

TRAUMATIC BRAIN INJURY (I). BRAIN CONCUSSION. TRAUMATIC INTRACRANIAL HEMATOMAS

34484 Pathology of the nervous system

Neurosurgery

Topic 17

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Key points



- **Traumatic brain injury (TBI)**
- **Cranial and brain lesions**
- **Evaluation and staging in emergency room**
- **Intracranial haemorrhage**
 - *Subdural hematoma (SDH) and epidural hematoma (EDH)*
- **Progressive injury: Hypoxia and hypotension in TBI**
- **Severe TBI**
 - *Edema, congestion, ischemia, and brain herniation*
 - *Treatment of severe TBI*
- **Complications and sequelae**



TRAUMATIC BRAIN INJURY (TBI)

- What is a TBI?

- “A traumatically induced structural injury and/or physiologic disruption of brain function as a result of an external force”
 - Result of a traumatic action on the brain and its coverings
- Loss of consciousness may NOT happen (**conscience** ≠ **consciousness**)

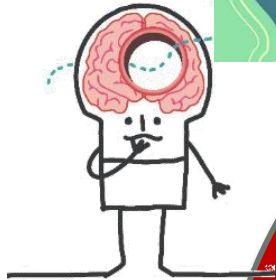


- Importance

- First cause of loss of consciousness in general population
- Most frequent cause of epilepsy 18-25 years
- High mortality
 - 1st cause of death < 20 years and 15-45 years
- Morbidity: sequelae (many and severe) ⇒ 15 % result in disability
 - 20 cases/million people severe disability
 - 40 cases/million people moderate disability
 - Long-term brain deterioration
(memory, movement, senses, emotions)



Sequelae

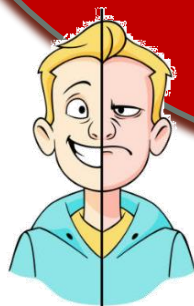


COGNITIVE DEFICITS
Short term memory loss, concentration problems, slow thinking, limited attention, deterioration of different cognitive abilities

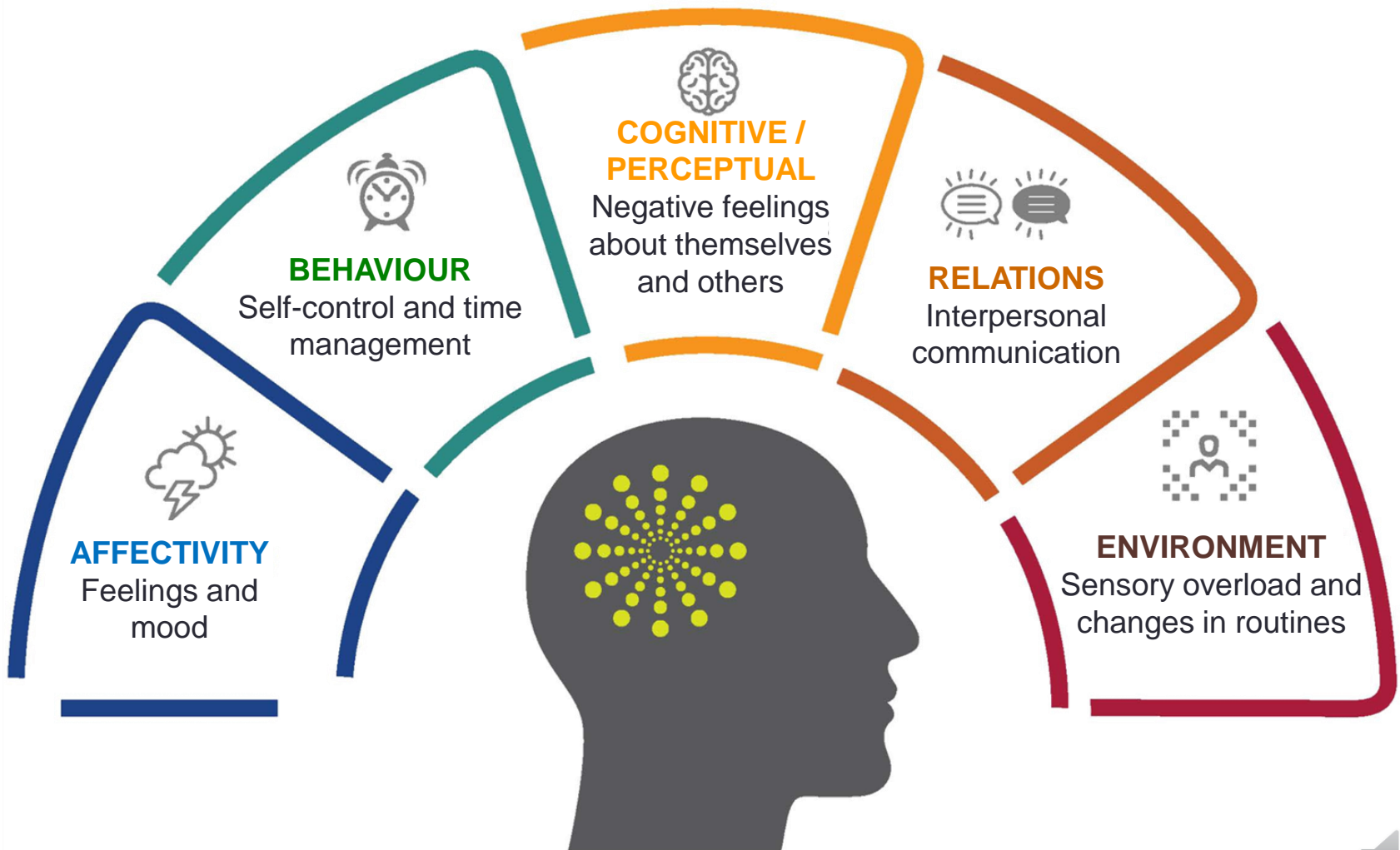
PHYSICAL DEFICITS
Speech, visual, hearing, deficits. Low motor coordination, spasticity, paralysis, epilepsy, loss of balance, and fatigue

TBI

EMOTIONAL DEFICITS
Mood swings, egocentrism. Anxiety, depression, low self-esteem, sexual dysfunction, nervousness, loss of motivation, and uncontrolled emotions



Sequelae: Irritability

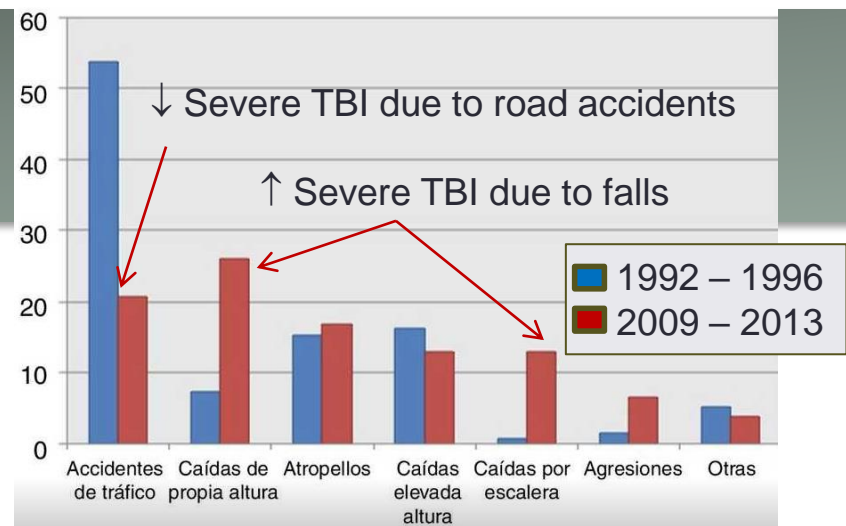


Incidence

- Incidence of TBI per 100,000 people:
 - 2000 emergencies
 - 300 admissions (125 severe)
 - 10-20 deaths
- High mortality (42 % of TBI are severe):
 - 15-17 % TBI result in death
 - Increasing cohort: fall of patients on anticoagulants

TBI as cause of death:

- 1 % deaths
- 25 % deaths due to trauma
- 50 % deaths due to road accidents



Severe trauma with pre-hospital death:

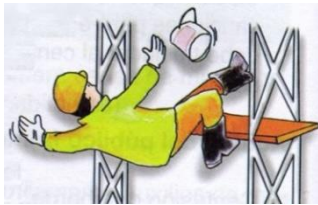
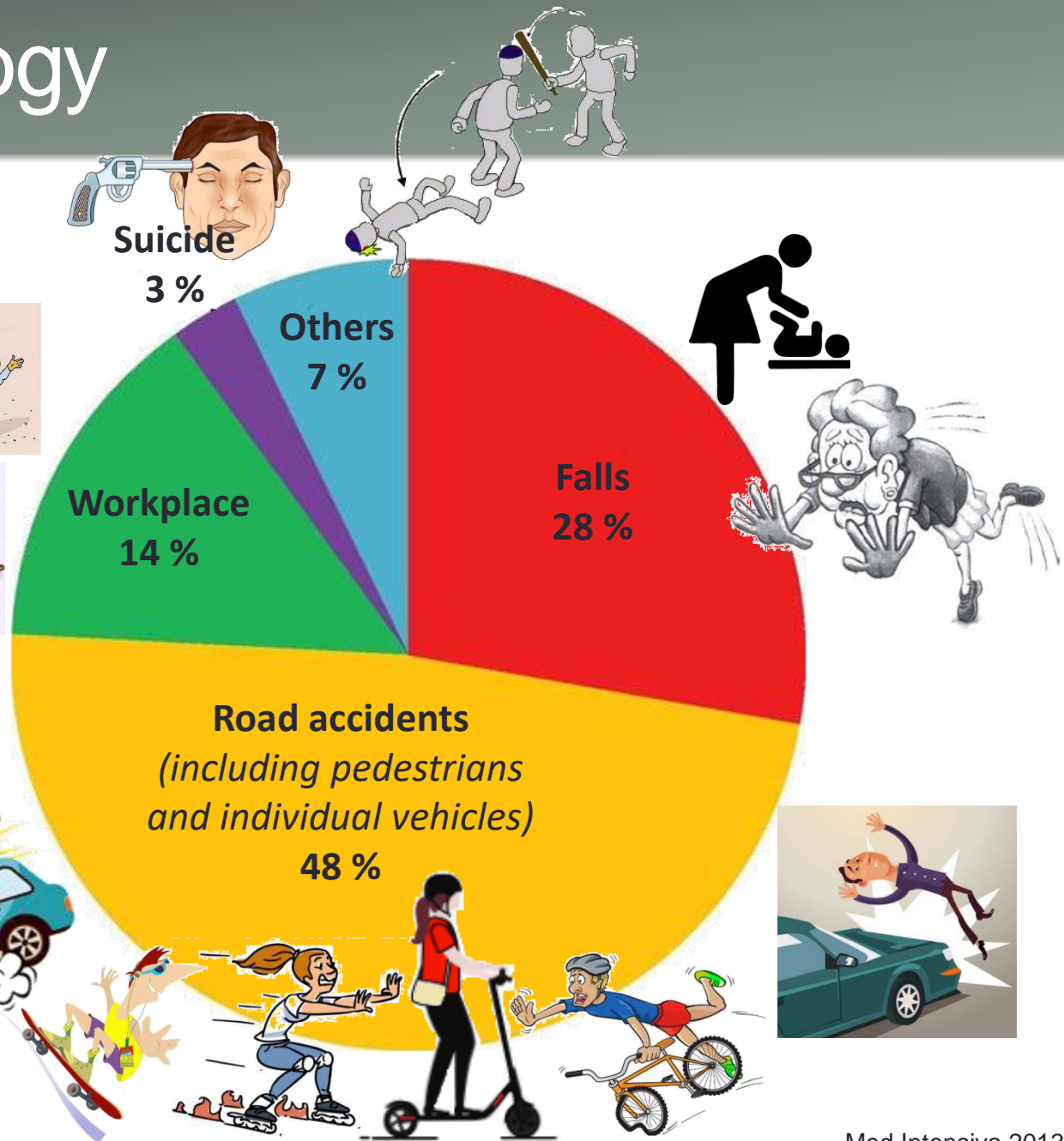
- 2/3 due to polytrauma
- 10 % due to TBI

Mortality of severe TBI that arrives to hospital:

- 35 % due to primary lesions
- 50 % due to secondary lesions
- 8 % due to extracranial complications

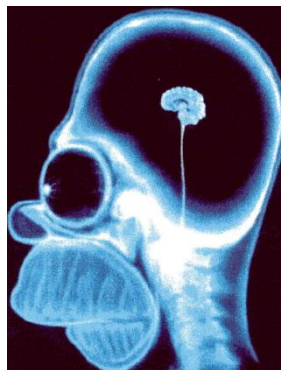
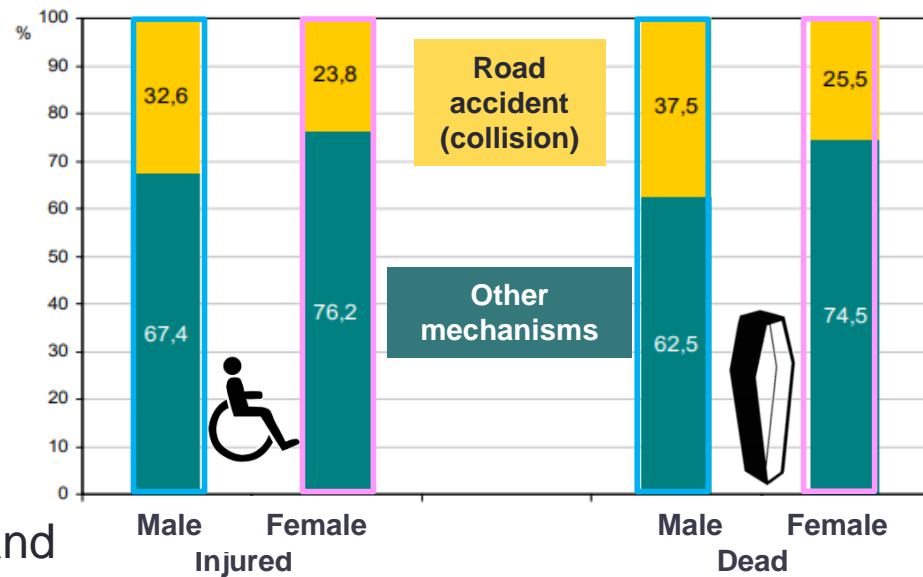


