TRAUMATIC BRAIN INJURY (TBI)

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KEEP IT STUPIDLY SIMPLE !

- KISS principle – US navy 1960
- Originally ‘keep it simple, stupid’

Contents

- TBI and importance
- Relevant anatomy and physiology
- Relevant radiology
- Brain, skull and scalp trauma types and identification
- Diagnosis and management
- Outcome

TBI?

- Injuries to brain or skull of traumatic nature

- Injuries to the face – maxillofacial injuries
- Atraumatic head injury – stroke/ abscess

- Classifications;
  - Blunt vs penetrating
  - GCS – mild, moderate or severe
  - Radiological – Marshall or Rotterdam score

Why is TBI important?

- 1.4million cases in UK – 10% moderate to severe
- Expensive hospital stay
  - Paralyze and ventilation, extensive monitoring – ICU care
  - Craniotomy, tracheostomy
  - High end antibiotics
- Rehabilitation
  - Long-term process
  - Lot of supportive care – NG,PEG, tracheostomy care/ physiotherapy/ pressure sores
  - Heavy financial burden
- Overall return to society is bleak

TBI

- PRIMARY BRAIN INJURY
  - What happens at initial incident
  - Public health measures help out in prevention

- SECONDARY BRAIN INJURY
  - Any brain injury occurring after the primary insult – PREVENTABLE!
  - Causes
    - Hypoxia – obstructed airway
    - Hypercarbia/ hypocarbia
    - Hypotension – brain ischaemia due to hypovolaemia
    - High ICP – by cerebral oedema or blood clot

GOAL IN TREATING TBI?

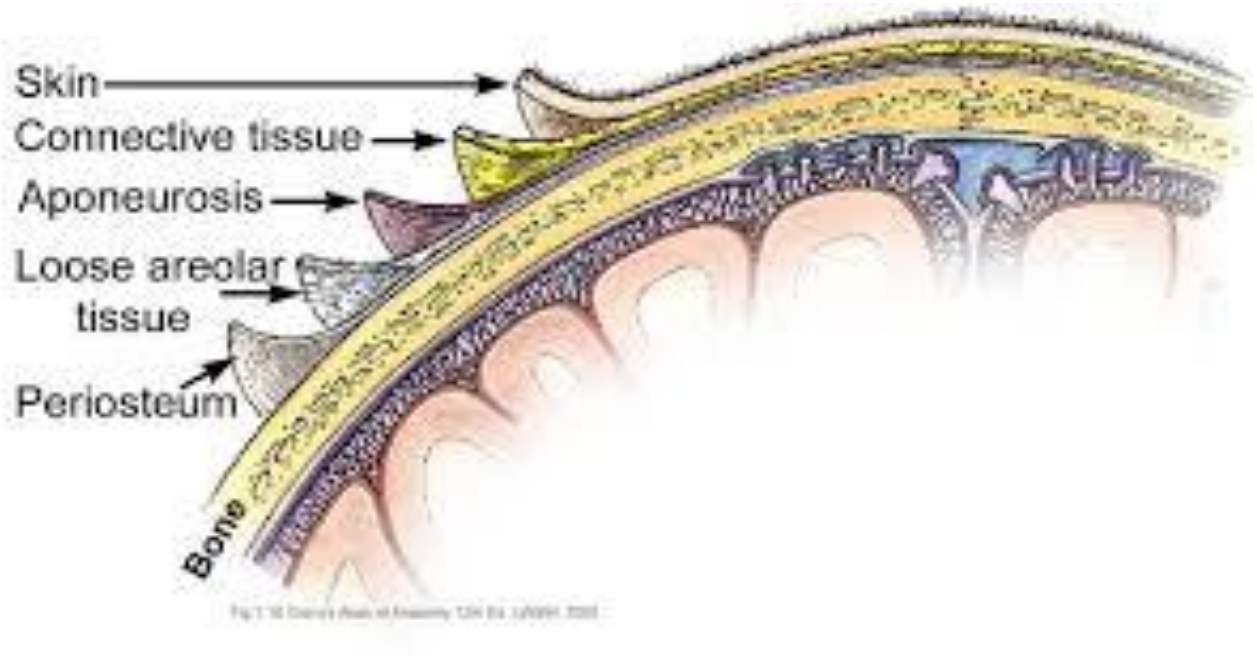
- TO AVOID SECONDARY INJURY
  - Optimise and transport to proper centre quickly
    - In SDH/EDH - when time from primary insult >2h – mortality rate doubles
- TO DIAGNOSE OTHER LIFE THREATENING TRAUMA
  - Abdominal ,thoracic trauma, etc.

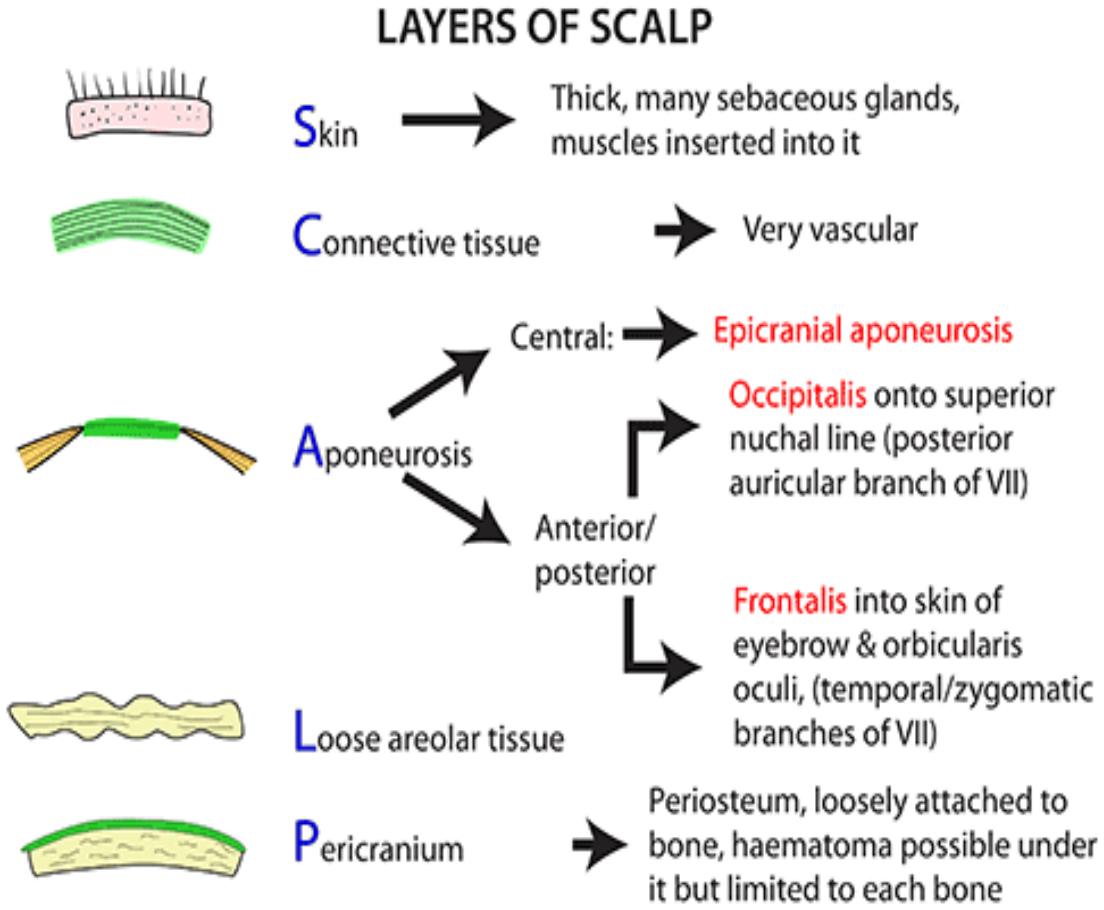
YOUR GOALS...

- Understand how to assess a TBI patient
  - Understand ***<u>mechanism of injury</u>***
  - Assess GCS
  - Pickup special signs of TBI
- Interpret a NCCT brain in a TBI situation
- Have a plan of management ;
  - To avoid secondary brain injury
  - Transferring to a suitable centre
- Pass finals

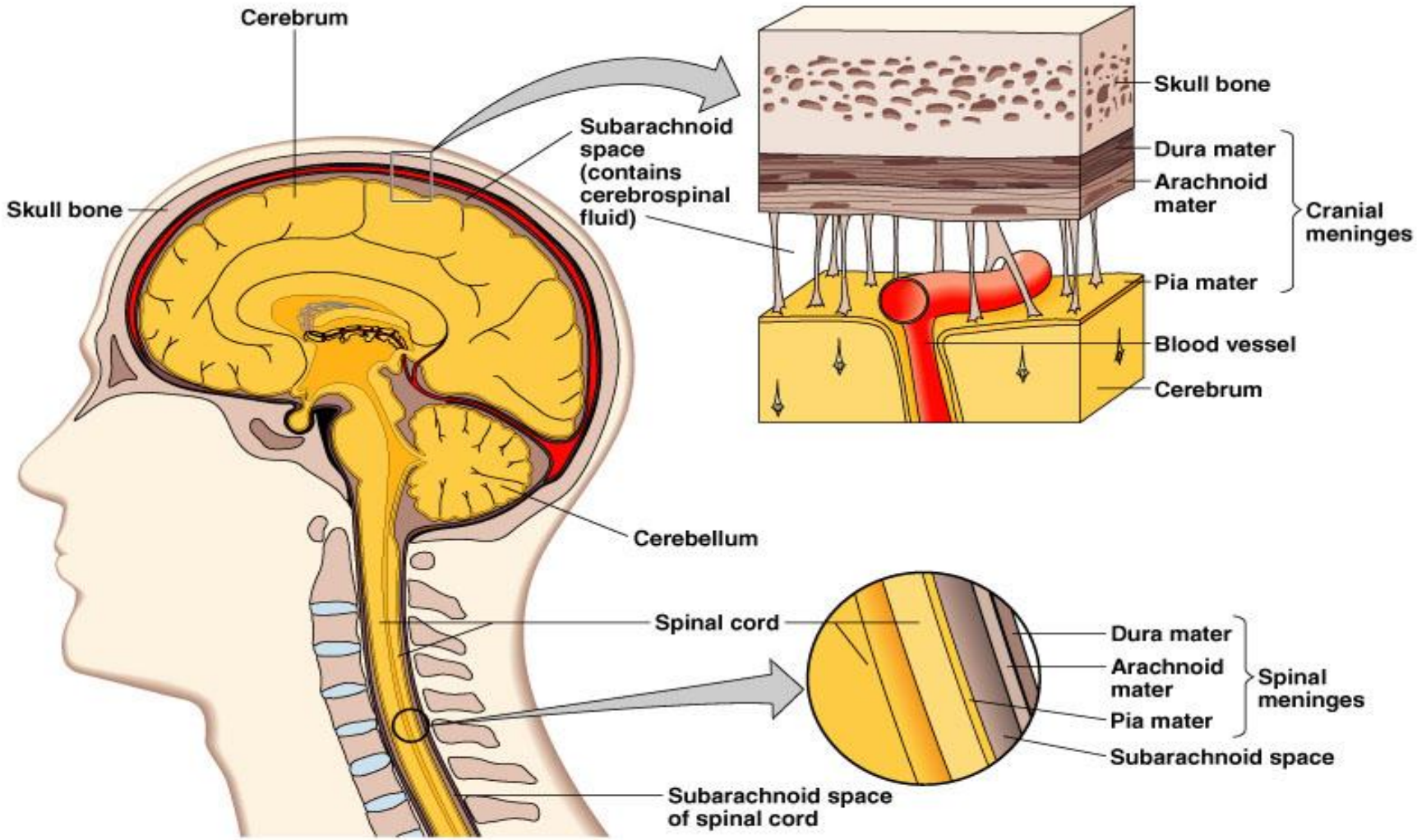
Save from death or life long misery for patient and caretakers

