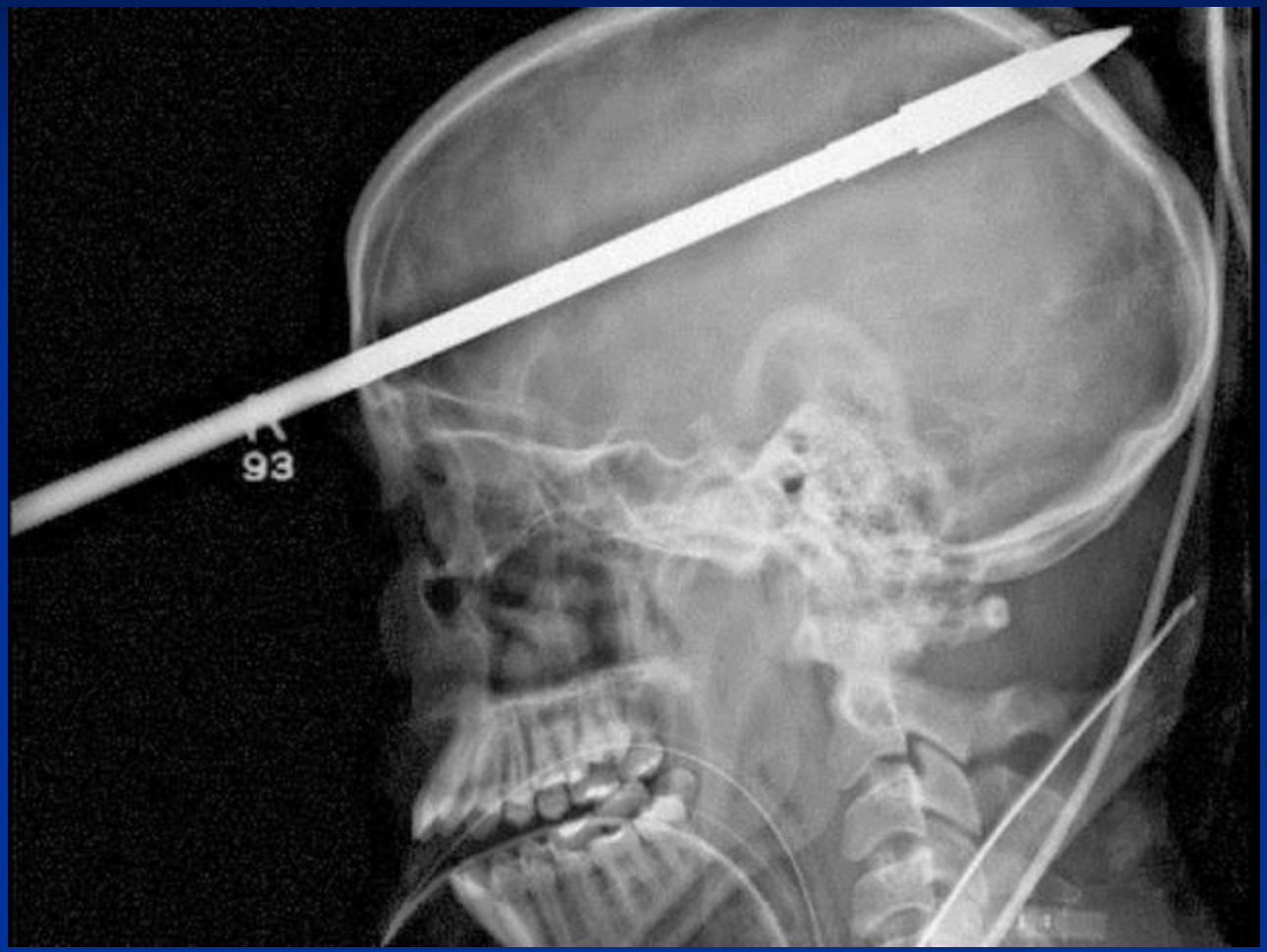
Neurotraumatology



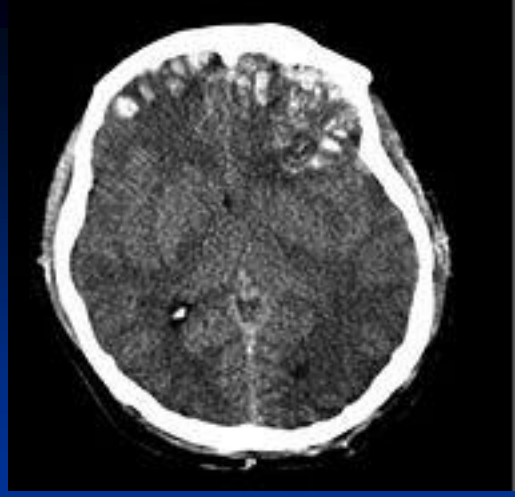
National Institute of Clinical Neuroscience, Budapest

