

# HYDROCEPHALUS

- a condition characterised by accumulation of excessive CSF within the cranial cavity
  - dilatation of cerebral ventricles
  - $\pm \uparrow$  ICP
  - enlargement of cranium (infants)
  - brain atrophy

## FLOW OF CSF

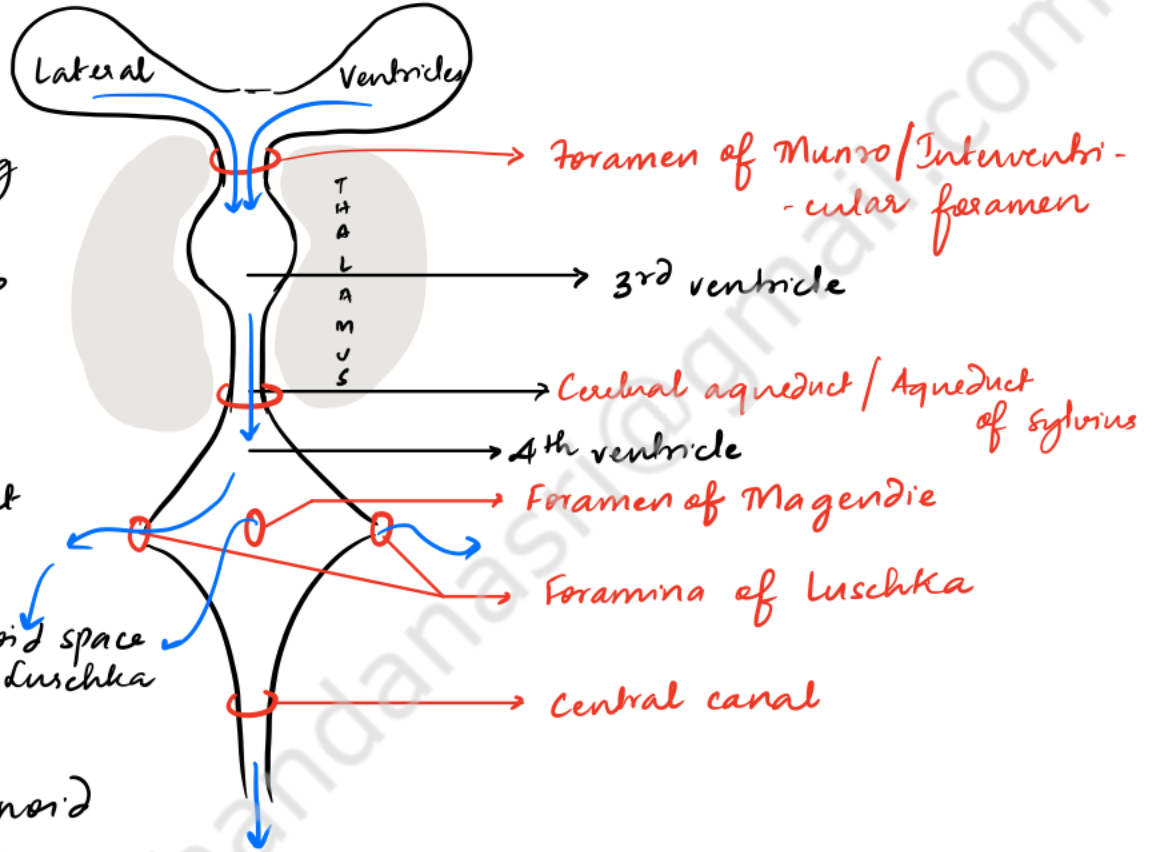
CSF is produced by the choroid plexus lining the ventricles

↓  
travels from lateral to 3rd ventricle via Foramen of Munro

↓  
Passes to 4th ventricle via Sylvian aqueduct

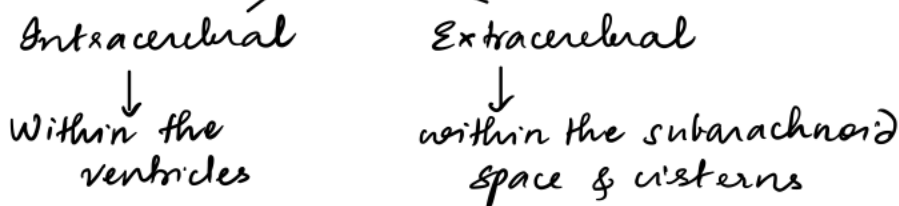
↓  
Passes from 4th ventricle to subarachnoid space via foramina of Luschka & Magendie

↓  
Absorbed by arachnoid villi lining the subarachnoid space



Total volume of CSF = 150 ml  
Produced at the rate of 20 ml/hr  
Absorbed within 8 hrs

## Accumulation of CSF in hydrocephalus



# CAUSES OF HYDROCEPHALUS

## ↑ CSF PRODUCTION

- Choroid plexus papillomas

## OBSSTRUCTION TO FLOW OF CSF

## ↓ CSF absorption

### NON-COMMUNICATING HYDROCEPHALUS

No communication between the ventricular system and the subarachnoid space

Obstruction at/proximal to Foramina of Luschka & Magendie

(i.e., obstruction in ventricular system)

- Eg. Aqueductal stenosis / gliosis
- Intraventricular hemorrhage
- Tumors

### COMMUNICATING HYDROCEPHALUS

communication between the ventricular system and the subarachnoid space

obstruction distal to the level of Foramina of Luschka & Magendie

- Eg. Subarachnoid hemorrhage
- Meningitis
- Leptomeningeal carcinomatosis
- Basilar impression

## CLASSIFICATION IS SEVERAL DECADES OLD

- Inject a dye into the ventricles

Do lumbar puncture

Dye appears in tapped CSF

Communicating

Dye does not appear in tapped CSF

Non communicating

based on ventriculogram & pneumoencephalogram findings

## TYPES BASED ON DURATION

### Acute Hydrocephalus

Days to weeks

- Eg: Hydrocephalus d/t tumor / meningitis

### Chronic Hydrocephalus

Months to years

Normal pressure hydrocephalus

## TYPES BASED ON PRESENTATION

### CONGENITAL

at birth

M/C - Obstructive

Non obstructive - Toxoplasma / CMV infection

### ACQUIRED

Post traumatic  
tumors  
NPH

## SPECIAL TYPES

HYDROCEPHALUS EX VACUO - ventricles enlarge as a compensatory response to overall shrinking of brain tissue

- Senile Cerebral atrophy
- Neurodegenerative disorders

BENIGN EXTERNAL HYDROCEPHALUS -  $\uparrow$  relative immaturity of arachnoid villi  $\rightarrow$   $\downarrow$  absorption of CSF  
seen exclusively in children - macrocrania, mild developmental delay  
usually self limiting; shunting if not relieved by 2y of age

NORMAL PRESSURE HYDROCEPHALUS - a type of communicating hydrocephalus  
( $\uparrow$  resistance to CSF outflow -  $\downarrow$  absorption at arachnoid granulations)  
with normal ICP

ICP is  $\uparrow$   $\rightarrow$   $\uparrow$  compensations  $\rightarrow$  HOWEVER, transient elevations occur

$\uparrow$  Ventricle size  
 $\downarrow$   
New fluid equilibrium

No identified cause - meningitis, trauma, SAH etc may contribute

### CLASSICAL TRIAD

- Gait ataxia
- Cognitive impairment
- Urinary incontinence

insidious onset  $> 40y$  (elderly)  
min duration  $> 3-6m$   
progressive

PORENCEPHALY - focal loss of volume in an area of the brain  
( $\uparrow$  stroke / surgery)

$\downarrow$   
localised collection of CSF in the cavity

ARRESTED HYDROCEPHALUS - Ventriculomegaly

+  
No clinical features that warrant

- symptoms may develop eventually / after precipitating event  
 $\downarrow$   
intervention  
trauma / infection

# CLINICAL FEATURES OF HYDROCEPHALUS

## Infants (unfused cranium)

- ↑↑ Head circumference
- Fullness of anterior fontanelle ± bulge
- Prominent dilated scalp veins
- Sunset sign - due to tectal compression by dilated 3<sup>rd</sup> ventricle - downward deviation of eyeballs (PARINAUD'S SIGNS)
- irritability, refusal of feeds, vomiting, lethargy, drowsiness, coma

## Older children & adults

- no cranial enlargement as fontanelles are fused.
- ↓
- ↑ ICP, brain compression

### RAPIDLY PROGRESSIVE

- New onset headache
- vomiting
- visual disturbances
- papilloedema
- optic atrophy
- Seizures - acute transtentorial herniation
- Survivors
- hemianopia

### CHRONIC

- slower accumulation
- ↓
- Progressive dullness, apathy
- Memory impairment
- Gait disturbance
- Urinary incontinence

## EVALUATION OF HYDROCEPHALUS

### CT/MRI

Cranial USG in newborns / infants i open fontanelle

CT - Dilated ventricles - degree of ventriculomegaly  
tumor  
shunt  
central parenchyma - atrophy

MRI - IOC in newly detected hydrocephalus

→ better identification of site of obstruction  
small tumors / cysts  
as a prep inv before endoscopic 3<sup>rd</sup> ventriculostomy  
aqueductoplasty

Plain X Ray skull - sutural separation  
erosion of posterior clinoids  
'Beaten silver' skull - ↑ Convolutional markings

SPECT acetazolamide challenge

# MANAGEMENT OF HYDROCEPHALUS

Goal: reverse/halt the damage due to ↑ ICP  
reconstitute cerebral mantle to allow normal development  
(try to ↓ shunt dependency)  
redirect CSF flow if restoration is not possible

Medical management → only a temporary measure & used in combination w/ surgery  
ACETAZOLAMIDE - to ↓ CSF secretion  
FUROSEMIDE

Other temporary measures: → serial LPs in IVH in neonates until blood is absorbed & normal flow resumes  
→ ventriculostomy - until tumor is resected

## SURGERY FOR HYDROCEPHALUS

### NON-SHUNT PROCEDURES

1) ENDOSCOPIC THIRD VENTRICULOSTOMY  
- for obstruction at or beyond the aqueduct  
(AQUEDUCTAL STENOSIS / GLIOSIS  
4<sup>th</sup> VENTRICULAR TUMORS  
4<sup>th</sup> VENTRICULAR OUTLET OBSTRUCTION)  
creation of a FENESTRATION in the floor of the 3<sup>rd</sup> ventricle (between mammillary bodies & pituitary recess)  
↓  
Obstructed CSF is diverted into the BASAL CISTERNS

2) ENDOSCOPIC AQUEDUCTOPLASTY  
endoscopic recanalisation of the stenosed aqueduct

3) ENDOSCOPIC AQUEDUCTAL STENTING  
stent is placed & connected to subcutaneous reservoir

### SHUNT PROCEDURES

1) VENTRICULOPERITONEAL SHUNT  
Frontal / occipital horn of lat. vent  
↓ subcutaneous tunnel  
abdominal cavity

2) VENTRICULOATRIAL SHUNT

3) VENTRICULOPLEURAL SHUNT

4) LUMBOPERITONEAL SHUNT  
in communicating hydrocephalus  
· Pseudotumor cerebri

Lumbar → Peritoneal  
subarachnoid space → cavity

Shunts - Catheters  
+  
Valves  
+  
CSF reservoirs

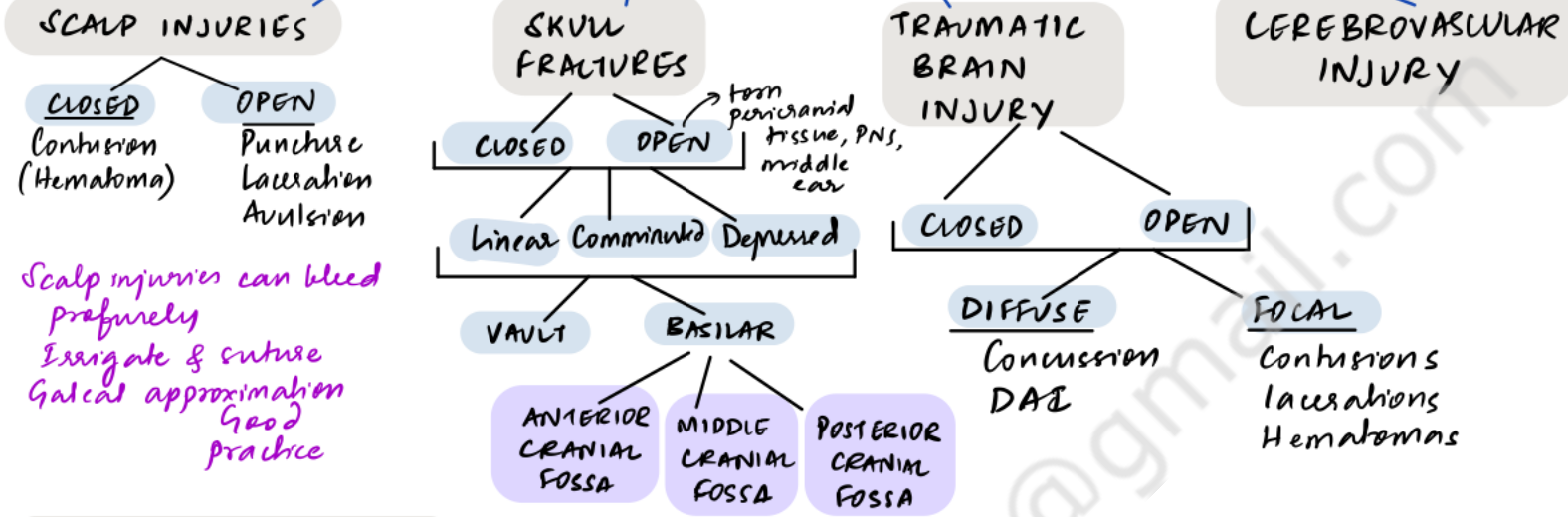
### SHUNT COMPLICATIONS

- 1) Overdrainage - low pressure headaches, accumulation of fluid/blood (subdural hygroma/hematoma)
- 2) Shunt ventricle syndrome - in children
- 3) Disconnection
- 4) Infection - ventriculitis, meningitis, peritonitis, wound inf, Staph aureus, epidermidis
- 5) Blockage
- 6) Distal complications - ascites, pseudoperitoneal cyst

# HEAD INJURY

## Causus

- MVA / RTA
- Falls
- Personal Violence
- Sports



Scalp injuries can bleed profusely  
 Irrigate & suture  
 Gagal approximation  
 Good practice

