Neurosurgery Final

🔊 **Podcast Style Review (Experimental Feature) - Need to be on Wi-Fi not 4G to work properly!**

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- **NOTE:** Highlighted in **bold** are the important key info!
- Topics are arranged in order of most to least commonly tested
- Check the table of contents below for easier navigation
- Good luck 🍀

Intracranial Pressure (ICP)

- **Definition and Physiology:**
  - Normal ICP: 0-10 mmHg (or 0-140 mm CSF).
  - Monroe-Kellie Doctrine: The sum of volumes (brain, blood, CSF) inside the rigid skull is constant. An increase in one component must be compensated by a decrease in another, or ICP will rise.
  - Pressure-Volume Relationship: Initially, small volume increases cause minimal ICP change (compensation). Once compensatory mechanisms are exhausted, small volume increases cause **large ICP increases**.
- **Signs and Symptoms of Increased ICP:**
  - Early: Headache (often worse in morning), vomiting (may be projectile), visual disturbances (e.g., blurred vision, papilledema).
  - Late: **Decreased level of consciousness (LOC)**, pupillary changes (e.g., unilateral dilation suggesting herniation), motor deficits, vital sign changes (**Cushing's Triad**: **hypertension**, **bradycardia**, **irregular respirations**), abnormal posturing (decorticate, decerebrate).
  - **Papilledema**: Swelling of the optic disc due to raised ICP.
- **Radiological Signs of Increased ICP:**

- Plain Skull X-ray (Chronic): **"Thumb printing" or "beaten silver" appearance**, **erosion of posterior clinoid processes**, widening of sutures (in children), enlarged sella turcica.
- CT/MRI: Effacement of cortical sulci, compression of ventricles, midline shift, signs of herniation. **Intracranial calcification is NOT a sign of raised ICP.**

- **Management of Increased ICP:**
  - **General Measures**:
    - **Elevate head of bed 30 degrees**.
    - Avoid neck flexion/compression of jugular veins.
    - Maintain normal blood pressure, control pain, ensure adequate airway.
  - **Specific Measures**:
    - Osmotic diuretics (**Mannitol**).
    - Hyperventilation (to induce vasoconstriction - **a specific measure**, not general). Use with caution.
    - Sedation (e.g., propofol, barbiturates) to decrease metabolic demand.
    - **CSF drainage** (e.g., external ventricular drain - EVD).
    - **Steroids** (e.g., Dexamethasone) are primarily used for edema around tumors, **not typically for traumatic ICP**.
    - **Decompressive craniectomy**.
- **Complications:**
  - **Herniation**: Displacement of brain tissue (e.g., Uncal - CN III palsy, Central, Subfalcine, Tonsillar - brainstem compression/respiratory arrest).
- **Monitoring:**
  - Sites: Intraventricular (gold standard, allows drainage), intraparenchymal, epidural, subdural, subarachnoid. **Diploic space (skull) is NOT a site.**
  - Waveforms: Lundberg A (plateau) waves indicate pathologically high ICP (>50 mmHg, lasting 5-20 min).

Subarachnoid Hemorrhage (SAH)

- **Definition:** Bleeding into the subarachnoid space.
- **Causes:**
  - **Most common non-traumatic cause: Rupture of a saccular (berry) aneurysm** (~80%).
  - Arteriovenous malformation (AVM).
  - Extension from intracerebral hemorrhage.
  - Trauma (Traumatic SAH).
- **Aneurysms:**
  - Type: **Saccular (Berry) are most common**. Others: fusiform, mycotic, traumatic.
  - Location: **Most common at bifurcations in the Circle of Willis**. **Anterior communicating artery (ACOM)** is the single most common site.
  - Risk Factors for Rupture: Hypertension, smoking, certain genetic conditions (e.g., Polycystic Kidney Disease, Ehlers-Danlos, Marfan - mentioned as risk factors for aneurysms).
- **Presentation:**
  - **Sudden onset, severe headache ("worst headache of my life")**.
  - Neck stiffness (meningismus).
  - Decreased LOC, vomiting, photophobia.
  - Focal neurological deficits (e.g., CN III palsy with PCOM aneurysm).
  - Seizures.
- **Diagnosis:**
  - **Non-contrast CT Brain**: Initial test of choice. Shows hyperdensity (blood) in sulci, fissures, basal cisterns.