Causes of traumatic brain injury (TBI)

- Auto, motorcycle or bicycle crashes
- Falls
- Violence: gun shots, abuse
- Explosive blasts

- Clssification: Scalp injury, skull injury, brain injury

Traumatic brain injury



- **Primary**: direct force, skull fracture, contusion, haematomas, laceration, nerve damage
- **Secondary(consequence)**: brain swelling (oedema), increased pressure inside of the skull (high intracranial pressure), epilepsy, haematomas
- The aim: management of secondary brain injury to decrease of mortality

(the primary has already occured)

Traumatic brain injury

- Healthy , young patients,
  The major cause of death and disability worldwide under 45 y
- Serious consequences (personal and social)
- Importance of primary injury supply

Statistics on TBI

14 000/year patients hospitalized in Hungary cause of TBI

Symptoms
-71,3% mild
-19,4% moderate
-9,4% severe

55% of patients die with severe TBI

40% of survivors have lasting damage or vegetative state.
60%-of survivors have mild symptoms or asymptomatic.

Symptoms of TBI

- Light-headedness, dizziness
- Feeling tired, with no motivation
- Urge to vomit
- Changes in sleep patterns (more sleeping)
- Mood changes (sad or angry for no reason)
- Increased sensitivity to lights, sounds,
- Loss of sense of smell or taste, ringing in the ear

Prehospital treatment for TBI

- Checking of vital parameters
- **Oxygenation and blood pressure**

Correct CPP (brain perfusion). management of secondary brain injury to decrease of mortality

- Inspektion – cranium
- Palpation – cranium
- Neurological examination
- „Glasgow Coma Scale” determining
- Known of anamnesis
- Polytrauma (80% the probability of head injury)!

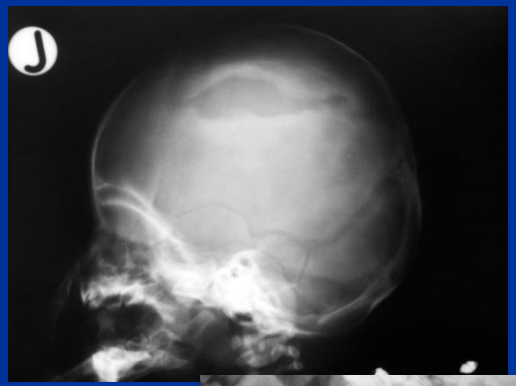
- **Always need to exclusion the injury of cervical spine**

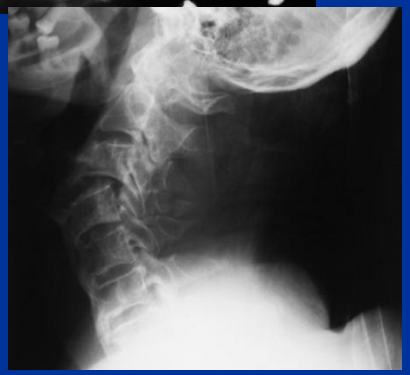
Glasgow Coma Scale (GCS)

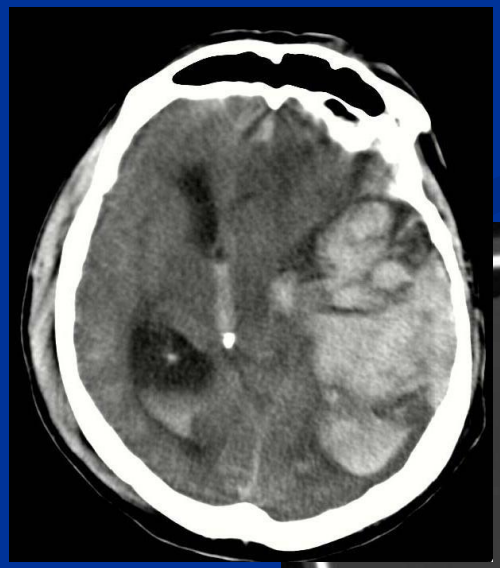
Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		

