

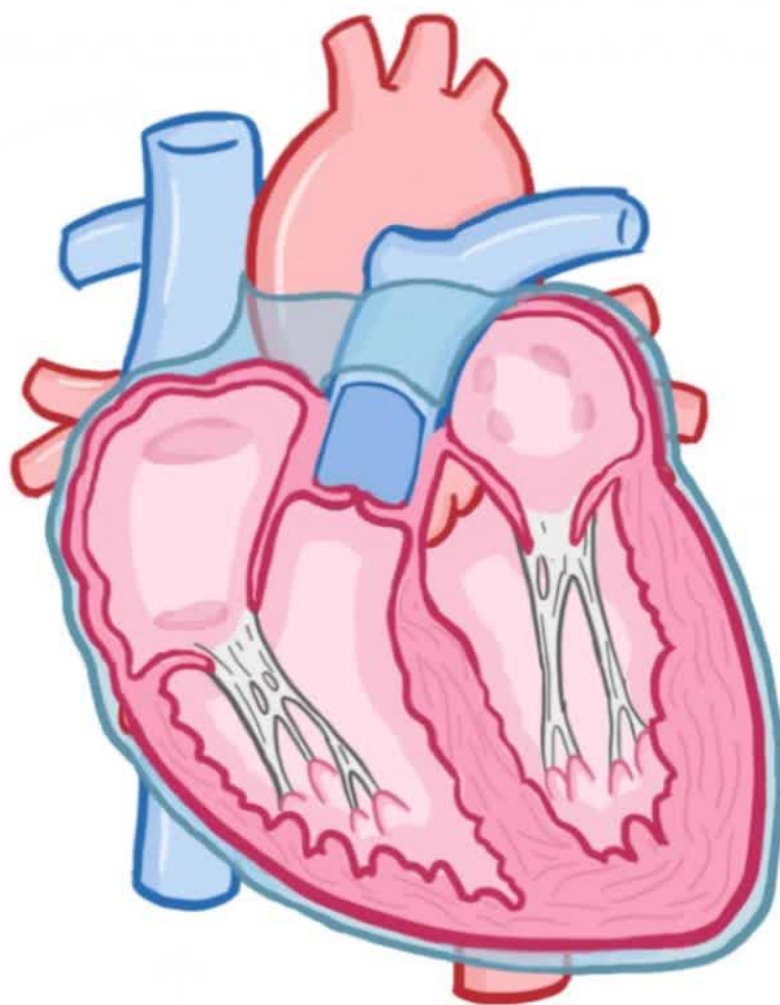
Internal medicine OSCE

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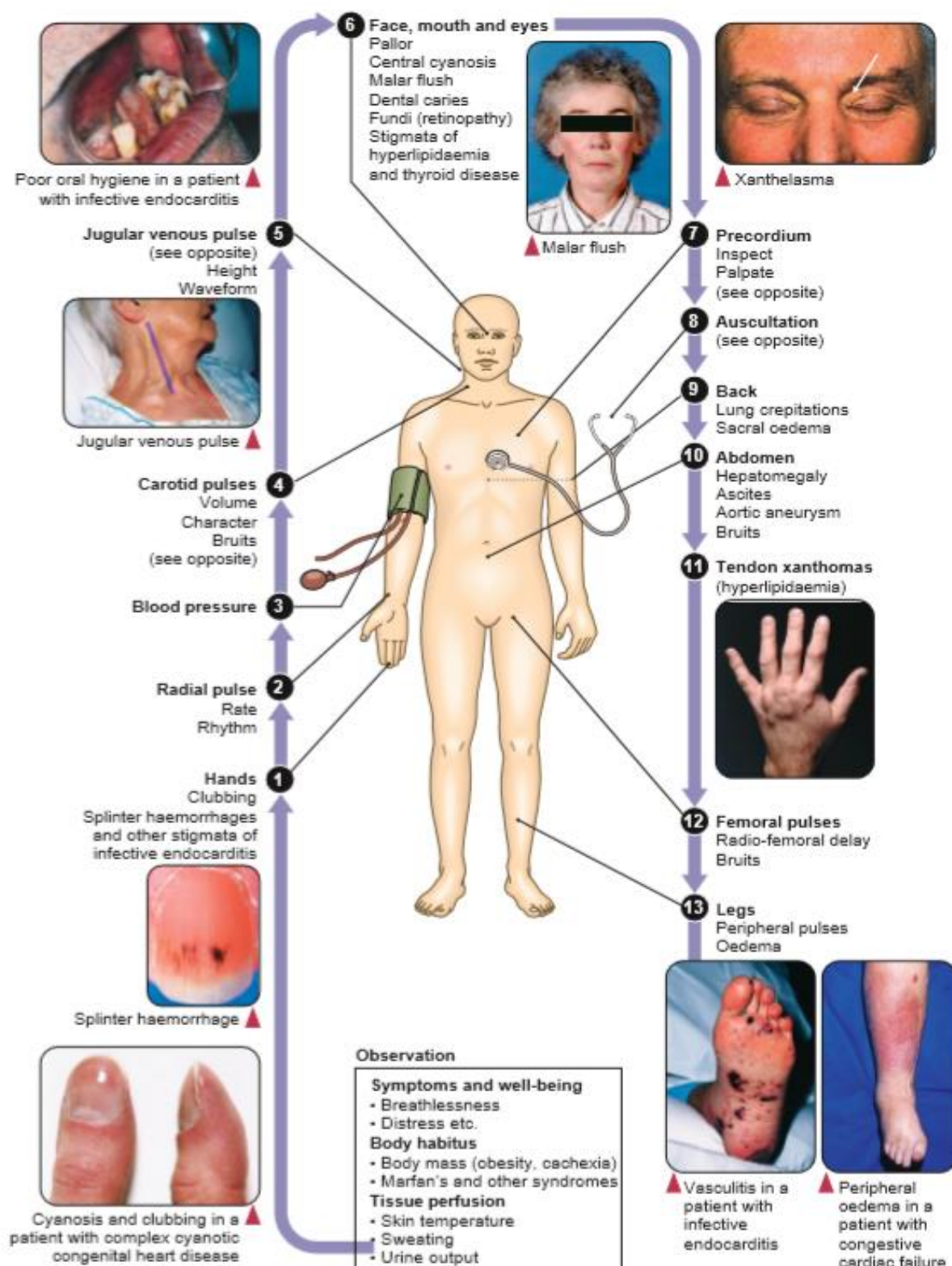
School of medicine Al-Quds university

1. Cardiovascular System
2. Respiratory System
3. GIT
4. Thyroid
5. Rheumatoid hand
6. Deep venous thrombosis
7. Endocrine System (Diabetes mellitus)

Chapter 1: Cardiology



CLINICAL EXAMINATION OF THE CARDIOVASCULAR SYSTEM





History Taking

Chief Complaint

Chest pain:

Case: a 60 year old male patient, presents to the ER with chest pain. Take history to know the cause.

-Introduce yourself, take the patient's profile (Age, occupation, marital status). Take the duration and ask the pt. what he was doing immediately before

Cardiac causes (List of differentials)				
	Angina	MI	Aortic dissection	Acute pericarditis
Site	Diffuse retrosternal	Retrosternal	Retrosternal, between shoulder blades	Retrosternal
Onset	Recent	Sudden, intense	Very Sudden	Gradual
Character	Tight, Heavy, pressure	Crushing, very heavy	Tearing, ripping	Sharp, stabbing
Radiation	To the Lt (or Rt) shoulder or arm, throat, jaw, back.	To the Lt. Arm, back, epigastrium	To the back	To the Lt. shoulder, arm, back
Associated symptoms	-----	Sweating, pallor, nausea, vomiting, diarrhea, Angor Animi	Sweating, pallor, nausea, syncope, focal neurological deficit	Fever, chills, rigors, cough
Timing	Intermittent each episode lasts <10 mins	Persistent for more than 30 mins.	Prolonged	Variable duration
Exacerbating factors	Exertion, Emotion, Cold, Exercise after meals	Stress, Exercise	-----	Inspiration, Coughing, Lying down
Relieving factors	Rest, glyceryl nitrate	Not relieved by glyceryl nitrate	-----	Sitting up, Leaning forward, Analgesics, NSAIDs
Severity	-----	Very severe	Very severe	-----

Notes	If unstable angina the pain is more severe, occurs at rest, increasing in frequency and duration	May be silent in elderly, diabetics and females	-----	Viral etiologies may be preceded by flu-like respiratory or GI symptoms
Risk factors	Age (♂>45 ♀>55), smoking, HTN, Hyperlipidemia, DM, previous attacks, Family history of premature CAD (♂<55 ♀<65)		HTN, Marfan syndrome, Ehlers-Danlos syndrome, Weight lifting, Pregnancy in 3 rd trimester	-----

Non-cardiac causes				
Take the duration and ask the pt. what he was doing immediately before				
	Pleural pain	Chest wall pain	Mediastinal pain	Esophageal pain
Site	Well localized	Wide area	Retrosternal. Central	Retrosternal or epigastric
Onset	Sudden	Sudden	Variable	Sudden
Character	Sharp, Stabbing	Generalized tightness	Dull, aching, gnawing	Dull, burning
Radiation	To the neck and shoulder tip (if diaphragmatic area is involved) To the Epigastrium (if the pleura overlying the lower six ribs is involved)	-----	To the medial side of the arm (in Pancoast's tumor)	To the arms and back
Associated symptoms	-----	Cough	-----	Heartburn and acid reflux
Timing	-----	Wakes the pt from sleep	Wakes the pt. from sleep	Wakes the pt. from sleep
Exacerbating factors	Inspiration and coughing	Coughing	Coughing (in tracheobronchial infections)	Lying flat
Relieving factors	-----	-----	-----	Nitrites sometimes relieve
Severity	-----	-----	-----	Usually mild
DDx	Pneumonia, PE, pneumothorax, fractured ribs	Rib fractures, intercostal muscle injury, HZV, malignancy	Lymphoma, thymoma, infection of tracheobronchial tree)	Esophageal spasm, GERD, hiatus hernia

Review of systems	• Ask about SOB, Palpitation, Syncope, Claudication, Ankle swelling • Cough, Wheezes, Hemoptysis • Fever, Chills, Wt. loss, Anorexia, Nausea, Vomiting
Past medical and surgical	HTN, hyperlipidemia, DM, previous caths and stents, recent infections, previous heart surgeries
Drug Hx	NSAIDs, B-blockers, Thyroxine, Cocaine
Family Hx	Family Hx of heart disease or premature CAD
Social Hx	Smoking history (# of pack years), alcohol, travel history

Shortness of breath (SOB)/ Dyspnea:

Case: a 53 year-old man presents to your clinic complaining of SOB, take detailed history to know the cause.

-Introduce yourself, take the patient's profile (age, occupation, marital status).

List of common differentials	Cardiac	Heart Failure: -Take the duration -Timing (At night, With exertion, At rest) -if at night: does it occur immediately when lying flat (Orthopnea), or does it occur later during sleep waking the patient gasping for air (PND)? -ask about the # of pillows used to sleep -If with exertion: ask about baseline, how it progressed? how does it affect daily life? (Check box 7.7 in the next page to assess severity) -Is it relieved with diuretics? (Ask about compliance to drugs and diet) -Associated symptoms: Fatigue, coughing with frothy blood sputum, ankle swelling, abdominal distention, oliguria, chest pain, palpitations.
		IHD: -Take the duration -Is it exacerbated with exercise? Is it relieved by glyceryl nitrate? -Associated symptoms: chest pain, palpitation, sweating, nausea, pallor.
		Others: -Ask about Fever, chills, rigors, cough
	Respiratory	Ask about the duration (Check box 7.6 in the next page for DDx).
		Asthma: -Typically wakes the pt. from sleep around 3-5 am. -Associated with wheezes and cough (either dry or productive with white viscid mucoid sputum). -Relieved by inhalers. -Ask about exposure to allergens (shaking bedding, hoovering, and mowing the lawn. Exposure to cats, dogs, horses and tree pollens), smoke, perfumes, fumes, cold air or drugs (Aspirin, NSAIDs). -Ask if related to exercise and if it continues to worsen 5-10 minutes after stopping activity (Exercise-induced asthma). -Ask if it improves on weekends or holidays (occupational asthma). -Causes of exacerbation: Stress (emotion and exercise) Infection (runny nose, fever, Chills, sore throat, chest pain, yellow-green sputum). Exposure to irritants (allergens, smoking, cold air, drugs).
		COPD: -Typically is worse upon waking in the morning. Improved after coughing clear, grey mucoid sputum. -Associated with wheezes and productive cough.
		PE: -Very sudden in onset. -Exacerbated with upright posture, relieved by lying flat, Associated with pleuritic chest pain, , cough with hemoptysis, tachycardia, pallor, sweating.

		Pneumonia: -Onset within hours to days. -Associated with: Acute productive cough (purulent green sputum), Fever, Chills, Pleuritic chest pain, Wt. loss and history of URTI. -Ask about abdominal pain, nausea and vomiting to consider lower lobe pneumonia.
	Hematology	Anemia: -SOB <u>without</u> chest pain. -Associated with: headache, fatigue, loss of concentration, dizziness upon standing, palpitation, bone pain, Wt. loss, hx of bleeding or easy bruising.
	Psychogenic	-Occurs suddenly at rest or while talking. -Associated with: Lightheadedness, Dizziness, Tingling in the fingers and around the mouth, Chest tightness.

Past Medical hx:	DM, HTN, Hyperlipidemia, Malignancy, hx of recent surgery or bed rest, Alpha-1-antitrypsin deficiency.
Drug hx:	Aspirin, NSAIDs, B-blockers, Ca ⁺² Channel blockers, Inhalers.
Family hx:	Family Hx of atopy (Allergic Rhinitis, sinusitis, Eczema, Conjunctivitis, Asthma), Hx of Lung Ca. or IHD.
Social hx:	Smoking Hx (calculate pack years), travel Hx, Hx. of vaccination



7.6 Breathlessness: modes of onset, duration and progression

Minutes	
- Pulmonary thromboembolism - Pneumothorax	- Asthma - Inhaled foreign body - Acute left ventricular failure
Hours to days	
- Pneumonia - Asthma	Exacerbation of COPD
Weeks to months	
- Anaemia - Pleural effusion	Respiratory neuromuscular disorders
Months to years	
- COPD - Pulmonary fibrosis	Pulmonary tuberculosis



7.7 Medical Research Council (MRC) breathlessness scale

Grade 1	Breathless when hurrying on the level or walking up a slight hill
Grade 2	Breathlessness when walking with people of own age or on level ground
Grade 3	Walks slower than peers, or stops when walking on the flat at own pace
Grade 4	Stops after walking 100 metres, or a few minutes, on the level
Grade 5	Too breathless to leave the house
(Grade 5b)	Too breathless to wash or dress

Palpitations:

Case: a 40 year-old lady presents to ER complaining of palpitations, ask relevant questions to know the cause of her complaint.

-Introduce yourself, take the patient's profile (age, occupation, marital status). Take the duration and ask what she was doing immediately before.

	Sinus tachycardia	SV Tach	Extra-systoles	A Fib	V tach
Onset	Gradual	Sudden with jump	Sudden	Sudden	Sudden
Character	Regular, fast	Regular, fast	Jump or missed beat followed by a strong beat	Irregular, fast (slow in elderly)	Regular, fast
Associated symptoms	-----	Polyuria, chest tightness, lightheadedness	-----	Polyuria, SOB	Presyncope, syncope, chest tightness
Timing	Lasts for a few mins	Lasts for mins-hours	Brief	Variable	Variable
Exacerbating factors	Anxiety, stress, exercise, caffeine, alcohol, smoking	Bending Usually occur at rest	Fatigue, caffeine, alcohol,	Exercise, alcohol	Exercise
Relieving factors	-----	-Valsalva maneuver -Carotid sinus pressure	Walking or exercise	-----	-----
Severity	Mild to moderate	Moderate to severe	Mild	Very variable	Severe
Causes	-Drugs (B2-agonists, Thyroxine, Antihistamines, Decongestants, Amphetamine, Cocaine, Ecstasy) -Infection and sepsis (Fever) -Thyrotoxicosis (Wt. Loss, Heat intolerance, Sweating) -Active bleeding (hematemesis, melena, hematuria) -Anemia (fatigue, weakness, SOB) -Pregnancy - - Pheochromocytoma (headache, sweating)	<u>No underlying heart disease</u>	-Excessive alcohol, caffeine, or tobacco consumption -Drugs (Tricyclic antidepressants, digoxin)	-IHD -Valvular heart disease (Mitral stenosis) -HTN - - Cardiomyopathy -Sick sinus syndrome -Alcohol excess -Congenital heart disease -Constrictive pericarditis - - Hyperthyroidism -OSA -Asthma/COPD -Idiopathic	-<u>People with cardiomyopathy or previous MI</u> -<u>People with pacemakers or Intra-cardiac devices have increased risk of V. Tach</u>

Review of systems	SOB, Chest pain, Syncope, Claudication, Ankle swelling, Fever, Headache, Dizziness.
Past medical and surgical Hx	Rheumatic fever, previous heart attacks, anemia, hyperthyroidism, HTN, DM, HF, endocarditis, previous caths or stents, previous heart surgeries.
Drug Hx	Thyroxine, B-agonists, Digoxin, Anti-depressants, Diuretics
Family Hx	Family hx of heart disease or sudden death
Social Hx	Smoking (#of pack years), alcohol, caffeine intake

Syncope:

Case: a 35 year-old man presents to ER after a brief LOC, take history to know the cause.

-Introduce yourself, take the patient's profile (age, occupation, marital status). Take the duration and ask what he was doing immediately before.

	Postural Hypotension	Neuro-cardiogenic syncope (Vasovagal attack)	Arrhythmias	Mechanical Obstruction to CO
Onset	Suddenly upon standing	Sudden	-----	-----
Prodrome	-----	Light headedness, tinnitus, dark vision, sweating, pallor. When he wakes up he is flushed and vomiting.	Palpitations, chest pain, SOB	-----
Duration	1-2 mins	<60 seconds	-----	
Exacerbating factors	Standing	Standing for long periods in warm weather, emotions, anxiety, cough	-----	Exertion, during exercise
Relieving factors	-----	Elevation of legs	-----	-----
DDx	Hypovolemia, septicemia, autonomic neuropathy, Drugs (diuretics, anti-hypertensives, vasodilators)	Vasovagal attack If frequent: hypersensitive carotid sinus syndrome	Tachyarrhythmia: V. Tach Brady-arrhythmia: Complete heart block	Severe aortic stenosis, HOCM, PE, Aortic dissection
Review of systems	- Chest pain, SOB, palpitation, claudication, ankle swelling, cough - Nausea, vomiting, diarrhea, melena, hematemesis - Fever, fatigue, pallor, anxiety - Weakness, numbness, jerky moves, tongue biting, amnesia after attack			
Past medical and surgical Hx	Anemia, Bleeding, prolonged vomiting or diarrhea, prolonged fasting, seizures, HTN, DM			
Drug hx	Vasodilators, Nitrates, ACEIs			
Family Hx	Bleeding, seizures			

Box 29.5 Clinical features strongly suggestive of syncope

- LOC preceded by chest pain, palpitation, dyspnoea, light-headedness or typical 'pre-syncopal' prodrome
- LOC after standing up, after prolonged standing, during exertion or following a typical precipitant:
 - unpleasant sight, sound, smell or pain
 - venepuncture
 - micturition
 - cough
 - large meal
- Brief duration of LOC (<1 minute)
- Rapid return of clear-headedness after LOC

Cardiovascular Examination

Hatem RJOOB

General Examination:

- 1- Tell the patient your name, your job, what you want to do, and introduce the examiner.
- 2- Wash your hands.
- 3- Put the patient at 45 degree.
- 4- Expose the patient pericardium and legs.
- 5- General appearance:
 - Stand at the end of the bed and look for:
 - Is the patient conscious, cooperative , alert oriented to time place and person .
 - Is the patient look well or ill?
 - Body mass (obese, cachexic).
 - Respiratory distress (tachypnea, dyspnea, cough, cyanosis).
 - Sweating.
 - Cyanosed , jaundice , pale ?
 - IV line
- 6- Examine both hands together, inspect the dorsal then palmar aspect of both hands, and look for:
 - Cyanosis (Heart failure due to decrease CO and vasoconstriction/ COPD)
 - Staining (TOBACCO)
 - Splinter hemorrhages (up to two is normal)- sign of endocarditis
 - Clubbing
 - Koilonychias (brittle, flat, spoon shaped- nails, occurs in iron deficiency anemia)
 - Capillary refill time
 - Janway lesion (painless , on the palms and soles)
 - Osler node (painful , raised , on peds of fingers and toes)
 - Temperature (cold in peripheral vascular disease/ cold in heart failure due to vasoconstriction/ warm in COPD)
 - Sweating (occurs in Hyperthyroidism- risk factor for cardiomyopathy, AF, and HF)
 - Palmar erythema (in chronic hepatic congestion, and hyperthyroidism)
 - Changes in crease color (pallor)
 - Dupuytren's contracture (occurs with DM- risk factor for cardiac diseases)
 - Muscle wasting (carpal tunnel syndrome)
 - Tremor (flapping tremor due to CO2 retention)

Causes of carpal tunnel syndrome :

1. Idiopathic
2. Obesity , pregnancy
3. ESRD(hemodialysis)
4. RA , amyloidosis
5. Endocrine (DM , hypothyroidism , Acromegaly)

Causes of Dupuytren contraction

1. Family hx (autosomal dominant)
2. DM , smoking , hyperlipidemia
3. Alcohol excess
4. HIV

Causes of palmar erythema

1. P: pregnancy , polycythemia
2. A: Alcohol intake , Autoimmune (RA , SLE)
3. L: liver Failure , leukemia
4. M: medication (steroids)
5. Thyrotoxicosis

Causes of CLUBBING

1. Tumors

- mesothelioma
- lung cancer
- Esophageal CA
- thymoma
- Atrial myxoma

2. Pulmonary (BILE)

- B: Bronchiectasis
- I: interstitial lung disease
- L: lung abscess
- E: Empyema

3. Cardiology

- cyanotic heart disease
- infective endocarditis

4. GIT

- IBD
- celiac
- liver disease

5. Endocrine (thyroid acropachy)

6. Familial (%5)

7. Idiopathic

7- Check both radial pulses together:

- Site: is found lateral to flexor carpi radialis tendon.
- Technique: use your three middle fingers.
- Assess the rate, rhythm, and symmetry (for radio- radial delay, this could occur in aortic dissection, and vascular diseases like takayasu).
- Don't forget to check for **collapsing pulse** by raising the pt's arm and feeling across the pulse with your fingers (**water hammer**).

8- If radial pulses are ok, don't examine the brachial pulses, if you are not sure then examine the brachial pulse

- Technique: use your right thumb for the patient right brachial artery.
- Site: it is found in the antecubital fossa medial to the biceps tendon.
- Assess rate , rhythm , volume , character .

9- Measure the BP using the sphygmomanometer.

- Take BP then look for pulsus paradoxus and postural hypotension.
- measure it at sitting position and standing (to see if there is postural hypotension).
- Where to measure: measure both arms.

10- Inspect the face for:

- Eyes: pallor, jaundice, arcus cornea, periorbital xantholasma, hypertensive and diabetic retinopathy.
- Malar flush (occurs in MS).
- Central cyanosis (lips, and under tongue) - the most common cardiac cause is pulmonary edema, other cause: right-left shunt.
- Angular stomatitis, smooth tongue (iron deficiency anemia).
- Sweaty face.

11- Neck: (visible masses , scars , distended veins , JVP , carotid Pulse)

Check the carotid pulse (mainly for volume).

- Site: is found at the angle of the jaw, anterior to sternocleidomastoid muscle.
- Assess: volume and character.
- Technique: use your left thumb for the patient right carotid artery.
- Caution: Never compress both carotid arteries.

Volume:

- Normal
- Large volume (AR, high CO (exercise, fever, pregnancy, and anemia))
- Low volume (HF, peripheral vascular disease, and hypovolemia (thready))

Character:

- Normal
- Slow rising (AS)
- Collapsing (AR)
- Bisferiens ((AS+ AR)// or HOCM)
- **Alternans** as in Advanced Heart failure

Inspect the JVP:

- Anatomy: the internal jugular vein enters the neck behind the mastoid runs deep the sternocleidomastoid and goes between its two head.
- Why important: It is important because it reflects the pressure in the right atrium because there is no valve in the superior vena cava.
- Position: You should examine it at 45 degree. In the normal person when he is in the supine position the vein will distend and no pulsation will be seen. In the sitting position the pulse is hidden behind the clavicle and sternum. At 45 degree it will be at the level of the clavicle.
- Normal JVP is less than 4 cm above the sternal angle. (normal atrial pressure is less than 9 cm).
- When the patient at 45 degree then the sternal angle = clavicle (both are at the same level), then you can measure from the sternal angle . if the patient is not 45 degree then you must measure JVP from the clavicle
- If you can't see the JVP, then change patient position , if you still cant see JVP then do Abdomenjugular reflux

- Abdomenjugular reflux : ask the patient if there is pain , then press firmly over the center of the abdomen for few seconds after explaining to the patient what you want to do... (Increase VR> increase RA pressure> raised JVP by 2-3 cm).
- It is raised in: HF, volume overload, major pulmonary embolism, superior vena cava obstruction (non Pulsatile), pericardial constriction (with Kussmaul sign- with inspiration the JVP raise instead of being lower)
-

To differentiate between carotid pulsation and jugular pulsation:

Carotid	Jugular
Rapid outward movement	Rapid inward movement
One peak per beat	Two peaks
Palpable	Impalpable
Unaffected by pressure at the root of the neck	Diminished by pressure
Independent of abdominal pressure	Rises
Independent of position	Varies
Independent with respiration	Decrease with inspiration

Wave abnormality	Cause
Absent ,a, wave	AF
Giant ,a, wave	TS, PS, P. HTN
Cannon waves	Complete heart block
Giant ,v, waves	TR

Note: we use internal jugular vein not external because external has tortuous course

JVP change with respiration

-JVP normally falls down with inspiration due to decrease intrathoracic pressure, when its not decrease with inspiration then this is called kussmaul sign

-kussmaul sign: it's a paradoxical rise of JVP with inspiration. its seen in pericardial constriction, sever right ventricular failure, restrictive cardiomyopathy but not in cardiac tamponade.

- inspiration increase venous return , but normally the ventricle can accommodate to this increase , however in the above pathologies , ventricle can't accommodate and so causes paradoxical raise in JVP (kussmaul sign)

JVP examination:

Pt should be in semi-recumbent position, slightly turn his head to the lt side

1. By inspection (use your torch):

- 2 pulses were visible, an outward single peaked arterial pulse and an inward double-waved venous pulse.
- Ask the pt to hold breath in deep inspiration and comment that the JVP decreases with inspiration.
- Ask the pt to sit up and comment that the JVP disappears when sitting upright.

2. By palpation:

- ask for any site of pain in the neck, warm your hands and palpate the visible pulse, comment that the JVP pulse is impalpable.
- Do neck obliteration at the root of the neck and comment that the JVP disappears with neck obliteration.
- Ask for any site of abdominal pain, do the abdomino-jugular reflex. Comment that the JVP increases with abdomino-jugular reflex.

3. Measure: place a ruler vertically at the sternal angle and place any straight object horizontally at the highest point of venous pulsation. Record the height on the ruler and add 5cm to obtain the length of the JVP. It's normally less than 9cm.

4. Auscultate: for venous hum using diaphragm.

Pericardium Examination:

➤ Inspection:

o from the foot of the bed: Ask the patient to take a deep breath and comment:

- Symmetrical movement with respiration.
- Chest deformity
- Breathing pattern

Symmetrical bilateral chest expansion , No chest deformities such as : pectus excavatum or carinatum, Pattern of breathing: 1. Male: Abdominothoracic 2. Female: thoracoabdominal

o Now continue from the Right side:

- Scars (midline sternotomy- CAPG or valve replacement, left submammary- mitral valvotomy, infraclavicular- pacemaker implantation)
- Superficial Hair distribution
- Swelling (masses)
- Gynecomastia (SE of cardiac medications →ACEI, spironolactone, Ca channel blockers, digoxin, and cirrhosis)
- Superficial Distended veins (Vena cava obstruction)
- Visible pulsation : The apex beat (normal site: 5th intercostals space, within the left midclavicular line. In left ventricular dilatation it will be displaced laterally).

- in Young age patient with sternotomy scar , its most probably valve replacement >>> you may hear metallic click
- in old age patient with sternotomy scar >> may be its CABG >> look for scar in his leg (doctor take graft from great saphenous for CABG)

-Graft can be taken from left internal mammary artery (its also called internal thoracic), its better as Graft than great saphenous because :

1. its an artery not vein , and it's the lastest artery in the body that affected by atherosclerosis
2. its connected from one side
3. it can accommodate better with the pressure found in the chest

➤ **Palpation:**

A- Palpate the apex beat (apex beat is the outermost and lower most part of the apical impulse).

✓ **Technique:**

- Put your whole hand flat over the pericardium to obtain a general impression of cardiac activity.
- Locate the apex beat by laying your **fingers** on the chest parallel to the rib spaces.
- If you can't feel it; ask the patient to roll onto the left side.

✓ **Feel for:**

- Site (Normal: 5th ICS, Mid-clavicular line)
- Size (normally 2-3cm, localized)
- Character (normal or tapping (MS) or hyperdynamic (volume overload) or sustained (pressure overload))
- Rate and rhythm.

✓ **Abnormal apex beat:**

1. **Tapping** (small and quick with palpable S1) → occur in mitral stenosis
2. **Thrusting (Heave or Sustained)**, it lifts your hand from the pt chest)) → occurs in pressure overload → LVH → such as in HTN and AS
3. **Hyperdynamic** (unsustained, large, quick) → aortic regurgitation

- ✓ If you couldn't feel the apex then this maybe due to: obesity, lung causes (COPD, pneumothorax , pleural effusion), Heart (pericardial effusion , dextrocardia), the apex beat behind the rib

B- Palpate for: heaves and thrills

Heave:

- Ask the patient to do expiration and hold it , to approximate chest wall to mediastinal structure
- by the heel of the hand palpate at the apex and left parasternal
- If u feel it at the apex – left ventricular hypertrophy.
- If you feel it at the left parasternal area- right ventricular hypertrophy or dilatation.
- Heave maybe the earliest sign of hypertrophy

Thrill:

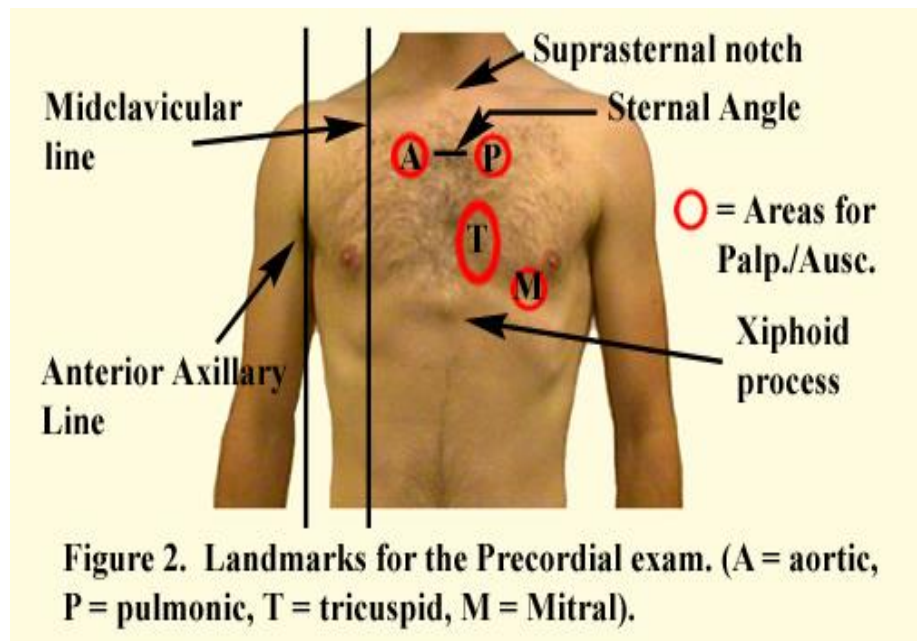
- It is a palpable murmur(grade 4 or more), most common: AS at the apex, VSD at the left sternal edge.
- by the pads of the fingers palpate at the 4th areas of the valves (apex , left lower sternal border ,then 2nd left intercostal space , and finally the 2nd right intercostal space)

➤ Auscultation:

- 1- Put your hand on the pulse (carotid or radial)
- 2- Auscultate the heart, at the 4 areas , while laying

Apex: for mitral valve
Lower sternum: for tricuspid
Left 2nd ICS: pulmonary
Right 2nd ICS: aorta

- Use the diaphragm then
The bell in the 4 areas



- 3- So you start at the apex, by the diaphragm , determine S1 and S2 , systolic and diastolic phases and if there is murmur , then go to the left axilla by the diaphragm: for MR radiation (even if there is no murmur heard , go to radiation sites always)
- 4- Put the diaphragm on the tricuspid area
- 5- Put the diaphragm on the pulmonary area
- 6- Put the diaphragm on the aortic area , then go to radiation site (carotid) (always go to the radiation site even if there is no heard murmur)
- 7- Ask the pt to sit and to hold his breath after expiration and listen at the Right 2nd ICS and lower sternum(A2 area) by the diaphragm for AR (early-diastolic murmur).

- 8- Ask the patient to lay down and repeat the auscultation using the Bell , start from the apex then while the bell on the apex roll the pt onto the left side, for MS (mid-diastolic murmur).
- 9- Use the bell on the other areas (tricuspid , pulmonary and aortic) .
- 10- Note : All heart sounds heard by diaphragm except S3 ,S4 and MS
- 11- Note : Inspiration result in a –ve pressure in the chest and so increases the venous return to the right heart but expiration increases the flow in the left side of the heart and so makes Lt sided heart murmur more obvious) , so inspiration increase the right hear murmurs and expiration increase the left heart murmurs .

➤ **Listen for:**

- S1, S2.
- Splitting.
- Added sounds (S3, S4, midsystolic click).
- Murmurs.

✓ **S1, S2:**

- S1 best heard at the apex, and S2 at the left sternal edge, S2 is louder

✓ **Splitting:**

- **Physiological:** aortic component precedes the pulmonary component, heard only during inspiration, disappear on expiration, and use the *diaphragm*, at the *left sternal edge*.
- **Pathological:**
 - **Wide splitting:** RBBB, pulmonary stenosis, pulmonary hypertension, VSD
 - **Fixed splitting:** occurs during inspiration and expiration (ASD)
 - **Reversed splitting:** AS, hypertrophic CMP, LBBB, and pacemaker.

✓ **Added sounds:**

➤ **S3:**

- Is normal in children, young adult, and pregnancy.
- Caused by rapid ventricular filling.
- Early diastolic sound (just after S2)
- Heard with the bell at the apex
- Abnormal after the age 40, the cause: left ventricular failure, Mitral regurgitation
- Gallop rhythm= S3+ tachycardia

➤ **S4:**

- Is a late diastolic murmur, heard just before S1.
- Always abnormal
- Caused by forceful atrial contraction against non-compliant or stiff ventricle.
- Causes: HTN, AS, LVH

➤ **Murmurs:** systolic, and diastolic

- **Systolic: (heard between S1 and S2)**

1. Ejection systolic:

- Pregnancy, fever, anemia, and innocent murmur.
- AS (all over the pericardium, and there is radiation to the carotids), PS
- ASD
- High pitch- heard with diaphragm

2. Pansystolic:

- MR (at the apex, and there is radiation to the axilla), TR
- VSD (left 2nd ICS) – may radiate to the right side.
- Heard with diaphragm

- **Diastolic murmurs: (heard between S2 and S1)**

1. Early:

- Causes: AR (left or right sternal border), PR (graham steel murmur)
- Heard with diaphragm.

2. Mid-diastolic:

- Causes: MS, TS
- Low pitch (by bell)
- MS: at the apex, and ask the pt to roll onto his left side
- Can be accentuated by exercise

- **Continuous murmur:**

- Cause: PDA
- Site: upper left sternal border
- Character: machinery
- Radiation: left scapula

Note:

- ✓ Multiple murmurs: think about rheumatic fever
- ✓ Rapidly changing murmur: endocarditis
- ✓ Graham steel murmur: pulmonary regurgitation
- ✓ The bell is used for: S3, S4, MS, and breath sounds.
- ✓ Kausmaul sign: with inspiration the JVP raised
- ✓ Pulsus paradoxus: the pulse that increase in volume with expiration, and decrease in inspiration (this is Normal) but in pulsus paradoxus is exaggerated, can assess it by BP in inspiration is less by 15mmHg or more than expiration BP. Cause: acute asthma, cardiac tamponade.
- ✓ Postural Hypotension: standing BP is less by 20mmHg or more than sitting BP. Cause: antihypertensive drugs, neuropathy.

❖ During inspiration:

- HR increases.
- **Pulse volume** (the difference between systolic and diastolic BP) decreases
- JVP decreases.

13. Listen to the back: for basal crackles

14. Examine for sacral edema, assess the depth, and the level (How high)

15. Examine the abdomen for:

- Liver palpation and span
- Ascites :
- ✓ Shifting dullness
- ✓ Fluid thrill

16. Examine lower limbs for edema: press for **5-10 sec** across a bony area (tibia) , assess the depth and the level (How high)

Summary of common conditions seen in OSCEs

	Aortic stenosis	Aortic regurgitation	Mitral stenosis	Mitral regurgitation	Tricuspid regurgitation
Location of murmur (loudest heard)	Aortic area	Aortic area	Mitral area	Mitral area	Tricuspid area
Type of murmur	Ejection systolic Radiating to carotids	End- diastolic	Mid- diastolic	Pan- systolic Radiating to axilla	Systolic
Manoeuvres to enhance murmur	None	Sit forward and expire	Roll on left side and expire	None	None
Pulse	Slow rising	Collapsing Carotid pulsations	Irregular if atrial fibrillation	Normal	Normal
Peripheral features	Narrow pulse pressure Commonly have CABG scar	Quincke's sign Corrigan's pulsation De Musset's sign	Atrial fibrillation on auscultation Right-sided heart failure	None	JVP increased to earlobes
Key management points	Aortic valve replacement if severe Beta-blockers Diuretics Treat heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures	Aortic valve replacement Treat any heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures	Mitral valvotomy Treat atrial fibrillation and heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures Anticoagulate (with warfarin)	Mitral valve replacement Treat any heart failure Antibiotic prophylaxis for gastrointestinal/genitourinary/dental procedures	Tricuspid valvotomy Treat right heart failure

MS	MR
Bell	Diaphragm
Not radiated	Radiated to the axilla
Mid-diastolic murmur	Pansystolic
Opening snap	-----

MR	AS
Pansystolic	Ejection systolic
Diaphragm	Diaphragm
Radiated to the axilla	Radiated to the carotid
-----	Ejection click
Increase with hand grip	Decrease with hand grip

❖ Its important to know indication of severity for each valvular lesion

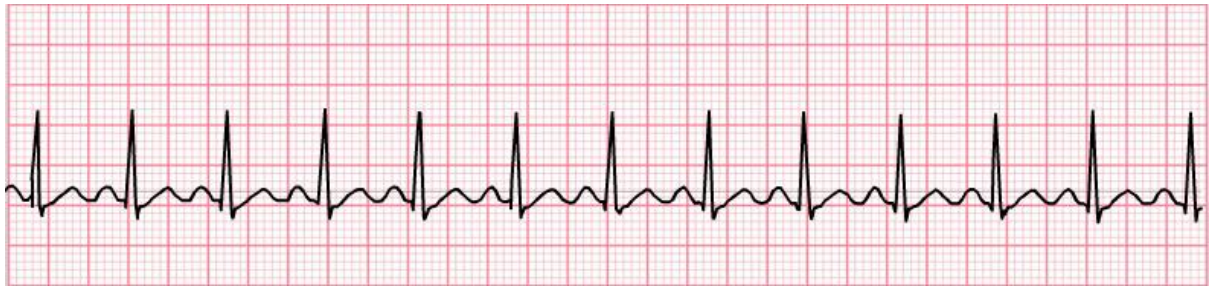
❖ ECG interpretation:

1-STEMI

- ST-elevation in leads **II, III, aVF** → **Inferior** wall MI → Occlusion of **Rt. Coronary artery**.
- ST- elevation in leads **I, aVL, V5, V6** → **Lateral** wall MI → Occlusion of **circumflex artery**.
- ST-elevation in leads **V1,V2,V3,V4** → **Anterior** wall MI → Occlusion of **LAD**.
- ST-depression in leads **V1, V2** → **Posterior** wall MI.

