



Round : Emergency medicine

Group no : B3

Done By : Inas wail

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 Airway Breathing Circulation , Stable Unstable

In stable Initial Management is : 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

FOCUSED HISTORY : 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

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

Abdomen Inspection, palpation and auscultation

Extremities Unilateral extremity enlargement, pain, and erythema Limb pulse asymmetry

INVESTIGATIONS ECG LABS cxr

P - Pulmonary Embolism
E - Esophageal Rupture
T - Tension Pneumothorax
M - Myocardial Infarction
A - Aortic Dissection
C - Cardiac Tamponade

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

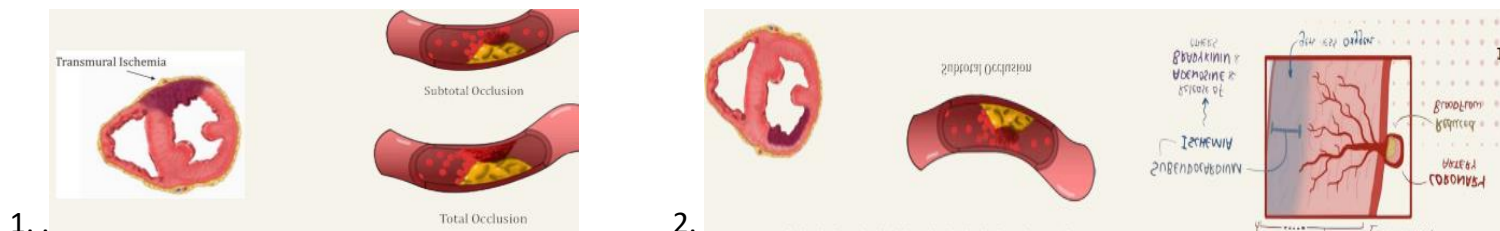
CAUSES OF ACS 1. Atherosclerosis: thickening of intima due to plaque 2. Plaque Rupture or Erosion: Triggers clot formation, obstructing blood flow. 3. Other Causes: coronary artery dissection, and cocaine use

RISK FACTORS, Prior coronary , DM, Peripheral artery disease, CKD, Hyperlipidemia, htn , family history M less 50 , f less 60) obesity, smoking

"Anginal chest pain is retrosternal, radiating to the jaw/arm. Exacerbated by exertion and relieved by rest. If not relieved by rest or nitroglycerin then it could be an MI due to angina. Atypical ACS occurs in three categories of patients (women, diabetic patients and the elderly). Their most common symptom is acute onset of dyspnea. In emergency cases if we suspect murmurs we need to take the patient to a quiet room, if not possible then we need to perform bed-side echocardiogram. Murmurs are musical audible sounds on auscultation, where the heart rate is abnormally heard"