## SKULL FRACTURES

### Management

Evaluation - CT & bone window  
 R/o C-spine injuries, other injuries

### Treatment

- Linear #s - no special Rx
- Basilar #s - monitor & Rx of complications - CSF leak etc.
- Open #s - Irrig. TT  
 • Irrigate & debride  
 • Absx
- Depressed #s - result from ↑ force focal impact

- Elevation of #
- Repair of Dura
- Hemostasis

Be careful if # overlies Dural venous sinus

- Prophylactic anticonvulsants
- Surgical Rx - indications
  - Depth of depression thicker than calvaria > 3-10mm inward displacements
  - FND
  - Cosmetic defect (Frontal bone)
  - Open contaminated #s

## Complications of Skull Fractures

- Bleeding, CSF leak
- Pneumocephalus
- CSF leaks
- Meningitis
- CN Palsies (Basilar #s)
- Post traumatic epilepsy
- Caroticoavernous fistula - in sphenoid #s
- Traumatic aneurysms

• Temporal #s - Facial Palsy  
 Hearing loss  
 Vertigo  
 → Battle Sign

# TRAUMATIC BRAIN INJURY (TBI)

Non degenerative brain insult  $\Delta$  acute external mechanical force with associated altered state of consciousness, & temporary/permanent impairments of COGNITIVE, PHYSICAL, PSYCHOSOCIAL FUNCTIONS

## PATHOPHYSIOLOGY

Final Neurological status is a result of Primary & Secondary Brain injury

### PRIMARY BRAIN INJURY

- Immediate injury to neurons due to transmission of force of impact AT THE TIME OF INJURY

↓  
Mechanical cellular disruption  
microvascular damage

#### TYPES

- Diffuse shearing injuries
  - CONCUSSION
  - DIFFUSE AXONAL INJURY
- Contusions
- Lacerations
- Tears - Brainstem, Pituitary stalk, cranial nerves

### SECONDARY BRAIN INJURY

- Subsequent neuronal injury  $\Delta$  sequelae of trauma
- insults that occur AFTER TRAUMA & worsen the neurological status

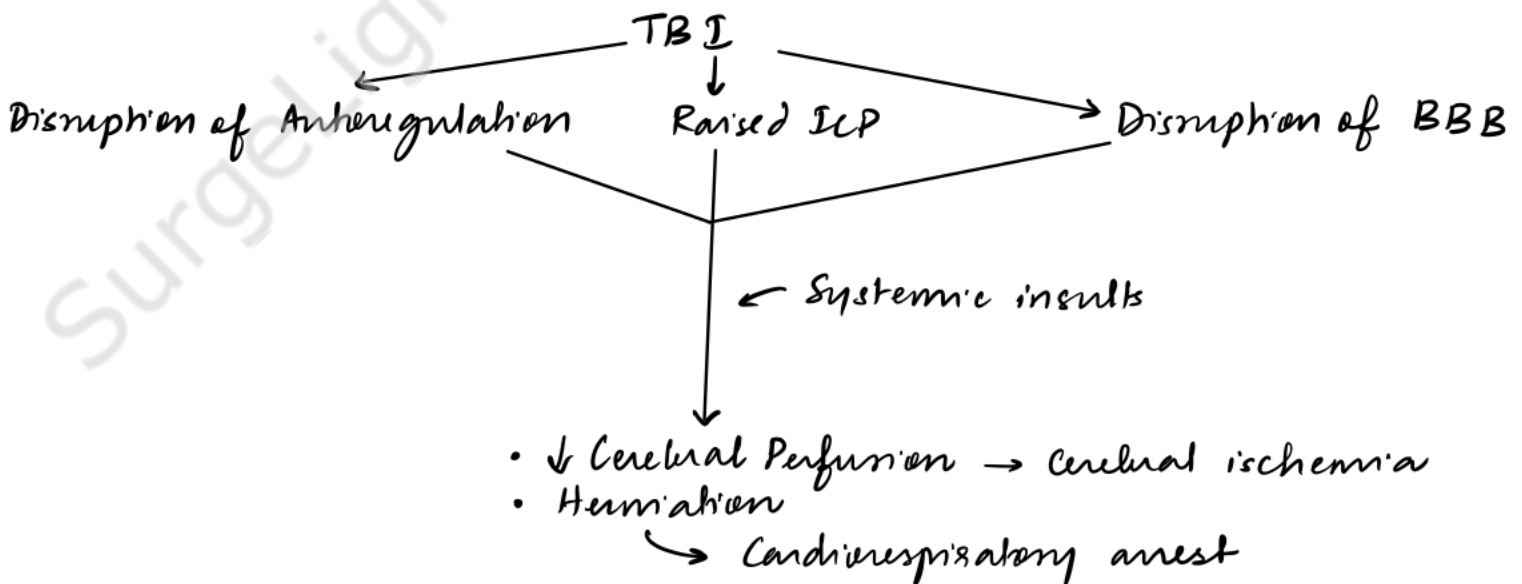
#### INSULTS

##### INTRACRANIAL

- RAISED ICP
  - Intracranial hematomas
  - Cerebral edema
  - Pneumocephalus
- VASOSPASM ( $\Delta$  SAH)

##### SYSTEMIC

- Hypotension
- Hypoxia
- Anemia
- Fever



## CONCUSSION

Temporary neuronal dysfunction following head trauma  
Biomechanically induced

### Clinically,

- Immediate & brief alteration of consciousness
- Brief disorientation
- Antegrade amnesia
- Dizziness, nausea, single episode of vomiting
- Lethargy/ irritability
- Headache
- Temporary FNDs - Asymmetry of corneal reflexes  
Nystagmus, Abn vestibular reflexes

Most pts are neurologically (N) (GCS-15) at presentation  
⇒ Dx is retrospective

### GRADES

- I - Confused temporarily; BVI, no memory changes
- II - Brief disorientation; Anterograde amnesia < 5 min
- III - LOC < 5 min; Retrograde amnesia
- IV - LOC - 5-10 min
- V - LOC - > 10 min

Repeated concussions → Traumatic encephalopathy



# DIFFUSE AXONAL INJURY

shear/stretch

Injury to axons throughout the brain due to rotational acceleration and deceleration

- can be considered a more severe form of concussion, often with irreversible consequences

## CHANGES

Stretch injury - at nodes of Ranvier - blebbing of nodal axolemma

Membrane injury → ↑ Ca<sup>2+</sup> influx

Axonal disruption → retraction of axons - Retraction balls

Axonal damage - **DURET HEMORRHAGES**

## PRESENTATION - LOC lasting > 6h

6-24h

mild DAI

usually only  
SUBCORTICAL AXONS  
in parasagittal white  
matter are  
involved

>24h

Severe DAI

↳ SUBCORTICAL AXONAL LESION  
+  
**CORPUS CALLOSUM** involvement  
+  
**BRAINSTEM INVOLVEMENT**

Imaging - CT is USUALLY NORMAL (small hemorrhages) → Delayed CT - edema/atrophy ±

∴ Poor neurological status → no prepositional CT abnormality

MRI - Abnormal signal in white matter  
Hemorrhages

R/O DAI

Mild DAI - good prognosis

Severe DAI - poor neurological outcomes  
Death

## CONTUSIONS

foci of hemorrhagic necrosis ON BRAIN SURFACE

as a result of contact/impact injury

COUP      CONTRECOUP

↓  
at site of impact

↓  
"# contusion"

↓  
at point at the poles opposite of impact

Edema around contusion → mass effect

## INTRACEREBRAL HEMATOMA

Focal collection of blood within the brain parenchyma

Usually a/c

- Hypertensive hemorrhage
- AVM Malformations
- Delayed traumatic intracerebral hemorrhage can occur within first 24h in pts w/ contusions

↓ - after 24h  
Reimaging - if slow worsening neuro status

## LACERATIONS

Focal area of brain trauma w/ torn

pia & arachnoid

↓  
Can occur due to penetrating trauma

↓  
missile      Non missile

Pial laceration → a/c SAH

## INDICATIONS FOR CRANIOTOMY in intra-axial hematoma / H'gic contusion

- Clot volume > 30 ml (50) irrespective of GCS
- Clot volume > 20ml w/ GCS (6-8)  
MLS > 5mm  
Basal Cistern Compression  
Posterior fossa
- Drop in GCS ≥ 2 points  
FNDS
- ICP > 20 mmHg

## SEVERITY OF HEAD INJURY

Determined by INITIAL (within 6-48 h of TBI) POST RESUSCITATION

[ No hypoxia  
hypotension  
hypothermia  
hypoglycemia  
Intoxication  
Sedation ] } GCS

Minor - GCS 15, no LOC  
Mild - 14, 15 + LOC  
Moderate - 9-13  
Severe - ≤ 8 } GCS

# MANAGEMENT OF HEAD INJURY

## A. INITIAL MANAGEMENT - ABC

### 1) AIRWAY

- Clear the mouth - FBs, vomitus, blood

- Oxygen

- Don't extend neck (C-spine!)

Jaw thrust & chin lift to improve airway

- OROPHARYNGEAL TUBE - GUEDEL'S AIRWAY

↳ to maintain airway patency

- ETT - intubation → RSS indicated to avoid ↑ ICP

to protect airway  
prevent aspiration  
allow hyperventilation  
SOS

Indications: • GCS ≤ 8  
• Penetrating head injury

Nasotracheal intubation

↳ ↓ neck manipulation

↳ avoid in • Facial #  
• Skull base #

- Cricothyrotomy / Tracheostomy

- Stabilize mandibular fractures

### 2) BREATHING

Spontaneous breathing - 100% O<sub>2</sub>

Poor / No spontaneous breathing - mechanical ventilation

### 3) CIRCULATION

Fluid Resuscitation - Target SBP >100 mmHg, <160 mmHg

Manage actively bleeding open wounds - Pressure / Suture

### 4) C-spine stabilisation - Collar

## 5) Primary assessment

- GCS
- Pupils
- Extremity motorics → spontaneous movements  
commands  
Reaction to pain

## 6) Secondary assessment

### Neurological exam

- Motor
- Brainstem
- Signs of ↑ ICP
- Sensory
- Cognition - MMSE

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## 7) IMAGING

- NCC head - Indications (NICE guidelines)

CT within  
1 hr

- 1) GCS < 13 at any point
- 2) GCS < 15 at 2 h
- 3) FND
- 4) Suspected Open / Depressed / Basal skull #
- 5) > 1 episode of vomiting
- 6) Seizure

1) Age > 65y

2) Coagulopathy - Antiplatelets / Anticoagulants

3) Dangerous mechanism - RTA, Fall from height

4) Retrograde amnesia > 30min

CT  
within  
8 hrs

# ICP management in head injury

ICP monitoring recommended in severe TBI (GCS < 8)

EVD > Intraparenchymal monitor

Mannitol vs Hypertonic Saline (20%)

↓  
Barbiturate high dose

↓  
Propofol

↓  
Therapeutic hypothermia

## Seizure management & Prophylaxis

### POST TRAUMATIC SEIZURES

#### IMPACT SEIZURES

at the time of injury

↓  
Acute brain reaction to trauma

↓  
Generalized convulsions

↓  
Do not ↑ risk of epilepsy

#### EARLY

within first day-1<sup>st</sup> week

- Requires urgent imaging

↑ Risk of post traumatic epilepsy

#### LATE

After 1 week

Gen - 6-18 m

↓  
single episode

#### EPILEPSY

Recurrent late seizures after trauma

Actively seizing: ANTICONVULSANTS: BZDs

### INDICATIONS FOR ANTISEIZURE PROPHYLAXIS

- GCS ≤ 10, Severe TBI
  - Cortical injury - acute SDH / ICH / contusions / lacerations / SAH
  - Depressed skull # & parenchymal injury
  - Early seizures, esp within 24h
  - Intubated pts
  - Post craniotomy
  - h/o - alcohol abuse  
past seizures
- } ↑ risk → may require continued AED

Phenytoin, Levetiracetam,  
Carbamazepine  
x 7d

## Discharge Criteria in Head injury - Minor/Mild

- GCS-15/15 & no FND
- Normal CT brain
- Not under influence of alcohol/drugs
- Accompanied by responsible adult

Admit  
the  
rest!

## ADVICE ON DISCHARGE

- Seek advice if/cto
- Persistent / worsening headache
  - Persistent vomiting
  - Drowsiness
  - Visual disturbance
  - Limb weakness / numbness

## COMPLICATIONS OF HEAD INJURY

### NEUROLOGICAL

- SAH - vasospasm - ischemia
- Cerebral thrombosis
- Delayed hemorrhage
- CNS infections
- CSF leaks
- Post traumatic hydrocephalus
- Post traumatic epilepsy
- Post concussion SO & Psychiatric disorders
- Long term neurological deficits

### NON-NEUROLOGICAL

- EARLY:
- Pneumonia
  - Atelectasis
  - Neurogenic Pulm edema
  - Stress ulcers
  - Bowel disturbances
  - Urinary disturbances

### LONG TERM:

- DVT, VTE
- Pressure sores
- Nutritional disturbances
- Contractures

Rehab ↓

Early mobilization

Nutritional rehab

Physiotherapy

Rehab — Speech, disability  
— Vocational

## INDICATIONS FOR SURGERY IN HEAD INJURY

- 1) Open skull fractures - scalp wound communicates w skull #
- 2) Depressed skull fractures
  - Depth of indentation  $>$  Calvarial thickness ( $> 3-10\text{mm}$ )
  - FNDs ⊕
  - Commis - Frontal bone #s
- 3) EDH →  $> 30\text{ml}$   
           $> 15\text{mm}$   
           $> 5\text{mm MLS}$  } irrespective of GCS
  - GCS  $\leq 8$  & anisocoria
  - Deterioration in GCS during conservative Rx
  - Posterior from EDH
- 4) SDH
  - Acute SDH -  $> 10\text{mm}$   
                  MLS  $> 5\text{mm}$   
                  Drop in GCS  $\geq 2$  points  
                                  since time  
                                  of injury
  - Chronic SDH  
                   $> 1\text{cm}$   
                  Any symptomatic  
                                  SDH
- 5) Intra-axial hematoma / hemorrhagic contusion
  - Volume  $> 20-30\text{ml}$   
          MLS  $> 5\text{mm}$   
          esp in temporal / posterior cranial fossa
- 6) Tension pneumocephalus
- 7) Medically refractory ↑ ICP

► Surgical intervention → usually pointless if GCS  $< 5$

# EPIDURAL HEMORRHAGE

Accumulation of blood between  $\leftarrow$  inner table of skull  
dural membrane

