

5th year

Neurosurgery

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Head Injuries

Basic anatomy of the head include: Scalp, Skull, Meninges, Brain Tissue, CSF and Blood

Head injuries are considered a major cause of death and disability all around the world

Head injuries are usually more common in males (occupation, motor vehicle accidents) and extremes of age (falls)

Head injuries are classified into:

1. SCALP INJURIES
2. SKULL INJURIES
3. BRAIN INJURIES

SCALP INJURIES :

- Scalp injuries causes a profuse bleeding due to the huge number of blood vessels found in the scalp and due to their poor constrictive ability, so scalp injuries are a cause of hypovolemic shock in both adults and children
- It is considered as a MAJOR cause of hypovolemic shock in children. When encountering a hypovolemic shock in children , 3 things must come into mind: Cephalohematoma, Subdural hematoma and Scalp injuries
- Types of scalp injuries:
 1. ABRASION (superficial or deep) : rare due to thick skin of the scalp and hair
 2. CONTUSION
 3. WOUNDS (Cuts or Lacerations)
 4. AVULSION
 5. SUBGALEAL (SUB-APONEUROTIC)
 6. SUBPERIOSTEAL (CEPHALOHEMATOMA)
- Wounds in general are classified into Cut or Laceration wounds according to the Margin. Cut wounds have regular margins whereas Laceration wounds have irregular margins
- Cut wounds are classified into:
 1. Incised cut wound () : length is greater than depth
 2. Punctured cut wound ()
 3. Stab cut wound : depth is greater than length

- If the presentation of the cut wound is less than 12 hours: close primary but first look/feel for fracture. If more than 12 hours/ infection: granulation
- In case of ischemia and infection do NOT close by tension
- Avulsion is an injury in which a body or a structure is forcibly detached from its insertion and it may be complete or incomplete.
- Scalp Avulsion is most commonly found in women (long hair)
- Scalp avulsion causes a severe PROFUSE bleeding. Best way to stop it is compression.
- Infection is a major complication but is very rare in the face and scalp (hypervascularized area)

Subgaleal (sub-aponeurotic)	Subperiosteal (cephalohematoma)
Vacuum (labor)	Prolonged 2 nd stage of labor, instrumental
Crosses midline Fluctuating Superficial bruising, swelling (12-72 h later)	Does NOT cross midline Solid (firmer) Less extensive

- Subgaleal scalp injury is a clinical diagnosis
- Subgaleal hematoma result from bleeding of emissary veins
- Subgaleal injuries may be associated with other types of brain injuries
- We must NOT interfere with subgaleal hematomas in babies as it may lead to infection
- Treatment is usually conservative in subperiosteal injuries

SKULL INJURIES/ FRACTURES :

They are classified in two ways:

- A. Open vs. Closed
- B. Penetrating vs. Blunt Trauma (cause)

Blunt Skull Injuries are classified into :

A. Linear fractures which are caused from wide blunt skull injury ; it is the most common skull fracture, NO displacement.

Linear fractures may be : at the vault (hairline), diastatic (suture), basilar (base)

Diastatic fractures are usually found in infants and young children, if found in adults it will usually involve the lambdoid suture (closes at 60 years)

B. Depressed fractures which result from small blunt injuries.

They may be open or closed, one piece or comminuted

C. Pond () : not really a fracture, mostly in babies

Does not need any intervention as it will be corrected alone by the ingrowing brain

- There is no need to do anything or intervene in the linear skull fracture unless it is complicated (parallel in close proximity, transverse a suture, involves a venous sinus groove or a vascular channel)
- Basilar skull fractures are rare due to huge amount of energy they need to be caused by
- Basilar skull fracture most important complication is : CSF leak (rhinorrhea, otorrhea) so we always give antibiotics.
- Basilar fractures:
 1. Blood in the sinuses
 2. Raccoon eyes (subconjunctival hemorrhage, periorbital ecchymosis)
 3. Battle's sign (retro auricular ecchymosis); bruising over mastoid process
- Traumatic vs. Hypertensive subconjunctival hemorrhage: you can see the posterior limit of the hemorrhage
- Usual sites for basilar fractures:
 1. Petrous part of temporal bone
 2. Orbital part of frontal bone
 3. Basiooccipital
- Basilar fractures may be an isolated fracture or it may be caused by extensions from fractures in the vault
- Complications of skull fractures:

1. Linear fractures: growing fracture due to pulsations forming a leptomenigeal cyst which is a cystic mass filled with CSF (meninges)
Most common in children less than 3 years. It is a rare complication
 2. Depressed fracture: high risk of epilepsy (15%) in addition to other complications such as increasing the ICP, hemorrhage
 3. Basilar fractures: CSF leak
- Cases requiring surgical intervention (elevation surgery) in depressed skull fracture:
 1. **ALL** Open depressed fracture (compound)
 2. Closed depressed fracture(simple) **IF**:
 - a) the fracture \geq thickness of the skull
 - b) associated with neurological signs
 - c) associated with speech problems
 - d) associated with seizures
 - e) cosmetic
 - In open (compound) fractures presenting less than 12 hours clean and close immediately, in those presenting after more than 12 hours do a craniotomy ,leave for 6 months and then follow by a delayed cranioplasty
 - In the case of comminuted depressed fractures do a craniotomy and an immediate cranioplasty (we do not wait 6 months)
 - The most serious complication of open (compound) fractures is infection.

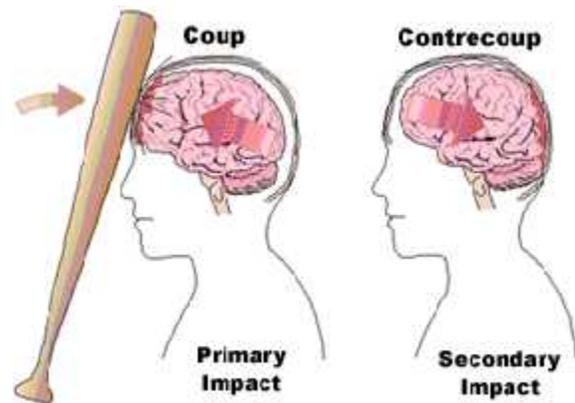
BRAIN INJURIES :

Brain injuries refer to the direct injury of the brain tissue, as not all head injuries are associated with brain tissue injury.

Brain injuries causes may be classified into:

1. Direct brain injury (open or closed)
 2. Accelerating / Decelerating injury (Coup – Contrecoup)
 3. Shearing forces
- Coup – Contrecoup injury: is an injury when the brain is thrust against the skull opposite to the blow side due to sudden movement of the brain inside the skull.

- Coup – Contrecoup injury causes subdural hematoma as well but the main problem is the brain injury not the hemorrhage
- It is more severe in elderly due to brain atrophy and more empty space in the skull



- Shearing forces : is an injury that occurs when the grey matter slides over the white matter in the brain, leading to diffuse axonal injury (DAI) as the nerve cell body usually originate in the grey matter and the axon travel from the grey to the white matter and these shearing forces (sliding) will lead to the damage of those axons (example: boxers)
- It is grossly silent (Normal CT& ICP) but microscopically there are axonal swellings (spheroids)
 - What is the diagnosis of a patient with a 3/15 GCS and a normal CT/ICP? DAI



- Types of 1ry brain injuries include:

1. Concussion

Most common type of 1ry brain injuries

It includes loss of consciousness, amnesia and memory dysfunction

Usually resulting from blunt trauma

The cause is unknown but it may be due to a structural or a physiological cause (most likely it is physiological)

It may be mild (treatment: only observe), severe (anti-inflammatory drugs)

2. Contusion بية

It includes: Bleeding, Brain Edema (edema is what usually kills the patient)

We may find on CT: compressed shifted falx, no ventricles, effacement

If small then use mannitol, if large it needs evacuation

If it persists more than 24 hours it may cause neurological deficits

3. Laceration

Damage is permanent in this case

Not necessarily associated with fractures

- In head injury patient always check for other traumas (multi-trauma patients) and for cervical injuries (spinal) even if the patient is not complaining
- Always do a cervical xray , if you see any abnormality then do a CT , In children do a CT

Assessment of head injury severity :

It depends on the GLASCOW COMA SCALE (glasgow-Scotland)

14-15: mild / 9-13: moderate / ≤8: severe (unconscious)

Criteria for doing a CT in a head injury patient:

Headache	Persistent anterograde amnesia
Vomiting	Age ≥ 60 years
GCS deteriorating	Drugs / alcohol intoxication
Seizure	Depressed fracture
Coma	Visible trauma above clavicle
Confusion more than 2 hour	Unsure / in doubt

Criteria for an admission in a head injury patient:

LOC or change in the level of cons ≥ 5 minutes	Extremes of age (less than 2, more than 65)
Post- traumatic amnesia ≥ 5 minutes	Multi- trauma patient
Vomiting	Skull fracture / CSF leak
Seizure	Abnormal CT
Focal neurological signs	

- Skull , lateral cervical spine chest XRAY are required on admission
- 3 views needs : AP, Lateral, Town's
- In stable patients a 2nd CT is needed 3-6 days later (1 week) and a 3rd CT 10-14 days later (2 weeks)
- NO role for MRI imaging in head injuries unless the injury is chronic

Patients are divided into :

Low risk	Normal CT Asymptomatic or only headache and dizziness	14-15
Moderate risk	CT- hematoma	9-13
High risk	LOC, Focal signs, Penetrating skull injury	Less than 8

- No radiological exam is needed in low risk patients
- Admit patients with moderate and high risk
- TREATMENT :

MILD	Elevation of head Analgesia (codeine sulphate) IV line and drain Any deterioration : repeat CT NO ADMISSION
MODERATE	Mannitol Closer observation No ICP monitoring
SEVERE	ICU admission ICP monitoring Burr hole

THE END

Done by : Lana Daoud

Complications of head injury

Morbidity and mortality of head injury result from 2 causes:

1. Primary head injury (at time of impactation- no benefit of medical or surgical intervention)
2. Secondary head injury (subsequent damage from 1ry insult and the intervention improve the outcome) → preventable

Complications of head injury:

Early complications:

1. Hypoxia.
2. Electrolyte imbalance.
3. Cerebral edema.
4. CSF leak.
5. Pyrexia.
6. Intracranial bleeding.
7. Increased intracranial pressure.

Late complications:

1. Chronic subdural hematoma.
2. Hydrocephalus.
3. Epilepsy.
4. Post-concussion syndrome.
5. Infection.

* starting with early complication:

1. Hypoxia: it's the most common and most preventable causes of patient deterioration after primary injury.

Can be due to (airway obstruction, lung injury, central respiratory depression, ARDS..etc)

management: (ABC, maintain patency of airway, ventilate if GSC=<8)

2.Electrolytes imbalance:

- * after severe head injury * particularly in children
- * most common is Na imbalance
- * Trauma or stress → ADH → volume retention and hyponatremia
- * symptoms= confusion – lethargy – dizziness – nausea – vomiting

- * if Na concentration <120 mmol/l it will lead to seizures and LOC.
- * Don't correct it rapidly bcz this will result in central pontine myelinolysis.

3. Cerebral edema:

- * commonly in children
- * can be: 1. A focal pattern around an intracerebral hematoma following contusion.
2. Diffusely throughout the cerebrum and cerebellum.
- * It can lead to { raised ICP and herniation + decrease LOC }
- * Types (vasogenic – cytotoxic – osmotic – interstitial)
- * ttt → osmotherapy using MANNITOL, Diuretics, surgical decompression.

a. Vasogenic edema:

- it caused by breakdown of BBB.
- Causes → Trauma, tumor, abscess, late stages of cerebral ischemia.
- finger like projection.

b. cytotoxic edema:

- BBB is intact
- result from cellular retention of sodium and water.
- All cells of brain are affected.
- can be seen in (early ischemia, early stroke, cardiac arrest, various intoxications)

Herniation of the brain

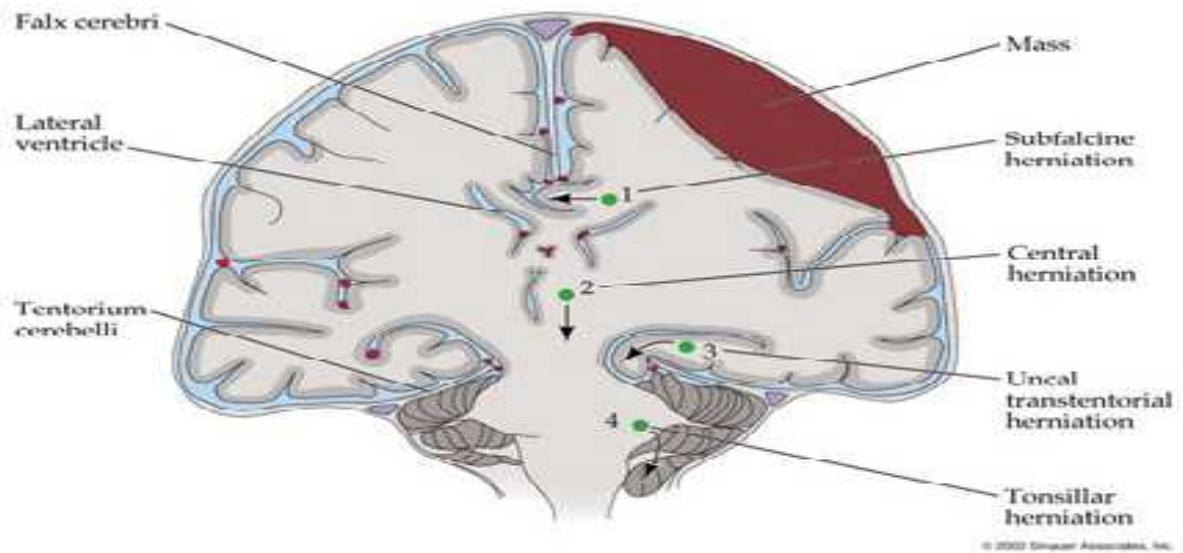
can be divided into:

a. Supra tentorial:

- 1. Uncal (transtentorial):** the inner most part of temporal lobe (uncus) squeezed so much that it moves towards the tentorium and puts pressure on the brainstem, most notably the midbrain the midbrain.
- 2. Central:** the diencephalon and parts of the temporal lobes of both of the cerebral hemispheres are squeezed through a notch in the tentorium cerebelli.
- 3. Cingulate (subfalcine):** the most common type, in which the innermost part of the frontal lobe is scraped under part of the falx cerebri.cerebri.
- 4. Transcalvarial:** the brain squeezes through a fracture or a surgical site in the skull.

b. Infratentorial:

1. Upward
2. Downward (tonsillar)



4. CSF Leak:

a. CSF otorrhea → fracture in petrous bone and damage TM.

b. CSF rhinorrhea

** associated with risk of meningitis (more with otorrhea)

*spontaneously resolve within one week. If not resolve we do 1. Lumbar subarachnoid drain to allow the fistula to close 2. radiographic dye to localize the fistula.

5. pyrexia:

* most commonly due to infxn

* less likely occur due to medication (phenothiazine) or damage in hypothalamus.

**• Bromocriptine has been used and has helped with the hyperthermia.

6. Intracranial bleeding

1. Extradural (epidural) hematoma. 2. Subdural hematoma. 3. Subarachnoid hemorrhage. 4. Intracerebral hematoma.

1. EPIDURAL HEMATOMA	2. SUBDURAL HEMATOMA
Present in 1 st 24 hr.. raised ICP.. LOC post trauma as result of concussion followed by lucid interval the 2ndry decline in consciousness (vomiting and papilloedma)+ fixed dilated pupil ipsilaterally and hemiparesis controlaterally.	The most common intracranial bleeding * Acute (less than 3 days) *subacute(4-21 day) * chronic (more than 3 weeks. {Headache---restlessness—neurological deterioration)
Site = between skull and dura (mostly temporal region)	Site= between the dura and arachnoid
Most commonly arterial 90% (middle meningeal artery) less commonly from dural sinuses 10%.	Mostly it is a venous blood – less likely from cortical artery
Children and young adult	Elderly especially those on anticoagulant
On CT without contrast→ hyper-dense biconvex	On CT without contrast → crescent hyperdense collection.
Mortality about 30%	Mortality about 50%
Treatment by emergent surgery (evacuation) 1. Burr hole 2. Flap craniotomy	Same
• Indications for surgery: 1. Thickness more than that of the skull. 2. Symptomatic. 3. Pediatric.	
Can't cross the suture	Can cross the suture

3. Subarachnoid hemorrhage:

- Subarachnoid hemorrhage can be caused by trauma and often does not require surgery.
- Blood accumulates in the space beneath the arachnoid layer that surrounds the brain
- While this can irritate the brain and cause symptoms of headache, vomiting, and stiff neck, treatment may be watchful waiting without surgery unless symptoms worsen.

