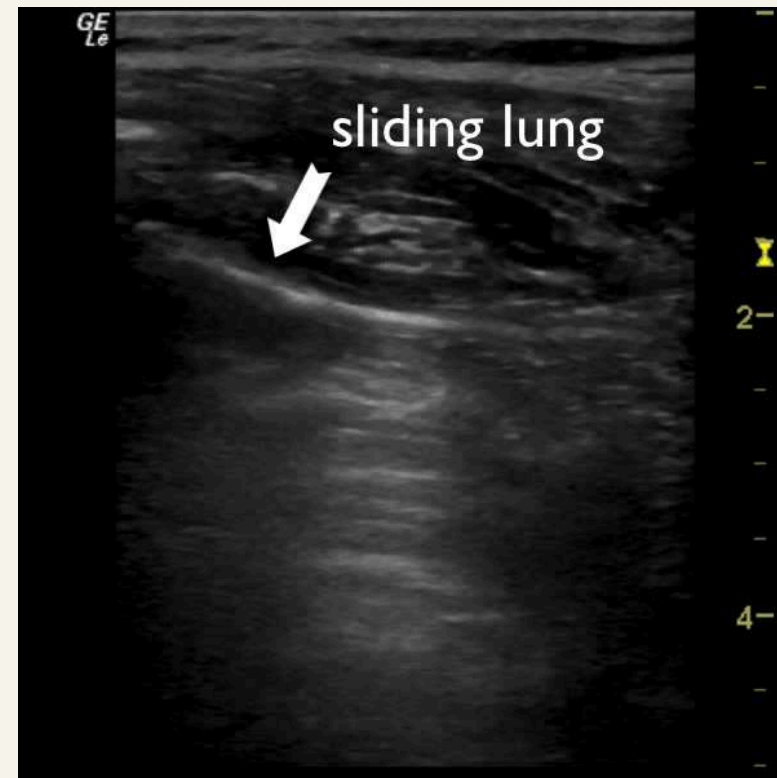


no lung markings are seen

lung may completely collapse

mediastinum **shift away** from the pneumothorax

Ultrasound

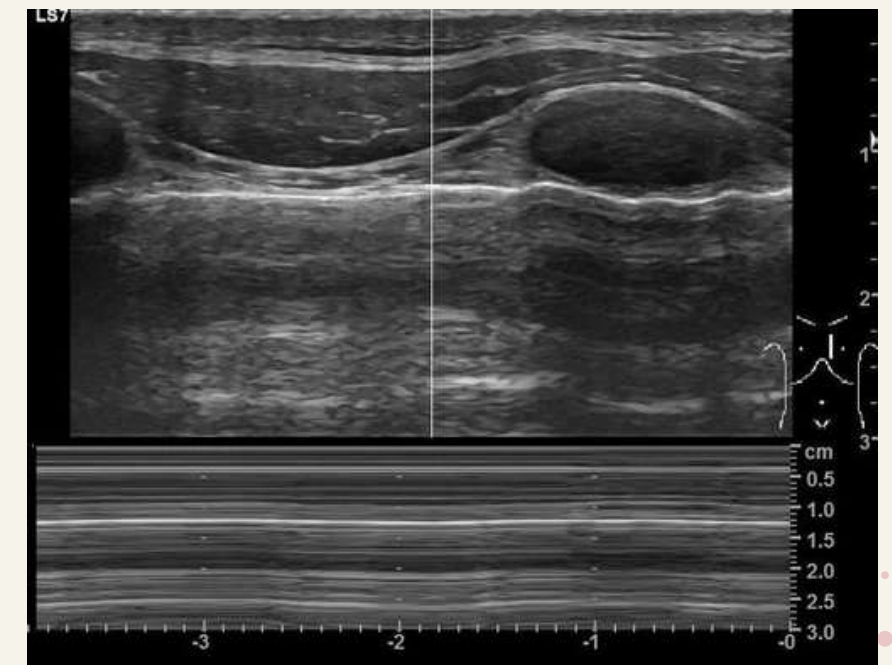


Visualising the junction between sliding lung and absent sliding is known as the **lung point sign** and is near 100% specific for pneumothorax

M mode ultrasound shows, normal lung sliding on right side resulting in a seashore sign / beach wave sign.

There is left-sided pneumothorax resulting in barcode/stratosphere sign.

Lung point could not be localised



The background features three vertical bars on the left: a wide pink bar, a medium blue bar, and a narrow beige bar. The right side of the slide is a light beige background with two rectangular areas of small pink dots, one in the top right and one in the bottom right.

THANK YOU