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



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

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

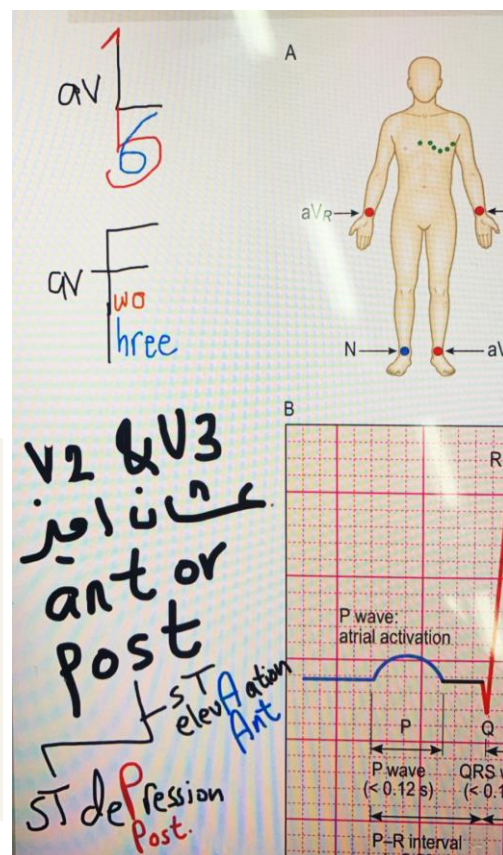
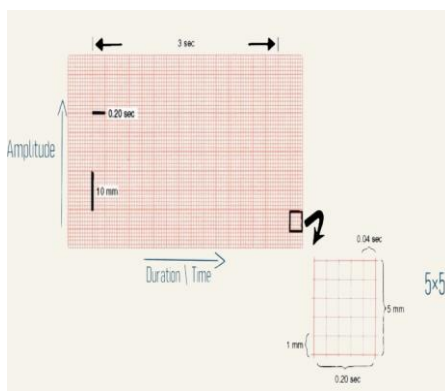
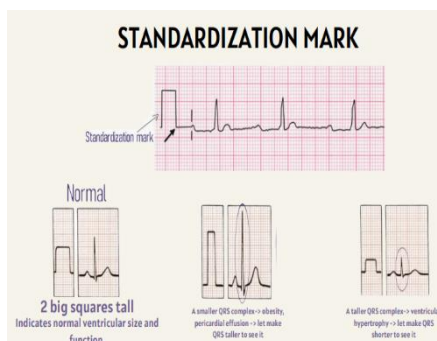
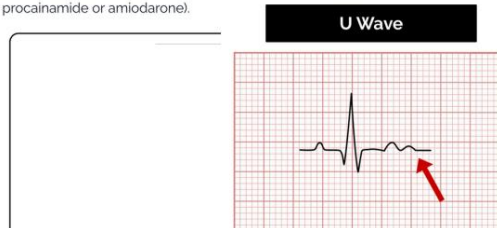
What is an ECG? Measures heart's electrical activity, using leads placed on the chest and limbs. • Purpose : To detect ischemia, infarction, arrhythmia , electrolytes abnormalities, pericardial diseases , valvular diseases and other cardiac abnormalities, Contraction = Depolarization Relaxation = Repolarization

- 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

U waves are not a common finding.

The U wave is a $> 0.5\text{mm}$ 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).



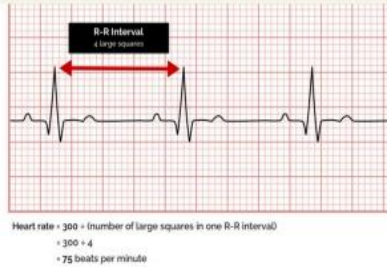
Regular heart rhythm

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



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

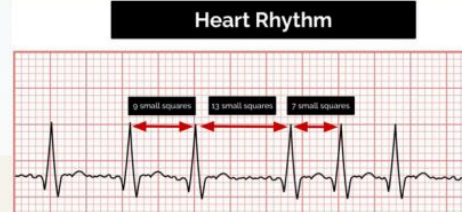
Irregular heart rhythm

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



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).

First Degree Heart Block



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 Heart Block Mobitz type 1 (Wenckebach)



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**.



P waves

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

- Are P waves present?
- If so, is each P wave followed by a QRS complex?
- Do the P waves look normal? – check duration, direction and shape
- 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

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

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**.

Third Degree Heart Block Complete Heart Block



ST depression

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



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 .

- **STEMI**: ST Elevation in specific leads. Q Waves: Develop overtime, indicating full-thickness infarction.

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.

"we need to have ST elevation in at least two contiguous leads on the 12 lead ECG. (leads that assess the same wall) - example: ST elevation seen in V1 and V2 is considered STEMI. but ST elevation in V2 and V3 is not indicative of STEMI, for STEMI, 1 small square (1 mm) is enough for limb leads. 2 small squares (2 mm) is enough for chest leads. - we need only 1 mm for limb leads but more in chest leads because limb leads are further than chest leads to the heart so they cover a wider area of the heart, so a wider area involved is needed to elevate the ST in these leads."

