Neurosurgery Final Summary

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Outlines

Highlighted in Yellow: mentioned in the P.P

Highlighted in Blue: V2 **Pages:** 4,9 -14 - 22,23,24 - 32,33,35 - 47,49 - 50,55,56

دعاء قبل المذاكرة

اللَّهُمَّ إِنِّي أَسْأَلُكَ فَهْمَ النَّبِيِّينَ، وَحِفْظَ الْمُرْسَلِينَ وَالْمَلَائِكَةِ الْمُقَرَّبِينَ،
اللَّهُمَّ اجْعَلْ أَلْسِنَتَنَا عَامِرَةً بِذِكْرِكَ، وَقُلُوبَنَا بِخَشْيَتِكَ، وَأَسْرَارَنَا بِطَاعَتِكَ،
إِنَّكَ عَلَى كُلِّ شَيْءٍ قَدِيرٌ، وَحَسْبُنَا اللَّهُ وَنِعْمَ الْوَكِيلُ

Neuroanatomy & Meningitis

1. Functional Neuroanatomy: Reflexes and Cranial Nerve Pathways

Reflex Type	Afferent/Efferent Cranial Nerves (CN)	Clinical Key
Corneal Reflex	CN V (Trigeminal) / CN VII (Facial)	Evaluates the pontine integrity and protective facial response.
Cough Reflex	CN X (Vagus)	Critical for lower airway protection and medullary function.
Gag Reflex	CN IX (Glossopharyngeal) / CN X (Vagus)	Assesses the pharyngeal motor response and medullary health.
Salivary Reflex	CN VII (Facial)	High-yield indicator of parasympathetic autonomic regulation.
Vestibulo-ocular Reflex (VOR)	CN VIII / CN III, IV, VI	**DO NOT SIMPLIFY** as CN II and III. VOR involves the vestibulocochlear and extraocular motor nerves to maintain gaze stability.

Parasympathetic Analysis

The **Facial Nerve (CN VII)** is the specific cranial nerve identified as a carrier of parasympathetic fibers. This carries profound functional significance in autonomic regulation, particularly through the salivary reflex, and serves as a key diagnostic landmark for autonomic pathway patency.

2. Ocular Mechanics and Pupillary Clinical Indicators

Ocular Movement Matrix

When the eyeball is **adducted (turned inward)**, the mechanics of vertical movement shift. While multiple muscles contribute to depression, the **Superior oblique muscle** is the **primary depressor** of the eye *only* when it is in the adducted position.

Failure in this specific movement is a pathognomonic sign of Trochlear nerve (CN IV) dysfunction, distinguishing it from general Inferior Rectus deficits.

Pupillary Dilation

Finding	Clinical Association
Optic Nerve Injury	Failure of the afferent limb prevents pupillary response.
Brain Stem Herniation	Compressive emergency; requires immediate neurosurgical intervention.
Oculomotor Nerve Injury	Disrupts the efferent parasympathetic fibers responsible for constriction.

Abducent nerve injury (CN VI) and **loss of sympathetic tone** (Horner's Syndrome) are frequently misidentified as causes of a dilated non-responsive pupil. They must be **excluded**; sympathetic loss actually causes pupillary *constriction* (miosis), not dilation.

3. Neuro-Vasculature and Cellular Anatomy

Cerebral vascular architecture and cellular reception form the metabolic and communicative baseline of the Central Nervous System (CNS).

The External Carotid Artery provides the primary supply to the head and neck. Its definitive branches include:

- Ascending pharyngeal artery
- Lingual artery
- Facial artery
- Superior thyroid artery

Note: The **Ophthalmic artery** is the critical outlier; it does *not* originate from the external carotid artery.

Cerebral Circulation Dynamics

The regulation of cerebral blood flow is governed by two fundamental principles:

1. Cerebral circulation is primarily controlled by the **local metabolic factors and autoregulation**.
2. **Protective Vasoconstriction:** In response to rising systemic blood pressure, the cerebral vessels undergo **vasoconstriction** to prevent hypertensive damage to neural tissue.

Cellular Structure

At the microscopic level, the **Dendrites** are the specialized neuronal components dedicated to message reception. They initiate the communicative cascade by receiving signals from other cells.

4. Peripheral Nerve Supply and Vagal Nerve Pathophysiology

Muscular Innervation

In hand function assessments, the **Abductor pollicis brevis** is specifically supplied by the **Median nerve**. This muscle is the sentinel marker for median nerve health at the wrist.

Vagal Nerve Stimulation (VNS) Complications

Complication	Clinical Presentation
Dysphonia	Alteration in voice quality or volume.
Dyspnea	Shortness of breath during stimulation.
Cough	Reflexive irritation of the laryngeal pathway.
Hoarseness	Persistent pitch or quality changes in speech.

Must distinguish **Dysphonia** (a VNS complication) from **Dysphasia** (language/speech processing impairment). **Dysphasia is NOT a complication of VNS.**

5. Clinical Assessment of Meningitis

Contraindications for Lumbar Puncture:

When neuroimaging is unavailable, the following signs indicate that a Lumbar Puncture is unsafe.

1- Signs of Increased Intracranial Pressure (ICP):

- Papilledema
- Decreased level of consciousness

2- Focal Neurological Deficits:

- Right-sided hemiparesis (or other localizing signs)
- Seizures occurring during the current illness

CRITICAL SAFETY NOTE: Daily Aspirin Use is **NOT** a contraindication for performing a Lumbar Puncture.

General Management:

The gold standard of care dictates that **treatment for meningitis should NOT be deferred** while waiting for culture and sensitivity results.

Empiric antibiotics must be started immediately upon clinical suspicion.

6. CSF Analysis and Differential Diagnosis of Meningeal Infections

Etiology	Appearance	Pressure	WBC Count/Type	Protein	Glucose (Blood Sugar = 105 mg%)
Viral Meningitis	Clear	Normal	20 (**Lymphocytes**)	63 mg%	65 mg% (Normal ratio)
Viral Meningoencephalitis	Clear	Variable	9 (**Lymphocytes**)	63 mg%	65 mg% (Normal ratio)
Bacterial Meningitis	Turbid	Increased	High (**Polymorphic Neutrophils**)	**Increased**	**Low Glucose (< 40 mg%)**

Clinical Note: The presence of **RBCs** and clinical **signs of meningism** (neck stiffness, fever, confusion) further point toward an aggressive **Bacterial Meningitis** presentation.

7. Specialized Management: Bacterial Brain Abscesses

Brain abscesses represent a localized, encapsulated parenchymal infection.

The definitive treatment for bacterial brain abscesses is **Stereotactic aspiration followed by a 4–6 week course of IV antibiotics.**

- **Rationale:** Aspiration provides source control and culture-directed therapy, while the prolonged IV course ensures adequate penetration of the blood-brain barrier.
- **Excluded Options:** Empiric treatment without biopsy, oral-only regimens, or short two-week courses are inadequate and considered incorrect management for this localized pathology.

Head Injuries

1. Introduction

Head injuries serving as a leading cause of global morbidity and mortality. **1% of all deaths**.

Jordan: 4th most common cause of death.

lives are lost to trauma in Jordan, with head injuries contributing to over 50% of these trauma-related fatalities.

Epidemiological Core: Prevalence is highest among men compared to women and significantly higher in young populations compared to the elderly.

2. Clinical Epidemiology and Civil Etiology

Causes in Civil Life:

- **Road Traffic Accidents (60%):** The predominant cause of high-kinetic energy trauma.
- **Domestic Incidents (30%):** Primarily falls and household injuries.
- **Industrial Accidents:** Occupational trauma often involving falls from height or machinery.
- **Assaults:** Intentional blunt or penetrating force.
- **Sports:** Impact injuries occurring during recreational or competitive activities.

3. Primary and Secondary Pathological Frameworks

I. Primary Injuries (Immediate)

- **Closed (Simple) vs. Open (Compound)**
- **Anatomical Classification:**
    - **Scalp:** Lacerations, hematomas, and avulsions.
    - **Skull:** Geographic or morphological fractures.
    - **Brain:** Concussion, contusion, laceration, and diffuse axonal injury.

II. Secondary Events (Complications)

- **Timing:** Early vs. Late complications.

4. Clinical Assessment: History and Physical Examination

The "Golden Hour" demands strategic adherence to the **ABCDE protocol**.

Clinical History

Requirement	Significance
Time and Type of Trauma	Essential for determining energy transfer and timeline for secondary edema.
History of Convulsions	Indicates cortical irritation and immediate risk of secondary hypoxia.
Loss of Consciousness (LOC)	Must assess for the "Lucid Interval," a hallmark of evolving hematomas.
Amnesia Types	Differentiate between Post-traumatic (PTA) and Retrograde amnesia.

Examination:

- **Airway Patency:** Immediate priority to prevent secondary hypoxic insult.
- **Blood Pressure and Respiration: Clinical Pearl:** Shock is rare in adult head injuries unless associated with severe scalp exsanguination; however, it is a common finding in infants.
- **Level of Consciousness:** Via **Glasgow Coma Scale (GCS)** and **Trauma Scale (Score)**.
- **Pupillary Size:** A critical indicator of herniation syndromes and brainstem function.
- **Scalp examination:** Scalp wounds, Scalp hematomas, Battle's sign, Racoon eye.
- **Neurological examination**
- **Examination of other systems**

The Glasgow Coma Scale (GCS) -The most prognostic sign in acute head injury-

Points	Eye Opening	Best Verbal	Best Motor
6	...	...	Follows commands
5	...	Oriented	Localizes pain
4	Spontaneous	Confused	Withdraws to pain
3	In response to voice	Inappropriate words	Flexion (decorticate)
2	In response to pain	Incomprehensible Sounds	Extension (decerebrate)
1	None	None	None

Severity Classification:

Mild: GCS 14–15

Moderate: GCS 9–13

Severe: GCS 8 and below **(Mandatory airway protection).**

5. Initial Management:

1. ABC Assessment.
2. Establish patent airway.
3. Insert large-bore IV line.
4. **Skull X-rays: 3 views required.**
5. Cervical spine X-rays.
6. CT-scan AS INDICATED (The best modality for evaluation of head injury)

Note: 16% of head injuries are associated with cervical spine injuries, necessitating mandatory cervical stabilization and X-rays.

Administering corticosteroids isn't part of the management plan

6. CT Indications and Admission Criteria

Indications for CT Scan	Indications for Admission
Age < 5 or > 65 years	Age < 5 or > 65 years
Drug/alcohol consumption	Drug/alcohol consumption
Witnessed LOC > 5 minutes	LOC > 5 minutes
Amnesia ?	**Amnesia > 5 minutes**
GCS 14 and below	GCS 14 and below
Abnormal neurological signs	Abnormal neurological signs
Skull fractures	Skull fractures
Signs of skull base fractures	Signs of skull base fractures
Post-traumatic seizures	Presence of seizures
Abnormal skull X-rays	Abnormal brain CT scan
	Multi-trauma or Co-morbidity
	If the clinician is in doubt

7. Scalp Anatomy and Soft Tissue Injuries

SCALP acronym (**S**kin, **C**onnective tissue, **A**poneurosis/Galea, **L**oose areolar tissue, **P**ericranium)

Scalp Hematomas:

- **Sub-galeal Hematoma:** Caused by torn emissary veins. It is a soft, boggy swelling that **crosses midline sutures**. In infants, this can lead to hypovolemic shock.
- **Sub-periosteal (Subpericranial) Hematoma:** A firm perinatal injury localized underneath the pericranium. It is **limited by sutures** and does not cross the midline.

Management: Treat both with compression; avoid aspiration to prevent iatrogenic infection.

Injury Type	Management Protocol
Abrasions	Clean with antiseptic, apply antibacterial ointment, gauze.
Contusions	Cold compresses and analgesia; no active surgical treatment.
Wounds (Cut/Lacerated)	Shave, **feel the floor for fractures**, debride foreign material, stop bleeding, and **suture in two layers using a deep inverted suture** (Lacerated: Approximate edges if you can . You may need to rotate flaps or graft)
Avulsions (Degloving scalp)	**Partial:** Stop bleeding, antiseptic solution, Do debridement, Approximate edges if you can, You may need to rotate flaps or graft, Suture in two layers using a deep inverted suture **Complete:** Hemorrhage control, debridement, and flap rotation or grafting.

8. Skull Fractures: Classification and Surgical Management

Fractures are markers of significant force and predictors of intracranial hematoma; specifically, they carry a **5% risk of associated extradural hematoma**.

• Diffuse trauma causes **linear fractures**

• Localized trauma causes **depressed fractures** (One fragment, Comminuted (multiple))

• **Radiology Note:** Fractures should be evaluated on CT using **"Bone Windows"** for precision.

• THE PATIENT SHOULD BE ADMITTED (Compound depressed skull fractures require emergency treatment)

Linear Fractures Geographic Classification:

- **Vault:** Hair-line fractures in hair-bearing areas.
- **Base:** Basilar fractures involve the base of the cranium, involving the cranial fossae (more likely to injure cranial nerves).
- **Suture:** Diastatic fractures running along or initiating within sutures.

Basilar Fracture Presentation:

- **Anterior Fossa: Bilateral raccoon (panda) eyes**, subconjunctival hemorrhage (often left-sided in clinical case studies), and **Occasionally** CSF rhinorrhea, Epistaxis, Blindness.
- **Middle Fossa: Battle's sign** (post-auricular bruising), hemotympanum, and **Occasionally** CSF otorrhea (Temporal bone petrous part). Diagnosed as CSF leak by B2 transferrin.

Management Logic:

- **Linear:** Observation only unless they are complicated (and if the patient deteriorates do CT scan to rule out hematomas). They heal spontaneously over weeks. Open basal fractures require mandatory antibiotic coverage.
- A growing skull fracture is a complication of linear fractures in babies.

- **Depressed:** Surgical intervention is required if:
    - Depth > thickness of adjacent skull.
    - Compound/Open wound.
    - Associated seizures or focal neurological signs.
    - Cosmetic deformity or overlying eloquent brain areas.

- **Surgical Options (Depressed):** Simple elevation, craniectomy, or cranioplasty (using acrylic for immediate or delayed repair if the fracture was compound).
- Pond fractures of babies do not require elevation.

9. PRIMARY Brain Injuries: Mechanisms and Clinical Entities

Injury Type	Clinical Characteristics
Concussion	A **traumatically induced transient disturbance** of brain function. It is **diffuse and physiological**. Brief LOC, amnesia, but normal examination and CT.
Contusion	Focal structural injury; typically **temporal/frontal lobes**; coup/contre-coup (Acceleration-deceleration). High risk of secondary brain edema. Children are highly susceptible to brain contusions.
Laceration / Burst Lobe	Results from direct or penetrating trauma, acceleration-deceleration, or shearing forces. Causes permanent neurological deficits.
Diffuse Axonal Injury (DAI)	Extensive shearing at the grey-white interface (white tract hemorrhages, brain stem punctate hemorrhage). Results in deep coma **(GCS 3/15)** -Because DAI often involves the **Reticular Activating System (RAS)-**. Transition from prolonged coma to a stable or a transient vegetative state. Graded via MRI.

Management of these injuries is categorized by GCS: Mild (14-15), Moderate (9-13), and Severe (8 and below).

