DNB Dec 2020 – Paper 1

PART - A

1.

- a) Imaging evaluation of a patient with Cushing's syndrome (5 marks)
- b) Role of imaging in differentiating adrenal adenoma from adrenal metastasis (5 marks)

2.

- a) Approach for incidental thyroid nodules detected on imaging (4 marks)
- b) **TI-RADS and its clinical utility** (6 marks)

PART - B

3.

- What is lipoma arborescens? (2 marks)
- Enumerate imaging findings on US and MRI (4 marks)
- Discuss any two differential diagnoses (4 marks)

4.

- **Indications** of MRI in breast cancer (2 marks)
- Technique (3 marks)
- Advantages (3 marks)
- Limitations (2 marks)

PART - C

5.

- Etiology of femoroacetabular impingement
- Imaging findings on plain radiographs and MRI (10 marks)

6.

- Enumerate the various posterior fossa tumours in paediatric age group

 – (2 marks)
- Brief protocol for evaluation (2 marks)
- Salient imaging features of the three most common paediatric posterior fossa tumours (6 marks)

PART - D

7.

- Salient CT and MRI features in acute ischemic stroke within 6 hours of onset – (5 marks)
- Algorithm-based approach for diagnosis and management guidance (5 marks)

8.

- Imaging protocol of paranasal sinuses for FESS (3 marks)
- Important anatomical landmarks & anatomical variants for FESS evaluation (3 marks)
- Radiological patterns and features of Aspergillus infection of paranasal sinuses – (4 marks)

PART – E

9.

- a) Radiological anatomy of ankle ligaments (3 marks)
- b) MR sequences and planes for ankle joint evaluation -(3 + 4 marks)

10.

- Imaging modalities in intractable epilepsy (3 marks)
- Imaging protocols (3 marks)
- Imaging findings of mesial temporal sclerosis (4 marks)

Q1. a) Imaging evaluation of a patient with Cushing's syndrome.

b) Role of imaging in differentiating adrenal adenoma from adrenal metastasis.

Answer

Imaging Evaluation of Cushing's Syndrome

1. Pre-Imaging Clinical & Laboratory Workup

(Confirm diagnosis before imaging)

- 24-hr urinary free cortisol
- Late-night salivary cortisol
- Low-dose dexamethasone suppression test
- Plasma ACTH levels → Differentiate ACTH-dependent vs ACTH-independent

2. Imaging Approach Based on ACTH Status

A. ACTH-Dependent Cushing's Syndrome (Pituitary source – Cushing's disease)

MRI Pituitary – Modality of choice

- High-resolution with **gadolinium contrast**
- Dynamic contrast-enhanced MRI → improves detection of microadenomas (<10 mm)
- Sensitivity: **50–60%** for microadenomas

CT Sella -

- Alternative when MRI contraindicated
- Less sensitive for small adenomas

B. ACTH-Independent Cushing's Syndrome (Adrenal source)

CT Adrenals - First-line

- Adenoma: usually <4 cm, homogeneous, smooth borders
- Carcinoma: larger, irregular margins, heterogeneous enhancement

MRI Adrenals -

- Problem-solving tool
- Better soft tissue contrast
- Chemical shift imaging for adenoma detection (signal loss on out-of-phase images)

C. Ectopic ACTH Production

CT Chest - First-line for bronchial carcinoid / SCLC

High-resolution for small nodules

MRI Chest / Mediastinum – For further characterization

Somatostatin Receptor Scintigraphy (SRS) or PET-CT

- Detect occult neuroendocrine tumors
- SRS: radiolabeled octreotide binding to somatostatin receptors
- FDG PET-CT: for aggressive tumors

3. Advanced / Functional Imaging

Inferior Petrosal Sinus Sampling (IPSS) -

- Indicated when MRI is negative or equivocal
- Compares central vs peripheral ACTH levels
- Gold standard for distinguishing pituitary vs ectopic ACTH source

4. Follow-Up Imaging

- Post-surgery: assess completeness of resection
- Long-term surveillance for recurrence (especially adrenal carcinoma / ectopic tumors)

Role of Imaging in Differentiating Adrenal Adenoma from Metastasis

- 1. Computed Tomography (CT)
- a. Hounsfield Units (HU) Unenhanced CT
 - Adenoma: ≤10 HU → high intracellular lipid content (diagnostic)
 - Metastasis: Usually >10 HU (lipid-poor)

b. Contrast-Enhanced CT - Washout Calculations

Absolute Washout (%) =

Enhanced-DelayedEnhanced-Unenhanced×100\frac{Enhanced -

Delayed}{Enhanced - Unenhanced} \times

100Enhanced-UnenhancedEnhanced-Delayed×100

≥ 60% → Adenoma

Relative Washout (%) = Enhanced-DelayedEnhanced×100\frac{Enhanced - Delayed}{Enhanced} \times 100EnhancedEnhanced-Delayed×100
 ≥ 40% → Adenoma

c. Morphology

- Adenoma: Small (<4 cm), homogeneous, smooth borders
- Metastasis: Larger, irregular margins, heterogeneous ± necrosis

2. Magnetic Resonance Imaging (MRI)

a. Chemical Shift Imaging (CSI)

• Adenoma: ↓ signal on **out-of-phase** vs **in-phase** (intracellular lipid)

• Metastasis: No significant signal loss

b. T2-Weighted Imaging

• Adenoma: Typically low-intermediate signal

Metastasis: Often higher signal (↑ water content)

3. PET-CT with 18F-FDG

• Adenoma: Low metabolic activity → low FDG uptake

• **Metastasis:** High FDG uptake (SUV↑) due to malignant metabolism

4. Ultrasound (limited role)

• Adenoma: Hypoechoic, homogeneous

• **Metastasis:** Heterogeneous, irregular

• CEUS: Can add functional information but less commonly used

Summary: Imaging Features

Feature	Adenoma	Metastasis	
Unenhanced CT HU	≤ 10 HU	> 10 HU	
Absolute Washout	≥ 60%	< 60%	
Relative Washout	≥ 40%	< 40%	
CSI (MRI)	Signal loss on out-of-phase	No signal loss	
T2WI (MRI)	Low/intermediate signal	Higher signal	
FDG PET-CT	Low uptake	High uptake	
Morphology	Small, smooth, homogeneous	Large, irregular, heterogeneous	

2. a) Approach for incidental thyroid nodules detected on imaging.

b) TI-RADS and its clinical utility.

