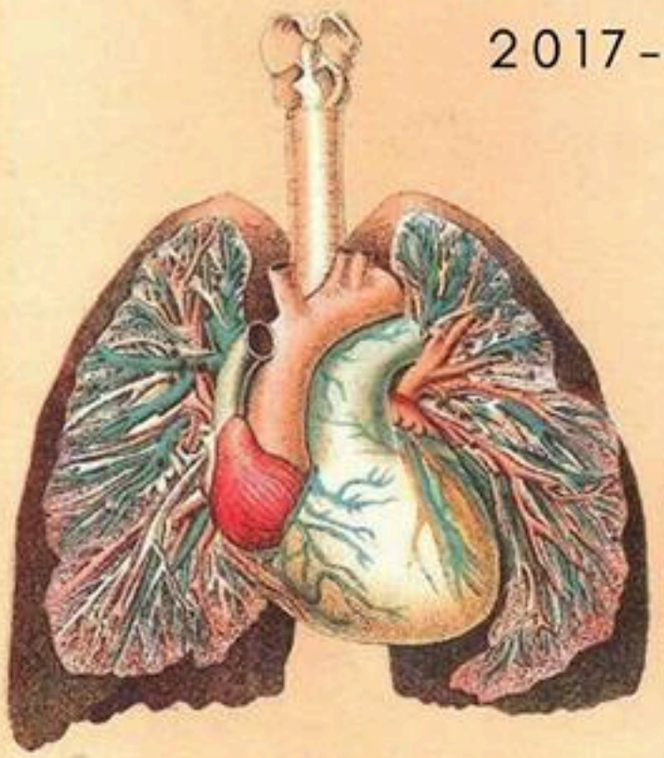
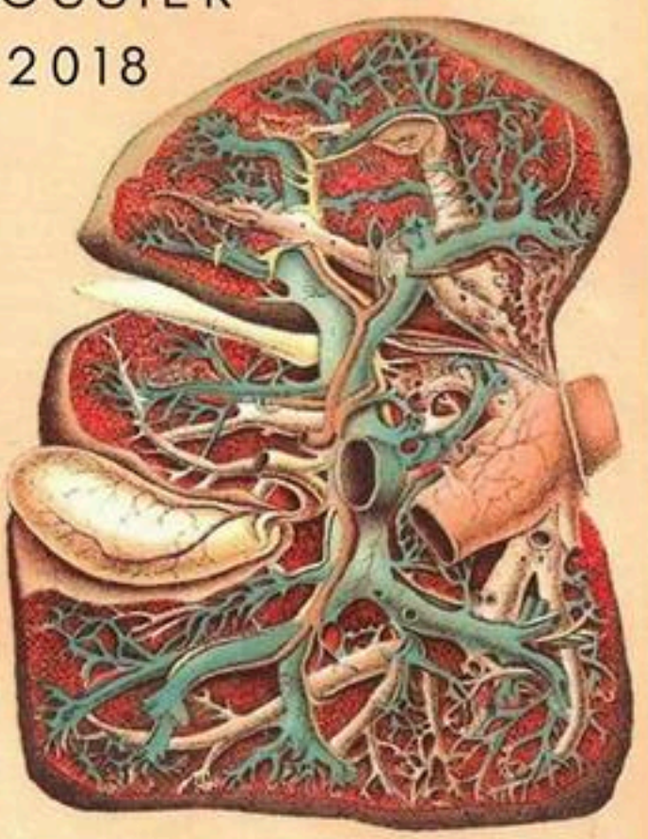
INTERNAL MEDICINE

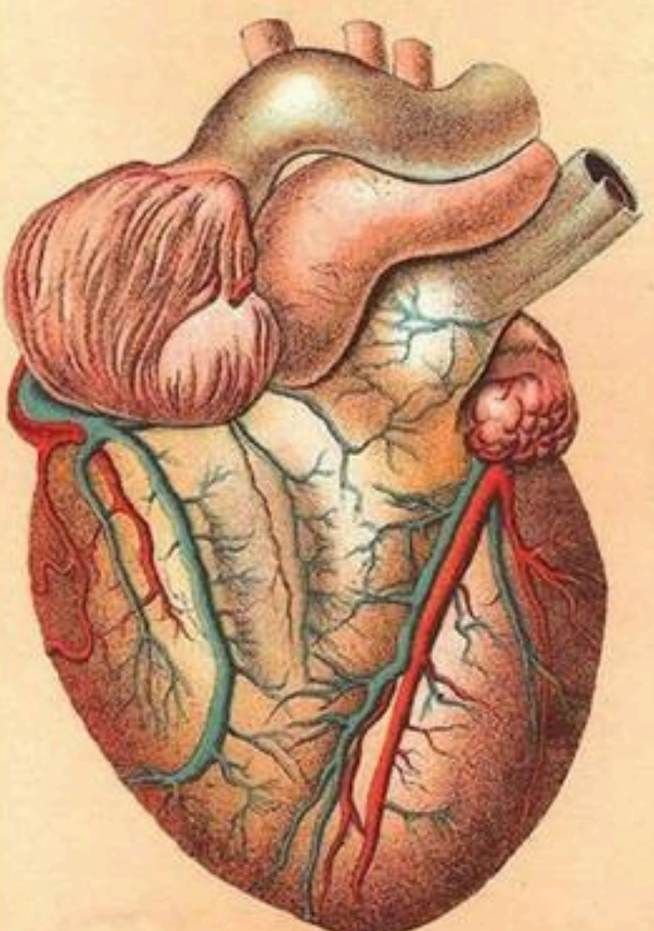
OSCE DOSSIER
2017-2018



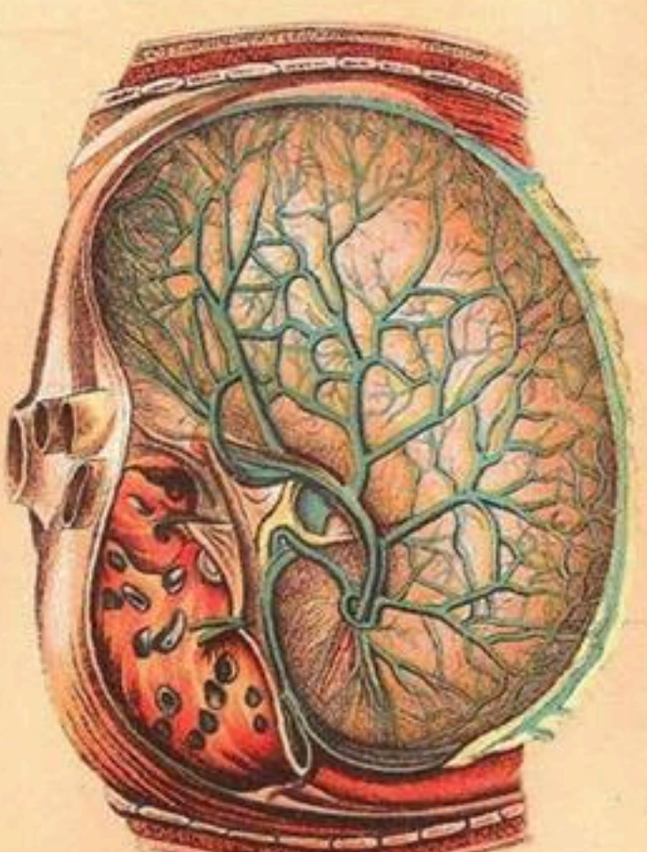
Lungs & Heart in Position



Section of Liver



The Heart Showing the Great Arteris.



The Stomach – Front view Figure in Reclining Position.

Human Internal organs.

OSCE Dossier

2018/2019 Edition

Authors

Ayat Al-Zghoul

Bushra Arafa Zayed

Hashim Ahmad Mohammad

Haya Yanes

Linda Al-bsoul

Majd Rawashdeh

Mohammad Qussay Al-Sabbagh

Raghad Bataineh

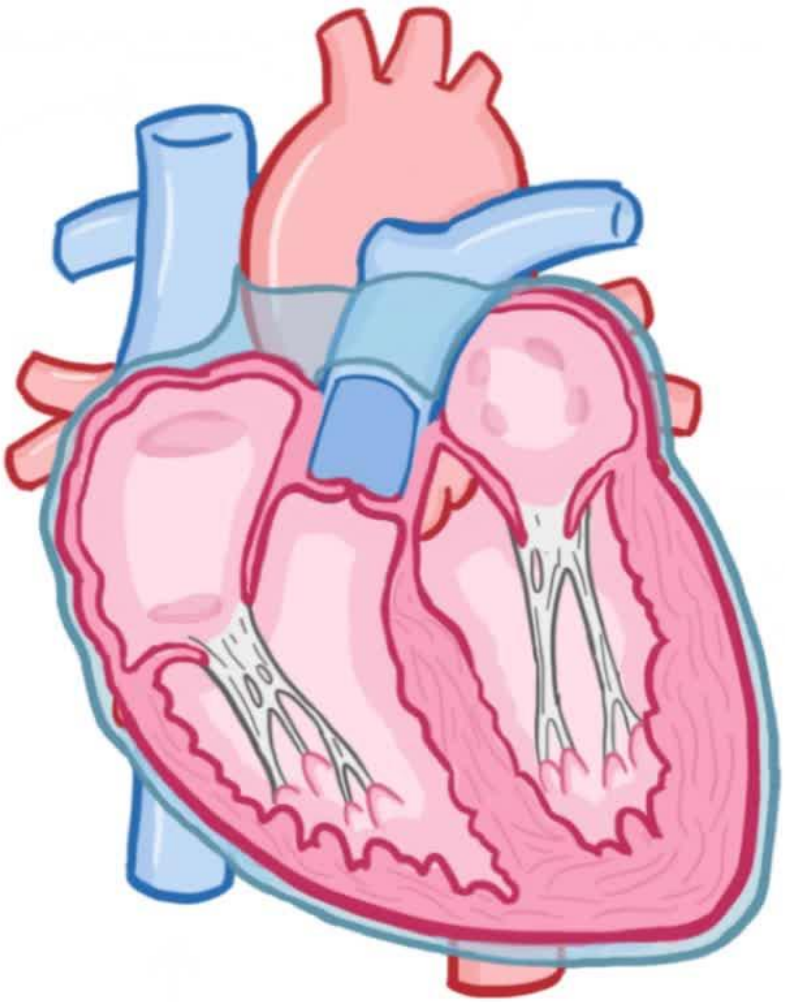
Reem Akiely

Acknowledgment

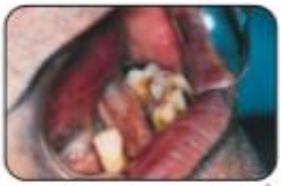
Special thanks to Bushra Arafa and Majd Rawashdeh for their significant contribution on editing and adding new content to the 2017/2018 edition of the dossier.

Contents

Chapter 1: Cardiology



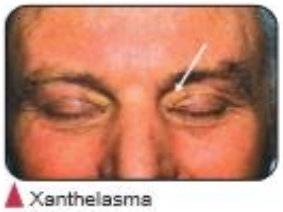
CLINICAL EXAMINATION OF THE CARDIOVASCULAR SYSTEM



Poor oral hygiene in a patient with infective endocarditis

6 Face, mouth and eyes
Pallor
Central cyanosis
Malar flush
Dental caries
Fundi (retinopathy)
Stigmata of hyperlipidaemia and thyroid disease

Malar flush



Xanthelasma

5 Jugular venous pulse
(see opposite)
Height
Waveform

Jugular venous pulse

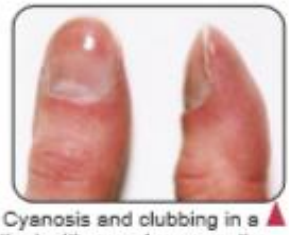
4 Carotid pulses
Volume
Character
Bruits
(see opposite)

3 Blood pressure

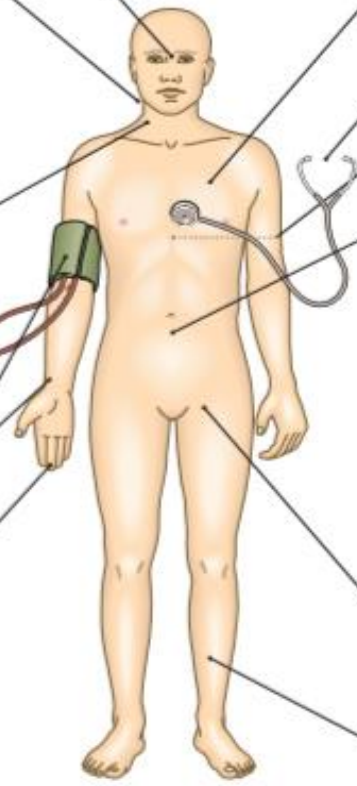
2 Radial pulse
Rate
Rhythm

1 Hands
Clubbing
Splinter haemorrhages and other stigmata of infective endocarditis

Splinter haemorrhage



Cyanosis and clubbing in a patient with complex cyanotic congenital heart disease



7 Precordium
Inspect
Palpate
(see opposite)

8 Auscultation
(see opposite)

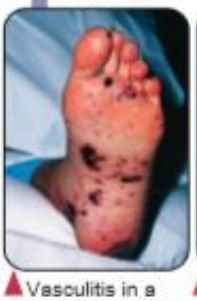
9 Back
Lung crepitations
Sacral oedema

10 Abdomen
Hepatomegaly
Ascites
Aortic aneurysm
Bruits

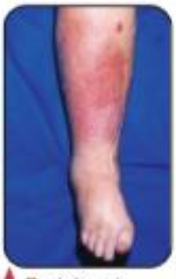
11 Tendon xanthomas
(hyperlipidaemia)

12 Femoral pulses
Radio-femoral delay
Bruits

13 Legs
Peripheral pulses
Oedema



Vasculitis in a patient with infective endocarditis



Peripheral oedema in a patient with congestive cardiac failure

Observation

Symptoms and well-being
- Breathlessness
- Distress etc.

Body habitus
- Body mass (obesity, cachexia)
- Marfan's and other syndromes

Tissue perfusion
- Skin temperature
- Sweating
- Urine output

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 3rd 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 t 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). -**<u>Causes of exacerbation</u>**: 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 Rhinits, 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)	No underlying heart disease	-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	-People with cardiomyopathy or previous MI -People with pacemakers or Intra-cardiac devices have increased risk of V. Tach

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

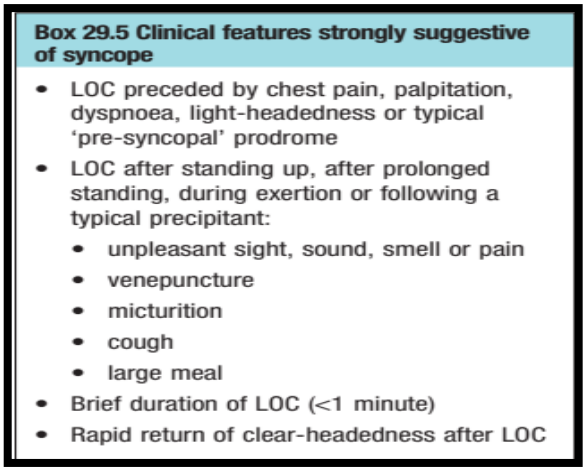
Syncope:

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

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:	**Unilateral 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	1- DVT: Ask about all risk factors in box 6.43 (Check next page)
2- Chronic venous insufficiency: Hx of varicose veins, DVT.	2- Soft tissue infection (ex: Cellulitis): Fever, chills, redness, hotness, tenderness, site of entry
3- Nephrotic syndrome: Frothy urine, oliguria, Wt. gain	3- Trauma
4- Liver disease: Jaundice, Pruritis, Abdominal distention, Anorexia, GI bleeding, Vomiting, Diarrhea, Easy Bruising, Alcohol consumption	4- Immobility: Recent hospital stay, hx of stroke or Parkinson's disease
5- Lymphatic obstruction: Hx. Of malignancy, If ♀ ask about pregnancy	5- Lymphedema: Hx of previous lymph node resection and radiotherapy
6- Dietary: Protein deficiency, Thiamine deficiency	6- Venous Obstruction: Hx of pelvic tumor, AV fistula
7- Drugs: NSAIDs, steroids, Ca^{+2} Ch. Blockers (Nifedipine, Amlodipine)	7- Joint disease: Pain, hotness, redness, skin rash, decreased range of movement
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?)	

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

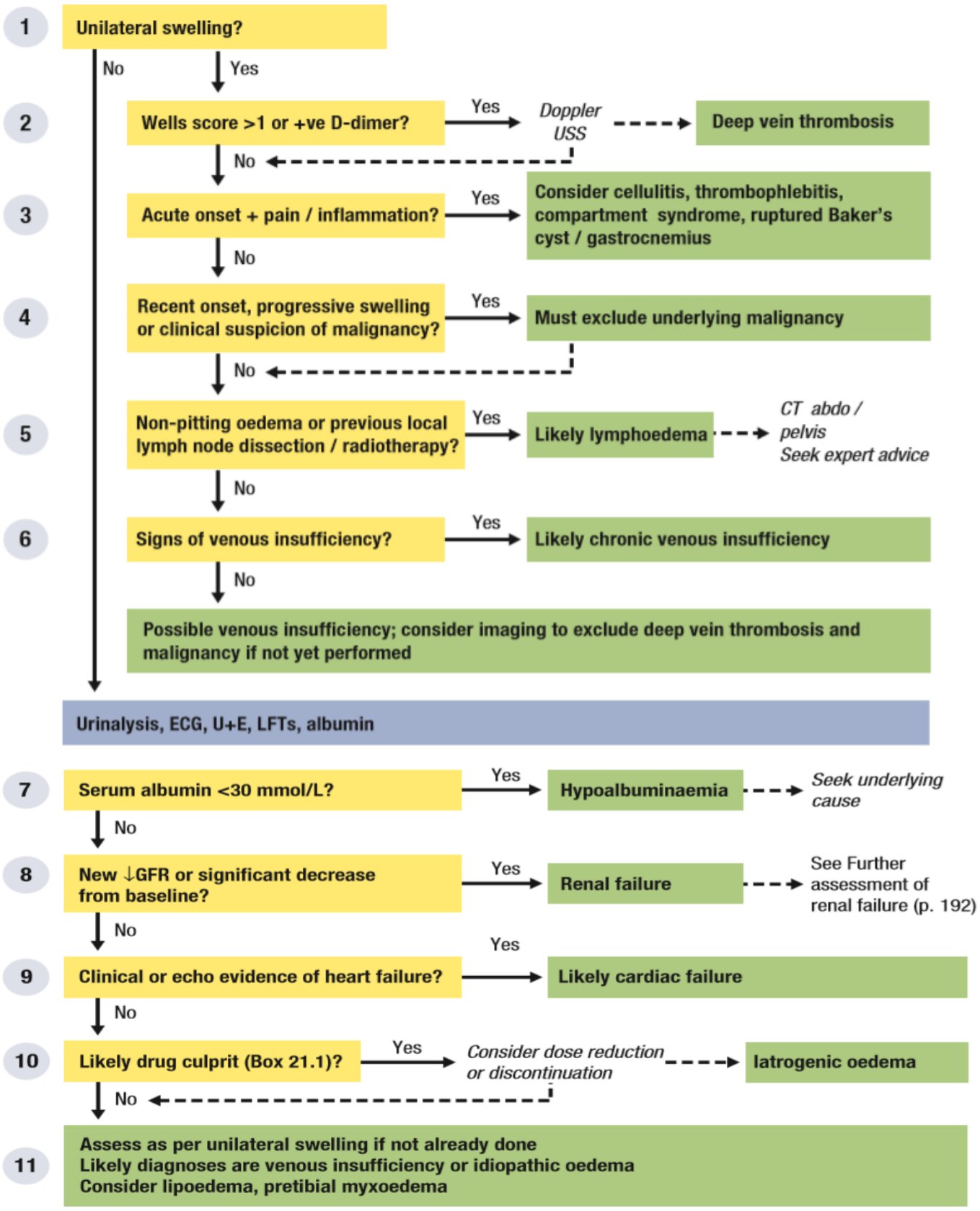
history to know the cause.

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


1
Unilateral swelling?
No
Yes
2
Wells score >1 or +ve D-dimer?
Yes
Doppler USS
Deep vein thrombosis
No
3
Acute onset + pain / inflammation?
Yes
Consider cellulitis, thrombophlebitis, compartment syndrome, ruptured Baker's cyst / gastrocnemius
No
4
Recent onset, progressive swelling or clinical suspicion of malignancy?
Yes
Must exclude underlying malignancy
No
5
Non-pitting oedema or previous local lymph node dissection / radiotherapy?
Yes
Likely lymphoedema
CT abdo / pelvis
Seek expert advice
No
6
Signs of venous insufficiency?
Yes
Likely chronic venous insufficiency
No
Possible venous insufficiency; consider imaging to exclude deep vein thrombosis and malignancy if not yet performed
Urinalysis, ECG, U+E, LFTs, albumin
7
Serum albumin <30 mmol/L?
Yes
Hypoalbuminaemia
Seek underlying cause
No
8
New ↓GFR or significant decrease from baseline?
Yes
Renal failure
See Further assessment of renal failure (p. 192)
No
9
Clinical or echo evidence of heart failure?
Yes
Likely cardiac failure
No
10
Likely drug culprit (Box 21.1)?
Yes
Consider dose reduction or discontinuation
Iatrogenic oedema
No
11
Assess as per unilateral swelling if not already done
Likely diagnoses are venous insufficiency or idiopathic oedema
Consider lipoedema, pretibial myxoedema

Examination of CVS

- Greet your patient, introduce yourself then ask for permission to examine him.

  Wash your hands and ensure adequate privacy, warmth and illumination.

  Stand to the Rt. Side of the patient.

  Expose the anterior chest to the umbilicus, position the patient in semi recombinant position (45^{o}).

- **General examination:**

  First impression: The pt is conscious, alert, oriented to time, place and person.

  Not in distress, no hoarseness of voice, not cyanosed, no IV lines.

  Vitals: RR, Temp, BMI, BP, Pulses

  Pulses:

  -Radial → Comment on rate, rhythm, compressibility, radio-radial asymmetry (as in subclavian or aortic disease), ask if he has pain in his shoulder then examine for collapsing pulse (occurs in Aortic Regurgitation And Patent Ductus Arteriosus), radio-femoral delay (as in Coarctation of the aorta), pulse deficit (Difference between radial pulse and HR by auscultation more than 10, occurs in A. Fib).

  -Brachial → Comment on rate, rhythm, volume (Small, Large, Normal),

  character (**Collapsing** as in Aortic Regurgitation and Patent Ductus Arteriosus, **Slow-Rising** as in Aortic Stenosis, **Bisferens** as in Concomitant Aortic Regurg. and Aortic Stenosis or HOCM, **Alternans** as in Advanced Heart failure), compressibility, brachio-brachial delay.

  Take BP then look for pulsus paradoxus and postural hypotension.

  -Carotid → NEVER assess both simultaneously, use the thumb that's contralateral to the side examined, comment on rate, rhythm, volume, character, compressibility.

  -Lower limb pulses → (Femoral, Popliteal, Posterior tibial, Dorsalis pedis) comment if palpable or not.

- **Hands**: inspect for Clubbing, Splinter hemorrhages, Peripheral cyanosis, Tar stains, skin and tendon Xanthomata, Janeway's lesions, Osler's nodes.

  Palpate for temperature (using the dorsum of your hand) and comment if sweaty or dry.

  Then examine for Fine tremor and Flapping tremor (Asterexis).

- **Face**: comment on Conjunctival Pallor, Jaundice, Conjunctival Hemorrhage, Corneal Urcus, Xanthelasma, Malar flush, Central cyanosis, Dental carries, Angular stomatitis, Glossitis. Ask to perform fundoscopy looking for Roth's spots or hypertensive retinopathy.

- **Neck**: Examine for scars, visible masses, distended veins.

❖ **JVP examination**:

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

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.

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.

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.

Auscultate for venous hum using diaphragm.

❖ **Examination of the Precordium:**

Inspection: start from the foot of the bed and comment on chest deformities and body hair distribution. Move to the Rt. Side of the pt. (make sure to lean forward to look at the lt. axilla) and comment on scars or masses, visible pulsations, dilated veins.

Palpation: Ask for permission, ask for any area of pain, warm your hands and maintain eye-to-eye contact.

Assess for tenderness all over the chest.

Examine for the apex beat first using your entire palm then locate it using 2 fingers. If impalpable tilt the pt. to his Lt. side.

Comment that it's gentle tapping localized at Lt. 5th intercostal space, midclavicular line.

Examine for thrills using the pulps of your fingers at the apex, Rt. Parasternal area and Lt. Parasternal area.

Ask the pt. to hold his breath on expiration then examine for heaves using your palm at the apex and Lt. Parasternal area.

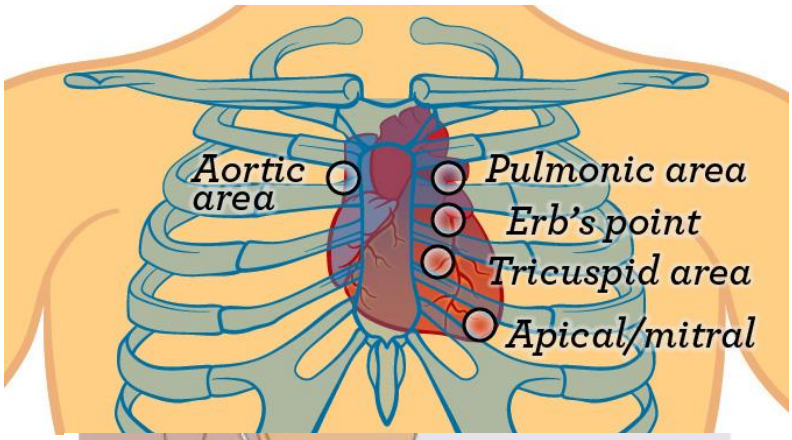
Auscultation:

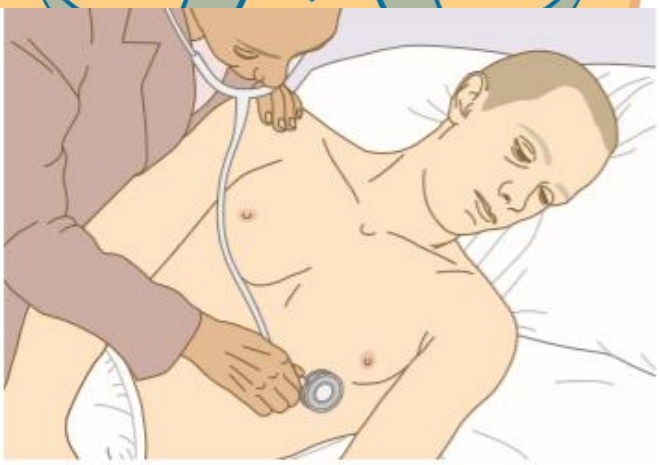
1- Use the diaphragm to auscultate in the 4 areas (Aortic, Pulmonic, Tricuspid and Mitral), palpate the carotid pulse while doing so to time any murmurs. Ask the pt. to take a deep breath while on the pulmonic area to auscultate for splitting.

2- Switch to the bell and auscultate over the apical area looking for added sounds (S3, S4).

Ask the pt. to roll to his Lt. side and auscultate for murmurs of mitral stenosis. (Figure A)

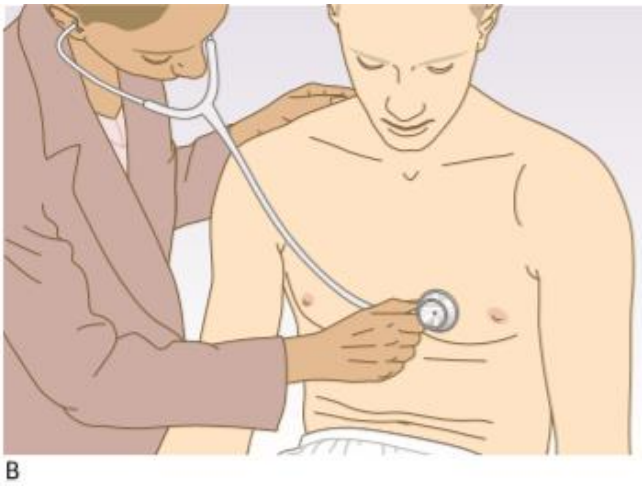
Ask the pt. to hold his breath on inspiration and auscultate for carotid bruits.





A

3- Switch to the diaphragm again and ask the pt. to sit up, lean forward and hold his breath in full expiration. Place the stethoscope's diaphragm on "Erb's area" to auscultate for murmur of aortic regurgitation. (Figure B)



B

While the pt. is sitting upright auscultate for basal lung crackles.

Test for sacral edema.

❖ Lower Limbs

Exposure should be all the way up to the umbilicus (for cultural concerns expose to the mid-thigh).

Inspection:	Look at all leg aspects for signs of **ischemia**: Amputated Limb or toe, Thick hypertrophied nails, Peripheral cyanosis, Pallor, Hair loss, Skin discoloration, Skin changes (Dry skin or Shiny skin), decreased muscle bulk, Charcot's joint deformity, Obvious swelling. Look at the heel and between the toes for ulcers.
	Look at all leg aspects for signs of **chronic venous insufficiency**: Visible distended / tortuous veins, Obvious scars, Obvious swelling, Redness or Hyperpigmentation, Thick skin, Ulcers.
Palpation:	-Feel for Temperature difference. -Feel for palpable masses, Coarse skin, Tenderness on squeezing the muscle. -Test for capillary refill by compressing a toenail (normally it's restored in 2 seconds maximum). -Feel the pulses: Dorsalis Pedis: Just lateral to the extensor halluces longus muscle, in the middle of the foot. Posterior Tibial: 2cm below and 2cm behind the medial malleolus or midway between the medial malleolus and the heel. Popliteal: Press firmly behind the 30^{o} flexed knee. Femoral: Midway between the ASIS and the symphysis pubis; just below the inguinal ligament. -Do neurological exam for power, sensation and vibration sense. -Check for edema by pressing using your thumb on the shaft of tibia then above the medial malleolus. -Measure the circumference of both legs at the same point (10cm below the tibial tuberosity).
Special tests:	-When examining for PAD: Perform Buerger's test (Next page). -When examining for Venous disease : Perform Trendelenburg test (Next page).
Auscultation:	-Auscultate over femoral arteries for bruits.

Buerger's test

Examination sequence

- With the patient lying supine, stand at the foot of the bed. Raise the patient's feet and support the legs at 45° to the horizontal for 2–3 minutes.
- Watch for pallor with emptying or 'guttering' of the superficial veins.
- Ask the patient to sit up and hang the legs over the edge of the bed.
- Watch for reactive hyperaemia on dependency; the loss of pallor and spreading redness is a positive test.

The Trendelenburg test

Examination sequence

- Ask the patient to sit on the edge of the examination couch.
- Elevate the limb as far as is comfortable for the patient and empty the superficial veins by 'milking' the leg towards the groin.
- With the patient's leg still elevated, press with your thumb over the sapheno-femoral junction (2–3 cm below and 2–3 cm lateral to the pubic tubercle). A high thigh tourniquet can be used instead.
- Ask the patient to stand while you maintain pressure over the saphenofemoral junction.
- If saphenofemoral junction reflux is present, the patient's varicose veins will not fill until your digital pressure, or the tourniquet, is removed.

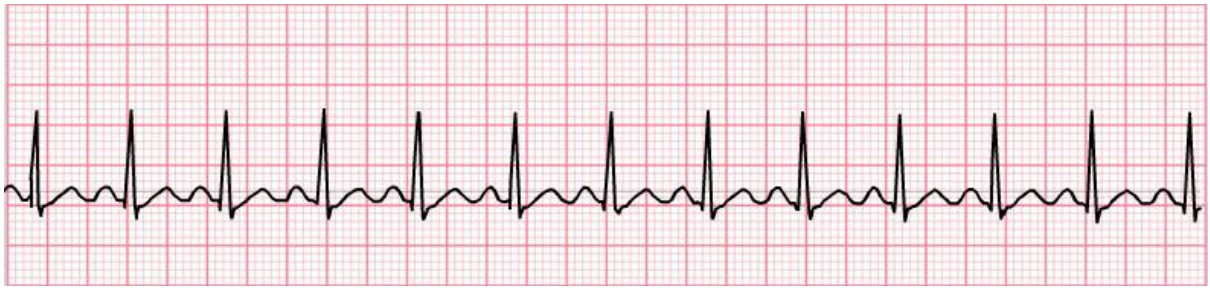
❖ **ECG interpretation:**

1-STEMI

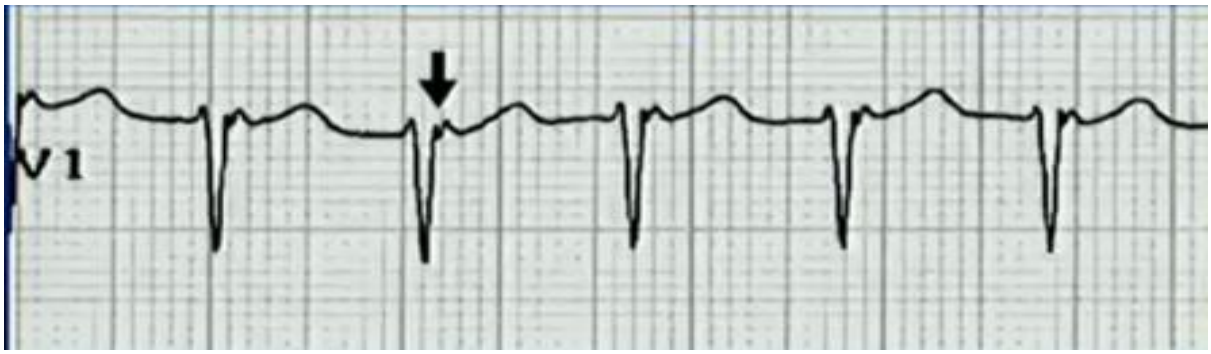
- ST-elevation in leads **II, III, aVF → Inferior** wall MI → Occlusion of **Rt. Coronary artery**.
- ST- elevation in leads **I, aVL, V5, V6 → Lateral** wall MI → Occlusion of **circumflex artery**.
- ST-elevation in leads **V1,V2,V3,V4 → Anterior** wall MI → Occlusion of **LAD**.
- ST-depression in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

2- Tachycardia (HR >100)

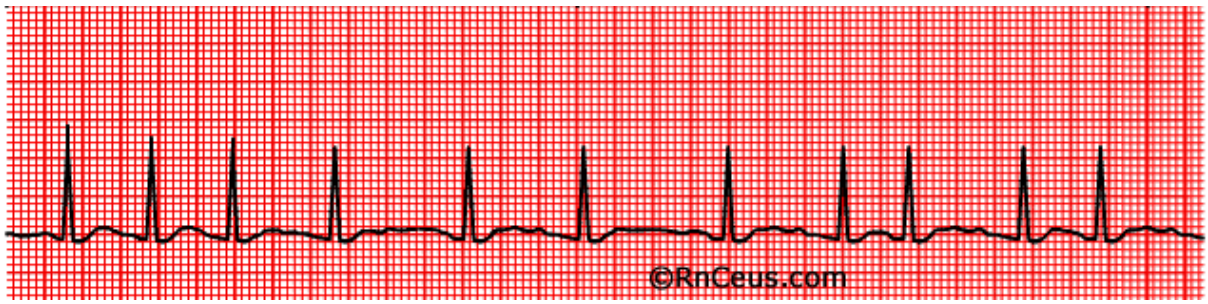
Is it fast?	**Is it regular?**	**QRS (normally ≤3 small squares)**	**P wave present?**	**------**	**Diagnosis**
✔	✔	Narrow	✔		Sinus tachycardia
✔	✔	Narrow	***X*** (Instead there's a retrograde P wave in V1)		SVT
✔	***X***	Narrow	***X***		A. Fib
✔	✔	Narrow	✔✔✔ موجودة وبقوة (more than one P wave for each 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



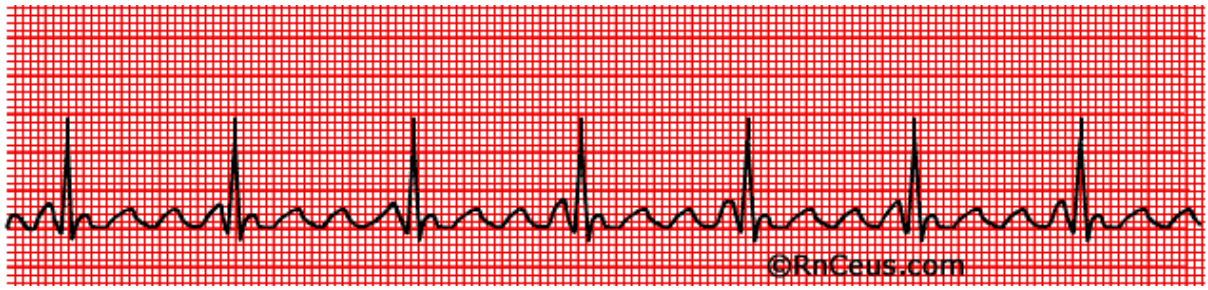
Sinus Tachycardia



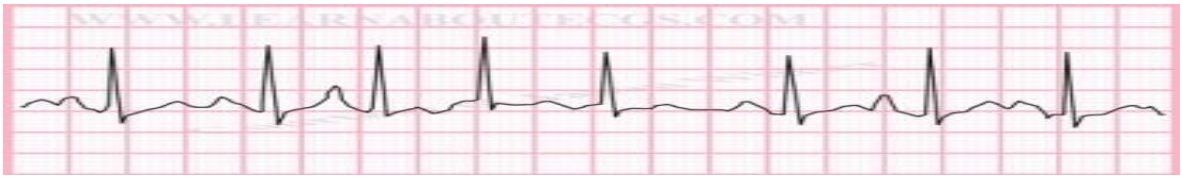
SVT



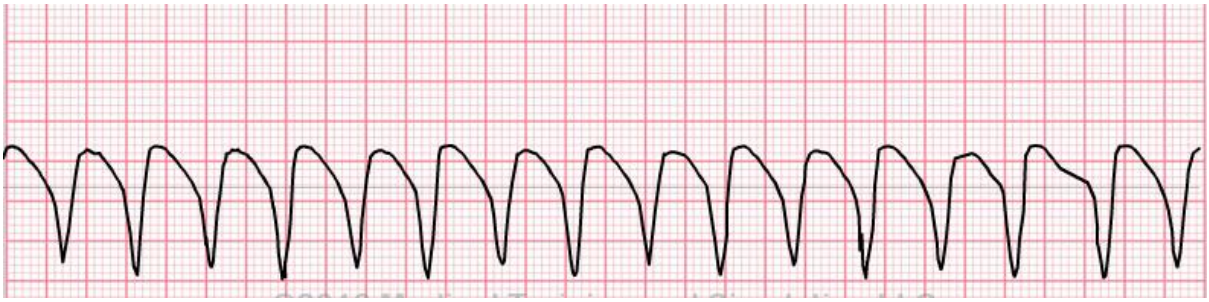
A-Fib



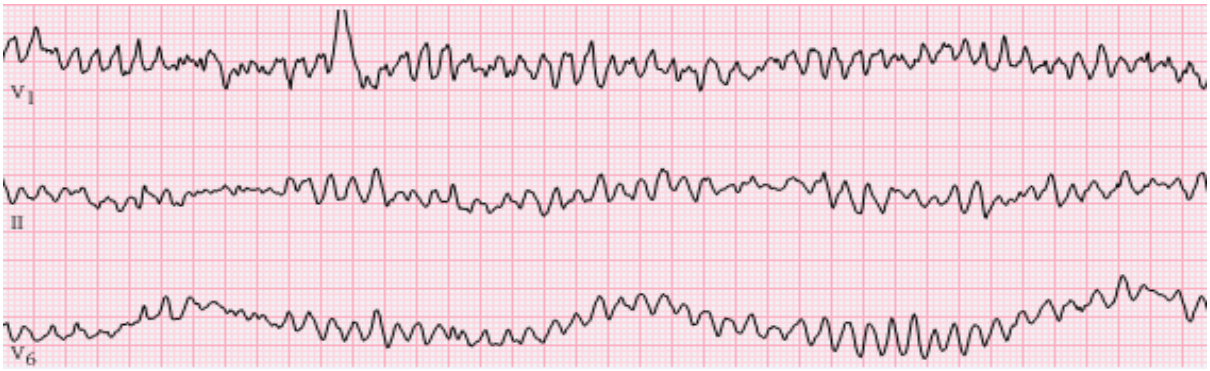
A-Flutter



MAT



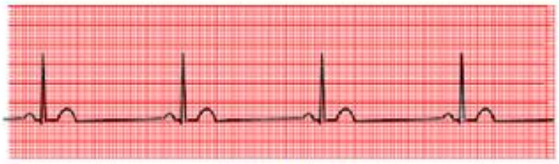
V-Tach



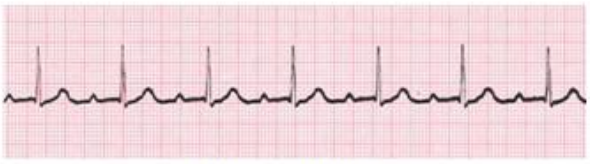
V-Fib

3- Bradycardia (HR<60)

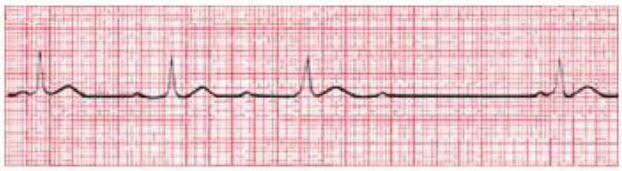
Is it slow?	Look at the PR intervals	Are there dropped beats?	Dx
✔	They're of normal length (< 5 small squares) and they're fixed	***X***	Sinus bradycardia
✔	They're elongated (>5 small squares) but fixed	***X***	1st degree AV block
✔	There's progressive prolongation of PR intervals then a dropped beat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



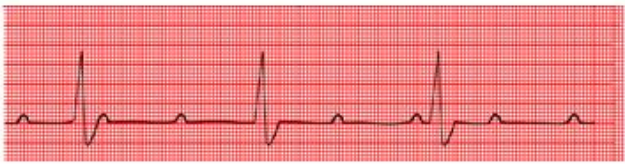
Sinus Bradycardia



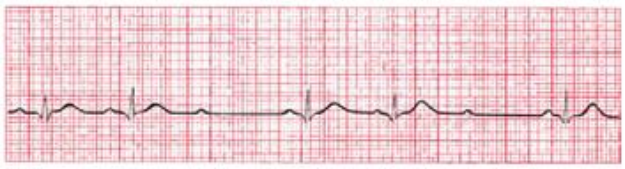
1st Degree AV Block



2nd degree AV block type 1 (Wenckebach block)