ANATOMY - SCALP

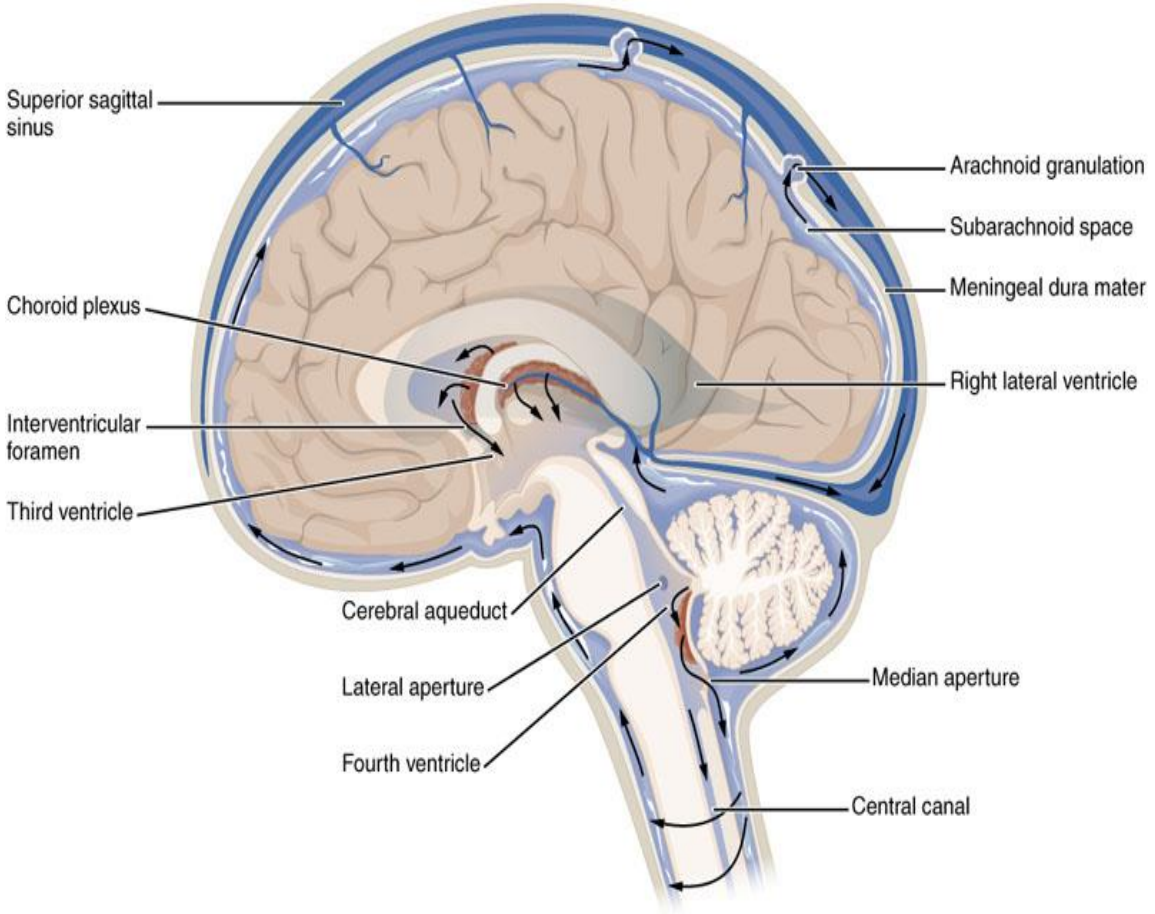


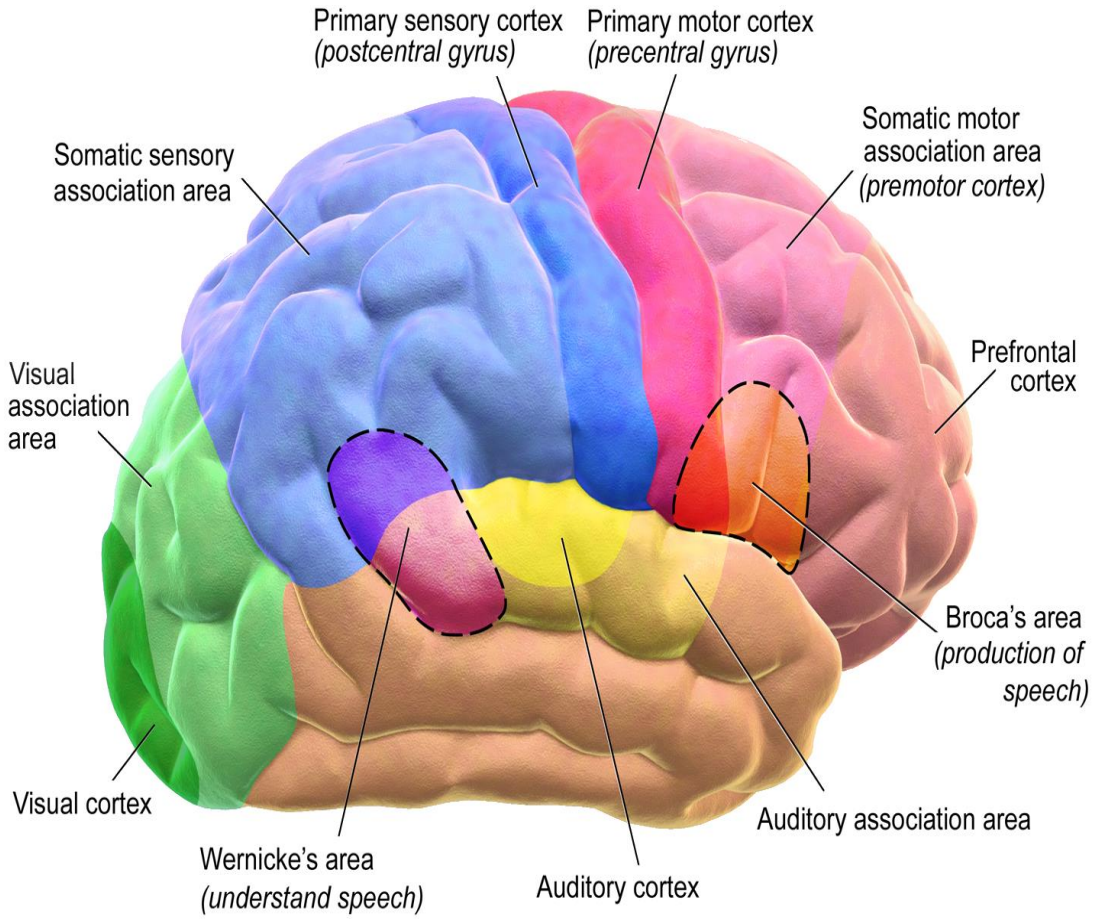


ANATOMY - MENINGES



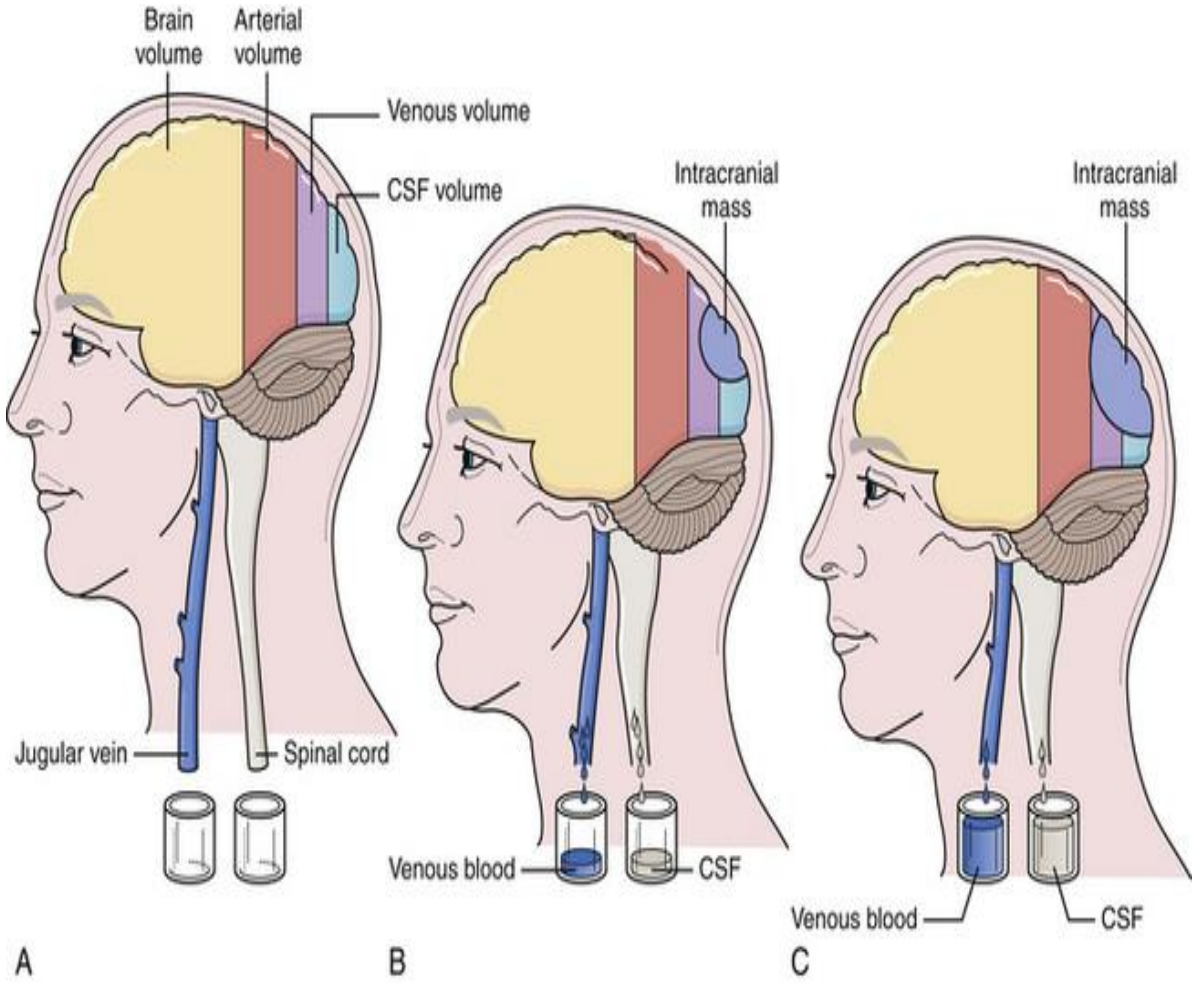
ANATOMY – BRAIN/VENTRICULAR SYSTEMS





PHYSIOLOGY - ICP

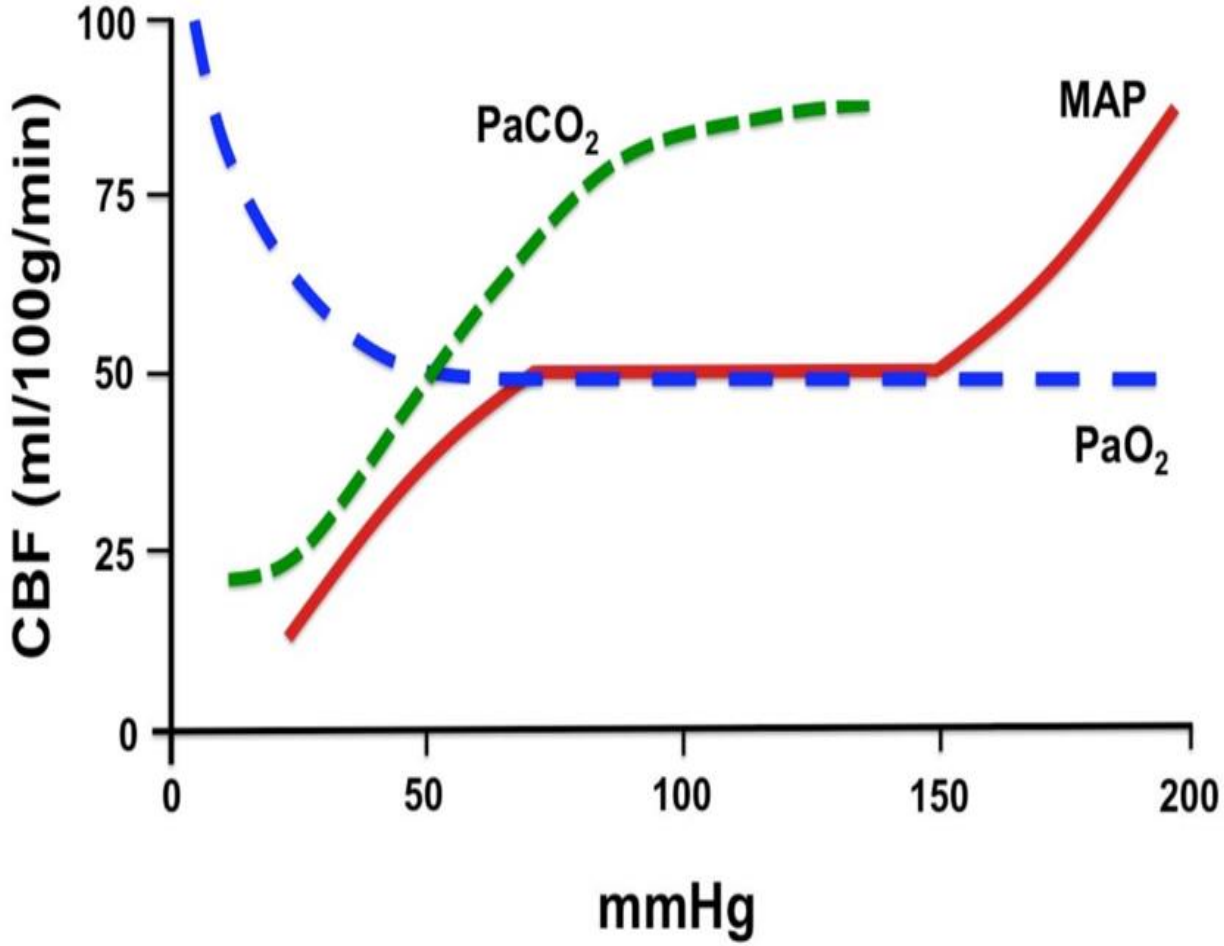
- Normal 0-10mmHg
- When increased (SOL or cerebral oedema) → regulations by,
  - Intracranial reservoirs compression to extracranial reservoirs
  - CSF - ventricles to lumbar cisterns
  - Blood - Venous sinuses to IJV
- Monro-kellie doctrine!



PHYSIOLOGY – CEREBRAL BLOOD FLOW

- Brain receives 15% of resting CO
- Autoregulation at MAP 60-140mmHg
- Hypertensive patient has a higher MAP and regulation has adjusted to a higher level – more susceptible for ischaemia at normal MAP range

- Goal is to have an adequate Cerebral perfusion pressure (CPP)
- CPP = MAP- ICP

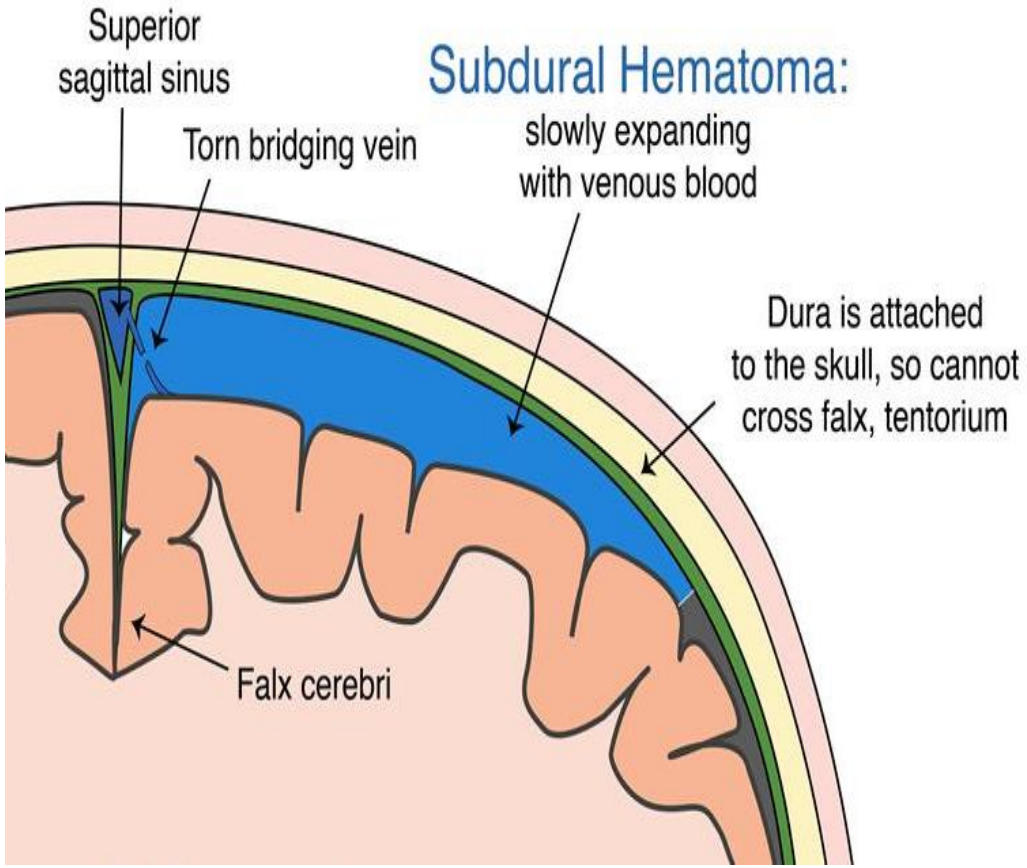


TYPES – INTRACRANIAL LESIONS

- Diffuse brain injury
- Focal brain injuries
  - SDH
  - EDH
  - CONTUSION/ INTRACEREBRAL HAEMATOMA
  - IVH
  - SAH