Etiology - Focussed blunt trauma to skull (DIRECT BLOW)

usually a/c skull # i/c line crossing vascular groove

BLEEDING FROM DURAL VESSEL

85% - high pressure arterial blood from lacerated meningeal artery

m/c  $\rightarrow$  Middle meningeal artery

Dissects dura away from the skull

15% - venous bleeding  
 $\downarrow$   
torn dural sinuses  
diploic veins  
meningeal veins

- slower course  
- m/c a/c depressed #s

Bleeding  $\rightarrow$  Dural separation from inner table  $\rightarrow$  Progression  $\rightarrow$  Brain compression  $\rightarrow$  Herniation

EDHs - attain max. size in min-hrs of injury - limited by tamponade effect  
clot in ruptured vessel

Hematoma extension is LIMITED by periosteal dural insertion at suture lines

## LOCATION

m/c - 60-80% - Temporoparietal - middle meningeal artery

10% - frontal - anterior meningeal artery, anterior ethmoidal artery

5% - Occipital

5% - posterior fossa - transverse/sigmoid sinus

m/c over the cerebral convexities

vast majority - occur ON THE SIDE OF IMPACT

## Presentation

Acute  
(60%)

Subacute  
(40%)

Chronic  
(10%)



## Clinical features

- Classical presentation (10-50%)

- initial transient LOC — due to 'concussion'



Lucid interval - several hours - subclinical expansion



Deterioration - due to ↑ ICP → herniation → Coma  
(↓ GCS)                      decompensation

- Uncal herniation from EDH

→ Blown pupil  
→ Ipsilateral third nerve palsy  
→ Contralateral hemiparesis } Not a rule

- Commonly a/i skull fracture, scalp hematoma

## IMAGING

### NECT head

- Bright, lentiform (biconvex), does not follow sulci, smoothly marginated



- heterogenous - active bleed

- Air in acute EDH ⇒ # of PNS / mastoid air cells

- midline shift, cerebral edema, herniation

Other inv - CBC, coag, RFT & S/E → to identify metabolic issues that may complicate Rx

## INDICATIONS FOR SURGERY

- EDH > 30 cc  
  > 15mm  
  > 5mm MLS } irrespective of GCS
- GCS ≤ 8 & anisocoria
- Deterioration during conservative management
- Posterior fossa EDH

Rx - Initial resuscitation

- ABC
- Spine care
- Maintain euolemia
- if ↑ ICP - Osmotic diuretics  
hyperventilation  
Elevation of head end
- correct coagulopathy

SURGICAL EVACUATION → definitive management

- Emergency burr hole decompression if neurosurgical facilities not available

- Procedure of choice - CRANIOTOMY → EVACUATION OF HEMATOMA  
↓  
Coagulation of bleeding sites  
↓  
Dura is hitched to bone

?? Endovascular embolisation of bleeding vessel

# SUBDURAL HEMORRHAGE

Collection of blood in the plane between Dura & arachnoid

M > F (3:1)

Older age  
m/c than EDH

## ETIOLOGICAL MECHANISMS

- 1) Vigorous head motion - acceleration-deceleration injury - esp older people  
Battered/Shaken baby syndrome
- 2) Spontaneous - coagulopathies  
Dural AVFs  
extension of intracerebral hemorrhage

NEED NOT BE A/C SKULL FRACTURE  
DIRECT IMPACT NOT NECESSARY

Movement of brain relative to skull

↓  
Shearing mechanism

Rupture of BRIDGING VEINS - cross subdural space from cortical surface to dural sinuses

[rarely - cortical artery  
oozing brain laceration]

↓  
Expansion of hematoma → ↑ ICP

Not limited by sutures

## RISK FACTORS

Brain atrophy

more space for

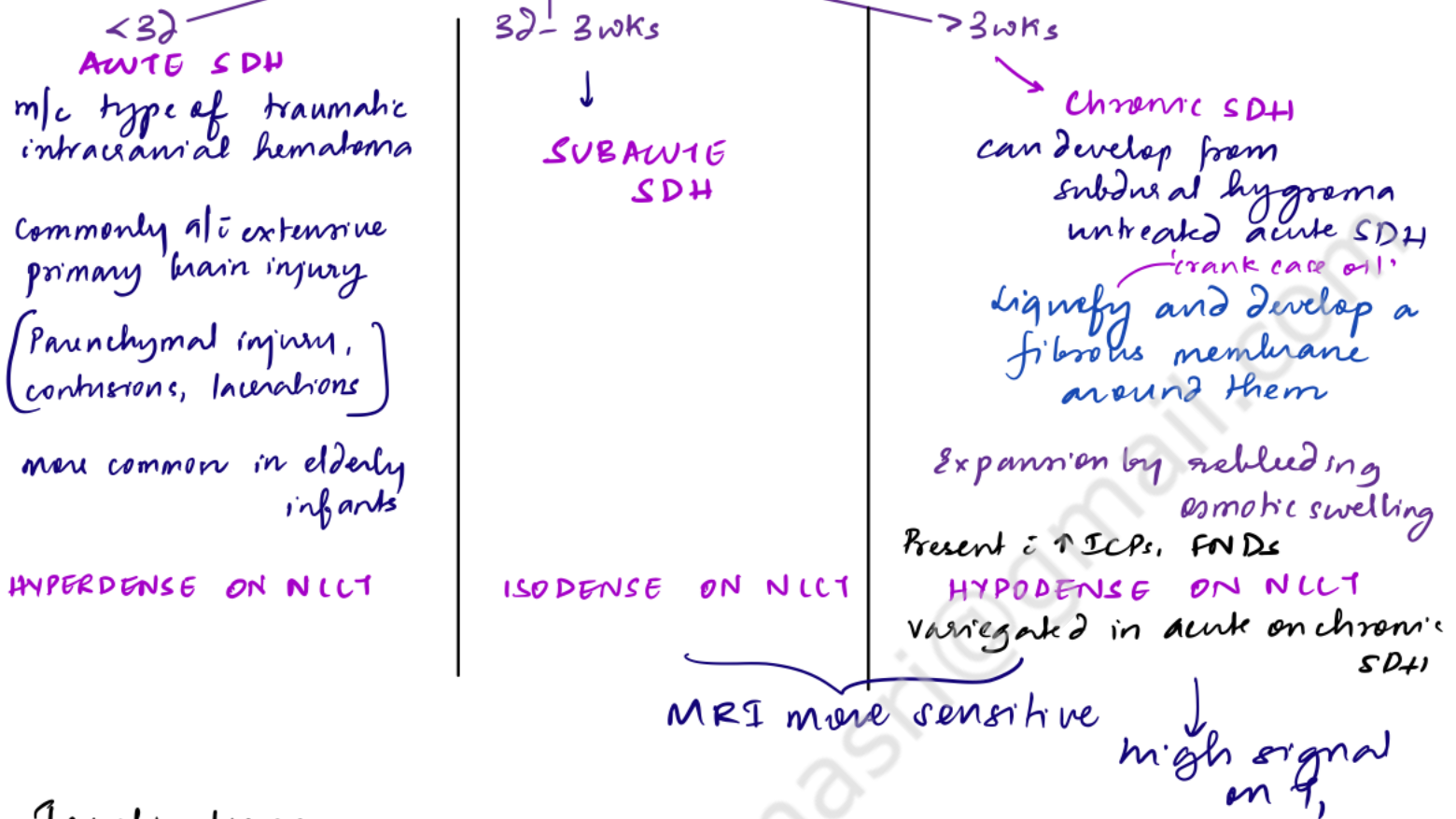
- brain to move
- hematoma to accumulate

Elderly  
Chronic alcoholism  
Dementia

## LOCATION

- along cerebral convexities - m/c frontotemporal
- along interhemispheric fissure & tentorium - Shaken baby
- posterior fossa

# PRESENTATION



## Investigations

- NCLT → concavoconvex configuration
- MRI - esp in subacute SDH

## Management

### Acute SDH

- Antiedema measures
- Seizure prophylaxis

Operative craniotomy for evacuation if thickness > 10mm

MCS > 5mm  
Drop in GCS by ≥ 2 point since time of injury

Non operatively managed

Rupture → Evolution to chronic SDH

Chronic SDH > 1cm  
any symptomatic SDH

↓  
Evacuate

Burr hole Craniostomy

active bleeding | Failure complex membranes (oculated)

↓  
Craniotomy + Evacuation ± Subdural/Subgaleal drains

# INTRACRANIAL ANEURYSMS & SUBARACHNOID HEMORRHAGE

## SUBARACHNOID HEMORRHAGE

Extravasation of blood into the subarachnoid space (between arachnoid & pia)

### ETIOLOGY

#### TRAUMATIC SAH

m/c type of traumatic intracranial hemorrhage

d/t

- rupture of subarachnoid vessels
- extension of an intraventricular hemorrhage along CSF flow

located typically in the - central sulci  
interhemispheric fissure  
sylvian fissure

#### NON TRAUMATIC SAH

- Intracranial aneurysms 75-90%
- AVM malformations 5-10%
- Benign perimesencephalic SAH rupture of small pontine/perimesencephalic veins
- Tumor bleeding
- Amyloid Angiopathy
- Drug abuse Cocaine

located typically in the **BASAL CISTERNS**  
Sylvian fissure - MCA / PComA

### ANEURYSM

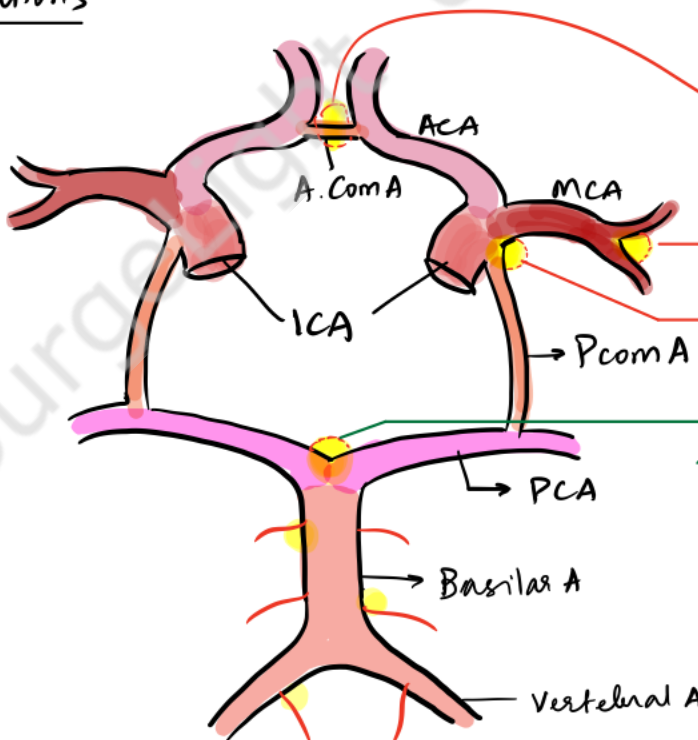
- focal dilatation in vessel wall

Saccular fusiform  
atherosclerotic / mycotic

dissecting infectious traumatic

SACULAR / BERRY ANEURYSMS - m/c - balloon-like outpouching (>90%)

### Locations



#### ANTERIOR CIRCULATION

- A Com A - 30-35% (85%)
- MCA bifurcation - 20%
- ICA - P. Com A 30-35%

#### VERTEBROBASILAR - 15%

- Basilar - 10%

Posterior fossa - 5%  
Vertebral artery (usually at PICA)

## Causes & risk factors of Berry aneurysms

ADPKD  
FMD  
NF-1  
Ehler's Danlos  
Marfan Syndrome

Advanced age  
Female gender  
Hypertension  
Smoking  
Cocaine abuse  
Family history

Intracranial As are susceptible to aneurysm development  
ditto  
: lack of external elastic lamina  
: very thin adventitia  
: unsupported location in subarachnoid space

Aneurysm sac is composed of only intima & adventitia  
media ends at junction of neck & parent vessel

### Aneurysm growth

- wall shear stress
- hypertension

Intracranial aneurysms are multiple in 10-30% cases; ↑ in females

Giant saccular aneurysm → > 2.5cm diameter

## CLINICAL FEATURES

- Aneurysms may be asymptomatic
- Minor aneurysmal hemorrhage - **WARNING LEAK / SEMINEL BLEED**  
headache, meningeal signs
- SAH → m/c presentation of intracranial aneurysm  
    ↳ peak incidence of aneurysmal SAH - 50-60y
  - Premonitory symptoms
  - Sudden excruciating headache - 'THUNDERCLAP' - localised / generalised
  - Altered mental status
  - Meningeal irritation
  - FND
  - Ocular hemorrhage - Subhyaloid / Prechinal hemorrhage
- Terson 50% - SAH + Vitreous hemorrhage
- Non hemorrhagic symptoms
  - Mass effect
    - Cranial neuropathies - III CN palsy by PCom A aneurysms
    - II CN compression by ophthalmic A aneurysms
    - Pituitary dysfunction - intrasellar aneurysms
    - Seizures
    - FNDs
- Emboli → TIAs

# GRADING OF SAH

## HUNT-HESS GRADING SYSTEM

- 0 - Asymptomatic, unruptured aneurysm
- 1 - AWAKE : Mild headache & nuchal rigidity / Asymptomatic
- 2 - AWAKE : Moderate-severe headache, CN palsy, nuchal rigidity
- 3 - LETHARGY : Mild FND (Pronator drift)
- 4 - STUPOROUS - Significant FND - hemiplegia
- 5 - COMA - posturing