    - **Lumbar Puncture (LP)**: **Perform if CT is negative but clinical suspicion is high** (esp. >6-12 hrs post-onset). Shows blood, **xanthochromia** (yellowish CSF supernatant due to bilirubin, appears after several hours, lasts up to 2 weeks).
    - Cerebral Angiography (DSA - Digital Subtraction Angiography): **Gold standard** to identify the source (aneurysm, AVM). CT Angiography (CTA) or MR Angiography (MRA) can also be used.
- **Grading Systems:**
    - Clinical: Hunt & Hess, World Federation of Neurological Surgeons (WFNS).
    - Radiological: Fischer scale (based on CT appearance/amount of blood, predicts vasospasm risk).
- **Complications:**
    - **Rebleeding**: Highest risk within **first 24 hours**. Major cause of mortality/morbidity. Surgery/coiling aims to prevent this.
    - **Cerebral Vasospasm**: Delayed narrowing of cerebral arteries. Typically occurs **4-14 days** after SAH. Can cause ischemic stroke. Diagnosed with angiography/TCD. Treated with "Triple H" therapy (Hypertension, Hypervolemia, Hemodilution - use with caution) and nimodipine. Radiographic evidence in 50-70%, symptomatic in ~30%.
    - **Hydrocephalus**: Acute or delayed. Communicating type most common (impaired CSF absorption). May require EVD or permanent shunt.
    - **Hyponatremia**: Due to SIADH or cerebral salt wasting.
    - Seizures.
    - Cardiac complications (ECG changes, arrhythmias).
- **Management:**
    - Secure the aneurysm (prevent rebleeding) via **surgical clipping** or **endovascular coiling**. Grades 1-3 typically treated early.
    - Manage complications (vasospasm, hydrocephalus, etc.).
    - Intensive care unit monitoring.
- **AVM vs Aneurysm:** AVMs have lower annual bleed risk (~1-2%) and lower mortality (~10%) per bleed compared to aneurysms. AVMs have poorer prognosis than aneurysms.

Intracranial Hematomas (Epidural & Subdural)

- **Epidural Hematoma (EDH):**
    - Location: Potential space **between the inner surface of the skull and the dura mater**.
    - Source: **Middle meningeal artery** rupture (most common, ~90%), dural sinus, diploic veins. Often associated with **temporal bone fracture**.
    - CT Appearance: **Lens-shaped (lenticular), biconvex hyperdensity**. **Does NOT cross suture lines** (dura adherent at sutures).
    - Presentation: Can have a **"lucid interval"** (initial LOC → recovery → subsequent decline) in ~1/3 of cases. Rapid deterioration possible due to arterial bleeding.
    - Epidemiology: Represents ~3% of head injuries. **Rare before age 2 and after age 60**.
    - Management: **Surgical emergency**. Evacuation via burr holes or craniotomy.
    - Prognosis: Generally better than acute SDH if evacuated promptly before significant herniation. Outcome significantly better when compared to acute subdural hematoma.
- **Subdural Hematoma (SDH):**
    - Location: Potential space **between the dura mater and the arachnoid mater**.
    - Source: **Tearing of bridging veins** crossing from cortex to dural sinuses (most common), cortical artery laceration, severe brain injury.
    - CT Appearance: **Crescent-shaped hyperdensity** (acute). **CAN cross suture lines** but limited by dural reflections (falx, tentorium). May show associated brain injury (contusions, edema).
        - *Subacute:* Isodense (difficult to see).