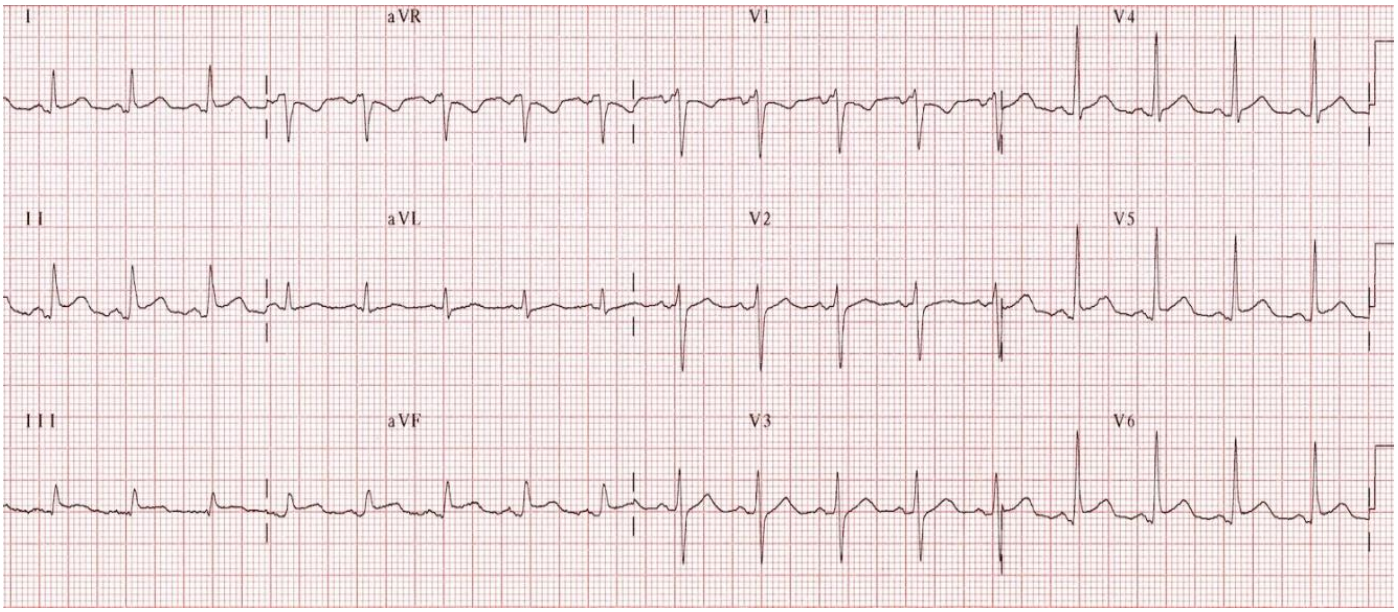
3rd degree AV block (complete heart block)



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

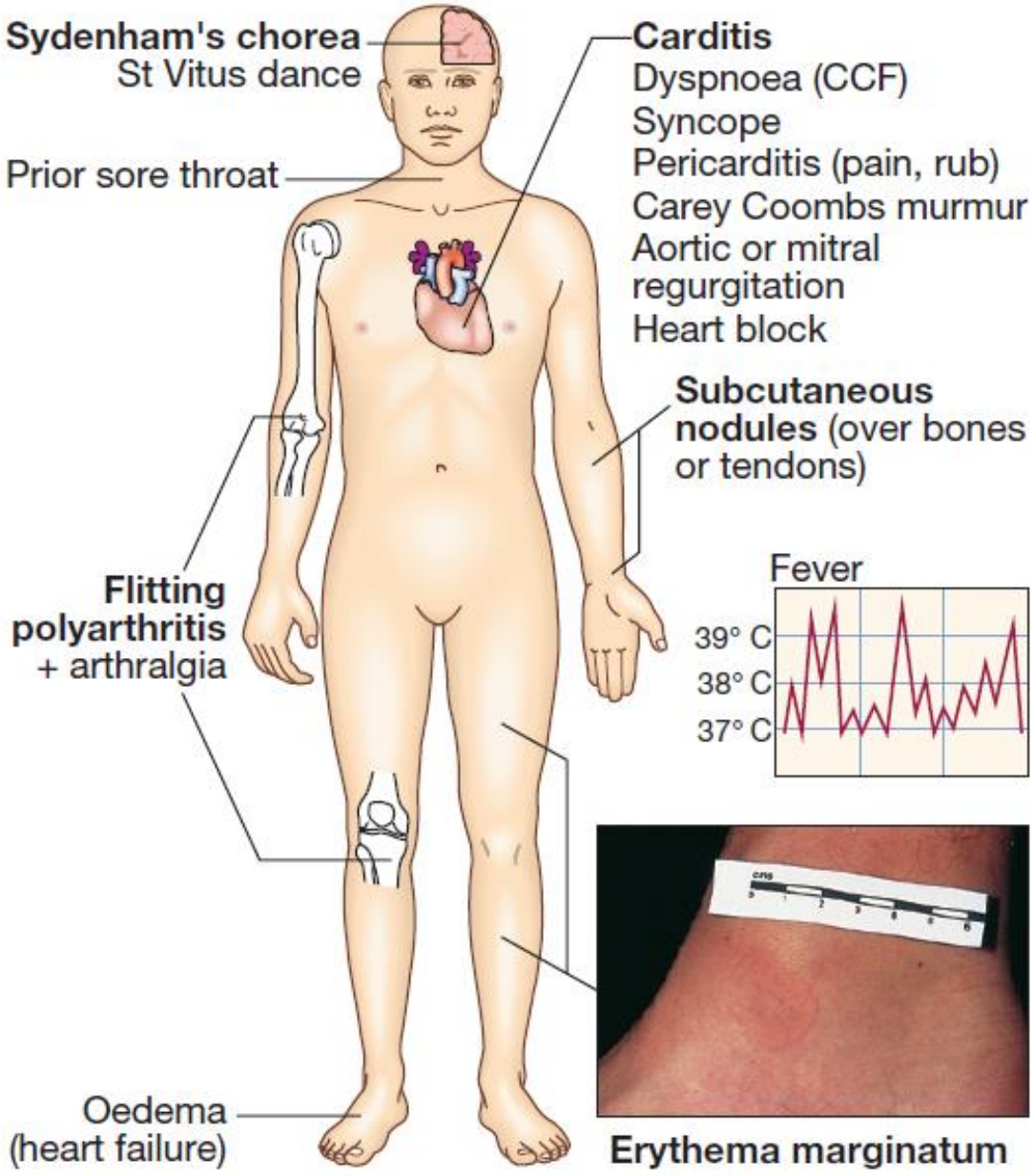


Fig. 18.86 Clinical features of rheumatic fever. Bold labels indicate Jones major criteria (CCF = congestive cardiac failure). *Inset (Erythema marginatum)* From Savin et al. 1997 – see p. 641.

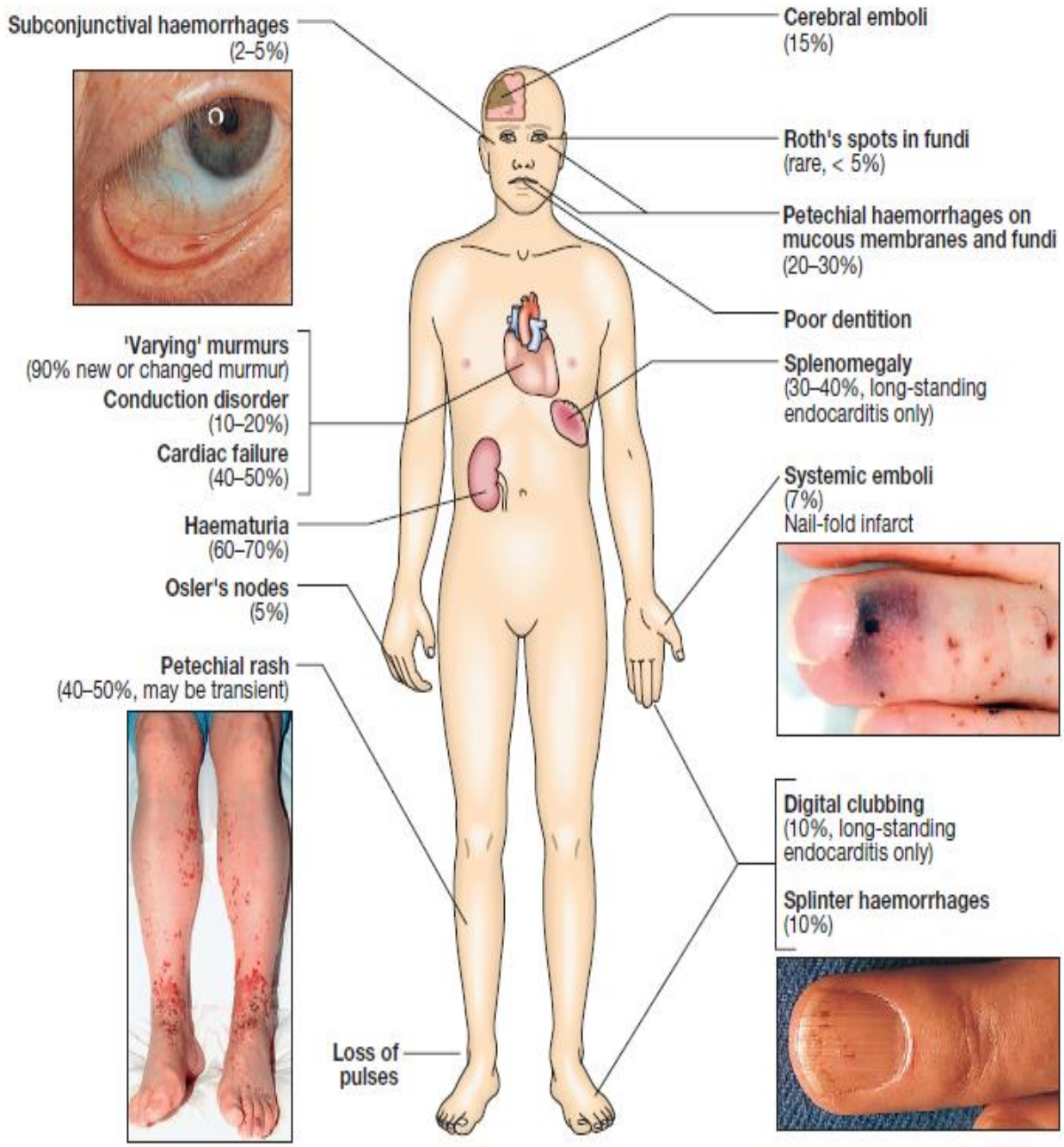


Fig. 18.93 Clinical features which may be present in endocarditis. *Insets (Petechial rash, nail-fold infarct)* From Newby and Grubb 2005 – see p. 641.

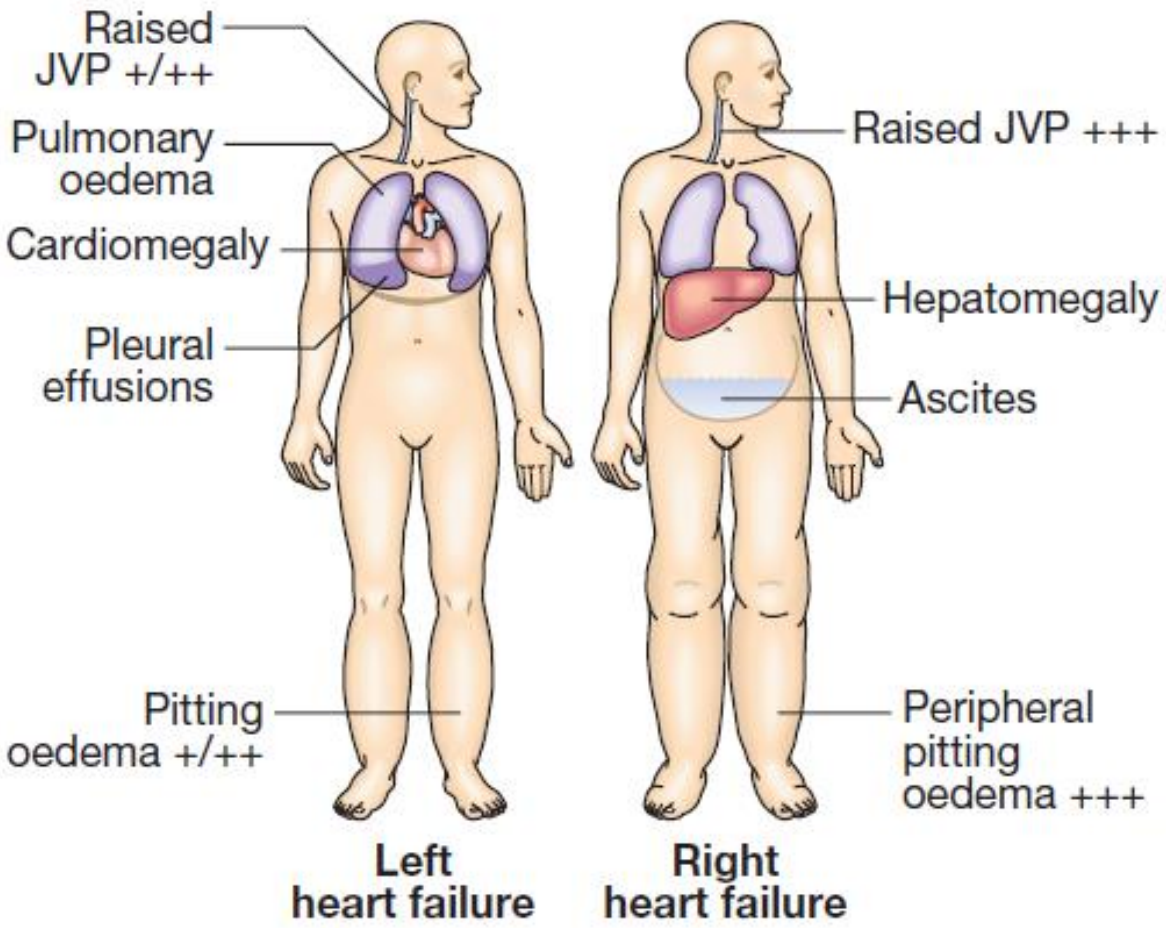


Fig. 18.24 Clinical features of left and right heart failure.
(JVP = jugular venous pressure)

List of Resources:

1- Davidson's Principles and Practice of Medicine, 22nd edition.

2- Macleod's Clinical Examination, 13th edition.

3- Macleod's Clinical Diagnosis.

4- The Only EKG Book You'll Ever Need, 7th edition.

5- Hanan Mansour's History Taking and OSCE Examination Dossier.

6- Aiman Juma'a's OSCE Dossier.

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.

ꕥ 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

ꕥ History

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

❖ **<u>Clarify the chief complaint :</u>**

» **<u>Productive or non productive</u> (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, aspergellosis**
- ✓ ***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

» **<u>Special sounds with cough</u> (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**),

» **<u>Timing</u>:** nocturnal cough **(asthma**), less in holidays **(occupational asthma**), during a drug course **(dry cough due to ACEI**), cough during swallowing **(neuromuscular abnormality**).

» **<u>Any preceding event</u>:** runny nose and fever/chills **(infection),** sore throat 2 weeks ago then resolved **(reactivation), exercise (exercise induced asthma)**

» **<u>Does it change or increase with specific conditions</u>**: 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 pervious attack ?

» **<u>Noisy breathing</u>**:
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), α1- antitrypsin deficiency (AR), other lung diseases in the family.

7.13 Examples of drug-induced respiratory conditions

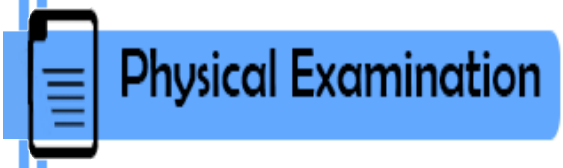
Respiratory condition	Drug
Bronchoconstriction	Beta-blockers Opioids NSAIDs
Cough	Angiotensin-converting enzyme inhibitors
Bronchiolitis obliterans	Penicillamine
Diffuse parenchymal lung disease	Cytotoxic agents: bleomycin, methotrexate Anti-inflammatory agents: sulfasalazine, penicillamine, gold salts, 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 compliant (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, W*egener'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.



Introduce yourself, take permission, insure privacy, warmth and light, make sure of good exposure and position >>>>The patient must be semi sitting and exposure from the neck to the umbilicus.

➢ **General look :**
- The patient is conscious, alert and oriented to time, place and person.
- The patient is lying/sitting/semi sitting.
- Oxygen mask or ventilator.
- Comfortable or distressed.
- Using accessory muscles for respiration (sternocleidomastoid, platysma and trapezius Muscles) .
- Audible sounds (wheezes, stridor…) Ask the patient to cough and then breathe deeply in and out with the mouth wide open.
- Cyanosis of the lips and underside of the tongue.
- Obese, thin, catechetic ?

➢ **Vital signs** (HR, BP, respiratory rate, temperature, O2 sat and BMI)
- Diastolic pressure of <60 mmHg is associated with increased mortality in community-acquired pneumonia
- In pneumothorax, hypotension may indicate the development of 'tension' with reduction in venous return to the heart and risk of cardiac arrest.

- Pulsus paradoxus: a fall in diastolic blood pressure of more than 10mmHg can occur in cardiac tamponade and asthma .

➢ **Hands**

- Hot/cold.
- Sweaty/dry.
- Palmar erythema (indicates CO2 retention as in COPD, cirrhosis, hyperthyroidism and polycythemia).
- Cyanosis.
- Tar stains on fingers and nails, Nicotine stains on dominant index and middle finger.
- Fine tremor (seen in patients taking β-agonist or theophylline bronchodilator inhalers).
- 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. Unilateral asterixis is due to a structural abnormality in the contralateral cerebral hemisphere. Patients with a flapping tremor cannot maintain their grip.
- 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).
- Yellow-nail syndrome is associated with lymphedema and an exudative pleural effusion.
-

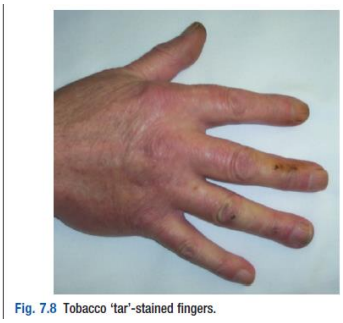


Fig. 7.8 Tobacco 'tar'-stained fingers.

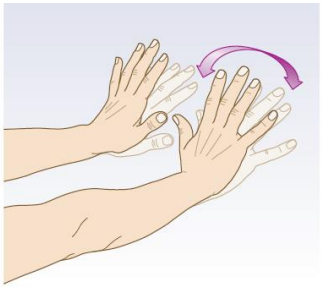
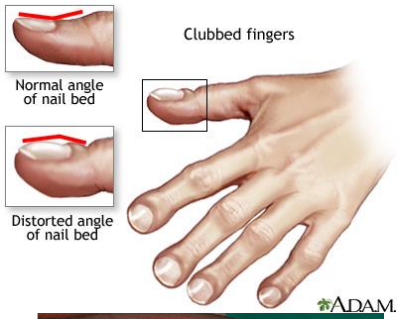


Fig. 7.10 Asterixis. Hand and arm position for observing the 'flapping tremor' of CO_2 retention.



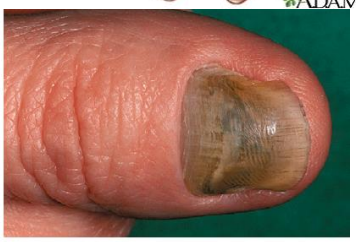


Fig. 7.9 Yellow nail syndrome.

➢ **Face**

- 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.
- Ptosis ,Horner's syndrome in Pancoast lung cancer.

- Tongue, mouth ulcers and dental hygiene.

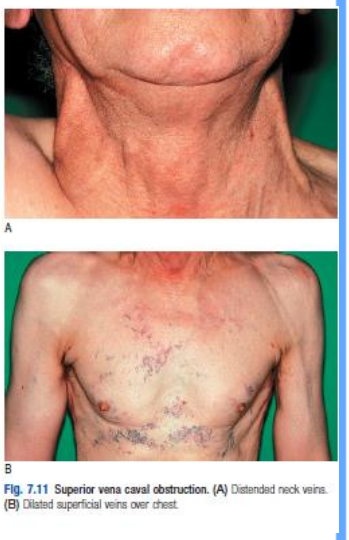


Fig. 7.11 Superior vena caval obstruction. (A) Distended neck veins. (B) Dilated superficial veins over chest.

➢ **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.

➢ **Chest**

➢ **Inspection**
- When you inspect the posterior thorax, make sure to ask the patient to sit up and cross his hands.
- Symmetrical, bilateral, abdominothoracic/thoracoabdominal breathing.
- Check for paradoxical breathing (abdomen moves inward; seen in bilateral phrenic nerve damage or severe COPD).
- Scars for surgeries (make sure you ask the patient to lift his arms to check the sides).
- Swellings, dilated superficial veins and subcutaneous nodules.
- Antero-posterior: transverse diameter ratio (normally 5:8).
- Check for deformities:
  - ✓ Barrel-shaped hyperinflated chest with intercostal indrawing in COPD patients.
  - ✓ Kyphoscoliosis (can be due to childhood poliomyelitis or spinal TB) causes CO2 retention and cor pulmonale.
  - ✓ Pectus carinatum (pigeon chest) with prominent Harrison's sulci can be caused by uncontrolled childhood asthma, osteomalacia or rickets.
  - ✓ Pectus excavatum (funnel chest) is usually asymptomatic but in severe cases, it may cause left heart displacement and reduced ventilator capacity.

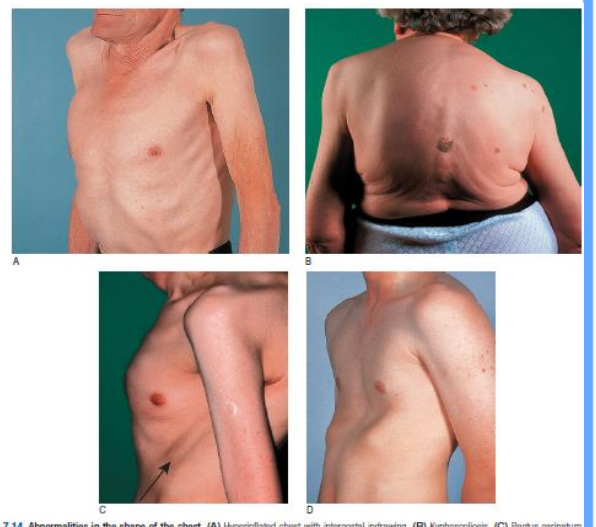


Fig. 7.14 Abnormalities in the shape of the chest. (A) Hyperinflated chest with intercostal indrawing. (B) Kyphoscoliosis. (C) Pectus carinatum prominent Harrison's sulcus (arrow). (D) Pectus excavatum.

➢ **Palpation**
- Make sure you warm your hands and palpate with your hands horizontally in a continuous pattern .
- Ask the patient if he is feeling any pain; start from the area furthest from the pain and gently palpate it at the end.
- Superficial palpation for tenderness (maintain eye contact to observe any discomfort or pain), subcutaneous emphysema and superficial masses (lipomas).

- Mediastinum:
  - ✓ Tracheal location >>fix your index and ring fingers on the heads of the patient's clavicle and localize the trachea with your middle finger (normally central or slightly deviated to the right).
  - ✓ Palpate the apex beat to confirm that the heart is not deviated (especially if the trachea is deviated to the left) , normally in the midclavicular fifth intercostal space.
  - ✓ Corticosternal distance (from cricoid cartilage just below the thyroid cartilage to the suprasternal notch) , normally 3-4 fingers (5cm).
  - ✓ Tracheal tug (gently place your index finger on the trachea and ask the patient to take a breath; normally the index should not go down).
- Tactile vocal fremitus (palpate using your metacarpophalangeal bony prominence while the patient says ninety nine or أربعة وأربعين), normally, symmetrical bilateral tactile vocal fremitus; increased vibration indicates consolidation.
- Chest expansion (place your hands with your fingers extended around the sides of the patient's chest and ask him to take a deep breath) normally the chest expands up to 2.5 cm on each side (symmetrical bilateral chest expansion).

7.20 Common causes of tracheal deviation

Towards the side of the lung lesion	
• Upper lobe or lung collapse • Upper lobe fibrosis	• Pneumonectomy
Away from the side of the lung lesion	
• Tension pneumothorax	• Massive pleural effusion
Upper mediastinal mass	
• Retrosternal goitre • Lymphoma	• Lung cancer

➢ Percussion

- Normally, it is bilateral symmetrical resonant.
- Locate the upper border of the liver at the right 5th intercostal space.

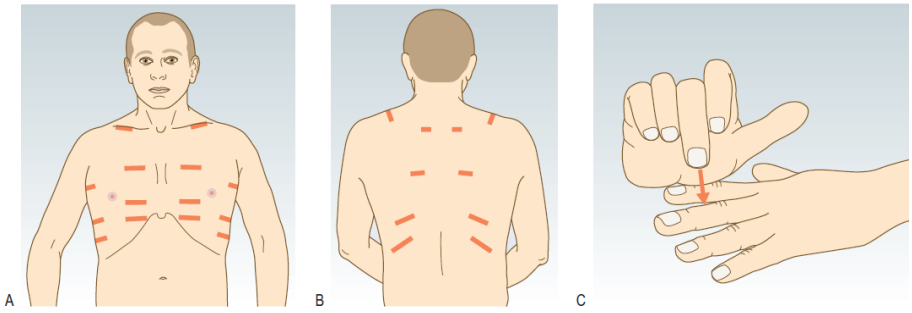


Fig. 7.18 Sites for percussion. (A) Anterior and lateral chest wall. (B) Posterior chest wall. (C) Technique of percussion.

- From the posterior chest, percuss for diaphragmatic excursion (locate the diaphragm with full expiration and then full inspiration) normally the distance should be between 5 and 8 cm (less than 5 cm indicates hyperinflation or bilateral phrenic nerve palsy).

7.21 Percussion note

Type	Detected over
Resonant	Normal lung
Hyperresonant	Pneumothorax
Dull	Pulmonary consolidation
	Pulmonary collapse
	Severe pulmonary fibrosis
Stony dull	Pleural effusion
	Haemothorax

➢ **Auscultation**

- Good or diminished air entry.
- Symmetrical bilateral vesicular breathing (abnormally can be bronchial in cases of lung consolidation, localized pulmonary fibrosis, non-occluding collapsed lung and at the top of a pleural effusion).
- Do not auscultate the clavicle.
- Added sounds (wheeze, crepitation, pleural rub, crackling and pneumothorax click) ask the patient to clear his throat by cough as crackling decreases after that in bronchiectasis
- Vocal resonance (ask the patient to say ninety nine or أربعة وأربعين) normally, it is symmetrical bilateral vocal resonance; the numbers are only clearly audible in cases of consolidation.
- Whispering pectoriloquy (higher sounds indicate consolidation).
- Egophony (ask the patient to say the letter 'e', normally it is heard as an 'e'; in pneumonia, the letter 'a' is heard).

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)

➢ Other

- Liver.
- Ascites.
- Pitting edema (can result from cor pulmonale).
- DVT.
- Erythema nodosum (present in sarcoidosis, SLE and TB).
- Non-tender subcutaneous nodules may occur in patients with disseminated cancer.

7.22 Causes of diminished vesicular breathing

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

7.27 Causes of bronchial breath sounds

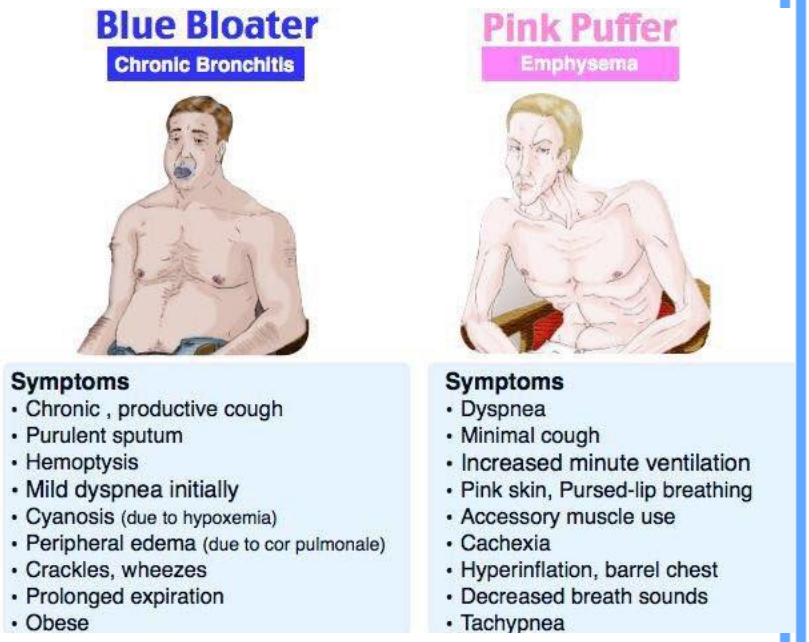
Common	
• Lung consolidation (pneumonia)	
Uncommon	
• Localised pulmonary fibrosis • At the top of a pleural effusion	• Collapsed lung (where the underlying major bronchus is patent)

knock the door, take patient permission, insure privacy and illumination		Chest examination:	
Exposure: above the umbilicus Position: 45 degree		➢ **Inspection** From the foot of the bed • Comment on mode of breathing, symmetry of expansion, chest deformity. From the side of the bed • Scars, swellings, dilated superficial veins	
Wash your hand			
General examination: • Conscious, oriented, alert • In pain or respiratory distress, use of accessory muscles for respiration • Cyanotic, lip pursing, audible sound • Any attached device: O2 mask, nasal cannula...		➢ **Palpation** • Keep eye to eye contact • Tenderness, palpable masses or subcutaneous emphysema • Measures chest expansion (6–8 cm is normal) at three places on the anterior and three on the posterior chest • Palpates apex beat	
Mention you want to check vitals			
Hand: • Clubbing (suppurative conditions, lung cancer, fibrosis) • Tar staining • Wasting of small muscles • Tremor + CO2 retention flap		➢ **Percussion** • Percusses at three positions on the anterior and three on the posterior chest • percuss for diaphragmatic excursion	
Eyes: • Sclera and conjunctiva for pallor and jaundice • Horner syndrome (Pancoast syndrome)		➢ **Auscultation** • Good or diminished air entry. • Symmetrical bilateral vesicular breathing • Added sounds • Vocal resonance • Whispering pectoriloquy • Egophony	
Mouth • central cyanosis under the tongue			
Neck: • JVP (raised in cor pulmonale) • Tracheal deviation and tracheal tug • Cricoid–sternal distance • Palpates lymph nodes		Palpates shins or ankles for peripheral edema	
		Thank the patient	

COPD

A 50-year-old gentle man presented with SOB m productive cough with audible breathing sounds he is smoker (45 pack year). Give differential diagnosis. Examine him looking for signs of COPD.

- **General look :**
  - Conscious alert oriented (if the patient came with co2 retention he may be drossy)
  - Posture of the patient (If the patient sits forward with the hands/arms on the thighs or knees to 'fix' the shoulder girdle, he raises the clavicles and upper chest, increasing lung volume and negative intrathoracic pressure then think of emphysema)
  - Thin or obese (thin >>emphysema , obese >> chronic bronchitis)
  - Cyanosis (central under the tongue and peripheral lips , conjunctiva) >> (blue colored patients >> chronic bronchitis , pink colored patient >>> emphysema)



  - Level of consciousness as mentioned before .
  - Tachypnea and Use of accessory muscles mainly the sternocleidomastoid .
  - Pursed lips .
  - Use of extra equipments : o2 support, inhalers .
  - Note any unusual noisy breathing sounds
- **Vital signs :**
  - RR (> 25 tachypnea , > 35 respiratory distress)
  - HR (could be tachycardia)
  - temperature .
  - o2 sat (patient hypoxic)
  - blood pressure .

- **hands :**
  - tarry stain , NO clubbing if it's found then think of lung malignancy , familial or another cause (COPD by itself DONOT cause Clubbing !)
  - palmar erythema in co2 retention in COPD
  - flapping tremor (Co2 toxicity) .
- **neck** :
  - use of accessory muscles ,distended vessels , JVP (elevated then think for Core Pulmonale) , look for tracheal tug (because of hyperinflation) ,
- **chest** :
  - ✓ **inspection** : breathing pattern , barrel chest (anteroposterior : transverse >5:7) with intercostal indrawing in COPD patients.
  - **palpation**: the chest expansion may be reduced .
  - **percussion :** hyperresonance chest .decrease the dullness of the liver and heart .
  - **Auscultation :** Diminished air entry , vesicular breathing , prolonged expiratory time , added sounds (end _expiratory wheeze ,coarse crackles with inspiration due to sputum accumulation if found) .

- Then mention that you are going to look for sign **of cor pulmonale :** Symptoms of fluid overload : ankle swelling , ascites , increase the JVP . hepatomegaly, Parasternal heave , loud P2 +/- tricuspid incompetency murmurs .

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 lover 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:

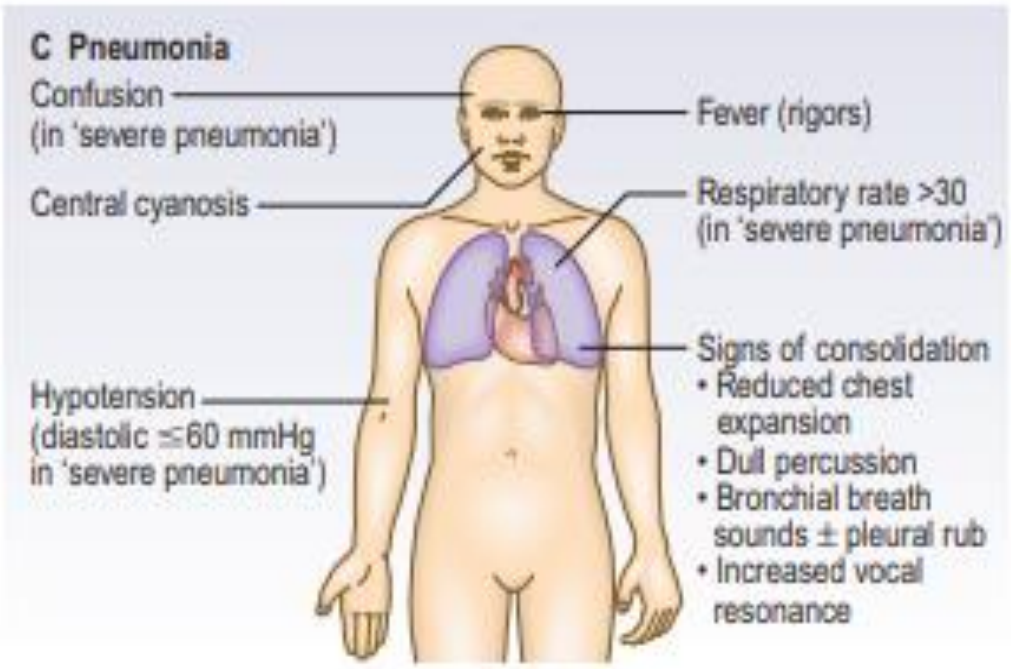
Risk factor: (for both types)

* 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.

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

❖ 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.

- 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 >= 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)

❖ <u>Other causes of PE without DVT:</u>

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.

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**

ꕥ **history**

✓ **Symptoms while sleeping (seek for witness)**
Snoring [partial airway obstruction], **apnea** [complete airway obstruction], **arousal** or **chocking** [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

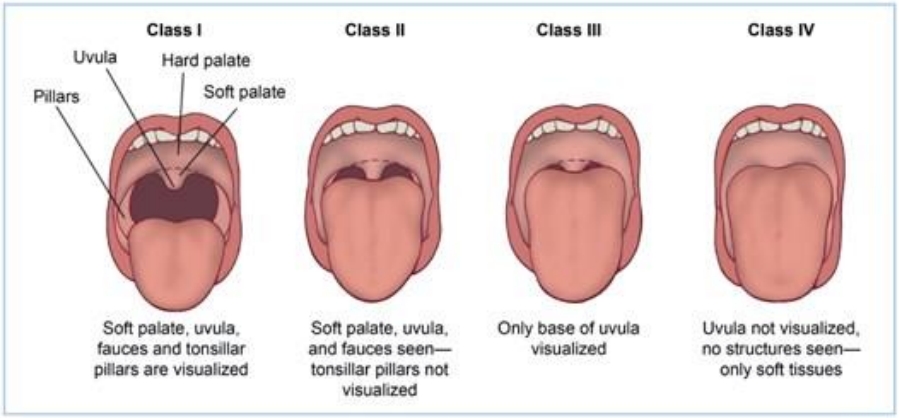
» Can be associated with nasal polyps, acromegaly and hypothyroidism.
» Increased risk for DM, systemic and pulmonary HTN, IHD, cardiac arrhythmia and strokes.

Drugs, alcohol

ꕥ **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:

→ Sings of hyperinflation:

* On inspection: barrel chest, intercostal retraction and paradoxical breathing (if sever 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).

→ Sings 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.

ꕥ Diagnosis

Polysomnography (overnight sleep study) confirms the diagnosis.

Pneumothorax (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.

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).

Labs: CXR is diagnostic (a line demarcating a hyperlucent area).

Pulmonary HTN (complication)

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, abd. mass, leg swelling
- ✓ **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, Fenfluramine, tryptophan.
- ✓ Social Hx: smoking, occupation (interstitial lung disease), alcohol, travel.

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.

Labs: CXR (increased vascular markings), ECG, PFTs.

Pleural effusion (complication)

Chief complaint: pleuritic chest pain

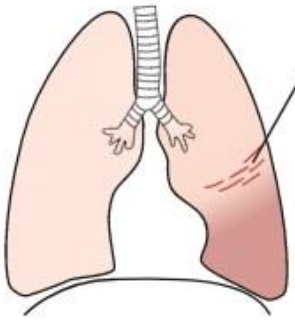
Diagnostic approach to pleural effusion:

- ✓ 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.
- ✓ Past Medical: SLE, Polyarteritis (Autoimmune)

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 (blunting of the costophrenic angles), CT scan.
Pleural fluid aspiration (Exudate VS Transudate)
Pleural biopsy: Abrahem's needle, pleuroscopy.

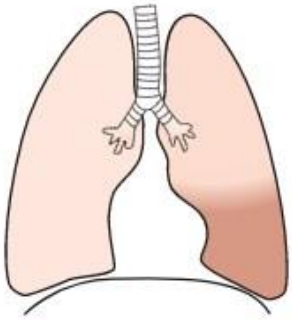
Some physical signs



(There may be brochial breathing at the top of an effusion)

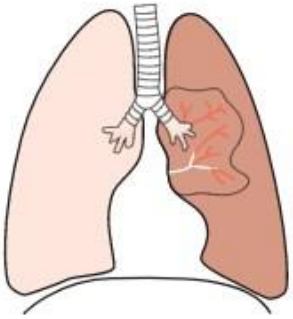
Expansion: ↓
Percussion: ↓ (Stony dull)
Air entry: ↓
Vocal resonance: ↓
Trachea + mediastinum central (shift away from affected side only with massive effusions ≥1000ml

PLEURAL EFFUSION



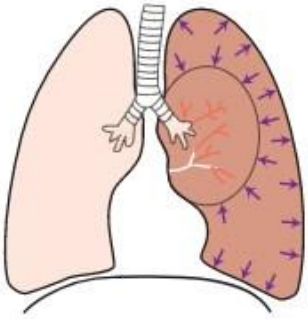
Expansion ↓
Percussion note ↓
Vocal resonance ↑
Bronchial breathing ± coarse crackles (with whispering pectoriloquy)
Trachea + mediastinum central

CONSOLIDATION



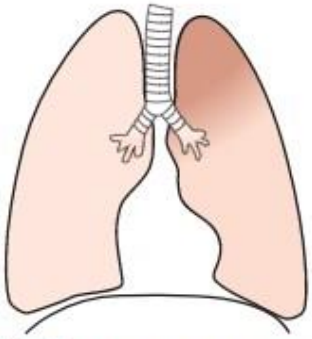
Expansion ↓
Percussion note ↑
Breath sounds ↓
Trachea + mediastinum shift Away from the affected side

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.

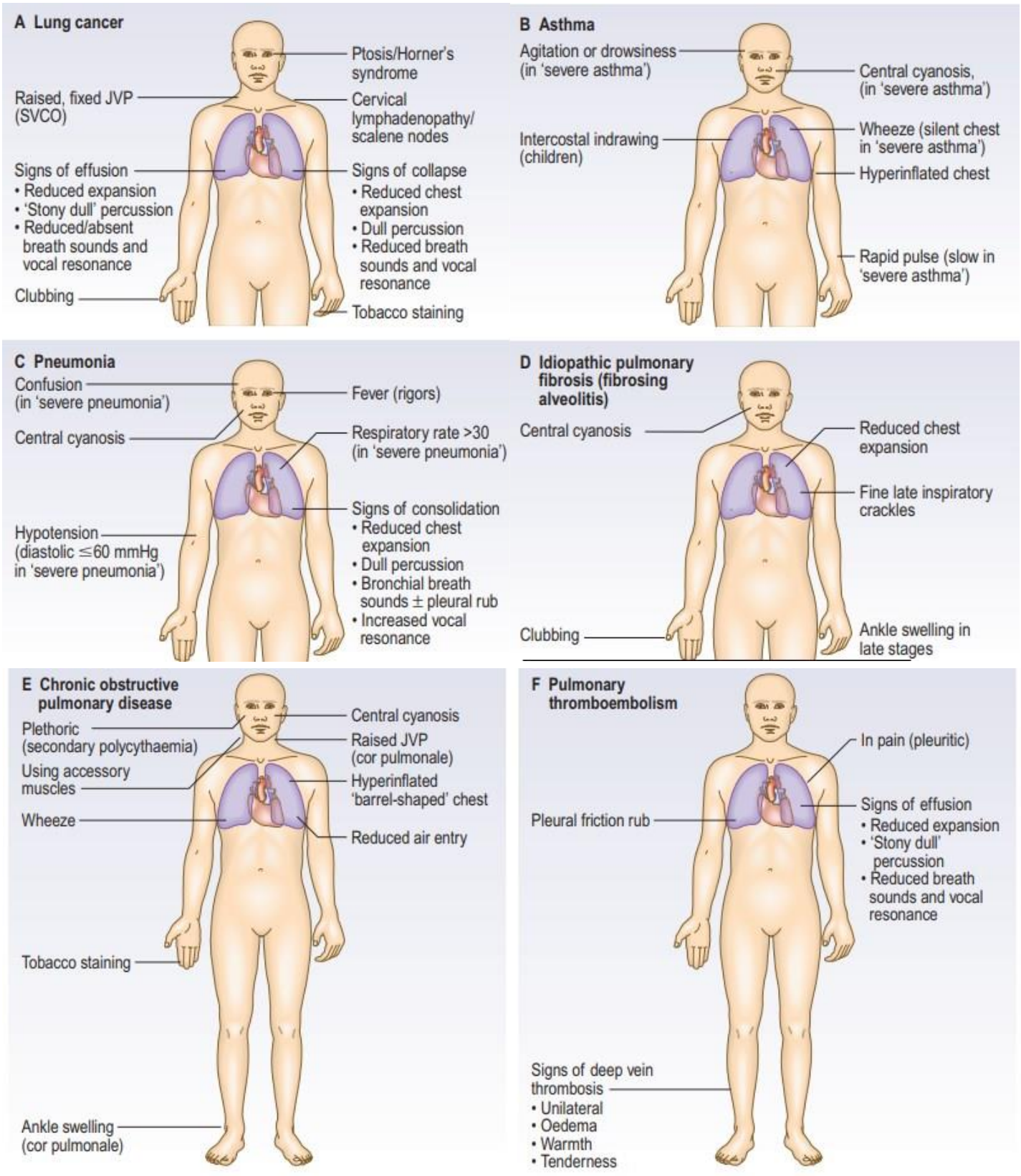


Fig. 7.20 Clinical signs of common respiratory conditions. JVP, jugular venous pressure; SVCO, superior vena caval obstruction.