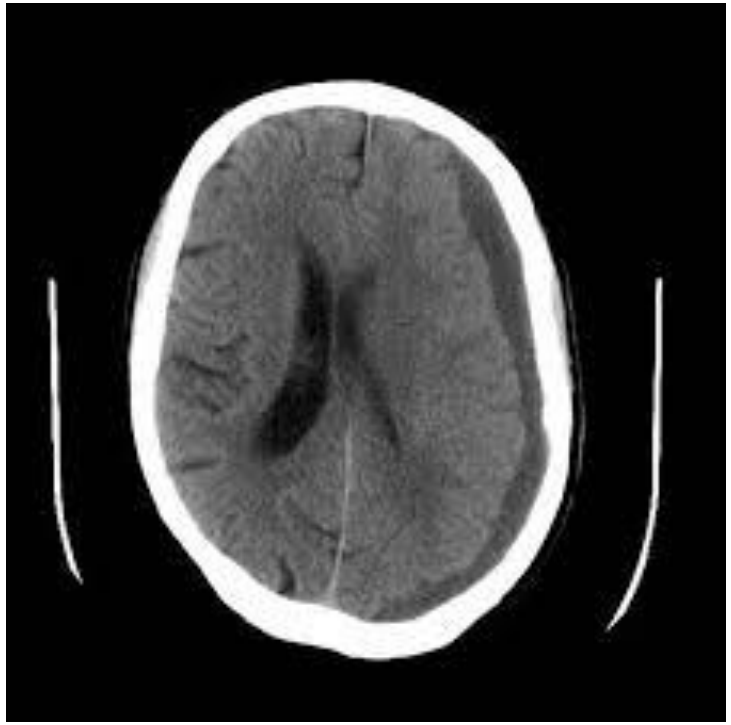
SDH

- Shearing of brain from the inside of the dura
  - Small cortical bridging veins are broken
  - Venous - slow bleed
- Semilunar (crescentic)?
  - No dural attachments-covers the brain convexity <u>across sutures</u>
- ACUTE – white (high attenuation of Hb)
  - Rotational injuries in young
- CHRONIC – black
  - Minor knock on adult – development of confusion or palsy over days-weeks



Acute SDH

- Hyperdense – dense clot
- Within 3days

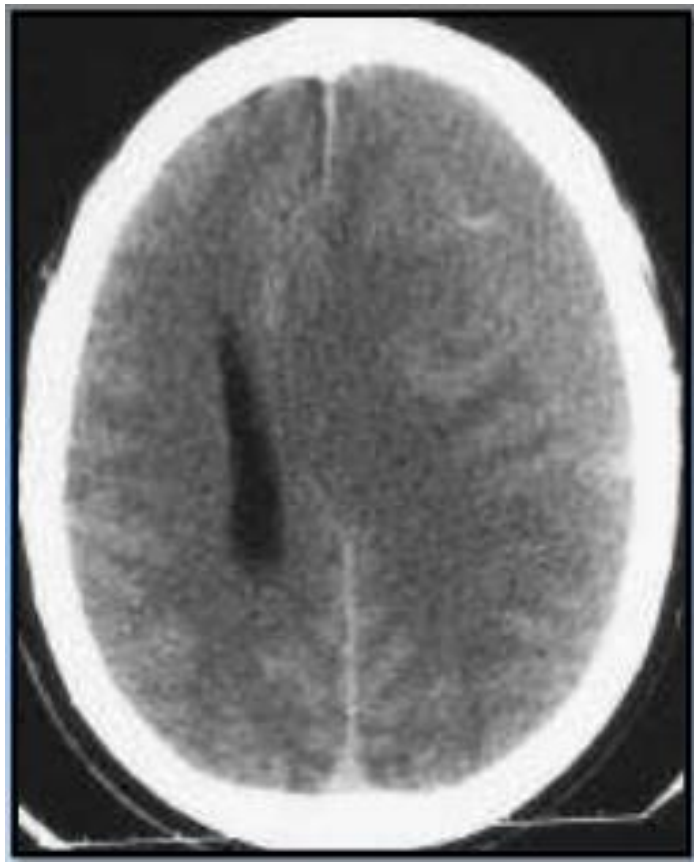


Chronic SDH

- Hypodense as density of clot reduces due to protein degradation – capsulated by a neomembrane with capillaries which are liable to rupture
- By definition > 3 weeks
- Main presentation with behavioral changes
- History of anticoagulants use

Acute on chronic SDH

- Re-bleed

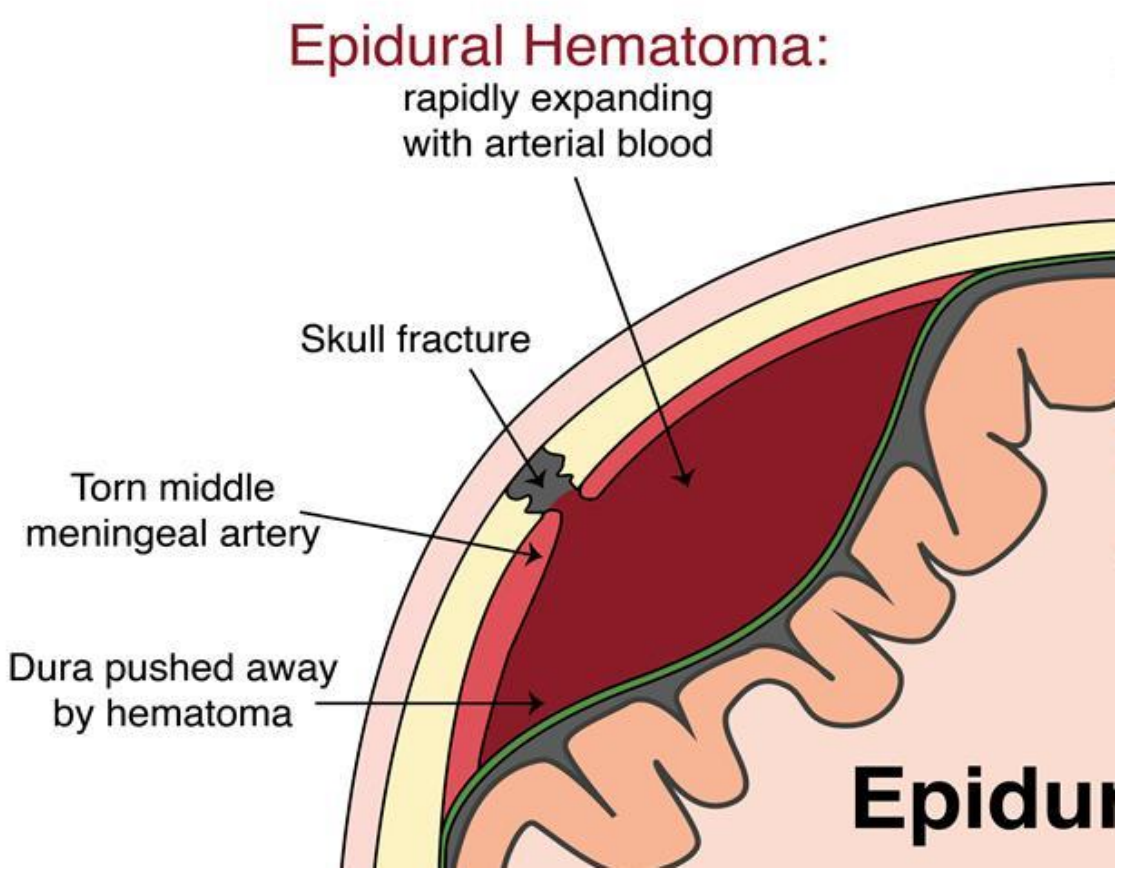


Subacute SDH

- L/fronto-parietal SDH
- Isodense
- 4-21 days presentation

EDH (epidural)

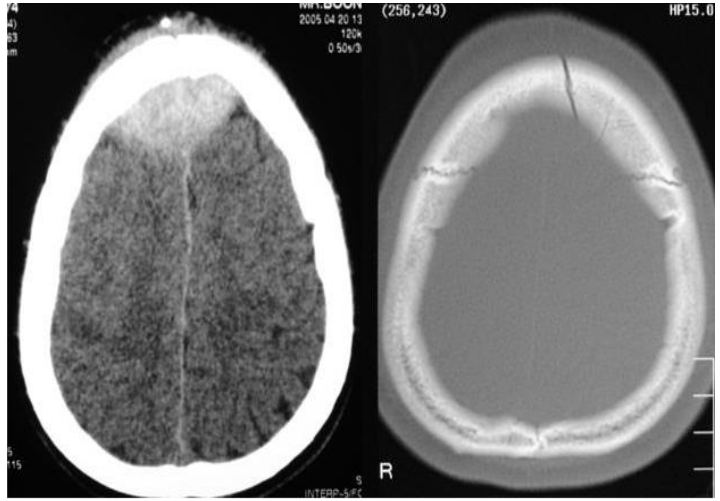
- Arterial bleed
- Direct trauma - Mainly with skull fracture on pterion with middle meningeal artery damage
- Biconvex/lenticular?
  - Dural attachments to sutures prevent extension – rapidly increase ICP supported by arterial bleeding



EDH

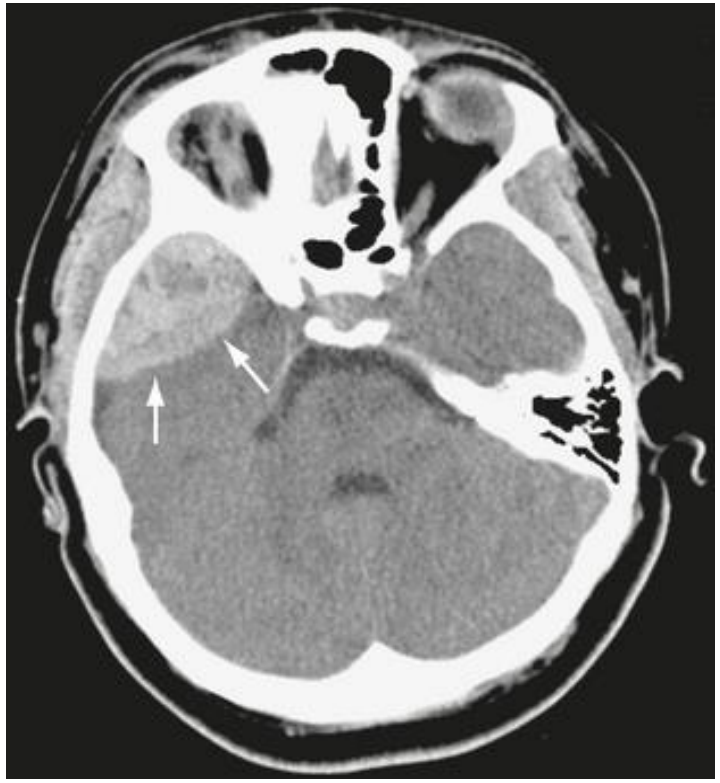
- Lucid interval?
  - Injury →brief LOC → recovery → progressive deterioration
  - Arterial blood shunted via veins → slows development of haematoma = interval from symptoms
  - Arterial bleed and pressure in the haematoma exceeds venous drainage = deterioration
    - Jonathan, Walter Hutchison

1. Lenticular parietal EDH
2. MLS
3. Collapsed lateral ventricles
4. Oedematous brain (absent sulci)
5. Scalp haematoma



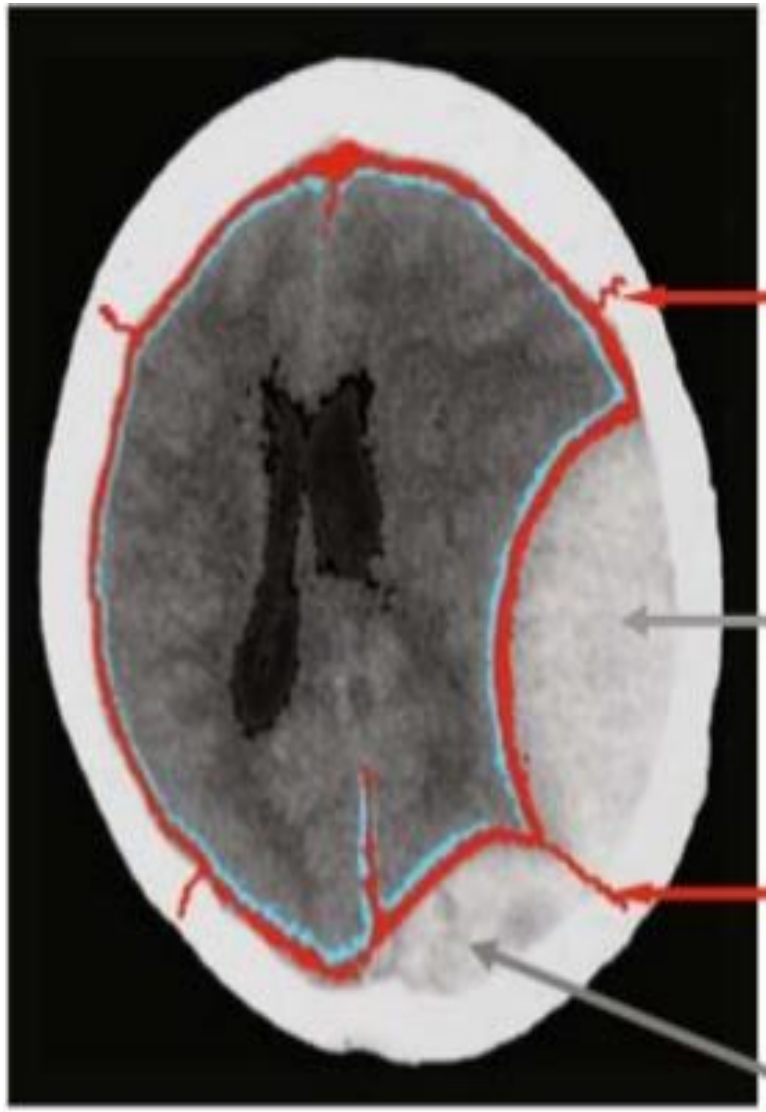
Bi-frontal EDH

- Frontal bone fracture on bone window



R/ Temporal EDH

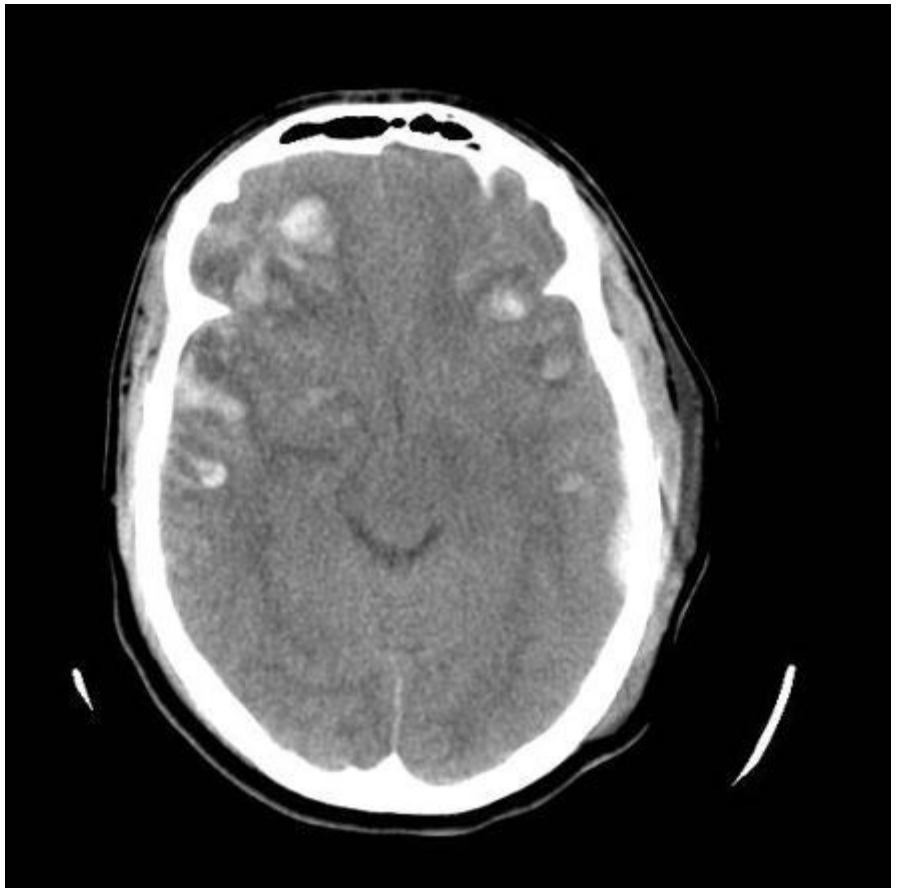
Involved vessel	Temperoparietal (most likely) - Middle meningeal artery Frontal - anterior ethmoidal artery Occipital - transverse or sigmoid sinuses Vertex - superior sagittal sinus