Etiology



Etiology

- Most affected population:
 - Infants: falls > abuse
 - Schoolchildren: bikes, falls, and collisions (road)
 - Teenagers (male): risky attitudes and activities
 - Young male: road accident > workplace and risk activities
 - Older women: other causes (**falls + anticoagulants**)

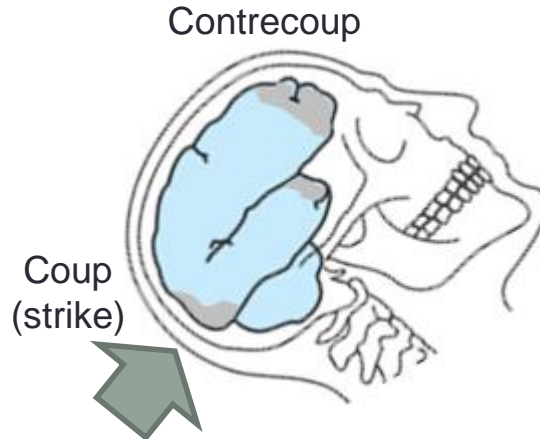


Basic mechanisms of TBI

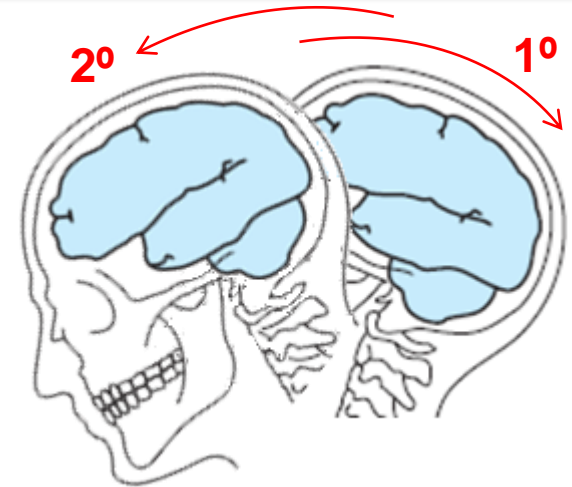
Blunt contusion



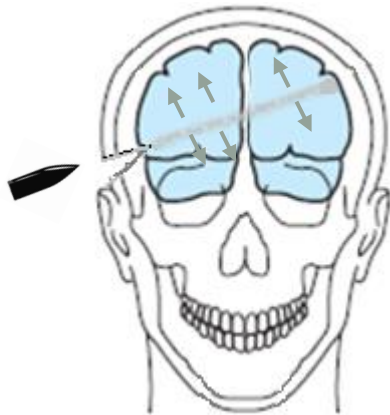
Collision
(direct impact)



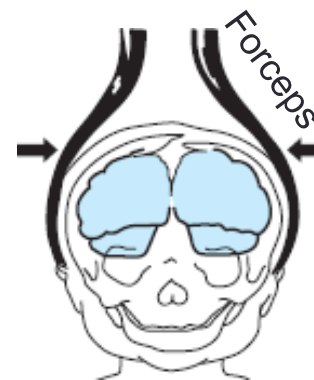
Coup – contrecoup



Acceleration - Deceleration



Penetrating / perforating
(low / high speed)



Compression



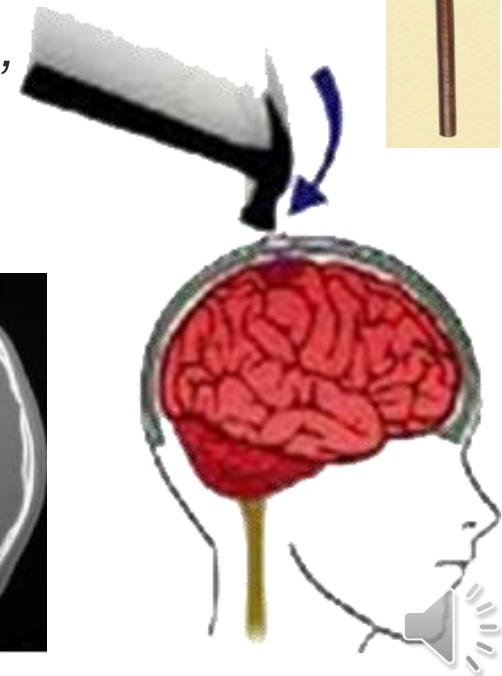
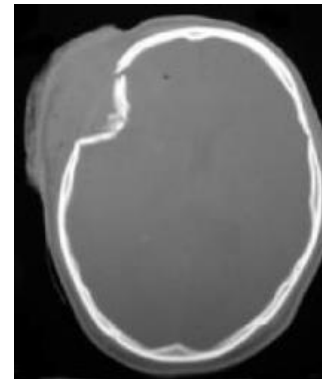
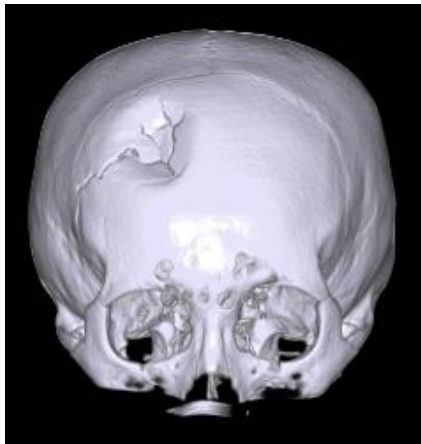
Basic mechanisms of TBI



Collision
(direct impact)

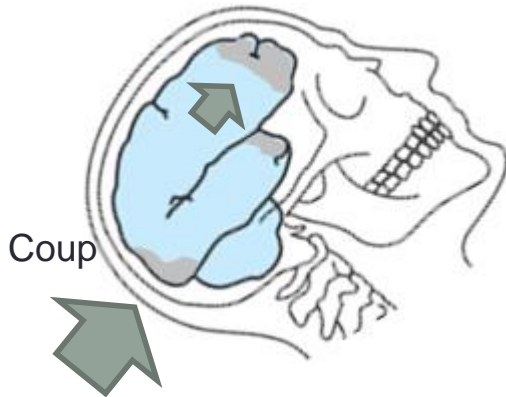
- Direct impact = inelastic collision
 - Impact object against head
 - **Skull** lesions (fracture)
 - *Direct lesion of subjacent brain*
 - *Indirect lesion (contrecoup, haemorrhage)*

Iwisa zulú
(knobkerrie)



Basic mechanisms of TBI

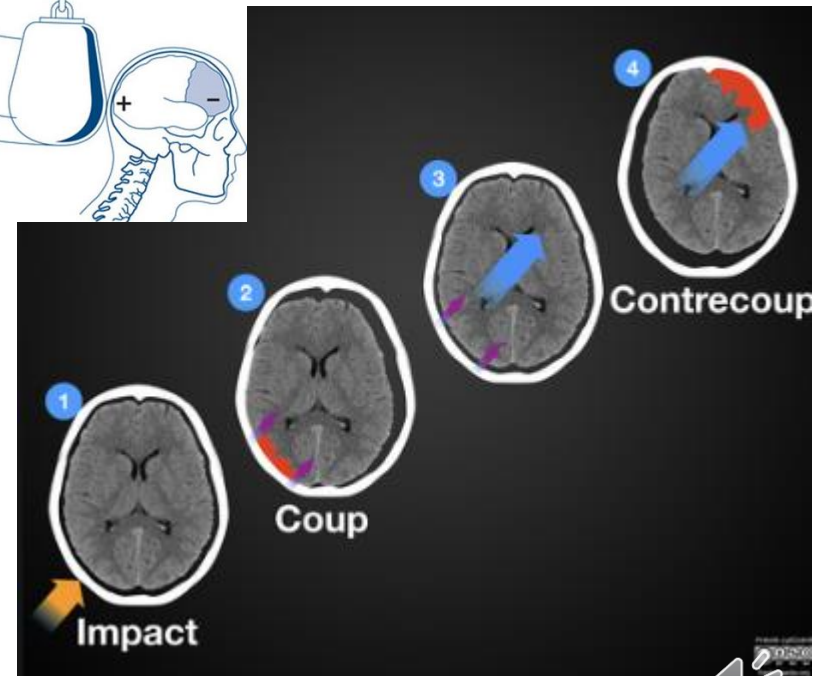
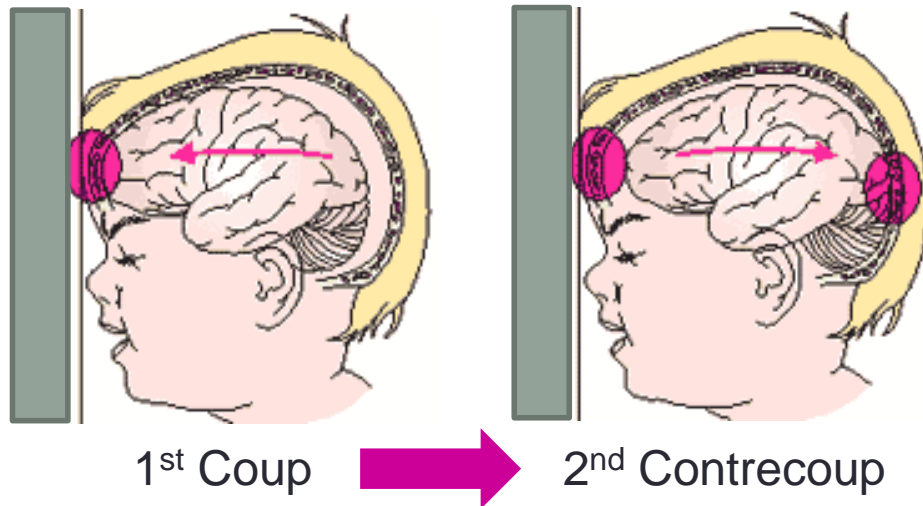
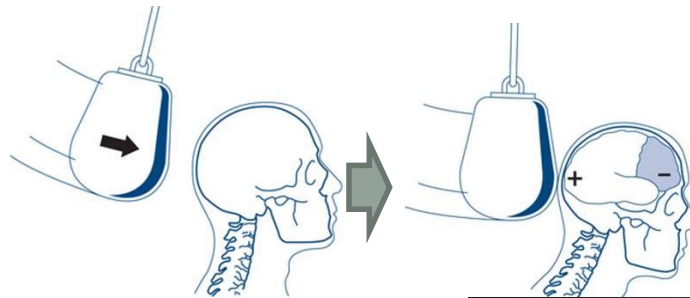
Contrecoup



Coup-contrecoup

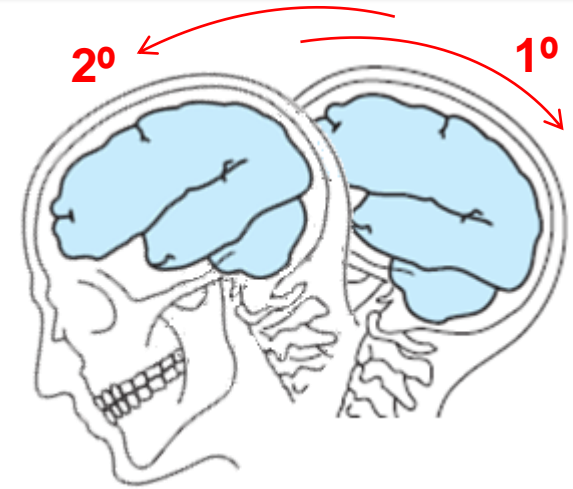
- Coup-contrecoup = elastic collision

- Strike against a stationary object
- Linear or rotational acceleration brain inside skull
- **Brain and vascular lesions**

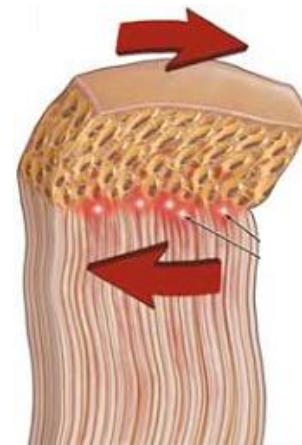
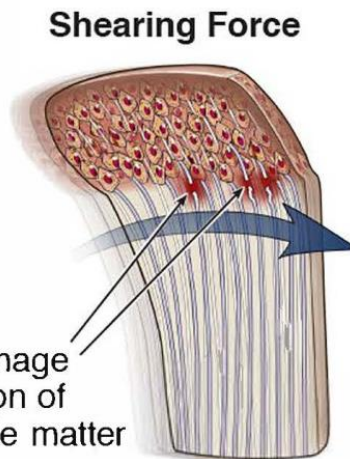
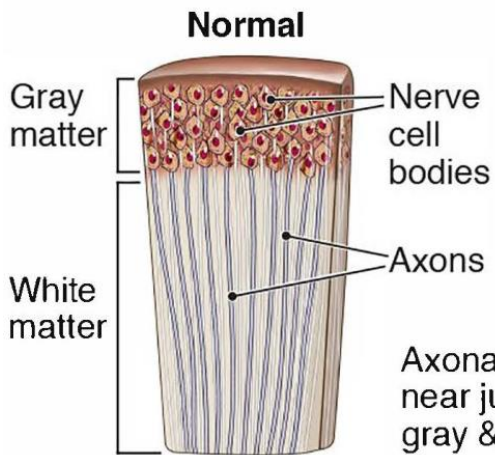


Basic mechanisms of TBI

- Acceleration – deceleration
 - Brisk flexion extension (punch, shaking infants)
 - Shear grey / white matter
 - **Diffuse axonal injury**
 - *Both in brain and in brainstem*
 - *BAD PROGNOSIS*



Acceleration – deceleration



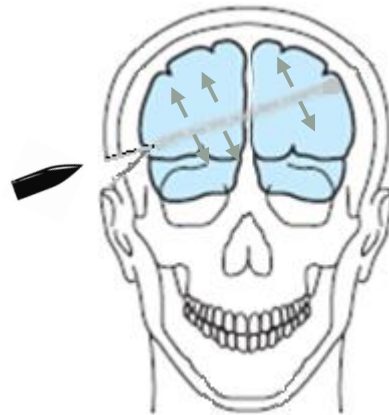
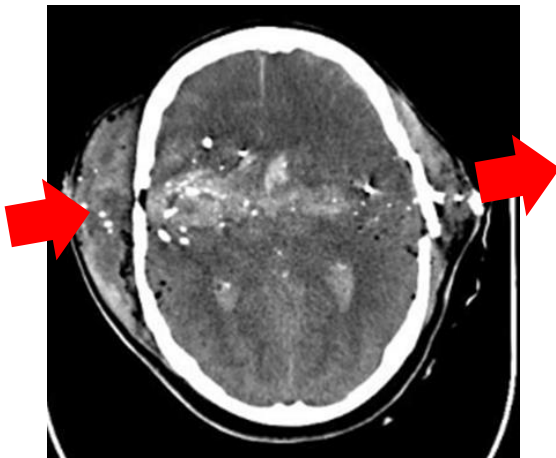
Basic mechanisms of TBI