Management and Therapeutic Aims

The ultimate neurosurgical goal is to treat the primary pathology while aggressively preventing secondary injury, ABC then-depends on clinical picture and Ct-scan findings and pathology

- **Ventilation:** Secured based on clinical indications to prevent hypoxia.
- **Intracranial Pressure (ICP):** Active monitoring and surgical/medical management.
- **Medications:** Strategic use of Steroids, Anticonvulsants, and Antibiotics.
- **Surgery:**
    - **Burr hole** (Initial diagnostic/therapeutic intervention)
    - Craniotomy
    - Craniectomy

- In severe cranio-cerebral injuries, the recommended measure to reduce increased pressure, in their order of safety are:

  Mannitol, hyperventilation, barbiturates, hypothermia, decompressive craniotomy

Decompressive Craniectomy: This involves removing a large section of the skull (the bone flap) and **leaving it off** intentionally. The dura mater is usually augmented with a graft to allow the brain to expand beyond the limits of the skull, which immediately lowers intracranial pressure (ICP) and prevents brainstem herniation.

10. Physiological Secondary Events (the primary preventable causes of post-injury decline)

A- Hypoxia

Hypoxia—the decrease in oxygen supply to brain tissue—acts as a catalyst for rapid deterioration. It starves neurons and triggers a cycle of brain swelling.

- **Airway Obstruction**
- **ARDS (acute respiratory distress syndrome)**
- **Central Respiratory Depression**
- **Chest Injury:** Component of multi-system trauma limiting ventilation.
- **Unobserved Epileptic Attack:** increasing metabolic demand while compromising the airway.
- **Shock State:** Systemic failure of oxygen delivery.

B- Ischemia

Ischemia occurs when poor tissue perfusion fails to meet cellular metabolic needs, typically due to significant blood loss.

	Hypovolemic Shock
Adults	Rare in isolation; necessitates severe scalp lacerations or associated multi-system injury.
Infants	High risk from subgaleal or subdural hematomas due to their disproportionately small total blood volume.

Failure of tissue perfusion forces neurons into anaerobic metabolism, leading to rapid cellular failure. This systemic impairment is the primary catalyst for the development of cerebral edema.

C- Brain Edema

Brain edema is a critical secondary event where fluid accumulation within the brain parenchyma increases intracranial pressure.

Cytotoxic Edema In this mechanism**, the BBB remains intact**. The failure originates in cellular metabolism, leading to the collapse of the sodium/potassium pump in the glial cell membrane.

Pathophysiology: Sodium and water are retained intracellularly, **creating "edematous" astrocytes and neurons.**

Imaging: CT without contrast typically shows diffuse edema across territories supplied by the internal carotid arteries (ICA).

Vascular Impact: Critically, the capillary endothelial cells also become edematous. This narrows the vessel lumen, further restricting blood flow and creating a vicious cycle of worsening ischemia.

Interstitial Edema This results from a **failure of the ependymal lining (the CSF-brain barrier),** usually as a result of hydrocephalus.

Mechanism: Increased intraventricular pressure forces protein-free CSF to seep into the periventricular brain tissue.

Imaging: On MRI FLAIR sequences**, this is pathognomonic**, appearing as a hyperintense signal or "halo" immediately surrounding the ventricles.

Mechanisms of Brain Edema

Type	Problem in	Key Features	Imaging/Pathological Hallmark
Vasogenic	Blood-Brain Barrier (BBB)	Barrier breakdown	General post-traumatic swelling
Cytotoxic	Na/K Pump	Intracellular fluid retention	Edematous capillary endothelial cells
Osmotic	Plasma	Blood osmolality imbalance	Dilutional hyponatremia
Interstitial	CSF-Brain Barrier	Ependymal lining seepage	Periventricular hyperintense signal

11. Secondary Brain Injuries (Complications):

	Primary Complications
Early Complications	**Hyponatremia** **Intracranial Hematomas (EDH, SDH, SAH, ICH)** **CSF Leaks** Early Epilepsy (<1 week)
Late Complications	**Chronic SDH**, **Post-Traumatic Hydrocephalus** **Late Epilepsy (>1 week)** **Post-Traumatic Syndromes**

A- Early Complications:

1. Hyponatremia and Electrolyte Disturbance:

The systemic response to trauma often triggers inappropriate (ADH) secretion, leading to water retention and dilutional hyponatremia.

- **General Clinical Picture:** Confusion, nausea, and lethargy.
- **Level < 120 mmol/l:** Seizure.
- **Level < 105 mmol/l:** Risk of status epilepticus.

Treatment mandates the slow infusion of normal or hypertonic saline. Correction speed must be strictly calibrated based on the patient's age and weight.

The Risk: Rapid correction of chronic hyponatremia leads to **osmotic demyelination**, specifically **pontine myelinolysis** (irreversible destruction of the myelin sheath in the brainstem caused by the sudden shift in osmolality)

2. Cerebro-spinal Fluid (CSF) Leaks

A CSF leak indicates a structural breach requiring both a skull fracture (Usually skull base fracture) and a dural tear. This creates a bi-directional risk: the loss of intracranial volume and the entry of pathogens.

Categorization and Diagnosis

- **Rhinorrhea:** CSF drainage from the nose.
- **Otorrhea:** CSF drainage from the ear.

Diagnostic Protocol:

1. **Beta 2 Transferrin:** The gold-standard protein marker used to confirm fluid is indeed CSF.
2. **Pneumocele:** Intracranial air on imaging, proving a dural breach.
3. **Cisternogram:** High-resolution coronal CT **with contrast** used to localize the dural defect.
4. **Meningitis:** Often the first clinical sign of a previously "silent" leak.

Prognostic Statistics

- **General Leaks:** 80% stop spontaneously within 2 weeks.
- **Otorrhea:** Occurs in 2% of basilar temporal fractures; 95% dry up within 2 weeks. However, these patients carry a 4% risk of infection (meningitis).

3. Intracranial Hematomas: Acute Pathologies

Classification of Hematomas by Time

- **Acute: 0 to 3 days** (72 hours). These typically consist of fresh, clotted blood and appear hyperdense (bright white) on a CT scan.
- **Subacute: 4 to 21 days**. During this phase, the blood begins to liquefy and break down. On a CT scan, it often appears isodense
- **Chronic: More than 21 days**. These consist of liquefied blood and "old" breakdown products. They appear hypodense (darker than the brain, similar to CSF) on a CT scan.

Acute traumatic intracranial hematomas frequently manifest with: Seizures

Classic presentations of Traumatic intracranial hematoma:

Bradycardia, hypertension, hemiparesis and anisocoria

	Extradural (EDH)	Subdural (SDH)	Subarachnoid (SAH)	Intracerebral (ICH)
Location	Dura and bone	Brain and dura	Subarachnoid space	Within parenchyma
Source	**Arterial** (MMA) (Temporal region)	Bridging **veins**	Brain vessels / laceration	Intraparenchymal vessels
CT Imaging	Biconvex / Lens-shaped	Crescentic / Semilunar	Diffuse / Sulcal density	Focal hyperdense clot
Treatment	Burr-holes /Craniotomy evacuation	Evacuation (if mass effect)	Conservative Analgesia / Shunt	Monitoring / Surgery

Extradural Hematoma (EDH):

Usually limited by Skull sutures

Has a **mortality rate** (around 5–20%)

The sources of bleeding for an extradural hematoma include:

- Middle Meningeal Artery (Most Common)
- Middle Meningeal Vein
- Dural Venous Sinuses: superior sagittal sinus or transverse sinus (This is more common in pediatric cases where the dura is less tightly adhered to the bone).
- Diploic Veins

The timing of an EDH is dictated by the branch of the Middle Meningeal Artery (MMA) involved:

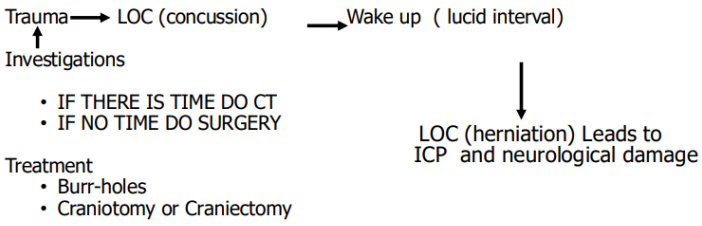
- **Stem of MMA:** Presentation within < 6 hours.
- **Anterior Branch:** 6–24 hours.
- **Posterior Branch:** 24–36 hours.

In adults (mainly <40), 90% of EDHs are associated with a skull fracture (fracture line).

In children, however, only 25% are associated with fractures, meaning a clear X-ray does **not** rule out an EDH in pediatric patients. (3 times as common in adults when compared to children)

Clinical Presentations:

- **Classical (25%):**



- Lucid interval corresponds to the period of accumulation of blood
- The best time of operation is before the ensuing of the second loss of consciousness which denotes herniation

- **Non-Classical (75%):** initial traumatic brain injury (TBI) without a lucid interval.

Acute Subdural hematoma:

The source of bleeding is the Pial/bridging veins, The blood clot develops between the brain and outer layer of the meninges

Does cross suture lines (but doesn't cross midline falx)

Subdural is associated with more severe injury, more dangerous than extradural hematoma (worse prognosis) and the highest mortality

The surgical treatment includes mainly evacuation of the hematoma and excision of the lacerated brain via a craniotomy

Subarachnoid Hemorrhage (SAH):

- SAH is managed conservatively with analgesia for headache unless complications arise.
- Its primary danger is the obstruction of arachnoid granulations, which prevents CSF absorption and causes hydrocephalus.

- Could be seen on CT as a separate entity or accompanying other injuries.

B- Late Complications:

Marked by a "delayed recovery" or a secondary plateau in the patient's progress.

1. Chronic Subdural Hematoma (CSDH)

CSDH typically presents **in the elderly or those on anticoagulants, often appearing 6 weeks after a minor or forgotten trauma.**

- **The Osmotic Theory:** This explains the characteristic 6-week delay. A semipermeable membrane forms around the initial clot; the organized clot then enlarges over time by absorbing CSF via osmosis.
- **Symptoms:** Headache, memory disturbances, unsteadiness, and eventually urinary incontinence.
- Associated with brain atrophy
- Can appear either hyperintense, isointense, or hypointense on MRI
- **Surgical removal:** Burr hole and subdural drain insertion for evacuation

2. Post-Traumatic Hydrocephalus

Usually a **communicating type** caused by blood in the CSF blocking absorption, SHOULD BE SUSPECTED IN DELAYED RECOVERY.

- **Red Flags:** Mental decline, ataxia (unsteady gait), and incontinence.
- **Management:** Often necessitates the surgical placement of a shunt.

3. Post-Traumatic Epilepsy

Most common symptom of intracranial hemorrhage

Categorized by the timing of the first seizure:

- **Early (< 1 week):** Occurs in 5% of cases. The risk doubles to 10% in children under age 5.
- **Late (> 1 week):** Occurs in 5% of cases; 50% of these patients will develop their first seizure within the first-year post-injury.

Occur due to: Depressed fracture, Intracranial hematoma, Brain contusion, Dural tear.

4. Post-Concussion Syndrome (PCS)

PCS remains a controversial clinical entity following minor head trauma, debated as having either organic (micro-structural) or psychological origins.

Symptom Classification

Somatic	Cognitive	Psychological
Headache	Concentration difficulty	Easy fatigability
Dizziness	Memory problems	Personality changes
Visual/Hearing problems	Dementia	Loss of libido
Balance difficulties		Insomnia / Photophobia

Brain Tumors

1. Introduction

Concerning brain tumors, all of the following data:

- ✓ The prevalence of brain tumors is between 5–18/100,000 population
- ✓ In Jordan, the prevalence is 5/100,000 population
- ✓ They are slightly more common in males than females
- ✓ Median age of presentation is 38–42
- ✓ Malignant brain tumors rarely metastasize outside CNS
- ✓ **Percentage of brain tumors within all body tumors:** 2%
- ✓ **Most common type of tumor to metastasize to the brain:** Lung

Core Essentials of Tumor Classification

Category	Primary Dichotomies / Standards
Origin	Primary Brain Tumor vs. Metastasis
Anatomical Location	Supratentorial vs. Infratentorial
Compartment	Intra-axial (parenchymal) vs. Extra-axial (meninges/nerves)
Demographics	Adult (mostly supratentorial) vs. Pediatric (mostly infratentorial)
Biological Behavior	Benign (non-invasive) vs. Malignant (invasive/rapid growth)
Diagnostic Gold Standard	**Magnetic Resonance Imaging (MRI)**

2. Principles of Classification and Pathogenesis

The clinical management of brain tumors has evolved beyond simple mass effect relief to a sophisticated understanding of the "Integrated molecular Diagnosis." (**WHO 2021)**

Classification by Cell of Origin

Cell of Origin	Tumor Types
Glial Cells	Astrocytoma, Oligodendroglioma, Ependymoma, Choroid plexus tumors
Neurons	Ganglioglioma, Gangliocytoma, Neuroblastoma
Pineal Gland	Pineal tumors (e.g., Pineoblastoma)
Embryonal	Medulloblastoma
Nerve Sheath	Schwannoma, Neurofibroma
Meningeal	Meningioma
Microglial Cells	Primary CNS Lymphoma
Pituitary	Pituitary Adenoma
Germ Cells	Germinoma, Teratoma
Malformations	Craniopharyngioma, Dermoid/Epidermoid cysts, Colloid cysts

Molecular Pathogenesis Triad

Carcinogenesis follows a specific molecular progression: **Induction, Promotion, and Progression**. This involves the activation of **oncogenes** (growth drivers) and the inactivation of **tumor suppressor genes** (biological brakes).

3. Etiology and Hereditary Risk Factors

Neuro-oncogenesis is multifactorial, yet genetic predisposition is exceptionally rare, usually manifesting within the context of specific germline mutations.

Inherited Syndromes and Associated Tumors

Syndrome	Primary Associated CNS Neoplasms
Neurofibromatosis Type 1 (NF1)	Optic nerve glioma, peripheral neurofibroma Arise from lateral or posterior side of spinal cord May lead to an increase of intravertebral foramen
Neurofibromatosis Type 2 (NF2)	**Bilateral acoustic neuromas (pathognomonic)**, multiple meningiomas
Tuberous Sclerosis	Subependymal glioma
Li-Fraumeni Disease	Glioma, ependymoma, medulloblastoma
Von Hippel-Lindau (VHL)	Hemangioma, hemangioblastoma

Environmental and External Risk Factors

- **Radiation:** secondary tumors.
- **Immunosuppression:** Primary CNS Lymphoma.
- **Viral Infection**
- **Chemical Exposure:** Specifically anthracen and nitrosurea derivatives.
- **Head Trauma:** meningiomas.

4. Clinical Presentation and Functional Localization

Symptoms arise from two mechanisms: generalized effects of increased ICP (headache, nausea) and focal deficits caused by mass effect on eloquent brain regions.

Key Manifestations

- **Headache:** Resulting from ICP elevation, traction on pain-sensitive dura/vessels, or secondary vision difficulties.
- **Epilepsy:** Occurs in 30% of tumor patients. Adult-onset epilepsy (>30 years old) is a "red flag" for intracranial mass.
- **Personality/Behavior:** Subtle changes often precede overt motor or sensory deficits.

Localization-Based Symptoms

Clinical signs are largely defined by their relationship to the **tentorium cerebelli**:

Region	Primary Signs and Symptoms
Supratentorial	Focal deficits based on lobar anatomy: motor/sensory loss, aphasia, visual field cuts (hemianopsia), and CN I/II palsies, hypothalamus and pituitary
Infratentorial	Hydrocephalus due to 4th ventricle compression, cerebellum sings (ataxia), and brain stem signs (CN III-XII palsies, long tract signs, altered consciousness).