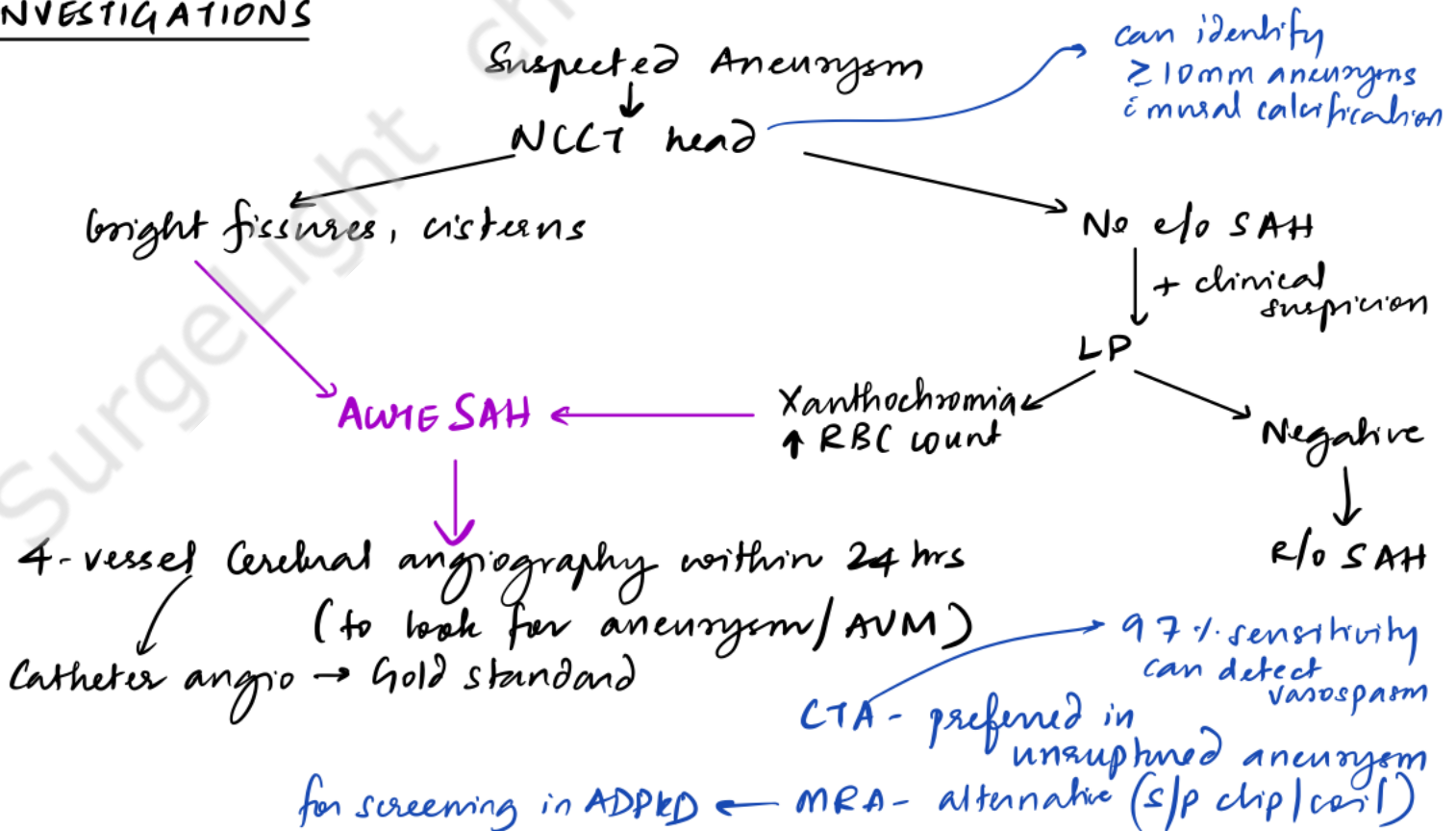
## WORLD FEDERATION OF NEUROLOGICAL SURGERY (WFNS) GRADING

- |   |              |                    |
|---|--------------|--------------------|
| 1 | GCS - 15     | } No motor deficit |
| 2 | GCS - 13, 14 |                    |
| 3 | GCS - 13, 14 | → Motor deficit    |
| 4 | GCS - ≤ 12   | } ± Motor deficit  |
| 5 | GCS - ≤ 6    |                    |

## FISHER - CT GRADING

- 1 No evident haemorrhage
- 2 Diffuse SAH < 1mm vertical thickness
- 3 Localised clots > 1mm thick
- 4 Diffuse / no SAH + ICH / IVH

## INVESTIGATIONS



## MANAGEMENT

### Rx of SAH

- ICU care, O<sub>2</sub>
  - Strict bed rest
  - Head end elevation 30°
  - Avoid straining
  - Anxiolytics, Analgesics
  - Seizure prophylaxis
- } General

### Low GCS

- EVD
- Mannitol if ↑ ICP

IV fluids - use only isotonic solutions (to ↓ cerebral edema)  
early aggressive fluid therapy can mitigate cerebral salt wasting  
Avoid hypovolemia

### Rx of VASOSPASM

- Nimodipine - CCB - 60mg Q4h  
or 30mg Q2h

→ start within 4d (96h) of onset

continue for 21d / until pt discharge in good neurological condition

- Statins
- Neuroprotective - MgSO<sub>4</sub>

### TRIPLE H THERAPY → for Symptomatic Vasospasm - controversial

Hypertension  
↓  
SBP ~ 180-220 mmHg  
MAP ~ 70-130 mmHg

Hypovolemia  
↓  
NS > 200ml/hr

Hemodilution  
↓  
hematocrit  
30-33%

Refractory vasospasm - intraarterial Verapamil / Nicardipine



## Rx of ANEURYSM

- within 24h → new consensus

### INTERVENTIONS

#### COILING

- Endovascular procedure
- Catheter advanced into parent artery
- Coils deployed to obliterate the aneurysm cavity
- Stent assisted coiling
- Balloon assisted coiling
- Flow diverting stents

#### CLIPPING

Aneurysmal vessel is dissected out & clip is placed across neck of vessel

#### Indications

- Wide aneurysm neck
- at bifurcations

#### Advantages

Better addresses mass effect of aneurysm

Excision & anastomosis may be done for giant aneurysms

## Management of Unruptured aneurysms

< 5 mm asymptomatic & no h/o SAH, ↓ risk of rupture

→ close observation

### Indications for intervention

- > 7mm
- 5-7mm aneurysm & risk factors for rupture - smoking, HTN, posterior circulation aneurysm, h/o SAH, family hx, multiple
- Young pts regardless of size
- Symptomatic aneurysm
- e/o aneurysm expansion -  $\geq 1\text{mm}$  ↑, change in configuration
- h/o previous rupture

# INTRACRANIAL PRESSURE

## 1) MONRO-KELIE DOCTRINE

Cranial Cavity - Fixed Volume

Components: Brain tissue, CSF, blood vessels, intravascular blood

↑ in one component → proportional decrease in one/more of the other components

if this fails

↓  
↑ ICP (↑ pressure per unit volume)

2) CSF is produced at a more or less constant rate of 20ml/min (almost independent of ICP)

## 3) Cerebral blood flow

30-100ml / 100g of brain tissue / min

- Autoregulation

If tissue demand exceeds autoregulation

↑ O<sub>2</sub> extraction → (Arteriovenous oxygen difference ↑)

↓  
< 0.25 ml / 100g of brain tissue / min  
: dysfunction

↓  
0.15 - 0.2 ml / 100g / min : reversible ischemia

↓  
< 0.15 ml / 100g / min - infarction

Complete cessation - Infarction within minutes

4) Ischemic injury → Tissue swelling → ICP → Vicious mechanism

Cerebral Perfusion Pressure = (Mean Arterial Pressure) - (ICP)

Must be maintained  
~ 50-70 mmHg

Must be kept  
below 20 mmHg

(N) - 0-20 mmHg

# NATURAL HISTORY OF RAISED ICP - 'Retrocaudal Delay'

- Focal distortion
- Effacement of Gyri and sulci
- Compression of ventricles
- Midline shift
- Subfalcine herniation
- Temporal lobe tentorial herniation
  - Third nerve compression
  - Obliteration of basal cisterns
  - Midbrain compression
  - Midbrain infarction
  - 'Duret hemorrhages' - small linear hemorrhages in midbrain & upper pons
- Further brainstem compression

## - Loss of Brainstem reflexes

Flexor  
↓  
Extensor response

Loss of: Corneal reflex  
Oculocephalic reflex  
Vestibuloocular reflex

## - Medullary Compression Syndrome

Respiratory reflexes

Vasomotor reflexes

CUSHING REFLEX - Bradycardia, hypertension, wide pulse pressure  
apnea → tachypnea

## - Foramen magnum herniation

## CLINICAL FEATURES OF ↑ ICP

- Headache, nausea, vomiting, visual disturbances
- Papilloedema (late sign)
- Abducens palsy - pseudolocalising sign

## CAUSES OF RAISED ICP

- 1) Space occupying lesions
  - Bleeding
  - Tumor
  - Abscess
- 2) Cerebral edema
- 3) Hydrocephalus
- 4) Venous sinus thrombosis, AVM → ↑ Intracranial blood volume
- 5) Miscellaneous
  - Pseudotumor cerebri
  - Craniostenosis
  - Altitude sickness
  - Hypervitaminosis A

## EVALUATION

- CT head → Erosion of lamina dura of Dorsum sellae
- Lumbar puncture X
- ICP monitoring
  - EVD / Ventriculostomy (lateral ventricle - frontal / occipital horn)
  - Subarachnoid bolt - Richmond screw / Philly bolt
  - Intraparenchymal device - microsensors

## MEASURES TO LOWER ICP

- 1) Elevate head end to 30° - improves venous outflow
- 2) Hyperventilation → hypocarbia → vasoconstriction
- 3) Antiedema
  - Mannitol
  - Hypertonic saline
  - Sodium Lactate
  - Glycerol

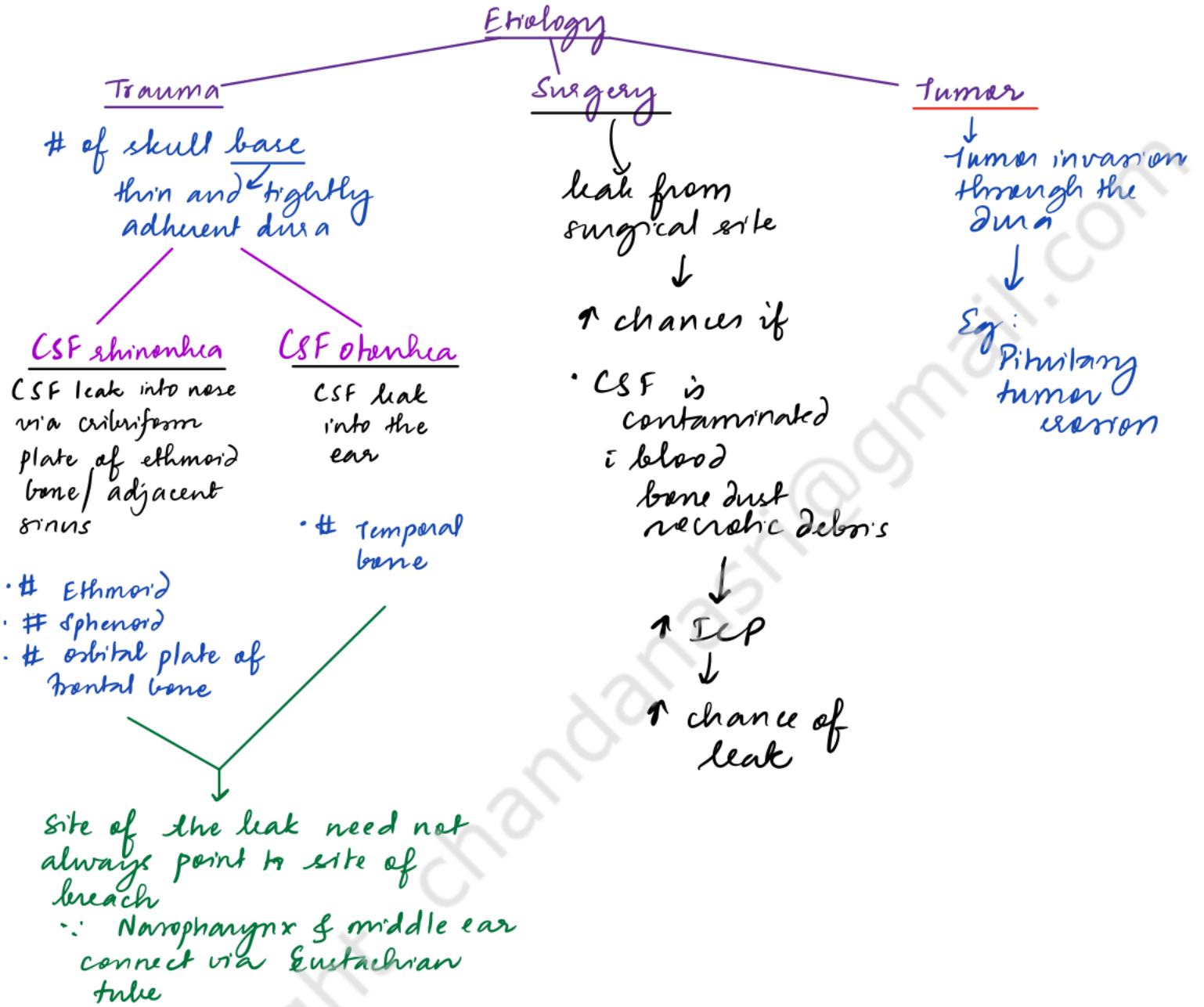
Acetazolamide  
Furosemide

↓ Production

Historically - urea
- 4) Drainage - EVD, Decompressive craniectomy

# CSF LEAKS

CSF leaks occur when DURA is violated. Predisposing factor: ↑ ICP



## Clinical Features

1) Watery discharge from nose / ear / surgical wound

↳ profuse  
'salty'

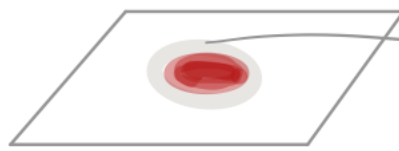
starts ~48h after dural breach. Traumatic → mucosal edema

may delay presentation

2) Orthostatic headache

## EVALUATION

1) Ring / Halo test - drop of discharge on paper



CSF - lower density forms a ring

2) CSF rhinorrhoea

- profuse, postural, cannot be sniffed back

Glucose concentration  $\left\{ \begin{array}{l} \text{CSF} - \geq 30 \text{ mg/dL} \\ \text{Nasal / Lacrimal secretions} - < 5 \text{ mg/dL} \end{array} \right.$

Chloride concentration  $\rightarrow$  CSF  $\gg$  nasal secretion

3)  $\beta_2$ -transferin - CSF specific  $\rightarrow$  most accurate

$\rightarrow$  takes 1-2 weeks

4) Radiology

CT - bone window

$\downarrow$   
#s, fluid in PNS

CT / MR / Radionuclide CSF cisternography to localise leak

## Complications of CSF leak

Poor wound healing  
Severe headaches -  $\uparrow$  ICP  $\downarrow$  ICP  
Recurrent bacterial meningitis

Prevention of post-op CSF leak - overscrubbing, vascularised pericranial flaps, dural sealants, prophylactic EVD