4. Intracerebral hematoma:

- Hemorrhage into the brain parenchyma due to areas of traumatic contusion, and the brain tissue usually aggravates it by releasing thromboplastin as a response.
- If the lesion was large and expanding it should be evacuated, otherwise if the patient is improving it may not require evacuation.
- On CT scan it appears as a **hyperdense area with midline shift.**

7. raised ICP:

3-10 days followed the trauma
caused by: cerebral edema—intracranial bleeding – hyponatremia—hypoventilation

NOW → LATE COMPLICATIONS

1. Hydrocephalus:

- * it may develop → a. acutely due to intraventricular hemorrhage and block CSF flow (less common) b. Insidiously, which is the most common.
- Usually its communicating hydrocephalus
- may present as either
 1. Ventriculomegaly with raised ICP (vomiting, headache, visual disturb.)
 2. Normal pressure hydrocephalus → memory problem. Gait ataxia and urinary incontinence.

2. Chronic subdural hematoma:

- * collection of blood break down product.
- * they are **hypodense** on CT scan
- * alcoholic, elderly, anticoagulant are at higher risk.
- * symptoms: headache, seizures, confusion, vomiting
- * indx of surgery is the same of acute subdural hematoma.
- * burr hole is effective.
- * follow up by CT scan for one month.

3. Epilepsy:

- *early (1st 24 hrs), intermediate (1-7 days), late (more than 7 days)
- * mostly with penetrating injury.
- * Mostly in young adult.
- *after trauma neural circuit become hyperexcitable so epilepsy will increase brain metabolism so increase blood flow so raised ICP
- * Risk factors: GSC<10 – hx of alcohol – hematoma – post-traumatic amnesia (>24hr)

- Early and intermediate epilepsy usually respond to prophylactic anticonvulsants and has no prognostic effects, but they don't prevent the occurrence of the late ones.
- These are usually given for 2 weeks.
- Longer periods showed no benefit

4. Post-concussion syndrome:

* no clear etiology (organic or psychological)

* symptoms

Somatic;; headache – dizziness – hearing and visual difficulty

cognitive:: impair memory and concentration

psycho: emotional instability- personal change – insomnia – loss of libido

Best of Luck

-Maher Odeh

Subarachnoid Hemorrhage

- extravasation of blood into the space between pia and arachnoid.
- Many of surviving patients are living with disability.
- 20% of stroke are hemorrhagic, 10% of them SAH and 10% intracerebral hemorrhage.
- Mean age is 55 yo. **But** aneurysmal SAH (40-60 years old)
- Female > male (3:2) ,, , black > white
- Increase risk of SAH in 3rd trimester of pregnancy.
- 15% die at home.
- 15-20% of patients prove negative for a cause.
- 15% multiple
- **Risk Factors:** SMOKING, HTN, Antithrombotic therapy, Statin, Alcohol and estrogen def. , family hx, advanced age.
- **Most common cause of SAH is → TRAUMA**
- Non-traumatic cause:
 1. Rupture of berry aneurysm (70%) → most common non-traumatic SAH
 2. Rupture of AV malformation (10%) → but in children it's the most common.
 3. Less common causes (mycotic aneurysm, infection, neoplasm, vasculitis)
 4. 15% due to unknown cause.
 - * **Aneurysm:** - 3% chance for rupture per a year
 - Risk of surgery → > 3% chance for rupture → don't do surgery
 - m.c locations: Anterior communicating artery > Posterior communicating artery > middle meningeal artery
 - 80% are in ant. Circulation and 20% in post. Circulation.
 - **Types=** 1. Saccular 2. Fusiform 3. Mycotic (m.c.c is staph)
 - Marfan syndrome is not correlated with Berry aneurysm.
 - Risk of rupture = size > 7mm
(cavernous (lowest risk) < anterior < posterior)
- **CLINICAL PRESENTATION:**
 1. **Headache:** {{sudden, severe, "worst headache of my life"}}
 - minor leakage of the aneurysm → sentinel leaks. And leads to Sentinel headache.
 - Sentinel headache occur few hours prior to aneurysmal rupture.

- In AVM there is no sentinel headache

2. Decrease level of consciousness

3. Meningism: (blood irritate the meninges) → neck stiffness, photophobia, headache and fever

4. Focal neurological signs.

5. Optic Fundi → mild papilledema, fundal hemorrhage, retinal hemorrhage

6. orbital pain and diplopia

**** Signs present before SAH include the following:**

- **Sensory or motor disturbance (6%)**
- **Seizures**
- **Ptosis (3~)**
- **Bruits (3%)**

- History: (clinical triad of SAH) →

1. Sudden, severe headache
2. Decrease level of consciousness
3. Meningism

We have 2 clinical scales (hunt and hess + WFNS), and other scale depend on the appearance of SAH on CT scan called 'Fischer'

Table 9.3 Subarachnoid haemorrhage grading systems.

Hunt and Hess grading system*

Grade	Description
1	Asymptomatic, or minimal headache and slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurological deficit (except cranial nerve palsy)
3	Drowsiness, confusion or mild focal deficit
4	Stupor, moderate to severe hemiparesis, possible early decerebrate rigidity and vegetative disturbances
5	Deep coma, decerebrate rigidity, moribund

WFNS grading system

Grade	Glasgow Coma Score (GCS)	Motor deficit
1	15	No deficit except a cranial nerve palsy
2	14-13	No deficit
3	14-13	Any deficit
4	12-7	With or without focal neurodeficit
5	6-3	Coma with or without abnormal posturing

* Serious systemic disease such as hypertension, diabetes, severe arteriosclerosis, chronic pulmonary disease, and vasospasm on angiography result in placement in next less favourable category.

Fischer scale:

Points	Description
0	Unruptured
1	No blood detected
2	Diffuse or vertical layers <1 mm thick
3	Clot and/or vertical layer >1 mm thick
4	Intracerebral or intraventricular clot

Investigations: (depends on high index of clinical suspicion).

1. CT scan without contrast.
2. Lumbar puncture → presence of the blood cells in subarachnoid space.
it should performed after CT scan to exclude significant intracranial mass.
its most sensitive after 12 hrs after bleed.
3. Angiography (CT or MRI) → to know the exact source of hemorrhage.
4. Lab invx → CBC (infx or hemorrhagic problem), PT time, cardiac enzymes, ABG
...

• **Complications:**

1. **Re-bleeding** → re-bleeding SAH is more severe than initial SAH.
It's the most early complication of SAH.
The greatest risk of re-bleeding occur in the 1st 24 hours.
Mortality rate is 78%.
2. **Cerebral vasospasm:** Delayed narrowing of large capacitance vessels at the base of the brain. ☹ occur in 70% of SAH. Occur 4-14 days after hemorrhage.
symptoms → decrease level of consciousness – focal neurological deficit
mostly involve → Terminal internal carotid artery + proximal portion of anterior and middle cerebral artery
*** Angiography should performed to differentiate vasospasm from re-bleed.
3. **Hydrocephalus**
4. **Seizures** (given phenytoin as prophylaxis)
5. **Hyponatraemia**

MANAGEMENT:

1. Patient stabilization, ABC, intubation (if present in coma or high ICP or decr. Level of consciousness). “Now its recommended to use antihypertensive only when MAP>130 mmhg””

2. Patient with signs of raised ICP or herniation should be treated accordingly (intubation, hyperventilation, osmotic agents [mannitol], loop diuretics, IV steroids)

3. Treat and prevent the complication;

* **re-bleeding** → Bedrest, analgesia, stool softeners, antifibrinolytics

* **vasospasm** → oral nimodipine (CCB); subarachnoid clot removal; hypertensive, hypervolemic, and hemodilutional (HHH) therapy; transluminal balloon angioplasty; intra-arterial injection of papaverine.

* **Hydrocephalus** → external ventricular drainage

* **Hyponatremia** → hypertonic sodium chloride, avoid fluid restriction

* **Seizures** → prophylactic anticonvulsants (phenytoin)

4. Surgical management:

• **Direct aneurysmal clipping** is still considered first-line treatment in the *US* → the aneurysmal neck is obliterated via application of a clip that occludes blood flow to the aneurysmal dome without compromising flow to the parent.

• **Guglielmi detachable coil (GDC)** is first-line therapy in *Europe* _It is a form of embolization where the aneurysm is filled with coils of decreasing size until densely packed artery.

• **Balloon embolization** is successful in some patients, but it has a higher incidence of complications than coil embolization.

• Other surgical options include the following:

–**Proximal ligation** of the parent artery or **trapping of aneurysms with or without bypass**

–**Wrapping or coating of aneurysms**(in rare cases of dissecting or fusiform aneurysm)

Notes:

* early and delayed surgery has advantages and disadvantages:

Early → decrease risk of re-bleeding but cause edematous brain tissue or intraoperative aneurysmal rupture

Late→ increase risk of re-bleeding but less edematous and aneurysmal rupture.

• For patients with Hunt and Hess/WFNS grades 1-3, surgical treatment is strongly recommended and has good outcome.

• In patients with a poor grade of SAH (grades 4-5), the outcome is poor, with or without surgical intervention.

• **Complications of surgical clipping include the following:**

- Hemorrhagic complications
- Ischemic complications
- Damage to parent artery or perforating arteries
- Acute or delayed neurological deficits from iatrogenic trauma
- Meningitis
- Cellulitis and wound infection

• **Common complications of endovascular therapy include the following:**

- Aneurysm rupture (GDCs, balloons)
- Thromboembolism (GDCs) with acute or delayed neurologic defici.
- Balloon rupture or deflation

Best of luck

-Maher Odeh

Spina bifida

etiology → 1. Environmental and genetic 2. Teratogenic 3. Nutritional (folic acid def.)

Definition: birth defect resulting from incomplete closure of embryonic neural tube.

Spina bifida: aperta and occulta → (Swelling – Tuft of hair- Redness- Dimple)



- Myelomeningocele = m.c and contain neural elements
- Meningocele= only meninges
- Lipomeningocele = contain lipid
- Spina bifida ventralis

Note → Mongolian spot= Dark blue discoloration in the back of the baby.

A. **Spina bifida occulta:**

- * bony vertebra is open but the spine is within the spinal canal.
- * there is skin discoloration, tuft hair or swelling.
- * may associated with tethering of the spinal cord

B. **Spina bifida aperta:**

1. **Lipomeningocele:**

- * lipoma located over the lumbosacral spine associated with bowel or bladder dysfunction.

2. **Meningocele:**

- * fluid filled sac
- * meninges involved
- * neural tissue un-affected
- * rarest type

3. **Myelomeningocele= spina bifida**

- * protruding of part of the meninges and part or all of spinal cord through opened vertebra.
- * Involved part of spinal cord is often not fully developed.

symptoms →

1. Pain, weakness, paralysis
2. bowel and bladder problems
3. Skin ulceration

Complications:

1. Club foot (m.c)
2. Spinal curvature abnormality (scoliosis or kyphosis)
3. Osteoporosis
4. Hydrocephalus

How spina bifida managed?

1. Detection: Triple screening (maternal alpha fetoprotein + amniocentesis+ US for bone)

- we do MRI to →
1. To know if there is neural elements or not.
 2. Brain MRI (because myelomeningocele → 80% associated with hydrocephalus and the percentage increase to 90% after surgery)

2. Antibiotics

3. Surgery:

two types, the 1st is within the 1st 48 hrs of delivery ((cover the spinal cord by muscle and skin))and 2nd is in uterosurgery (which improve the prognosis).

Benefits of the surgery:

- a. The mother can hold her baby
- b. Cosmetic
- c. fear of rupture and infection

4. Observation

5. Physical therapy (walking and speech)

C. **Spina bifida ventralis:**

- * less common than other
- * the protrusion is into retroperitoneal space
- * affect retroperitoneal organs (duodenum, kidneys, adrenal , aorta, inferior vena cava)

Done by: Maher Odeh

DEGENERATIVE CERVICAL DISEASES

Anatomy

- Atlas is the first cervical vertebra and it has no body; it allows you to nod.
- axis is the second one, acts as a pivot for the rotation (dens is the pivot point)
- thoracic vertebra are larger than cervical and articulates with the ribs and have long spinous processes
- lumbar spines have massive block like body, short spinous processes and are sturdiest.
- sacral vertebrae are fusion of 5 vertebra
 - connect to the hip to form posterior pelvic wall
 - superior to coccyx
 - ends the vertebral canal
- neck vertebrae are very mobile > lumbar > thoracic
- inter-vertebral disc
 - pads of flexible fibro-cartilage
 - function:
 - cushion vertebrae
 - absorb shocks
 - allow for spine flexibility
 - composed of:
 - annulus fibrosus (fibrous)
 - nucleus pulposus (gelatinous—80% water)
 - structural changes with age
 - young—90% water making it spongy and flexible
 - old age less water, harder more susceptibility to herniate
- ligaments
 - anterior and posterior longitudinal ligaments
 - inter-spinous ligament
 - supraspinous ligament
 - intertransverse ligament
 - ligamentum flavum
- curvatures
 - C-shaped in infant and with age becomes S shaped
 - Scoliosis: side-ways curvature
 - Lordosis: inward curve of the lumbar spine
 - Kyphosis: outward of the thoracic spine

Note: the question about this lecture → he will give you a case scenario about a patient who has weakness in a certain muscle group and absence of one of the reflexes and you should know the nerve root that has been affected (where is the compression).