- **Frontal Lobe:**
  - **Prefrontal Area:** Personality, judgment, and social behavior.
  - **Frontal Eye Field:** Voluntary eye movements.
  - **Broca's Area (Left Hemisphere):** Motor speech production (expressive aphasia).
- **Parietal Lobe:**
  - **Sensormotor Area:** Post-central gyrus for somatosensory processing.
- **Temporal Lobe:**
  - **Auditory Area:** Primary sound processing.
  - **Wernicke's Area (Left Hemisphere):** Speech comprehension (receptive aphasia).
- **Occipital Lobe:**
  - **Visual & Visual Association Areas:** Primary visual processing and interpretation.

Intracranial lesion (mass lesion) can present with:

Increased intracranial pressure,

Motor deficits

Fits

Cranial nerve lesion

Decerebrate rigidity/posture results because of a lesion in: Midbrain

Concerning the non-surgical treatment of brain tumor:

Antiepileptics

Analgesia

Head elevation 30

5. Diagnostic Investigations and Differential Diagnosis

MRI with gadolinium is the gold standard, particularly for the posterior fossa and skull base.

Modality	High-Yield Findings
Skull X-Ray	Calcifications (Oligo, Meningioma, Ependymoma, Craniopharyngioma ->) **Hyperostosis** (Meningioma) Bone destruction (Mets, Chordoma, Craniopharyngioma) **Erosion** of sella tursica **Sings of ICP** **Midline shift of a calcified pineal gland**.
Brain CT	Best for acute hemorrhage and bone destruction; identifies site and multiplicity. Enhancement tumors • High-grade gliomas (non-uniform/ring-enhancing) • Meningioma (uniform/dural tail) • Metastatic lesions (often multiple/well-circumscribed) • Acoustic neuroma and large pituitary tumors
Brain MRI	Superior for small masses, brain stem, and CPA. Shows decreased T1 and increased T2 signals. **T2 FIESTA** is preferred for Schwannomas.
Angiography or MRA	
PET scan	
CSF cytology	remember the contraindications
Biopsy	needle biopsy thru burr hole ,or stereo tactic biopsy image guided, or at time of treatment.
Tumor markers	

Enhancement tumors: indicates a breakdown of the blood-brain barrier (BBB).

Differential Diagnosis (Non-Neoplastic Mimics)

- **Vascular:** Hematoma, Aneurysm, AVM.
- **Infection:** Abscess, Tuberculoma, Hydatid cyst.
- **Parasellar Specifics:** Craniopharyngioma, tuberculum sellae meningioma, carotid aneurysm.
- **Cysts:** Arachnoid, Dermoid, Epidermoid.

6. Treatment Modalities

- **Medical Therapy:** medical treatment doesn't affect tumor itself. Primarily steroids (Dexamethasone) to reduce peritumoral edema; essential in GBM and metastases.
- **Surgical Treatment:**
  - Recommended for most tumors. Approaches include **Craniotomy**, **Craniectomy**, **Trans-sphenoidal**, and **Trans-oral**.
  - Management is multimodal, aimed at histological diagnosis (biopsy), cytoreduction (debulking), and relief of complications like hydrocephalus.
- **Radiotherapy:**
  - **Conventional:** Adjuvant for residual disease.
  - **Neuroaxis Radiation:** Specifically indicated for tumors with high CSF seeding potential (**Medulloblastoma, Ependymoma**).
  - **Complication:** increase edema, demyelination, radio-necrosis, affect cognitive functions, may induce other kind of tumors as meningioma.
  - **Radiosurgery (SRS):** High-dose, precise delivery (e.g., Gamma Knife).
- **Chemotherapy:**
  - Limited by the BBB.
  - **Temozolomide:** Standard alkylating agent for Glioblastoma.
  - **PCV:** Procarbazine, Lomustine, Vincristine combination.
- **New Frontiers:** Immunotherapy (LAK cells), hyperthermia, and gene therapy.
- **Posterior Fossa Tumors:**
  - May need shunting or EVD prior to definitive surgery
  - Risk: possible peritoneal seeding, prolonged hospitalization, risk of shunt complications

7. The Gliomas

Gliomas comprise **52% of all brain tumors**. They are classified into four main types arising from neuroectoderm.

1- Astrocytoma -more common in males-

The most common primary brain tumor (45%). Peak incidence occurs between **40–60 years**.

- **Distribution:** Equally common in frontal, temporal, parietal lobes, and thalamus; less common in the occipital lobe.
- **Function in:**
  - Support neurons
  - Absorb neurotransmitter
  - Release neuroactive molecules
  - Aid in formation of BBB
- **Classification systems (WHO)**:
  - Grade 1: Pilocytic astrocytoma
  - Grade 2: Diffuse astrocytoma
  - Grade 3: Anaplastic astocytoma
  - Grade 4: Glioblastoma multiforme -more common in males-

- **Grading & Prognosis:**
    - **Low Grade (WHO I-II):** Avascular, well-differentiated, fibrous, and 15% calcified. Average survival: **8 years**. In adults usually in cerebral hemispheres, In children: in cerebellum
    - **High Grade (WHO III-IV):** WHO IV is **Glioblastoma (GBM)**. Highly vascular, necrotic, and may present as a **"Butterfly Glioma"** crossing the corpus callosum.
    - **Survival (High Grade):**
        - ✓ Surgery alone = **17 weeks**
        - ✓ Surgery + Adjuvant Radiation = **37 weeks**.
- **Clinical features:**
  1. Epilepsy
  2. Feature if increase ICP
  3. Focal neurological deficit.
- **Investigations:**
    - **CT (Low grade):**
        - ✓ Small hypodense mass
        - ✓ Little surrounding edema
        - ✓ No enhancement
        - ✓ Calcification may present
        - ✓ High grade
        - ✓ Large mass
        - ✓ Marked edema
        - ✓ Enhance in non-uniform manner.
    - **MRI, More sensitive than CT specially:**
        - ✓ Posterior fossa, brain stem and skull base tumor and for small tumor mass.
        - ✓ Usually both low and high appear decrease T1 signal increase T2 signal
    - **Angiograph**
    - **Skull X-RAY**
- **Spread:**
    - ➢ Systemic: rare
    - ➢ CSF seeding: 10 -25% of high grades
    - ➢ Tracing thru white matter
- **Management:**
    - **Surgical; aim is to:**
        - ✓ Take biopsy
        - ✓ Decrease tumor size
        - ✓ Reduce tumor mass prior to adjuvant therapy
    - **Radiotherapy as adjuvant therapy**
    - **Other therapy:** Chemotherapy, Immunotherapy, Hyperthermia

2- Oligodendroglioma

- **Peak Age:** 5th decade.
- **Features:** Supratentorial and well-demarcated.
- Most are well differentiated, 40 % are mixed glioma with astrocytoma or ependymoma
- **Clinical features** as astrocytoma
- Chicken wires appearance on histology
- **Pathognomonic: 90% show calcification**; 50% show contrast enhancement. (CT, MRI).

- **Treatment:** Aggressive resection followed by radiotherapy
- **Prognosis:** 5-year survival is 30 – 50%.

3- Ependymoma -more common in males-

- **Distribution:** 70% Infratentorial (children); 30% Supratentorial (adults).
- **Histology:** Papillary **(Rosette and pseudorosette** patterns). **Subependymoma** variant is often heavily calcified, Anaplastic.
- **Clinically:**
  - **Supratentorial:**
    - ✓ Presented with increased ICP
    - ✓ Focal neurological deficit
  - **Infratentorial:**
    - ✓ Increased ICP due to hydrocephalus
    - ✓ Ataxia due to cerebellum involvement
- **Investigation:**
  - **CT**
  - **MRI**
    - ✓ Tumor arises in ventricle and enhances
    - ✓ Calcification in 90% especially supratentorial
    - ✓ The most common posterior fossa tumor with calcification
  - **Spread by:**
    - ✓ Seeding through CSF
    - ✓ Systemic spread is rare
- **Treatment:**
  - **Surgical resection**
  - **Radiation of whole neuroaxis:** Second most radio sensitive tumor after medulloblastoma
  - **Prognosis:** 5 years survival 20 -50% Adults and supratentorial tumors have better prognosis

8. Specialized Tumors:

1- Vestibular Schwannoma (Acoustic Neuroma)

Benign posterior fossa tumor of CN VIII growing at **1-2 mm/year**.

- **Early Triad:** Progressive unilateral hearing loss (98%), tinnitus, disequilibrium.
- **Progression:** >2cm (CN V/VII compression—facial numbness/weakness); >4cm (Brainstem compression/ataxia).
- **Diagnosis:** MRI with gadolinium or **T2 FIESTA**. CT with contrast 2nd choice.
- **Implications for testing** family members of NF2 mutation carrier
- **Radiation:** Stereotactic Radiosurgery (Gamma Knife) SRS or XRT.
- **Surgery:** lesion>3cm, brain stem compression, edema, hydrocephalus.
- **Curable if** complete resection (almost always possible).

2- Chondromas: Are tumors arising from remnants of Notochord tissue

3- Medulloblastoma -more common in males-

The most common midline posterior fossa tumor in children (Peak age 5). Highly malignant (most common malignant brain tumor in children)

CT: Isodense midline lesion compressing 4th ventricle, with strong enhancement, **MRI**

Homer Wright Rosettes, appears blue on microscope

Treatment:

- ✓ Treat hydrocephalus (Drainage procedure)
- ✓ Surgery requires **whole neuroaxis radiation** due to aggressive CSF seeding and hematogenous spread (drop metastasis) to the spine. (One of the most **chemo-sensitive** and **radiosensitive** tumors of the CNS). You can't repeat surgery for residual mass.

4- Meningioma -more common in females-

The most common benign tumor (15%). Tumor arise from arachnoids layer of meninges, Occur at any age, peak in middle age

Metastasis is rare, The "atypical" variant shows increased mitotic activity or specific histological features that make it more likely to recur, earning it a Grade II status.

- **Histology:** Syncytial (meningio-thelio-matous), Transitional, Fibroblastic, and Angiomatous.
- **Clinical Syndromes by Site:**
  - **Parasagittal (M.C):** Epilepsy, contralateral lower limb paresis, urinary incontinence if bilateral, ICP in bilateral tumors.
  - **Sphenoid Ridge/Olfactory Groove:** Optic atrophy/anosmia; **Foster Kennedy Syndrome** (ipsilateral optic atrophy, contralateral papilledema, Olfactory groove).
  - **Suprasellar:** Bitemporal hemianopia without endocrine dysfunction.
- **Possible risk factors:** head trauma, Low levels of radiation, Nf2, Sex hormones are important (**hormone-sensitive**: progesterone & Estrogen receptors)
- **Investigations:** CT → Hyperdense, Enhance uniformly, Hyperostosis of cranial vault
  MRI → Isointense in T1.
- **Treatment:**
  - ✓ Total surgical excision
  - ✓ Radiation may be used to treat residual tumors
  - ✓ Risk of recurrence
  - ✓ Most common source tumor invaded dural sinus and not removed by surgery and in malignant variant
  - ✓ Has the best prognosis in general

5- Craniopharyngiomas

Benign (WHO Grade 1) but locally aggressive tumors that arise from remnants of **Rathke's pouch.** They are the most common suprasellar tumor in children.

Growth Hormone (GH) deficiency → short stature

Located in the suprasellar region, they compress the **optic chiasm** from above or below, causing **bitemporal hemianopia.,** Fits (Seizures), Headache, and Anosmia.

Contain a thick, brownish fluid rich in **cholesterol crystals**.

Calcification on a CT or MRI in a pediatric suprasellar mass is highly suggestive

6- Pituitary Adenoma

Arising from the anterior pituitary (20% incidence in autopsy studies). Associated with **MEN-1 syndrome**. Pituitary adenomas are more commonly diagnosed in younger adults, especially 20–40 years, particularly prolactinomas.

Prolactinoma is the most common secreting pituitary tumor

- **Classification:** Microadenoma (<1cm) vs. Macroadenoma (≥1cm).
- **Functional Types:** Prolactinoma (amenorrhea/libido loss), GH (Acromegaly), ACTH (Cushing's).
- **Differential diagnosis:** parasellar tumours (e.g. craniopharyngioma, tuberculum sellae meningioma), carotid aneurysm
- **Clinical Features:**
  - ✓ Mass effects
  - ✓ H/A
  - ✓ Bitemporal hemianopsia (compression of optic chiasm); hydrocephalus (3rd ventricle compression)
  - ✓ Invasive adenomas: CNIII,IV,V1,V2,VI palsy (cavernous sinus compression); proptosis and chemosis. (Ophthalmoplegia (paralysis of eye movements) rather than causing a visual field deficit)
  - ✓ Cavernous sinus occlusion.
- **Endocrine effects:**
  - ✓ **Hyperprolactinemia (prolactinoma):** infertility, amenorrhea, galactorrhea, decreased libido. ACTH production: Cushing's disease, hyperpigmentation
  - ✓ **GH production:** acromegaly/gigantism
  - ✓ **Panhypopituitarism:** due to compression of pituitary (hypothyroidism, hypoadrenalism, hypogonadism)
  - ✓ **DI** – rare, except in apoplexy
- **Pituitary Apoplexy:** Emergency necrosis/hemorrhage. Presents with abrupt headache, visual loss, and panhypopituitarism.
- **Cushing's syndrome can be due to:** Adrenal, Pituitary, and Lung tumors.
- **Investigations:**
  - Formal visual fields, CN testing.
  - Endocrine tests (prolactin level, TSH, 8 AM cortisol, fasting glucose, FSH/LH, IGF-1).
  - Electrolytes, urine electrolytes, and osmolarity.
  - Imaging (MRI with and without contrast)
- **Treatment:**
  - **Medical:**
    - ✓ Apoplexy: rapid corticosteroid administration ± surgical decompression
    - ✓ Prolactinoma: dopamine agonists (e.g. bromocriptine)
    - ✓ Cushing's: serotonin antagonist (cyproheptadine), inhibition of cortisol production (ketoconazole) -can be due to: Adrenal tumor, Pituitary tumor, Lung tumor, Breast carcinoma)
    - ✓ Acromegaly: somatostatin analogue (octreotide) ± bromocriptine ■ endocrine replacement therapy
  - **Surgical (endoscopic):**
    - ✓ Trans-sphenoidal (M.C)
    - ✓ Trans-ethmoidal
    - ✓ Transcranial - less commonly- (for significant suprasellar extension)

7- Hemangioblastoma:

Is a benign brain tumor

The most common posterior fossa tumor in adults

Not neuroepithelial origin

Child comes with vomiting and nausea of 3 months duration and has cerebellar signs and otitis media 2 weeks ago. What is the differential diagnosis & What is the next step?

Answer: Abscess Cerebellar tumor, CT scan

Absolute contraindication for a lumbar puncture? Brain space occupying lesion

Surgery is usually not the primary treatment option in:

Microadenoma with acromegaly

Nonfunctioning pituitary adenoma

Invasive pituitary adenoma

Microadenoma with Cushing

Intracranial Pressure (ICP)

1. Introduction

The management of intracranial pressure (ICP) is a strategic race against secondary ischemic injury.