Answer

Approach to Incidental Thyroid Nodules on Imaging

1. Initial Clinical Assessment

- History & Risk Factors:
 - Age (<20 or >60 years ↑ risk)
 - Male gender
 - Family history of thyroid cancer
 - Prior head/neck irradiation
 - Rapid nodule growth
 - Hoarseness, dysphagia, compressive symptoms
- Physical Examination:
 - Nodule size, consistency
 - Fixity to adjacent structures
 - Cervical lymphadenopathy
- Previous Imaging:
 - Compare with prior scans to assess growth/change

2. Imaging Evaluation & Risk Stratification

Primary Modality: High-resolution Ultrasound

Key US Features:

Feature	Low Suspicion	High Suspicion	
Composition	Spongiform / cystic	Solid hypoechoic	
Echogenicity	Iso / hyperechoic	Markedly hypoechoic	
Margins	Smooth	Irregular / microlobulated / extrathyroidal	
Calcifications	None / coarse	Microcalcifications	
Shape	Wider-than-tall	Taller-than-wide	
Vascularity	Peripheral	Marked intranodular	

Risk Stratification Systems:

ACR TI-RADS

Assign points for composition, echogenicity, shape, margins, echogenic foci

TI-RADS category → guides FNA vs follow-up

• ATA Guidelines

- High / intermediate / low suspicion categories
- o Corresponding size thresholds for FNA

3. Indications for Fine-Needle Aspiration (FNA)

Risk Level (US)	ATA Threshold for FNA
High suspicion	≥ 1 cm
Intermediate	≥ 1 cm
Low suspicion	≥ 1.5–2 cm
Very low suspicion	≥ 2 cm (or consider observation)
Pure cystic	No FNA unless symptomatic
Additional: Any size if suspicious cervical lymphadenopathy or high-risk patient	

4. FNA Procedure & Cytology

• Technique: US-guided, multiple passes

• Reporting System: Bethesda System

- 1. Non-diagnostic
- 2. Benign
- 3. AUS/FLUS
- 4. Follicular neoplasm / suspicious for follicular neoplasm
- 5. Suspicious for malignancy
- 6. Malignant

5. Management Based on FNA

Bethesda Category	Risk of Malignancy	Management
1. Non-diagnostic	5–10%	Repeat FNA
2. Benign	0–3%	US follow-up (6–18 months)
3. AUS/FLUS	10–30%	Repeat FNA / molecular testing
4. Follicular neoplasm	25–40%	Molecular testing / diagnostic surgery
5. Suspicious	50–75%	Surgery
6. Malignant	97–99%	Surgery

6. Follow-up & Special Considerations

- Benign nodules: US at 6–18 months, then 3–5 years if stable
- **High-risk patient:** Closer surveillance
- **PET-positive thyroid nodules:** FNA regardless of size
- **Pregnancy:** FNA indicated for suspicious nodules; defer surgery until postpartum unless aggressive tumor

ACR TI-RADS (Thyroid Imaging Reporting and Data System)

1. Scoring System

Features & Points (Assign points per category → sum total → determine TI-RADS category)

Category Subcategory		
Composition	Cystic / spongiform	0
	Mixed cystic-solid	1
	Solid or almost solid	2
Echogenicity	Anechoic	0
	Isoechoic / hyperechoic	1
	Hypoechoic	2
	Very hypoechoic	3
Shape	Wider-than-tall	0
	Taller-than-wide	3
Margins	Smooth	0
	III-defined	0
	Lobulated / irregular	2
	Extrathyroidal extension	3
Echogenic Foci	None	0
	Large comet-tail artifact	0
	Macrocalcifications	1
	Peripheral (rim) calcifications	2
	Punctate echogenic foci (microcalcifications)	3

2. TI-RADS Categories & Management

Category	Points	Risk of Malignancy	FNA Recommendation	Follow-up if No FNA
TR1	0	Benign	None	None
TR2	2	Not suspicious	None	None
TR3	3	Mildly suspicious	≥ 2.5 cm	US at 1, 3, 5 years
TR4	4–6	Moderately suspicious	≥ 1.5 cm	US at 1, 2, 3, 5 years
TR5	≥ 7	Highly suspicious	≥ 1.0 cm	Annual US up to 5 years

3. Clinical Utility

- **1. Standardization** Reduces inter-reader variability in reporting thyroid nodules.
- **2. Risk Stratification** Objective scoring helps determine malignancy risk.
- **3. Guidance for FNA** Avoids unnecessary biopsies; prioritizes high-risk nodules.
- **4. Follow-up Protocol** Defines surveillance intervals for non-biopsied nodules.
- **5. Resource Optimization** Minimizes patient anxiety, reduces cost.

4. Limitations

- **Inter-observer variability** in subjective features (margins, echogenicity, calcifications)
- False positives benign nodules sometimes appear suspicious
- False negatives some malignant nodules may appear benign
- Not a substitute for **clinical judgment** or **high-risk history** consideration

3. What is lipoma arborescens? Enumerate imaging findings on US and MRI. Discuss any two differential diagnosis.

Answer

Lipoma arborescens (LA) is a **rare**, **benign intra-articular synovial lesion** characterized by:

- Villous lipomatous proliferation of the synovium
- Replacement of subsynovial tissue by mature adipocytes
- Macroscopically resembles "tree-like" fronds floating in joint fluid

Epidemiology

- Prevalence: <1% of all lipomatous lesions
- Typical age: **5th–7th decade** (but reported in children and young adults)
- Sex: No strong predilection
- Usually unilateral (bilateral rare)
- **Most common site**: Knee joint → suprapatellar bursa > other compartments
- Rare sites: Hip, shoulder, wrist, elbow, tendon sheaths

Etiology & Associations

- **Primary (idiopathic)** no underlying joint disease
- **Secondary** associated with chronic joint irritation:
 - Osteoarthritis (most common association)
 - Rheumatoid arthritis
 - Psoriatic arthritis
 - Prior trauma or surgery
 - Chronic synovitis

Clinical Presentation

- Chronic, painless joint swelling
- Intermittent or persistent **effusion**
- Mild to moderate arthralgia
- Gradual onset
- Usually no locking (unless associated loose bodies)
- Limited range of motion possible in advanced cases

Pathology

- Gross: Yellow, villous, and fatty fronds protruding from synovium
- Microscopy:
 - Hypertrophied villi
 - Core replaced by mature fat cells
 - Overlying synovial lining intact

Minimal inflammation

Imaging Features

1. Plain Radiography

- May be normal
- Soft tissue density due to effusion
- Occasionally fatty lucencies
- Degenerative changes common
- Bone erosions: rare

2. Ultrasound

- Joint effusion with echogenic frond-like projections
- Projections move with joint manipulation
- No posterior acoustic shadowing
- Doppler: usually no significant hypervascularity

3. CT

- Intra-articular frond-like mass of **fat density** (–80 to –120 HU)
- Surrounded by joint fluid
- Minimal or no enhancement post-contrast
- Coexistent degenerative changes may be seen

4. MRI (Gold Standard for Diagnosis)

Pathognomonic appearance:

• Frond-like synovial mass of fat signal outlined by joint effusion

Signal Characteristics:

Sequence	Appearance	
T1	High signal (matches subcutaneous fat)	
T2	High signal (matches fat)	
Fat-suppressed (T1 FS, STIR)	Signal dropout (fat suppression)	
GRE	Chemical shift artifact at fat–fluid interface possible	
Post-contrast	No enhancement of fatty component; mild synovial rin enhancement possible if inflamed	

Additional MRI Findings:

- Moderate-to-large joint effusion
- Associated osteoarthritic changes
- Minimal synovial thickening beyond fronds

Treatment

- Synovectomy (arthroscopic or open) is curative
- Recurrence is **uncommon**
- Address underlying joint pathology to prevent recurrence

Prognosis

- Benign, non-aggressive
- Excellent outcome post-surgery

Differential Diagnosis – Imaging

Condition	MRI Signal	Distinguishing Features
Lipoma Arborescens	Fat signal on all sequences; frond-like; fat-sat suppression	Villous fronds; associated effusion
Synovial Lipomatosis	Fat signal; diffuse synovial infiltration	No villous projections
PVNS	Low T2 (hemosiderin), blooming on GRE Hemosiderin-laden thickened synovium signal	
Synovial Chondromatosis	Nodules with cartilage signal; possible calcifications	Multiple intra-articular loose bodies; may calcify
Synovial Hemangioma	High T2; avid enhancement	Vascular channels; phleboliths possible
Nonspecific Synovitis	Thickened synovium; variable T2	Lacks fat signal

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4. Indications, technique, advantages and limitations of MRI in imaging of breast cancer.

Answer

1. MRI Acquisition

Breast MRI requires a **high-field strength scanner** (≥1.5T, preferably 3T) with a **dedicated breast coil**. The patient is positioned **prone** to:

- Minimize motion artifacts.
- Optimize lesion conspicuity by allowing the breast to be fully extended.

2. Breast MRI Sequences

A multiparametric approach using various MRI sequences enhances lesion characterization and differentiation of benign from malignant pathology.

Sequence	Purpose
T1-weighted (Pre-contrast)	Evaluates fat, hemorrhage, post-surgical changes
DCE MRI (Dynamic Contrast-Enhanced)	Detects neoangiogenesis using gadolinium (hallmark of malignancy)
Diffusion-Weighted Imaging (DWI)	Assesses tumor cellularity ; ADC maps help in malignancy grading
T2-weighted (Fat-sat or STIR)	Differentiates cystic vs. solid lesions
Kinetic Curve Analysis	Time-signal intensity curve; washout = malignancy

Protocol

Parameter	Value	
Gadolinium dose	0.1 mmol/kg	
Rate	2 mL/sec	
Slice thickness	3 mm, 2D or 3D spoiled gradient echo (SPGR)	
Peak enhancement	~90 seconds post-injection	
Dynamic imaging	Every 1 minute for 8 minutes (8 + 1 pre)	
Total sequences	~9 (1 pre-contrast + 8 post-contrast)	

Breast MRI Protocols

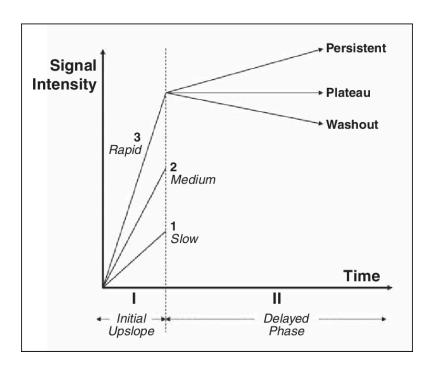
Parameter	Standard Protocol	Short Protocol	Abbreviated Protocol	Ultrafast Protocol
Total Sequences	9 (1 pre + 8 dynamic)	~3	2 (Pre + 1 Post)	2–3 rapid sequences
Pre-contras t T1	Yes	Yes	Yes	Yes
Dynamic Imaging	8 post-contrast (every 1 min till 8 min)	2 post-contrast (at 4 and 8 min)	1 post-contrast (at 4 min)	Multiple post-contrast (every 7–15 sec within 1 min)
T2-weighte d / STIR	Done	Optional	Optional	
DWI with ADC map	Done	Optional		
Kinetic Curve Analysis	Full curves: wash-in, wash-out, plateau)		Limited: early enhancement on 1st post-contrast image)	High-resolution: early enhancement rate, time to enhancement, SER)
Scan Duration	~15–20 mins	~8–10 mins	~5–6 mins	<2–3 mins
Gadolinium Dose	0.1 mmol/kg	Same	Same	Same
Goal	Comprehensive diagnostic/staging	Efficient lesion evaluation with reduced acquisition	Screening with morphology + early enhancement info	Early lesion enhancement kinetics for rapid triage
Use Case	Diagnostic, staging, surgical planning	Intermediate centers, triage	High-throughput screening, surveillance	Rapid detection, kinetic characterization within seconds

3. Post-processing Techniques

- **Subtraction Imaging**: Removes pre-contrast background noise to highlight enhancement.
- **Maximum Intensity Projection (MIP)**: Aids in rapid detection of enhancing lesions.

• **Kinetic Curve Analysis**: Determines enhancement characteristics (persistent, plateau, washout patterns).

Kinetic Curve Analysis in DCE-MRI



Kinetic curve analysis plays a pivotal role in **differentiating benign from malignant lesions** by assessing **the pattern of contrast uptake and washout**.

Types of Enhancement Curves

Enhancement Pattern	Initial	Later	Significance
Type I	Gradual enhancement	Gradual enhancement	Suggests benign lesions (e.g., fibroadenoma, cysts, fat necrosis).
Type II	Rapid	Plateau	Indeterminate—requires further evaluation.
Type III	Rapid	Washout	Highly suspicious for malignancy.

Key Features of Malignant Lesions on DCE-MRI

- Early, intense contrast uptake (peak within 90 sec).
- Heterogeneous or rim enhancement.
- Type III washout pattern in kinetic curve analysis.

Indications for Breast MRI

Breast MRI is **not a first-line screening tool for all women** due to cost and accessibility concerns. However, it plays a crucial role in several clinical scenarios.

1. High-Risk Screening

- Women with BRCA1/BRCA2 mutations.
- Strong family history (lifetime risk >20-25%).
- History of radiation to the chest (e.g., Hodgkin's lymphoma survivors).
- Li-Fraumeni, Cowden, or Bannayan-Riley-Ruvalcaba syndrome.

Benefit: Detects **cancers at an earlier stage** than mammography in these populations.

2. Preoperative Staging of Breast Cancer

- Multifocality (same quadrant) and multicentricity (different quadrants).
- Detection of contralateral breast cancer (seen in ~5% of cases).
- Extent of ductal carcinoma in situ (DCIS).

Benefit: Helps guide breast-conserving therapy (BCT) vs. mastectomy decisions.