2- Tachycardia (HR >100)

Is it fast?	Is it regular?	QRS (normally ≤3 small squares)	P wave present?	-----	Diagnosis
✓	✓	Narrow	✓		Sinus tachycardia
✓	✓	Narrow	X (Instead there's a retrograde P wave in V1)		SVT
✓	X	Narrow	X		A. Fib
✓	✓	Narrow	✓✓✓ موجودة وبقوة (more than one P wave for each QRS complex)		A. Flutter
✓	X	Narrow	✓ and each P wave has a distinct morphology		MAT
✓	✓	Wide	X	Looks good	V. Tach
✓	X	Wide	X	Looks like a total mess	V. Fib



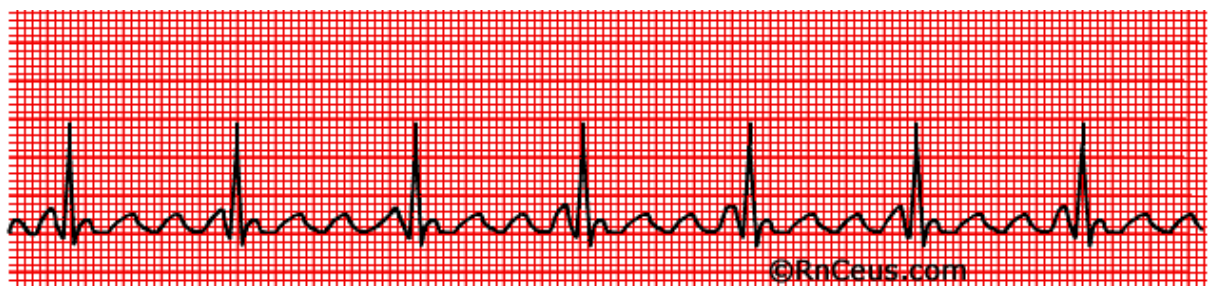
Sinus Tachycardia



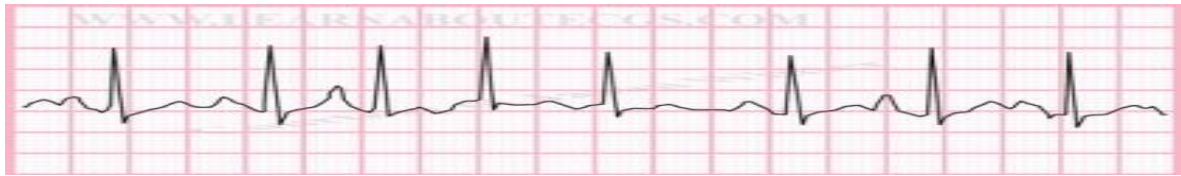
SVT



A-Fib



A-Flutter



MAT



V-Tach



V-Fib

3- Bradycardia (HR<60)

Is it slow?	Look at the PR intervals	Are there dropped beats?	Dx
✓	They're of normal length (< 5 small squares) and they're fixed	X	Sinus bradycardia
✓	They're elongated (>5 small squares) but fixed	X	1 st degree AV block
✓	There's progressive prolongation of PR intervals then a dropped beat	✓	2 nd degree AV block type 1 (Wenckebach block)
✓	PR intervals are of normal length and fixed then a dropped beat	✓	2 nd degree AV block type 2 (Mobitz type)
✓	No relation between PR intervals	✓✓✓ (A LOT)	3 rd degree AV block (complete heart block)



Sinus Bradycardia



1st Degree AV Block



2nd degree AV block type 1 (Wenckebach block)



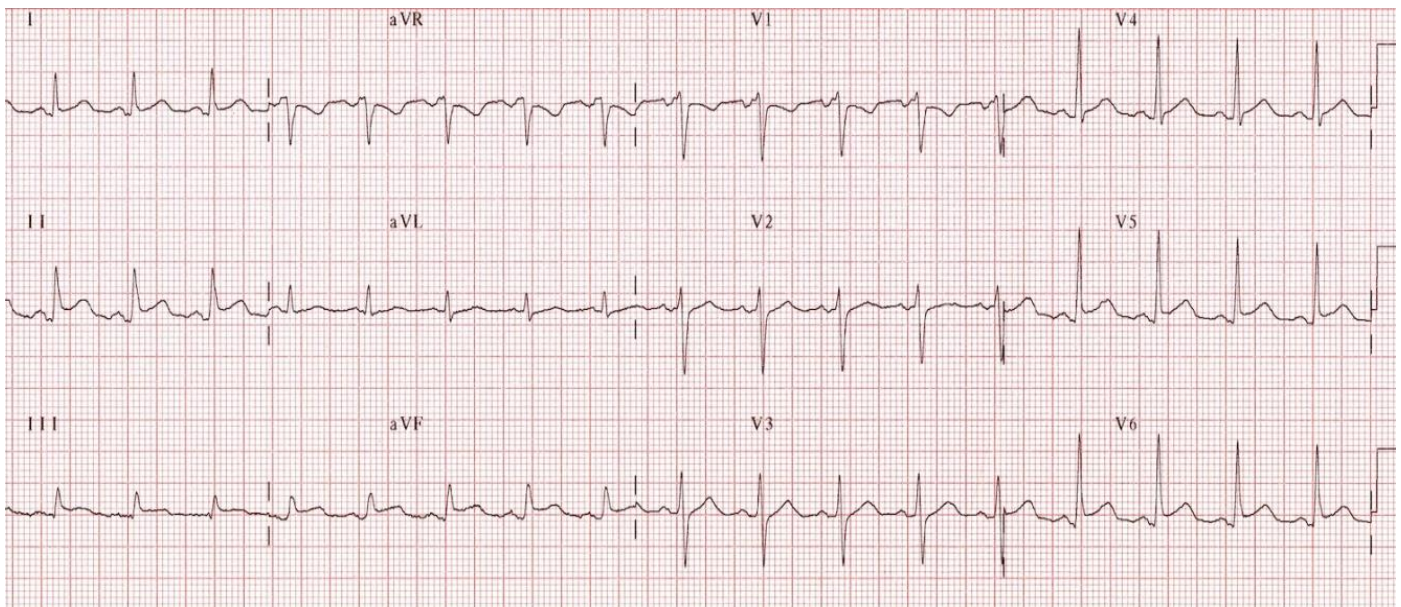
3rd degree AV block (complete heart block)



2nd degree AV block type 2 (Mobitz type)

4- Acute Pericarditis

Diffuse ST-elevation in all leads except leads V1 and aVR (these have ST depression), with PR depression in all leads except aVR (it has PR elevation).



Acute Pericarditis

5- Axis deviation

Always look at Leads I and aVF		Dx
Lead I	Lead aVF	
+ve	+ve	Normal axis
-ve	+ve	Rt. Axis deviation
+ve	-ve	Lt. Axis deviation
-ve	-ve	Severe axis deviation mostly to the right



Checklists

Question: Examine JVP	
Steps (total 26)	Check if done
Introduce himself/herself	☐
Ask for permission to do examination	☐
Comment on the room settings	☐ privacy ☐ warmth ☐ adequate light
Comment on hand hygiene	☐
Position the Pt in semi-recumbent (45°)	☐
ask pt to slightly turn his head to the Lt side	☐
Stand at the right side of the pt	☐
Inspect the neck for visible pulsation	☐
Comment on the visible pulse; 2 pulses were visible an outward single peaked arterial pulse and an inward double waved venous pulse.	☐
Ask pt to hold his breath at deep inspiration	☐
Comment that JVP decreases on inspiration	☐
Ask the pt to sit up, while he observe the effect on the pulse	☐
Comment that JVP disappears on sitting upright	☐
Ask for any site of pain in the neck before palpation	☐
Warm his hands before palpation	☐
Palpate the visible pulse	☐
Comment that JVP pulse is impalpable	☐
Do neck obliteration test	☐
Comment that JVP disappears on neck obliteration	☐
Warn the pt that he's going to push his abdomen and ask if the abdomen is tender	☐
Perform abdomino-jagular reflex	☐
Comment that JVP increases with abdomino-jagular reflex	☐
Comment on the need to measure JVP	☐
Auscultate the neck for venous hum	☐

Question: Do Inspection and Palpation of the Precordium:	
Steps (total 28)	Check if done
Introduce himself/herself	☐
Ask for permission to do examination	☐
Comment on the room settings	☐ privacy ☐ warmth ☐ adequate light
Comment on hand hygiene	☐
Position the Pt in semi-recumbent (45°)	☐
Exposure of anterior chest to the umbilicus	☐
Stand at the foot of the bed	☐
At the foot of the bed comment on chest deformities and body hair distribution	☐ chest deformities ☐ body hair distribution
Stand at the right side of the pt	☐
At the right side of the pt inspect for scars	☐
Look at the axilla at the left of the pt for scars	☐
At the right side comment on visible pulsation (location)	☐
Warm his hands	☐
Ask for any tender area on the chest	☐
Maintain eye-to-eye contact throughout palpation	☐
Comment on chest tenderness	☐
Locate the apex beat	☐
Comment on the apex beat;	☐ gently tapping ☐ location
Ask the pt to hold his breath at expiration after a deep inspiration for palpation of heaves	☐
Palpate for left ventricular heave at the apex using palm	☐
Palpate for right ventricular heaves at the left sternal margin	☐
Palpate for thrills using the pulps of your fingers at apex and both sides of sternum	☐ at apex ☐ at right side of sternum ☐ at left side of sternum

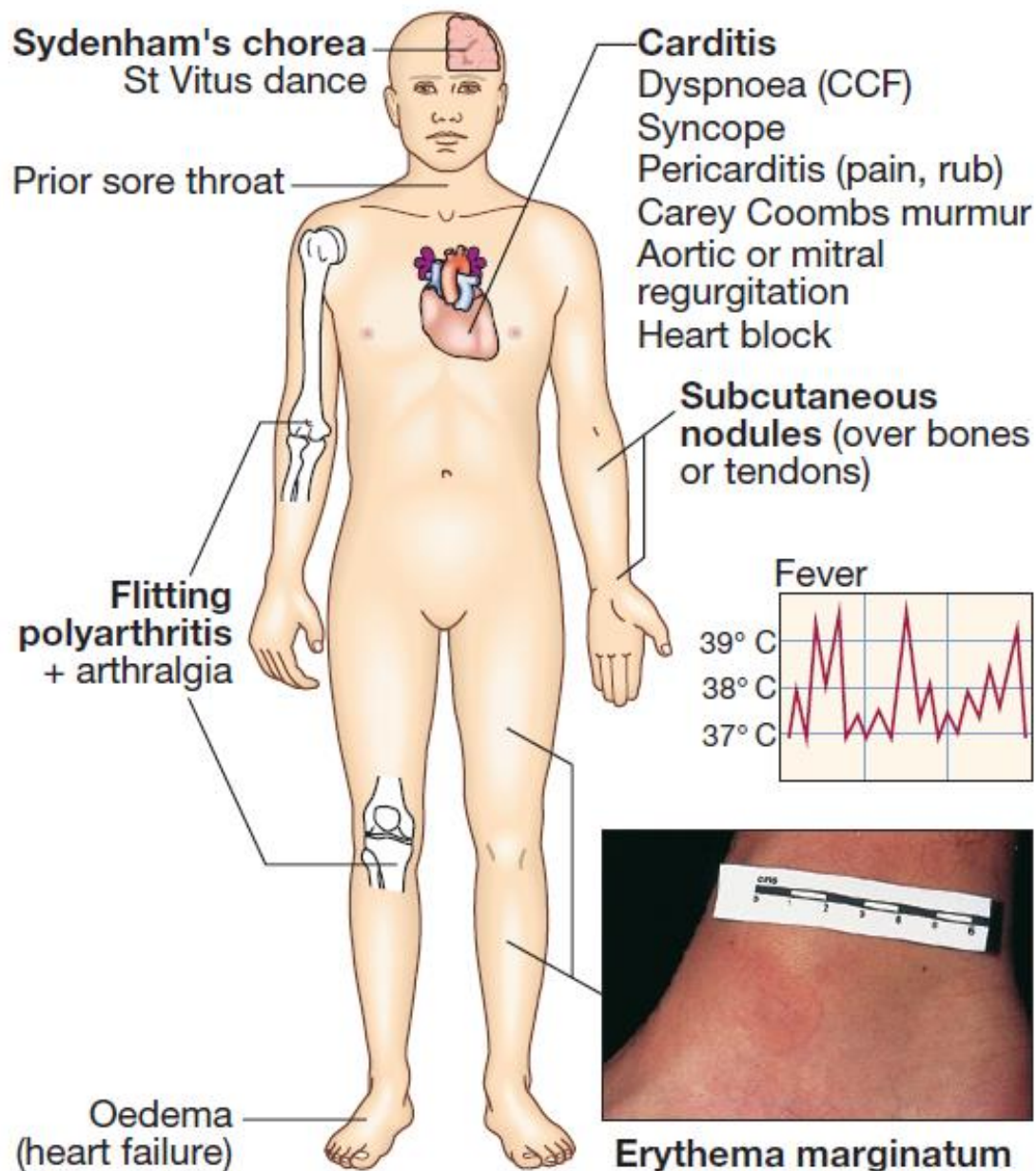


Fig. 18.86 Clinical features of rheumatic fever. Bold labels indicate Jones major criteria (CCF = congestive cardiac failure). *Inset (Erythema marginatum)* From Savin et al. 1997 – see p. 641.

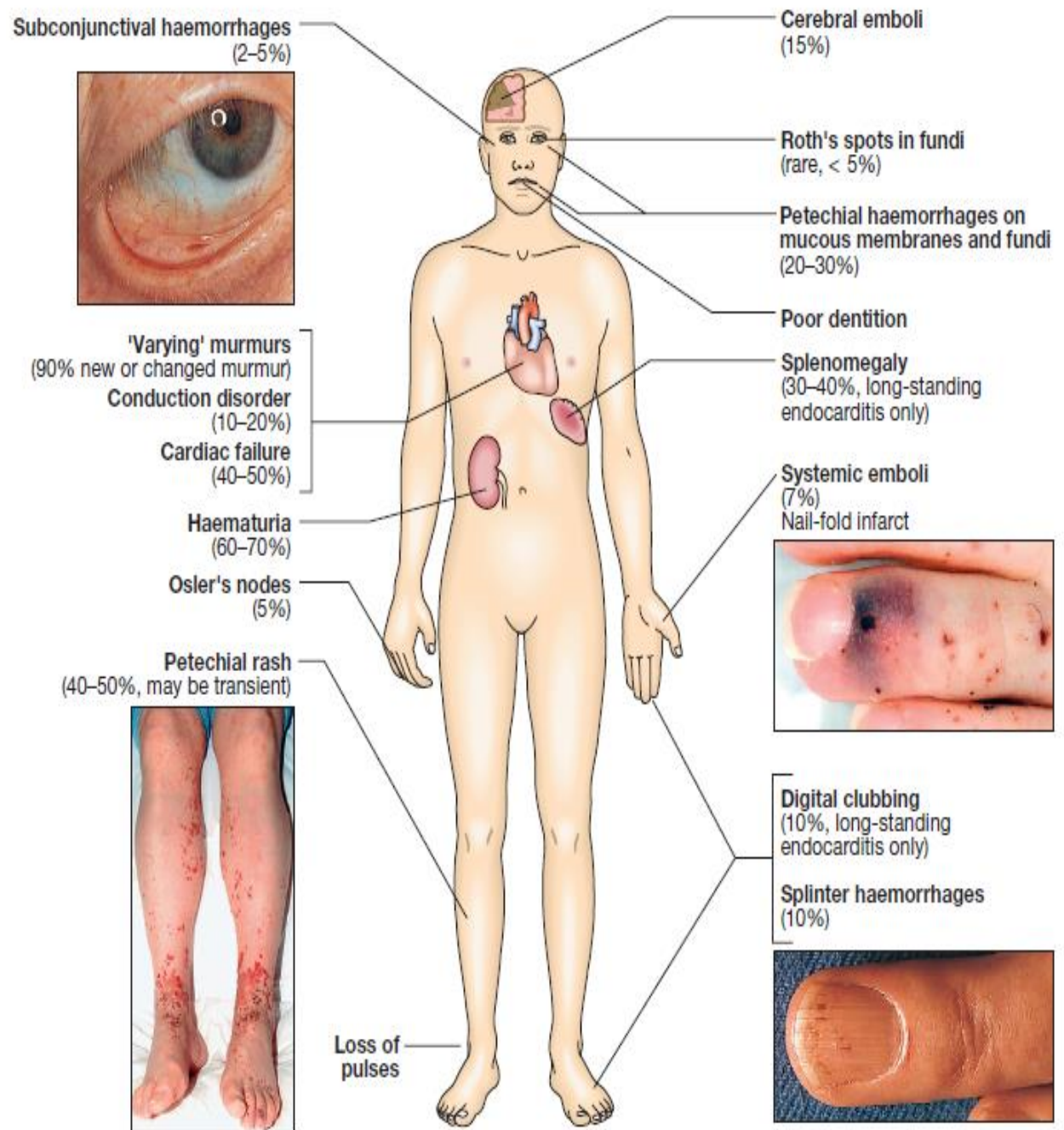


Fig. 18.93 Clinical features which may be present in endocarditis. *Insets (Petechial rash, nail-fold infarct)* From Newby and Grubb 2005 – see p. 641.

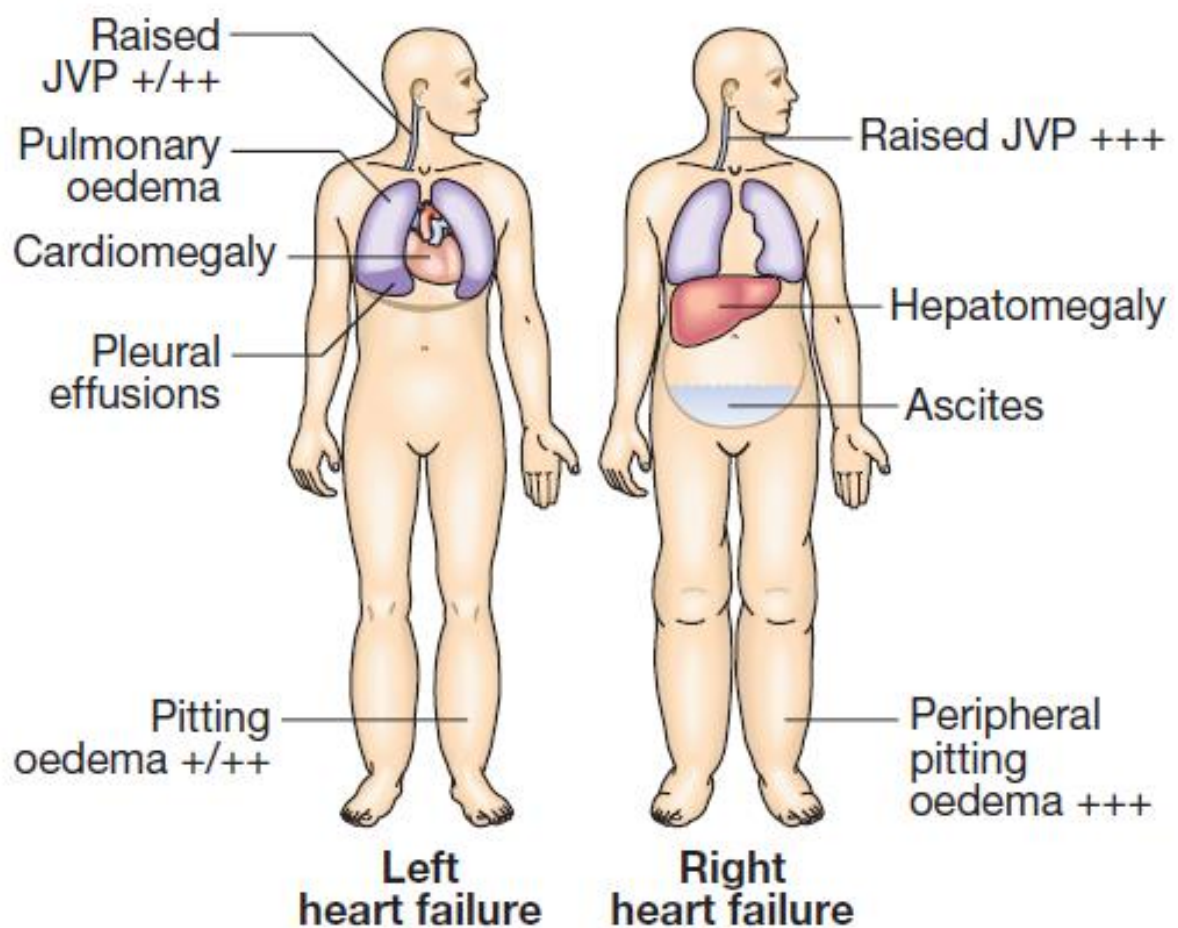


Fig. 18.24 Clinical features of left and right heart failure.
(JVP = jugular venous pressure)

Respiratory System

Hatem Rjoob

Chapter 2: Respiratory System



History Taking

Cough; productive, non-productive & hemoptysis

-X year old male pt presented with a history of cough of X days duration, take a good history and list the differential diagnosis.

∞ Differential diagnosis:

- Acute cough (<3 weeks): **foreign body aspiration**, **upper respiratory tract infection** (yellow sputum), **Lower respiratory tract infections** (pneumonia, TB) (purulent sputum, can be blood stained).
- Chronic cough (>8 weeks):
Productive: COPD (in adult only, clear mucoid sputum, purulent if active infection is present), **TB** (blood stained, where TB is endemic), **bronchiectasis** (purulent sputum), **pulmonary edema - HF** (pink watery sputum), **lung CA** (blood stained sputum), **pulmonary embolism** (frothy pink sputum), **cystic fibrosis** (purulent sputum, in children), **lung abscess** (purulent sputum).
Non productive: asthma, post nasal drip, GERD, drugs (ACEI), sarcoidosis, and neuromuscular disease.
- 3-8 weeks is called subacute or acute persistent

∞ History

- » Knock the door, introduce yourself, take permission and start taking patient profile (age, occupation, admission details). Listen to the case carefully.
- » **Chief complaint: cough and Duration:** to know if acute or chronic

❖ **Clarify the chief complaint :**

- » **Productive or non productive (sputum):** if productive ask about :
 - ✓ **The consistency of the sputum, is it easy to evacuate or hard mass?**
 - ✓ **Amount:**
large volume (**bronchiectasis**), large volume on single occasion (**lung abscess rupture**), large volume of watery sputum (**pulmonary edema, if pink: bronchioalveolar ca**), mucous plug (**asthma**).

- ✓ **Taste or smell:** foul (infection, bronchiectasis, abscess).
- ✓ **Solids:** foreign body, aspergillosis
- ✓ **color:**

7.3 Types of sputum		
Type	Appearance	Cause
Serous	Clear, watery	Acute pulmonary oedema
	Frothy, pink	Alveolar cell cancer
Mucoid	Clear, grey	Chronic bronchitis/chronic obstructive pulmonary disease
	White, viscid	Asthma
Purulent	Yellow	Acute bronchopulmonary infection
		Asthma (eosinophils)
	Green	Longer-standing infection
		Pneumonia
		Bronchiectasis
		Cystic fibrosis
		Lung abscess
Rusty	Rusty red	Pneumococcal pneumonia

- » **Special sounds with cough** (does it have a special sound?) :
bovine cough (left recurrent laryngeal paralysis or invasion),
violent repetitive cough in children (foreign body),
painful parking cough (laryngeal infection, inflammation),
- » **Timing:** nocturnal cough (asthma), less in holidays (occupational asthma), during a drug course (dry cough due to ACEI), cough during swallowing (neuromuscular abnormality).
- » **Any preceding event:** runny nose and fever/chills (infection), sore throat 2 weeks ago then resolved (reactivation), exercise (exercise induced asthma)
- » **Does it change or increase with specific conditions:** recumbent posture (COPD,GERD), temperature (bronchiectasis) , after exercise or exposure to allergens , irritants (asthma).
- » moist cough (bronchiectasis), smoker's moist cough at morning (chronic bronchitis), paroxysmal dry cough (bronchial hyperactivity after viral infection with asthma).
- » How severe is it ?! Ask if the patient experience vomiting, fatigue or dizziness after cough?!
- » Any similar previous attack ?
- » **Noisy breathing:**
Stridor (infection/inflammation, e.g. acute epiglottitis, tumors of the trachea and main bronchi, extrinsic compression by lymph nodes, anaphylaxis and foreign body),
wheezy breathing (mainly asthma and COPD).

- » **Associated symptoms:** feeling of hotness, chills ,rigor, fatigue, dyspnea, chest pain, hemoptysis, anorexia , weight loss

➔ **Red flag symptoms:** (important)

• Hemoptysis • Breathlessness • Fever • Chest pain • Weight loss

➔ pleuritic chest pain: sharp, stabbing, non-central chest pain, increased by inspiration and coughing, feature of PE, pneumonia, pneumothorax.

*** since it's a respiratory case then the smoking is very important , ask about active and passive smoking ? pack yeas ? make sure that the patient understand that water pipes also involved in smoking?

*** if you are suspecting asthma , it's very important to ask about family history , atopy, allergy ?

*** if you suspect malignancy it is good to ask about metz symptoms to the liver, brain , bone and adrenal .also try to ask about paraneoplastic syndrome symptoms such as hypercalcemia symptoms (bones, stones, groans and mental overtone) , ask about hyponatremia symptoms due to SIADH , Cushing syndrome ,,etc.

❖ **Review of system**

- » Review the systems focusing on GI symptoms : abdominal pain (can be seen in lower lobe pneumonia), nausea, vomiting, diarrhea, anorexia..

❖ **Past medical and surgical history**

- » Previous similar attack (asthmatic attacks) , other lung disease, heart diseases or heart failure (pulmonary HTN), allergy, GERD, chronic sinusitis (bronchiectasis), DVT with PE.

❖ **Drug history**

- » ACEI, NSAIDs, B-blockers...

❖ **Family history**

- » Asthma, eczema and hay fever (atopy), Cystic fibrosis is (AR), α 1- antitrypsin deficiency (AR), other lung diseases in the family.

7.13 Examples of drug-induced respiratory conditions	
Respiratory condition	Drug
Bronchoconstriction	Beta-blockers Opioids NSAIDs
Cough	Angiotensin-converting enzyme inhibitors
Bronchiolitis obliterans	Penicillamine
Diffuse parenchymal lung disease	Cytotoxic agents: bleomycin, methotrexate Anti-inflammatory agents: sulfasalazine, penicillamine, gold salts, aspirin Cardiovascular drugs: amiodarone, hydralazine Antibiotics: nitrofurantoin Intravenous drug misuse Radiation
Pulmonary thromboembolism	Oestrogens
Pulmonary hypertension	Oestrogens Dexfenfluramine, fenfluramine
Pleural effusion	Amiodarone Nitrofurantoin Phenytoin Methotrexate Pergolide
Respiratory depression	Opioids Benzodiazepines

❖ **Social history**

- » Smoking with pack years (COPD), alcohol (neuromuscular), occupation (occupational asthma), recent travel (TB), pets (allergy, asthma).
- ❖ If the pt has **hemoptysis** as chief complaint (not as a color of sputum only),ask about the following:
 - ✓ Make sure that it is hemoptysis not hematemesis?!
 - ✓ Is the blood fresh or mixed with sputum ?!
 - ✓ Amount and appearance: large (lung CA, bronchiectasis, TB), blood clots (lung ca)
 - ✓ Frequency: intermittent (bronchiectasis), daily for a week (CA,TB, lung, abscess), sudden single episode (PE and infarction) associated with hematuria (Goodpasture's syndrome, *Wegener's* granulomatosis
 - ✓ Ask the patient if there's bleeding elsewhere from other body orifices (bleeding tendency), if he's taking any antiplatelet or anticoagulant, or if there's family history of recurrent bleeding.



Physical Examination

Examination of the Respiratory System

Examination:

- 1- Introduce yourself, take permission, insure privacy, warmth and light, make sure of good exposure and position
- 2- exposure from the neck to the umbilicus.
- 3- Put the patient at 45 degree.

1- General appearance:

Stand at the end of the bed and look for:

- Is the patient conscious, cooperative, or confused?
- Is the patient look well or ill?
- Oxygen mask or ventilator.
- Comfortable or distressed.
- Use the accessory muscles (sternocleidomastoid, platysma, pectoral muscles) → elevation of shoulders > **Characteristic of COPD.**
- Body mass (obese, cachexic).
- **Tachypnea (RR > 15/min).**

Causes:

1. Increased ventilatory drive (fever, asthma, COPD).
2. Reduced ventilatory capacity (pneumonia, pulmonary edema, interstitial lung dz)

((RR > 30 → the most important prognostic sign associated with death in pneumonia))

- ✓ Slow RR occurs in (opioid toxicity, hypothyroidism, increased ICP, hypothalamic lesions, hypercapnia).
- Special breathing patterns:
 - ✓ Cheyne-Stokes breathing (periods of increasing rate and depth of breathing followed by diminishing respiratory effort and rate, usually ending in a period of apnea → seen in pt with **stroke in the Brain Stem, severe cardiac failure**, and may be **normal in elderly during sleep**).
 - ✓ Kussmaul respiration (hyperventilation with deep sighing respiration) a response to reduced arterial PH in metabolic acidosis → occur in ARF, lactic acidosis, DKA, Salicylate and methanol poisoning.

- Cough.
 - Cyanosis is an abnormal blue discoloration of the skin and mucous membrane
DD: hypoxemia, methemoglobinemia, sulphemoglobinemia.
 - * Hypoxemia: defined as **an absolute concentration of deoxygenated Hb of >50g/l**
 - ✓ In pt with normal Hb, central cyanosis is detected when the O₂ sat is below 90% (Pa O₂<60mmHg).
 - ✓ In anemic or hypovolemic pt cyanosis rarely seen because severe hypoxia is needed.
 - ✓ In polycythemic pt cyanosis occurs at a higher saturation.
 - ✓ Look carefully at the tongue and the lips to see central cyanosis.
-
- Hoarseness, vary from dysphonia to aphonia (causes: usually viral infection, invasion of the left recurrent laryngeal nerve at the hilum in lung cancer).
 - Stridor, a harsh and rasping noise on inspiration (causes are foreign body or tumor occluding the URT, larynx, trachea or main bronchus),, if **u suspect ask the pt to cough and then breathe deeply in and out with the mouth open.**
-
- 2- Examine **both hands** together, inspect the dorsal then palmar aspect of both hands, and look for: (Cyanosis , Clubbing , Temperature , Tobacco , Tremor)
- Cyanosis (Heart failure due to decrease CO and vasoconstriction/ COPD)
 - **Yellow-nail syndrome** is associated with lymphedema and an exudative pleural effusion.



Fig. 7.9 Yellow nail syndrome.

- Clubbing : It rarely develops quickly over several weeks with empyema., If tenderness of the wrist is also present, suspect hypertrophic pulmonary osteoarthropathy (squamous cell cancer).

Hypertrophic pulmonary osteoarthropathy (almost always associated with lung cancer, usually squamous cancer, x-ray shows subperiosteal new bone formation, ALK phosphatase is raised)

- Tobacco (tar) staining
- Temperature (warm in COPD)
- Hand (Hot /cold ,, sweaty /dry)
- Tremor :
 1. Fine tremor (seen in patients taking β -agonist or theophylline bronchodilator inhalers).
 2. Flapping tremor (Asterixis): Ask the patient to hyperextend his wrists and abduct his fingers for 30 seconds and observe for flapping tremor. Seen in cases of CO2 retention and severe ventilator failure in end organ damage, other causes: **liver failure, advance renal failure, acute thalamic lesion**. Unilateral asterixis is due to a structural abnormality in the contralateral cerebral hemisphere. Patients with a flapping tremor cannot maintain their grip.

- **Note: the causes of CLUBBING are:**

Cyanotic heart disease

Lung diseases (CA, fibrosis, restrictive diseases)

Ulcerative colitis

Biliary atresia

Birth defect (familial)

Infective endocarditis

Neoplasm

GI malabsorption (e.g: celiac disease)

Rarely can develop over several weeks of life as in **empyema**

- If the underlying cause is successfully treated clubbing may resolve.

3. **Measure the BP.**

- Hypotension (diastolic BP < 60 is associated with increased mortality in pneumonia)
- In pneumothorax hypotension may indicate the development of tension pneumothorax.
- Hypotension may be a feature of life-threatening asthma.



4. **Inspect the face for:**

- Horner syndrome (ipsilateral ptosis, miosis, and anhidrosis, enophthalmus may occur), occur with apical lung cancer (pancost) invading the sympathetic chain.
- Iridocyclitis (in TB and sarcoidosis).
- Sclera and conjunctiva for pallor and jaundice. Jaundice is first seen in the mucosa beneath the tongue; it results from a congested liver failure due to a chronic pulmonary disease.
- Pallor is seen from anemia.
- Chemosis, conjunctival and retinal vein dilatation. (In CO₂ retention, in SVC obstruction).
- Central cyanosis (lips, and under tongue).

5. **Neck:**

- Scars.
- Vein engorgements.
- Lymph nodes (a palpable supraclavicular node strongly suggests metastatic spread of lung cancer; localized cervical lymphadenopathy is a common presenting feature of lymphoma).
- Pamperton's sign >>> ask the patient to raise both hands and observe for Facial flushing, distension of neck veins and stridor ; positive in SVC obstruction
- JVP.

➤ **Inspect the JVP:**

- Anatomy: the internal jugular vein runs between the two ends of sternocleidomastoid and goes above toward the ear.
- Why important: It is important because it reflects the pressure in the right atrium because there is no valve in the superior vena cava.
- Position: You should examine it at 45 degree, in the normal person when he is in the supine position the vein will distend and no pulsation will be seen,

In the sitting position the pulse is hidden behind the clavicle and sternum, at 45 degree it will be at the level of the clavicle.

- Normal JVP is less than 4 cm above the sternal angle. (Normal atrial pressure is less than 9 cm).



❖ JVP raised in:

- Cor pulmonale (e.g: due to COPD).
- Tension pneumothorax.
- Severe acute asthma.
- Major pulmonary embolism.
- Superior vena cava obstruction (non-Pulsatile) caused by: lung cancer, lymphoma, thymoma, and mediastinal fibrosis.
- Constrictive pericarditis caused by TB or RA. (With Kussmaul sign).

-PULSATILE : REFLECT RIGHT ATRIAL PRESSURE

-NON-PULSATILE : NOT REFLECT RIGHT ATRIAL PRESSURE

➤ Neck lymph nodes:

- Groups: Submental, submandibular, Preauricular, postauricular, upper cervical, middle cervical, lower cervical, posterior triangle, pretracheal, supraclavicular.
 - Scalene lymph node enlargement may be the first evidence of metastatic lung cancer.
 - Localized LN is a common presenting feature of lymphoma.
 - To palpate the scalene LN: place your index finger between the clavicle and sternocleidomastoid muscle and press down toward the first rib; you may feel a soft mobile mass just above the hard first rib.
 - Note the palpable LN: number, size, consistency, mobile or fixed.
-

6. Examination of the thorax

(Expose the chest and the upper abdomen fully)

➤ **Inspection:** look for

(6S and Hair distribution)

- Symmetry (Chest symmetry , movement with respiration ,Chest deformity)
- Scars
- Swellings or Visible masses
- Superficial Dilated veins. (SVC obstruction or coarctation of aorta)
- Skin changes (Spider nevi ,Marks and spots on the skin
- Subcutaneous lesions (metastatic nodules, lipoma, and neurofibroma).
- Hair distribution
- Localized indrawing of the chest wall (**paradoxical movement**) seen in pt with double fracture of series of ribs, called **flail chest**.
- Look if there's paradoxical inward movement of the abdomen during inspiration which indicate **diaphragmatic paralysis** or **severe COPD**.

-Asymmetry (bulge or retraction) :to know bulge or retraction , then ask the patient to breath from his mouth , the part moves with respiration is normal , and the other part is abnormal .

-Superficial dilated vein >> due to SVC obstruction >> raised JVP

-Causes of SVC : intraluminal(thrombosis) and extraluminal (retrosternal goiter , Pancoast tumor)

- thrombosis in SVC : its unusual site for thrombosis and may occur due to thrombophilia , vasculitis, malignancy

Abnormalities in the shape of the chest:

1. Increase in the anteroposterior compared with the lateral diameter → called **barrel chest** , seen in severe COPD (the degree of deformity doesn't correlate with the severity of dz)

NORMALLY (A/P)/TRANSVERSE = 5/8 , IN BARREL CHEST ITS HIGHER

2. Kyphosis and scoliosis

→ may be **idiopathic** or secondary to childhood **poliomyelitis** or **spinal TB**

- i. Kyphosis: exaggerated anterior curvature of the spine
- ii. scoliosis: lateral curvature
- iii. kyphoscoliosis.
- iv. Severe kyphoscoliosis may cause reduced ventilatory capacity and pt develop ventilatory failure with CO₂ retention and cor pulmonale at early stage.

3. **Pectus carinatum (pigeon chest):** localized prominence of the sternum and adjacent costal cartilage with indrawing of the ribs to form horizontal grooves called Harrison sulci

→ Result from **lung hyperinflation** while the bony thorax still pliable such as in poorly controlled asthma, **osteomalacia** and **rickets**.

4. **Pectus excavatum (funnel chest):** developmental deformity with localized depression of the lower end of sternum, pt usually asymptomatic, in severe cases the heart is displaced and the ventilatory capacity is reduced.

➤ Palpation:

✓ *ASK THE PATIENT FOR ANY PAIN*

✓ *Position of mediastinum → by examination of **trachea** and **cardiac apex beat** , Tracheae for upper mediastinum and apex beat for lower mediastinum*

- 1- For the **Trachea** from in front the pt place the tip of your right index finger into the suprasternal notch and press gently against the trachea / slight deviation to the right is common in normal people.

- Deviation of trachea occurs in upper mediastinal shift

Toward the side of lesion	Away from the lesion
Upper lobe collapse	Tension pneumothorax
Upper lobe fibrosis	Massive pleural effusion
Pneumonectomy	Tumor

2- Distance b/w the suprasternal notch and cricoid cartilage:

- Normally is 3 to 4 fingers, if reduced suggest lung hyperinflation.
- A tracheal tug : can be found in **severe hyperinflation** where fingers resting on the trachea move inferiorly with each inspiration.

3- Apex beat:

- Palpate the trachea to confirm that the heart is not deviated (especially if the trachea is deviated to the left)
- Deviation of the apex indicates shift in the lower mediastinum.
- Deviation of the apex without trachea occurs in LV dilatation, scoliosis, kyphoscoliosis, and severe pectus excavatum

4- Chest expansion:

- Upper lobes assessed by observation of the clavicles from behind.
- Lower lobes assessed by placing the hands firmly on the chest with the fingers extending around the side of the chest, the thumbs should almost meet in the midline and should be slightly lifted off the chest so they are free to move with respiration
- Ask the pt to take deep breath → the thumbs should move **symmetrically** apart at **least 5 cm. (if < 2 cm then the lung is restricted)**

Reduced expansion unilaterally	Bilaterally
Pleural effusion (one side)	Bronchiectasis, Asthma ,Advanced COPD
Pneumothorax	Diffused pulmonary fibrosis
Lung or lobar collapse	Obesity
Unilateral fibrosis	ILD
	Res muscle paralysis
	Ankylosing spondylitis
	Pleurisy

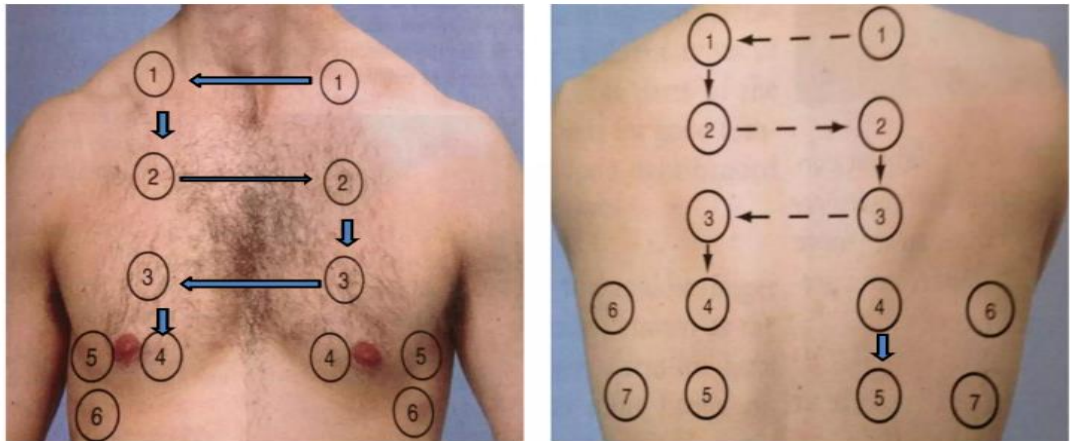


5- **Tenderness** over the costal cartilage found in costochondritis of Tietze's syndrome, also in rib fracture, and areas over pulmonary infarction.