Degenerative cervical Disc Diseases

- loss of normal structure and function in the spine
- pressure on spinal cord or nerve root could rise by
 - o disc displacement
 - o disc herniation
 - o spinal stenosis
 - o osteoarthritis
 - o cartilage breakdown
- prolapsed disc stages: bulge, protrusion, herniation extruded disc)
- {proteoglycans, water, non-collagenous protein} → all decreased with age but collages increases with age
- cascade of degenerative changes causes dysfunction, instability and then re-stabilization
- cervical disc disorders
 - o herniated nucleus pulposus
 - o degenerative disc disease
 - o internal disc disruption
- herniation
 - o herniation occurs secondary to posterolateral annular stress
 - o herniation is either focal 35% or broad based 25-50%
 - o risk factors
 - age related degeneration
 - lifestyle
 - genetics
 - smoking
 - nutrition
 - physical activity
 - note: the nucleus pulposus appears to be the first site of degeneration while the annulus fibrosis being the primary pain generator once injury or degeneration occurs
 - most commonly: c6/c7 > c5/c6/ c4-c5 / c7-t1
 - o radiculopathy
 - lateral prolapse toward the nerve root
 - o myelopathy: central prolapse compressing nerve root, spinal cord, the anterior spinal artery and vein
 - o examples
 - C5/C6 prolapse (C6 nerve root)

- Motor: mild weakness of elbow extension
 - Sensory: numbness in the thumb or index finger
 - Reflex: depressed supinator reflex
- C6/C7 prolapse (C7 nerve root)
 - Motor: weakness of elbow extension
 - Sensory: numbness in the middle or index finger
 - Reflex: absence triceps jerk
- C7/T1 Prolapse (C8 nerve root)
 - Motor: weakness may involve long flexor muscles, triceps, finger extension or small hand muscles
 - Sensory: decreased sensation in ring and middle finger and medial side of the hand and forearm
 - Reflex: triceps jerk may be depressed
- Examination
 - Spurling maneuver: patient's neck is extended, laterally bend toward site of pain in seated position will produce radiculopathy
 - Shoulder abduction sign: active abduction of symptomatic arm placing the patient's hand on head in seated position decreases pain and symptoms
 - Neck distraction: axial traction forces on the neck in supine position pulling it upward by force of about 10-15 kg
- Investigations: x-ray, post-myelogram CT and MRI being the most important
- Conservative treatment:
 - Bed rest
 - Cervical collar
 - Analgesia
 - Muscle relaxant
 - Physiotherapy
 - Avoid extension and lifting heavy objects
 - Note: manipulation for the neck is hazardous and contraindicated
- Usually you give the patient a trial of conservative treatment except in these cases, you go immediately for surgery
 - Significant weakness
 - Evidence of central disc prolapse
 - Pain not resolving with conservative treatment for more than 10 days or chronic/ relapsing pain
- Surgery:
 - Anterior cervical discectomy with fusion
 - Poster cervical fusion (cervical spondylotic myelopathy)

THORACIC DISC DISEASE

- least common due to decreased mobility and increased stability of the vertebra
- accounts of only (0.25-0.75) of all disc herniation
- causes are the same for cervical
- it's self-limiting and rarely requires surgical intervention

LUMBAR DISC DISEASE

- dryness of nucleus pulposus will lead to herniation
- SCATICA
 - o definition: neuralgia along the course of the sciatic nerve (L4 to S3)
 - o 1-10% of the population
 - o back pain is usually self-limited
 - o female = male
 - o MC below 25-45 year old
 - o MC site of herniation L5-S1
- Risk factors
 - o Age
 - o Activity
 - o Sedentary lifestyle
 - o Smoking obesity
 - o Vibration
 - o Psychosocial factors
- Herniation caused by: mostly trauma 80% and degeneration 20%
- Most common direction of herniation is posterolateral
- Examples
 - o L3/L4 (L4 Root)
 - Motor: weakness of quadriceps
 - Sensory: decrease sensation over anterior thigh and medial aspect of lower leg
 - Reflex: decreased or absent knee jerk
 - o L4/L5 (L5 root)
 - Motor: weak dorsiflexion
 - Sensory: parasthesia on dorsum of the foot and great toe
 - Reflex: decreased or absent medial hamstring reflex
 - o L5/S1 (S1 root)
 - Motor: weak plantar flexion

- Sensory: parasthesia in lateral root
- Reflex absent ankle jerk

CAUDA EQUINA SYNDROME

- compression of lumbosacral nerve roots below conus medularis (below L2)
- presentation
 - low back pain
 - bowel and bladder dysfunction
 - numbness in saddle area
 - weakness
 - sexual dysfunction
 - foot drop
 - absent ankle jerks on both sides
- physical
 - straight leg raising test
 - femoral nerve stretch test
 - naffziger test
- investigations
 - x-ray
 - MRI: excellent for lumbar disc herniation and scarf formation
 - CT scan: for evaluating patients with severely spondylitic changes
- Management (for lumbar)
 - You start with a trial of conservative therapy except those with cauda equina syndrome
 - The same as that of the cervical
- Surgical indications
 - Cauda equina
 - Progressive neurological deficit during a period of observation
 - Persistent bothersome sciatica
 - Pain despite conservative management or a period of 6-12 weeks
- Procedure
 - Fenestration
 - laminectomy
 - Standard of care is lumbo micro-discectomy
 - Percutaneous automated discectomy

LUMBAR SPONGYLOSIS

- degenerative arthritis and osteophyte formation (most commonly anteriorly)
- clinical presentation: no symptoms
- no investigations needed or treatment unless complicated
- incidental finding

LUMBAR SPONDYLOLYSIS AND SPONDYLSTHESIS

- spondylolysis: defect in pars interarticularis
- spondylolisthesis: when spondylolysis is accompanied with forward translation of one vertebrae relative to another
- classification
 - o I: congenital: sacral-facet dysplasia
 - o II: isthmic: stress fracture of the pars inter-articularis
 - o III: degenerative: intersegmental instability (produced by facet arthropathy)
 - o IV: traumatic: acute stress
 - o V: pathologic --bone disease
- Grading
 - o I: 1-25%
 - o II: 26-50%
 - o III: 51-75%
 - o IV: 76-100%
 - o V: >100%
- Treatment
 - o Physical therapy (rehabilitation and medication)
 - o Surgical therapy (lumbar spine fusion surgery)
 - o When to treat:
 - Accompanying neuro deficits
 - Persistent back pain after conservative treatment
 - Slippage > Grade II (50%)
 - Traumatic spondylolysis

LUMBAR STENOSIS

- part of the aging process that could be primary or secondary
- primary is congenital and secondary is degenerative
- usually occurs in cervical and lumbar spine more than others
- clinically:
 - o lower back pain, radiating leg pain, possible bladder or bowel difficulties

- neurogenic claudication (pain associated with walking relieved by sitting or leaning forward)
- rarely, cauda equina
- treatment:
 - laminectomy
 - posterior laminotomy
- component of lumbar canal stenosis
 - osteophyte formation
 - facet hypertrophy
 - diffuse bulging disc
 - hypertrophy of ligamentum flavum
- What is lateral recess stenosis? Narrowing reduces the available space within the exit forway (foramen) of the spinal canal

Table 6
Clinical differentiation between neurogenic claudication and vascular claudication

Description	Neurogenic claudication	Vascular claudication
Quality of pain	Cramping	Burning, cramping
Low back pain	Frequently present	Absent
Sensory symptoms	Frequently present	Absent
Muscle weakness	Frequently present	Absent
Reflex changes	Frequently present	Absent
Arterial pulses	Normal	Decreased or absent
Arterial bruits	Absent	Frequently present
Skin/dystrophic changes (cyanosis, hair loss, and so forth)	Absent	Frequently present
Aggravating factors	Erect posture, ambulation extension of spine	Any leg exercise
Relieving factors	Sitting, bending forward squatting	Rest
Walking uphill	Symptoms produced later	Symptoms produced earlier
Walking downhill	Symptoms produced earlier	Symptoms produced later
Bicycle test	No symptoms provoked unless erect	Provokes symptoms

Table 7
 Comparison of spinal stenosis with disc herniation

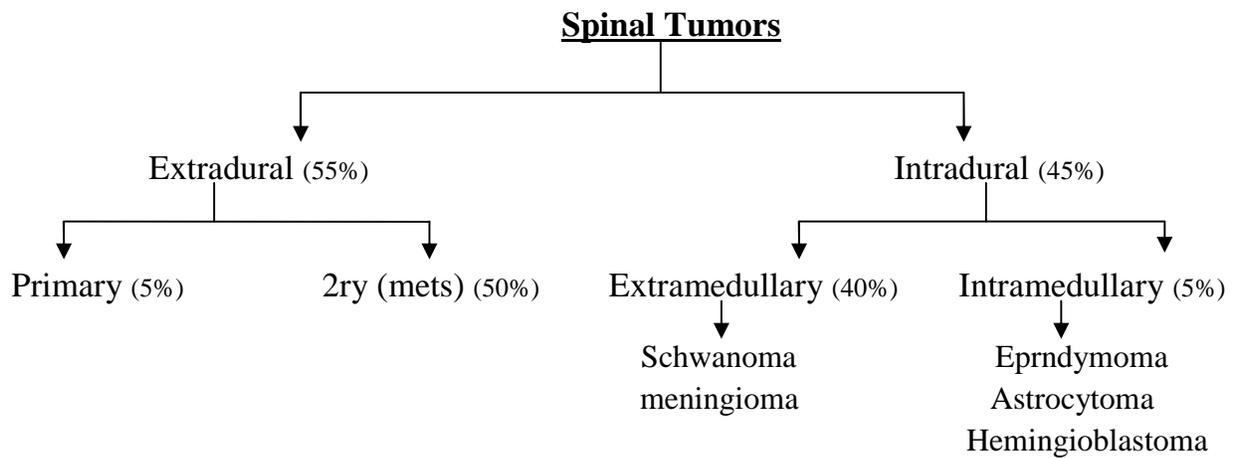
Description	Stenosis	Disc herniation
Age	Usually >50 years	Usually <50 years
Onset	Insidious	Sudden
Positional changes		
Sitting (flexion)	Better	Worse
Extension	Worse	Better
Focal motor weakness	Less common	Common
Dural tension signs	Less common	Common
Focal muscle stretch	Less common	Common
Reflex changes		

Done by: Mohammed Raed

Spinal Tumors

Spinal cord:

- * Connection between brain and periphery of the body.
- * White matter periphery, gray matter inside, CSF- filled canal in the center.
- * Same layers of meninges as the brain (dura, arachnoid, pia)
- * Sheath of the canal is the ependymal.
- * Cord until L1,L2 then we have the filum terminale (ependyma, pia, but not cord)
- * Filum terminale ends at S1.
- * Dura ends at S2
- * Denticulate ligament is a series of thin connections exiting from the pia through the arachnoid, and attaches to the dura. These ligaments “centralize” the cord in its place. When we want to remove a tumor, we have to cut through the denticulate ligaments.
- * Roots exit below the disc (except in the cervical region)
- * Ligamentum flavum between the two vertebrae.



- ❖ Malignant tumors are the commonest, of those:
 - 50% are mets
- ❖ Of all primary tumors 1-2% are malignant.

Presentation:

Presentation: symptoms of nerve or cord compression and loss of function due to:

1. Direct compression by the tumor.
2. Or instability and destruction of the vertebrae.

First presenting symptom is Pain:

It's a night pain (due to decrease cortisol and CO₂ retention during the night)

The location of pain strongly correlated to the site of the tumor.

DDx: Disc prolapse.

Differentiated by enhancement on imaging.

The most important single prognostic factor is the ambulatory state at presentation

Imaging:

1. Signs on plain x-ray:

- a. General signs of bone destruction, deformity (scoliosis)
- b. Mass
- c. Increase intervertebral foramen size (dumbbell → (like in schwannoma))
- d. Increase interpedicular distance (normal 22-25 mm)
- e. Scalloping of the vertebrae.

2. CT scan:

- to differentiate between extramedullary Vs. Intramedullary
- cystic enhancement lesion → Hemangioblastoma

3. MRI:

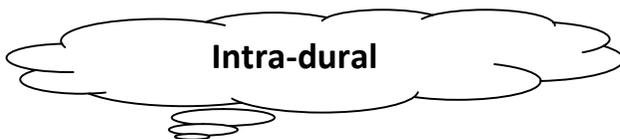
- The most important

4. Lumbar puncture:

- increase protein in 95% of intramedullary tumors (esp. ependymoma)

5. Bone Scan

Now the details about the types of spinal tumors:



a. Intradural Intramedullary:

- * primary is uncommon , mets are rare
- * most are gliomas, and most of them are ependymoma.
- * Ependymoma > Astrocytoma is spinal cord (the opposite in brain tumors)

1. Ependymoma:

- ❖ Most common primary (65%)
- ❖ Types:
 - Myxopapillary → Mostly in filum terminale
 - Anaplastic → very rare and very malignant
- ❖ 30- 40 with male predilection

- ❖ Separated from the spinal cord by a plane of cleavage → allows complete resection (sausage-like).
- ❖ Tx: Surgical resection and no role of radiotherapy.

2. Astrocytoma:

- ❖ 2nd – 5th decades
- ❖ Male > female
- ❖ The most common intramedullary tumors in peds.
- ❖ Thoracic > cervical
- ❖ 4 types
- ❖ Pilocytic type – almost benign
- ❖ 1/3 are malignant (glioblastoma-multiforme “GBM” and anaplastic)
- ❖ Less enhancing than ependymoma
- ❖ Prognosis depends on the grade.

3. Hemangioblastoma:

- ❖ Benign
- ❖ Uncommon (2% of all spinal cord tumors)
- ❖ 5% in VHL syndrome
- ❖ Well circumscribed but not capsulated.
- ❖ Intramedullary > extramedullary
- ❖ Associated with polycythemia
- ❖ If it hemorrhages → presents acutely

4. Intramedullary mets: are very very rare!

b. Intradural Extramedullary:

1. Spinal Meningioma:

- ❖ Middle age (30-50), Female > male
- ❖ Chronic slowly growing tumor eroding the bone and compressing the SC.
- ❖ Mostly in thoracic spine (80%) >upper cervical>foramen magnum.
- ❖ Typical patient: elderly lady with dorsal pain over years associated with weakness progressing over months + sensory level.
- ❖ Adherent to spinal dura → base attached to the dura give the appearance of dural tail on MRI (pathognomic)
- ❖ Associated with Neurofibromatosis.
- ❖ On x-ray → bone erosion of the pedicles and **NO** hyperostosis (unlike meningioma in the brain)

2. Neural sheath tumor (NST):

- ❖ Slowly growing tumors
- ❖ Common in low thoracic and upper lumbar.
- ❖ Adult 20-50
- ❖ Erodes the bony foramina
- ❖ 2 types: neurofibroma (extends into the nerve sheath, infiltrate, with NF I, multiple) and Schwannoma (surrounds the nerve sheath without internal extension, with NF II, solitary)
- ❖ Most are indural, but can occur extradurally which is very rare!

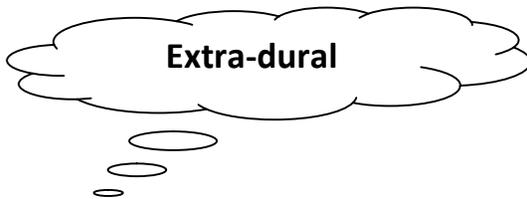
Meningioma Vs NST

- Meningioma compresses the cord → myelopathy.

- NST compresses the nerve root → radiculopathy, But later it may affect the cord leading to → myeloradiculopathy.

3. Filumterminale tumor:

- ❖ 4th – 5th decade
- ❖ M > F
- ❖ Mostly in the prox. portion.



- ❖ Primary tumors arise from any structure; bone, BM, nerve root, soft tissue.
- ❖ Mets are the most common.
- ❖ Mets are mostly from Lungs > breast/prostate > kidney > Lymphoma > Thyroid.
- ❖ Most common site is thoracic region
- ❖ Metastasis is mainly hematogenous.

Water shed area of the spinal cord:

1. Between the blood supply coming from superiorly (ant. and post. Vertebral arteries) and that coming from inferior.
2. At T4 level.

General Notes:

- ❖ Presentation depends on the affected level.
- ❖ Intradural extramedullary tumors:
 - hydrocephalus and increase ICP → mostly in the upper cervical tumors

- Asymmetry in early S&S.
 - Weakness begins distally
 - Sensory ataxia
 - Sphincters are affected LATE.
 - Pain esp. in filum terminale → increase with recumbency, neck flexion, movement and relieved by paracetamol!
- ❖ Mets: 15% present paraplegic, 70% have weakness at dx.
- ➔ Cord pain: dull/aching
- ➔ Radicular pain; electrical/ radiating

Treatment:

All are treated by surgery: “laminectomy + (excision vs. debulking)”
 If the vertebra is involved: “vertebrectomy + fusion”

Spinal tumors Notes:

- ☞ Chronic loss of neurological doesn't mean lifelong deficit while acute ones usually causes that.
- ☞ Disc and dura resist metastasis, so if you see two damaged vertebrae and an intact disc in between, this is mets.
- ☞ The pain of the mets is usually described as boring pain increased at night (because of low cortisol level). Remember that cortisol decreases the edema so when its low, the edema is more, so as the pain.
- ☞ In schwannoma, the management is complete excision but if this is not possible, you don't have to do complete excision, just remove as much as you can and leave the rest as its very slowly growing tumor.
- ☞ There is no role for radiotherapy in schwannoma.
- ☞ Schwannoma causes radicular pain.
- ☞ While meningioma causes ill-defined type.
- ☞ Meningioma is associated with neurofibromatosis II while schwannoma with I.
- ☞ There's no role for radiotherapy in meningioma
- ☞ Spinal ependymoma is common in adults while cranial type is common in children.

Best of luck

- Maher Odeh

Brain tumors I

Primary Brain Tumors:

Introduction:

To understand the primary brain tumors we have to cover first the main cells in the brain that give rise to the tumor.

1. **Astrocytes:** they are the most common tumor-giving cells in the brain, whether in children (pilocytic astrocytoma) or adults (Glioblastoma Multiforme).
2. **Oligodendrocytes:** give oligodendroglioma which arises in adult.
3. **Ependymal cells:** which lineup the ventricles, and give rise to ependymoma that happens in children.
4. **Meninges:** they give meningioma, which almost exclusively occurs in women. Meningiomas are the most common **benign** CNS tumors in adult.