## TREATMENT

- Bed rest; Head elevation to  $\geq 45^\circ$  C to  $\downarrow$  Cranial CSF hydrostatic pressure
- $\downarrow$  CSF production - ACETAZOLAMIDE - controversial
- Pressure dressing
- Local Abs
- CSF drainage if leak persistent

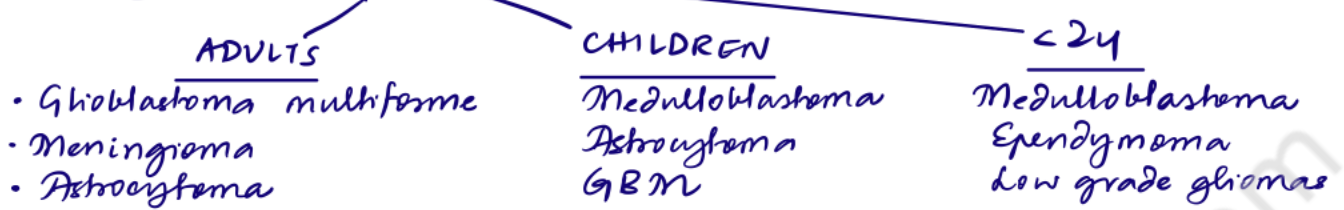
UNSUCCESSFUL CONSERVATIVE  $\rightarrow$  Sx - Craniotomy + reapproximation of torn dura & reinforced closure - fascial/pericranium, muscle graft

fails  $\rightarrow$  VP shunt

# BRAIN TUMORS

• Metastatic tumors are to 5-10x m/c than primary brain tumors

• 3 m/c Primary CNS tumors



## WHO CLASSIFICATION (2016) OF BRAIN TUMORS - SIMPLIFIED

1) Diffuse Astrocytic / Oligodendroglial tumors

Astrocytoma  
Glioblastoma

2) Other Astrocytic tumors - Pilocytic Astrocytoma

3) Other Gliomas - Choroid Gliomas

4) Ependymal cell tumors - Ependymomas

5) Choroid Plexus tumors - Choroid Plexus Papillomas / Carcinomas

6) Neuronal / Mixed Neuroglial tumors - Ganglioma

7) Pineal tumors - Pineocytoma / Pineoblastoma

8) Embryonal tumors - Medulloblastoma

9) Tumors of Cranial & Paraspinal nerve - Schwannomas, Neurofibromas  
MPNST

10) Meningiomas

11) Mesenchymal tumors - hemangiomas, Leiomyomas, Chondromas

12) Sellar Origin tumors - Craniopharyngioma

13) Melanocytic tumors - Melanoma (Meningeal)

14) Lymphomas - DLBCL, MALT lymphoma of Dura, ALCL

15) Histiocytic tumors - LCH, Erdheim Chester

16) Germ cell tumors - Teratoma, Germinoma, Yolk sac tumor,  
Choriocarcinoma

17) Metastatic tumors

## Cells of Origin

- 1) Neuroglia - Glial cells → Gliomas (50-60%)
  - Astrocytes → Astrocytoma, Glioblastoma multiforme
  - Oligodendrocytes → Oligodendroglioma
  - Ependymocytes → Ependymoma
- 2) Neurons / Neuroblasts
  - Ganglioneuroma
  - Neuroblastoma
- 3) Primitive neuroectoderm - Medulloblastoma
- 4) Choroid epithelial cells - choroid plexus papilloma
- 5) ARACHNOIDAL FIBROBLASTS - Meningioma
- 6) Endothelial cell - Hemangioma
- 7) Primitive notochord remnant - Chordoma
- 8) Pituitary cell - Adenoma
- 9) Pineal Parenchymal cell - Pinealocytoma
- 10) Schwann cell - Neurinoma / Schwannoma
- 11) Lymphocytes - CNS lymphoma
- 12) Primitive Germ cells - Germ cell tumors
- 13) Melanocytes - Melanoma

---

## HEREDITARY SYNDROMES & BRAIN TUMORS

(< 5% of 1° CNS tumors)

- |  |  |
|--|--|
| 1) Neurofibromatosis 1 & 2                 | 6) Retinoblastoma  |
| 2) VHL - Hemangioma                        | 7) Turcot Syndrome (GBM, medulloblastoma, colorectal polyps) |
| 3) Tuberosclerosis - Astrocytoma           | 8) Werner's Syndrome - Meningioma                            |
| 4) Li Fraumeni 5° - Gliomas, PNETs         | 9) Cowden's 5° - Cerebellar gangliocytoma                    |
| 5) MEN 1 - Pituitary adenomas, Schwannomas | 10) Gorlin 5°  |
|  | 11) Muir Torre 5°  |



# PRIMARY BRAIN TUMORS

## INTRA-AXIAL BRAIN TUMORS

- arise from within the brain parenchyma

Produce symptoms by 3 mechanisms

1) Tumor cells infiltrate along nerve cells & tracts  
↓  
Seizures

2) Tumor cells grow as a mass

Generalised symptoms  $\xrightarrow{SOL}$  Focal (FND) symptoms  
 $\Delta \uparrow$  ICP  $\downarrow$  compression

These symptoms resolve when tumor is resected

3) Destruction of adjacent neuroparenchyma  
- symptoms do not improve after treatment

## EXTRA-AXIAL BRAIN TUMORS

- arise from outside the brain parenchyma

- Compress adjacent brain parenchyma

- FNDs  
- Seizures

- Growth  $\rightarrow$  Mass

$\downarrow$   
SOL  
 $\uparrow$  ICP  
 $\downarrow$   
Symptoms

Tumor resection often restores the patient to normal neurologic state

Sy: Meningiomas

## Mechanisms of $\uparrow$ ICP in Intracranial neoplasms

1) Tumor mass - SOL

2) Cerebral edema adjacent to neoplasm

3) Obstruction of CSF pathways - hydrocephalus

4) Obstruction of venous pathways

$\rightarrow$  occurs earlier in posterior cranial fossa tumors

## CLINICAL FEATURES

### A. SYMPTOMS $\Delta$ + $\uparrow$ ICP

- Headache
- Vomiting
- Altered mental status
- Cushing reflex
- Mass shift  $\rightarrow$  False localising signs
  - Abducens palsy
  - CN 3 palsy
  - Hemiplegiaon one side } Kernohan's notch
- visual field defects
- Children  $\rightarrow$  tense fontanelle  
Macrocephaly

### B. SYMPTOMS $\Delta$ + tumor itself - focal brain dysfunction

- Seizures - Focal  $\rightarrow$  Generalised
- FNDs
  - hemiparesis/plegia
  - Sensory loss
  - aphasia
  - CN palsies
  - VFDs
  - Personality changes
- Pituitary/Pineal tumor - endocrinal issues
- Choroid plexus papilloma - CSF overproduction

### BASED ON TUMOR LOCATION

#### Supratentorial:

Frontal lobe: Seizures  
Intellectual &  
Personality changes  
Motor disturbances - Aphasia  
Anosmia

Temporal lobe - Personality changes  
Sensory aphasia  
Memory impairment

Parietal lobe - Gerstmann's, Apraxia

Occipital lobe - VFDs, Cortical blindness

Thalamus - Sensory disturbances

#### Posterior Cranial Fossa

• Early CSF flow obstruction  
- hydrocephalus

• Projectile vomiting

• CN dysfunction

• Long tract signs

m/c brain stem tumor

- astrocytoma

## INVESTIGATIONS

- CT
- MRI  $\pm$  Gadolinium, DWI
- MR Spectroscopy
- Angiography

## Pathology

### Gliomas

WHO Grades	
↓ By grade of anaplasia	<u>I</u> - Pilocytic astrocytoma - almost only in kids
	<u>II</u> - Astrocytoma / Oligodendroma
	<u>III</u> - Anaplastic Astrocytoma / Oligodendroglioma
	<u>IV</u> - Glioblastoma multiforme

## TREATMENT

- 1) Dexamethasone  $\rightarrow$   $\downarrow$  cerebral edema
- 2) Surgical Resection - whenever feasible
  - $\rightarrow$  adequate tissue sampling for Dx
  - Neurological improvement  $\Delta$   $\downarrow$  mass effect
  - Degree of resection correlates  $\pm$  survival
- 3) RT - improves survival
  - 50-60 Gy
  - Stereotactic Radio Surgery**
- 4) Chemo - Temozolomide for GBM

# MENINGIOMA - 15-20% of intracranial tumors

- derived from Arachnoid Cap cells - Fibroblasts of the arachnoid matter

F > M 2:1 ; adults > children → NF-1

Intracranial locations : Along the falx cerebri  
Along the convexities  
Sphenoid wing

Others: Foramen magnum  
Olfactory groove  
inside lateral ventricles

Slow growing, encapsulated, benign tumors

Atypical aggressive meningiomas - invade adjacent bone / cerebral cortex

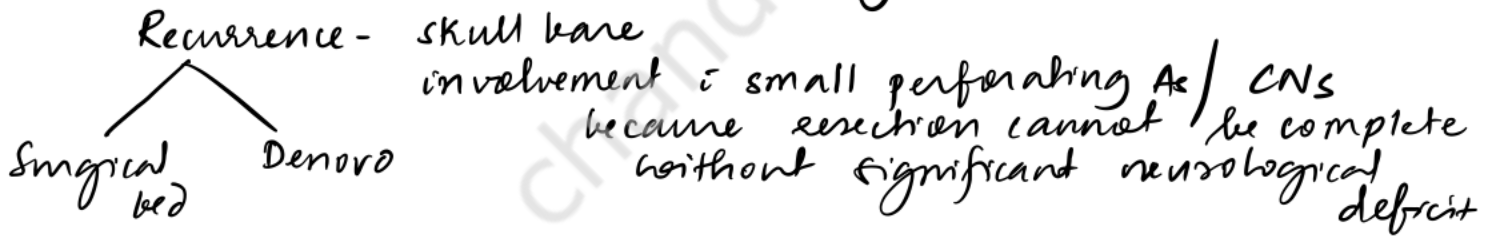
→ < 1%

Require post-op RT

MRI - 'Dural based' tumors

Meningioma 10% pk - multiple meningiomatosis

Surgical resection is essentially curative



\* Simpson grading scale - for extent of resection

I - Complete (including dural attachment + abnormal bone)

II - Complete & cauterization of dural attachment

III - Complete - cont dural attachment

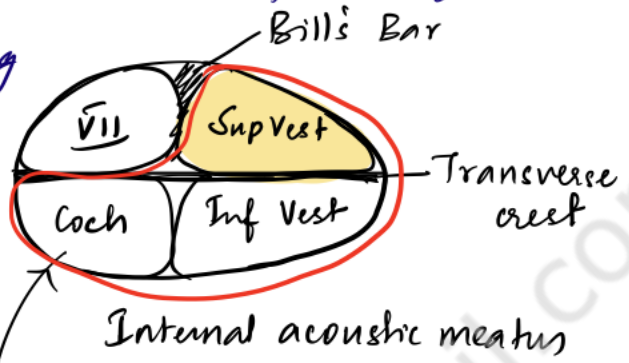
IV - Incomplete

V - Biopsy

# ACOUSTIC NEUROMA

## Vestibular Schwannomas

- predominantly arise from **SCHWANN CELLS** of the superior vestibular portion of the vestibulocochlear nerve
- Benign, encapsulated, slow growing
- 80% of all CP angle tumors
- 10% of all brain tumors
- B/L in NF-2



## Tumors

### Intracanalicular

Erosion of the canal

CN VII involvement

CN VII involvement

Sensory > Motor

- Posterior meatal wall numbness
- ↓ taste

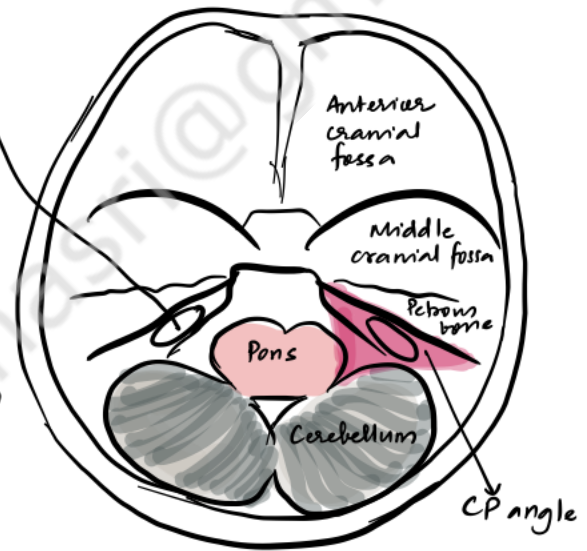
### CP angle

Other cranial nerves IX, X, XI

Brainstem displacement

Cerebellar displacement

↑ ICP, Hydrocephalus



Evaluation: MRI w/ gadolinium - Gold std

✓ PTA, BERA

✓ CN examination

### APPROACHES

- Middle Cranial Fossa
- Translabryithine
- Suboccipital

Rx → Microsurgery - preferred for tumors > 3cm

SRS / Gamma Knife Surgery - < 3cm

# FACIAL NERVE

- Spl visceral efferent (Motor)
- General visceral efferent (Parasympathetic)
- Special visceral afferent (Taste)
- General somatic afferent (Touch)

Stylomastoid foramen

Facial canal

Internal auditory meatus

Facial (N) Nuclei

Abducens nucleus

Motor nucleus

Superior salivatory nucleus

Nucleus tractus Solitarius

Spinal nucleus of CN V

Nervus intermedius

GENICULATE GANGLION

Greater Petrosal N

Pterygo Palatine ganglion

Lacrimal gland  
(Nasal, palatine, pharyngeal glands)