3. Assessment of Neoadjuvant Chemotherapy (NACT) Response

- Monitors tumor shrinkage before surgery.
- Helps differentiate residual tumor vs. fibrosis.
- Predicts pathologic complete response (pCR).

4. Differentiation Between Scar vs. Recurrence

- Post-lumpectomy or post-radiation changes can mimic recurrence.
- Persistent or increasing enhancement on MRI suggests tumor recurrence.

5. Evaluation of Breast Implants

- Silicone implant rupture (intracapsular vs. extracapsular rupture).
- MRI is the gold standard for implant integrity assessment.

6. Occult Primary Breast Cancer

 Axillary metastases with no identifiable primary tumor on mammogram or ultrasound.

7. Problem-Solving Modality

- Inconclusive mammography/ultrasound findings.
- Suspicious nipple discharge with negative mammogram and ultrasound.

Advantages of Breast MRI

Highest Sensitivity for Breast Cancer Detection (~90-99%):

- Detects small tumors missed on mammography.
- Superior for dense breast tissue evaluation.

Multiparametric Functional Imaging:

- Differentiates between benign and malignant lesions.
- Provides information on vascularity, cellularity, and diffusion.

No lonizing Radiation:

• Suitable for young women and high-risk groups.

Accurate Preoperative Staging:

- Reduces re-excision rates and improves surgical planning.
 Superior Evaluation of Breast Implants:
- Best modality for detecting silent implant ruptures.

Limitations of Breast MRI

Lower Specificity (False Positives) (~20-30%):

- Benign enhancing lesions (fibroadenomas, papillomas, fat necrosis) can mimic cancer.
- Biopsy confirmation is often needed.

Expensive and Limited Availability:

- Not widely available, especially in resource-limited settings.
- Cost can be a barrier for routine use.

Time-Consuming:

- Standard MRI: ~30–45 minutes.
- Shortened Protocols (Abbreviated MRI): ~10 minutes.

Requires Gadolinium Contrast:

- Contraindicated in renal impairment (eGFR <30 mL/min/1.73 m²).
- Risk of nephrogenic systemic fibrosis (NSF).

Not a First-Line Screening Tool:

 Mammography remains the primary screening tool due to its cost-effectiveness.

Recent Advances in Breast MRI

- 1. Abbreviated Breast MRI (AB-MRI)
 - Shortens MRI exam time (<10 minutes).
 - Retains high sensitivity while improving accessibility.
 - Promising for high-risk population screening.

2. Diffusion-Weighted Imaging (DWI)

- Non-contrast technique that evaluates tumor cellularity.
- High ADC values → Benign lesion.
- Low ADC values → Malignant lesion.

3. Ultrafast Breast MRI

- Captures early contrast enhancement within 10-15 seconds.
- Helps differentiate benign vs. malignant lesions based on contrast uptake kinetics.

4. MR Spectroscopy

- Detects choline peak, a marker of malignancy.
- Reduces unnecessary biopsies.

5. Artificial Intelligence (AI) in Breast MRI

- Automated lesion detection and characterization.
- Reduces interpretation time and false positives.

5. Discuss the etiology of femoroacetabular impingement and imaging findings on plain radiographs and MRI.

Answer

FAI syndrome is **hip pain and motion restriction** caused by abnormal contact between the femoral head—neck junction and the acetabular rim during physiological range of motion. It may lead to **labral and cartilage injury** and predispose to **early hip osteoarthritis**.

Epidemiology

- Active young & middle-aged adults most affected.
- Cam type → young athletic men.
- Pincer type → middle-aged women.
- Often seen in athletes in sports involving repetitive hip flexion (e.g., soccer, hockey, martial arts).

Risk Factors

- **High-impact sports during adolescence** (risk ↑ during physeal closure).
- Overuse activity.
- Previous hip disorders:
 - Slipped Capital Femoral Epiphysis (SCFE)
 - o Perthes disease
- Anatomic variations:
 - Coxa profunda
 - o Protrusio acetabuli
 - Acetabular retroversion
- Post-traumatic deformities.

Clinical Features

- Pain location: Groin (most common), buttock, lateral hip, thigh, or back.
- Pain character: Motion or position-related; worsens with flexion, adduction, internal rotation.
- **Mechanical symptoms:** Stiffness, clicking, catching, locking.
- Key test: FADIR (flexion, adduction, internal rotation) sensitive, not specific.

Pathophysiology

- Cam type:
 - Loss of femoral head sphericity → abnormal shear stress at chondrolabral junction → anterosuperior labral tear and cartilage delamination.

Pincer type:

- Acetabular overcoverage → labral compression anterosuperiorly → cartilage damage, with possible posteroinferior contrecoup chondral lesions.
- Mixed type: Combination of both morphologies.

Locations

- Cam bump: Anterosuperior head—neck junction (lateral to physeal scar).
- Pincer overcoverage: Often anterosuperior acetabulum (acetabular retroversion, os acetabuli).
- Labral injury: Anterosuperior acetabular rim.
- Contrecoup lesion: Posteroinferior cartilage injury in pincer type.

Imaging Approach

1. Plain Radiographs

- Pelvis AP: Neutral tilt/rotation essential.
 - Lateral center-edge angle (↑ = overcoverage).
 - Acetabular index (↓ = overcoverage).
 - Ilioischial line (medial overlap = coxa profunda/protrusio).
 - Crossover sign, ischial spine sign (acetabular retroversion).
 - Posterior wall sign.
 - Pistol-grip deformity (cam).
- Lateral views (Dunn 45°, Dunn 90°, frog-leg, Meyer lateral, Lequesne false profile, cross-table lateral):
 - Femoral head-neck offset ↓ in cam.
 - Anterior center-edge angle (↑ = overcoverage).

2. CT

- Excellent for osseous morphology & 3D surgical planning.
- Radial reconstructions along femoral neck.
- Cam morphology:
 - Osseous bump anterosuperiorly, cysts, herniation pits.
 - Alpha angle >55° (anterior) or >60° (anterosuperior).
 - Femoral head–neck offset <6–7 mm.

• Pincer morphology:

Acetabular depth ↑, retroversion, ossicles.

3. MRI

- Morphology + labral and cartilage pathology.
- 3D radial sequences along femoral neck.
- Findings:

- o Cam bump, decreased offset.
- o Labral tears, detachment.
- o Chondrolabral separation, cartilage delamination ("carpet lesion").
- Paralabral cysts.
- Measure alpha angle, femoral head-neck offset, acetabular retroversion.

4. MR Arthrography

- ↑ Sensitivity for labral tears & chondral lesions.
- Direct intra-articular contrast outlines labral detachment, carpet lesions.
- CT arthrography may be most accurate for labral tears.