Because the adult cranium is a rigid, non-expanding vault, any pathological volume increase (be it hemorrhage, tumor, or edema) threatens the very perfusion required to keep cerebral tissue viable.

High-yield principles:

- **The Monro-Kellie Doctrine:** The physical law governing the "rigid box" model, where total intracranial volume is constant (V_{brain} + V_{blood} + V_{CSF} = K constant).
- **Cerebral Perfusion Pressure (CPP):** The primary driver of cerebral blood flow, calculated as CPP = MAP – ICP, (MAP = (SBP + (2 x DBP))/3)
- **Intracranial Compliance:** The dynamic relationship between volume changes and pressure spikes.
- **Stepwise Tiered Management:** A mandatory 4-tier escalation system (Tier 0 to Tier 3) designed to stabilize and rescue the injured brain.

2. Monro-Kellie Doctrine

The foundation of neurocritical care is the "rigid box" model. Unlike other anatomical compartments that can expand to accommodate swelling, the adult skull is unyielding.

This rigidity serves to protect the brain but also creates a system where minor volume expansions can cause explosive rises in pressure, leading to global ischemia or mechanical displacement (herniation).

The Monro-Kellie Doctrine

This doctrine states that the sum of volumes of the three intracranial components is constant. Any increase in the volume of one must be offset by an equal decrease in another, or the pressure will skyrocket. (V_{brain} + V_{blood} + V_{CSF} = K)

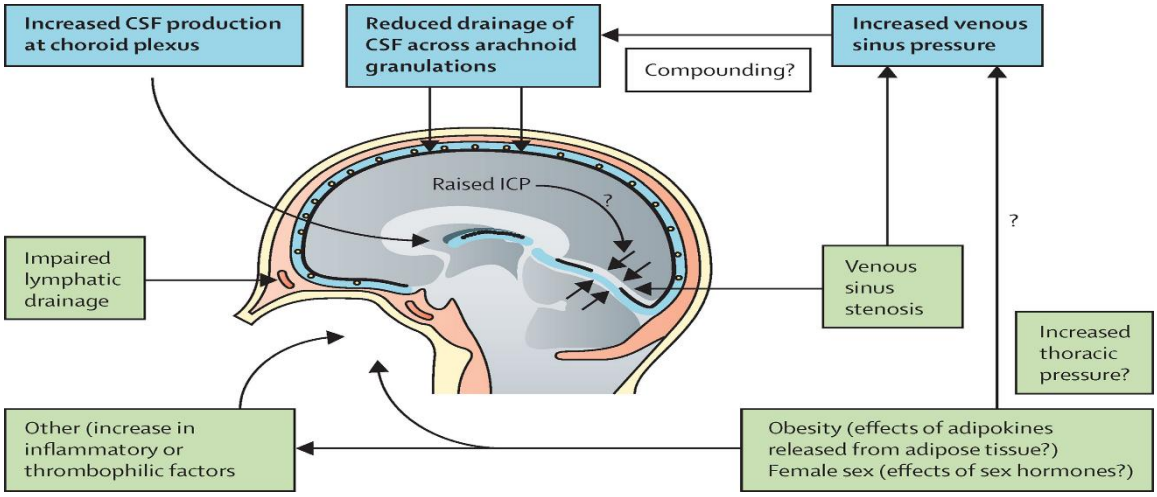
Component	Characteristics	Impact of Volume Expansion
Brain Tissue	Semi-solid, non-compressible.	Primary mass (~80\% volume). Increases via tumors, abscesses, or edema.
Blood	Arterial and venous systems.	Venous blood is the first to be displaced to maintain equilibrium.
CSF	Fluid in ventricles/subarachnoid space.	Displaced into the spinal canal; serves as the primary early buffer.

The "Vicious Cycle" of Rising ICP

When pathological volume is added, the body attempts a compensatory response that often accelerates the injury:

1. **Compression of Low-Pressure Systems:** Rising ICP first collapses thin-walled veins, decreasing venous outflow and causing congestion.
2. **Compensatory Vasodilation:** In an attempt to maintain blood flow, the body retains CO2, which dilates cerebral vessels. This actually increases intracranial blood volume, further raising ICP.

3. **CPP Compromise:** As ICP rises, the pressure gradient for blood flow (CPP = MAP - ICP) narrows.
4. **Ischemia and Swelling:** Reduced flow leads to cerebral ischemia, which triggers cytotoxic edema (swelling), adding more volume to the system and repeating the cycle.



Cerebral Perfusion Pressure (CPP) Relationship

Systemic blood pressure (MAP) and intracranial pressure (ICP) work in opposition to dictate brain health.

- **Rising ICP:** Directly reduces CPP if MAP is static.
- **Falling MAP:** Hypotension (from shock or sedation) reduces CPP even if ICP is normal.
- **The Clinical Goal:** We aim to maintain ICP < 20 mmHg and CPP > 65 mmHg to ensure neuronal survival.

These physical constraints dictate the brain's "compliance," a concept that explains why some lesions are silent while others are deadly.

3. Intracranial Compliance and Pathophysiological Phases

Compliance is the brain's ability to maintain stable pressure despite volume changes. In clinical practice, the *rate* of volume change is as critical as the volume itself; the brain can accommodate a slow-growing meningioma far better than an acute epidural hematoma.

Phase	ICP Status	Clinical State / Symptoms
Phase 1: High Compliance	Normal	Asymptomatic
Phase 2: Decreasing Compliance	Rising	Warning Zone: Headache, nausea, mild confusion
Phase 3: Minimal Compliance	Very High	**Emergency:** Herniation, coma, microvascular collapse

Phase 1: Compensation

- **Displacement Mechanisms:** Volume is added, but ICP remains near normal because CSF is shunted to the spinal canal and venous blood is pushed out.
- **Clinical Pearl:** This is why large tumors may be discovered incidentally without symptoms.

Phase 2: Warning Zone (The Danger Zone)

- **Exhausted Reserve:** Compensatory mechanisms are nearly spent. Small increases in volume now cause disproportionately larger rises in ICP.
- **Early Symptoms:** The patient becomes symptomatic (headache, vomiting). This state is critical because it remains reversible if the underlying cause is addressed.

Phase 3: Decompensation

- **System Failure:** The compliance curve becomes vertical; a tiny volume change leads to a massive pressure spike, Loss of consciousness is a late sign of increased ICP (after all compensatory mechanisms have been exhausted)
- **Fatal Progression:** This phase leads to the collapse of microvasculature as ICP approaches MAP, resulting in global ischemia and brain herniation.

4. ICP Measurement and Monitoring Methodologies

Invasive Monitoring Methods

Method	Location	Accuracy	CSF Drainage	Infection Risk
Intraventricular	Lateral Ventricle	⭐⭐⭐⭐	✅ Yes	Highest
Intraparenchymal	Brain Tissue	⭐⭐⭐	❌ No	Low (ICU)
Subdural	Subdural Space	⭐⭐	❌ No	Low-Moderate
Epidural	Between Skull/Dura	⭐	❌ No	Lowest

The "Gold Standard": Intraventricular Catheter (EVD):

Advantages: Provides the most accurate measurement, allows for therapeutic CSF drainage to lower ICP, and permits CSF sampling.

Disadvantages: Highest risk of ventriculitis and hemorrhage; placement is difficult if ventricles are collapsed or shifted.

The Lumbar Puncture (LP) Warning: While an LP can measure pressure, it is **strictly contraindicated** in the presence of an "expanding lesion" (mass effect) due to the high risk of precipitating herniation.

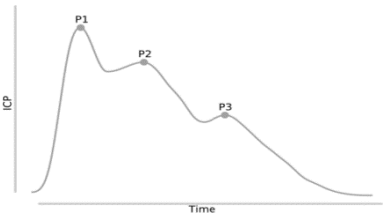
However, numbers alone do not tell the whole story; we must analyze the "pulse" of the brain through its waveform.

5. Waveform Analysis and Normal Values (herniation long before)

Age Group	Normal Range (mmHg)
Adults	< 10–15
Children	3–7
Term Infants	1.5–6

The ICP Pulse Wave (P1, P2, P3)

A single ICP pulse comprises three distinct peaks:



1. **P1 (Percussion Wave):** Originates from arterial pulsations of the choroid plexus. Normally the highest peak.
2. **P2 (Tidal Wave):** Reflects intracranial compliance. **This is the most important clinical wave.**
3. **P3 (Dicrotic Wave):** Originates from aortic valve closure and venous pulsations.

Trend Waves (Lundberg Waves)

- **A Waves (Plateau):** Sudden, massive rises to 50 mmHg lasting 5–20 minutes. This is a neurosurgical emergency indicating near-total loss of compliance.
- **B Waves:** Rhythmic spikes (every 30–120 seconds) suggesting cerebral vasospasm or reduced compliance.
- **C Waves:** Small, frequent oscillations related to normal autonomic regulation.

6. Clinical Manifestations

X-Ray: "Thumb impression" "Beaten copper" or "beaten silver" appearance, erosion of the posterior clinoid processes, and widening (diastasis) of skull sutures, Enlargement of sella turcica, Erosion of dorsal Sella, Bone vascular invagination

CT/MRI: Midline shift, compressed ventricles, and effacement of the cortical sulci.

Lumbar puncture cannot be used to measure the ICP if the brain CT scan is abnormal

Trans-tentorial herniation at level of midbrain will affect: Pupillary size, Motor system Eye movement, Level of consciousness.

The presentation is classic for raised (ICP): Morning headache (due to CO_2 retention → vasodilation overnight), Vomiting (projectile), Occipital headache → **papilledema**

M.C location for spontaneous intracerebral hemorrhage 2^{ry} to HTN is: Basal ganglia

Cushing's triad: Bradycardia, bradypnea, and hypertension

Note: The uncus is the medial part of the temporal lobe

Complications are: infections and hemorrhage (in that order).

7. Management Tiers for Increased ICP

Management follows the "Escalation Principle." Treatment must proceed stepwise; we do not jump to Tier 3 unless lower-tier interventions have failed.

Tier	Purpose	Strategies
0	**Stabilization**	ABCs, head elevation (>30 degrees), midline positioning, **cervical-collar fit check**, normothermia (<38 degrees C), pain control, **steroids for tumor-induced vasogenic edema**, avoid hyponatremia.
1	**Reduction**	Hyperosmolar therapy (Mannitol), **PaCO_2 goal 35–38 mmHg**, EVD drainage, decompressive craniotomy, increased analgesia/sedation.

2	**Metabolism**	Heavy sedation (Propofol/Midazolam), **Optimize Serum Sodium Goal**, Hypocapnia (**PaCO_2 30–35 mmHg**), **Neuromuscular Blocking Agent Challenge**.
3	**Rescue**	**High-dose Barbiturates** (Pentobarbital to target EEG burst suppression), **moderate hypothermia** (32–34 degrees C).

Exam Tip: Always escalate sequentially. **Tier 3 (Barbiturate coma/Hypothermia) is a rescue therapy for refractory ICP only.**

Conclusion: Successful neurosurgical management requires maintaining ICP below 20 mmHg and CPP above 65 mmHg.

By adhering to the tiered escalation system and monitoring both absolute values and waveform morphology, we protect the brain from the irreversible consequences of secondary injury.

8. Idiopathic Intracranial Hypertension (IIH)

IIH is a diagnosis of exclusion characterized by high ICP in the absence of a mass or hydrocephalus, most common in obese females of childbearing age.

Diagnostic Criteria for IIH (Dandy criteria)

Criteria	Description
1. Clinical Signs	Papilledema (striking feature).
2. Neuro Exam	Normal except for cranial nerve abnormalities (specifically **6th nerve/abducens palsy**).
3. Imaging	Normal CT/MRI (no mass/hydrocephalus); MRV used to exclude venous sinus thrombosis.
4. CSF Composition	Normal constituents (no infection or hemorrhage).
5. CSF Pressure	Elevated opening pressure on Lumbar Puncture.
Note: If papilledema is absent, diagnosis requires criteria 2-5 plus unilateral or bilateral abducens palsy.	

Associated Risk Factors: OCPs, Tetracycline, Vitamin A, and Nalidixic acid.

Clinical Striking Features: Papilledema and "amaurosis fugax" (transient visual loss). Chronic cases lead to scotomas and optic atrophy.

Optic Nerve Fenestration: A surgical procedure where small "windows" are cut into the sheath of the optic nerve to relieve pressure and protect the patient's vision.

Therapeutic Lumbar Puncture often provides dramatic relief.

Safety Rule: During therapeutic LP, the ICP must be reduced to **half of the initial reading or to the normal level, whichever is higher**.

Management:

1. Lifestyle Modifications: Weight Loss:

2. Medical Management: reduce the production of (CSF) or manage associated symptoms:

- ✓ **Acetazolamide:** The first-line medical treatment. It is a carbonic anhydrase inhibitor that decreases CSF production.
- ✓ **Topiramate:** carbonic anhydrase inhibitory effect and its benefits in treating associated migraine-like headaches; it also aids in weight loss.
- ✓ **Furosemide**: A loop diuretic sometimes used as an adjunct to acetazolamide to further reduce fluid volume.
- ✓ Intravenous Methylprednisolone: Typically reserved for acute, rapidly progressive visual loss (fulminant IIH) to bridge the patient to urgent surgery.

3. Interventions & Surgical Procedures

- ✓ Repeated Lumbar Punctures
- ✓ Cerebral Transverse Sinus Stenting: evidence of venous sinus stenosis (narrowing).
- ✓ Shunts: Permanent drainage systems to divert CSF:
  - ➢ Ventriculoperitoneal (VP) Shunt: Diverts fluid from the brain ventricles to the abdomen.
  - ➢ Lumboperitoneal (LP) Shunt: Diverts fluid from the lumbar spine to the abdomen.
  - ➢ Lumbar Drain: Usually a temporary external drain.

Subarachnoid Hemorrhage (SAH)

1. Introduction

Subarachnoid Hemorrhage (SAH) is a neurosurgical emergency, characterized by the extravasation of blood into the subarachnoid space, the anatomical compartment between the pia mater and the arachnoid membrane.

While the **etiology** varies, the rupture of saccular (berry) aneurysms is the primary driver, accounting for 77% of spontaneous cases.

The clinical urgency of SAH cannot be overstated; the first 24 hours represent a critical window where mortality and the risk of catastrophic rebleeding are at their zenith.

CAUSES: Traumatic, Spontaneous/Nontraumatic, Unidentified cause 15%.

Spontaneous/Nontraumatic: Arteriovenous Malformations (AVMs) represent the second most identifiable cause of spontaneous SAH.

Saccular aneurysm origin of SAH is worse than AVM

Cerebral aneurysm of grade 1 after 3 months, incidence of death is **2–5%**.

2. Epidemiology of SAH

Demographic Factor	Epidemiological Data and Statistics
Age	Incidence peaks at age 50; 80% of cases occur between ages 40–65. Rare in children <10 years (0.5% of cases).
Sex	Predominantly female (3:2 ratio). Risk is significantly elevated during the third trimester of pregnancy.
Race	Higher incidence in Black populations compared to White populations.
Geography/Stats	**US:** 6–16 cases per 100,000 (approx. 30,000 cases/year). **Worldwide:** 2–49 per 100,000 (Highest in Japan and Finland).

A female patient in her fifth decade presenting with a sudden-onset headache must be treated as a ruptured aneurysm until proven otherwise.