Astrocytes and oligodendrocytes, together, are called the supportive neural tissue, or simply, Glial cells. **Gliomas** constitute the majority of primary brain tumors.

5. **Neuronal cells** → these have the potential to give **medulloblastoma**.

	Children	Adult
Astrocytes	PCA (m.c benign tumor in children)	GBM (m.c adult cancer)
Oligodendrocytes	-	ODG
Ependymal cells	Ependymoma	-
Neurons	Medulloblastoma	-
Meninges	-	Meningioma

- In primary brain tumors, there are some major differences between children and adults. In adults, tumors most often are supratentorial, while in children they are infratentorial.
- The presentation generally doesn't differentiate between brain tumors, as they all can present with features of a space-occupying lesion.
- Previously we had an old classification of primary brain tumors based on "Grade" of the tumor, dividing it into grade 1 through 4. Currently, we consider grade 1

and 2 under one title called “low grade” tumors, and grade 3 and 4 under “high grade” tumors.

- The major differences between those two are:
 1. Low grade tumors tend to have distinct margins (capsule), high grade tumors don't
 2. Low grade tend to have low vascularization, if ever vascular, unlike high grade.

Now Notes about each type

The most common brain tumors are neuroepithelial (astrocytoma, ependymoma, medulloblastoma, oligodendroglioma) and the mc type of neuroepithelial tumors → glioma

1. Astrocytoma:

- a. 45% of primary brain tumors
- b. occipital region rare, other areas are equal.
- c. Grading based on histopathology:
 - grade I → pilocytic (high cellularity- doesn't upgrade, good prognosis)
 - grade II → low grade (pleomorphism)
 - grade III → anaplastic (mitosis)
 - grade IV → Glioblastoma Multiforme (necrosis and hemorrhage)
- d. The presence of mitotic activity suggest high grade lesion.
- e. Low grade in brainstem is fatal.
- f. High grade usually supratentorial, Low grade subcortical.
- g. Histological → not capsulated, calcium deposition, low grade would be avascular.
- h. Glioblastoma (grade IV) → shows an enhancement on CT which indicates active tumor, palisaded necrosis typical of this type.
- I. signs and symptoms of raised ICP, epilepsy, focal deficits.
- J. On CT scan:
 - a. Low grade:
 1. Hypodense (black)
 2. Edema surrounding tumor.
 3. Calcification
 4. Intact BBB
 - b. High grade:
 1. Larger
 2. Marked edema
 3. Enhancement

- 4. BBB distorted
- K. MRI though is more specific.
- L. Life expectancy in grade IV is less than a year.
- M. Treatment options:
 - i. Aims of surgery: biopsy, determine tumor size and decrease it.
 - ii. If it's superficial → depulk
 - iii. Radiation of two types: conventional or stereotactic (gamma)
 - iv. Chemotherapy doesn't cross BBB (Telmazolamide may be used)
 - v. other options → hyperthermia, immunotherapy and phototherapy

2. Oligodendroglioma:

- a. 5th decade of life.
- b. 5% of all gliomas
- c. Most common to present with seizure.
- d. The most common calcifying primary tumor
- e. better outcome.

3. Ependymoma:

- a. The most common site in children → is the 4th ventricle.
- b. The most common site in adult → is the lateral ventricle.
- c. has so much things in common with medulloblastoma (site, presentation, appearance) but is less chemosensitive.
- d. Two categories:
 - i. Anaplastic
 - ii. Non-anaplastic → papillary, myxopapillary and subependymoma.
 - * Anaplastic and papillary are most symptomatic.
- e. Treatment:
 - i. surgical resection → seeding is an issue therefore radiotherapy used post. Op.

4. Medulloblastoma:

- a. Usually midline, in vermis.
- b. more malignant than ependymoma
- c. highly chemo-sensitive and radio-sensitive → So it has better prognosis than ependymoma.
- d. 4th ventricle involvement → hydrocephalus, truncal ataxia, CNS deficit.
- e. spread either through CSF seeding which is common or hematogenous spread.

5. Meningiomas:

- a. these with acoustic schwannoma and pituitary adenoma have a **female predominance**.
- b. Extra-axial tumor
- c. RF: radiation, trauma, neurofibromatosis II, sex hormones.
- d. locations: parasagittal, suprasellar, olfactory groove, cavernous sinus.
- e. Grading depends on position not histopathological ID.
Grade II considered atypical and III is the malignant from the criteria is to have invasion to the brain.
- f. In sphenoidal ridge → Fooster Kennedy syndrome (optic atrophy and contralateral papilledma)
- g. Recurrence if → Incomplete resection
- h. surgery aim → total resection with meninges according to Simpson grading
 - Grade 0: recurrence 0% → remove tumor with 3cm of dura free margin.
 - Grade 1: total resection with dura.

6. Pituitary adenoma:

- a. either functioning or non-functioning
- b. age of presentation → 20-34
- c. usually benign
- d. clinical: compression, apoplexy (hemorrhage), hormonal effects.
- e. mass effects: headache, visual disturbances.
- f. endocrine effect either hyper-secretion or hypo-secretion.
- g. hyper-secretion → prolactinoma is the most common.
- h. hypo-secretion → usually with large tumors and GH/LH/FSH are the first to be affected but PRL is most resistance.
- i. Dx → MRI – CT – x-ray – hormonal assay

NOTES:

* **female predominance tumors:**

- a. acoustic schwannoma
- b. pituitary adenoma
- c. meningioma

*** Drop metastasis (seeding):**

- a. ependymoma
- b. medulloblastoma
- c. glioblastoma

The Most common:

- m.c brain tumors → mets
- m.c primary brain tumors → those of astrocytes (PCA vs GBM)
- m.c benign brain tumor in adults → meningioma
- the pediatric primary brain tumor → PCA
- m.c calcifying brain tumors is → oligodendroglioma
- m.c 4th ventricle- involving tumor → ependymoma
- Exclusively common in female → meningioma

Best of luck

-Maher Odeh

WHO classification of the brain tumors

Neuroepithelial tumors:

- Astrocytomas:
 - All grade I/II except anaplastic (III) and glioblastoma (IV)
 - Glioblastoma has two subtypes: giant cell glioblastoma and gliosarcoma.
- Oligodendroglial tumors:
 - Grade II unless anaplastic, grade III
- Oligoastrocytic:
 - Grade II unless anaplastic, grade III
- Ependymal:
 - Subependymoma and myxopapillary are grade I.
 - Ependymoma → grade II
 - Anaplastic → grade III
- Choroid plexus:
 - One in each grade:
 - I: choroid plexus papilloma.
 - II: atypical choroid plexus papilloma.
 - III: choroid plexus carcinoma.
- Other neuroepithelial tumors:
 - Astroblastoma and angiocentric glioma → grade I
 - Choroid glioma of the 3rd ventricle → grade II
- Neuronal and mixed Neuronal-Glial tumors:
 - Rosette-forming glioneuronal tumor of the 4th ventricle, ganglioglioma, gangliocytoma, desmoplastic infantile astrocytoma/ganglioglioma, dysembryoplastic neuroepithelial tumor, paraganglioma → Grade I
 - Central neurocytoma, cerebellar liponeurocytoma, extraventricular neurocytoma → Grade II
 - Anaplastic ganglioglioma → grade III
- Tumors of the pineal region:
 - Mixed grade, I don't think they'll ask about it.
- Embryonal tumors → All grade IV.
 - Medulloblastoma → grade IV ALWAYS.
 - Either with extensive nodularity or anaplastic.
 - CNS primitive neuroectodermal tumor.
 - Atypical teratoid/rhabdoid tumor

- Tumors of Cranial and Paraspinal Nerves:
 - Schwannoma and Neurofibroma → Grade I
 - Perineuroma → grade I, II, III.
 - Malignant peripheral nerve sheath tumor, any grade except grade I.
- Tumors of the meninges:
 - Meningiothelial cells
 - Meningioma I
 - Atypical II
 - Anaplastic III
 - Mesenchymal: mostly grade 0
 - Lipoma, angiolioma, solitary fibrous tumor, leiomyoma, rhabdomyoma, chondroma, osteoma, hemangioma, osteochondroma → **Grade 0**
 - Rhabdomyosarcoma, anaplastic, Kaposi, Ewing → grade 3
 - Primary melanocytic lesions:
 - Two grade III → malignant melanoma, meningeal melanomatosis.
 - Diffuse melanocytosis → grade 0
 - Melanocytoma → Grade 1
- Tumors of the Hematopoietic system:
 - All grade III: malignant lymphoma, plasmocytoma and granulocytic sarcoma.
- Germ cell Tumors: All Grade III except teratoma (grade I)
 - Germinoma, embryonal carcinoma, yolk sac, choriocarcinoma, mixed germ cell.
- Tumors of Sellar region: (All grade I)
 - Pituicytoma
 - Granular cell tumor
 - Craniopharyngioma
 - Spindle cell oncocytoma of the adenohypophysis.

Done by: Shaimaa Shahin

INCREASED INTRACRANIAL PRESSURE

Before starting to talk about increased intracranial pressure (ICP) we have to be familiar with the normal intracranial pressure.

CSF value = Less than 200 cmH₂O is considered normal

The production of CSF = 500ml/day , 150 ml is in your brain at all times (average adult) , so we can conclude that CSF is changed 3 times/day (500/150)

Determinants of the ICP include components found inside the skull:

1. CSF
2. BLOOD
3. BRAIN TISSUE

$$\text{mmHg} \times 13.6 = \text{cmH}_2\text{O}$$

According to the Monro-Kellie hypothesis if any of those components increase, the other must decrease in order to maintain a normal ICP at all times.

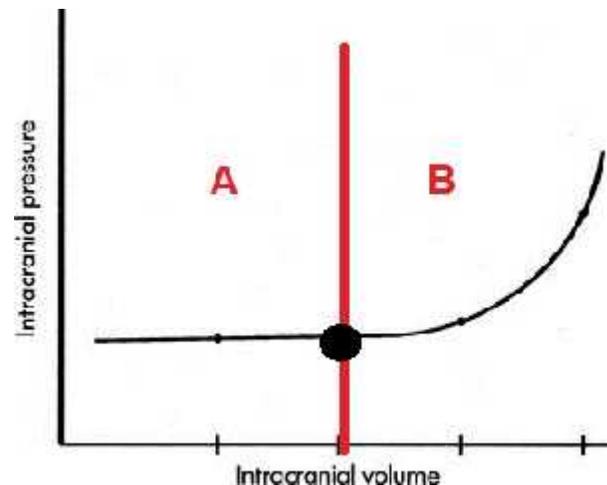
Assuming an increased ICP, the body starts compensating for this increase by a decrease in the other components. It starts by decreasing the CSF around the brain, followed by venous blood along the skull (rigid) and finally by pushing the brain tissue outside the skull (herniation)

1. CSF
2. VENOUS BLOOD (ALONG THE SKULL)
3. BRAIN TISSUE (HERNIATION)

This curve demonstrates the relationship between the rise in intracranial volume (CSF,BLOOD,BRAIN TISSUE) and the rise in intracranial pressure.

We can notice that for a certain point or for a certain rise in intracranial volume, the intracranial pressure isn't affected much (by means of compensating according to the Monro-Kellie hypothesis we talked abt before).

Reaching a certain point (point of discrimination) an exponential rise in ICP occurs with every rise in the volume. Its an exponential relationship and not a linear one.



- Phase A : compensating, compliant, compliance
- Phase B: non-compensating, non-compliant, elasticity
- Beyond point of discrimination: derranged cerebrovascular reactivity

Now, we're gonna talk about another important subject before starting talking about increased ICP (MAP, CPR, CBF) :

MAP (mean arterial pressure) = 1/3 systolic + 2/3 diastolic as we know it, but for making things easier

We are gonna assume that **MAP= (systolic + diastolic)/2**

CPP (cerebral perfusion pressure) = MAP – ICP

- So for a normal CPP value (60-120) we need a normal MAP (pump/heart & blood) and a normal ICP
- CPP values less than 60 indicate an ischemic problem

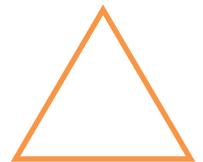
CBF (cerebral blood flow) = CPP/ CVR (cerebral vascular resistance)

- Ventilators control the CO2 levels , causing vasodilation and decreasing the CVR which increases the CBF

INCREASED INTRACRANIAL PRESSURE:

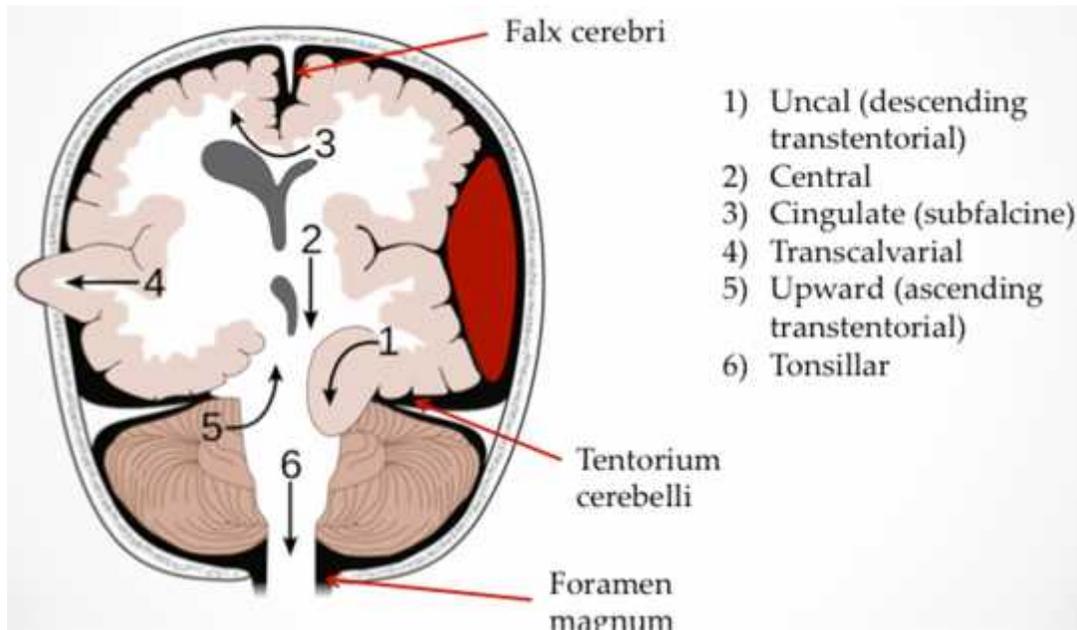
When talking about increased intracranial pressure we are talking about a triad including ONLY:

1. HEADACHE
2. PAPILLEDEMA
3. VOMITING



When talking about S&Sx of increased intracranial pressure we are talking about :

1. Increased intracranial pressure triad
 2. Herniation
 3. Tumor or lesion (pressure symptoms)
- H/A is caused by traction of blood vessels and compression of dura at base of cranium
 - Morning H/A is a characteristic and is caused by : hypoventilation (sleep), cortisol levels (increased at early morning – circadian rhythm)
 - Papilledema alone is NOT a characteristic for an increased ICP as it may be also seen in retinopathies
 - Morning H/A and vomiting are also found in migraine (ddx)
 - Optic neuritis : H/A , papilledema, unilateral (ddx)
 - There are many types of herniation as shown below :



- Tonsillar herniation is the most dangerous type and is caused by herniation of cerebellar tonsils, which pushes the medulla oblongata leading to a CUSHING TRIAD :
 1. Increased blood pressure (hypertension)
 2. Bradycardia
 3. **CHEYNE STOKE** breathing (increasingly deep respirations followed by a steady diminution of breathing until an apneic episode occurs)
- Uncal/transtentorial herniation causes:
 1. Loss of consciousness (compression of RAS)
 2. Ipsilateral mydriasis (compression of ipsilateral CN3)
 3. Contralateral homo. hemianopia (compression of ipsilateral posterior cerebral artery - PCA)
 4. Contralateral hemiparesis (compression of ipsilateral crus cerebri)
 5. Contralateral mydriasis (compression of contralateral CN3)
 6. Ipsilateral homo. hemianopia (compression of contralateral PCA)
 7. Ipsilateral hemiparesis (compression of contralateral crus cerebri)
- Kernohan notch /phenomenon: is known as an indentation in the contralateral cerebral crus by the tentorium cerebe. Signs of compression of the contralateral side of the injury are more obvious :
 1. Contralateral mydriasis

2. Ipsilateral homonymous hemianopia
 3. Ipsilateral hemiparesis
- We may also see a 6th nerve palsy which is called a false localizing sign. The cause is unknown but it may be due to its long intracranial course
 - Transcalvarial herniation : needs craniotomy

DIAGNOSIS AND TREATMENT

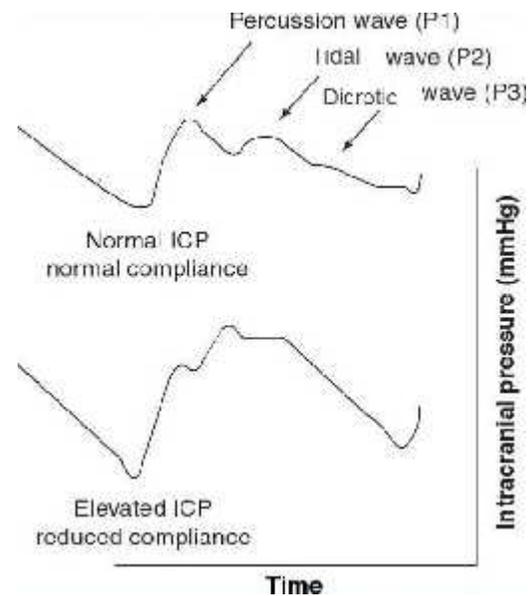
Lumbar puncture (LP) is a diagnostic method and a treatment at the same time. But always make sure to do a CT in order to rule out an intracranial mass before doing a LP as it is a contraindication. In the past when there was no CT available, the doctors used to check for papilloedema before performing a LP in order to rule out an intracranial mass

Fröin syndrome: yellowish CSF caused by increased protein content (more than 40 mg) and it usually indicates obstruction

Intraventricular measuring is the GOLDEN STANDARD for diagnosing increased ICP (at the level of foramen of Monro)

Those are the ICP waveform known as LUNDBERG waves seen in intraventricular measuring. They are 3 waves (exam question) : percussion (P1), tidal (P2), and dicrotic (P3).

