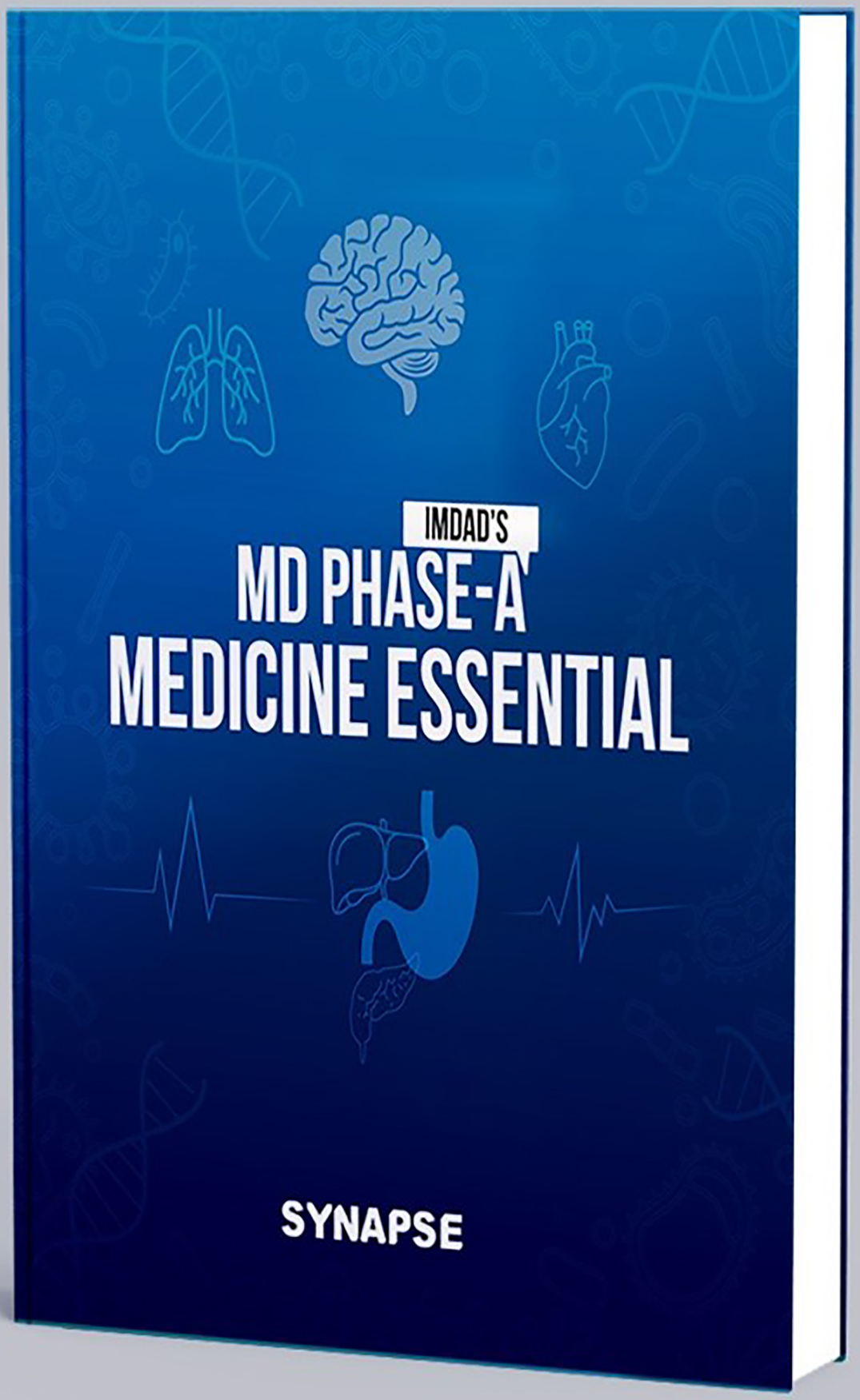

IMDAD'S
MD PHASE-A
MEDICINE ESSENTIAL
SYNAPSE

Tips for Written Exam

Question Pattern

২ টা পেপার পরীক্ষা, প্রতিটি পেপারে Group -A, Group -ই থাকবে। প্রতিটি গ্রুপে ১০ টি করে প্রশ্ন।

Question Type

পরীক্ষায় যে সকল প্রশ্ন আসে, সেগুলা একটা প্যাটার্ন ফলো করে।প্রশ্নগুলা সাধারণ হয়:

1) Evaluation type

- Clinical evaluation
- Only Evaluation

2) Management related

- Emergency management
- Non-Emergency management

3) Investigation related

4) Examination related

- Pulse
- JVP
- Fundoscopic findings
- Hand examination etc.

5) Theoretical question

- Differences
- Diagnostic Criteria
- Short note type question

Answer Pattern

1st Rule - Must Answer all questions

(Uncommon question ও কখনো ছেড়ে আসা যাবে না)

প্রথমে প্রশ্নটি সুন্দর করে ঠান্ডা মাথায় পড়বেন। key points গুলা মার্ক করবেন।প্রশ্ন পড়তে ভূল হলে পুরা উত্তরটাই ভুল হয়ে যাবে।

Evaluation

যে কোন প্রশ্নে শুধুমাত্র Evaluation চাইলে key points, Differentials, Clinical Evaluation (Clinical features of each differentials) এবং Investigation পর্যন্ত লিখতে হবে।

Clinical evaluation / Clinical checklist চাইলে Clinical evaluation পর্যন্ত লিখতে হবে

Answer to the Q NO- 1

Key points :

.....

.....

From this scenario my differentials are-

a).....

b).....

c).....

কোন scenario তে যদি key points গুলা কোন specific disease কে বেশি indicate করে, সেক্ষেত্রে Provisional diagnosis দিয়ে বাকিগুলা differentials হিসেবে দিতে পারেন।

From this scenario my provisional diagnosis is……Others possible differentials are-

a).....

b).....

c).....

Clinical Evaluation:

▶প্রতিটা diseases এর important symptoms, Examination findings গুলা লিখে ডান দিকে Disease name টা দিয়ে দিবেন। চাইলে এটা বক্স করেও দিতে পারেন।

- —— —— —— —— —— ——
- —— —— —— —— —— —— Disease name
- —— —— —— —— —— ——
- —— —— —— —— —— ——

- —— —— —— —— —— ——
- —— —— —— —— —— —— Disease name
- —— —— —— —— —— ——

Investigation:

▶Investigation লেখার সময় scenario অনুযায়ী 1st line, second line investigation অথবা General, specific investigation, অথবা investigation to confirm diagnosis,investigation to see etiology, to see complications, investigation for follow-up যে কোন একটা প্যাটার্ন ফলো করতে পারেন। Differentials অনেক বেশি থাকলে সেক্ষেত্রে আলাদা না করে একবারেই সকল investigation দিয়ে দিতে পারেন।

▶Specific / 2nd line investigation এ invasive / advanced / confirmatory investigation গুলা দিবেন।

1st line investigation / General investigation

Investigation Name	Findings

2nd Line / Specific

Investigation Name	Findings

Endocrinology

SL. No.	Topic	Page	Written	OSPE
1.	Investigation			
2.	Radioactive Iodine Uptake (RAIU) Test		**	**
3.	Thyroid Disease			
4.	Atrial Fibrillation in Thyrotoxicosis		**	
5.	Thyrotoxic Crisis (Thyroid Storm)		***	*
6.	Hashimoto's Thyroiditis (Chronic Autoimmune Thyroiditis)		**	**
7.	Subacute (de Quervain's) Thyroiditis		***	***
8.	Post-partum Thyroiditis		***	
9.	Riedel's Thyroiditis		*	
10.	Hypothyroid		***	**
11.	Myxoedema Coma		***	*
12.	Subclinical Thyrotoxicosis			*
13.	Subclinical Hypothyroidism			*
14.	Non-Thyroidal Illness (Sick Euthyroidism / Euthyroid Sick Syndrome)		*	
15.	Graves' Disease		***	***
16.	Graves' Ophthalmopathy		***	**
17.	Thyroid Nodule & Swelling			
18.	Multinodular Goiter		**	**
19.	Thyroid Neoplasia		*	*
20.	Delayed puberty		**	
21.	Amenorrhoea		**	
22.	Gynaecomastia		***	
23.	Hirsutism		***	***
24.	PCOS		***	***
25.	Turner's syndrome		*	**
26.	Klinefelter		*	*
27.	Short stature		***	
28.	Parathyroid Diseases		**	***
29.	Hypercalcaemia		***	*
30.	Hypoparathyroid disease		**	**
31.	Hypocalcaemia		**	**
32.	Cushing syndrome		***	***
33.	Pseudo cushing		***	
34.	Adrenal insufficiency		***	**
35.	Incidental Adrenal Mass		**	
36.	Primary hyperaldosteronism		***	*
37.	Phaeochromocytoma		**	*
38.	Spontaneous hypoglycaemia		*	*
39.	Hypopituitary		**	*
40.	Galactorrhoea		*	
41.	Acromegaly		**	***

Previous Question

Thyroid Disease

Q. A young man has presented with chronic diarrhoea, weight loss and palpitation. How would you evaluate the patient?
Q. How will you evaluate and manage a case of thyrotoxic crisis (thyroid storm)?
****Q. A 40-year-old man, a case of thyrotoxicosis underwent thyroid surgery. On first POD, he developed high grade fever, palpitation and altered consciousness. What is the most probable cause of this presentation and outline the management plan for him. *(July 2024)***
Q. How will you manage a 34 year old woman presenting with features of thyrotoxicosis and low I^{131} uptake
*****Q.1 A 35-year-old woman presented with fever, pain in front of her neck, palpitation, tremor and sweating for 2 (two) weeks. How would you investigate and treat her? *(January 2026)***
*****Q. A 25-year-old woman presented with fever, palpitation and painful neck swelling. How would you investigate and manage her? *(July 2023)***
Q: How would you evaluate and manage a case of myxoedema coma.
Q. How would you evaluate a 20 year old girl complaining of 10 kg weight gain in last 2 months. or How would you clinically evaluate a 20 year old woman present with weight gain.
***Q. How would you evaluate a 35 year old woman presenting with extreme weakness having low FT3, FT4 but near normal TSH.**
Q. How would you evaluate and manage pain and tingling sensation in fingers in a patient with hypothyroidism?*(July 2022)

Graves' disease

Q. How would you manage a woman with thyrotoxicosis at her 8 weeks of pregnancy? or How will you manage thyrotoxicosis due to Graves disease during pregnancy?
Q. Graves' ophthalmopathy disea

Thyroid nodule & swelling

Q: How will you clinically evaluate a 20-year-old woman presenting with thyromegaly? *(January 2017)*
Q. How would you evaluate and manage a 30-year-old woman with solitary thyroid nodule?