3. Natural History and Mortality Progression

- **Pre-hospitalization:** 15% of patients expire before reaching medical care.
- **Within 24 Hours:** 25% mortality rate, with or without medical intervention.
- **At 1 Week:** Mortality reaches approximately 40%.
- **At 6 Months:** 50% of all patients have died.
- **Survivor Morbidity:** 40% of survivors suffer major neurologic deficits; most experience transient or permanent cognitive impairment.

4. Etiology and Pathogenesis of Intracranial Aneurysms

The arterial wall's integrity relies on the media layer, which contains the smooth muscle and is bounded by the internal and external elastic membranes. The primary pathological step in saccular formation is the failure of the **internal elastic membrane**. When this structural layer fails, the vessel cannot resist luminal pressure, leading to dilation. This failure typically manifests in high-stress regions where the vessel is most vulnerable.

Factor Category	Pathogenesis and Statistics
Congenital	Muscle and elastic tissue defects (Marfan) in the arterial media of the Circle of Willis (found in 80% of autopsy vessels).
Micro-dilation	These defects lead to microaneurysms (<2 mm) in 20% of the population.
Clinical Aneurysm	Dilations >5 mm occur in 5% of the population.
Acquired	Atherosclerosis, Hypertension, and Hemodynamic stress.

5. Classification and Localization of Aneurysms

Aneurysms are categorized by morphology and cause, each appearing distinct on imaging:

1. **Saccular (Berry) -M.C-** -90% in anterior circulation & Mostly at bifurcation of arteries in the base of the brain-
2. **Fusiform**
3. **Mycotic:** infectious process
4. **Atherosclerotic**
5. **Traumatic**

The high incidence at the AComA (40%) and MCA (34%) bifurcations is due to the intense **hemodynamic stress** at these junctions. Anatomical location serves as the primary predictor of clinical impact and the surgical approach required once external triggers cause the vessel to fail.

6. Rupture: Risk Factors and Clinical Presentation

Rupture is the transition from a latent vascular defect to a life-threatening emergency, often triggered by hemodynamic instability.

Rupture Risk Factors:

- Hypertension and Atherosclerosis
- Smoking and Oral Contraceptive Pills (OCPs)
- Vigorous exercise and Hemodynamic stress
- Pregnancy

Clinical Presentation:

- **Headache:** **The classic "worst headache of my life."**
- **Altered Consciousness:** From drowsiness to profound coma.
- **Meningism:** Neck rigidity, vomiting, photophobia, and fever + Kernig's & Brudzinski's. This complex arises because blood in the subarachnoid space acts as a **chemical irritant**, causing "chemical meningitis."
- **Seizures:** Most common with MCA aneurysm ruptures.
- **Focal Signs:** **Third Cranial Nerve Palsy (PCoA)** (complete eyelid ptosis, pupil dilation, and outward eye deviation) is a critical sign of focal pressure from an aneurysm.

Meningism and **oculomotor nerve palsy** are **red flags** indicating the hemorrhage has already breached the vessel wall, necessitating immediate diagnostic confirmation.

7. Diagnostic Modalities and Severity Grading

Diagnosis:

A. Clinical suspicion

↓

B. Non-contrasted CT (showing bright white areas of blood).

↓

C. If CT is negative but suspicion remains, a **lumbar puncture** is performed to check for **Xanthochromic CSF**.

Hunt and Hess Scale (Clinical Acuity)

Grade	Description
1	Asymptomatic or minimal headache and slight nuchal rigidity.
2	Moderate/severe headache, nuchal rigidity, no neuro deficit (except CN palsy).
3	Drowsiness, confusion, or mild focal deficit.
4	Stupor, moderate/severe hemiparesis, early decerebrate rigidity, and **vegetative disturbances**.
5	Deep coma, decerebrate rigidity, moribund.

Grade 1 patient has a 70% survival rate, which drops to 10% for Grade 5.

WFNS Scale (GCS Based)

Grade	Glasgow Coma Score (GCS)	Motor Deficit
1	15	No deficit (except CN palsy)
2	14–13	No deficit
3	14–13	Any deficit
4	12–7	With or without focal neurodeficit
5	6–3 (Managed in the ICU)	Coma with or without abnormal posturing

Fisher CT Grading Scale

Group	Blood Pattern on Non-enhanced CT	DCI/Vasospasm Risk
1	No subarachnoid blood detected.	21%
2	Diffuse or vertical layers <1 mm thick.	25%
3	Localized clot or vertical layers ≥1 mm thick.	31%
4	Intracerebral or intraventricular clot with diffuse or no SAH.	37%

8. Management and Treatment Strategies

Management focuses on stabilization and exclusion of the bleed source.

Conservative management is recommended in stage 1

- **Stabilization:** ICU admission, dark room (to counteract photophobia), and head elevation of 30 degrees.
- **Supportive Care:** Administration of **Codeine phosphate** for headache, laxatives to prevent straining, anxiolytics, and IV normal saline, Foley's catheter should be inserted.
- **Finding the cause:**
  - **(DSA) is the gold standard,** CT Angiography, MRA.
  - **MRI:** has high sensitivity for aneurysms ≥5 mm, unruptured cerebral aneurysms, AVMs that are not detected by cerebral angiography or spinal AVMs causing SAH
- **Intervention: (preventing rebleeding)**
  - **Clipping:** Surgical placement of a metal clip across the aneurysm neck.
  - **Coiling:** Endovascular packing of the aneurysm with wire coils to induce clotting.

The primary goal is preventing secondary insults and complications, which represent the most critical post-rupture period.

9. Complications and Special Considerations

Complication	Timing and Impact
Rebleeding	Highest risk in first 24h (4.1%); 19% at 2 weeks. **Mortality is 78%.** Partly occurs due to fibrinolytic activity in CSF (give Antifibrinolytics)
Vasospasm	Occurs 4–14 days post-hemorrhage (treated with nimodipine) Percentage of radiographic evidence: 60% Considered a late complication (after 72 hours)
Hydrocephalus	Acute (20%) within 24h; Chronic (10–15%) develops later.
Hyponatremia	10–34% occurrence (linked to SIADH or ANF).
Seizures	25% of cases; most frequent in MCA ruptures.
Cardiac/Pulm	90% arrhythmias; pulmonary edema is universal in severe SAH.

Multiple Aneurysms: Present in 15-20% of cases, creating a diagnostic dilemma regarding which vessel ruptured and how to manage unruptured lesions. **Rebleeding and vasospasm remain the most critical hurdles for survival.**

10. Arteriovenous Malformations (AVM)

AVMs are congenital anomalies where the capillary bed is missing, allowing blood to pass directly from artery to vein. This causes **"arterialized" veins** (veins subjected to high arterial pressure) and **"steal syndrome"** where surrounding brain tissue is deprived of oxygenated blood.

AVMs tend to **occur in** cortical/subcortical regions, which are **predominantly** supplied by **MCA**

AVM Epidemiology:

- Occur in 4-5% of the population, but only **10-15% are symptomatic**.
- Less common than aneurysms (1:5 ratio).
- Typically present in younger patients; 10% mortality rate.
- The annual risk of bleeding is 1–2%. Secondary aneurysms develop in 5–10% of AVM cases.

Modes of Presentation:

1. **Hemorrhage - ICH or SAH- (50%):** Usually from smaller lesions. (CT then angiograph)
2. **Seizures (45%):** Usually from larger lesions.
3. **Headache (30%):** Recurrent.
4. **Neurological Deficits (20%):** Ischemia (shunting or steal syndrome) or pressure.

   2+3+4 = Diagnosis by MRI and angiography

Classification:

The **Spetzler-Martin Grading System** assesses surgical risk based on three characteristics.

Characteristic	Points Assigned
Nidus Size	<3 cm (1 pt) small 3–6 cm (2 pts) medium >6 cm (3 pts) large
Eloquence of Brain	Non-eloquent (0 pts) Eloquent (1 pt)
Venous Drainage	Superficial only (0 pts) Deep veins (1 pt)

The total points determine the grade (1–5). A **Grade 6** lesion is considered inoperable.

Treatment:

- **Surgical Excision:** Physical removal of the AVM.
- **Embolization:** Blocking abnormal vessels via endovascular materials.
- **Radiotherapy:** Using Gamma Knife technology.

The Spetzler-Martin scale is the definitive tool for management; size and eloquence determine whether a lesion is a candidate for safe excision or if the risks to critical brain tissue necessitate embolization or radiotherapy.

Hydrocephalus

1. Introduction

Hydrocephalus is the pathological accumulation of CSF within the ventricular space at an inappropriate pressure. Volume alone is insufficient for diagnosis; it is the pressure-driven impact on cerebral tissue that dictates the urgency of treatment.

Key Epidemiological Data:

- **Estimated Prevalence:** ~1–1.5% (Note: Some literature erroneously suggests higher ranges due to reporting artifacts; clinical consensus remains in this narrower band).
- **Congenital Incidence:** 0.9–1.8 per 1,000 live births.

2. Physiology and Dynamics of Cerebrospinal Fluid (CSF)

Production Mechanism

CSF is actively produced within the **choroid plexus**, where a single layer of cuboidal epithelial cells filters plasma through a vascularized core. (**interstitial fluid** and the **ependymal lining**)

This process is metabolically driven by **sodium-potassium ATPase** and **carbonic anhydrase**.

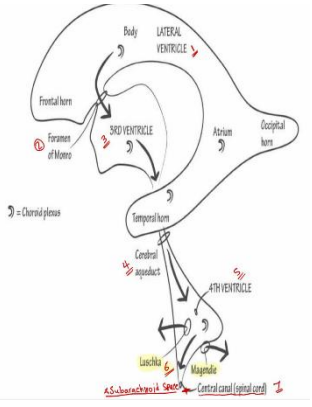
The production rate is constant at **0.30–0.35 mL/min** (~500 mL/day), regardless of intracranial pressure. (active secretion and filtration)

Clinical Pearl: CSF is not static. It turns over **3–4 times per day**, a critical fact when calculating the clearance of infections or metabolic byproducts.

Characteristic	Clinical Values/Description
Appearance	Clear and colorless
Total Adult Volume	150 mL (25 mL in ventricles; 125 mL in subarachnoid space)
Neonatal Volume	40–50 mL
Childhood Volume	65–140 mL
Cellularity	Acellular (< 5 lymphocytes; > 5 is termed Pleocytosis)
Protein Levels	15–40 mg/dL (Site/age dependent) Less of the plasma
Sugar Concentration	>40 or 2/3 of the plasma sugar concentration
Electrolytes	Similar Na+, higher Cl-(salty taste), lower K+ compared to plasma

The Circulation Pathway

1. **Lateral Ventricles** (Frontal, Temporal, Occipital horns; Atrium; Body)
2. **Foramina of Monro** (Interventricular foramina)
3. **Third Ventricle**
4. **Cerebral Aqueduct** (Sylvian Aqueduct)
5. **Fourth Ventricle**
6. **Foramina of Luschka and Magendie**
7. **Subarachnoid Space** and **Central Canal of the spinal cord**



Absorption Mechanisms

CSF returns to the venous system through **arachnoid villi**—diverticula of the arachnoid that invaginate into the **superior sagittal sinus**. Large clusters of arachnoid villi, known as **arachnoid granulations**, become grossly visible with age.

Normal ICP Ranges by Age

Age Group	Normal ICP Range (mmHg)
Neonate	< 2
Infant	1.5–6
Young Child	3–7
Adolescent (> 15 years)	< 15
Adult	< 15

3. Pathophysiology and Intracranial Pressure (ICP)

Hydrocephalus arises from a triad of mechanisms:

1. Increased production (rarely seen outside of choroid plexus tumors),
2. Decreased absorption,
3. **Obstruction to flow (the most common).**

Tissue Impact and Interstitial Edema

When pressure rises, the brain's compliance is eventually overwhelmed. Progressive ventricular dilatation leads to the physical separation of **ependymal cells**. This breach allows CSF to enter the white matter via **bulk flow**, creating **interstitial cerebral edema**. This is a physiological tipping point where pressure becomes a direct tissue-injury state.

This explains:

- Periventricular T2 hyperintensity on MRI
- Gait and motor symptoms in chronic hydrocephalus

4. Clinical Classifications of Hydrocephalus

Classification	Definition / Clinical Dichotomy
Communicating vs. non-communicating	Bacterial meningitis Choroid plexus papilloma
Compensated vs. non-compensated	Based on the physiological success of compensatory mechanisms (symptomatology).
Acute vs. Chronic	Speed of progression and onset of symptoms.
Congenital vs. Acquired	Developmental origin vs. secondary to trauma, hemorrhage, or tumor.
Internal vs. External	Fluid localization relative to brain parenchyma.

Classification dictates the surgical trajectory: bypass, diversion, or resection.

5. Etiology and Site-Specific Obstructions

Anatomical site of obstruction is the primary determinant of the radiological phenotype and surgical approach.

Site	Associated Pathologies and Clinical Pearls
Lateral Ventricles	**Choroid plexus tumors:** Rare (0.4–0.6% of CNS tumors) but cause production rates 3–4x normal. Endoscopic coagulation of the choroid plexus or *surgical removal of the papilloma.*
Foramina of Monro	Atresia, congenital membranes, or gliosis post-hemorrhage /ventriculitis.
Third Ventricle	**1- Colloid cysts** (superior/anterior location); **2- Ependymal/Arachnoid cysts. → Bobble-head doll syndrome:** Rhythmic head nodding (2-3 times/sec). → The endoscopic fenestration is a treatment option. **3-** Craniopharyngiomas & Chiasmal-hypothalamic gliomas. (M.C pediatric neoplasms that obstruct the third ventricle) **pretectal area** and the **superior colliculi** are located in the dorsal midbrain (the tectum) responsible for coordinating vertical eye movements, particularly upward gaze.
Sylvian Aqueduct	**Congenital aqueductal stenosis:** 12–13 mm long and only **0.2–0.5 mm** wide in neonates. (true stenosis, forking, septum, or subependymal gliosis) The most common cause of hydrocephalus in children **Bickers-Adams-Edwards syndrome:** X-linked recessive. **Pathognomonic sign:** "Cortical thumbs" (flexion-adduction). **Secondary:** in utero infections (e.g. toxoplasmosis), intraventricular hemorrhage, or mumps encephalitis
Pineal Region	**Germinomas**; highly radiosensitive and common causes of obstructive hydrocephalus. **Low-grade astrocytomas:** the most common periaqueductal pediatric neoplasms that cause hydrocephalus.
Fourth Ventricle	**In infants: Dandy-Walker cysts:** >85% incidence of hydrocephalus. (Can be associated with polydactyly) **Core anatomical triad:** 1. Agenesis or hypoplasia of the cerebellar vermis 2. Cystic dilatation of the 4th ventricle 3. Enlarged posterior fossa (often with elevated tentorium) **In older children: Tumors:** Medulloblastoma (85%), Posterior fossa Astrocytoma (65%), Ependymoma (75%), brainstem gliomas (25%)
Basilar/Arachnoid	**Absorptive obstruction:** Post-meningitic/post-hemorrhagic sclerosis; **Meningeal carcinomatosis.**

6. Etiology and Clinical Presentation by Age Group

Etiology by Age

Age Group	Common Causes
Premature Infants	**Intraventricular Hemorrhage (IVH)** (Grades 1–4) in the germinal matrix.
Full-term Infants	Aqueductal stenosis, Chiari II, Dandy-Walker, Encephaloceles, Vein of Galen malformations.
Older Children	Trauma, meningitis, neoplasms (secondary).
Adults	30% Idiopathic **others:** SAH, head injury, tumors, cranial surgery.