6- **Subcutaneous emphysema** → crackling sensation over gas containing tissue can be a result of severe acute asthma, ruptured esophagus, pneumothorax.

- 7- **Tactile vocal fremitus (TVF)**, anteriorly and posteriorly (ask the patient to say 44).
 If decreased: effusion or collapse.
 If increased: consolidation , fibrosis , top of pleural effusion

Tactile Vocal fremitus (TVF)



Decrease TVF Decrease vocal resonance	Increase TVF , increase vocal resonance causes bronchial breathing
Pleural effusion	Consolidation
Pneumothorax , COPD(??)	Cavitation (large)
Lung collapse with complete obstruction	Lung collapse with partial obstruction
Mass lesion (hydatid cyst , bronchial CA)	Fibrosis
	Top of pleural effusion

➤ **From posterior, palpate for:**

- Tenderness.
- Emphysema.
- Chest expansion : 3 sites from front (supra-mammary , mammary line and costal cartilage)and 3 sites from behind (shoulder , scapula , below scapula angel)

Normal chest expansion (3-5) , if less than 2 then its restricted

➤ **Percussion**

Performed in sequence over equivalent areas on the two sides of the chest



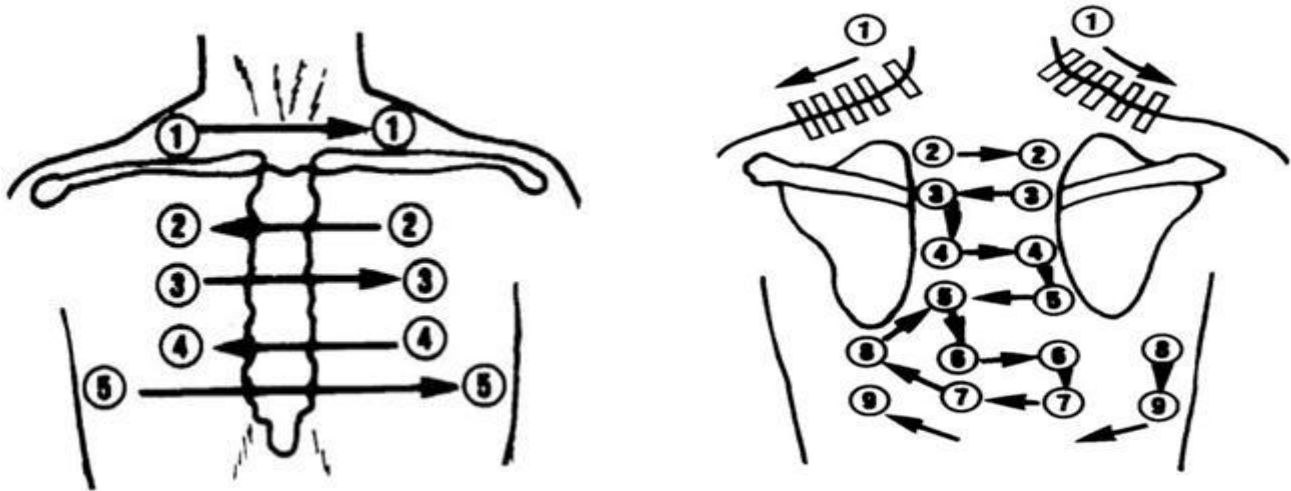
From posterior:

1. Percuss the lung apices by placing the left middle finger across the anterior border of the trapezius overlapping the supraclavicular fossa.
2. Percuss the clavicle directly within its medial third.
3. During percussion of the upper posterior chest move the scapula laterally by asking the pt to fold their arms across the front of their chest.
4. Avoid percussion near the midline as this produce dullness from solid structures of spine and muscles.

From anterior:

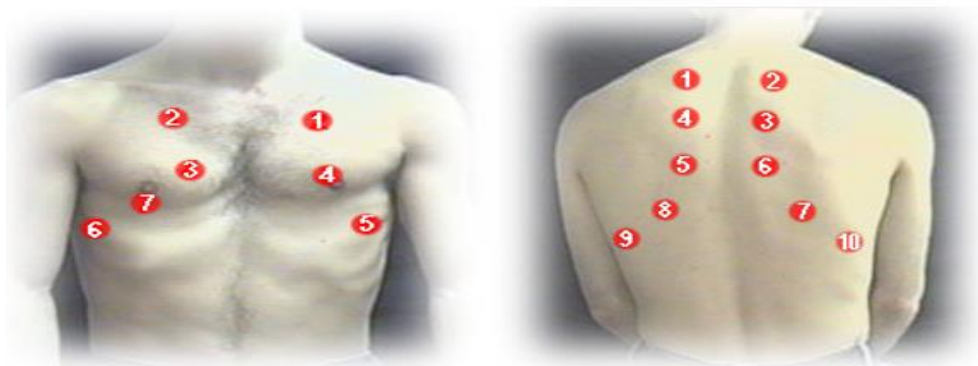
- 1- Percussion over solid structure like heart, liver or consolidation area is dull.
- 2- Normally the upper level of the liver dullness is at the level of the 5th rib in the right midclavicular line, resonance below this indicates a hyperinflated lung.
- 3- The area of cardiac dullness present on the left anterior chest.

Type of percussion note	Detected over
Resonant	Normal lung
Hyperresonant	Pneumothorax , obstructive lung disease (bronchiectasis , asthma , COPD, emphysema)
Dull	Pulmonary consolidation
	Pulmonary collapse
	Severe pulmonary fibrosis
Stony dull	Pleural effusion
	Hemothorax



➤ **Auscultation**

- ✓ Start from back (because most pathologies are in the posterior)
 - Ask the patient to cross his arms, to turn the scapula outward.
 - Start at the clavicles.
 - Auscultate both sides alternately, avoid the midline.
 - Down to the level of 11th rib
 - Identify the breath sound, and if there is any gap between inspiration and expiration.
 - Listen for any added sounds (wheeze, crackles)
 - Assess the **vocal resonance** by asking the patient to say one, one, one.
- ✓ Then from the front:
 - From above the clavicle down to the 6th rib
 - And laterally from axilla to the 8th rib
 - Assess as the above.

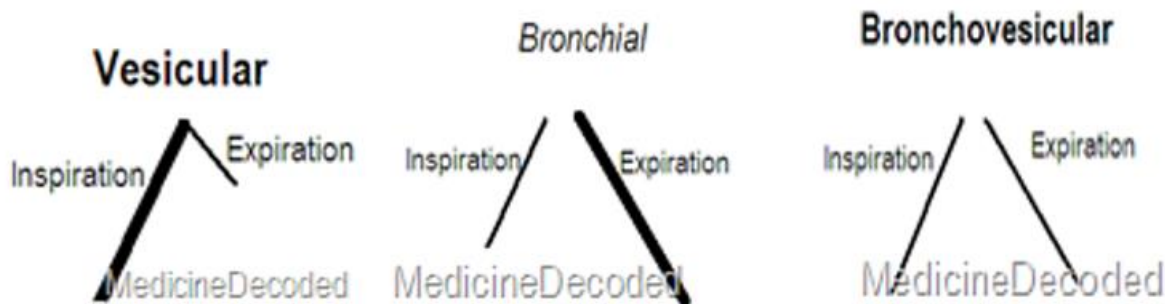


Normal breath sounds:

- Use the bell because most sound reach the chest wall are low frequency.
- **Vesicular** (low-pitched, soft intensity sound, inspiration is more intense and has longer duration than expiration, there is no gap).
- **Tracheal**: only over the trachea.
- **Bronchial**: over the large airways (heard over the sternum)
- **Bronchovesicular**: heard in the posterior chest between scapula, and in the first and the second intercostals spaces anteriorly.

Abnormal breath sounds:

- **Bronchial** (expiration more intense and has longer duration than inspiration, and there is gap).
- Causes: **consolidation, fibrosis, collapse**, and at the top of pleural **effusion**. > All if the underlying major bronchus is patent.
- The presence of bronchial breathing, exclude the possibility of an obstructing lung cancer.
- **Bronchovesicular**: suspect that air-filled lung has been replaced by **fluid-filled** or **solid** lung tissue.



7.27 Causes of bronchial breath sounds

Common

- Lung consolidation (pneumonia)

Uncommon

- Localised pulmonary fibrosis
- At the top of a pleural effusion
- Collapsed lung (where the underlying major bronchus is patent)

Diminish breath sounds:

- Causes: obesity, pleural effusion, marked pleural thickening, pneumothorax, hyperinflation, collapse, obstruction.
- If the breath sounds are diminished, **ask the patient to cough**, if there is obstruction by secretions, the breath sounds will become more audible after coughing.

7.22 Causes of diminished vesicular breathing	
Reduced conduction	
• Obesity/thick chest wall	• Pneumothorax
• Pleural effusion or thickening	
Reduced airflow	
• Generalised, e.g. COPD	• Localised, e.g. collapsed lung due to occluding lung cancer

- Consolidation , fibrosis and top of pleural effusion >>> increase TVF , increase vocal resonance , dullness , make a bronchial breathing

Aegophony:

- Transmitted nasal sounds heard over **consolidation** lung or at the upper level of pleural **effusion**.

Added sounds:

- Crackles: bubbling sounds occur during the passage of air through fluid in alveoli
- Non- musical sounds.
- Pathology: collapsing of peripheral airways on expiration, so with high **inspiratory** pressure air enters rapidly and cause abrupt opening of alveoli and small bronchi.
- Causes:
 - ❖ Early inspiratory: small airway disease, or bronchiolitis
 - ❖ Mid inspiration: pulmonary edema
 - ❖ Late inspiration: pulmonary fibrosis, bronchial secretion (COPD, pneumonia, lung abscess, TB)
 - ❖ Coarse, change with coughing: bronchiectasis, or pulmonary cavities.
 - ❖ Throughout inspiration and expiration (**Biphasic**): bronchiectasis, sepsis.

7.25 Causes of crackles	
Phase of inspiration	Cause
Early	Small airways disease, as in bronchiolitis
Middle	Pulmonary oedema
Late	Pulmonary fibrosis (fine) Pulmonary oedema (medium) Bronchial secretions in COPD, pneumonia, lung abscess, tubercular lung cavities (coarse)
Biphasic	Bronchiectasis (coarse)

Wheeze:

- Musical sounds.
- Pathology: continuous oscillation of opposing airways and implies airway narrowing
- Tends to be louder on **expiration**, because airways are narrow on expiration.
- Inspiratory wheezes implies severe airway narrowing
- High pitched wheeze arises from smaller airways
- Low pitched wheeze originates from larger bronchi.
- In severe airway obstruction, wheeze may be absent (silent chest).

Causes:

- Wheeze is characteristic for **asthma** and **COPD**. **Bronchiolitis** in children.
- Lung cancer: localized wheeze (may cause diffused wheezing if the tumor in the center).

CLINICAL PEARL

2-2

“All That Wheezes Is Not Asthma”

The most common cause of wheezing is asthma. However, any condition that mimics large-airway bronchospasm can cause wheezing.

- CHF—due to edema of airways and congestion of bronchial mucosa
- COPD—inflamed airways may be narrowed, or bronchospasm may be present
- Cardiomyopathies, pericardial diseases can lead to edema around the bronchi
- Lung cancer—due to obstruction of airways (central tumor or mediastinal invasion)

Pleural friction rub:

- Pathology: inflammation of parietal and visceral pleura, when they move over one another.
- Causes: pneumonia, P. embolism, P. vacuities , bronchial cancer .
- Heard best at the *end of inspiration* and *beginning of expiration*.
- Disappear when breathing hold (to differentiate it from pericardial rub)

Pneumothorax click:

- Rhythmical sound synchronous with cardiac cycle.
- Causes: air between the two layers of pleura overlying the heart.

➤ To complete my examination:

- CXR
 - O2 sat
 - Peak flow meter
-

Hyperinflated chest finding on examination

a. General

1. Use accessory muscle
2. Tachypnea (may be emphysema)
3. Cyanosis (chronic bronchitis)
4. Thin (emphysema) , obese (chronic bronchitis)
5. Pursed lips , leaning forward
6. Indrawing of supraclavicular fossa and intercostal space during inspiration
7. bulge of supraclavicular fossa (emphysema)
8. raised JVP (pulsatile)
9. cor-pulmonal >> lower limb edema

b. inspection of the chest

1. bulge (emphysema)
2. Barrel-shape chest

c. Palpation

1. Tracheal tug
2. Decrease distance (between sternal angel and cricoid)
3. Apex beat not palpable
4. Decrease chest expansion
5. Subcutaneous emphysema
6. Decrease TVF on both sides

d. Percussion

1. Hyperresonance
2. Obliteration of liver and cardiac dullness
3. Liver may be pushed down
4. Over the heart we do light percussion (should be dullness) , if it became resonance >>then this called (sure sign of hyperinflation)

e. Auscultation

1. Breath sound diminished
 2. Vesicular breathing with prolonged expiration
 3. Rhonchi at expiration
 4. Course crepitation at late inspiratory phase or may be biphasic
 5. Vocal resonance (increase or decrease ???)
-

COPD

Blue Bloater

Chronic Bronchitis



Symptoms

- Chronic , productive cough
- Purulent sputum
- Hemoptysis
- Mild dyspnea initially
- Cyanosis (due to hypoxemia)
- Peripheral edema (due to cor pulmonale)
- Crackles, wheezes
- Prolonged expiration
- Obese

Pink Puffer

Emphysema



Symptoms

- Dyspnea
- Minimal cough
- Increased minute ventilation
- Pink skin, Pursed-lip breathing
- Accessory muscle use
- Cachexia
- Hyperinflation, barrel chest
- Decreased breath sounds
- Tachypnea

Pink puffer and blue bloater

According to their clinical appearance, patients with COPD are often categorized as either “Pink Puffer” or “Blue Bloater”.

	Pink Puffer	Blue Bloater
Main pathomechanism	Emphysema (prominent)	Chronic bronchitis (prominent)
Clinical features	- Breathlessness but Noncyanotic - Cachectic or thin due to increase energy expenditure during breathing - Sitting ,lean foreword - Pursed-lip breathing - Mild cough - Tachypnea - Patient appear distressed and use of accessory muscles	- Cyanotic - Chronic Productive cough - Overweight - Peripheral edema - Normal RR or slightly increase - There is no apparent distress and no use of accessory
PaO ₂	Slightly reduced	Markedly reduced
PaCO ₂	Normal (possibly in late hypercapnia)	Increased (early hypercapnia)

Pursed lip breathing

- The patient breathes in through the nose and breathes out slowly through pursed lips.
- This style of breathing increases airway pressure and prevents bronchial collapse during the last phase of expiration.
- More commonly seen in patients with emphysema

- Prolonged expiratory phase, end-expiratory wheezing, crackles, muffled breath sounds, and/or coarse rhonchi on auscultation

Blue bloaters (chronic bronchitis prominent)	Pink Puffers (emphysema predominant)
• Major complaint is chronic cough, productive of mucopurulent sputum, with frequent exacerbations due to chest infections. • Often presents in late 30s and 40s. • Dyspnea usually mild. • Patient usually over weight. • Cyanosis is common, peripheral edema also occurs due to <u>cor pulmonale</u>, therefore called blue bloaters. • Rhonchi are common on auscultation.	• Major complaint is dyspnea, often severe. • Usually presenting after age 50. • Patients are usually thin, with recent weight loss common. • Patient is uncomfortable with use of accessory muscles of respiration but there is no cyanosis or peripheral edema, therefore called pink puffers. • Breath sounds are very reduced. No rhonchi.
• Hemoglobin usually elevated (15-18 g/dl) • PaO₂ reduced (45-60 mmHg) • PaCO₂ slightly to markedly elevated • Chest X-ray shows increased interstitial markings (dirty lungs) especially at bases. Diaphragms are not flattened.	• Hemoglobin usually normal (12-15 g/dl) • PaO₂ normal to slightly reduced (65-75 mmHg). • PaCO₂ normal to slightly reduced (35-40 mmHg). • Chest x-ray shows hyperinflation with flattened diaphragm. Vascular markings are diminished, particularly at the apices.
• Pulmonary function shows normal total lung capacity TLC; may be slightly increased	• TLC markedly increased.

Features of advanced COPD

- Congested neck veins "Sign of increased jugular venous pressure, first due to pulmonary hypertension and later to cor pulmonale"
- **Barrel chest:** This deformity is most commonly seen in individuals with emphysema. " It is caused by long-term use of the accessory respiratory muscles along with the flattening of the diaphragm in normal and quiet inspiration; this flattening is due to hyperinflation of the lungs."
- Asynchronous movement of the chest and abdomen during respiration
- Use of accessory respiratory muscles due to diaphragmatic dysfunction "On inspiration, the jugular notch and flanks retract."
- Hyperresonant lungs, reduced diaphragmatic excursion, and relative cardiac dullness on percussion
- Decreased breath sounds on auscultation: "silent lung"
- **Peripheral edema (most often ankle edema) "Edema occurs in COPD as a result of water retention (secondary aldosteronism due to sympathetic activation) and pulmonary vasoconstriction (due to chronic hypoxemia). Heart failure is not always the cause. Diuretic treatment is often unnecessary, as edema improves with increased pulmonary function."**
- Right ventricular hypertrophy with signs of right heart failure and cor pulmonale(more with chronic bronchitis)
- Hepatomegaly
- Often weight loss and cachexia "The main causes are negative energy balance (increased work of breathing and systemic inflammation raise basal metabolism), muscle atrophy (glucocorticoid therapy), and increased oxidative stress (chronic hypoxemia). However, some patients experience weight gain because their overall activity level is reduced"
- Secondary polycythemia
- Confusion: due to hypoxemia and hypercapnia
- Nail clubbing in the case of certain comorbidities (e.g., bronchiectasis, pulmonary fibrosis, lung cancer)

Nail clubbing is not a finding specific to COPD; its presence usually suggests comorbidities such as bronchiectasis, pulmonary fibrosis, or lung cancer.

Acute exacerbation of COPD is considered acute worsening of the patient's respiratory symptoms (increased dyspnea, increased sputum volume, production of purulent sputum) that necessitates a change in medications

On examination

Inspection (*signs of advanced airway obstruction*)

- Patient is dyspnoic
 - Accessory muscles of respiration are used. Sternomastoid and scalene muscles become prominent during inspiration.
 - Pursing of lips: during expiration patient closes his lips tightly to aid expiration by breathing out against a resistance in attempt to prevent air trapping by maintaining intrabronchial pressure that prevents sudden collapse of small airways during expiration.
 - Reduction in the length of trachea above the sternal notch.
 - Tracheal tug: tracheal descent during inspiration.
 - Indrawing of supraclavicular fossae and intercostal spaces during inspiration.
 - Jugular venous distension during expiration.
-

- Chest becomes barrel-shaped i.e. anteroposterior diameter becomes more than the transverse diameter, due to air trapping.
- Central cyanosis.

Palpation

- Apex beat not palpable
- Chest expansion becomes decreased.

Percussion

- Percussion note is hyper-resonant
- Loss of normal area of cardiac & liver dullness.

Auscultation

- Breath sounds are decreased
- Normal vesicular breathing with prolonged expiration.
- Coarse crepitations at lung bases heard during inspiration and expiration both phases and change in character or disappear after coughing.
- Rhonchi at expiration.

Look features of respiratory failure and heart failures.

Features of type II respiratory failure

- Central cyanosis
- Flapping tremor
- Bounding pulse.

Features of right heart failure (Cor pulmonale)

- Raised JVP
- Right ventricular heave
- Loud P2
- Enlarged liver
- Pedal edema

BLUE BLOATERS AND PINK PUFFERS

Although chronic bronchitis and emphysema mostly occur in combination, yet the patient may present with predominant features of chronic bronchitis or emphysema.

When the chronic bronchitis is predominant with cough, sputum, cyanosis, hypercapnia, pulmonary hypertension, right ventricular failure & peripheral edema occurring at an early stage, patient is called "BLUE BLOATER"

When emphysema is pure, patient is always breathless but not cyanosed. Cough, edema & heart failure are not prominent, and the patient is termed as "PINK PUFFER". These patients are not cyanosed because hypoxia increases respiratory rate that compensates hypoxia.

Asthma

A 20-year-old gentle man presented with SOB ,chest tightness, cough with audible breathing sounds , nonsmoker , the patient mentioned that the symptoms become worse at night specially 4:00 am and with exercise . give differential diagnosis . Examine him looking for signs of Asthma.

➤ **General look:**

- Conscious alert oriented to place, time and people (severe cases patient >> agitation / drowsiness).
- Cyanosis (central under the tongue and peripheral lips, conjunctiva) >> central cyanosis in severe cases .
- Tachypnea and use of accessory muscles mainly the sternocleidomastoid .
- Use of extra equipment: o2 support, inhalers.
- Note any unusual noisy breathing sounds
- If there is any skin rash and eczema.
- Look for any sign of acute attack:
diaphoresis, tachypnea, wheezing, speaking in incomplete sentences , use accessory muscles . Paradoxical movement of the chest and abdomen during the inspiration.
Hemodynamically instability.

➤ **Vital signs :**

- RR (> 25 tachypnea , > 30 respiratory distress)
- HR (could be tachycardia)
- Temperature.
- o2 sat (patient may be hypoxic)
- Blood pressure (may be hypotensive and pulsus paradoxus) .

➤ **hands :**

- no specific findings .but you have to inspect looking for any sign of allergy such as eczema

➤ **neck :**

- Tracheal tug >> hyperinflation, may have elevated JVP , no other specific findings .

➤ **chest :**

➤ **inspection :**

- ✓ breathing pattern ,
- ✓ Pectus carinatum (pigeon chest) with prominent Harrison's sulci can be caused by uncontrolled childhood asthma, osteomalacia or rickets.

➤ **Palpation:** the chest expansion may be reduced.

➤ **percussion :** hyperresonance chest .decrease the dullness of the lower and heart

➤ **Auscultation:** Diminished air entry, vesicular breathing, prolonged expiratory time, added sounds (end expiratory wheeze).

- Mention that you are going for signs of atopy: eczema, rhinitis and nasal polyps.

Pneumonia

Pneumonia patient will present with acute cough as a chief complain.
Pneumonia syndrome could be typical vs Atypical.

Typical (S. pneumonia , H-influenza , G-ve)	Atypical (mycoplasma , chlamydia m viruses)
Sudden onset High grade fever >39 wit chills Productive cough with green / yellow sputum - purulent (could be dry at first) . Toxic looking patient Loss of appetite Pleuritic chest pain SOB (plural effusion)	Gradual (headache , sore throat , fatigue, myalgia) Low grade fever Dry cough Non- toxic look Good appetite
On physical exam: tachycardia , tachypnea , late inspiratory crackles , bronchial breathing , increased TVF , plural friction rub , dullness	Pulse-temperature dissociation (normal pulse despite fever) , wheeze , crackles
On X-RAY : Lobar consolidation	Diffuse bilateral infiltrates with minimal consolidation

IN history, ask about:

Risk factor: (for both types)

- * weight loss
- * DM, chronic liver disease, lung diseases
- * current smoker (more than 1 pack –year)
- * Previous Respiratory infections
- * Hospital stay
- * increased risk of aspiration (stroke, epilepsy, surgeries)
- * Malignancy > chemotherapy > neutropenia > infection.

Social history: travel history, vaccination

For investigation:

CBC

CXR

Gram stain and sputum culture

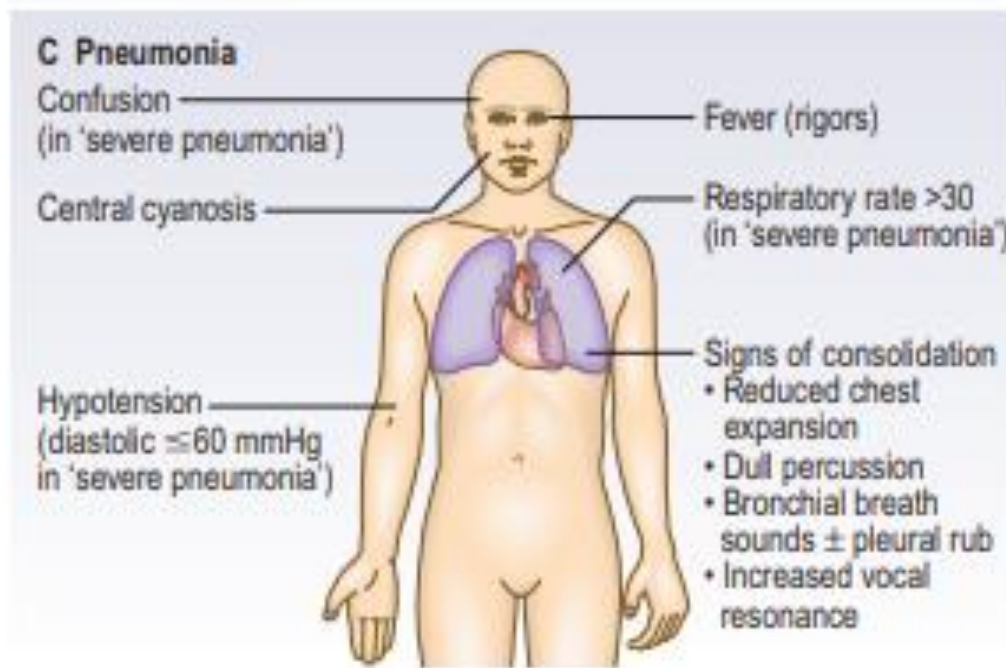
Blood culture

serology

} not useful in acute case.

Note:

pt. could have upper abdominal pain m if the pneumonia in the lower lobe.



Pulmonary Embolism

❖ Introduction:

Pulmonary embolism is a common cause death in the hospital, especially in patients with cancer, stroke and pregnancy. PE has a wide variety of presenting features, ranging from no symptoms to shock or sudden death. The most common presenting symptom is dyspnea followed by chest pain and cough. However, many patients, including those with large PE, have mild or nonspecific symptoms or are asymptomatic. Being difficult to suspect based only on the symptoms and difficult to diagnose, knowledge of PE risk factors is a must to know when to suspect it.

The majority (80%) of pulmonary emboli arise from the propagation of lower limb DVT. Rare causes include septic emboli (from endocarditis affecting the tricuspid or pulmonary valves), tumor (especially choriocarcinoma), fat, air, amniotic fluid and placenta.

A risk factor for DVT/PE can now be identified in over 80 percent of patients with venous thrombosis. Furthermore, there is often more than one factor at play in a given patient.

❖ Clinical Vignette:

A 21-year-old pregnant lady presented to the ER with chest pain and cough. Investigations showed that she had PE. Ask the patient relevant questions to assess her for causes and risk factors of PE.

>>> It is very important to understand this subject and to memorize these risk factors.

- As most cases of PE are due to lower limb DVT, the best way to think of its causes and risk factors is through the famous Virchow's triad:
 - 1- Stasis: Anything causing stasis of venous blood in lower limb veins increases the risk of DVT/PE.
e.g.: Prolonged immobilization, long travel, obesity ... etc.
 - 2- Endothelial injury
 - 3- Hypercoagulable state
 - a) Inherited: factor V Leiden, protein C and S deficiency.
 - b) Acquired: Malignancy, nephrotic syndrome

❖ Risk factors of DVT/PE:

- Age $\geq$ 65 years
- Smoking
- Malignancy
- Prior history of DVT/PE
- Hereditary hypercoagulable state
- Prolonged immobilization (being bed-ridden, long-distance travel)
- Obesity
- Nephrotic Syndrome
- Cardiac disease, especially CHF and congenital heart disease

- Major trauma
- Previous surgery (especially pelvic surgery)
- Estrogen exposure (pregnancy, OCPs, hormone-replacement therapy).
- Certain cancer therapies (e.g. tamoxifen, thalidomide, lenalidomide)
- Diseases:
 - Anti-phospholipid syndrome
 - Inflammatory bowel disease
 - Nephrotic syndrome
 - Severe liver disease
 - Myeloproliferative neoplasms (polycythemia Vera and essential thrombocythemia)

❖ Other causes of PE without DVT:

- 1- Fat embolism (long bone fracture)
- 2- Amniotic fluid embolism (during or after delivery)
- 3- Air embolism (trauma to thorax, indwelling venous/arterial lines)
- 4- Septic emboli (IV drug use)
- 5- Schistosomiasis

Wells criteria and modified Wells criteria: clinical assessment for pulmonary embolism

Clinical symptoms of DVT (leg swelling, pain with palpation)	3.0
Other diagnosis less likely than pulmonary embolism	3.0
Heart rate >100	1.5
Immobilization (≥3 days) or surgery in the previous four weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Probability	Score
Traditional clinical probability assessment (Wells criteria)	
High	>6.0
Moderate	2.0 to 6.0
Low	<2.0
Simplified clinical probability assessment (Modified Wells criteria)	
PE likely	>4.0
PE unlikely	≤4.0

DVT: deep vein thrombosis; PE: pulmonary embolism.

1 19.94 Risk factors for venous thromboembolism	
Surgery	
• Major abdominal/pelvic surgery • Hip/knee surgery	• Post-operative intensive care
Obstetrics	
• Pregnancy/puerperium	
Cardiorespiratory disease	
• COPD • Congestive cardiac failure	• Other disabling disease
Lower limb problems	
• Fracture • Varicose veins	• Stroke/spinal cord injury
Malignant disease	
• Abdominal/pelvic • Advanced/metastatic	• Concurrent chemotherapy
Miscellaneous	
• Increasing age • Previous proven VTE • Immobility	• Thrombotic disorders (p. 1054) • Trauma

Obstructive sleep apnea (OSA)

A 40-year-old gentle man presented with excessive day time sleepiness and recurrent airway obstruction, take full history.

❖ **Patient profile:** M>F, middle aged - usually overweight.

❖ **Diagnostic approach to OSA**

∞ **history**

✓ **Symptoms while sleeping (seek for witness)**

Snoring [partial airway obstruction], **apnea** [complete airway obstruction], **arousal** or **choking** [arousal is seen on EEG or the pt move his limbs, while in choking there is full awakening], choking episode is described as snoring → pausing → grunting noise → snoring again, all in addition to **Nocturia**.

✓ **Symptoms in the morning**

Headache at morning, Unrefreshing sleep, restless sleep.

✓ **Symptoms during the day**

Impaired cognitive function (concentration and memory), irritability/personality change, decreased libido, excessive daytime sleepiness [due to sleep fragmentation].
→ Increased risk for RTA

Past medical and surgical history

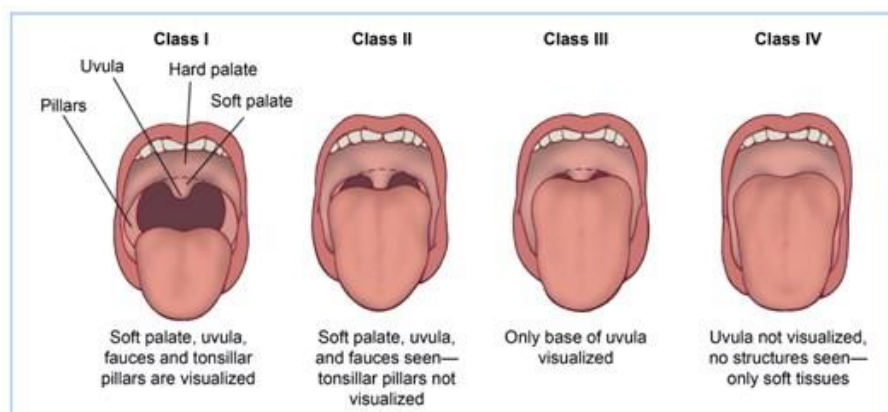
- » Can be associated with nasal polyps, acromegaly and hypothyroidism.
- » Increased risk for DM, systemic and pulmonary HTN, IHD, cardiac arrhythmia and strokes.

Drugs, alcohol

∞ **Physical exam**

Perform full physical examination **focusing** on the following:

- ✓ Neck : JVP examination, thyroid exam (pt with hypothyroidism can develop OSA), neck circumference, cricosternal distance
- ✓ Mouth: mallampati classification



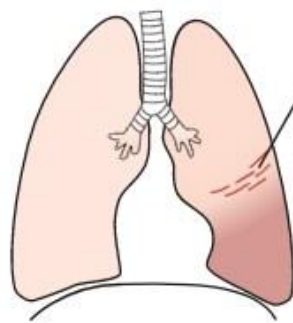
- ✓ Chest:
 - Signs of hyperinflation:
 - * On inspection: barrel chest, intercostal retraction and paradoxical breathing (if severe hyperinflation).
 - * On palpation: increased AP: transverse diameter (more than 5:7), symmetrical decreased chest movement
 - * Hyper resonant percussion note
 - * On auscultation: Decreased air entry, prolonged expiratory phase, wheezes, loud A2 (if associated with HTN).
 - Signs of pulmonary HTN: (don't forget the increased risk of pulmonary HTN in these pts)
 - * Left parasternal heave (due to RT ventricular hypertrophy)
 - * Wide S2 splitting, pansystolic murmur in case of tricuspid regurgitation.

∞ Diagnosis

Polysomnography (overnight sleep study) confirms the diagnosis.

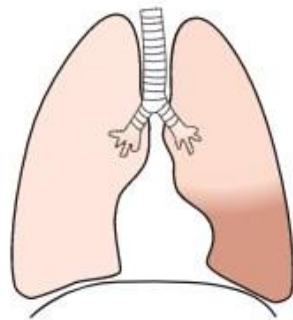
Pneumothorax (complication)	Pulmonary HTN (complication)	Pleural effusion (complication)
Chief complaint: pleuritic chest pain Diagnostic approach to pneumothorax: ✓ Pleuritic chest pain (sharp, stabbing and severe, might be dull, increase with inspiration and coughing), SOB. ✓ It's a complication of: Trauma, COPD, pneumocystic carinii infection, cystic fibrosis, pneumonia, TB, or might be spontaneous.	Chief complaint: cough, chest pain, hemoptysis Diagnostic approach to pulmonary HTN: ✓ Chest pain (can be pleuritic or central if 'HF' ensues), SOB, cough, hemoptysis, fatigue, hoarseness, wheeze, <i>abd. mass, leg swelling</i> ✓ It's a complication of decreased ventilation (the body compensates by increasing perfusion) or of right ventricular hypertrophy. ✓ Ask about trauma, pregnancy, tumors ✓ Past medical history: CT diseases, vasculitis, hepatitis, DVT, COPD, major surgeries. ✓ Drugs: oral contraceptives, chemotherapy, amphetamines, <i>penthramine, tryptophan.</i> ✓ <i>Social Hx: smoking, occupation (interstitial lung disease), alcohol, travel.</i>	Chief complaint: pleuritic chest pain Diagnostic approach to pleural effusion: ✓ Pleuritic chest pain (mostly with inflammation, sharp, stabbing, friction rub, intensifies with inspiration and cough), SOB, feeling of compressed lung, cough, wheeze. ✓ Causes: HF, renal diseases, hypothyroidism, peritoneal dialysis, liver cirrhosis, mitral stenosis, trauma, post MI, recent RTIs esp. pneumonia, TB, PE, cancer. ✓ Drugs: amiodarone, phenytoin, methotrexate. ✓ <i>Past Medical: SLE, Polyarteritis (Autoimmune)</i>
Physical signs: hyperresonance and decreased breath sounds over the area. If tension pneumothorax takes place, there will be be SHIFTING signs (lung collapses at the same side, trachea is shifted to the other side).	Physical signs: right ventricular heave, loud S2 (P component), systolic ejection murmur, raised JVP, hepatomegaly, ascites, LL edema with calf pain, pulsatile liver, tricuspid regurgitation (pan systolic), right ventricular S4.	Physical signs: tachypnea, decreased expansion, deviation of trachea, decreased TVF, stony dull on percussion, decrease breath sound, bronchial breathing, friction rub, crackles, crepitations, decreased vocal resonance.
Labs: CXR is diagnostic (a line demarcating a hyperlucent area).	Labs: CXR (increased vascular markings), ECG, PFTs.	Labs: CXR (blunting of the costophrenic angles), CT scan. <i>Pleural fluid aspiration (Exudate VS Transudate)</i> <i>Pleural biopsy: Abraham's needle, pleuroscopy.</i>

Some physical signs



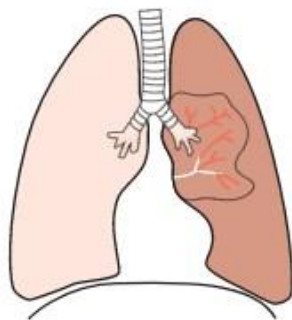
(There may be
brochial breathing
at the top of an effusion)
Expansion: ↓
Percussion: ↓ (Stony dull)
Air entry: ↓
Vocal resonance: ↓
Trachea + mediastinum central
(shift away from affected side
only with massive effusions
≥ 1000ml)

PLEURAL
EFFUSION



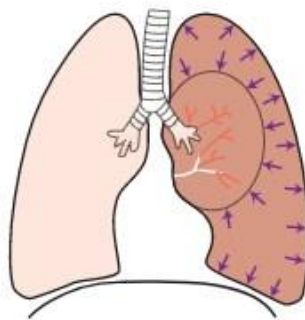
Expansion ↓
Percussion note ↓
Vocal resonance ↑
Bronchial breathing ±
coarse crackles (with
whispering pectoriloquy)
Trachea + mediastinum central

CONSOLIDATION



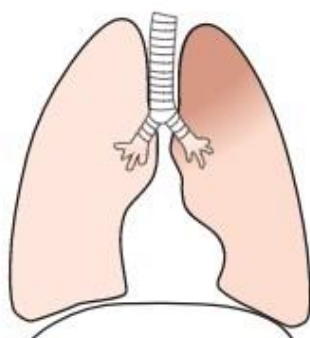
Expansion ↓
Percussion note ↑
Breath sounds ↓
Trachea + mediastinum shift
Away from
the affected side

SPONTANEOUS
PNEUMOTHORAX/
EXTENSIVE COLLAPSE
(ΔΔ LOBECTOMY/
PNEUMONECTOMY)



Expansion ↓
Percussion note ↑
Breath sounds ↓
Trachea + mediastinum shift
towards the affected side

TENSION
PNEUMOTHORAX
(See fig 1 p763 for
chest X-ray image)

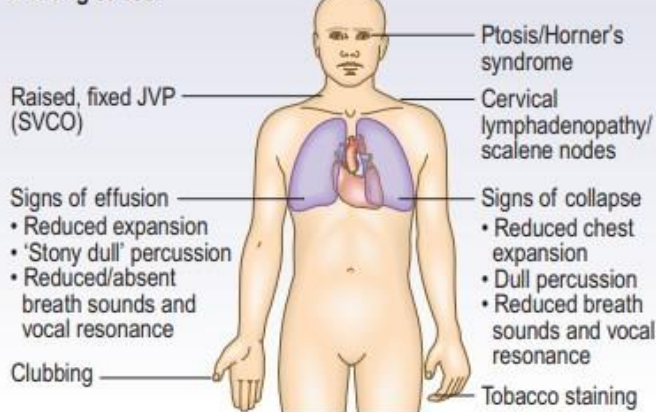


Expansion ↓
Percussion note ↓
Breath sounds bronchial
± crackles
Trachea + mediastinum
central or pulled towards
the area of fibrosis

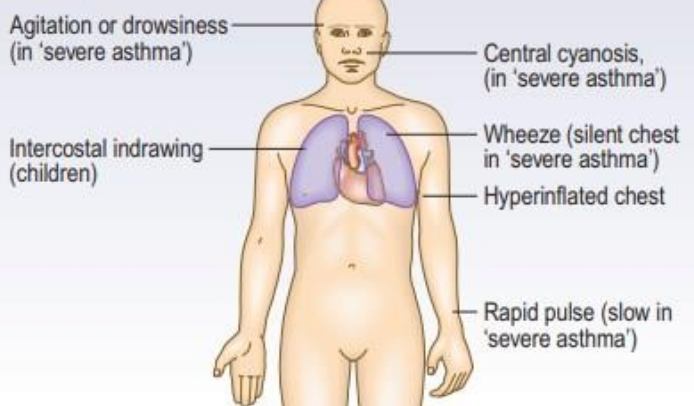
FIBROSIS

Fig 1. Physical signs on chest examination.