Gynaecomastia

*****Q. How would you evaluate and investigate a 56-year-old man presented with gynaecomastia? *(July 2024, 2022, January 2023)***

Hirsutism

*****Q.A 20-year-old woman presented with progressive weight gain, menstrual irregularity and excessive growth of facial hairs for 6 (six) months. Make a list of clinical information and necessary investigations with expected findings to reach a diagnosis. *(January 2024, 2026)***
****Q.A 20-year-old woman presented with irregular menstruation and hirsutism. How would you evaluate her? *(July 2023)***

Investigations

Radioactive Iodine Uptake (RAIU) Test

Principle:

Measures the thyroid gland's ability to trap and concentrate **radioactive iodine** (usually **I-123** or low-dose **I-131**) from the blood via the sodium-iodide symporter (NIS). It reflects thyroid function and iodine organification/turnover.

Procedure:

- Patient swallows a small dose of radioactive iodine (capsule/liquid).
- Uptake measured using a gamma probe over the neck at **4–6 hours** and **24 hours** (sometimes only 24 h).
- Expressed as **percentage** of administered dose taken up by thyroid.
- Often combined with thyroid scan (for imaging distribution).

Indications:

- Differential diagnosis of **thyrotoxicosis/hyperthyroidism**
- Distinguish Graves' disease vs toxic nodule vs thyroiditis
- Evaluate cause of low/high thyroid function
- Calculate dose for radioactive iodine therapy
- Assess thyroid autonomy or organification defects

Normal Values (approximate; lab-specific, influenced by dietary iodine):

- 4–6 hours: **3–16%** (or 5–15%)
- 24 hours: **8–35%** (commonly 10–30%)

Interpretation:

- **High uptake** (> normal range) → Increased trapping/function
  - ✓ Graves' disease (diffuse high uptake)
  - ✓ Toxic multinodular goiter
  - ✓ Toxic adenoma
  - ✓ TSH-secreting pituitary tumor

- **Low uptake** (< normal range) → Decreased trapping/function
  - ✓ Subacute thyroiditis / Silent thyroiditis (destructive release)
  - ✓ Factitious thyrotoxicosis
  - ✓ Struma ovarii
  - ✓ Exogenous thyroid hormone intake
  - ✓ Hypothyroidism (primary)

Thyroid Disease

How to interpret thyroid function test results

TSH	T_4	T_3	Most likely interpretation
Undetectable	Raised	Raised	Primary thyrotoxicosis
Undetectable or low	Raised	Normal	Extra-thyroidal thyroxine with thyrotoxicosis
Undetectable or low	Normal	Raised	Primary T_3 toxicosis
Undetectable or low	Normal	Normal	Subclinical thyrotoxicosis

Undetectable or low	Raised	Low or normal	Non-thyroidal illness
Undetectable or low	Low	Raised	Over-treatment of hypothyroidism with levothyroxine (↓ TSH)
Undetectable or low	Low	Low	Secondary hypothyroidism
Raised	Low	Low	Primary hypothyroidism
Mildly elevated (5–20 mU/L)	Low	Low	Primary hypothyroidism
Elevated (>20 mU/L)	Low	Low	Severe primary hypothyroidism
Mildly elevated (5–20 mU/L)	Normal	Normal	Subclinical hypothyroidism
Elevated (>20 mU/L)	Normal	Normal	Athetoid antibodies with affinity to the animal assay used in TSH assay
Elevated	Raised	Raised	TSH-secreting tumour / thyroid hormone resistance

Usually upper part of reference range. T_3 is not a sensitive indicator of hypothyroidism and should not be requested. Usually lower part of reference range. i.e. Secondary to pituitary or hypothalamic disease. Note that TSH assays may report detectable TSH.
(TSH = thyroid-stimulating hormone)

(Ref: Davidson 24th P-654, Box:30.5)

Clinical features of thyroid dysfunction (OSPE)

Category	Thyrotoxicosis – Symptoms	Thyrotoxicosis – Signs	Hypothyroidism – Symptoms	Hypothyroidism – Signs
Common	Weight loss despite normal or increased appetite Heat intolerance, sweating Palpitations, tremor Dyspnoea, fatigue Irritability, emotional lability	Weight loss Tremor Palmar erythema Sinus tachycardia Lid retraction, lid lag	Weight gain Cold intolerance Fatigue, somnolence Dry skin Dry hair Menorrhagia	Weight gain
Less common	Osteoporosis (fracture, loss of height) Diarrhoea, steatorrhoea Angina Ankle swelling Anxiety, psychosis Muscle weakness Periodic paralysis (predominantly in Chinese and other Asian groups) Pruritus, alopecia Amenorrhoea/oligomenorrhoea Infertility, spontaneous abortion Loss of libido, impotence Excessive lacrimation	Goitre with bruit[1] Atrial fibrillation[2] Systolic hypertension/increased pulse pressure Cardiac failure Hyper-reflexia Ill-sustained clonus Proximal myopathy Bulbar myopathy[2]	Constipation Hoarseness Carpal tunnel syndrome Alopecia Aches and pains Muscle stiffness Deafness Depression Infertility	Hoarse voice Facial features Purplish lips Malar flush Periorbital oedema Loss of lateral eyebrows Anaemia Carotenaemia Erythema ab igne Diastolic hypertension Delayed relaxation of reflexes Dermal myxoedema

Rare	Vomiting Apathy Anorexia Exacerbation of asthma	Gynaecomastia Spider naevi Onycholysis Pigmentation	Psychosis (myxoedema madness) Galactorrhoea Impotence	Ileus, ascites Pericardial and pleural effusions Cerebellar ataxia Myotonia

[1] In Graves' disease only
[2] Features found particularly in older patients

(Ref: Davidson 24th P-655, Box:20.7)

Prevalence of thyroid autoantibodies (%)

	Antibodies to:		
	Thyroid peroxidase[1]	**Thyroglobulin**	**TSH receptor[2]**
Normal population	8–27	5–20	0
Graves' disease	50–80	50–70	>95
Autoimmune hypothyroidism	90–100	80–90	10–20
Multinodular goitre	~30–40	~30–40	0
Transient thyroiditis	~30–40	~30–40	0

[1] Thyroid peroxidase (TPO) antibodies are the principal component of what was previously measured as thyroid microsomal antibodies.
[2] Thyroid-stimulating hormone receptor antibodies (TRAb) can be agonists (stimulating, causing Graves' thyrotoxicosis) or antagonists (blocking, causing hypothyroidism).

Non-specific laboratory abnormalities in thyroid dysfunction (OSPE)

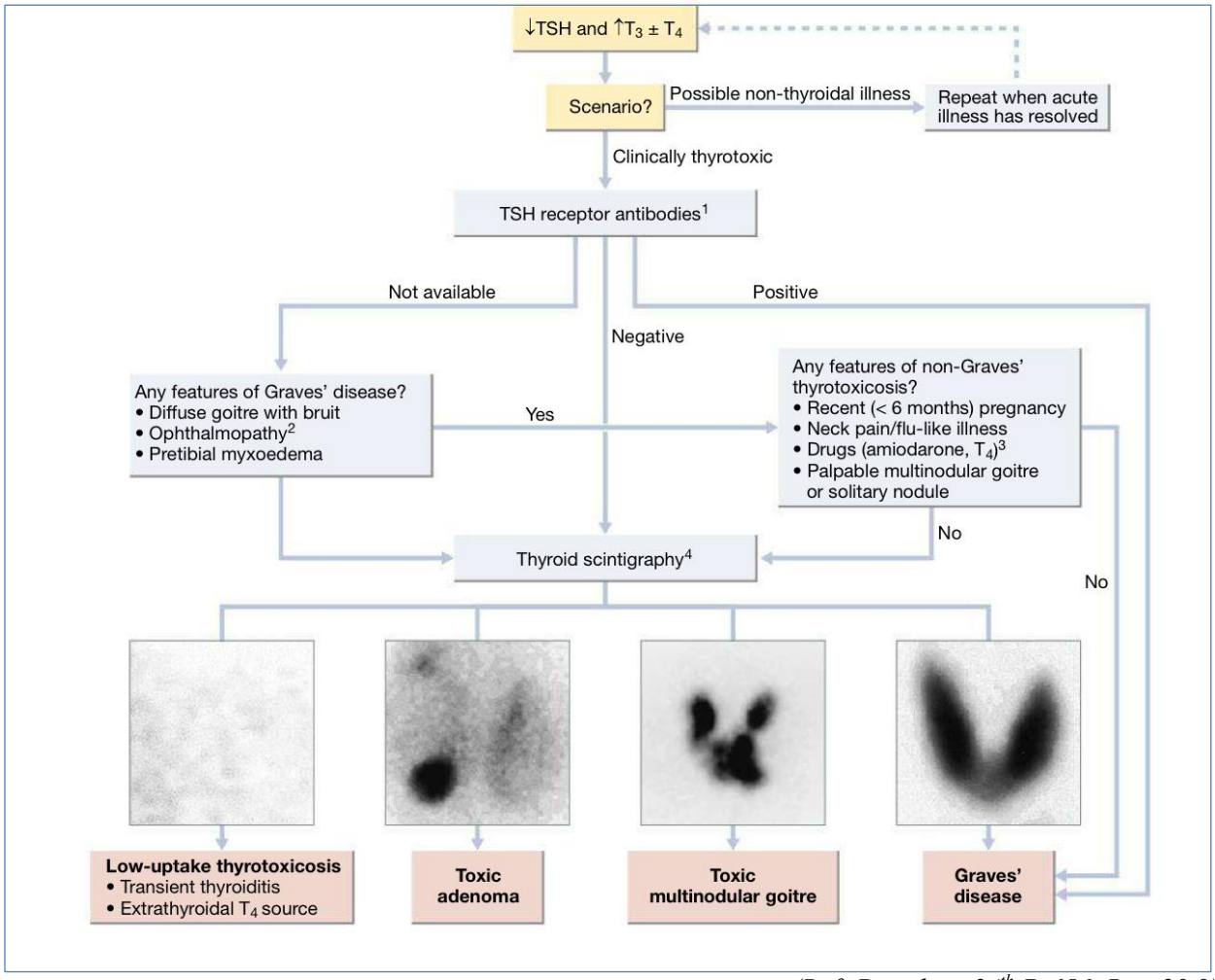
Thyrotoxicosis

- Serum enzymes: raised alkaline aminotransferase, γ-glutamyl transferase (GGT), and alkaline phosphatase from liver and bone
- Raised bilirubin
- Mild hypercalcaemia
- Glycosuria: associated diabetes mellitus, 'lag storage' glycosuria

