DNB Dec 2020 - Paper II

PART A

1. a) Enumerate causes of inferior rib notching. — **2 marks**
   b) Describe X-ray, CT, and MRI findings in **Coarctation of Aorta**. — **(2 + 2 + 2 marks)**
   c) Briefly discuss the role of **Interventional Radiology** in its management. — **2 marks**

---

2. a) **Radiological patterns** of pneumonias on **Chest CT** in immunocompromised patients. — **5 marks**
   b) **Imaging features** of fungal infections. — **5 marks**

PART B

3. Enumerate the causes of hemoptysis in an adult patient. — **2 marks**
   Briefly discuss:

- Indications — **2 marks**
- Techniques — **4 marks**
- Complications — **2 marks**
  of radiological interventions in hemoptysis.

---

4. a) **Arterial and venous anatomy** of lower limb (with diagram). — **5 marks**
   b) Imaging features of **Deep Venous Thrombosis (DVT)**. — **5 marks**

PART C

5.

- Define **Acute Aortic Syndrome** — **1 mark**
- Classify **Aortic Dissection** — **2 marks**
- Describe:
  - Imaging features on **CT Angiography** — **4 marks**
  - Management — **3 marks**

---

6. Role of:

- Nuclear Imaging — **2 marks**
- Cardiac MRI — **4 marks**
- MDCT — **4 marks**
  in evaluation of **Ischemic Heart Disease**

PART D

7. a) Enumerate causes of **Neonatal Respiratory Distress** — **2 marks**
b) Describe in detail:

- Pathophysiology — **2 marks**
- Imaging findings in **Hyaline Membrane Disease** — **6 marks**

---

8. Draw a schematic diagram describing **Anatomy of Diaphragm** — **2 marks**
Imaging features of **Diaphragmatic Injury** on:

- Chest X-ray — **2 marks**
- Ultrasound — **2 marks**
- CT — **2 marks**
- MRI — **2 marks**

PART E

9.

- Describe anatomy of **Mediastinum** (with diagram) — **5 marks**
- Discuss imaging features of **Anterior Mediastinal Masses** — **5 marks**

10.

- Define **Pulmonary Edema** — **2 marks**
- Describe its pathophysiology — **2 marks**
- Enumerate causes — **2 marks**
- Imaging features — **4 marks**

Q1. a) Enumerate causes of inferior rib notching.

b) Describe X-ray, CT and MRI findings in Coarctation of aorta.

c) Briefly discuss role of interventional radiology in its management.

Answer

Inferior Rib Notching

Definition: Smooth concave erosions of the **inferior rib margins** on chest X-ray due to **chronic enlargement of intercostal arteries or veins**.

1. Most Common Cause
Coarctation of the Aorta

- **Mechanism**: Narrowing distal to origin of left subclavian artery → increased collateral flow via internal thoracic → intercostal arteries → inferior rib erosion.
- **Laterality**: Usually **bilateral**, sparing **1st and 2nd ribs** (supplied by costocervical trunk, not affected).
- **Key Imaging Tip**: Look for figure-3 sign on CXR and post-stenotic aortic dilatation.

2. Vascular Causes

- **Arteriovenous Malformations / Fistulas**
  ↑ arterial flow through intercostals → bone erosion.
- **Aortic Aneurysm**
  Mass effect → collateral circulation activation.
- **Tetralogy of Fallot (rare)**
  Secondary to increased collaterals (especially post-shunt).
- **Blalock–Taussig Shunt**
  Iatrogenic ↑ flow via subclavian–pulmonary artery connection.
- **Aortic Coarctation (repaired)**
  Persistent collaterals may still notch ribs.
- **Subclavian Steal Syndrome**
  Retrograde vertebral flow → intercostal artery hypertrophy.

3. Non-Vascular Causes

- **Neurofibromatosis Type 1 (NF1)**
  Intercostal neurofibromas cause rib scalloping—notching may be irregular/asymmetric.
- **Superior Vena Cava Syndrome (rare)**
  Collaterals via azygos & intercostals → venous erosion.

4. Key Radiographic Points

- **Location**: Inferior rib margin, typically ribs 3–9.
- **Pattern**:
    - **Bilateral, symmetric** → usually vascular (e.g., coarctation).
    - **Unilateral** → local cause (e.g., post-surgical shunt, neurofibroma).
- **First two ribs spared** in vascular causes (different blood supply).
- **Superior rib notching** suggests venous etiology (e.g., SVC obstruction).

Coarctation of the Aorta (CoA)

1. X-ray Findings

Feature	Description	Pathophysiology
Inferior Rib Notching	Concave erosions of inferior margins of ribs 3–9	Hypertrophied intercostal arteries (collateral circulation) erode bone
Figure-3 Sign	Pre-stenotic dilatation → coarctation → post-stenotic dilatation	Produces “3” contour on mediastinum
LVH Shadow	Enlarged cardiac silhouette with LV prominence	Increased afterload from narrowing

2. CT Angiography

- **Direct Coarctation Visualization**: Focal narrowing, usually distal to left subclavian artery (near ligamentum arteriosum).
- **Post-stenotic Dilatation**: Immediately distal aortic enlargement.
- **Collateral Vessels**: Hypertrophied internal mammary and intercostal arteries.
- **Cardiac/Aortic Measurements**: Exact lumen diameter, cross-sectional area, and gradients.
- **Associated Findings**: Bicuspid aortic valve, aneurysm formation.

3. MRI / MR Angiography

- **High-Resolution Narrowed Segment**: Accurate length and severity measurement.
- **Flow Dynamics**: Phase-contrast MRI → velocity mapping, pressure gradients, turbulence visualization.
- **Collateral Mapping**: Internal mammary, scapular, and intercostal artery networks.
- **Cardiac Function**: LV wall thickness, ejection fraction, diastolic/systolic performance.
- **No Radiation**: Preferred in younger patients for follow-up.

4. Interventional Radiology
Balloon Angioplasty

- **Indications**: Native CoA (older child/adolescent) or recoarctation.
- **Pros**: Minimally invasive, quick recovery.
- **Cons**: Risk of dissection, aneurysm, or re-stenosis.

Stent Placement

- **Indications**: Adolescents/adults, recurrent CoA, long segment narrowing.
- **Pros**: More durable lumen patency vs. balloon alone.
- **Cons**: Needs larger access vessel; possible stent migration/fracture.

5. Comparison – Imaging Modality Roles

Modality	**Strength**	**Limitation**
X-ray	Suggestive signs (rib notching, figure-3)	No direct lumen visualization
CT Angio	Excellent lumen & collateral detail	Radiation exposure
MRI / MRA	Flow quantification + anatomy without radiation	Longer scan time, less available
IR	Definitive treatment (balloon/stent)	Invasive, risk of complications

---

Q2. a) Radiological patterns of pneumonias on chest computed tomography in immunocompromised patients.

b) Imaging features of fungal infections.

Answer

CT Patterns of Pneumonia in Immunocompromised Patients

1. Bacterial Pneumonia

CT Feature	Description	Typical Pathogens
Lobar Consolidation	Dense, homogeneous, often segmental/lobar; **air bronchograms** common	*S. pneumoniae*, *H. influenzae*
Cavitation	Irregular low-attenuation areas within consolidation	*S. aureus*, *P. aeruginosa*, anaerobes
Pleural Effusion	Often reactive; may be empyema	Gram-positive/negative bacteria

2. Viral Pneumonia

CT Feature	Description	Typical Pathogens
Ground-Glass Opacities (GGO)	Patchy/diffuse increased attenuation without vessel obscuration	CMV, RSV, influenza
Interstitial Thickening	Reticular or septal thickening	CMV, adenovirus
Peribronchial Thickening	Bronchial wall prominence ± peribronchial cuffing	RSV, influenza

3. Fungal Pneumonia

a. Invasive Aspergillosis

Feature	Early	Late
Halo Sign	Nodule + surrounding GGO (hemorrhage)	—

Air Crescent Sign	—	Crescent-shaped air within necrotic core

b. Pneumocystis jirovecii Pneumonia (PJP)

- **Diffuse GGO**: Often perihilar, sparing periphery initially.
- **Crazy-Paving Pattern**: GGO + interlobular septal thickening.
- ± thin-walled cysts.

c. Cryptococcosis

- **Nodules**: ± cavitation.
- **Mass-like Consolidation**: May mimic tumor.

4. Mycobacterial Pneumonia

a. Tuberculosis (TB)

- **Upper Lobe Predominance**: Consolidation, cavitation, satellite nodules.
- **Tree-in-Bud Pattern**: Centrilobular nodules + branching opacities (endobronchial spread).

b. Non-Tuberculous Mycobacteria (NTM)

- **Nodular–Bronchiectatic Pattern**: RML & lingula involvement.
- **Tree-in-Bud**: Often more diffuse than TB.

5. Parasitic Pneumonia

Feature	Example
Focal/Diffuse Consolidation	Toxoplasmosis, Strongyloidiasis
Nodules ± Cavitation	Chronic parasitic infections

6. Other Opportunistic Infections

a. Nocardia

- Multiple nodules ± thick-walled cavities.
- Adjacent consolidation.
- Pleural effusion common.

b. Actinomycosis

- Chronic consolidation ± central low attenuation.
- Peripheral enhancement (“mass-like”).
- Can cross fissures.