Treatment & Prognosis

- Conservative: Activity modification, NSAIDs, physiotherapy.
- **Surgical:** Arthroscopic/open osteochondroplasty, acetabular rim trimming, labral repair, possible osteotomy.
- Prognosis: Good if treated before advanced cartilage loss; untreated → early OA.

Complications

- Labral tear.
- Chondrolabral separation.
- Cartilage injury (carpet lesion, delamination).
- Early osteoarthritis.

Q6. Enumerate the various posterior fossa tumours in the paediatric age group. Describe in brief the protocol for evaluation of these patients. Discuss the salient imaging features of the three commonest paediatric posterior fossa tumours.

Answer

Posterior Fossa Tumors in Pediatric Patients

Posterior fossa tumors are the most common brain tumors in children, comprising ~60% of pediatric intracranial neoplasms. Their presentation is often due to **mass effect on the brainstem and cerebellum**, and **obstructive hydrocephalus** from 4th ventricle compression.

1. Medulloblastoma

- **Epidemiology:** Most common malignant brain tumor in children, peak age 3–8 years.
- Location: Midline cerebellar vermis, often extending into the 4th ventricle.
- MRI features:
 - **T1:** Iso- to hypointense.
 - o **T2:** Variable (often slightly hyperintense).
 - o **DWI:** Restricted diffusion (high cellularity).
 - o **Post-contrast:** Homogeneous or heterogeneous enhancement.
 - Additional: May cause hydrocephalus, drop metastases (evaluate spine MRI).
- **Key point:** Belongs to **embryonal tumors**; aggressive, but radiation-sensitive.

2. Pilocytic Astrocytoma (PA)

- **Epidemiology:** Most common benign brain tumor in children; peak 5–15 years.
- **Location:** Cerebellar hemispheres > vermis.
- MRI features:
 - Classic: Cystic lesion with an enhancing mural nodule.
 - **T1:** Cyst is hypointense; nodule iso- to hypointense.
 - **T2:** Cyst hyperintense; nodule mildly hyperintense.
 - Post-contrast: Vivid enhancement of mural nodule.
- **Key point:** WHO grade 1; favorable prognosis after complete excision.

3. Ependymoma

• **Epidemiology:** 2nd most common malignant posterior fossa tumor in children <5 years.

- **Location:** Arises from floor of the 4th ventricle; often extends through foramina of Luschka/Magendie.
- MRI features:
 - **T1:** Iso- to hypointense.
 - **T2:** Hyperintense with heterogeneous signal.
 - o Calcifications: Common, better seen on CT.
 - Post-contrast: Heterogeneous enhancement.
- **Key point:** Tumor plasticity allows extension through foramina into cisterns.

4. Brainstem Glioma

- **Epidemiology:** Common in children 5–10 years; DIPG is most aggressive.
- Location: Pons (DIPG), midbrain, medulla.
- MRI features:
 - o **DIPG:** Expansile, T2 hyperintense, minimal enhancement.
 - Focal gliomas: Better circumscribed, often low-grade.
- **Key point:** Biopsy often avoided in DIPG; diagnosis is radiologic.

5. Atypical Teratoid/Rhabdoid Tumor (AT/RT)

- **Epidemiology:** Highly malignant, usually <3 years.
- MRI features:
 - Heterogeneous due to hemorrhage, necrosis.
 - Variable enhancement.
 - May have calcifications.
- **Key point:** Associated with **INI1/SMARCB1 deletion**.

6. Choroid Plexus Papilloma/Carcinoma

- **Epidemiology:** Rare, can occur in 4th ventricle in children.
- MRI features:
 - Lobulated, frond-like enhancing mass.
 - Associated with hydrocephalus due to CSF overproduction/obstruction.
- **Key point:** Papillomas benign; carcinomas malignant.

7. Hemangioblastoma

- Rare in children; consider in VHL.
- MRI: Cyst with enhancing mural nodule; flow voids from feeding vessels.

8. Cerebellar Lipoma

• Rare; high T1/T2 signal; fat-saturated sequences confirm diagnosis.

9. Other PNETs

• Similar to medulloblastoma but different histology; aggressive.

10. Metastases

• Rare in children; think of leukemia, lymphoma, neuroblastoma.

Radiological Evaluation Workflow

- **1. MRI brain with contrast** mainstay for lesion characterization.
 - Include DWI/ADC, SWI, T1/T2/FLAIR, post-contrast.
 - Evaluate hydrocephalus, brainstem compression, spinal drop metastases (if medulloblastoma/PNET/ATRT suspected).
- **2. CT brain** useful for calcification, acute bleed, bone changes.
- 3. Spinal MRI to detect CSF dissemination.
- **4. CSF cytology** after ruling out mass effect risk.
- **5. Genetic testing** for tumor predisposition syndromes.

Differential Clues on Imaging

Feature	Medulloblasto ma	Pilocytic Astrocytoma	Ependymoma	AT/RT
Age peak	3–8 yrs	5–15 yrs	<5 yrs	<3 yrs
Location	Midline vermis	Cerebellar hemisphere		
Cystic with mural nodule	Rare	Common	Possible	Uncommo n
Calcification (CT)	±	Rare	Common	Possible
Diffusion restriction	Strong	No	Mild-moderate	Often

Q7. Discuss in brief the salient CT and MRI features in an acute ischemic stroke patient arriving to the hospital within 6 hours of onset. Suggest an algorithm based approach for confirmation of diagnosis and guiding the management of such a patient.

Answer

Imaging Features in Acute Ischemic Stroke (< 6 hours)

1. Non-Contrast CT (NCCT)

Purpose: Rapid, first-line — exclude hemorrhage, detect early ischemic changes. **Early (< 6 hrs) CT Findings** — often subtle:

- 1. Loss of Gray-White Differentiation:
 - Insular ribbon sign blurring of insular cortex.
 - Obscuration of lentiform nucleus (caudate/putamen indistinct).
- 2. Sulcal Effacement:
 - Loss of normal CSF density in sulci due to cytotoxic edema.
- 3. Hyperdense Vessel Sign:
 - Hyperdense MCA/basilar intraluminal thrombus.
- 4. Subtle Hypoattenuation:
 - Low density in affected vascular territory; early cytotoxic edema.
- **5. ASPECTS score** (Alberta Stroke Program Early CT Score):
 - Assesses extent of early ischemia in MCA territory.
 - Lower score = worse prognosis.

2. MRI (if rapidly available, especially in stroke centers)

Purpose: More sensitive for hyperacute ischemia, detects lesions missed on CT. **Diffusion-Weighted Imaging (DWI)**

- Bright signal in ischemic tissue within minutes of onset.
- **Restricted diffusion** → confirmed by low signal on ADC map.

Apparent Diffusion Coefficient (ADC)

 Low ADC in core infarct region (due to restricted water motion in cytotoxic edema).