Hypothyroidism

- Serum enzymes: raised creatine kinase, aspartate aminotransferase, lactate dehydrogenase (LDH)
- Hypercholesterolaemia
- Anaemia: normochromic normocytic or macrocytic
- Hyponatraemia

These abnormalities are not useful in differential diagnosis, so the tests should be avoided and any further investigation undertaken only if abnormalities persist when the patient is euthyroid.



(Ref: Davidson 24th P-656, Box:20.9)

Thyrotoxicosis

Causes of thyrotoxicosis and their relative frequencies

Cause	Frequency (%)
Graves' disease	70
Multinodular goitre	14
Solitary toxic adenoma	5
Thyroiditis	3
Post-partum thyroiditis	0.5
Subacute thyroiditis	0.5
Drug-induced	1
Iodine-induced	1
Trophoblastic tumour	1
Factitious thyrotoxicosis	0.2
Struma ovarii	<0.1
TSH-secreting tumour	<0.2
Thyroid hormone resistance	<0.1

In a series of 2087 patients presenting to the Royal Infirmary of Edinburgh over a 10-year period, characterised by negligible radioisotope uptake; i.e. de Quervain sarcoid containing thyroid tissue. *Human chorionic gonadotrophin has thyroid-stimulating activity.*
(TSH = thyroid-stimulating hormone)

(Ref: Davidson 24th P-656, Box:20.6)

Q. A young man has presented with chronic diarrhoea, weight loss and palpitation. How would you evaluate the patient?

From above scenario most likely diagnosis is – Thyrotoxicosis

Evaluation:

History –

- Heat intolerance
- Excessive sweating
- Increase or normal appetite with weight loss
- Tremor of hands
- Restlessness
- Irritability
- Dyspnoea
- Fatigue
- Anxiety
- Emotional lability
- Angina
- Osteoporosis – fracture or loss of height
- Muscle weakness
- Steatorrhoea
- Loss of libido
- Impotence
- Excessive lacrimation
- Apathy

Examination:

- Weight loss or low BMI
- Fine tremor on outstretched hand
- Palmar erythema
- Irregularly irregular pulse
- Systolic hypertension
- Lid lag
- Lid retraction
- Goitre with or without bruits
- Hyper-reflexia
- Proximal myopathy
- Gynaecomastia
- Pigmentation
- Spider naevi

Investigation:

Name of investigation — Aim / Interpretation

1. **FT3, FT4, TSH**
   - ✓ Low TSH
   - ✓ High FT3, FT4

2. **Determine the cause**
    - Thyroid antibody
    - Radioactive iodine uptake
    - Thyroid scan
    - Thyroid ultrasound
3. **Other test**
    - ECG

Here are the **key high-yield points** from the passage, condensed for quick exam revision:

Atrial Fibrillation in Thyrotoxicosis

- Occurs in ~**10%** of thyrotoxicosis patients
- Much higher incidence in **men** and strongly age-related → up to **~50%** in males >60 years
- **Subclinical thyrotoxicosis** is also a risk factor for AF
- Ventricular rate: poorly controlled by **digoxin** alone → good response to added **β-blocker**
- High risk of **thromboembolism** → **anticoagulation** required (unless contraindicated)
- After euthyroidism restored: ~**50%** spontaneously revert to sinus rhythm; rest may need **cardioversion**

Thyrotoxic Crisis (Thyroid Storm) **(OSPE)**

- Rare, life-threatening emergency; mortality ~**10%** even with treatment
- Classic features: **fever**, **agitation/delirium**, **tachycardia** / **AF**, and in elderly → **cardiac failure**
- Common precipitants:
    1. Infection (most frequent)
    2. Recent **thyroidectomy** (ill-prepared patient)
    3. Early post-^{131}**I** therapy (radiation-induced hormone release)

- Management (urgent endocrine input essential):
    1. **Supportive**: rehydration
    2. **β-blocker**: **propranolol** (preferred) – oral 80 mg q6h or IV 1–5 mg q6h
    3. **Glucocorticoids**: hydrocortisone 100 mg IV q8h (↓ peripheral T_4 → T_3 conversion)
    4. **Inhibition of new hormone synthesis**: **propylthiouracil (PTU)** 200 mg q4h (preferred over carbimazole because it also blocks T_4 → T_3 conversion)
    5. **Iodine** (after PTU started): sodium iodate, radiographic contrast (500 mg/day oral), or alternatives (KI / Lugol's solution) → normalizes T_3 in 48–72 h
    6. If unconscious/uncooperative → PTU + propranolol via **nasogastric tube**
    7. After 10–14 days → usually switch to **carbimazole** maintenance

(Ref: Davidson 24th P-654-655)

Q. How will you evaluate and manage a case of thyrotoxic crisis (thyroid storm)?

Precipitating factors:

- Infection
- Inadequate treatment of thyrotoxicosis
- Ill-prepared thyroidectomy
- Following radio-iodine ablation therapy

Clinical features:

- Fever
- Agitation
- Delirium

- Tachycardia
- Atrial fibrillation
- Cardiac failure

****Q. A 40-year-old man, a case of thyrotoxicosis underwent thyroid surgery. On first POD, he developed high grade fever, palpitation and altered consciousness. What is the most probable cause of this presentation and outline the management plan for him.** ***(July 2024)***

Key points:

- 40-year-old thyrotoxic patient underwent thyroid surgery
- 1st POD developed:
  - ✓ High fever
  - ✓ Palpitation
  - ✓ Altered consciousness

From the history and clinical presentation, this is a case of **thyrotoxic crisis**, mostly due to **ill-prepared surgery**.

Treatment:

It is a **medical emergency**, treat promptly.

General:

- Hospitalization in **HDU**
- Treat promptly, it is a medical emergency
- Adequate hydration
- Control of temperature
- Resuscitation as appropriate
- Broad spectrum antibiotics

Specific treatment:

- **Propranolol** orally 80 mg 4 times or intravenously 1–5 mg
- **Sodium iopanoate** 500 mg/day orally
- **Radioactive iodine**
- **Potassium iodide or Lugol's iodine**
- **Oral carbimazole** 40–60 mg daily for 5 days
- Then 45 mg daily for 5 days
- After 10 days maintenance dose 5–20 mg/day
- **Dexamethasone** 2 mg 4 times daily
- **Amiodarone**
- **Plasma exchange**

Hashimoto's Thyroiditis (Chronic Autoimmune Thyroiditis)

- Most common cause of hypothyroidism in iodine-sufficient areas
- Destructive lymphocytic infiltration → fibrosis ± goitre (goitrous or atrophic variants)
- Incidence: ~5–8/1000 women/year, ~0.8/1000 men/year; increases with age
- Clinical: firm/rubbery diffuse goitre; ~25% hypothyroid at presentation
- Key marker: **high-titre anti-TPO antibodies** (very strong diagnostic indicator)
- Natural history: initially euthyroid (TSH normal or slightly raised) → progressive risk of overt hypothyroidism
- Treatment: Levothyroxine
  - ✓ For hypothyroidism
  - ✓ To shrink goitre (dose to suppress TSH to low but detectable levels)

Mechanism of action	Drug	Dose	Hazards
Androgen receptor antagonism	Cyproterone acetate	50–200 mg on days 1–11 of 28-day cycle with ethinylestradiol 30 µg on days 1–21	Hepatic dysfunction, feminisation of male fetus, progesterone receptor agonist
	Spironolactone	100–200 mg daily	Menstrual irregularity, postural hypotension, hyperkalaemia
	Flutamide	Not recommended	Hepatic dysfunction
5α-reductase inhibition	Finasteride	5 mg daily	Limited clinical experience; possibly less efficacious than other treatments
Suppression of ovarian steroid production and elevation of sex hormone-binding globulin	Oestrogen	See combination with cyproterone acetate above; conventional oestrogen-containing contraceptive	Venous thromboembolism, hypertension, weight gain, dyslipidaemia, increased breast and endometrial carcinoma risk

(Ref: Davidson 24th P-674, Box:20.29)