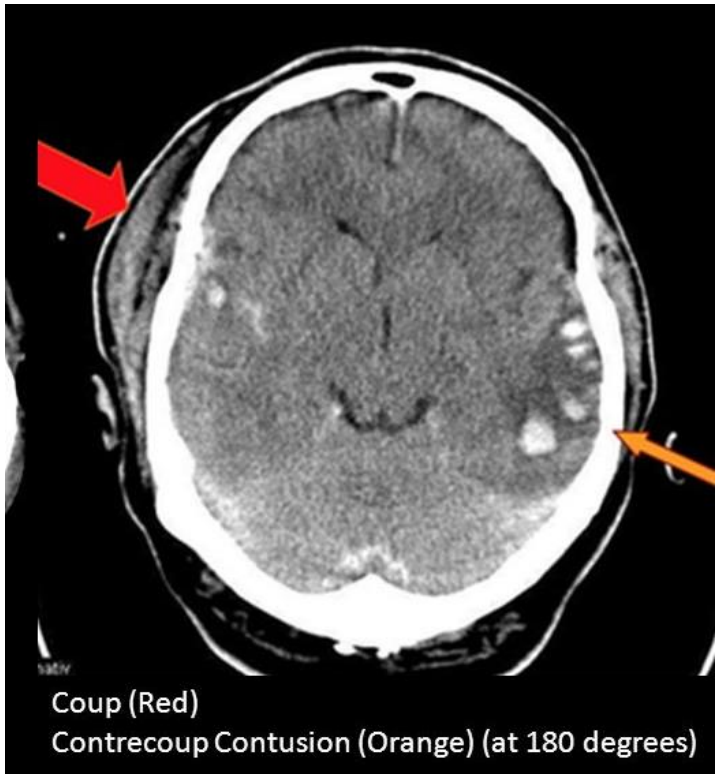
Involved vessel	Temperoparietal (most likely) - Middle meningeal artery Frontal - anterior ethmoidal artery Occipital - transverse or sigmoid sinuses Vertex - superior sagittal sinus

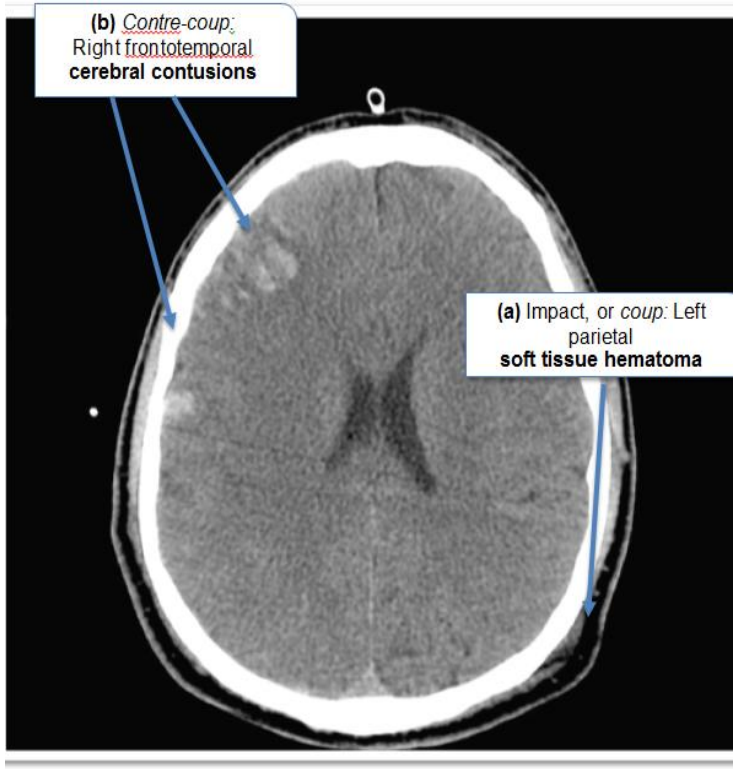
CONTUSION/INTRACEREBRAL HAEMATOMA

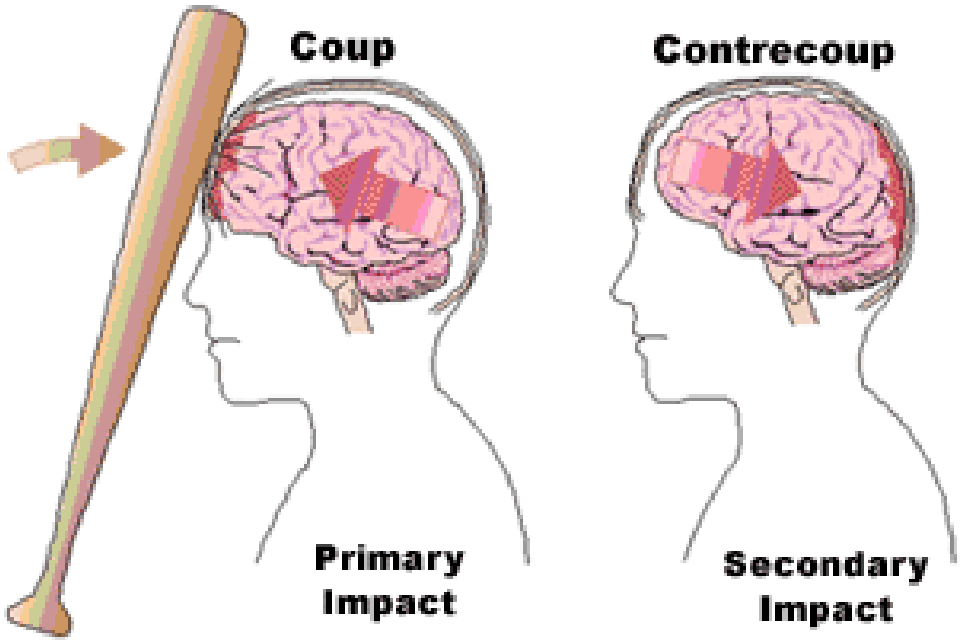
- Acceleration- deceleration injury (Contre-coup injury)
- Petechial haemorrhage from gyri vessels → small haematoma
  - Show as hypodense areas (surrounding oedema) and hyperdense (blood) areas
- Sometimes coalesce in to large intracerebral haematomas in 1-2days
  - Show as hyperdense lesions



- Contre-coup injury?
  - Injury to exact opposite site of impact
  - Initial acceleration of skull and impact of stationary brain – Coup
  - Subsequent acceleration of brain and deceleration of skull – Contre-coup

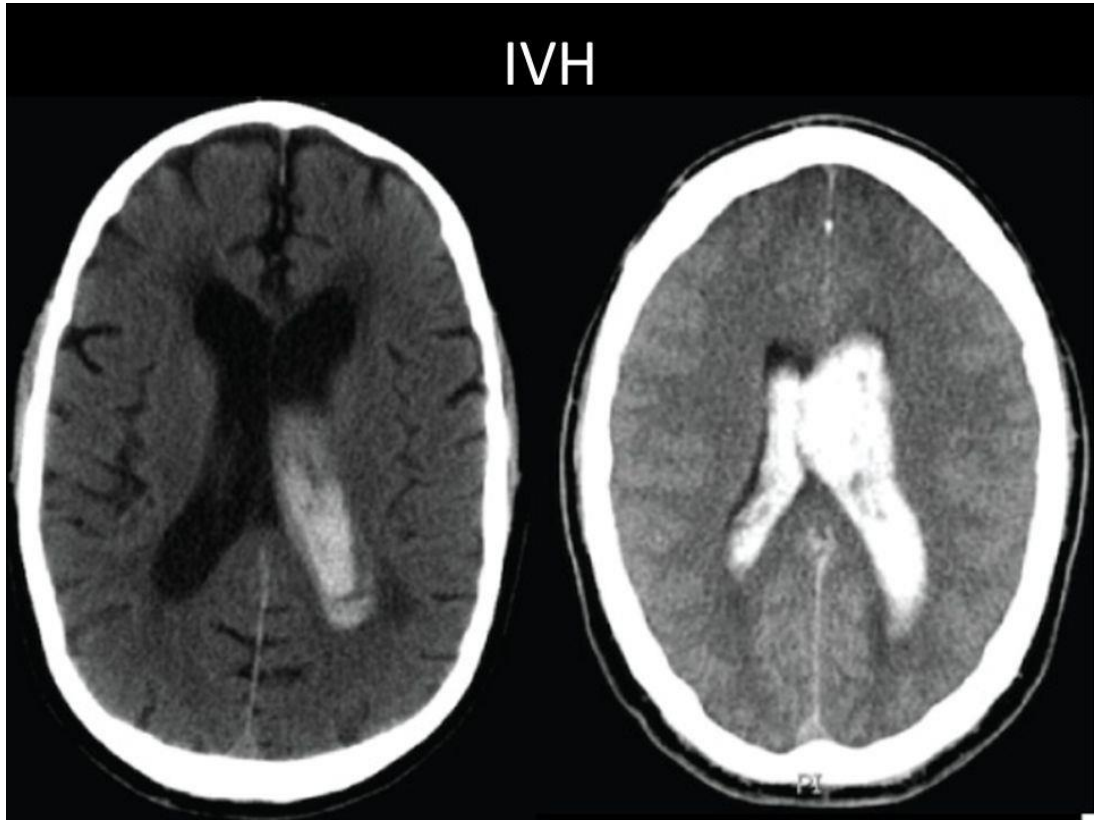






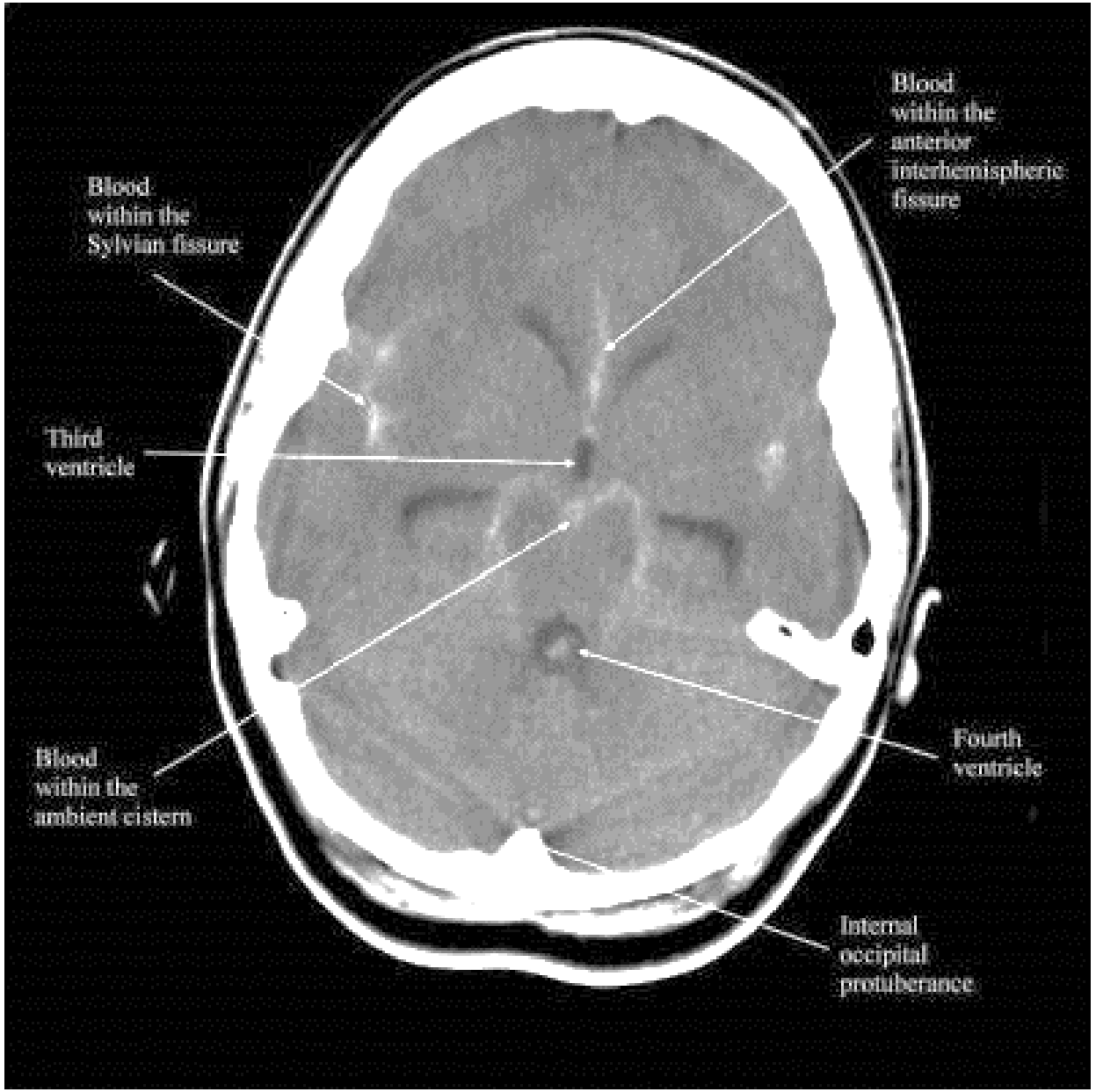
INTRAVENTRICULAR HAEMORRHAGE

- Bleeding into ventricular system causes hydrocephalus as outflow blocked by clotted blood → rise in ICP



SAH

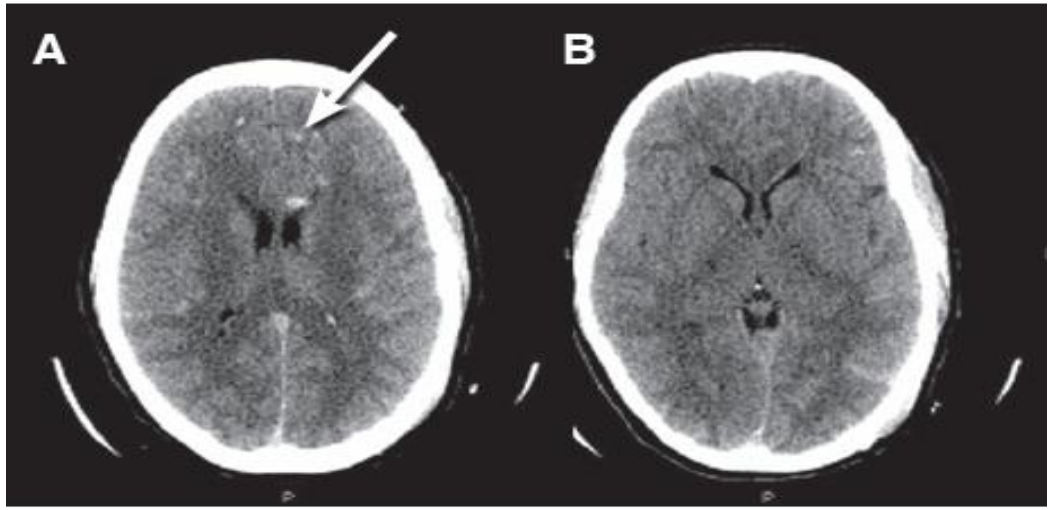
- Small SAH is inevitable in blunt trauma to head
- SAH per say doesn’t need any surgical intervention
- Usually atraumatic following
  - Ruptured AVM
  - Ruptured aneurysm