High speed (bullet)



Low speed (knife, pen)



Perforating / penetrating
(high / low speed)

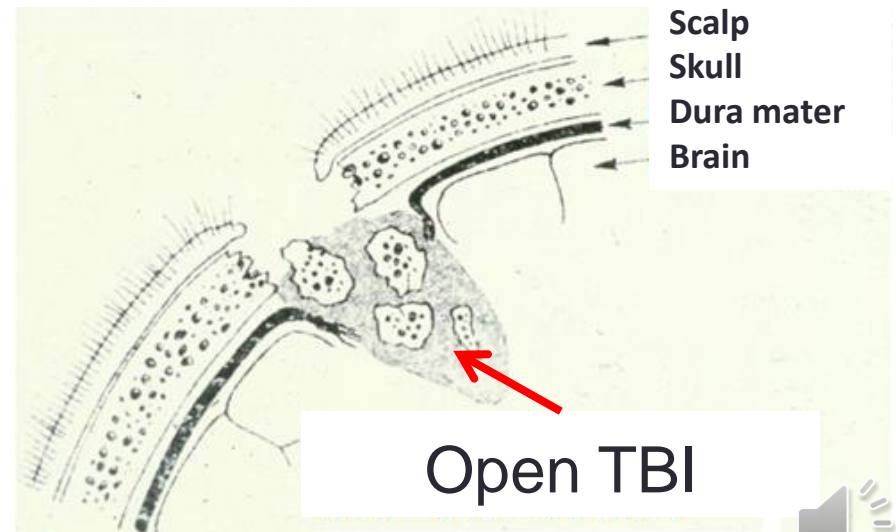
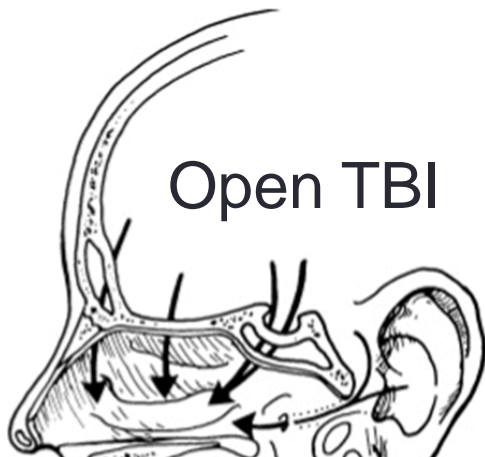


Compression
(forceps, entrapment)

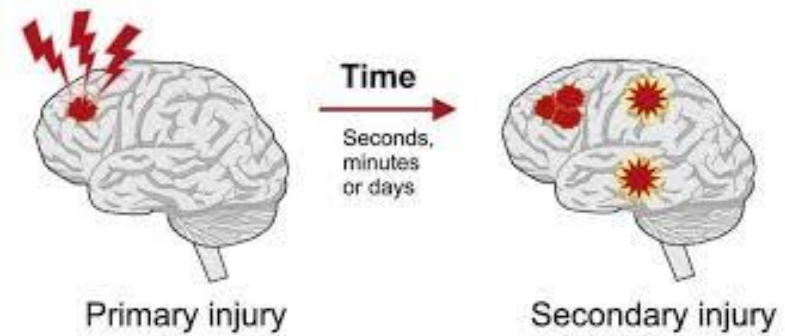


Types of TBI

- According to dura mater:
 - Closed TBI = Dura mater intact
 - Open TBI = Dura mater opened
 - *Possible infection*
 - *Skull base → Direct communication with paranasal sinuses, mastoid, and middle ear*



Types of lesions



- Primary

- Contusion and scalp wounds
- Skull / facial fracture
- Brain concussion
- Brain contusion
- Brain laceration
- Diffuse axonal injury

- Secondary ⇒ ↑ ICP

- Intracranial hematomas
- Brain edema / herniation
- Brain congestion (hyperaemia)
- Extracerebral causes: cerebral ischemia / hypoxia

- Tertiary
(complications and sequelae)

- Hydrocephalus
- Epilepsy
- CSF fistula
- Septic (meningitis, empyema, abscess)
- Post-traumatic encephalopathy



SCALP LESIONS

- Skin abrasion or scrapes (impact)
- Contusion: blood and edema in skin
- Subcutaneous hematoma
 - Children: difficult to differentiate from depressed fracture
- Subgalea hematoma
 - Under the galea, soft, fluctuating
 - DO NOT PUNCTURE
- Subperiosteal hematoma
 - Between periosteum and bone
 - Obstetric trauma (cephalohematoma)
 - *DO NOT PUNCTURE*

• Wounds

– *Not always with TBI*

– Types:

- *Perforating*
- *Incised-blunt*
- *Partial scalp detachment*
 - **S**kin, **C**onnective tissue, **A**poneurosis, **L**oose connective tissue, **P**eriosteum

– Heavy haemorrhage

– Compression

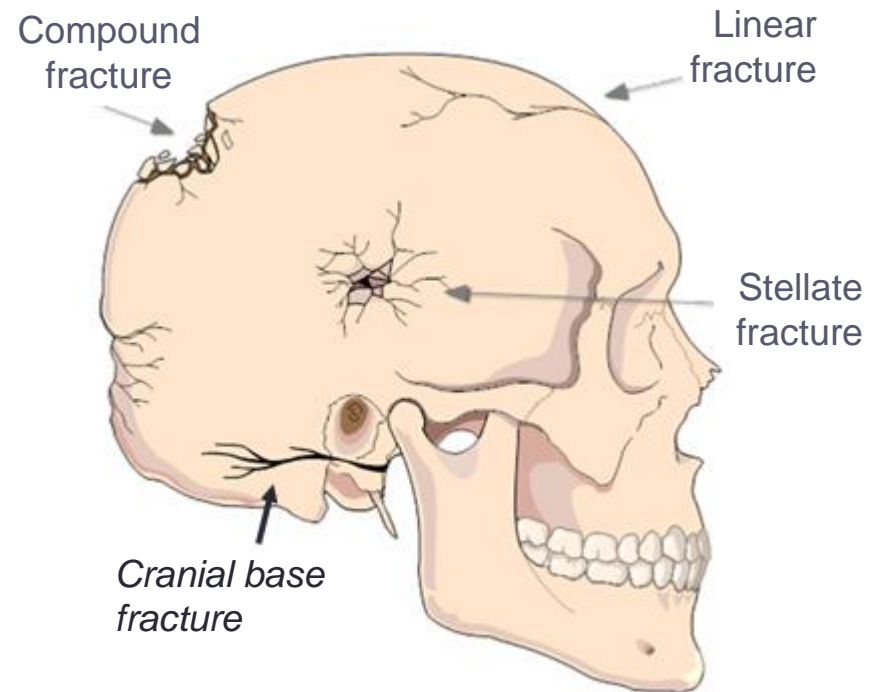
– Rx: fracture and foreign bodies ruled out

– Shaving, cleaning, and suturing



SKULL FRACTURES

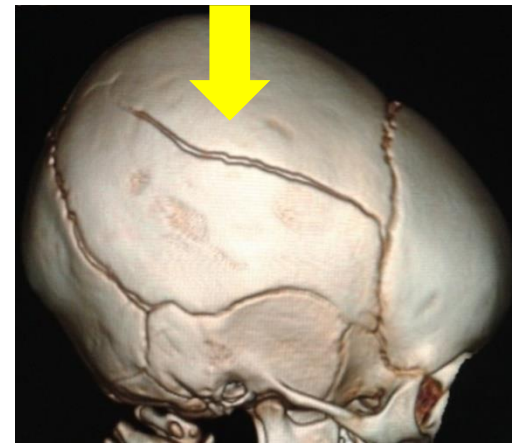
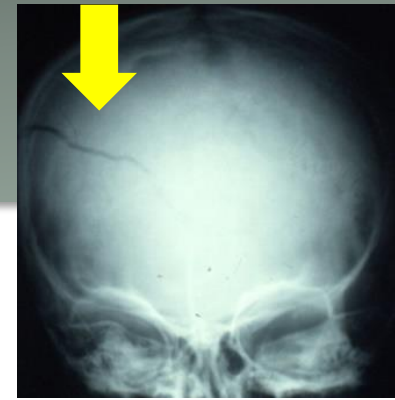
- High-energy impact
- Prognosis determined by brain lesion (NOT by bone)
- Localisation:
 - Vault
 - *Linear and stellate*
 - *Depressed and with sinking of fragments*
 - *Compound*
 - Skull base



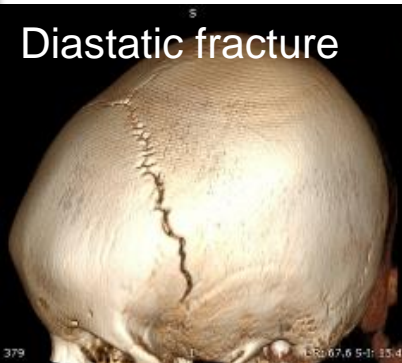
Skull fractures

1. Cranial vault fractures

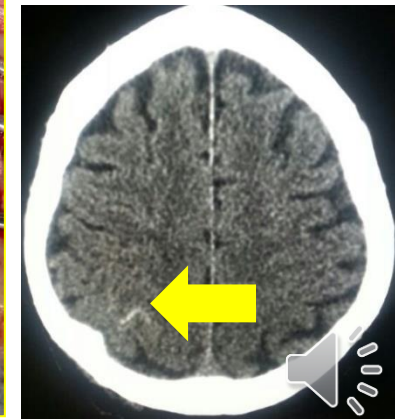
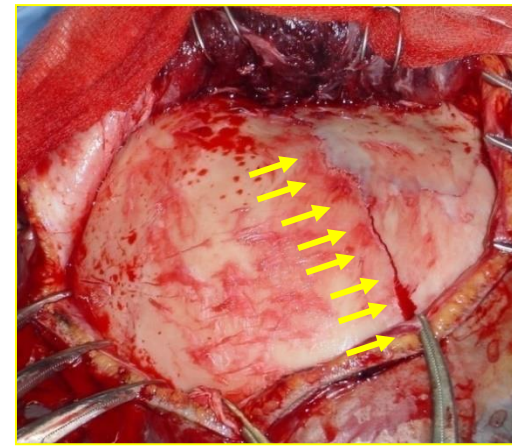
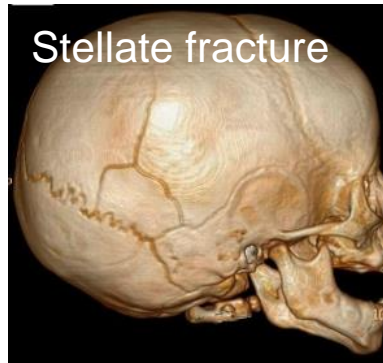
- Linear fracture (80% of fractures)
 - Differentiate from sutures and vessels
 - Diastatic = fracture of a suture
 - Stellate = higher impact
 - Underlying vessels lesion?
- Depressed fracture
- Compound fracture



Diastatic fracture



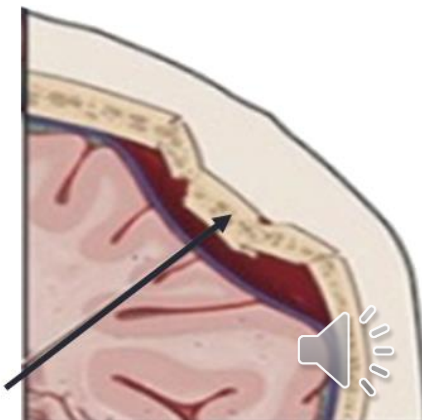
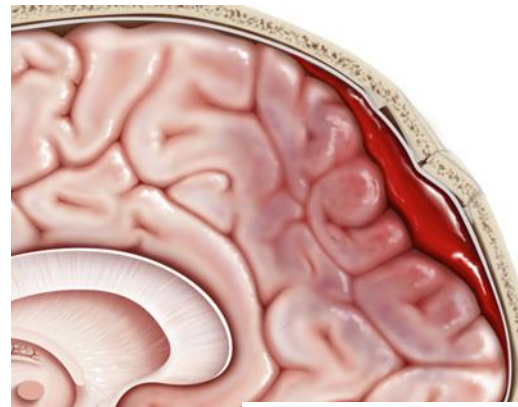
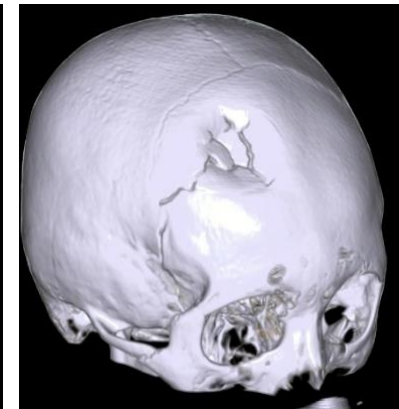
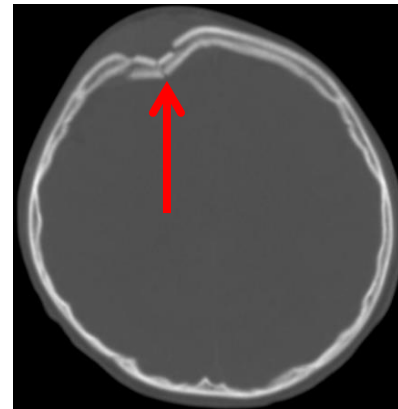
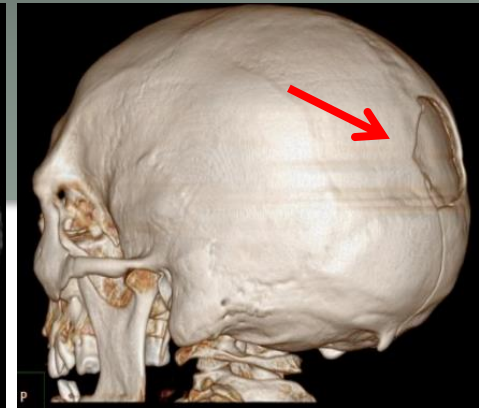
Stellate fracture