How to read chest x-ray

- Always make sure of the patient's profile, date and time of x-ray.
- Check the *technique* by assessing the following
  - **Positioning**: AP vs PA (scapula is out of the field in PA)
  - **Adequacy** : both shoulders , diagram and upper abdomen should be included in the CXR.
  - **Inspiration**: pt should take deep breath, if there is adequate inspiratory effort at least **5-6 Ant. Ribs** above the diaphragm AND **8-10 Post. Ribs** should be viewed.
  - **Penetration**: intervertebral spaces above and below the heart should be radiolucent, and the outline of vertebra behind the heart should be identified.
  - **Rotation**:
    check if the CXR is centralized→ equal distance between medial end of clavicles and central line (midline).
  - **Orientation:**
    verify right and left sides (gastric bubble should be on the left side).

- Start *Interpretation* of the CXR, use any systemic approach you like, here we will discuss ABCDEF approach:
  - **Airway** (trachea): Midline vs. deviated or rotated, foreign body in trachea, endotracheal tube position.
  - **Bones** (clavicles, ribs, humeri, etc): move with the external border of the bone, any steepness indicate fracture.
  - **Cardio**mediastinal silhouette: check cardiac size (more than 50% of the transverse diameter of the thorax is cardiomegaly)
  - **Diaphragms** and the costophrenic angles: put in your mind that RT diaphragm is slightly higher than left diaphragm.
  - **Expanded lungs** (lung fields, soft tissues)
  - **Foreign bodies**(tubes, lines, wires, devices, etc)

- Lung is the most important part to comment on, compare Rt & Lt, look for:
  - Pulmonary infiltrates or consolidation: **pneumonia**
  - Cavitations: **TB, lung CA especially squamous cell carcinoma**
  - Dilated airways: **bronchiectasis**
  - Hyperinflation and emphysema: **COPD**

» Line demarcating a hyperlucent space: **pnemothorax**
» Prominent pulmonary vessels >>> pulmonary HTN
» Blunted costophrenic angel: plural effusion
» If you found cardiomegaly with prominent pulm. vessels: **Pulm. HTN & CHF**

Chapter 3: Gastroenterology and Hepatology

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.

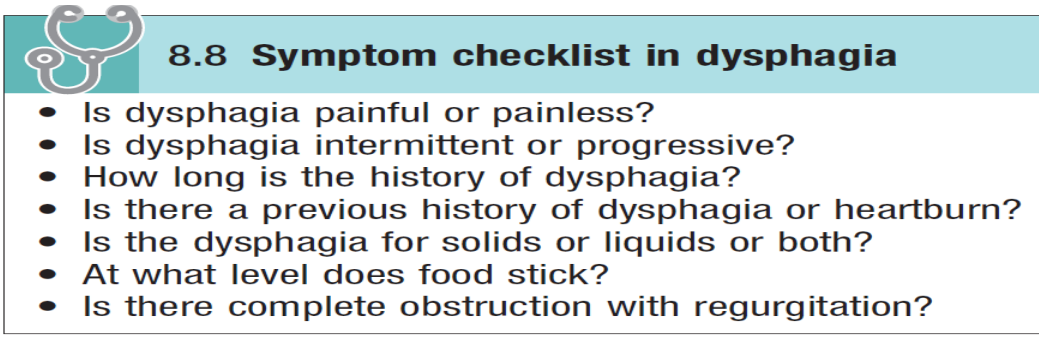
Dysphagia

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**.



- 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.

- **<u>Ask about red flag symptoms:</u>**
  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
Neuromuscular Dysmotility	**Liquids more than Solids and Liquids**
• Achalasia • **Systemic Sclerosis**	• Myasthenia gravis
Mechanical Structural	
• Oesophageal cancer • Peptic oesophagitis • Other benign strictures, e.g. after prolonged nasogastric intubation	• Extrinsic compression, e.g. lung cancer

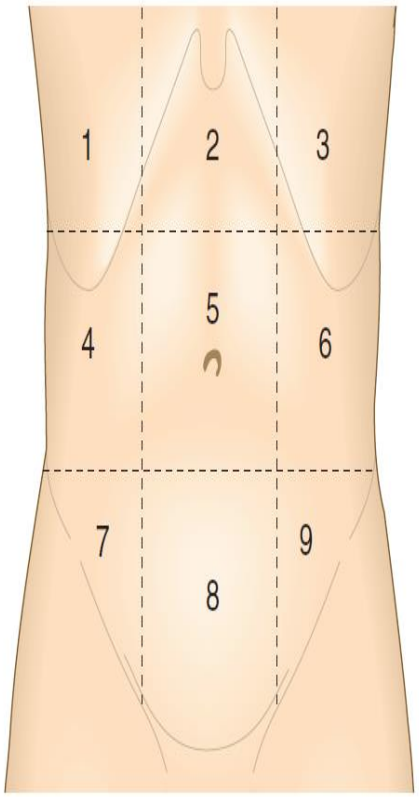
Checklists

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, *Yersina* (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).

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

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

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
- Mallory–Weiss oesophageal tear
- Oesophagitis, gastritis, duodenitis
- Oesophagogastric varices
- Oesophageal or gastric cancer
- 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

➔ Jaundice can be:

a. Pre-hepatic: caused by hemolysis (so we should ask about sickle cell anemia and thalassemia in the history).

b. Hepatic: caused by any hepatic disease (so we should ask about symptoms related to cirrhosis, hepatitis, and autoimmune liver diseases like autoimmune hepatitis, PBC and PSC).

c. Post-hepatic: caused by obstruction to bile flow (so we should ask about symptoms of gallstones and bile duct stones).

➔ See the checklist below (next page), and go through it while looking at the following tables.

Questions box 13.7

Questions to ask the patient presenting with jaundice

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

1. Is your urine dark? Are your stools pale? (Obstructive jaundice)
2. Do you have any skin itching (pruritus)?
3. ! Have you had any fever? (Cholangitis)
4. ! Have you had a change in your appetite or weight? (Malignancy)
5. Have you had any abdominal pain or change in bowel habit?
6. ! Have you had any vomiting of blood or passage of dark stools?
7. Do you drink alcohol? How much? How long? (CAGE questions, page 12)
8. Have you ever used intravenous drugs?
9. Do you have any tattoos?
10. Have you ever had a blood transfusion?
11. Have you started any new medications recently?
12. Have you had any recent contact with patients with jaundice or liver problems?
13. Have you any history of recent high-risk sexual behaviours?
14. Have you travelled overseas to areas where hepatitis A is endemic?
15. Have you been immunised against hepatitis B?
16. Have you any history of inflammatory bowel disease? (Primary sclerosing cholangitis)
17. Have you had any surgeries (e.g. pancreatic or biliary)?
18. What is your occupation (contact with hepatotoxins)?
19. Is there any family history of liver disease?

TABLE 14.2 Changes in urine and faeces with jaundice

		Causes of jaundice	
Substance and site	**Haemolysis**	**Obstruction or cholestasis**	**Hepatocellular liver disease**
URINE			
Bilirubin (conjugated)	Normal*	Raised	Normal or raised
Urobilinogen	Raised	Absent or decreased	Normal or raised
FAECES			
Stercobilinogen	Raised	Absent or decreased	Normal
Causes	Haemologytic anaemia	Extrahepatic biliary obstruction (e.g. gallstones, carcinoma of pancreas or bile duct, strictures of the bile duct), intrahepatic cholestasis (e.g. drugs, recurrent jaundice of pregnancy)	Hepatitis, cirrhosis, drugs, venous obstruction

*Unconjugated bilirubin levels are elevated in the serum.

8.22 Common causes of jaundice

Increased bilirubin production

- Haemolysis (unconjugated hyperbilirubinaemia)

Impaired bilirubin excretion

- Congenital
  - Gilbert's syndrome (unconjugated)
- Hepatocellular
  - Viral hepatitis
  - Cirrhosis
  - Drugs
  - Autoimmune hepatitis
- Intrahepatic cholestasis
  - Drugs
  - Primary biliary cirrhosis
- Extrahepatic cholestasis
  - Gallstones
  - Cancer: pancreas, cholangiocarcinoma

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

Wilson's disease (rare)	Hemochromatosis (rare)	Celiac disease
➢ Chief complaint/ duaration ➢ 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 intenstion) 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/ duaration ➢ Deposition of iron in skin , liver , heart ➢ **Symptoms:** Vertigo, memory changes, behavioural chenges, 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/ duaration ➢ 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

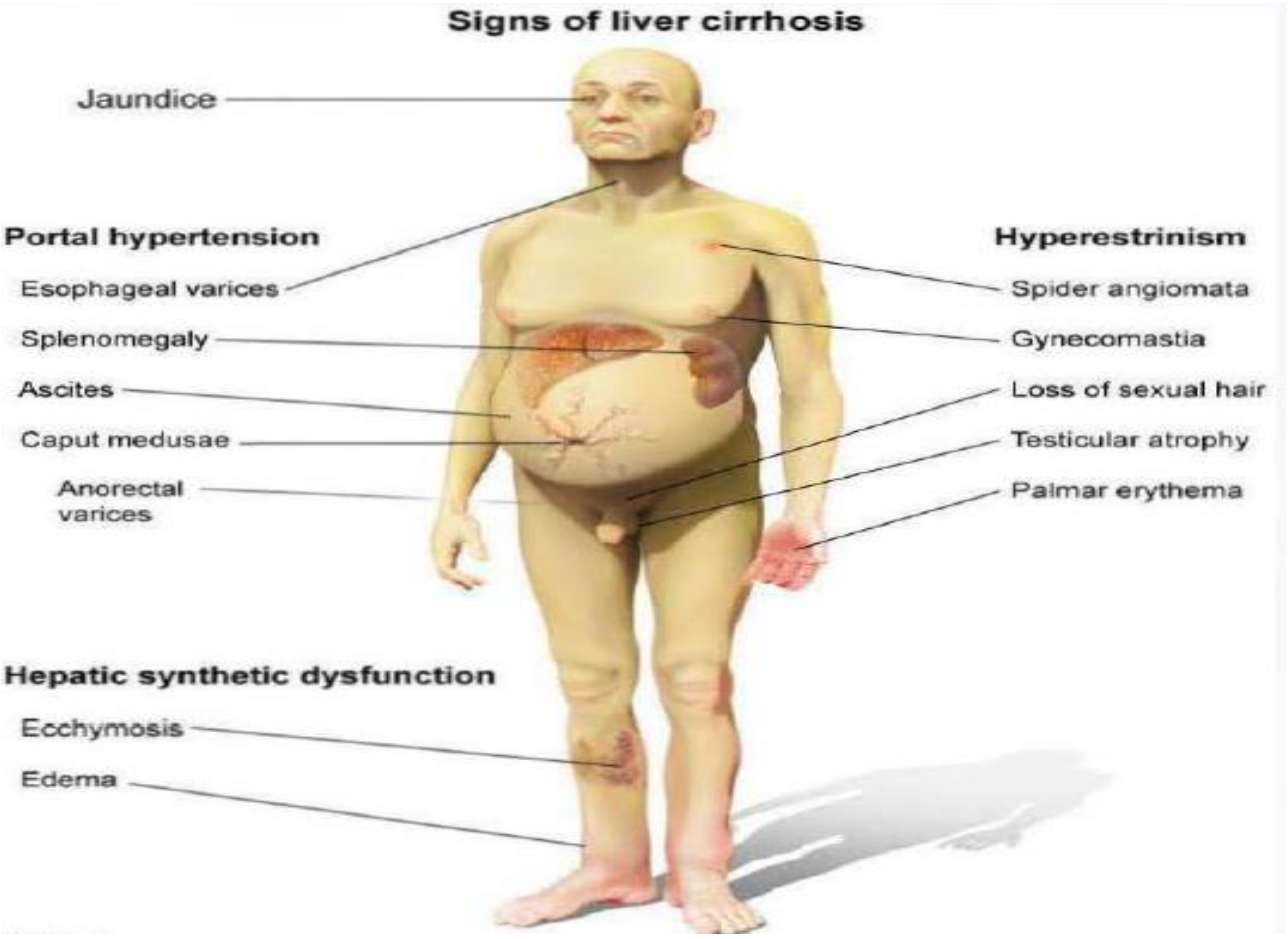
Crohn's disease (regional enteritis)	UC	IBS
➢ **Chief complaint** 'abdominal pain with duarrhea'/ duration ➢ **Symptoms**: Diarrhea (usually bloody), abdominal pain (RLQ), nausea, vomiting, malabsorption, weight loss, fever, malaise. ➢ **Extraintestinal features:** uveitis, episcleritis, ankylosing spondylitis, sacroiilitis, monoarticular migratory arthritis, DVT, PE, erythema nodosum, pyoderma gangrenosum, aphthous oral ulcers, cholelithiasis, nephrolithiasis(stones) ➢ **Complications**: fistula, abscess, anorectal disease, malignancy, malabsorbtion 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 diturbances, dehydration, cancer, sclerosing cholangitis, growth retardation ➢ **mucosa and submucosa, no skip lesions, sites sre 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

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.

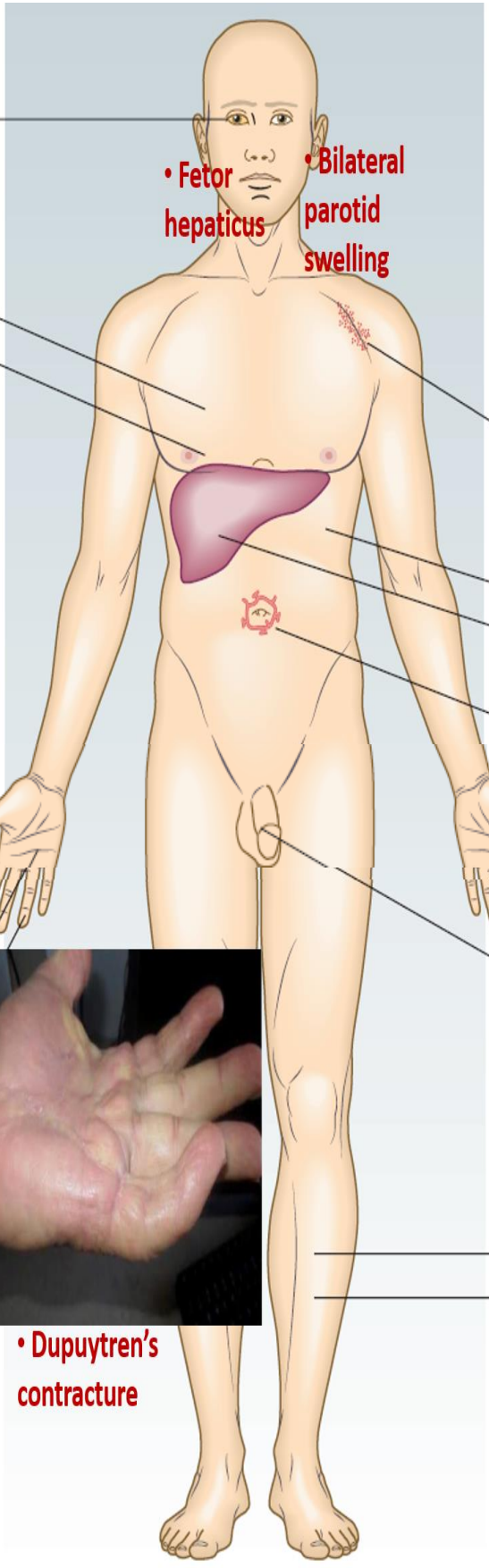
- All types of chronic liver disease will ultimately lead to liver cirrhosis.
- Liver cirrhosis causes four types of signs:

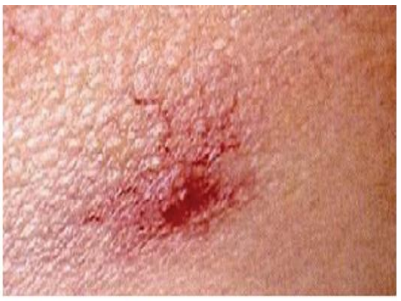
1. Signs related to impaired liver function (decreased synthesis of albumin> edema, clotting factors> easy bruisability... etc.).
2. Signs related to hyperestrinism (impaired breakdown of estrogen causes gynecomastia, spider angiomata, loss of sexual hair, testicular atrophy, and palmar erythema).
3. Signs related to portal hypertension.
4. Signs related to hepatic encephalopathy and liver failure (asterixis and fetor hepaticus)



Eyes
- Jaundice

Chest
- Gynaecomastia (in men)
- Breast atrophy (in women)





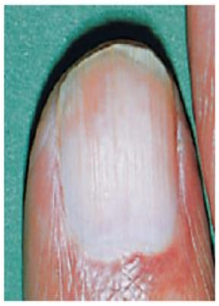
A Spider naevi

Upper half of body (above umbilicus)
- Spider naevi (A)

Abdomen
- Splenomegaly
- Hepatomegaly (but liver may be small)
- Dilated collateral vessels around umbilicus

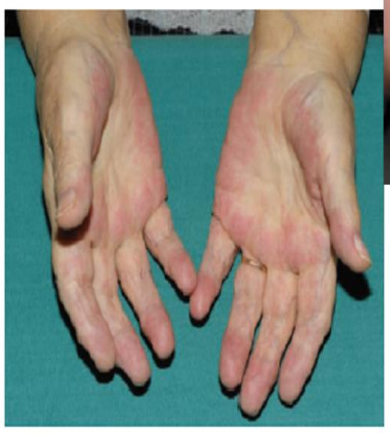
• Caput medusae

Ascites



C Leukonychia

Hands
- Leukonychia (white nails) (C)
- Palmar erythema (B)
- Clubbing
- Asterixis



B Palmar erythema

• Dupuytren's contracture

Genitalia
- Testicular atrophy

Legs
- Oedema
- Hair loss

General
- Skin pigmentation
- Loss of body hair
- Bruising

Precipitants of hepatic encephalopathy

[Renal failure, GI bleed, Infection, Hypokalemia and alkalosis due to diuretics, High protein intake, Constipation, CNS drugs, Acetaminophen and methotrexate, Alcohol, Cirrhotic patient with TIPS)

1- Renal failure: Renal failure leads to decreased clearance of urea, ammonia, and other nitrogenous compounds.

Ask about CKD, elevated creatinine in the labs, causes of AKI.

2- Gastrointestinal bleeding:
A) The presence of blood in the upper gastrointestinal tract results in increased ammonia and nitrogen absorption from the gut.
B) Bleeding may predispose to kidney hypoperfusion and impaired renal function.
C) Blood transfusions may result in mild hemolysis, with resulting elevated blood ammonia levels.

Ask about melena, hematochezia, hematemesis (esophageal varices).

3) Infection: Infection may predispose to impaired renal function and to increased tissue catabolism, both of which increase blood ammonia levels.

Ask about: Fever, night sweats, productive cough, dysuria, nausea, vomiting and diarrhea.

4) Constipation: Constipation increases intestinal production and absorption of ammonia.

5) Medications: Drugs that act upon the central nervous system, such as opiates, benzodiazepines, antidepressants, and antipsychotic agents, may worsen hepatic encephalopathy.

6) Diuretic therapy: Decreased serum potassium levels and alkalosis may facilitate the conversion of NH_4^+ to NH_3. At the author's institution, diuretic-induced hypovolemia is the most common reason for patients with previously well-controlled hepatic encephalopathy to present to the emergency room with worsening mental function.

Ask about diuretics: Furosemide, and thiazides.

7) Dietary protein overload: This is an infrequent cause of hepatic encephalopathy.
In liver cirrhosis, protein intake should not exceed 40-50 g/day.

From medscape + notes

How to differentiate between the spleen and the kidney?

8.36 Differentiating a palpable spleen from the left kidney

Distinguishing feature	Spleen	Kidney
Mass is smooth and regular in shape	More likely	Polycystic kidneys are bilateral irregular masses
Mass descends in inspiration	Yes, travels superficially and diagonally	Yes, moves deeply and vertically
Able to feel deep to the mass	Yes	No
Palpable notch on the medial surface	Yes	No
Bilateral masses palpable	No	Sometimes, e.g. polycystic kidneys
Percussion resonant over the mass	No	Sometimes
Mass extends beyond the midline	Sometimes	No (except with horseshoe kidney)

Ascites Examination

- **Inspection:**
  Comment on abdominal distention.
  See whether the flanks are full or not.
- **Shifting dullness:**
  - With the patient supine, percuss from the midline out to the flanks. Note any change from resonant to dull, along with areas of dullness and resonance.
  - Keep your finger on the site of dullness in the flank and ask the patient to turn on to his opposite side.
  - Wait for 10 seconds to allow any ascites to gravitate, then percuss again. If the area of dullness is now resonant, shifting dullness is present, indicating ascites.

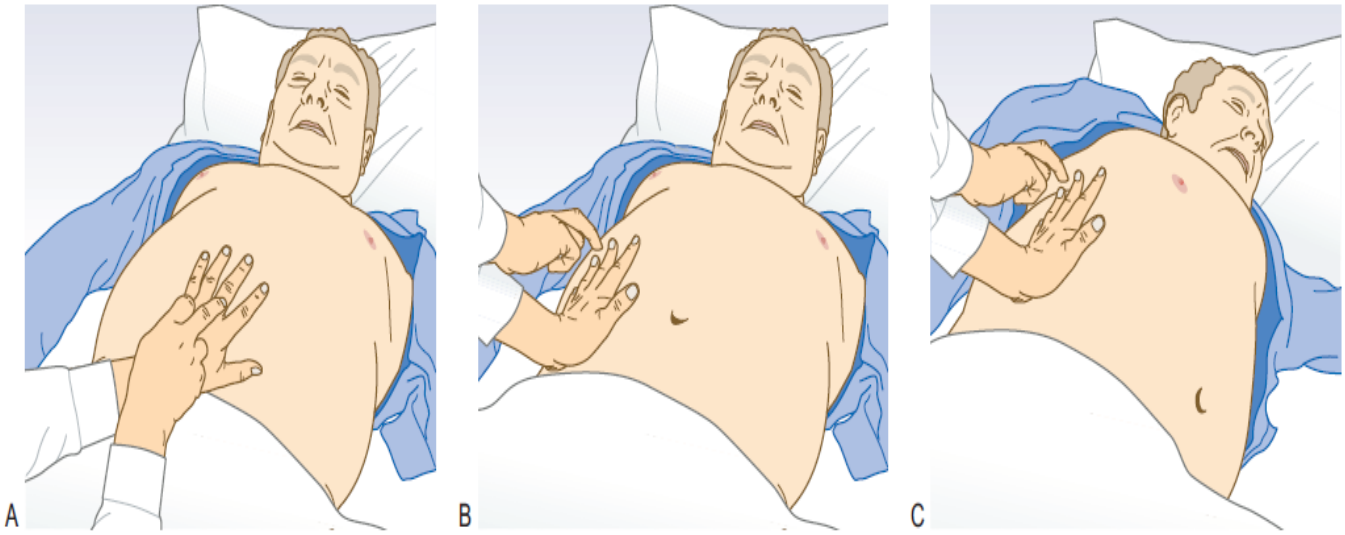


Fig. 8.19 Percussing for ascites. (A and B) Percuss towards the flank from resonant to dull. (C) Then ask the patient to roll on to his other side. In ascites, the note then becomes resonant.

- **Fluid thrill:**
  - If the abdomen is tensely distended and you are not certain whether ascites is present, feel for a fluid thrill.
  - Place the palm of your left hand flat against the left side of the patient's abdomen and flick a finger of your right hand against the right side of the abdomen.

- If you feel a ripple against your left hand, ask an assistant or the patient to place the edge of his hand on the midline of the abdomen. This prevents transmission of the impulse via the skin rather than through the ascites. If you still feel a ripple against your left hand, a fluid thrill is present (only detected in gross ascites).

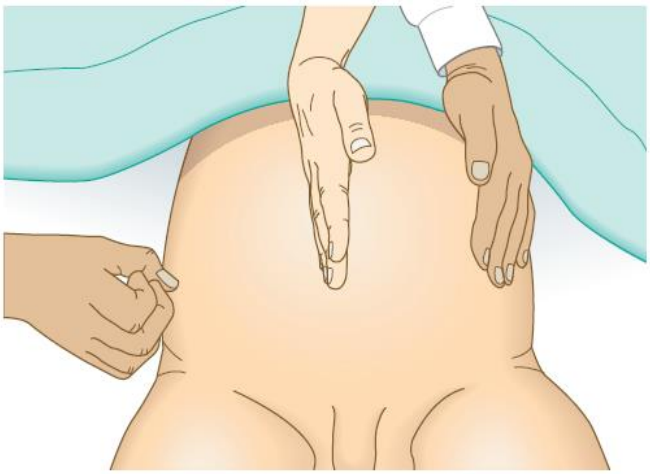


Fig. 8.20 Eliciting a fluid thrill.

-

Abdominal Examination

Position of the patient

Either you keep the back rest of the bed 15-20% or you put one or two pillows under the head and both arms are by side and not behind. you may flex the hip 45% and the knee 90 to get more relaxation of the abdominal wall if pt. still not relaxed

Exposure

Ideally from nipples to the mid thigh but in respect to the pt. sensitivity **expose the abdomen from the xiphisternum to the symphysis pubis, leaving the chest and legs covered.**

(Examination of the gastrointestinal system)

General Examination

Conscious , oriented (impaired in hepatic encephalopathy), cachectic or obese. Not in pain , looks well (in acute abd. Pt. lies very still)(in renal colic pt. rolls around with each bout) , vital signs and BMI.

Hands

Clubbing, koilonychia (IDA),leukonychia (hypoalbuminemia), Dupuytren's contracture (contracture of the palmar fascia),tar stain , palmer erythema (centrally spared),muscle wasting and flapping tremor(Asterixis).

Face

Examine for jaundice and pallor

If jaundice is not obvious, ask the patient to look down and retract the upper eyelid to expose the sclera; look to see if it is yellow in natural light and look for jaundice under the tongue.

angular cheilitis, atrophic glossitis, The tongue has a beefy, raw appearance in folate and vitamin B_{12} deficiency, aphthous ulcers, dental hygiene.

Bilateral parotid swelling

Fetor hepaticus (mousy smell) in liver failure, alcohol smell

Lymph nodes

Cervical , axillary and inguinal L.N

(Lt. supraclavicular LN –virchow's lymph node- gastric and pancreatic cancer) which called (Troisier's sign)

Chest :

Spider naevi

Gyenaecomastia

Breast atrophy in female

Hair distribution according to the gender(ex. Normal hair distribution according to the male pattern)(loss of hair in chronic liver disease)

scratch marks

Abdominal Examination:

1- Inspection

Start from the foot of the bed

- Note any abdominal distension (remember the 5Fs – flatus, faeces, foetus, fat, fluid),flat or scaphoid abd.
- abdominal respiration(in peritonitis no abd. movement)
- umbilicus in the midline and inverted

From the rt. Side of the patient

bruising, scars, strias ,stoma,dilated veins (spider neavi and caput medusa)(and direction of blood is outward in portal HTN or upward in IVC obstruction or downward in SVC obstruction) herniae, and any visible peristalsis.

- A mass may be apparent. To exaggerate the presence of a mass, inspect with the head raised from the bed to tense the abdominal muscles

Two questions :1- ask the pt . to cough (and looking for hernia orifices. And cough may increase pain in case of peritonitis)

2-ask pt. to raise his head looking for divercation of recti

2-Palpation and percussion

•Ensure your hands are warm. Ask patient if they have any pain or tenderness.

• Begin with light palpation of the nine segments. If patient has complained of pain begin at opposite side. Observe patient's face throughout palpation to ensure that you are not causing pain.

• ***Light palpation*** is used to assess:

1- tenderness and guarding (a sign of irritation of the peritoneum).

2- Superficial masses

3- Gain patient confidence

• ***deep palpation*** of the same nine segments. Deep palpation is used to assess for masses and tenderness

• If appropriate, test for rebound tenderness (a sign of intra-abdominal pathology)

Palpation for enlarged organs

- **Liver palpation:**
- Place your hand flat on the skin of the right iliac fossa.
    - Ask the patient to breathe in deeply through the mouth.
    - Feel for the liver edge as it descends on inspiration.
    - Move your hand progressively up the abdomen, 1 cm at a time, between each breath the patient takes, until you reach the costal margin or detect the liver edge.

Liver percussion

- Ask the patient to hold his breath in full expiration.
- Percuss downwards from the right fifth intercostal space in the mid-clavicular line, listening for the dullness that indicates the upper border of the liver.
- Measure the distance in cm below the costal margin in the mid-clavicular line or from the upper border of dullness to the palpable liver edge.

Palpation of the spleen

- Starting from RIF Move your hand diagonally upwards towards the left hypochondrium 1 cm at a time between each breath the patient takes. **roll on to his right side** and repeat the above. Palpate with your right hand, placing your left hand behind the patient's left lower ribs, pulling the ribcage forward.

Percussion for spleen for dullness from abdomen upward (same direction of palpation)and from mid axillary line on ribs and down.

Palpate for kidneys (bimanual examination)

Renal angle tenderness-

Percuss for urinary bladder (dull)

Examine for ascites

shifting dullness

3-Auscultation :

- place your stethoscope diaphragm to the right of the umbilicus and do not move it.
- Listen for up to 2 minutes before concluding that bowel sounds are absent.
- Listen above the umbilicus over the aorta for arterial bruits.
- Now listen 2–3 cm above and lateral to the umbilicus for bruits from renal artery stenosis.
- Listen over the liver and spleen for bruits and friction rub

4-succussion splash like a half-filled water bottle being shaken. Explain the procedure to the patient, then shake the patient's abdomen by lifting him with both hands under his pelvis(normally within 4 hours from meal)

5-don't forget to mentioned that you have to examine

1-digital rectal examination

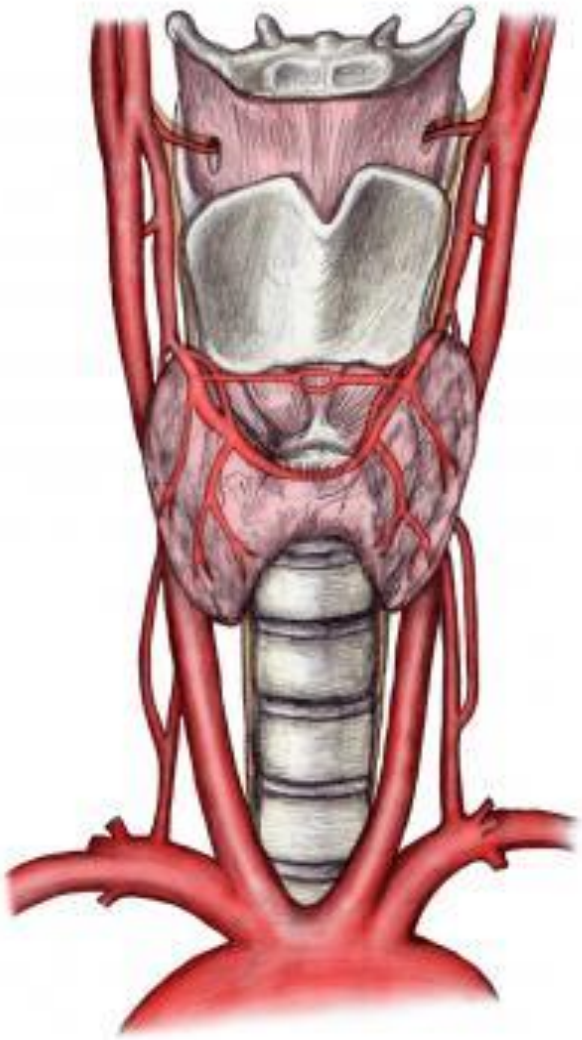
2-genetalia

3-back

4-virchow's LN

5-lower limbs: (edema, loss of hair , pyoderma gangrenosum and auscult for bruit above femoral art.)

Chapter 4: Endocrine System

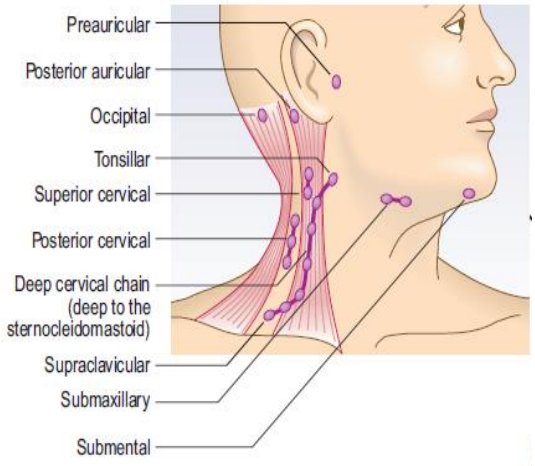


This part includes the most probable topics that show up in 4th year OSCE, these are:

- *Hyper/ hypothyroidism history and physical exam.*
- *Cushing's syndrome history and physical exam.*
- *Lower limb examination in diabetic patient and DM follow up.*
- *History of hirsutism.*
- *Acromegaly physical exam.*
- *Calcium disorders history.*

Before we start, few notes related to more than one topic you should remember:

❖ **Cervical lymph nodes:**



❖ **visual fields examination:**

Peripheral visual fields

- Test each eye separately.
- Ask the patient to cover one eye and look directly into your opposite eye.
- Shut your eye that is opposite the patient's covered eye.
- Test each quadrant separately with a wiggling finger or white-tipped hatpin. Hold the target equidistant between you and the patient.
- Start peripherally and move the target along the diagonal towards the centre of vision until the patient detects it.
- Repeat for the other quadrants.
- Compare your visual field with the patient's.

Central visual field

- Test each eye separately using a red hatpin.
- Shut your eye that is opposite the patient's covered eye.
- Ask the patient to cover one eye and look directly at your open eye.
- Hold the hatpin in the centre of the visual field, as close to fixation as possible.
- Ask the patient what colour the hatpin is. A 'pale' or 'pink' response implies colour desaturation, usually because of a lesion affecting the optic nerve.
- Compare the four quadrants of the visual field centrally; each time ask about colour desaturation. Note that the visual field for red may be smaller than for white.

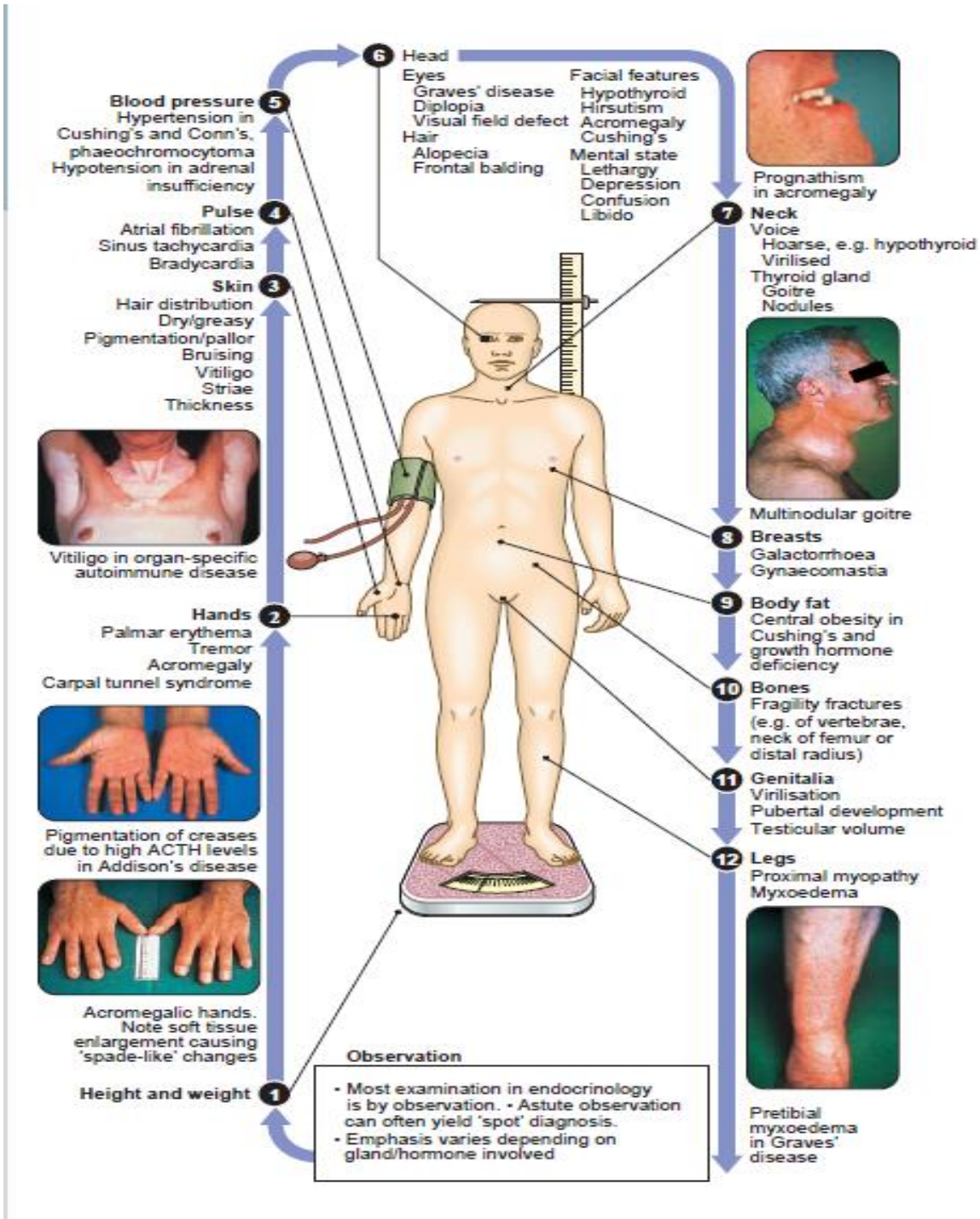
❖ **Testing for proximal myopathy:** ask the patient to stand from a sitting position with arms crossed; an inability to do this suggests proximal muscle wasting.

❖ **Diplopia:** test this by asking the patient to follow your finger by his eyes, without moving the head, while you are drawing H shape with your finger in the air.

❖ **Testing for carpal tunnel syndrome (CTS):**

1- Tinel's sign: by percussion with tip of your index over median nerve close to wrist. Ask the patient for any shooting tingling sensation elicited upon percussion
2- Phalen's sign: ask patient to do reverse prayer's sign, ask whether he felt any pain elicited
3- Look for thenar muscle wasting

❖ **General endocrine findings on physical exam are summarized in the following picture:**



❁ Thyroid gland ❁

History Taking

OSCE stations may be as the following:

- *Take a proper history differentiating between hyper and hypothyroidism.*
- *Ask relevant questions to know the cause of hyper OR hypothyroidism.*

➢ ***Quick review:***

The thyroid axis is involved in the regulation of cellular differentiation and metabolism in virtually all nucleated cells, so that disorders of thyroid function have diverse manifestations. Follicular epithelial cells synthesize thyroid hormones by incorporating iodine into the amino acid tyrosine. The thyroid secretes predominantly thyroxine (T4) and only a small amount of triiodothyronine (T3), the more active hormone; ~85% of T3 in blood is produced from peripheral conversion of T4. They both circulate in plasma almost entirely (> 99%) bound to transport proteins, mainly thyroxine-binding globulin (TBG).

➢ ***Plan:***

I. *Ask about symptoms of hyper/hypothyroidism regardless the cause.*
II. *Symptoms caused by anatomical location of goiter -if present-.*
III. *Past thyroid history and family history.*
IV. *Ask by differentials.*

I. Symptoms of hyper or hypothyroidism:

When asking to differentiate, you need to ask many questions related to almost all systems of the body; thus, you need to be systematic in order not to miss any:

System	Symptoms in hypothyroidism	Symptoms in hyperthyroidism
CNS	Depressed mood, apathy	Nervousness, anxiety, irritability and tremor
CVS		Palpitation, SOB.
GI	Constipation, decreased appetite.	Diarrhea, increased appetite.
GUS	menorrhagia	Oligomenorrhea or amenorrhea **In males: there may be gynecomastia, decreased libido and erectile dysfunction.*
MSS	Dry hands, symptoms of carpel tunnel syndrome, brittle nails and coarse hair or alopecia.	Sweaty hands, oncholysis, alopecia and muscle weakness

Others *-very important-*	Cold intolerance, husky voice and weight gain.	Heat intolerance and weight loss.
		Ask about eye problems: exophthalmos, diplopia, lid lag. *Seen mostly in Grave's.*

II. Symptoms caused by anatomical location of goiter -if present-:

- Dysphagia -compression on the esophagus-.
- Dyspnea and SOB -compression on the trachea-.
- Dysphonia.

III. Past thyroid history and family history:

- Previous thyroid diseases in general.
- Family history of any thyroid diseases.

IV. Ask by differentials:

Causes of hypothyroidism	**Causes of hyperthyroidism**
Hashimoto thyroiditis	Toxic MNG/adenoma
Postpartum thyroiditis	
Post total thyroidectomy.	Grave's disease
Post I131 treatment	HCG-induced hyperthyroidism -Associated with pregnancy or Trophoblastic Tumors
Congenital, i.e. Thyroid agenesis or dysplasia.	Hyperthyroidism due to thyrotropin secretion (TSH-oma).
Medications, i.e. Lithium and Amiodarone	Inherited non-immune hyperthyroidism
Iodine deficiency	Thyroxine induced or amiodarone induced.
Central hypothyroidism	Subacute thyroiditis "daQuevarian":
Thyroid infiltration, i.e. Riedel's struma, amyloidosis, and hemochromatosis	

How to ask:

- Causes of hypothyroidism:
  - **I. Hashimoto thyroiditis:**
    It an autoimmune disorder so you have to ask:
    → If the patient has any other autoimmune diseases especially: vitiligo, DM type 1, primary adrenal insufficiency and pernicious anemia.
    → Family history of any autoimmune diseases.
  - **II. Postpartum thyroiditis:**
    → Ask about last pregnancy and whether the symptoms are related to the postpartum period or not -symptoms arise within 1-6 months after childbirth-.
  - **III. Post total thyroidectomy:** did the patient undergo thyroid surgery?