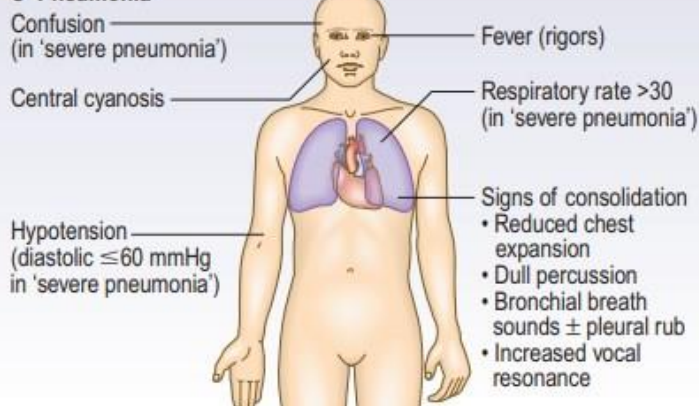
A Lung cancer



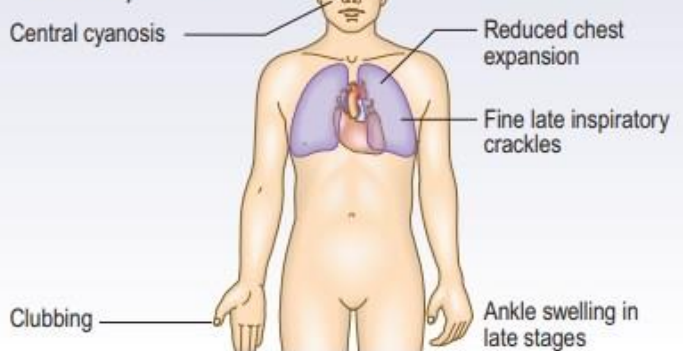
B Asthma



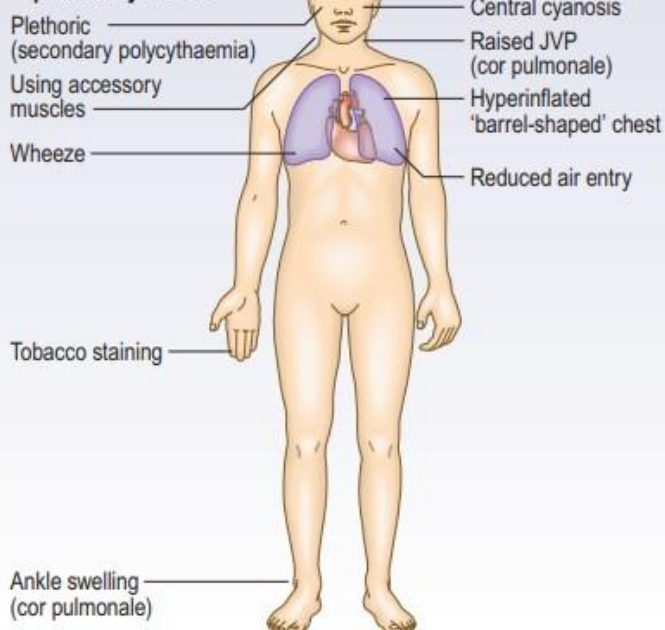
C Pneumonia



D Idiopathic pulmonary fibrosis (fibrosing alveolitis)



E Chronic obstructive pulmonary disease



F Pulmonary thromboembolism

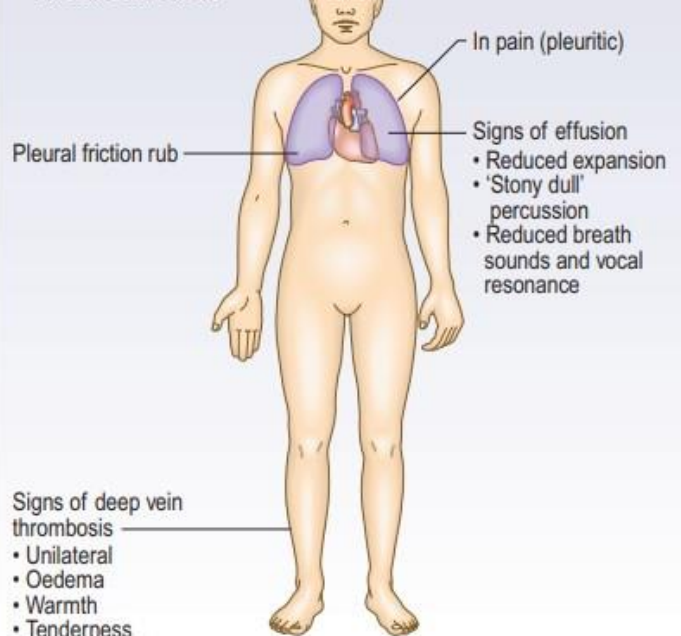


Fig. 7.20 Clinical signs of common respiratory conditions. JVP, jugular venous pressure; SVCO, superior vena caval obstruction.

Important presentations

Pneumonia

Inspection

- Look for sputum pot at bedside.
- Tachypnoeic, tachycardic, or hypotensive?
- Warm peripheries.
- Bounding pulse.
- Sweaty and clammy.

Palpation

- Reduced expansion on the affected side.
- Increased tactile vocal fremitus if consolidation.

Percussion

- Dull.

Auscultation

- Coarse crackles, localized.
- Bronchial breathing (possible).
- Whispering pectoriloquy.
- Reduced air entry.
- Increased vocal resonance.

Lobar collapse

Palpation

- Mediastinal shift towards the abnormality.
- Potentially ↓ chest wall movement locally.

Percussion

- Dullness to percussion restricted to affected lobe.

Auscultation

- ↓ breath sounds usually.

Pleural effusion

Inspection

- Reduced chest expansion unilaterally (if large).

Palpation

- Trachea may be pushed away from the effusion.
- Apex beat:
 - A large right effusion will displace the cardiac apex to the left
 - A large left effusion may make the apex beat difficult to palpate.

Percussion

- 'Stony dull'.

Auscultation

- Markedly reduced breath sounds.
- Reduced vocal resonance.
- Collapsed or consolidated lung above the effusion may produce an overlying region of bronchial breathing.

Pneumothorax

Inspection

- No mediastinal shift (only occurs with a tension pneumothorax).
- Chest wall asymmetry may be evident with a large pneumothorax (greater volume on affected side).

Percussion

- Hyper-resonant.

Auscultation

- ↓ breath sounds on affected side.
- ↓ vocal resonance on affected side.

Interstitial fibrosis

Inspection

- Patients may be cyanosed.
- There may also be signs of connective tissue disease or skin changes of radiotherapy.
- Clubbing is common.

Palpation

- Trachea may move towards the fibrosis in upper lobe disease.
- ↔ or ↓ chest wall movement.

Percussion

- ↔ percussion note.

Auscultation

- ↔ breath sounds.
- ↔ vocal resonance usually, may be increased if dense fibrosis.
- Fine 'Velcro®' crackles.

COPD

Inspection

- Inhalers at the bedside.
- Sputum pot?
- Thin skin with bruising (use of steroids).
- Use of accessory muscles/brace position.
- Tachypnoea.
- No mediastinal shift.
- Chest hyper-expanded with little additional excursion.
- Prolonged expiration and pursed lip breathing.

Percussion

- May be globally hyper-resonant to percussion.

Auscultation

- ↓ breath sounds globally, may be additional polyphonic wheeze.
- ↓ vocal resonance usually in the upper lobes (where bullae are commonest).
- Heart sounds often quiet.

Bronchiectasis

Inspection

- Often copious sputum (usually purulent, may contain blood).
- Digital clubbing may be present.
- Low BMI.

Palpation

- No mediastinal shift.
- Chest wall expansion equal.

Percussion

- Percussion resonant.

Auscultation

- Mixed, predominant coarse crackles.
- Often additional polyphonic wheeze.
- Vocal resonance normal.

Neuromuscular insufficiency

Intrinsic muscle weakness or damaged innervations.

Inspection

- Non-respiratory signs of neuromuscular illness (e.g. altered phonation, limited mobility).
- Rapid shallow breathing, sometimes with abdominal paradox.

Palpation

- Chest wall expansion equal but limited excursion.

Percussion

- Percussion note resonant.

Auscultation

- Breath sounds normal.
- Basal crackles common from atelectasis (impaired cough).

A. Common in the OSCE						
	Clubbing	Expansion	Trachea	Percussion	Breath sounds	Added sounds
Normal	No	Normal	Central (slightly right deviated)	Resonant	Vesicular	–
COPD	No	Reduced	Normal	Hyper-resonant (with emphysema)	Reduced vesicular	Early inspiratory crackles, coarse, widespread. Expiratory wheeze may be present
Pulmonary fibrosis	Common	Normal	Normal/ towards affected side	Normal, dull	Reduced vesicular	Fine late inspiratory crackles
Bronchiectasis	Common	Normal/ reduced	Normal	Normal	Reduced vesicular	Coarse inspiratory and expiratory crackles, and wheeze
Asthma (note the patient may have normal signs)	No	Reduced	Normal (unless there is a pneumothorax)	Normal/slightly hyper-resonant	Reduced vesicular (beware the silent chest – this is immediately life threatening)	Widespread expiratory wheeze (there may be some inspiratory wheeze if this is chronic asthma)
Pulmonary embolus (few abnormal signs – thus can put a normal simulated patient into an OSCE station)	No	Normal	Normal	Normal	Normal (may be reduced in areas affected by large PEs)	Unusual

B. Unlikely in the OSCE (but you may be asked about)

	Clubbing	Expansion	Trachea	Percussion	Breath sounds	Added sounds
Pleural effusion Fig. 2.10	No (may be present if there is an underlying tumour)	Reduced with large effusion	Normal (deviated away in large effusions)	Stony dull	Reduced or absent	–
Consolidation (pneumonia) Fig. 2.7	No	Normal or ↓	Normal	Dull	Bronchial	Localized crackles
Pneumonectomy	Depends on underlying cause	Decreased on removed side	Deviated away from remaining lung	Hyper-resonant over affected side	Absent over affected side; bronchial sounds may be transferred	Bronchial sounds may be audible
Lobar collapse	No	Normal	Normal/ towards	Dull (slight)	Slightly reduced vesicular	Possibly a few crackles
Pneumothorax	No	Reduced on the affected side	Away (a late sign of tension)	Hyper-resonant	Reduced vesicular	–

Gastroenterology

Hatem Rjoob

Alimentary system

History

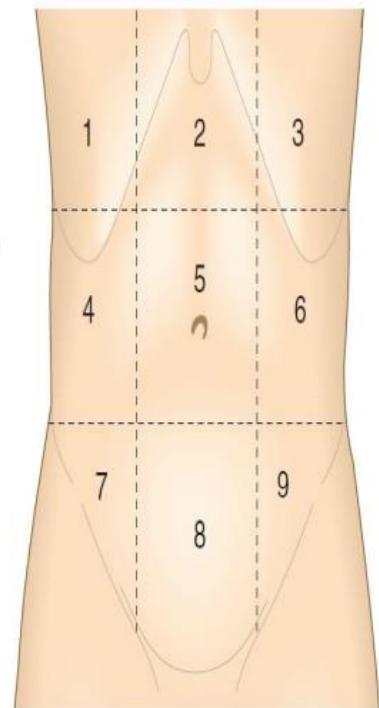
o **Patient's Profile:** Name; Age; Marital Status; Place of Residence; Occupation

o **Chief Complaint:** Acute Pain (of less than a few days duration that has worsened progressively until the time of presentation.)

o **History of Present Complaint:**

1)site of pain

- cholecystitis/cholangitis (1)
- biliary colic (1, 2)
- hepatitis (1, 2)
- pneumonia (1 or 3)
- peptic ulcer disease/gastritis (2)
- acute coronary syndrome (2)
- pancreatitis (2, 5)
- ruptured abdominal aortic aneurysm (AAA) (2, 5)
- splenic rupture (3, diffuse)
- renal calculus (4, 7 or 6, 9)
- pyelonephritis (4 or 6)
- early appendicitis (5, diffuse)
- established appendicitis (7)
- terminal ileitis, e.g. Crohn's disease, *Yersinia* (7)
- mesenteric adenitis (7, diffuse)
- diverticulitis (7 or 9)
- colitis (7, 8, 9)
- ectopic pregnancy (7, 8, 9)
- pelvic inflammatory disease/endometriosis (7, 8, 9)
- ovarian torsion/cyst rupture (7, 8, 9)
- lower urinary tract infection (UTI)/cystitis (8)
- intestinal obstruction (diffuse)
- perforation (diffuse)
- mesenteric ischaemia (diffuse)
- gastroenteritis (diffuse mid-/upper abdominal)
- diabetic ketoacidosis/hypercalcaemia/adrenal crisis (diffuse)
- functional abdominal pain (any region or diffuse).



2) Onset and Duration of Pain

How does the pain come about? How long does it last? How does it fade away? Gradually or suddenly?

o Sudden:

- Perforation >> peptic ulcer
- Rupture >> AAA
- Infarction >> MI
- Mesenteric occlusion

o Rapidly Accelerating:

- Colicky syndromes
- Inflammation
- Ischemia

o Gradual:

- Inflammation >> persist until underlying cause subsides
- Obstruction >> non-strangulated bowel obstruction, urinary retention

3) Character and severity of Pain

Describe the pain to me: Is it continuous or on and off? Is it sharp or dull?

Severity: 1-10. Does it vary in severity? How did it progress?

o Colicky:

- Hyperperistalsis of smooth muscle against a mechanical site obstruction of a muscular conducting tube (bowels, ureter) produces a cramping pain
- The pain fluctuates in severity at frequent intervals and described as 'gripping'
- The interval between bouts helps locate the cause:
 - # Short interval: Jejunum, Intermediate: Ileum, Long: Colon
- Muscular dysfunction - GI mobility disorders (IBD) cause colicky pain but it's not true colic.
- Biliary/Renal colic: short peaks of pain which doesn't resolve completely in between exacerbations

o Persistent:

- Sharp, severe
- Steadily increases in intensity
- Suggests infection or inflammation

o Burning or Gnawing:

- GERD and PUD

1. **Visceral:** Dull and aching, can be colicky. Poorly localized. (distention or spasm of a hollow organ)
2. **Parietal:** Sharp and localized. (peritoneal irritation or spread of inflammation to the parietal peritoneum)
3. **Referred:** Aching and perceived to be near the surface of the body

4) Radiation

- **Shoulder Pain:**
 - Diaphragmatic irritation
 - Spleen
 - Perforated ulcer or abscess
- **Loin/Groin:**
 - Renal colic
- **Back:**
 - Pancreatic pain
 - Ruptured or dissecting AAA
 - Peptic ulcer in posterior wall
- **Scapula:**
 - Gallbladder (Right)
- **Periumbilical:**
 - Small bowel
 - Appendicitis

5) Shifting or Migration

Did the pain shift from its initial site? After how long?

Did that increase or decrease its severity?

Did it become more localized?

Can you point out the point of maximal pain?

6) Timing (Temporal Sequence)

Did you do anything that may have initiated the pain?

- Relation to food, meals, alcohol or bowel movements. Recent trauma?

7) Alleviating and Exacerbating Factors *Does anything increase or decrease the severity?*

<i>Factor</i>	<i>Relieves</i>	<i>Exacerbates</i>
• Movement • Breathing	- Patients with Renal Colic or Stones are restless and unable to lie still since they find no position comfortable	- Inflammation: - Diffuse peritonitis (relieved by rest and flexion of knees) - Cholecystitis, Pancreatitis
• Food	- Duodenal Ulcer	- Gastric Ulcer - Cholecystitis; Pancreatitis - Mesenteric Ischemia
• Sitting up, Leaning Forward	- Pancreatitis	-
• Vomiting	- Visceral pain in intestinal obstruction (transient) - Gastric Ulcer pain	-

8) Associated Gastrointestinal Symptoms

1. Anorexia:

- It is common in many upper gastrointestinal and liver diseases but does not specifically indicate gastrointestinal disease.

2. Diet

3. Weight

- The most common cause for Weight Loss is diabetes mellitus. Others like hyperthyroidism, malabsorption, malignancies and fever are common.
- loss of 10% of the weight over 6 month is an alarming sign
- Very rapid weight loss (> 0.5 kg/day) invariably indicates fluid loss. The causes include diuretic therapy, severe diarrhea, vomiting, heat illness or severe burns.

4. Mouth Ulcers (Crohn's, Celiac)

5. Dysphagia :

- Could be caused by painful mouth ulcers, mouth or throat infections, cerebrovascular accidents, bulbar palsy, benign stricture or CA of the esophagus...etc
- Neurological Dysphagia → worse for liquids than for solids.
- Neuromuscular Dysphagia → worse for solids than for liquids.
- Ask if its for Solids, liquids or both? List in chronological order
- Ask about Level at which the food sticks
- Duration, Onset and Progression

6. Odynophagia

- It may be present with or without Dysphagia.
- often an indication for active esophageal ulceration from peptic esophagitis or esophageal candidiasis.

7. Regurgitation

- What comes up? If food, is it digested or recognizable and undigested?
- How often? Precipitating factors?

8. Heartburn:

- Burning sensation behind the sternum caused by the reflux of acid into the esophagus
- How often does it occur and what makes it happen, e.g. lying flat or bending over?

Heartburn and other reflux symptoms :

- If heartburn is the main symptom, GERD is the most likely diagnosis.
- It is often accompanied by acid reflux due to regurgitation of acid producing a sour taste in the mouth.
- The occurrence of reflux on lying flat or bending forward help to differentiate it from retrosternal chest pain.
- *Water brash*: the sudden appearance of excessive saliva in the mouth due to reflex salivation and suggests GERD.

9. Dyspepsia:

- Pain or discomfort centered in the upper abdomen
- 'Fat intolerance': nausea and abdominal fullness which is worse after fatty or spicy meals.
- Could be Reflux, Ulcer or Dysmotility-like

10. Nausea and Vomiting:

- Nausea without vomiting is common in dyspepsia.
- Vomiting may be caused by GI disorders or by severe pain, e.g. renal or biliary colic, myocardial infarction or from systemic disease, metabolic disorders and drug therapy.
- *On taking history of vomiting*, remember to cover 9 questions : how often (say 3 times daily), amount (one cup or so), nature of vomitus (recognizable food, digested food, clear acidic fluid..), color (green, yellow, brown..), smell (foul smell or normal) projectile or non-projectile, association with nausea, association with pain (preceded or followed by pain, and if pain is relieved by vomiting), and lastly, association with blood.

11. Hematemesis:

- Coffee ground → upper GI bleeding that have been slowed or stopped with conversion of Hb to brown by gastric acid e.g. peptic ulcer
- Red fresh blood → more acute or more severe GI bleeding and may arise from arterial source e.g. Mallory-Weiss tear.
- Dark blood → esophageal varices.
- Cancers of the stomach and esophagus rarely present with haematemesis.

12. Indigestion or abdominal pain:

- A term commonly used for ill-defined symptoms from the upper GI tract

13. Distension:

- Duration and Progression
- Is it constant or variable? (Functional bloating is fluctuating distension that develops during the day and resolves overnight in IBS)
- What factors are associated with any variations?
- Is it painful? (IBS)
- Does it affect their breathing?
- Is it relieved by belching, vomiting or defecation?

14. Altered Bowel Habits:

- normal frequency ranges from 3 times daily to once every 3 days.
- *On taking history of defecation*, remember to cover 6 questions: how often (say one or two daily), color (brown, black, white...), consistence (hard, soft, watery..), size (bulky, string..), smell and specific gravity.
- With constipation or diarrhea, ask if the patient means that he is moving his bowels less or more frequently than usual, or if stool has been changed in consistence.
- **Constipation:** infrequent passage of hard stools
 - Lifelong or recent? How often do the bowels empty each week? How much time is spent straining? Is it Associated abdominal pain, anal pain on defecation or rectal bleeding?
- **Diarrhea:** frequent passage of loose stools
 - Is it acute, chronic or intermittent? Is there tenesmus, urgency or incontinence?

15. Defecation:

- Colour: brown, black, pale, white or silver?
- Consistence: hard, soft or watery?
- Size: bulky, pellets, string or tape like?
- Specific gravity: does it float or sink?
- Smell?
- Does it contain mucus or slime?

16. Melena:

- Passage of tarry, shiny black stools with a characteristic odor and results from upper GI bleeding

17. Rectal Bleeding (hematochezia):

- *On taking history of bleeding*, remember to cover these questions: how often, amount, color, bright or dark ? Blood may be mixed with stool, coat the surface of otherwise normal stool, or be seen on the toilet paper or in the pan , pre/post or during defecation , Anal pain?.
- Causes : Hemorrhoids, anal fissure, colorectal polyps, colorectal cancer, inflammatory bowel disease, ischemic colitis, complicated diverticular disease, in massive upper gastrointestinal bleeding blood may pass through the intestine unaltered, causing fresh rectal bleeding...etc

19. Prolapse and Incontinence:

- Does anything come out of the anus on straining? Does it return spontaneously or have to be pushed back?
- Is the patient continent of feces and flatus?
- Have they had any injuries or anal operations in the past?
- **In Incontinence** Determine whether the cause is neurological or muscular.

20 Tenesmus:

- Unproductive desire to pass stool indicates the presence of lesion in the sigmoid-rectal region.
- Usually accompanied by pain, cramping and involuntary straining efforts
- Is it true Tenesmus or only due to fear of pain?

21. Change of Skin color or jaundice:

22. Pain: “SOCRATES”

- *Visceral* abdominal pain results from distension of hollow organs, mesenteric traction or excessive smooth muscle contraction. Visceral pain is often experienced as a deep poorly localized sensation in the midline.
- *Somatic* pain is caused by irritation of the parietal peritoneum and is usually localized to the area of inflammation.

9) Associated Gastrointestinal Symptoms

<i>General</i>	<i>Urinary</i>	<i>Gynecological</i>	<i>Neurological</i>	<i>Respiratory and Cardiac</i>
• Fever - any inflamed organ, high grade if there is perforation • Chills or Rigors - Cholangitis; Choledocolithiasis • Sweating or thirst • Arthralgias, weakness, fatigue • Masses or Lumps recently noticed in the abdomen or groin? • Dehydration • Anemia Symptoms	• Dysuria • Polyuria • Hematuria • Nocturia • Slow flow, hesitancy or dribbling • Urine Color changes • Pruritis • Ankle Swelling	• Menstrual pattern: last period, regularity • Contraceptives • Sexual Hx • Pregnancies: normal or ectopic • Previous gynecological or tubal surgeries • Mid-cycle pain • Vaginal bleeding	• Fainting or Dizziness • Altered mental status • Vertigo • Headaches	• Cough - Symptoms of pneumonia: RUQ pain + cough + fever • Palpitations • SOB • Chest pain - Symptoms of thoracic disease

10) Past Medical, Family and Surgical History

- **Chronic diseases:**
 - **Hypertension**
 - **Hypercholesteremia**
 - **GI Diseases**
 - **Cardiovascular Diseases:** Risk of abdominal vascular disease (AAA, mesenteric ischemia)
 - **Diabetes** : Predisposes to myocardial ischemia and sepsis and may mask abdominal pain
 - **Cancer Hx** : Risk of bowel obstruction and perforation from recurrence.
 - **Immune diseases**
 - **Liver diseases or portal hypertension**
 - Thyroid dysfunction
 - **Blood disorders**
 - **Neurological or neuromuscular disorders** (GI dysmotility)
- **Recent Dietary Hx:** food-borne illness
- **Past Hx**
 - Similar condition or symptoms
 - Recent infections
 - Previous radiation or chemotherapy
 - Previous Blood transfusions
- **Previous procedures:** endoscopy; ERCP; colonoscopy... etc

- **Previous Surgeries:**
 - Adhesions may lead to intestinal obstruction
 - Previous removal of any organ excludes it from consideration
- **Family Hx**
 - Cancer
 - Chronic diseases
 - Celiac disease or IBD

11) Medications

- **NSAIDs:** Risk of peptic ulcer
- **Corticosteroids:** May mask signs of inflammation (fever, peritoneal irritation)
- **Antibiotics:**
 - May decrease the pain in pts with peritonitis due to partial treatment.
 - Also, if a patient has diarrhea >> suspect AB induced pseudomembranous colitis.
- **OCP**
- **Statins**
- **Vitamins and supplements**

12) Social

- Smoking (heartburn, PUD, Crohn's, GI malignancies)
- Travel Hx
- Alcohol (heartburn, dyspepsia, nausea, diarrhea, chronic liver disease)
- Sexual Hx
- Drug use
- Obesity
- Dietary Hx: fiber or fat rich; any food intolerance
- Allergies
- Recent stressors

Physical examination of the GIT

General Appearance

1. Wash hands, introduce yourself, confirm pt details
2. Take permission , ensure good light and privacy
3. Appearance :
 1. Position (flat , supine , 45, left lateral)
 2. conscious , Cooperative
 3. Patient looks well/ill
 4. Respiratory distress (dyspnea , tachypnea ,...)
 5. Sweaty
 6. Jaundice , cyanosis , pale
 7. Body mass (obese or cachexia)
4. Canula attached to his arm or not , nothing or there is something dropping in it
5. Look around the patient (vomiting , sputum ,...)
6. False catheter ?
7. Read the patient labs and vital signs if presents
8. In General Exam , patients should be in Supine Position 15 degree

Hands

1. Fingers:
 - a. Clubbing – *IBD / cirrhosis / coeliac disease/ lymphoma/ amyloidosis*
 - b. Koilonychia – chronic iron deficiency
 - c. Leukonychia - hypoalbuminemia (*cirrhosis / celiac disease/ nephrotic syndrome/ malnutrition/ burns*)
 - d. capillary refill time
 - e. cyanosis
2. Palms
 - a. Palmar erythema – *cirrhosis / pregnancy/ polycythemia*
 - b. Dupuytren's contracture:
 - i. Thickening and contraction of the palmar fascia→ can't fully extend 4th and 5th fingers
 - ii. *Associated with alcohol excess / family history (autosomal dominant)/ DM/ smoking/ hyperlipidemia/ HIV infection*
3. Back of the hands
 - a. Asterixis - *hepatic encephalopathy/ uremia/ CO2 retention*
 - b. Spider nevi
 - i. In men→ any are pathologic
 - ii. In women→ greater than 5 are pathologic
 - iii. Appear in the distribution of SVC (chest, arms, face)

Palmer erythema :

P: polycythemia and pregnancy

A: alcohol intake and autoimmune disease (RA, SLE)

L: liver failure and leukemia

M: medication (steroid)

+ thyrotoxicosis

Eyes

- 1) Xanthelasma - *hyperlipidemia*
- 2) Corneal arcus - *hyperlipidemia*
- 3) Conjunctival pallor - *anemia*
- 4) Jaundice
- 5) Kayser–Fleischer rings - *Wilson's disease*

Mouth/ Cheeks

- 1) Angular stomatitis - *Candida/ IDA/ B12 deficiency*
- 2) Oral candidiasis - *IDA, immunodeficiency*
- 3) Mouth ulcers - *Crohn's/ Celiac disease*
- 4) Glossitis - *Iron, Folate, or B12 deficiency*
- 5) Hepatic fetor - *chronic liver disease*
- 6) Parotid swelling - *chronic alcohol abuse/ bulimia*
-Due to sialadenosis of the salivary gland
- 7) central cyanosis
- 8) Gum bleeding
- 9) Pigmentation >> pseud-jugular syndrome
- 10) Dental caries

Neck

- 1) Cervical lymph nodes - *lymphadenopathy* → *infection/ malignancy*
- 2) Virchow's node - **Troisier's sign** - *left supraclavicular* - *gastric/ pancreatic CA (or lung/ breast)*

Chest

1) Spider nevi

2) Gynecomastia

a) Physiologic: *puberty/ old age*

b) Pathologic: *cirrhosis/ digoxin/ spironolactone/ starvation/ thyrotoxicosis/ renal failure/ adrenal CA/ testicular abnormalities*

c) Meanwhile, in women, *breast atrophy* is a sign of liver disease

3) Loss of body hair - *chronic liver disease*

Focused abdominal exam

Expose the patient from the nipple to the mid-thigh

Take your position in front of the patient (tip of the bed) while he is laying down in supine position with one or two pillows under the head.

1. General Inspection

1) Symmetry - *asymmetry w/ enlarged liver or bladder*

2) Shape

a) Distended

i) Fat → Distension is central - Umbilicus is inverted

ii) Fluid (ascites) → Distension of the flanks, Umbilicus flat or everted

iii) Feces → usually associated with abdominal pain.

iv) Flatus → usually associated with abdominal pain.

v) Fetus.

b) Flat (*normal*)

c) Scaphoid (*normal or diaphragmatic hernia*)

3) umbilicus :

-Site (centrally located or not)

-Shape (inverted ,everted , crescent), inverted is normal , everted means hernia , pregnancy or normal

-Masses (para-umbilical hernia , LN)

-Discharge

4) respiration , visible pulsation and visible peristalsis (انزل على مستوى المريض)

-type of breathing (abdominothoracic or thoracoabdominal), if no movement then think of peritonitis , diaphragmatic paralysis , tense ascites .

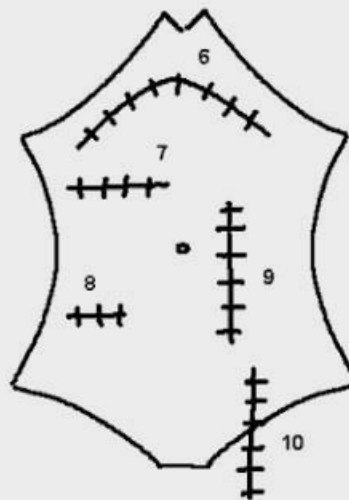
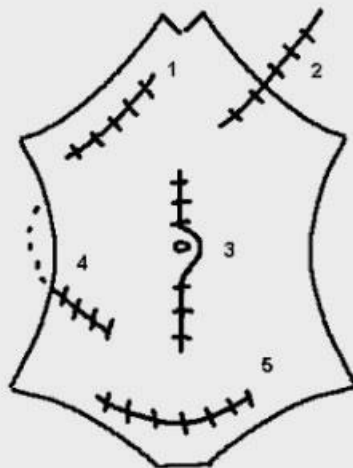
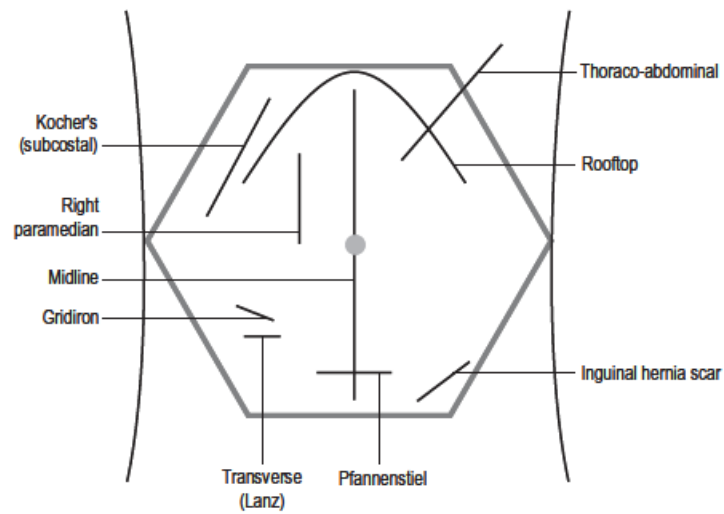
5) Visible pulsation

-expansile pulsation >> AAA

-transmitted pulsation >> mass transmit aortic pulsation such as pseudocyst

Go to the right side of the patient :

1. Skin
 - superficial discoloration**
 - a) Cullen's sign – bruising surrounding umbilicus– retroperitoneal bleed (*pancreatitis/ruptured AAA*)
 - b) Grey-Turner's sign – bruising in the flanks – retroperitoneal bleed (*pancreatitis/ruptured AAA*)
 - rash** :shingles
 - pigmentation**
 - scratch sign due to pruritis**
2. Scars: describe site, size, well-healed or not , and if there is incisional hernia or not (ask patient to sit 45 without your help ,so hernia appear while doing this)
 - a) Midline scars (*laparotomy*)
 - b) RIF Gridiron/ McBurney (*appendectomy*)
 - c) Right subcostal/ Kocher (*cholecystectomy*)
 - d) Suprapubic/ pfannenstiel scar (*C-section*)
3. S triae
 - a) White - chronic- rapid weight gain/ previous pregnancy
 - b) Pink – new
 - c) Purple - cushing's' syndrome
4. Umbilicus confirm
5. Swelling (masses) :(site , shape , size , symmetry , discoloration , skin changes , scars , visible pulsation)
6. Superficial dilated vein (either IVC obstruction or cubit medusae , so do milki test to differentiate), (Milki test : if flow away from the umbilicus its portal HNT , if its toward the umbilicus then its IVC obstruction)
7. Superficial hair distribution
8. Stomas or drains
9. Complete the inspection by observing the hernial orifices. Ask the patient to cough and then to tense the abdominal muscles by lifting the head to reveal any defect.
10. cough impulse for hernia



- 1 = Kocher
- 2 = Thoracoabdominal
- 3 = Midline
- 4 = Muscle splitting loin
- 5 = Pfannenstiel
- 6 = Gable
- 7 = Transverse muscle splitting
- 8 = Lanz
- 9 = Paramedian
- 10 = McEvedy



2. Superficial (Light) palpation

- clean and warm your hands , ask the patient if there is any pain , start away from pain / masses . G or S shape starting from the LIF keep eye contact with the patient .
- Use the whole flat of your hand (not just fingertips)
- Palpate all 9 regions

1) Tenderness

- a) Note area and severity
 - i) Epigastrium→ PUD
 - ii) RUQ→ cholecystitis
 - iii) LIF→ diverticulitis
 - iv) RIF→ Crohn's or appendicitis
- b) *In several areas on minimal pressure→ peritonitis or anxiety*
 - i) Anxiety "pain" disappears if pt is distracted

2) Rebound tenderness

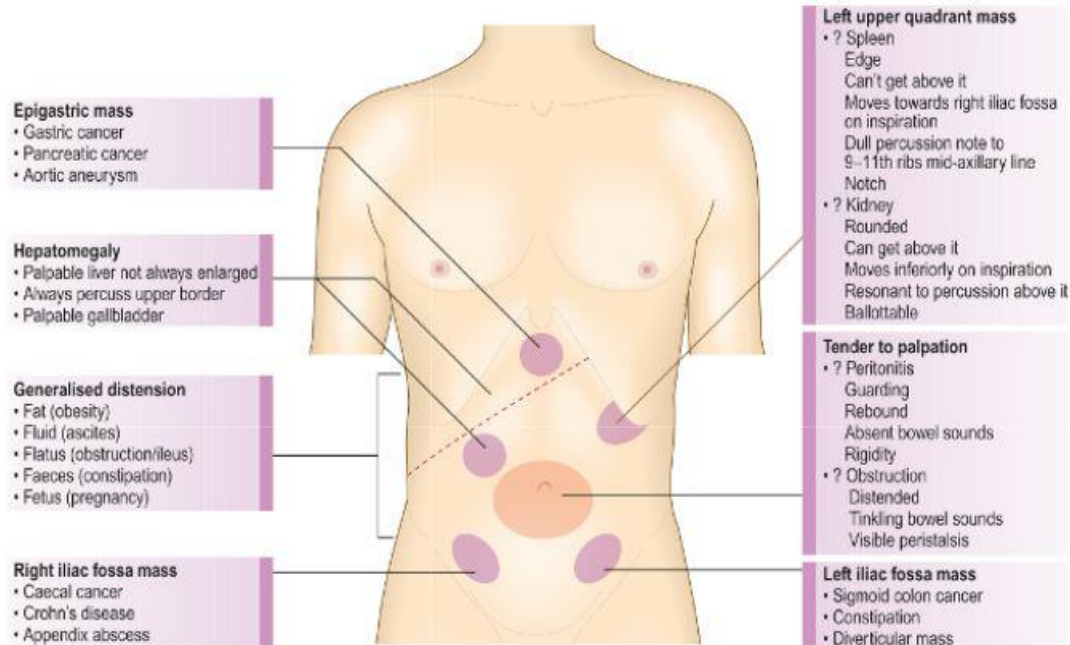
- a) Pain worse upon releasing pressure
- b) *Peritonitis*

3) temperature

4)Guarding (tighting of the pateints abdominal muscle)

- a) Voluntary→ voluntary contraction in the abdominal muscles when palpation (pressure)provokes pain
- b) Involuntary→ reflex contraction of the abdominal muscles when there is inflammation of the parietal peritoneum
- c) Ask pt to bend knees to relax abdominal muscles
- d) Note if localized or generalized
- i) Generalized (due to a perforated viscus)→ board-like rigidity w/ thoracic breathing

5) Masses :light/superficial ones are noted here .Ask the patient to tense his abdominal muscle to see the mass in the wall or deep . *If any mass is palpated*, describe: site, size, shape, surface, edge, temperature, tenderness, consistence, fluctuation, pulsations, regional lymph nodes, mobility/ fixity



3.11 Features to note in any lump or swelling (SPACESPIT)

- **S**ize
- **P**osition
- **A**ttachments
- **C**onsistency
- **E**dge
- **S**urface and shape
- **P**ulsation, thrills and bruits
- **I**nflammation
 - Redness
 - Tenderness
 - Warmth
- **T**ransillumination

3. Deep palpation

- “Pulsatile mass in upper abdomen”
 - Normal aortic pulsation in thin person
 - gastric/ pancreatic tumor transmitting an underlying aortic pulsation
 - AAA
- “A hard subcutaneous nodule palpable at the umbilicus”
 - may indicate metastatic cancer (‘Sister MaryJoseph’s nodule’)
- Faecal scybala may be palpable in the sigmoid colon in the left iliac fossa (normal)

- *Palpation for organs:*

Liver : *The liver can be palpable normally in 25% of the population, so a palpable liver edge does not equal hepatomegaly .The liver may be enlarged or displaced downwards by hyperinflated lungs .*

1. Begin palpation in the right iliac fossa using the flat edge of your hand (radial side of your right index finger)

2. Press your hand into the abdomen as you ask the patient to take a deep breath (press with your hand against the abdominal wall while the patient expires, then wait until the patient inspires. If the liver is enlarged its edge will hit your hand.)