CT Imaging Features of Fungal Lung Infections

1. Invasive Aspergillosis

Feature	Description	Phase

Halo Sign	Central nodule/mass with surrounding GGO (perilesional hemorrhage)	**Early**
Air Crescent Sign	Crescent-shaped air within lesion (necrosis + cavitation)	**Late**
Nodules / Consolidation	Solitary or multiple; often peripheral	Any
Cavitation	Progressive parenchymal destruction	Advanced

2. Chronic Pulmonary Aspergillosis

Subtype	CT Findings
Aspergilloma (Fungal Ball)	Mobile soft-tissue mass in pre-existing cavity; air crescent may be seen
Chronic Cavitary Pulmonary Aspergillosis (CCPA)	Multiple thick-walled cavities ± aspergillomas; pericavitary consolidation; pleural thickening

3. Pneumocystis jirovecii Pneumonia (PJP)

Feature	Description
Diffuse GGO	Bilateral, symmetric, perihilar-predominant
Crazy-Paving Pattern	GGO + interlobular septal and intralobular line thickening
Cysts / Pneumatoceles	Thin-walled, upper-lobe predilection
Pneumothorax	Due to rupture of cysts/pneumatoceles

4. Cryptococcosis

Feature	Description
Nodules	Solitary or multiple; ± cavitation
Mass-like Consolidation	Tumor mimic
Miliary Pattern	Diffuse micronodules in disseminated disease
Lymphadenopathy	Mediastinal/hilar, sometimes necrotic

5. Histoplasmosis

Feature	Description

Mediastinal / Hilar Lymphadenopathy	± calcifications
Nodules	Solitary/multiple; often calcified in healed disease
Cavitary Lesions	Chronic fibrocavitary form
Miliary Pattern	Disseminated small nodules in severe cases

6. Coccidioidomycosis

Feature	Description
Nodules	Solitary or multiple; ± cavitation
Consolidation	Lobar/segmental; acute phase
Cavitation	Thin-walled; chronic stage
Pleural Effusions	With consolidation or nodules
Miliary Pattern	Disseminated form

7. Mucormycosis

Feature	Description
Reverse Halo Sign	Central GGO with surrounding denser consolidation ring
Nodules / Consolidation	Rapidly progressive; may cavitate
Pleural Effusions	In severe infection

Q3. Enumerate the causes of hemoptysis in adult patient. Briefly discuss indications, techniques and complications of radiological interventions in them.

Answer

Q5. Haemoptysis
Answer:

Haemoptysis is defined as the expectoration of blood originating **below the glottis** (from tracheobronchial or pulmonary vasculature).
It must be distinguished from:

- **Pseudohaemoptysis**: Blood from upper airway or oropharynx
- **Haematemesis**: Vomiting of blood (gastrointestinal source)

Classification by Volume

Type	**Estimated Volume**	**Clinical Implication**
Mild	<30 mL/day	Often self-limited
Moderate	30–100 mL/day	Needs evaluation and follow-up
Severe	>100 mL/day or respiratory compromise	Requires urgent management
Massive	>300 mL/24 hr or any volume with airway compromise	**Medical emergency**

Etiology of Haemoptysis
Grouped based on **pathophysiological system involvement**:

Category	**Examples**
Infective	Tuberculosis, bronchiectasis, chronic fungal (aspergilloma), abscess
Neoplastic	Bronchogenic carcinoma, metastases
Vascular	Pulmonary embolism, AVMs, pseudoaneurysms, vasculitis (GPA)
Autoimmune	Goodpasture's syndrome, SLE, Wegener's granulomatosis
Iatrogenic	Post-biopsy, bronchoscopic procedures
Cardiac	Mitral stenosis (elevated pulmonary venous pressure)
Hematologic	Anticoagulation, thrombocytopenia, DIC
Idiopathic	Cryptogenic haemoptysis (~10–15%)

Clinical Evaluation

- **History**: Onset, volume, duration, associated symptoms (fever, weight loss, dyspnea)
- **Physical Exam**: Chest auscultation, ENT/oral cavity check (rule out pseudohaemoptysis)
- **Lab Tests**: CBC, INR/PT, sputum AFB and culture, renal function (esp. if contrast CT planned)

Imaging Approach in Haemoptysis

Modality	**Role**
Chest X-ray	First-line; low sensitivity but may localize side of pathology
CT Chest (HRCT/CTA)	**Gold standard** for localization, cause, and vascular evaluation
Bronchoscopy	Localizes site, therapeutic (tamponade, cautery), collects samples
DSA	Used for **intervention** (bronchial artery embolization)
MRI Chest	Rare; used when neoplastic or vascular etiology suspected

CT Angiography Protocol

Preferred Technique:
Split Bolus Dual-Phase CT Angiography (Bronchial + Pulmonary Arteries)
Typical Findings on CTA:

Finding	**Interpretation**
Hypertrophied bronchial artery	Diameter >2 mm; indicates active supply to diseased segment
Tortuosity	Seen in chronic inflammatory or neoplastic states
Bronchopulmonary shunting	Pulmonary artery supplying systemic lesion (non-tapering vessel)
Pseudoaneurysm	Focal contrast-filled outpouching; may rupture
Parenchymal cavitation	TB, aspergilloma, abscess
Non-bronchial systemic arteries	Internal mammary, subclavian, intercostals; seen in chronic disease

Definitive Management: Bronchial Artery Embolization (BAE)

Bronchial Artery Embolization (BAE) is a **minimally invasive image-guided interventional radiology procedure** that targets **systemic arteries (mostly**

bronchial) supplying abnormal lung segments to control **moderate to massive haemoptysis**.

Indications for BAE

Category	Details
Recurrent haemoptysis	≥100 mL blood for ≥3 episodes within 1 week
Progressive	Increasing volume/frequency of haemoptysis
Airway compromise	Respiratory distress or oxygen desaturation due to bleeding
Failed conservative tx	No response to antibiotics or antitubercular therapy

Contraindications to BAE

Absolute	Relative
Arteries supplying spinal cord (radiculomedullary artery with hairpin turn)	Pulmonary artery stenosis
Coronary branches from culprit artery	Contrast allergy
Cerebral supply (vertebral or intracranial branch) from culprit artery	Pregnancy (relative, case-specific)

*Always assess on **oblique/lateral DSA views** for suspicious branches.*

Pre-Procedural Evaluation

Clinical

- Confirm haemoptysis origin
- Evaluate volume/severity, oxygenation, and airway risk
- Rule out upper airway or GI bleed

Laboratory

Test	Acceptable Level (CIRSE)
Hemoglobin	>7 g/dL
Platelets	>20,000/mm³
INR	<2.0 (if on vitamin K antagonists)

Imaging

Modality	Role
Chest X-ray	First-line to localize side or detect cavity
CT Angiography	Essential – Split bolus CTA evaluates **bronchial + pulmonary** systems
Bronchoscopy	For diagnosis, clot removal, or biopsy if source unclear

CT Angiography Planning

- Use **lung window first** → identify parenchymal disease
- Switch to **mediastinal window** → trace hypertrophied arteries
- Look for:
    - **Bronchial arteries** (>2 mm, tortuous)
    - **Ectopic bronchial arteries** (from subclavian or thyrocervical trunks)
    - **Non-bronchial systemic collaterals** (IMA, LTA, intercostals)
    - **Shunting**: Pulmonary artery branches without tapering

Arteries to Prioritize for Embolization

Artery	**Landmark**
R. Intercostobronchial trunk	9 o'clock at L main bronchus upper border
L. Bronchial artery	12 o'clock at L main bronchus lower border
L. Internal mammary branches	1 cm distal to clavicle; posterior to subclavian artery
L. Lateral thoracic branches	Near 2nd rib lateral border (subclavian inferior branch)

Procedure Technique

Access & Setup

- **Common femoral artery** (standard) or **radial** (subclavian access)
- 6F arterial sheath, 5F catheter, Progreat 2.7F microcatheter

Steps

Step	**Description**
Hook culprit artery	End-hole catheter via roadmap DSA
Confirm position	50–60% diluted contrast injection under fluoroscopy
Advance microcatheter	Beyond origin of non-target branches
Embolize	**PVA particles** (355–500 μm), optionally **glue** (NBCA) if trained
Injection technique	Slow, pulsed under fluoro, parallel to flow velocity
Achieve stasis	Until blush disappears; confirm with post-embolization DSA
Flush & proceed	NS flush catheter, repeat for next artery

Goal: ***Embolize abnormal vascular bed & shunts, not main artery trunk.***

Angiographic Signs of Culprit Arteries

Sign	**Imaging Finding**
Hypertrophy	Diameter >2 mm

Tortuosity	Coiled, irregular course
Parenchymal blush	Persistent staining post-injection
Neovascularity	Fine proliferative vascularity near lesion
Pseudoaneurysm	Localized saccular outpouching
Bronchopulmonary shunting	Early opacification of pulmonary vein or artery

Post-Procedural Care

- Check **limb pulses and neurologic status**
- **Observe for 24 hours** for rebleeding or complications
- Resume treatment of underlying condition (e.g., TB)
- Serial follow-up imaging if indicated

Complications

Common (self-limited)	**Serious (rare)**
Chest/back pain	Spinal cord infarction (<1%)
Transient fever or PEs	Stroke, cortical blindness
Post-embolization syndrome	Bronchial infarction, esophagobronchial fistula, MI, ischemic colitis