Turner's syndrome

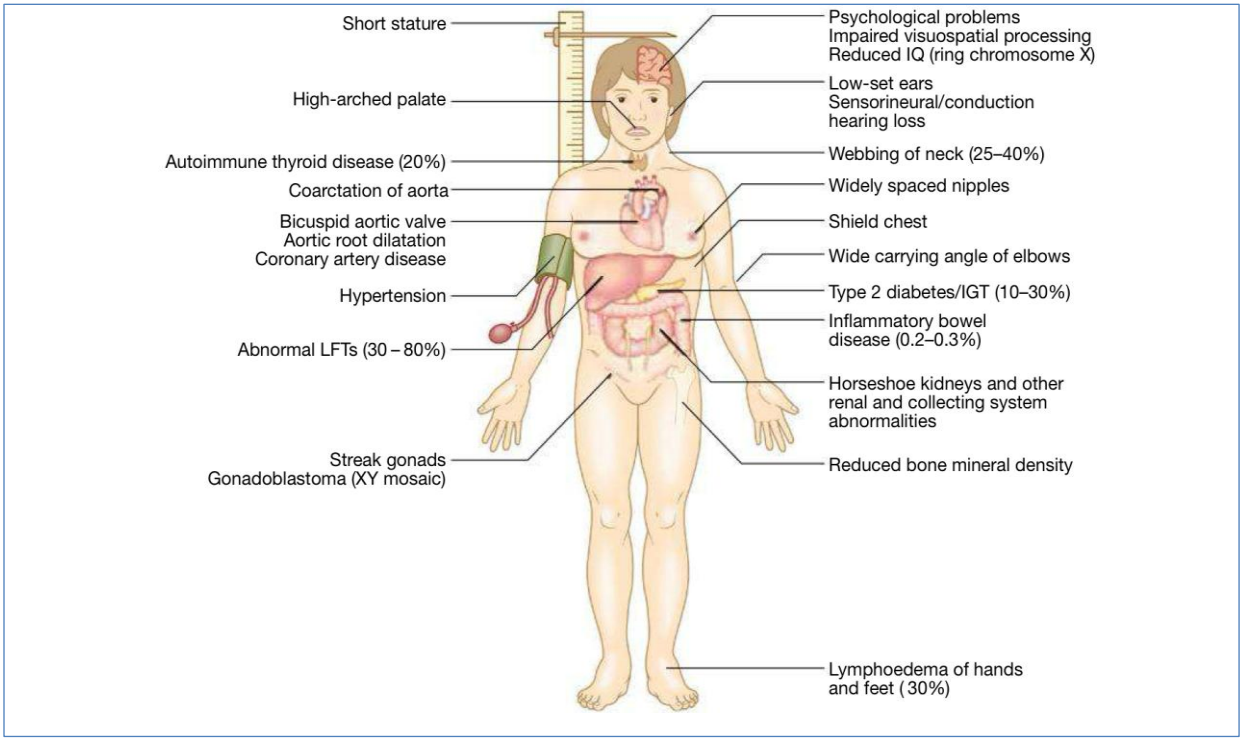
(OSPE)

Q. Briefly discuss about Turner's syndrome.

Incidence

- 1 in 2500 female
- Karyotype – **45XO**

Clinical features —



Investigation: Karyotyping analysis

Acute Severe Hypercalcaemia Management

1. **First-line**: Aggressive IV 0.9% saline rehydration → improves renal function & ↑ urinary Ca excretion
2. **Bisphosphonates** (e.g. **zoledronic acid**) IV → inhibit bone resorption (onset 2–4 days, lasts ~2–4 weeks)
3. **Calcitonin** (short-term) → rapid action (hours), used in life-threatening cases for first 24–48 h
4. **Denosumab** → alternative in refractory cases or renal failure
5. **Haemodialysis** → for very severe / resistant cases or renal failure

(Ref: Davidson 24th P-676)

*****Q. How would you evaluate and manage a patient with serum calcium level 16 mg/dl**

Possibilities:

Causes of hypercalcaemia (OSPE)

With normal or elevated parathyroid hormone (PTH) levels

- Primary or tertiary hyperparathyroidism
- Lithium-induced hyperparathyroidism
- Familial hypocalciuric hypercalcaemia

With low PTH levels

- Malignancy (lung, breast, myeloma, renal, lymphoma, thyroid)
- Elevated 1,25-dihydroxyvitamin D (vitamin D intoxication, sarcoidosis, human immunodeficiency virus, other granulomatous disease)
- Thyrotoxicosis
- Paget's disease with immobilisation
- Milk-alkali syndrome
- Thiazide diuretics
- Glucocorticoid deficiency

(Ref: Davidson 24th P-676, Box:20.32)

Clinical features – Bones, stones, abdominal groans, psychiatric

Symptoms –

- Polyuria
- Polydipsia
- Generalized aches and pain
- Renal colic
- Anorexia
- Nausea
- Dyspepsia
- Peptic ulceration
- Constipation
- Lethargy
- Depression
- Drowsiness
- Impaired cognition
- Fatigue

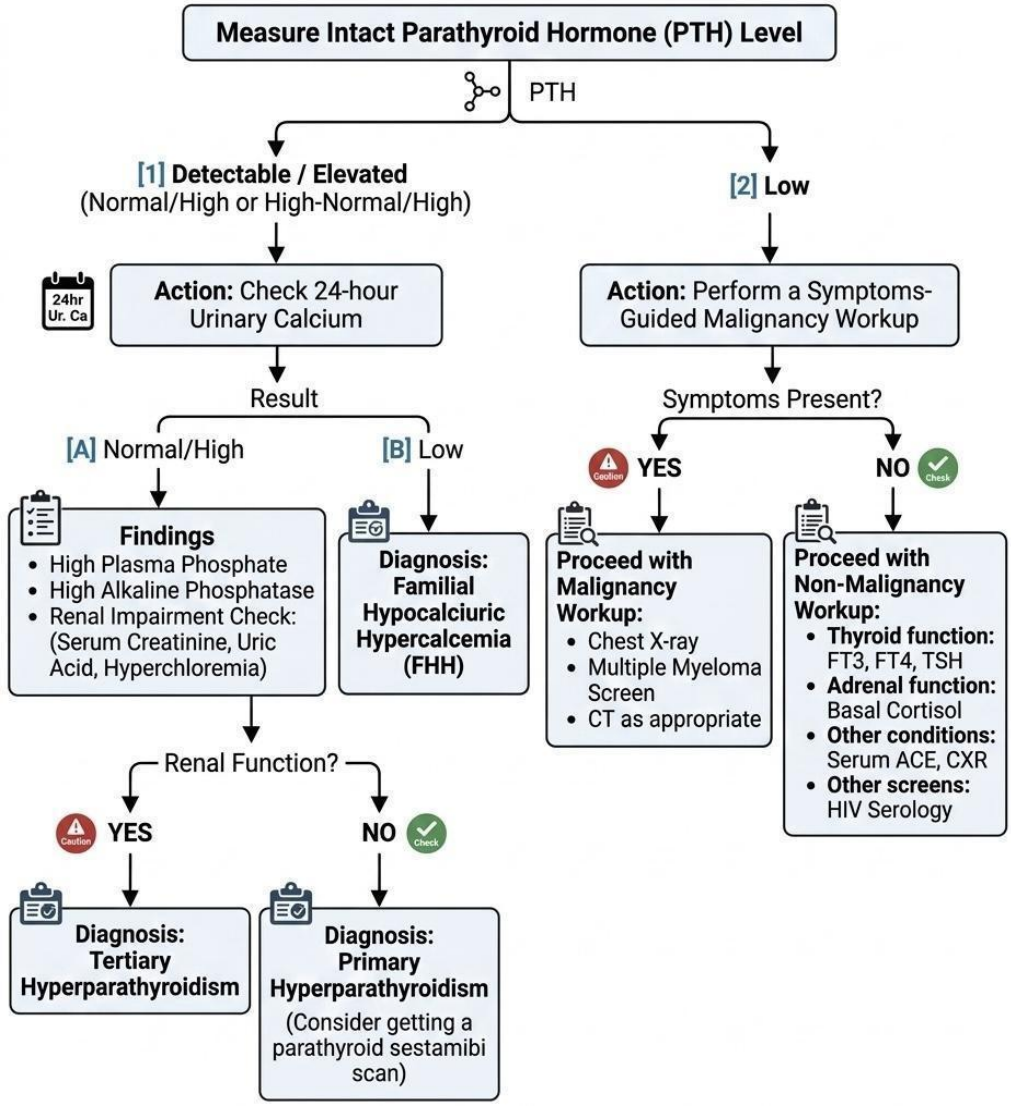
Sign –

- Parathyroid tumours are almost never palpable
- Hypertension
- Oedema
- Anaemia } Evidence of CKD

Family history of –

- Familial hypocalciuric hypercalcaemia
- Multiple endocrine neoplasia

Investigation:



(A) High / Normal iPTH

Investigation	Aim / Interpretation
24 hours urinary calcium level	Low → **Familial hypocalciuric hypercalcemia (FHH)**
	Normal / High → **Primary hyperparathyroidism**
	Normal / High → **Tertiary hyperparathyroidism**
Inorganic phosphate, ALP,	**Renal impairment →**
Serum creatinine, uric acid,	Abnormal / Elevated → **Tertiary hyperparathyroidism**
Electrolytes	Normal → **Primary hyperparathyroidism**
Parathyroid sestamibi scan	Localized parathyroid tumour → If surgery indicated

Cardiology

No	Topic	Page	Written	OSPE
1.	Examination			
2.	• Pulse		**	
3.	• JVP		***	
4.	• Heart Sounds		*	
5.	Investigation			
6.	• ETT		*	
7.	• NT Pro BNP			
8.	• CXR in Cardiology			
9.	• Echocardiography			
10.	• TEE			
11.	• Stress Echo		*	
12.	• MRI			
13.	• Cardiac Catheterization			
14.	Palpitations		***	
15.	Syncope		***	
16.	Chest pain		***	
17.	Cardiogenic Shock		*	
18.	HF		*	
19.	Arrythmia			
20.	• AF		**	
21.	• Temporary Pacemaker			
22.	• Implantable Defibrillator			
23.	• CRT			
24.	• ARNI			
25.	Cardiac arrest		**	
26.	Peripheral vascular disease			
27.	Acute limb Ischemia		*	**
28.	Aortic aneurysm			*
29.	Aortic dissection			
30.	Marfan Syndrome			*
31.	HTN		***	
32.	Accelerated HTN		**	
33.	Acute Rheumatic fever		**	
34.	Valvular heart disease			*
35.	• MS			*
36.	• MR			
37.	• AS			
38.	• AR			
39.	• TS			
40.	Infective endocarditis		**	***
41.	Coarctation of aorta			**
42.	Cardiomyopathy			
43.	• Hypertrophic Cardiomyopathy		*	**
44.	• Takotsubo Cardiomyopathy			
45.	Pericardial disease			
46.	• Tuberculous Pericarditis		**	***
47.	• Cardiac Tamponade		**	***

Previous Question

Examination

**Q: Describe different types of pulse with clinical importance *(July 2022)*
Q. Write in brief about the importance of pulse examination in a patient with fever. *(January 2026)*
***Q. Mention the importance of examination of jugular venous pulsation in clinical medicine. *(January 2023, 2025, July 2025)*
**Q. On examination of a 30-year-old woman, prominent V wave of Jugular venous pulsation was found. *(January 2024, 2025, July 2025)*
a) Formulate a list of causes of this abnormal finding.
b) Enumerate differentiating points between "a" wave and "v" wave of JVP.
Q. What information can you gather by examining the JVP?