Skull fractures

1. Cranial vault fractures

- Linear fracture (80 %)
- Depressed fracture
 - *Outer table sunk below inner table*
 - *Possible contusion of subjacent brain → ↑ Risk of post-traumatic epilepsy*
 - *Usually accompanied by laceration of scalp and dura mater*
- Compound fracture

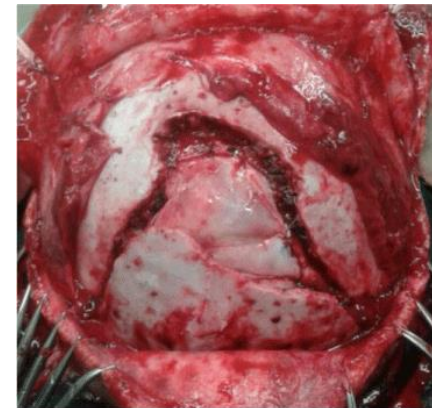
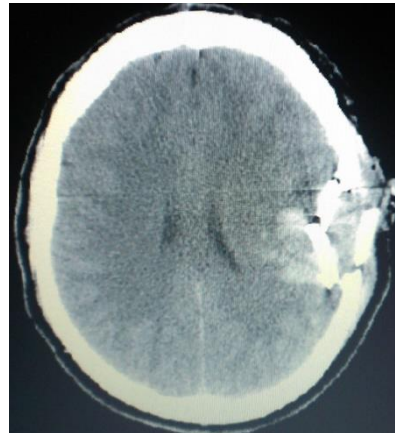
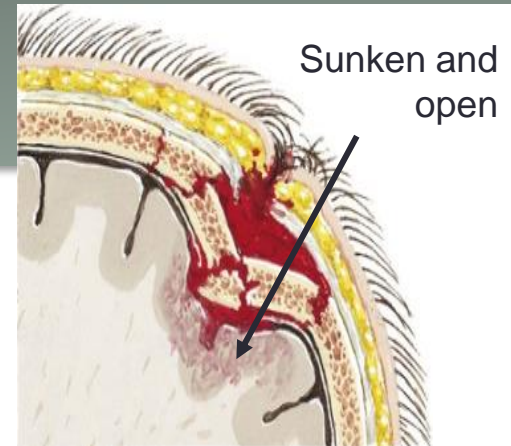


Depressed
Fracture closed

Skull fractures

1. Cranial vault fractures

- Linear fracture (80 %)
- Depressed fracture
- Compound fracture
 - *With bone chips and splinters that exit through skin*
 - *Bone impacts in dura ⇒ Lesion of dura (= OPEN fracture) ⇒ Possible lesion of brain tissue*
 - *Related to severe brain damage*



Skull fractures

1. Cranial vault fractures

- Clinical features: depend on BRAIN lesion
 - *Examination may be normal, especially in linear fractures*
- Diagnosis:

Linear fracture	Depressed or compound fracture
Examination with no findings: head plain x-rays	Urgent head CT
If clinical suspicion of brain lesion, or plain x-ray shows fracture, request urgent head CT	
Consider risk of haematoma	Consider risk of opened dura mater (meningitis)

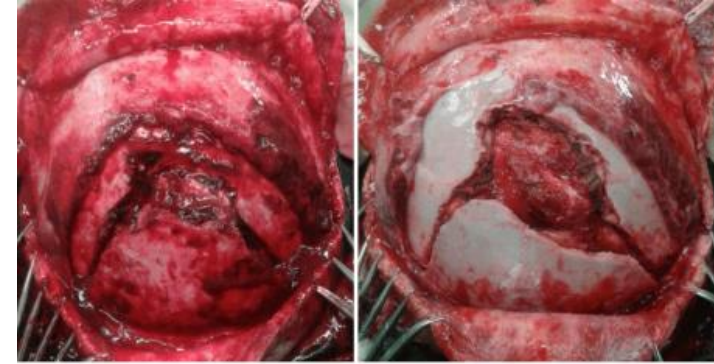
- Treatment



Skull fractures

1. Cranial vault fractures

- Clinical features
- Diagnosis
- Treatment:



Linear fracture

24-hour observation

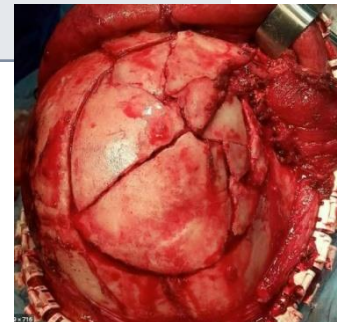
No treatment needed

Depressed or compound fracture

Hospital admission

Surgical intervention: elevate fragments, remove splinters, and bruised brain area (avoid possible epilepsy foci). Antibiotic.

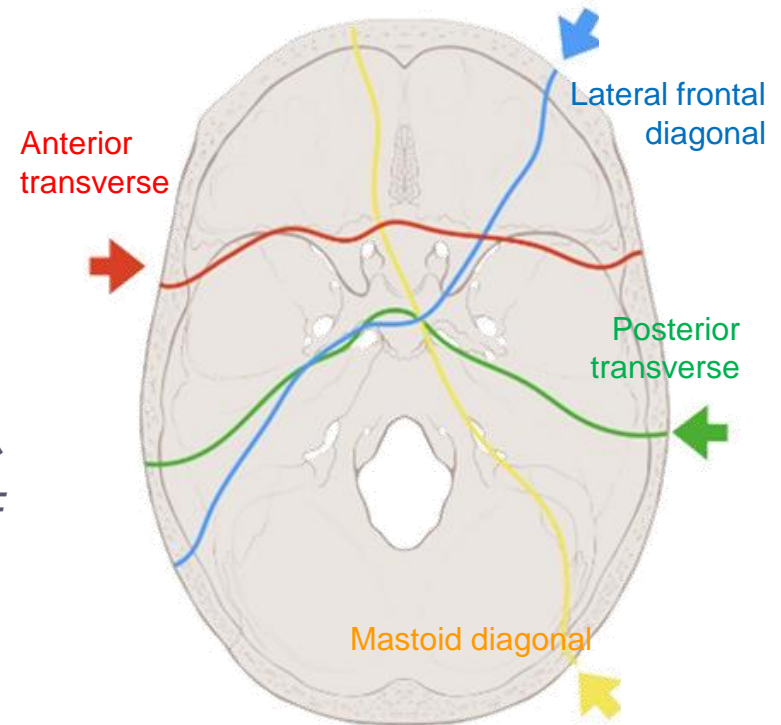
Prognosis depends on brain injury



Skull fractures

2. Skull base fractures

- Very strong traumatism
- Most frequent: frontoethmoidal and petrous bone (anterior and middle fossae)
- Affect structures:
 - *Paranasal sinuses or petrous bone* ⇒ rupture of dura mater ⇒ leakage CSF or blood (rhinorrhoea, otorrhea), pneumocephalus
 - *Cranial nerves*
 - I-VI in anterior fossa
 - VII and VIII > V-VI in petrous bone Fracture (Graneligo syndrome)
 - IX-XII in posterior fossa
 - *Large vessels (carotid), less common*



Transsphenoidal fracture



Skull fractures

2. Skull base fractures

– Clinical features

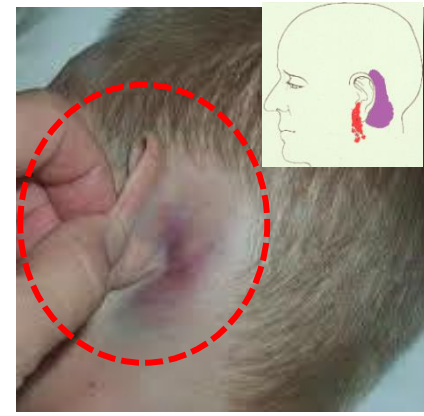
Periorbital
ecchymosis
(*racoon
eyes*)



Subconjunctival hemorrhage



*Retromastoid ecchymosis
(Battle sign)*



LCR

CSF leak
(*rinoliquorrhea*)



Hemotympanum,
otorrhagia



V-XII cranial
nerves palsy

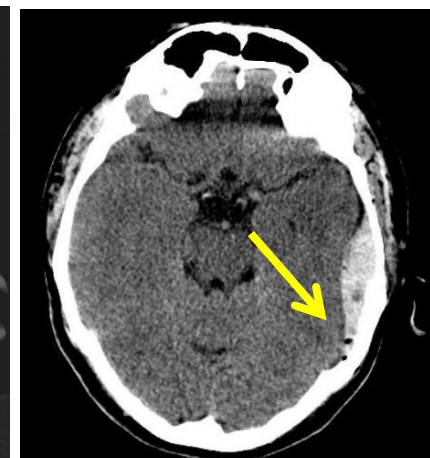
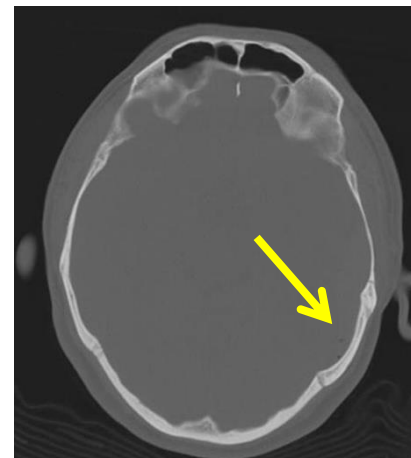
Skull fractures



Pneumocephalus (X-ray and CT scan)

2. Skull base fractures

- Clinical features
- Diagnosis: CT
 - *Suspicion: periorbital or retromastoid hematoma*
 - *May associate dural tear → risk of meningitis (pneumococcus)*
 - *Pneumocephalus = open traumatic brain injury*
- Treatment



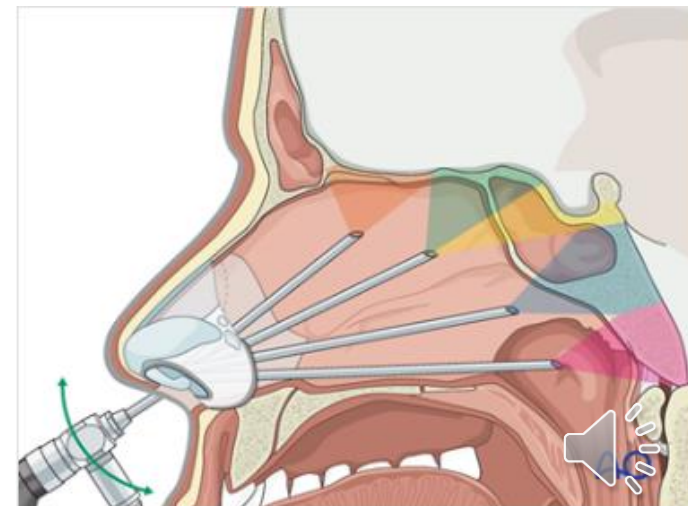
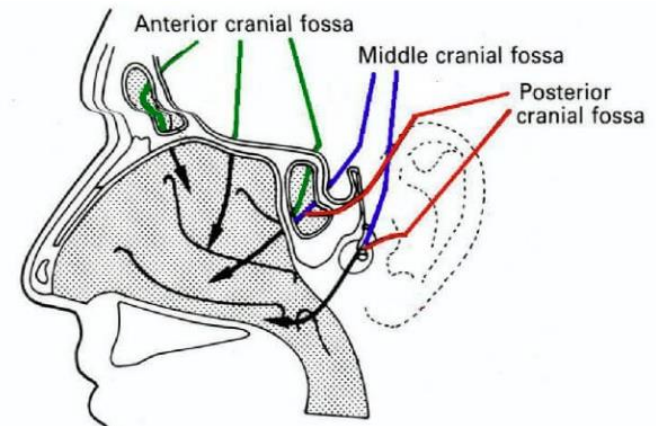
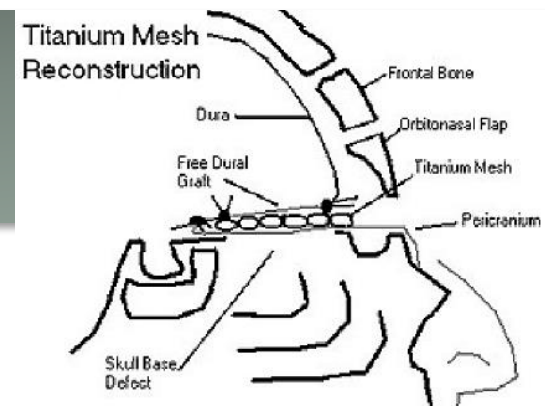
*Left: Right petrous-temporal fracture (carotid channel → request angiography to rule out vascular lesion)
Right: Same patient, left temporoparietal fracture with left epidural hematoma (air inside)*



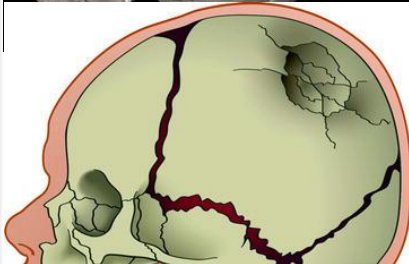
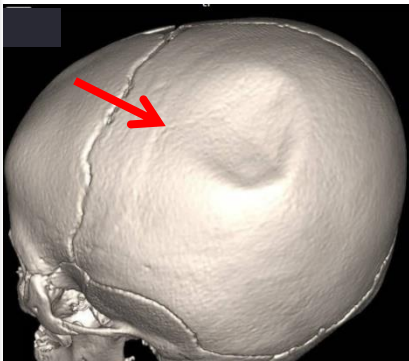
Skull fractures

2. Cranial base fractures

- Clinical features
- Diagnosis
- Treatment
 - *24-h observation, even if no focal signs or loss of consciousness*
 - *Most do not need treatment*
 - *If liquorrhea, do NOT plug (↑ risk meningitis)*
 - *If CSF leak:*
 - Antibiotics
 - Conservative treatment: bed rest 7-10 days
→ repeated lumbar punctures or lumbar drain
 - Surgical treatment (after 3 weeks): dural repair (endoscopic or craniotomy)