        - *Chronic:* Hypodense (dark), may have membranes.
    - Presentation: Variable, often presents with decreased LOC, focal deficits. Often associated with **more severe underlying brain injury** than EDH. Acute SDH has **higher mortality/morbidity than EDH**.
    - Epidemiology: More common than EDH. Acute SDH often in trauma. Chronic SDH common in **elderly**, alcoholics, those on anticoagulants (due to brain atrophy, stretched bridging veins).
    - Management: Surgical evacuation (craniotomy for acute; burr holes often sufficient for chronic) if significant mass effect or neurological decline.
- **Key Differences:**
    - Shape: EDH = Lens, SDH = Crescent.
    - Sutures: EDH = Stops, SDH = Crosses.
    - Source: EDH = Mostly Arterial (MMA), SDH = Mostly Venous (Bridging Veins).
    - Prognosis: EDH = Better (if treated early), SDH = Worse (due to associated injury).

Brain Tumors

- **General:**
    - **Metastases are the most common intracranial tumors overall**. Common primary sources: Lung > breast > melanoma > renal > colon. Spinal mets most commonly from: Lungs > breast/prostate > kidney > Lymphoma > Thyroid.
    - **Meningioma is the most common *primary* brain tumor in adults**.
    - **Glioblastoma (GBM) is the most common *malignant* primary brain tumor in adults**.
    - **Pilocytic astrocytoma and Medulloblastoma are common *pediatric* primary brain tumors**.
    - **Medulloblastoma** is the most common *malignant* pediatric brain tumor.
    - Location: Adults - mostly supratentorial (~70%). Children - mostly infratentorial (posterior fossa, ~60%).
    - Posterior Fossa Tumors (Adults): Metastases, Acoustic neuroma (schwannoma), **Hemangioblastoma**, Meningioma.
    - Gender: Most brain tumors slightly more common in males, **EXCEPT Meningioma and Pineocytoma (more common in females)**.
    - CSF Seeding (Drop Metastases): **Medulloblastoma, Ependymoma, Glioblastoma**.
    - Calcification: **Oligodendroglioma** is the most common primary tumor to calcify. Craniopharyngioma also commonly calcifies.
- **Specific Tumor Types:**
    - **Meningioma**:
        - Origin: **Arachnoid cap cells**. Extra-axial (outside brain parenchyma).
        - Epidemiology: Most common primary brain tumor, **F>M**, peak age 30-50s.
        - Risk Factors: **Ionizing radiation**, Neurofibromatosis Type 2 (NF2), hormones.
        - Grading: WHO Grade I (**benign**, >90%), Grade II (atypical), Grade III (malignant/anaplastic).
        - Imaging: Often round, well-defined, dural-based, enhance brightly and homogeneously with contrast, may have **"dural tail"**.
        - Treatment: **Surgical resection** (Simpson grading for completeness). Can be curative. Radiotherapy for higher grades or residual/recurrent tumor.
        - Spinal Meningioma: Common in **thoracic spine**, F>M, slow-growing, presents with myelopathy/radiculopathy.
    - **Astrocytoma**:
        - Origin: Astrocytes (glial cells). Intra-axial.
        - WHO Grading:
            - Grade I: **Pilocytic Astrocytoma** (common in children, cerebellum, often cystic with enhancing nodule, good prognosis).
            - Grade II: **Diffuse Astrocytoma** (low-grade, infiltrative).