Tips for Successful BAE

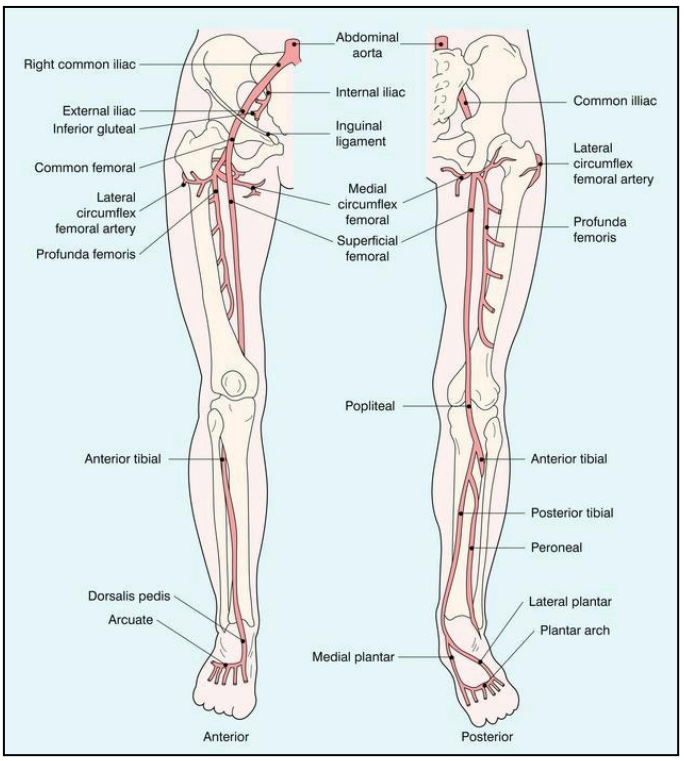
- Always **pre-plan with CTA**: identify all culprit and ectopic arteries
- Use **radial access** if subclavian difficult to cannulate
- Use **superselective embolization** to prevent non-target ischemia
- In **persistent bleeding**, consider:
    - Re-embolization
    - Pulmonary artery embolization (for shunting)
    - Surgery (rare, for malignancy or failure)

Q4. a) Arterial and venous anatomy of lower limb with help of diagram.

b) Imaging features of deep venous thrombosis.

Answer

Arterial Anatomy of the Lower Limb



1. Aorta and Iliac Arteries

- **Abdominal Aorta**
  - Bifurcates at **L4** into **right and left common iliac arteries**.
  - Visible on **axial CT/MR** at the level of the umbilicus.
- **Common Iliac Arteries**
  - Each divides into:
    - **Internal Iliac Artery** → Pelvic viscera, gluteal region, medial thigh.
    - **External Iliac Artery** → Main arterial supply to lower limb.

2. External Iliac Artery

- Passes **beneath inguinal ligament** → becomes **Femoral Artery**.
- Landmark for transition: **mid-inguinal point**.

- Radiological note: often evaluated in **CTA runoff studies** for peripheral vascular disease.

3. Femoral Artery

- **Course**: From inguinal ligament along anterior thigh in femoral triangle.
- **Key Branches**:
  - **Superficial Circumflex Iliac Artery**
  - **Superficial Epigastric Artery**
  - **External Pudendal Arteries**
  - **Profunda Femoris Artery (Deep Femoral)**
    - Large branch early in course.
    - Supplies deep thigh muscles and femur via **perforating branches**.
- Imaging note: Frequent site for **atherosclerotic stenosis**; easily seen on **duplex ultrasound** or **MRA**.

4. Popliteal Artery

- Continuation of femoral artery after **adductor hiatus**.
- **Location**: Popliteal fossa, posterior to knee.
- **Branches**:
  - **Genicular Arteries** → Knee joint collateral supply.
- Imaging note: On CTA/MRA, popliteal artery is prone to **entrapment syndrome** or **traumatic injury**.

5. Anterior Tibial Artery

- Arises from **popliteal artery** at lower border of popliteus muscle.
- Passes through **interosseous membrane** → anterior compartment.
- Continues on dorsum of foot as **Dorsalis Pedis Artery** (palpable pulse at 1st–2nd metatarsal space).
- Imaging note: Often evaluated for **critical limb ischemia** in diabetics.

6. Posterior Tibial Artery

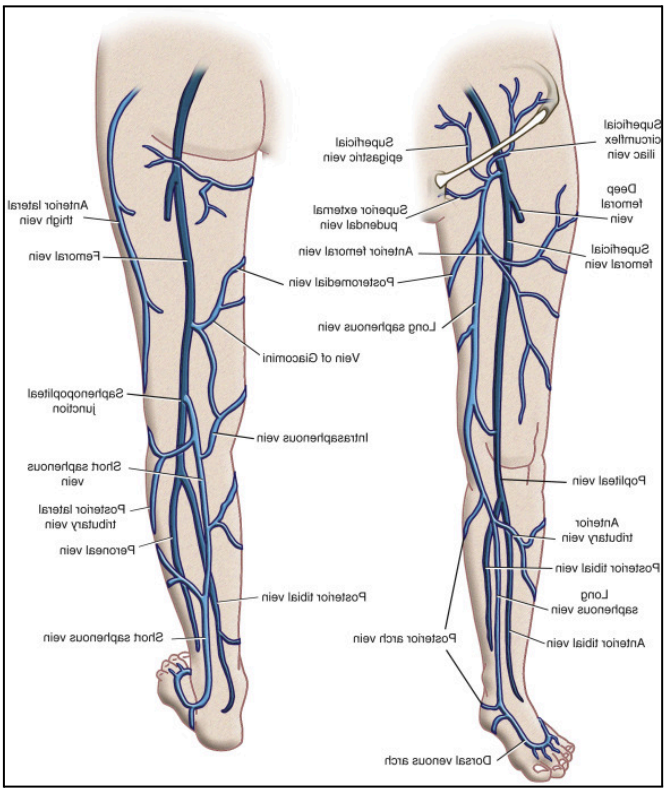
- Continuation of popliteal artery in posterior compartment.
- Gives off:
  - **Peroneal (Fibular) Artery** → Lateral compartment supply.
- Divides behind medial malleolus into:
  - **Medial Plantar Artery**
  - **Lateral Plantar Artery**
- Imaging note: Posterior tibial artery pulse is key in **peripheral vascular disease assessment**.

Landmarks for Cross-sectional Imaging

Artery	Key Level on CT/MR	Landmark

Common Iliac bifurcation	L4	Umbilicus level
External Iliac → Femoral	Mid-inguinal point	Under inguinal ligament
Femoral → Popliteal	Adductor hiatus	Distal thigh
Popliteal bifurcation	Below knee joint	Popliteus level
Posterior Tibial bifurcation	Medial malleolus	Ankle

Venous Anatomy of the Lower Limb



1. Superficial Veins

- **Great Saphenous Vein (GSV)**
  - Longest vein in the body.
  - Origin: **Medial dorsal venous arch** of foot.
  - Course: Medial leg → thigh → drains into **femoral vein** at saphenofemoral junction (groin).
  - Imaging note: Common in **varicose veins**, site for **venous harvesting** (e.g., CABG).
- **Small Saphenous Vein (SSV)**
  - Origin: **Lateral dorsal venous arch**.
  - Course: Posterior leg → drains into **popliteal vein** at saphenopopliteal junction.

    - Imaging note: Better seen on **duplex ultrasound** with posterior approach.

2. Deep Veins

- **Femoral Vein**
    - Continuation of **popliteal vein** after passing through adductor canal.
    - Receives **profunda femoris vein** + **great saphenous vein**.
    - Clinical: Common site for **proximal DVT**.
- **Popliteal Vein**
    - Formed by union of **anterior tibial** + **posterior tibial veins**.
    - Located in popliteal fossa behind the knee.
    - Imaging: Easily assessed on US; thrombus here is a high-risk for **PE**.
- **Anterior Tibial Vein**
    - Paired venae comitantes along **anterior tibial artery**.
    - Drains anterior leg compartment.
- **Posterior Tibial Vein**
    - Paired venae comitantes along **posterior tibial artery**.
    - Drains posterior leg compartment.
- **Peroneal (Fibular) Vein**
    - Paired venae comitantes along **peroneal artery**.
    - Drains lateral leg compartment.

3. Perforating Veins

- Connect superficial → deep venous system.
- Contain valves to ensure **one-way flow toward deep veins**.
- Imaging: Incompetent perforators often seen in **chronic venous insufficiency** on Doppler.

Deep Venous Thrombosis (DVT)

A. Ultrasound – First-line modality

Key Signs:

1. **Non-compressibility** – most reliable sign.
2. **Direct thrombus visualization** – echogenic/hypoechoic intraluminal material.
3. **Dilated vein** – larger than accompanying artery.
4. **Absence of color Doppler flow** – or disturbed flow.
5. **No augmentation** – distal compression fails to increase flow.

Advantages:

- No radiation, bedside capability.
- Excellent for femoral, popliteal, tibial veins.

B. CT Venography

Indications:

- Inconclusive US, pelvic/iliac vein thrombosis, trauma, pre-intervention mapping.

Key Signs:

1. **Filling defect** within contrast-filled lumen.
2. **Venous enlargement** relative to contralateral side.
3. **Contrast halo sign** – peripheral contrast around thrombus.

Advantages:

- Excellent pelvic/abdominal coverage.
- Detects alternative causes of leg swelling.

C. MR Venography

Indications:

- Contraindication to iodinated contrast, detailed pelvic mapping, pregnancy.