DIFFUSE BRAIN INJURY (DAI)

- Disruption of white matter fibre tracts
  - Specially at grey-white margin where densities are different
- Specially in deceleration injuries
- Corpus callosum and brain stem mostly affected
- Difficult to see in CT
  - Maybe negative initially
- Best seen in delayed MRI

Figure 5. Diffuse Axonal Injury On Computed Tomography



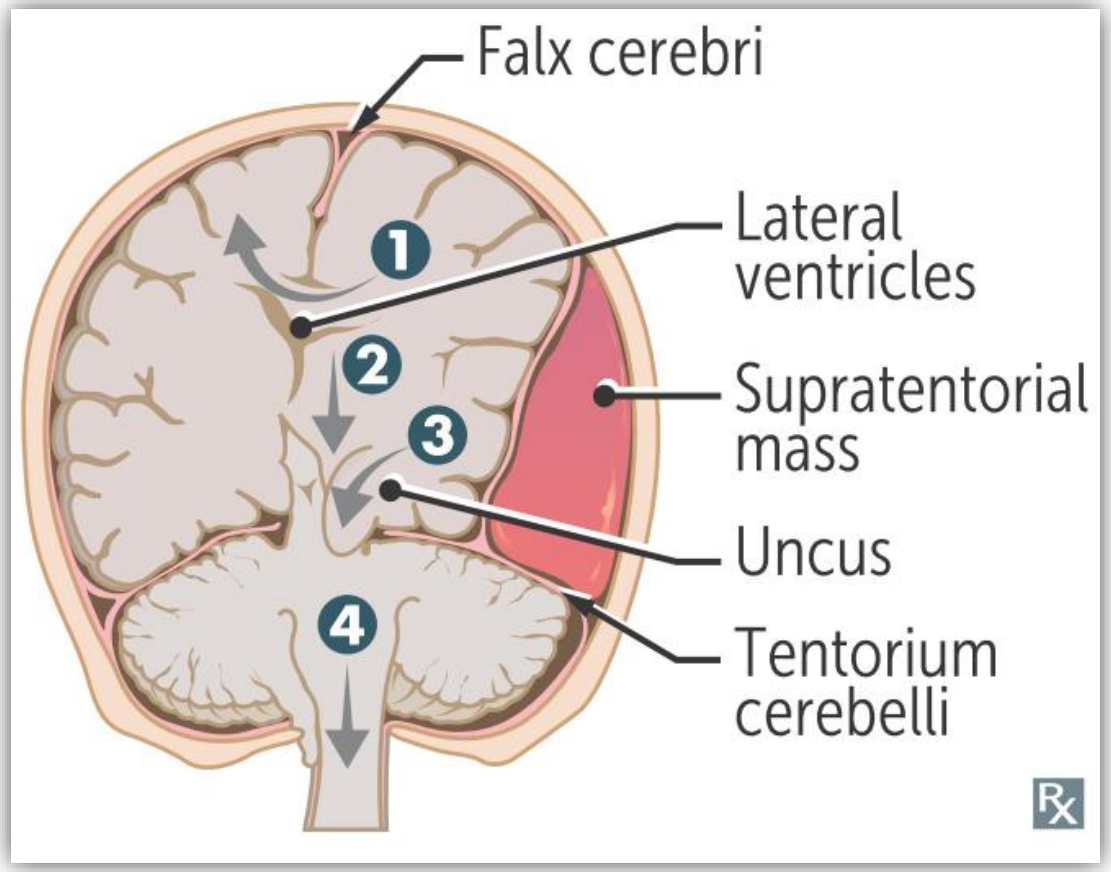
In view A, arrow points to areas of hemorrhage that often accompany diffuse axonal injury. In view B, note that the CT is negative.

1. Petechial haemorrhages
2. Poor grey-white differentiation
3. Tight brain

Brain herniation

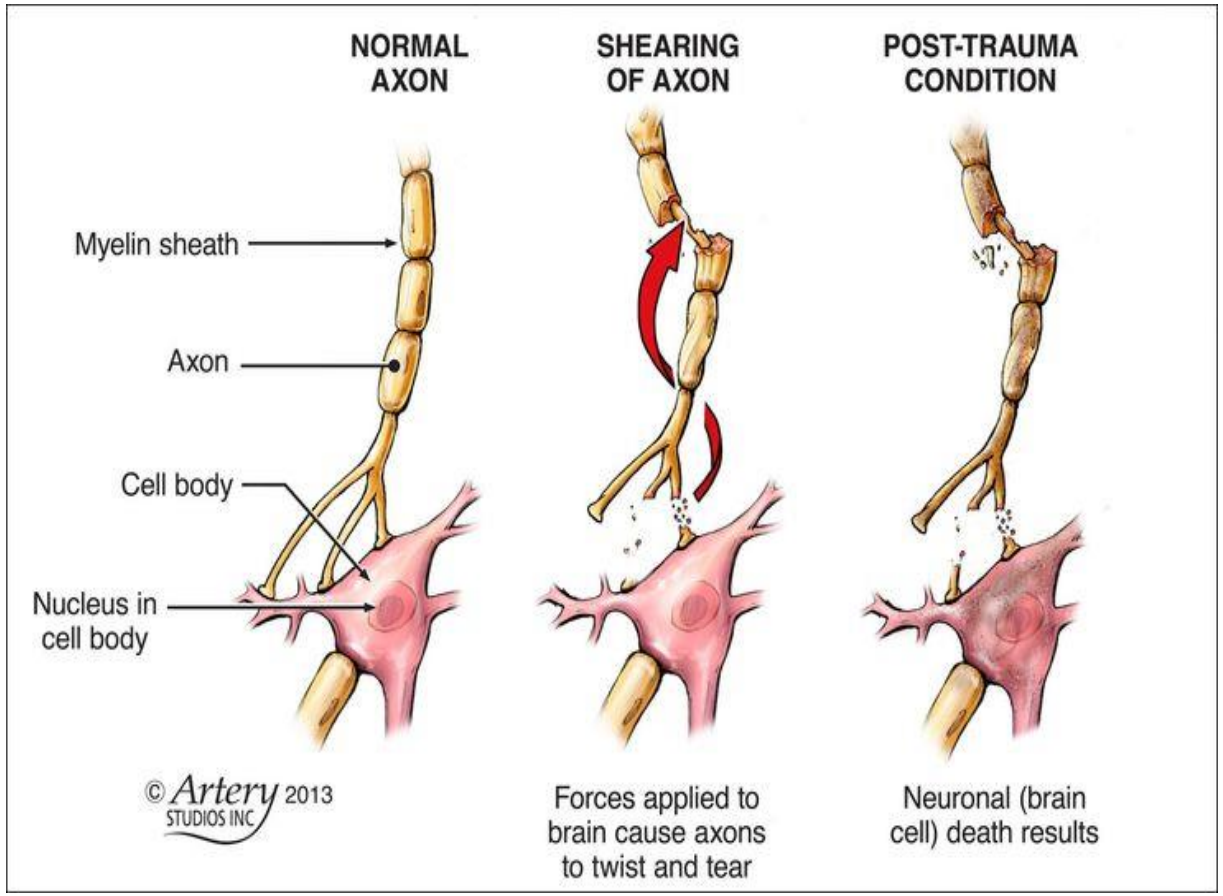
- Expanding haematoma → ICP rising → CSF into cisterns/ venous blood out of cranium → compensation limits maximum → brain herniates

1. Subfalcine herniation
2. Transtentorial herniation
3. Uncal herniation
   - Press on III – parasympathetic sphincter pupillae gone – unopposed dilator pupillae (V1) → dilated pupil
   - ***Dilated pupil means severe brain injury***
4. Tonsillar herniation

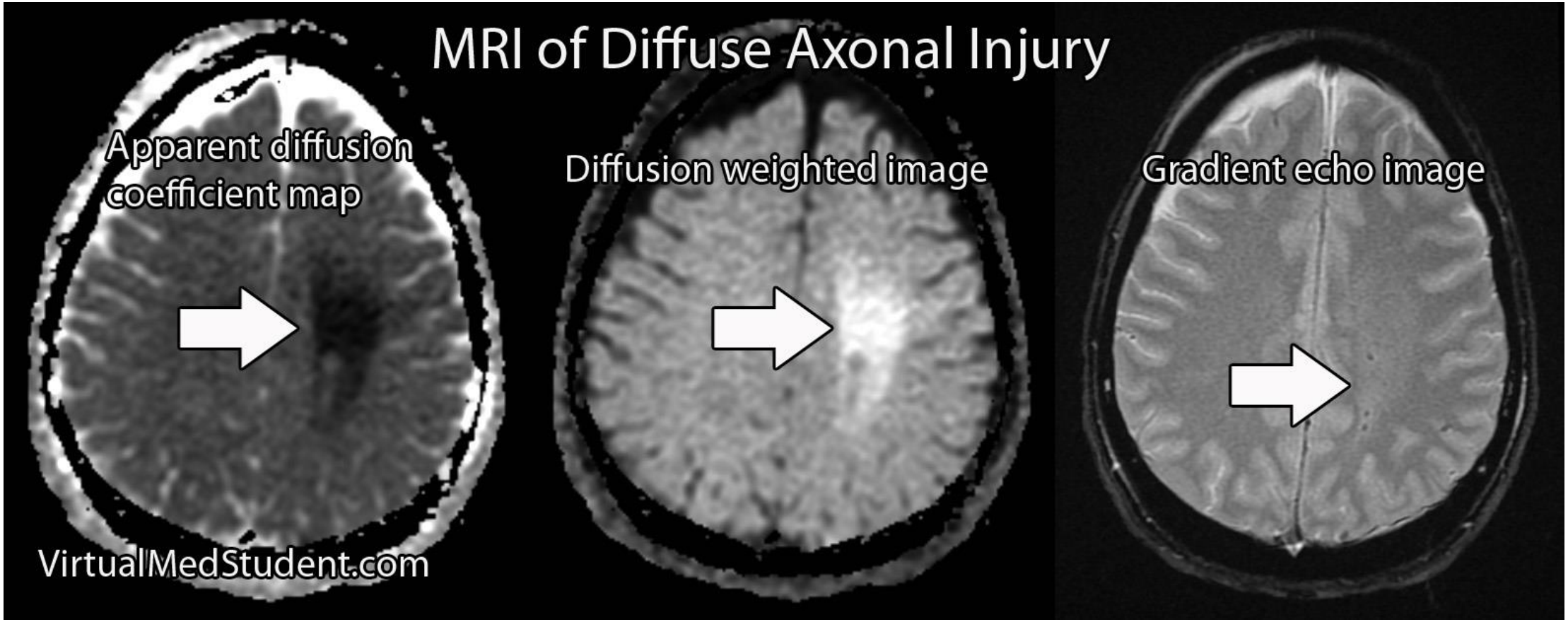


DAI

- Persistent vegetative /unconscious states
  - Difficult to wean off the ventilator
- Multiple lesions – poor prognosis



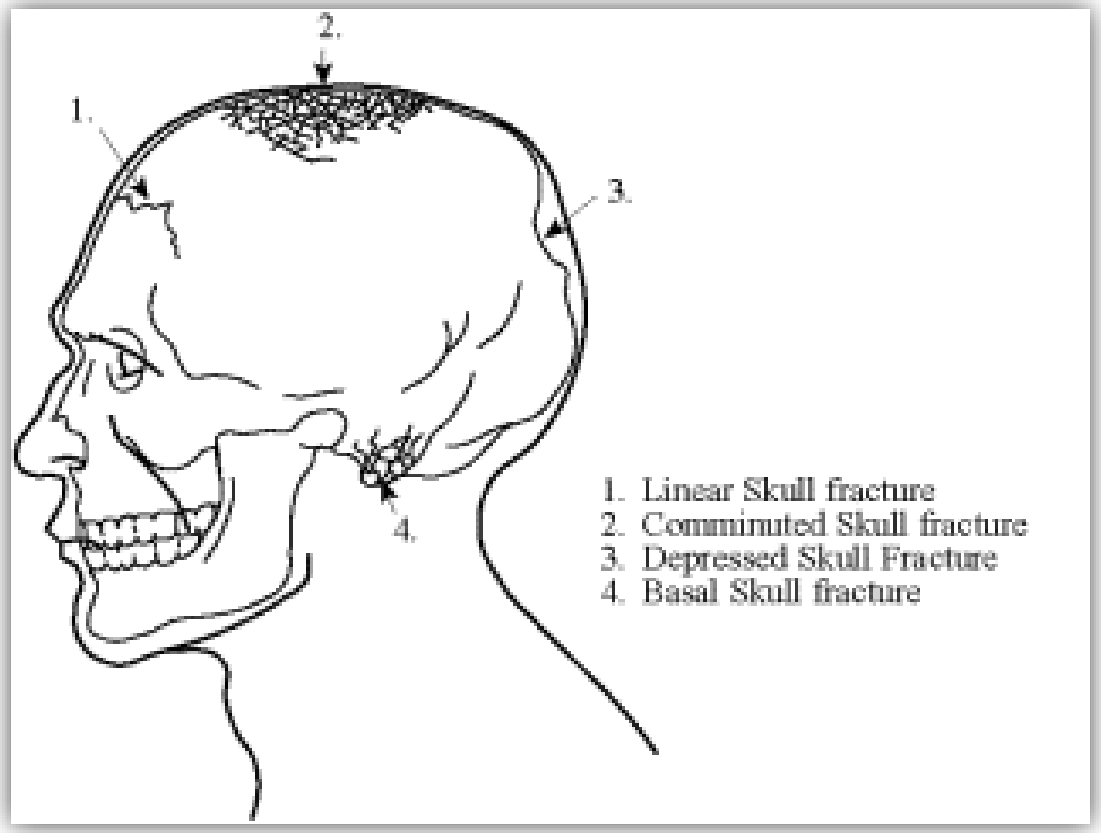
MRI


MRI of Diffuse Axonal Injury
Apparent diffusion coefficient map
Diffusion weighted image
Gradient echo image
VirtualMedStudent.com