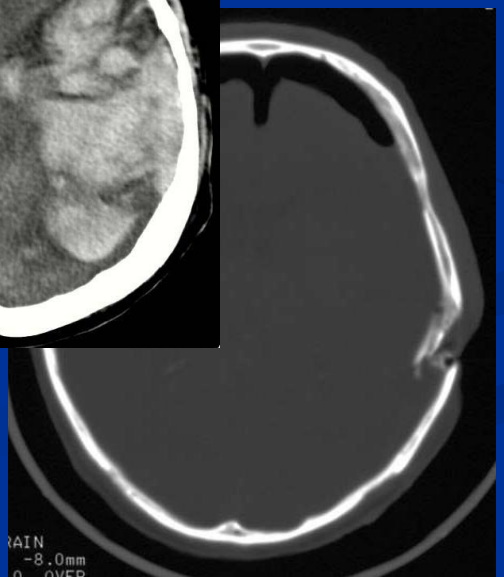
Radiological methods

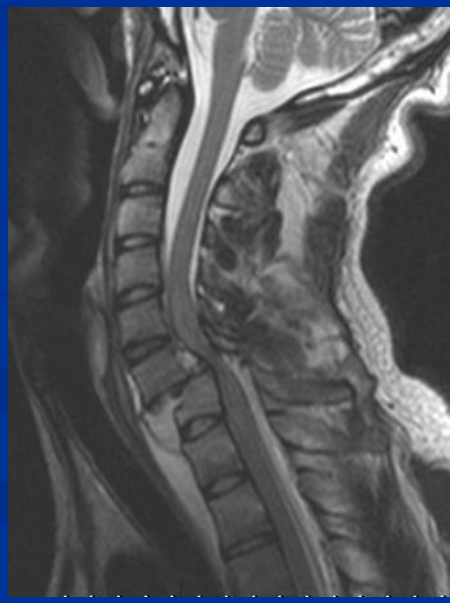
X-ray
CT
MRI (rare)