3. If you feel the liver edge, note:

- a) Degree of extension below the costal margin
- b) edges(sharp /round)
- c)surface (*smooth/ irregular*)
- d)Consistency of the liver edge (soft /firm/hard)
- e) Tenderness (*hepatitis/ RHF*)
- f) Pulsatility (*tricuspid regurgitation*)

- | |
|--|
| <ul style="list-style-type: none">- If you don't feel liver edges :<ul style="list-style-type: none">1. Normal2. Shrunked liver- If you feel liver edges :<ul style="list-style-type: none">1. Enlarged2. Displaced (due to hyperinflated lung)- In both cases do liver span |
|--|

Palpation of the Gallbladder:

1. it is not usually palpable (only when gallbladder enlarged as a rounded mass moving w/ inspiration)
2. Perform palpation at the right costal margin, midclavicular line (9th rib tip).
3. An enlarged gallbladder suggests obstruction to biliary flow/infection (cholecystitis/ mucocele/pancreatic CA).
4. **Murphy's sign:**
 1. Place your hand in the right costal margin, midclavicular line
 2. Ask the patient to take a deep breath
 3. As the gallbladder is pushed down into your hand the patient may suddenly develop pain and stop inspiring.
 4. If this occurs and there is *no discomfort in the same location on the left side of the abdomen* then this is known as a positive Murphy's sign, which is suggestive of cholecystitis
5. **Courvoisier's sign:**
 1. in the presence of a palpable gallbladder which is nontender and accompanied with mild painless jaundice , the cause is *unlikely to be gallstones* .
 2. Probably pancreatic cancer

Spleen: Only palpable when it's 3x its normal size

1. *Start in right iliac fossa – massive splenomegaly can extend this far!*
2. *Align your fingers in the same direction as the left costal margin*
3. *Press your right hand into the abdomen as you ask the patient to take a deep breath*
4. *Feel for a step, as the splenic edge passes under your hand (a notch may be noted)*
5. *If you don't feel anything, repeat process with your hand 1-2 cm closer to the left hypochondrium*

Kidney: Only ballotable w/ cysts/ malignancy/ hydronephrosis

1. *Place your left hand behind the patient's back, at the right flank*
2. *Place your right hand just below the right costal margin in the right flank*
3. *Press your right hand's fingers deep into the abdomen*
4. *At the same time press upwards with your left hand*
5. *Ask the patient to take a deep breath*
6. *You may feel the lower pole of the kidney moving inferiorly during inspiration*
7. *Repeat this process on the opposite side to assess the left kidney*
(normally, the lower pole of the right kidney may be palpable in the right flank)

Palpation of the aorta:

1. Palpate using fingers from both hands

2. Palpate *just above the umbilicus* at the border of the aortic pulsation
3. Note the movement of your fingers:
 - **Upward** movement = **pulsatile**
 - **Outward** movement = **expansile** (suggestive of AAA)

Palpation of the bladder:

Empty→ not palpable Full (urinary retention)→ felt arising from behind the pubic symphysis

4. Percussion

- By using the right hand, and by moving only the wrist joint back and forth, percuss the nine quadrants by the middle finger. Normally the abdomen is tympanic due to the gas content of the intestine while Solid or fluid-filled structures are dull to percussion
- Assess the liver and spleen span by percussion
- An area of dullness in the pelvis below the umbilicus in the suprapubic region is usually a full bladder

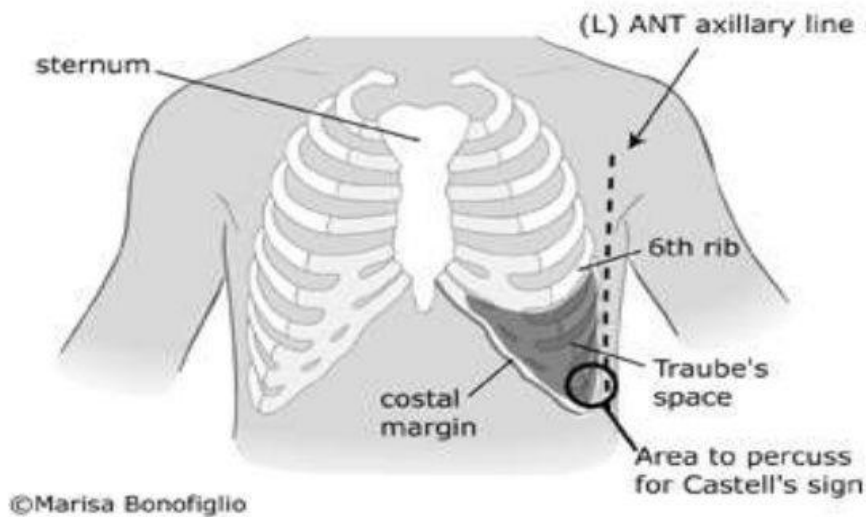
Percussion of the liver

- The normal liver is identified as an area of dullness to percussion over the right anterior chest between the fifth rib and the costal margin . (8-12 cm)
- percuss up from RIF(light percussion)then down from 2nd intercostal space midclavicular line (heavy percussion)
- Resonance below the fifth intercostal space suggests emphysema or occasionally the interposition of the transverse colon between the liver and the diaphragm (Chilaiditi's sign), or shrunken liver

Percussion of the spleen

(do it if there is splenic enlargement only)

- The normal spleen is identified as an area of dullness to percussion posterior to the left mid-axillary line beneath the ninth, 10th and 11th ribs.
- percuss up from RIF moving towards the left hypochondrium to assess for splenomegaly
- Normal→ resonant
- Traube's space→ dull in splenomegaly
 - A triangle between the left sixth rib superiorly, the left mid axillary line laterally, and the left costal margin inferiorly.



***One common question is : how can you differentiate between the spleen and kidney on examination ?!**

- *On palpation*: you can't get above the spleen while you may get above the kidney.
- *On palpation*: the spleen can't be palpated normally while the kidney is palpable by balloting.
- A kidney is ballotable ; the spleen is not.
- *On percussion*: the spleen is dull while the kidney lies behind the colon (retroperitoneal) and so it is tympanic.
- The spleen is characterized by its palpable splenic notch on the edge.
- There is splenic rub auscultation while kidney no

Feature	Spleen	Kidney
Mass is smooth & regular in shape	More likely	Polycystic kidney are bilateral irregular masses
Mass descends on inspiration	Yes , travels superficially and diagonally (early)	Yes , moves deeply and vertically (later)
Able to feel deep to the mass	Yes	No
Palpable notch on the medial surface	Yes	No
Bilateral masse palpable	No	Sometimes
Percussion resonant over mass	No	Sometimes
Mass extends beyond midline	Sometimes	No(except horseshoe kidney)
Balloted	No	Yes
Get above it	No	yes

Tests to detect ascites

(Causes include intra-abdominal malignancy, chronic liver disease, severe heart failure, nephrotic syndrome and hypoproteinemia.)

1. **Shifting dullness** → more reliable, + even with small amount of fluid, if its positive then this indicate moderate ascites (700-1000)cc
 - a) Percuss from the center of the abdomen to the flank until dullness is noted
 - b) Keep your finger on the spot at which the percussion note became dull
 - c) Ask patient to roll onto the opposite side to which you have detected the dullness
 - d) Keep the patient on their side for 30 seconds
 - e) Repeat your percussion in the same spot
 - f) If fluid was present (ascites) then the area that was previously dull should now be resonant
 - g) If the flank is now resonant, percuss back to the midline, which if ascites is present, will now be dull (i.e. the dullness has shifted)
2. **Fluid thrill** → more useful in gross ascites where the percussion note is dull all over
 - a) Ask the patient to put their hand on their abdomen.(to prevent transition of impulse via skin)
 - b) Place one hand flat on one side of the abdomen.
 - c) Tap the other side quickly and you may feel the shift of fluid with your flat hand.
 - d) The patient's hand prevents the force being simply transmitted through the subcutaneous fat.
 - e) If you still feel a ripple against your left hand this is a fluid thrill.
 - f) Positive fluid thrill indicate severe ascites

succussion splash : A sounds like shaking a half-filled water bottle. Shake the abdomen by lifting the patient with both hands under the pelvis. An audible splash more than 4 hours after the patient has eaten or drunk anything, indicates delayed gastric emptying, e.g. pyloric stenosis .

5. Auscultation

1) Bowel sounds (You should listen in two places)

- a) Best heard at McBurney's point (at the ileocecal valve)
- b) Normal: low-pitched gurgling, **3-12 per minute**, once every 5-10 seconds
- c) Abnormal: tinkling, with increased frequency and volume (*mechanical bowel obstruction*)
- d) Absent: (*functional bowel obstruction: ileus/ peritonitis*)
- i) Must listen for 2 additional minutes (total of 3 minutes) before concluding absence

2) Bruits

- a) Aortic bruits – auscultate just above the umbilicus (over aorta)– AAA, atherosclerosis
- b) Renal bruits – auscultate 2-3 cm superior and lateral to the umbilicus - renal artery stenosis
- c) Liver bruits(right hypochondrial) - may be heard in
 - i) acute alcoholic hepatitis
 - ii) hepatocellular cancer
 - iii) arteriovenous malformation

3) Abdominal succussion splash

- a) shake the patient's abdomen by lifting him with both hands under his pelvis, with stethoscope placed on epigastrium
- b) sounds like a half-filled water bottle being shaken.
- c) Indicates *gastric outlet obstruction*
- d) An audible splash more than 4 hours after the patient has eaten or drunk anything indicates delayed gastric emptying, e.g. *pyloric stenosis*.

4) Friction rub

- a) Sounds like rubbing your dry fingers together,
- b) may be heard over the liver (*perihepatitis*) or spleen (*perisplenitis*)

***DON'T forget to finish your examination without :**

- *PR*
- *Hernial Orifices and genitalia*
- *Supraclavicular Lymph Nodes*

Causes of hepatomegaly:

Physiological:

- Reidel's lobe
- Hyperexpanded chest

Infections:

- Viral: viral hepatitis, Epstein–Barr virus, cytomegalovirus
- Bacterial: tuberculosis, liver abscess
- Protozoal: malaria, histoplasmosis, amoebiasis, hydatid, schistosomiasis

Alcoholic liver disease:

- Fatty liver (can also be caused by diabetes mellitus)
- Cirrhosis (other causes of cirrhosis also lead to hepatomegaly but these are less common)

Metabolic diseases:

- Wilson's disease
- Haemochromatosis
- Cellular infiltration, e.g. amyloid

Malignant disease:

- Primary/secondary solid tumours (secondary are more common)
- Lymphoma
- Leukaemia

Congestive cardiac disease:

- Right heart failure
- Tricuspid regurgitation (causes a pulsatile liver)

Budd–Chiari syndrome.

 8.33 Causes of hepatomegaly	
Chronic parenchymal liver disease	
• Alcoholic liver disease • Hepatic steatosis • Autoimmune hepatitis	• Viral hepatitis • Primary biliary cirrhosis
Malignancy	
• Primary hepatocellular cancer	• Secondary metastatic cancer
Right heart failure	
Haematological disorders	
• Lymphoma • Leukaemia	• Myelofibrosis • Polycythaemia
Rarities	
• Amyloidosis • Budd–Chiari syndrome	• Sarcoidosis • Glycogen storage disorders

-In early cirrhosis the liver is enlarged , latter on it shrunken

-In cirrhosis ,liver is usually small .However , the liver enlarge if cirrhosis is due to hemochromatosis and primary biliary cirrhosis also in early stage of alcoholic cirrhosis

-budd-chiari syndrome : doesn't raise JVP and the hepatojugular reflux is -ve

While the congestive heart failure does

- In HCC >> you may hear audible bruit
- Right HF >> soft and tender liver
- TR >> pulsatile liver

Causes of splenomegaly:

Infective:

- Acute: Epstein–Barr virus, cytomegalovirus, HIV, infective endocarditis
- Chronic: toxoplasmosis, malaria, brucella, leishmaniasis, schistosomiasis

Haematological disease:

- Haemolytic anaemia
- Myeloproliferative disorders (especially myelofibrosis)
- Sickle cell disease/thalassaemia
- Leukaemia (especially chronic myeloid leukaemia, CML)
- Lymphoma

Portal hypertension:

- Cirrhosis
- Hepatic, portal or splenic vein thrombosis

Systemic diseases:

- Amyloidosis
- Sarcoidosis
- Rheumatoid arthritis



8.37 Causes of hepatosplenomegaly

- Lymphoma
- Myeloproliferative diseases
- Cirrhosis with portal hypertension
- Amyloidosis, sarcoidosis, glycogen storage disease

Causes of tender hepatomegaly:

Infective- viral hepatitis,
infectious mononucleosis
leptospirosis
amoebiasis

congestive- CCF

Budd-chiari synd

misc-hepatoma,hydatid cyst

• ***Massive splenomegaly***

- chronic myeloid leukemia
- myelofibrosis
- gaucher disease
- lymphoma
- Kala-azar (visceral leishmaniasis)
- malaria
- beta-thalassemia major
- AIDS with mycobacterium avium complex



Chapter 3: Gastroenterology and Hepatology



History Taking

General Considerations

Whenever you take a history, try to divide it into the following parts:

- 1- History of Present Illness:
 - a) General questions that should be asked to all patients with a certain symptom.
 - b) Asking relevant questions that will either support or refute a certain differential diagnosis.
- 2- Past medical history and medications:
Ask about previous diseases, drugs or risk factors that might be associated with the differential diagnosis.
- 3- Social history and family history.

So, in the history station you have to bear these things in mind:

- Be wide! Don't focus your questions on one differential diagnosis. Even uncommon entities causing the symptom in the question are listed in the checklist of the examiner.
- Know the system involved in the station. If you get lost, ask about all symptoms, risk factors, or any associations related to that system.
- Don't memorize checklists or questions. Try to understand the differential diagnoses and formulate your own way of approaching symptoms.

Don't study history taking disease by disease, study it chief complaint by chief complaint.

Dysphagia

OSCE Vignette: A 56-year-old man presented with difficulty swallowing. He is losing weight because he is unable to eat. Obtain a detailed history trying to know the cause of his swallowing problem.

Dysphagia is difficulty swallowing. The most important thing in the history is to know whether it is due to oropharyngeal or esophageal problem, and to know whether it comes with solids, liquids or both.

General Questions:

- Always ask for **clarification** (Do not confuse dysphagia with early satiety, the inability to complete a full meal because of premature fullness, or globus, the feeling of a lump in the throat. Globus does not interfere with swallowing and is not related to eating).
- Dysphagia is an **alarming GI symptom**, so it always indicates a serious pathology. **Always investigate it.**



8.8 Symptom checklist in dysphagia

- Is dysphagia painful or painless?
 - Is dysphagia intermittent or progressive?
 - How long is the history of dysphagia?
 - Is there a previous history of dysphagia or heartburn?
 - Is the dysphagia for solids or liquids or both?
 - At what level does food stick?
 - Is there complete obstruction with regurgitation?
-
- Dysphagia is either (1) **Oropharyngeal**: bulbar palsy, pseudobulbar palsy, myasthenia gravis, and pharyngeal pouch) or (2) **Esophageal**: esophageal dysphagia can be (a) structural (i.e. dysphagia due to complete or partial occlusion of the esophageal lumen) or (b) dysmotility (i.e. impairment of peristalsis).
 - **How to differentiate between these types of dysphagia based on the history?**
 - **Was there difficulty swallowing solids, liquids or both?**
 - Only liquids: Neurological → If it's neurological, ask about diplopia, tremor, and weakness.
 - Both: Motility disorder (e.g achalasia, CNS, or pharyngeal causes).
 - Solids then liquids: suspect a stricture (benign or malignant).
 - **Is it difficult to initiate a swallowing movement?**
 - Yes: Suspect bulbar palsy, especially if patient coughs on swallowing.
 - **Is swallowing painful (odynophagia)?**
 - Yes: Suspect ulceration (malignancy, esophagitis, viral infection or Candida in immunocompromised, or poor steroid inhaler technique) or spasm.
 - **Is the dysphagia intermittent or is it constant and getting worse?**

- Intermittent: suspect esophageal spasm.
- Constant and worsening: suspect malignant stricture.

- **Does the neck bulge or gurgle on drinking?**

- Yes: Suspect a pharyngeal pouch.

- **Ask about red flag symptoms:**

- 1- Weight loss
- 2- Loss of appetite
- 3- Hematemesis, melaena
- 4- Progressive and persistent

- **Past medical history:**

GERD, PUD, scleroderma, iron deficiency.

- **Medications:**

Taking pills without water (erosive esophagitis), use of antacids (related to GERD and PUD).

- **Social history:**

Smoking, alcohol intake (risk factors of esophageal cancer and GERD).

- **Family history:**

Family history of esophageal cancer, neuromuscular diseases.



8.7 Causes of dysphagia

Oral

- Tonsillitis, glandular fever, pharyngitis, peritonsillar abscess
- Painful mouth ulcers

Neurological Oropharyngeal

- Bulbar or pseudobulbar palsy
- Cerebrovascular accident

Liquids more than

Neuromuscular Dysmotility **Solids and Liquids**

- Achalasia
- Myasthenia gravis
- **Systemic Sclerosis**

Mechanical Structural

- Oesophageal cancer
- Peptic oesophagitis
- Other benign strictures, e.g. after prolonged nasogastric intubation
- Extrinsic compression, e.g. lung cancer



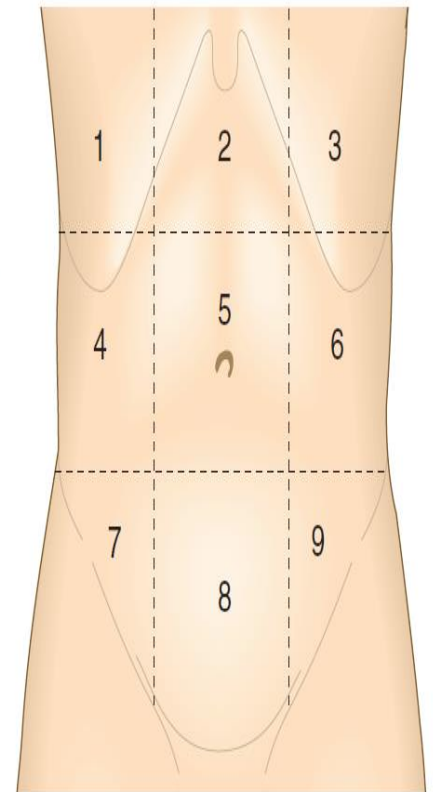
Checklists

Introduces himself, gains consent	
Ask for clarification	
Onset (how it started): • Sudden • Gradual	
• Character: • Fluids • Solids or both	
Time: Duration • Intermittent, continuous, progressive	
Level: where does food/liquid feel like it is getting 'stuck'	
• Exacerbating or relieving factors	
Define at which stage the dysphagia occurs: When initiating swallowing • After swallowing has been initiated	
• Pain (esophageal/abdominal), odynophagia	
• Trauma, foreign body	
• Feeling of a lump in the throat	
• Previous episodes of dysphagia	
• Family members/contacts with similar	
'Red flags': Weight loss Loss of appetite Hematemesis, melaena Progressive and persistent	
Past medical history: • Stroke • Thyroid problems • PUD and GERD	
Family history: • Upper gastrointestinal tract cancer	
Drug history: • NSAIDs • Bisphosphonates	
Social history: • Alcohol (peptic ulcer disease, gastritis) • Smoking • Illicit drug use • Diet: spicy foods (peptic ulcer disease)	

Abdominal Pain

Differential diagnosis of acute abdominal pain based on the site

- cholecystitis/cholangitis (1)
- biliary colic (1, 2)
- hepatitis (1, 2)
- pneumonia (1 or 3)
- peptic ulcer disease/gastritis (2)
- acute coronary syndrome (2)
- pancreatitis (2, 5)
- ruptured abdominal aortic aneurysm (AAA) (2, 5)
- splenic rupture (3, diffuse)
- renal calculus (4, 7 or 6, 9)
- pyelonephritis (4 or 6)
- early appendicitis (5, diffuse)
- established appendicitis (7)
- terminal ileitis, e.g. Crohn's disease, *Yersinia* (7)
- mesenteric adenitis (7, diffuse)
- diverticulitis (7 or 9)
- colitis (7, 8, 9)
- ectopic pregnancy (7, 8, 9)
- pelvic inflammatory disease/endometriosis (7, 8, 9)
- ovarian torsion/cyst rupture (7, 8, 9)
- lower urinary tract infection (UTI)/cystitis (8)
- intestinal obstruction (diffuse)
- perforation (diffuse)
- mesenteric ischaemia (diffuse)
- gastroenteritis (diffuse mid-/upper abdominal)
- diabetic ketoacidosis/hypercalcaemia/adrenal crisis (diffuse)
- functional abdominal pain (any region or diffuse).



- **History of Present Illness:**

- Ask about SOCRATES of pain.
- Associated symptoms: (this depends on the type of abdominal pain described by SOCRATES).

- ❖ **Gastrointestinal/colorectal symptoms:**

- Nausea/vomiting • Bowel habit, diarrhoea/constipation • Flatulence
- Fevers • Dysphagia • Dyspepsia
- Bloating/abdominal swelling (generalized/localized)
- IBD symptoms: arthralgia, eye symptoms, skin features, oral ulcers, bloody diarrhea.

- ❖ **Liver/hepatic symptoms:**

- Right upper quadrant pain • Jaundice • Ankle swelling

❖ Gallstone symptoms:

- Jaundice
- Right upper quadrant pain radiating to shoulders
- Dark stools
- Pale urine

❖ Renal symptoms:

- Location and character:
- Loin to groin + flank + colicky: renal stones
- Flank + burning dysuria: pyelonephritis
- Generalized lethargy
- Pruritus
- Ankle swelling

If the patient is female:

❖ Gynecological symptoms

- Correlation with menstrual periods
- Menorrhagia
- Irregular periods
- Vaginal discharge

❖ Obstetric symptoms

- Possibility of patient being pregnant
- Last menstrual period
- Contraception
- Vaginal bleeding (with severe abdominal pain = ectopic pregnancy until proven otherwise).

1

8.6 Non-alimentary causes of abdominal pain

Disorder	Clinical features
Myocardial infarction	Epigastric pain without tenderness Angor animi (feeling of impending death), hypotension, cardiac arrhythmias
Dissecting aortic aneurysm	Tearing interscapular pain Angor animi, hypotension Asymmetry of femoral pulses
Acute vertebral collapse	Lateralised pain restricting movement Tenderness overlying the involved vertebra
Cord compression	Pain on percussion of thoracic spine Hyperaesthesia in dermatomal distribution Spinal cord signs
Pleurisy	Lateralised pain on coughing Chest signs, e.g. pleural rub
Herpes zoster	Hyperaesthesia in dermatomal distribution Vesicular eruption
Diabetic ketoacidosis	Cramp-like pain, vomiting, air hunger Tachycardia, ketotic breath
Salpingitis or tubal pregnancy	Suprapubic and iliac fossa pain, localised tenderness, nausea, vomiting, fever

1

8.5 Diagnosing abdominal pain

	Disorder			
	Peptic ulcer	Biliary colic	Acute pancreatitis	Renal colic
Site	Epigastrium	Epigastrium/right hypochondrium	Epigastrium/left hypochondrium	Loin
Onset	Gradual	Rapidly increasing	Sudden	Rapidly increasing
Character	Gnawing	Constant	Constant	Constant
Radiation	Into back	Below right scapula	Into back	Into genitalia and inner thigh
Timing				
Frequency/periodicity	Remission for weeks/months	Able to enumerate attacks	Able to enumerate attacks	Usually a discrete episode
Special times	Nocturnal and especially when hungry	Unpredictable	After heavy drinking	Following periods of dehydration
Duration	½–2 hours	4–24 hours	>24 hours	4–24 hours
Exacerbating factors	Stress, spicy foods, alcohol, non-steroidal anti-inflammatory drugs (NSAIDs)	Unable to eat during bouts	Alcohol Unable to eat during bouts	
Relieving factors	Food, antacids, vomiting		Eased by sitting upright	
Severity	Mild to moderate	Severe	Severe	Severe



Checklists

Introduces himself, gains consent	
Ask for clarification	
History of Present Illness: • Site • Onset (how it started): - Sudden - Gradual • Character: - Colicky (renal stones) - Sharp/sudden (rupture of viscus) - Burning (peptic ulcer disease) - Dull • Radiation: - To back (abdominal aortic aneurysm, ruptured duodenal ulcer) - To testicles/groin (hernia) - To shoulders (gallbladder) - Loin to groin (renal stone) • Time: Duration/ Intermittent, continuous, progressive. • Alleviating factors: Dietary factors, relieved by bowel motion?, relieved by a certain position (lying on one side, or leaning forward), relieved by abstinence from food?. • Exacerbating factors: Dietary factors, increased by swallowing? (esophagus/stomach), fatty foods (gallstones), acidic/spicy foods, hot drinks (peptic ulcer disease). - Does it increase by movement or breathing? • Severity (from 0-10)	
Associated symptoms: As described above.	
Red Flags of acute abdominal pain: Weight loss, bleeding (upper GI bleed or lower GI bleed), loss of appetite.	
Past medical and surgical history: • Previous surgeries • Hepatitis, or history of blood transfusions • IBD, IBS.	
Social history: Smoking, alcohol intake	
Medications: NSAIDs, antacids, use of laxatives.	
Family history: Ask about relevant conditions related to the history (IBD, colon cancer ... etc.).	

Irritable Bowel Syndrome:

Rome III criteria	Rome IV criteria
At least 3 months, with onset at least 6 months previously of recurrent (at least 3 days/month) abdominal pain or discomfort associated with 2 or more of the followings	Recurrent abdominal pain, on average, at least 1 day per week in the last 3 months, associated with 2 or more of the followings
Improvement with defecation	Related to defecation
Onset associated with a change in frequency of stool	Associated with a change in frequency of stool
Onset associated with a change in form of stool	Associated with a change in form (appearance) of stool

Abdominal Distension

OSCE vignette: A 55-year-old gentleman presented with a 2-month history of weight loss and increased abdominal girth. Examine him looking for causes of abdominal distension.

Here, you have to examine the patient as if you are examining for ascites.

-Examination steps are found at the end of this chapter-



8.11 Causes of abdominal distension

Factor	Consider
Fat	Obesity
Flatus	Pseudo-obstruction, obstruction
Faeces	Subacute obstruction, constipation
Fluid	Ascites, tumours (especially ovarian), distended bladder
Fetus	Check date of the last menstrual period
Functional	Bloating, often associated with irritable bowel syndrome

Hematemesis

- Hematemesis is vomiting of blood from the GI tract. It indicates upper GI bleed.
- Upper GI bleed could be:
 - a) **Active bleeding**: presents with **bright red blood or clots**.
 - b) **Modest bleeding** or bleeding that **has ceased**: presents with **coffee-ground blood** (blood gets degraded by gastric pepsin and becomes dark and granular just like coffee).
- ➔ Ask about the **character** of bleeding to know whether it's active or old.
 - Upper GI bleed is any bleeding that occurs above the ligament of Treitz (above the duodenojejunal junction). So, blood may come from:
 - 1- Esophagus: esophageal varices, Mallory-Weiss tear, esophagitis.
 - 2- Stomach: PUD, gastritis, gastric cancer.
 - 3- Duodenum: duodenitis.
 - Each of these differentials has a characteristic history that you should be familiar with;
- PUD: Usually presents with a history of epigastric pain and hematemesis. There is an association with H. pylori, NSAIDs, steroids and alcohol.
- Mallory-Weiss tear: Presents with hematemesis that was preceded by forceful retching with non-bloody vomit.
- Cancer: Associated with weight loss and generalized weakness and fatigue.

History of Present Illness:

• General questions about the chief complaint:

- Ask about the character of the blood, quantity, and history of previous episodes.
- Ask if there's bleeding from other sites.
- Duration.

• Ask relevant questions to rule in/out differential diagnoses:

- **Esophageal:**
 - ✓ Esophageal Varices → Does the patient have a history of chronic liver disease/cirrhosis? History of previous hematemesis or varices? Does he/she have ascites or lower limb edema?
 - ✓ Esophagitis → Does the patient have a history of heartburn (GERD)?
 - ✓ Mallory-Weiss tear → was the bleeding preceded by retching without blood? Was the onset preceded by binge alcohol drinking?



8.18 Symptom checklist in haematemesis and melaena

- Is there a previous history of dyspepsia, peptic ulceration, gastrointestinal bleeding or liver disease?
- Is there a history of alcohol, NSAIDs or corticosteroid ingestion?
- Did the vomitus comprise fresh blood or coffee ground-stained fluid?
- Was the haematemesis preceded by intense retching?
- Was blood staining of the vomitus apparent in the first vomit?

- **Gastric:**
- ✓ PUD → Was it preceded by epigastric pain, or discomfort? NSAIDs? Steroids?
If there's pain, ask whether it was relieved by food antacids or not?
Was that pain awakening you at night?
- ✓ Gastric cancer → Weight loss? Early satiety? Anorexia? Dysphagia?

Past medical and surgical history:

PUD, GERD, liver disease, coagulopathy, esophageal varices, previous GI bleed, previous GI surgery, AAA repair (aortoenteric fistula).

Medications:

NSAIDs, steroids, anticoagulants (ask about them in any OSCE stations with bleeding), aspirin -ask about aspirin as specifically even if you have asked about NSAID-.

Family history:

Ask about family history of coagulopathy and GI malignancy.

Social history:

Smoking and alcohol intake.



8.19 Causes of upper gastrointestinal bleeding

- | | |
|---------------------------------------|---------------------------------|
| • Gastric or duodenal ulcer | • Oesophagogastric varices |
| • Mallory–Weiss oesophageal tear | • Oesophageal or gastric cancer |
| • Oesophagitis, gastritis, duodenitis | • Vascular malformation |



Checklists

Introduces himself, gains consent	
Asks for clarification (distinguishes hematemesis from hemoptysis)	
• Details about the chief complaint: 1- Volume: If large volume, patient needs to be assessed and resuscitated immediately. 2- Number of episodes 3- Character/color - Coffee grounds - Dark clots - Fresh, bright red - Mixed with vomitus 4- Onset (what brought it on): - Medications, alcohol - Vomiting/retching (Mallory–Weiss tear) - Sudden -esophageal varices- 5- Precipitating factors: - Alcohol → Mallory-Weiss tear - NSAIDs - Trauma to abdomen 6- Previous episodes of hematemesis	
• Associated symptoms: - Melena - Abdominal/chest pain - Symptoms of shock (faintness, shortness of breath) - Bleeding, bruising elsewhere, epistaxis - Anemia (fatigue, dyspnea on exertion) - Dysphagia, odynophagia - Vomiting - Liver/hepatic symptoms: • Right upper quadrant pain • Jaundice • Ankle swelling and ascites - Weight loss.	
• PMH: GERD, PUD, liver problems, coagulopathy.	
• Family history: coagulopathies, cancer	
• Medications: NSAIDs, steroid, aspirin, warfarin.	
• Social history: Smoking, alcohol	

Jaundice (yellowish discoloration of tissues (skin, mucus membranes & sclera))

History

1) Prehepatic jaundice:

If bilirubin is not taken up by the liver or if it is produced in excess, unconjugated bilirubin is deposited in extra-hepatic tissues.

History findings:

- Weakness (a consequence of Anemia if present).
- Icterus.
- normal urine and stool color.

Common causes: RBC disorders, Hereditary spherocytosis, Sickle cell anemia, Auto-immune, Mismatched blood transfusion, Infective Sepsis and Malaria.

2) Hepatic jaundice:

Disorders of bilirubin uptake or conjugation either congenital or acquired.

History findings:

- Weakness.
- Anorexia.
- Icterus.
- dark urine color.
- normal stool color.

Common causes

Congenital: Gilbert's syndrome, Crigler-Najjar Syndrome, Dubin-Johnson Syndrome and Rotor syndrome.

Acquired: *Impaired Hepatic Bilirubin Uptake* (CHF, Portosystemic shunts, Drug inhibition: rifampin, probenecid), *Impaired Bilirubin Conjugation* (Neonatal jaundice, liver disease, hyperthyroidism), *Impaired Excretion* (Hepatitis, Primary biliary cirrhosis, Sepsis, Drugs/toxins...).

3) Obstructive jaundice:

Failure of bilirubin to reach the gut

History findings:

- Icterus.
- Dark urine color
- Pale stool color.
- **Pruritus(itching)**

Common causes: Choledocholithiasis, Cancer/Neoplasm: Pancreatic CA, Cholangiocarcinoma (rare), Gallbladder CA, Metastatic CA., Strictures after invasive procedures, Acute and chronic pancreatitis, Primary sclerosing cholangitis (PSC), Parasitic infections.

*In addition to the four main questions (Icterus, urine & stool color and pruritus),

	Icterus	Stool color	Urine color	pruritus
Prehepatic	+	Normal	Normal	-
Hepatic	+	Normal	Dark	-
Obstructive	+	Pale	Dark	+

The following should be asked about for patient presenting with jaundice :

- ✓ *Urine and stool color : is your urine dark ? are your stools pale ? (obstructive jaundice)*
- ✓ *Skin itching*
- ✓ *Appetite and weight loss. (malignancy)*
- ✓ *Signs of inflammation: Fever and chills as in cholangitis.*
- ✓ *Abdominal pain (RUQ pain).*
- ✓ *altered bowel habit.*
- ✓ *GI bleeding (vomiting of blood or passage of dark stool)*
- ✓ *Hepatitis B or C risk factors: Blood transfusion, sexual and contact history and skin tattooing, immunized against hepatitis B or not.*
- ✓ *Hepatitis A risk factors: travel to endemic areas and immunization history.*
- ✓ *Drugs (Paracetamol, chlorpromazine, methyldopa.) and alcohol history.*
- ✓ *Family History: Inherited disorders including liver diseases and hemolytic conditions (congenital spherocytosis, haemochromatosis...).*
- ✓ *Past medical history (biliary surgery, pancreatitis, hepatitis.).*
- ✓ *Have you had any recent contact with patients with jaundice or liver problems*
- ✓ *Surgical history: Have you had any surgeries (eg:pancreatic or biliary?)*
- ✓ *Occupation: what is the occupation (contact with hepatotoxin?)*

Physical examination

1) General assessment of the patient

- Jaundice, pallor, cyanosis and obvious distress (in pain or not).
- Confusion and diminished mental state → signs of liver failure.
- Pale lemon complexion (anemia + Icterus) → hemolytic causes.
- Cachexia → malignancy with liver metastases.

2) Start from the hand

- Finger clubbing, palmar erythema and leukonychia → signs of chronic liver disease.
- Flapping tremor of outstretched hands → signs of liver failure.
- Pallor at palmar creases → sign of hemolytic anemia.
- Needle track marks, tattoos and body piercing → evidence of drug misuse.
- Scratch marks of itching → sign of obstructive jaundice

3) Face and neck

- Pallor in the conjunctiva
- Smell breath for fetor hepaticus or alcohol → signs of liver failure.

4) Chest and abdomen

- Spider naevi, gynaecomastia → signs of chronic liver disease.
- Hepatomegaly → hepatic jaundice.
- Splenomegaly → prehepatic jaundice (hemolytic causes) .
- Ascites (shifting dullness) and periumbilical caput medusa.
- Palpable painless gallbladder + obstructive jaundice = Courvoisier's Law → pancreatic CA or distal cholangiocarcinoma.

5) Examine ankles and sacrum for edema → hypoalbuminaemia.

6) Certain findings suggest specific diseases such as:

- Hyper-pigmentation → hemochromatosis.
- A Kayser-Fleischer ring in the eye → Wilson's disease.
- Xanthomas → primary biliary cirrhosis.



Checklists

• Introduces himself, gains consent	
• History of present illness: - How was the jaundice discovered – did the patient notice it, or was it someone else? - Onset (what brought it on, how it started) - Time: • Duration • Intermittent (e.g. Gilbert’s syndrome), continuous, progressive • Fevers • Previous episodes of jaundice • Family members/contacts with similar symptoms	
• Associated symptoms: - Gallstones, biliary duct obstruction: Abdominal pain, pale stools, itching, steatorrhea, dark urine - Liver symptoms: Abdominal swelling, ankle swelling, bleeding, bruising (liver failure and impaired synthetic function) - Autoimmune conditions: Arthralgia, vitiligo, skin rashes (systemic lupus erythematosus) - Risk factors for viral hepatitis • Hepatitis B and C: Contaminated needles, intravenous drug abuse, blood transfusions, tattoos. • Foreign travel/contacts	
• PMH: previous jaundice, hemolytic anemias (sickle cell anemia and thalassemia), IBD (linked to PSC)	
• Family history: any family member with similar disease (may indicate acute viral hepatitis)	
• Medications: Hepatitis B immunization, antibiotics, statins, acetaminophen, anti-TB medications.	
• Social history: alcohol intake, IV drug abuse.	

Details about Important GI Diseases

<u>Wilson's disease (rare)</u>	<u>Hemochromatosis (rare)</u>	<u>Celiac disease</u>
➤ Chief complaint/ duration ➤ Deposition of copper in liver and basal ganglia ➤ Symptoms: Tremor, dysarthria, slow movement, dementia, behavioural changes, vision problems, abdominal distension, dysphagia, LL edema, joint problems, fatigue. ➤ Physical exam: 1. ask the patient to talk (look for dystonia, dyarthria) 2. test posture 3. look for memory (say 3 names and let him repeat) 4. tremor (rest or intension) 5. kayser fischer rings in cornea (green brown) 6. chronic liver disease examination	➤ Patient's profile (40-60: congenital, 10-30: juvenile), acquired: iron ➤ Chief complaint/ duration ➤ Deposition of iron in skin , liver , heart ➤ Symptoms: Vertigo, memory changes, behavioural changes, dizziness, fatigue, weakness, impotency, sexual dysfunction, amenorrhea, changes in breast size (increase in males, decreases in females), cold intolerance, arthralgia, abdominal swelling, lower limb swelling. ➤ Past: thalassemia, sickle cell anemia, any disease that require chronic blood transfusion, DM, chronic liver disease ,surgical ➤ Drugs: Iron ➤ Family: bleeding tendency, similar condition ➤ Social: smoking, sex, alcohol ➤ Physical: chronic liver disease exam, decreased pulses, skin bronze, testicular atrophy, arthralgia, heart and thyroid exam	➤ Chief complaint/ duration ➤ Hypersensitivity to gluten (wheat products), abdominal pain, bloating, nausea, vomiting, fatigue, weight loss, weakness, steatorrhea, osteoporosis (decrease in height), osteomalacia, vit. Deficiency ➤ Past: cancer ➤ Drugs ➤ Family ➤ Social
Labs: Liver enzymes, liver biopsy, decreased serum ceruloplasmin	Labs: Elevated serum iron and ferritin, decreased TIBC, liver biopsy	Labs: Biopsy of proximal small bowel
<u>Crohn's disease (regional enteritis)</u>	<u>UC</u>	<u>IBS</u>
➤ Chief complaint 'abdominal pain with diarrhea' / duration ➤ Symptoms: Diarrhea (usually bloody), abdominal pain (RLQ), nausea, vomiting, malabsorption, weight loss, fever, malaise. ➤ Extraintestinal features: uveitis, episcleritis, ankylosing spondylitis, sacroiliitis, monoarticular migratory arthritis, DVT, PE, erythema nodosum, pyoderma gangrenosum, aphthous oral ulcers, cholelithiasis, nephrolithiasis(stones) ➤ Complications: fistula, abscess, anorectal disease, malignancy, malabsorption of vit. B12 and bile acids, stones. ➤ involvement is transmural 'the whole wall', skip lesions, all GI, Smokers	➤ Chief complaint 'hematochezia, bloody diarrhea' / duration ➤ Symptoms: Hematochezia, abdominal pain, small bowel movements, fever, anorexia, weight loss in severe cases, tenesmus (rectal dry heaves) ➤ Extraintestinal symptoms: jaundice, uveitis, arthritis, skin lesions. ➤ Complications: iron deficiency anemia, haemorrhage, electrolyte disturbances, dehydration, cancer, sclerosing cholangitis, growth retardation ➤ mucosa and submucosa, no skip lesions, sites are colon and rectum, smoking is a preventive factor.	➤ Chief complaint ' intermittent abdominal pain for 6 months' with alteration of bowel habits/ duration ➤ Symptoms: site of pain (lower abdomen), onset and character (colicky or cramping), exacerbation and relieving (defecation), alternating bowel habits (for diarrhea ask about blood or mucus, for constipation ask about hardness). ➤ Cover all other symptoms (it's a diagnosis of exclusion): fever, chills, rigors, fatigue, weight loss, abdominal distension, back pain, stress, depression , anxiety, heat intolerance , polyuria, tenesmus, bloating, dyspepsia, lactose intolerance, dysmenorrhea. ➤ Past: gastroenteritis, surgical resection ➤ Drugs: laxatives, iron, opioids ➤ Family: cancer ➤ Social: smoking, alcohol, stress, sex

Budd-Chiari syndrome

Summary

Budd-Chiari syndrome is a rare condition resulting from hepatic vein obstruction that leads to hepatomegaly, ascites, and abdominal discomfort. It is most commonly due to a thrombotic occlusion secondary to a chronic myeloproliferative neoplasm (e.g., polycythemia vera), but may be caused by other conditions associated with hypercoagulable states. The obstruction of blood flow causes congestion of the liver with subsequent liver cell damage. If left untreated, it may result in progressive liver failure. Doppler ultrasound confirms the diagnosis. Management involves preventing further clotting with anticoagulation therapy, restoring blood flow with radiologic or surgical procedures, and treating the underlying condition.

Etiology

- Secondary to conditions associated with **hypercoagulability** (most common) ^[1]
 - **Polycythemia vera** (most important cause)
 - Paraneoplastic thrombocytosis
 - Pregnancy and postpartum period
 - Clotting disorders (e.g., factor V Leiden thrombophilia, antiphospholipid syndrome)
 - Chronic inflammatory diseases (e.g., Behcet's disease)
 - Side effect of medication (e.g., hormonal contraception) ^[2]
- Secondary to invasion or compression of the hepatic veins (less common)
 - Hepatocellular carcinoma, renal carcinoma
 - Chronic infections and hepatic lesions (e.g., amebiasis, aspergillosis, syphilis, tuberculosis)

Pathophysiology

- **Obstruction** (e.g., due to thrombosis or compression) of hepatic veins → ↓ blood outflow → hepatic venous congestion → increased sinusoidal pressure, cellular hypoxia, centrilobular necrosis → **congestive hepatopathy**
- May develop into “**nutmeg liver**”: Because of ischemia and fatty degeneration, the tissue appears speckled with dark spots.
- Clinical symptoms depend on the extent of hepatic injury and portal hypertension.