        - Grade III: **Anaplastic Astrocytoma**.
        - Grade IV: **Glioblastoma Multiforme (GBM)** (highly malignant, common in adults, poor prognosis, often shows central necrosis, ring enhancement, significant edema).
    - **Medulloblastoma**:
        - Epidemiology: Most common **malignant** pediatric brain tumor.
        - Location: **Posterior fossa**, typically **midline (vermis)**.
        - Characteristics: **Spreads via CSF** ("drop metastases"). Highly cellular (PNET family). Radiosensitive and chemosensitive.
        - Presentation: Often hydrocephalus, truncal ataxia.
    - **Ependymoma**:
        - Origin: Ependymal cells lining ventricles and central canal.
        - Location: **4th ventricle** (children, common cause of hydrocephalus), spinal cord (adults).
        - Characteristics: Can seed via CSF.
    - **Oligodendroglioma**:
        - Origin: Oligodendrocytes.
        - Characteristics: Often **calcified**, typically presents with **seizures**. Better prognosis than high-grade astrocytomas.
    - **Schwannoma (Neurinoma)**:
        - Origin: Schwann cells. Extra-axial.
        - Location: Commonly affect cranial nerves (e.g., **Vestibular Schwannoma/Acoustic Neuroma** at CPA - CN VIII), spinal nerve roots. Associated with NF2.
        - Spinal Schwannoma: Often causes radicular pain, may enlarge intervertebral foramen ("dumbbell tumor"). More easily excised than neurofibromas.
    - **Pituitary Adenoma**:
        - Location: Sella turcica.
        - Classification: Microadenoma (<10 mm), Macroadenoma (>10 mm). Functional (hormone-secreting) vs Non-functional.
        - Most common functional type: **Prolactinoma** (causes amenorrhea, galactorrhea, infertility). Others: GH-secreting (acromegaly/gigantism), ACTH-secreting (Cushing's disease).
        - Presentation: Hormonal effects, mass effect (**bitemporal hemianopia** from optic chiasm compression), headache, hypopituitarism.
        - Treatment: **Medical (Dopamine agonists like bromocriptine) is first-line for prolactinomas**. Surgery (typically **transsphenoidal approach**) for others or non-responsive prolactinomas/mass effect.
    - **Hemangioblastoma**: Vascular tumor, often cystic with enhancing mural nodule. Common in posterior fossa in adults. Associated with von Hippel-Lindau disease.
- **Non-Surgical Treatment:**
    - **Dexamethasone** (steroid) for vasogenic edema.
    - Antiepileptics for seizures.
    - Analgesia.
    - Head elevation (if raised ICP).
    - Radiotherapy and Chemotherapy (depending on tumor type/grade).

Spinal Disc Herniation and Spinal Stenosis

- **Lumbar Disc Herniation:**
    - Definition: Protrusion of nucleus pulposus through annulus fibrosus.
    - Most Common Levels: **L5-S1 (affects S1 root)** and **L4-L5 (affects L5 root)** account for ~95%.

    - Nerve Root Compression: Typically affects the **traversing nerve root** (the one exiting one level below the disc). E.g., L4-L5 disc herniation usually compresses the L5 nerve root. **Far lateral** herniations compress the **exiting nerve root** at the same level (e.g., far lateral L4-L5 herniation compresses L4 root).
    - Clinical Features:
        - Low back pain.
        - **Sciatica**: Radicular pain radiating down the leg along the nerve root distribution.
        - Specific Neurological Deficits:
            - **L5 Root**: Pain down lateral leg/dorsum of foot/big toe; Weakness in **foot dorsiflexion** (esp. Extensor Hallucis Longus - EHL), foot eversion; Sensory loss lateral leg/dorsum foot. Reflexes usually normal.
            - **S1 Root**: Pain down posterior leg/lateral foot; Weakness in **plantar flexion** (gastrocnemius - difficulty toe walking), foot inversion; Sensory loss lateral foot/sole; **Absent or diminished ankle jerk (Achilles reflex)**.
    - Diagnosis: **MRI** is the investigation of choice. Straight leg raise test often positive.
    - Management:
        - **Conservative**: Majority improve (e.g., **90%** of low-risk patients improve in **one month**). Includes rest, analgesia, physiotherapy.
        - **Surgical**: Indications include **Cauda Equina Syndrome (CES)**, **progressive/significant neurological deficit**, intractable pain unresponsive to conservative therapy. Procedures: Microdiscectomy.
- **Cauda Equina Syndrome (CES):**
    - Cause: Compression of multiple lumbosacral nerve roots below the conus medullaris. Often due to large central disc herniation.
    - Clinical Triad: **Bilateral sciatica/leg weakness**, **saddle anesthesia** (perineal numbness), **bowel/bladder dysfunction** (urinary retention/incontinence). May have decreased anal tone, absent ankle jerks.
    - Management: **SURGICAL EMERGENCY** requiring urgent decompression.
- **Conus Medullaris Syndrome:** Injury to the sacral cord (conus). Features UMN (hyperreflexia initially) and LMN signs. Early bowel/bladder/sexual dysfunction. Symmetric deficits.
- **Spinal Stenosis:**
    - Definition: Narrowing of the spinal canal, lateral recesses, or neural foramina.
    - Causes: Mostly **degenerative** changes in older adults (>65). Includes **facet joint hypertrophy (osteoarthritis)**, **ligamentum flavum hypertrophy/infolding**, **disc bulging/herniation**, osteophytes, spondylolisthesis. Congenital causes are less common.
    - Most Common Level: L4-L5.
    - Clinical Features: **Neurogenic claudication** (leg pain/numbness/weakness exacerbated by walking/standing, relieved by sitting/flexing forward), back pain. Neurological exam may be normal at rest.
    - Management: Conservative initially, surgery (laminectomy/decompression) for refractory symptoms.