- **NSTEMI**: ST Depression or T Wave Inversion; no ST elevation). (management Medical therapy: 1. DAPT (Oral antiplatelet). 2. Anticoagulant • 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)

"In NSTEMI, patients could present with normal ECG, inverted T wave or even ST depression. That's why we can NOT only rely on ECG for diagnosis. So if we suspect NSTEMI then we need to perform cardiac enzymes test and admit the patient putting him/her on a continuous cardiac monitoring (3 leads maximum)"

- **Stable and Unstable Angina**: May show normal ECG at rest; stress may reveal ST depression (long term management of stable : • Lifestyle Modifications: Smoking cessation, diet, and exercise. • Medications: Beta-blockers, calcium channel blockers, nitrates, and aspirin. • Elective PCI or CABG: Considered if symptoms persist)

INITIAL **ACS** MANAGEMENT (MONA-B) Morphine: For severe pain. • Oxygen: If saturation less than 90 • Nitroglycerin: Unless contraindicated. • Aspirin: Antiplatelet effect. • Beta-blockers: To reduce heart workload

"☆ IF any patient experienced a second attack of chest pain we need to do a 12 leads ECG again. ☆ Most sensitive cardiac enzyme is troponin I. However, chronic kidney disease can lead to elevation in troponin levels (mainly CHF)

☆ We could use CPK- MB enzyme, however it is not specific. It can be elevated in any heart damage, or in cases of renal failure. CPK in general could be elevated in muscle disorders, like rhabdomyolysis or even IM injections. In any case of ACS, we need to return some kind of perfusion to prevent ischemia and necrosis. We also need to give aspirin (75 mg is enough as anti-platelets dose; > 300 mg dose is anti-inflammatory)

☆ If a patient has an MI and is hypotensive, do NOT give morphine and nitroglycerin as they can exacerbate hypotension. ☆ When managing patients with STEMI, the first thing we need to do always is to restore perfusion to prevent further damage and necrosis. If 90 mins have already passed, give clopidogrel (plavix) and aspirin. - if CATH isn't available/ possible then also give medications. MI is better handled by the elderly as they have collaterals that will decrease their mortality rates unlike the young."

AORTIC DISSECTION

Definition: is a life-threatening emergency that requires prompt recognition in the Emergency Department(ED). An aortic dissection is a tearing the inner layer of the aorta that leads to a progressively growing hematoma in the intima-media space.

"Typical presentation is someone coming to the ER complaining of sudden severe chest pain radiating to the back following heavy weight lifting. ☆ Other symptoms depend on the site of the deficit, if the ascending part is involved then we get stroke-like symptoms and syncope. If the arch is involved, we get a radio-radial delay associated with chest pain. If the descending part is involved, we get abdominal pain/ groin pain, as well as radio-femoral delay.

☆ stroke- mimics include hypoglycaemia, sepsis and aortic dissection involving the ascending aorta. (This was just mentioned briefly, I added an explanation) One of the risk factors for aortic dissection is bicuspid aortic valve, which is a congenital anomaly where the aortic valve has two leaflets instead of three, which makes this valve ineffective at stopping blood from leaking back into the heart. This leakage is called aortic regurgitation. The aortic valve may also become stiff and not open up, which is called aortic stenosis, which causes the heart to pump harder than usual to get blood through the valve. The aorta may become enlarged with this condition and this increases the risk of aortic dissection."

"X-ray findings that we can see in cases of AD are: loss of the heart knuckle (knob) Widening of the mediastinum Calcium sign Plural effusion and sometimes displacement of the trachea/esophagus"

"One of the most important complications of aortic dissection is cardiac tamponade. Beck's triad is very important. Muffled heart sound occurs because of the weak sound transmission. Hypotension occurs due to the fluid restricting the heart expansion (no preload) which interferes with its contractility too (no afterload) JVD is due to the retention of the blood back to SVC and up the jugular vein as the heart is unable to pump the blood in its chambers."

Causes , **Acquired**

1. 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.
2. Trauma e.g. deceleration injury in a motor vehicle collision, iatrogenic injury during valve replacements , graft surgery (traumatic aortic dissection)
3. Vasculitis with aortic involvement (e.g., syphilis, Takayasu arteritis) 4. Use of amphetamines and cocaine ,
4. pregnancy Third-trimester pregnancy (or early postpartum period) 6. Atherosclerosis)

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

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.