IV. **Post radioactive iodine therapy:** did the patient has been treated with radioactive iodine previously?

V. **Iodine deficiency or excess:** ask about the patient's diet.

VI. **Amiodarone/ lithium therapy "i.e. drugs".**

Causes of Hyperthyroidism:

1. **Grave's disease:**
   → You need to ask about autoimmune diseases like addision's or DMT1
2. **Subacute thyroiditis "de Quervaian":**
   It is caused by viral infection.
   → Associated with fever, systemic upset and pain that may radiate to the ear.
3. **Thyroxine induced or by amiodarone "i.e. drugs"**
4. **Toxic MNG/adenoma:** suggested when there is a palpable mass over in the thyroid.
5. **HCG-induced hyperthyroidism:** associated with pregnancy or Trophoblastic Tumors.
6. Hyperthyroidism due to thyrotropin secretion (TSH-oma): headache, visual symptoms may be present.
7. Inherited non-immune hyperthyroidism

NOTES:

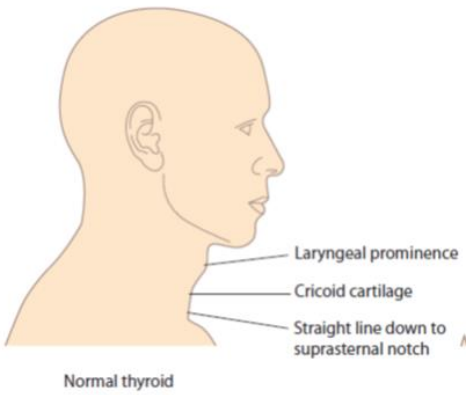
1- If a station came and you suspected that it might be related to thyroid carcinoma, ask the following:
- *Ask about rate of growth; if rapid think of thyroid lymphoma.*
- *Ask about symptoms due to metastasis:*
  - *bone: pain, pathological fractures*
  - *Lungs: cough, SOB...*
  - *CNS: headache, dizziness…*
- *Family history of thyroid malignancy.*
- *Constitutional symptoms of any malignancy: anorexia, significant weight loss, fever and night sweats.*

2- Note that amiodarone may cause hypo or hyperthyroidism.

Physical Examination

- **Position and exposure:** the patient should be sitting with neck fully exposed.
- **General inspection**:
  → General appearance of the patient: agitated, nervous, apathy.
  → His clothes may indicate heat or cold intolerance.
  → Husky voice.
- **Systematic examination:**
  1- **Vitals:** do not forget to mention that you need to check the vitals.

NOTE: *when palpating thyroid gland it is important to place your hand on the correct place of thyroid –as shown in the figure-. NOT on the laryngeal prominence.*



Normal thyroid

→ Pulse: *Assess the radial pulse for…*

- Rate:
  - → Tachycardia (hyperthyroidism)
  - → Bradycardia (hypothyroidism)
- Rhythm – irregular (atrial fibrillation) – thyrotoxicosis

2- <u>Hands:</u>

Inspect the patients' hands for…

→ Dry skin (hypothyroid) /Increased sweating (hyperthyroid)
→ Temperature: Cold -in hypo-/ -hyper-warm
→ Thyroid acropachy "in Graves' disease".
→ Palmar erythema in hyperthyroidism.
→ Peripheral tremor in hyperthyroidism.
→ Test for carpel tunnel syndrome -seen in hypothyroidism-

3- <u>Face:</u>

Inspect the face for…

- Dry skin – hypothyroidism / Sweating – hyperthyroidism
- Thinning of the hair –hypothyroidism-
- Eyebrows: loss of the outer third in hypothyroidism (rare)
- Periorbital edema.
- Eyes:

I. <u>Exophthalmos</u> (anterior displacement of the eye out of the orbit) in Graves': inspect from the front, side and above.
II. <u>Lid retraction:</u> note if the sclera is visible above the iris -seen in Graves' disease-
III. Inspect for <u>any redness / inflammation</u> of the conjunctiva -seen in hyperthyroidism-.
IV. <u>Diplopia:</u> Eye movement can be restricted in Graves' disease due to abnormal connective tissue deposition in the orbit and extra-ocular muscles and this may result in diplopia.
V. <u>Lid lag</u>.

4- <u>Thyroid gland examination:</u>

- **Inspection:**

Inspect the neck for any:

→ Skin changes.
→ Scars -may indicate thyroid surgery.
→ Mass -if found describe its site, size, shape and skin over it *"the 4 Ss'"*
→ Ask the patient to:

- Sallow -thyroid normally moves with swallowing-
- Protrude the tongue –thyroid does NOT move while protruding the tongue, thyroglossal cyst does-.
- Open his mouth and inspect if there is a lingual thyroid.
- To raise his arms above the head notice any facial congestion and cyanosis - ***Pemberton's sign-.***

- **Palpation:** *do NOT forget to ask the patient if there is any pain.*

→ From Anterior: note any tenderness.

→ From posterior with slight flexion of the neck:

- Palpate each thyroid lobe by itself, and comment on the symmetry of the lobes, if there is any masses then describe them (4 Ss, tenderness, temperature, consistency, attachment, pulsation, surface, edges, mobility), any thrills.
- Ask the patient once again to sallow and protrude the tongue while palpating this time.
- Cervical Lymph nodes.
- Trachea for deviation.
- Carotid pulse: to check if there is berry sign which is the absence of carotid pulsation on palpation.

- **Percussion:**
  Percuss the clavicle bilaterally to see if there is any dullness -retrosternal goiter-.

- **Auscultate:**
  For bruits -found in Grave's' disease-.

- **finish your examination with:**

→ Testing the **deep tendon reflexes** –delayed relaxation in hypothyroidism/ hyperreflexia in hyperthyroidism-.

→ Inspect the lower limbs for **pretibial myxedema** -in Graves'-

→ **Test for Proximal myopathy;** proximal myopathy is associated with hyperthyroidism.

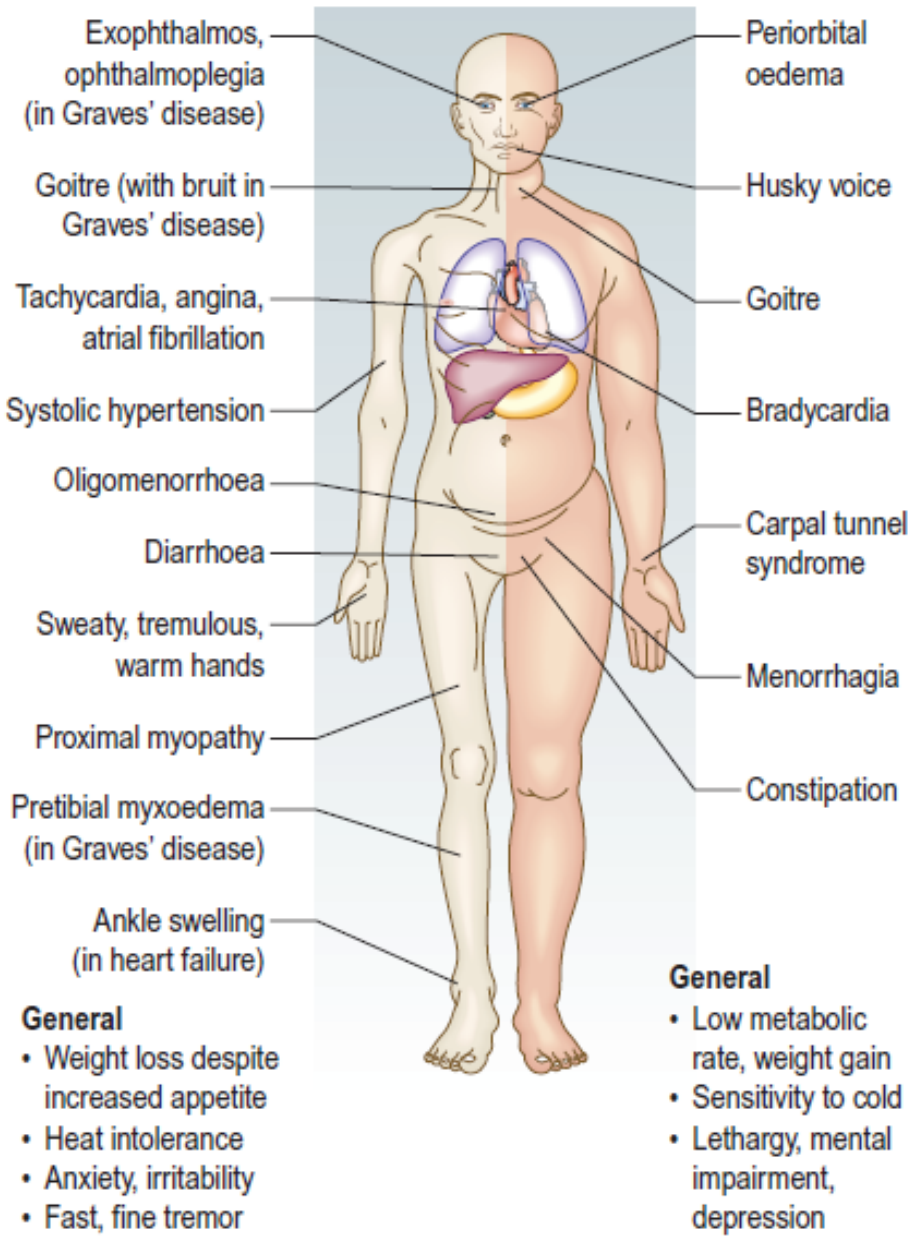
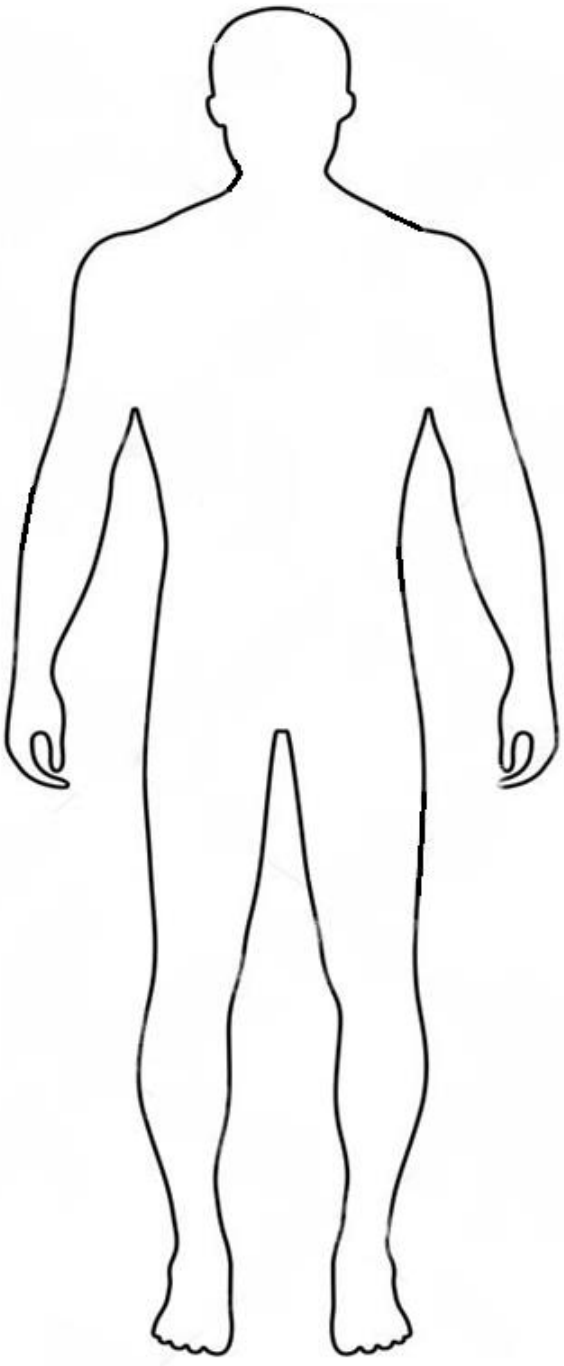


Fig. 5.3 Features of hyper- and hypothyroidism.

Here is a simple picture; you may revise the examination by writing the steps in your own way



❁ Cushing's Syndrome ❁

History Taking

NOTE: this part will be brief as it is unlikely to show up in the OSCE as a history-.

➢ ***Quick review:***

Cushing's syndrome is due to a chronic excess of glucocorticoids. Glucocorticoids (e.g., cortisol) stimulate lipolysis, the release of amino acids from the muscles, and gluconeogenesis by the liver. Cortisol inhibits all stages of the inflammatory process and also affects the bones by decreasing the protein matrix. Its immunosuppressive effect is on T cells and their associated cell-mediated immunity and delayed hypersensitivity. Excess cortisol can additionally stimulate mineralocorticoid and androgen receptors with the clinical appearance of aldosterone excess (hypertension, hypokalemia, and alkalosis). Cortisol does not bind androgen receptors. So glucocorticoids have multiple effects on the human body, so when taking history or examining a patient with Cushing's we are supposed to look for all effects of steroids excess.

➢ ***Plan:***

I. *You need to ask about the symptoms of excess steroids regardless the cause.*

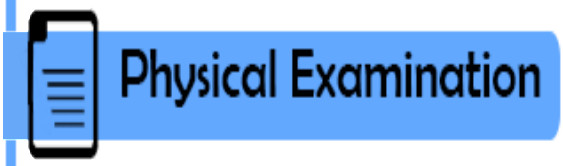
II. *Then, ask by differentials.*

I. Symptoms of excess steroids regardless the cause:

1- Ask about recent weight gain and how much?
2- Ask the patient if he/she bruises easily?
3- Has skin become thin recently? Any striae?
4- Ask if the patient has recently been more susceptible to infections?
5- Ask about visual problems as steroids could cause cataract.
6- Ask about problems of excess androgens: acne and excess hair in females.
7- In females, ask about menstrual irregularity or amenorrhea.
8- Ask about proximal myopathy: you may ask for example "if the patient is facing difficulty getting up from the chair?"
9- Ask whether the patient has been diagnosed with diabetes or not, or symptoms of hyperglycemia.
10- Ask about pathological fractures.

II. Ask by differential:

1- **Exogenous administration of steroids:** so ask the patient if he is taking steroids for any reason or not. It is extremely important to ask about this, as the most common cause of Cushing's is iatrogenic.
2- **Cushing disease: i.e.** excess ACTH production from the pituitary. Ask the patient if he has visiual problems suggesting a pituitary mass and ask whether the patient has noted any hyperpigmentation over his body.
3- **ACTH secreting tumors**: ask whether the patient has been diagnosed with malignancy and ask about general constitutional symptoms of malignancy.
4- **Adrenal neoplasia:** suggested when the symptoms develops rapidly and ask about flank pain or heaviness, however these two are not common symptoms to be found.
5- Finally, it is important to exclude things that causes **pseudo-Cushing's** and these include: depression, alcoholism and primary obesity.



Examining a patient for signs of Cushing's syndrome involves a general inspection of the patient, along with a systematic examination of the main organs involved in the disease.

- **Position and exposure:** inspect the patient generally while standing then ask him/ her kindly to lie down in supine position.
  Exposure: hands, face, chest, abdomen and lower limbs.

- **General Inspection:**

Look at the patient as a whole; remember that patients with Cushing's have a characteristic appearance that may be noted easily:

→ central obesity
→ stooped posture (due to osteoporotic damage)
→ Generalized changes over the body, including:
  ♦ bruising
  ♦ Striae -purple and wider than 1 cm are the characteristic striae of Cushing's-
  ♦ Thin skin

- **Systemic Examination:**

Examine each of the following body parts separately:

1- **Vitals:** blood pressure may be raised.
2- **Hands and arms:**
→ Bruising
→ Thin arms.
→ Hyperpigmentation in skin creases -REM: seen when Cushing's is caused by excess ACTH-.
→ Note whether there is changes suggesting rheumatoid diseases indicating an iatrogenic cause of the Cushingoid features.
→ Test for proximal myopathy.

3- Face:

→ Rounded face (moon face).
→ Visible vessels on the cheeks suggesting thin skin or telangiectasia.
→ Greasy skin.
→ Acne.
→ Hirsutism.
→ Plethora: *"important as this is only seen in true Cushing's not in pseudocushing's"*
→ At this point you may tell the examiner that you want to test Visual fields (looking for a bitemporal hemianopia that may be the result of an ACTH producing pituitary tumor pressing on the optic chiasm)

4- Shoulders:

→ Supra-clavicular fat pads.
→ Buffalo hump.

5- Chest: gynecomastia in males.

6- Abdomen:

→ Central obesity
→ **Purple** Striae.
→ Bruising.
→ Renal transplant scars (patient may be on long term steroids causing the Cushing's syndrome).
→ Palpate the abdomen for any adrenal masses: rarely found.

7- Legs:

→ Skin ulceration "poor wound healing".
→ Palpate for edema -due to hypertension secondary to Cushing's-
→ test for proximal myopathy

8- Finally, you may examine **the Spine**: for spinal tenderness (may occur with osteoporosis if vertebral fractures are found).

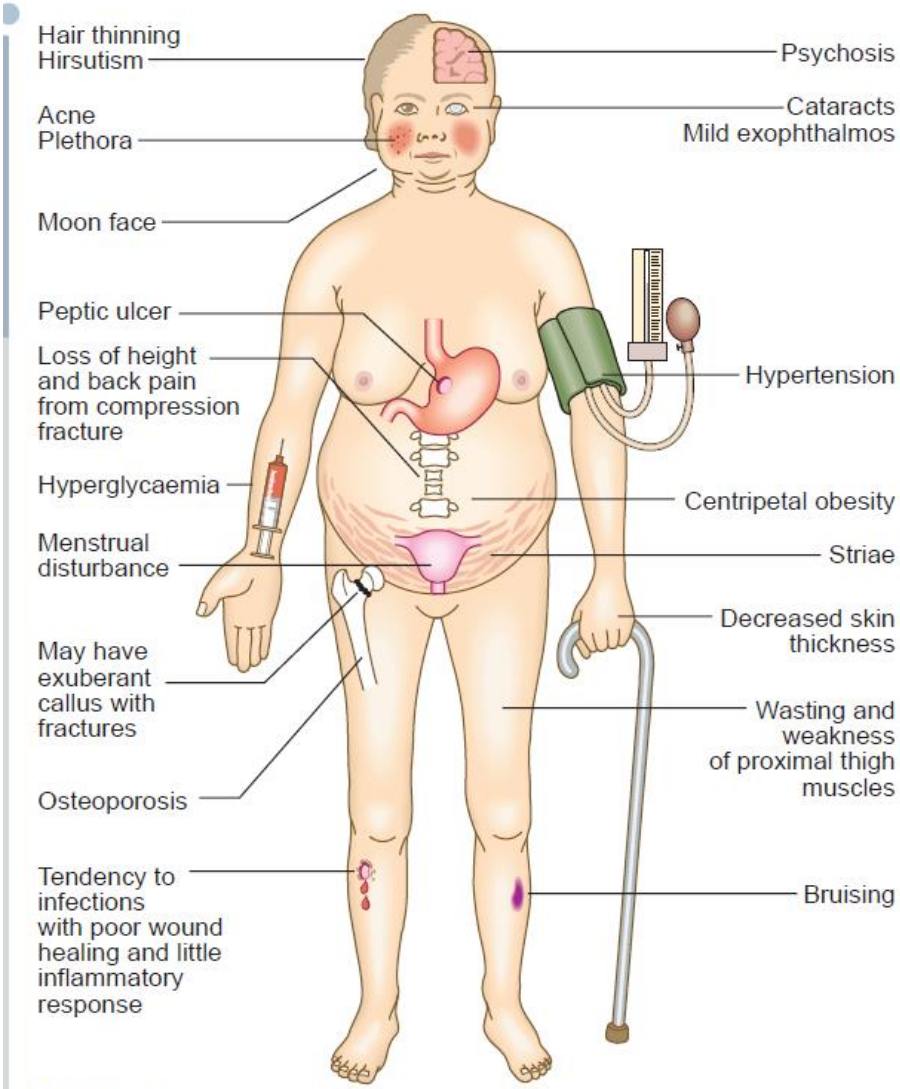
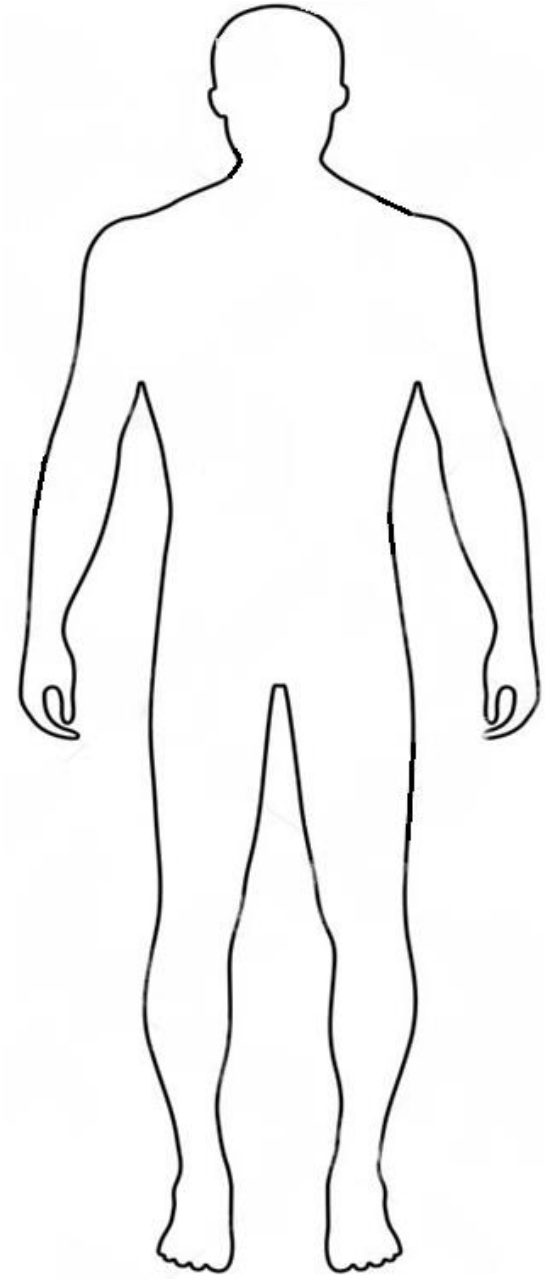


Fig. 10.6 Clinical features of Cushing's syndrome.

Here is a simple picture; you may revise the examination by writing steps in your own way



Hirsutism

History Taking

➢ ***Quick review:***

Normally in females, both the ovaries and the adrenals contribute to androgen production, almost equally. Usually, normal androgen production causes only growth of pubic and axillary hair.

Thus, hirsutism is excessive hairiness in a woman beyond what is considered normal for her race. It is caused by androgen (including testosterone) excess.

➢ ***Plan:***

I. *Ask about hirsutism regardless the cause.*
II. *Ask about symptoms of excess androgens in general.*
III. *Ask by differentials.*

I. Ask about hirsutism regardless the cause:

→ The onset of hirsutism, is it from the puberty or not? Since when developed?
→ Severity and rate of growth.

II. Ask about symptoms of excess androgens in general.

→ Deepening of the voice.
→ Blading.
→ Acne.
→ Decreased breast size.
→ Increased muscle mass.
→ Menstrual irregularities.

III. Ask by differential:

1- **Polycystic ovarian syndrome:** associated with gradual increase in hirsutism since puberty, **irregular** menstrual cycles, if the female is married ask her whether she has problems with fertility, ask whether if she has been diagnosed with diabetes recently, if she have sleep apnea and ask about her weight.
2- **Racial variation:** this is suggested when the patient has normal menstruation and has a family history of hirsutism.
3- **Cushing's syndrome:** suggested when there are symptoms of Cushing's-mentioned previously- such as weight gain, easily bruising and thin skin….
4- **Ovarian or adrenal carcinoma:** suggested by hirsutism developed over few months, amenorrhea and other androgen excess symptoms are found in such case.
5- **Drugs:** fluoxetine, pheyntoin, minoxidil and cyclosporine.

LIST 29.5 Causes of hirsutism

Polycystic ovary syndrome (most common cause)
Idiopathic
Adrenal: androgen-secreting tumours (e.g. Cushing's syndrome, congenital adrenal hyperplasia, virilising tumour—more often a carcinoma than an adenoma)
Ovarian: androgen-secreting tumour
Drugs: phenytoin, diazoxide, streptomycin, minoxidil, anabolic steroids (e.g. testosterone)
Other: acromegaly, porphyria cutanea tarda

- *Note that menstrual cycle:*
  - → *Irregular since puberty in PCOS*
  - → *Amenorrhea in ovarian or adrenal carcinoma.*
  - → *Normal and regular in racial variation*

❁ 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.

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?

➢ ***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

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…

→ Glucangonoma: 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:**

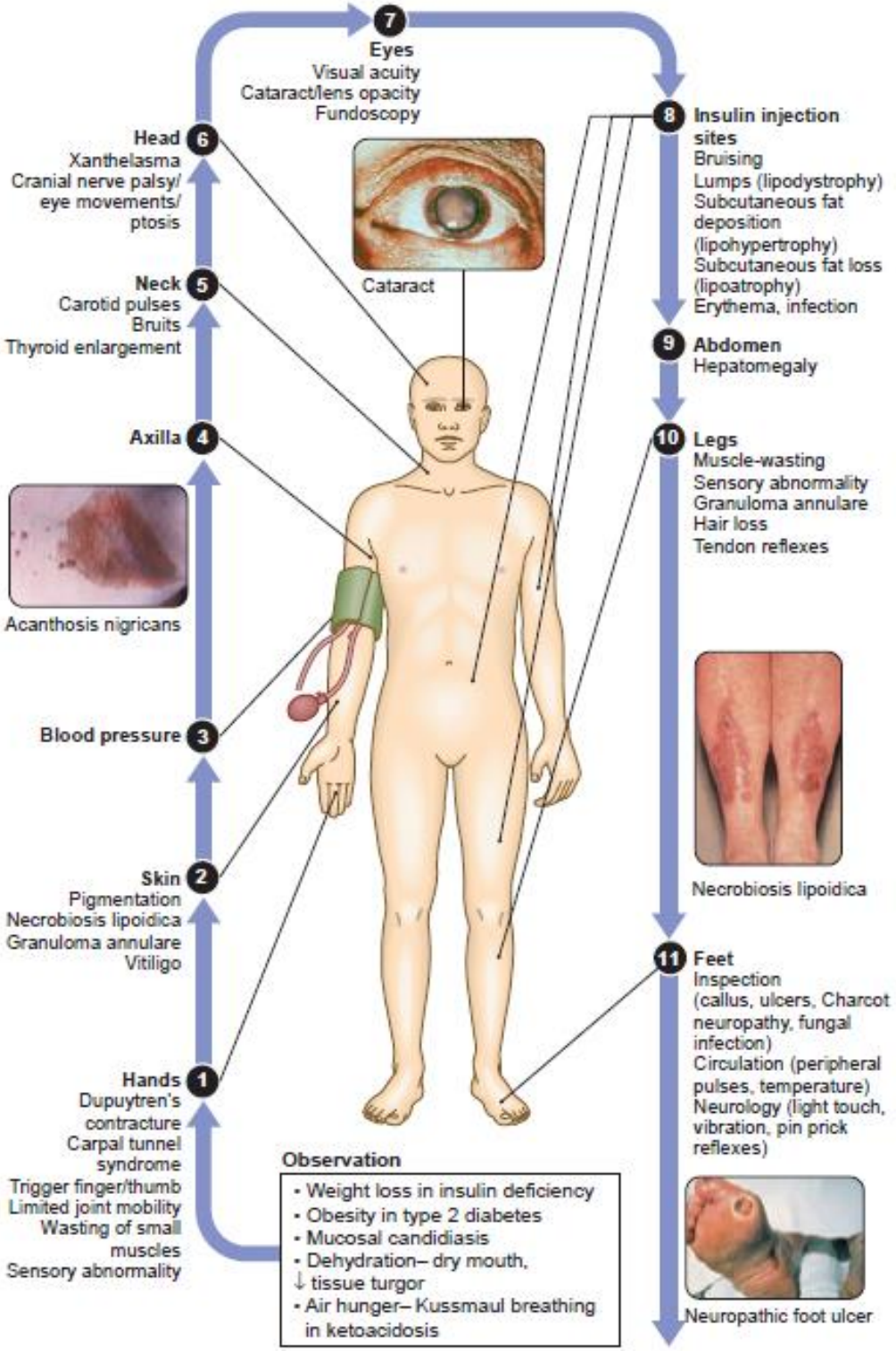
→ Sensation (do not forget to ask the patient to close his eyes then test for it).
→ Vibration (tested with tuning fork over a bony prominence).
→ Proprioception (do not forget to ask the patient to close his eyes then test for it).
→ Test the reflexes.

All of these may be absent or reduced.

- **<u>Finishing your exam:</u>**

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

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



Acromegaly

Physical Examination

➢ ***Quick review:***

Acromegaly is caused by excess GH secretion, usually from pituitary macroadenoma. If GH hypersecretion occurs before epiphyseal fusion, then gigantism will result. More commonly, GH excess occurs after epiphyseal closure and acromegaly ensues. Acromegaly is associated with cardiomyopathy, increased incidence of diabetes mellitus, hypertension and colonic cancer.

- **Position and exposure:** at 45° and expose chest fully and abdomen.
- **General Inspection:**

Patient Well or unwell?

Voice (deep and slow): deepening of the voice is a sign of acromegaly.

- **Systemic examination:**

1- Vitals:

→ BP (acromegaly is associated with increased incidence of hypertension).
→ Palpate the Pulse.

2- Hands:

♦ Inspection:
→ Large hands; coarse, sweaty skin.
→ Diabetic finger prick marks. -indicating DM-
→ Clubbing.
→ Changes of osteoarthritis are common due to skeletal overgrowth.
→ Examine for carpel tunnel syndrome.

3- Arms :

→ Palpate behind the medial epicondyle looking for ulnar nerve thickening, which is found in acromegaly.
→ Test for proximal myopathy

4- Face:

→ Prominent supraorbital ridges.
→ Large nose and lips.
→ Prognathism (prominent mandible) and malocclusion.
→ Wide spaces between the teeth.
→ Macroglossia.
→ Scars from previous surgery.

5- Eyes:

→ Test the Visual fields (looking for bitemporal hemianopia). This is due to mass effect from the pituitary gland.

→ Fundoscopy (papilloedema secondary to raised ICP, diabetic and hypertensive changes): just mention.

6- Axilla:

→ Acanthosis nigricans.

→ Skin tags (called *molluscum fibrosum*, which are non-tender skin colored protrusions)

7- Neck:

→ Goiter (increased thyroid size and vascularity)

→ Acanthosis nigricans: in the back of the neck.

8- Chest:

→ Coarse body hair.

→ Gynecomastia. –if associated with hyperprolactinemia-

→ Feel for displaced apex.

→ Listen for any murmurs.

→ Listen to lung bases if any suspicion of heart failure.

9- Abdomen:

→ Organomegaly (examine for hepatomegaly and splenomegaly).

10- Legs:

→ Large feet.

→ Proximal myopathy.

→ Peripheral edema (if associated heart failure).

→ Foot drop may be present due to common peroneal nerve entrapment.

11- Finally mention that you should look for testicular atrophy, as acromegaly may be associated with mixed pituitary tumors, with hyperprolactinemia or due to gonadotropin deficiency due to enlarging pituitary.

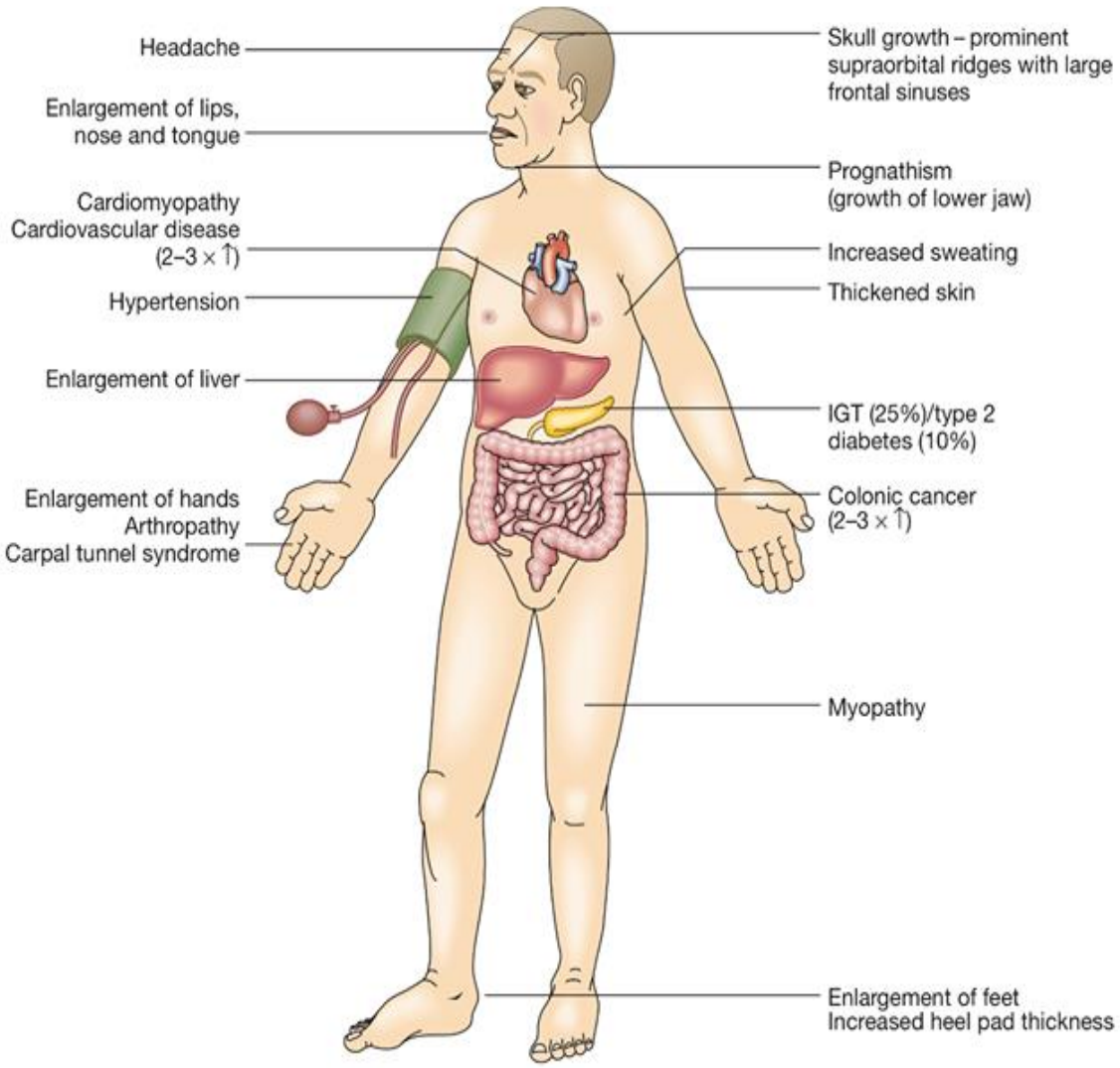
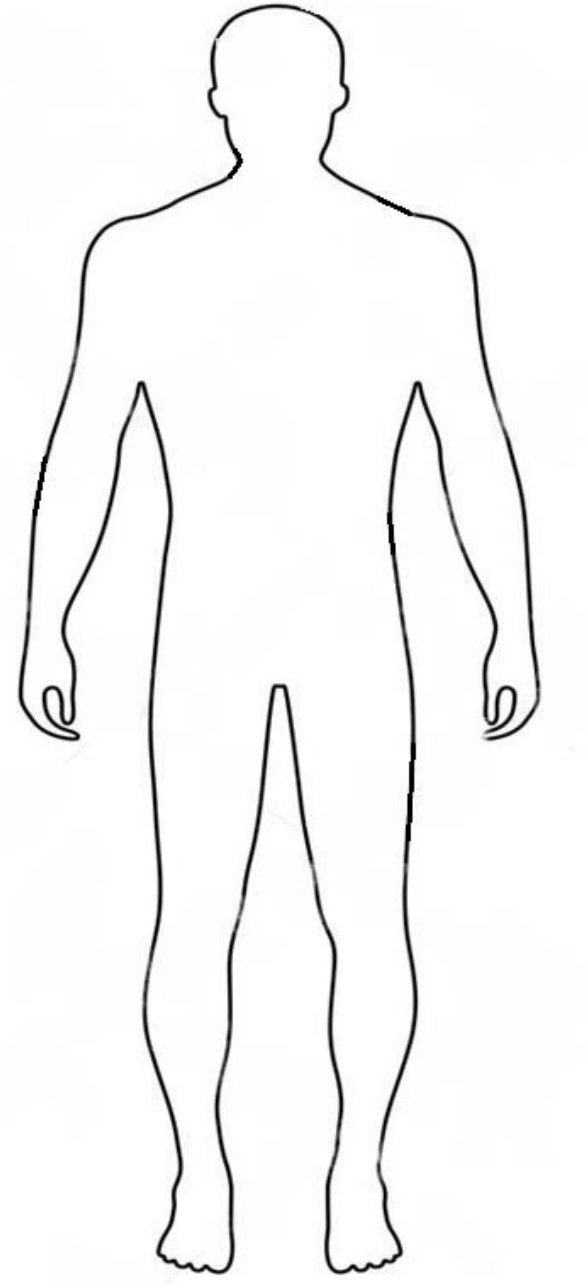


Fig. 20.29 Clinical features of acromegaly. (IGT = impaired glucose tolerance)



Calcium Disorders

History Taking

❖ Hypercalcemia:

Remember that symptoms of hypercalcemia are:

Stones, bones, abdominal moans and psychic groans

➢ *Plan:*

I. Ask about symptoms of hypercalcemia regardless the cause.
II. Ask by differentials.

I. Symptoms of hypercalcemia:

1. Renal symptoms:
   - Polyuria
   - Nocturia
   - Dehydration
   - Stones
2. Skeletal symptoms:
   - Ask about bone pain and pathological fractures.
3. Neurological manifestations:
   - Lethargy
   - Weakness
   - Loss of consciousness
   - Confusion
4. GI:
   - Nausea
   - Anorexia
   - Constipation
   - History of peptic ulcer

II. Ask by differentials:

Most common causes you need to ask about are:

1- Primary hyperparathyroidism: *has no specific symptoms other than the mentioned above.*
2- Hypercalcemia due to bone Mets: ask about bone pain or any diagnosed cancer.
3- Malignancy related hypercalcemia: most commonly associated with lung cancer so ask about general respiratory symptoms, general symptoms of any malignancy (like weight loss) or history of malignancy.
4- Multiple myeloma: associated with weight loss, back pain and symptoms of decreased immunity - recurrent infections-.
5- Drugs: like thiazides.
6- Familial hypercalcemia hypocalciuria.

❖ Hypocalcemia:

➢ *Plan:*

I. Ask about symptoms of hypocalcemia regardless the cause.
II. Ask by differentials.

I. Symptoms of hypercalcemia:

- Tingling around mouth and the fingers
- Muscle cramps
- Fits or seizures

II. Ask by differential :

1- Recent surgery of thyroid or parathyroid.
2- Diseases of malabsorption.
3- Renal diseases.
4- History of recent acute pancreatitis.
5- Hypocalcemia of malignancy.
6- Hypomagnesemia.
7- Pseudo-hypoparathyroidism: this disease is associated with skeletal deformity in general and short 5^{th} or 4^{th} finger or toe specially.
8- Vitamin D deficiency.

Chapter 5: Hematology

** You will notice that this system is not presented in the usual way as the CVS, RS, or GI (simply because it's very different). However, we hope you find it simple and easy.

** Contents

Anemia

❖ Revision of some basics

I. Anemia is defined as "reduction in circulating RBC mass". Since hemoglobin (Hb), hematocrit (Hct) and RBC count are used as surrogates for RBC mass (which is difficult to measure), we can say that anemia is defined as "reduction in Hct or Hb concentration".

➔ Anemia is defined as Hb <13.5 g/dL in males, and < 12.5 g/dL in females (normal Hb is 13.5-17.5 g/dL in males and 12.5-16.0 g/dL in females). *These values are according to Pathoma.*

II. Anemia could present as isolated anemia or as part of pancytopenia (Anemia + Thrombocytopenia + neutropenia).

III. Classification

- Based on mean corpuscular volume (MCV), anemia can be classified as Microcytic (MCV < 80 μm^3), Normocytic (MCV = 80 -100 μm^3), or Macrocytic (MCV > 100 μm^3).
- If Microcytic anemia (MCV < 80), the differential diagnosis includes the following:
  - Iron deficiency anemia,
  - Anemia of chronic disease,
  - Thalassemia,
  - Sideroblastic anemia (includes lead poisoning, pyridoxine deficiency, toxic effects of alcohol).
- If Macrocytic anemia (MCV > 100), the differential diagnosis includes the following:
  - Vitamin B12 deficiency and/or folate deficiency,
  - Liver disease.
  - Others; alcoholism, drugs (e.g. Fluorouracil (5-FU))
- If Normocytic anemia, the differential diagnosis includes the following: {"TARAB"+**H**}
  - Tumor
  - Aplastic anemia
  - Renal failure (decreased erythropoietin production)
  - Anemia of chronic disease (chronic inflammation, malignancy)
  - Bone marrow fibrosis
  - **H**EMOLYSIS (Hereditary spherocytosis, Sickle cell anemia, Paroxysmal nocturnal hemoglobinuria (PNH), G6PD deficiency, Immune hemolytic anemia (IHA), Microangiopathic hemolytic anemia (MAHA), Malaria).

- For the classification to be more accurate and more clinically relevant, we should look at the reticulocyte index.
  - The reticulocyte count is an important initial test in evaluation of anemia because it indicates whether effective erythropoiesis is occurring in the bone marrow. *(in fact, you should look at the reticulocyte index before the MCV as you'll see in a minute).*
  - A reticulocyte index > 2% implies excessive RBC destruction or blood loss. The bone marrow is responding to increased RBC requirements (the bone marrow is trying to compensate what has been lost/destroyed).
  - A reticulocyte count < 2%, in the presence of anemia, implies inadequate RBC production by the bone marrow.