P1 is always the highest wave followed by P2 and finally P3 in the normal ICP waveform. In case of elevated ICP this changes.



Changes in the Brain XRAY include:

1. Widened sutures (children)
2. Thumb impressions – Beaten copper/silver (gyrus impressions on the skull due to pulsations)
3. Decrease in the post-clinoid processes
4. Increase in the sella turcica

Changes in the Brain CT include :

1. We may see a lesion (brain tumor/edema/..)
2. Small, shifted ventricles
3. Effacement (brain tissue directly on the skull ; no CSF)

MOST IMPORTANT thing when it comes to the treatment of elevated ICP is treating the primary cause

If we couldn't decrease ICP in all means we do a decompressive craniotomy.

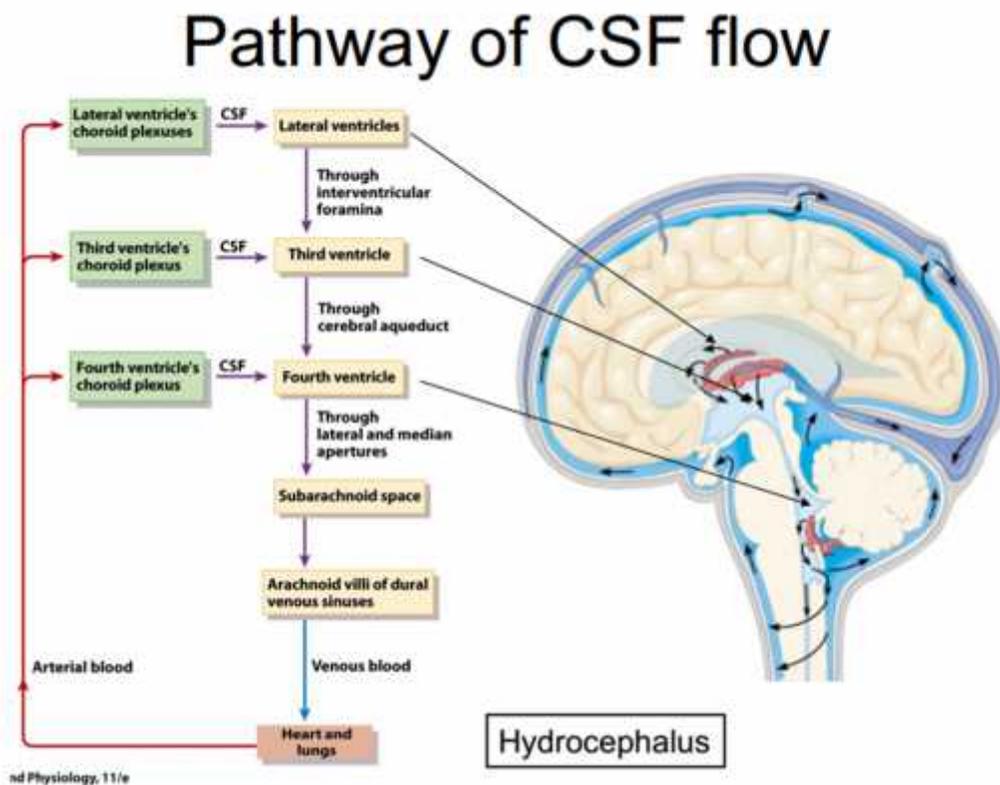
- Idiopathic intracranial hypertension (IIH) is considered the purest form of increased ICP. When doing a CT (normal +/- small ventricles), CSF (normal +/- low protein)
We treat it by treating obesity (decreasing the BMI) or Lumbar Puncture(LP) according to the severity
- Lumboperitoneal shunt is a continuous LP
- Optic sheath fenestration is another way for treatment
- Benign intracranial hypertension (BIH); tortuous optic nerve

THE END
DONE BY : LANA DAOUD

HYDROCEPHALUS

We're going to start talking about CSF in general, its production, tract and finally drainage before talking about hydrocephalus

- The ventricular system is a set of four interconnected cavities (ventricles) in the brain, where the cerebrospinal fluid (CSF) is produced. The ventricular system is continuous with the central canal of the spinal cord allowing for the flow of CSF to circulate.
- CSF acts as a cushion or buffer for the brain's cortex, providing basic mechanical and immunological protection to the brain inside the skull
- Production of CSF : Choroid Plexus (another 20% comes out from the ependymal lining of the ventricles)
- Tract of CSF:
Choroid Plexus – Lateral Ventricles (one in each hemisphere) – Foramen of Monro (interventricular foramina) – 3rd Ventricle – Cerebral Aqueduct (Sylvius) – 4th Ventricle – 1 Magendie/2 Luschka (1 median and 2 lateral apertures) – Subarachnoid Space – Sagittal Sinus
- Drainage of CSF : through the Arachnoid villi to the Sagittal Sinus
- Normal CSF production = 500 mL / day
- Normal CSF value = 150 mL in an average adult , so that means that CSF is changed 3 times / day (500/150)



Hydrocephalus

refers to the abnormal condition consisting of an increased volume of the CSF along with distention of the CSF spaces (ventricular system) (increased ICP and ventricular dilatation)

- Hydrocephalus causes are classified into :
 1. Increased CSF production
 2. Decreased CSF absorption
 3. Obstruction (communicating or non-communicating) (most common cause)
- Increased CSF production is a rare cause of hydrocephalus and is usually due to Choroid Plexus Papilloma (it looks like)
- Decreased absorption of CSF through the arachnoid villi to the sagittal sinus is caused by either infection or hemorrhage (subarachnoid hemorrhage)
- Any Obstruction beyond the 4th ventricle (outside the ventricular system) is called a communicating hydrocephalus whereas any obstruction before the 4th ventricle or within the ventricular system is called a non-communicating hydrocephalus
- We no longer use the terms obstructive/non-obstructive hydrocephalus
- Obstruction may be due to congenital (aqueductal stenosis) or acquired (tumors) causes
- Most common cause of pediatric hydrocephalus is aqueductal stenosis whereas in adults the most common cause is idiopathic (1/3 of adult cases), other causes include hemorrhage, head injury, infection and tumors

- Diagnosis of hydrocephalus is either through CT, MRI or head circumference
- Changes on the CT include ventricular dilatation (ventriculomegaly) mainly and other signs of increased ICP
- Signs and Symptoms include those of an increased ICP (triad of headache, vomiting and papilledema) , mass symptoms, ataxia/urine incontinence/dementia, increased head circumference and most importantly failure to thrive (children)
- The presentation depends on whether the hydrocephalus is acute or chronic
- One of the signs of acute hydrocephalus is 'sunset eyes' which is an impaired upward gaze
- Treatment :
 1. Treat the underlying cause if found (remove a tumor by surgery for example)
 2. Bypass methods (shunts and 3rd ventriculocisternostomy)
- Hydrocephalus treatment is necessary and should be done as soon as possible because if left untreated it leads to high levels of morbidity and mortality (bad prognosis)
- Communicating hydrocephalus is usually treated by shunts whereas non-communicating hydrocephalus is usually treated by endoscopic 3rd ventriculocisternostomy and shunts

Types of Bypass treatments :

A. 3rd ventriculocisternostomy:

Surgical creation of a communication between the 3rd ventricle and the interpeduncular cistern, for drainage of CSF in hydrocephalus

Pay attention not to injure the basilar artery or the mammary bodies during the operation

Used to treat non-communicating hydrocephalus

Advantages over shunts include:

1. No foreign body used
2. Once obstruction is gone everything goes back to normal

Disadvantages over shunts include:

1. High failure rates
2. Fast closure
3. It is only a temporary solution

B. Shunts :

Ventricular shunt is a surgical procedure in which a tube is placed in one of the fluid filled chambers inside the brain (ventricles) in order to relieve the pressure and it may be described as a divergence from the lateral ventricle.

A shunt is composed of a ventricular catheter, valve and a catheter elsewhere.

Types of shunts include:

1. Ventriculoperitoneal shunt (VP shunt) ; most common
2. Ventriculoatreal shunt
3. Ventriculopleural shunt

Complications of shunts include:

1. Proximal obstruction ; due to hemorrhage, increased protein in CSF or the choroid plexus itself
If you try to push the valve, it doesn't come back or it comes back slowly
2. Distal obstruction
You can't push the valve inside due to increased pressure inside the shunt
3. Shunt infection ; in the case of infection do the following:
 - a) Use an external ventricular drain
 - b) Calculate the amount of CSF from the drain
 - c) Perform a CSF culture
 - d) Monitor ICP
 - e) Give antibiotics (gentamycin, vancomycin)
4. Infection along the tract (skin)
5. Hemorrhage
Due to over drainage, bridging veins

6. Valve malfunction
7. Shunt fracture
8. Migration or disconnection

Drowsiness, Headache, Loss of consciousness and coma are signs of shunt malfunction

Following up a hydrocephalus patient with a valve include performing a:

1. Shunt survey/series : which is a series of x-rays covering the entire shunt length
2. CT scan : to check for hydrocephalus

NORMAL PRESSURE HYDROCEPHALUS

- Is defined as normal pressure (ICP) and dilated ventricles on CT scan.
- The classic triad of NPH consists of :
 - 1) Abnormal gait :
Earliest feature and most responsive to treatment; bradykinetic, broad-based, magnetic, and shuffling gait
 - 2) Urinary incontinence :
Urinary frequency, urgency, or frank incontinence
 - 3) Dementia :
Prominent memory loss and bradyphrenia; forgetfulness, decreased attention, inertia
- Differential diagnosis include : Dementia (Alzheimer's), chronic subdural hematoma
- Diagnosis is made by exclusion
- Pathogenesis: production is more than absorption
- Treatment : Lumbar Puncture (40 cc) , but don't forget to do a CT before performing the LP
- Recovery : Ataxia \geq Incontinence \geq Dementia
- NOTE when doing a Lumbar Puncture you do 3 things; measure the opening pressure, take two samples (for analysis and culture), make specific specimens.

THE END
Done by : Lana Daoud

Epilepsy Surgery

Epilepsy Surgery

- Epilepsy is not a safe disease.
- morbidity in epileptic pt is 10 times more than normal pp.
- mortality is 3 times more than normal pp.
- Most important is refractory/intractable epilepsy; i.e., epilepsy that doesn't respond to medical management by at least 2 conventional and 1 newer epilepsy drug, for at least 6 months in adults, and ASAP in newborns.
- Refractory epilepsy accounts for ~30% of epilepsy patients (i.e. 1/3 of epilepsy patients); 2/3 can be medically managed
- Epilepsy occurs in ~0.5-1% of entire population
- Epilepsy is the most common outpatient condition in neurology
- Presentation of refractory epilepsy:
 - Mostly in young people
 - 50% in <25 y/o
 - 75% of these are <5 y/o
 - Each seizure attack is a minor insult to the brain; over time these insults lead to brain damage
 - SEs of anti-epileptics along with recurrent attacks during development means a lot problems at a young age → early diagnosis is best
- Management with 1 antiepileptic drug is best.
 - BUT, only 50% of patients will be managed (assuming appropriate drug, dose, therapeutic level)
- With 2 drugs, 62% will be managed
- With 3 drugs, 65% will be managed (i.e. 2/3 of patients will be managed)
- More than 3 drugs, the patient will not be responsive → refractory epilepsy
- How to evaluate the patient with refractory epilepsy:
 - MRI (not a standard MRI, here we focus on temporal lobe) to R/O:
 - Organic lesion
 - Congenital anomaly
 - Temporal sclerosis
 - Standard EEG:
 - Abnormal bioelectrical activity
 - Normal EEG does not R/O epilepsy
 - Abnormal EEG does not confirm epilepsy
 - Video-EEG:
 - Localization of the focus
 - Correlation of physical and bioelectrical changes
 - Confirmation of epilepsy (can differentiate between epilepsy/psychogenic/simulated seizures)
 - There should be congruence between bioelectrical activity of the brain and the clinical findings
 - Patient may be both epileptic and have psychogenic seizures because many epileptic patients have

- Can be done:
 - Ideally in < 5 y/o because of the highest level of neuroplasticity (the ability to change the neuronal function based on demand; i.e. the other hemisphere can take over some of the functions); e.g. in Wernicke, Broca, Motor areas;
- In adults, completely contraindicated because of low neuroplasticity
- Can be:
 - Functional
 - Most commonly done
 - Temporal and Parietal removed
 - Disconnect frontal and occipital
 - Complications are hydrocephalus and hemo sclerosis
 - Anatomical
- Palliative Surgery (not curative):
 - Multifocal seizure or nonlocalizable seizure
 - Options(never cure the patient):
 - Callosotomy (dissect anterior 2/3 of corpus callosum and disconnect the two hemispheres to spread the seizure), improvement rate is about 40-50% but transient mutism may occur post op
 - VNS (vagal nerve stimulation):
 - Last resort
 - Improvement is about 50%
 - Complication (all of them are transient):
 - Dysphonia (temporary on insertion of electrodes)
 - Dyspnea
 - Pain
 - Sensation of abnormal respiration.

Epilepsy surgery Notes

- 70% of patients with epilepsy are well controlled with drugs (50% with one drug, increases to 62% with the addition of a second drug)
- in the definition of medically intractable epilepsy, the duration should be at least 6 months, and the number of drugs used are 3 (2 classical and 1 new)
- epilepsy patients have 3x mortality than normal population
- temporal lobe epilepsy accounts for 80% of all epilepsies
- corpus callostomy and vagal nerve stimulation are palliative not curative and their efficacy is around 50%
- Corpus callostomy: you remove the ant. 2/3 of it

Done

OSCE

&

Notes

* Rule of 5Cs in oligodendroglioma :-

2

- ① cerebral cortex C supratentorial, $\frac{1}{2}$ of these are in Frontal lobes
- ② calcification 90%
- ③ Convulsion 80%
- ④ curable
- ⑤ chemotherapy

* ependymoma

- 2/3 occur in Infratentorial / mostly in children
- 1/3 occur in supratentorial / mostly in adults
- predominantly Intra-ventricular
- rosettes and pseudorosettes (diagnostic features)

* most frequent malignant brain tumors in children \Rightarrow Medulloblastoma
↳ and Truncal ataxia is typically present.