Signs and Symptoms

Patient Category	Clinical Presentation
Premature Infants	Apnea, bradycardia, tense fontanelle, globoid head shape.
Infants	**"Setting-sun" eyes**, Macewen's sign (cracked-pot sound), frontal bossing, poor head control. Dilated scalp veins Suture widening
Toddlers/Older	Headache, lethargy, **papilledema**, lateral rectus (abducent) palsy, hyperreflexia. (Fundoscopic examination showing advanced optic disc swelling)

Head Circumference Norms

- **Birth Norm:** 33–36 cm.
- **0–3 Months:** +2 cm/month | **4–6 Months:** +1 cm/month | **7–12 Months:** +0.5 cm/month.
- **Abnormal:** Any growth above the **95th percentile**.

7. Diagnostic Imaging and Studies (limiting radiation while maximizing resolution)

Modality	Clinical Utility and Historical Context
Skull X-ray	Primarily "shunt surveys" for hardware integrity or disconnection.
Cranial Ultrasound	Essential for premature infants; ideal for transfontanelle bedside monitoring.
CT Scanning	**Historical Gold Standard (1976–1986).** Shows periventricular fluids/edema but limited to axial planes and involves radiation & have less resolution than MRI.
Brain MRI	**Current Gold Standard (Since 1986).** Detects transependymal resorption and identifies low-grade gliomas with superior resolution. Determine CSF flow across the aqueduct.

8. Management: Non-Surgical and Surgical Interventions

Non-Surgical Options

- **Pharmacological:** Acetazolamide and Furosemide (temporary reduction of production).
- **Procedural:** Serial Lumbar Punctures or ultrasound-guided ventricular taps.
- **Archaic/Historical:** Head wrapping and radioactive gold (rarely, if ever, utilized in modern practice).

Surgical Interventions

1. **Lesion Resection:** The definitive treatment for obstructive masses.
2. **Endoscopic Third Ventriculostomy (ETV):** Bypasses obstructions at or distal to the aqueduct. (A small perforation is made in the tuber cinereum (the thin floor of the third ventricle) This opening connects the third ventricle directly to the **interpeduncular cistern**
   - **Success Rate:** 75% in pediatric aqueductal stenosis (Kamikawa et al.).
3. **Choroid Plexus Coagulation (CPC):** Often adjunct to ETV.
4. **CSF Shunts:** Silastic diversion to the **peritoneal cavity** (m.c), **atrium**, or **pleural space**.(where normal physiologic processes can absorb the fluid)
5. **Ventriculo-subarachnoid shunt:** Also known as a Torkildsen shunt.

9. Shunt Complications and Failure Analysis

Failure Mode	Description / Site-Specific Uncommon Complications
1- Mechanical	Fracture, migration, or hardware disconnection.
2- Functional	**Malfunction/Overdrainage:** Leads to subdural hematoma or hygroma.
3- Cranial	Subdural hygroma/hematoma, intracerebral hematoma at insertion site.
4- Subcutaneous	Shunt migration, disconnection, or fracture.
5- Peritoneal	**Peritonitis, pseudocysts**, perforation, or hernias.
6- Atrial	**Endocarditis, Nephritis.**

1+2+7=common complications, 3+4+5+6= uncommon complications

7- Shunt Infection Protocol

Infection occurs in 1–15% of cases, primarily introduced at the time of surgery.

Premature infants have an increased risk. • Evident within 1 month of placement. Nearly 90% of all shunt infections are recognized within 12 month

- **Pathogens:** **S. epidermidis (60%)**, **S. aureus (30%)**.

  Remainder (10%) caused by coliforms, propionibacteria, streptococci, or **H. influenzae**.
- **Diagnosis**: is confirmed by CSF sampling from the shunt reservoir; with the findings of leukocytosis and positive culture.
- **Treatment:** Hardware removal + EVD placement + IV antibiotics.
- **Re-implantation Criteria:** 3 consecutive negative cultures, WBC < 50, Protein < 500 mg/dL.

Shunt obstruction:

Intracerebral Hematoma around the catheter

Swelling around the valve of the shunt highly suggestive of shunt malfunction

• Often presents with signs and symptoms of increased ICP:

- ✓ Headache,
- ✓ Irritability,
- ✓ Lethargy,
- ✓ Nausea, and/or vomiting.

• The shunt:

- ➢ It itself can be examined for evidence of obstruction..
- ➢ Head CT
- ➢ Shunt survey X-rays

• Treated by:

- ✓ Replacing the occluded or nonfunctioning components
- ✓ Replacing the entire system.

10. Normal Pressure Hydrocephalus (NPH)

NPH is a chronic adult condition often presenting insidiously with a normal ICP profile on spot checks, despite pathological dynamics.

The Hakim-Adams Triad:

- **Motor Dysfunction:** "Magnet gait" (difficulty initiating gait, feet appear stuck to floor).
- **Urinary Incontinence:** Early on, the patient is aware of the urge (uninhibited bladder) but cannot reach the bathroom due to the gait deficit.
- **Dementia:** Progressive cognitive decline.

Advanced Clinical Indicators:

- **Frontal Release Signs:** Positive suck and grasp reflexes.
- **Dyskinesias:** Parkinsonian-like movements.

Cause communicating hydrocephalus:	**Cause non-communicating hydrocephalus**
Meningeal carcinomatosis Subarachnoid hemorrhage Encephalocele Normal pressure hydrocephalus	Fourth ventricle ependymoma Colloid cyst of the third ventricle Cerebellar pilocytic astrocytoma Brain stem glioma Craniopharyngioma

A child had VP shunt surgery when he was 40 days old, presented with fever and hypoactivity Do CSF examination if other causes of fever are excluded

Functional and Epilepsy Neurosurgery

1. Foundations of Functional Neurosurgery

Functional neurosurgery is defined by the principle of "function over pathology." functional neurosurgery intervenes in a brain that may appear structurally normal on standard imaging but is physiologically dysfunctional.

Historical perspective: early lesioning procedures → advent of stereotactic surgery → modern neuromodulation.

2. Lesioning techniques

🧠 1. Thalamotomy

✅ **What is it?**

A **surgical lesion of the ventral intermediate nucleus (VIM)** of the **thalamus**.

🧠 **Why this target?**

The **VIM nucleus** is a major relay between:

- **Cerebellum → motor cortex**

This pathway is responsible for **tremor generation**.

🎯 **Indications**

- **Essential tremor**
- **Parkinsonian tremor** (especially unilateral)

❗ Does **NOT** help rigidity or bradykinesia.

🧬 **Mechanism**

- Interrupts abnormal oscillatory signals
- Stops tremor transmission to motor cortex

⚠️ **Side effects (if bilateral)**

- Dysarthria
- Ataxia
- Weakness

➡️ Usually done **unilaterally**

🧠 **Exam pearl:**

Thalamotomy treats tremor only.

2. Pallidotomy

✅ **What is it?**

A lesion made in the **globus pallidus internus (GPi)**.

🧠 **Why this target?**

In Parkinson's disease:

- GPi is **overactive**
- Sends excessive inhibitory signals to the thalamus
- Leads to **bradykinesia and rigidity**

🎯 **Indications:**

- **Parkinson's disease**
- Especially for:
  - Rigidity
  - Bradykinesia
  - Levodopa-induced dyskinesia

🧬 **Mechanism:**

- Reduces excessive inhibitory output
- Improves movement initiation and control

🧠 **Clinical note:**

More effective for **rigidity and bradykinesia** than tremor.

🧠 **Exam pearl:**

Pallidotomy → Parkinson rigidity & dyskinesia

🧠 3. Anterior Cingulotomy

✅ **What is it?**

A lesion in the **anterior cingulate cortex**, part of the **limbic system**.

🧠 **Why this target?**

The anterior cingulate:

- Processes **emotion, pain perception, attention**
- Plays a role in obsessive thoughts and affective responses

🎯 **Indications:**

- **Severe refractory psychiatric disorders**, such as:
  - OCD
  - Major depression
  - Chronic pain syndromes

🧬 **Mechanism:**

- Reduces emotional response to distress
- Does **not** remove cognition or consciousness

🧠 **Exam pearl:**

Cingulotomy treats psychiatric disease, not movement disorders.

3. Ablative Approaches

1️⃣ Radiofrequency Ablation (RFA)

🔹 **What it is:**

- A **heated electrode** is inserted into a precise brain target.
- Heat (≈70–90°C) destroys abnormal neural tissue.

🔹 **How it works:**

- Electrical current → heat → **coagulative necrosis**
- Immediate effect (unlike radiosurgery).

🔹 **Common targets:**

- **VIM nucleus** → tremor
- **GPi** → Parkinson's rigidity
- **Cingulate gyrus** → psychiatric disorders

🔹 **Advantages:**

✅ Precise
✅ Immediate clinical effect
❌ Irreversible
❌ Risk of hemorrhage

🧠 **Summary:**

Radiofrequency ablation = heat-based lesioning with immediate effect.

2️⃣ MR-Guided Focused Ultrasound (MRgFUS)

(Used mainly for Essential Tremor)

🔹 **What it is:**

- Uses **high-intensity ultrasound waves**
- Guided by **real-time MRI**
- No incision, no skull opening

🔹 **How it works:**

- Ultrasound beams converge on a single brain target
- Heat causes **precise thermal ablation**

🔹 **Target:**

- **Ventral intermediate nucleus (VIM) of thalamus**

🔹 **Advantages:**

✅ Non-invasive
✅ Immediate tremor relief
✅ No craniotomy

🔹 **Disadvantages:**

❌ Limited to unilateral treatment
❌ Not reversible
❌ Skull density can limit effectiveness

🧠 **Exam pearl:**

MRgFUS = non-invasive thalamotomy for essential tremor

3️⃣ Radiosurgery (Gamma Knife)

🔹 **What it is:**

- Delivers **focused gamma radiation**
- Causes **delayed tissue necrosis** (weeks–months)

🔹 **Used for:**

- **Subthalamotomy** in Parkinson's disease
- Some tremor cases

🔹 **Target:**

- **Subthalamic nucleus (STN)**

🔹 **Advantages:**

✅ Non-invasive
✅ Precise

🔹 **Disadvantages:**

❌ Delayed effect
❌ Risk of radiation necrosis
❌ Less predictable than DBS

4. Functional neurosurgery in Parkinson' s Disease

The dopaminergic neuron loss in the **Substantia Nigra (SN)** leads to a catastrophic loss of dopaminergic tone in the striatum, which fundamentally disrupts the balance between the direct and indirect motor pathways (imbalance in the basal ganglia circuitry).

In the Parkinsonian state, the loss of dopamine results in the **pathological hyper-activity** of the Subthalamic Nucleus (STN) and the Globus Pallidus Internus (GPi). This "overactive" state causes a chronic, excessive inhibition of the motor thalamus.

Because the thalamus is responsible for "driving" the motor cortex, its hyper-inhibition manifests as the classic motor triad: rigidity, bradykinesia, and tremor.

The Circuit Breaker

The rationale for controlled lesioning or Deep Brain Stimulation (DBS) is to act as a **functional circuit breaker**. By disrupting the excessive inhibitory output from the STN or GPi, we alleviate the pathological hyper-inhibition of the motor thalamus.

This "rebalances" the circuitry, allowing the thalamo-cortical projections to fire appropriately and restoring fluid motor function. Lesioning in PD works by destroying overactive basal ganglia targets (thalamus, GPi, STN) to restore motor balance.

Targeted Lesioning Techniques

Target Nucleus	Primary Indication /Symptom Improved	Clinical Considerations /Risks
Ventral Intermediate Nucleus (VIM) Thalamotomy	Parkinsonian Tremor and **Essential Tremor** (often via MRgFUS).	Primarily unilateral; bilateral procedures carry high risk of dysarthria and cognitive slowing.
Globus Pallidus Internus (GPi) Pallidotomy	Dyskinesias, Rigidity, and **Bradykinesia**.	-
Subthalamic Nucleus (STN) Subthalamotomy	Akinesia and Rigidity.	**High-stakes risk:** Permanent hemiballismus if the lesion is inaccurately placed.

Limitations

Irreversibility: If a lesion is misplaced or too large, the resulting deficits—specifically speech dysarthria, cognitive changes, and swallowing dysfunction (dysphagia)—are permanent.

This risk profile is significantly amplified in bilateral cases, which is why DBS has become the preferred standard for patients requiring bilateral circuit modulation.

Stereotactic neurosurgery:

Used to precisely localize the target.

Methods of creating the lesion:

- Radiofrequency thermocoagulation.
- Focused ultrasound (MRgFUS – incisionless).
- Gamma Knife radiosurgery.

Ablative Lesioning vs. Deep Brain Stimulation (DBS)

	Ablative Lesioning	Deep Brain Stimulation (DBS)
Cost	Significantly lower; accessible in resource-limited settings.	Higher; high-cost hardware and maintenance.
Reversibility	Irreversible; permanent destruction.	Reversible and programmable post-operatively.
Bilateral Capability	High risk; usually limited to unilateral.	Safe and standard for bilateral symptoms.
Implanted Hardware	None.	Permanent leads, extensions, and pulse generators.
Speed of Effect	Immediate clinical response.	Requires weeks/months of programming for optimization.

5. Epilepsy Surgery

Surgical intervention is a strategic necessity in drug-resistant epilepsy (DRE), as chronic uncontrolled seizures carry significant risks of sudden death (SUDEP) and cognitive decline.

The goal of surgery is the complete removal or disconnection of the **epileptogenic zone**—the specific region of cortex essential for seizure generation (seizure freedom or reduction with preservation of neurological function).

Earlier surgery = better outcomes (especially in children)

Patient Selection

- **Inclusion Criteria**
  - **Drug-Resistant Epilepsy (DRE):** (failure of ≥2 appropriate medications)
  - **Localized Zone:** (via MRI, EEG, or SEEG/depth electrodes).
  - **Acceptable Risk Profile:** Favorable risk-to-benefit ratio.
- **Contraindications**
  - **Generalized Epilepsy**
  - **Poor Localization**
  - **Major Comorbidities**

Surgical Procedures

Procedures are classified by their intent: curative resections aim for the total elimination of seizures, whereas palliative procedures aim to reduce seizure frequency or intensity.

A- Resective Surgery (Curative Intent)

1. Anterior Temporal Lobectomy (ATL):

The most common and successful epilepsy procedure. It is the gold standard for **Mesial Temporal Lobe Epilepsy (MTLE)**, with **60–80%** of patients achieving seizure freedom.

Consultant Pearl: **Left Temporal Lobectomy** often requires **Awake Surgery** with intraoperative language mapping to protect eloquent speech centers.

2. Lesionectomy:

Targeted removal of a specific structural pathology (e.g., cavernoma, tumor, or focal cortical dysplasia) identified on imaging.

3. less common, more complex: Frontal, parietal, occipital resections

B- Palliative Procedures

	Indication/Target	Goal
Corpus Callosotomy	Drop attacks/Atonic seizures.	Palliative; prevents rapid secondary generalization to reduce fall-related injuries.
Multiple Subpial Transections (MST)	Seizures in eloquent cortex (motor/speech).	Disrupts horizontal seizure spread while preserving vertical functional columns.
Neuromodulation	VNS, RNS, or DBS (Anterior Thalamus).	Palliative; typically aimed at a **50% reduction** in seizure frequency.