IV. Clinical features of anemia:

- A variety of non-specific complaints: **fatigue/weakness, shortness of breath,** palpitations**, headache, dizziness/lightheadedness**, poor concentration, diarrhea, nausea, vague abdominal discomfort.
- Pallor of conjunctiva or skin ("Have you -or someone you know- noticed that you look pale?")
- Angina, especially with preexisting coronary artery disease.
- Hypotension (also ask about postural lightheadedness) and tachycardia.
- Signs of the underlying cause- jaundice if hemolytic anemia, blood in stool if GI bleeding,…

V. Important investigations when it comes to anemia include:

- CBC (we're mostly concerned with Hb/Hct, reticulocyte index -if present-, and RBC indices especially MCV)
- Blood smear
- Specific tests according to your suspicions; like Iron studies, Vitamin B12 and folate levels,..

VI. Evaluation of anemia (general approach) - *from Step-Up:*

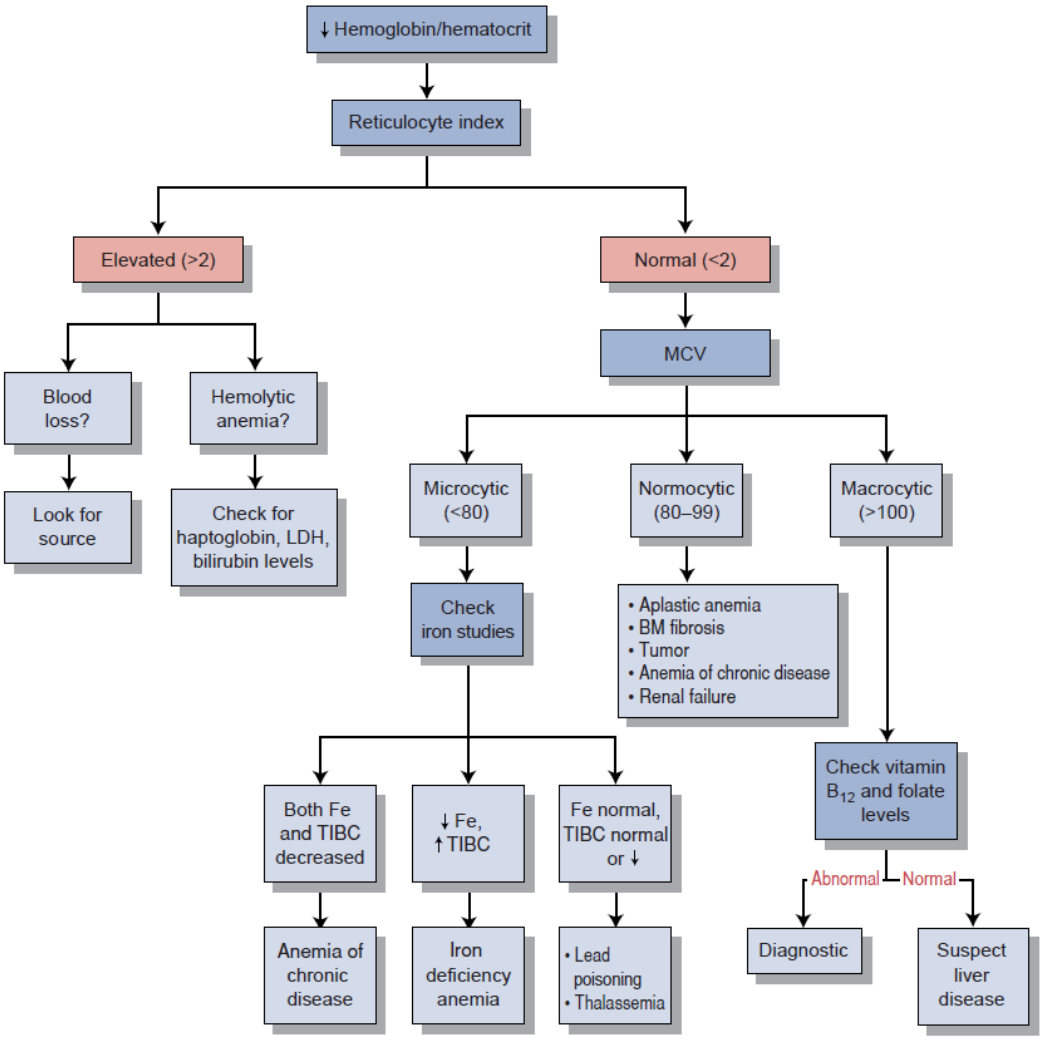


FIGURE 9-1 **Evaluation of anemia.**

Note: the following tables include **some** key points about the most important -but not all- diseases causing anemia. If you did not take the hematology rotation or did not study the system yet, these will help in making "History taking" (the following section) easier. However, if you do have the time, it's better to read these topics from Step-Up (chapter 9) or any other similar source.

Microcytic Anemias

(1) Iron Deficiency Anemia

Causes:

- **Chronic blood loss** (think of menstrual bleeding or GI blood loss)
- **Dietary insufficiency/increased iron requirements**, primarily seen in these three groups:
  - Infants and toddlers (especially if the diet is predominantly human milk [low in iron]. + Children in this age group have an increased requirement for iron because of accelerated growth).
  - Adolescents (rapid growth increases iron requirements). Adolescent women are particularly at risk due to loss of menstrual blood.
  - Pregnant women (pregnancy increases iron requirements).

NOTE: the RDW measures the variation in RBC size. The RDW is usually abnormal (high) in iron deficiency anemia, but is generally normal in other microcytic anemias.

(2) Anemia of Chronic Disease

- Anemia associated with **chronic inflammation** (e.g. endocarditis or autoimmune conditions) or **cancer**.
- The anemia can be normocytic or microcytic.
- Why can it cause microcytic anemia? Because in chronic disease, iron is present in the body but it's trapped in macrophages thus not available for hemoglobin synthesis.

(3) Thalassemias

- **Inherited** disorders *(thus family Hx is of high significance)* characterized by inadequate production of either the alpha or beta globin chain of hemoglobin.
- Divided according to the chain that is deficient into α– and β- thalassemia.

(4) Sideroblastic Anemia

- Hereditary (due to certain enzyme deficiency) or acquired.
  Acquired causes include drugs (e.g. chloramphenicol, **alcohol**), exposure to **lead**, collagen vascular disease, **vitamin B6 [pyridoxine] deficiency** *(most commonly seen as a side effect of isoniazid treatment of TB).*

TABLE 9-1 Iron Studies in Microcytic Anemias

	Serum Ferritin	Serum Iron	TIBC	RDW
Iron Deficiency Anemia	Low	Low	High	High
Anemia of Chronic Disease	Normal/high	Low	Normal/low	Normal
Thalassemia	Normal/high	Normal/high	Normal	Normal/high
Sideroblastic Anemia	High	Normal/High	Normal/ Low	------

Macrocytic Anemias

(1) Vitamin B12 Deficiency

- The main dietary sources of vitamin B12 are meat and fish.
- Vitamin B12 is bound to intrinsic factor (produced by **gastric** parietal cells), so it can be absorbed by the **terminal ileum**.
- Causes: *(almost all causes are due to impaired absorption)*
  - Pernicious Anemia (lack of intrinsic factor due to an autoimmune process).
    Thus, the presence of other autoimmune processes (e.g. vitiligo or Hashimoto thyroiditis) in the history might be a hint.
  - Gastrectomy
  - Poor diet (e.g. STRICT vegetarianism); alcoholism.
  - Crohn disease, ileal resection
  - Other organisms competing for vitamin B12 (e.g. fish tapeworm or bacterial overgrowth).

Special clinical features (in addition to the usual signs and symptoms of anemia):

- Sore tongue (stomatitis and glossitis)
- **Neuropathy** (subacute combined degeneration of the spinal cord (manifests as poor proprioception and vibratory sensation {posterior column} and spastic paresis {lateral corticospinal tract}, other possibilities: Urinary and fecal incontinence, impotence, dementia).
  Neuropathy can distinguish between vitamin B12 deficiency and folate deficiency.

(2) Folate Deficiency

- Green vegetables are the main source of folate. Overcooking of vegetables can remove folate.
- Causes: *(almost all causes are due to impaired absorption)*
  - Inadequate dietary intake
  - Alcoholism
  - Increased demand/ pregnancy
  - **Long term use of oral antibiotics**
  - **Folate antagonists such as methotrexate**
  - **Anticonvulsant medications (phenytoin)**
  - Hemolysis
  - Hemodialysis

The clinical features of folate deficiency are similar to those in vitamin B12 deficiency, **without the neurologic symptoms.**

Normocytic Anemias (Hemolytic anemias will be in a separate table)

(1) Aplastic Anemia

- Bone marrow failure leading to pancytopenia (anemia, neutropenia, thrombocytopenia).
- Causes:
  - Idiopathic
  - Radiation exposure

(2) Anemia of Chronic Disease

- The release of inflammatory cytokines has a suppressive effect on erythropoiesis.
- The anemia can be normocytic or microcytic.

- Medications (e.g. chloramphenicol, sulfonamides, gold, carbamazepine).
- Viral infection (Human parvovirus B19, Hep.C, Hep.B, EBV, CMV, HIV, herpes zoster varicella).
- Chemicals (e.g. benzene, insecticides)

For **aplastic anemia** and **Bone marrow fibrosis:**

Special clinical features (in addition to the usual signs and symptoms of anemia):

- Signs and symptoms of thrombocytopenia (e.g. petechiae, easy bruising).
- Increased incidence of infection (due to neutropenia).

(3) <u>Anemia of CKD and Renal failure</u>

The main -*but not only*- mechanism is decreased EPO production.

However, the etiology of anemia in patients with advanced stages of CKD tends to be multifactorial (e.g. decreased RBC production due to lack of EPO, increased blood loss due to multiple venipunctures, chronic inflammation,..).

Hemolytic Anemias

Clinical Features

1. Signs and symptoms of anemia
2. Signs and symptoms of underlying disease (e.g. bone crisis in sickle cell disease)
3. **Jaundice** (due to unconjugated bilirubin), bilirubin gallstones (cholelithiasis), hepatosplenomegaly. *These are more seen with extravascular hemolysis* (mainly in spleen and liver).
4. Dark urine color (due to hemoglobinuria, not bilirubin) may be present. *This indicates intravascular hemolysis.*
5. Lymphadenopathy (in chronic cases).

(1) <u>Sickle Cell Anemia</u>

- **Autosomal recessive** disorder that results when the normal Hb A is replaced by the mutant Hb S. Sickle cell disease is caused by inheritance of two Hb S genes (homozygous).
- Hb S may be distinguished from Hb A by electrophoresis because of the substitution of an uncharged valine for a negatively charged glutamic acid at the sixth position of the β-chain.
- Under reduced oxygen conditions (e.g., acidosis, hypoxia, changes in temperature, dehydration, and infection) the Hb molecules polymerize, causing the RBCs to sickle. Sickled RBCs obstruct small vessels, leading to ischemia (see Clinical Pearl 9-3).
- Hemolysis is predominantly extravascular (so keep jaundice and gallstones in your mind).

Note: I recommend reading "clinical features of sickle cell anemia" from page 336 in Step-Up.

(2) <u>Hereditary Spherocytosis</u>

- **Autosomal dominant** inheritance of a defect in the gene coding for spectrin and other RBC proteins.
- There is a loss of RBC membrane surface area without a reduction in RBC volume, necessitating a spherical shape. The spherical RBCs become trapped and destroyed in the spleen (by macrophages)—hence the term extravascular hemolysis.

→We expect jaundice, gallstones, splenomegaly.

CLINICAL PEARL 9-3

Almost Every Organ Can be Involved in Sickle Cell Disease

- Blood—chronic hemolytic anemia, aplastic crises
- Heart—high-output CHF due to anemia
- CNS—stroke
- GI tract—gallbladder disease (stones), splenic infarctions, abdominal crises
- Bones—painful crises, osteomyelitis, avascular necrosis
- Lungs—infections, acute chest syndrome
- Kidneys—hematuria, papillary necrosis, renal failure
- Eyes—proliferative retinopathy, retinal infarcts
- Genitalia—priapism

(3) G6PD Deficiency

- An X-linked recessive disorder that primarily affects men.
- Known precipitants include; **fava beans, sulfonamides,** nitrofurantoin, primaquine, dimercaprol, and **infection**.
- Presents as episodic hemolytic anemia that is usually drug-induced.
- Hemolysis is predominantly (but not exclusively) intravascular.
- Dark urine and jaundice are usually present.

(4) Autoimmune Hemolytic Anemia (AIHA)

- Production of auto-antibodies toward RBC membrane antigen(s) which leads to destruction of these RBCs.
- Two types;
  1) Warm AIHA (autoantibody is IgG which binds optimally to RBC membranes at 37^{O}).
     Produces usually extravascular hemolysis – the primary site of RBC sequestration is the spleen. Splenomegaly is a common feature.
     Causes: primary (idiopathic), secondary to lymphomas, leukemias (**CLL**), other malignancies, **SLE** *(most common cause)*, drugs (**penicillin, cephalosporins, α -methyldopa**)
  2) Cold AIHA (autoantibody is IgM which binds optimally to the RBC membrane at cold temperatures (usually 0^{O}C TO 5^{O}C).
     Produces mainly intravascular hemolysis. *(but extravascular hemolysis is also possible).*
     Jaundice is expected if significant hemolysis is present.
     Causes: can be idiopathic or due to infection (such as **Mycoplasma pneumoniae** or **infectious mononucleosis**).

**** Other causes of hemolysis include:**

- Microangiopathic Hemolytic Anemia (MAHA)
  key points about this entity will be mentioned in the "Bleeding Tendency" section).
- Malaria

(5) Paroxysmal Nocturnal Hemoglobinuria (PNH)

- An acquired disorder that affects hematopoietic stem cells and cells of all blood linages.
- This is caused by deficiency of anchor proteins that link complement-inactivating proteins to blood cell membranes → the deficiency of this anchoring mechanism results in an unusual susceptibility to complement mediated lysis of RBCs, WBCs, and platelets.
- Results in intravascular hemolysis that usually occurs episodically, often at night during sleep.

Special clinical features (in addition to the usual signs and symptoms of anemia):

- **Pancytopenia**
- Thrombosis of venous systems can occur (e.g. of the hepatic veins [Budd-Chiari syndrome]).
  ***destroyed platelets release their cytoplasmic contents into circulation, inducing thrombosis).*

History Taking

Note: the question will NOT simply be "Take brief history from a patient with anemia to know the cause of the anemia". Instead, it is usually given in a way that is similar to the following:

["Mr. ~~~ presented to you complaining of fatigue, headache, and dizziness *(or SOB, or poor concentration, or diarrhea, … and so on)*. Take brief/ focused history to reach a diagnosis**.**" **+/- a clinical aid (CBC results for example)].**

The question/examiner might want you to reach a specific diagnosis in the end (and in this case, his answers would be directed towards one specific entity), or he might just want you to ask the questions that would cover the possible differential diagnoses (in this case, he'll be mostly "listening" to your questions rather than giving you precise answers).

Either way, I recommend using the following three steps while taking history:

The first step: Knowing that the case is anemia

The second step: knowing if the anemia is isolated or is part of pancytopenia (to narrow down the possibilities of the diagnosis)

The third step: Proceed with history taking (according to the general outline) to know the exact cause of anemia.

Please keep the **general outline** for history taking always in your mind.

- Patient profile
- Chief complaint and its details.
- Associated symptoms
  - Constitutional symptoms (**fever, malaise, weight loss,** chills and rigors, night sweats).
  - Other symptoms of the same system
- Review of systems
- Past medical (including blood transfusion), past surgical, OBG.
- Drug history
- Family history
- Social history
  - Life style; including diet
  - Smoking
  - Alcohol
  - Occupation
  - Address
  - Travel

(1) Knowing that the case is anemia

How:

1. By asking about symptoms of anemia. For example, if Mr.~~~ came complaining of fatigue and dizziness, you should ask about other symptoms like SOB (+ its relation to exertion), pallor, poor concentration,…
   If many – not necessarily all – are positive, this is probably anemia.
2. If there is a clinical aid in the question (for example, the CBC results), it will usually provide a hint that this is anemia (for example, Hb level = 10 g/dL).

(2) Knowing if the anemia is isolated or is part of pancytopenia

1. By asking about the following
   - **Recurrent infections** *(a result of neutropenia)*
   - Symptoms of skin bleeding → **Petechiae** (ask about small red spots on the skin) and **easy bruising**.
     Symptoms of mucosal bleeding → **epistaxis**, hemoptysis, GI bleeding, hematuria, and menorrhagia.

(results of thrombocytopenia)

2. Look at the WBC and platelet counts in the CBC, if present.
   Normal WBC count: 5,000-10,000 /µL (5-10 K/µL)
   Normal platelet count: 150,000-450,000 /µL (150-450 K/µL).

Note: usually -not always- the question will be more towards isolated anemia to get closer to a specific diagnosis. However even if it is isolated anemia, you usually get points for asking about the previous two points. [let the examiner see that you understand what you're asking about ;)].

Note 2: multiple myeloma (discussed in a later section) causes anemia due to BM infiltration and renal failure. It also causes recurrent infections due to deprivation of normal immunoglobulins.

Causes of pancytopenia include:

- Damage to bone marrow precursors e.g. **Aplastic Anemia**.
- Myelophthesic process (pathologic process (e.g. metastatic cancer) that replaces the bone marrow resulting in pancytopenia.
- **Myelodysplastic syndromes** (increased blasts -but less than 20%- in the bone marrow)
  & Acute leukemias (>20% blasts in the bone marrow)
  → increased blasts "crowd out" normal hematopoiesis resulting in pancytopenia.
- **Myelofibrosis** (bone marrow fibrosis)
- **Drugs** (e.g. certain chemotherapy drugs cause BM suppression)

(3) Reach the exact cause of anemia (the diagnosis)

According to the general outline:

- **Patient profile**, if you didn't ask about it in the beginning
- **Chief complaint** (whatever it was, ask about its onset, duration, course, and progression, and any other details that come to your mind)
- **Associated symptoms**
  - Constitutional symptoms (fever, malaise, weight loss, …)
  - Other symptoms of the same system [The presence of other symptoms of anemia, Recurrent infections, petechiae and easily bruising, epistaxis, … *(if you still haven't asked about them)*].
- **Review of systems**
  Be fast when asking, and if certain diagnoses are in your mind, focus on them.
  For example, if you're suspecting:
  - Iron deficiency anemia → ask about dyspnea on exertion, postural (orthostatic) lightheadedness, melena, hematemesis, epistaxis, pica (cravings for non-nutritive substances like sand, ice, or paint or others), injury, menstruation in females.
  - Pernicious anemia → ask about the symptoms of other autoimmune conditions like vitiligo or Hashimoto thyroiditis.
  - Hemolytic anemia → ask about jaundice, gall stones, dark urine, …
  - Sickle cell anemia → ask about severe bone pain (pain crisis), acute severe chest pain and SOB (acute chest syndrome), painful swelling at the dorsa of the hand and feet (dactylitis), joint pain, priapism, ophthalmologic complications, abdominal pain, … *(There are many*

Important Note: it might look like there's a lot to ask about (especially in the review of systems, past medical, and drug history), however, the hints in the case (like the CBC or the answers of the examiner usually reduce the possibilities, so pay good attention to the hints to save time and make your questions directed towards a specific group of diagnoses.

For example: if the question asks you find out the exact diagnosis, and the CBC reveals the presence of microcytic anemia *(this scenario came in the year 2017/2018- first semester)*, your questions should focus on: Iron deficiency anemia, thalassemia, sideroblastic anemia, and anemia of chronic disease, in an attempt to know which one of them is the right diagnosis.

(This is why studying the causes/risk factors of each disease -*which were mentioned in the previous tables*- is of high significance).

others. This is why reading the clinical features of sickle cell anemia from page 336 in Step-Up was recommended ;p)

- *The previous are only some examples, but apply the same principle whatever the disease you're thinking of, and remember that for the purpose of history taking in the OSCE, it's better to know the key points about **many** diseases, than to know every single detail about **few** diseases.*

- **Past medical history**
  - **Chronic diseases** (SLE, RA, chronic renal disease, chronic infections like TB, **Malignancies**, …)
    Note: if you ask "Do you have SLE?" or "Do you have chronic renal disease?" .., the examiner will sometimes NOT give you a direct yes/no, so you might need to elaborate and ask about SYMPTOMS of SLE or SYMPTOMS of chronic renal disease, and so on.
  - Don't forget to ask about history of **blood transfusion** as this might provide a clue to the diagnosis (for example, a patient with thalassemia or sickle cell anemia might be dependent on blood transfusions).
- **Past surgical history**
  - Ask about any surgery done, and focus on GI surgeries (e.g. colon resection due to malignancy, ileal resection, gastric sleeve,…)
- **OBG**
  - Asking about the severity of menses in female patients is super important when it comes to anemia.
    - Regularity of period and the frequency (ask about the number of days between the beginning of each menses and the next; *i.e. length of the cycle*).
    - Heaviness (ask about the numbers of days of menses, **the presence of clots in blood**, the frequency of changing pads)
  - Ask about current pregnancy
- **Drug history**
  - Aspirin, NSAIDs, anti-platelets, anti-coagulants (e.g. warfarin).
  - Chemotherapy
  - Carbamazepine, phenytoin, chloramphenicol → can cause aplastic anemia
  - Penicillin, cephalosporins → can cause drug-induced immune hemolytic anemia

  There are many other drugs. The previous are only some important examples
- **Family history** (very important)
  - Thalassemia
  - Sickle cell anemia
  - G6PD deficiency [X-linked recessive → so if the patient is a male *(which is most probably the case)* don't forget to ask about the maternal uncles and maternal grandfather of the patient].
  - Hereditary spherocytosis
- **Social history**
  - Life style including diet → ask about meat consumption, vegetables consumption, **fava beans sensitivity,** raw fish consumption [might cause fish tapeworm (*diphyllobothrium latum*) infestation → vit.B12 deficiency].
  - Smoking and Alcohol
  - Occupation and address (example: radiation exposure → aplastic anemia)
  - Travel (significant if you're suspecting malaria for example).

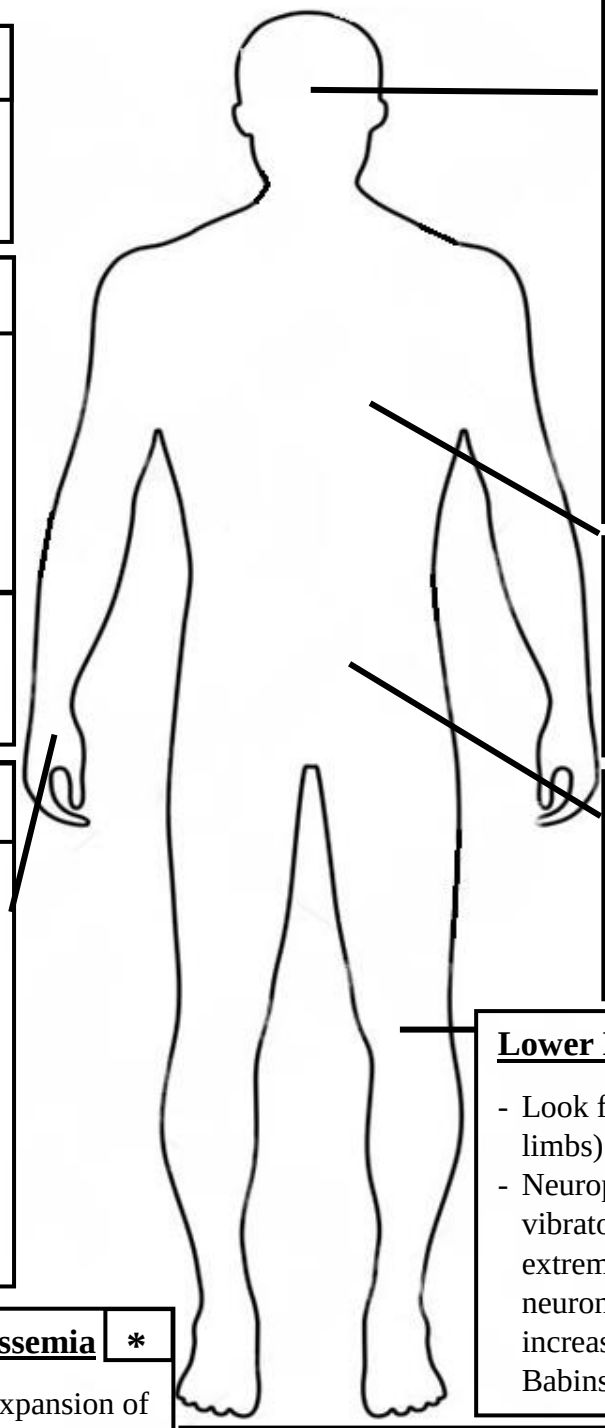
Physical Examination

(Signs of anemia on examination)

Please follow the numbers in order to be systematic and not forget important points.

The following figure is mostly about "anemia" in general with some points about specific diseases. However, keep in your mind that the specific cause of anemia might have its own signs.

IDA: Iron deficiency anemia HA: Hemolytic anemia



General 1

- Fatigue and in distress
- Pallor
- Jaundice (HA)

Vital Signs 2

- Heart Rate: **tachycardia**
- Blood pressure: assess for **hypotension**
- Respiratory rate
- Temperature: assess for **fever**

Always check for orthostatic changes in anemia.

Hands 3

- Koilonychia (IDA)
- Changes consistent with certain chronic diseases (example: Palmar erythema, leukonychia, and half and half nails in CLD,, clubbing in lung cancer or chronic IBD,, etc.)
- Vitiligo (pernicious anemia)

Head and Neck 4

- Eyes: look for pallor, or jaundice (HA)
- Mouth: look for angular stomatitis, and atrophic glossitis (IDA), hypertrophied tongue/beefy raw appearance (folate or B12 def.)
- Lymphadenopathy (chronic disease)
- Raised JVP

Chest 5

- S3
- Palpitations
- Tachycardia

Abdomen 6

- Hepatomegaly/ splenomegaly (in certain hemolytic anemias).

Lower Limbs 7

- Look for bruising (also in upper limbs) – pancytopenia
- Neuropathy: loss of position and vibratory sensation in the lower extremities, ataxia, upper motor neuron signs [spasticity, weakness, increased deep tendon reflexes, Babinski sign] – **B12 deficiency.**

Specific points for β-thalassemia *

- Distortion of bones due to expansion of bone marrow space → short limbs, rib deformities, chipmunk face.
- Growth retardation and failure to thrive
- Skull has a crew cut appearance on skull X-ray.

If the patient is likely getting blood transfusions (e.g. sickle cell anemia or β-thalassemia , you should mention that you need to examine for signs of iron over load; bronzed skin, liver disease signs, diabetes signs, hypothyroidism signs,… *(clinical features of hemochromatosis).*

Bleeding Tendency (the following text focuses on non-traumatic causes of bleeding)

❖ Revision of some basics

- ❖ Hemostasis occurs in two stages: primary and secondary
  - Primary hemostasis forms a weak platelet plug and is mediated by interaction between **platelets** and the vessel wall.
  - Secondary hemostasis stabilizes the platelet plug and is mediated by the **coagulation cascade.**

Disorders of primary hemostasis

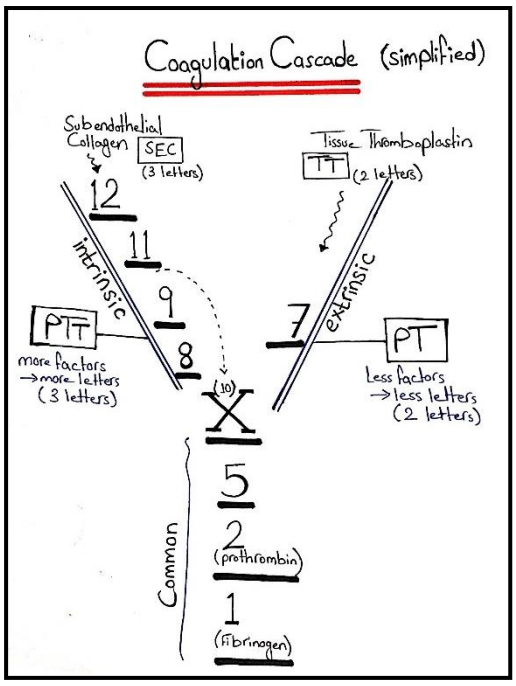
- ❖ Are usually due to abnormalities of platelets; they're divided into quantitative disorders or qualitative disorders.
- ❖ Platelets abnormalities usually cause mucocutaneous bleeding (skin and mucous membranes)
  - Symptoms of mucosal bleeding include epistaxis, hemoptysis, GI bleeding (including gum bleeding), hematuria, and menorrhagia. Intracranial bleeding occurs with severe thrombocytopenia.
  - Symptoms of skin bleeding include petechiae (1-2 mm), purpura (>3mm), ecchymosis (>1cm) and easy bruising. Petechiae are a sign of thrombocytopenia and are not usually seen with qualitative disorders.
- ❖ Useful laboratory studies include:
  - Platelet count – normal: 150,000-450,000 /µL (150-450 K/µL).
  - Bleeding time – normal: 2-7 minutes; prolonged with quantitative or qualitative platelet disorders.
  - Blood smear – used to assess the number, size, and shape of platelets.
  - Bone marrow biopsy (often not done) – used to assess megakaryocytes, which produce platelets.

Thrombocytopenia is defined as platelet count **less than 150 K/µL** (normal : 150-450 K/µL).

As in anemia, thrombocytopenia could present as isolated thrombocytopenia or as part of pancytopenia (Anemia + thrombocytopenia + neutropenia).

Disorders of secondary hemostasis

- ❖ Are usually due to factor abnormalities.
- ❖ Coagulation factors abnormalities usually cause deep tissue bleeding into muscles and joints (hemarthrosis) and re-bleeding after surgical procedures (e.g. circumcision and wisdom tooth extraction). Deep tissue bleeding is more severe than mucocutaneous bleeding.
- ❖ Useful laboratory studies include:
  - Prothrombin time (PT), measures extrinsic (factor VII) and common (factor II, V, X, and fibrinogen) pathways of the coagulation cascade.
    INR is derived of PT
  - Partial thromboplastin time (PTT) measures intrinsic (factors XII, XI, IX, VIII) and common (factor II, V, X, and fibrinogen) pathways of the coagulation cascade.



The following tables contain key points about important -but not all- diseases related to this topic.

Qualitative Disorders of primary hemostasis (normal platelet count, abnormal platelet function)

Von Willebrand Disease

- Considered a qualitative platelet disorder and a coagulation disorder at the same time.
- Due to **genetic** vWF deficiency.

→ Platelet adhesion is impaired.

- Autosomal dominant

Bernard-Soulier Syndrome

- Qualitative platelet disorder.
- Due to **genetic** GPIb deficiency.

→ Platelet **adhesion** is impaired.

- Autosomal recessive.
- Mild thrombocytopenia.
- Blood film shows enlarged platelets.

Glanzmann Thrombasthenia

- Qualitative platelet disorder.
- Due to **genetic** GPIIb/IIIa deficiency.

→ Platelet **aggregation** is impaired.

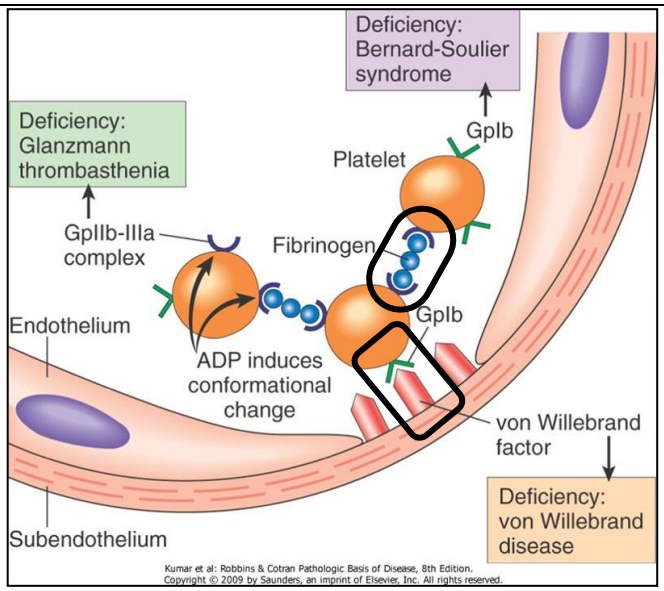
- Autosomal recessive

Remember:

Platelet adhesion is dependent on vWF and GPIb.

Platlet aggregation is dependent on GPIIb/IIIa, and fibrinogen as a linking molecule.

See the figure to the right.



Aspirin

Aspirin irreversibly inactivates cyclooxygenase → lack of TXA2 impairs aggregation.

Uremia

- Disrupts platelet function; both adhesion and aggregation are impaired.
- Look for signs of uremia (nausea, vomiting, chest pain, itching, uremic smell, decreased level of consciousness,…)

Quantitative Disorders of primary hemostasis

Idiopathic/Immune Thrombocytopenic Purpura (ITP)

- Autoimmune production of IgG directed against platelet antigens (e.g. GPIIb/IIIa).
- Usually a diagnosis of exclusion.

Heparin Induced Thrombocytopenia (HIT)

- Platelet destruction that arises secondary to heparin therapy.
- Fragments of destroyed platelets may activate remaining platelets leading to thrombosis. (so the main feared complication of Heparin-Induced Thrombocytopenia is actually THROMBOSIS ;p)

HUS and TTP *discussed in the following page (under the title Microangiopathic Hemolytic Anemia)*

<table>
<tr><th colspan="2">Disorders of secondary hemostasis (disorders of coagulation)</th></tr>
<tr><td><u>Hemophilia A</u>
• Genetic deficiency of factor 8.
• X-linked recessive disorder. Affects male patients primarily.
<u>Hemophilia B</u>
• Genetic deficiency of factor 9.
• X-linked recessive disorder. Affects male patients primarily.</td><td><u>Vitamin K Deficiency</u>
• Vitamin K is activated in the liver by epoxide reductase.
• Clotting factors 10, 9, 7, 2, and proteins C &S depend on activated vitamin K for their activation in the liver.
• Sources of vitamin K include diet (e.g. leafy green vegetables) and synthesis by gut flora.
• Causes of vitamin K deficiency:
- Long-term antibiotic therapy (suppression of gut flora)
- Malabsorption of fat soluble vitamins (e.g. small bowel disease, obstructive jaundice)
- Newborns (due to lack of GI colonization by bacteria)
- Warfarin (blocks epoxide reductase, thus, vitamin K will not be activated).</td></tr>
<tr><td rowspan="2"><u>Von Willebrand Disease</u>
Books put this disease with disorders of coagulation since it resembles a genetic deficiency of the Von Willebrand factor. However, remember what we said in the previous page (It also causes qualitative platelet problem since its deficiency impairs platelet adhesion).
In fact, it’s worth mentioning that patients with Von Willebrand disease present with skin and mucosal bleeding. Deep bleeding is usually <u>not</u> seen.</td><td><u>Liver disease</u>
<u>1.</u> Decreased production of most coagulation factors (most clotting factors are produced in the liver).
<u>2.</u> Decreased activation of vitamin K by epoxide reductase.</td></tr>
<tr><td><u>DIC</u> discussed in the following text (under the title Microangiopathic Hemolytic Anemia)</td></tr>
</table>

❖ **<u>Microangiopathic Hemolytic Anemia (MAHA)</u>**

- Pathologic formation of platelet microthrombi in small vessels.
- Results:
  - Platelets are consumed in the formation of microthrombi→ thrombocytopenia→ mucocutaneous bleeding.
  - In certain cases (like DIC), coagulation factors are also consumed → deep bleeding.
  - RBCs are ‘sheared’ as they cross microthrombi, resulting in hemolytic anemia with schistocytes (sheared RBCs) seen on blood film.
- MAHA is not a single disease, it’s in fact seen in several disorders such as:

  <u>(1). HUS</u> (Hemolytic Uremic Syndrome)
  - Classically seen in <u>children</u> with *E coli O157:H7* infection which results from exposure to <u>undercooked beef</u>.
  - Most important clinical features are the triad:
    <u>Hemolytic anemia</u> + **<u>thrombocytopenia</u>** + **<u>uremia/renal failure</u>**
    Some say that HUS is the most common cause of acute renal failure in pediatric age group.

  <u>(2). TTP</u> (Thrombotic Thrombocytopenic Purpura)
  - Patients with TTP lack functional ‘ADAM-TS-13 enzyme’.
  - Clinical features:
    <u>The triad of HUS</u> + **<u>fever</u>** + **<u>neurological symptoms</u>**

Quick HIT

TTP
- There is no consumption of clotting factors in TTP, so PT and PTT are normal.
- TTP = HUS + fever + altered mental status
- HUS = microangiopathic hemolytic anemia + thrombocytopenia + renal failure

(3). HELLP (Hemolysis, Elevated Liver enzymes & Low Platelet count)

- A life-threatening condition that can occur in pregnancy, where microthrombi mainly occur in the liver vasculature.

(4). DIC (Disseminated Intravascular Coagulation)

- Pathologic activation of the coagulation cascade leading to widespread microthrombi that result ischemia and infarction.
- The consumption of platelets and factors can result in either type of bleeding.
- DIC is almost always secondary to another disease process (e.g. Obstetric complications, Sepsis, Malignancy, Major tissue injury, Shock/circulatory collapse, Snake venom).

Note: another cause of MAHA is prosthetic heart valves.

Note: As in anemia, The question will NOT simply be "Take brief history from a patient with [thrombocytopenia or hemophilia, or …] to know the cause".

The question/examiner might want you to reach a specific diagnosis in the end (and in this case, his answers would be directed towards one specific entity), or he might just want you to ask the questions that would cover the possible differential diagnoses (in this case, he'll be mostly "listening" to your questions rather than giving you precise answers).

Either way, I recommend using the following steps while taking history:

The first step: Knowing that the case is "bleeding tendency", and what type (mucocutaneous Vs. deep).

The second step: Proceed with history taking to know the exact cause of the bleeding.

(1) Knowing that the case is 'bleeding tendency'

You should know that from the symptoms that the patient comes with.

- Epistaxis, purpura (often described as "small red spots on the skin"), hemoptysis, GI bleeding, menorrhagia, hematuria → mucocutaneous bleeding.
- Bleeding after surgeries or bleeding into joints → deep bleeding.

Important note *(regarding hemoptysis, hematuria, hematemesis, and hematochezia/melena)*: You should be very careful when it comes to these symptoms, because their presence might signify platelet disorders (e.g. thrombocytopenia), or a problem in the system they belong to (e.g. TB in the case of hemoptysis, or GN in the case of hematuria).

- How to tell? *see next page*

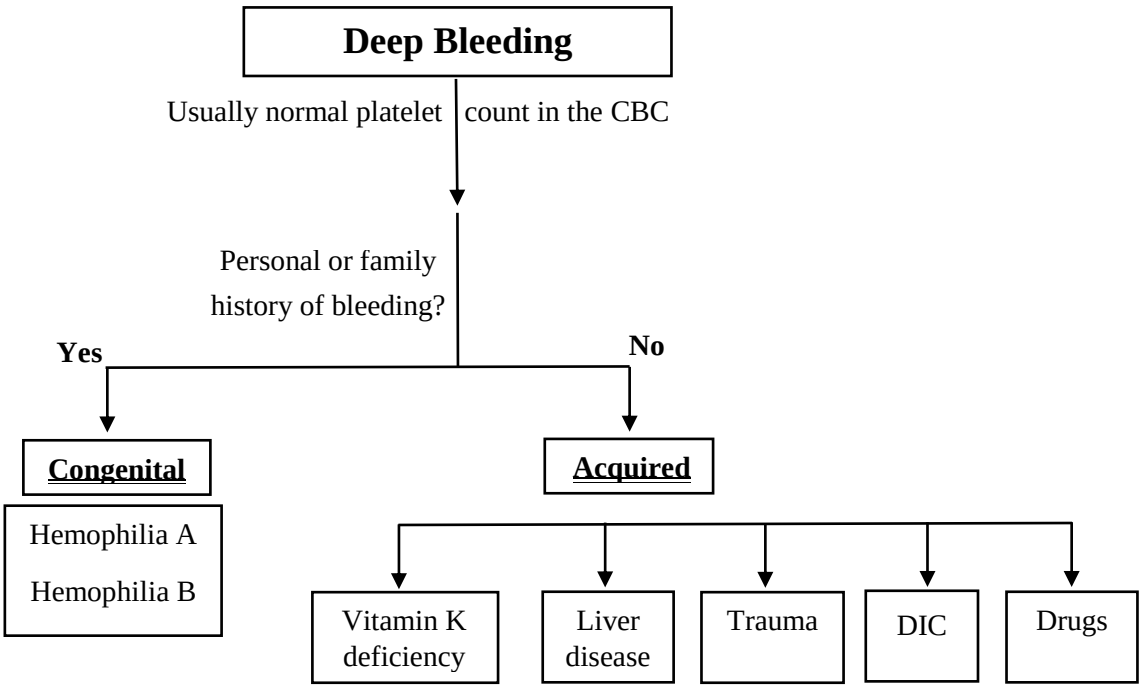
Note:

Anemia, followed by thrombocytopenia, are high yield topics when it comes to hematology history stations in OSCE.

- **Always ASK about bleeding from other sites**. For example, if a patient comes complaining of coughing blood, you should ask him about nose bleed, gum bleeding, small red spots on the skin, vomiting blood, blood in urine, heavy menses in females, and so on. If the answer is negative regarding the other sites, most probably the answer is towards diseases of the respiratory system rather than a bleeding tendency.
  Of course, other parts of the history like associated symptoms will also help you, but asking first about bleeding from other sites will be a good start to know which system to focus on.
  Please keep in your mind that the patient does not have to bleed from all mentioned sites to consider the case a bleeding tendency. For example, the patient might come with purpura only, or purpura with epistaxis and so on, but the note was added to remind you to be careful when it comes to the 'H' symptoms (hemoptysis, hematuria, hematemesis, and hematochezia/melena).