TYPES – SKULL FRACTURE

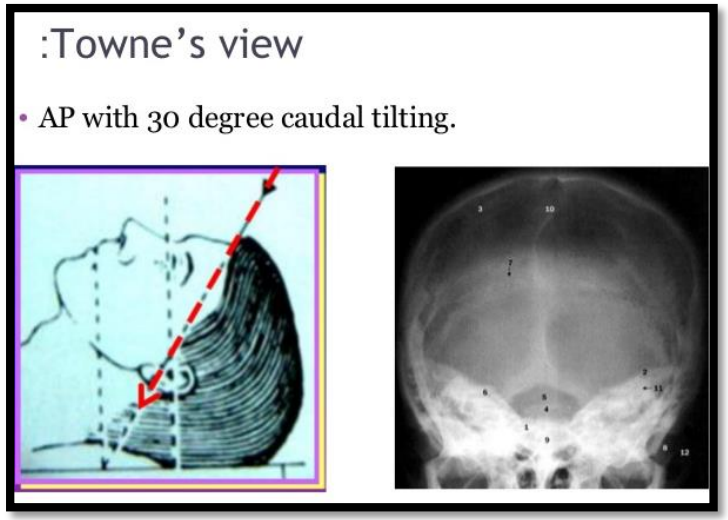
- Linear
  - simple, difficult to detect
- Comminuted
  - Need to fix together
- Depressed
  - Need to elevate
- Basal/basilar
  - Linear fractures in base of the skull
- Diastatic
  - Widened sutures in aged <3y

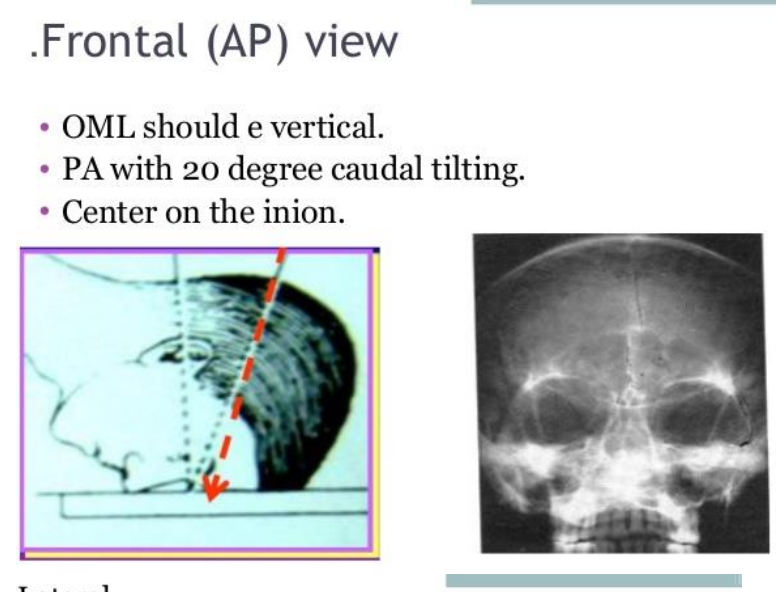
- **OPEN VS CLOSED FRACTURES**

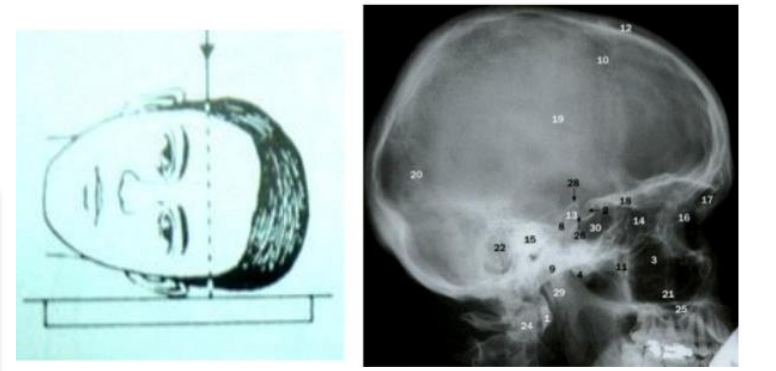


Radiology for skull fractures

- Traditionally a x-ray AP & Lateral view
- Impact on occiput
  - Towne's view to see posterior fossa
- Moderate to severe TBI
  - NCCT bone window

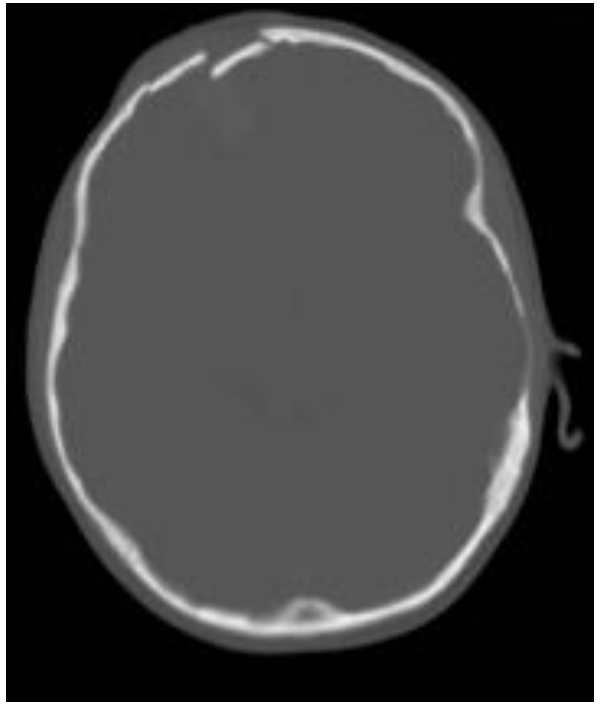






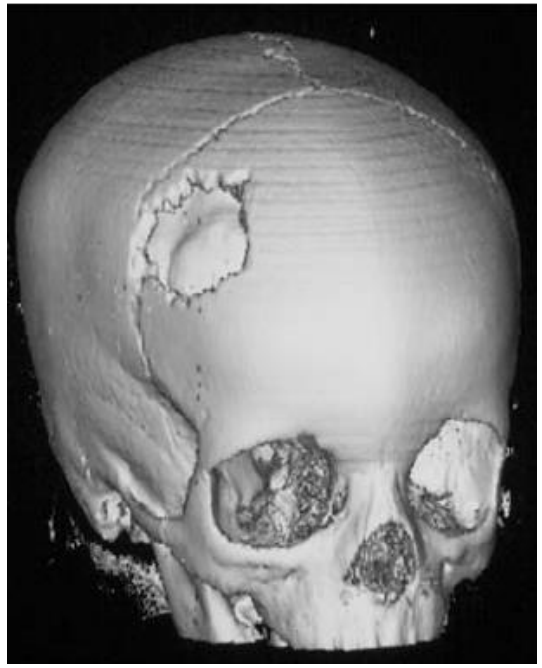
Linear #

Depressed #



Depressed # on bone window

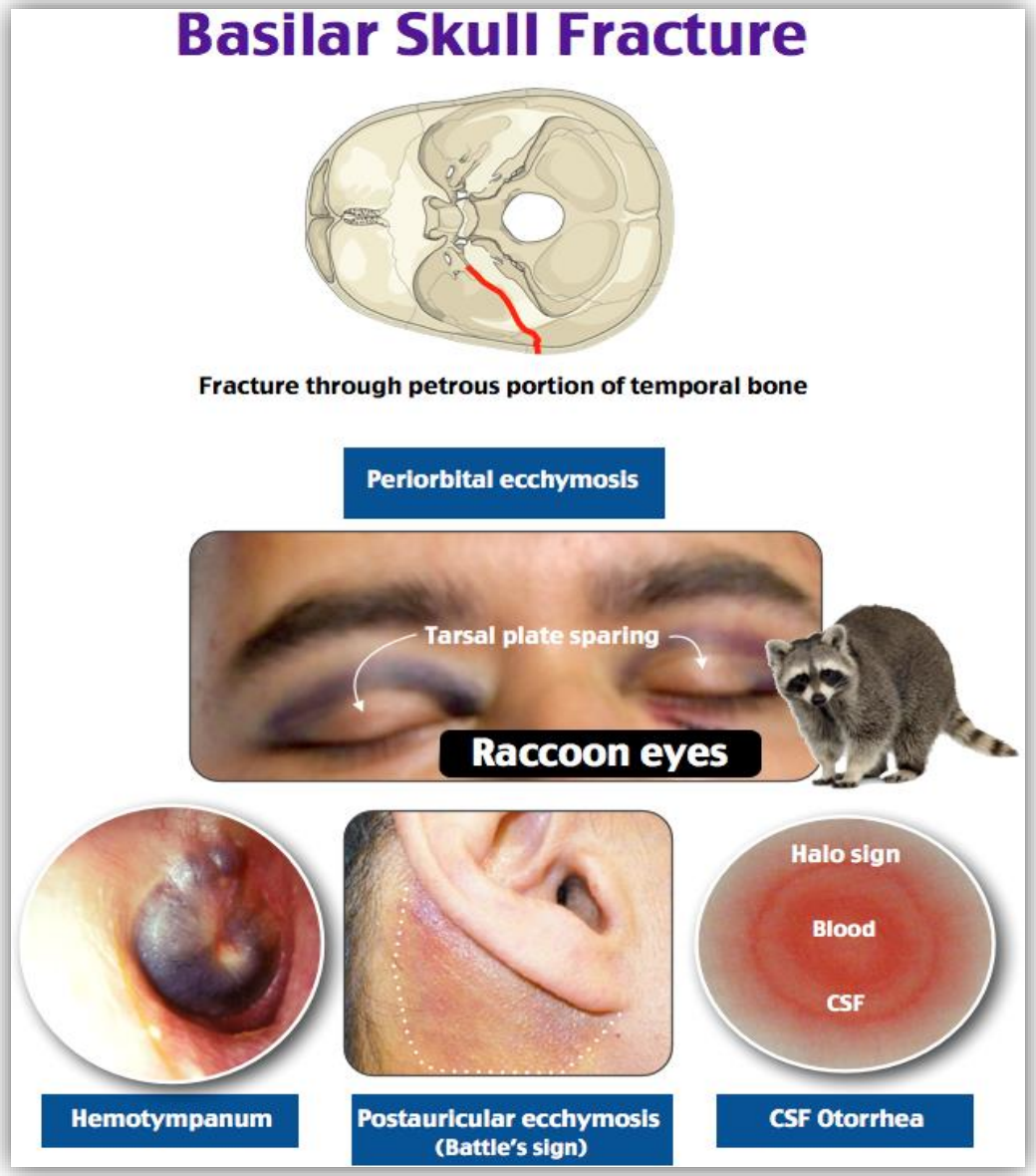
3D reconstruction of a depressed #



Basilar fracture identification

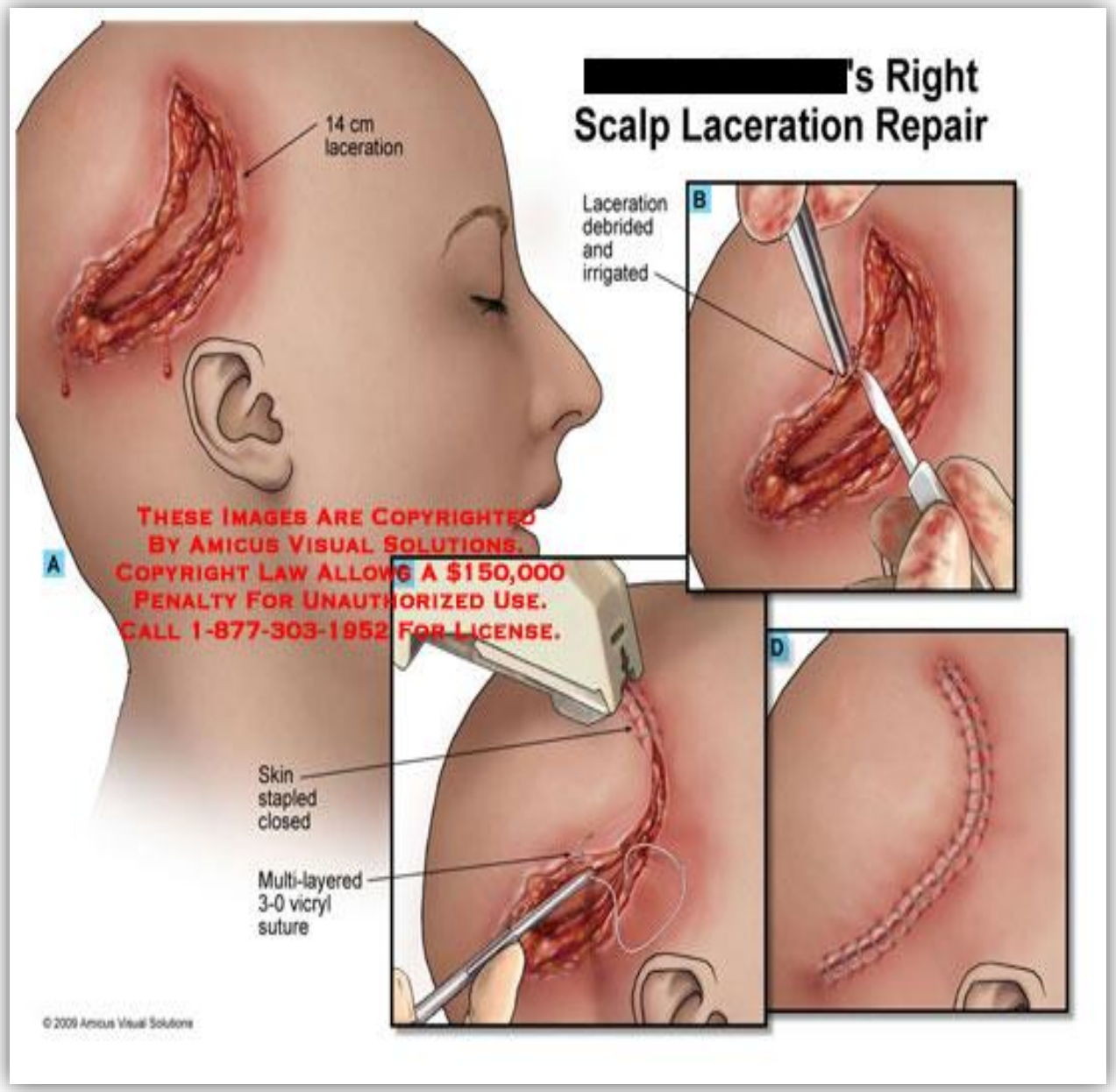
- Clinical suspicion
  - Raccoon eyes – haematoma around eyes sparing tarsal plates
    - Can be due to orbital # as well
  - Battle sign – bruising behind the ear
    - blood collection in Loose areolar tissue layer
  - CSF rhinorrhea or otorrhoea
  - Halo sign from nasal discharge
  - Epistaxis
    - Commonly from nasal bone fracture
  - Bleeding from ear
    - Commonly from lacerations of EAC
    - Check for haemotympanum