General Sensation from

- Concha
- Posterior superior EAC
- Tympanic Membrane

Submandibular ganglion

Submandibular gland

Sublingual gland

Terminal branches

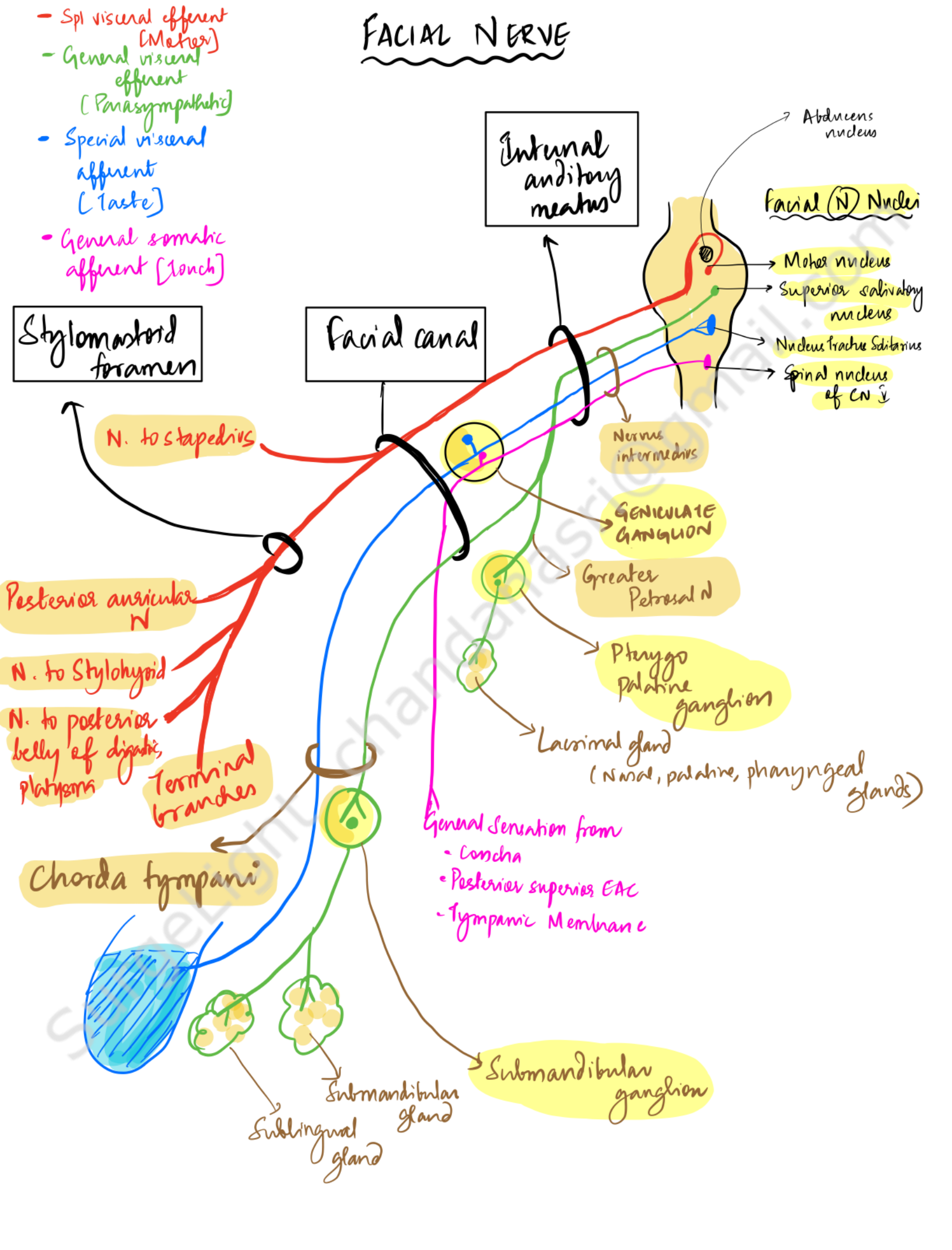
Chorda tympani

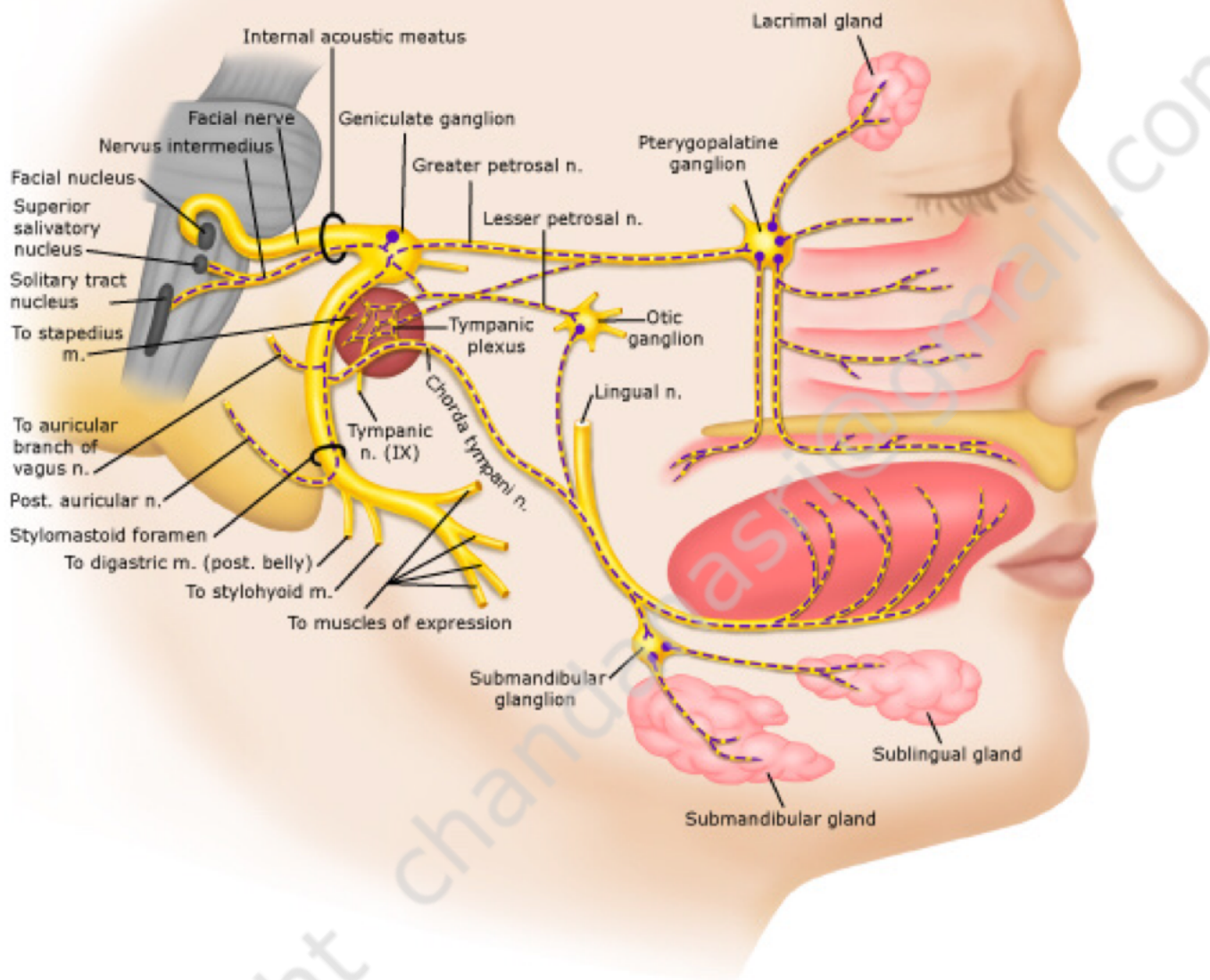
Posterior auricular N

N. to Stylohyoid

N. to posterior belly of digastric, platysma

N. to stapedius





SurgeLight chandrashekar.com

# MANAGEMENT OF FACIAL NERVE INJURY (SURGICAL)

## STATIC

- Suspension surgeries using temporal fascia
  - suspension of angle of mouth from zygomatic bone using temporal fascia
- Medial canthus reconstruction to reduce epiphora
- Lateral tarsorrhaphy to prevent exposure keratitis due to widened palpebral fissure
- Upper eye lid weights - gold weights

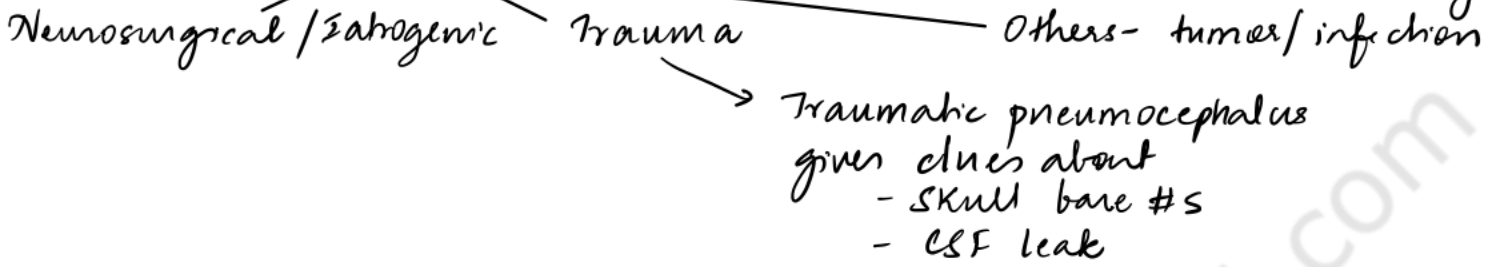
## DYNAMIC

- Muscle transfer
  - temporalis to masseter
- Free muscle graft
  - ↓
  - Gracilis neurovascular transfer
- Cross facial nerve transplant
- Nerve grafts
  - Greater auricular N
  - Sural nerve
  - Lateral cutaneous nerve of thigh



# PNEUMOCEPHALUS

- air in the intracranial cavity
- Results from a defect in the cranium that allows air into the cranial cavity



## Mechanisms of development of pneumocephalus

### Ball valve mechanism

Defect in the skull acts as one-way valve

Rx - avoid positive pressure ventilation

Flat position to minimize air travelling upwards into cranial cavity

### Inverted bottle mechanism

Air enters the intracranial space  
d/t negative pressure generated by traumatic / iatrogenic CSF leak

- Barian skull #s involving mastoid air cells

Rx - Head end elevation  
Controlled CSF diversion  
Definitive repair of skull base defect

Non tension pneumocephalus → resolves on its own by absorption into blood stream

Supplemental 100% O<sub>2</sub> → ↑ rate of resorption by ↑ diffusion gradient of

N<sub>2</sub> - predominant intracranial air pocket & the blood stream

→ Neurosurgical emergency

TENSION PNEUMOCEPHALUS: Intracranial air pocket under tension

↓  
MOUNT FUJI SIGN

AIR BUBBLE SIGN

- focal air pockets in subarachnoid spaces

↓  
↑↑ ICP → herniation (worsening) (CS)

Rx - Burr hole

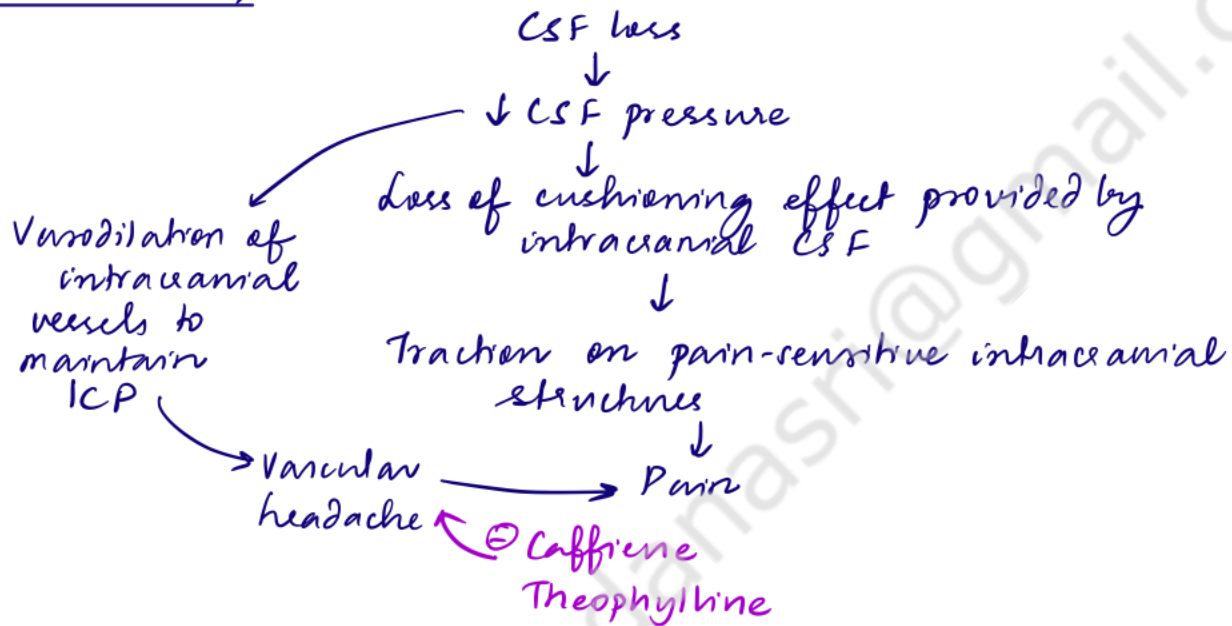
# POST DURAL PUNCTURE HEADACHE

- complication of neuraxial anaesthesia - can occur following
  - spinal anaesthesia / subarachnoid block
  - inadvertent dural puncture during epidural analgesia

RISK FACTORS: Female gender  
Young age  
Obstetric procedures

Larger dural tears  
Cutting needles > Pencil/Blunt needles

## PATHOPHYSIOLOGY



## CLINICAL FEATURES

- Dull throbbing pain - frontal-occipital distribution
  - Aggravated by erect posture
  - Relieved by recumbency
  - Appears within 5 days of Dural puncture
  - Relieved within 1 week
  - within 48h of epidural blood patch
  - Headache may be a/c - tinnitus, photophobia, nausea
- R/o - Subdural hematoma, Cerebral thrombosis

TREATMENT: Bed rest, hydration, Caffeine, Analgesics - PCT  
Gabapentin  
Sumatriptan  
Cerebral vasoconstrictors

Invasive - Epidural blood patch / Epidural injection of colloids

## SPINAL DYSRAPHISM

Neural tube forms from the neural plate at 21 days of gestation

Failure → Neural tube defects: Spinal dysraphism

Folate supplementation starting before conception - helps prevent  
Screening - USG, amniotic fluid ↑ AFP

## SPINAL DYSRAPHISM

### SPINA BIFIDA APERTA

↓  
'Open' defects

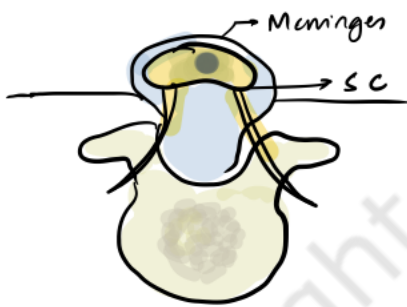
- Meningocele
- Meningomyelocele
- Myelocele
- Syringomyelocele