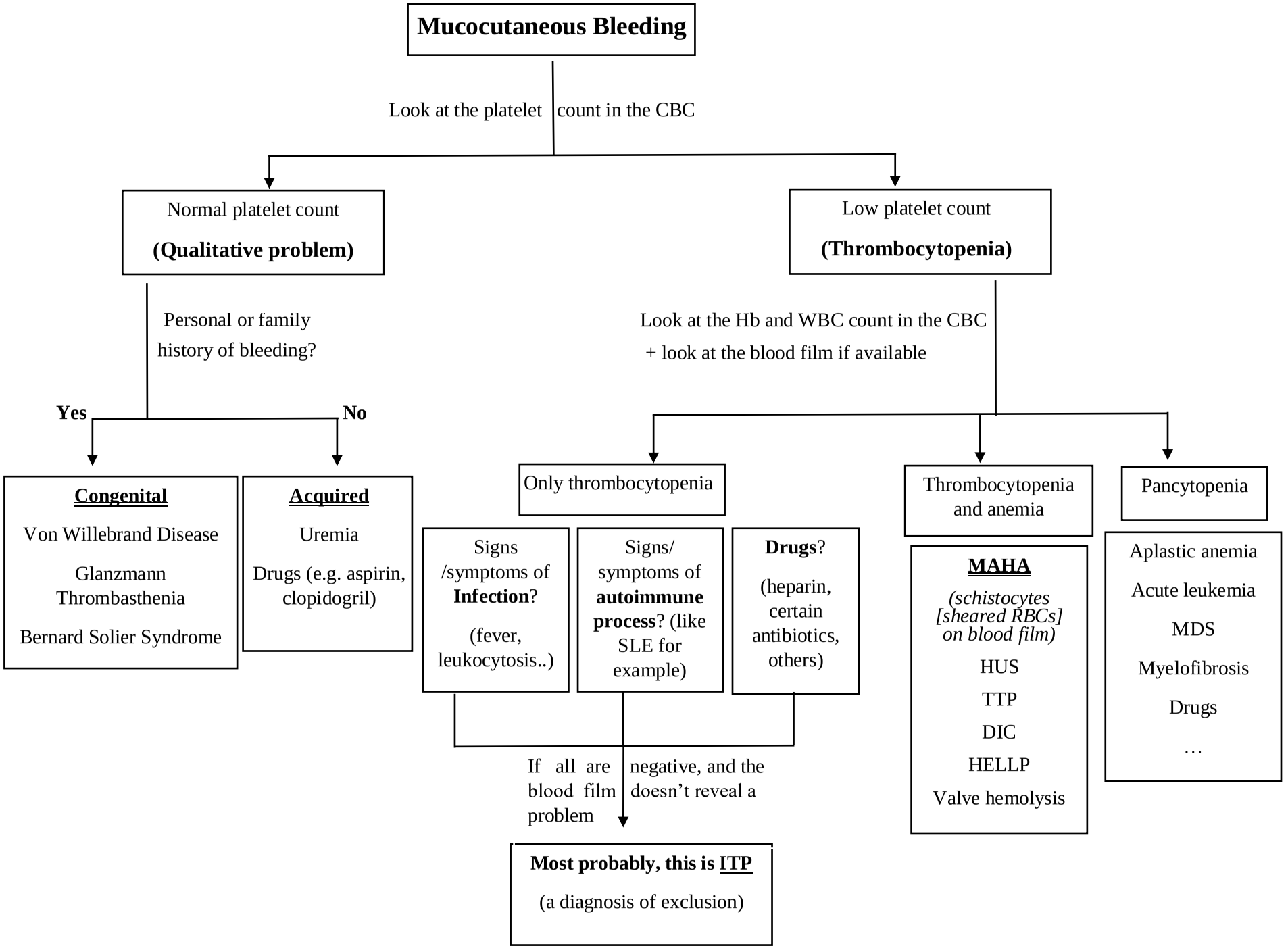
(2) Knowing the cause of bleeding

The following two schemes are according to Dr. Hussam Al-Qaisi's seminar. They were added because they are very useful in narrowing the possibilities when it comes to bleeding (both; clinically and in exams). You can use them as an aid in addition to the general outline for history taking, and the few points you know about each disease.



How to ask about personal or family history of bleeding *(bleeding in general)*:

Easy bruising, menorrhagia, prolonged bleeding after tooth extraction, required blood transfusions in previous surgery,… in the patient himself and his relatives; most importantly, first degree relatives (and maternal uncles/maternal grandfather in X-linked recessive disorders).


Mucocutaneous Bleeding
Look at the platelet count in the CBC
Normal platelet count
(Qualitative problem)
Low platelet count
(Thrombocytopenia)
Personal or family history of bleeding?
Yes
No
Congenital
Von Willebrand Disease
Glanzmann Thrombasthenia
Bernard Solier Syndrome
Acquired
Uremia
Drugs (e.g. aspirin, clopidogril)
Look at the Hb and WBC count in the CBC
+ look at the blood film if available
Only thrombocytopenia
Thrombocytopenia and anemia
Pancytopenia
Signs /symptoms of Infection?
(fever, leukocytosis..)
Signs/ symptoms of autoimmune process? (like SLE for example)
Drugs?
(heparin, certain antibiotics, others)
MAHA
(schistocytes [sheared RBCs] on blood film)
HUS
TTP
DIC
HELLP
Valve hemolysis
Aplastic anemia
Acute leukemia
MDS
Myelofibrosis
Drugs
…
If all are negative, and the blood film doesn't reveal a problem
Most probably, this is ITP
(a diagnosis of exclusion)

Physical Examination

(Signs of thrombocytopenia on examination)

The following figure is mostly about "thrombocytopenia" in general with some points about specific diseases. However, keep in your mind that the specific cause of thrombocytopenia might have its own signs.

MAHA: Microangiopathic Hemolytic anemia

General **1**

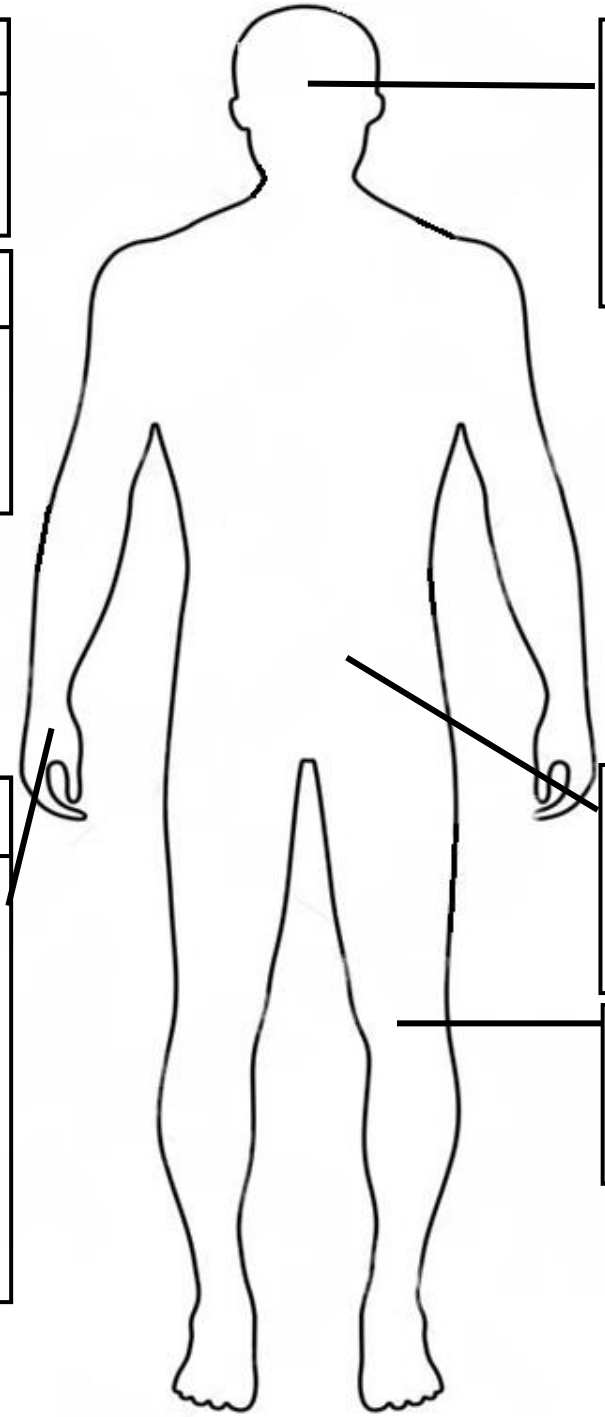
Look for:
- Pallor (MAHA)
- Jaundice (MAHA)

Vital Signs **2**

- Heart Rate
- Blood pressure
- Respiratory rate
- Temperature

Hands and Upper Limbs **3**

- Petechia/ bruising
- Splinter hemorrhages
- (remember that thrombocytopenia is present in several rheumatological diseases like SLE, RA, and vasculitis)



Head and Neck **4**

- Eyes: look for conjunctival pallor, or jaundice (MAHA)
- Gum bleed, epistaxis

Abdomen **5**

Look for splenomegaly

Lower Limbs **6**

- Bruising and petechiae.

Multiple Myeloma

❖ Revision of some basics

I. Multiple myeloma is a neoplastic proliferation of a single plasma line that produces monoclonal immunoglobulin. This leads to enormous copies of one specific immunoglobulin (usually of the IgG or IgA type).

II. Incidence is increased after the age 50.

III. As the disease process advances, bone marrow elements are replaced by malignant plasma cells. Therefore, anemia, leukopenia, and thrombocytopenia may be present in advanced disease.

IV. Clinical features *(the following are the most important, but there are others)*

- Skeletal manifestations
  - **Bone pain** with hypercalcemia – due to osteolytic lesions, fractures, and vertebral collapse. Occurs especially in the low back or chest (ribs) and jaw (mandible).
  - Pathologic fractures
  - Loss of height secondary to collapse of vertebrae.
- **Anemia** (normocytic normochromic) – due to BM infiltration and renal failure.
- **Renal failure** (myeloma kidney)
- **Recurrent infections**
- **Cord compression** (rare but may occur secondary to a plasmacytoma or fractured bone fragment).

Quick HIT

The signs of multiple myeloma can be remembered using the mnemonic CRAB for **C**alcium (hypercalcemia), **R**enal failure, **A**nemia, and **B**one lesions (lytic)

Quick HIT

Low hemoglobin, high calcium, high serum protein, and poor renal function suggest multiple myeloma.

V. Investigations

BLOOD

- **Serum protein electrophoresis (SPEP) and immunofixation.** *(to look for monoclonal spike and identify the type of the monoclonal Ig).*
- **CBC** (leukopenia, thrombocytopenia, and anemia may be present especially in advanced disease) and **peripheral blood smear** (RBCs are in Rouleaux formation)
- **Serum Calcium** (hypercalcemia)
- Serum albumin, serum total protein.
- Serum Creatinine
- Serum free light chain
- ESR

URINE

- **Urine protein electrophoresis and immunofixation.**
- **Assess for the presence Bence Jones protein.**

BONE

- **Bone marrow biopsy**
- **Bone imaging (X-ray to detect lytic lesions, MRI for spinal cord compression)**

Again, IF multiple myeloma comes as a history station, the question will probably NOT be direct, so the first step would be to know that the case is multiple myeloma (you would know from the presenting symptoms or the lab results in the question, and so on).

Follow the general outline for history taking but focus on the clinical features of multiple myeloma (ask about bone pain with its SOCRATES, symptoms of anemia and fatigue, symptoms of chronic renal failure [see nephrology part], recurrent infections, neurological symptoms).

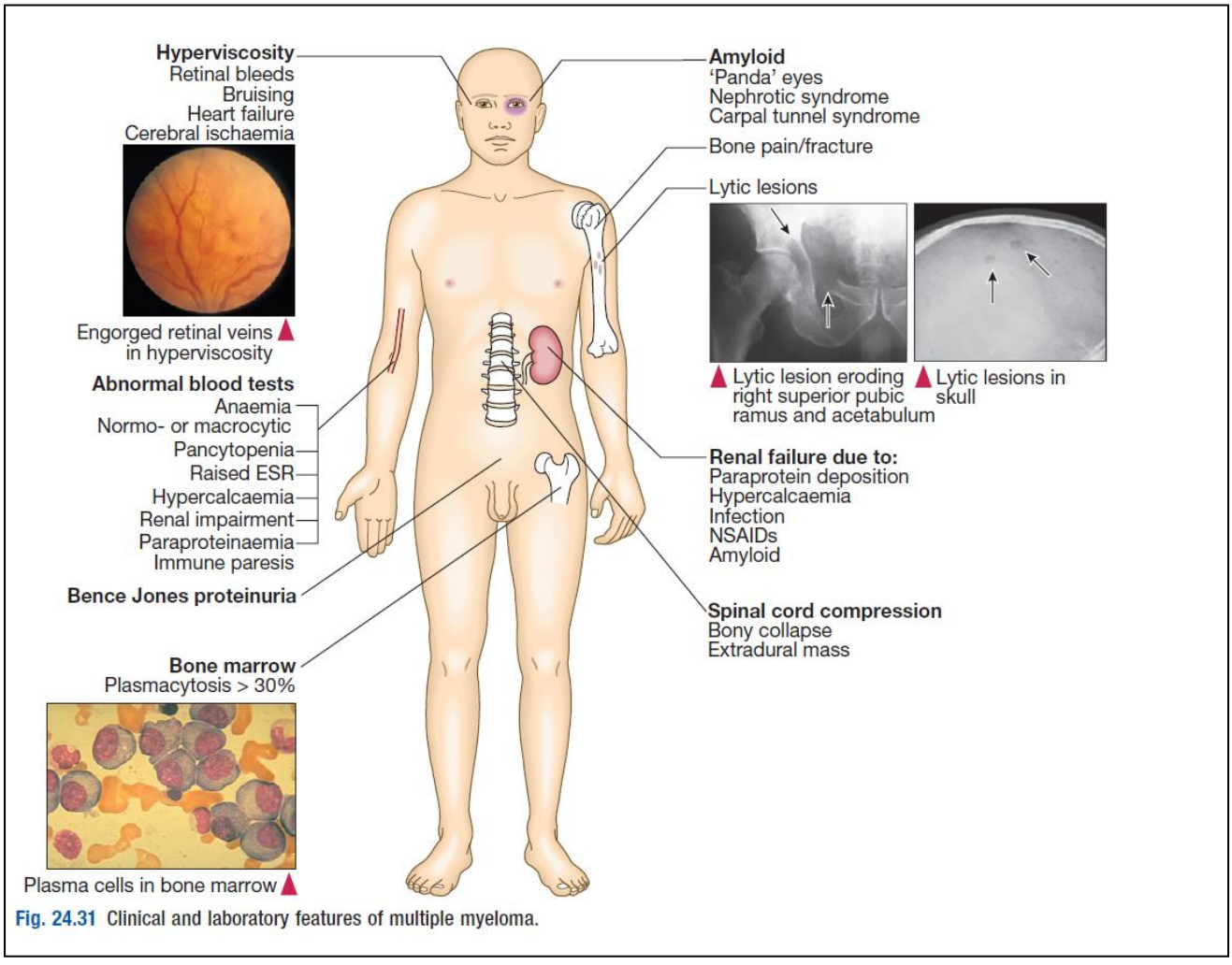
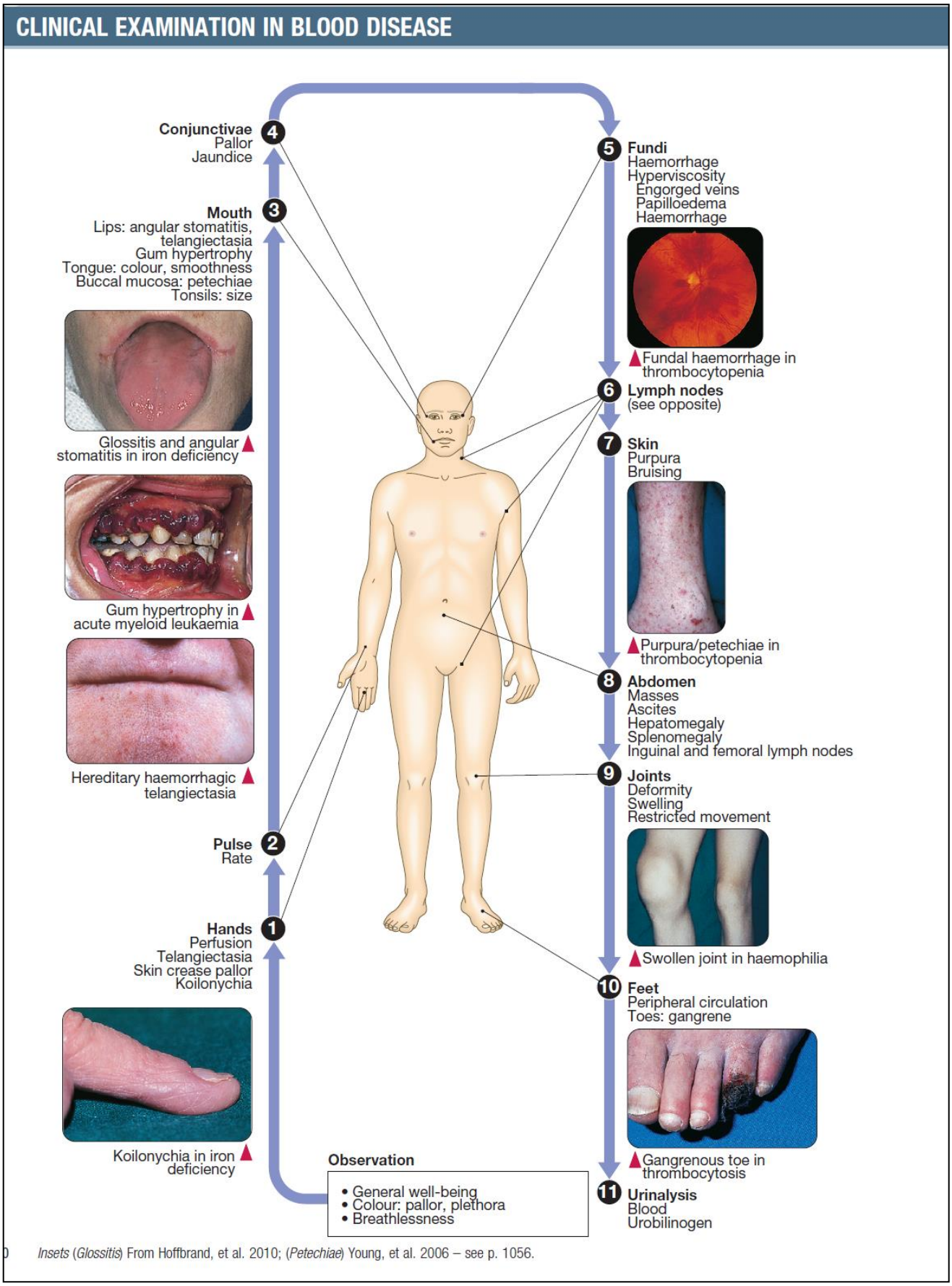


Fig. 24.31 Clinical and laboratory features of multiple myeloma.

Three important final notes:

- Please revise **lymph nodes examination** *(from chapter 3 of Macleod's)* and **examination of hepatomegaly and splenomegaly** *(from Macleod's or the GI section of the dossier),* as these two topics are very very very important in hematology and other systems. They're also considered high yield topics in the OSCE.
- The following 4 figures are useful so please study them :).
- I really hope you found this section enjoyable, and I apologize for any mistake I may have made. Wish you all best of luck :).

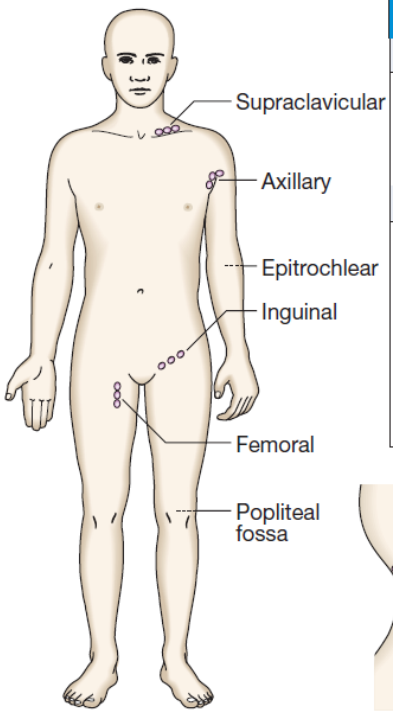
Some Useful Images

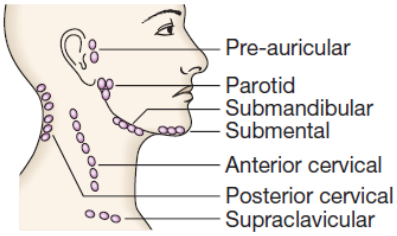


Abnormalities detected in the blood are caused not only by primary diseases of the blood and lymphoreticular systems, but also by diseases affecting other systems of the body. The clinical assessment of patients with haematological abnormalities must include a general history and examination, as well as a search for symptoms and signs of abnormalities of red cells, white cells, platelets, haemostatic systems, lymph nodes and lymphoreticular tissues.

6 Lymphadenopathy

Lymphadenopathy can be caused by benign or malignant disease. The clinical points to clarify are shown in the box.





Lymphadenopathy

History

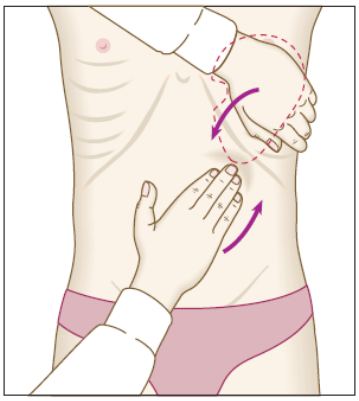
- Speed of onset, rate of enlargement
- Painful or painless
- Associated symptoms: weight loss, night sweats, itch

Examination

- Sites: localised, generalised
- Size (cm)
- Character: hard, soft, rubbery
- Fixed, mobile
- Search area that node drains for abnormalities (e.g. dental abscess)
- Other general examination (e.g. joints, rashes, finger clubbing)

8 Examination of the spleen

- Move hand up from right iliac fossa, towards left upper quadrant on expiration.
- Keep hand still and ask patient to take a deep breath through the mouth to feel spleen edge being displaced downwards.
- Place your left hand around patient's lower ribs and approach costal margin to pull spleen forwards.
- To help palpate small spleens, roll the patient on to the right side and examine as before.



Characteristics of the spleen

- Notch
- Superficial
- Dull to percussion
- Cannot get examining hand between ribs and spleen
- Moves well with respiration

Anaemia

Symptoms and signs help to indicate the clinical severity of anaemia. A full history and examination is needed to identify the underlying cause.

Anaemia

Non-specific symptoms

- Tiredness
- Lightheadedness
- Breathlessness
- Development/worsening of ischaemic symptoms, e.g. angina or claudication

Non-specific signs

- Mucous membrane pallor
- Tachypnoea
- Raised jugular venous pressure
- Tachycardia
- Flow murmurs
- Ankle oedema
- Postural hypotension

Bleeding

Bleeding can be due to congenital or acquired abnormalities in the clotting system. History and examination help to clarify the severity and underlying cause of the bleeding.

Bleeding

History

- Site of bleed
- Duration of bleed
- Precipitating causes, including previous surgery or trauma
- Family history
- Drug history
- Age at presentation
- Other medical conditions, e.g. liver disease

Examination

There are two main patterns of bleeding:

1. **Mucosal bleeding**
   Reduced number or function of platelets (e.g. bone marrow failure or aspirin) or von Willebrand factor (e.g. von Willebrand disease)
   Skin: petechiae, bruises
   Gum and mucous membrane bleeding
   Fundal haemorrhage
   Post-surgical bleeding
2. **Coagulation factor deficiency** (e.g. haemophilia or warfarin)
   Bleeding into joints (haemarthrosis) or muscles
   Bleeding into soft tissues
   Retroperitoneal haemorrhage
   Intracranial haemorrhage
   Post-surgical bleeding

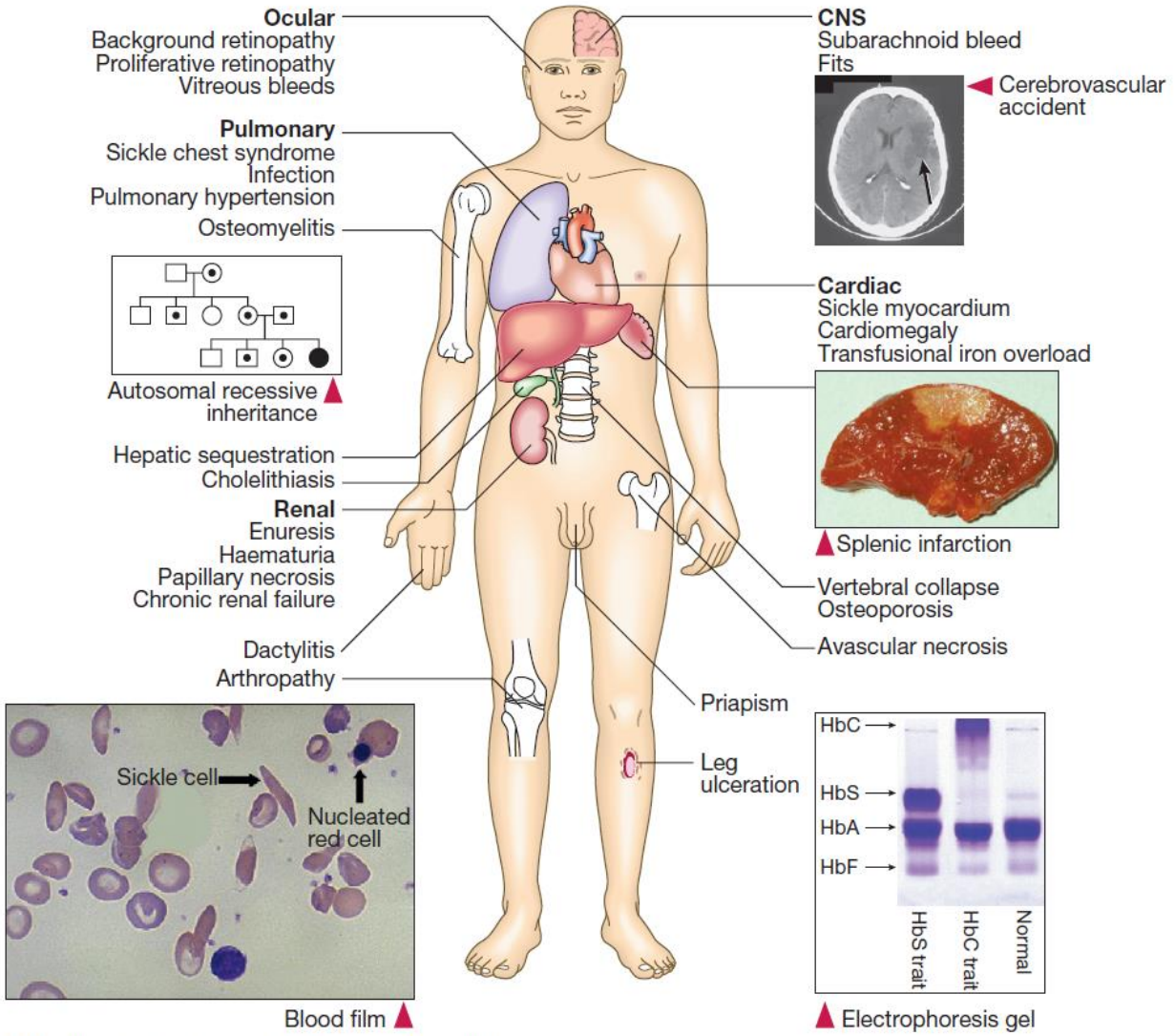
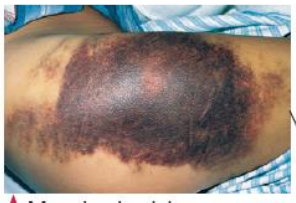
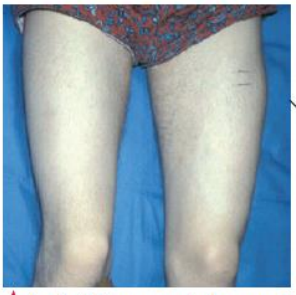


Fig. 24.24 Clinical and laboratory features of sickle-cell disease.

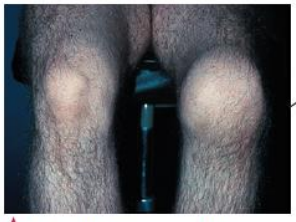


Massive bruising

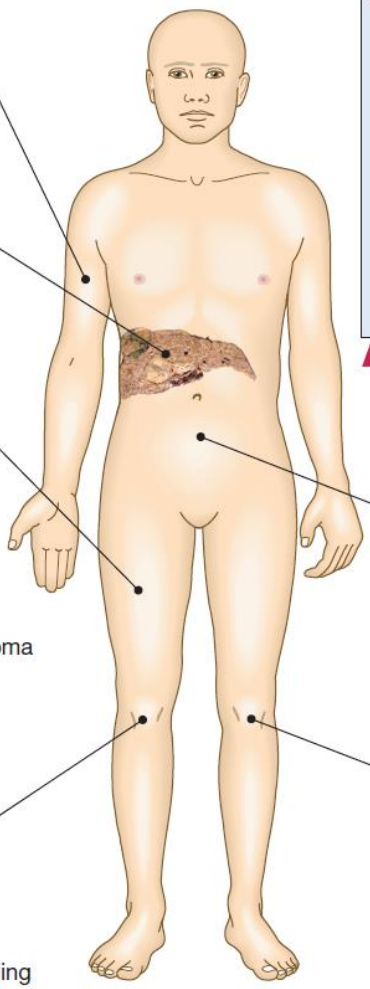
Hepatoma in cirrhotic liver secondary to HCV infection contracted from coagulation factor concentrate

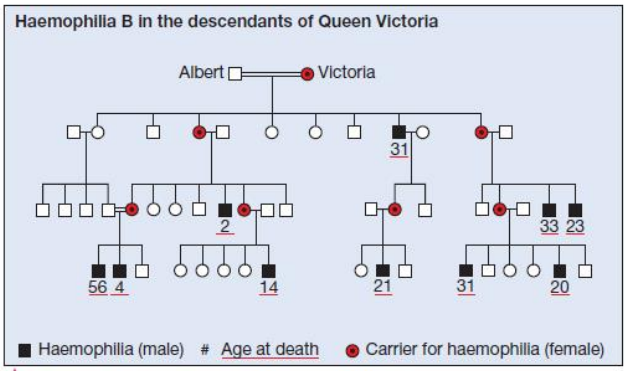


Left thigh muscle haematoma in severe haemophilia

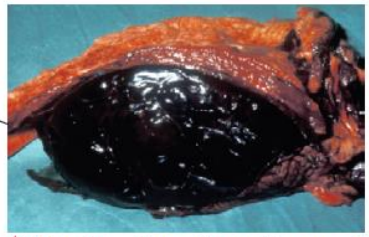


Chronic haemophilic arthropathy with joint swelling and muscle wasting on left

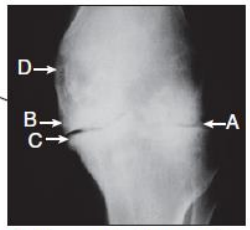




X-linked inheritance of haemophilia B



Massive retroperitoneal haemorrhage



X-ray of advanced haemophilic arthropathy

Fig. 24.32 Clinical manifestations of haemophilia. On the knee X-ray, repeated bleeds have led to broadening of the femoral epicondyles, and there is no cartilage present, as evidenced by the close proximity of the femur and tibia (A); sclerosis (B), osteophyte (C) and bony cysts (D) are present. (HCV = hepatitis C virus). *Inset (Massive bruising)* From Hoffbrand 2000 – see p. 1056.

Past Paper

(These are just the available ones (from the past two or three years [2016-2018] only))

* Take history form a patient "with symptoms of anemia" (+ hints like CBC) to know the diagnosis.

* Take history from a patient "with symptoms of anemia" (+ hints like CBC) to know the diagnosis. What is the diagnosis?

  The answer was one of the hemolytic anemias.

* "A female patient presented to you complaining of fatigue, lightheadedness, and shortness of breath since ---. Her Hemoglobin level was 8, MCV = 70….."

  1. Take brief history to know the cause of the symptoms
  2. What is the diagnosis?
     The answer was "Iron deficiency anemia, due to heavy menses" (after excluding other causes while taking the history).

* "A man presented to you with fatigue, dyspnea, and disorientation. His hemoglobin level = 5.4 and MCV =119, *and probably some other hints*."

  1. What is the diagnosis? *It was vitamin B12 deficiency*
  2. Examine him specifically for the type of anemia he has. *Don't forget neurological exam*.

* Examine for hepatosplenomegaly.

Chapter 6: Nephrology

History Taking

Hematuria

Case scenario: A 40-year old lady presented with red colored urine. Take a focused history of her complaint.

Start by introducing yourself and asking about patient profile (age, gender,..)

HPI:

Characterize the hematuria

- Take the duration and frequency, was this the first time?
- Establish the color of the urine and character of blood (fresh vs clots), clots are more typical of glomerular disease while fresh of trauma or infection
- At what point during urination is the blood seen? (initial-urethra, end-bladder neck or prostate , throughout – bladder or kidney.
- Associated with pain? If yes, ask SOCRATES. Site: Flank (renal stones), Loin (Loin pain-hematuria), Suprapubic (cystitis)
- Apperance of the urine? Frothy? Indicates GN
- Exacerbated by cold or exercise?
- Constitutional symptoms (fever, chills, weight loss,fatigue,nightsweats) TB, Malignancy
- Other urinary symptoms (dysuria, frequency, urgency)
- Specific symptoms: CVS (heart failure), rheumatological (joint pain) (SLE),hearing loss (Alport), bleeding tendecy and bruising
- Recent trauma(rhabdomyolysis), infection (URTI, skin, pharyingitis), or menustration.
- Strenous exercise or ingestion of food causing pseudohematuria (blackberries, beetroot).

Meds: NSAIDs, Warfarin, Heparin, Aspirin, Penicillin,Rifampicin, Rifabutin, Clofazamine, Entacapone

PMHx: HTN, kidney stones, heart disease, SLE, bleeding disorders

FHx: alport disease, HTN, malignancy, adult polycystic kidney disease

SHx: smoking, occupational exposure to rubber, dyes

9.8 Causes of haematuria

Painless	
• Glomerulonephritis • Tumours of the kidney, ureter, bladder or prostate* • Tuberculosis* • Schistosomiasis* • Hypertensive nephrosclerosis • Interstitial nephritis (unless very acute/severe)	• Acute tubular necrosis • Renal ischaemia (renovascular disease) • Distance running or other severe exercise • Coagulation disorders, anticoagulant therapy
Associated with pain	
• Urinary tract infection • Renal stones with obstruction	• Loin pain-haematuria syndrome
May be either	
• Urinary tract infection • Reflux nephropathy and	• Adult polycystic kidney disease

9.7 Abnormalities of urine colour

Orange-brown	
• Conjugated bilirubin • Rhubarb, senna • Concentrated normal urine, e.g. very low fluid intake • Drugs: sulfasalazine	
Red-brown	
• Blood, myoglobin, free haemoglobin, porphyrins • Beetroot, blackberries	• Drugs: rifampicin, rifabutin, clofazimine, entacapone
Brown-black	
• Conjugated bilirubin • Drugs: L-dopa, metronidazole, nitrofurantoin, chloroquine, primaquine	• Homogentisic acid (in alkaptonuria or ochronosis)
Blue-green	
• Drugs/dyes, e.g. propofol, fluorescein, triamterene	

Notes:

- Make sure to differentiate between hematuria and contamination by menstrual blood or urine color abnormalities
- If new onset of painless hematuria in adults > 40 y, suspect malignancy
- Causes of hematuria not listed in the table: SLE, Alport syndrome, heart failure.

Urinary incontinence

Case scenario: A 60-year old man presented with difficulty controlling his urine. Take a focused history regarding the possible causes of his complaint.

Notes:

There are different types of urinary incontinence:

- Urge incontinence (preceded by an sudden strong need to urinate)
- Stress incontinence (caused by an increase in the intra-abdominal pressure due to laughing, coughing, sneezing, exercise)
- Mixed incontinence
- Continual incontinence (caused by a fistula between the bladder/urethra and the vagina, a complication of obstetric surgery)

Start by introducing yourself and asking about patient profile (age, gender,…)

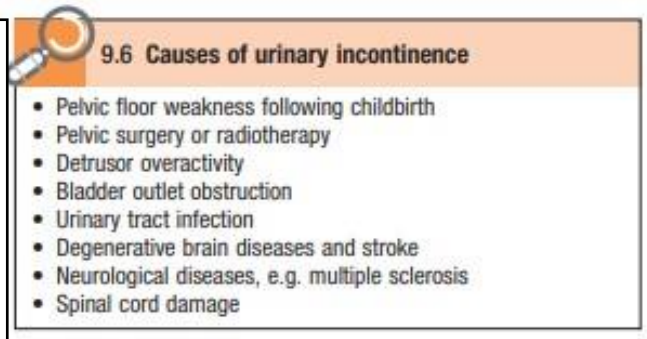
HPI:

- Ask about onset and frequency
- Does it occur during sleep?
- Number of pads used and if they are wet or fully soaked
- Provocative factors? Laughing, sneezing, coughing, exercise
- Constitutional symptoms: fever, chills,.. (UTI)
- Other urinary symptoms: dysuria, frequency, urgency (UTI), slow flow , hesitancy, terminal dribbling (BPH)

PMHx: Stroke, MS, spinal cord damage

PSHx: Pelvic surgery or radiation

POHx: Previous pregnancies, mode of delivery, obstetric surgeries



9.6 Causes of urinary incontinence

- Pelvic floor weakness following childbirth
- Pelvic surgery or radiotherapy
- Detrusor overactivity
- Bladder outlet obstruction
- Urinary tract infection
- Degenerative brain diseases and stroke
- Neurological diseases, e.g. multiple sclerosis
- Spinal cord damage

Nephrotic Syndrome

Case scenario: A 6-year old boy presented with facial puffiness. Urine dipstick showed +3 protein, take a focused history on the possible causes of his condition. What is your diagnosis?

- Classical features:

1. Nephrotic range proteinuria >3.5 g/day
2. Hypoalbuminemia
3. Hyperlipidemia
4. Edema

Other features: hypertension, recurrent infections and hypercoagulable state

- UA:
  Inspection: frothy urine
  Dipstick: +3 protein
  Microscopy: fatty/waxy casts

Primary

1. **Minimal Change Disease**
-Commonly seen in children
-Either idiopathic or secondary to Hodgkin's lymphoma or drugs (NSAIDs, Lithium)
-Not associated with HTN
2. **Focal Segmental Glomerulosclerosis**
-Either idiopathic or secondary to diseases (sickle cell disease, lymphoma, HIV, or other GN) or drugs (heroin, lithium)
3. **Membranous Glomerulonephritis**
-Most commonly seen in adults
-Can be idiopathic **OR**
-Secondary to:
1. Drugs (gold, penicillamine)
2. Solid Malignancies (lung, breast, colon, prostate)
3. Infections (hepatitis B/C, malaria, streptococcal, schistosomiasis)
4. Autoimmune diseases (SLE, RA, sjogren, sarcoidosis)

Secondary

1. **Diabetic nephropathy**
-Occurs in both type 1 and 2

2. **Amyloidosis**
-Causes:
1. Familial Mediterranean Fever (FMF)
Symptoms:
Paroxysmal attacks of fever, abdominal pain, chest pain.
*Ask if the patient takes colchicine to prevent amyloidosis.
2. Multiple Myeloma
Symptoms:
CRAB
C: Hypercalcemia (nausea, vomiting, abdominal pain, constipation…. Etc)
R: Renal failure
A: Anemia (dizziness, SOB on exertion, fatigue)
B: Bone lesions (bone pain)

Nephritic Syndrome

Case scenario: A 24 year old lady complaining of fatigue and malaise, her CR level is 2, previously normal. Urinalysis showed +3 protein, +2 blood, RBC casts. Take a history of the possible causes of her condition. What is your diagnosis?

Presents with:
1. Hematuria
2. Variable proteinuria usually < 2g/day
3. Hypertension
4. Oliguria
5. Edema
6. Azotemia

UA:
Dipstick: + blood
Microscopy: dysmorphic RBC, RBC casts

Types of GN –Nephritic Syndrome:

Primary					***Secondary***		
Postinfectious GN	**Membrano-prolefrative GN**	**IgA Nephropathy (Berger's disease)**	**Alport Syndrome**	**ANCA+ renal limited GN**	**Good pasture Syndrome (anti-GBM disease)**	**Vasculitis**	**Lupus Nephritis**
-Mostly seen in children -Preceded by streptococcal infections (URTI 1-2 weeks) **OR** (Skin infection 6 weeks)	-It is both nephritic and nephrotic -Hepatitis C is the main cause	-Develops during URTI	Presents with kidney disease, hearing loss, and eye abnormalities	-Disease is limited to kidneys alone -Causes rapidly progressive GN	-Affects both lungs and kidneys. -lung symptoms usually precede kidney symptoms e.g. cough , hemoptysis , SOB	- Wegener's granulomatosis (sinusitis, epistaxis, SOB) -Churg-Strauss syndrome (ask about asthma, atopy) -Henoch Schonlein Purpura (rash on extensor surfaces of legs, arthritis, abdominal pain)	Ask about the 11 criteria of SLE, patient should have at least **4**

**Other causes: endocarditis and cryglobulinemia (secondary), rapidly progressive GN(primary)

Acute Kidney Injury

Case scenario: A 37 year old lady presented to the ER with 48-hour diarrhea and vomiting. She can't remember passing urine today. Her labs showed CR level of 1.9, it was previously normal. What is your diagnosis? What are the diagnostic criteria? Take a detailed history of other possible causes of her condition.

Kidney Disease: Improving Global Outcomes (KDIGO) defines AKI as any one of the following

1- Increase in serum creatinine by 0.3 mg/dL or more from baseline within 48 hours **OR**
2- Increase in serum creatinine to 1.5 times baseline within one week **OR**
3- Urine output less than 0.5 mL/kg/h for 6 hours.