Key Signs:

1. **T1 hypointensity**, **T2 hyperintensity** in acute thrombus.
2. **Filling defects** on contrast-enhanced sequences.
3. **Vein wall enhancement** – inflammation in subacute/chronic cases.

Advantages:

- No ionizing radiation.
- Superior pelvic & IVC evaluation.

Imaging Landmark

Vein	**Key Ultrasound Approach**	**Common DVT Sites**
Common femoral	Groin transverse probe	Saphenofemoral junction
Femoral vein	Medial thigh	Proximal extension of calf DVT
Popliteal vein	Posterior knee fossa	High embolic risk
Tibial veins	Mid-calf	Often extension from foot DVT

Q5. Define acute aortic syndrome. Classify aortic dissection. Describe its imaging features on CT angiography and its management.

Answer

AAS includes a group of life-threatening pathologies involving the aortic wall with overlapping clinical features (e.g., sudden, severe chest/back pain) but differing pathophysiology and management.

Components of Acute Aortic Syndrome

Condition	Description	Pathophysiology
Aortic Dissection	Tear in intima → blood enters media → true & false lumen	**Intimal tear** with separation of media layers
Intramural Hematoma (IMH)	Hemorrhage into media **without intimal tear**	Rupture of **vasa vasorum** (microvessels in aortic wall)
Penetrating Atherosclerotic Ulcer (PAU)	Ulceration of atherosclerotic plaque into media	Erosion through intima due to plaque
Aortic Rupture	Full-thickness aortic wall disruption with hemorrhage	Can be due to progression of any of the above
Limited/Localized Dissection	Small focal dissection, often stable	Partial medial disruption, usually contained

Aortic Dissection

Aortic dissection is a life-threatening condition involving a **tear in the intima** of the aorta, allowing blood to enter the **medial layer**, creating a **false lumen**. It is the most common acute aortic syndrome (AAS), alongside **intramural hematoma** and **penetrating atherosclerotic ulcer**.

Pathophysiology

- Initiated by **intimal tear** → blood tracks into the media → **separation of layers**.
- Formation of **true and false lumens**; false lumen may re-enter the true lumen (re-entry tear) or be blind-ended (cul-de-sac).

- False lumen can lead to **malperfusion**, **rupture**, or **aneurysmal degeneration**.

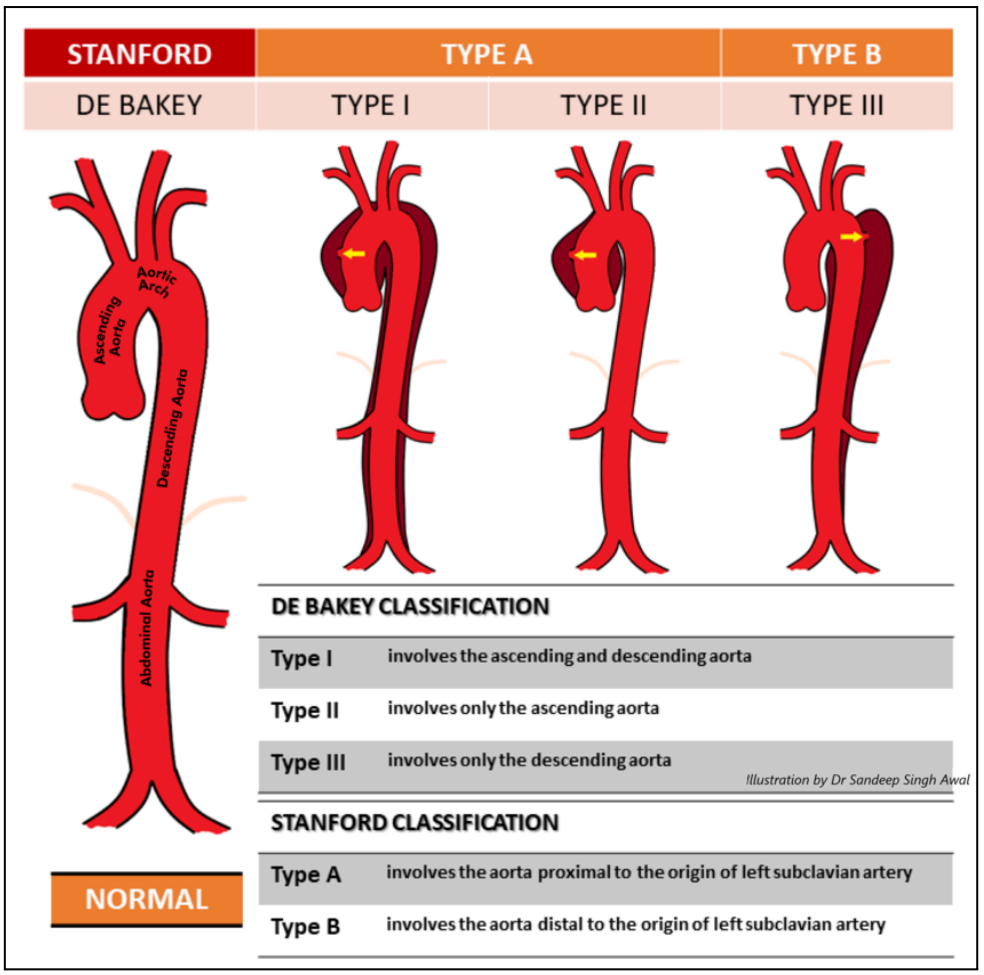
Classification Systems

A. Stanford Classification (Clinically most relevant)

Type	Involvement	Management
A	Involves ascending aorta (± descending)	**Surgical emergency**
B	Only descending aorta (distal to left subclavian artery)	**Medical ± endovascular**

B. DeBakey Classification (Anatomy based)

Type	Origin	Extent
I	Ascending aorta	Propagates to arch & descending
II	Ascending aorta only	Confined proximally
IIIa	Descending thoracic aorta	Limited to thorax
IIIb	Descending aorta	Extends to abdomen/pelvis



Imaging Modalities and Findings

A. CT Angiography (CTA) – Gold Standard

Parameter	CTA Findings
Intimal flap	Linear structure separating true and false lumen
False lumen signs	Larger, delayed enhancement, “beak sign”, “cobweb sign”
Entry tear	Seen as contrast-filled defect in intima
Branch vessel involvement	Malperfusion, vessel origin from false lumen
Complications	Rupture, hemothorax, pericardial effusion, organ ischemia

B. MRI Angiography

- Used in **stable patients or chronic dissection**.

- Phase contrast cine MRI can demonstrate **flow dynamics** in true vs. false lumens.
- Delayed enhancement helps identify **thrombosed false lumen**.

C. Transesophageal Echocardiography (TEE)

- Useful in unstable patients.
- Shows **intimal flap**, pericardial effusion, aortic valve involvement.

D. Chest Radiograph (CXR)

Finding	**Description**
Widened mediastinum	Most common
Obliteration of aortic knob	Suggestive of ascending involvement
Pleural effusion (left)	Suggestive of rupture

Key Imaging Differentiators – True vs. False Lumen (on CT/MRI)

Feature	**True Lumen**	**False Lumen**
Size	Smaller	Larger
Enhancement	Enhances first	Delayed
Location	Outer curvature	Inner curvature
Beak sign		Acute angle at flap
Cobweb sign		Linear structures (residual media)
Origin of visceral arteries	Often from true lumen	Can be from false (risk of malperfusion)

Complications of Aortic Dissection

Complication	**Imaging Features**
Aortic rupture	Hemomediastinum, hemothorax, contrast extravasation
Pericardial tamponade	Pericardial effusion with RA/RV compression
Organ malperfusion	Non-enhancing kidneys/bowel, SMA/celiac/renal artery involvement
Stroke	Carotid artery involvement
Acute aortic regurgitation	TEE shows diastolic regurgitation
Chronic aneurysmal degeneration	False lumen expansion, sac formation

Chronic vs. Acute Dissection (Imaging Clues)

Feature	Acute	Chronic
Flap	Mobile, thin	Thickened, immobile
Lumen contrast	Delayed, heterogenous	Uniform
False lumen	May thrombose partially	May show calcification
Symptoms	Severe pain	Asymptomatic or vague symptoms

Management Overview

Stanford Type	Management
Type A	**Immediate surgery** (ascending aorta replacement ± root/valve)
Type B – uncomplicated	**Medical** (BP control: β-blockers, target SBP <120)
Type B – complicated	**TEVAR** (Thoracic Endovascular Aortic Repair) if malperfusion, persistent pain, aneurysm, rupture

Summary Points

- Aortic dissection is a critical **acute aortic syndrome** requiring prompt diagnosis.
- **CTA is the gold standard**, with MRI as a preferred modality for follow-up or in renal impairment.
- Correct identification of **true vs. false lumen**, **extent**, and **complications** dictates therapy.
- Use the **DISSECTION mnemonic** to systematically assess dissection features.
- Type A requires **surgery**; Type B often **medical**, unless complicated.

Q6 Role of nuclear imaging, cardiac MRI and MDCT in evaluation of ischemic heart disease patient.

Answer

Ischemic Heart Disease (IHD)

Ischemic Heart Disease refers to myocardial dysfunction resulting from reduced coronary blood supply, typically due to atherosclerotic coronary artery disease (CAD). Imaging plays a central role in **diagnosis**, **risk stratification**, and **treatment planning**, with different modalities addressing anatomy, perfusion, viability, and prognostication.