Skull fractures



*Ping pong fracture
(pond fracture)*

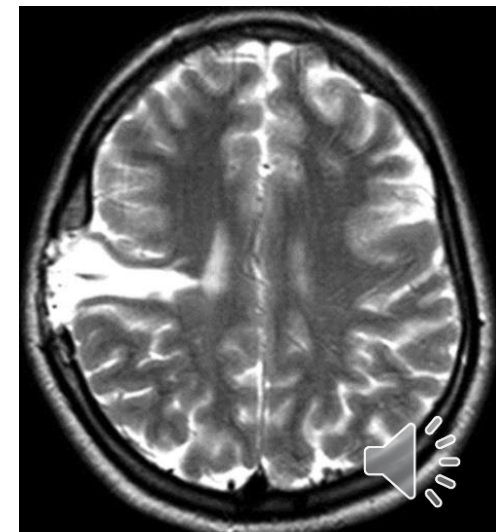
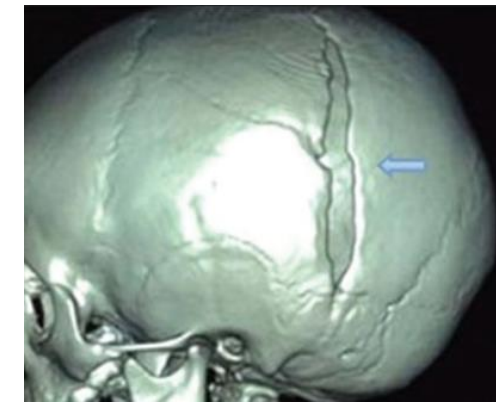
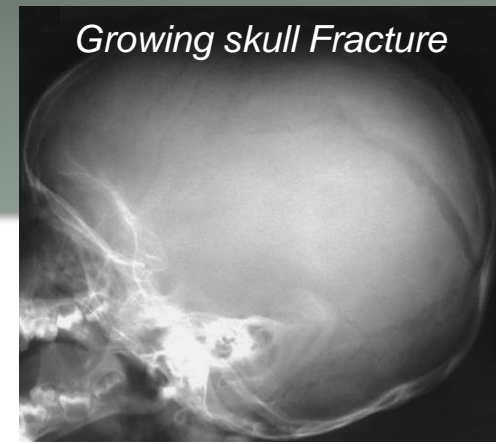
3. Skull fractures in paediatrics

– Ping pong skull fracture

- *Depressed skull fracture with no skin or dura lesions*
- *Surgical treatment*

– Growing skull fracture

- *Enlarging fracture, post-traumatic leptomenigeal cyst*
- *Fractures with dural tear*
- ⇒ *Arachnoid herniates through the fracture*
- ⇒ *Pulsing CSF → Progressive enlargement of the fracture*
- *Surgical treatment*



PRIMARY BRAIN DAMAGE

- General or focal
- Cortex or brainstem
- According to loss of consciousness (OBSOLETE):
 - *Commotion (< 6 hours, no pathological lesions)*
 - *Concussion (> 6 hours, with microscopic lesions)*
 - *Laceration (with macroscopic lesions)*

Not reported in
international literature

Consciousness ↔ brainstem damage (due to swaying or secondary ICHT)

Amnesia ↔ diffuse cortical damage (depends on energy of traumatic agent and severity of TBI)



Brain concussion

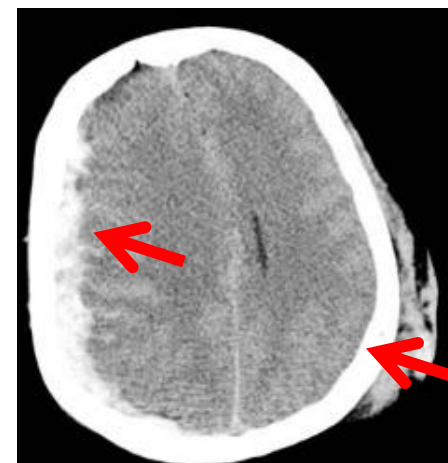
- “Immediate and transitory loss of consciousness, of variable duration (<6 hours), secondary to non-penetrating traumatic brain injury” (violent shaking of the brain)
 - Patient immobile, shallow breathing, light pulse, no response to external stimuli, mydriasis, no swallowing / cough / pupillary reflex
 - Short period of amnesia, no focal deficits
 - Other symptoms: blurred vision, nausea/vomiting, headache, light-headedness, strange behaviour, lack of coordination and concentration...
- No image or pathology lesions
 - *Biochemical disfunction with ↓ mitochondrial ATP ± alterations excitatory neurotransmitters*
- Does not need specific treatment



Brain contusion



- Produced by mechanical forces that move the brain → areas of coup and contrecoup
 - Direct impact, acceleration-deceleration
- From petechiae to haemorrhagic destruction and extensive necrosis (CT, MRI)
 - Possible deep haemorrhage due to torsion and shearing of brain and vessels
 - Rupture blood brain barrier → vasogenic edema, ↑ICP
- Clinical features: anodyne ⇒ focal lesion ⇒ complex and severe picture
- May require treatment to avoid secondary lesions and/or ↑ICP

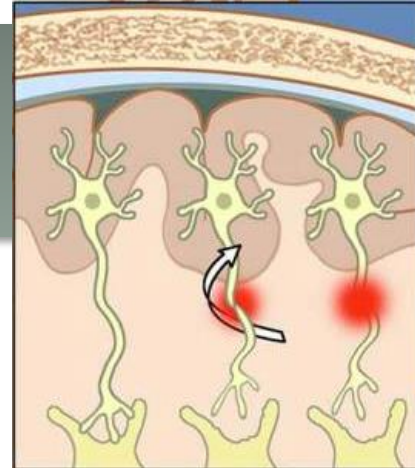


Coup and contrecoup

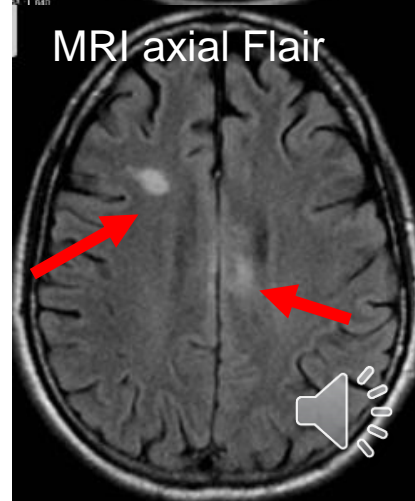


Diffuse axonal injury

- Primary lesion due to sudden rotational acceleration-deceleration movement
 - Shear between grey and white matter → diffuse injury in axons
 - Early and sustained deterioration of level of consciousness
- CT / MRI
 - Microhaemorrhages in corpus callosum, cortico-subcortical junction, and brainstem
 - MRI: diagnosis of choice, even with normal CT
- Bad prognosis
 - First cause of post-traumatic vegetative state (occurs in 40-50 % of severe traumatic brain injury)

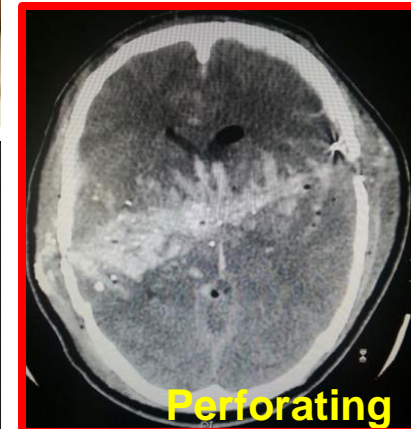
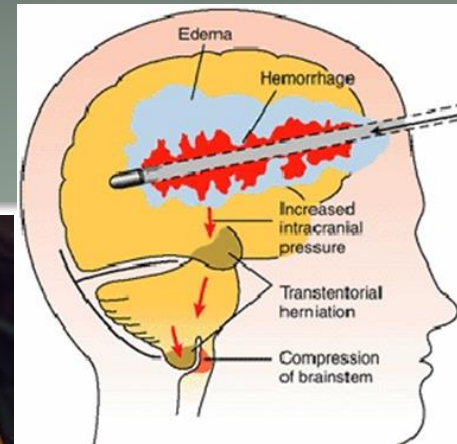
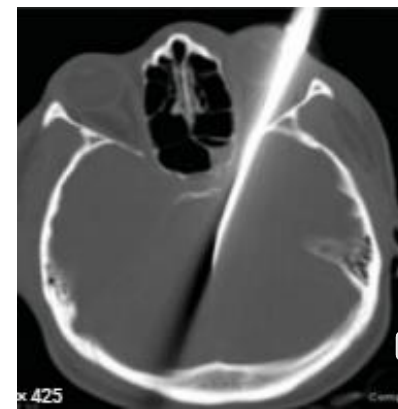
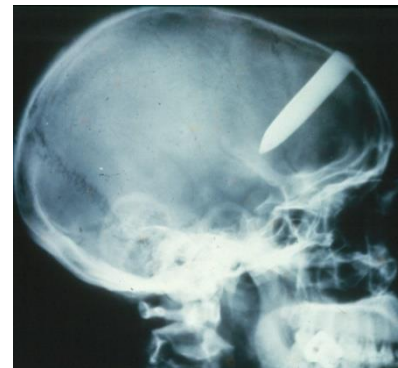


MRI axial Flair



Brain laceration

- Affects skull, dura, and brain
- Types:
 - Stab or sharp item (penetrating, slow speed) ⇒ focal lesion
 - *Knife, nail, harpoon, scissors, pen*
 - *Squama temporalis, orbit, or anterior fossa floor*
 - Firearm (penetrating or perforating, high velocity) ⇒ expansive wave ⇒ Greater destruction of nervous tissue
 - *20 % survive initial impact, only half of these individuals survive the consequences*



INITIAL EVALUATION OF TBI

- History of the facts
 - Hit, run over, fall
 - Driver, accompanying person, passenger
 - Car, motorbike, bike, bus, individual 2-wheel vehicle
 - Fall, sports accident, aggression, self-aggression
- Recent personal history
 - Drug, food (latex-fruit), environmental allergies
 - Last intake of solids, liquids, medication, drugs
- Level of consciousness
 - How has it changed since the accident
 - Normal consciousness, bradypsychia, space-time disorientation, stupor, coma
- Consider polytrauma



Initial exam: "visual exam"



Headache



Vomiting



Confusion



Shock and other lesions



Signs of skull fracture

**RED FLAG
SIGNS?**

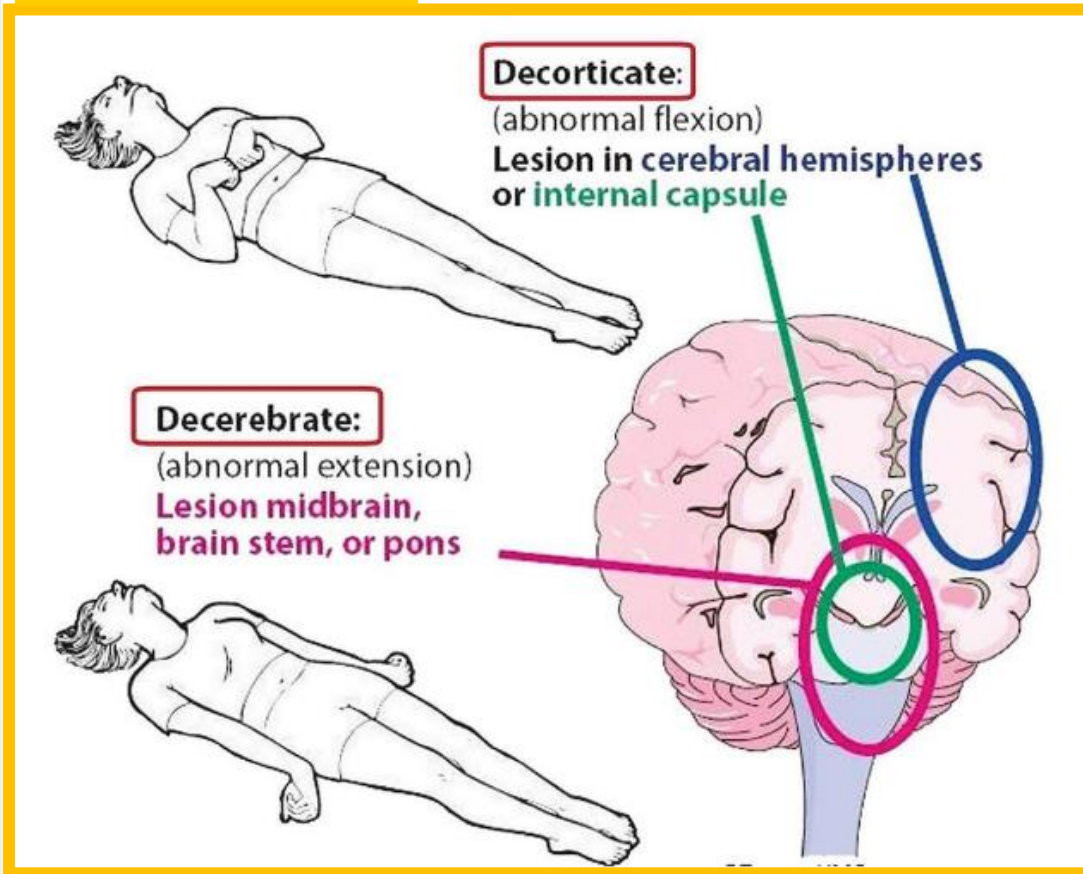
Normal consciousness
Bradypsychia
Disorientation
Stupor
Coma



Initial exam: "red flag signs"



*"Upper, arms up.
 Lower, arms down"*



Neurological focal signs



Mydriasis / anisocoria

Normal consciousness
 Bradypsychia
 Disorientation
Stupor
Coma



Cushing's triad:

- Hypertension
- Bradycardia
- Irregular breathing



Initial exam: “fast examination”

• Patient with stupor or coma:

- Response to pain
- Facial
- Movement of limbs
- Pupils
- Neck stiffness
- Plantar cutaneous reflex