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 categorizes dissections according to their origin and extent

DEBAKEY CLASSIFICATION

Type I	Type II	Type III
- 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	- Dissections originate in, and are restricted to, the ascending aorta. - Generally requires surgery	- 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 (syst-diastolic)
5. Syncope, diaphoresis, confusion, or agitation

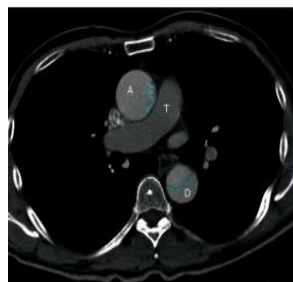
C O M P L I C A T I O N S

1. 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
2. Aortic rupture and acute blood loss (shock & death)
3. Complications of Stanford A MI, Aortic regurgitation, Cardiac tamponade, Stroke

DIAGNOSIS

Clinical assessment(evaluation of symptoms and risk factors) , Physical Examination((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)

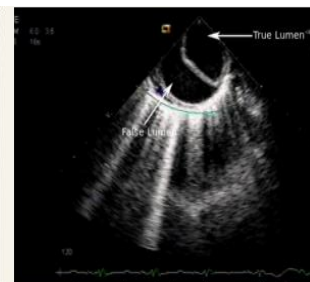
,Laboratory Assessment (D-dimer) , Imaging (CT angiography : the modality of choice, careful in renal insufficiency TEE and MRA : initial study for high risk patients, confirmatory if uncertain CXR : abnormal in 80% , may show : abnormal aortic contour, widening of mediastinum , deviation of trachea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



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.

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.

Management :

1. Hemodynamically unstable : 1.Resuscitate with ACLS (ABCDE approach) 2.Perform simultaneous bedside investigation 3.treat life threatening complications (hemodynamic support) 4.Expedite surgical management
2. 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
3. Medical (Hypotensive pt IV fluids Vasopressor : norepinephrine, phylephrine) 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)
4. Supportive care: (Management of pain and anxiety which lowers sympathetic tone) 1.Pain management (morphine) 2.Procedural sedation 3.Anticoagulant reversal

Deep Vein Thrombosis (DVT)

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.

- " Risk factors are components of Virchow's triad (picture aside) Mostly it involves lower limbs. It occurs in upper limbs mainly in cancer patients. In lower limbs, we will find edema. There are two types of edema, pitting and nonpitting edema.
- most common cause of nonpitting edema is myxedema (hypothyroidism) shin of tibia
- Most common cause of pitting edema is heart failure and fluid accumulation due to excess fluids.
- D-dimer is used to determine whether there is a dvt/pe or not (indicative of a clot) however if it was positive then the diagnosis is not confirmed, we still need further testing. (it could be normally elevated in pregnant women and cancer patients.)
- ☆ Most common ECG finding in PE patients is sinus tachycardia (abnormal change) // normal change is finding the ECG normal. A rare but unique and classic pattern known as Mcginn-White sign "S1Q3T3" can be seen. (a deep S wave in lead 1, deep Q wave in lead 3 and an inverted T wave in lead 3) X- ray findings in PE patients include 7 signs, sometimes we find none so
- x-ray is not the best investigation to do. We need to examine popliteal as well as femoral pulses.
- Treatment is to give two-drugs therapy (warfarin and heparin) they should be started together as warfarin has a slow onset of action.
- 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) Virchows triad : hypercoagulability – endothelial injury- venous stasis

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 2. 3. Redness and Warmth Enlarged Veins: Surface veins may appear more prominent 4. Palpable Cord: Firm, 5. 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)