1. Nuclear Imaging

Modalities:

- **Single Photon Emission Computed Tomography (SPECT)** – Widely available, uses gamma emitters (e.g., Tc-99m sestamibi, Tl-201).
- **Positron Emission Tomography (PET)** – Uses positron emitters (e.g., Rb-82, N-13 ammonia, F-18 FDG), higher resolution and quantitative capability.

Mechanism:

- **Myocardial Perfusion Imaging (MPI):** Compares perfusion at stress vs. rest.
  - **Reversible defect** → ischemia.
  - **Fixed defect** → infarction/scar.
- **PET Viability Assessment:**
  - Preserved FDG uptake but reduced perfusion → hibernating myocardium.
  - No uptake → scar/non-viable tissue.

Key Roles in IHD:

- **Diagnosis:** Detects flow-limiting CAD when anatomical imaging is equivocal.
- **Risk Stratification:** Extent/severity of defects predicts MACE (Major Adverse Cardiac Events).
- **Prognosis:** Large ischemic burden (>10% LV myocardium) → improved outcomes with revascularization.
- **Guiding Management:** PET helps decide if revascularization is worthwhile.

Radiological Signs:

- SPECT/PET polar maps showing stress-induced perfusion defects.
- Quantitative PET: Coronary flow reserve (CFR) < 2 indicates abnormal microvascular function.

Advantages:

- Proven clinical utility.
- PET has superior resolution and quantitative capability.

Limitations:

- Ionizing radiation.
- Lower spatial resolution vs. CMR/CT.
- Dependent on adequate stress induction (exercise or pharmacological).

2. Cardiac Magnetic Resonance Imaging (CMR)
Mechanism:
- Uses magnetic field and radiofrequency pulses; gadolinium contrast for perfusion and scar assessment.
- Cine sequences for ventricular function; late gadolinium enhancement (LGE) for scar/fibrosis; T1/T2 mapping for tissue characterization.

Key Roles in IHD:
- **Functional Analysis:** Gold standard for LV and RV volume, EF, wall motion.
- **Viability Assessment:**
  - LGE detects myocardial infarction (hyperintense signal due to gadolinium retention in fibrosis/necrosis).
  - Infarct transmurality <50% → higher likelihood of functional recovery after revascularization.
- **Ischemia Detection:** Stress perfusion CMR reveals subendocardial ischemia with high sensitivity.
- **Acute MI:** Detects microvascular obstruction (dark core in hyperenhanced infarct zone), intramyocardial hemorrhage (T2* hypointensity).
- **Chronic IHD:** Quantifies scar burden for prognosis.

Radiological Signs:
- **LGE:** Hyperintense in infarcted myocardium, conforming to coronary territory.
- **Perfusion Deficit:** Hypoenhancement during stress, resolving at rest in ischemia.
- **Edema (Acute MI):** Hyperintense on T2-weighted/T2 mapping.

Advantages:
- No ionizing radiation.
- High spatial resolution, multiplanar capability.
- Comprehensive single-session assessment.

Limitations:
- Less available; requires expertise.
- Contraindicated in some metallic implants.
- Caution in severe renal impairment (risk of NSF with gadolinium).

3. Multidetector Computed Tomography (MDCT)
Modalities:
- **Coronary CT Angiography (CCTA)** – Contrast-enhanced, ECG-gated high-resolution imaging of coronary arteries.
- **CT Calcium Scoring (CAC):** Non-contrast scan quantifying calcified plaque burden (Agatston score).

Key Roles in IHD:
- **Anatomical Evaluation:**

    - CCTA: Sensitivity >95% for detecting >50% coronary stenosis.
    - Excellent **rule-out** test in low-intermediate risk patients.
- **Risk Prediction:** CAC >400 → high risk for coronary events.
- **Plaque Characterization:**
    - Calcified, non-calcified, or mixed.
    - Vulnerable plaque features: positive remodeling, low attenuation (<30 HU), napkin-ring sign.
- **Post-intervention:** Evaluates bypass graft patency, stent assessment (limited for small diameter stents).

Radiological Signs:

- **Stenosis:** Luminal narrowing >50% with contrast filling defect.
- **Plaque Morphology:** Low-attenuation plaques suggesting lipid-rich necrotic core.

Advantages:

- Non-invasive, rapid.
- High spatial resolution.
- Excellent negative predictive value.

Limitations:

- Ionizing radiation.
- Iodinated contrast → nephrotoxicity/allergic risk.
- Limited in high heart rates, irregular rhythm, heavy calcification.

Comparative Table

Modality	Primary Role	Key Strengths	Main Limitations
SPECT / PET	Perfusion & viability	Proven outcomes data, PET quantitative	Radiation, lower spatial resolution
CMR	Structure, function, viability, perfusion	No radiation, high spatial resolution, tissue characterization	Limited availability, MRI contraindications
MDCT (CCTA)	Coronary anatomy, calcium score	Excellent NPV, plaque characterization	Radiation, contrast use, arrhythmia limitations

---

Q7. Enumerate causes of neonatal respiratory distress. Describe in detail pathophysiology and imaging findings in hyaline membrane disease.

Answer

Neonatal Respiratory Distress

Respiratory distress in the newborn is a clinical emergency with a broad differential. Imaging — primarily **chest radiography** — plays a crucial role in diagnosis, differentiation, and guiding management.

Pulmonary Causes

1. Respiratory Distress Syndrome (RDS / Hyaline Membrane Disease)

- **Etiology**: Surfactant deficiency → alveolar collapse → diffuse atelectasis.
- **Radiographic Findings**:
  - **Diffuse, symmetric "ground-glass" / reticulogranular opacities**.
  - **Air bronchograms**: Lucent branching lines against opaque lung background.
  - **Low lung volumes**: Diaphragm elevated, small lung fields.
  - No pleural effusion.
- **Pearls**:
  - Onset within minutes to hours after birth.
  - Often worsens over first 24–48 hrs if untreated.
  - Improves after surfactant therapy.

2. Transient Tachypnea of the Newborn (TTN)

- **Etiology**: Delayed resorption of fetal lung fluid, common in C-section births.
- **Radiographic Findings**:
  - **Hyperinflated lungs** (flattened diaphragms).
  - **Prominent vascular markings** extending to periphery.
  - **Perihilar streaking** and interlobar fissural fluid (linear or wedge-shaped opacity).
  - Occasionally small pleural effusion.
- **Pearls**:
  - Appears soon after birth; resolves in 24–72 hrs.
  - "Wet lung" appearance.

3. Meconium Aspiration Syndrome (MAS)

- **Etiology**: Inhalation of meconium-stained amniotic fluid → chemical pneumonitis + airway obstruction.
- **Radiographic Findings**:

    - **Patchy, coarse, asymmetric opacities** with areas of hyperinflation.
    - Possible air trapping and focal atelectasis.
    - Complications: Pneumothorax, pneumomediastinum.
- **Pearls**:
    - Term or post-term infant with meconium-stained fluid.
    - Mixed pattern of consolidation and hyperlucency.

4. Neonatal Pneumonia

- **Etiology**: Bacterial/viral (e.g., GBS, E. coli, CMV).
- **Radiographic Findings**:
    - Variable: From focal consolidation to diffuse opacities.
    - May mimic RDS but often **with pleural effusion**.
    - Hyperinflation possible in viral causes.
- **Pearls**:
    - Clinical + lab correlation important.

5. Pulmonary Hypoplasia

- **Etiology**: Small lungs (e.g., CDH, oligohydramnios).
- **Radiographic Findings**:
    - **Small lung fields** with elevated diaphragm.
    - Sparse vascular markings.
    - Associated anomalies visible (e.g., bowel loops in chest for CDH).

6. Persistent Pulmonary Hypertension of the Newborn (PPHN)

- **Radiographic Findings**:
    - Often normal or nonspecific.
    - Can show mild interstitial markings.
- **Pearls**:
    - Echo more diagnostic; radiology rules out other causes.

7. Pneumothorax

- **Radiographic Findings**:
    - Lucent hemithorax without lung markings peripherally.
    - Lung edge visible; mediastinal shift if tension pneumothorax.

8. Congenital Pulmonary Airway Malformation (CPAM)

- **Radiographic Findings**:
    - Multiple cystic lucencies or a single large cyst.
    - Mass effect possible.

Cardiac Causes (CHD, CHF)

- **Radiographic Clues**:
    - Cardiomegaly (CTR > 0.6 in neonates).
    - Pulmonary plethora (↑ vascular markings) in left-to-right shunts.

- Oligemia in obstructive lesions (e.g., pulmonary atresia).

Hyaline Membrane Disease – Pathophysiology & Imaging Integration
Pathophysiology Recap

1. **Surfactant deficiency** → high surface tension → alveolar collapse.
2. **V/Q mismatch** → hypoxemia + hypercapnia.
3. **Inflammation + capillary leak** → proteinaceous exudate in alveoli.
4. **Hyaline membranes** line alveoli, further impairing gas exchange.