FLAIR

- Usually **normal in first 6 hrs** ("DWI-FLAIR mismatch" suggests hyperacute stroke, useful for wake-up stroke selection).
- Mild hyperintensity may develop after ~4–6 hrs.

T2 / T2 / SWI*

- SWI may show susceptibility vessel sign (thrombus blooming artifact).
- Detects small hemorrhagic transformation.

MRA (TOF or contrast-enhanced)

• Shows vessel occlusion without contrast (TOF) or with contrast (CE-MRA).

Comparative Table (< 6 hrs)

Feature	CT (NCCT)	MRI (DWI/FLAIR/SWI)	
Hemorrhage detection	Excellent (primary goal)	Good on GRE/SWI	
Early ischemia	Loss of gray–white differentiation, sulcal effacement	DWI bright, ADC low	
Intravascular thrombus	Hyperdense vessel sign	SWI blooming, TOF/MRA occlusion	
Posterior fossa stroke	Poor sensitivity	Excellent (DWI)	
Hyperacute infarct detection	Limited sensitivity	Highly sensitive within minutes	
Perfusion assessment	Needs CTP	MRI perfusion available	

Algorithm: Acute Ischemic Stroke (< 6 hrs from onset)

Step 1: Immediate Clinical Assessment

- Confirm stroke suspicion:
 - Sudden focal neuro deficit (FAST, NIHSS scoring).
- Establish onset time (or last known well).
- Check contraindications for thrombolysis/thrombectomy.
- ABC stabilization.

Step 2: Emergent Imaging

Goal — Exclude hemorrhage, confirm ischemia, identify occlusion, assess penumbra.

A. First-line — Non-Contrast CT (NCCT)

- Rule out intracranial hemorrhage (contraindication to tPA).
- Assess early ischemic signs → ASPECTS scoring.
- Look for hyperdense vessel sign (suggests thrombus).

B. Add if available immediately:

- CT Angiography (CTA):
 - Detect large vessel occlusion (LVO).
 - Guide thrombectomy decision.
- CT Perfusion (CTP) (optional in <6 hrs, more useful in 6–24 hrs window):

o Core vs penumbra assessment.

Step 3: MRI Pathway (if available without delay)

- MRI-DWI + ADC: Confirm ischemia within minutes.
- MRA/TOF: Show vessel occlusion.
- **SWI**: Detect thrombus & hemorrhage.
- **DWI–FLAIR mismatch**: Suggests hyperacute onset.

Step 4: Imaging-Based Decision Making

Imaging Result	Action	
Hemorrhage on NCCT/SWI	No tPA → Treat as hemorrhagic stroke	
No hemorrhage + ischemic changes ≤1/3 MCA territory + onset ≤4.5h	IV tPA candidate	
Large vessel occlusion on CTA/MRA + onset ≤6h	Direct to Mechanical Thrombectomy (with or without tPA)	
Small core, large penumbra (CTP/MR perfusion) beyond standard window	Consider extended thrombectomy window (up to 24h in select cases)	

Step 5: Example Workflow (< 6 hrs)

- 1. Arrival & ABC → Neuro exam + NIHSS.
- 2. Immediate NCCT (goal: <20 min door-to-scan).
- 3. If no bleed:
 - Perform CTA (head + neck) immediately.
 - o Consider CTP or MRI if available and rapid.
- 4. Interpret findings:
 - Hemorrhage → neurosurgery/ICU.
 - o Ischemia, small core:
 - **■** <4.5 hrs → IV tPA.
 - LVO & <6 hrs \rightarrow Mechanical thrombectomy ± IV tPA.
- **5. Post-treatment**: ICU/stroke unit care.

Q8. Describe the imaging protocol of paranasal sinuses for FESS (Functional Endoscopic Sinus Surgery). What are the important anatomical landmarks and anatomical variants which should be looked for while evaluating for

FESS? Discuss the radiological patterns and features of sinuses.

Answer

Imaging Protocol of Paranasal Sinuses for Functional Endoscopic Sinus Surgery (FESS)

1. Preferred Imaging Modality

High-Resolution Computed Tomography (HRCT)

- Rationale: Gold standard for pre-FESS evaluation because it delineates bony anatomy and sinonasal variants critical for surgical planning.
- Acquisition:
 - Thin collimation: 0.5–1 mm slice thickness.
 - Multiplanar reconstruction (MPR): Axial, coronal, sagittal planes.
 - **FOV:** Covers frontal sinuses anteriorly to sphenoid sinus posteriorly, including nasal cavity and skull base.
 - o Radiation dose optimization: Low-dose protocols where possible.

Non-contrast HRCT is sufficient in most cases.

Contrast-enhanced CT is reserved for suspected:

- Complicated sinusitis (e.g., orbital or intracranial extension)
- Sinonasal tumors
- Postoperative complications

2. Recommended Planes & Their Utility

- Axial: Overall anatomy, sphenoid sinus, posterior ethmoid air cells, lamina papyracea, skull base.
- **Coronal:** Ostiomeatal complex (OMC) anatomy, frontal recess, relationship to orbit and cribriform plate.
- Sagittal: Sphenoid sinus ostium, frontal recess pathway, skull base slope.

3. Key Imaging Parameters

- **Slice thickness:** 0.5–1 mm (thin slices critical for surgical navigation systems).
- **Reconstruction interval:** ≤ slice thickness for isotropic voxels.
- **Kernel:** Bone algorithm for sharp detail.
- Matrix: 512 × 512 for optimal spatial resolution.

4. Interpretation Checklist for Radiologist (FESS-Oriented)

A. Sinonasal Disease

- Mucosal thickening, polyps, retention cysts.
- Complete/partial opacification of sinuses.
- Patterns suggestive of chronic sinusitis, fungal sinusitis, or neoplasia.

B. Critical Structures to Highlight

- **Orbit:** Lamina papyracea integrity
- **Skull base:** Keros classification (depth of olfactory fossa)
- Internal carotid artery and optic nerve canal dehiscence
- Cribriform plate asymmetry
- Any bone dehiscence or erosion

5. Role of 3D Reconstruction

- Optional but useful for surgical navigation and patient-specific surgical planning.
- Volume-rendered and surface-shaded reconstructions can depict complex anatomy and variants.

6. Radiology Reporting Pearls for FESS

- Always describe disease extent and relevant anatomical variants in the same report.
- Use a structured template for consistency and to avoid missing surgically critical details.
- Mention any anatomical hazard zones (low cribriform plate, dehiscent ICA, optic nerve exposure).
- Clearly identify **side dominance** of disease for surgical planning.

Anatomical Variants Relevant to FESS

Recognizing these on **preoperative HRCT** is crucial, as they can alter drainage pathways, predispose to sinusitis, and increase surgical risk.

1. Concha Bullosa

- Pneumatization of the middle turbinate.
- May narrow or obstruct the Ostiomeatal Complex (OMC) or ethmoid infundibulum.
- Report size, laterality, and effect on adjacent structures.