### SPINA BIFIDA OCCULTA

- Simple spina bifida occulta
- Spinal dorsal sinus
- Lipomyelomeningocele
- Diastematomyelia
- Tethered spinal cord
- Several types may co-exist

MENINGOMYELOCELE → m/c type of Spina bifida aperta

- Protrusion of varying amount of spinal neural tissue outside the confines of the spinal canal



- Deficiency of skin, muscle & bony elements

- Open neural placode - exposed anywhere from thoracic to sacral level

- Neurological deficit - sensory, motor, autonomic

Thoracic → Extensive defects  
Sacral → Depends on level  
↳ Only bladder involvement

• 80% a/i hydrocephalus → ↑ in thoracic defect, ↓ in sacral defects

• 90-95% a/i Chiari II malformation

• Associated brain anomalies - Corpus Callosal anomalies  
Fused tectal plates  
Thalamic fusion

Rx - Surgical closure of meningocele is undertaken within 24-48hr → to prevent infection (meningitis, ventriculitis)

Before closure - child is nursed prone  
Moist sterile dressings  
Prophylactic antibiotics

Sx - All exposed neural tissue is considered viable unless proven otherwise

Neural elements must be carefully separated from the cutaneous elements (to prevent inclusion hernia)

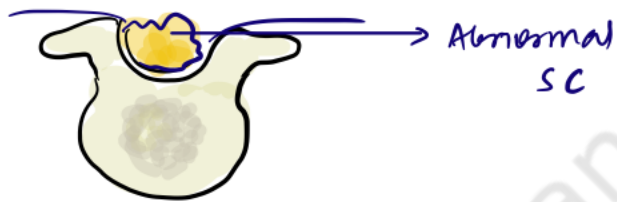
Dura is closed in watertight fashion

Myofascial closure

Skin closure — Large defect - SSG  
— Small defect - 1° closure

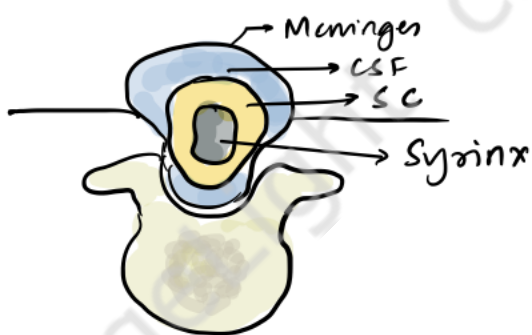
Combined c shunt placement & Chiari decompression if required

MYELOCELE: a/c arrested development of spinal cord



most cases are still-born  
↑ mortality

SYRINGOMYELOCELE



central canal of SC is dilated

rarest variant of spina bifida

gross neurological deficits ⊕

MENINGOCELE = protrusion of dura & arachnoid outside the confines of the spinal cord

Neural tissue remains within spinal canal

No neural elements = No neurological deficits = Simpler repair

m/c in lumbar region

# SPINA BIFIDA OCCULTA

## 1) SIMPLE SPINA BIFIDA OCCULTA

Posterior lumbar bone defect  
No symptoms/deficits

## 2) DERMAL SINUS

Track from skin to subarachnoid space  
- cutaneous 'dimple' / pit  
m/c in lumbosacral region

Complications - ascending infection  
tethering of cord as child grows

may be a/c intraspinal tumors - dermoids  
lipomas

Rx - track excision & untethering of cord

## 3) DIASTEMATOMYELIA - Spinal cord is SPLIT into 2 HEMICORDS by a bone / fibrous band that tethers the cord

↓  
prevents free movement / ascent of cord

a/c tuft of hair

Needs surgical repair

## 4) LIPOMENINGOMYELOCELE - fatty tissue within spinal canal

↑ incidence of  
post-op neurological  
deficits

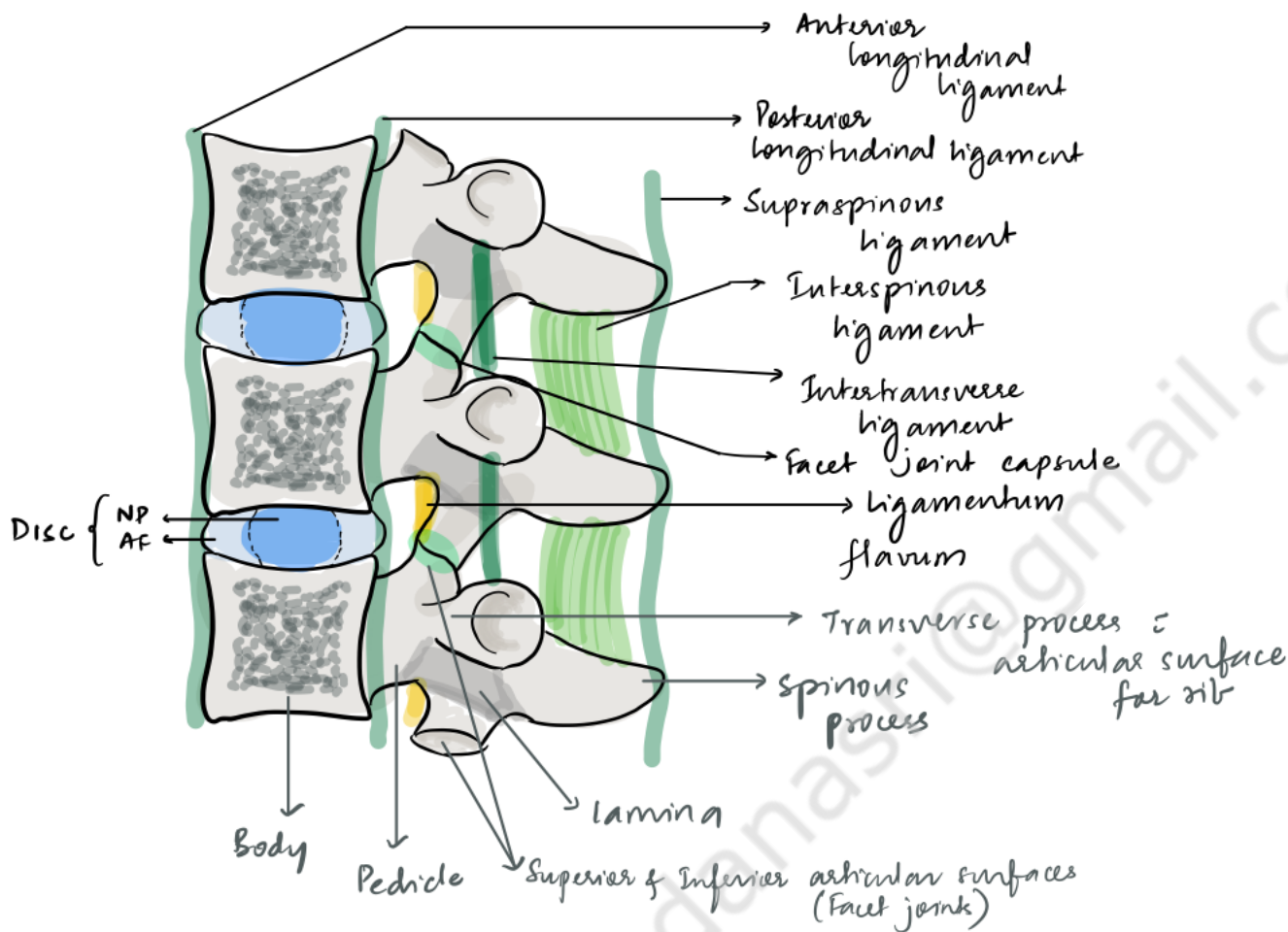
cord tethering  
Large dural defect  
Neurological defects

MEMBRANA REUNIENS - a fibrous band connecting skin to the spinal theca

↓  
cord tethering

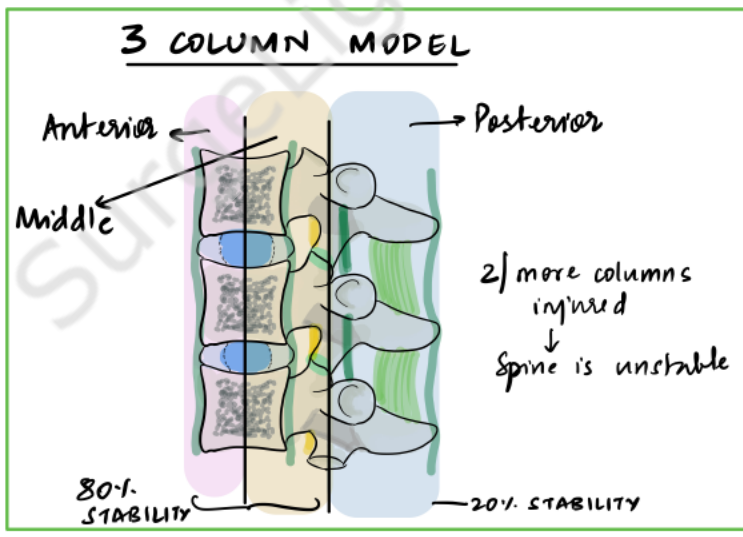
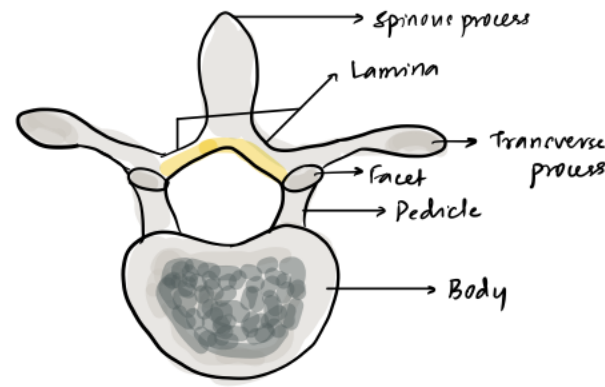
# SPINAL TRAUMA

## ANATOMY



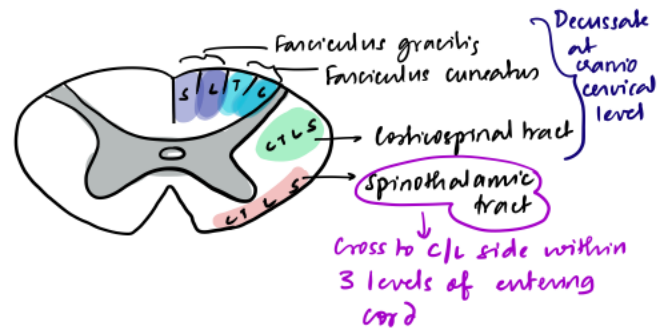
7	CERVICAL
12	THORACIC
5	LUMBAR
5	SACRAL
4	COCCYGEAL
<b>33</b>	<b>VERTEBRAE</b>

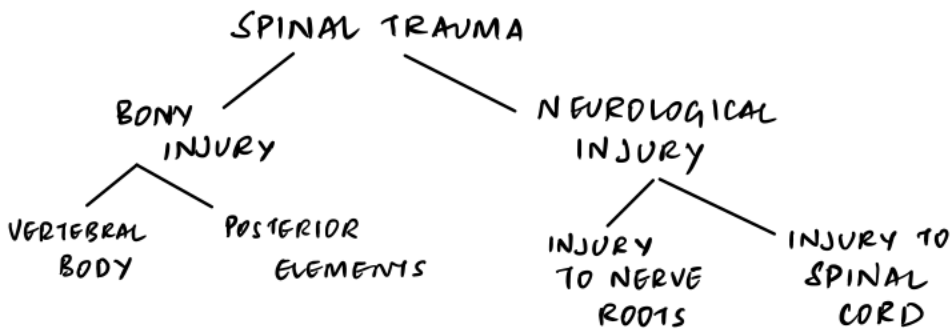
Spinal level	Vertebral level
Upper cervical	Same level
Lower cervical	Spinal level - 1
Upper thoracic	Spinal level - 2
Lower thoracic	Spinal level - 3
Lumbar	Spinal level - (3-5)
Sacral / Cx	Spinal level - (6-10)



DENIS- HOLDSWORTH CONCEPT

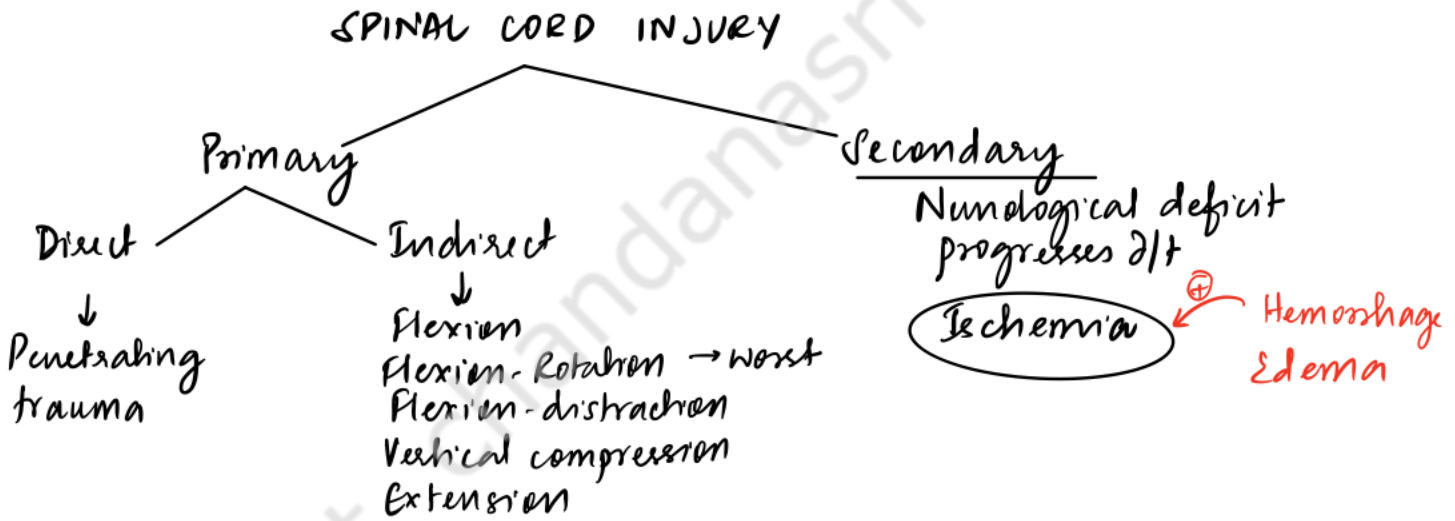
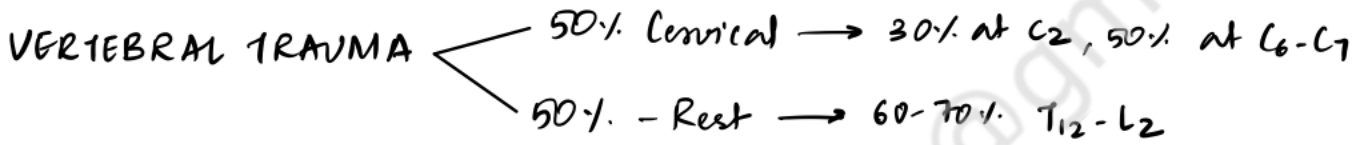
Spinal Cord - extends from Foramen magnum to L1-L2 level  
 T12-L1 - Conus medullaris