Investigation

Q. Mention the indication, contraindication and high-risk findings of ETT.
Q. How will you evaluate elevated troponin-I?
Q. Give a brief account on NT-ProBNP
Q. Role of transoesophageal echocardiography in clinical practice
**Q. Write in brief about dobutamine stress echocardiography and mention its role in the evaluation of ischaemic heart disease. *(July 2022)*

Palpitation

Q. How would you evaluate a 35-year-old woman presented with palpitation?
***Q. A 30-year-old woman presented with recurrent episodes of palpitation associated with dizziness. How would you evaluate her clinically? *(January 2023)*
***Q.5 A 25-year-old man presented with recurrent episodes of palpitation for 3 (three) months. Make a list of clinical information hinting the underlying diagnosis. *(July 2023, January 2026)*

Syncope

***Q: How would you evaluate a 60-year-old male with recurrent syncope? *(January 2024, 2026, July 2025)*
Q. A 50-year-old man presented with recurrent syncope and his pulse rate was 40/minute. How would you evaluate and investigate him? *(July 2022)*

Chest Pain

**Q: How will you evaluate a patient presented with exertional chest pain? *(July 2025, January 2024)*
Q: How will you evaluate a patient presented with sudden severe chest pain?
**Q. A 54-year-old man presented with severe central chest pain for 4 (four) hours. His ECG and chest X-ray were normal. Write a check-list of further clinical information and investigations to diagnose the underline condition. *(January 2026)*
Q. A 55 years old diabetic male presented with central chest pain for 06 hours. How will you manage this patient at emergency department?

Effect of respiration	Minimal change (slight ↑ on inspiration)	Normally ↓ on inspiration (abnormal ↑ = Kussmaul's sign)
Effect of posture	Little change when limb raised	Height changes markedly with position (best at 45°)
Pressure occlusion	Not abolished by light pressure	Easily obliterated by gentle pressure on neck
Hepatojugular reflux	No significant change	Increases height of venous pulse
Abdominal compression	No effect	Positive hepatojugular reflux in right heart failure
Clinical reflection	Left ventricular systolic function & peripheral perfusion	Right heart filling pressures & function
Common abnormalities	Bounding, thready, pulsus paradoxus, bisferiens, etc.	Giant a waves, cannon a waves, absent y descent, elevated JVP, etc.

JVP

*****Q. Mention the importance of examination of jugular venous pulsation in clinical medicine *(January 2023, 2025, July 2025)***

Clinical Condition	Abnormalities in JVP
1) Heart failure – Particularly right heart failure	Elevated, sustained abdominojugular reflux >10 seconds
2) Pulmonary embolism	Elevated
3) Absent 'a' wave	Atrial fibrillation
4) Giant 'a' wave	Tricuspid stenosis
5) Cannon wave	Complete heart block
6) Giant 'v' wave	Tricuspid regurgitation
7) Superior vena caval obstruction	Elevated, loss of pulsation
8) Constrictive pericarditis	Elevated, Kussmaul's sign
9) Pericardial effusion	Elevated, prominent 'y' descent

****Q. On examination of a 30-year-old woman, prominent V wave of Jugular venous pulsation was found. *(January 2024, 2025, July 2025)***
a) Formulate a list of causes of this abnormal finding.
b) Enumerate differentiating points between "a" wave and "v" wave of JVP.

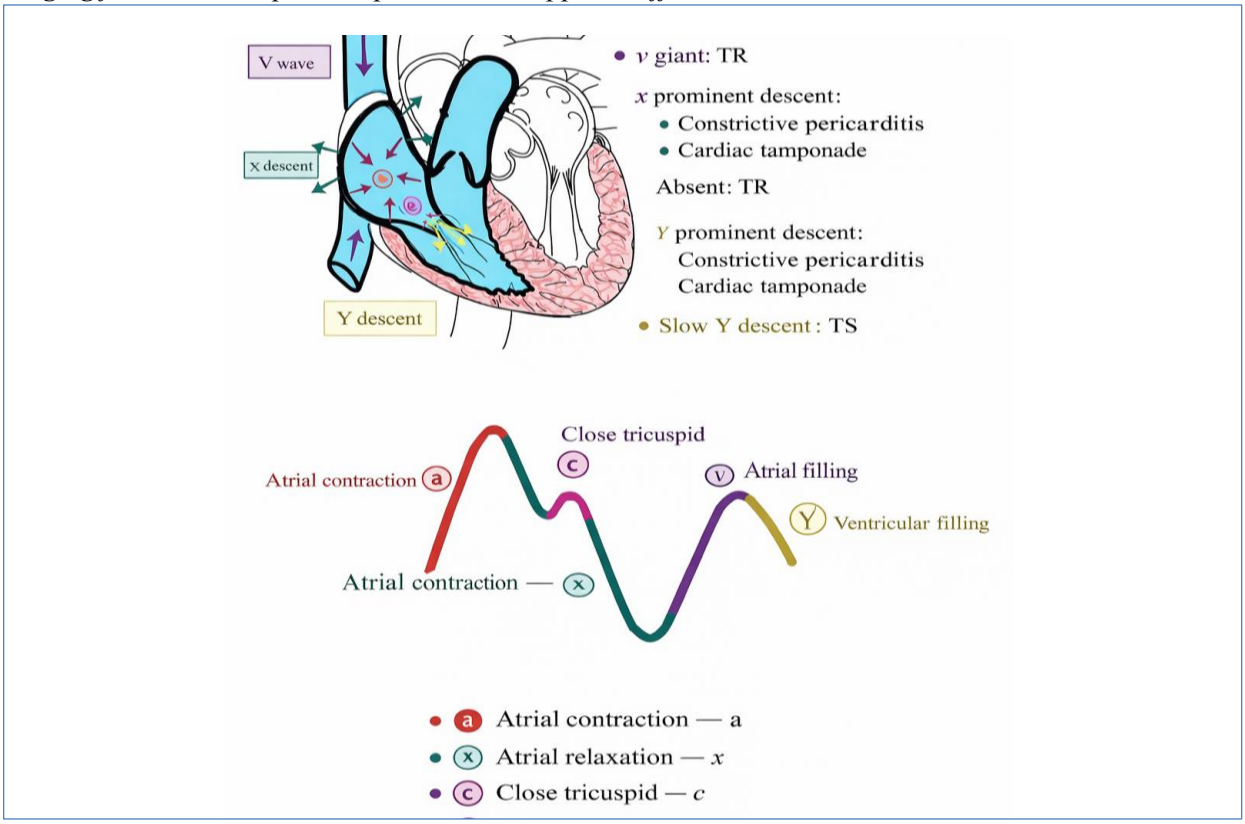
Differentiating Points Between "a" Wave and "v" Wave of JVP

Feature	"a" Wave	"v" Wave
Cause	Atrial contraction	Atrial filling against closed tricuspid valve
Timing	Just before first heart sound (S1)	After second heart sound (S2), during systole
Relation to ECG	After P wave	After T wave
Clinical Correlation	Prominent in tricuspid stenosis, pulmonary hypertension	Prominent in tricuspid regurgitation
Effect of Atrial Fibrillation	Absent (no organized atrial contraction)	Present

Haemodynamic effects of respiration

Parameter	Inspiration	Expiration
Jugular venous pressure	Falls	Rises
Blood pressure	Falls (up to 10 mmHg)	Rises
Heart rate	Accelerates	Slows
Second heart sound	Splits*	Fuses*

** Inspiration prolongs right ventricular ejection, delaying P_2 and shortens left ventricular ejection, bringing forward A_2; expiration produces the opposite effects.*



Q. What information can you gather by examining the JVP?

Information by examining the jugular venous pressure (JVP):
Raised JVP:

- CCF
- Constrictive pericarditis
- Cor pulmonale
- Cardiac tamponade
- Fluid overload
- SVC obstruction

Non-pulsatile:

- SVC obstruction

Respiratory

SL. No.	Topic	Page	Written	OSPE
1.	Investigations			
2.	Bronchoscopy			
3.	PET scan			
4.	CXR			***
5.	POCUS			
6.	Sputum examination		**	
7.	MT		*	
8.	IGRA		*	
9.	Cough		***	
10.	Shortness of breath		***	
11.	Pleural effusion		**	***
12.	Haemoptysis		***	
13.	Respiratory Failure		*	
14.	Pulmonary nodules			
15.	Bronchiectasis		*	***
16.	Lung abscess			***
17.	Asthma		***	
18.	COPD • GOLD guideline		***	
19.	Pneumonia		***	**
20.	Tuberculosis		***	***
21.	National TB Guideline		***	
22.	Fungal infection		*	**
23.	Carcinoma		**	***
24.	DPLD		**	***
25.	Sarcoidosis		***	***
26.	Connective tissue disease associated ILD		*	*
27.	Pulmonary embolism		***	***
28.	Pulmonary HTN		***	
29.	OSA		**	
30.	ARDS			
31.	Pneumothorax		**	***

Previous Question

Cough

Q. A 40 years old man has presented with cough for 6 months. Make a check list for etiological pertinent clinical information that you may obtained from general examination.
*****Q. A 55-year-old man, smoker, presented with cough and progressive shortness of breath for 6 months. Make a list of clinical information with expected findings to reach the diagnosis. *(July 2023)***
Q.A 30-year-old woman presented with fever and cough for the last 2 months. On Physical examination there is anaemia. CXR reveals mediastinal lymphadenopathy. How will you evaluate this woman to reach a diagnosis?
****Q. How will you evaluate a 30-year-old man with chronic cough with normal chest X-ray? *(July 2024)***
Q. A 45-year-old male smoker presents to you with three months' history of episodic nocturnal cough.
How would you proceed to make a logical diagnosis?