Hydrocephalus and Shunts

- **Definition:** Abnormal accumulation of CSF within the ventricles, leading to ventricular enlargement and potentially raised ICP.
- **Classification:**
    - **Non-communicating (Obstructive):** CSF flow blocked *within* the ventricular system (e.g., between ventricles or at outlets).
        - Causes: **Congenital Aqueductal Stenosis** (most common cause of congenital hydrocephalus), tumors (e.g., colloid cyst of 3rd ventricle, ependymoma of 4th ventricle), Chiari malformation, Dandy-Walker malformation.
    - **Communicating:** Impaired CSF absorption at arachnoid granulations or blockage in subarachnoid space *after* CSF has exited the ventricles.
        - Causes: **Subarachnoid hemorrhage (SAH)**, **meningitis**, intraventricular hemorrhage, meningeal carcinomatosis.
- **Specific Causes/Associations:**

- **Aqueductal Stenosis:** Narrowing of the cerebral aqueduct (of Sylvius) connecting 3rd and 4th ventricles. **Most common cause of congenital hydrocephalus**.
- **Chiari II Malformation:** Associated with **myelomeningocele**. Hindbrain herniation obstructs CSF flow.
- **Dandy-Walker Malformation:** Cystic dilatation of 4th ventricle, agenesis/hypoplasia of cerebellar vermis, enlarged posterior fossa.
- **Normal Pressure Hydrocephalus (NPH):** Idiopathic communicating hydrocephalus in elderly. Classic triad: **Gait disturbance ("magnetic gait")**, **Urinary incontinence**, **Dementia**. CSF pressure is normal or intermittently elevated. Potentially reversible with shunting.

- **Clinical Features (Infants):** Rapid head growth, bulging fontanelle, **"setting sun" sign** (downward gaze deviation), irritability, poor feeding, vomiting, developmental delay.
- **Management:**
  - Treat underlying cause if possible (e.g., tumor resection).
  - **CSF Diversion Procedures:**
    - **Ventriculoperitoneal (VP) Shunt**: Most common. Drains CSF from ventricle to peritoneal cavity.
    - Ventriculoatrial (VA) Shunt: Drains to atrium (less common now).
    - Ventriculopleural Shunt.
    - Lumboperitoneal (LP) Shunt: Used for communicating hydrocephalus or IIH (contraindicated in non-communicating).
  - **Endoscopic Third Ventriculostomy (ETV)**: Creates an opening in the floor of the 3rd ventricle to bypass obstruction (e.g., aqueductal stenosis). Often preferred over shunt if feasible.
- **Shunt Complications:**
  - **Infection (Ventriculitis/Meningitis)**: Most common organism **Staphylococcus epidermidis**. Requires shunt removal, external ventricular drain (EVD), antibiotics.
  - **Obstruction/Malfunction**: Most common complication overall. Proximal (ventricular catheter), valve, or distal (peritoneal) obstruction. Requires shunt revision.
  - Overdrainage (can lead to slit ventricles, subdural hematomas).
  - Underdrainage.
  - Mechanical failure/disconnection/migration.
  - Seizures.
  - Subdural hematoma.