Causes of Spinal trauma

- RTA
- Fall from height
- Violence



**SHOCK IN SPINAL TRAUMA**

Hypovolemic shock : d/t hemorrhage - requires volume resuscitation

Neurogenic shock -  $\uparrow$ HR/Bradycardia + Warm peripheries  $\rightarrow$   $\downarrow$  Vascular tone - due to  $\uparrow$ /UNAPPPOSED VAGAL TONE

d/t sympathetic interruption - trauma at/above the level of symp outflow

**Spinal shock** - temporary physiological disorganisation of spinal cord function

PARALYSIS  
HYPOTONIA

HYPOREFLEXIA

Starts within minutes of injury  
can persist upto 6 weeks

resolution

Bulbo cavernosus - first reflex to return

## INITIAL MANAGEMENT

- ABCs

- Immobilization  $\left\{ \begin{array}{l} \text{Spine Board} \\ \text{Rigid cervical collar} \end{array} \right.$

- Careful transportation

- Spinal log roll - i min 3 people

- Take care while removing helmet

- Hypotension correction

- ↓
- LL elevation
- IV fluids

- Airway - Nasotracheal int

↓  
Orotacheal int

↓  
Cricothyrotomy → Tracheostomy

- PR - anal tone

perianal sensation → ⊕ → incomplete spinal injury

In every case of suspected C-spine injury

↓  
Predictors of C-spine trauma

- Impaired consciousness / intoxication
- significant head / facial trauma
- suggestive mechanisms of injury
  - RTA, FHT
- neck deformation / tenderness
- unexplained hypotension
- Diaphragmatic breathing
- Extremity FNDs

## CLINICAL FEATURES

• External injuries - s/o mechanism of trauma

• Spinal Exam - deformity / gap / step / tenderness

## NEUROLOGICAL EXAMINATION

- Respiratory paralysis
  - hemi / para / quadriplegia / plegia
  - Reflexes: Areflexia → Hyperreflexia
  - Sensory level
  - Autonomic - Bowel & Bladder disturbances
- } Motor

Spinal syndromes : Ant / Post / Central Cord S<sup>o</sup>

Brown Sequard S<sup>o</sup>

Spinal transection  $\left\{ \begin{array}{l} \text{Complete} \\ \text{Incomplete} \end{array} \right.$

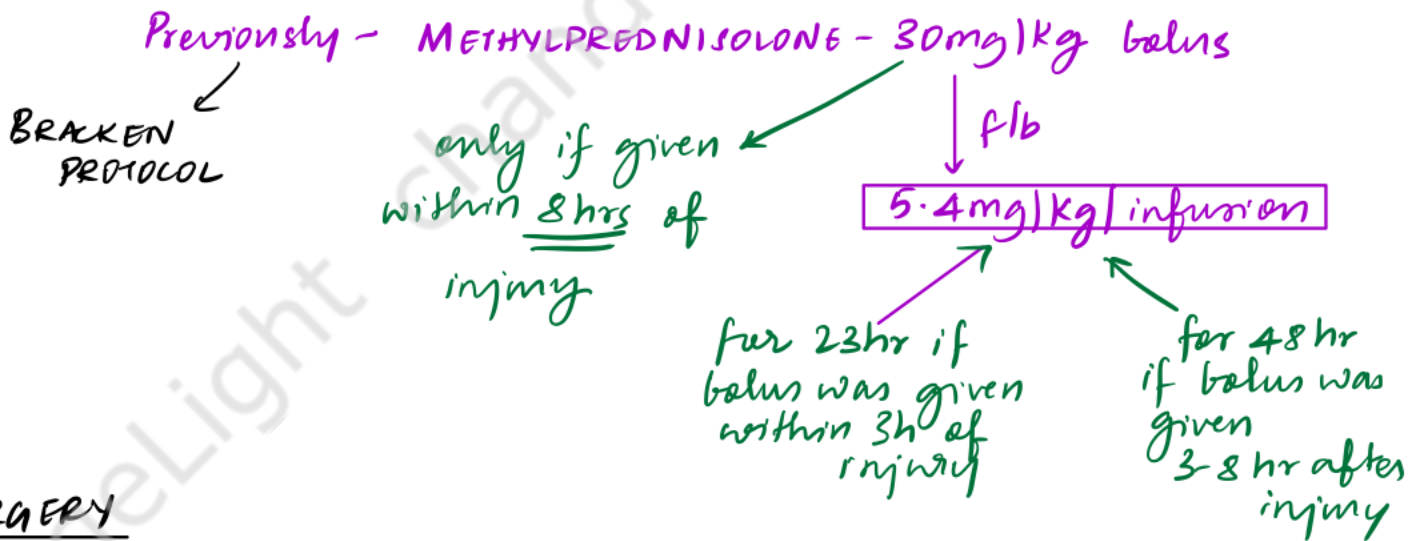


## IMAGING

- XRay Spine
  - AP
  - Lateral - Assess prevertebral soft tissue swelling
  - CTIS
  - sagittal alignment
  - translation
  - Angulation
  - Chematoma
- CT - bone & soft tissue changes, myelography - cord compression
- MRI - cord injury
  - indicated when neurological deficits ⊕
  - Cord edema
  - contusions

## TREATMENT

- ABCs etc
- Neuroprotection → currently, data does NOT recommend spinal dural steroids



## SURGERY

- Decompression → Realignment → Stabilization
  - ↑ Orthotic devices

## REHAB

COMPLICATIONS - Progressive neurologic deficit  
Post traumatic syringomyelia

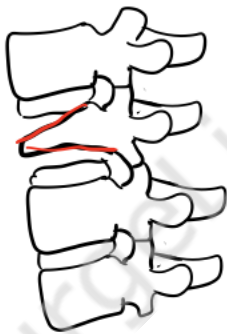
## SPECIFIC INJURIES

### C-SPINE

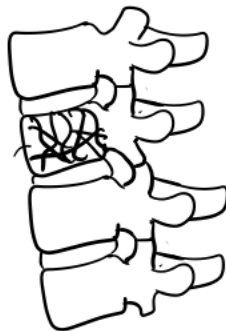
- 1) Jefferson # - Bursting # of ring of C1 (atlas) -  $\geq 7\text{mm}$  dislocation  
     $\Rightarrow$  transverse ligament disruption (Rule of Spence)  
    Sx - not indicated
- 2) DONTOID #s - C2 (axis) protrusion  
    Sx for widely displaced #s
- 3) HANGMAN'S # - hyperextension/distraction injury  
    B/L C2 pars interarticularis #s  
    Sx - only if no spinal cord compression / failed external mobilization
- 4) Jumped Facets - Hyperflexion + rotation/distraction C-spine injury
- 5) Clay Shoveller's # - Avulsion # of C7 spinous process

### T-L SPINE

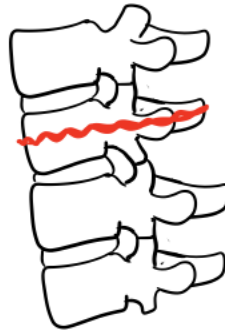
Compression #



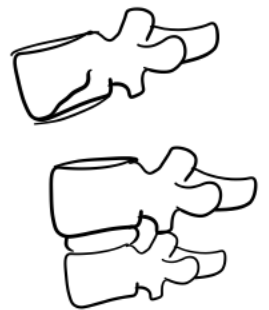
Burst #



Chance #



# - Dislocation



Due to axial compression forces

Due to distracting forces

↓  
due to translational + distracting forces

# SPINAL TUMORS

- ~20% of CNS tumors occur in the spine
- Majority - histologically benign

## SPINAL TUMORS

### EXTRADURAL TUMORS (55%)

- **BONY VERTEBRAL TUMORS** ← vertebral body  
Posterior elements
  - Metastases (m/c)
  - Multiple Myeloma
  - Lymphoma
  - Chordoma
  - Hemangioma
  - Giant Cell tumor
  - Osteosarcoma/Osteoma
- **PERIPHERAL NERVE SHEATH TUMORS**

### CLINICAL FEATURES

- Bony Vertebral lesions - gradually worsening focal back pain, unresponsive to rest, ↑ in night recumbency, worsened by Valsalva, percussion tenderness ⊕
- Bone pain - Bony destruction  
Pathological fxs
- Neuropathic pain - root/meningeal irritation
- PNSTs - Extramedullary nerve root > Cord compression  
↓  
Radiculopathy (earlier) >>> Myelopathy (later)

### INTRADURAL TUMORS

#### EXTRAMEDULLARY (40%)

- Meningioma
- Papillary ependymoma
- Schwannoma
- Neurofibroma
- Dermoid

### CLINICAL FEATURES

- Extramedullary Cord / Root compression  
↓  
• Myelopathy (Meningiomas)
  - Brown Sequard s<sup>o</sup>
  - Complete loss
- Compression first affects long tracts lying in peripheries of the cord
- Radiculopathy (PNST)  
Pain & Paresthesias

#### INTRAMEDULLARY (5-10%)

- Ependymoma
- Subependymoma
- Astrocytoma
- Glioblastoma
- Hemangioma
- Metastasis

(>90% - benign)

### CLINICAL FEATURES

- Progressive myelopathy  
↓  
mimics syringomyelia
- CENTRAL CORD s<sup>o</sup>
- U/L (Brown Sequard)  
↓ progresses  
B/L (when tumor enlarges)
- Neurological deficit below level of lesion
- Sensory loss & sacral sparing
- Sphincter dysfunction
- HYDROCEPHALUS ⊕ in malignant tumors

Evaluation -> Plain X-Rays - insensitive, non specific

#### EXTRADURAL

- Osteoblastic / Osteolytic lesions (VB)
- Pedicular erosion - enlarged intervertebral foramen
- VB Collapse

#### EXTRAMEDULLARY

- Enlarged spinal canal
- Calcification

#### INTRAMEDULLARY

- Spinal canal widening
- Posterior scalloping of vertebral bodies

2) MRI spine (MRI > CT)

3) CT - delineates primary bone tumors

4) Myelography - helps distinguish extra & intradural

5) Bone scans - when VB lesions suspected

6) Spinal angiography

when hemangioma is suspected  
planned angioembolization of vascular lesions

LP - only if leptomeningeal spread is being evaluated

## TREATMENT

Rx of Spinal Cord Compression

1) Steroids - Dexamethasone - 4-24mg Q6h  
or

Methyl prednisolone

2) Emergency RT - 30Gy in 10 fractions

3) Surgical decompression

Indications - Cord compression ↑ despite steroids  
& RT  
Bony compression contributing  
to ND

• Total Surgical excision - TOC for benign tumors & some malignant tumors

↓  
Laminectomy approach

excise involved dura & nerve roots (if infiltrated)

After extensive surgery, spine may be stabilised by

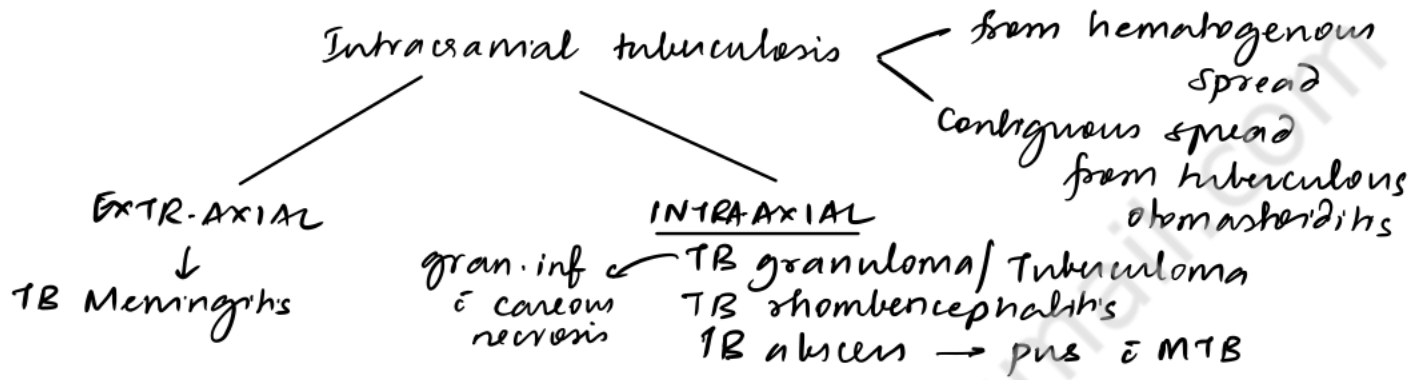
- H-fixation
- Laminoplasty

LEPTOMENINGEAL METS → CT/RT

Vertebral mets - Palliation, Bisphosphonates, Rx of Metabolic complications

# TUBERCULOMA

Tuberculomas / Tuberculous granulomas - well defined focal masses that result from *Mycobacterium tuberculosis* infection  
seen in lung  
• CNS



Tuberculomas - Ring enhancing lesions & surrounding vasogenic edema

Ddx - Neurocysticercosis  
Cerebral toxoplasmosis  
CNS cryptococcosis  
Bacterial abscess

Neurosarcoïdosis  
Cerebral mets  
CNS lymphoma

Rx - ATT → 12m → continue till e/o resolution

Systemic corticosteroids - for: perilesional edema  
paradoxical progression during Rx

Surgical intervention - for acute complications  
• when dx is uncertain

# PROIN'S SYNDROME

CSF Xanthochromia + ↑ CSF Protein level + Marked Coagulation of CSF

Caused by - Meningeal irritation - Meningitis  
Blockage of CSF flow by - tumor mass  
- abscess - Epidural abscess

Stagnation of CSF within thecal sac → exudation from  
tumor/abscess  
↓  
activation of coagulation factors

Pseudo-Proin SD - stagnation of CSF distal a spinal block 2/+  
spinal disc bulge  
Tumour

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