Radiologic Features in HMD

Feature	Mechanism	Imaging Appearance
Diffuse ground-glass opacity	Collapsed alveoli + interstitial fluid	Fine reticulogranular pattern
Air bronchograms	Air-filled bronchi amidst collapsed alveoli	Lucent tubular branching lines
Low lung volumes	Widespread atelectasis	Elevated diaphragms, narrow intercostal spaces
Symmetry	Uniform surfactant deficiency	Bilateral equal involvement
Progression	Worsening alveolar collapse over hours	Increasing opacity density

Radiographic Differentials in Neonates

Condition	Lung Volume	Opacity Pattern	Other Clues
RDS	↓	Fine granular, air bronchograms	Preterm, symmetric
TTN	↑	Perihilar streaks, fissural fluid	Term, resolves quickly
MAS	Variable	Patchy consolidation + hyperinflation	Term/post-term, meconium
Pneumonia	Variable	Focal/multifocal opacities	Effusions, sepsis signs
Pulmonary Hypoplasia	↓	Small lung fields	CDH, oligohydramnios
CHD	Variable	Vascular changes	Cardiomegaly, echo correlation

Q8. Draw a schematic diagram describing anatomy of diaphragm and imaging features of diaphragmatic injury on chest X-ray, ultrasound, CT and MRI.

Answer

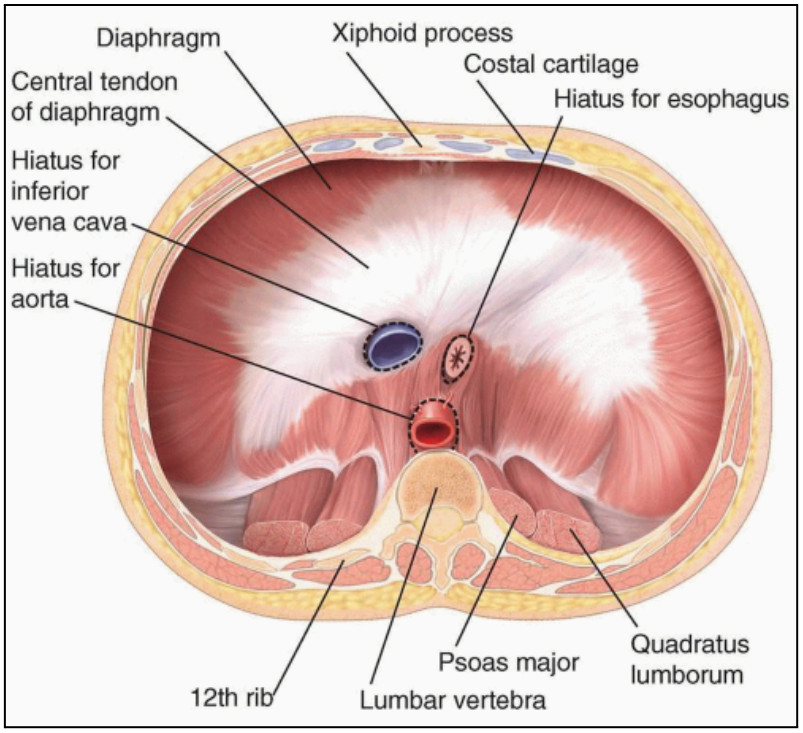
Embryology of the Diaphragm

The diaphragm develops between the **4th to 12th weeks of gestation** and forms from **four embryologic components**:

Component	Contribution
Septum transversum	Forms **central tendon**
Pleuroperitoneal folds	Forms **posterolateral parts** of diaphragm
Esophageal mesentery	Contributes to **crura** and surrounds esophagus
Muscular body wall	Contributes to **peripheral muscular rim**

Defects in fusion can result in **congenital diaphragmatic hernias**:

- **Bochdalek hernia** (posterolateral): 90%, usually left-sided
- **Morgagni hernia** (anteromedial): rare, right-sided



Gross Anatomy

The diaphragm is a **thin, dome-shaped musculotendinous structure** that separates the **thoracic** and **abdominal** cavities. It has **three parts based on attachments**:

1. Sternal Part

- Origin: Xiphoid process
- Smallest and most anterior part

2. Costal Part

- Origin: Lower six ribs and their costal cartilages
- Forms the largest part of the muscular dome

3. Lumbar Part (Crura)

- Origin: Upper lumbar vertebrae (L1-L3 on right, L1-L2 on left)
- Forms the **right and left crura**
    - **Right crus**: larger and longer, loops around the esophagus forming the **esophageal hiatus**

Arcuate Ligaments

- **Median arcuate ligament**: Joins the right and left crura anterior to aorta
- **Medial arcuate ligament**: Thickening over the **psoas major**
- **Lateral arcuate ligament**: Thickening over the **quadratus lumborum**

Hiatuses in the Diaphragm

Hiatus	Vertebral Level	Structures Passing Through
Caval (IVC)	T8	IVC, branches of **right phrenic nerve**
Oesophageal	T10	Oesophagus, **vagus nerves**, lymphatics
Aortic	T12	Aorta, **thoracic duct**, azygos and hemiazygos veins

Note: **Aortic hiatus is retrocrural**—not affected by diaphragmatic contraction.

Muscle-Tendon Structure

- **Central tendon**: Strong aponeurosis where all muscle fibers insert; **non-contractile**, lies beneath the pericardium
- **Muscular periphery**: Contractile zones that pull the central tendon downward during inspiration

Nerve Supply

- **Motor**: Phrenic nerve (C3–C5)
- **Sensory**:
    - Central part: **Phrenic nerve**
    - Peripheral part: **Lower 6 intercostal nerves** and **subcostal nerves**

Blood Supply

Region	Arterial Supply	Venous Drainage
Superior	Pericardiophrenic and musculophrenic (from internal thoracic artery)	Drain into brachiocephalic veins
Inferior	Inferior phrenic arteries (from abdominal aorta or celiac)	Drain into IVC, adrenal, and hepatic veins

Functional Anatomy

- **Inspiration**: Contraction → central tendon moves caudally → increases vertical dimension of thorax → negative intrathoracic pressure → air inflow
- **Expiration**: Passive recoil of lungs and diaphragm
- **Additional roles**: Increases intra-abdominal pressure for **coughing, vomiting, defecation, micturition, and childbirth**

Clinical Relevance in Radiology

Condition	Imaging Finding
Paralysis	Elevated dome, **paradoxical motion** on sniff test
Eventration	Localized dome elevation with intact contour
Hernia (Bochdalek)	Posterolateral defect, fat/bowel in thorax
Tumor infiltration	Focal thickening, loss of fat planes, mass effect
Trauma	Discontinuity, herniation of abdominal contents

Diaphragmatic Injury – Imaging Approach

1. Chest X-ray (CXR)

Mechanism: Initial screening in trauma; sensitivity limited (~30–60%), but specific findings can be diagnostic.

Key Signs:

1. **Abnormal diaphragmatic contour or elevation**
    - **One side elevated, irregular, or obscured.**
    - **Loss of crisp diaphragmatic outline.**
2. **Visceral herniation into thorax**
    - **Stomach, bowel loops, or liver silhouette visible above diaphragm.**
3. **NG tube in chest**
    - **Pathognomonic if tip curls in intrathoracic stomach.**
4. **Hemothorax / pneumothorax**
    - **Often with associated rib fractures or chest trauma.**
5. **Bowel gas above diaphragm**
    - **Multiple air-fluid levels in thoracic cavity.**

Pearl: Left-sided injuries are more common (liver protects right side), but right injuries are harder to detect on CXR.

2. Ultrasound (Focused Assessment – eFAST / Thoracoabdominal)
Mechanism: Bedside, rapid assessment in unstable trauma patients.
Key Signs:

1. **Diaphragm discontinuity** – Direct gap in echogenic diaphragm line.
2. **Herniation of abdominal contents** – Liver, spleen, bowel loops visualized above diaphragm.
3. **Dynamic evaluation** – Absent or paradoxical movement on injured side during respiration.
4. **Hemothorax detection** – Anechoic/hypoechoic collection in thoracic cavity.

Pearl: Operator-dependent; best for anterior or right-sided injuries, limited for posterior tears.

3. Computed Tomography (CT)
Mechanism: Gold standard for stable patients; high spatial resolution.
Direct Signs:

1. **Diaphragmatic defect** – Clear tear or focal discontinuity.
2. **Visceral herniation** – Stomach, bowel, liver, spleen in chest cavity.

Classic CT Signs:

- **Collar sign** – Waist-like constriction of herniated viscus at tear site.
- **Dependent viscera sign** – Herniated abdominal organ rests directly against posterior ribs (diaphragm absent).

Indirect Signs:

- **Hemoperitoneum / hemothorax**.
- **Contralateral mediastinal shift** from herniated mass effect.

Advanced:

- **CT angiography** – Evaluates associated vascular injury (especially in penetrating trauma).