Skull Fractures

- **Types:**
  - **Linear:** Simple break in the bone, no displacement. Most common type. Usually requires no specific treatment unless crossing a vascular groove (risk of EDH) or sinus, or associated with complications.
  - **Depressed:** Bone fragment pushed inward. Requires surgical elevation if: **compound (overlying scalp laceration)**, depression depth > thickness of skull, associated with significant intracranial hematoma, overlies a dural venous sinus, causes neurological deficit, or for cosmetic reasons. Risk of infection and epilepsy.
  - **Basal (Base of Skull):** Fracture involving the skull base (anterior, middle, or posterior fossa). Difficult to see on plain X-ray, better on CT. Often associated with **CSF leak** and **cranial nerve injuries**.
    - Signs:
      - Anterior Fossa: **Raccoon eyes** (periorbital ecchymosis), **CSF rhinorrhea**, anosmia (CN I injury), epistaxis.
      - Middle Fossa: **CSF otorrhea**, **hemotympanum** (blood behind eardrum), deafness/tinnitus/vertigo (CN VIII injury), facial palsy (CN VII injury).
      - Posterior Fossa: **Battle's sign** (mastoid ecchymosis - appears late), potential injury to lower cranial nerves.
    - Management: Often conservative. Observation for CSF leak (most resolve spontaneously within 5-10 days). Prophylactic antibiotics debated, may be given if leak persists. Surgery if leak is persistent/recurrent or

meningitis occurs. LP shunt is NOT treatment of choice for CSF leak.

- **Pond:** Indentation fracture seen in infants (like a dent in a ping-pong ball). Often requires no treatment unless significant depression.
- **Growing Skull Fracture (Leptomeningeal Cyst):** Rare complication of linear fracture in infants/young children where dura is torn, allowing brain/arachnoid to herniate through fracture, preventing healing and causing progressive enlargement. Requires surgical repair.

- **Management Principles:**
  - Assess for associated intracranial injury (CT scan crucial).
  - Compound fractures require cleaning, debridement, antibiotics, and often surgical repair/elevation.
  - Admit patients with significant fractures (depressed, basal, linear crossing vessel/sinus) for observation.

Spinal Cord Injury (SCI)

- **Assessment:**
  - In unconscious patient: Assess for spinal tenderness (less reliable), absence of deep reflexes (part of spinal shock), flaccid paralysis, loss of sphincter tone, inspection of the back. Motor response absence is not definitive. Maintain spinal immobilisation.
  - Glasgow Coma Scale (GCS) used for head injury severity.
- **Complete SCI:** Total loss of motor and sensory function below the level of injury.
  - **Spinal Shock**: Temporary physiological state following SCI characterized by flaccid paralysis, areflexia, loss of sensation, and autonomic dysfunction below the injury level. Can last hours to weeks.
  - Features of complete transection syndrome: Quadriplegia/paraplegia, areflexia, anesthesia below level, **loss of sphincter tone**, respiratory insufficiency (if high cervical), **neurogenic shock**.
- **Incomplete SCI Syndromes:**
  - **Central Cord Syndrome**: **Most common type**. Typically from **hyperextension injury** in patients with underlying cervical spondylosis. Characterized by **greater weakness in upper limbs than lower limbs**. Sensory loss variable (often pain/temperature loss in "cape" distribution). Prognosis often good for ambulation.
  - **Anterior Cord Syndrome**: Injury to anterior 2/3 of cord, often due to anterior spinal artery occlusion or flexion injury. Results in loss of motor function and pain/temperature sensation below the lesion. **Proprioception, vibration, and deep touch are preserved** (dorsal columns intact). Poor prognosis.
  - **Brown-Séquard Syndrome (Hemicord Lesion)**: Results in **ipsilateral loss of motor function, proprioception, and vibration** below the lesion, and **contralateral loss of pain and temperature sensation** starting 1-2 levels below the lesion. Good prognosis.
  - **Posterior Cord Syndrome**: Rare. Damage to dorsal columns. Loss of proprioception and vibration below lesion. Motor function, pain, and temperature preserved. Results in sensory ataxia.
- **Neurogenic Shock:**
  - Hemodynamic instability seen in injuries above T6 due to loss of sympathetic tone.
  - Characterized by **hypotension**, **bradycardia**, and peripheral vasodilation (**warm, dry skin** below injury initially, though hypothermia can occur due to inability to regulate temperature). Distinct from hypovolemic shock (which has tachycardia).
- **Cervical Spine Injury:**
  - Most common level for traumatic SCI.
  - **Most common overall cause of cervical injury is trauma**.
  - Head injury patients (5-15%) have associated spinal injury. Spinal injury patients (~50%) have associated head injury.
  - High index of suspicion needed. Immobilisation is crucial.