* Meningioma

- arise from arachnoid layer
- RF (radiation, trauma, NF2).
- psammoma bodies
- surrounded by severe cerebral edema
- no radiotherapy

Neurosurgery notes

Morning reports

Day 1:

- * most common sites of CSF leak : lower cervical and thoracic region.
- * caffeine causes vasoconstriction and decreases the CBF
- * epidural blood patch : used to treat CSF leak ... we take small amount of Pt. blood ..extract the fibrin and use it as a gel to close the site of leak.
- * most common cause of decreased ICP
 - 1- epidural anesthesia
 - 2- LP
 - 3- trauma
- * *In Pt. with highly suspicion of decreased ICP due to leak ... we do valsalva maneuver to enhance the CSF leak.*
- * enhancement on MRI :
 1. decreased ICP
 2. meningeal carcinomatosis
 3. TB-meningitis
 4. idiopathic

Day 2:

- ****Trigonocephaly**: change in the appearance of the skull in the form of a triangle, this is cosmetically bad. And usually occurs in premature closure of the sutures of the skull.
- **One unusual complication of shunting is Rupture of the shunt because silicon usually doesn't rupture. A shunt could undergo rupture or disconnection.

****Now new fracture appears straight as if cut by a knife and head injury is considered a dynamic process in which several changes could appear post-trauma.**

**** *In head injury we could see the following:***

- Contusion (on prognosis we depend on edema and bleeding) shown as white dots
- Edema that will cause narrowing of the ventricles.
- Scalp hematoma and subdural hematoma.
- Physical symmetrical calcification in the lateral ventricles but these have nothing to do with injury but with but start at age 18 (calcification of choroidal plexus)

Day 3:

In brucellosis: epidural abscess and pus might develop causing spinal canal stenosis. Here we have no surgical treatment but only medical with IV antibiotics.

Inflammation on axial cut MRI appears as a cluster of white dots.

Sometimes patient can tolerate significant EDH with minimal or no symptoms at all.

Day 4:

Hyperintense signals on MRI appear white and could indicate lacunar infarcts that might cause parasthesia, paralysis or the possibility of MS.

Stenosis of the lumbar canal: typical history:

1- Chronic back pain 2- bilateral sciatica 3-neurogenic claudication.

****Most cases of falling down in elderly are cardiac in origin.**

****17% of head injury associated with cervical spine injury.**

Day 5:

In an elderly individual already diagnosed with brain metastasis we have no indication to do brain MRI, why?

- 1- Patient is already diagnosed.
- 2- We have no role for radiotherapy.
- 3- we could only do it for confirmation.

In neonates overlapping of the occipital bone over the parietal bone is mostly due to trauma caused by vacuum delivery.

****If the patient prefers to lie flat: think in intracranial hypotension.**

****Cluster headache: less than 4 hours, associated rhinorrhea, lacrimation.**

****All metabolic causes: gradual onset not sudden.**

Day 6:

****In normal situation the temporal horn of the lateral ventricles are not seen but if dilated by increase ICP then they will become apparent.**

****Patients with pontine calcification usually have deafness tinnitus and usually die from otorrhagia of the affected ear**

OSCE question :

in patient with odontoid fracture we must do MRA to make sure that the vertebral arteries are not affected

Day 7:

**Neurofibroma come from nerve root bilaterally and thus cause bilateral compression of the spinal cord.

**To recognize the site of spinal cord compression we can see enlargement of the IVD area on Xray .

Day 8:

**blood in intra hemispheric area appear hyper dense and wideness this area

**Traumatic birth could cause brachial plexus damage ... Erb-Duchenne (proximal) and Klumpke (distal) this could appear as calcification of the teared nerve roots

Day 9:

**Always count the lumbar vertebra from the last rib until sacral area because could have some lumbarization anomalies extra or lost lumbar vertebra

** Sign of degenerative diseases could include irregular endplates and bridging of osteophytes

** *we do dynamic studies to see if the spine stable or not*

**if the disc is central in protrusion then it will only appear at mid line on axial cut

**in stepping of the vertebra we care about the post alignment bcs of its relation to the Dural sac

common question by dr tamimi :

we do T1 MRI bcz it will enable us to see the posterior edge of IVD space

→ flat or concave is normal

→convex is abnormal

****DD of Hyperdense area on CT :**

if chronic is calcification @ if acute blood

****subdural hematoma :**

- hypodense = chronic
- isodense = subacute
- hyperdense = acute fresh blood

Day 10

No additional notes

Round notes:

****Patient with myelocoele have:**

→Hydrocephalus: treatment with shunt series.

→Urine incontinence: those patients usually die from urosepsis.

****CT is more accurate and faster than MRI.**

In Ct we say density, but MRI we say intensity.

- Bone window show skull + bone + air + and calcification.

- Bone window: to see fractures

- Anything white is either: bone + calcification + blood..

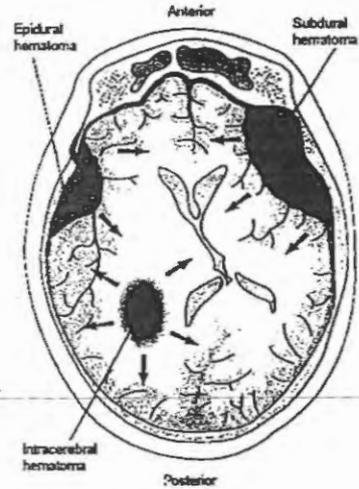
On MRI :

T1: CSF is black: anatomical.

T2: CSF is white: pathological

Hematomas :

- 1) epidural (lens shape)
- 2) subdural (crescentic)
- 3) intracerebral



On CT subdural hematoma :

1. hyperdense indicates acute bleeding
2. isodense same density as brain which indicates subacute bleeding
3. hypodense in chronic hematoma

****Scout is the part of the CT that shows where the cut has been taken.**

****To know if the brain MRI is sagittal or parasagittal (the reference is 4th ventricle)**

****On MRI: first vertebrae is C2 (odontoid process) we don't see C1**

****On Ct if the disc is white = (water)**

****The disc is black = dehydration**

****Commonest spine tumor: metastasis from breast CA or prostate CA**

Epilepsy: 80% treated medically

20%: intractable epilepsy, meaning failure of medical treatment of 3 anti-epileptic agents, in maximum dose for 1 year to treat the condition. This 20% is treated via surgery??

1) Resection: brain lesion, tumors, AV malformation.

2) Vagal stimulation: at the left side not the right, because right will stimulate the AV node and cause bradycardia. It is palliative, decrease the frequency of seizures 65%. This mode of ttt is also used to treat severe depression.

3) Disconnection: separation of the 2 hemispheres from one another via callosotomy. Usually indicated in multifocal epileptic foci.

In sciatica:-

- 1- Positive patrick sign: hip problem.
- 2- Straight leg sign.

DDx of sciatica:

- 1) Trauma: mostly in young*
- 2) Spinal metastasis*
- 3) Spinal abscess*
- 4) Degenerative spinal disease: mostly in elderly*
- 5) Spinal Dural hematoma and AVM : Rare*

**Concerning the GCS:

This gives you an idea about the cortical function and level of consciousness.

In terms of motor response examination:

→ If patient has transection of the spine ask him/her to protrude tongue, if he/she did it then the GCS of motor function is 6 because of command obeying.

→ Concerning flexion reflex to pain:

It is a primitive reflex that is stereotypic with no localization "any where you apply pain patient will flex the arms". Its aim is to protect the heart.

Extension to pain: also stereotypic with no localization. This indicates that only the medulla is functioning here upon applying pain both upper and lower limbs will extend.

A disc always compresses on the lower nerve except in one case only

... if the disc was far lateral than it will compress on the upper

Spondyloleucosis:

- Create a pseudo-disc.
- Affect the root above.
- Considered a degenerative disease.
- Right feels worse when going down stairs
Due to buckling of the ligamentum flavum.

**Radiculopathy doesn't affect deep tendon reflexes.

**Pain sensitive parts of the vertebra:

- 1) Facet joint
- 2) Paraspinous muscle.
- 3) PLL "most important"

All these can be stimulated by disc prolapsed.

We prefer angio > MRI for aneurysms why?

- 1) Angio specificity 85% while that of MRI 20%*
- 2) Angio can detect bilateral aneurysms that occur in 20% of cases.*
- 3) We can do embolization in angio and find the feeding vessel of AVM.*

****Concerning disc prolapsed:**

- Always ask if valsalva "coughing" make symptoms worse.
- Usually trauma triggers it.
- Most patients become better with pregnancy.
- The reason central disc prolapsed is rare is the fact that PLL is hypertrophied at the center.
- When we say central roots we mean S1-S4.

If patient has urinary incontinence and loss of bladder control, we must examine the perianal area sensation supplied by S2.

****Sciatica:**

L4: medial leg + lateral thigh + anterior knee and the back.

L5: The back + back of thigh + lateral leg.

S1: all the back.

Cauda equine syndrome:

Always involvement of the bladder plus/minus sensory and motor symptoms.

→ And usually affect the action of dorsiflex + cause saddle paralysis and impotence and sexual dysfunction.

→ Usually patient presents with foot drop and absent ankle jerk in both sides.

(MRI) is the best investigation??

Because it will show soft tissue and disc is a soft tissue.

If you don't have MRI and you want to make CT more accurate we do CT myelogram: we add contrast to epidural space then do CT.

(2) SENSORY FIBERS are LARGE and are the FIRST to be affected by COMPRESSION, while MOTOR FIBERS are SMALL and are the FIRST to be affected by ISCHEMIA.



Subsidence of pain from disc prolapse ISN'T ALWAYS GOOD ; because it could mean that the sensory fibers are compressed leading to numbness (subsiding of pain) but weakness will progress.

- Indications of conservative treatment of disc prolapse :

1. 1st attack
2. No Cauda Equina Syndrome
3. No deterioration of neurological deficits
4. Tolerated pain

What to give :

1. NSAIDs
2. Muscle relaxants
3. Bed rest → most important
4. +/- physiotherapy

- Complications of disc prolapse surgery :

1. Infection of the disc → discitis , adhesions
2. Infection of the wound
3. Bleeding (IVC, aorta) / (6) also recurrence (5%)
4. Paralysis : very very rare

(5) - Instability

out come
+ 80% cure
+ 15% (pain)
+ 5% recurrence

On MRI the 1st to be seen is C2 (odontoid process) while C1 is the first to be seen on X-Ray. (OSCE question)

- Stenosis of the canal is due to

- 1) the bulging of the disc (anteriorly)
- 2) Ligamentum flavum (posteriorly)
- 3) Facet joint hypertrophy (laterally)



Posterior stenosis can be treated by Flavectomy.

Stenosis of the canal is mostly seen in L4/5 > L3/4 > L2/3 > E1/2

Notes from Dr. Waleed Al Ma'ani ☺

Empty Cella Torsica can cause RHINORRHEA → spontaneous mostly in EM. this (rhinorrhea) due to atrophy of dural coverings of the cella could also occur in spontaneous age related atrophy of the Olfactory Plate in elderly women.

Rhinorrhea → traumatic / spontaneous → empty cella Torsica atrophy of olfactory bulb.

- Causes of recurrent meningitis :

1. CSF Leak
2. Abscess
- (a) 3. Inadequate treatment.
4. Immune deficiency.
- (a) 5. Sinus connecting with skin with theca inside (the tuft of hair in myelomeningocele).

- If the pressure is equal in all 3 compartments (Blood, CSF, Brain matter) herniation will NOT occur when we do Lumbar Puncture. Ex: Pseudotumor Cerebri (Benign Intracranial Hypertension) is treated by LP and NO herniation develops. *BH* (Diagnostic & therapeutic)
- How can we differentiate between turbulent blood flow and coagulated blood on CT? *Coagulated blood appears WHITE while turbulent flow appears BLACK.*

- Epilepsy caused by head injury is 1st treated with Phenytonin in adults while in children the 1st choice is Phenobarbital.

- Posterior extradural hematoma :
 1. ~~More dangerous~~ than anterior extradural and subdural hematomas with and increased mortality. The reason is that it is close to the medulla and brain stem.
 2. Difficult to diagnose.
 3. Mostly occurs in infants and children
 4. MUST be treated IMMEDIATELY
- ~~In CSF leak we give the patient Antibiotics and ask him/her to sleep on side of the leak to prevent CSF welling if the previous steps didn't work we can perform Dural Pasty.~~
- ~~It is quite difficult to identify the site of a CSF leak even with the use of dyes.~~
- Skull fractures can be categorized into:
 - Simple : closed
 - Compound (more than one piece)
 - : open
 - Both being a description of the SCALP.
 - Linear or depressed which is a description of the SKULL. This could be either simple or compound

- Concerning depressed fractures :



Trap Door Fracture

Not common

If the depression is more than the thickness of the skull we do surgery otherwise NO SURGERY.



The deepest point is the site of impact

This type is more common than the Trap Door Fracture.

- *Basal Skull Fractures : linear fracture but with distinct complications associated with the formation of Battle Sign , Raccoon Eyes "Bilateral", Otorrhea and Rhinorrhea.*

- Concerning coup and counter-coup, here a contusion will develop under the surface of the temporal and frontal bones and also the frontal and occipital poles due to the ridges found in the skull in these areas.

- *The weakest part of the skull is the occipital bone due to the presence of multiple foramina and openings causing more cranial damage.*

- Frontal fossa fractures will be associated with :

1. Bleeding and Periorbital edema.
2. Battle sign
3. *Subconjunctival Hemorrhage: if it came from basal skull trauma you won't be able to see beyond the hemorrhage, i.e. you can't see the sclera.*
4. Rhinorrhea and Otorrhea: usually mixed with blood but the blood doesn't clot (due to the fibrinolytic action of the CSF) which helps differentiate it from actual bleeding.

The most important complication of frontal fossa fractures is bleeding.

- Depressed fractures in infants (POND fractures) at the site of a suture that still didn't ossify from pressure, the brain will grow and push it out.

- To diagnose a patient as BRAIN DEAD :

1. 3 doctors : an anesthesiologist, a neurologist and another specialist, the treating physician should be present.
2. The patient must be on cardiopulmonary bypass
3. His/Her temperature should be 37.5 °C
4. Flat EEG.
5. No Spinocilliary reflex : if you pinch the patient no dilation of the pupil will occur.
6. Negative COWS test
7. Negative ANGIO test (only don't in Sweden), ANGIO test : if a clot sticks to the base of the skull there will be no circulation → mimics brain death.

- False depressed fractures : a hematoma started to ossify from the sides giving a boggy swelling, usually iatrogenic from the physician's fingers. We confirm the diagnosis via CT Scan.

- Treatment of cerebral edema :

1. Mannitol
2. Hypertonic glucose.
3. Mg Sulfate
4. Furosamide (Lasix) : but here we must monitor the patient's electrolytes and K+ homeostasis
*** While giving the treatment we need to monitor the patient's ICP via a monitor that is applied Intraparynchomal, Subdural or Extradural in order to maintain ICP homeostasis.

- The pain sensitive parts of the head :

- 1) Scalp.
- 2) Blood Vessels.
- 3) Basal Dura

The rest of the head is sensitive to STRECH but NOT to PAIN.

- Sub-Arachnoid Hemorrhage : blood vessels in the brain move in CSF so if the vessels bleed the 1st thing to happen is mixing of blood with CSF causing SEVERE OCCIPITAL HEADACHE and 50% die IMMEDIATELY.

- An aneurysm could rupture due to increased ICP from the following :
1. Trauma 2)Constipation 3)Intercourse.

- *Once an aneurysm ruptures, blood will start to coagulate but the CSF will cause fibrinolysis and remove to coagulated blood about 4 days later. So rebleeding will occur and death will occur at an even higher percentage than the 1st SAH*

- In the past, they used to give the anti-fibrinolytic agents after the first bleed but it resulted in more harm than good.