- ***Difficult to detect in x ray***



Scalp trauma

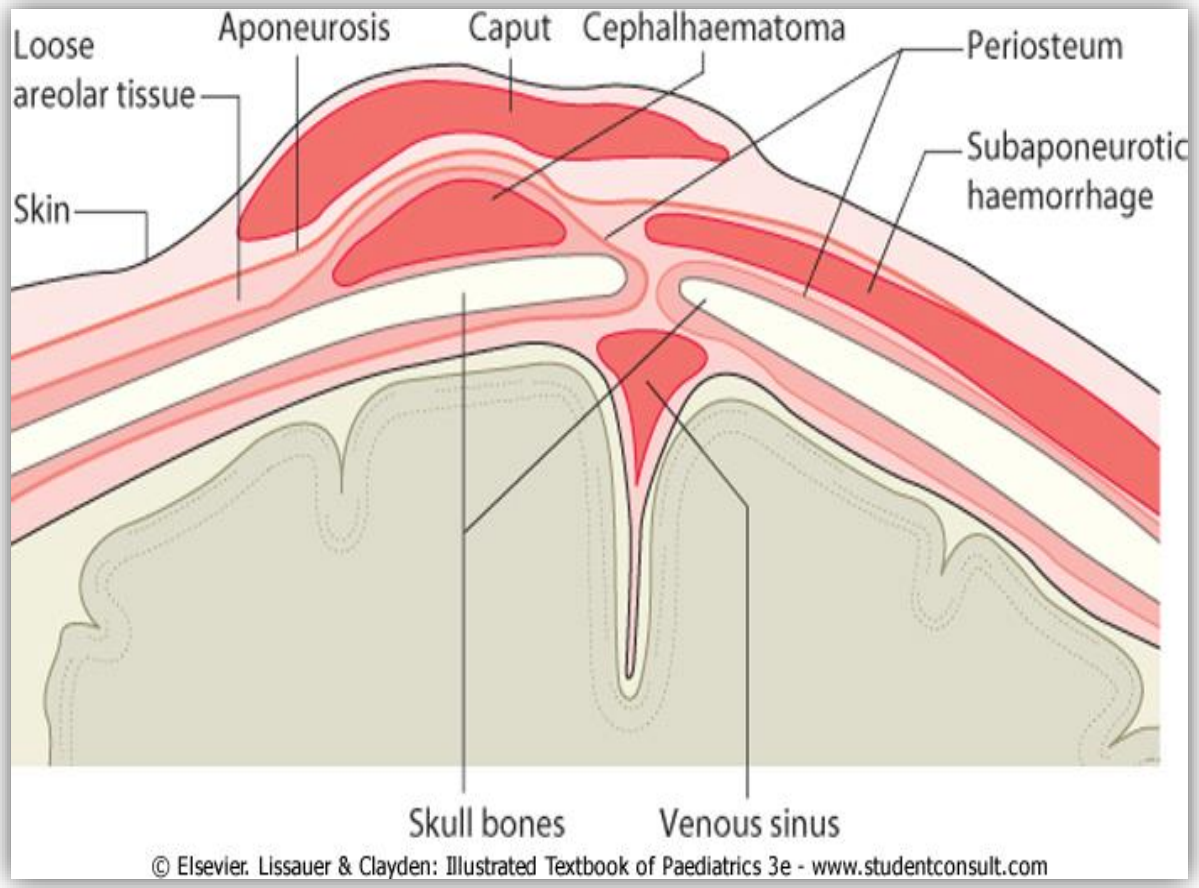
- **Scalp lacerations**
  - Severe bleeding
    - vessels in the Connective tissue are torn and retract – pressure controls
  - Temporary suturing before transferring
  - Proper wound excision – layered closure
    - Aponeurosis
    - Skin and connective tissue



Scalp trauma

- **Subgaleal haematoma**
  - Subaponeurotic haematoma
  - In Loose areolar tissue layer
  - Boggy mass

- Cephal haematoma
  - Under periosteum in newborns

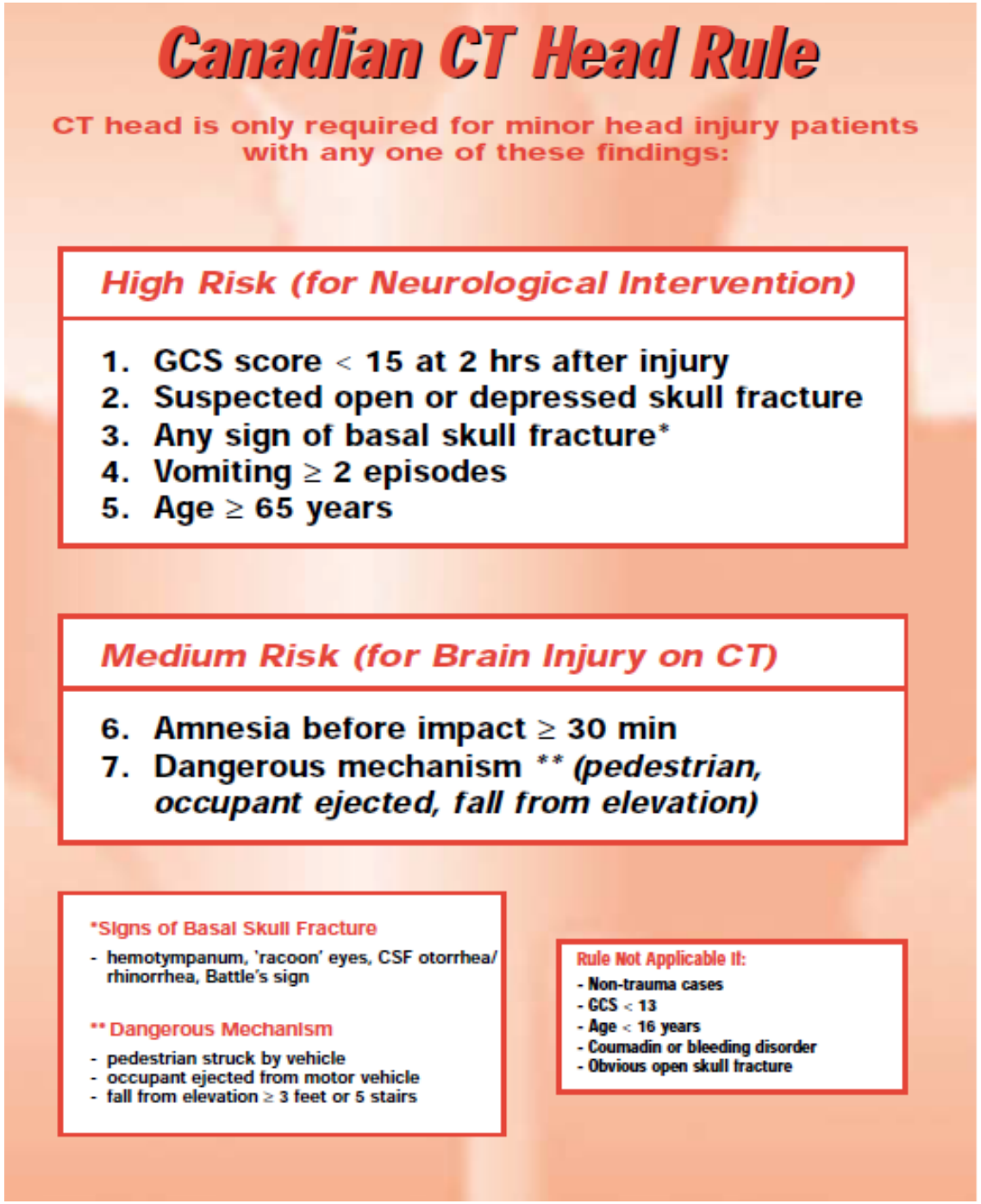


Severity of TBI

- Mild (GCS 13-15)
- Moderate (GCS 9-12)
- Severe (GCS 8 or lower)

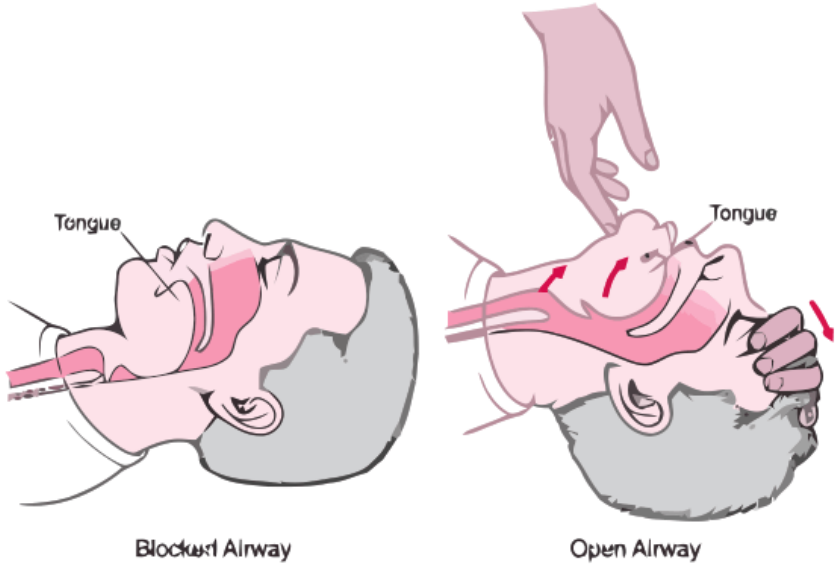
When to do a NCCTB?

1. New onset seizures after head injury
2. Seizures in a previous epileptic after head injury
3. Under drug/ alcohol influence coming with head injury



Diagnosis & Management - simultaneously

- **A**IRWAY
  - After GCS /C spine– intubation, paralysis & ventilation
    - GCS equal or <8
    - Secures airway to avoid aspiration → pneumonia
    - Good oxygenation to avoid hypoxic brain damage
    - Minimise patient exhaustion in a combatant patient



- C- spine
  - Assume injured until proven otherwise
  - Hard cervical collar
  - Avoid head tilt and chin lift while intubating

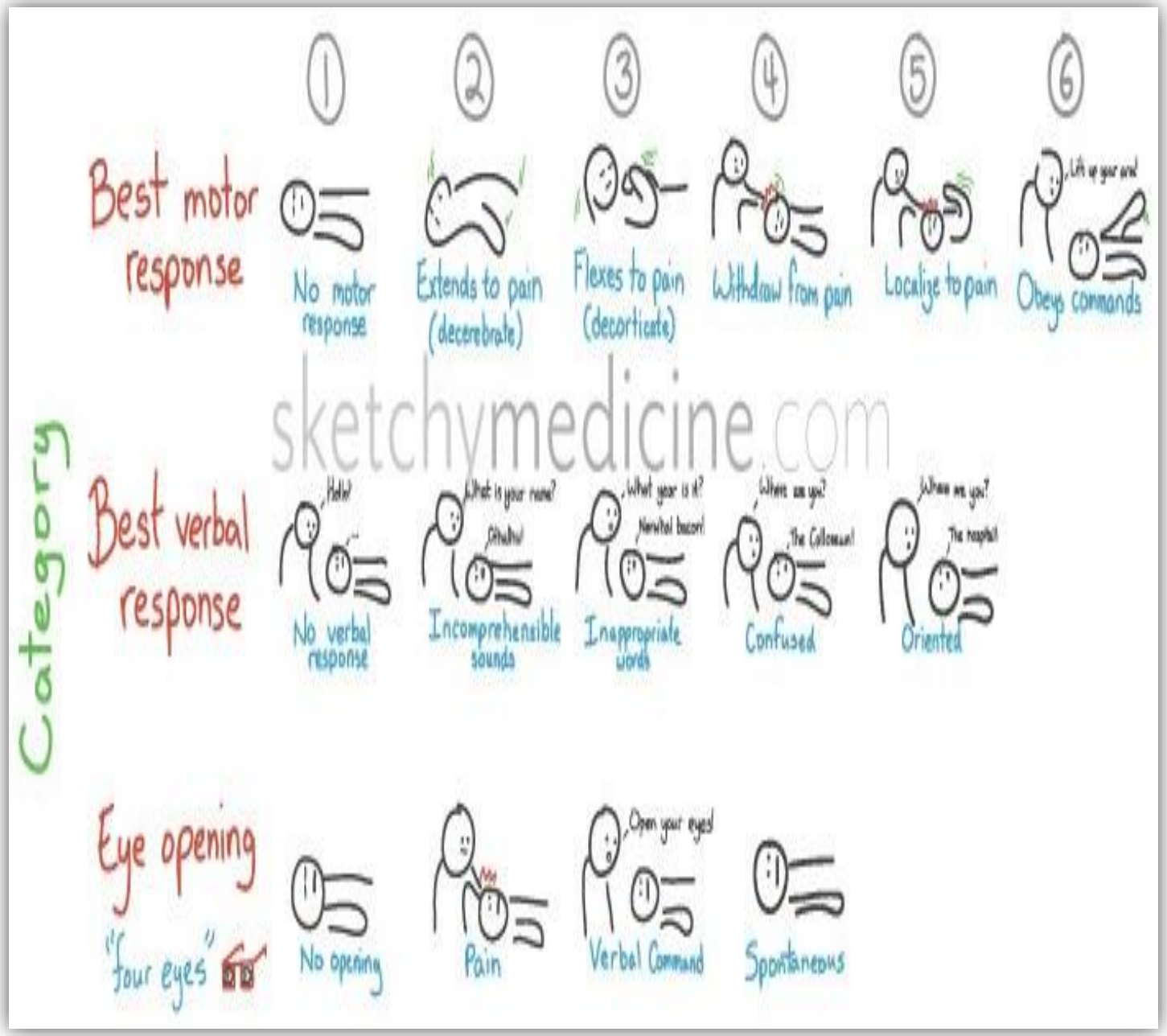
Diagnosis & Management - simultaneously

- **B**REATHING
  - Intubated and ventilated
  - Target for normocapnia
  - SpO2 > 93%
- **C**IRCULATION
  - Minimise hypotension (reduce/ splint fractures, avoid bleeding from wounds)
- **D**ISABILITY
  - GCS
- **E**XPOSURE
  - Skull lacerations, fractures, pupils

- *Normocapnia:* $PaCO_2$ between 35 and 45 mm Hg
- *Hypocapnia:* $PaCO_2 < 35$ mm Hg
- *Hypercapnia:* $PaCO_2 > 45$ mm Hg
- *Hypoxemia:* $PaO_2 < 80$ mm Hg
- *Note:* significant activation of carotid bodies occurs only when $PaO_2 < 60$ mm Hg

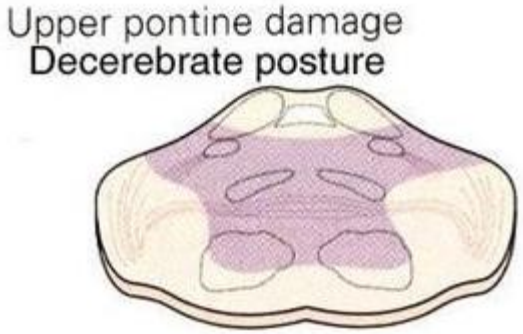
GCS

- Repeatable monitoring of patient's 'level of consciousness'
- Easy to perform
- Motor component has more weight
  - Prognostic indicators
- Say as E4V5M6
- Different Paed GCS available



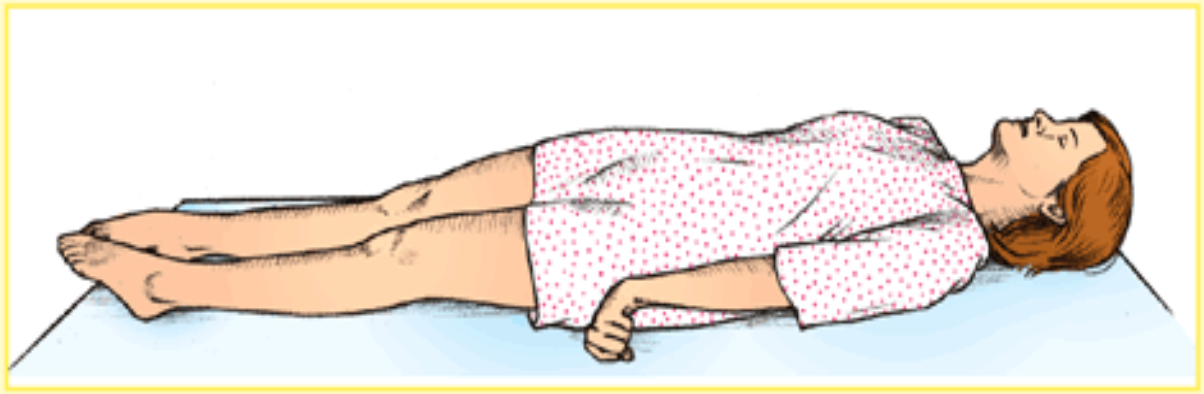
Decorticate, decerebrate?