Causes of AKI		
Pre-renal (caused by underperfusion of the kidneys, either from true loss of volume or decreased effective arterial blood flow)	**Renal**(caused by a dysfunction in any part of the kidney, either the glomeruli, tubules, interstitium or vasculature)	**Post-renal**(caused by obstruction to the urine flow)
1.Hypovolemia Causes: a. Dehydration which could be due to poor fluid intake, working in hot conditions, vomiting*, diarrhea*, polyuria, burns, third spacing (e.g. ascites) or excessive diuretic use *Symptoms of gastroenteritis b. Blood loss	1.Acute tubular necrosis Caused by either ischemia or a nephrotoxin a. Ischemic ATN can be due to shock or any condition that causes sudden hypoperfusion b. Nephrotoxic ATN causes include: free myoglobin (rhabdomyolysis) , free hemoglobin (intravascular hemolysis), contrast, antibiotics (e.g. aminoglycosides,vancomycin) Antifungal (e.g. amphotericin B) chemotherapeutic agents (e.g. cisplastin) , NSAIDs	1.Urethral obstruction It commonly occurs due to benign prostatic hyperplasia, symptoms are: hesitancy, slow flow, incomplete emptying ,frequency, , terminal dribbling, and nocturia Other causes: kidney stones, tumors, vaginal or uterine prolapse
2.Hypotension Causes: a. Sepsis b. Hypovolemia d. Excessive use of antihypertensive medications	2. Glomerulonephritis Symptoms include frothy urine, red colored urine, swelling	2. Bilateral ureteral obstruction or unilateral obstruction of a solitary functioning kidney Ask about symptoms of kidney stones or family history of renal tubular acidosis type 1 which predisposes to formation of calcium kidney stones Other causes: retroperitoneal fibrosis

3.Decreased cardiac output Occurs in CHF.	3. Acute interstitial disease It is usually a result of an allergic rxn to a drug e.g. antibiotics, NSAIDs, or PPI Symptoms include: fever, skin rash, swelling Less commonly it can be due to an infection e.g. pyelonephritis	3. Medications which crystalize in the urine: 1.Methotroxate 2.Ganciclovir 3.Acyclovir
4.Vascular disease limiting renal blood flow (embolus) Does not present with any specific symptom. It usually causes elevated blood pressure that is poorly controlled with drugs	4. Vascular disease a. Renal artery/vein thrombosis b. Thrombotic thrombocytopenic purpura (TTP). Symptoms include: neurological (e.g. confusion, seizures) , bruising, pallor and fatigue c. Hemolytic-uremic syndrome (HUS), it is the most common cause of AKI in children	
5.Medications which impair renal autoregulation a.NSAIDs b.ACE-I/ARB c.Cyclosporin		
6.Hepatorenal syndrome Ask about causes of cirrhosis and symptoms of portal hypertension e.g. abdominal distention, vomiting blood, black tarry stool, bleeding tendency, infections, dizziness, SOB on exertion		

****Exclude pre-renal and post-renal causes before moving to renal causes of AKI***

**** How to ask about these causes in history? NEXT PAGE…*

Causes of AKI – HISTORY

Pre-renal

Past medical history	Drug histroy
Gastroenteritis N & V , diarrhea	diuretics
GI bleeding	NSAIDS
CHF	ACEI/ARBs
pancreatitis	cyclosporine
renal art. stenosis	
Liver cirrhosis	

Post renal

Past medical history	Drug histroy
Kidney stones; Flank pain & urinary symptoms	Acyclovir
BPH; Nocturia, overflow incontinence	MTX
Urogental prolapse	
RTA	
Malignancy; wt loss, anorexia	

Renal

ATN		AIN	GN
ischemia	nephrotoxins	Fever + rash, SLE	Flu like sx with hematurea , frothy urine
Sepsis	Exogenous : contrast material, laxatives, antifungal		
Blood loss	Endogenous: myoglobin, ask about BURN		

Chronic Kidney Disease

Case scenario: A 72 year old man referred to you after routine monitoring of his renal function demonstrated steadily increasing CR levels. Renal ultrasound showed small kidneys and his BP is 150/92. Take a focused history regarding the possible causes of his condition.

CKD is defined as any of the following:

1. Kidney damage for > 3 months, with or without decrease in GFR, with either pathological abnormalities or markers of kidney damage.

2. GFR < 60 mL/min/1.73m^2 for >3 months, with or without kidney damage.

Stages of CKD:

Stage	Description	GFR
CKD1	Kidney damage with normal or high GFR	=>90
CKD2	Kidney damage with mild decrease in GFR	60-89
CKD3	Moderate decrease in GFR	30-59
CKD4	Severe decrease in GFR	15-30
CKD5	End-stage kidney disease (dialysis requiring)	<15

Most common causes of CKD:

1. Diabetes

2. Hypertension

3. Chronic GN

4. Vascular diseases e.g. renal artery stenosis, renal vein thrombosis

4. Unrecovered AKI

5. Polycystic kidney disease (consanguineous family)

6. Congenital defects in the kidney or bladder

7. Recurrent kidney stone formation

6. Long-term use of drugs that affect the kidneys e.g. NSAIDs

Clinical presentation:

Patients with CKD stages 1 – 3 are frequently asymptomatic, symptoms generally manifest in stages 4 and 5.

1. Manifestation related to decreased erythropoietin production: Anemia (fatigue, dizziness, SOB on exertion, inability to concentrate)

2. Manifestations related to uremia:

Neurological	Cardiac	Gastrointestinal	Hematological	Cutaneous	Other
• Altered mental status , confusion • Lethargy • Coma • Peripheral neuropathy • Restless leg syndrome • Asterixis	• Pericarditis ,can be complicated by cardiac tamponade • HTN	• Metallic taste • Uremic fetor • Anorexia, weight loss • Nausea , vomiting • Diarrhea	• Thrombocytopenia (bruising, tendency to bleed)	• Fluid retention • Pruritus • Nail atrophy	• Fatigue, failure to thrive • Malnutrition • Increased thirst • Decreased libido, erectile dysfunction, amenorrhea

3. Manifestations related to abnormalities in calcium and phosphorus homeostasis:

1. Ostetis fibrosa cystica (bone pain, fractures)

2. Adynamic bone (increased risk of fractures)

3. Osteomalacia (bone pain, fractures, proximal myopathy)

How to differentiate between AKI and CKD?

Tests	AKI	CKD
PTH	normal	high
Ca++ level	normal	Low
EPO level	Normal/slightly low	Markedly low
Evidence of normocytic normochromic anemia	No	Yes
Presence of cell casts in UA	Yes	No or very few

Hypertension

Case scenario: A 26 year old lady, diagnosed with HTN 8 months ago, is complaining of poorly controlled hypertension despite her compliance to treatment. Take a focused history on the possible causes of her condition.

Classification of blood pressure ranges (2017):

- Normal =< 120/<80
- Prehypertension 120-139/80-89
- HTN, Stage 1: 140-159/90-99
- HTN, Stage 2: >160/>100

Primary HTN

Risk factors:
1. Age : >55 y for males, >65 y for females
2. Gender: males>females
3. Race: African Americans
4. Obesity: BMI =>30
6. Alcohol
7. Smoking
8. Dyslipidemia
9. Salt intake
10. Diabetes
11. Heart disease
12. CKD
13. Family history
14. Stress
15. Sedentary lifestyle

Secondary HTN

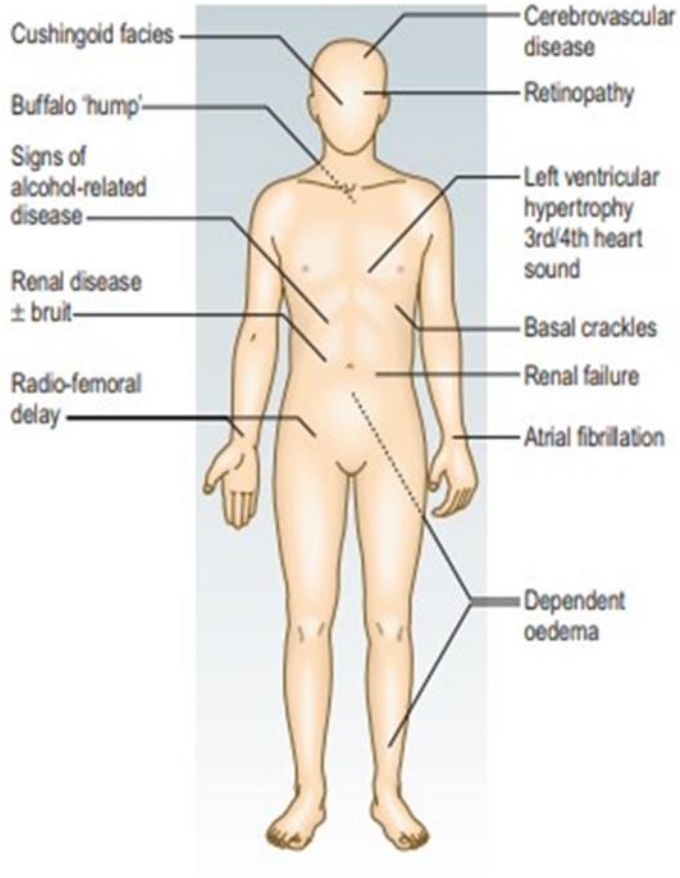
When to suspect secondary HTN?
1. If new onset of HTN at a young age <30 y or at an old age >50 y
2. If poorly controlled with drugs (>3-4 drugs)
3. If there are clinical or lab features specific for a disease e.g. episodic HTN/flushing/palpitations, low TSH, hypokalemia

Causes:
1. Renal diseases: adult polycystic kidney disease, renal artery stenosis, glomerular disease.
2. Endocrine diseases: cushing syndrome, conn's syndrome, pheochromocytoma, hyper/ hypothyroisidm, hyperparathyroidism
3. Coarctation of the aorta
4. Obstructive sleep apnea
5. Pregnancy
6. Medications: OCP, NSAIDS, TCA, cyclosporine, steroids.

Few hints to help you ask about these possible causes are found in the next page.

Causes of secondary HTN that you can ask about it:
Cushing's syndrome: ask about weight gain, striae (تشققات بالجلد), proximal myopathy, having a moon face.
Pheochromocytoma: palpitations, headaches, dizziness, flushing.
Hyperthyroidism: heat intolerance, weight loss, tremor, excessive sweating.
Hypothyroidism: cold intolerance, weight gain, hair loss.
OSA: snoring, restless sleep, increased daytime sleepiness.
Ask about anxiety or recurrent panic attacks (حالات ذعر من غير سبب)

Physical signs related to secondary causes and complications of HTN



Urine analysis

General principles:

- Clean catch (midstream urine sample)
- < 4 hours old

Sequence of urinalysis
Inspection of urine sample
Assessment of color and clarity: o Normal fresh urine is clear but varies in color o Cloudy urine indicates presence of leukocytes (bacterial infection) o Abnormalities of urine color are mentioned in table 9.7
Dipstick test
Mentioned in the table
Microscopy
o Bacteria: distinguish infecting organism from amorphous material, use gram stain and culture to identify organism $>10^5$ CFU/mL of urine (UTI) o Cells: dysmorphic RBCs (glomerular disease), monomorphic RBCs (renal stones or UTI), WBCs (UTI) o Casts: RBC casts (glomerular disease), WBC casts (pyelonephritis, interstitial nephritis), Muddy brown (epithelial) casts (ATN) o Crystals: rarely diagnostic. Hexagonal cysteine crystals (cystinuria)

9.16 Urine dipstick test*

Investigation	Comment
Specific gravity	Reflects urine solute concentration. Varies between 1.002 and 1.035; ↑ when kidneys actively reabsorb water, e.g. fluid depletion or renal failure due to ↓ perfusion. Abnormally low values indicate failure to concentrate urine
pH	Normally 4.5–8.0. In renal tubular acidosis pH never falls <5.3 despite acidaemia
Glucose	Small amounts may be excreted by normal kidneys
Ketones	Test is specific for acetoacetate and does not detect other ketones, e.g. β-OH butyrate, acetone. Ketonuria occurs in diabetic ketoacidosis, starvation, alcohol use and very-low-carbohydrate diets
Protein	Readings > 'trace' (300 mg/L) indicate significant proteinuria. Proteinuria >2 g/day suggests glomerular disease
Blood	The test does not differentiate between haemoglobin and myoglobin. If you suspect rhabdomyolysis, measure myoglobin with specific laboratory test
Bilirubin and urobilinogen	Bilirubin not normally present. Urobilinogen may be up to 33 µmol/L in health. Abnormalities of bilirubin and urobilinogen require investigation for possible haemolysis or hepatobiliary disease
Leukocyte esterase	Indicates presence of leukocytes in urine. Seen in urinary tract infection or inflammation, stone disease and urothelial cancers
Nitrite	Most Gram-ve bacteria convert urinary nitrate to nitrite. A positive result indicates bacteriuria, but a negative result does not exclude its presence

*Use freshly passed urine (Fig. 9.12).

9.9 Causes of proteinuria

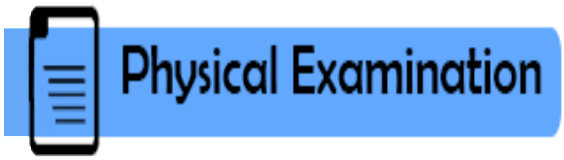
Renal disease

- Glomerulonephritis
- Diabetes mellitus
- Amyloidosis
- Systemic lupus erythematosus
- Drugs, e.g. gold, penicillamine
- Malignancy, e.g. myeloma
- Infection

Non-renal disease

- Fever
- Severe exertion
- Severe hypertension
- Burns
- Heart failure
- Orthostatic proteinuria*

*Occurs when a patient is upright but not lying down; the first morning sample will not show proteinuria.



General:

- **Assess the general appearance and consciousness level**

Look for fatigue, pallor, breathlessness, uremic (lemon-yellow) complexion, cushingoid appearance and hirsutism (related to steroid therapy and cyclosporine respectively)

- **Vital signs**

Measure temperature and blood pressure and check for pulsus paradoxus (do not use the arm with the AV fistula) (usually elevated except in tubulointerstitial disease and uremic pericarditis)

- **Head**

1. Look at the eyes for conjuctival pallor of anemia and across the cornea for band keratopathy or at the edge for limbic calcifications (related to hypercalcemia)
2. Smell the patient's breath for uremic fetor

- **Hands**

1. Inspect for nail changes: half and half nails, Leukonychia, Muehrecke's nails, Beau's lines, Splinter hemorrhage
2. Examine for asterixis
3. Inspect for AV fistula
4. Examine for Carpal Tunnel Syndrome: Phalen's and Tinel's test

Abdominal Examination:

Position: supine with one pillow

Exposure: Nipple to mid-thigh, for social concerns from xiphisternum to symphysis pubis

- **Inspection**

1. Look for distention (from enlarged kidneys in APKD or rarely obstructive uropathy) , or suprapubically from bladder distention
2. Look in the loin for scars of renal tract surgeries or in the iliac fossa for those of transplant surgery. You may see a peritoneal catheter or small scars left in the midline and hypochondrium by one.

- **Palpation**

1. Use the fingers of your right hand and start and the right lower quadrant

- A distended bladder is felt as a smooth firm mass arising from the pelvis which disappears with urethral catheterization
- Polycystic kidneys have a distinctive nodular surface

2. For lesser degrees of kidney enlargement, place one hand behind the patient's back below the ribs, and one hand anteriorly over the upper quadrant and gently but firmly push your hands together as the patient breathes in and out
3. Ask the patient to sit up and palpate for renal angle tenderness (pyelonephritis)

- **Percussion**

Percussion of kidneys is not helpful

- **Auscultation**

Auscultate for renal artery bruit over both loins posteriorly and in the epigastrium

- **Test for Ascites**

- **In men, examine external genitalia and do PR** to assess the prostate for benign and malignant causes
- **In women, perform vaginal examination** to exclude pelvis malignancy and to assess prolapse

Cardiovascular examination:

- **Look for pitting edema in the ankle, sacrum and the back of the thighs in recumbent patient**
- **Asses the JVP**

High JVP in cardiac tamponade due to uremic pericarditis and low JVP in nephrotic syndrome although edema is present

- **Palpate the apex beat**

Displaced in fluid overload or heart failure

- **Auscultate for:**

1. Mid-systolic flow murmur
2. $3^{rd}/4^{th}$ heart sounds
3. Pericardial friction rubs

Respiratory examination:

- **Percuss the chest to detect pleural effusion**
- **Auscultate for bilateral lung crackles indicating fluid overload or heart failure.**

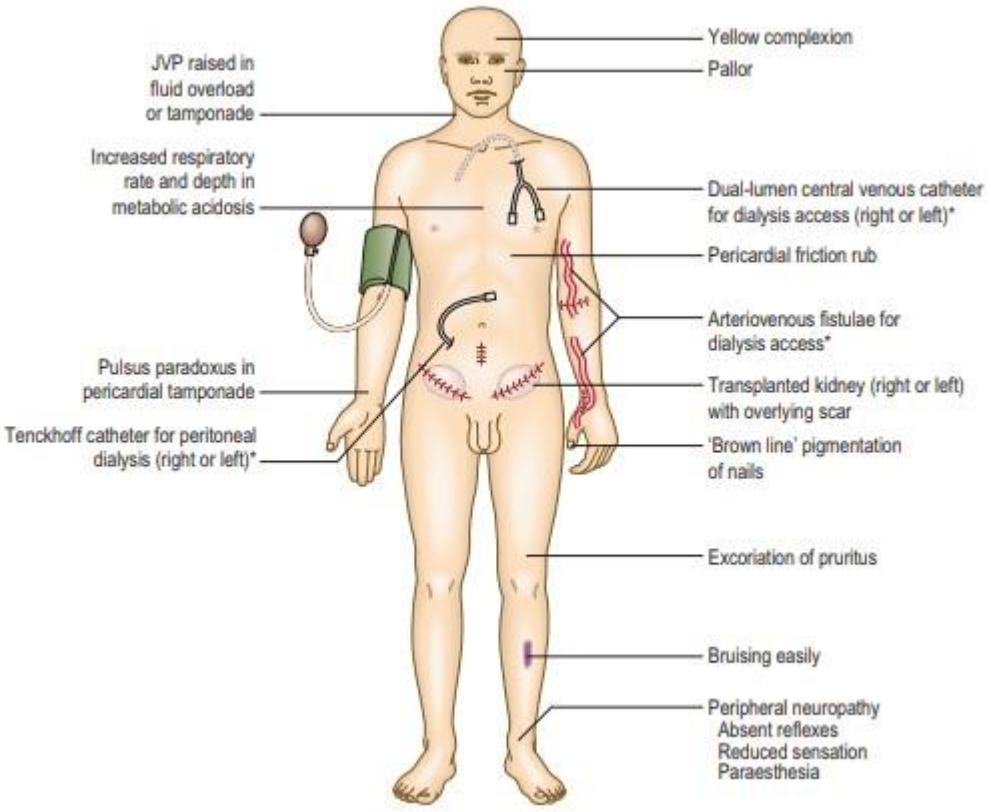
Neurological examination:

- **Assess the level of consciousness**
- **Test sensation and tendon reflexes.**

Peripheral neuropathy occurs in advanced CKD

- **Examine the optic fundi**

Retinopathy is found in DM and HTN



References:

1. Macloed clinical examination 13th edition
2. Medstudy 16th edition
3. Medscape
4. ACA/AHA High Blood Pressure Guidelines – American College of Cardiology
5. OSCE dossier
6. Hashem Ahmad, Sharifeh Masaid and Amer Abu Shanab notes

Chapter 7: Rheumatology

History Taking

RA

INTRODUCTION

- RA is a chronic inflammatory disease results from immune-complex mediated vasculitis (type III hypersensitivity), it affects almost any organ system:
- ➢ The commonest presentation is symmetrical polyarticular joint pain.
- ➢ It affects females more than males.

OSCE Vignette: A 45 years old female presented to your clinic complaining of stiffness in her shoulders, hands, knees, and hips. The stiffness lasted for about an hour every morning. After her wrists and fingers began to swell she decided to visit your clinic, take a focused history looking for the symptoms of RA.

- **Patient profile:** Sex (F>M) & Age (40-60),
- **Chief complaint:** Symmetrical polyarticular (large & small joint pain) for X duration
- **History of presenting illness:**

➢ Ask about articular symptoms:

1. Joint pain (SOCRATES), usually symmetrical, affecting the hands (except DIP), elbows, knees, ankle & feet.
2. Morning stiffness (for 1 hour)
3. Gel phenomenon (stiffness after long period of rest).
4. Locking, weakness, swelling
5. Joint hotness, redness, swelling (usually absent).
6. Neck fractures

➢ Ask about extraarticular symptoms:

1. Neuropathy (weakness, numbness & paralysis). “mononeuritis multiplex”
2. Dry eyes, dry mouth. “Sjogren's /Sicca Syndrome”
3. Eye problems (redness, burning, blurry vision) “uveitis, episcleritis”
4. Chest pain, pericardial pain, SOB, cough, wheeze, hemoptysis “pericarditis. Pericardial effusion, plural effusion, pulmonary fibrosis”

5. Raynaud's phenomenon, skin rash.
6. Signs of anemia.

- Ask about the constitutional symptoms:
  1. Low grade fever.
  2. Fatigue.
  3. Anorexia.
  4. Wight loss.
  5. Depression.
  6. Restriction of normal life activities.

- **Previous medical & surgical Hx**: DM, HTN, IHD, strokes.
- **Drugs Hx**: Steroids, ACEIs, Antiepileptics.
- **Family history of:** RA & other autoimmune disease.
- **Social history:**
  - Smoking, alcohol, drug misuse.
- **Work-up:**
  - RF, ESR, CRP.
  - Normocytic normochromic anemia.
  - Radiography: loss of juxta articular bone, narrowing of joint space, bony erosions at the margin of the joint, synovial fluid analysis.

Proximal Muscle Weakness

OSCE Vignette:

A 47-year-old woman presents with a 2 month history of progressive weakness in her thighs and upper arms. On examination, proximal muscle strength is symmetrically reduced but distal muscle strength is normal. Ask the patient relevant questions to distinguish between polymyositis and dermatomyositis.

Both polymyositis and dermatomyositis cause proximal muscle weakness. The difference is that polymyositis does not involve skin, whereas dermatomyositis is associated with characteristic skin rash.

- History is directed at:
  1- Common features between these two conditions.
  2- The characteristic skin rashes associated with dermatomyositis.
  3- Asking relevant questions to help exclude other differential diagnoses.

PM	DM (presents with PM features and dermatological features)
Commonly present with insidious, progressive and **symmetrical proximal muscle weakness** (weeks–months). The patient particularly experiences difficulties in walking up stairs or rising from a chair.	**Gottron's papules**: scaly, erythematous eruptions particularly over the extensor surfaces of the MCP, PIP and DIP joints (*Fig. 2.13.2*). Macular erythema (without scaly eruption) can occur in other extensor surfaces e.g. of the elbows and knees, known as **Gottron's sign** (*Fig. 2.13.3*).
Muscle pain (approximately 1/3).	**Heliotrope rash**: violet discoloration of the eyelids, occasionally accompanied by periorbital oedema (*Fig. 2.13.4*).
Systemic features: fever, fatigue, and weight loss (due to oesophageal dysmotility).	**Photosensitivity**
Aspiration pneumonia, dysphagia, dysphonia and **respiratory failure** (if there is involvement of the respiratory and pharyngeal muscles).	**Nail-fold erythema**
Pulmonary fibrosis (30%)	

History Points: (Try to memorize them in your way and order, so as to remember them in the exam).

- **General questions about the complaint and common features of PM and DM:**

1- Make sure that it's a chronic muscle weakness (acute muscle weakness is more likely to be stroke).

2- Ask about muscle weakness:

a) Do you have: difficulty getting out of bed (neck muscles weakness), difficulty combing your hair or getting out of a chair unaided (proximal muscle weakness of arms and thighs, respectively)?

b) Make sure the patient does not have features suggestive of distal muscle weakness. Do you have problems with writing or holding things with you hands?

3- Ask about common features of both PM and DM.

a) Myalgia (all of patients have weakness but only 1/3 have pain)

b) Dysphagia (remember the upper third of the esophagus is formed of skeletal muscles).

- **Questions related to the characteristic signs of dermatomyositis:**
  1- Do you have any skin rash?
  If yes, ask him about the specific skin rashes.
  a) **Heliotrope rash (butterfly)**—around eyes, bridge of nose, cheeks.
  b) **Gottron papules**—papular, erythematous, scaly lesions over the knuckles (MCP, PIP, DIP).
  c) **V sign**—rash on the face, neck, and anterior chest.
  d) **Shawl sign**—rash on shoulders and upper back, elbows, and knees.
  e) Periungual erythema with telangiectasia.
  f) Subcutaneous calcifications in children—can be extremely painful.
- Always remember that **dermatomyositis is associated with malignancy**. Once you diagnose dermatomyositis, you have to exclude an occult malignancy in lung, breast, ovary, GI tract, and myeloproliferative neoplasms).

OSCE tips: Malignancy in DM

DM may occur 2° to a malignancy:
- Ask about **non-specific features of malignancy** e.g. weight loss and malaise
- Perform **systems review**
- Consider performing **whole body CT**, **GI tract imaging** and **mammography**

- **Drug history:**

- Ask about steroid and statins (they both cause myalgia and proximal muscle weakness).

- **Ask about any significant family history.**

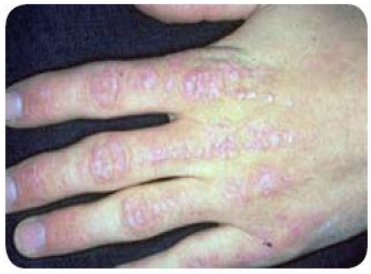


Fig. 2.13.2: Gottron's papules.

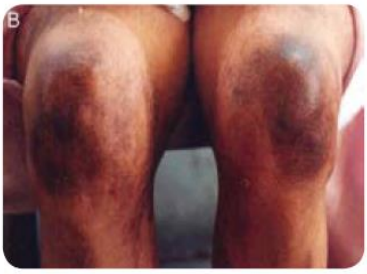


Fig. 2.13.3: Gottron's sign.

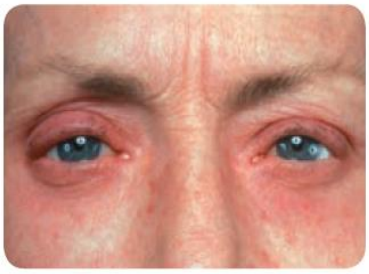


Fig. 2.13.4: Heliotrope rash.

Systemic lupus Erythematosis (SLE)

Clinical features of this disease involve mostly every system thus you should ask about these clinical features by systems so that you do not miss any:
NOTE: things that are underlined and bolded are of the 11 criteria used to diagnose SLE.

- *The plan*
  - ***I.*** *Ask about the clinical features of the disease itself.*
  - ***II.*** *Ask about the relation of the symptoms to triggers.*
  - ***III.*** *Ask about past medical and family history of autoimmune diseases.*

I. Ask about the clinical features of the disease itself:

1- Constitutional symptoms: Fatigue (often the sign of an impending exacerbation and a prominent finding in most patients), malaise, fever, weight loss.
2- Cutaneous: **Butterfly rash** (erythematous rash over cheeks and bridge of nose),**photosensitivity, discoid lesions** (erythematous raised patches with keratotic scaling), **oral or nasopharyngeal ulcers**, alopecia, Raynaud phenomenon.
3- Musculoskeletal: Joint pain (may be the first symptom of the disease—found in 90% of patients), **arthritis** (inflammatory and symmetric, not erosive as in rheumatoid arthritis [RA]), arthralgias, myalgia with or without myositis.

Pericarditis or pleuritis are considered as one entity in the 11 criteria under the name serositis

4- Cardiac: **Pericarditis**, endocarditis (Libman–Sacks endocarditis is a serious but rare complication), Myocarditis. Ask about chest pain,SOB,palpitations…
5- Pulmonary: **Pleuritis** (most common pulmonary finding), pleural effusion, pneumonitis(may lead to fibrosis), pulmonary HTN (rare). Ask about chest pain, SOB, cough, sputum.
6- Hematologic: **Hemolytic anemia with anemia or reticulocytosis** of chronic disease (ask whether the patient complains of SOB, pallor, headache, lightheadedness, dizziness upon standing up.),**leukopenia, lymphopenia** (recurrent infections that has been noticed recently), **thrombocytopenia** (mucosal bleeding).
7- Renal: **Proteinuria >0.5 g/day** -ask about frothy urine- ,**cellular casts**, glomerulonephritis(ask about hematuria, edema), azotemia, pyuria, uremia -refer to nephrology chapter for symptoms of uremia-, HTN.
8- Immunologic: often associated with antiphospholipid syndrome (ask about recurrent DVT, arterial thrombosis and recurrent abortions).
9- GI: Nausea/vomiting, dyspepsia, dysphagia, peptic ulcer disease.
10- CNS: **Seizures, psychosis** (may be subtle), depression, headaches, TIA, cerebrovascular accident
11- Other findings include conjunctivitis and Sjögren syndrome -ask about dry mouth and eyes-

The rest of the criteria are based on investigations and labs, and these are:

1- Immunologic manifestations -positive LE preparation, false-positive test result for syphilis, anti-ds DNA, anti-Sm Ab.
2- ANAs

II. Ask about the relation of the symptoms to triggers:

- Are the symptoms worse after pregnancy?
- Are the symptoms related to the use of some drugs, namely; procainamide, hydralazine, isoniazid, chlorpromazine, methyldopa and quinidine?

III. Ask about past medical and family history of autoimmune diseases.

Seronegative Spondarthritis

➢ *Quick review:*

This term is applied to a group of inflammatory joint diseases distinct from RA that share a number of clinical features. These are:

1- Ankylosing spondylitis (AS)
2- Peripheral SPA
3- Psoriatic arthritis
4- IBD associated (AKA entropathic)
5- Reactive arthritis
6- Juvenile onset

➢ *Plan:*

I. Ask about the symptoms common in these diseases.
II. Ask about specific symptoms in each to reach the diagnosis.

I. Ask about the symptoms common in these diseases:

- Enthesitis: ask about any episodes of swelling in Achilles tendon or heel.
- Axial arthritis: may be scaroillitis or spondylitis that present as low back pain that have the following features:
  - → Insidious onset
  - → Ascending pattern; that may eventually involve the whole spine.
  - → Pain improves with exercise and NSAID
  - → Pain increases with rest thus it comes usually at night

  Not to get lost, simply ask SOCRATES and with the examiner answering you can reach the diagnosis.
- Asymmetrical peripheral arthritis, usually affecting the large joints.
- Extra articular symptoms:
  - → Mucosal: conjunctivitis, buccal ulceration, urethritis, prostatitis, bowel ulceration.
  - → Pustular skin lesions and nail dystrophy.
  - → Anterior uveitis.
  - → Erythema nodosum.

II. Ask about specific symptoms in each disease to reach the diagnosis:

1- Ankylosing spondylitis:

- → CVS involvement thus ask about chest pain, SOB, palpitations…
- → Lung fibrosis: SOB, dry cough…
- → Due to the involvement of the costovertebral joints patients complain of rigid chest wall and pleuritic chest pain.

2- Reactive arthritis:

- → This type comes following urogenital or gastrointestinal infection (1-3 weeks) and characterized with triad of: urethritis, conjunctivitis, arthritis that is additive in its nature. There may also be skin lesions.

3- Entropathic:

- → This type is associated with IBD, so ask about the symptoms of IBD *(you can find these in GI chapter*). It is also important to know that the peripheral arthritis correlates with the activity of the diseases while the axial does not.

4- Psoriatic:

- → Usually the cutaneous psoriasis precedes arthritis but the opposite can occur.
- → Ask the patient whether he/she has psoriatic lesions (*which are well-defined erythematous scaly plaques, particularly affecting the extensor surfaces and scalp.)* or not.
- → Psoriasis means الصدفية.

Approach to joint pain

History Taking

It is important to establish the pattern of joint pain from the following points; so you reach to a diagnosis more easily:

- *Articular vs non-articular (joint itself or surrounding structures such as tendons)*
- *Inflammatory vs non-inflammatory (e.g. rheumatoid vs osteoarthritis)*
- *Acute vs chronic*
- *Peripheral vs axial*
- *Additive vs migratory (joints improve and new ones become involved) vs palindromic (recurrences and relapses)*
- *Inflammatory monoarthritis vs oligoarthritis vs polyarthritis*

Joint pain is a pain! So:

I. Simply start with SOCRATES!

1- <u>Site:</u>

→ Firstly, make sure that the pain is joint pain (i.e. arthralgia) not pain originating from other structures.

One joint is a monoarthritis.
2–4 joints oligoarthritis.
>4 is polyarthritis

→ Secondly, ask the patient about the total number of the joints causing the pain so that you know is it mono/oligo/polyarthritis as this will guide you to the differential.

→ Notice whether the involved joints are mainly small joints, large. Peripheral vs axial. Is it symmetrical involvement or asymmetrical.

2- <u>Onset:</u>
Is it chronic and gradual OR acute (which goes more with septic, gouty arthritis or traumatic)

3- **<u>Character</u>**.

4- **<u>Radiation:</u>** radiation of pain is most likely a sign of nerve involvement.

5- <u>Associated symptoms:</u>

I. Morning stiffness: Ask about the presence of early-morning stiffness and for <u>how long stiffness lasts</u>: is it for few minutes →osteoarthritis , less than 30 minutes →inflammatory arthritis, around 1 hour→RA.
II. Redness and hotness: may be seen in septic joint or inflammatory arthritis.
III. Swelling: can be inflammatory, septic or may be seen in hemarthrosis.

6- **<u>Timing</u>** (frequency, duration and periodicity of symptoms)

→ A history of several weeks of pain, early-morning stiffness and loss of function is likely to be an inflammatory arthritis.

→ 'Flitting' pain starting in one joint and moving to others over a period of days is a feature of rheumatic fever and gonococcal arthritis.

→ If intermittent with resolution between episodes, it is likely to be palindromic rheumatism.

7- <u>Exacerbating and relieving factors:</u>

→ Is it relieved by moving the joint→ inflammatory?

→ Is it exacerbated by moving the joint→ of non-inflammatory (most likely osteoarthritis)?

→ Pain is there upon movement and rest→ septic arthritis is a possible cause.

8- <u>Severity.</u>

Then ask about:

II. Ask about loss of function.
III. Deformities.
IV. History of trauma or injection into a joint.
V. Ask about history of same complaint.
VI. Drugs: NSAID and thiazides (both associated with gout).

→ Here is a table summarizing the pattern of joint pain with other symptoms according to the differential *-most common ones-*.

Rheumatological	Rheumatoid arthritis	•Slowly progressive symmetrical polyarthritis •Small joints (commonly of hand) •Deforming •Early morning stiffness
	Gout	•First MTP joint most commonly affected •Isolated swollen, hot, painful joint •Hyperuricaemia risk factors e.g. diuretics, alcohol excess (esp beer), renal disease
	Psoriatic arthritis	•Associated skin plaques and nail changes •Early morning stiffness •Many patterns of joint involvement
	SLE	•Systemically illness with intermittent fevers •Photosensitive rash •Generalised myalgia and arthralgia •Other systemic Sx (e.g. psych disturbance, pleurisy, ulcers)
	Enteropathic arthritis	•Symmetrical arthritis of lower limb joints and SI joints •Early morning stiffness •Symptoms/diagnosis of Crohn's or UC
Orthopaedic	Osteoarthritis	•Elderly •Worse on movement (rest helps) and at end of day
	Septic arthritis	•Isolated hot, red, swollen joint •Agonizingly painful •Systemically unwell with fever

→ Look at the following table , just be familiar with important causes of joint pain

14.2 Differential diagnosis of monoarthritis, oligoarthritis and polyarthritis

	Type	Examples
Monoarthritis (single joint involvement)	Infective	*Staphylococcus aureus*, *Staphylococcus epidermidis*, *Salmonella*, tuberculosis, *Neisseria gonorrhoeae*, *Escherichia coli*, *Haemophilus*
	Traumatic	Haemarthrosis
	Bleeding diathesis	Acute exacerbation of underlying state
	Post-traumatic	
	Degenerative	Osteoarthritis, Charcot joint
	Metabolic	Crystal arthropathies: gout, pseudogout
	Inflammatory polyarthritis presenting as monoarthritis	Rheumatoid arthritis
Oligoarthritis (involvement of 2–4 joints)	Infective	Bacterial endocarditis, *Neisseria gonorrhoeae*, *Mycobacterium tuberculosis*
	Degenerative	Osteoarthritis
	Inflammatory oligoarthritis	Sarcoidosis, reactive arthritis, psoriatic arthritis, ankylosing spondylitis
	Inflammatory polyarthritis presenting as oligoarthritis	Rheumatoid arthritis
Polyarthritis (involvement of ≥5 joints)	Infective	Bacterial: Lyme disease, subacute bacterial endocarditis Viral: rubella, mumps, glandular fever, chickenpox, hepatitis B and C, human immunodeficiency virus (HIV)
	Post-infective	Rheumatic fever
	Degenerative	Osteoarthritis: nodal with Heberden's/Bouchard's nodes
	Metabolic	Haemochromatosis, gout
	Inflammatory	Rheumatoid arthritis, SLE, psoriatic arthritis
	Other	Hypertrophic pulmonary osteoarthropathy

Hand Examination

Physical Examination

Case: A 40 year-old lady has a history of joint pain for the last 2 months. Examine her hands for signs suggestive of the diagnosis.

-Greet your patient, Introduce yourself and ask for permission to perform the examination.

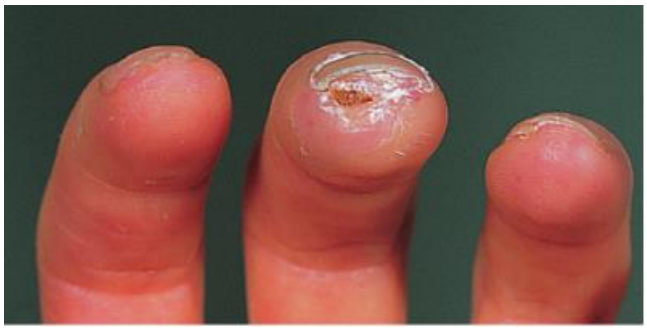
-Wash your hands and ensure adequate privacy, warmth and illumination.

-Position the pt. seated upright, expose both hands up to the elbows and rest the hands on a pillow.

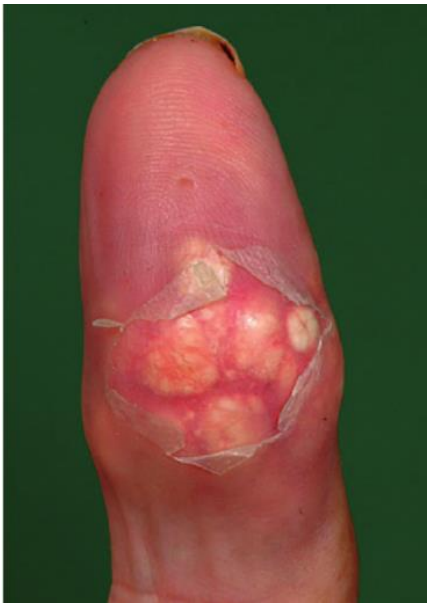
-Ask for any site of pain to avoid touching or moving it.

Look	• Look at the palmar aspect then at the dorsal aspect of both hands. • At the palmar aspect look for palmar erythema and visible muscle wasting of the thenar, hypothenar and small muscles of the hand. • Look for psoriatic nail changes, onycholysis and telangiectasias at nail folds. • Look for obvious deformities such as Arachnodactyly, Ulnar deviation, Anterior displacement of the wrist and Duputyren's contracture. • Examine the lateral side of the fingers for Mallet finger deformity, Boutonniere deformity, Swan Neck deformity and Z-thumb. • Look at the finger tips for calcinosis suggestive of systemic sclerosis. • Look at the interphalangeal joints for Gouty Tophi of Gouty arthritis. • Look for Bouchard's nodes (at PIPs) Heberden's nodes (at DIPs) and squaring of the wrist, all suggestive of Osteoarthritis. • Look for swelling at metacarpophalangeal (MCP) joints and interphalangeal (IP) joints. Ask the pt. to make a fist and look for hill-valley-hill-valley aspect of the knuckles which is lost in Rheumatoid Arthritis (RA). • Ask the pt. to flex the fingers pointing toward the scaphoid bone to look for Rotational deformity. • **Note**: if you detect any deformity, support the patient's hand against a hard surface and try to reverse it. If the deformity is reversible it's due to SLE (Jaccoud Arthropathy). If it's not reversible it's due to RA.
Feel	• Examine the dorsum of the patient's hand with the dorsum of yours to assess temperature. Compare bilateral hands and assess the temperature of swellings –if present-. • Feel the muscle bulk of the thenar and hypothenar eminences for muscle wasting. • Feel the consistency of swellings –if present- and determine if hard or soft.

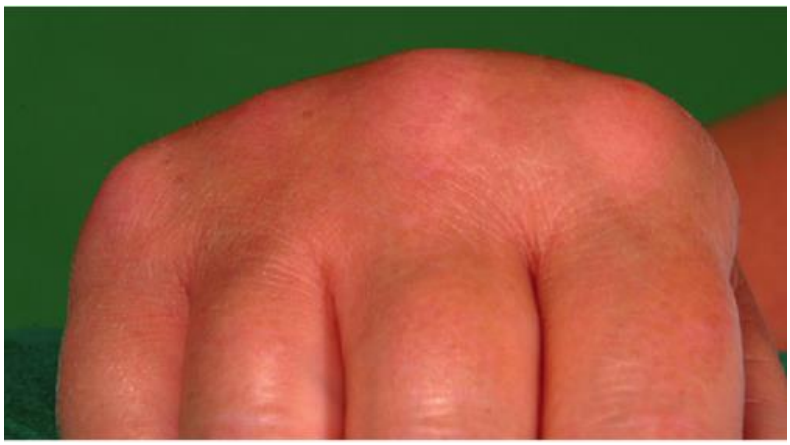
	• Squeeze with your thumb and index across each IP joint to detect sponginess and/or tenderness. • Squeeze with your thumb and index across each MCP joint to detect sponginess and/ or tenderness. Ask the pt to move his fingers while you do so as to detect crepitations. • Palpate the flexor tendon sheaths for swelling or tenderness. • Feel the extensor surface of the forearm for Rheumatoid Nodules.
Move	(Always start with active movement, then examine passively if the pt. can't move actively) • Ask the pt. to make a fist then fully extend his fingers. • Ask the pt. to perform Prayer's sign (to assess extension of the wrist) and Reverse Prayer's sign (to assess flexion of the wrist), Ulnar deviation (to assess adduction) and Radial deviation (to assess abduction). • Test the patient's grip by asking him to squeeze your index and middle finger and compare bilaterally. • To detect triggering, move one joint while fixating the other joints, one at a time.