Shortness of breath

Q.Evaluation of acute breathlessness / with normal chest x-ray
*****Q. A 25-year-old woman presented with severe respiratory distress with normal chest on clinical examination. How would you evaluate her? *(July 2024)***
****Q. A 55-year-old man presented with acute breathlessness for 1 *(one)* day. On auscultation of his chest revealed no abnormalities. *(July 2025)***
a) What are the clinical possibilities of this presentation?
b) Mention the plan of investigations with expected findings.
*****Q. A 55-year-old smoker male presented with progressive exertional breathlessness for last 1 year.How would you evaluate him to make a diagnosis? *(Janu-2023)***

Pleural effusion

Q. A 65-year-old man presented with shortness of breath, cough for 3 *(three)* weeks. On clinical examination he had left sided pleural effusion. Make a list of further clinical information hinting the diagnosis. *(Jan-2026)
Q. How will you investigate a 60 years old man with right sided pleural effusion.
****Q. A 55-year-old man presented with right sided hemorrhagic pleural effusion. How would you assess clinically and investigate him? *(Jan–2023)***
****Q: How would you evaluate a 35 years old woman presenting with bilateral pleural effusion *(July–2019)***
Q. Treatment of malignant pleural effusion?

Hemoptysis

Q. Management of acute massive haemoptysis
*****Q. 39 year old man presented with haemoptysis with normal chest X-ray. How would you evaluate him?**

Q. A 40 years old man has presented with cough for 6 months. Make a check list for etiological pertinent clinical information that you may obtained from general examination.

Possibilities:

- **Pulmonary tuberculosis**
- **Bronchial Asthma**
- **Chronic obstructive pulmonary disease (COPD)**
- **Bronchiectasis**
- **Post nasal drip**
- **GERD**
- **DPLD**
- **Drug induced – ACEI**
- **Bronchial carcinoma**

Evaluation:

- Low grade irregular fever with evening rise of temperature - Weight loss - Lymphadenopathy - Contact with TB patient	**Pulmonary tuberculosis**
- Breathlessness - Chest tightness - Worse at night - Wheeze - Family history of Asthma - Seasonal variation - Vehicular with prolonged expiration, Rhonchi	**Bronchial asthma**
- History: smoker, chronic cough, exertional breathlessness, h/o infective exacerbation, sputum production - Signs: cachexia, pursed lip breathing, tripod position, vesicular with prolonged expiration, rhonchi, Cyanosis. - Evidence of CO2 retention - Evidence of cor pulmonale	**COPD**
Dry, distressing, persistent cough: - Breathlessness - Cyanosis - Clubbing - Bilateral basal end inspiratory crepitation not altered by coughing	**DPLD**
Chronic persistent daily cough: - Profuse purulent sputum - Chest pain - Massive hemoptysis - Halitosis - Weight loss - Fever, malaise, anorexia Signs: clubbing, central cyanosis, coarse crepitations altered by cough	**Bronchiectasis**

Lower Limb Doppler Ultrasound:
- Detects DVT as source of emboli in suspected PE

Spirometry:
- Obstructive pattern in asthma (low FEV_1, reduced FEV_1/FVC), reversible with bronchodilator

Urinalysis (for ketones):
- Positive in diabetic ketoacidosis, indicating ketonuria

Serum Lactate:
- Elevated in lactic acidosis, a form of metabolic acidosis

****Q. A 55-year-old man presented with acute breathlessness for 1 (one) day. On auscultation of his chest revealed no abnormalities. *(July 2025)***
a) What are the clinical possibilities of this presentation?
b) Mention the plan of investigations with expected findings.

a) Differential diagnoses
In a 55-year-old man with acute breathlessness for 1 day, normal chest auscultation:

- **Pulmonary embolism (PE)**
- **Acute severe asthma (silent chest)**
- **Metabolic acidosis (DKA, renal failure, sepsis)**
- **Severe anemia**
- **Anxiety / panic attack**
- **Acute myocardial infarction (AMI)**

b) Plan of Investigations with Expected Findings
First-line Investigations

Investigation	Expected Findings (short)
Pulse oximetry / ABG	PE: hypoxemia; Asthma: hypoxemia ± hypercapnia; Metabolic acidosis: ↓pH ↓HCO_3; Anemia/Anxiety: near-normal; MI: mild hypoxemia
ECG	PE: sinus tachy, S1Q3T3; Asthma/Anxiety: sinus tachy; DKA/Sepsis: tachycardia; MI: ST/T changes
Chest X-ray	PE/Anemia/Anxiety: normal; Asthma: hyperinflated; MI: pulmonary congestion/edema
CBC	Anemia: ↓Hb; Sepsis: leukocytosis; others: normal
Blood glucose, ketones, electrolytes, renal function	DKA: ↑glucose/ketones; Renal failure: ↑urea/creatinine; Sepsis: acidosis/lactate ↑
Troponin	↑ in MI or massive PE

Second-line / Directed Investigations

Investigation	Expected Findings
D-dimer	Elevated in PE (but nonspecific)
CT Pulmonary Angiography (CTPA)	Diagnostic for PE; intraluminal filling defect
Echocardiography	RV dilation/strain in PE, LV dysfunction/wall motion abnormality in MI
BNP / NT-proBNP	Elevated in LV dysfunction/MI
Arterial venous lactate	Elevated in sepsis/metabolic acidosis
Thyroid profile (if arrhythmia suspected with MI-like picture)	Abnormal in thyrotoxic AF
Spirometry/peak flow (later, if stable)	↓ Peak flow in asthma
Iron studies (if anemia suspected chronic)	↓ Iron, ↓ ferritin in iron deficiency anemia

Pleural effusion

Light's criteria for distinguishing pleural transudate from exudate (OSPE)

Exudate is likely if one or more of the following criteria are met:

- **Pleural fluid/serum protein ratio >0.5**
- **Pleural fluid LDH/serum LDH ratio >0.6**
- **Pleural fluid LDH > two-thirds of the upper limit of normal serum LDH**

(LDH = lactate dehydrogenase)

(Ref:Davidson 24th P-497,Box:17.14)

Causes of pleural effusion (OSPE)

Common causes:

- Pneumonia *(parapneumonic effusion)*
- Tuberculosis
- Pulmonary infarction*
- Malignant disease
- Cardiac failure*
- Subdiaphragmatic disorders *(subphrenic abscess, pancreatitis etc)*

Uncommon causes:

- Hypoproteinaemia* *(nephrotic syndrome, liver failure, malnutrition)*
- Connective tissue disease *(particularly systemic lupus erythematosus)*
- Post-myocardial infarction syndrome
- Acute rheumatic fever
- Meigs syndrome *(ovarian tumour with pleural effusion)*
- Myxoedema*
- Asbestos-related benign pleural effusion

* *May cause bilateral effusions.*

(Ref: Davidson 24th P-496, Box:17.12)

Causes of right sided pleural effusion:

- Liver abscess
- Meig's syndrome
- Dengue haemorrhagic fever

Causes of left sided pleural effusion:

- Acute pancreatitis
- Rheumatoid arthritis
- Dressler's syndrome
- Oesophageal rupture (Boerhaave syndrome)
- Dissecting aneurysm

Q. A 65-year-old man presented with shortness of breath, cough for 3 (three) weeks. On clinical examination he had left sided pleural effusion. Make a list of further clinical information hinting the diagnosis. *(January 2026)

From this scenario differentials are :left sided pleural effusion due toPossibilities

- Bronchial carcinoma
- Tuberculosis
- Lymphoma
- Parapneumonic effusion

Evaluation:

• Elderly age • Progressive dyspnoea • Persistent cough • Significant weight loss • Chest pain (dull / pleuritic) • Hemoptysis • Smoking history • Hoarseness / dysphagia • History of known malignancy	**Malignant Pleural Effusion**
• Low-grade fever (evening rise) • Night sweats • Weight loss, anorexia • Chronic cough ± scanty sputum • Past or contact history of TB • Young contact / overcrowded living • No cardiac symptoms	**Tuberculous Pleural Effusion**
• Progressive shortness of breath • Weight loss • Low-grade fever (may mimic TB) • Night sweats (B symptoms) • Painless lymphadenopathy (cervical / axillary / inguinal) • Hepatosplenomegaly • Chest pain usually mild or absent • History of recurrent infections	**Lymphoma**
• Acute onset fever with chills • Productive cough • Pleuritic chest pain • Toxic look • History of recent pneumonia • Rapid worsening of dyspnoea	**Parapneumonic Effusion**

Gastroenterology

No	Topic	Page	Written	OSPE
1.	Investigation			
2.	Dysphagia		***	
3.	Dyspepsia		***	
4.	Vomiting		*	
5.	Upper GI bleeding		**	
6.	Lower GI bleeding		*	
7.	Diarrhoea / Malabsorption		***	*
8.	Weight loss		*	
9.	Constipation			
10.	Abdominal pain		*	
11.	Oral ulcer		*	**
12.	Hiccough			
13.	GERD			
14.	Barrett's			
15.	CA oesophagus			*
16.	Achalasia cardia			*
17.	Duodenal ulcer			*
18.	Gastric CA			*
19.	Zollinger Ellison syndrome			
20.	Coeliac disease		**	**
	● Dermatitis Herpetiformis			**
21.	SIBO		*	
22.	Whipple disease			
23.	Miscellaneous disease of small intestine			
	● Protein Losing Enteropathy			
	● Intestinal Lymphangiectasia			
	● Lactose Intolerance			
	● Meckel's Diverticulum			
	● Bile Acid Diarrhoea			
24.	Colorectal CA			
	● Familial Adenomatous Polyposis			
	● Peutz Jeghers Syndrome			**
25.	IBD		***	***
26.	IBS		**	
27.	Acute pancreatitis		***	**
28.	Autoimmune Pancreatitis			
29.	Chronic pancreatitis		**	***
30.	Adenocarcinoma Of the Pancreas		*	***

Previous Question

Investigations

Q. Describe the role of UGE in evaluating an adult patient presenting with acute abdomen.
Describe the role of upper GI endoscopy in clinical practice.
Q. Role of endoscopic ultrasound in medical disorders

Dysphagia

Q. A 60-year-old man presented with dysphagia for 3 (three) months. How would you evaluate him? *(Jan-2025)
Q. A 55-year-old man presents with difficulty in swallowing for 6 months. Mention the plan of investigations with reasoning of their selection. *(Jul-2024)

Dyspepsia

Q. Mention the alarming signs of dyspepsia. How would you investigate a 60-year-old man presenting with dyspepsia? *(Jan-2026)*
Q. How will you clinically evaluate a 36-year-old male with dyspepsia and repeatedly normal upper GI endoscopies?