- *Fibrinolytic activity is found in bodily fluids that undergo filtration:*

1. Semen
2. Urine
3. Aqueous Humor.
4. CSF

-Brain tumors :

**tuberous sclerosis : mental retardation + multiple facial anomalies + finger agnosia

** increase ICP that is caused by mass occupying lesion (edema surrounding the mass is highly reactive to steroid)

**headache is more with post fossa tumor bcs it will cause compression of 4th ventricle causing hydrocephalus & this need rapid surgical treatment

** non projectile vomiting (not proceeded by nausea) indicate tumor in the post fossa.

**if parietal lobe was affected :

→ dominant = Gersman syndrome

→ non dominant = acalculia @ hemi neglect

**we must do tumor marker if suspected tumor of germ cell origin

***cytoreduction conversion to the active form so chemo would work properly!*

**we do transoral approach for any skull based tumor that are usually very malignant

**germinoma is the most common in japan

***gamma knife results will begin to appear 3 years post
procedure , so if we do AVM gamma knife ttt we still have risk
of bleeding for 3 years at least*

***chemo is better to be given to children especially with
oligodendroglioma ,*

** usually ttt with surgery + chemo+radio bcs radio will affect child growth and
cause mental retardation

***brain tumor usually invade and mets but
spinal tumor usually cause compression*

****signs indicating the presence of mass :**

- 1) shifting of the midline
- 2) sulci are ~~not~~ apparent in comparison to other side
- 3) partially collapsed ipsilateral ventricle

~~**meningioma never cross midline + has increase incidence of calcification (similar to oligo + ependymoma)~~

****most tumor with enhancement is meningioma (2)**

****COCERNING CPP :**

$$\overset{100}{CPP} = \overset{10}{MAP} - \overset{10}{ICP}$$

CPP NL = 90 MM HG = 1000 MM H2O

CPP cant be reduced beyond certain point 60 mmhg

MAP decrease or ICP increase will cause decrease CPP and hypoxia to brain will

develop ... MAP will affect CPP more than ICP because it is (10X THE ICP) SO

BE CAREFUL TO HYPOVOLEMIA

NOTES FROM DR WALEED LECTURES ☺ :

****Most common cause of BIH (although it is not totally benign as it may cause blindness due to optic nerve atrophy):**

- | | |
|----------------------|---|
| 1) Stroke | 2) Low central venous pressure |
| 3) OCP | 4) Hypervitaminosis A |
| 5) Steroid | 6) Stress |
| 7) Idiopathic | |

20
OCP
Jaw
MRV

Important :

status epilepticus will cause hypoxia & thus lead to inc ICP

****vomiting + HA in morning = visual flashes = migraine or occipital lobe tumor??***

(to differentiate we look for papilledema which absent in migraine)*

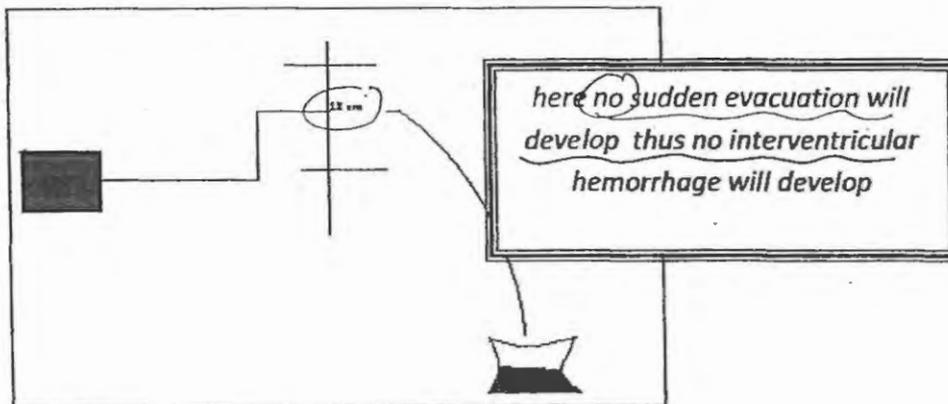
*LP is considered ttt of choice in BIH due to equalize pressure in all brain compartment other wise is contraindicated bcs it will cause herniation.

****When to apply EVD ?**

- 1) pt has normal ventricle
- 2) Child with hydrocephalus
- 3) Adult with obstructive hydrocephalus

***Most important complication is meningitis** if bag was put bellow the level of pt then over drainage will occur

Over drainage in a chronically constricted skull will cause intraventricular hemorrhage why ? bcs the ventricle wall is accustomed to flow under increased pressure and the wall is also atrophied . this condition is sometimes lethal



- Occipital trauma is more dangerous than Frontal trauma as the frontal is protected by air sinuses.

- The 2 cases that show basal cistern closure are Traumatic SAH and TB Meningitis.

CSF leak could be

- 1) Traumatic: due to either a penetrating injury or a basal skull fracture → it leaks from the nose or the ears or BOTH.
- 2) Spontaneous: due to either empty Cella Torsica or atrophy of the olfactory bulbs.

* In shunt obstruction its either:

1 proximal (usually caused by choroid plexus)

(منوع نحلول ثقيله لانه ممكن نطلع plexus و المريض يموت) (2)

2 distal

to differentiate we press on the valves if its compressible then the obstruction is upper, if its not compressible then its distal obstruction

EXTERNAL SHUNTS COMPLICATIONS

1) Ventriculo-pleural:

infection + over drainage ((due to +ve pressure of chest))

2) Ventriculo-peritoneal:

infection + slipping + perforation + kinking + block + over drainage + coiling.

→ (Intestinal obstruction)

(but it is easy and vast surface absorption) > also easy to repair

inguinal hernia
peritoneal cyst
volvulus

} * Accomodable axial growth

3) Ventriculo-jugular :

infection + CLOTTING + slipping + kinking + block + coiling

(here we do it through facial vein)

4) Ventriculo- atrial : (usually in Infancy)

infection + atrial fibrillation and is difficult to administer in a child

You know it is in the right position if u see splitting of P wave

shunt embol:
retrograde flow.

****INTERNAL SHUNTS:****

1- 3rd ventriculostomy : through a fenestration in the floor of the 3rd ventricle

FOM : foramen of Monro
AOS : aqueduct of Sylvian

*This can be done via endoscope
**We need to be careful bcz it is associated w increased % of Hemorrhage .



2- Tankildsen operation :tract from the lat. Ventricles to the cisterns .

****Candida albicans is the hardest to treat in shunts infection**

**** In shunt application if the whole operation took out more than 30 minutes then infection rate is 100% !!!**

**** The shunt valve is located behind the ear .**

If the shunt got infected :

- 1- REMOVE it and take cultures and CSF analysis .
- 2- Apply external Drain (ED) .
- 3- AB and observe CSF from ED till WBC < 3 .
- 4- Remove ED and apply a new shunt in other site .
- 5- IV cloxacillin & floxacillin for 2 wks

**Over-drainage will cause slit ventricle syndrome : subdural hygroma bcz fluid will take the place of CSF .

Ventricles with very minimal increase in ICP causing unconsciousness of the patient & requires adjusting to shunt .

**Best shunt is done in the first time , and each time of redoing increases the complications ---

**Oblique X-ray cut is aimed to visualize the IV foramina : if dilated this is a sign of compression on nerve roots,, These foramina should be compared bilaterally bcz usually compression is unilateral but can be bilateral ..

**Commonest spinal tumor is METS from organs :

A-composed of lobes : kidney + lungs + breast + prostate (osteoblastic)

B-bilateral organs .

All other than prostate are osteolytic mets .

***Filum terminale ependymoma is the easiest to be resected .*

****Most mets tumors arise from vertebral pedicles and upon growing the abut to the canal making stenosis .**

dura is an excellent barrier to prevent mets , so if the tumor is Extradural it will stay that and never become intradural !!

****in the past they used to treat BIH via cutting the filum terminale and pia matter bcz this leads to CSF leak .**

****dura has the capability to absorb CSF (4%) so if we had a complete obstruction CSF will eventually be absorbed by dura .**

the CSF that remains unabsorbed by the dura will :

1-xanthochromic & yellow due to very high protein level .

2-if exposed to air > clotted .

this is called Froins' syndrome > indicates complete obst. In CSF flow .

****CSF protein levels depends on the gravity & site of CSF extraction**

****Sites of CSF extraction :**

1-ventricles 2- spinal tab

3-cisterna magna

****cisterna magna :triangular shaped area bet. Cerebellum and medulla , here we can extract CSF w no complications by approaching this area below skull laterally**

-intra-medullary intra-dural block will give inverted Champaign glass appearance .

-pain from compression is due to :

- 1. nerve. root compression.*
- 2. mets of vertebra*

-water shed area of the spinal cord is found at the lower border of T4 & is very poor w blood supply .

***at the level of T4 there are :*

- 1. carina*
- 2. aortic arch*
- 3. azygos vein*
- 4. sternal angle of lewis*
- 5. water shed area*
- 6. many others*

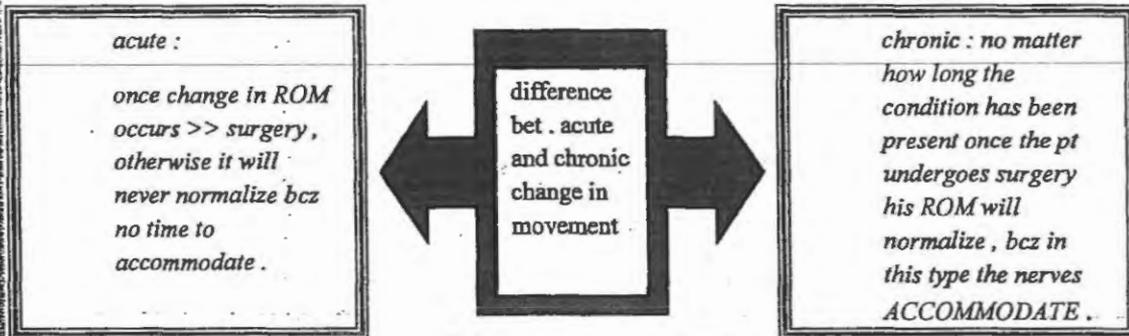
*spinal segment x = spinous process x + 1 or 2 or 3 (depends in location)
bcz the cord is shorter than the vertebral column and the spinous
processes will no be directly behind its vertebra (its directed downward)*

***so if sensation is lost in umbilicus =T10 then the tumor is at 7 or 8 or 9
level of spinal processes .*

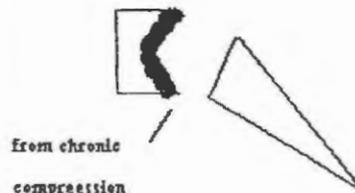
*any orifice in the body in the body has dermatome
of 5 or 10 , how ?
anus = S5
umbilicus=T10
breast =T5
eyes =V5*

- any patient presenting with LL pain must undergo PR exam !!
bcz rectal tumors can cause LL pain .

- also ask the patient to cough and whether the pain increases then it is typical of mass occupying lesion compressing nerve roots .



- if we have a tumor :
 - 1- erosion of the pedicles .
 - 2- increased interpedicular space (NL = 22 mm)
 - 3- collapsed vertebra .
 - 4- osteolytic % osteoblastic lesions .
 - 5- wide foramina . scalloping of vertebra



- best operation to be done is decompressive laminectomy bcz most tumors occur in pedicles .
- for chronic cases we do vertebrotoomy that always followed by fusion (not for acute)
- concerning Shwannoma , we must remove it completely , otherwise it might regrow .
 this is done by removing the myelin sheath or if the n. isn't very important such as T10 we remove it !!

Kocher point

Located at the point of meeting of 2 lines :

- 1- line drawn from pupil up to the hair line .
- 2- line in half way bet. Lateral canthus (of the eye) and tragus (of ear) upward .

is a common entry point for an intraventricular catheter to drain cerebral spinal fluid

** indications to apply an ICP monitor :*

1) *neurological criteria :*

a. severe head injury

b. abnormal admitting head CT

c. normal CT but ≥ 2 risk factors { age > 40 , SBP < 90 mmHg , unilateral or bilateral decorticate posturing

2) *altered LOC accompanying multi-system injury*

3) *following removal of intracranial mass esp: after post.fossa surgery (high risk to develop hydrocephalus)*

*ICP monitor is also used to differentiate between normal pressure hydrocephalus(NPHC) and dementia (how? First we measure Pt. ICP then we inject fluid intrathecally and observe the changes in the ICP , in both cases it will be elevated initially then in dementia it will normalize quickly but in NPHC it will normalize later on.

* we measure ICP every 15 min and if it is high we make sure that the patient is not doing any valsalva maneuver (crying , straining , coughing)

* we keep a drain in the disc surgery to prevent collection + formation of epidural hematoma

* aim of spina bifida surgery :

1. avoid deterioration of neurological function
2. avoid infection formation
3. cosmetic correction

But no improvement will occur

* all forms of spina bifida except meningeomyelocele is static bcz the last will have micro-trauma to the nervous tissue every time patient moves causing deterioration of neurological function

* straight raise test is specific for (lower lumbar n. disease) for upper lumbar n. disease we do →femoral test :-

Ask patient to lie on his belly and raise his leg if +ve means upper lumbar n. disease

* Patrick's test >>>>>>> sacroillitis

* when do we do surgery after medically treating the patient ?

1. failure of medical treatment 1-2 months later
2. progressive weakness
3. parasthesia development

* surgery is done acutely in :

1. intractable pain
2. acute weakness
3. cauda equine syndrome

* L5 and S1 anomalies present similarly except for eversion abnormality found in S1

* hyperpathia >>>> simple touch will cause severe pain

* Sensation :

1. sup. >>> touch and temperature
2. deep >>> proprioception + pain + vibration

* radiculopathy >>> distribution along the myotome or dermatome is always treated surgically

* Para central L4/ L5 disc could give S1 symptoms

* *disc surgery complications :*

A) intra-op :

Bleeding, damage to aorta through the PLL , Dural damage causing meningitis , visceral damage

B) post-op :

Arachnoiditis , diskitis , hematoma , recurrence , FBSS " failed back surgery syndrome "

* reflexes :

1. monosynaptic >>> deep tendon reflexes
2. poly synaptic >>> plantar + abdominal + hoffmans + cremasteric

* *why recurrence ?*

- *disability and spondylothesis*
- *disc not completely removed*
- *disc is degenerative disease*
- *epidural fibrosis developed*

* what is the difference between Neurogenic and vascular claudication ?

Neuro >>>>> 1. Patient feels better if he leans forward

2. both have same mechanism Ischemia due to increase metabolism

3. patient will be able to walk the same distance after rest.

Vascular >>>> pt will walk less at each time, even after rest

* In prolonged esp. uncontrolled DM knee reflexes are reduced

* stereotactic MRI >>>> MRI used to guide the surgeon during surgery

* discectomy , different approach :

(laminectomy, fenestration through lig.flavum or bone)

- * ant. And middle fossa are separated by sphenoid wings
- * in some cases ,mass in the middle fossa can cause exophthalmos
- * subdural hematoma can result in mid line shifting and collapsed lateral ventricle .