Clinical features

- **Abdominal pain**
- **Tender hepatomegaly**
- **Ascites** and abdominal distention
- Jaundice
- Signs of increased perfusion of portocaval anastomoses (e.g., esophageal varices, or caput medusae)
- In severe cases: ankle edema, splenomegaly, and renal impairment

In contrast to congestive heart failure, which can also cause hepatic congestion, Budd-Chiari syndrome does not lead to jugular venous distension.

Diagnostics

- **Blood analysis:** often nonspecific findings; possible elevated aminotransferases
- **Ascites fluid analysis**
 - WBC count $< 500 /\mu\text{L}$
 - Serum ascites albumin gradient > 1.1
- **Imaging** (confirmatory test): A visible occlusion of the hepatic vein confirms the diagnosis.
 - Doppler ultrasound
 - CT, MRI with contrast

Treatment

- Treat the underlying disease
- Anticoagulation (to prevent propagation of the thrombus)
- **Restore blood flow**
 - Localized thrombolysis
 - Balloon angioplasty and stenting
- TIPS (transjugular intrahepatic portosystemic shunt) if complications of portal hypertension occur
- Liver transplantation in the event of liver failure

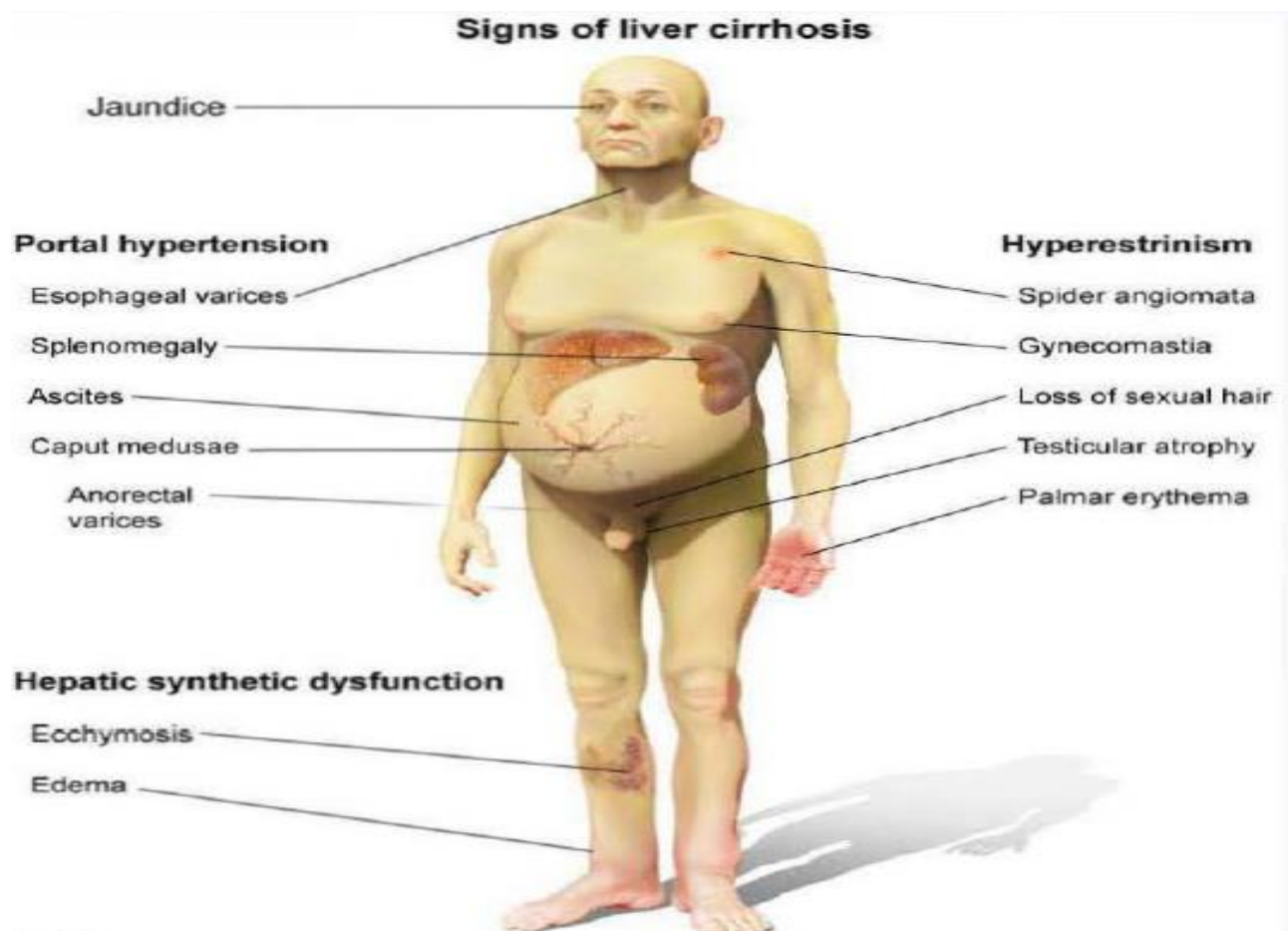
Complications

- Portal hypertension
- Acute liver failure
- Cirrhosis
- Hepatocellular carcinoma (HCC)

Chronic Liver disease

OSCE Vignette: A 62-year-old gentleman presented with a change in mental status. He was an alcoholic and an IV-drug abuser in his twenties, and was diagnosed with hepatitis B after that. Examine this patient looking for signs of chronic liver disease.

- All types of chronic liver disease will ultimately lead to liver cirrhosis.
- Liver cirrhosis causes four types of signs:
 1. Signs related to impaired liver function (decreased synthesis of albumin> edema, clotting factors> easy bruisability... etc.).
 2. Signs related to hyperestrogenism (impaired breakdown of estrogen causes gynecomastia, spider angiomas, loss of sexual hair, testicular atrophy, and palmar erythema).
 3. Signs related to portal hypertension.
 4. Signs related to hepatic encephalopathy and liver failure (asterixis and fetor hepaticus)



Q: What are the stigmata or signs of CLD?

A: As follows:

1. In the face:

- Parotid enlargement (bilateral).
- Xanthelasma (common in PBC).
- Spider angioma (also in neck, arm, forearm, hand and any part above the nipple line).
- Pigmentation.
- Hepatic facies.
- Jaundice.
- Cyanosis (in hepato-pulmonary syndrome).

2. In hands:

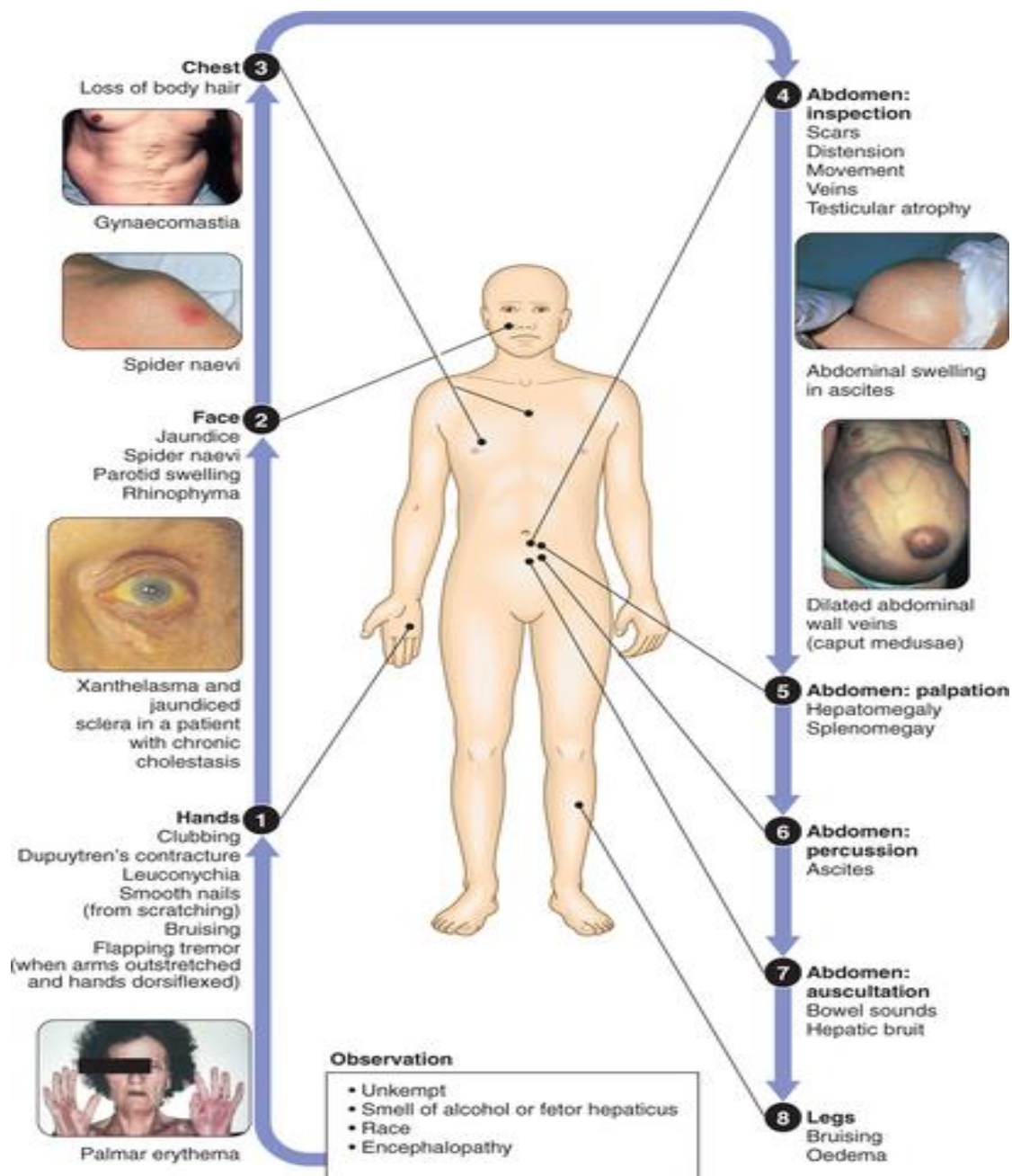
- Palmar erythema (also called liver palm).
- Dupuytren's contracture.
- Leuconychia.
- Clubbing.
- Flapping tremor.
- Others: scratch marks, spider angioma, xanthoma, pigmentation, jaundice and cyanosis.

3. Chest:

- Gynaecomastia (also spider angioma).
- Less hair in chest or body, scanty axillary (also pubic hair).
- Engorged veins in chest and also abdomen (due to portal hypertension).

4. Others:

- Abdomen: ascites, splenomegaly, engorged veins and caput medusae.
- Testis (small and atrophied).
- Generalized pigmentation (any CLD can cause pigmentation, but commonly in haemochromatosis and primary biliary cirrhosis).
- Purpura and ecchymosis



Thyroid examination

Hatem Rjoob

General thyroid Examination

Introduction - Permission - Environment (light/temp/privacy) - Position -Exposure - Hygiene

- Position , the patient should be setting on a chair with enough space behind him to be able to examine him
- Exposure , from nipples to all upward

First impression

- The patient is conscious, alert and oriented to time, place and person.
- Do they seem abnormally hyper- or hypoactive?
 - o Hyperthyroidism: restless and agitated
 - o Hypothyroidism: slow motion and apathy
- Note if there is hoarseness or stridor after talking to the patient
- Describe facial expression (apathy/startled) & abnormal ticks.
- Clothing (any suggestion of heat or cold intolerance)
- Obese or cachectic(cachectic as in malignancy)
- Obvious tubes or drains

-Hand shake (temp and sweat; cold & dry in hypothyroidism, sweaty & warm in hyperthyroidism.)

Vital signs

- Pulse:
 - o Hyperthyroidism: resting tachycardia, there may be irregular irregularity (A Fib).
 - o Hypothyroidism: bradycardia and first degree heart block

Test for collapsing pulse (for Graves).

- Respiratory rate
- Blood pressure: wide pulse pressure(hyperthyroidism)
 - o hyperthyroidism >> isolated systolic HNT
 - o hypothyroidism >> diastolic HNT
- BMI:
 - o Hypothyroidism: increased.
 - o Hyperthyroidism: decreased.
- Temperature

Hands

- **Palms:**

- o Hyperthyroidism: Sweaty and warm. Look for **palmar erythema**
- o Hypothyroidism: Cold and dry. May be coarse, yellow and cold due to peripheral vasoconstriction, thenar muscle wasting.(thenar muscle wasting >>carpal tunnel syndrome in hypothyroidism)

- **Thyroid Acropachy:** tender wrist, clubbing and periosteal bone formation. Found in *Graves*

- Acropachy :
 - digital clubbing
 - swelling of digits and toes
 - & periosteal reaction of extremity bones

- **Fine Tremor** in *hyperthyroidism*: ask the patient to extend their arms . You may place a paper over the patient's hand to make it more visible.

- **Onycholysis** in *hyperthyroidism*.

Onycholysis is the medical term for when your nail separates from the skin underneath it.

- vitiligo +/- hyperpigmented borders (Grave's Dz)

Face and Eyes

1-Inspect for:

- Texture of hair (dry-coarse in hypothyroidism and greasy-sweaty and diffuse thinning of hair in the scalp in hyperthyroidism)
- Hair loss (occurs in both) or eyebrow thinning (outer 1/3 in hypothyroidism its sign of Hertoghe) its also called queen anne's sign "loss of the outer 1/3 of the eyebrow's" (hypo)

2. **Exophthalmos:** examine from above or the lateral side, sclera is visible all over (graves)

3.**inspect sclera for jaundice** (if there is an autoimmune disease affecting the liver and the thyroid , or from thyroid drug)(use the sunlight not the torch)

4. **upper Lid retraction:** the sclera is visible **above** the superior corneal limbus , its called dalrymple's sign (Grave's Dz)

5. **Ophthalmoplegia:** ask the patient to follow the letter H; test for diplopia and nystagmus. "paralysis or weakness of the eye muscles that affect mostly superior recti and inferior oblique" positive in graves

6. **Lid lag:** ask the patient to follow your finger or an object moving down vertically. Positive when a rim of sclera is visible between the upper eyelid and the superior iris on downward gaze.(because of the delay in the eyelid) Occurs in *hyperthyroidism.it occur due to sympathetic hyperstimulation*

7-**mobius sign:** lack of convergence on looking to near object due to weak medial recti muscle (graves)

8- **Joffroys sign:** The patient able to look up without any frontal wrinkles (head should be fixed)(graves)

9- Large tongue >>hypothyroidism

o Corneal Ulcer: due to prolonged eye opening and infrequent blinking>>hyperthyrodism

o Conjunctivitis

o Chemosis (redness and edema of eye globe)>>hyper

o Periorbital edema >>hypothyroidism

Local Thyroid examination

Inspection

From the front of the patient, your eyes at the levels of the patient's neck, inspect the following starting with positive findings:

- Symmetry
- swelling: Masses (Site, Size, Shape, Surface, Skin changes, Scars over it, Pulsation, Moving with protrusion of the tongue or swallowing of water)
- Scars (Site, length, width, orientation, healing, skin changes etc...)
- superficial Dilated veins
- skin changes: Hyper/Hypo pigmentations, Discoloration of the skin (redness, bluish etc..)
- When asking the patient to swallow water, ask him to hold then swallow
- When describe the site of neck mass, identify in which triangle, and its relation to fixed anatomical structure (SCM "3 thirds", Mastoid, Sternoclavicular joint, clavicle, suprasternal notch, mandibular angle, Submental notch, Hyoid bone, Midline)

- size: measure about ...bycm in diameter.
- shape: if diffuse (butterfly) if localized (oval, round) ☐ more extend to right side or left side of neck.
- surface: nodular or smooth
- skin changes (Vein engorgement, visible pulsations, edema, redness, hyper/hypopigmentations and sinus.)
- **superficial veins**:- may occur 2nd to comparison of thyroid swelling on SVC
- **Sinus (fistula)**:- its occur in top of infected thyroglossal cyst.
- **Skin discoloration** (redness) signifies inflammation
- scars (neck collar scar at the crease of the neck indicates previous thyroid surgery, **if present look for sign of hypocalcaemia (Chvostek's and Trousseau's signs)**, describe (Site, length, width, orientation, healing, skin changes) of the scar.

-special character: Perform Four Maneuvers, ask the patient to:

1) **Swallow** (you may give them some water) and observe any abnormalities. Ask the patient to have some **sips of water**, hold it in his/her mouth, and then swallow. Observe if lump moves, if so it's likely originated from the thyroid. This is because the thyroid, cricoids, the larynx are enclosed by the pretracheal fascia which moves upon swallowing.

2) **Protrude their tongue**. Ask the patient to extend the neck, open the mouth and protrude his/her **tongue** as far as possible, and observe if a lump moves. If this happens, the lump is likely a **thyroglossal cyst** because persistence of the thyroglossal duct is attached to the base of the tongue by a fibrous track through the hyoid (also thyroglossal fistula will move up and down when protruding the tongue)

3) Open their mouth and use the torch to check for a **lingual thyroid**.

e.g.:- there is single symmetrical swelling in anterior triangle of neck measures about 4 by 6cm in diameter butterfly in appearance with smooth surface and I can't determine any abnormality in skin over mass this mass is move up and down with swallowing and I can't recognize other mass in neck.

Palpation:

Explain the exam, warm your hands and ask if there is any pain & maintain eye-to-eye contact throughout palpation .

From front: (neck should be slightly extended)- temp(use the dorsum of the hand and compare it with surrounding structure), tenderness and tracheal deviation .

From back:(The neck should be slightly flexed)- first be ensure Patient has glass of water in his hand then put neck of the patient in natural position to relax muscle of neck,(**Please examine each lobe alone, don't move both hands at same time.** Fix one hand on one lobe to stabilize it and palpate the other lobe.)

-(4S , CEA , M)>> site , size , shape , surface , constancy , edges , attachments , mobility

-**Number, site, size** [measure it with tape measure], **shape, surface, consistency**(firm ,soft ,Hard),**Edges** (lower border if you can get below it or no, by palpating the tracheal ring ,regular or not), **attachment to skin & sternomastoid** [by asking the patient to push his head against your hand then feel for the other side. **mobility**(ask the patient to swallow ,and try to move the mass in vertical and horizontal direction . fixity in any direction suggests malignant infiltration or thyroiditis) (if its single nodule in the gland then comment on its location in the gland (lobe/isthmus)).in the thyroid **nodule** the cyst feels firm and solid swelling(adenoma) feels soft because adenoma is highly cellular?!!!

-Comment on whether there is a palpable **thrill**

-do **special character** :ask the pt to drink water and to protrude his tongue while palpating

-palpate the carotid artery pulsation against the transvers process of the cervical vertebra .between the posterior Border of the thyroid and SCM. In unilateral goiter ,first palpate the normal side for normal feel of carotid pulsation and then compare it with the affected side . In benign goiter, the pulsation will always be felt ,though displaced backward. BUT, in a malignant goiter ,carotid pulsation is weak or absent due to malignant infiltration of the carotid sheath(**Berry Sign positive : an absence of a carotid pulsation in malignant thyroid**)

NOTE: when you palpate the carotid ,**don't** do it bilaterally at the same time .if the pt has carotid stenosis then bilateral palpation can cause fainting(syncope),also you should do it gently to avoid dislodgment an atheroma (that maybe found in the pt carotid artery)

- **Kocher test** ask the pt to extend his/her neck and to take deep breathing with open mouth .now compress the swelling from both sides . Appearance of stridor when you press the lateral lobes >>this suggest scabbard trachea (positive kocher test). (it occur in large ,long standing multinodular goiter and malignant thyroid infiltrating the trachea)

-palpate LN:

-Continue with the “**up and down technique**”. Start from chin and proceed backwards to below ears, palpating the submental, submandibular, parotid and preauricular nodes. Palpate post-auricular (mastoid) nodes behind the ears and then continue down the ant. border of the SCM and feel ant. triangular nodes, such as the jugulodigastric node (tonsillar). Move laterally along the clavicles feeling for both supra and infraclavicular nodes. Return up the post. border of the SCM feeling for post. triangular nodes. Complete by palpating occipital nodes on the back of the neck

-Remember **LIST** as the most common causes of Cervical Lymphadenopathy

L= lymphoma and leukemia

I=infection either bacterial (b-hemolytic strep., TB), viral (CMV, EBV, HIV), protozoal and Toxoplasmosis

S=sarcoidosis

T=tumors

-Remember also **MIAMI** as the most common causes of Cervical Lymphadenopathy

- **M= Malignancies**
 - Leukemias
 - Lymphoma
 - Kaposi sarcoma
 - Metastasis
- **I = infection**
- **A= autoimmune** : SLE , RA , dermatomyositis , Sjogren syndrome
- **M= miscellaneous** : Kawasaki , sarcoidosis ,histiocytosis x
- **I = Iatrogenic**
 - Allopurinol
 - Antibiotic (eg: penicillins , trimethoprim/sulfamethoxazole)
 - Antihypertensive (hydralazine , captopril)

Notes :

*** In the case of lymphadenopathy :(site , size , constancy , tenderness , attachment)**

- firm or rubbery

- solitary and may be multiple

- fixed with skin in cases of malignancy or tuberculous nodule

In percussion:

1. do it over manubrium of sternum, note (do it in all cases even tracheal wright was palpable) to detect retrosternal extension of goiter: Becomes dull if a goiter extends retrosternal or if the whole thyroid is displaced.
2. percuss medial parts of clavicles

In auscultation:

1. with diaphragm over the thyroid lump try to hear any Bruit which is present in graves as a result of hypervascularity
2. also on carotid artery to hear if there is Bruit (result if there is compression from the lump on the carotid or when there is atherosclerosis in the artery)
3. Bruit over the thyroid is characteristic of Graves' disease. To reduce transmission, ask the patient to:
 - Hold his breath (ask the pt to take deep inspiration and Hold it)
 - Place your hand on the root of the neck to reduce transmission from the jugular vein

o Deep tendon reflexes

- Hyperthyroidism: Hyper-reflexia
- Hypothyroidism: Delayed relaxation

o Proximal Myopathy(ask the patient about weakness when go upstairs and do squatting many times):

in hypothyroidism >> high ck , normal ESR

in hyperthyroidism >>normal to high ck

Thank your patient

Differential Diagnoses:

Midline Mass	Anterior Triangle and SCM	Posterior Triangle
-thyroid swelling -thyroglossal cyst -submental lymphadenopathy -dermoid cyst (<i>teratoma</i>) -laryngocele (<i>common in glassblowers and saxophone players</i>) -paramedian lobe of thyroid tract (<i>80% on left</i>) -delphian node (<i>enlarges in thyroid disease, it drains the thyroid and larynx</i>) -prehyoid bursitis -lipoma -cartilage tumor -thymus -CA of larynx, trachea or esophagus -sebaceous cyst -isthmus	-lymphadenopathy (<i>see below</i>) -chemodectoma (<i>solid paraganglioma of chemoreceptor tissue</i>) -branchial cyst (<i>epithelial cyst due to failure of fusion of 2^{ry} branchial cleft</i>) -cold abscess (<i>aka collar-stud abscess 2^{ry} to TB</i>) -carotid aneurysm -parathyroid tumor -SCM tumor (<i>seen in the 1st 2 weeks of life after a complicated delivery, disappearing in 4-6 months</i>)	-lymphadenopathy (<i>see below</i>) -pharyngeal pouch (<i>aka zenker's diverticulum, it's the #1 diverticulum of the esophagus, cystic in nature</i>) -cystic hygroma (<i>#1 lymphangioma in pediatrics, its transilluminable</i>) -clavicle tumor -subclavian aneurysm

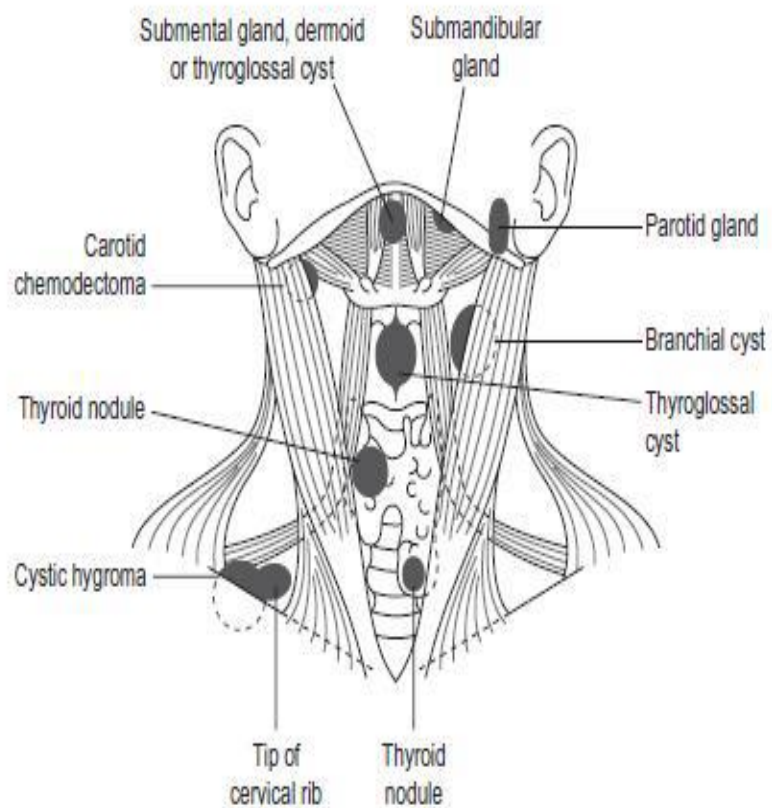


Figure 2 Locations of the most common swellings in the neck.

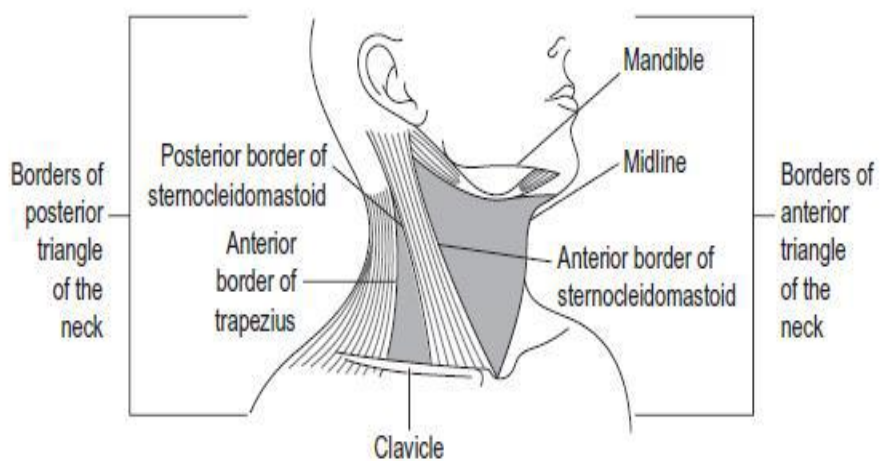


Figure 1 Posterior and anterior triangles of the neck.

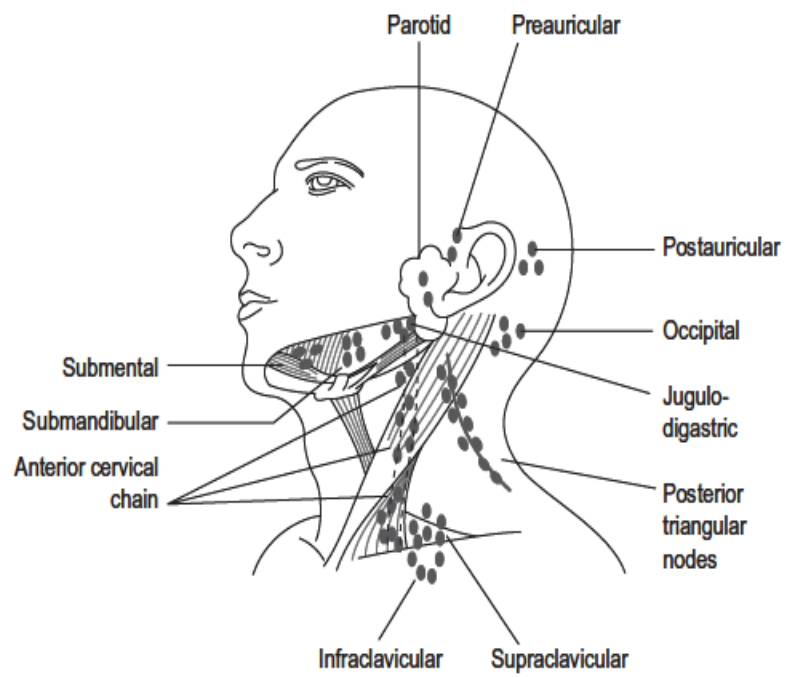


Figure 4 Typical grouping of lymph nodes.

Neck regions in details

-Before initiating a neck examination, make sure you understand the cervical regions.

1) SCM Regions:

The SCM is a key muscular landmark as it divides each side of the neck into anterior and lateral segments. The SCM has 2 heads: **sternal head** (attached to manubrium by anterior surface) and **clavicular head** (attached to superior surface of medial 1/3 of clavicle). The SCM is superiorly attached to the mastoid process. Contents include: SCM muscle, Greater Auricular Nerve, Transverse Nerve of neck and superior part of Ext. Jugular Vein.

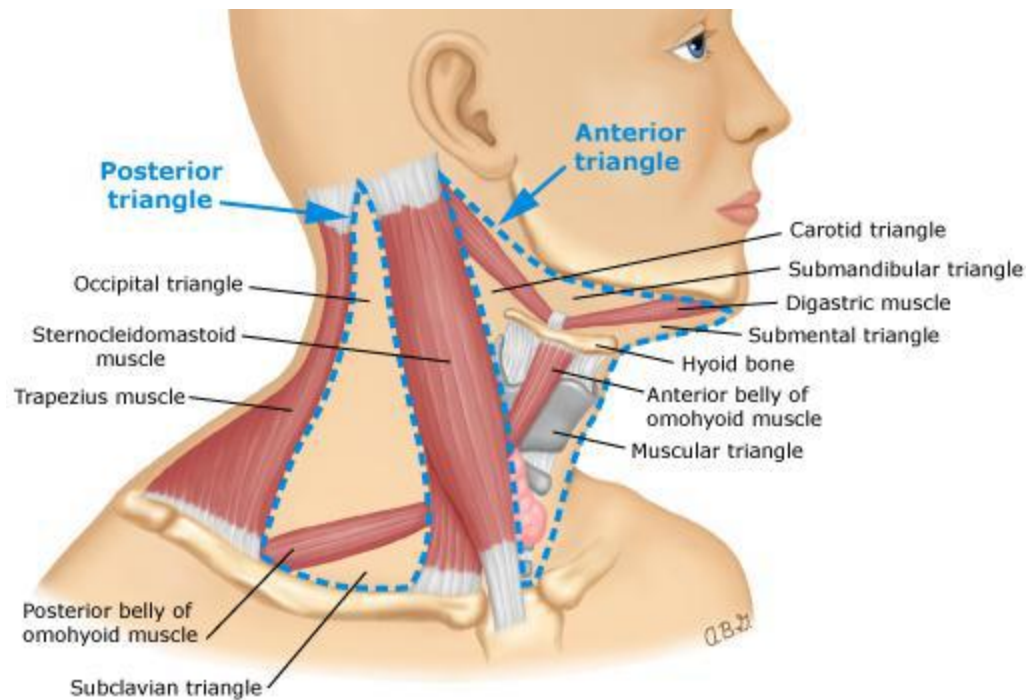
2) Anterior Triangle (Anterior cervical region):

The anterior triangle is bound anteriorly by the **median line**, posteriorly by the ant. border of the **SCM**, superiorly by the inf. border of the **mandible**. The floor is formed by the pharynx, larynx and thyroid. The **digastric** and **omohyoid** muscles divide the triangle further into 4 regions.

- a) **Submental Triangle:** bound inf. by body of the hyoid and laterally by the ant. bellies of the digastrics muscles. The submental triangle contains the submental lymph nodes and anterior jugular veins.
- b) **Submandibular Triangle (digastrics triangle):** bound by mandible and ant. + post. Bellies of digastrics muscle. It contains the submandibular gland and lymph nodes. The hypoglossal nerve and part of the facial artery and vein.
- c) **Carotid Triangle:** bounded by sup. belly of omohyoid, post. digastrics belly and SCM. The carotid triangle contains the common carotid artery, external carotid artery, internal jugular vein, vagus nerve, carotid body and sinus.
- d) **Muscular Triangle (omotracheal triangle):** bound by sup. belly of omohyoid, SCM and median plane. Contains thyroid and parathyroids.

3) Posterior Triangle (lateral cervical region):

Bound by SCM, Trapezius and middle 1/3 clavicle inferiorly (between trapezius and SCM). The posterior triangle contains part of the trapezius muscle. The inferior belly of the **omohyoid** muscles divides it into occipital and **omoclavicular** triangles. The occipital triangle contains the external jugular vein and cervical lymph nodes. The omoclavicular triangle (Supraclavicular triangle) contains the subclavian artery (3rd part), subclavian vein, supraclavicular artery and lymph nodes.



Note :subclavian triangle = supraclavicular triangle =omoclavicular triangle = Ho's triangle

- Remember how to deal with any mass.

- 1) Inspect by verifying **site, size, shape, skin changes, symmetry, scars and color.**
- 2) Palpate to characterize the **surface** (smooth.irregular), **edge** (well/ill defined), consistency (soft/firm/hard), **temperature** (use dorsal surface of hand), **tenderness**, **transillumination**, **pulsatility** (expansile/transmitted), **compressibility**, **fluctuation** (use 2 fingers of same hand), **fixation** (to skin/underlying tissue), **mobility** (in what planes), and **fluid thrill**.
- 3) Percussion (dull/resonance)
- 4) Auscultation

signs specific to Graves

- exophthalmos
- pretibial myxedema
- Thyroid bruit
- Clubbing
- ophthalmoplegia

hypothyroidism

- Apathetic face
- Bilateral ptosis
- Loss of eyebrows laterally (Hertoge's sign)
- Periorbital edema

1. Hyperthyroidism can cause :
AFib , Heart failure so causing
S3 and pulmonary edema .it
also can cause collapsing pulse
and ejection systolic murmur
due to hyperdynamic circulation
2. Pre-tibial mxedema is an
autoimmune phenomena >> so
can occur in Hashimoto and
graves
3. Peritibial myxedema is a non-
pitting edema , but can cause
pitting edema if caused heart
failure

Types of thyroid malignancies	
Epithelial (thyroid follicular cells) (90%-95%)	• Papillary (>70%) • Follicular • Anaplastic
Parafollicular C-cells (3%-4%)	• Medullary thyroid cancer
Other cells (<5%)	• Lymphoma • Sarcoma • Metastatic (renal, breast, melanoma, colon)

Differential diagnosis of myopathy			
Disorder	Clinical features	ESR	CK
Glucocorticoid-induced myopathy	- Progressive proximal muscle weakness & atrophy without pain or tenderness - Lower extremity muscles are more involved	Normal	Normal
Polymyalgia rheumatica	- Muscle pain & stiffness in the shoulder & pelvic girdle - Tenderness with decreased range of motion at shoulder, neck & hip Responds rapidly to glucocorticoids	↑	Normal
Inflammatory myopathies	- Muscle pain, tenderness & proximal muscle weakness Skin rash & inflammatory arthritis may be present	↑	↑
Statin-induced myopathy	- Prominent muscle pain/tenderness with or without weakness - Rare rhabdomyolysis	Normal	↑
Hypothyroid myopathy	- Muscle pain, cramps & weakness involving the proximal muscles - Delayed tendon reflexes & myoedema - Occasional rhabdomyolysis Features of hypothyroidism are present	Normal	↑

CK = creatine kinase; **ESR** = erythrocyte sedimentation rate.

MRC Muscle Power Scale

Score	Description
0	No contraction
1	Flicker or trace of contraction
2	Active movement, with gravity eliminated
3	Active movement against gravity
4	Active movement against gravity and resistance
5	Normal power

Notes :

***Goiter**

- grade 0 : non palpable or visible
- grade 1 : palpable
- grade 1A : detected on palpation only
- grade 1B : palpable and visible with neck distended
- grade 2 : visible with neck in normal position
- grade 3 : visible large goiter visible from a distant

***Thyroid eye dz classification**

Class 0	N	No signs or symptoms
Class 1	O	Only signs of upper lid retraction and stare, with or without lid lag and exophthalmos
Class 2	S	Soft-tissue involvement
Class 3	P	Proptosis
Class 4	E	Exophthalmos
Class 5	C	Corneal involvement
Class 6	S	Sight loss due to optic nerve involvement

*** differences between Toxic multinodular goiter and Graves' Dz**

Toxic multinodular goitre	Graves' disease
Older age-group	Younger age-group
Nodular enlargement	Diffuse enlargement
Eye signs not present	Eye signs present
Atrial fibrillation present in 40% of patients	Atrial fibrillation uncommon
No associated autoimmune diseases	Autoimmune diseases commonly associated

*** In case of simple diffuse goiter**

- diffuse enlargement
- non tender
- may deviate the trachea
- may be retrosternal ex.

*** Graves Dz**

- more common in female
- immunoglobulin against TSH receptors
- hyper with goiter
- thyroid eye dz
- acropachy
- pretibial myxoedema
- normochromic normocytic anemia , raised ESR , hypercalcemia
- associated with other autoimmune dzs such as type 1 DM

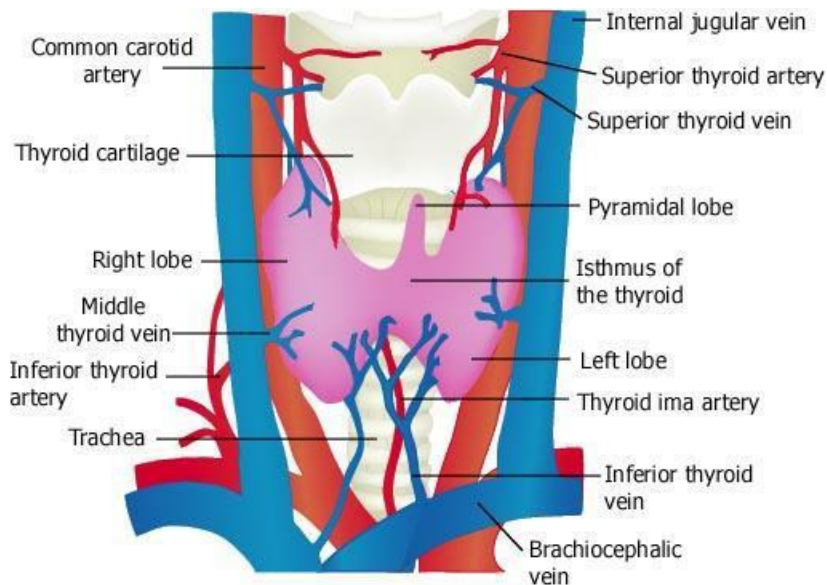
*** 5 Ms for surgery**

- Mechanical problems such as obstruction
- Malignancy
- Marred beauty – cosmetic
- Medical treatment failure
- Mediastinal extension (unable to perform FNAC)

*** Complications of thyroidectomy**

- Immediate (24 h) : hemorrhage , hoarseness , hyperthyroidism
- Early (30 d) : infection , hypoparathyroidism , hypocalcemia .
post operatively , if hypocalcemic – Chvosteks and trousseueau's signs are positive .
- Late (after 30 d) : hyperthyroidism – recurrence , hypothyroidism , hypertrophic scarring .

* Arterial Supply of the thyroid



Blood supply:

1) Superior Thyroid artery:

-The 1st branch of the external carotid artery.

-It is related to the external laryngeal nerve (when injured, it leads to loss of high pitched voice & voice fatigue).

2) Inferior Thyroid artery (inverted L-shaped course):

From the thyro-cervical trunk which is a branch of 1st part of subclavian artery. Its terminal branches near the gland are in close relation to recurrent laryngeal nerve (in between, above or below terminal branches).

3) Thyroid ima artery:

From the arch of aorta or innominate artery (present in 1-3% of people) (severe bleeding during thyroidectomy).