Outcome Analysis

- **Temporal Lobe Cases:** best results (up to 70% seizure freedom). Lt tempral lobectomy (awake surgery)
- **Extra-temporal Resections:** lower but still meaningful benefit.
- **Neuromodulation (VNS/RNS/DBS):** usually 50% reduction rather than full seizure freedom.

Risk Profile

- **Cognitive/Psychological:** Potential for memory decline (especially with dominant temporal resections) or language impairment.
- **Neurological:** Visual field cuts (superior quadrantanopia) or hemiparesis depending on resection proximity to the internal capsule or optic radiations.
- **General Surgical:** Standard risks of infection, hemorrhage, and stroke.

Degenerative Spinal Disease

1. Spinal anatomy

Ligamentum Flavum:

- Attached to the inner part (anterior surface) of the lamina above and the outer part (upper border) of the lamina below.
- Covers the interlaminar space; it is present throughout the spine, including the cervical region.

Naming Conventions:

- **Intervertebral discs** are named according to the vertebrae immediately above and below them.
- **The neural foramen** is named after the exiting nerve root.
- **The Uncus:** The specific anatomical structure prone to damage in cases of lateral herniation.

Low Back Pain:

- Defined as any pain located between the costal margin and the buttock crease.
- 90% of low-risk back pain patients show improvement within one month of non-surgical treatment.

2. UMN vs. LMN

To determine if the "problem" is central (brain/cord) or peripheral (nerve root/nerve).

LMN Lesion (Peripheral)	UMN / Central Problem
Hypotonia (Flaccidity)	Spasticity / Hypertonia
Fasciculations and Fibrillations	Neurogenic Atrophy
Hyporeflexia / Absent Deep Tendon Reflexes	Hyperreflexia
Note: Disuse atrophy is NOT a primary LMN component.	

Babinski Sign: Its presence indicates a central (UMN) problem. It is absent in pure radiculopathy.

Romberg Sign: A positive test indicates a lesion in the Dorsal Column, affecting proprioception.

Spinal Cord Hemisection: paralysis is ipsilateral to the lesion.

Distribution of Prolapsed Discs by Spinal Region

Spinal Region	Percentage of Total Prolapses	Most Common (M.C.) Vertebral Levels
Lumbar	79%	L4-5 and L5-S1 (95% of all lumbar)
Cervical	19%	C5-6
Thoracic (Dorsal)	2%	D11-12

3. Cervical Disc Prolapse

Prolapse Location	Affected Structure	Resulting Clinical Condition
Central	Spinal Cord	Myelopathy
Mediolateral (Rt/Lt)	Nerve Root	Radiculopathy (Pain in the distribution of a particular nerve root) or Brachialgia (Upper Limb pain)

Comparative Localization: C5-C6 vs. C6-C7

Feature	C5-C6 Pathology (C6 Root)	C6-C7 Pathology (C7 Root)
Muscle Weakness	Biceps	Triceps and Wrist Flexion
Reflex Status	Absent/Diminished Biceps reflex	Diminished/Absent Triceps jerk
Sensory Findings	Index finger (Medial arm)	Index and Long fingers

Signs of Cervical Myelopathy:

Hoffman Sign: A reflex contraction of the thumb and index finger upon flicking the nail of the middle finger.

Inverted Supinator Jerk: indicating a cord level lesion.

Torticollis: as a protective muscular response.

Golden standard diagnostic image: Cervical MRI.

Surgery: Anterior Cervical Discectomy and Fusion =ACDF.

(Total removal of the disc through an anterior approach and the placement of an implanted cage, which is essential for maintaining post-operative disc height and segmental stability).

4. Thoracic (Dorsal) Disc Prolapse

Representing only 2% of spinal prolapses (unique due to its rarity), these often occur at levels such as D11-12.

- **Clinical Manifestations:**
  - Central compression leads to myelopathy.
  - Notably, nerve root compression in this region is rarely symptomatic, the intercostal nerves are supported by the thoracic cage, often masking radicular symptoms.. (Unlike the limb-bound nerves of the cervical and lumbar regions)
- **Treatment Options:**
  - **Conservative:** The majority of cases
  - **Surgical:** If neurological deficits progress, options include decompression or specialized Endoscopic Thoracoscopic Discectomy.

5. Lumbar Disc Prolapse: Sciatica and Clinical Correlation

Typically presents as a para-central protrusion, frequently impacting the sciatic nerve (composed of roots L4 through S3). This resulting in radiculopathy, or sciatica. CES is characterized by initial sciatica followed by progressive urinary retention.

Nerve Root Compression: depends on the direction of the herniation:

Posterolateral (Standard) Prolapse: Typically compresses the traversing nerve root at that level, because it affects the root that has already exited the spinal canal, e.g., an L4-L5 posterolateral herniation affects the L5 root).

Far Lateral Prolapse: Compresses the exiting nerve root at the same level, e.g., an L3-L4 far lateral herniation affects the L3 root. more painful than medial discs and are frequently associated with fragmented discs.

Disc Level	Direction	Nerve Root	Motor Deficit	Reflex Change	Sensory/Pain
L3-L4	Far Lateral	L3	Knee Extension	Absent/Weak Knee Jerk	Medial thigh numbness
L4-L5	Posterolateral	L5	Foot Dorsiflexion	N/A (Ankle Jerk intact)	Big toe distribution
L4-L5	Far Lateral	L4	Foot Inversion	Absent/Weak Knee Jerk	Paresthesia to the knee
L5-S1	Posterolateral	S1	Plantar flexion	Absent/Diminished Ankle Jerk	Right or Left Sciatica

Conus Medullaris vs. Cauda Equina Syndrome:

	Conus Medullaris Syndrome (CMS)	Cauda Equina Syndrome (CES)
Vertebral Level	Restricted to L1-2 (Terminal Cord)	Any level between L2 and S1
Pathological Trigger	Typically a "huge" disc volume at L1-2	Multi-level or large lower lumbar prolapse
Primary/First Symptom	Bladder dysfunction	Sciatica
Bladder Progression	Early overflow incontinence	Retention precedes overflow incontinence

Neurosurgical Emergencies:

- **Acute foot weakness**, specifically the loss of **Plantar Flexion** (indicative of S1 nerve root compromise).
- **Autonomic failure**, presenting as **overflow incontinence** (the terminal stage of Cauda Equina Syndrome).

Surgical Standards:

Microscopic or Endoscopic: **Interlaminar Fenestration** is paired with **Sequestrectomy.**

Sciatica Recurrence: The most common cause of sciatica returning after a successful lumbar surgery is a recurrence of the disc prolapse.

Note that gynecological injuries are not considered a standard complication of lumbar disc surgery.

Indications for Surgery

1. **Cauda Equina Syndrome**
2. **Progressive Motor Deficit (M.C):** Deteriorating strength (power dropping to 3/5 or 1/5).
3. **Quality of Life:** Intractable pain that fails to respond to conservative measures.

Diagnostic Logic: The Sacralized L5 Challenge

- **Standard Observation:** An L5-S1 down-migrating disc typically results in S1 sciatica.
- If a patient presents with L5 sciatica but the MRI indicates an L5-S1 disc prolapse, the clinician must suspect the presence of a **sacralized L5 vertebra** (a transitional vertebra).
- **Mandatory Action:** In this scenario, an L-S X-ray (AP and lateral views) is mandatory to confirm the vertebral numbering. This often reveals that what appeared to be L5-S1 on MRI is functionally the L4-5 level.

6. Spinal Canal Stenosis

Combination of mechanical stressors and inflammatory processes that progressively narrow the space available for the spinal cord and nerve roots.

The presentation of stenosis is often chronic, with the L4-L5 level being the most common site of lumbar involvement. pain is a hallmark, frequently exacerbated by back flexion.

Contributing Factors	Non-Contributing Factors
Multiple Osteophytes	Denticulate Ligament Hypertrophy
Ligamentum Flavum Hypertrophy	
Facet Joint Hypertrophy	
Multiple Disc Bulges	

7. MRI: The Modic Classification

Type	Process	T1 Signal	T2 Signal
Type 1	**Edema / Inflammation**	**Low**	**High**
Type 2	**Fatty Replacement**	**High**	**High**
Type 3	**Sclerosis**	**Low**	**Low**

A motor power of 1/5 on the MRC scale requires immediate MRI investigation; absolute surgical indications include Cauda Equina and progressive deficits.

Spinal Cord Injuries (SCI)

1. Epidemiology and Etiologies

Gender Ratio	4:1 (Male to Female)
Peak Age Demographic	60% of cases occur between ages 15–25
Anatomical Distribution	55% Cervical; 15% Thoracic (distributed 1/3 each in upper, middle, lower); 15% Thoracolumbar junction; 15% Lumbar
Head-Spine Correlation	5–15% of head injuries have spinal injury; 5% of spinal injuries have head injury
Cervical Sentinel Factor	15% of injuries above the clavicle involve a C-spine injury
Multi-level Fracture Risk	5–15% of C-spine fractures involve a second, non-contiguous vertebral fracture
Cervical Neuro-Involvement	>50% of cervical spine trauma involves neurologic injury

Primary Etiologies:

1. **Road Traffic Accidents (RTA):** The most common cervical injury.
2. **Sports:** Often associated with high-energy axial loading (e.g., diving, rugby).
3. **Falls:** geriatric populations and occupational accidents.
4. **Assaults:** penetrating injuries or direct focal cord compromise.

2. Anatomy and Pathophysiological Foundations

Bony/Structural Components ("The Container")	**Neural Components ("The Content")**
Fractures: Disruption of vertebral bodies or posterior elements.	**Complete Cord Injury:** Total absence of motor and sensory function below the lesion level.
Dislocations: causing acute canal narrowing.	**Incomplete Cord Injury:** Partial preservation of neurological pathways (sensory or motor).
Ligamentous Injury: Tears leading to instability.	

Pathophysiological Drivers of Damage:

1. **Traction and Compression:** Disruption of neural axons and microvasculature.
2. **Loss of Auto-regulation:** The cord loses its ability to maintain constant blood flow despite changes in systemic pressure.
3. **Secondary Ischemic Cascade:** Because auto-regulation is lost, systemic hypotension leads directly to cord ischemia, escalating the initial injury.
4. **Spinal Shock:** A transient state of reflex depression (areflexia) that masks the true extent of permanent neurological deficit.

4. Mechanisms of Injury (MOI)

1. **Hyperflexion:** resulting in anterior wedge fractures and posterior ligamentous disruption.
2. **Hyperextension:** elderly patients with pre-existing stenosis or falls hitting the chin.
3. **Axial Loading:** result in **burst fractures**.
4. **Direct Trauma / Penetrating Injuries:** typically causing hemi-sections of the cord.

5. Clinical Syndromes of Spinal Cord Injury

A- Complete Spinal Cord Transection Syndrome:

Mechanism: Total cord disruption

Clinical Presentation / Sensory-Motor Loss:

- Quadriplegia; upper/lower extremity areflexia; anesthesia below level.
- Neurogenic Shock: Hypotension and hypothermia *without* compensatory tachycardia
- Loss of rectal and bladder sphincter tone.
- Respiratory insufficiency (above C4, damage to Phrenic nerve)
- Spinal shock

B- Incomplete Spinal Cord Injuries:

Name	Mechanism / Cause	Clinical Presentation
Central Cord Syndrome ***(M.C)***	Severe neck hyperextension	Arm weakness > leg weakness. **Anatomical Hallmark:** Pain/temp affected because lateral spinothalamic fibers cross just ventral to the central canal. "Cape-like" dissociated with sensory loss.
Anterior Cord Syndrome	Anterior spinal artery infarction (Trauma, Disc prolapse)	Paralysis and loss of pain/temp below lesion. Touch, vibration, and proprioception are spared (dorsal columns intact).
Brown-Séquard Syndrome	Cord hemi-section (e.g., stab)	**Ipsilateral:** Paralysis, pyramidal signs and loss of vibration/position sense below lesion level. **Contralateral:** Loss of pain/temp below lesion level.
Posterior Cord Syndrome	Injury to dorsal columns (least common)	Loss of proprioception, ataxia, and faltering gait. power and general sensation remain intact. Pain, Temp, Sensory, Motor, remain intact below the level
Cauda Equina Syndrome	Polyradiculopathy (L2 and below)	Asymmetric **(LMN)** weakness; Perianal numbness; radicular pain; saddle anesthesia; late sphincter disturbance.
Conus Medullaris Syndrome	Terminal cord injury (T12-L1)	Characterized by sudden, early disturbance of bowel and bladder function. Anal areflexia

6. Acute Management: Field and Hospital

Field Management:

1. **Immobilization:** Rigid C-spine stabilization is the absolute priority.
2. **Maintain blood pressure:** counteract the loss of cord auto-regulation.
3. **Oxygenation**
4. **Brief Motor Exam**

Maintain active bleeding, Awake? Immobilize, Unconscious? might need CPR

In-Hospital Management:

1. **Immobilization**
2. **Systemic Stabilization:** CVS, respiratory, GIT, bladder, and thermoregulation.
3. **Detailed Neuro-Evaluation:** Formal grading of motor/sensory levels.
4. **Radiological Evaluation:** Most importantly Thorax (Airway > Bleeding…)
5. **Steroid Protocols:** to mitigate the inflammatory secondary cascade.

5–10% of patients worsen after ER. It is driven by escalating edema, secondary ischemia, and the unforgivable failure of inadequate immobilization.

7. Radiological Evaluation

Imaging Modality	
X-ray (1st step)	**Cross-table:** 85% Sensitive; a rapid but insufficient screen for clearance. **AP / Lateral X-ray:** 92% Sensitive; For any fracture or dislocation. **Swimmer's View:** Mandatory for visualizing the **C7-T1 junction** (when the shoulders obscure a standard lateral view). The most important **Flexion-Extension:** Used to rule out dynamic ligamentous instability in the awake, cooperative patient.
CT-Scan	**The Gold Standard** for bony architecture, burst fractures, and surgical planning. (not in slides)
MRI	Most useful for visualizing soft tissue structures For bleeding, Disc prolapse… Not used in ER

Spinal instrumentation (plates, screws, and cages) is required for internal fixation.

The transition from life-saving surgical intervention to long-term **rehabilitation** is the defining factor in a patient's eventual quality of life and functional independence.

8. Important P.Ps Information

Brown-Sequard at T6:

- Dissociative sensation
- 90% can walk

Component of Horner's syndrome:

1. Miosis
2. Enophthalmos
3. Anhidrosis
4. Correctible ptosis

Nerve fibers share in the control of normal bladder function:

A. Sacral
B. Lumbar
C. Descending cortical

The MRC Scale for Muscle Strength:

Grade	Description
0	No muscle contraction (Total paralysis).
1	Flicker or trace of contraction (Visible or palpable contraction, but no joint movement).
2	Active movement with gravity eliminated (The patient can move the limb across a flat surface, but not lift it).
3	Active movement against gravity (The patient can lift the limb, but it falls with even slight resistance).
4	Active movement against gravity and resistance (Subdivided into 4-, 4, and 4+ in clinical practice).
5	Normal power (Full strength against heavy resistance).

Guillain-Barré Syndrome (GBS):

Acute inflammatory demyelinating polyradiculoneuropathy.

Because it affects the Peripheral Nervous System (PNS), it classically presents with (LMN) signs.