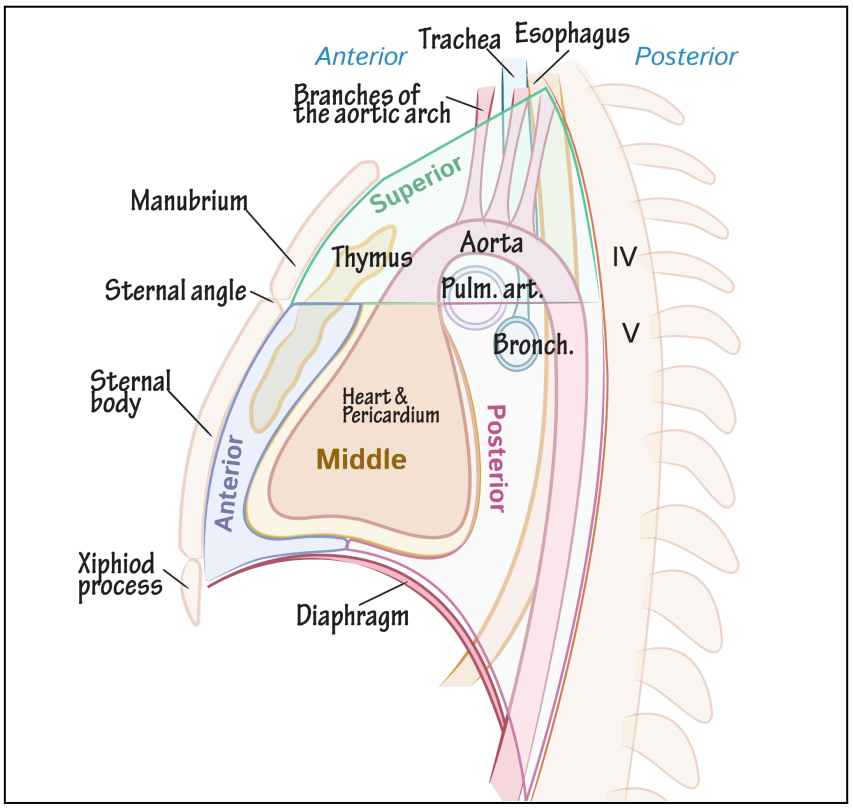
4. Magnetic Resonance Imaging (MRI)
Mechanism: Problem-solving tool, non-acute evaluation, high soft-tissue contrast.
Key Signs:

1. **High-resolution diaphragm anatomy** – Multiplanar visualization of tear.
2. **Multiplanar reconstructions** – Axial, coronal, sagittal views for full tear assessment.
3. **Tissue characterization** – Distinguish fat, bowel, liver with different signal intensities.
4. **Functional imaging** – Cine MRI for motion analysis in chronic or subtle injuries.

Pearl: Rarely used acutely; useful for chronic diaphragmatic hernias or equivocal CT.

Q9. Describe anatomy of mediastinum with the help of diagram. Discuss imaging features of anterior mediastinum masses.

Answer



The **ITMIG (International Thymic Malignancy Interest Group) classification of mediastinal compartments** is a **multidetector CT-based** classification system.

Feature	Prevascular (Anterior)	Visceral (Middle)	Paravertebral (Posterior)

Boundaries	**Anterior**: Sternum **Posterior**: Pericardium **Lateral**: Mediastinal pleura **Superior:** Thoracic inlet **Inferior:** Diaphragm	**Anterior**: Posterior pericardium **Posterior**: 1 cm posterior Anterior surface of vertebral bodies **Lateral**: Mediastinal pleura **Superior:** Thoracic inlet **Inferior:** Diaphragm	**Anterior**: Posterior margin of visceral compartment **Posterior**: Vertebral bodies **Lateral**: Costovertebral junction **Superior:** Thoracic inlet **Inferior:** Diaphragm
Major Contents	Thymus Retrosternal fat Left brachiocephalic vein Internal mammary vessels Prevascular lymph nodes	Heart and pericardium Ascending and descending aorta Superior vena cava Trachea and main bronchi Esophagus Pulmonary arteries and veins Visceral group lymph nodes	Thoracic vertebrae Spinal cord Paraspinal soft tissue Nerve roots Sympathetic chains
Common Masses	Thymoma Germ cell tumor Lymphoma Thyroid extension	Lymphadenopathy Tracheal or bronchogenic tumors Esophageal lesions Vascular anomalies	Neurogenic tumors Paravertebral abscess Extramedullary hematopoiesis
Imaging Modality	Best seen on axial contrast-enhanced CT and MRI	Axial CT with mediastinal and lung windows	MRI (for spinal extension), CT with bone windows
Clinical Significance	Key site for anterior mediastinal tumors Common biopsy approach via anterior route	Includes central thoracic viscera; common site for lymphadenopathy and airway/esophageal pathology	Important in neurogenic tumor evaluation and spine pathology

Anterior Mediastinal Mass

Anterior mediastinal masses (AMMs) arise in the prevascular compartment, which lies **anterior to the pericardium and great vessels**, bounded **anteriorly by the sternum and posteriorly by the pericardium** (ITMIG classification). Though broad in etiology, these masses are often grouped using the classical mnemonic **"4 Ts"**:

- **Thymic** lesions (thymoma, thymic carcinoma, hyperplasia, cysts)
- **Teratoma** (germ cell tumors)
- **Thyroid** (substernal goiter)
- **Terrible lymphoma** (Hodgkin and Non-Hodgkin lymphoma)

Imaging Approach

I. Clinical and Laboratory Correlation

- **Age & Sex**:
  - <30 years: Germ cell tumors, lymphoma
  - 40–60 years: Thymoma
  - 60 years: Thymic carcinoma, lymphoma
- **Symptoms**:
  - **Mass effect**: Dyspnea, dysphagia, SVC syndrome
  - **Paraneoplastic syndromes**:
    - Myasthenia gravis (thymoma)
    - Cushing's syndrome, SIADH (thymic NETs)
  - **B symptoms** (fever, weight loss, night sweats): lymphoma
- **Tumor markers**:
  - **AFP, β-hCG** → Germ cell tumors
  - **Thyroid function tests** → Retrosternal goiter

IMAGING MODALITIES

1. Chest Radiograph (CXR)

Role:

- **Initial screening tool** for mediastinal masses.
- Often the **first imaging modality** performed in patients with nonspecific chest symptoms.

Findings:

Sign	Description	Typical Findings in Anterior Mediastinal Mass

Broad-based margin	Interface of mass with mediastinum forms an obtuse angle	Mass appears to arise from mediastinum, not lung parenchyma
Silhouette Sign	Loss of normal borders when two structures of the same radiodensity touch	Loss of heart border (right or left) suggests mass in anterior mediastinum (e.g., right heart border with thymoma)
Cervicothoracic Sign	Differentiates anterior from posterior mediastinal mass based on visibility above clavicles	**Cervicothoracic sign = Positive** Mass margin not seen above clavicle → anterior; sharp margin extending above clavicle → posterior
Hilum Overlay Sign	Relation between lateral border of cardiac silhouette and pulmonary vessels are not maintained suggesting. Hilar vessels seen through the mass suggest the mass is not in the hilum	○ Mass is not of cardiac origin ○ Preserved visibility of hilar vessels → mass is anterior or posterior to hilum
Hilum Convergence	To differentiate enlarged PA from juxta-hilar mediastinal mass	Pulmonary artery branches converge towards the waist of heart – juxta-hilar mediastinal mass
Abdomino thoracic Sign	Differentiates diaphragmatic from mediastinal masses based on interface with diaphragm	**Negative**
Both Sides of Mediastinum	Mass projects beyond midline, extending into both hemithoraces	Common in lymphomas or large thymomas; may shift trachea or compress adjacent structures
Radiographic Sign	**Description**	**Implication in Anterior Mediastinal Mass**
Broad-based interface	Mass shows a wide attachment to mediastinum with obtuse angle with pleura	Typical of mediastinal (vs intrapulmonary) origin

Mediastinal silhouette	Obliteration of adjacent heart or vessel borders (silhouette sign)	Loss of right heart border → right anterior mass; loss of left heart border → left anterior mass
Cervicothoracic sign	Superior margin of mass is **ill-defined** if anterior, **well-defined** if posterior	Anterior masses do **not** extend above clavicle; posterior ones **do**
Hilum overlay sign	Pulmonary vessels are visualized through the mass	Mass is not at hilum but **anterior or posterior to it**
Hilum convergence sign	Vessels converge into a central mass	Suggests **hilar lymphadenopathy**, not anterior mediastinal mass
Abdomino-thoracic sign	Interface between thoracic mass and abdominal contents	Diaphragmatic continuity helps localize mass to thorax or abdomen
Thymic sail sign	Triangular soft tissue shadow in right upper mediastinum in neonates	Represents **normal thymus**, not pathology
Spinnaker sail sign	Thymic lobes lifted and outlined by air due to pneumomediastinum (like spinnaker sail)	Sign of **pneumomediastinum**, not mass — but thymus prominently displaced anteriorly
Wave sign of Mulvey	Wavy or scalloped contour of the thymus due to adjacent rib indentation	Indicates **normal thymus** in infants and children
Notch sign	Concave or notched upper margin where mass meets mediastinal border	Seen in **anterior mediastinal masses**, suggesting origin and convex interface

2. Ultrasonography (USG)

Role:

- **Adjunct modality** for evaluating lesions that are **cervicomediastinal** in location or **thyroid in origin**.
- Primarily used when:
    - **Substernal goiter** is suspected.
    - There is **continuity with cervical mass**.
    - A **vascular origin or cystic nature** of lesion is suspected.
    - **Biopsy guidance** is needed for superficial components.
- The **thymus** appears as a **homogeneous, hypoechoic (dark gray) soft tissue structure** in the anterior mediastinum.

    - Scattered throughout this background are **multiple small, punctate echogenic (bright) foci**.
    - These echogenic spots represent:
        - **Thymic septations**
        - **Fat interspersed within thymic tissue**
        - **Small internal vessels**
    - The resulting image resembles a **night sky full of stars**, hence the name "**Starry Sky**" or **“Speckled Thymus”**.