4) Accessory Tracheal & Esophageal arteries:

In ligament of Berry's; which is a thickened part of the pretracheal fascia that joins trachea to the thyroid gland.

Venous Drainage

1. Superior Thyroid vein: to internal jugular vein (or common facial vein).

2. Middle Thyroid vein: crosses common carotid to join internal jugular vein (middle thyroid vein is the shortest vein; hence it should be the first vein to be tied & cut).

3. Inferior Thyroid veins (10-12 veins): pass from the isthmus over the front of the trachea to join the left innominate vein.

Nerve supply

1-sympathetic from superior and middle cervical

2-parasympathetic from the vagus, via branches of the laryngeal nerve

Vagus >>>> superior laryngeal and recurrent laryngeal nerve. Each of them has internal and external branches.

A.recurrent laryngeal nerve

- The nerve on the Rt. side turns around the 1st part of Rt. subclavian artery & on the left side turns around the arch of the aorta.

-The nerve runs in groove between the trachea & esophagus in close to the terminal branches of the inferior thyroid artery (in between, above or below it)

- It enters the larynx at the inferior horn of thyroid cartilage.

-it supplies sensory to the mucous membrane below the vocal cords

-it also supplies all intrinsic muscle of the larynx except cricothyroid muscle

Surgical importance:

o The nerve might be non recurrent in small percent of cases (2%) & in this situation it might be injured with ligation of **the middle thyroid vein**.

o Ligation of inferior thyroid artery should be performed away from the gland to the nerve.

Injury to RLN

Unilateral >>>>> hoarseness

Bilateral >>>>> airway obstruction

-if the injury is bilateral, airway obstruction will result. This requires emergency intubation or tracheostomy.

-if the nerves are intact and the injury is temporary, recovery usually occurs in (3-6) months

-if the injury is permanent, it will require either a permanent tracheostomy or lateral fixation of arytenoid

B.superior laryngeal nerve

1-external laryngeal nerve

-arises from the superior laryngeal n. of the vagus.

-It passes in close relation to superior thyroid artery .

It supplies:

- Cricothyroid muscle (responsible for high pitched voice – tenses vocal folds)

Injury : causes loss of high pitched voice.

2-internal laryngeal nerve

Sensory innervation to mucous membrane of the larynx above the vocal cord .

If injured will cause choking (يتشردق من السوائل)

Surgical Importance:

Superior thyroid artery should be ligated as near as possible to the gland to avoid injury of the nerve.

- o The lymph nodes around the pyramidal lobe are called Delphian lymph nodes
- o The ligament that connects the thyroid to the trachea is the ligament of Berry
- o The most posterior extension of the lateral thyroid lobes is called the tubercle of Zuckerkandle

Hand and Wrist Exam

Hatem RJOOB

A. Introduction

- Wash hands
- Introduce yourself
- Confirm pt details
- Explain the exam
- Gain consent
- Ask if pt has any pain
- Exposure and position Expose the hands, wrists and elbows(Position their hands on a pillow)

B. Inspection of the dorsum

Hand posture

- Note any abnormal posture (e.g. contracture)

Scars or swelling

- Scars→ *previous surgery*
- Swelling, comparing the hands and wrists

Skin colour

- Erythema of the soft tissue may indicate *cellulitis* or *joint sepsis*
- Pallor of the hands may indicate the presence of *peripheral vascular disease* and/or *anaemia*

Deformities

1. Bouchard's nodes:

- Occur at the proximal interphalangeal joints (PIPJ)
- Associated with osteoarthritis (OA)

2. Heberden's nodes:

- Occur at the distal interphalangeal joints (DIPJ)
- Associated with OA

3. Swan neck deformity:

- Occur at the distal interphalangeal joint (DIPJ)
- Features include DIPJ flexion with PIPJ hyperextension
- Associated with rheumatoid arthritis

4. Z-thumb:

- Hyperextension of the interphalangeal joint , in addition to fixed flexion and subluxation of the metacarpophalangeal joint (MCPJ)
- Associated with rheumatoid arthritis

5. Boutonnières deformity:

- PIPJ flexion with DIPJ hyperextension
- Associated with rheumatoid arthritis

Skin changes

- Skin thinning or bruising can be associated with long-term steroid use (e.g. common in patients with active inflammatory arthritis)
- Psoriatic plaques (salmon colored plaques with silvery scale)

Muscle wasting

- Muscle wasting can occur secondary to chronic joint pathology
- Consider lower motor neuron lesions (e.g. carpal tunnel syndrome)

Nail changes

- **Nailfold vasculitis** is a feature of **rheumatoid arthritis** (small vessel vasculitis)
 - Nail pitting and onycholysis are associated with psoriasis (and psoriatic arthritis)
 - Clubbing
 - Ridging: longitudinal lines over nail surface.
 - Hyperkeratosis: - thickening of nail.
 - Splinter hemorrhage as in SLE case. **D/D** (trauma , infective endocarditis , SLE , vitamin C def)
 - Periungual erythema (dilated capillary loop)>>> SLE.
- If you identify any clinical signs, note if they are symmetrical (e.g. affecting both limbs) or not.

Fig. 6.2 Causes of finger clubbing

System	Causes
Cardiac	Endocarditis Cyanotic heart disease (e.g. Down syndrome, right to left shunts) Atrial myxoma
Respiratory	Lung cancer Bronchiectasis Fibrosis Cystic fibrosis Mesothelioma Empyema
Gastrointestinal	Inflammatory bowel disease (e.g. Crohn's disease, ulcerative colitis) Lymphoma Malabsorption (coeliac disease) Cirrhosis
Thyroid disease (uncommon)	Called thyroid acropachy
Idiopathic	No known cause
Unilateral clubbing (very rare)	Axillary artery aneurysm; brachial arteriovenous malformation



Figure 10. Fingernails affected by psoriasis: (a) pitting; (b) onycholysis

C. Inspection of the palms

hand posture

- Note any asymmetry of the hands
- Note abnormal hand posture (e.g. *clawed hand* secondary to *Dupuytren's contracture*)

Scars or swelling

- Scars → previous surgery
- Swelling → comparing the hands and wrists

Skin colour

- Erythema of the soft tissue may indicate cellulitis or joint sepsis
- Pallor of the hands may indicate the presence of peripheral vascular disease and/or anaemia
- Pale hand creases at Hb < 7

Deformity

- **Dupuytren's contracture** presents as a thickening or painless nodule in the palm
- The most common finger to be affected is the **ring finger** ; the thumb and index finger are much less often affected
- In advanced disease, there is a **loss of range of motion** in the affected fingers



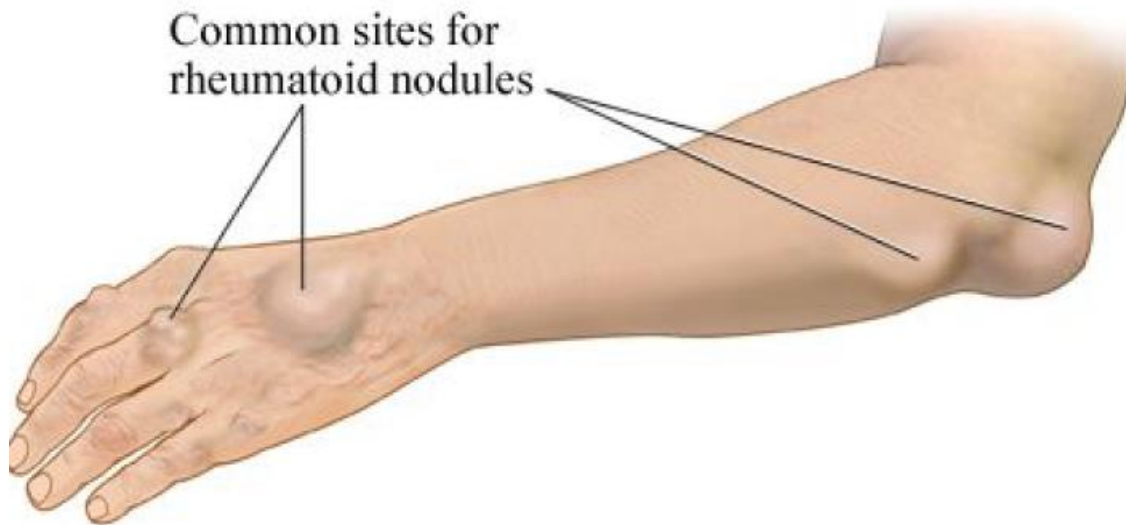
Thenar/hypothenar wasting

- Isolated wasting of the thenar eminence is suggestive of carpal tunnel syndrome



D. Inspection of the elbows

- Psoriatic plaques
- Rheumatoid nodules



E. Palpation of the palms

Temperature

- Check the wrists and small joints of the hand
- Increased warmth in a joint is suggestive of **inflammatory arthritis** or **joint sepsis**

Radial and ulnar pulse

- Confirm there is adequate blood supply to the hand

Thenar/hypothenar eminence bulk

- Wasting of these areas is often noted in ulnar/median nerve lesions

Palmar thickening

- Can be caused by Dupuytren's contracture

Assess median and ulnar nerve sensation

- Median→ thenar eminence and index finger
- Ulnar→ hypothenar eminence and little finger

F. Palpation of the dorsum

Assess radial nerve sensation

- Over the first dorsal web space

Assess and compare temperature using the back of your hand

- Forearm
- Wrist
- MCP joints

Gently squeeze across the metacarpophalangeal (MCP) joints

- Observe for non-verbal signs of discomfort
- Tenderness may indicate active inflammatory arthropathy



Bimanually palpate the joints of the hand (MCPJ/PIPJ/DIPJ/CMCJ)

Assess and compare joints for tenderness , irregularities and warmth :

-squaring of the CMCJ is associated with OA



Palpate the anatomical snuffbox

-Tenderness may suggest *scaphoid fracture*



Bimanually palpate the patient's wrists

-Feel for evidence of joint line *irregularities* or *tenderness*



Elbows

- Palpate the elbow
- Palpate the arm along the ulnar border to the elbow
- Note any rheumatoid nodules or psoriatic plaques (extensor surface)



G. Movements:

Must be done actively first (by the pt) then passively, feeling for crepitus and noting any pain (ROM→ range of motion)

- **Finger extension** – “Open your fist and splay your fingers”
- **Finger flexion** – “Make a fist”
- **Wrist extension** – “Put the palms of your hands together and extend your wrists fully” – ROM 90°
- **Wrist flexion** – “Put the backs of your hands together and flex your wrists fully” – ROM 90°

Motor assessment

(quickly assess the motor function of the radial, ulnar and median nerve.)

Ask the patient to carry out the following movements against resistance:

- Wrist/finger extension – radial nerve



- Finger Abduction of the index finger – ulnar nerve



- Thumb Abduction – median nerve



Function

Power grip – “Squeeze my fingers with your hands”

Pincer grip – “Squeeze my finger between your thumb and index finger “

Pick up a small object or undo a shirt button – “Can you pick up this small coin out of my hand?”

Tinel's test→ for carpal tunnel syndrome (nerve irritation)

- Tap over the carpal tunnel with your finger
- If the patient develops **tingling** in the **thumb** and **radial two and a half fingers** this is suggestive of median nerve irritation and compression

Phalen's test→ to support the diagnosis of carpal tunnel syndrome

- Ask the patient to hold their **wrist** in complete and forced **flexion** (pushing the dorsal surfaces of both hands together) for **60 seconds**
- If the patient's **symptoms** of carpal tunnel syndrome are **reproduced** then the test is **positive** (e.g burning, tingling or numb sensation in the thumb, index, middle and ring fingers)



Rheumatoid arthritis

Summary

Rheumatoid arthritis (RA) is an inflammatory autoimmune disorder characterized by joint pain, swelling, and synovial destruction. RA predominantly affects middle-aged women. The condition can also cause various extra-articular manifestations such as rheumatoid nodules and pulmonary fibrosis. Diagnosis is mainly based on clinical features (e.g., morning stiffness, symmetrical joint swelling) and laboratory tests (e.g., anti-CCP). X-ray findings (e.g., soft tissue swelling or joint space narrowing) occur late in the disease and are therefore not typically used for diagnosis. Early intervention with disease-modifying antirheumatic drugs (DMARDs) plays a decisive role in successful treatment. RA is not curable, but early effective treatment may help offset severe complications (e.g., permanent damage to the affected joints).

Epidemiology

- **Prevalence:** ~ 1% in Caucasians; lower in African and Chinese population ^[1] "Prevalence increases with age."
- **Sex:** ♀ > ♂ (3:1)
- **Peak incidence:** 30–50 years "or earlier onset, see juvenile idiopathic arthritis."

Around 70% of patients with rheumatoid arthritis are HLA-DR4. Patients with Felty's syndrome (a triad of rheumatoid arthritis, splenomegaly and neutropaenia) are even more strongly associated with 90% being HLA-DR4

Etiology

- Chronic **inflammatory autoimmune** disorder of unknown etiology
- Hypotheses suggest the etiology is multifactorial, with the following factors playing a role:
 - Genetic disposition: RA appears to be associated with specific HLA types (**HLA-DR4, HLA-DR1**). . " It is assumed that, because of genetic disposition, an immune response is triggered by an unknown infection."
 - Around 70% of patients with rheumatoid arthritis are HLA-DR4. Patients with Felty's syndrome (a triad of rheumatoid arthritis, splenomegaly and neutropaenia) are even more strongly associated with 90% being HLA-DR4
 - Environmental triggers (e.g., infection, tobacco)
 - Hormonal factors
 - A number of studies have suggested a link between *Proteus mirabilis* infection and the development of RA, and this may contribute to the increased incidence of RA in women, who are more susceptible to UTI.
 - Proteus mirabilis* is a bacterium in fecal flora.
 - Proteus mirabilis* is a Gram-negative, anaerobic, rod
 - molecular structure of *Proteus mirabilis* is very similar to cells in joints.
 - When the immune system develops antibodies to combat the bacteria, the antibodies inadvertently attack the similarly structured joint tissues.

Pathophysiology

- Initially, non-specific inflammation affects the synovial tissue, which is later amplified by activation of T cells (autoimmune response).
- With time, it may lead to inflammatory joint effusion and **synovial hypertrophy**, as well as progressive destruction and deterioration of cartilage and bone. " Ligaments and tendons may also be affected"
 - Synovial lining hyperplasia
 - Pannus formation along the synovial tissue → produce proteinases → destroy cartilage extracellular matrix
 - Rheumatoid arthritis involves pannus formation, which leads to cartilage destruction and joint ankylosis. Pannus is an abnormal layer of fibrovascular tissue or granulation tissue. Common sites for pannus formation include over the cornea, over a joint surface (as seen in rheumatoid arthritis), or on a prosthetic heart valve. Pannus may grow in a tumor-like fashion, as in joints where it may erode articular cartilage and bone.
- Patients with positive **rheumatoid factor (RF)** are more likely to develop extra-articular manifestations of rheumatoid arthritis

Clinical features

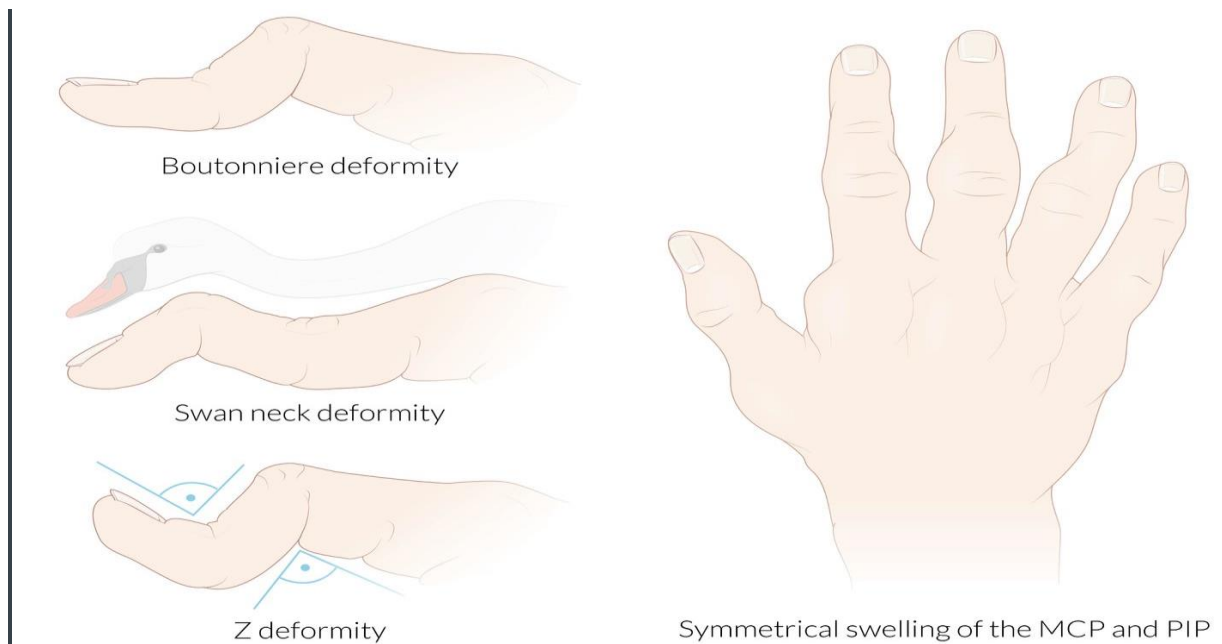
Articular manifestations

- **Polyarthralgia**
 - **Symmetrical pain and swelling** of affected joints (also at rest). "Redness is uncommon and indicates a different diagnosis!"
 - Frequently affected joints
 - **Metacarpophalangeal** joints (MCPJs)
 - **Proximal interphalangeal** joints (PIPJs)
 - Wrist joints
 - Knee joints
 - Usually not involved: DIP joints, first CMC joint, and the axial skeleton (except for the cervical spine, see RA of the cervical spine)
 - **The sacroiliac joint is spared in rheumatoid arthritis.**

symptoms may be more noticeable in the dominant hand.

- **Morning stiffness** (often > 60 min) that usually improves with activity
- **Joint deformities**
 - **"Rheumatoid hand"**
is characteristic, and can include the following deformities:
 - Deepening of the interosseous spaces of the dorsum of hand "Diminished function and inactivity lead to atrophy and interosseous muscle wasting."
 - **Swan neck deformity:** PIP hyperextension and DIP flexion
"Typically caused by contraction of the flexor tendon or tight surgical repair following tendon rupture; requires treatment with splinting or surgery"
 - **Boutonniere deformity:** PIP flexion and DIP hyperextension.
 - **Hitchhiker thumb deformity** (Z deformity of the thumb): hyperextension of the interphalangeal joint with fixed flexion of the MCP joint
 - **Ulnar deviation** of the fingers
 - Hammer toe
 - **Atlanto-axial subluxation**

DIP joints are not typically affected in RA!



Hand deformities in rheumatoid arthritis

Rheumatoid arthritis of the hand typically manifests as pain and swelling affecting the MCP and PIP joints symmetrically (i.e., bilaterally).

Joint deformities typically seen in rheumatoid arthritis include:

- Boutonniere deformity: PIP flexion and DIP hyperextension
- Swan neck deformity: PIP hyperextension and DIP flexion
- Z deformity of thumb (Hitchhiker thumb): IP hyperextension and MCP flexion

Carpal tunnel and tarsal tunnel syndromes can occur in RA. If a patient with inflammatory knee arthritis presents with a swollen calf, suspect a ruptured Baker cyst (popliteal cyst) causing pseudophlebitis. Occasionally, a Baker cyst can cause extrinsic venous compression that can simulate a deep vein thrombosis.

Extra-articular manifestations

- **Constitutional symptoms:** low-grade fever, myalgia, malaise, fatigue, weight loss, night sweats
- **Skin: rheumatoid nodules**
 - Non-tender, firm, subcutaneous swellings (2 mm–5 cm)
 - Commonly occur in areas exposed to higher pressure e.g., extensor side of the forearm, bony prominences
- **Lungs**
 - Fibrosis, pneumoconiosis (Caplan syndrome)
 - Rheumatoid pulmonary nodules
 - Pleuritis, pleural effusions " Often asymptomatic"
- **Eye:** keratoconjunctivitis sicca, scleritis, and episcleritis " Approximately 20% of cases of scleritis are associated with RA"
- **Endocrine and exocrine glands:** secondary Sjögren syndrome
- **Hematological**
 - Anemia of chronic disease
 - Neutropenia
 - Splenomegaly (Felty syndrome)
- **Other musculoskeletal**
 - Tenosynovitis and bursitis
 - **Carpal tunnel syndrome** (entrapment neuropathy)
 - Typical nocturnal paresthesia of volar hand and fingers I–III
 - Atrophy of thenar muscles → difficulty making a fist; inability to oppose the thumb
 - Tarsal tunnel syndrome

- **Heart:** pericarditis and myocarditis; higher risk of myocardial infarction, stroke, and CHF " The risk of death from a stroke or heart attack in patients with rheumatoid arthritis is three times higher than in the rest of the population"

The most common cause of death in RA is coronary artery disease.

- **Vascular:** peripheral vasculitis manifesting as livedo reticularis, **Raynaud phenomenon**, purpura, necrosing fingertips or peripheral neuropathy (Vasculitis: skin, bowel, and peripheral nerves)

Clinical features of rheumatoid arthritis	
Clinical presentation	Symptoms • Insidious onset, multiple joint pain, stiffness & swelling • Morning stiffness lasting hours, improves with activity • Small joints (eg, PIP, MCP, MTP) commonly involved • Monoarthritis (eg, knees, elbows) can also occur later • Spare the DIP joint, unlike osteoarthritis Examination • Affected joints are tender to the touch, swollen, with limited range of motion • Tenosynovitis of the palms → "trigger finger" • Rheumatoid nodules (especially on elbows) • Cervical joint involvement can lead to spine subluxation → spinal cord compression
Laboratory/ imaging studies	• Positive anti-CCP antibodies (diagnostic testing) • High IgM rheumatoid factor • High C-reactive protein & ESR correlate with disease activity • X-ray: Soft-tissue swelling, joint space narrowing & bony erosions

Anti-CCP = anti-cyclic citrullinated peptide; DIP = distal interphalangeal joints;
 ESR = erythrocyte sedimentation rate; IgM = Immunoglobulin M; MCP = metacarpophalangeal;
 MTP = metatarsophalangeal

Rheumatoid arthritis

Etiology

Autoimmune disorder,
Genetic predisposition (HLA-DR4, HLA-DR1)

Epidemiology

♀ > ♂
Peak age: 30–50 years

Serology

Anti-CCP antibodies
Rheumatoid factor

Laboratory findings

↑ CRP
↑ ESR

Complications

Joint disability (e.g., rheumatoid hand;
hammer toe)
increased risk of cardiovascular disease;
Felty syndrome

General symptoms

Night sweats
Low-grade fever
Myalgia

Keratoconjunctivitis sicca,
scleritis, episcleritis



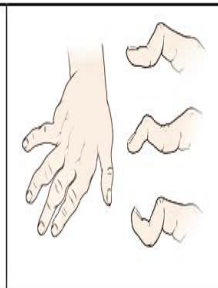
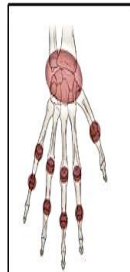
Morning stiffness

Pleuritis,
pulmonary fibrosis

Pericarditis and myocarditis

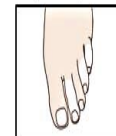


Bursitis,
Rheumatoid nodules



Symmetrical polyarthritis
(esp. of MCP and PIP joints);
joint deformities
(e.g., swan neck deformity,
boutonniere deformity,
Z deformity),
tenosynovitis,
carpal tunnel syndrome

Vasculitis
Purpura,
peripheral neuropathy,
Raynaud phenomenon



- If a patient with RA presents with a swollen painful calf, consider a ruptured Baker cyst. Baker cyst is the extension of inflamed synovium into the popliteal space.
- RA most commonly affects the cervical spine joints in the axial skeleton (particularly at C1 and C2) and can lead to cervical spine subluxation, which can also cause spinal cord compression.
- In RA, the incidence of cervical involvement has been reported to be 25-80% and results from pannus formation at the synovial joints between C1 and C2. Commonly, patients have subtle symptoms, which include neck pain (occipital), C2 radicular pain (paresthesias of the hands and feet), and myelopathy. Neurologic symptoms occur when the spinal cord is involved (paraplegia, quadriplegia).
- All patients with RA should be screened with a plain x-ray for C1-C2 subluxation before intubation or anesthesia is performed.

C-spine: Patients with chronic, severe disease may develop cervical instability at the atlanto-axial articulation (C1-C2). The rest of the axial skeleton is spared. While patients can be asymptomatic, suspect cervical (C1-C2) involvement when a patient with RA complains of:

- recurrent occipital headaches,
- limited neck range of motion, or
- paresthesias of the hands and feet.

If these symptoms are present, order cervical spine x-rays with flexion and extension views. In RA patients scheduled to undergo endotracheal intubation, an evaluation for cervical instability is mandatory. Acute subluxation, which may occur with extension of the neck for intubation, can cause spinal cord compression or vertebral artery compression leading to quadriplegia or sudden death.

Remember: All patients with long-standing RA should have flexion and extension neck films before surgery to assess for subluxation.

The thoracic, lumbar, and sacral spine and the SI joints are usually spared in RA (in contrast to ankylosing spondylitis and psoriatic arthritis). Again, RA does not present as lumbar spine pain. If you see a patient with RA and spine pain, think about the myriad other potential causes of spine pain; e.g., compression fractures, infections—not a flare of RA.

Felty syndrome

- **Definition:** Felty syndrome is a severe subtype of seropositive RA
 - " Patients are also frequently positive for HLA-DR4"
- **Clinical features**
 - Clinical triad consisting of **arthritis**, **splenomegaly**, and **neutropenia** (leads to ↑ risk of recurrent bacterial infections)
 - Other symptoms: skin ulcers of the lower limbs (indicating vasculitis), hepatomegaly, fever, and chest pain (indicating pleuritis or pericarditis)
 - Associated with increased risk of developing non-Hodgkin lymphoma
- **Diagnosis**
 - Leukopenia with selective neutropenia
 - ↑↑ RF
 - ↑↑ ACPA
- **Treatment**
 - DMARD: **methotrexate**
 - Alternative: cyclophosphamide
 - Prompt and aggressive antibiotic treatment for suspected infections " Isolated fever may be the only finding"
 - Consider splenectomy to treat leukopenia and improve the disease course. " Indicated in patients with a combination of severe granulocytopenia and recurrent infections despite receiving the recommended treatment"

If a patient has arthritis, splenomegaly, and neutropenia, consider Felty syndrome.

Felty syndrome	
Clinical features	• Rheumatoid arthritis ○ Severe erosive joint disease & deformity ○ Rheumatoid nodules ○ Vasculitis (mononeuritis multiplex, necrotizing skin lesions) • Neutropenia (ANC <1500/μL) • Splenomegaly
Diagnosis	• Anti-CCP & RF are positive in >90% of patients • Markedly elevated ESR, often >85 mm/hr • Peripheral smear & bone marrow biopsy to rule out other causes of neutropenia

ANC = absolute neutrophil count; anti-CCP = anti-cyclic citrullinated peptide;
ESR = erythrocyte sedimentation rate; RF = rheumatoid factor.

FELTY SYNDROME

- Felty syndrome is a clinical disorder seen in patients with severe, long-standing (>10 years) RA but rarely precedes the diagnosis.
- Characterized by neutropenia and splenomegaly. Splenomegaly without neutropenia can also occur in patients with RA, but the diagnosis of Felty syndrome should not be made in the absence of neutropenia
- Pts with FS have severe seropositive RA with $\uparrow$ risk of extra-articular manifestations
- More common in women—peak incidence in 4th and 5th decade
- Pathophysiology unknown
- Associated with HLA-DR4— $\uparrow$ incidence in pts with FH of RA—suggesting genetic component
- FS usually improves with treatment of underlying RA (eg. methotrexate)

Diagnostics

The diagnosis of RA is based on diagnostic criteria that include laboratory testing. Imaging may support the diagnosis, but radiological joint findings are no longer included in the criteria, as they often become evident only in late stages of disease.

ACR criteria for RA (ACR/EULAR classification criteria (2010))

- Rheumatoid arthritis = score of ≥ 6 points + confirmed **presence of synovitis** in at least one typical joint without an alternative, more probable diagnosis (e.g., trauma or degenerative joint conditions)
 - For every column, the highest score is taken into account. " For example: 4–10 small joints (3 points) + low positive rheumatoid factor (RF) (2 points) + normal CRP/ESR (0 points) + duration of symptoms ≥ 6 weeks (1 point) = 6 points"

TABLE 17.4. Criteria for the Classification of RA^a

CRITERION	DESCRIPTION	POINTS
Joint involvement (swollen, tender, or erosions seen on x-ray)	1 large joint	0
	2–10 large joints	1
	1–3 small joints	2
	4–10 small joints	3
	> 10 joints	5
Serology ^b	⊕ RF or anti-CCP at low level	2
	⊕ RF or anti-CCP at high level	3
Acute-phase reactants	Elevated CRP or ESR	1
Duration	≥ 6 weeks	1

^aThe diagnosis of RA requires a score of 6 or higher and no other disease to explain the symptoms.

^bA ⊕ serology is now required for diagnosis.

Seronegative RA is diagnosed when patients meet other criteria but lack both RF and anti-CCP. These patients tend to have less severe disease than what is seen in antibody-positive patients.

Laboratory tests

- **Non-specific parameters**

- ↑ Inflammatory markers: CRP, ESR correlate with inflammatory activity.
 - ↑ Ferritin as an acute phase protein
 - Possibly leukocytosis, thrombocytosis
- Anemia of chronic disease

Erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP)

- up to 40% of patients with RA may have normal levels
- In the early stages of an insidious onset of the RA disease, the acute phase markers CRP and ESR are often normal, particularly when small joints are involved.

- **Serology (specific parameters)**

- **Antinuclear antibodies (ANA):** elevated in 30% of cases " ANA are not specific for RA but are important for excluding differential diagnoses (especially systemic lupus erythematosus)."
- **ACPA " Anti-citrullinated peptide antibodies"**
(e.g., anti-CCP " Anti-cyclic citrullinated peptide")
 - Specificity > 90% " The test has about the same sensitivity as RF (60%) but has a higher specificity and enables earlier detection."

anti-cyclic citrullinated peptide (CCP) antibodies

- Anti-CCP positivity is a prognostic marker.
- Anti-cyclic citrullinated peptide antibody may be detectable up to 10 years before the development of rheumatoid arthritis.
- appears earlier than RF,
- is associated with more aggressive disease and extraarticular involvement.

- **Rheumatoid factor (RF)**
 - **IgM autoantibodies** against the Fc region of IgG
 - Low specificity " Elevated RF may be physiological or caused by chronic inflammation associated with RA or other autoimmune and infectious diseases. Typically, the amount of RFs in serum increases during the course of the disease."

Rheumatoid factor

- Rheumatoid factor (RF) is a circulating antibody (usually IgM) which reacts with the Fc portion of the patients own IgG.
- Rheumatoid factor is an antibody with reactivity to the heavy chain of IgG.
- The rheumatoid factor may be of IgM, IgG or IgA class.
- The conventional (agglutination) test, detects only IgM RF
- **high titre levels are associated with severe progressive disease (but NOT a marker of disease activity).**

A positive rheumatoid factor is associated with:

- More severe erosive disease
- Extra-articular manifestations including subcutaneous nodules and
- Increased mortality.

Conditions associated with a positive RF include:

- Sjogren's syndrome (around 100%)
- Felty's syndrome (around 100%)
- Mixed cryoglobulinemia (types II and III) - 40 to 100%
- rheumatoid arthritis (70-80%)
- Mixed connective tissue disease - 50 to 60%
- infective endocarditis (= 50%)

- **Synovial fluid analysis**

- Synovial fluid is collected by joint aspiration.
 - Findings
 - Cloudy yellow appearance
 - Sterile specimen with leukocytosis (**WBC: 5,000–50,000/μL**)
 - ↑ Neutrophils, granulocytes, and ragocytes " Leukocytes with inclusions of aggregated IgG, fibrin, complement, and RF"
 - ↑ Proteins, ↓ viscosity
 - Possibly rheumatoid factor
-

Imaging

- **Conventional x-ray**

" Radiographic changes are typical for RA. But even in aggressive cases, it takes 6–24 months until erosions are radiologically evident. As a result, classification criteria for diagnosing early RA no longer include radiological joint findings."

- Dorso-palmar x-ray of both hands
- Radiological findings
 - Early: soft tissue swelling, demineralization (juxta-articular)
 - Late: joint space narrowing, **erosions of cartilage and bone**, demineralization (generalized), subluxation

Even if radiographic findings are normal, RA is still possible!

- **MRI:** (with or without contrast), especially if cervical spine involvement is suspected or in early stages " More sensitive than conventional x-ray for detecting changes in joints and bones"
- **Ultrasound:** joint effusion, formation of pannus

Clinical features of rheumatoid arthritis	
Clinical presentation	• Pain, swelling & morning stiffness in multiple joints • Small joints (PIP, MCP, MTP); spares DIP joints • Systemic symptoms (fever, weight loss, anemia) • Cervical spine involvement: subluxation, cord compression
Laboratory/imaging studies	• Positive rheumatoid factor & anti-CCP antibodies • C-reactive protein & ESR correlate with disease activity • X-ray: soft tissue swelling, joint space narrowing, bony erosions

Anti-CCP = anti-cyclic citrullinated peptide; **DIP** = distal interphalangeal; **ESR** = erythrocyte sedimentation rate; **MCP** = metacarpophalangeal; **MTP** = metatarsophalangeal; **PIP** = proximal interphalangeal.

Condition	Osteoarthritis (most common)	Rheumatoid Arthritis (RA)	Psoriatic arthritis	Gout	Pseudogout
Specific groups	- Sex: ♀ > ♂ - Majority of people > 65 years have radiological evidence of osteoarthritis which may be asymptomatic	♀ > ♂ - Family history of RA	- Sex: ♀ = ♂ - Family or personal history of psoriasis - May be triggered by trauma or infections (e.g., HIV infection)	-Sex: ♀ < ♂ -Peak incidence: 30–60 years -Family history of Gout -Often in alcoholics	- Sex: ~ ♀ = ♂ -Mostly older patients (> 50 years)
Course of disease	Chronic, progressive course	Chronic course with varied patterns: either monocyclic, polycyclic or progressive	Chronic, progressive course	Chronic course with acute attacks (rapid onset of symptoms)	
Clinical features	- No constitutional symptoms - Morning stiffness < 30 minutes - Pain/stiffness worsens with increased activity and relieved by rest Non-inflammatory	- Constitutional symptoms - Morning stiffness > 30 minutes Pain/stiffness can be relieved by activity - Articular inflammation (swollen, warm joints)	-Psoriatic skin lesions and nail changes -Dactylitis with “sausage digits” -Enthesitis	-Articular inflammation (swollen, erythematous, warm joints) -Eventually development of tophi	Articular inflammation (swollen, erythematous, warm joints)
Symmetry of joint involvement	Usually asymmetrical	Symmetrical	Symmetrical	Asymmetrical	Asymmetrical
Pattern of disease	- Polyarthritis ○ Predominantly weight-	-Polyarthritis: predominantly wrists ,	Polyarthritis or oligoarthritis	-Usually monoarthritis:	-Monoarthritis (rarely

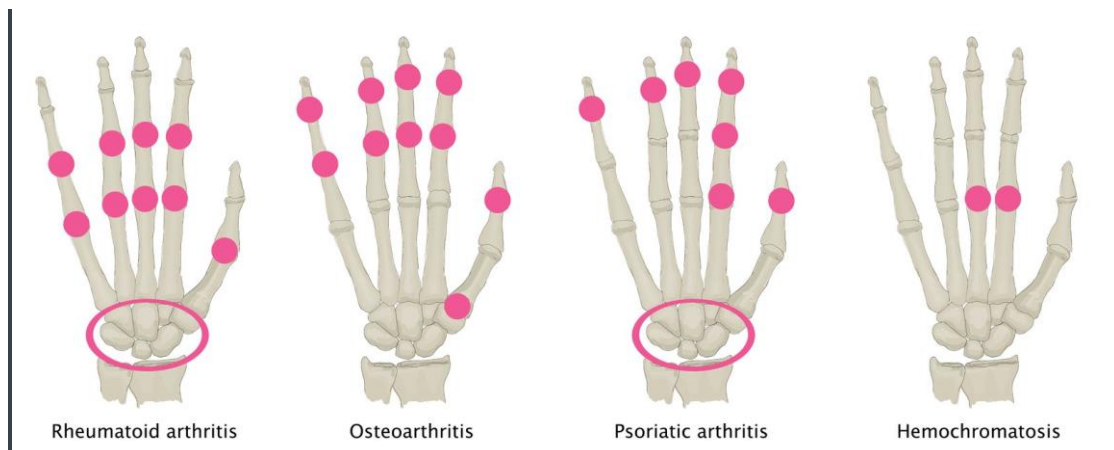
	bearing joints (e.g., knees) ○ Spares the wrist and the MCP joints	MCP and PIP joints	○ Commonly DIPJ and/or spinal involvement ○ Sometimes large joints	typical locations include the big toe (MTPJ) , knee and thumb (MCPJ)	oligoarthritis): commonly knees and wrist joints
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Other differential diagnoses:

- Autoimmune-related arthritis (e.g., sarcoidosis, SLE, rheumatic fever, mixed connective tissue disease, polymyalgia rheumatica)
- Arthritis associated with Inflammatory bowel disease
- Vasculitides
- Hemochromatosis
- Viral arthritis (e.g., parvovirus B19, hepatitis viruses)
- Lyme arthritis
- Reactive arthritis (post-urethritis, post-enteritis)
- Fibromyalgia

Hemochromatosis is another disease that commonly involves the 2nd and 3rd MCP and PIP joints, but the arthropathy of hemochromatosis is distinctly asymmetric. Also, hemochromatosis has hook-like osteophytes on the MCP joints and chondrocalcinosis—neither finding is seen in RA. Patients can easily be screened for hemochromatosis with iron studies. An elevated transferrin saturation (Fe/TIBC of > 45%) or elevated ferritin level suggests the diagnosis. Know these clues for differentiating RA and SLE from hemochromatosis.

	Rheumatoid arthritis	Osteoarthritis
pathophysiology	autoimmune (inflammatory)	degenerative due to ↑ wear and tear on joints → loss of cartilage (non-inflammatory)
Age of starting	At any age	Usually later in life
Speed of onset	Rapid, over weeks to months	Slow, over years
Pain	improves with movement	worse with movement and better with rest
Primary joint affected	Proximal interphalangeal	Distal interphalangeal
	Metacarpophalangeal	Carpometacarpal
Heberdens nodes	Absent	Present
Joint characteristics	Soft, warm and tender	Hard and bony (little or no swelling)
Stiffness	Worse after resting (morning stiffness)	If present, worse after effort, may be described as evening stiffness
	Usually > 1 hour	Usually < 1 hour
Systemic symptoms	Present (eg: fatigue)	Absent
RF and anti-CCP	Positive	Negative
ESR and C-reactive protein	Elevated	Normal
x-ray	Osteophytes absent	Osteophytes may be present



Differential diagnosis: Hand joint pain

Rheumatoid arthritis: symmetrical swelling of metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints;

Osteoarthritis: PIP, distal interphalangeal (DIP), and first carpometacarpal (CMC) joints;

Psoriatic arthritis: DIP and MCP joints, or whole digit ("sausage digits");

Hemochromatosis: asymmetrical arthropathy of the MCP joint of digits II and III

Treatment

General measures

- For acute episodes of inflammation: cryotherapy
- Physical and occupational therapy
- Physical activity

Acute anti-inflammatory therapy

- **Indication:** acute attack

Symptomatic Control of RA:

A. NSAIDs

- NSAIDs are the best initial therapy for the pain of RA. They work immediately to improve inflammation but do nothing to prevent the progression of disease.

- The dose should be reduced if the patient responds well to DMARDs

B. Steroids

- Steroids also work in a matter of hours to control the pain of RA secondary to inflammation. Steroids are used:

- When NSAIDs do not control symptoms immediately.
- As a bridge when waiting for DMARDs to take effect; DMARDs are much slower in onset of action than steroids.

- Steroids do not prevent the progression of RA. In fact, they can result in generalized bone loss (osteoporosis).

- PPIs are recommended because combining glucocorticoids with NSAIDs substantially increases the risk of GI ulcers.