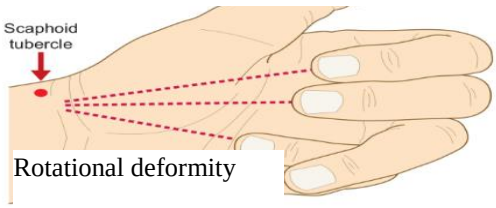
Calcinosis in systemic sclerosis



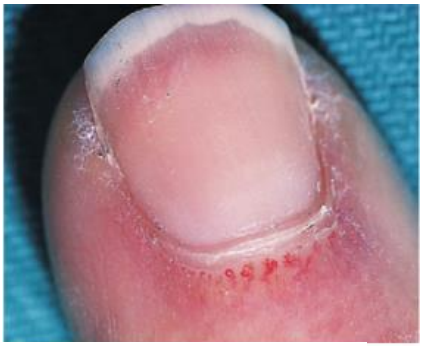
Gouty Tophus



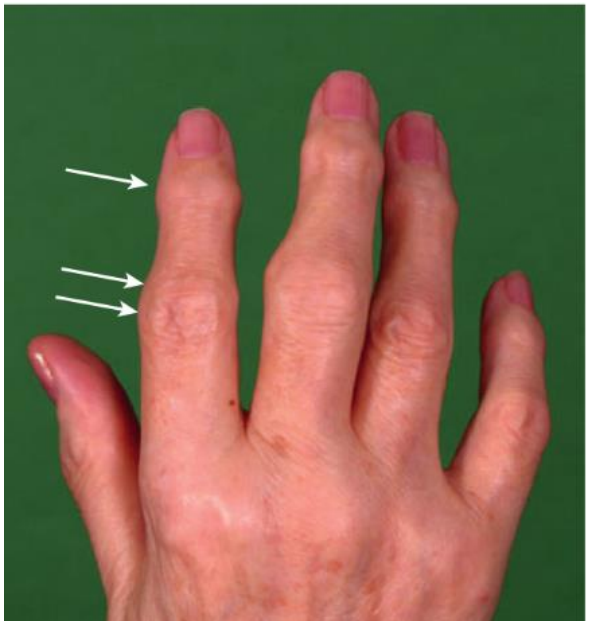
Loss of Hill-Valley-Hill-Valley aspect in RA

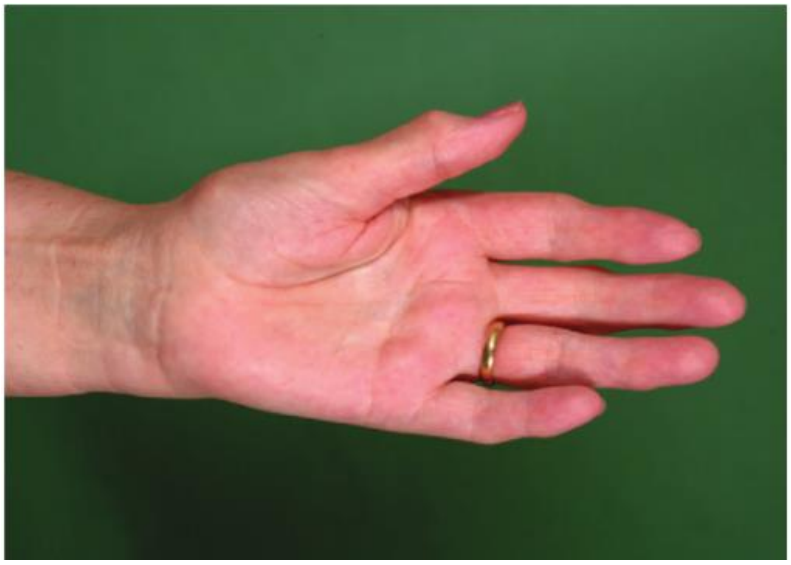


Rotational deformity

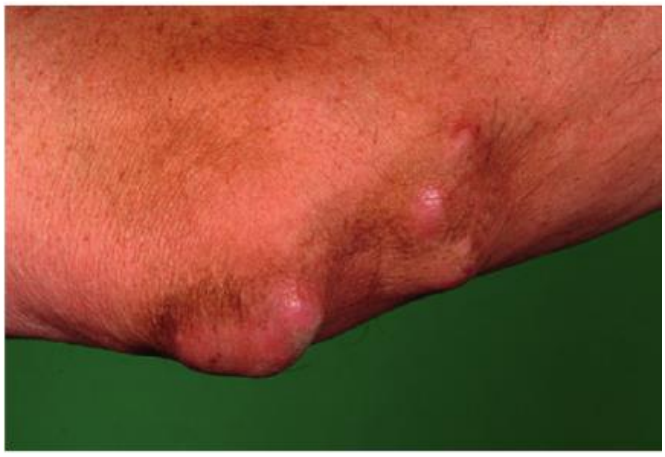


Telangiectasias at nail fold





Bouchard's nodes (at PIP joints), Heberden's node (at DIP joints) and Squaring of the wrist in OA



Rheumatoid nodules at extensor surfaces

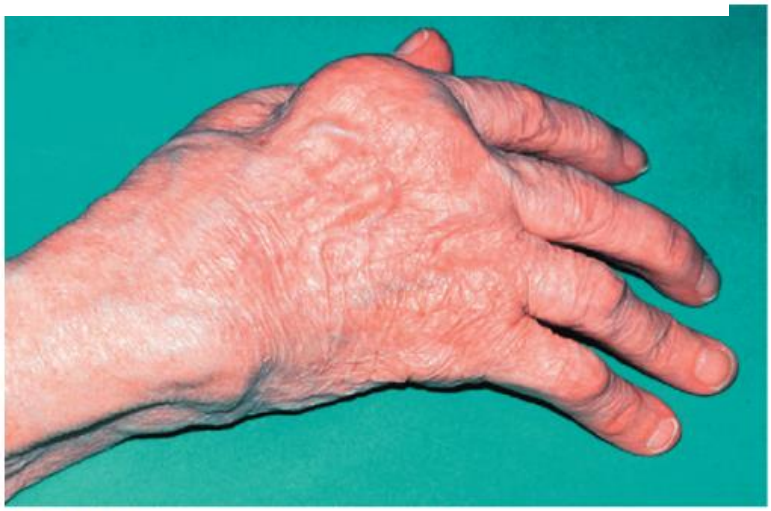
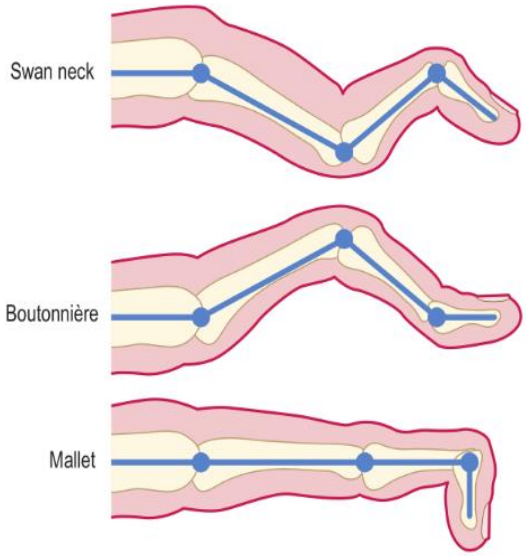
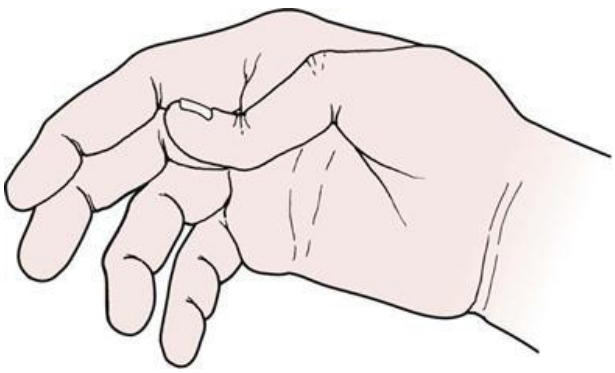


Fig. 14.34 Rheumatoid hand, showing ulnar deviation of the fingers, small-muscle wasting and synovial swelling at carpus, metacarpophalangeal and proximal interphalangeal joints.





Z thumb deformity in RA

Knee Examination

This subject is more likely to be in the orthopedic rotation not in the internal medicine OSCE. But it was included here just in case.

-Greet your patient, Introduce yourself and ask for permission to perform the examination.

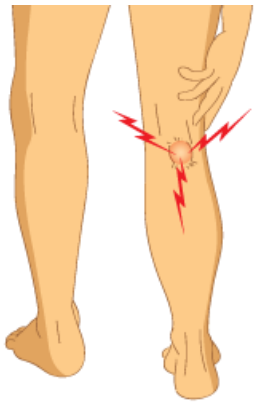
-Wash your hands and ensure adequate privacy, warmth and illumination.

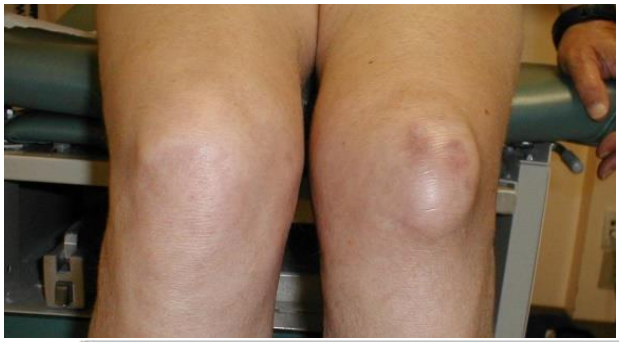
-Start your examination with the patient standing then in-supine position, expose both lower limbs up to iliac crests.

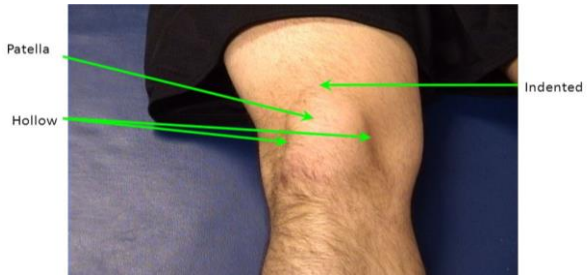
-Ask for any site of pain to avoid touching or moving it.

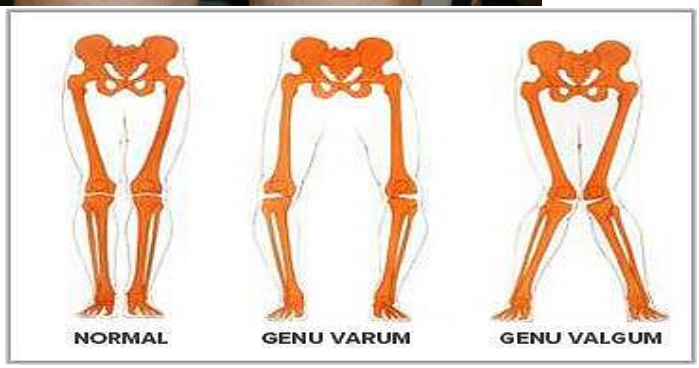
Look

- While the patient is standing examine his gait by asking him to walk a straight path head and forth.
- Inspect while the pt. is standing looking for Valgus or Varus deformity, Patellar hollow, obvious muscle wasting (Vastus medialis, calf muscles and quadriceps), Housemaid's knee and Baker's cyst.









- Ask the patient to lie in a supine position, stand to the right side of the pt. and comment on the position of the patella, redness, hotness, scars, sinuses, hair distribution, asymmetry, swelling and sutures.

Feel

- Feel the temperature of the joint with the dorsum of your hand and compare both limbs.
- Feel the patella, the femoral condyles, Joint lines, Head of fibula, the patellar tendon and the quadriceps tendon, the tibial tuberosity and comment on tenderness.
- Feel the posterior aspect of the knee and the hamstring muscles.

Move

- Start by examining the active range of motion then examine passively.
- Ask the pt. to flex his knee. Normal range of flexion is 0-140^{o}. Abnormal range of flexion is when the movement stops before the calf touches the thigh.
- Ask the pt. to extend his knee. Full extension is to 0 degrees.
- Listen for crepitus when the pt. flexes or extends the joint.

Special Tests

- McMurray's test (for medial and lateral menisci)
  Flex the knee to 90^{o}, feel the joint lines with your left hand (between the femoral condyles and the tibial head) and hold the foot with your right hand.

To examine the lateral meniscus, flex the knee then extend while internally rotating the foot and place the knee in a varus position (Figure A). A sensation of clunk on the lateral side of the joint is a positive test.
To examine the medial meniscus, flex the knee then extend while externally rotating the foot and place the knee in a valgus position (Figure B). A sensation of clunk on the medial side of the joint is a positive test.
Repeat the flexion-extension cycles a number of times, and reduce the degree of flexion each time.

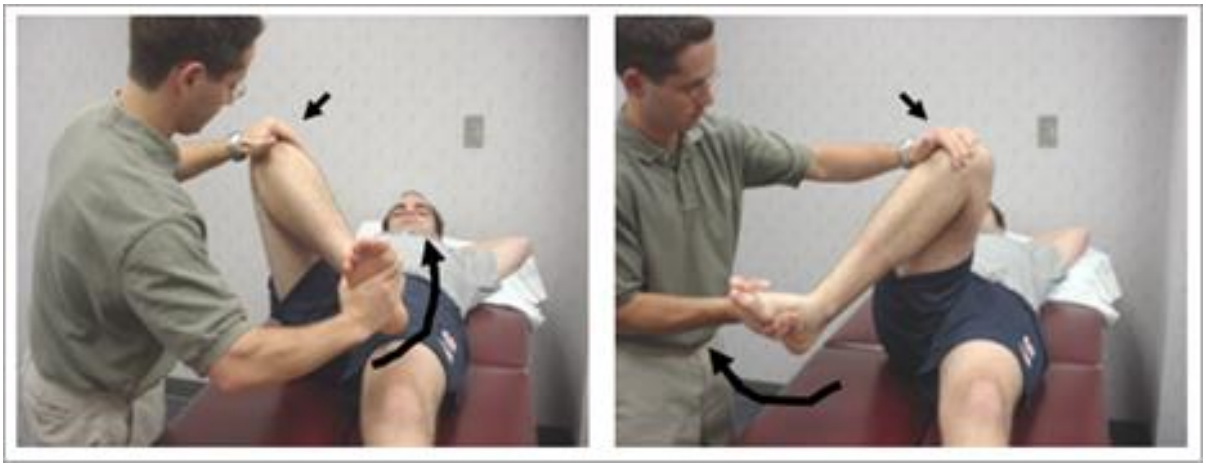


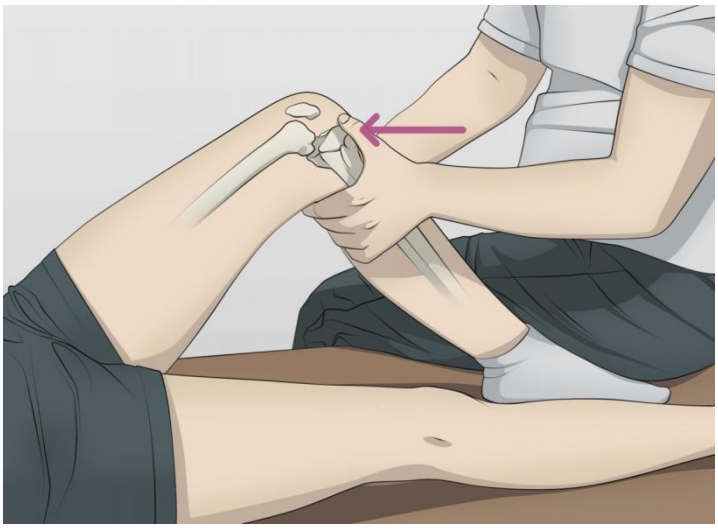
Figure A Figure B

- Medial and Lateral collateral test
  Extend the knee fully, hold the foot and place it between your elbow and waist. Place one hand on the lateral joint line and the other hand on the medial line.

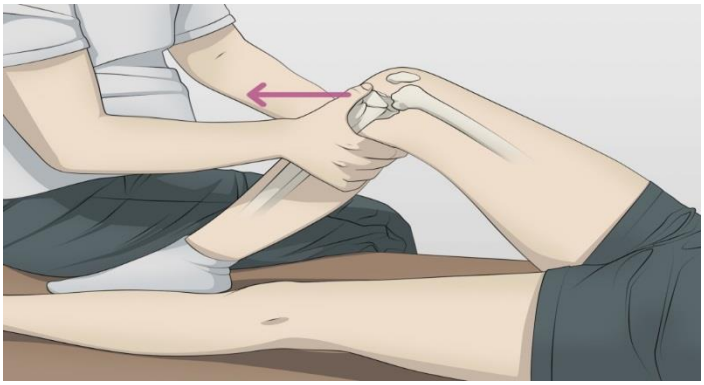
  To test the medial collateral ligament: Move your body to force a valgus stress and feel for an opening in the medial joint line. Repeat while flexing the knee to 30^{o}. If an opening could still be felt it indicates a defected medial collateral ligament.

  To test the lateral collateral ligament: Move your body to force a varus stress and feel for an opening in the lateral joint line. Repeat while flexing the knee to 30^{o}. If an opening could still be felt it indicates a defected lateral collateral ligament.

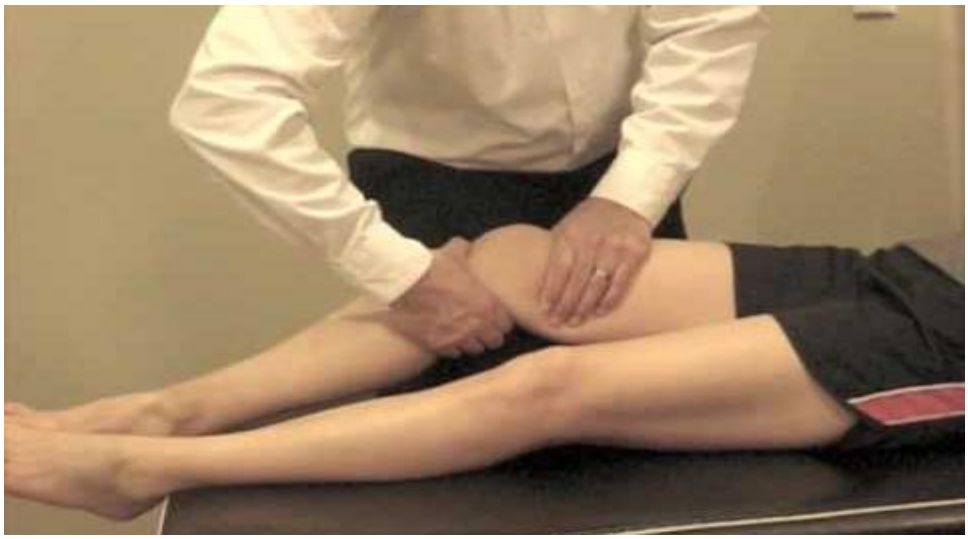
- Drawer's test (For Anterior and Posterior Cruciate Ligaments/ACL and PCL)
  Flex the patient's knee to 90o ask for their permission to sit on the foot to prevent it from rotating. Place both hands the tibia by placing your thumbs on the tibial tuberosity and your fingers along the popliteal fossa.
  Perform the posterior drawer test first by moving the tibia away from you, any translocation indicates possible PCL rupture.



Look for posterior sag before performing the anterior drawer test, then move the tibia towards you, any translocation that is >5mm indicates possible ACL rupture.



- Lachman's test (for ACL)
  Ask the patient to extend his knee and relax it. Place your knee on the bed under the patient's knee and hold the distal end of femur with your left hand and the proximal end of tibia with your right hand then try to translocate the tibia on the femur anteriorly. You should feel a stiff end point that indicates normal ACL. Absence of abrupt stiffness indicates possible ACL rupture (positive test).

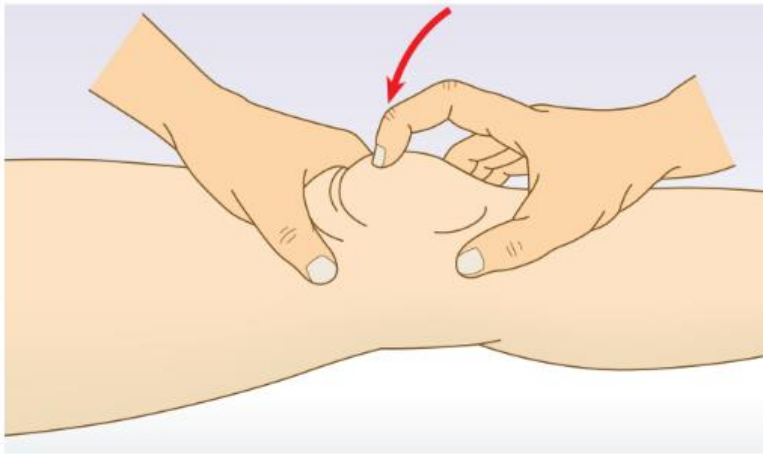


Testing for Knee effusion:

- The Patellar Tap:

With the patient's knee extended, empty the suprapatellar pouch by sliding your left hand down the thigh until you reach the upper edge of the patella.

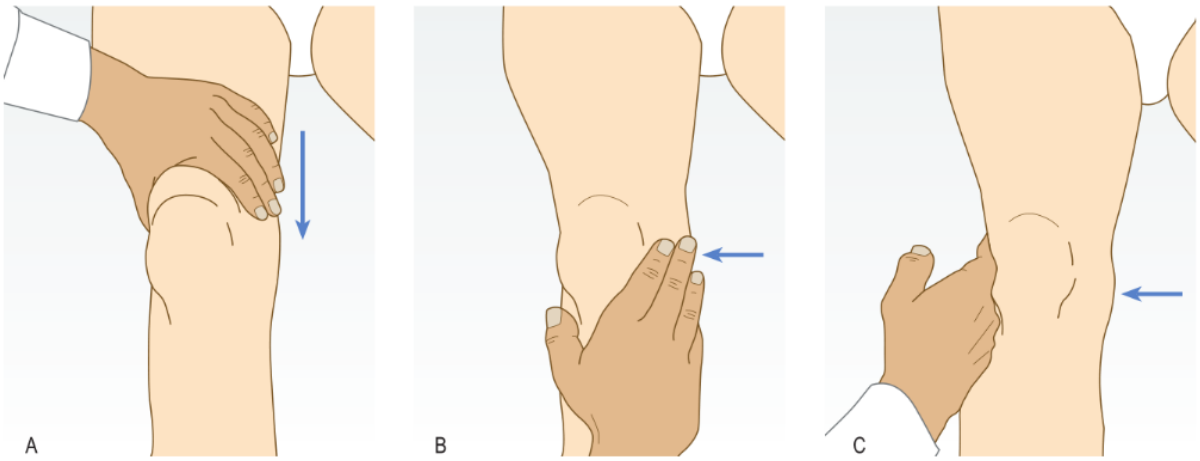
Keep your hand there and, with the fingertips of your right hand, press down briskly and firmly over the patella. In a moderate-sized effusion you will feel a tapping sensation as the patella strikes the femur. You may feel a fluid impulse in your left hand.



Testing for effusion by the patellar tap.

- The 'Bulge or Ripple test':

With the patient's knee extended and the quadriceps muscles relaxed, empty the medial side of the joint by milking the fluid to the suprapatellar pouch. Now empty the suprapatellar pouch as for the patellar tap. Stroke the lateral side of the joint. Watch the medial side for a bulge or ripple as fluid reaccumulates on that side.


A
B
C

Chapter 8: Infectious Disease

Fever, Hyperthermia & Hyperpyrexia

Note: most of temperature cut points written here are controversial and differ according to the resource. The cut points that are mentioned here are according to Dr. Jamal, so memorize these values for the sake of the exam.

INTRODUCTION

1- Normal body temperature:

- The 'normal' oral or ear temperature is 37°C but may range between **35.8°C and 37.2°C** (98–99°F).
  - Peripheral methods of monitoring temperature are: **tympanic membrane, temporal artery, axillary, and oral thermometry.**
  - Central methods are**: pulmonary artery catheter, urinary bladder, esophageal, and rectal thermometry**).
  - Peripheral methods of monitoring temperature are not as accurate as central methods, but central methods are less practical than peripheral methods.

2- Definition of Symptoms:

- **Fever (pyrexia)** is a body temperature >99th percentile of the healthy adult maximum. Fever is defined as a single spike of temperature above 38^{o}c per 24 hours, or two spikes of temperature between 37.2^{o}c and 38^{o}c per 24 hours, or sustained temperature between 37.2^{o}c and 38^{o}c for at least 2 hours per 24 hours.
  - Fever is a sign, which is the objective measure, the subjective measure (symptom) of fever is **"feeling of hotness".**
  - A patient with a fever is described to be **febrile.**
  - Fever occur due to elevation of the thermostat setting in the hypothalamic thermoregulatory center.
- **Chills & Rigors:** Most resources uses these terms interchangeably, but they are not the same:
  - **Rigors:** are bouts of uncontrollable muscular shaking, often with 'chattering' teeth, lasting for minutes. They are associated with rapid temperature rises and may be caused by cytokines and acute-phase proteins resetting the hypothalamic temperature set point. Subjectively the patient feels cold and unwell and the episode may be followed by sweating. A rigor may be associated with **bacteraemia or malaria** and is of poor diagnostic value.
  - **Chills (**goose skin) seen more commonly with **viral infections.**

- **Hyperpyrexia/Extreme fever**: is the term for an extraordinarily high fever (>41.5°C).
- **Low grade fever:** any fever below 38.2ºc
- **Hyperthermia:** In contradistinction to fever, the setting of the thermoregulatory center during hyperthermia remains unchanged at normothermic levels, while body temperature increases in an uncontrolled fashion and overrides the ability to lose heat. Exogenous heat exposure and endogenous heat production are two mechanisms by which hyperthermia can occur.
- Neutropenic fever: a single oral temperature of >38.3°C (101°F) or a temperature of >38.0°C (100.4°F) sustained for >1 hour in a patient with severe neutropenia (<500).

➢ If the patient has mild or moderate neutropenia (500-1500), it's described as febrile neutropenia.

3- Fever of unknown origin:

- Fever of unknown origin (FUO) is defined as:

1. Fever higher than 38.3ºC on several occasions.
2. lasting for at least three weeks Or 3 visits to the clinic.
3. without an established etiology despite intensive evaluation and diagnostic testing.

- Classified according to the occasion:

➢ **Immunodeficient FUO** (in immunocompromised patient)
➢ **Neutropenic FUO** (see neutropenic fever above)
➢ **HIV-related FUO** (in HIV +ve patients)
➢ **Nosocomial** (Hx of hospital admission)
➢ **Classical FUO** (not otherwise)

17.4 Causes of pyrexia of unknown origin

Infection (~30%)	Non-infectious causes (~70%)

• Tuberculosis (extrapulmonary and disseminated)
• Endocarditis
• Abdominal abscess
• Bone and joint infection
• Urinary and prostatic infection
• HIV-related
• Travel-related, e.g.
  ◦ Malaria
  ◦ Typhoid
  ◦ Dengue
  ◦ Rickettsial infections
• Healthcare-acquired infection
	• Malignancy
	• Connective tissue disorders

- Approach to patient with FUO:

1- Detailed Hx & Px (hx of fever as seen next, ROS, looking for patterns of certain Diseases, considering uncommon presentations).

2- Extensive investigations:

➢ ESR & CRP
➢ CBC with deferential
➢ LFT
➢ Urine analysis
➢ KFT
➢ TSH/T3/T4
➢ Serum lactate dehydrogenase

- Tuberculin skin test or interferon-gamma release assay
- HIV antibody assay and HIV viral load for patients at high risk.
- Three routine blood cultures drawn from different sites over a period of at least several hours without administering antibiotics, if not already performed
- Rheumatoid factor
- Creatine phosphokinase
- Heterophile antibody test in children and young adults
- Antinuclear antibodies
- Serum protein electrophoresis.
- Chest X-ray + CT
- Abdominal CT with contrast
- Echocardiogram.

Note: In FUO you're looking for needle in a haystack, so you should have very low threshold to think about a diagnosis.

3- If all these came negative, the patient is Saied to have FUO, what to do here?

- Give the patient antipyretics + hydration.
- Follow up every week (complete Hx or Px, to check for new complaints), no need to repeat the work up unless there are new findings.
- These follow ups continue until the fever is gone or after 16 Weeks.
- At week number 16, do extensive investigations, if not thing is found, repeat the cycle.

History Taking

Hx of fever

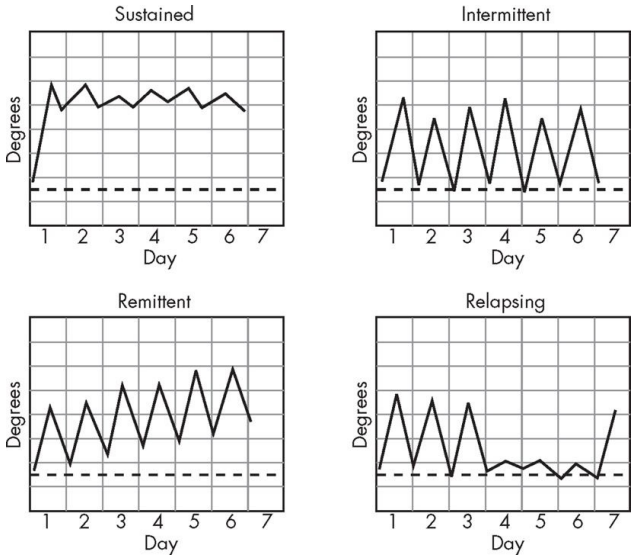
Patient's profile: Age, marital state, occupation & residency all give clues about the cause.

Chief complaint: Feeling of hotness for X days duration.

HPI:

- How do the patient know they had pyrexia?
- Documented (measured) or not? What was the temperature?
- Onset (how it started)?
- Pattern (Day-night variation, frequency, progression & previous attacks):

1. **Sustained fever:** ex: typhoid fever (caused by salmonella typhi, usually without sweating)
2. **Remittent fever:** ex: Brucella (usually associated with excessive sweating).
3. **Intermittent fever:** ex: the presence of abscess.
4. **Relapsing fever:** borrelia infection.
5. **Syndromic fever:** special patterns associated with special infections, ex: tertian fever in P. vivax and P. ovale malaria.



- Exacerbating/ Alleviating factors.
- Associated symptoms: Rigors, Chills, Lethargy, Night sweats, Weight loss, loss of appetite.

ROS:

- Systematically ask about localizing symptoms.

Past medical history:

- Immunization.
- Immunosuppression, DM, steroids use, HIV.
- Tuberculosis, Rheumatic fever.
- Valvular heart problems, CHD.
- Cancer.
- Hospital admissions.
- Hx of FUO or FMF.

Past surgical history:

- Recent surgeries (wound infection?)
- Dental procedures.
- Gynecological procedures.

Family history:

- Tuberculosis
- Cancer
- Immunosuppressive illnesses
- Familial Mediterranean fever.

Drug history (drug fever?)

Social history:

- Alcohol, smoking, illicit drug use.
- Recent travel history:
  Where/when/what country?
  Food, water, restaurants?
  Did others on holiday have same symptoms?
  Swimming in rivers, at coasts or in possible contaminated waters?
  Insect/tick bites?
- Recent diet.
- Recent contact with animals or pits.
- Sexual history (if appropriate and only after signposting clearly)

Brucella (Malta fever)

INTRODUCTION

Brucellosis has a very high incidence in Mediterranean regions.

- It's transmitted by Ingestion of non-pasteurized dairy products; most common route, Ingestion of infected tissue or Slaughterhouse workers exposed to animal meat through abraded skin.
- Fever is the most common symptom and sign, the infection spreads then and it can cause a focal disease (Spondylodiscitis, endocarditis, CNS infections, epididymo-orchitis)

History Taking

Patient profile: residency (rural area), occupation (Farmer, butcher, Diary products production)

Chief complaint: Fever for X duration.

History of presenting illness:

- Fever Hx (as seen before): usually gradually onset remittent fever, worst at night, with excessive sweating, chills, rigors, myalgia, Wight loss & loss of appetite.
- Focal symptoms (ROS):
  1. CNS involvement: Headache, photophobia, neck stiffness, seizures & confusion. (meningitis).
  2. MSS: Low back pain (Spondylodiscitis/sacroiliitis), bone & joint pain.
  3. GU: testicular swelling, Tenderness, painful/bloody ejaculation. (epididymo-orchitis)
  4. CVS & RS reviews, especially visitations (systemic thromboembolic events), that indicates endocarditis.

previous medical & surgical Hx (DM, AIDS, malignancy, malnutrition, vitamin deficiency, immunosuppression state .. etc)

Family history of Brucellosis.

Social history:

- Pets/animals.
- Feeding habits (unpasteurized milk, uncooked meat)
- Travel history.

Physical examination:

- General weakness, may be cachectic.
- Lymph node enlargement.
- GI exam: hepatosplenomegaly.
- MSS exam: Joint and bone tenderness, swelling, restriction of movement.
- CNS exam: signs of meningitis.
- CVS: new onset murmurs, janeway lesions, osler's nodes, roth's spots.
- GU: testicular swelling & tenderness.

DDx:

- TB.
- Typhoid fever.
- Endocarditis.
- Malignancy (Ex: lymphoma & multiple myeloma).
- HIV

Work up:

- CBC, LFT.
- Blood cultures (positive in 50% of cases)
- Serology (Standard tube agglutination or ELISA) → titers above 1/160 considered +Ve.
- PCR
- Echocardiogram (to rule out endocarditis).

Tuberculosis (TB)

INTRODUCTION

- TB is caused by MTB mycobacterium tuberculosis.
  - It is transmitted via an airborne droplet nucleus.
  - **Primary TB** usually starts in the middle lobes, it has one of 3 fates, it either regress & calcifies, or becomes latent TB or progresses into Primary pulmonary TB.
  - **Secondary TB:** Either exogenous (new infection) or endogenous infection (reactivation of a dormant primary lesion) in a person who has been sensitized by earlier exposure.
  - **Miliary TB:** Following a massive blood-borne dissemination of MTB, the microorganism will infiltrate any organ.
  - Extra-pulmonary TB: TB meningitis, MSS involvement (Pott's disease), GI lesions (mimics Crohn's disease), Lymphadenitis, pericardial involvement, and rarely, GU & Skin involvement.

History Taking

- **Patient profile:** Age (more common in children), residency & occupation (crowded places)
- **Chief complaint:** A Respiratory symptom, ex: cough for 3 months duration.
- **History of presenting illness:**
  - Ask about all respiratory symptoms (check the respiratory section):
    1. Cough
    2. Sputum production
    3. Hemoptysis
    4. Dyspnea
    5. Wheezing
    6. Chest pain
  - Ask about the constitutional symptoms:
    1. Fever.
    2. Night sweats.
    3. Malaise.
    4. Fatigue.
    5. Anorexia.
    6. Wight loss.
  - Focal symptoms (ROS):
    1. CNS involvement: Headache, photophobia, neck stiffness, seizures & confusion (Note: TB meningitis usually presents in a chronic pattern).
    2. MSS: Bone & joint pain, pathologic fractures (fracture with a trivial trauma), deformities (Kyphosis).

3. CVS: symptoms of constrictive pericarditis (Fatigue, chest pain, dyspnea, prominent veins in the neck, hepatomegaly, without lower limb edema).
4. GI: Intestinal obstruction (Constipation, vomiting, abdominal distension, with colicky abdominal pain), Anorectal ulcerations (anal fissure like symptoms; tearing pain worst after defecation and persists for hours), generally mimics Chron's disease.
5. GU: Dysuria, hematuria, loin pain, testicular swelling, painful ejaculation, bloody ejaculation.

previous medical & surgical Hx (Childhood TB, DM, AIDS, malignancy, malnutrition, vitamin deficiency, immunosuppression state .. etc)

Family history of TB & malignancy.

Social history:

- Travel history (if the patient is immigrant, or traveled to an endemic area)
- If he was a prisoner.

DDx:

- According to the system involved (malignancy, Brucella, etc ..)
- Hint: TB can present in any way that you can think about, so it's a part of any DDx list :p
- DDx for pulmonary TB (cavitary lesions in X-ray):
    1. Lung abscess/pneumonia
    2. Lung cancer
    3. Pulmonary infarction
    4. Wegner's granumalotosis
    5. Progressive massive fibrosis

Work up:

- Sputum stain & culture: 3 early morning samples or every 12 hours:
    1. Ziehl-Neelsen stain
    2. Culture: solid medium (Lowenstein-Jensen agar)
    3. PCR: to differentiate it from atypical mycobacteria.
- CXR.
- Bronchial biopsy.

UTI

INTRODUCTION

The urinary tract is sterile, if it's colonized by bacteria this is UTI.

- UTIs require anatomical problem to occur, it's the short urethra in females. In males, however, it always indicates an underlaying problem (obstruction, strictures, stones)
- UTIs are either **Cystitis** (Urgency, frequency, Dysuria & suprapubic pain) or **pyelonephritis** (all of the above symptoms + Fever & loin pain), the differentiation between these infections is critical, as pyelonephritis carries higher risk for the development od sepsis & MOF.
- The most common cause of UTI is E. Coli.

History Taking

Patient profile: Sex (more common in females)

Chief complaint: dysuria for X days duration.

History of presenting illness:

- Lower urinary symptoms (cystitis):
  1. **Dysuria.**
  2. **Suprapubic pain?**
  3. **Frequency?**
  4. **Urgency?**
  5. Pyuria (pus in urine? smell in urine?)
  6. Hematuria (can be seen in cystitis, pyelonephritis, stones, or tumors, so it's not specific)
  7. Hesitancy.
  8. Intermittency.
  9. Poor stream.
  10. Terminal drippling.
  11. Feeling of incomplete voiding.

Characteristic symptoms of cystitis

symptoms of the risk factors (prostatic hypertrophy for example)

- Upper urinary symptoms (pyelonephritis)
  1. Flank (loin) pain? Unilateral or bilateral? SOCRATES?
  2. Fever
  3. Rigors
  4. Chills
  5. Confusion
- ROS

Our goal now is to ask about the risk factors (all these may be absent in females, as being female is the most important risk factor)

- **Past medical:**
  1. **Kidney stones** (ask about fluid intake as well).
  2. History of **prostatic hypertrophy** and its symptoms (already covered above).
  3. History of **DM & immunosuppression**.
  4. History of **constipation** (independent risk factor for UTI), **appendicitis** (pelvic appendicitis may cause UTI) & **fistulas** (diverticulitis & Chron's disease may lead to colo-vesical fistula, resulting in fecalurea, pneumaturia & recurrent UTI).
  5. History of **STDs** (itching and burning sensation).
  6. History of **spinal cord injury, cauda equina, urinary retention.**
- **Past surgical:**
  1. History of instrumentation (Foley's catheter, urethral surgery, etc ..) → strictures.
- **Drugs history:**
  1. steroids, immunosuppressants.
  2. Cyclophosphamide (may cause hemorrhagic cystitis)
- **Family history:** of UTIs, Kidney stones, malignancy.
- **Social history:**
  1. Genital hygiene.
  2. Sexual history:
     - Some birth control devices increase the risk of UTI: **spermicidal** & **Diaphragms**.
     - Relationship between the intercourse & UTIs: some females develop "**honeymoon" UTI**".
     - Oral or anal intercourse: increases the risk for UTI, especially in males.

Gastroenteritis/ food poisoning

INTRODUCTION

Normal bowel movement is variable, but it can range from 3 times/day to 3 times/week.

- Diarrhea is defined as a change in the usual frequency of defecation accompanied by a change in the consistency.
- Classification:
  1. Acute diarrhea: diarrhea lasting less than 2 weeks (14 days)
  2. Chronic diarrhea: more than one month
  3. Persistent diarrhea: diarrhea lasting more than 2 weeks but less than 4 weeks.

The Ddx of acute diarrhea is:

1. **Food poisoning:** develops due to ingestion of a food contaminated with preformed bacterial toxin, incubation period is few hours, presents mainly with nausea, vomiting, diarrhea, without constitutional symptoms.
2. **Gastroenteritis**: due to active infection in the gut, usually due to ingestion of food-borne bacteria, presents mainly with diarrhea (could be bloody), nausea, vomiting, and general constitutional symptoms (fatigue, anorexia, fever, rigors & chills).
3. **Drug side effect**.

The Ddx of chronic diarrhea is:

1. **Infections causes:** Entamoeba histolytica & giardia lamblia.
2. **Malabsorption syndromes:** Celiac disease, lactose intolerance, steatorrhea… etc.
3. **Inflammatory causes:** IBD & microscopic colitis.
4. **Endocrine problems:** Hyperthyroidism, DM (autonomic neuropathy), carcinoid syndrome, VIPoma (WDHA syndrome).
5. **Others:** Colorectal CA, IBS, Laxative abuse, bacterial colonization, surgical resection, fecal impaction .. etc.

History Taking

NOTE: in this section, we will discuss acute (infections diarrhea), other causes of diarrhea could be found in the GI section, and could be distinguished through systemic history taking of GI symptoms and recognition of diseases patterns.

Patient profile: residency, occupation & age are important in establishing the etiology.

Chief complaint: Diarrhea/Vomiting for X duration (usually less than 2 weeks).

History of presenting illness:

1. **Diarrhea:**
   - **Clarify** what the patient means by diarrhea in their own words (frequent bowel habit Vs loose bowel habit?)
   - **Time:** Duration? Course? (Intermittent, continuous, progressive).
   - Onset (how it started)
   - **Consistency**: Watery? Loose? Greasy and difficult to flush away? Well formed? Mucus?
   - **Colour:** Black (melaena)? Red (blood)? Green?
   - **Smell**: offensive?
   - Alternate with constipation?
   - Exacerbating/ reliving factors? (especially diet)
   - Any anal symptoms? (tenesmus/ urgency)?
   - Severity (how it affects the quality of life)
2. **Vomiting:**
   - Preceded by nausea or not?
   - Amount? Color?
   - Related to abdominal discomfort/ dyspepsia? Relives this dyspepsia?
   - Related to food?
3. **Abdominal pain** (SOCRATES)

4. **Abdominal distension**: (related to or relieves pain/ vomiting/ diarrhea)
5. **General constitutional symptoms:** Fever, chills, rigors, malaise, loss of appetite.
6. **Dehydration:** postural hypotension, dry mouth, decreased urinary output
7. **Other GI symptoms & ROS**

Our goal now is to ask about the risk factors/ cause:

- Previous medical history: DM, HIV, immunosuppressed?
- Drug history: laxatives, other drugs??
- Family history: Of GI problems
- **Social history** (the most important one, if you have no time & you are suspecting Infectious cause from the symptoms, ask about this first):
    1. **Recent foreign travel/foreign contacts**
        - Similar symptoms in other members of travelling party
        - Rural/forest exposure
        - Water consumed (mineral water, tap water, boiled water)
    2. **Diet:**
        - Recent changes (Contaminated water→ cholera and parasitic infections/ uncooked meat → E. Coli / egg products (مايونيز-شاورما) → salmonella / reheated rice → Bacillus cereus … etc)
        - Takeaways
        - Barbecues
    3. **Contact with anyone suffering from diarrhea**
    4. **Incubation period:** The time between risky behavior & the onset of symptoms

• Smoking
• Alcohol
• Occupation (dusty environment)
• Accommodation: institution, residential home
• Activities of daily living

The End

References:

1- Macleod Clinical Examination, 13th edition.
2- Step-up to Medicine 4th Edition.
3- Macleod Clinical Diagnosis 1st edition.
4- The Old OSCE dossier.
5- Medstudy 17th edition.