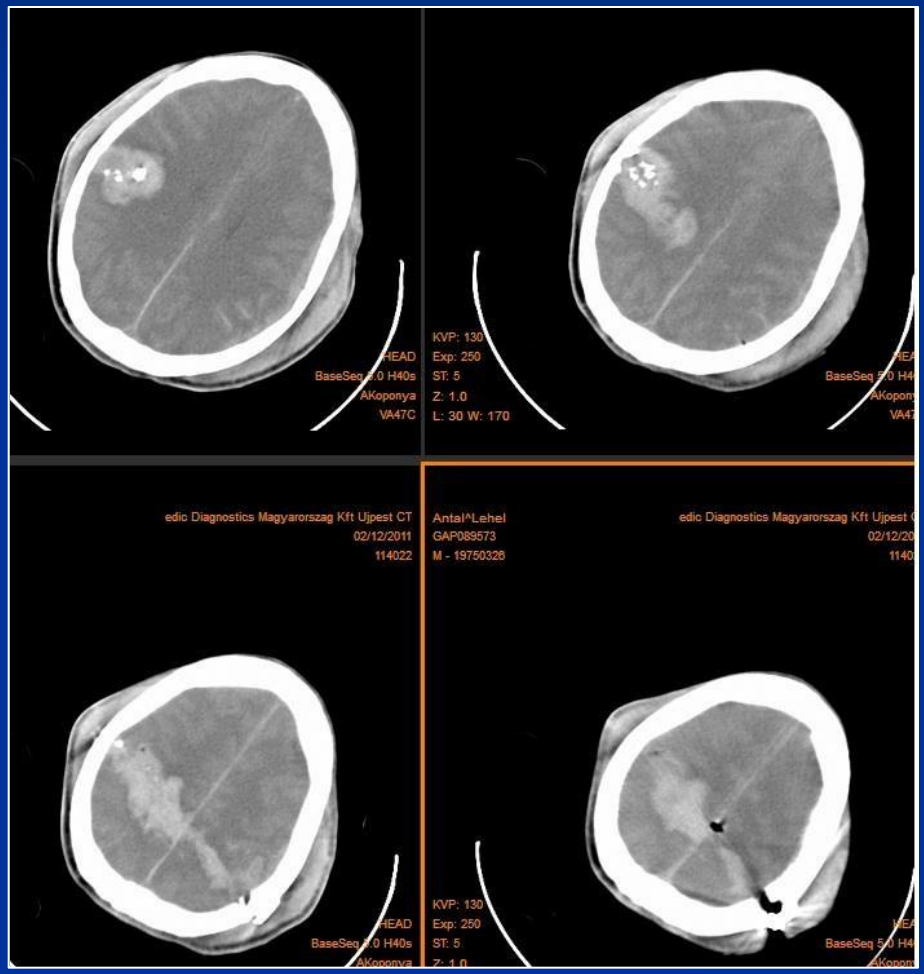


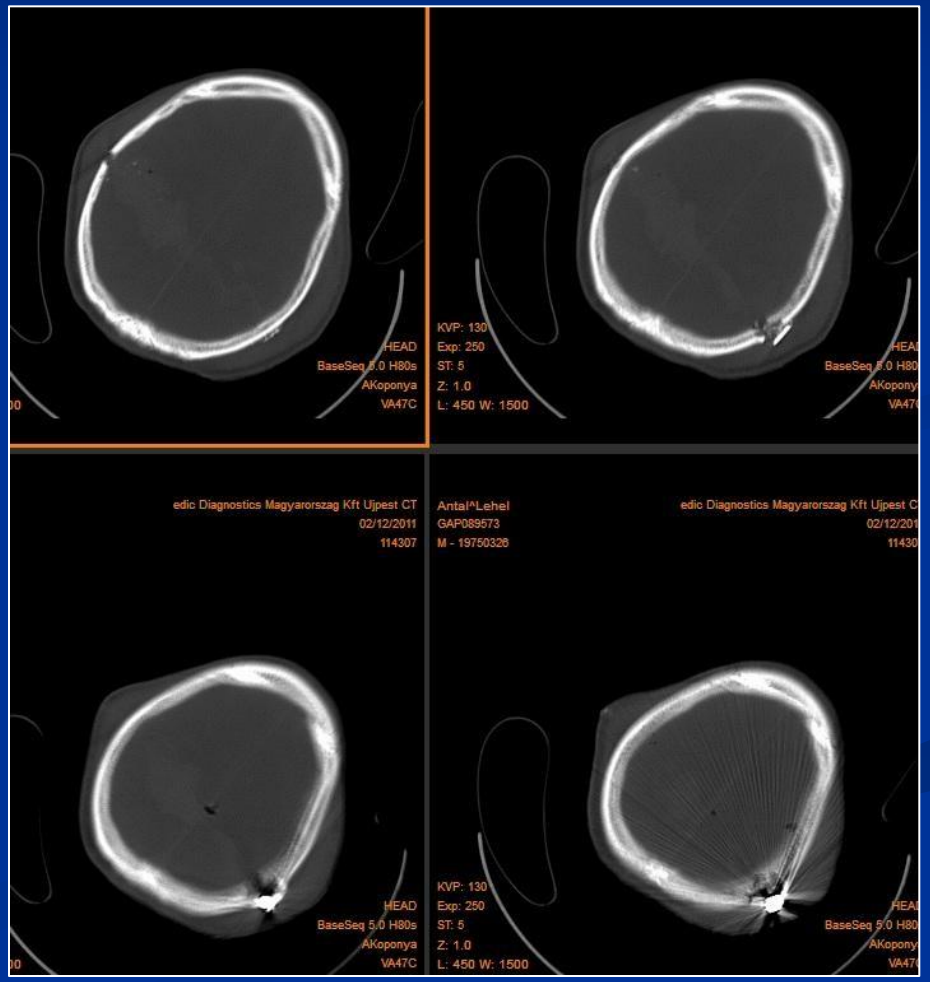




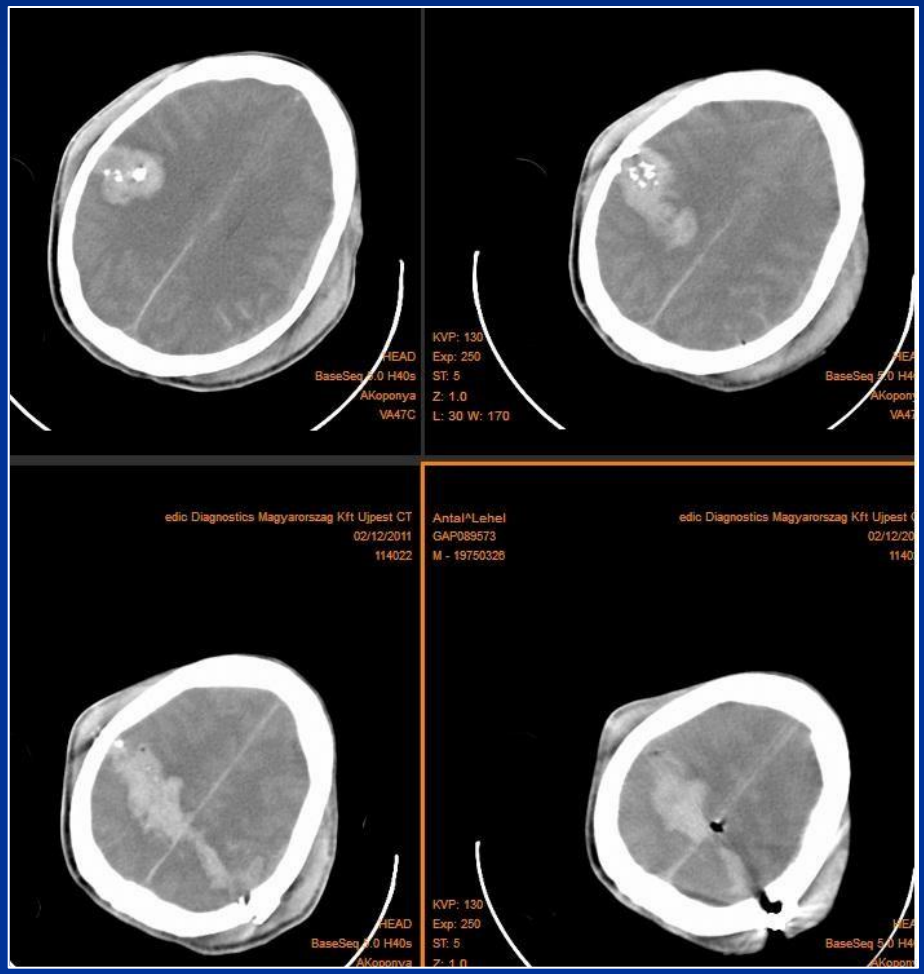