Steroids

- Systemic " Osteoporosis prophylaxis is necessary if a daily dose of prednisolone or its equivalent > 7.5 mg over a period > 3 months."
- Intra-articular injections of PRN " Allows for rapid and sometimes long-term relief of symptoms in the affected joint"
- Prevention of osteoporosis: optimization of sufficient calcium and vitamin D intake

Long-term anti-inflammatory therapy with disease-modifying antirheumatic drugs (DMARDs) " DMARDs are drugs that have the ability to slow down disease progression of rheumatoid arthritis; they are not necessarily pharmacologically related"

- Induce immunosuppression, leading to potential remission of RA
- Reduce mortality and morbidity by up to 30%
 - Slow progression of disease
 - Preserve joint function
 - Limit complications
- Slow onset of action (≥ 6 weeks), so symptomatic treatment with glucocorticoids and NSAIDs is often required

- **Non-biologic agents**

- Drug of choice: methotrexate (MTX)

- Methotrexate is the best initial DMARD
- First-line treatment for moderate to severe RA " MTX is given only once per week!"
- Benefits: highly effective, relatively well-tolerated, low cost, possibly life-prolonging
- Methotrexate works by inhibiting dihydrofolate reductase.
- Common side effects of MTX include gastrointestinal symptoms, oral ulcers or stomatitis, rash, alopecia, hepatotoxicity, interstitial pneumonitis and pulmonary fibrosis, and bone marrow suppression, increased risk of lymphoproliferative disorders, teratogenicity
- Macrocytic anemia is a common side effect.
- Methotrexate therapy for rheumatoid arthritis is associated with hepatotoxicity. Serum liver studies should be checked prior to initiation and periodically thereafter. Much of the toxicity of methotrexate, including hepatotoxicity, can be mitigated by concurrent administration of folic acid, which does not reduce the effectiveness of the drug.
- To minimize side effects, folic acid is recommended 24–48 hours after taking MTX.
- Do not give NSAIDs on the same day as MTX, as they can worsen the side effects of MTX by inhibiting its renal excretion .
- Methotrexate should not be used in patients who are pregnant or are planning to become pregnant in the near future and those with severe renal insufficiency, liver disease, or excessive alcohol intake.
- Patients who do not respond after 6 months may require biologic DMARDs such as tumor necrosis factor-alpha inhibitors (etanercept, infliximab) as step-up therapy.

In an RA patient being treated with methotrexate who develops new-onset dyspnea and interstitial infiltrates on CXR, consider opportunistic infections and hypersensitivity pneumonitis from methotrexate and stop the drug.

- Alternative drugs:
 - Leflunomide " Inhibits the enzyme orotate reductase, which catalyzes the oxidation of dihydroorotic acid (an essential step in pyrimidine synthesis)."
 - Hydroxychloroquine
 - Sulfasalazine: for use in pregnancy

Hydroxychloroquine:

- This agent can be used as monotherapy as a DMARD in cases of mild disease in which we wish to avoid the toxicity of methotrexate.
- More often hydroxychloroquine is used in combination with methotrexate as a DMARD.
- Hydroxychloroquine is toxic to the retina. Patients treated with hydroxychloroquine should have a baseline ophthalmologic evaluation and periodic reassessment.
- Both hydroxychloroquine and sulfasalazine are safe in pregnancy.

Sulfasalazine causes:

- Bone marrow toxicity.
- Hemolysis with G6 PD deficiency.
- Rash.

- **Biologic therapy**

- Indication: moderate or severe disease activity remaining after three months of DMARD therapy
- Should be combined with non-biologic DMARDs
- **Tumor necrosis factor (TNF) α inhibitors:** e.g., adalimumab, infliximab, etanercept
 - See contraindications to anti-TNF- α treatment.
- Others: rituximab (anti-CD20) and anakinra (interleukin-1 receptor antagonist, particularly for Still disease)

Early administration of DMARDs is crucial for a better outcome!

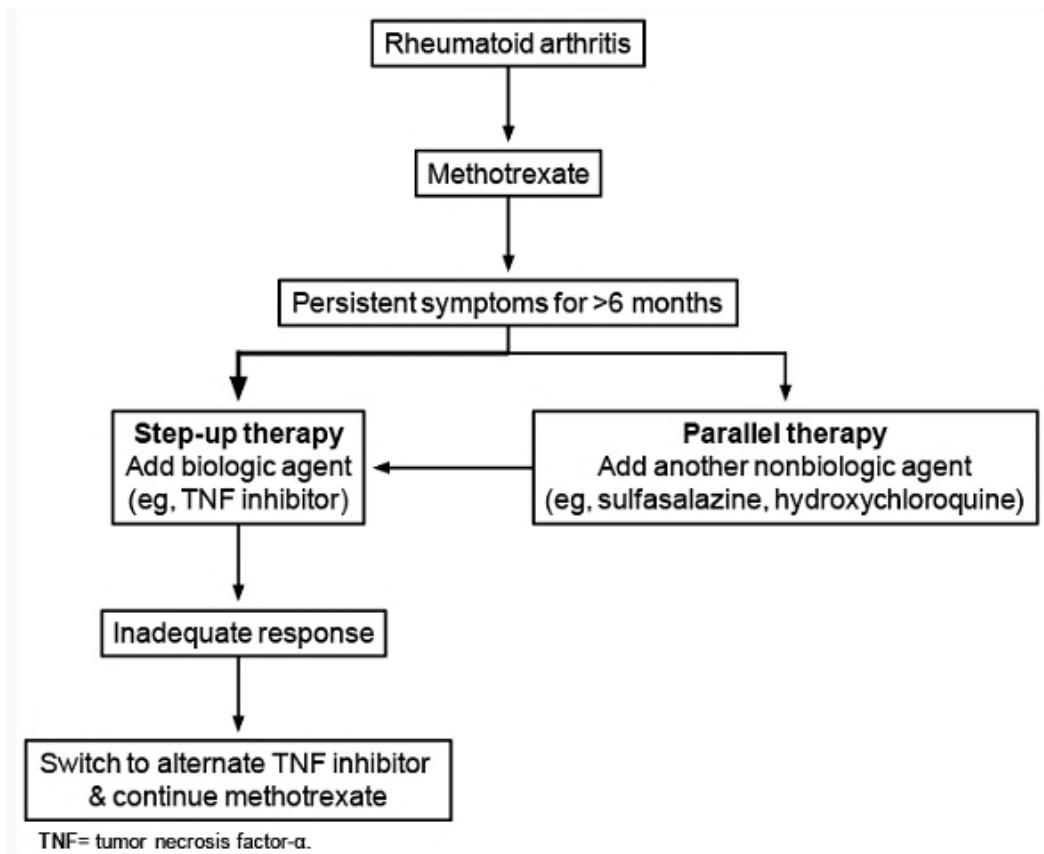
Tumor Necrosis Factor Inhibitors

- Tumor Necrosis Factor Inhibitors (infliximab, adalimumab, etanercept, golimumab, certolizumab):
- Tumor necrosis factor (TNF) inhibitors are the first line as DMARDs for those not responding to methotrexate or intolerant of methotrexate.
- They are often used initially in combination with methotrexate to prevent disease progression.
- **Toxicity of anti-TNF drugs:**
 - Reactivation of TB (screen with a PPD prior to their use).
 - Infection.

Rituximab

- This agent, originally developed for non-Hodgkin lymphoma, is effective in RA as a DMARD by removing CD20 positive lymphocytes from circulation. This leads to excellent long-term control of RA. Rituximab is used in combination with methotrexate in those not responding to anti-TNF medications.

Sulfasalazine, Leflunomide (pyrimidine synthesis inhibitor), Abatacept (interfering with the immune activity of T cells), and Anakinra (human IL-1 receptor antagonist protein): These agents are alternative DMARDs to add to methotrexate if anti-TNF agents do not control disease.



▪ Adverse Effects of RA Medications:

Drug	Adverse effect
Anti-TNF	Reactivation of tuberculosis
Hydroxychloroquine	Retinopathy
Sulfasalazine	Rash, hemolysis
Rituximab	Infection
Gold salts	Nephrotic syndrome
Methotrexate	Liver, lung, bone marrow

Disease-modifying antirheumatic drugs		
Agent	Mechanism	Adverse effects
Methotrexate	Purine antimetabolite	• Hepatotoxicity • Stomatitis • Cytopenias
Leflunomide	Pyrimidine synthesis inhibitor	• Hepatotoxicity • Cytopenias
Hydroxychloroquine	TNF & IL-1 suppressor	• Retinopathy
Sulfasalazine	TNF & IL-1 suppressor	• Hepatotoxicity • Stomatitis • Hemolytic anemia
TNF inhibitors • Adalimumab • Certolizumab • Etanercept • Golimumab • Infliximab		• Infection • Demyelination • Congestive heart failure • Malignancy

TNF = tumor necrosis factor.

NOTE :

- Patients with rheumatoid arthritis are at increased risk of developing osteopenia, osteoporosis, and bone fractures, especially if additional risk factors for osteoporosis are present (low body weight, female sex, family history of osteoporosis, cigarette smoking, postmenopausal state, excessive alcohol use, other comorbidities) are present. The degree of bone loss generally correlates with disease activity.
- Management includes adequate physical activity, optimization of calcium and vitamin D intake, minimization of corticosteroid therapy, and consideration for bisphosphonate treatment.

Complications

Untreated and/or severe cases can result in permanent damage to the joints with stiffening and deformity.

- **Complications in the upper limbs:** rheumatoid **hand deformities** (see “Clinical findings” above)
- **Complications in the lower limbs**
 - Baker cyst due to inflammatory joint effusion
 - Foot impairment: pes plano-valgus " Caused by erosive damage to the bones and tendons"

cord compression

- The recent limb weakness and the presence of pyramidal signs in the legs in a patient with rheumatoid arthritis are highly suggestive of spinal cord lesion at the cervical region.
- Although plain radiography of the cervical spine is the initial imaging assessment tool for neck pain in patient with rheumatoid arthritis, those with symptoms or signs of cord compression should undergo immediate MRI and be sent for surgical consultation.

Ocular: common, with 25% of patients having eye problems

- **keratoconjunctivitis sicca (most common)**
 - characterised by dry, burning and gritty eyes caused by decreased tear production
 - Other associated symptoms include irritation, redness, discharge, and easily fatigued eyes. Blurred vision may also occur. Scarring of the cornea may occur in some cases without treatment
 - do Tear-film integrity
 - A reflex response to irritation of the corneal surface is epiphora, or watering.
 - Symptoms will be worse when tear-film evaporation is greater,
- **episcleritis (erythema)**
- **scleritis (erythema and pain)**
- **corneal ulceration,**
- **keratitis,**
- **Iatrogenic**
 - **steroid-induced cataracts,**
 - ❖ 'steroid cataract' is typically posterior subcapsular, and causes constant and gradually progressive blur.
 - ❖ (Steroids >> raised intraocular pressure)
 - ❖ Steroids can cause raised blood glucose levels. Fluctuating blood sugar levels can cause osmotic swelling of the lens in the eye, resulting in fluctuations in vision. However, diabetic retinopathy will not affect vision unless maculopat
 - **chloroquine retinopathy**

- **Other complications**

- See "Subtypes and variants" above.
- **Muscle weakness** " May be due to myositis, drug induced myopathy (e.g., glucocorticoids), or synovial inflammation and subsequent diminished motion of joint"
- **Lung disease:** pleural " E.g., pleuritis and pleural effusion" and parenchymal lung diseases " E.g., pulmonary nodules, interstitial fibrosis, organizing pneumonia"

Respiratory:

- pulmonary fibrosis,
- pleural effusion (**rheumatoid pleural effusion: characterised by >> low glucose level**)
- pulmonary nodules,
- bronchiolitis obliterans,
- methotrexate pneumonitis,
- pleurisy
- cricoarytenoid arthritis

➤ **cricoarytenoid arthritis:**

- seen in up to 75% of patients with rheumatoid arthritis.
- It can cause sore throat, hoarse voice and stridor, but is often asymptomatic.
- symptoms can rapidly worsen in the post-operative period.
- It is unrelated to any lung fibrosis.
- the most helpful diagnostic test >> Spirometry with flow–volume loop
- Also: direct laryngoscopy and high-resolution computed tomography of the larynx.
- Patients can need urgent tracheostomy and steroids, both orally and via joint injection.

- **Cardiovascular disease:** increased risk of pericarditis, coronary artery disease, heart failure, atrial fibrillation, and stroke

➤ **Constrictive pericarditis is the commonest cardiac complication of rheumatoid arthritis**

➤ **ischaemic heart disease: RA carries a similar risk to type 2 diabetes mellitus.**

- **Vasculitis** involving the kidneys " Kidney disease directly as a result of rheumatoid arthritis are rare; they are more likely due to drug toxicity (e.g., NSAIDs and cyclosporine)" and skin
- Amyloid A (AA) **amyloidosis**
- **Septic arthritis** " Joint damage predisposes to infection. Empiric antibiotic therapy should be started if septic arthritis is suspected."
- Osteopenia, osteoporosis, and bone fractures
- Sjögren syndrome (secondary form)

- Osteoporosis: ↑ proinflammatory cytokines, corticosteroid therapy, and lack of physical activity>> local (around inflamed joint) and generalized loss of bone mass (↑ risk of osteopenia, osteoporosis, and bone fractures esp. if other risk factors (eg. low body weight, female sex, smoking, family history of osteoporosis, postmenopausal state, excessive alcohol intake, other comorbidities) are present—**degree of bone loss correlates with disease activity**

- Strategies to prevent bone loss: adequate physical activity, optimization of vitamin D and calcium intake, minimization of glucocorticoid dose, low threshold for starting bisphosphonates in RA pts.

Amyloidosis

- Poorly controlled rheumatoid arthritis + proteinuria and hypoalbuminaemia raises the possibility of systemic amyloidosis >> Rectal biopsy
- Secondary amyloid A (AA) amyloidosis is an important complication of rheumatoid arthritis (RA) >> (~20%).
- It is caused by extracellular accumulation of AA fibrils, derived from the acute-phase reactant serum amyloid A protein, within various tissues and organs.
- Any patient with longstanding RA who develops proteinuria, or intractable diarrhoea,

Prognosis

- **Factors associated with poor prognosis**
 - Prolonged disease progression without initiation of treatment
 - Late onset (> 60 years of age)
 - Insidious onset (acute or sudden is not a poor prognostic)
 - Sex: ♀
 - Smoking
 - Social factors (e.g., low socioeconomic status, low level of education)
 - Poor functional status at presentation
 - X-ray: early articular erosions (e.g. within the first 6 months of presentation and in less than < 2 years).
 - Extra articular features e.g. nodules ,lung involvement ,vasculitis
 - HLA DR4
- **Laboratory tests associated with worsened prognosis if abnormally elevated**
 - CRP
 - ESR
 - ACPA
 - RF titer
- **Anti-CCP antibodies (The poorest prognostic factor)**

Deep venous thrombosis

Peripheral Venous Disease – History Taking

❖ Patient Profile

❖ Chief Complaint: usually Lower Limb Pain (calf tenderness), redness and swelling.

❖ History of Present Illness:

Lower Limb Pain	
○ Site: Unilateral or Bilateral ○ Onset and Duration: DVT usually sudden - DDx: Cellulitis (fever, redness, hot, tender, shiny skin) ○ Character ○ Radiation ○ What do you think caused this? ○ Recent trauma?	○ Timing ○ Severity: Does it make walking difficult? ○ Progression of the pain and associated symptoms over time ○ Alleviating or Exacerbating Factors: Prolonged standing or lying down? ○ Previous Hx of same symptoms
Associated With	
● Fever ● Erythema: is it well demarcated? ● Color Changes: - Pale - Cyanosed - Hyperpigmentation ● Edema: - Extent - Progression - Changes related to the time of day?	● Itching ● Ulceration or Eczema ● A Venous Ulcer (take a short history) ● Heaviness ● Varicosities ● Symptoms of PE: - SOB - Pleuritic Chest Pain: increases on coughing or breathing in - Hemoptysis
❖ <u>Previous History and Risk Factors</u>	
● Immobility: - Bed rest or recent operation - Recent long flight - Pelvic or LL fractures ● Family History: - Thrombosis - Cancer - Deficiency ● Medications: - OCPs ● Social: - Smoking, Obesity (BMI), Prolonged standing	● Medical and Surgical History: - Heart Failure (stasis): SOB, PND, Chest Pain - Previous DVT or varicosities - Polycythmia Vera, Factor V leiden thrombophilia - Antithrombin III, Protein S or Protein C deficiency - APS: anti phospholipid syndrome - Connective Tissue Disease - Recent central venous catheter - HTN - DM - Liver or Renal disease ● Gynecological History: - Pregnancy - Multiple miscarriages

History taking station:

a 65 year old male has visited your clinic complaining of Lower limb swelling, ask him relevant questions to reach a diagnosis

Steps (total 28 points)	Check if done
Introduce him/her self, ask for permission, and ensure conversation is private	☐
skip taking patient profile	
Specify what patient mean by LL swelling	☐
duration	☐
Site ; unilateral or bilateral	☐
Extent ; up to what level	☐
Onset ; acute vs chronic	☐
Progression ; Throughout the day , with activity, Lying or standing	☐
Associated symptoms; Pain , itching	☐
Other site ; Periorbital, Abdomen, Genitalia, Back, Hands, ... etc	☐
Inquire about local causes ;	☐
1) Trauma	☐
2) DVT; calf tenderness, previous Hx of DVT, immobilization, recent surgery	☐
3) Cellulites: fever,redness,hotness,tenderness,shiny skin,site of entry	☐
4) Joint disease: swelling, pain, hotness, redness, ↓ROM, skin rash	☐
5) Venous obstruction; pelvic tumor, IVC obstruction , AV fistula	☐
6) Lymphedema	☐
Inquire about systemic causes ;	☐
1) CHF: SOB, PND, chest pain	☐
2) Liver disease: jaundice, itchiness , abdominal distention , anorexia, GI bleeding, dilated veins, vomiting , diarrhea, easily bruising	☐
3) Renal disease; polyurea, frequency, dysurea, Hx of HTN or DM	☐
4) Malnutrition	☐
5) Hypothyroidism: weight gain , decreased appetite, cold intolerance	☐
6) Drugs & Allergy: cortisol , NSAIDs, CCB, ... etc	☐
Previous history of leg swelling	☐
past medical history of DM	☐
past medical history of HTN	☐
Smoking status	☐
Alcohol intake status	☐
Drugs	☐
Thank the patient	☐

its common to have an OSCE station with young patient who have lower limb swelling , dont forget to ask about symptoms of rheumatological disease especially behcent disease (skin rash , genital ulcer ,...etc) , since they can cause DVT

Lower Limb swelling: Case: a 60 year-old lady presents with LL swelling. Take focused

-Introduce yourself, take the patient's profile (age, occupation, marital status)

-Ask general questions about the complaint:

Take the **duration**.

Site: Unilateral or Bilateral?

Extent: Up to what level?

Associated symptoms: Are there color changes? Are the limbs tender? Do they feel hot? Is there itching?

Progression: Do they progress with activity or throughout the day? Or with lying down?

Other sites: Periorbital? Abdomen? Genitalia? Back? Hands?

Bilateral lower limb swelling list of DDx:

1- Heart Failure: Ask about PND, orthopnea, chest pain, palpitation, claudication, abdominal swelling, oliguria, nocturia. Ask about compliance to diet and medications

2- Chronic venous insufficiency: Hx of varicose veins, DVT.

3- Nephrotic syndrome: Frothy urine, oliguria, Wt. gain

4- Liver disease: Jaundice, Pruritis, Abdominal distention, Anorexia, GI bleeding, Vomiting, Diarrhea, Easy Bruising, Alcohol consumption

5- Lymphatic obstruction: Hx. Of malignancy, If ♀ ask about pregnancy

6- Dietary: Protein deficiency, Thiamine deficiency

7- Drugs: NSAIDs, steroids, Ca⁺² Ch. Blockers (Nifedipine, Amlodipine)

8- Immobility: Recent surgeries and hospital stay, Hx of stroke or Parkinson's disease

9- Hypothyroidism (Myxedema): Cold intolerance, Wt. gain, Decreased Appetite

10- Increased capillary permeability: Septicemia (Fever, recent infections), allergy (is he allergic to something? Was there an exposure to the allergen?)

Unilateral lower limb swelling list of DDx:

1- DVT: Ask about all risk factors in box 6.43 (Check next page)

2- Soft tissue infection (ex: Cellulitis): Fever, chills, redness, hotness, tenderness, site of entry

3- Trauma

4- Immobility: Recent hospital stay, hx of stroke or Parkinson's disease

5- Lymphedema: Hx of previous lymph node resection and radiotherapy

6- Venous Obstruction: Hx of pelvic tumor, AV fistula

7- Joint disease: Pain, hotness, redness, skin rash, decreased range of movement

Past Medical Hx	Previous hx of lower limb swelling, HTN, DM, Hyperlipidemia, IHD
Drug Hx (Box 21.1)	NSAIDs, steroids, Ca+2 Ch. Blockers (Nifedipine, Amlodipine)
Social Hx	Smoking, Alcohol intake

history to know the cause.

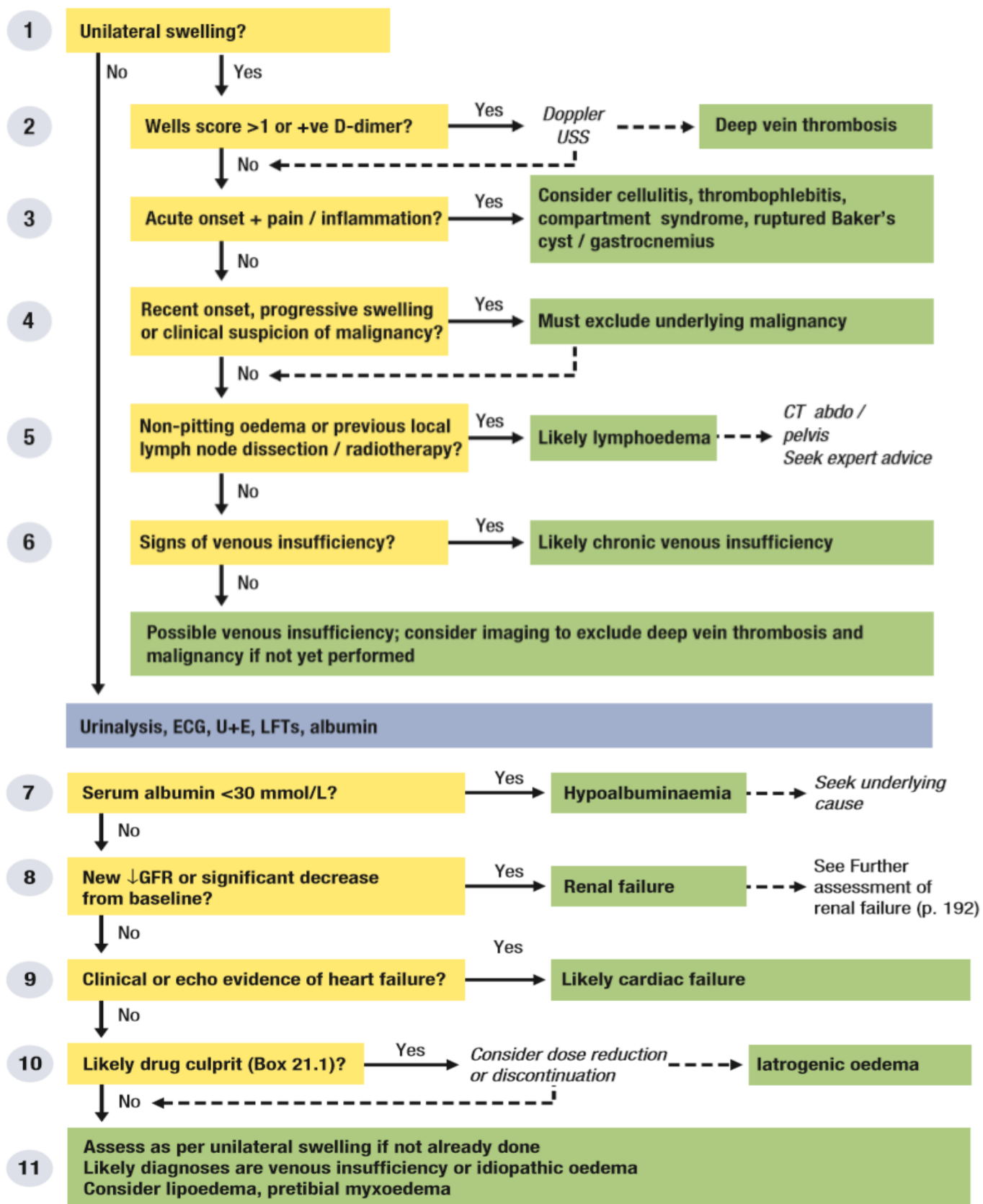


6.43 Risk factors for deep vein thrombosis

- Recent bed rest or operations (especially to the leg, pelvis or abdomen)
- Recent travel, especially long flights
- Previous trauma to the leg, especially long-bone fractures, plaster of Paris splintage and immobilisation
- Pregnancy or features to suggest pelvic disease
- Malignant disease
- Previous deep vein thrombosis
- Family history of thrombosis
- Recent central venous catheterisation, injection of drugs, etc.

Box 21.1 Drugs causing oedema

- Calcium channel blockers
- IV fluids
- Corticosteroids
- Mineralocorticoids (fludrocortisone)
- Thiazolidinediones ('glitazones')
- Withdrawal of diuretics
- NSAIDs
- Oestrogens



DVT (Examination & Management)

Inspection (30 degrees)

Wash hands. Introduce self & gain consent from patient

General Inspection

Fully expose the legs & thighs. Comment on:

Skin, legs, nails, trophic changes, ulceration, gangrene, scars, varicose veins, missing toes

Compare the colour of both legs – looking for cyanosis of the skin & toenails

Palpation (Ask if anywhere hurts first & whenever possible check expression)

Find out the patients' temperature

Compare the temperature of both feet and limbs

Start with the good limb then examine the affected limb

Look for venous distension - check to see if the veins empty promptly on elevation (if they do – its varicose). *This is caused by blood leaving via the collateral superficial veins*

Palpate the calf gently for evidence of heat & tenderness along the distribution of the deep venous system *but avoid unnecessary handling*. Look for hardening of the muscle. Watch the patients's face as you palpate

Look for pitting odema at the ankle confined to the symptomatic leg – note extent up the leg (check for odema at the ankle then move up the leg)

Look between the toes

Check for pulses. Start distally and follow the pulses proximally

Check groins for lymph nodes as well as pulses

Measure

Use a tape measure to make comparisons between both legs

Measure the diameter of the leg at mid calf (10cm below the tibial tuberosity)

A calf DVT results in calf swelling

An ileo-femoral DVT results in an entirely swollen leg

Rule out Differentials (match this findings to your history)

Cellulitis

Feels hot, appears erythematous & demarcated. Check between the toes for athletes foot (investigate by WBC & CRP)

Superficial thrombophlebitis

Localised pain with inflamed superficial veins. Lump is hard, tender and follows the vein (3-4cm) (investigate by WBC & CRP). Overlying skin may look brown

Ruptured Bakers Cyst

Painful lump below the knee and deep to gastronemius muscle. May feel hot & warm (investigate by ultrasound)

Muscle trauma/haematoma

Tender with swelling if there is a haematoma present (investigate by ultrasound)

Lymphodema

Swelling of the whole leg. Odema (long standing) is tense & non pitting. Check groins for lymph Nodes (investigate by ultrasound)

Assessing the Clinical Risk of DVT

1 point for each

Active cancer (ongoing treatment, within 6 months or palliative)

Paralysis, paresis or recent plaster immobilization of lower limb

Calf swelling 3cm> than asymptomatic side (10cm <tibial tuberosity)

Collateral superficial veins (non varicose)

Local tenderness along the distribution of the deep venous system

Pitting odema confined to the symptomatic leg

0 points

Recently bedridden for >3 days or major surgery <4 weeks

Entire leg swollen

-2 points

Alternative diagnosis at least as likely as a DVT

3+ High Probability

If D Dimer is negative – order ultrasound

1/2 Medium Probability

If D Dimer is negative or positive – order ultrasound and get a senior review

0 Low probability

If D Dimer is positive, order ultrasound and get senior review
If D Dimer is negative – consider other diagnosis or discharge after senior review

Tests for active DVT

1. tender calf swelling
2. homans sign (dorsiflex the foot)
3. mose's (squeeze calf muscles)

-Test for tenderness over deep veins over the calf . flex the knee 90 to rest foot on the table and press the calf muscle against the tibia to compress the posterior tibial veins ,

1. if its not tender then gentle squeeze the calf muscle from side to side if painful then its positive

2. if calf muscle is tender then don't squeeze , since it can dislodge the thrombus from DVT and result in pulmonary embolism

-Homan's sign : let the leg rest with the knee fully extended , then dorsiflex the foot , this will stretch the calf muscle and posterior tibial vein , if there is DVT this will cause pain and make homan test positive

-note: all these tests should perform very gently as they can dislodge thrombus and cause pulmonary embolism

DVT Exam

Tutor Notes

The student must be able to demonstrate practical skill in checking for DVT

On completion, ask the following questions (for answers – see the crib sheet)

1. What are the risk factors for developing a DVT?
2. How would you calculate the patients' risk of having a DVT?
3. How would you treat a post operative deep vein thrombosis (occurring 2 weeks post)?

Behcet disease

Definition

A systemic vasculitis that is characterized by the deposition of immune complexes in arteries and veins of all sizes

Epidemiology

- Most common from the Mediterranean region to eastern Asia, with the highest prevalence observed in Turkey and Japan
- Peak incidence: 20–40 years "The onset of Behcet disease is uncommon before puberty and after the age of 50 years."

Etiology

- Autoimmune and infectious triggers (e.g., precipitating HSV or parvovirus infection) have been suggested.
- Strong HLA-B51 association

Clinical features

- **Recurrent painful oral aphthous ulcers** (95–100%)
 - Usually last about 1–4 weeks
 - Typically the initial presenting symptom
- **Recurrent genital ulcerations** (60–90%)
 - Single or multiple ulcers that resemble oral aphthous ulcers and heal with scarring
 - Most commonly affect the vulva in female and the scrotum in male individuals
- **Ocular disease** (50–80%) "Initial presenting feature of Behcet disease in 10–20% of cases"
 - Uveitis (iridocyclitis, chorioretinitis), keratitis, and/or retinal vasculitis
 - Typically bilateral
 - More common and more severe among men
 - Usually occurs 2–3 years after the onset of oral and/or genital ulcers
- **Skin lesions** (35–85%)
 - Erythema nodosum
 - Papulopustular lesions
 - Pyoderma gangrenosum
 - Pseudofolliculitis or acneiform eruptions
 - Dermatographism: formation of urticaria after minor pressure is applied to the skin, likely mediated by local histamine release

- **Arthritis** (30–70%)
 - Non-erosive, non-deforming, asymmetric mono-/oligoarthritis
 - Usually affects the knees, ankles, hands, and/or wrists "The axial skeleton, more proximal joints such (e.g., hips, shoulder), and more distal joints (e.g., interphalangeal joints) are usually not affected. Arthritis that predominantly affects these joints suggests a different cause."
- **Gastrointestinal disease:** ileocecal ulceration with abdominal pain, anorexia, diarrhea, lower GI bleeding, nausea, vomiting "Gastrointestinal disease is more common in Japan and Korea. In Behcet disease, gastrointestinal lesions usually appear 4–6 years after the onset of oral ulcers."
- **Vasculopathy** "Although Behcet disease can affect both arteries and veins, the veins are affected more commonly than the arteries"
 - Superficial thrombophlebitis
 - Thrombosis of large veins (e.g., deep vein thrombosis, Budd-Chiari syndrome)
 - Arterial thrombosis
 - Aneurysms (e.g., pulmonary artery aneurysms) "Behcet disease is the only systemic vasculitis known to cause pulmonary artery aneurysms."
- **Neuro-Behcet syndrome** (5–10%)

"Neurological symptoms usually occur within 5 years of disease onset."

 - Parenchymal CNS disease: behavioral changes, ataxia, hemiparesis, sudden hearing loss "Vasculitis within the CNS can cause unifocal or multifocal parenchymal damage. The brain stem is often affected"
 - Extra-parenchymal CNS disease: cerebral venous thrombosis, intracranial hypertension

Diagnostics

- Positive **pathergy skin test:** erythematous papule or pustule 24–48 hours after a needle prick to a depth of 5 mm
- Autoantibodies (e.g., ANA, ANCA, rheumatoid factor) are usually absent.
- Nonspecific markers of inflammation may be present during flares (e.g., ↑ ESR, ↑ CRP).

PATHERGY: Positive pathergy test, Aphthous mouth ulcers, Thrombosis (arterial and venous), Hemoptysis (pulmonary artery aneurysm), Eye lesions (uveitis, retinal vasculitis), Recurrent Genital ulcers, Young at presentation (3rd decade)

Diagnostic criteria (International Study Group criteria)

- Recurrent oral ulceration at least three times within a 12-month period
AND
- ≥ 2 of the following
 - Recurrent genital ulceration
 - Eye lesions
 - Skin lesions
 - Positive pathergy test

Differential diagnosis

- Crohn disease
- Aphthous stomatitis (e.g., due to vitamin B₁₂/folate/iron deficiency, gluten-sensitive enteropathy)
- Herpes infection
- Sweet syndrome
- Reactive arthritis
- SLE

Treatment

- Oral ulcers and/or genital ulcers: topical corticosteroids
- Topical lidocaine for pain relief
- Skin lesions
 - Papulopustular lesions: See "Treatment" of "Acne vulgaris".
 - Erythema nodosum: colchicine
- Arthritis: colchicine
- Ocular disease, CNS disease, and/or vasculopathy
 - Systemic corticosteroids
 - Immunosuppressant therapy (e.g., azathioprine, infliximab, cyclosporine A, cyclophosphamide, IFN- α , methotrexate)

❁ *Diabetes mellitus* ❁



History Taking

❖ History taking: -in brief-

➤ Quick review:

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. Long-standing metabolic derangement can lead to the development of complications of diabetes, which characteristically affect the eye, kidney and nervous system.

➤ Symptoms of hyperglycemia:

Diabetes mellitus may present with a classical triad of symptoms:

- Polyuria (and nocturia): due to osmotic diuresis caused by glycosuria.
- Thirst: due to the resulting loss of fluid and electrolytes.
- Weight loss: due to fluid depletion and breakdown of fat and muscle secondary to insulin deficiency.

Other common symptoms are:

- Tiredness, mood changes, poor concentration and blurred vision (due to glucose-induced changes in lens refraction).
- Bacterial and fungal skin infection are common because of the combination of hyperglycemia impaired immune resistance and tissue ischemia. Itching of the genitalia (pruritus vulvae in women, balanitis in men) is due to Candida yeast infection (thrush).
- Headache.

➤ **Diabetic patient follow up:**

- Ask about sugar reading at home (at morning “ should be less than 120”, after launch “less than 200”).
- Hemoglobin A1c: should be less than 7.
- LDL: less than 100.
- Creatinine: within a normal level.
- Ask about symptoms of DM: Polydipsia, polyuria.
- Ask about symptoms of hypoglycemia:
Confusion, Dizziness ,Feeling shaky, Hunger, Headaches, Irritability, Pounding heart; racing pulse, Pale skin, Sweating , Trembling, Weakness ,Anxiety.
- Ask about the patient vision, is there any changes, any blurring?
- If the measured readings was not in the levels required, then check the patient compliance; if the patient is compliant then you may need to change or adjust the doses of the drugs and insulin.

➤ **Secondary diabetes:**

• **Overview:**

Causes of secondary DM can be grouped into three groups to make it easier to memorize and understand these groups are:

1- Endocrine diseases:

Cushing syndrome.
Acromegaly.
Thyrotoxicosis.
Pheochromocytoma
Glucangonoma
PCOS

2- Pancreatic disease:

Hemochromatosis.
Wilson’s disease.
Cystic fibrosis.
Chronic pancreatitis
Pancreatic cancer
Pancreatic surgery

Questions box 29.4

Questions to ask the diabetic patient

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. What was your age at the time the diabetes was diagnosed?
2. Did you require insulin from the start?
3. What was the problem that led to the diagnosis? (Polyuria, thirst, weight loss, recurrent skin infections, screening assessment)
4. What previous and current drug treatment are you taking for diabetes?
5. What diet has been prescribed? What do you understand about your diabetic diet?
6. What blood sugar testing do you do? What are the usual results?
- ! 7. Have you had any problems with hypoglycaemia (treatment-induced low blood sugar)? Have you had episodes of sweating, confusion, malaise or unconsciousness?
8. Do you know what action should be taken if these acute symptoms (of hypo- or hyperglycaemia) occur? (Check sugar level, take glucose tablet, go to hospital)
9. Have you had ketoacidosis (very high blood sugar associated with acidosis) and needed admission to hospital? (Polyuria, dehydration, confusion, unconsciousness)
10. Have you had complications of diabetes—eyes, nerves, blood vessels, kidneys?
11. What regular testing has been performed for these problems?
12. How do you and your family cope with this chronic condition?
13. Have you been able to work?

3- **Drug induced DM**

“Clinical scenario: 30-year-old male patient presented to endocrine clinic with fasting blood sugar of 200 and HbA1c of 8, take a proper history to know the secondary causes of his condition.”

- **History taking:**

- I. Endocrine diseases:**

- Cushing syndrome: does the patient take steroids, does the patient complain of central obesity, weight gain, moon face, and proximal muscle weakness....etc.
 - Acromegaly: did that patient note that his/her foot size increased? Does his/her ring become smaller? Hands, foot, nose any soft tissue enlargement, does he complain of visual field defect?
 - Thyrotoxicosis: does the patient complain of weight loss, increased appetite, palpitations or heat intolerance?
 - Pheochromocytoma: does the patient complain of recurrent episodes of headache, palpitation, sweating, tremor...
 - Glucagonoma: usually presents with nonspecific symptoms like fatigue.
 - Increased levels of estrogen: does the patient take exogenous estrogen? Diagnosed with or have the symptoms of PCOS (menstrual irregularities and hirsutism)?

- II. Pancreatic diseases:**

- Hemochromatosis: skin discoloration, fatigue, impotence in males or amenorrhea in females, arthralgia...etc.
 - Wilson's: is there any family history of Wilson's disease? Does the patient recently complain of neurological and liver failure symptoms?
 - Cystic fibrosis: young patient with recurrent chest infection, infertility in males.
 - Surgery: Did the patient have any pancreatic surgery?
 - Chronic pancreatitis: does he complains of recurrent attacks of epigastric pain with nausea and vomiting?
 - Pancreatic cancer: does he complain of weight loss, loss of appetite or night sweats.

- III. Drug induced DM:** these include thiazide diuretics, beta blockers



Physical Examination

❖ Diabetic foot examination:

Diabetic foot is a complication of both ischemic and neuropathic problems of diabetes. Most likely, the station would be examine the lower limbs for a patient with DM.

- **Position and exposure:** the patient is supine and the lower limbs are exposed ideally to the mid-thigh.

- **Inspection:**

- 1- **Skin changes:**

- Pallor
 - Erythema
 - Hair loss
 - Dry/ sweaty skin.
 - Necrobiosis lipoidica.
 - Is there any ulcers? If yes then examine them fully .
 - Is there any gangrene? If yes then examine them fully.
 - Inspect between the toes checking for any infection.
 - Inspect the heel; you may notice hyperkeratosis.

- 2- **Musculoskeletal changes:**

- Joint deformities –Charcot joint, claw toe, hammer toe or hallux valgus.
 - Notice any muscle wasting.

- 3- **Finally, the nails:** check for thickening of the nail or onchogryphosis.

- **Palpation:**

- Tenderness.
 - Temperature -always compare to the other side-
 - Capillary refill. *Normally capillary refill takes 2-3 seconds.*
 - Pulses of the lower limb –femoral, popliteal, dorsalis pedis and posterior tibial- .
 - Edema if found.

- **Neural exam:**

- Sensation (do not forget to ask the patient to close his eyes then test for it).
 - Vibration (tested with tuning fork over a bony prominence).
 - Proprioception (do not forget to ask the patient to close his eyes then test for it).
 - Test the reflexes.

All of these may be absent or reduced.

- **Finishing your exam:**

→ Check the patient's gait: as Charcot joint may affect the gait-

Mentioned that it is indicated to measure Ankle Brachial index "ABI".