3. Computed Tomography (CT)

- **Fat attenuation:** Suggestive of thymolipoma, dermoid/teratoma.
- **Calcifications:** Seen in teratomas (tooth/bone), treated lymphoma, old hemorrhage.
- **Cystic attenuation:** Thymic cyst, necrotic thymic carcinoma, cystic teratoma.
- **Homogeneous soft tissue mass:** Suggestive of lymphoma or thymoma.
- **Infiltrative, irregular, enhancing lesion:** Consider thymic carcinoma or lymphoma.

4. Magnetic Resonance Imaging (MRI)

Role:

- **Problem-solving modality** when CT findings are inconclusive.
- **Preferred in young patients or those with contrast allergy or renal dysfunction.**

Sequences Used:

- **T1-weighted imaging:** Fat shows high signal; helpful in fat-containing lesions.
- **T2-weighted imaging:** Cystic and highly cellular tumors appear hyperintense.
- **STIR:** Suppresses fat to highlight edema or inflammation.
- **DWI and ADC maps:** Restricted diffusion in malignancies (e.g., lymphoma, thymic carcinoma).
- **Chemical Shift Imaging (CSI):** Differentiates thymic hyperplasia (microscopic fat) from thymic neoplasms.

<u>Classification by Etiology</u>

A. Thymic Lesions

1. Thymoma (Most common anterior mediastinal tumor in adults)

- **WHO Classification**: A, AB, B1, B2, B3 (based on epithelial vs lymphocyte content)
- **Imaging**:

    - CT: Well-defined, lobulated, homogeneous, may have calcification
    - MRI: Isointense on T1, hyperintense T2; no fat drop on chemical shift
    - **Signs of invasiveness** (suggest thymic carcinoma): irregular margins, necrosis, invasion, lymphadenopathy
- **Associations**: Myasthenia gravis, pure red cell aplasia, hypogammaglobulinemia
- **Staging**:
    - **Masaoka-Koga** (surgical/pathological)
    - **TNM (ITMIG-IASLC)**: T1–T4, N0–N2, M0–M1b

2. Thymic Carcinoma

- More aggressive than thymoma
- Squamous cell is most common histology
- Imaging: Infiltrative, necrotic, irregular margins, frequent nodal/distant metastasis
- Paraneoplastic syndromes are rare

3. Thymic NETs

- <5% of TETs; may be associated with MEN1
- Imaging: Intense enhancement; can mimic carcinoid tumors
- Paraneoplastic: Cushing's (ectopic ACTH), SIADH, Acromegaly

B. Germ Cell Tumors (GCTs)

1. Teratoma (Mature cystic)

- Most common mediastinal GCT
- Imaging hallmark: **Fat + fluid + calcification (teeth)**
- Rupture: Air-fluid level, inflammation, trichoptysis

2. Immature Teratoma

- More solid, enhancing soft tissue
- Imaging can't reliably differentiate from mature forms

3. Seminoma

- Homogeneous, lobulated, mild enhancement, few/no calcifications
- Lymph node and lung metastasis common
- β-hCG ↑ (in ~30%)

4. Non-Seminomatous GCT (NSGCT)

- Includes yolk sac tumor, choriocarcinoma, embryonal carcinoma
- Large, heterogeneous, hemorrhage/necrosis common
- AFP and/or β-hCG elevated
- Aggressive with poor prognosis

C. Lymphoma

- **Hodgkin's (HL)**: Nodular sclerosis common in anterior mediastinum
    - Lobulated soft tissue mass, contiguous nodal spread, may cavitate
    - B symptoms common

- **Non-Hodgkin's (NHL)**: Often more aggressive (e.g., PMBCL, T-cell lymphoblastic)
    - Encases vessels more commonly than HL
    - Pleural/pericardial effusion, axillary/abdominal nodes

PET-CT is crucial for staging and treatment response.

D. Thyroid Lesions

- **Retrosternal Goiter**:
    - Continuity with cervical thyroid
    - Heterogeneous with calcifications
    - Enhances avidly post-contrast
    - May cause tracheal deviation/compression
- **Thyroid carcinoma (ectopic or invading)** is rare but should be considered

E. Neurogenic Tumors

(Rare in anterior mediastinum – typically posterior)

- Phrenic/vagus nerve origin
- Schwannomas, neurofibromas
- If in anterior mediastinum: follow nerve course, look for NF1

Imaging Differentiation

Feature	Thymoma	Germ Cell Tumor	Lymphoma	Goiter
Age	40–60 yrs	15–35 yrs	Any (HL <30 yrs)	>50 yrs
Shape	Lobulated, smooth	Cystic/fatty/necrotic	Lobulated or diffuse mass	Lobulated, contiguous with thyroid
Fat/Calcium	Rare (calcification)	Fat + calcification (teratoma)	Rare (calcification post-treatment)	Coarse calcification
Vessel Invasion	May occur (B2/B3)	NSGCT often invasive	Common in NHL	Rare
Enhancement	Homogeneous (low-grade)	Heterogeneous (NSGCT)	Homogeneous (HL), hetero (NHL)	Avid
Paraneoplastic	MG, PRCA	β-hCG, AFP syndromes	B symptoms	Hypothyroid/Hyperthyroid

PET-CT	Mild to moderate SUV	High in NSGCT	High in HL/NHL	Variable uptake

Role of ITMIG

- Defines **anatomical mediastinal compartments** to localize masses better
- Encourages **standardized reporting** using prevascular, visceral, paravertebral zones
- Assists in **surgical planning and biopsy approach**

Q10. Define pulmonary oedema and describe its pathophysiology. Enumerate causes and imaging features in pulmonary oedema.

Answer

Pulmonary Oedema

Pulmonary oedema is the abnormal accumulation of fluid in the **interstitial** and **alveolar spaces** of the lung, leading to impaired gas exchange and respiratory distress.

Pathophysiology

1. Cardiogenic Pulmonary Oedema

Mechanism:

- Due to **elevated pulmonary capillary hydrostatic pressure** from left-sided heart dysfunction.

Causes:

- **Left ventricular dysfunction** (systolic or diastolic heart failure)
- **Valvular heart disease** (mitral stenosis/regurgitation, aortic valve disease)
- **Acute myocardial infarction**
- **Volume overload** (renal failure, excessive IV fluids)

Sequence:

LV failure → ↑ LA pressure → ↑ pulmonary venous pressure → ↑ pulmonary capillary hydrostatic pressure → transudation of fluid into interstitial & alveolar spaces.

2. Non-Cardiogenic Pulmonary Oedema

Mechanism:

- Due to **increased alveolar-capillary membrane permeability** without elevation in pulmonary capillary pressure.

Causes:

- **ARDS** (sepsis, trauma, aspiration, pneumonia)
- **Inhalation injury** (smoke, toxins)
- **High-altitude pulmonary oedema (HAPE)**
- **Neurogenic pulmonary oedema** (CNS injury, SAH)
- **Re-expansion pulmonary oedema** (rapid drainage of pleural fluid or pneumothorax)

Sequence:

Injury/inflammation → ↑ permeability of alveolar-capillary barrier → protein-rich fluid leakage into interstitium & alveoli.

Causes

1. Cardiogenic (↑ Pulmonary capillary hydrostatic pressure)

- Left ventricular failure (systolic/diastolic dysfunction, CHF)
- Ischemic heart disease (acute MI)
- Mitral valve disease (stenosis/regurgitation)
- Volume overload (renal failure, aggressive IV fluids)
- Severe hypertension

2. Non-cardiogenic (↑ Alveolar-capillary membrane permeability)

- **ARDS** – sepsis, trauma, aspiration, pneumonia
- **HAPE** – high-altitude pulmonary oedema
- **Inhalation injury** – smoke, toxins
- **Neurogenic oedema** – head trauma, intracranial hemorrhage
- **Re-expansion oedema** – after pneumothorax/pleural effusion drainage
- **Toxin/drug exposure** – narcotics, chemotherapy agents
- **Acute kidney injury** with fluid overload
- **Sepsis / SIRS**
- **TRALI** – transfusion-related acute lung injury
- **Near-drowning** – aspiration of water
- **Anaphylaxis**

Imaging Features

1. Chest X-ray

Feature	**Cardiogenic**	**Non-cardiogenic**
Heart size	Enlarged (cardiomegaly)	Normal
Distribution	Perihilar "bat-wing" opacities	Diffuse, often peripheral
Interstitial oedema	Kerley B lines, peribronchial cuffing	May be present
Pleural effusion	Common, bilateral	Less common
Other	Cephalization of pulmonary vessels	Air bronchograms common

2. CT Scan

- **Cardiogenic:**
  - Smooth interlobular septal thickening
  - Peribronchial cuffing
  - Ground-glass opacities
  - Pleural effusions
  - Cardiomegaly
- **Non-cardiogenic:**
  - Ground-glass opacities ± patchy consolidation
  - Irregular interlobular septal thickening
  - Air bronchograms
  - Peripheral predominance (ARDS)

    - Minimal/absent cardiomegaly

3. Ultrasound

- Multiple vertical **B-lines** (>3 per space) in both lungs
- Pleural effusions (more in cardiogenic)

4. MRI (limited use)

- Increased T2 signal intensity in affected lung regions

Differential Diagnosis

- **Pneumonia** – especially early bilateral bronchopneumonia
- **Interstitial lung disease** – acute interstitial pneumonia (AIP)
- **Pulmonary hemorrhage** – diffuse alveolar haemorrhage syndromes
- **Lymphangitic carcinomatosis** – smooth/irregular septal thickening + nodularity