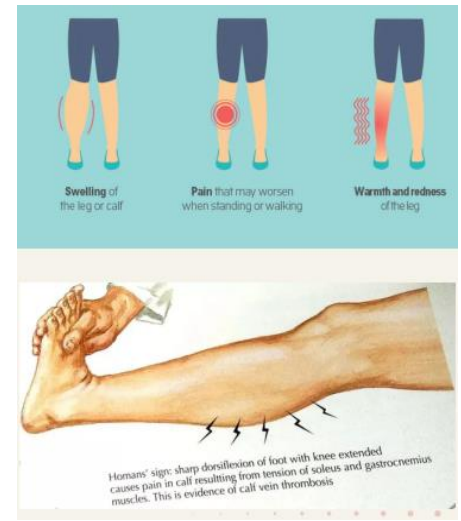
Diagnosis: Wells Criteria

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%)**



- 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 tests mostly useful when negative as its negative predictive value is >99%.)
- Imaging Modalities
 - A. Doppler Ultrasound: 1. 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
 - 2. 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)
 - B. Compression Ultrasound: mainstay for DVT diagnosis assess vein compressibility (non-compressibility indicates a clot) often used in conjunction with Doppler ultrasound

Treatment of Suspected/Confirmed DVT in ER

Anticoagulation Low Molecular Weight Heparin (LMWH) or Fondaparinux = indicated for immediate anticoagulation
Warfarin or Direct Oral Anticoagulants (DOAC) = 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

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.

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)

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 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.

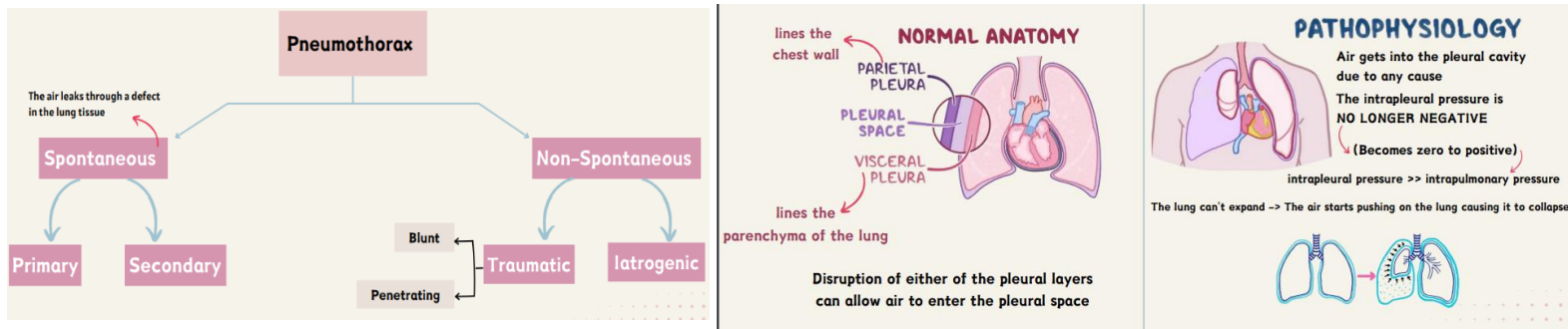
THE EMERGENCY TREATMENT

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.

PNEUMOTHORAX

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



Spontaneous Pneumothorax most likely results from subpleural bulla rupture. 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. 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: Tall, Thin, Young, Males, Smokers, Marfan).

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).

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 post procedure chest x-ray is indicated

" It could be either spontaneous or due to a cause. Mostly spontaneous in thin tall men, wise to a rupture of a small emphysematous bullae.

Iatrogenic pneumothorax could be due to therapeutic/ diagnostic procedures like biopsy taking, central vein cannula introduction as well as aspiration.

Tension pneumothorax is either closed or open. Closed in blunt injuries, like due to a seatbelt in car accidents or airbag, and open in gunshot and stabbing wounds. Triad of tension pneumothorax includes: absent breath sounds, jugular vein distension and hypotension.

Physical examination and related findings are very important to diagnose tension pneumothorax. We could find tracheal deviation, hyperresonance on percussion as well as absent/reduced breath sounds on one side"

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

Symptoms: SOB ,Acute Chest Pain (Pleuritic) ,Hypotension ,Tachycardia ,Hypoxia

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)

Management: 1) Needle Decompression (When unstable): o The pressure should be relieved immediately with needle decompression (2nd intercostal space midclavicular)

2) Chest Tube: o 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.