Congenital Anomalies

- **Spina Bifida:** Defect in vertebral arch closure.

    - **Spina Bifida Occulta:** Mildest form. Vertebral arch defect without protrusion of meninges or neural tissue. Often asymptomatic, may have overlying skin stigmata (hairy patch, dimple, lipoma). Rarely associated with tethered cord.
    - **Spina Bifida Cystica:** Defect with cystic protrusion.
        - **Meningocele:** Sac contains meninges and CSF, **but no neural tissue**. Spinal cord usually normal. May have minor deficits. Transillumination is helpful. Less common than myelomeningocele. **15%** have hydrocephalus.
        - **Myelomeningocele (MMC): Most common severe form**. Sac contains meninges, CSF, **and malformed spinal cord/nerve roots**. Results in significant neurological deficits below lesion (paralysis, sensory loss, bowel/bladder incontinence). **Strongly associated with Chiari II malformation** and **hydrocephalus (80-90%)**. Other associations: **tethered cord syndrome**, syringomyelia. Usually noted at birth as midline dorsal mass. **Folic acid supplementation pre-conceptionally and early pregnancy reduces risk**. Management requires urgent surgical closure (within 24-72 hrs) to prevent infection and further damage; does **not** reverse existing deficits. Requires multidisciplinary care.
- **Arnold-Chiari Malformation:** Hindbrain herniation through foramen magnum.
    - **Type I:** Cerebellar tonsils herniate (>5mm below foramen magnum). Often presents in adolescence/adulthood with headache, neck pain, syringomyelia.
    - **Type II:** Herniation of cerebellar tonsils, vermis, and brainstem. **Associated with myelomeningocele**. Often presents in infancy with hydrocephalus, brainstem dysfunction.
- **Dandy-Walker Malformation:** Triad of cystic dilatation of 4th ventricle, complete/partial agenesis of cerebellar vermis, and **enlarged posterior fossa**. Often associated with hydrocephalus. Small posterior fossa is **not** a feature.
- **Tethered Cord Syndrome:** Low-lying conus medullaris anchored by a thickened filum terminale, lipoma, or scar tissue. Causes progressive neurological deterioration (gait disturbance, leg weakness, urinary issues, foot deformities, back pain). Often associated with spina bifida. Requires surgical untethering.