Autonomic instability manifests as:

- **Fluctuating Blood Pressure:** Patients often experience both labile hypertension and orthostatic hypotension.
- **Cardiac Arrhythmias:** Tachycardia or bradycardia.
- **Ileus and Urinary Retention:** Due to involvement of the autonomic nerves.

- **Acute arterial insufficiency**:

Sudden Onset: Acute, sudden pain in a patient with multiple cardiovascular risk factors (Diabetes Mellitus, Ischemic Heart Disease with stents, and dyslipidemia) is a classic trigger to look for vascular compromise.

Refractory to Morphine: Pain from acute limb ischemia is notoriously "out of proportion" to physical findings and is often resistant to even high doses of opioids because the underlying tissue is actively dying from lack of oxygen (ischemia).

The "6 Ps":

1. **Pain:** Sudden, severe, and often "out of proportion" to physical findings.
2. **Pallor:** The limb appears pale or mottled.
3. **Pulselessness:** Absent pulses distal to the occlusion (the most reliable physical sign).
4. **Paresthesia:** "Pins and needles" or numbness (indicates early nerve ischemia).
5. **Paralysis:** Loss of motor function (indicates advanced, potentially irreversible ischemia).
6. **Poikilothermia:** The limb feels "perishingly cold" as it equilibrates with room temperature.

Lack of Relief with Position/NSAIDs

- **Artery of Adamkiewicz supplies:** Thoracic vertebrae
- **Associated with high velocity pelvic fracture:** Peripheral nerves injury
- **Most common affected cranial nerve by Spinal injury:** Abducent
- **Jefferson fracture:** Fracture of C7
- **Cause of cervical myelopathy in age above 50:** Cervical spondylosis
- **Which spinal cord injury should undergo surgical treatment?** Compression

Spinal Tumors

1. Introduction

The following table summarizes the fundamental surgical interventions utilized to access the cranial vault and manage intracranial pathology:

Procedure Name	Definition	Surgical Action/Objective
Trepanation (Burr hole)	Drilling a localized hole into the skull.	Exposure of the dura and brain to treat pathologies such as subdural hematoma drainage.
Craniotomy	Surgical removal of a bone flap.	Providing access to the dura or brain for definitive surgery (e.g., tumor resection); the bone flap is later fixed back into place.
Craniectomy	Removal of a bone flap without immediate replacement.	To allow for significant brain expansion and decompression in the setting of refractory edema.
Cranioplasty	Reconstruction of a skull defect. such as **titanium mesh** or **bone cement**.	Repairing a missing bone flap to restore protection and aesthetics.

2. Classification Spinal Cord Tumors:

The anatomical classification:

- **Extradural (55%)**
  - Metastases (Most common)
  - Primary cancers of the bone (e.g., Multiple Myeloma, Plasmacytoma)
- **Intradural (45%) - chronic progressive cord compression-**
  - **Extramedullary (40%):** Located within the dura but outside the cord substance.
    - Meningioma (NF2) , slow-growing, fits best, **females, *thoracic* (dorsal) spine (about 80% of cases)**
    - Neurofibroma
    - Schwannoma (NF2) (more easily excised than neurofibromas) more common in the lumbar region, often presenting with radicular pain
    - Subarachnoid metastasis
    - Lipoma
  - **Intramedullary (5%):** Located within the spinal cord itself.
    - *Incidence:* Accounts for 20% of intraspinal tumors in adults and 35% in children.

The classical classification of tumors:

- **Benign**
- **Cyst**
- **Malignant**
- **Metastasis**

3. Extradural Benign Tumors

Tumor Type	Surgical/Radiological Objective	Key Materials / Clinical Note
Hemangioma	Recognition of vascular vertebral invasion.	Displays a characteristic **"salt and pepper"** appearance on imaging.
Osteoid Osteoma	Identification of benign bone-forming lesions.	Often presents with nocturnal pain relieved by aspirin.
Osteochondroma	Monitoring for cord compression.	A **cartilage-capped** bone tumor; frequently associated with **multiple exostoses**.

4. Spinal Metastases

The most frequent extradural malignant neoplasms.

In adults, the initial site of involvement is typically the **vertebral body, specifically the posterior aspect**, which has significant implications for spinal stability.

More rapid and painful.

Ex; back pain followed by paraparesis then progressed to paraplegia over 6 hours.

Distribution of Metastatic Disease:

- Vertebral: 94% (May involve epidural extension)
- Intradural Extramedullary: 5%
- Intramedullary: 1%

Primary Cancer Sites (Ranked by Percentage):

1. **Breast:** 22% -Via Batson plexus (hematogenous spread)-
2. **Lung:** 15% (Hilar Mass) -Via Batson plexus (hematogenous spread)-
3. **Prostate:** 10% (Mostly lumbar spine) -Via Batson plexus (hematogenous spread)-
4. **Lymphoma:** 10%
5. **Kidney:** 7%
6. **GI Tract:** 5%
7. **Melanoma:** 4%
8. **Unknown/Others:** 28% (combined)

5. Specialized Lesions

- **Intracerebral Hematoma:** Brain Contusion is formally classified as an intracerebral hematoma once its diameter exceeds **3 cm**.
- **Brain Hydatid Cyst:** A unique parasitic lesion that is radiologically and surgically distinct due to its completely **non-vascularized** nature (No enhancement with contrast), CSF intensity fluid

6. Intramedullary Spinal Tumors

Intramedullary lesions are rare (4-10% of CNS tumors), 20% of all intraspinal tumors in adults and 35% in children

Intramedullary Neoplastic Lesions:

- **Glial Neoplasms (90-95%):**
  - **Ependymoma:** 60%, sausage shaped (common in the filum terminale).
  - **Astrocytoma:** 33% (often displays indistinct cord edema).
  - **Ganglioglioma:** 1%.
- **Non-Glial Neoplasms:**
  - **Highly vascular lesions:**
    - Hemangioblastoma
    - Paraganglioma
  - **Rare lesions:**
    - Primary lymphoma
    - Intramedullary metastasis
    - Soiltary fibrous tumor
    - Primitive neuroectodemal tumors

Intramedullary Benign (Non-Neoplastic) Masses:

- ✓ **Syringohydromyelia** (Cystic cavitation of the cord).
- ✓ Multiple Sclerosis (MS) plaques.
- ✓ Transverse myelitis.
- ✓ Arachnoid or ependymal cysts.

7. Froin's syndrome

Is a classic triad of findings in the cerebrospinal fluid (CSF) that indicates a complete blockage of the subarachnoid space, typically caused by a spinal tumor or a large inflammatory mass.

The Diagnostic Triad:

1. **Xanthochromia**
2. **Hyperalbuminosis (Extremely high protein):** exceeds **500 mg/dL** (normal: 15–45 mg/dL).
3. **Spontaneous Clotting:** "coagulum." This happens because the protein concentration is so high that clotting factors (like fibrinogen) are present in the stagnant fluid.

Rest of the file was talking about EDH, SDH, and SAH, I didn't add them since we already mentioned them.

Spina Bifida (Spinal Dysraphism)

1. Definition & Epidemiology

A major birth defect and a type of neural tube defect that involves an opening in the vertebral column caused by the failure of the neural tube to close properly during embryonic development

Neural tube defects are the second most common type of birth anomaly after congenital heart disease, an estimated prevalence of about one to three per 1000 live births.

Anatomic Region	Frequency
Lumbosacral Spine	90%
Thoracic Spine	6%–8%
Cervical Spine	2%–4%

2. Embryological Pathogenesis

Spinal dysraphism is the permanent structural consequence of a disrupted embryological timeline. The *timing* of the failure dictates the *type* of the defect.

Failure during primary neurulation results in **open** defects, while failures in secondary neurulation result in **closed** states.

1. **Gastrulation (Ends mid-3rd week):**
    - The transformation of the bilaminar embryonic disk into a trilaminar disk through the addition of the mesoderm.
2. **Primary Neurulation (Weeks 3 and 4):**
    - Begins with the formation of the neural plaque and groove in the ectoderm (Day 18).
    - Ends with the complete closure of the neural tube within the mesoderm.
    - **Clinical Pearl:** Disruptions here typically cause **Open Spinal Dysraphism** (e.g., Myelomeningocele).
3. **Secondary Neurulation (Weeks 5 and 6):**
    - The final step, occurring immediately after primary neurulation.
    - Disruptions here relate to **Closed Spinal Dysraphism** (e.g., Lipomyelomeningocele).

3. Etiology

- **Genetic Factors:**
    - The MTHFR (methylene tetrahydrofolate reductase)
    - **90–95% of myelomeningocele cases occur in low-risk couples** with no prior history.
    - higher prevalence in females.
- **Nutritional Deficiencies:**
    - ✓ Folic acid is the primary preventative tool. Supplementing at 4 mg/day in high-risk women reduces recurrence risk by 70%.
    - ✓ Other essential nutrients include Inositol, B12, Choline, and Retinoic Acid.
- **Maternal Factors:** Maternal diabetes, obesity, and early pregnancy fever.
- **Medications:** Anticonvulsants, specifically **Valproate**

4. Classification and Subtypes

1- Spina bifida occulta (closed)

Secondary neurulation failure, Congenital absence of a spinous process and variable amounts of lamina (some of the vertebrae -at least 2- are not completely closed).

Reported prevalence range of SBO: 5-30% of North Americans (5-10% is probably more realistic).

The defect may be palpable, and there may be overlying cutaneous manifestations, No visible exposure of meninges or neural tissue.

Often an incidental finding, usually no clinical importance when it occurs alone (Asymptomatic).

SBO may occasionally be associated with diastematomyelia, tethered cord, lipoma, or dermoid tumor → symptomatic: Tethered cord (gait disturbance, leg weakness and atrophy, urinary disturbance, foot deformities…,).

- **Clinical radiological classification:**
    - *With Subcutaneous Mass:* Lipo-myelomeningocele, Meningocele, Myelocystocele.
    - *Without Subcutaneous Mass:* Simple (Tight filum terminale) vs. Complex (Diastematomyelia, Dorsal dermal sinus).
- **The cutaneous stigmata:**
    - ✓ Midline lumbar capillary hemangioma
    - ✓ Focal hairy patch over the thoracolumbar spine
    - ✓ Dermal sinus located above the midsacrum
    - ✓ Midline subcutaneous lipoma

2- Spina bifida aperta/cystica (open)

Primary neurulation failure.

Clinical-radiological: Myelomeningocele, Myelocele/schisis, Hemi-myelomeningocele.

Meningocele: Protrusion of the meninges (filled with CSF) through a defect in skull or spine.

Myelomeningocele: Open spinal cord (with a meningeal cyst), Occurs during the first month of pregnancy, Associated with diastematomyelia

Lipo-myelomeningocele: Deficiency of at least 2 vertebral arches, here covered with lipoma.

5. Prenatal Detection Protocols

- **Serum Alpha-fetoprotein (AFP):** Measured at 15–20 weeks. A level ≥ 2 multiples of the median carries a staggering **relative risk of 224** for NTDs.
- **Ultrasound:** Detects 90–95% of cases; vital for differentiating NTDs from non-neurologic causes of elevated AFP (omphalocele), can help to more accurately estimate gestational age.
- **Amniocentesis:** Recommended for subsequent MM pregnancies if ultrasound is inconclusive; carries a ≈ 6% risk of fetal loss (even if abortion is not considered, it may allow for optimal post-partum care if MM is diagnosed).
- **Fetal MRI**
- **Planning for the mode of delivery:** After the antenatal diagnosis
- **Psychological support for the family:** After the antenatal diagnosis
- **In some centers in-utero surgery can be offered:** After the antenatal diagnosis

6. Clinical and Ancillary Assessment

- **Neurological (Spinal):** Observe spontaneous movement of lower limbs; determine the lowest level of function via painful stimuli response.
- **Chiari II/Hydrocephalus Pivot:** Measure Occipitofrontal Circumference (OFC) and perform a Head Ultrasound within 24 hours. Monitor for inspiratory stridor or apnea.
- **Ancillary Medical Review:** Neonatologist assessment for additional anomalies, especially those that may preclude surgery (e.g. pulmonary immaturity) (MM patients average 2–2.5 additional anomalies, some of which may preclude immediate surgery).
- **Bladder Management:** Initiate regular urinary catheterization and non-emergent urological consult.
- **Orthopedic consultation:** for severe kyphotic or scoliotic spine deformities and for hip or knee deformities.

7. Management and Surgical Intervention

ADMISSION (Acute Stabilization)

- Measure size of defect
- **Positioning:** Place the infant in the **Trendelenburg position**.
- **Lesion Care:** Cover the defect with **Telfa or a wet dressing** to prevent desiccation.
- **Antibiotics:** Mandatory for ruptured lesions; generally unnecessary for unruptured ones.

Surgical Intervention (cannot reverse the neurological deficits)

- **Standard Closure:** Myelomeningocele **must be closed within 24 hours** regardless of whether the membrane is intact. After 36 hours, the lesion is **colonized**, dramatically increasing the risk of postoperative infection.
- **Intrauterine Closure:** This remains controversial. While it may reduce the incidence of Chiari II and hydrocephalus, it provides no improvement in distal neurological function.

8. Associated Malformations and Long-term Outcomes

- **Hydrocephalus:** Occurs in 85–90% of MM cases & 15% of meningocele, typically as part of a Chiari II malformation. Most require a shunt by 6 months. May not be apparent after birth immediately, may be seen as far as 2 years, Deterioration in a MM patient should always be treated as a shunt malfunction until proven otherwise.
- **Tethered Cord:** .Abnormally low conus medullaris. Usually associated with a short, thickened filum terminale, or with an intradural lipoma. Present radiographically in 70% of MM patients (Most common); symptomatic in a minority
- **Other Complications:** Syringomyelia, Dermoid tumors at the repair site (16%), and significant urinary challenges (Neurogenic bladder) (only 3–10% achieve normal continence) (Requires monitoring but not "mandatory" incontinence for all).

- 80% will have normal IQ. Mental retardation is most closely linked to shunt infection.

- 40-85% are ambulatory with bracing, however, most choose to use wheelchairs for ease.

- **What is the management of newborn found to have myelomeningocele and hydrocephalus?** Simultaneous repair and shunt

- **Chiari II Malformation (Arnold-Chiari):** Distinguished from Chiari I by its hindbrain involvement (Small posterior fossa). It is the most common cause of early mortality due to cerebellar herniation & medullary compression at the foramen magnum, leading to respiratory arrest or aspiration.

Finding	Chiari Type 1	Chiari Type 2
Caudal dislocation of medulla	Unusual	Yes
Caudal dislocation into cervical canal	Tonsils	Inferior vermis, medulla, 4th ventricle (small, elongated, and displaced downward)
Spina bifida (myelomeningocele)	May be present	Rarely absent
Hydrocephalus	May be absent	Rarely absent
Medullary "kink"	Absent	Present in 55%
Course of upper cervical nerves	Usually normal	Usually cephalad
Usual age of presentation	Young adult	Infancy
Usual presentation	Cervical pain, suboccipital H/A	Progressive hydrocephalus, respiratory distress

Metric	Detail
Survival Rate	≈ 85% with modern multidisciplinary treatment. Without any treatment, only 14-30% of MM infants survive infancy
Early Mortality Cause	Chiari II (Arnold-Chiari) complications (e.g., respiratory arrest).
Late Mortality Cause	Shunt malfunction.

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