2. Deviation of Nasal Septum (DNS)

- Can cause asymmetrical nasal airflow, impeding mucociliary clearance.
- May displace turbinates laterally, narrowing drainage pathways.

3. Haller Cells

 Infraorbital ethmoid cells located along the orbital floor / maxillary sinus roof. May narrow the ethmoid infundibulum or contribute to maxillary sinus obstruction.

4. Supraorbital Ethmoid Cells

- Pneumatization of anterior ethmoid cells superiorly into the frontal bone above the orbit.
- Important for frontal recess anatomy mapping.

5. Onodi Cells (Sphenoethmoidal cells)

- Posterior ethmoid cells pneumatizing superolateral to the sphenoid sinus.
- Often intimately related to optic nerve and internal carotid artery (ICA)
 risk of injury during surgery.

Radiological Patterns & Features in Sinonasal Disease

1. Mucosal Thickening

- o On CT: Soft-tissue attenuation along sinus walls.
- o On MRI: Hyperintense on T2, hypointense on T1.
- Suggests inflammatory changes.

2. Sinus Opacification

- Partial or complete filling of sinus cavity.
- Etiologies: Chronic rhinosinusitis, polyposis, mucous retention cysts.

3. Air-Fluid Levels

- o Horizontal fluid density with air above, seen on CT.
- Sign of acute sinusitis or superimposed infection.

4. Polyps

- Smooth, expansile soft-tissue masses from mucosa.
- Common in nasal cavity, maxillary, ethmoid sinuses.
- Often bilateral in allergic fungal sinusitis.

5. Sinus Wall Erosion / Bone Remodeling

- Bone thinning, expansion, or erosion.
- Seen in:
 - Long-standing sinusitis (pressure remodeling)
 - Fungal sinusitis
 - Benign expansile lesions (mucocele)
 - Malignancy (aggressive destruction)

6. Pneumatization Variants

- Agger nasi cells: Most anterior ethmoid cells can narrow frontal recess.
- o Infraorbital cells: See Haller cells above.
- Supraorbital cells: Extension into frontal bone above orbit.

7. Frontal Recess Variants

- Shape and size variations affecting frontal sinus drainage.
- o Important for Draf procedure planning.

Q9. a) Discuss the radiological anatomy of ankle ligaments.

b) Briefly state various MR sequences and planes used to evaluate ankle joint on magnetic resonance imaging.

Answer

RADIOLOGICAL ANATOMY & IMAGING OF ANKLE LIGAMENTS

1. Lateral Ligament Complex

- Anterior Talofibular Ligament (ATFL)
 - o Origin: Anterior margin of lateral malleolus
 - **Insertion:** Neck of talus (non-articular surface)
 - Function: Resists inversion in plantarflexion; weakest and most frequently injured ligament in ankle sprains.
 - MRI appearance: Low-signal band on T1/T2; injury shows discontinuity, thickening, or high T2/STIR signal.

• Calcaneofibular Ligament (CFL)

- o **Origin:** Tip of lateral malleolus
- o **Insertion:** Lateral calcaneal wall (posterior to peroneal tendons)
- Function: Stabilizes ankle and subtalar joint in neutral/dorsiflexion.
- MRI: Best seen on coronal plane; injury often coexists with ATFL tear.

Posterior Talofibular Ligament (PTFL)

- Origin: Posterior lateral malleolus
- Insertion: Posterior talus (lateral tubercle)
- **Function:** Resists posterior talar translation; rarely injured except in high-energy trauma/dislocation.

2. Medial Ligament Complex (Deltoid Ligament)

Superficial Layer:

- o Components: Anterior tibiotalar, tibionavicular, tibiocalcaneal.
- Function: Prevents excessive eversion and external rotation.
- MRI: Low signal on all sequences; injury shows thickening, high T2/STIR signal.

Deep Layer:

- o Components: Posterior tibiotalar, deep anterior tibiotalar.
- **Function:** Stronger medial restraint; resists talar abduction.
- MRI: Shorter, thicker than superficial layer; injury is less common but more significant.

3. Imaging Modalities

A. Radiography

- Views: AP, lateral, mortise.
- Utility:
 - Detect avulsion fractures (e.g., tip of lateral malleolus = ATFL avulsion).
 - Assess talar tilt, mortise widening (>4 mm may suggest deltoid rupture).
 - o Identify associated fractures (Weber classification relevance).

B. Magnetic Resonance Imaging (MRI) – Gold standard for ligament evaluation MRI Sequences

- T1-weighted (T1WI): Anatomy, bone marrow fat, cortical detail.
- T2-weighted (T2WI): Fluid-sensitive; detects effusions, edema.
- Proton Density (PD): Balanced resolution and contrast; ideal for ligament/tendon evaluation.
- STIR / Fat-sat T2: Maximizes edema/fluid detection (acute injuries).
- Gradient Echo (GRE/SWI): Sensitive for hemorrhage, cartilage defects.

Imaging Planes

- **Axial:** Best for ATFL, PTFL, syndesmosis, peroneal tendons.
- Coronal: Best for CFL, deltoid ligament, subtalar joint.
- Sagittal: Best for anterior/posterior structures (Achilles, ATFL, PTFL).

C. Ultrasound

- **Advantages:** Dynamic assessment, immediate bedside evaluation, comparison with contralateral side.
- Findings: Hypoechoic gaps, discontinuity, thickening, hyperemia on Doppler.

4. Pathological Spectrum

Sprain Grading (MRI/US correlation)

- Grade I: Ligament thickening, mild edema; intact fibers.
- **Grade II:** Partial tear; fiber disruption with preserved continuity.
- **Grade III:** Complete tear; discontinuity, retraction, joint instability.

Avulsion Injuries

- ATFL → lateral malleolar avulsion fragment
- Deltoid → medial malleolar avulsion

Chronic Ankle Instability

• MRI: Attenuated/thickened ligaments, irregular low signal (fibrosis), talar tilt.

MRI Pitfalls

- Magic angle effect in tendons (avoid by proper foot positioning)
- Anisotropy in ultrasound mimicking hypoechoic tears
- Physiologic joint fluid misinterpreted as pathology in asymptomatic patients

MRI Appearance of Key Ankle Ligaments

Ligament	Best Plane	Normal MRI Signal	Acute Injury	Chronic Injury
ATFL	Axial	Low T1/T2	Thickening, high T2, discontinuity	Thinning, irregular low-signal band
CFL	Coronal	Low T1/T2	Discontinuity, edema around ligament	Irregular low signal, lax appearance
PTFL	Axial	Low T1/T2	Rarely torn unless dislocation	Usually intact
Deltoid (Superficial)	Coronal	Low T1/T2	High T2/STIR, fiber disruption	Thickened, low signal
Deltoid (Deep)	Axial/Coron al	Low T1/T2	High T2/STIR, fiber retraction	Fibrotic, attenuated

Q10. A 37-year-old female presents with history of intractable epilepsy. Discuss the imaging modalities and protocols used in workup of this patient. Describe imaging findings of mesial temporal sclerosis.