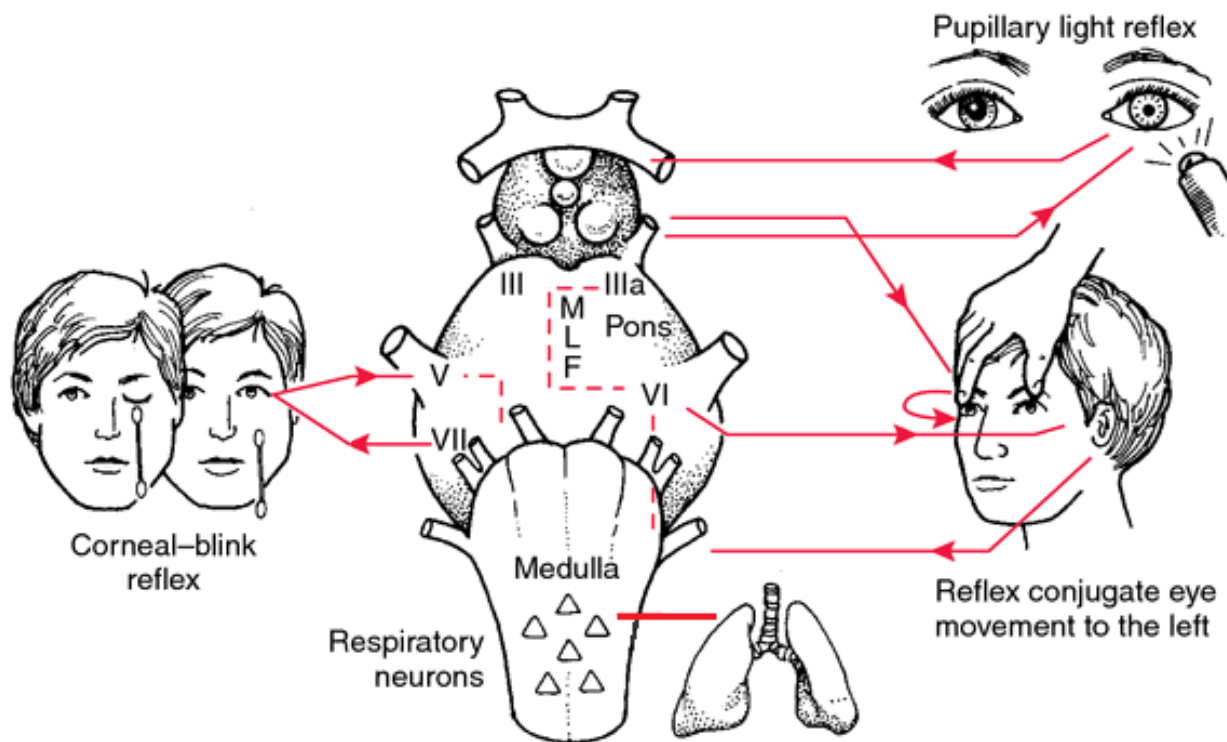
• How to do it:

- Press a fingernail
- Open an eyelid before a light
- Exam neck stiffness (except if there could be cervical fracture)
- Plantar cutaneous reflex (cortico-spinal pathway, Babinski)



Lesion site localization through neurological exam findings

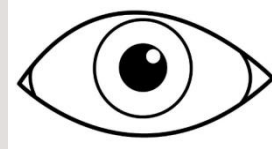
- Pupillary dilatation = mesencephalon
- Corneal reflex = fifth cranial nerve = pons
- Oculocephalic reflexes = eighth cranial nerve = pons
- Respiratory abnormalities = medulla



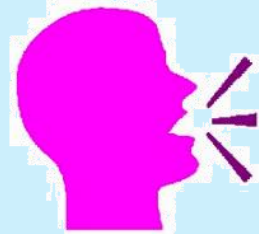
Initial exam

- *Glasgow coma scale*
 - Traumatic brain injury or any brain damage
 - **Best** ocular, verbal, and motor response
 - Routine in emergencies and ICU ⇒ **progression**
 - **3 to 15 points**
- *GCS-P (2017)*
 - Pupil reactivity score (subtract points)
 - 2 = 2 pupils unreactive
 - 1 = 1 pupil unreactive
 - 0 = normal pupils
- Modified for children and intubated patients

GCS



Spontaneous	4
To sound	3
To pressure	2
None	1



Oriented	5
Confused	4
Inappropriate	3
Sounds	2
None	1



Obey commands	6
Localising	5
Normal flexion	4
Abnormal flexion	3
Abnormal extension	2
None	1

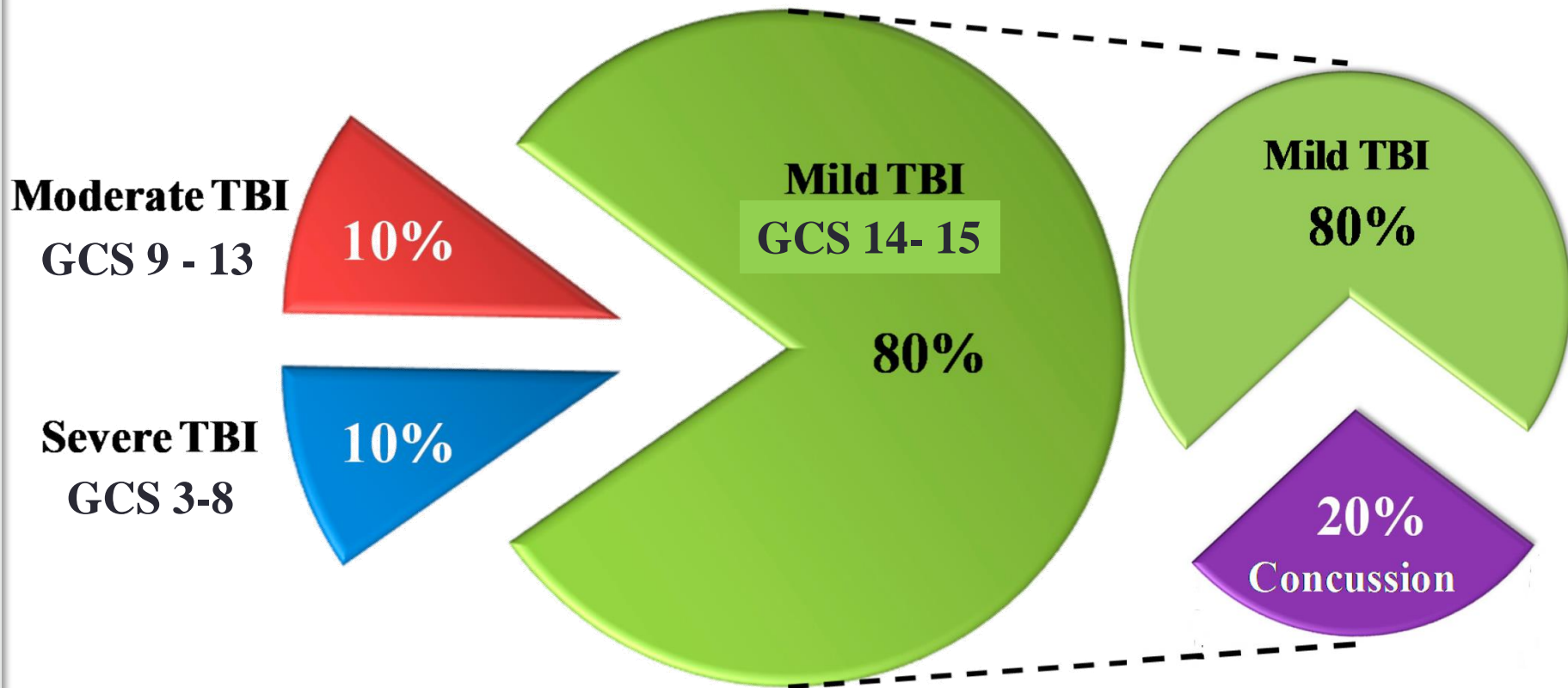


15-14 = Mild

13 – 9 = Moderate

≤ 8 = Severe

Initial exam: classification



- *GCS-pupillary reaction*
 - Subtract 1-2 points if there is 1-2 unreactive pupil → MAY MODIFY STAGING
 - Patients with GCS 14: if 1 pupil is unreactive, patient is GCS-P 13 ⇒ MODERATE Traumatic brain injury (ADMISSION TO HOSPITAL)



TRAUMATIC BRAIN INJURY MANAGEMENT IN EMERGENCY ROOM

- Low-risk patients
 - Headache, dizziness, abrasion, or contusion of the scalp
 - *Treatment: home observation by a responsible adult*
 - *Do not require image tests EXCEPT:*
 - *Coagulopathies, alcoholism, drug abuse, epilepsy, or elderly with some disability*
- Moderate-risk patients
- High-risk patients

Tabla V. Recomendaciones para observación domiciliaria después de un traumatismo craneoencefálico

- El niño debe ser vigilado por una persona responsable, al menos, durante las 24 horas siguientes, por si se detecta algún problema
- Debe acudir de nuevo al hospital para ser reevaluado, si observa alguna de las siguientes alteraciones:
 - Dolor de cabeza intenso o progresivo
 - Comportamiento anormal: confuso, irritabilidad inconsolable, somnoliento con dificultad para despertar
 - Vómitos
 - Movimientos anormales, dificultad para caminar, pérdida de fuerza en alguna extremidad
 - Alteraciones en la visión, pupilas de tamaño diferente
 - Salida de líquido o sangre por la nariz o los oídos
- Puede dejarle dormir, pero debe despertarle cada 2 a 4 horas y comprobar brevemente sus reacciones
- Puede tratar el dolor de cabeza con paracetamol o ibuprofeno a las dosis habituales



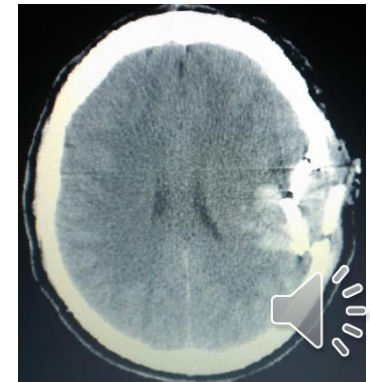
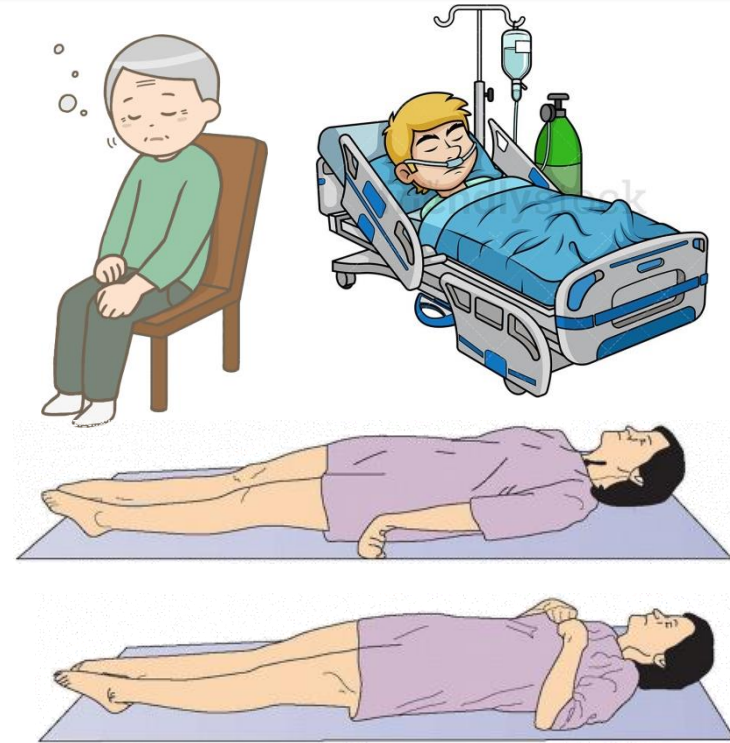
TRAUMATIC BRAIN INJURY MANAGEMENT IN EMERGENCY ROOM

- Low-risk patients
- Moderate-risk patients
 - Post-traumatic amnesia, loss of consciousness
 - Significant subgaleal swelling
 - Vomiting, seizures, progressive headache
 - Infants < 2 years
 - History of drug abuse
 - *Head CT and admission to 24-hour observation*
- High-risk patients

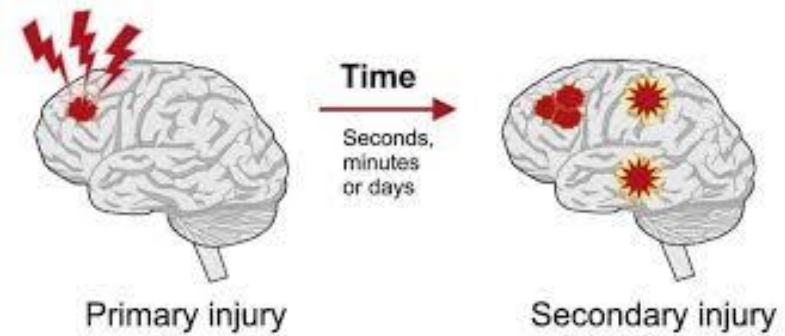


TRAUMATIC BRAIN INJURY MANAGEMENT IN EMERGENCY ROOM

- Low-risk patients
 - Moderate-risk patients
 - High-risk patients
 - Decreased or decreasing level of consciousness
 - GCS < 14
 - Neurological focal signs
 - Penetrating traumatic brain injury, skull depressed fracture
- *CT scan + admission (ICU?)*
- *Evaluation: neurosurgery*



Types of lesions



- Primary

- Contusion and scalp wounds
- Skull / facial fracture
- Brain concussion
- Brain contusion
- Brain laceration
- Diffuse axonal injury

- Secondary $\Rightarrow \uparrow$ ICP

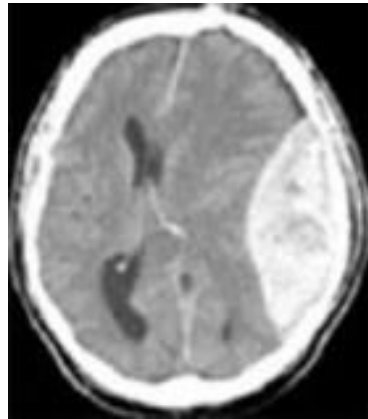
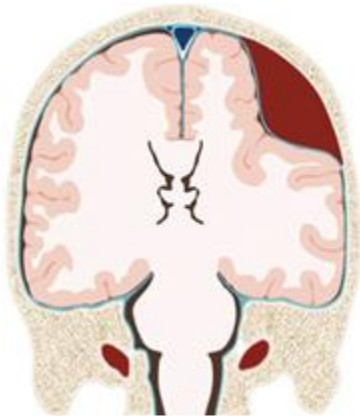
- Intracranial hematomas
- Brain edema / herniation
- Brain congestion (hyperaemia)
- Extracerebral causes: cerebral ischemia / hypoxia

- Tertiary
(complications and sequelae)