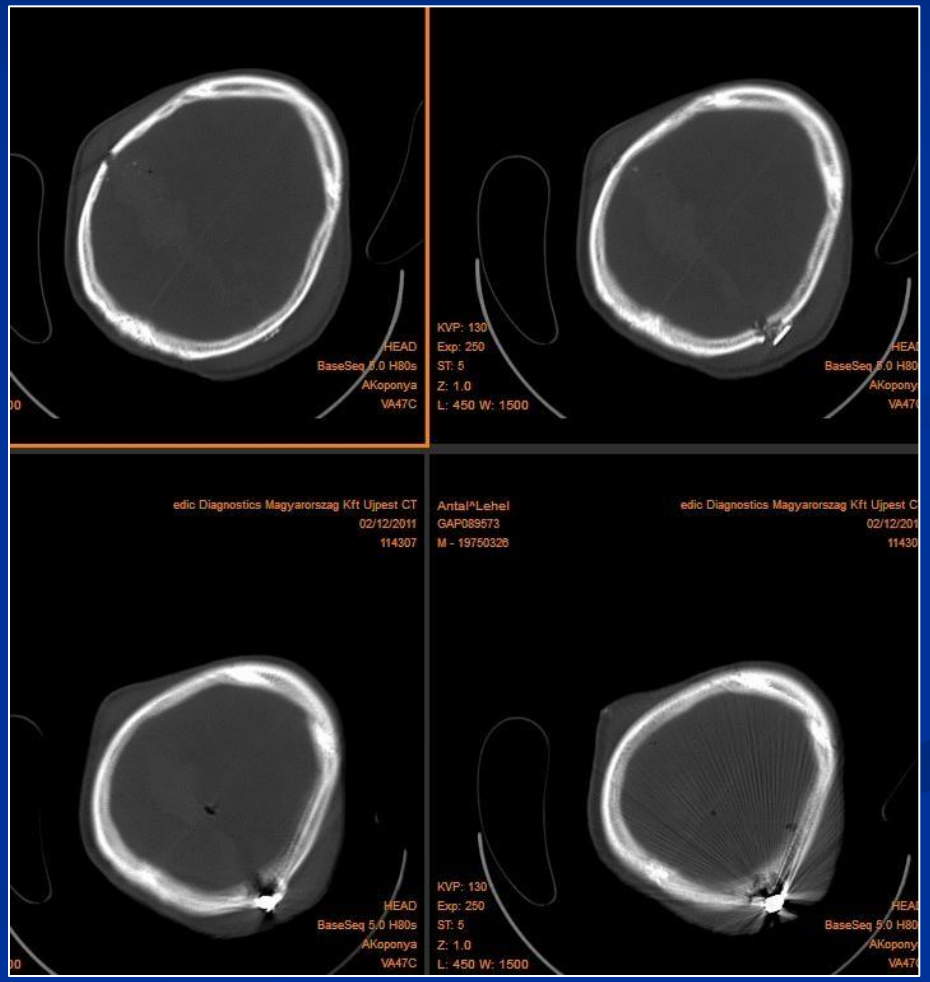
CT examination





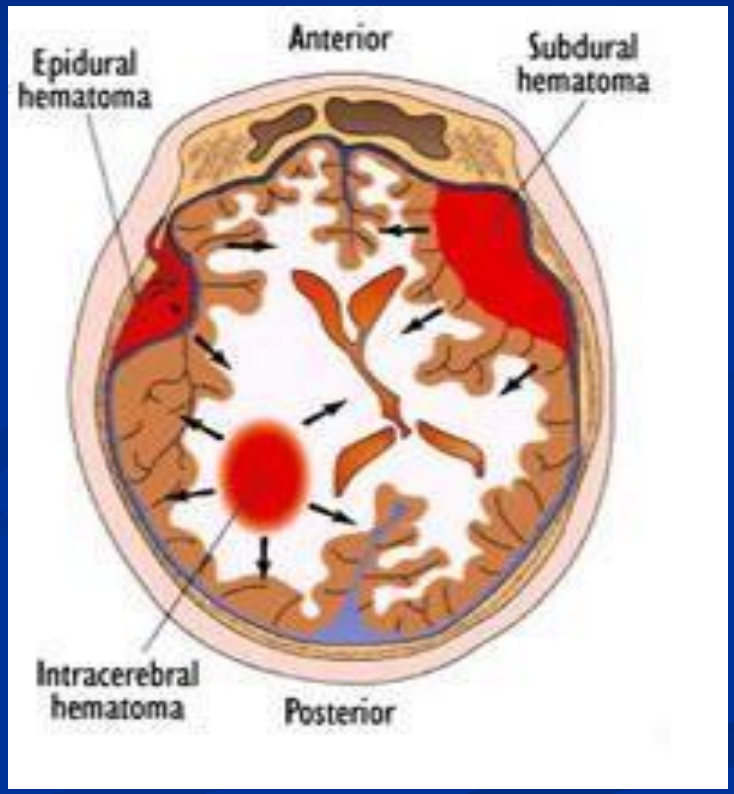