Idiopathic Intracranial Hypertension (IIH)

- Also known as Pseudotumor Cerebri or Benign Intracranial Hypertension.
- **Definition:** Syndrome of elevated ICP without hydrocephalus or intracranial mass lesion, and with normal CSF composition.
- **Epidemiology:** Typically affects **young, obese women** of childbearing age. Risk factors include obesity, certain medications (OCPs, tetracyclines, Vitamin A).
- **Clinical Features:** Headache (often pulsatile, worse with Valsalva), transient visual obscurations, pulsatile tinnitus, **papilledema** (hallmark sign, risk of permanent visual loss), **CN VI palsy** (false localizing sign).
- **Diagnosis (Modified Dandy Criteria):**
    1. Symptoms/Signs of increased ICP.
    2. **No localizing neurological signs (except CN VI palsy)**.
    3. **Normal neuroimaging (CT/MRI)** - ventricles may be normal or small. **No space-occupying lesion**.
    4. **Elevated Lumbar Puncture (LP) opening pressure** (>20-25 cmH2O in non-obese, >25 in obese).
    5. **Normal CSF composition**.
- **Management:**
    - Goal: Preserve vision and alleviate symptoms.
    - Weight loss (if obese).
    - Medications to reduce CSF production: **Acetazolamide** (carbonic anhydrase inhibitor) is first-line.
    - Therapeutic LPs (temporary relief).
    - Surgical options for refractory cases or progressive visual loss:
        - **Optic nerve sheath fenestration (ONSF)**.
        - **CSF shunting** (Lumboperitoneal (LP) shunt commonly used, or VP shunt).

Other Key Concepts

- **Cerebral Perfusion Pressure (CPP):**
  - **CPP = Mean Arterial Pressure (MAP) - Intracranial Pressure (ICP)**.
  - Represents the pressure gradient driving blood flow to the brain.
  - Maintaining adequate CPP (typically target >60-70 mmHg) is crucial in managing head injury/raised ICP. Low CPP can lead to ischemia.
  - MAP calculation: MAP = Diastolic Pressure + 1/3 (Systolic Pressure - Diastolic Pressure).
- **Glasgow Coma Scale (GCS):** Standardized tool for assessing level of consciousness. Scores range from 3 (deep coma) to 15 (fully awake).
  - Severity Classification: **Severe Head Injury: GCS ≤ 8**, **Moderate: GCS 9-13**, **Mild: GCS 14-15**.
  - Patients with GCS ≤ 8 typically require intubation.
- **Specific Syndromes:**
  - **Cushing's Triad:** Physiological response to severely raised ICP/brainstem compression. Consists of **Systemic Hypertension**, **Bradycardia**, and **Irregular Respirations (Bradypnea)**. A late and ominous sign. (Note: Cushing *reflex* involves hypertension and bradycardia).
  - **Horner's Syndrome:** Triad of **Ptosis** (mild eyelid droop), **Miosis** (pupil constriction), and **Anhidrosis** (decreased sweating) on one side of the face. Due to disruption of sympathetic pathway. Not typical for SAH.
- **Trauma:**
  - Primary Injury: Occurs at the moment of impact (e.g., contusion, laceration, DAI, fracture).
  - Secondary Injury: Develops subsequently due to physiological responses (e.g., **edema**, **hematoma expansion**, ischemia, **infection**, raised ICP). Occurs hours to days after initial injury. Management aims to prevent/minimize secondary injury.
  - **Diffuse Axonal Injury (DAI):** Shearing injury to axons, often from acceleration-deceleration forces. Can cause prolonged coma despite relatively normal initial CT scan.
- **Epilepsy:**
  - Causes: Can be post-traumatic, post-stroke, due to tumors, infections, congenital malformations, vascular lesions (SAH, AVM), etc. Multiple Sclerosis is **not** listed as a likely cause in the provided context.
  - Post-Traumatic Epilepsy: Risk increased with depressed skull fracture, intracranial hematoma, dural tear, brain contusion.
  - Temporal Lobe Epilepsy: Most common site for refractory epilepsy amenable to surgery.
- **Cranial Nerves:**
  - CN III (Oculomotor) Palsy: Can be caused by **PCOM aneurysm** compression (pupil often involved) or uncal herniation.
  - CN VI (Abducens) Palsy: Can be a non-specific sign of raised ICP (false localizing sign) as seen in IIH.
  - Parasympathetic Fibers: Carried by CN III, **CN VII**, CN IX, CN X.