Vomiting

Q. How would you evaluate a 25 years old woman presenting with vomiting for the last two weeks

Upper GI bleeding

Q. A 45-year-old woman presented with massive hematemesis. Write the principles of management for this patient. *(Jan-2023)*

Lower GI bleeding

*Q. A 50 year old man presented with chronic lower gastrointestinal bleeding. How would you evaluate him

Malabsorption / Diarrhoea

Q. What is steatorrhea? Enumerate the causes of malabsorption or
Q. How would you evaluate a patient presenting with malabsorption syndrome or
***Q. A 30 year old woman has presented with chronic diarrhoea. Give a checklist of clinical information to reach a diagnosis or**
Q. Enumerate the plan of investigation with findings for a patient with chronic diarrhoea *(July-2019)
Q. A 60-year-old woman presented with acute watery diarrhea with altered mental state. How would you assess her clinically and treat her? *(Jul-2022)*
Q. A 55-year-old woman presented with chronic diarrhea for 1 (one) year. *(Jul-2025)
a) Mention relevant points from history
b) Write important investigations that may aid in the diagnosis with rationale.

IBD

(OSPE)

Ulcerative Colitis (UC)

- Always starts in rectum (proctitis) → continuous proximal spread (no skips).
- Inflammation limited to mucosa; spares deeper layers.
- Hallmarks: Crypt abscesses, pseudopolyps (chronic), loss of goblet cells, crypt distortion.
- Symptoms: Bloody diarrhea, mucus, rectal bleeding, tenesmus.
- Severe: Toxic (fever, tachycardia, peritoneal signs).
- Cancer risk: Dysplasia → colorectal cancer.

Crohn's Disease (CD)

- Anywhere GI tract (mouth to anus); most common: terminal ileum + right colon.
- **Skip lesions, transmural inflammation, deep fissuring ulcers → cobblestone mucosa.**
- Hallmarks: Granulomas, fistulae, abscesses, perianal disease, rectal sparing.
- Symptoms: Abdominal pain, watery diarrhea (non-bloody), weight loss, malabsorption.
- Complications: Obstruction, fistulae, abscesses.

Comparison of ulcerative colitis and Crohn's disease (OSPE)

Feature	Ulcerative colitis	Crohn's disease
Age group	Any	Any
Gender	M = F	Slight female preponderance
Incidence	Stable	Increasing
Ethnic group	—	Any, more common in Ashkenazi Jews
Genetic factors	HLA-DR*103; onion skin barrier function (HNF4A, LAMB, CDH1)	Defective innate immunity and autophagy (NOD2, ATG16L1, IRGM)
Risk factors	More common in non-smokers	More common in smokers
Anatomical distribution	Colon only; begins at anorectal margin with variable proximal extension	Any part of gastrointestinal tract; perianal disease common; patchy distribution (skip lesions)
Extra-intestinal manifestations	Common	Common
Presentation	Bloody diarrhoea, tenesmus	Variable; pain, diarrhoea, weight loss common
Histology	Inflammation limited to mucosa; crypt distortion; cryptitis; loss of goblet cells	Submucosal or transmural inflammation; deep fissuring ulcers; granulomas
Management	5-ASA (suppositories, azathioprine, biologic therapy [anti-TNF, anti-α4β7 integrin, anti-p40, JAK inhibitors]); colectomy is curative	Glucocorticoids, azathioprine, biologic therapy (anti-TNF, anti-α4β7 integrin, anti-p40, nutritional therapy); smoking cessation; surgery for complications not curative; 5-ASA is not effective
(5-ASA = 5-aminosalicylic acid; TNF = tumour necrosis factor)		

(Ref: Davidson 24th P-836, Box:23.61)

*****Q. A 31-year-old woman presented with passage of blood mixed loose stool with abdominal cramps for last 6 months. Write important clinical clues and plan of investigations to reach the underlying cause.** ***(Jul-2024)***

*****Q. A 32-year-old man presented with recurrent bloody diarrhea for last 2 months. Write a plan of investigations with expected findings to reach the underlying diagnosis.** ***(Jan-2025, 2023, Jul-2024, Jan-2022)***

Possibilities:

From above scenario most likely diagnosis is –

• Ulcerative colitis

Other possibilities –
• Crohn's colitis
• Diverticulitis
• NSAID induced colitis
• Carcinoma colon

Evaluation:

Bloody diarrhea with mucus Tenesmus Abdominal cramps First attack usually not severe Relapses and remissions Provoked relapse by emotional stress, intercurrent infection Severe case – Toxic, fever, tachycardia Anorexia, malaise, weight loss Non smoker or ex-smoker	**Ulcerative colitis**
Bloody diarrhea Constipation During attack – Local tenderness, guarding, rigidity, palpable mass Intestinal obstruction	**Diverticulitis**
Symptoms depend on site of involvement – **Left colon:** Per-rectal bleeding, intestinal obstruction **Right colon:** Anaemia, altered bowel habit Palpable mass Hepatomegaly	**Carcinoma colon**
H/O long term NSAID use Concomitant corticosteroid use Bloody diarrhea Abdominal pain Hematemesis, melena	**NSAID induced colitis**
Bloody diarrhea with mucus No tenesmus	**Crohn's disease / Crohn's colitis**

Causes of pancytopenia (OSPE)

Bone marrow failure
- Hypoplastic/aplastic anaemia (p. 978): inherited, idiopathic, viral, drugs

Bone marrow infiltration
- Acute leukaemia
- Myeloma
- Lymphoma
- Carcinoma
- Haemophagocytic syndrome
- Myelodysplastic syndromes

Ineffective haematopoiesis
- Megaloblastic anaemia
- Acquired immunodeficiency syndrome (AIDS)

Peripheral pooling/destruction
- Hypersplenism: portal hypertension, Felty syndrome, malaria, myelofibrosis
- Systemic lupus erythematosus

(Ref: Davidson 24th P-939,Box:25.16)

Q. How will you evaluate a patient with platelet count about 65000/mm³

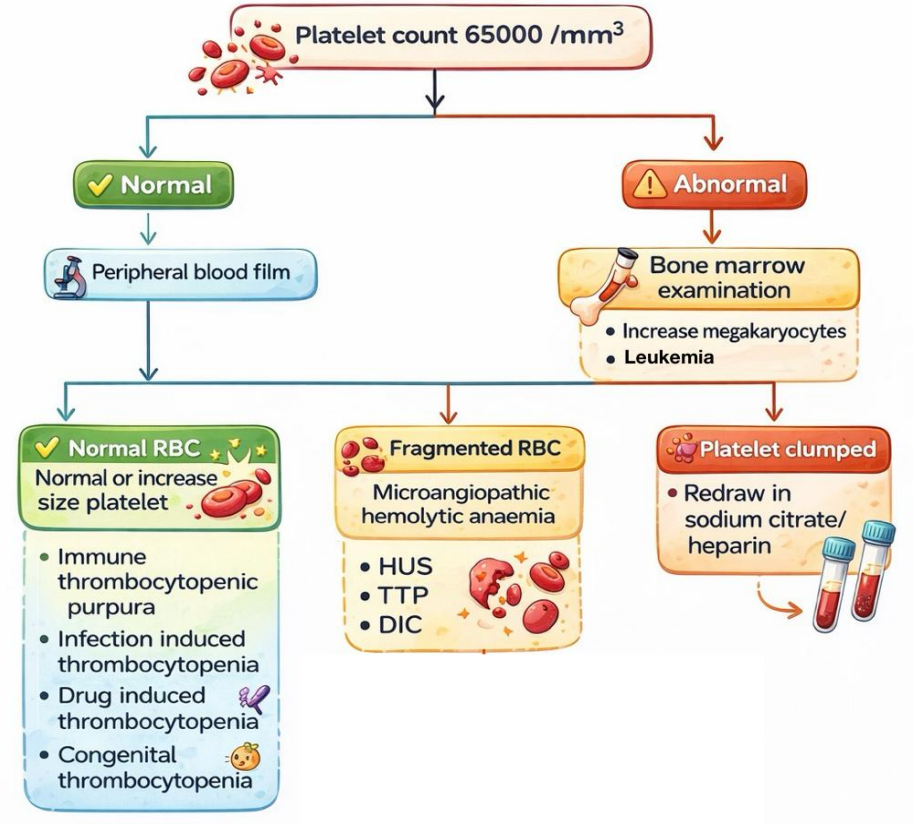
Possibilities

- **Immune thrombocytopenic purpura (ITP)**
- **Dengue hemorrhagic fever (DHF)**
- **Systemic lupus erythematosus (SLE)**
- **Acute leukemia**
- **Aplastic anemia**
- **Disseminated intravascular coagulation (DIC)**
- **Vitamin B_{12} and/or folate deficiency (Megaloblastic anemia)**