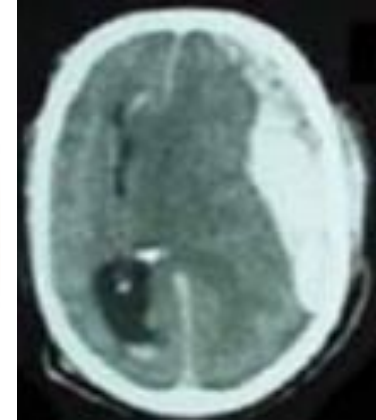
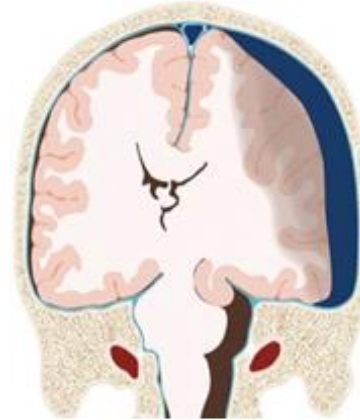
- Hydrocephalus
- Epilepsy
- CSF fistula
- Septic (meningitis, empyema, abscess)
- Post-traumatic encephalopathy



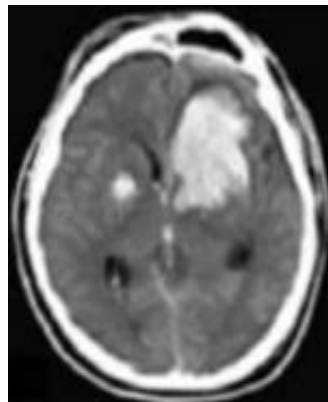
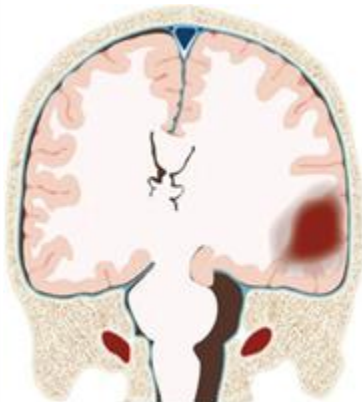
INTRACRANIAL HEMORRHAGE



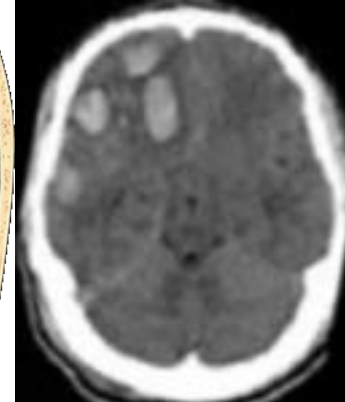
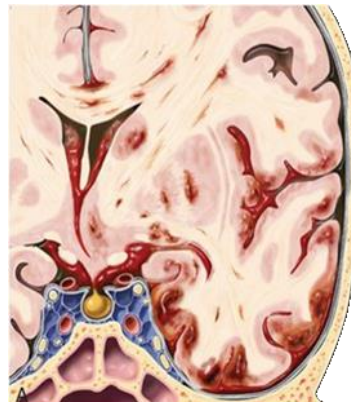
Epidural hematoma



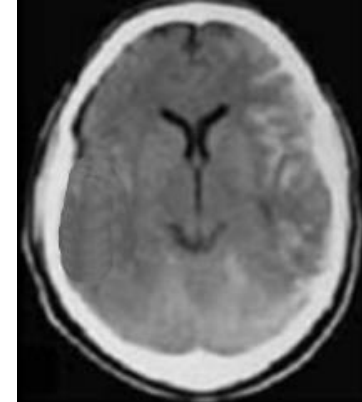
Subdural hematoma



Intracerebral haemorrhage



Brain contusion

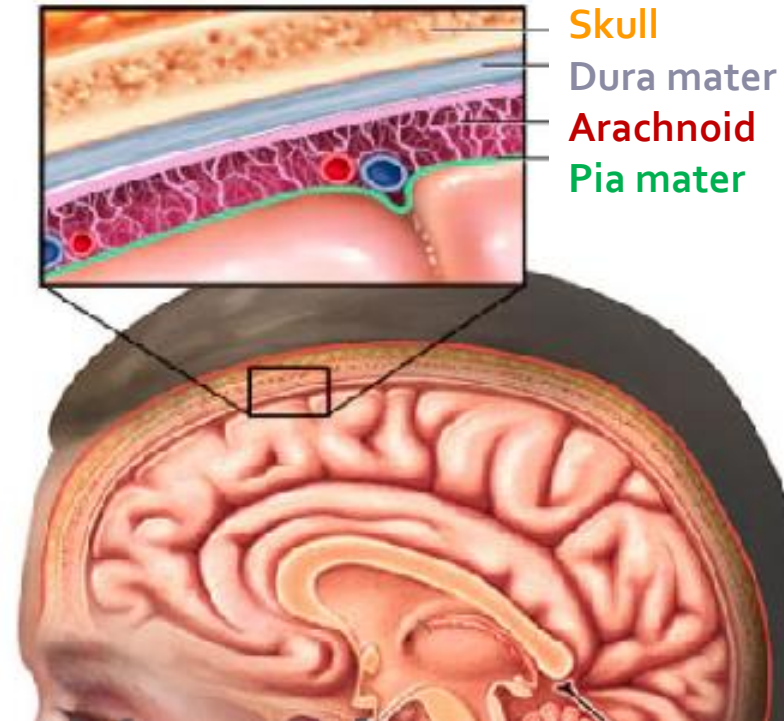


Traumatic SAH



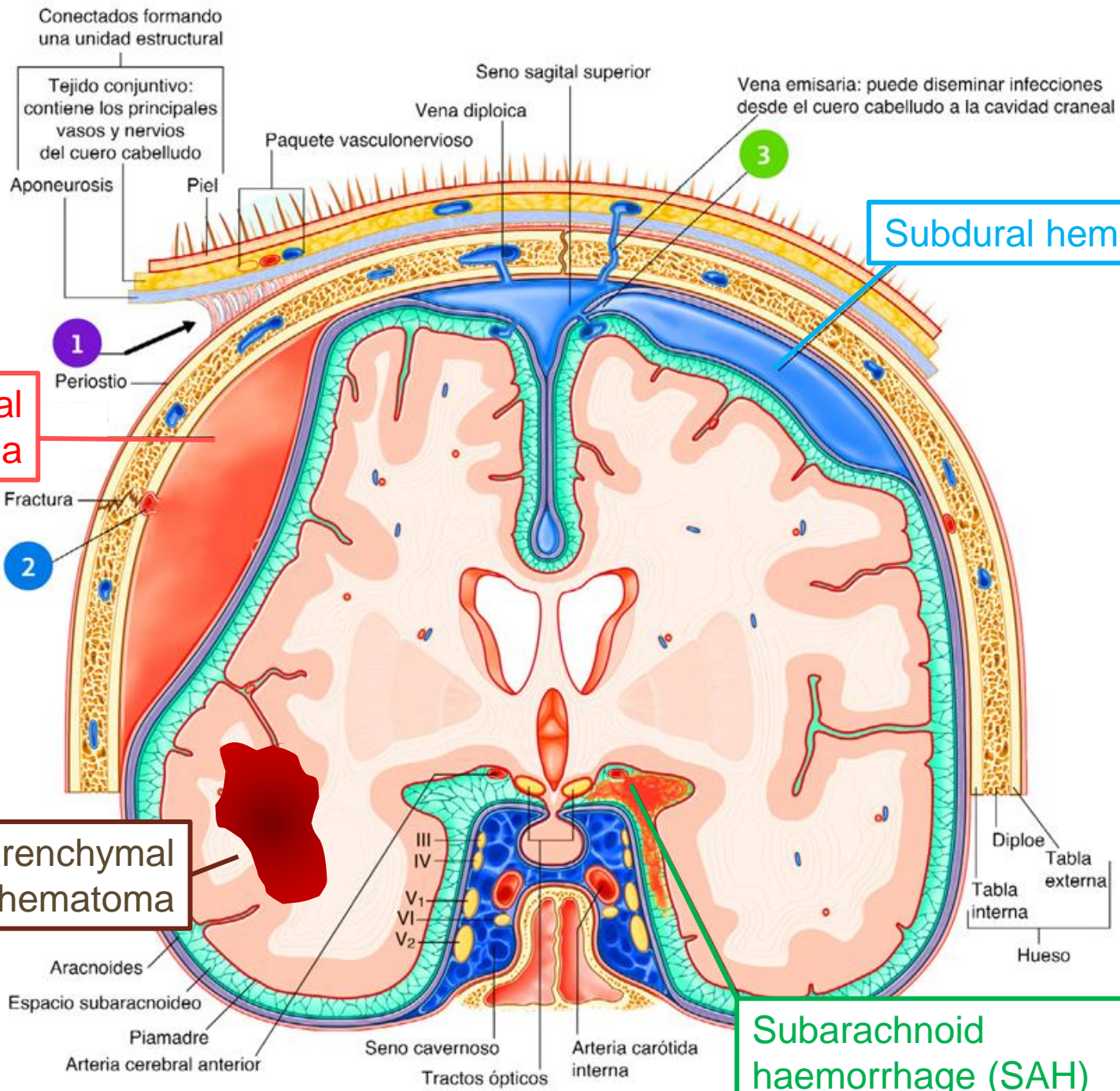
INTRACRANIAL HEMORRHAGE

- Epidural hematoma (2% traumatic brain injury): young people, severe
- Subdural hematoma: very frequent, torpid evolution
 - Acute
 - Subacute
 - Chronic
- Intracerebral hematoma (intraparenchymal): direct contusion, coup, contrecoup
- Subarachnoid haemorrhage: mainly aneurysmal



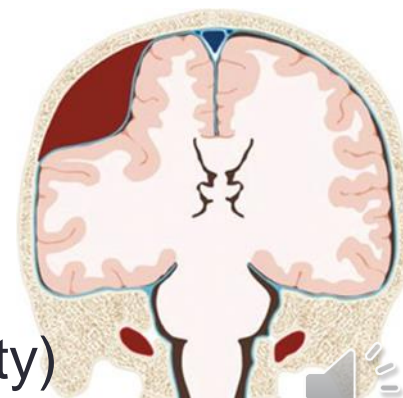
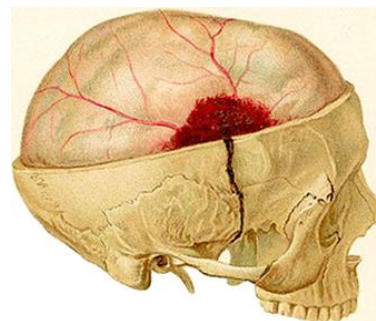
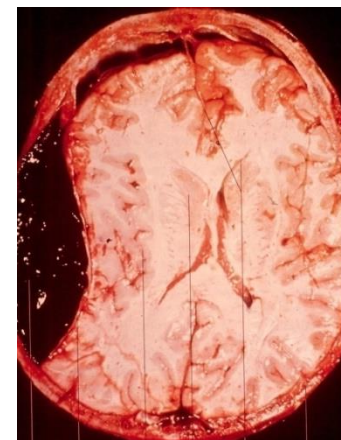
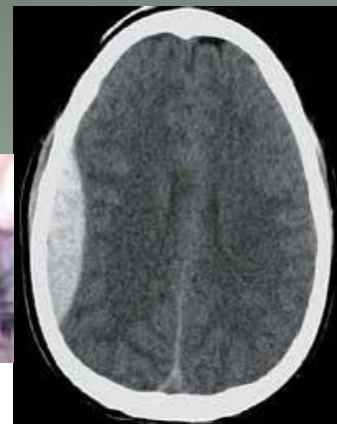
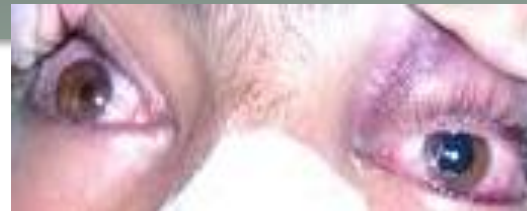
Diagnosis of bleeding = CT





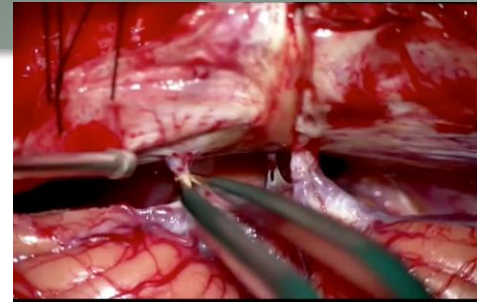
Epidural hematoma

- Between dura mater and skull
 - CT scan: shape of biconvex lens
- Incidence: 1-3 % TBI, young people
- Etiology:
 - 85 % arterial \Rightarrow rupture of middle meningeal artery in temporal squama
 - 15 % venous (tearing venous sinus)
 - *Location: temporal, parietal > frontal > posterior fossa*
- Clinical features: typical in 30 %
 1. Loss of consciousness
 2. Lucid interval
 3. Rapid neurological deterioration
- Treatment = emergency surgery (10-30 % mortality)

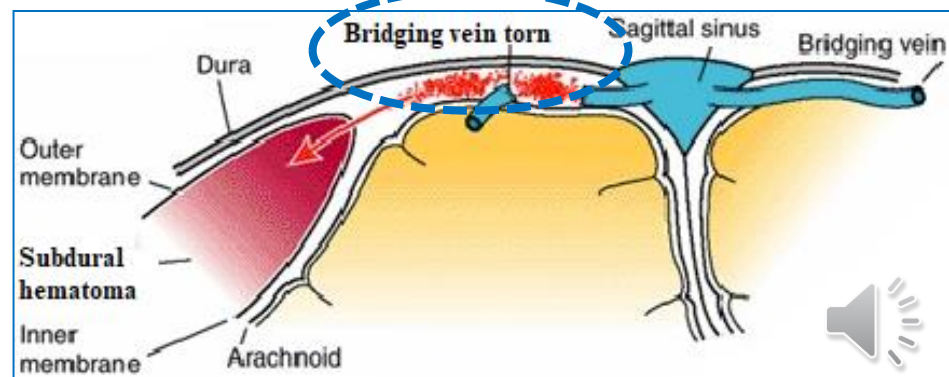
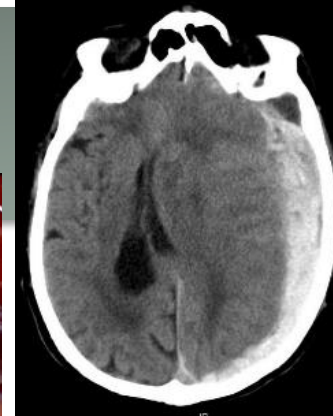
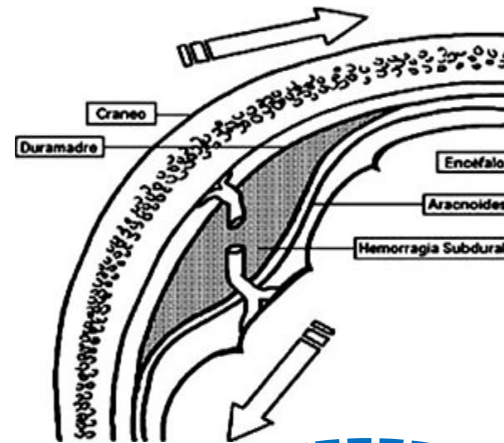