- Abnormal posturing
- Decorticate M3
  - Upper brain stem damage
- Decerebrate M2
  - Corticospinal tract damage
- Brain herniation!

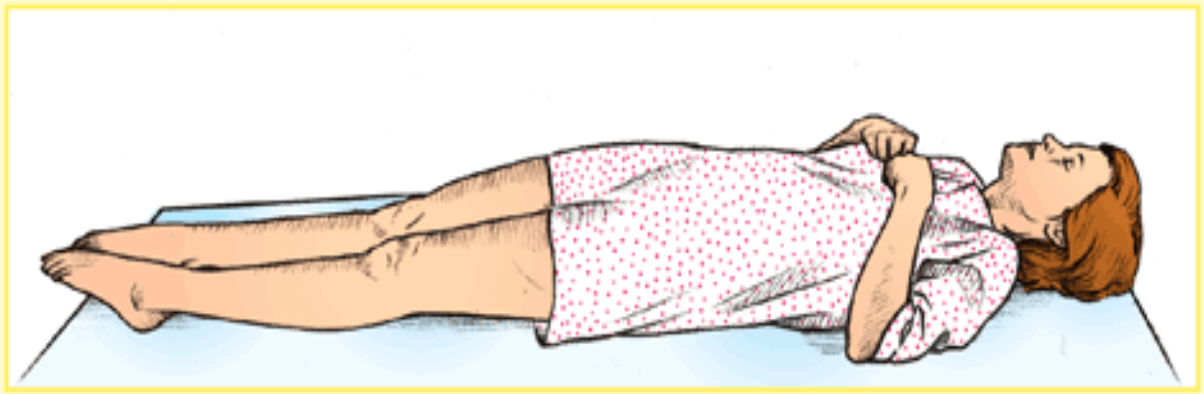


Comparing decerebrate and decorticate postures

Decerebrate posture results from damage to the upper brain stem. In this posture, the arms are adducted and extended, with the wrists pronated and the fingers flexed. The legs are stiffly extended, with plantar flexion of the feet.



Decorticate posture results from damage to one or both corticospinal tracts. In this posture, the arms are adducted and flexed, with the wrists and fingers flexed on the chest. The legs are stiffly extended and internally rotated, with plantar flexion of the feet.



Medical management

- Medical reduction of ICP
  - Position of nursing – head end 30^{o} elevated →Allow good jugular drainage
  - Diuretics – in normovolaemic patient! → reduces cerebral oedema
    - IV Mannitol
      - osmotic diuretic / temporary measure to reduce ICP while transferring
      - AE – hypotension, hyperkalemia/ rebound increase in ICP
    - Hypertonic saline
      - volume expander
      - AE – worsen pulmonary oedema/ Central Pontine Myelinosis
  - Sedation → reduce agitation to reduce ICP and brain metabolism
  - Cooling → 35^{o} C reduces ICP
  - Muscle relaxation (paralysis) → reduce ICP and metabolic rate
  - Anti-epileptics → reduce ICP
    - Phenytoin sodium for 1/52
    - Stop after 1/52 if no seizures
  - Barbiturate induced coma

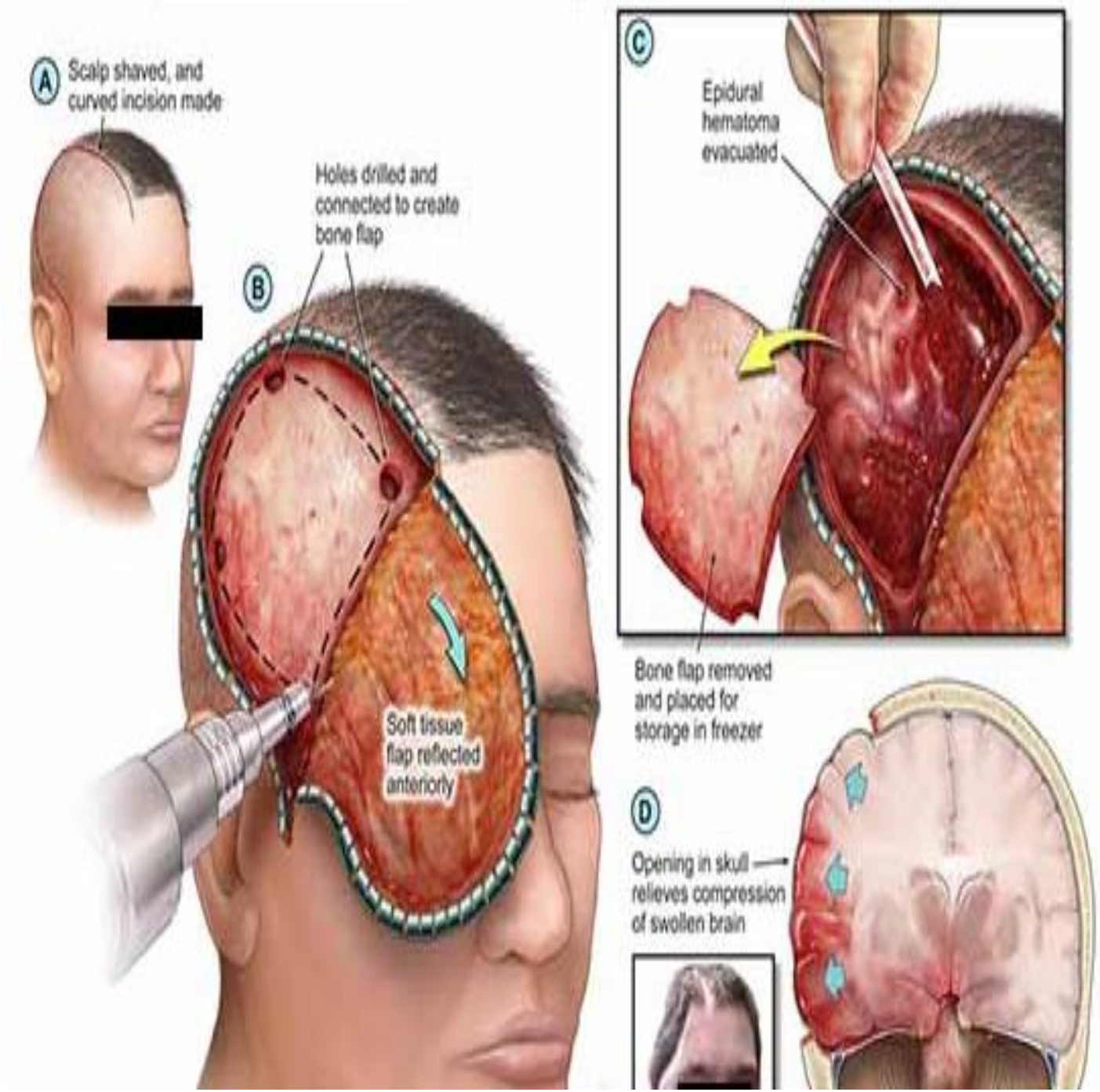
Medical management

- Intubation and ventilation
  - Avoid Valsalva →increase ICP
  - Avoid hypoxia and have normocarbia
  - Avoid respiratory arrest
- Normoglycaemia
  - CBS monitoring – soluble insulin infusions
- Antibiotics
  - Open skull fracture
  - Scalp laceration
  - CSF leak – give antibiotics or not = debatable
- Steroids are detrimental – CRASH trial 2004

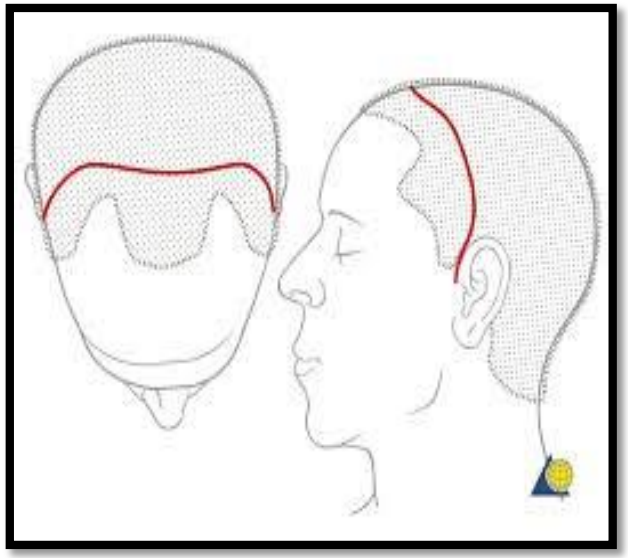
Surgical management

- Decompression craniectomy
  - Remove haematoma and part of the skull (break MK doctrine)
  - ‘trauma’ question mark incision
  - Replace bone flap later

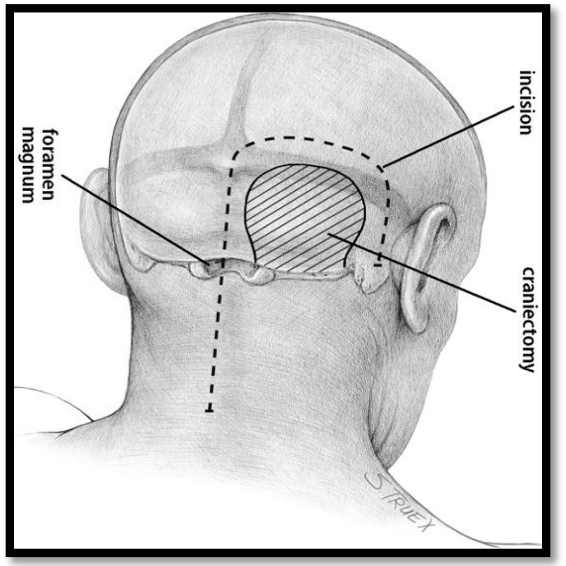
- Burr hole craniotomy
  - To remove a haematoma before transportation
  - **<u>obsolete</u>**



Large question mark incision for tempero-parietal drainage



Frontal craniectomy
Lazy S in the hairline

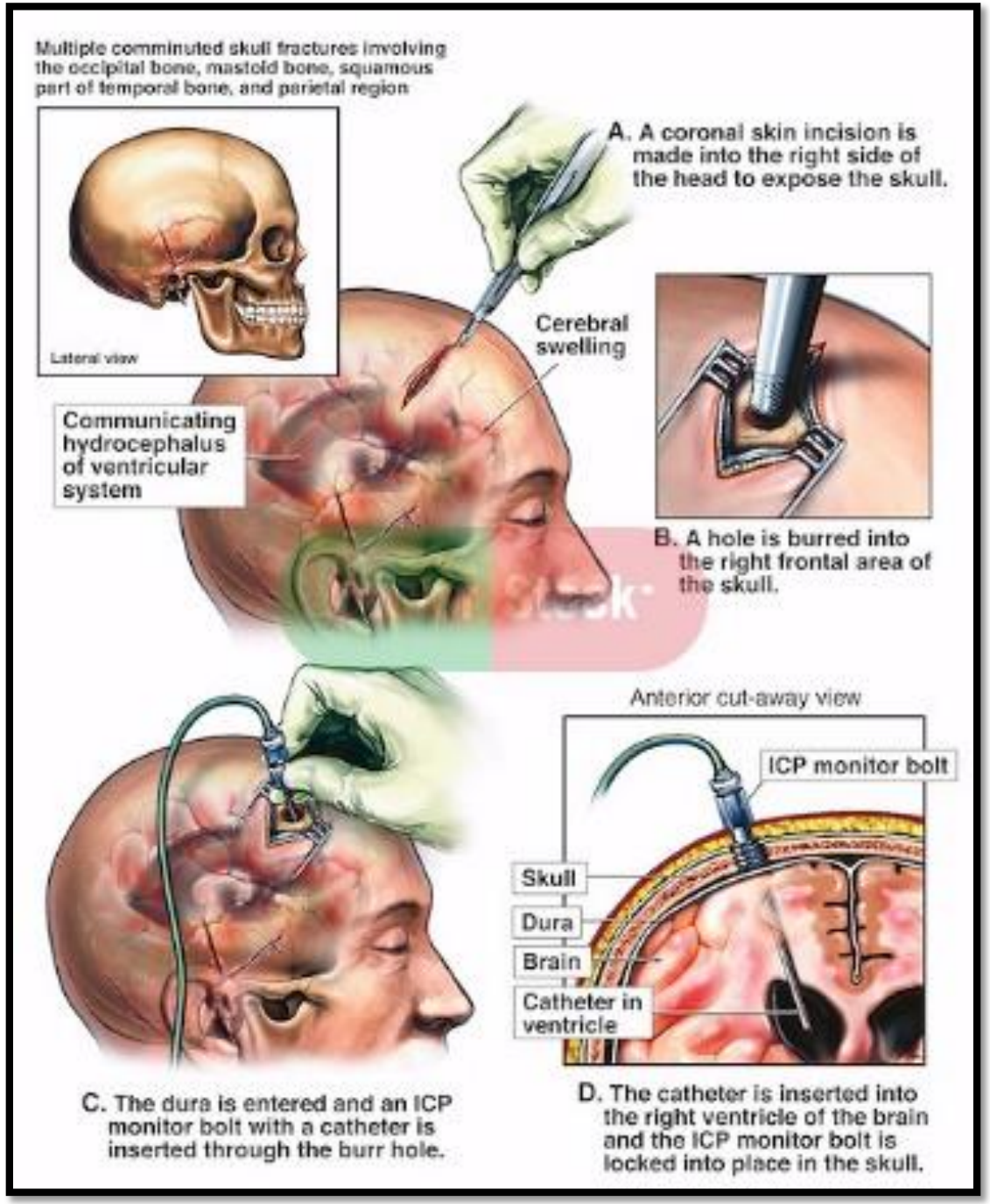


Occipital craniectomy

- Sunken flap or syndrome of the trephined
- A rare AE following craniectomy due to atmospheric pressure

ICP monitoring

- Ipsilateral insertion of an ICP bolt – to measure ICP
- Raid rise - ?rebleed
- Monitors response to treatment



Outcome

Extended Glasgow Outcome Scale (GOS-E)

GOS-E category	Abbreviation	Description
Death	Death	Dead
Vegetative state	VS	Unable to obey commands
Lower severe disability	LSD	Dependent on others for care
Upper severe disability	USD	Independent at home
Lower moderate disability	LMD	Independent at home and outside the home but with some physical or mental disability
Upper moderate disability	UMD	Independent at home and outside the home but with some physical or mental disability, with less disruption than LMD
Lower good recovery	LGR	Able to resume normal activities with some injury-related problems
Upper good recovery	UGR	No injury-related problems

Questions?

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