Evaluation

• Malaise • Breathlessness • Paraesthesia • Sore mouth • Smooth tongue • Glossy and stocking paraesthesia • Loss of ankle jerk • Optic atrophy	• Megaloblastic anemia

Investigation:



Purpura

Q: Enumerate the causes of purpura

Non-palpable

- Immune thrombocytopenic purpura (ITP)
- Dengue hemorrhagic fever
- Disseminated intravascular coagulation
- Systemic lupus erythematosus
- Acute leukemia
- Aplastic anemia
- Drugs — cytotoxic, antiplatelet
- Gestational thrombocytopenia
- HELLP syndrome
- Thrombotic thrombocytopenic purpura
- Hemolytic uremic syndrome (HUS)

Palpable

- Henoch–Schönlein purpura (HSP)
- Granulomatosis with polyangiitis (GPA)
- Eosinophilic granulomatosis with polyangiitis (EGPA)
- Microscopic polyangiitis (MPA)
- Cryoglobulinemic vasculitis
- Polyarteritis nodosa (PAN)

*****Q. A 24-year-old woman presented with pallor and recurrent episodes of jaundice for 3 (three) months. Write a list of clinical information and investigations hinting the underlying diagnosis. (Jan-26)**

From the scenario my differentials are:

- **Autoimmune hemolytic anemia (AIHA)**
- **Hereditary spherocytosis**
- **G6PD deficiency**
- **Thalassemia (intermedia/major)**
- **Wilson's disease**

• Pallor + intermittent jaundice • Mild fever, fatigue • Splenomegaly • History of autoimmune disease / drugs • No family history usually	**Autoimmune Hemolytic Anemia (AIHA)**
• Pallor + jaundice since childhood • Family history positive • Splenomegaly • Pigment gallstones • No drug trigger	**Hereditary Spherocytosis**
• Episodic jaundice after: ✓ Infection ✓ Drugs (antimalarial, sulfa) ✓ Fava beans • Dark urine during attacks • Male predominance (but females can be affected)	**G6PD Deficiency**
• Pallor out of proportion to anemia • Jaundice • Bone deformities • Hepatosplenomegaly • Family history common	**Thalassemia (Intermedia/Major)**
• Young adult consanguinity/family history of liver/neurologic issues. • Pallor + jaundice • Kayser-Fleischer (KF) rings (slit-lamp); subtle neurologic/psychiatric signs (tremor, personality change).	**Wilson disease**

First-Line Investigations

- CBC → ↓ Hb, ↑ reticulocyte count
- Peripheral blood film
- LDH → ↑

- Haptoglobin → ↓
- Urine urobilinogen → ↑
- Liver function tests
- Serum bilirubin → ↑ unconjugated
- Slit-lamp exam for KF rings

Second-Line investigation

Investigation	Suspected Condition
Direct Coombs test (DAT)	AIHA
Osmotic fragility / EMA binding test	Hereditary spherocytosis
G6PD enzyme assay	G6PD deficiency
Hb electrophoresis	Thalassemia
24-hour urinary copper (post-penicillamine challenge). Serum free copper	elevated in Wilson's disease

Q: Write down the laboratory evidence of intravascular hemolysis.

Laboratory evidence of intravascular hemolysis:

- Decrease hemoglobin
- Decrease haptoglobin
- Increase unconjugated bilirubin
- Increase LDH
- Increase reticulocytes
- Increase urinary urobilinogen
- Increase methaemalbumin
- Positive urinary hemosiderin
- Haemoglobinuria

Aplastic anaemia

Causes of secondary aplastic anaemia (OSPE)

Drugs

- Cytotoxic drugs
- Antibiotics (chloramphenicol, sulphonamides)
- Antirheumatics (penicillamine, gold, phenylbutazone, indometacin)
- Antithyroid drugs (carbimazole, propylthiouracil)
- Anticonvulsants
- Immunosuppressants

Chemicals

- Benzene, toluene (glue sniffing)
- Insecticides (chlorinated hydrocarbons, organophosphates and carbamates)

Radiation
Viral hepatitis
Pregnancy
Paroxysmal nocturnal haemoglobinuria

(Ref: Davidson 24th P-945-979, Box:25.62)

Sickle-cell anaemia

(OSPE)

Genetics & Types

- Caused by **single point mutation**: glutamic acid → valine at position 6 of **β-globin chain**.
- **Autosomal recessive**.
- **Homozygous (SS)** → sickle-cell disease (full clinical syndrome, only HbS).
- **Heterozygous (AS)** → sickle-cell trait (usually asymptomatic, mixture of HbA + HbS).

Epidemiology

- Very high frequency in **tropical Africa** (>20% SS, ~8% trait).
- **Heterozygote advantage**: sickle-cell trait protects against severe **falciparum malaria** in early childhood → explains high prevalence in malaria-endemic areas.
- **Homozygotes (SS)** do **not** have this malaria resistance.

Pathogenesis

- Deoxygenated **HbS polymerises** → forms **tactoids** → distorts RBC → **sickled cells**.
- Reversible on reoxygenation; repeated sickling → **irreversibly sickled cells**.
- Sickled cells: rigid, membrane damage, splenic removal, vaso-occlusion.
- **HbF strongly inhibits** polymerisation (protective).
- Triggers: **hypoxia, acidosis, dehydration, infection**.

Clinical Features / Crises

- **Vaso-occlusive (painful) crisis** — most common: severe bone pain, infarction (dactylitis in children, long bones/ribs/pelvis in adults), fever, tachycardia.
- **Stroke** — devastating (10–15% of children); screen with **transcranial Doppler**; prevent with transfusion/hydroxycarbamide/HSCT.
- **Acute chest syndrome** — leading cause of adult death: fever, chest pain, dyspnoea, new infiltrate; often follows vaso-occlusive crisis + fat emboli.
- **Sequestration crisis** — sudden organ swelling (spleen in children → severe anaemia/shock/death; liver in adults); autosplenectomy common in adults.
- **Aplastic crisis** — parvovirus B19 infection → severe transient red cell aplasia → profound anaemia, low reticulocytes (unlike other crises).

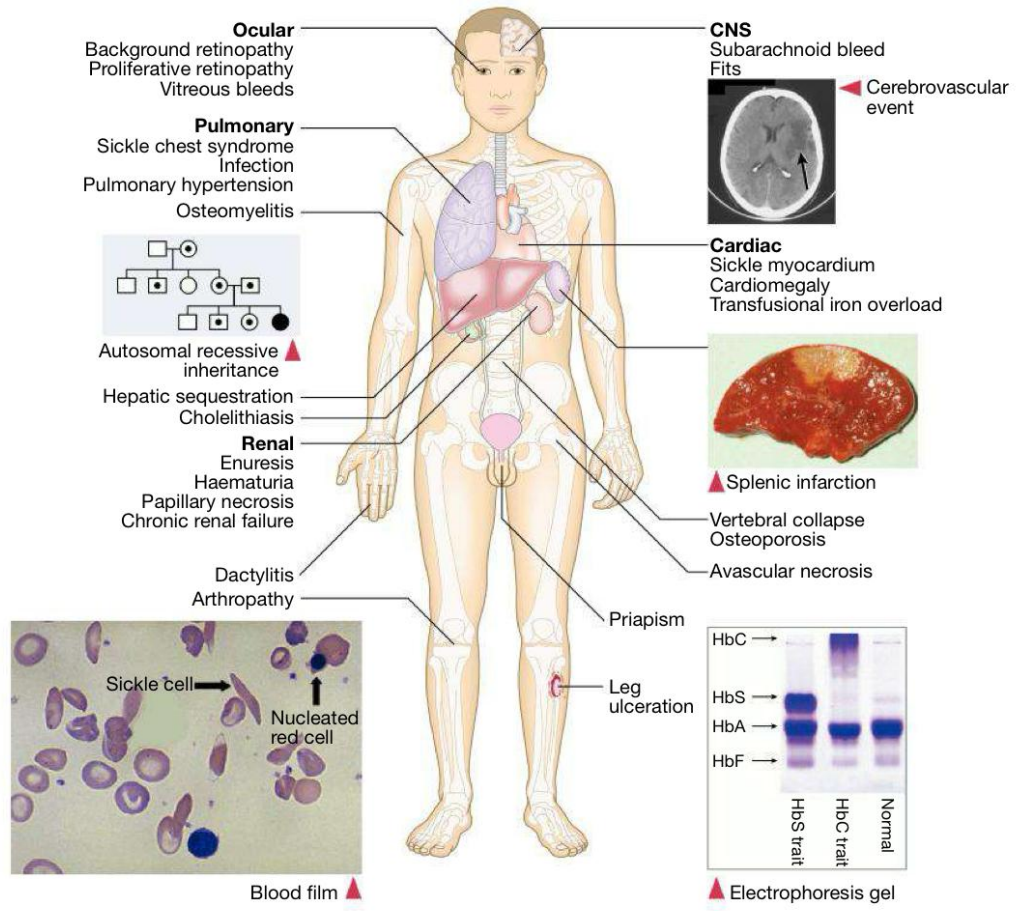


Fig. 25.24 Clinical and laboratory features of sickle-cell disease.

Investigations

- Anaemia (Hb ~60–80 g/L), **reticulocytosis**, sickle cells + target cells on film, hyposplenism features.
- **Solubility test** (sodium dithionite) → turbid with HbS.
- Definitive: **Hb electrophoresis** → HbS predominant, no HbA, ↑ HbF (2–20%).

Management

- **Prophylaxis**: daily **folic acid**, hyposplenism management (vaccinations: pneumococcal, etc.), influenza vaccine.
- **Vaso-occlusive crisis**: aggressive hydration, oxygen, strong analgesia (opiates), antibiotics.
- **Transfusion**: simple top-up or exchange (keep HbS <30% in severe/recurrent cases).
- **Hydroxycarbamide** (hydroxyurea) → ↑ HbF → ↓ crises frequency (used in recurrent severe cases).
- **Curative option**: allogeneic **HSCT** (HLA-matched sibling).
- **Pregnancy**: high-risk — multidisciplinary care; ↑ crises, placental issues, thromboembolism.