Subdural hematoma

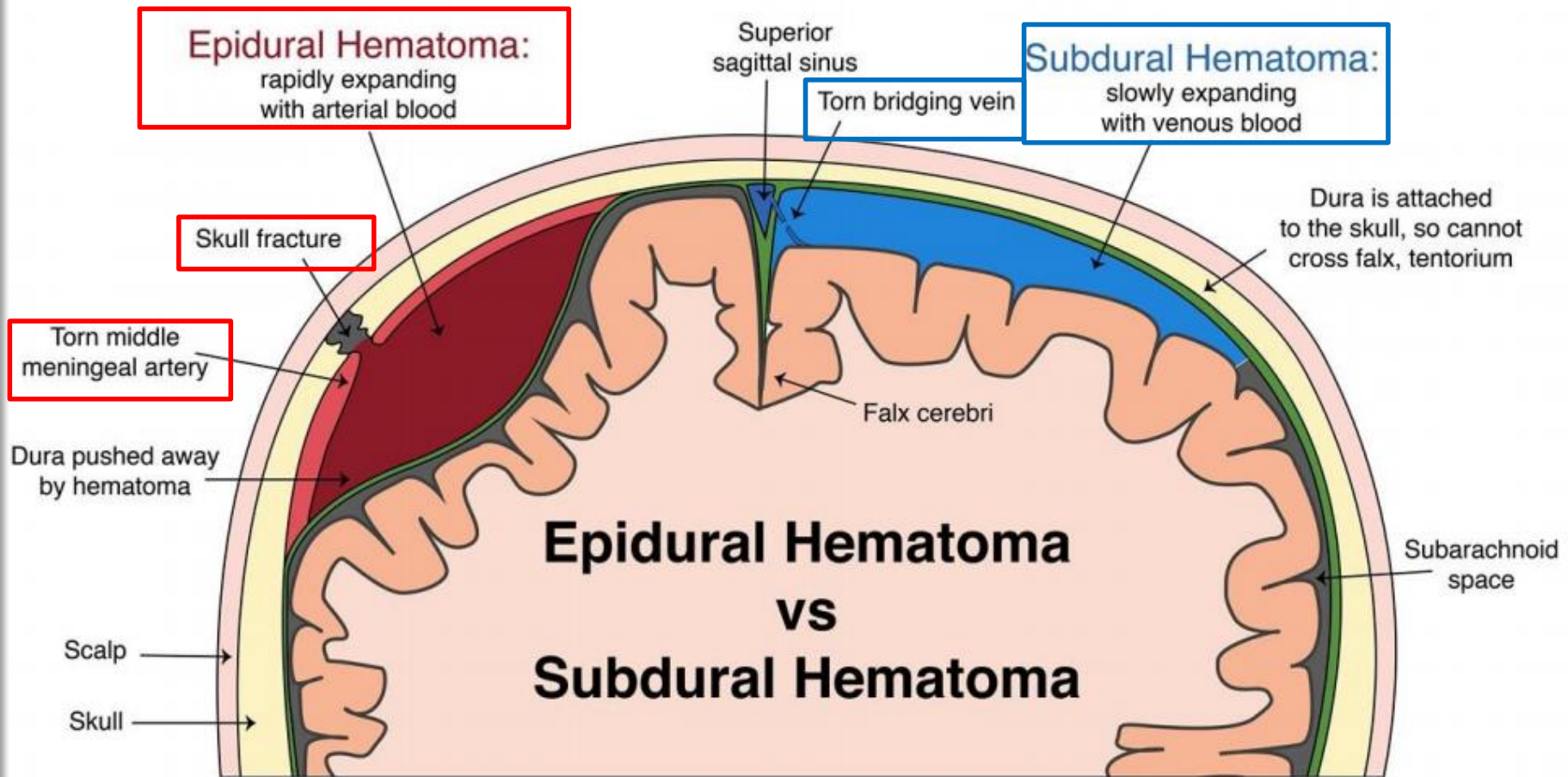
- In subdural space, between cortex and dura mater, usually frontotemporal
 - CT: crescent shape, expands directly on the cortex
 - MRI: different degrees of liquefaction depending on time of evolution
- Etiology:
 - Rupture of cortico-dural vessels (bridging veins >> cortical arteries) > cortical laceration
- Classification (time)
 - Acute 0 – 3 days
 - Subacute 3 days -3 weeks
 - Chronic > 3 weeks



Bridging veins



Epidural *versus* subdural hematoma



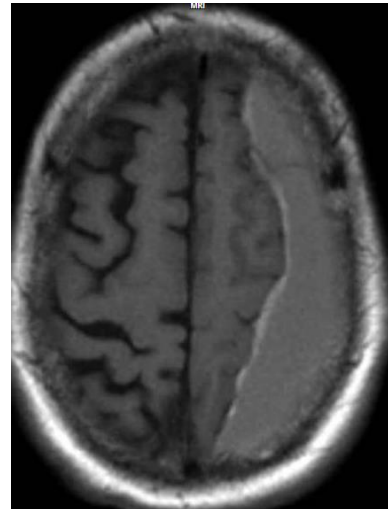
Subdural hematoma (SDH)



Acute SDH



Subacute SDH



Chronic SDH (MRI)

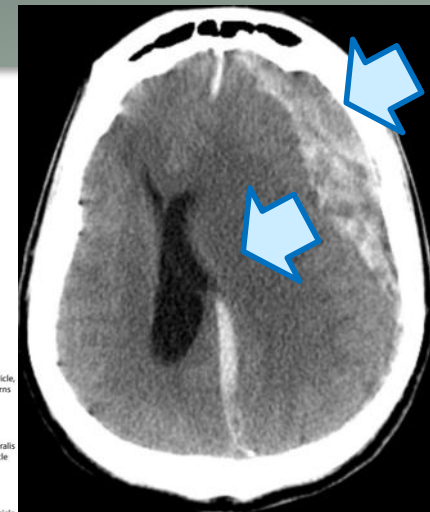
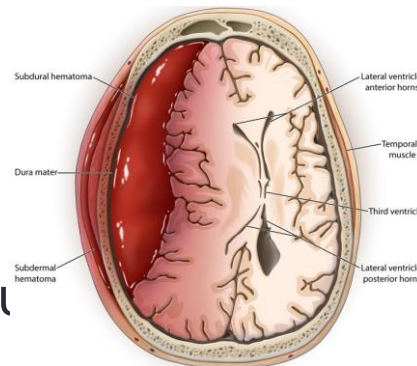


Acute on
chronic SDH

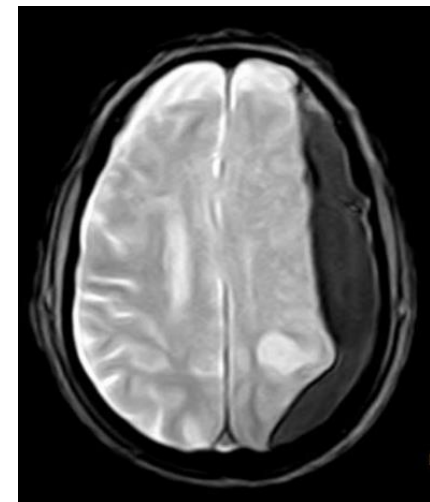


Acute subdural hematoma

- Etiology
 - Requires stronger impact than epidural
 - ⇒ Possible lesion of underlying brain parenchyma
 - ⇒ Worst prognosis
- Clinical presentation
 - Somnolence or coma minutes or hours after traumatic brain injury
 - Possible neurological focal signs
 - *Location, mass effect, transtentorial herniation*
- Treatment: emergency surgery (craniotomy)
 - Mortality 50 – 90 % depending on severity



CT: SDH + brain herniation

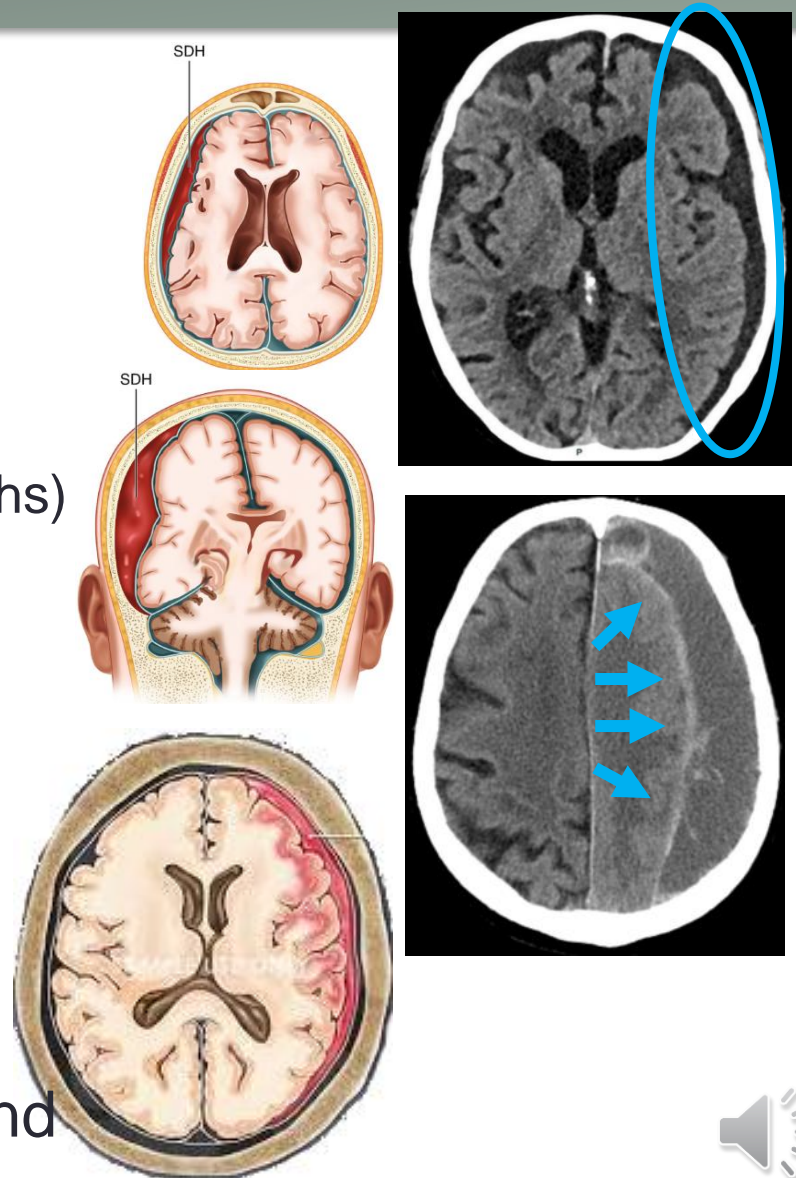


MRI echo gradient: SDH + infarction

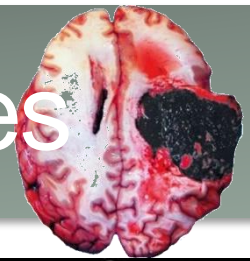


Chronic subdural hematoma

- Etiology
 - Trivial trauma, unnoticed
 - Elderly, diabetic, children, alcoholic, anticoagulated patients...
- Clinical presentation
 - Symptom-free interval (weeks, months)
 - Headache, neurological deficits, cognitive dysfunction
 - *May simulate stroke, tumours, dementia...*
- CT
 - Hypodense lesion, crescent shape, does not respect sutures
- Treatment: surgical (trephine and drainage)



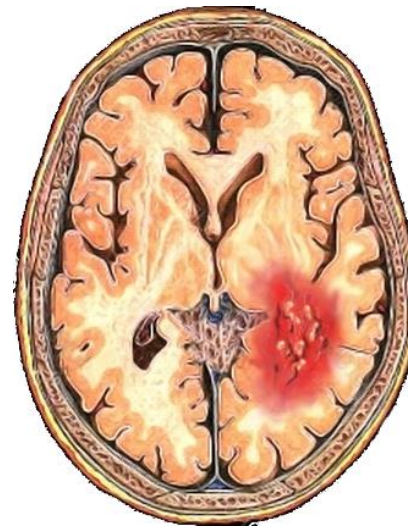
Other intracranial hemorrhages



- **Contusion – intraparenchymal hemorrhage**

- Major trauma \Rightarrow Contusive focus \pm brain laceration
- Focal neurological lesion and /or epilepsy
- General lesion due to \uparrow ICP (hemorrhage + vasogenic oedema – Blood brain barrier rupture-)

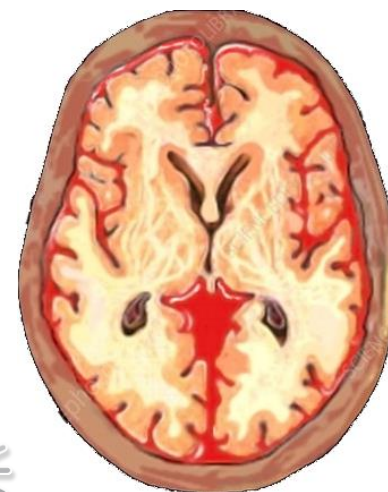
\triangleright *May require treatment*



Intracerebral contusion-hemorrhage

- **Traumatic subarachnoid hemorrhage (SAH)**

- Frequent
- Accompany other traumatic brain injury lesions
- \triangleright *Little importance, although may hinder clinical course*
- Does not require specific treatment



Traumatic subarachnoid hemorrhage



SUMMARY KEY CONCEPTS TOPIC 3

- **Traumatic brain injury**
 - The most important: BRAIN LESION
 - Types according to dura mater: closed *versus* open
- **Lesions**
 - Primary (immediate) > secondary (minutes-hours) > tertiary (days)
- **Evaluation in ER**
 - Facts and circumstances + rapid exam
 - Rule out other lesions
 - Staging Glasgow coma scale and GCS-P
- **Intracranial hematomas**
 - Secondary lesion that requires early diagnosis (CT) and treatment
 - Differential diagnosis epidural *versus* subdural hematoma



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