With all my best wishes ..w d3watkom ☺

Bayan al-Khdour

I want to thax my amazing group for their help

*(Abeer al-shmali , Nadia abu-thaimer, Asma2 mustfa, Zain el-3abdeen
Ayoob 3nnabe, Ndal mtani . faris 7jawe. Tariq el rfo3)*

Ma 89rto ☺

(16/9/2011)

BRAIN TUMORS

o Risk factors for brain tumors :

Radiation : apparent in patients working in atomic radiation (atomic bombs), or exposed to diagnostic radiation (especially if developing brain)

Single exposure to CT in a child <6 months increases the risk 100X, so it's not preferred to do CT scan to children <2 years

There's a relation between the ear used (for mobile) and acoustic shwannoma and glioma, but NO increased risk compared to general population

Surgery is not curative for glioma, it's an infiltrative tumor, even if resected there's a high risk for recurrence

Of all brain tumors : glioma (45%), meningioma (15%), metastasis (15%)

Surgery can be :

- 1) Curative : in benign tumors, extra-axial (medulloblastoma, meningioma, pituitary tumors)
- 2) Debulking or for biopsy : in infiltrative tumors, deep tumors (thalamus), optic nerve glioma

Types of radiotherapy :

- 1) Conventional radiotherapy
- 2) Brachytherapy : placing a radioactive material inside the tumor/brain
- 3) Stereotactic radiotherapy : a type of which is Gamma knife (stereotactic radiosurgery) : beams of radiation are focused stereotactically (3D orientation) into the tumor, the whole dose of radiation is given in one sitting
 - Gamma knife is most effective in vascular malformations (not tumors)
 - Meningioma is not radiosensitive

Calcification is more common in benign tumors

o Astrocytoma :

Presence of mitotic activity → high grade

Presence of necrosis + neovascularization → Glioblastoma

o **Oligodendroglioma :**

Rare tumor, commonly presents with epilepsy (the most common to present with epilepsy)

The commonest brain tumor to calcify

Anaplastic is more chemosensitive

o **Ependymoma :**

Usually inside the ventricles, rarely extraventricular

In children the most common site is the fourth ventricle, in adults it's the lateral ventricle

Grade 1 : myxopapillary, very benign, occurs in the spine not the brain

Grade 2 : papillary, cellular ependymoma

Grade 3 : anaplastic ependymoma, malignant

Grade 4 : ependymblastoma, similar to glioblastoma

ependymoma arises from *the floor* of the 4th ventricle, can be adherent to the brainstem → can't be excised completely

Treatment : resect the tumor, treat the hydrocephalus (shunt or endoscope), then radiotherapy or chemo+radiotherapy

Radiation is craniospinal or neuraxis because it has the tendency of CSF seeding

5y survival : 30-50%

ependymoma and medulloblastoma : have similar location, similar patterns of presentation, age, and almost similar appearance

o **medulloblastoma :**

arises from *the roof* of the 4th ventricle (vermis)

more chemosensitive and radiosensitive than ependymoma

it's more malignant than ependymoma but can be completely excised

treatment surgery ± shunt + radiation (craniospinal) ± chemotherapy

5% may have bone metastasis

5y survival : 40-60% (although it's more malignant)

o **Meningioma :**

Can be classified according to :

- site (suprasellar, parasagittal, olfactory groove)
- cells (fibroblastic, syncytial, chordoid)
- grades :
 - grade 1 : most are grade 1 (95%), benign
 - grade 2 : premalignant, atypical, benign with high risk of recurrence
 - grade 3 : <1.3%, malignant, invades the brain like any malignancy, high risk of recurrence

treatment : surgery (complete excision with the surrounding dura), you shouldn't excise any part of the brain (it arises from the meninges, compresses the brain)

o **Glioneural tumors :**

neuron cells and glioma, in the temporal lobe, cause seizures, rare

o **NOTES :**

- tumors more common in females : meningioma, pituitary adenoma, vestibular schwannoma (acoustic neuroma)
- commonest pediatric tumors : astrocytoma, medulloblastoma, ependymoma
- 4th ventricle tumors : ependymoma, medulloblastoma
- Cerebellopontine angle tumors : acoustic neuroma, meningioma, epidermoid

You have to know the WHO classification of brain tumors, and about epidermoid and pituitary adenomas

by: Rema Jundi

SPINAL CORD TUMORS

- Spinal tumors can be intra-dural (within the CNS) or extra-dural (mostly mets)
 - Intra-axial (Intradural) can be either intramedullary or extramedullary

 - Extradural 55%
 - o 1ry 5%
 - o 2ry (mets) 50%
 - Intradural 45%
 - o Extramedullary 40%
 - o Intramedullary 5%

 - Malignant tumors are the commonest, of those
 - 50% are mets
 - Of all 1ry cord tumors : 1-2% are malignant

 - Presentation : symptoms of nerve or cord compression & loss of fn.due to
 - o direct compression by the tumor
 - o or instability & destruction of the vertebra

 - First presenting symptom is **PAIN** :
 - o it's a night pain (due to ↓cortisol & CO2 retention during the night) ,the location of pain is **strongly correlated** to the site of the tumor
- ddx. : Disc prolapse
Differentiated by enhancement on imaging

- The most imp single prognostic factor is the ambulatory state at presentation

IMAGING

1) Signs on plain X-ray

- General signs of bone destruction & instability , deformity (scoliosis..),
- Mass
- ↑intervertebral foramen size.....dumbbell (schwannoma, neurofibroma)
- ↑interpedicular distance (nl distance 22-25 mm)
- Scalloping of the vertebrae

2) CT scan :

- diff. intramedullary Vs. extramedullary
- cystic enhancing lesion → hemangioblastoma

3) MRI :

- The most imp. !!4
- Lumbar puncture: ↑ protein in 95% of intramedullary tumors (esp. ependymoma)
- Bone scan..

Intramedullary

- Primary are uncommon , secondary are RARE!
- Most are gliomasof which most common is ependymoma
- Ependymoma > astrocytoma in the spinal cord (the opposite occurs in the brain)

1) Ependymoma

- Most common primary (65%)
- Types :
 - Myxopapillary.....mostly in filumterminale
 - Anaplastic type : VERY RARE & VERY MALIGNANT
- 30-40 yrs
- Separated from the SC by a plane of cleavage → allows complete resection / sausage -like
- Tx : surgical resection , no role for radiotherapy

2) Astrocytoma

- 2nd – 5th decades
- The most common intramedullary in peds
- M>F
- Thoracic >cervical
- 4 types
- Pilocytic type.....almost benign
- 1/3 is malignant (GBM or anaplastic)
- Less enhancing than ependymoma
- Prognosis depends on the grade

3) Hemangioblastoma

- Benign
- Uncommon (2% of all SC tumors)
- 5% in VHL syndrome
- Well-circumscribed but not encapsulated
- Intramedullary >extramedullary
- Ass. with polycythemia

- If it hemorrhages → presents acutely!
- 4) Intramedullary mets : VERY VERY RARE!

INTRADURAL EXTRAMEDULLARY

1) Meningioma

- F>M , middle age (30-50 yrs)
- Chronic slowly-growing tumor eroding the bone and compressing the SC
- Mostly in the thoracic spine (80%) >upper cervical > foramen magnum
- *Typical pt: elderly lady with mid dorsal pain over years associated with weakness progressing over months + sensory level*
- Adherent to spinal dura.....base attached to the dura giving the appearance of dural tail on MRI (*pathognomonic*)
- Imaging → calcification/Very enhancing !
- In addition to removing the tumor we have to remove the adjacent dura with a safety margin
- Associated with Neurofibromatosis

2) Neural sheath tumors (NST)

- Very slow-growing tumors
- Common in low-thoracic and upper lumbar
- Adults 20-50
- Erodes the bony foramina!
- 2 types : neurofibroma (extends into the nerve sheath , infiltrates , with NF I , multiple) & schwannoma (surrounds the nerve sheath without internal extension , with NF II , solitary)
- Most are intradural , but can occur extradurally which is very rare!

Meningioma Vs NST

- Meningioma compress the cord → myelopathy
- NST compress the nerve root → radiculopathy
But later it may affect the cord as well → myeloradiculopathy

3) Filumterminale tumor

- 4th – 5th decade
- M > F
- Mostly in the prox. Portion

EXTRADURAL

- Primary : may arise from any structure ; bone, BM , nerve roots, soft tissue
- Mets are most common !!
 - o Primary tumors (any bilateral organ or bi-lobar organ) + lymphoma + multiple myeloma
Lung, breast, prostate, thyroid, kidney, ovary..
- Metastasis is mainly **hematogenous**
 - Venousas in prostate CA (Batson plexus of veins)
 - Arterial (most, breast, lung)
- Thoracic > lumbar > cervical
- Suspected in CA pts presenting with : back pain , persisting in recumbency
- 5-10% of all malignancies present with SC compression

*** Water-shed area of the SC :

- between the blood supply coming from superiorly (ant.&post. Vertebral arteries) & that coming from inferior (artery of Adamkins?..originating from T10 or T11)
- at T4 level

GENERAL NOTES

- presentation depends on the affected level
 - intraduralextramedullary tumors
 - o hydroceph& ↑ ICP....mostly in upper cervical tumors
 - o asymmetry in early S&S
 - o weakness begins distally
 - o sensory ataxia
 - o sphincters are affected LATE
 - o pain esp. in filumterminale..
 - ↑ with recumbency , neck flexion & movement , relieved by paracetamol ! ,
 - mets : 15% present paraplegic, 70% have weakness at dx
- cord pain : dull / aching
- radicular pain : electrical / radiating

TREATMENT :

- All are treated by surgery : laminectomy + (excision Vs. debulking)
- If the vertebra is involved : vertebrectomy + fusion

* Neurosurgery:

Body fluids that have fibrinogenic Activity:

- 1- Urine
- 2- CSF
- 3- Aqueous Humor

↳ that's why there are red blood cells in SAH

Causes of Severe very bad edema:

- Mets
- Abscess
- Contusion
- Meningioma

Stable vs. Unstable Spine: Denis Classification

- 1- Post Column → Posterior bony arch of the vertebra
- 2- Ant Column → the ant long. lig. & the ant half of the disc
- 3- Middle Column → the post long. lig. & the post half of the disc

②

if > than one ¹ column is affected or if there was ²

= a canal compromise + 1 of the columns it's unstable

³ and if there's wedging of the vertebra > 50% on X-ray

Herniation: Rupture of the disc

Protrusion / Bulge: no disc rupture

The most common

most common spinal tumors are metastatic 50%

most common 1° = = : ependymoma in adults

astrocytoma in children

1° Supratentorial tumor: Astrocytoma in adults & children

1° Infratentorial tumor: Hemangioblastoma in adults

Medulloblastoma if malignant &

Pilocytic Astrocytoma if benign in children

level of lumbar disc prolapse: L5-S1 then L4-L5

lumbar canal stenosis: L4-L5 then L5-S1

(3)

Head injury is classified according to GCS:

Mild

Moderate

Severe

14-15

9-13

≤ 8 (pt must be intubated)

Aneurysm Ruptures:

80% Anterior circulation

Ant. communicating > Middle cerebral artery

20% Posterior circulation

Posterior cerebral artery

To SAH : complications are 1- Rebleeding (this 2nd time is lethal)

2- Vasospasm (2-3 days)

↳ give nimodipine to prevent it

↳ (CCB)

why not give anti-fibrinolytic agents to prevent CSF from causing

a rebleed?

it will cause: venous sinus thrombosis, aneurysm, Aneurysm

Hydrocephalus, glaucoma

Surgery is done after 2 weeks (waiting for the

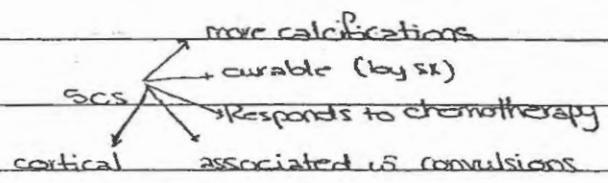
brain to stabilize)

Any cervical injury above C5 → fear of respiratory compromise.

Ischemia appears as a white signal on the spinal cord

Medulloblastoma	vs.	Ependymoma
in the vermis or roof of 4th ventricle		in the floor of the 4th ventricle

has a Banana Sign
 more sensitive to radiotherapy
 more risk of mets.



In post. fossa tumors, when putting a shunt, there's an increased risk of upward herniation

That's why we put a high pressure shunt.

Prolactin levels of > 200 are significant for Prolactinoma
 < 200 => drugs, lactation or stalk effect

⑤

Null Cell adenoma vs. nonfunctioning adenoma
no specific cell type have a specific cell type
but produce NO hormones

All hydrocephalus is obstructive except the hydrocephalus
due to ↑ production of CSF.

Obstructive → 1- Communicating: obstruction at arachnoid
granulation level.
↳ non-communicating: at any level
above the 4th ventricle

* the ventriculo-atrial shunt is the most physiological one

7- Lumbar degenerative diseases:

- Emergencies:
- ① Acute Cauda Equina
 - ② Acute Root drop.
 - ③ Rapid neurological deficit.

For disc surgery:

Intraop complications:

- 1- Bleeding
- 2- Root injury
- 3- Dural tear

Post-op complications:

- 1- Infection (discitis)
- 2- Persistent Pain.
- 3- Recurrence (5-30%)
- 4- Instability
- 5- Hematoma
- 6- Adhesions.
- 7- Arachnoiditis.

(B)

Seizures post brain injury are due to hyponatremia if the CT scan is normal. The hyponatremia is due to 2° use of diuretics in ↑ ICP or due to SIADH secretion post-trauma

Otorrhea: management 1- Antibiotics & don't examine (Prevent infection)
2- IV fluids.
3- Gauze & sleep on the side that leaks.

* How to know that the leaking fluid is CSF?

- ① Glucose level
- ② β -transferrin?? (not sure)

CSF = isodense on CT scan.

* In the case of bleeding in the ventricle: due to HTN

in the Sylvian fissure: due to aneurysm.

* ↑ in ICP doesn't cause change in the level of consciousness unless it's associated with herniation.

Cerebral Blood Flow = $\frac{\text{Cerebral Perfusion Pressure (CPP)}}{\text{Cerebral vascular Resistance}}$

CPP \geq 60 either by ↓ ICP (↓ Resistance) OR
↑ BP (↑ CBF) by ↑ MAP

⑦

\uparrow CO₂ \rightarrow \uparrow in CO₂ causes vasodilation

\downarrow in CO₂ = vasoconstriction

that's why when sleeping, \downarrow RR \rightarrow \uparrow CO₂ leads to \uparrow ICP

leading to the MORNING headache.

another cause is \downarrow coarctations at night: \uparrow edema:

\uparrow headache.

SAH Fisher Classification

Grade I no blood

I Blood $<$ 1mm.

II Blood $>$ 1mm.

III Traumatic/arterial Bleeding

\neq Spinal Tumors - X-ray findings \rightarrow

① Osteolytic lesion

② Scalloped vertebrae

③ wide intervertebral foramen

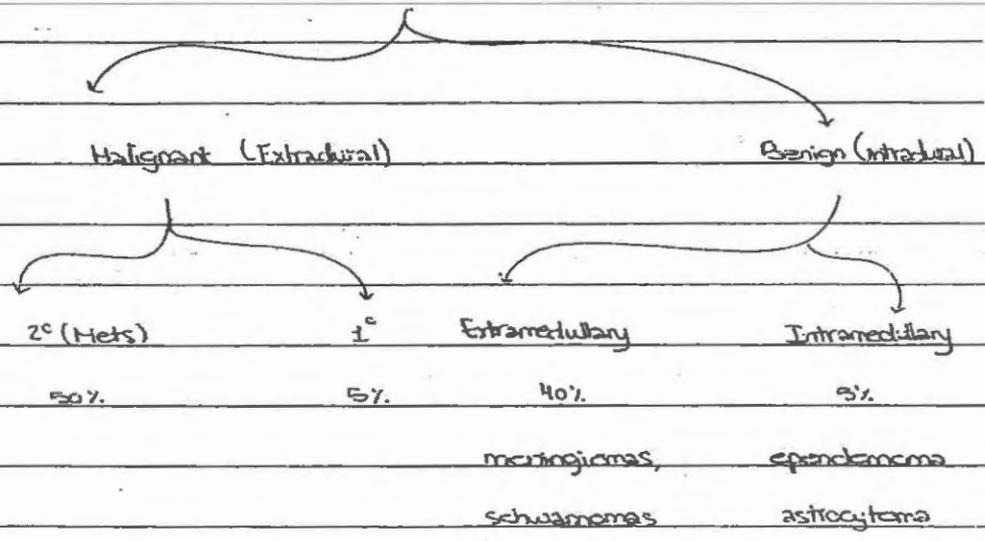
④ Fractures of the pedicles

⑤ loss of vertebral height

Boin Syndrome:

if there's an obstruction due to a spinal tumor, the distal CSF will be absorbed by the dura leaving a turbid, yellow mucinous fluid, that dot immediately if aspirated.

Spinal Cord Tumors



Management of myelomeningocele →

- ① Psychological Counsil to the parents. & deliver by C/S.
- ② Cover the sac & give Abx
- ③ Neurological Exam.
- ④ Surgery