Answer

Imaging in Intractable Epilepsy

1. Role of Imaging

- **Purpose**: Identify the epileptogenic focus, guide surgical planning, and detect associated structural or functional abnormalities.
- Approach: Multimodal combines structural imaging (MRI, CT) and functional/metabolic imaging (PET, SPECT, fMRI, MEG).

2. Structural Imaging

A. Magnetic Resonance Imaging (MRI) - Gold Standard

Why: Best soft-tissue resolution; detects subtle cortical malformations and hippocampal sclerosis often missed by CT.

Protocol - Epilepsy Dedicated MRI (3T preferred):

- 1. High-Resolution 3D T1-weighted (e.g., MPRAGE, 1 mm isotropic)
 - Detects hippocampal atrophy, cortical dysplasia, tumors, vascular malformations.
 - Coronal slices perpendicular to the hippocampus for mesial temporal lobe epilepsy (MTLE).
- 2. T2-weighted & FLAIR (axial + coronal oblique)
 - Highlights gliosis, cortical/subcortical signal changes, and periventricular nodular heterotopia.
- 3. Double Inversion Recovery (DIR)
 - Improves lesion conspicuity by suppressing both CSF and white matter signals (for subtle cortical dysplasia).
- 4. Susceptibility-weighted Imaging (SWI)
 - Detects cavernomas, microbleeds, and hemosiderin deposits.
- 5. Diffusion-weighted Imaging (DWI)
 - Acute seizure-related changes, postictal diffusion restriction, stroke-like lesions.
- 6. Diffusion Tensor Imaging (DTI) & Tractography
 - Maps white matter tracts for pre-surgical planning.
- 7. MR Volumetry & T2 relaxometry
 - Quantifies hippocampal volume; detects subtle sclerosis.
- 8. Functional MRI (fMRI)

 Localizes language, motor, and memory areas to avoid postoperative deficits.

B. Computed Tomography (CT)

Indications:

- MRI contraindicated (pacemaker, metallic implants)
- Emergency settings trauma, acute hemorrhage.

Findings:

- Calcified lesions (e.g., tuberous sclerosis, neurocysticercosis, oligodendroglioma)
- Large tumors, malformations, infarcts.

Limitations: Low sensitivity for subtle cortical dysplasia or hippocampal sclerosis.

3. Functional / Metabolic Imaging

A. Positron Emission Tomography (PET)

- **Tracer**: ^18F-FDG PET (most common).
- Interictal PET: Hypometabolism in epileptogenic cortex.
- **Sensitivity**: High in temporal lobe epilepsy (TLE), lower in extratemporal.
- Other tracers: ^11C-flumazenil (GABA receptor), amino acid PET for tumors.

B. Single-Photon Emission Computed Tomography (SPECT)

- Radiotracers: Tc-99m HMPAO, Tc-99m ECD.
- **Ictal SPECT**: Hyperperfusion at seizure onset (most accurate if injected within 30 sec of seizure onset).
- Interictal SPECT: Hypoperfusion in epileptogenic zone.
- Subtraction Ictal SPECT Co-registered to MRI (SISCOM):
 - Improves localization by subtracting interictal from ictal perfusion and overlaying on MRI.

C. Magnetoencephalography (MEG)

- Detects magnetic fields from cortical electrical activity.
- Localizes interictal epileptiform discharges with high spatial accuracy.
- Often fused with MRI for magnetic source imaging (MSI).

D. EEG-fMRI

- Combines EEG spike detection with BOLD MRI mapping.
- Identifies seizure-onset zones and network connectivity.

4. Common MRI Findings in Intractable Epilepsy

- 1. Mesial Temporal Sclerosis (MTS)
 - Hippocampal atrophy, T2/FLAIR hyperintensity, loss of internal architecture.
- 2. Focal Cortical Dysplasia (FCD)

- Cortical thickening, blurring of gray–white junction, transmantle sign.
- **3. Tumors** (Dysembryoplastic neuroepithelial tumor, ganglioglioma, low-grade glioma)
- **4. Vascular lesions** (Cavernomas, AVMs)
- 5. Post-traumatic / post-infective gliosis
- **6. Developmental malformations** (Heterotopia, polymicrogyria, schizencephaly)

5. Surgical Planning Integration

- **Step 1**: Structural MRI → lesion localization.
- **Step 2**: Functional imaging (PET/SPECT) → metabolic/perfusion confirmation.
- Step 3: fMRI, MEG, DTI → eloquent cortex & tract mapping.
- Step 4: Image fusion & neuronavigation for resection planning.

Imaging Findings in Mesial Temporal Sclerosis (MTS)

1. MRI Findings (Mainstay for MTS Diagnosis)

A. Volume Loss (Hippocampal Atrophy)

- Most characteristic feature: Reduction in hippocampal size, usually ipsilateral to the seizure focus.
- **Asymmetry**: Compare with the contralateral hippocampus (side-to-side comparison).
- Often progressive in chronic cases.

B. T2 / FLAIR Hyperintensity

- Increased signal intensity in the hippocampal head and body.
- Reflects gliosis and neuronal loss.
- Best appreciated on coronal oblique images perpendicular to the hippocampal axis.

C. T1 Hypointensity

- Seen on high-resolution T1-weighted images.
- Represents chronic structural damage and gliosis.

D. Loss of Internal Architecture

- Blurring of hippocampal internal structure.
- Loss of normal laminar differentiation (e.g., disappearance of the hyperintense SR/SLM layers).

E. Extension to Adjacent Structures

 In advanced cases: atrophy of amygdala, parahippocampal gyrus, and entorhinal cortex.

2. Sequence-Specific Highlights

• **FLAIR**: Best for detecting subtle hyperintensity; CSF suppression improves hippocampal visualization.

- **DWI**: Usually normal in chronic MTS; may show restricted diffusion in acute post-ictal states.
- **Contrast Enhancement**: Absent in typical MTS; enhancement suggests alternate pathology (e.g., inflammation, tumor).

3. Functional Imaging Correlates

- **FDG-PET**: Hypometabolism in the mesial temporal lobe ipsilateral to seizure focus.
- **SPECT**: Interictal hypoperfusion; ictal hyperperfusion.
- Helps in surgical planning when MRI findings are subtle.

4. MRI Protocol for MTS

- High-resolution coronal T1 (3D volumetric sequences like MPRAGE or SPGR).
- Coronal oblique T2 perpendicular to hippocampal axis.
- Coronal oblique FLAIR.
- Optional: DWI, volumetric hippocampal measurements.