The bullet comes from the.?..side





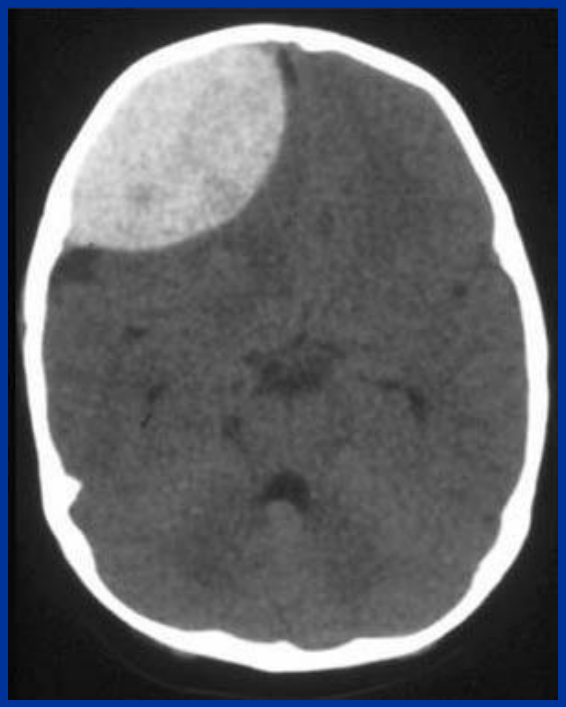
Consequences of TBI

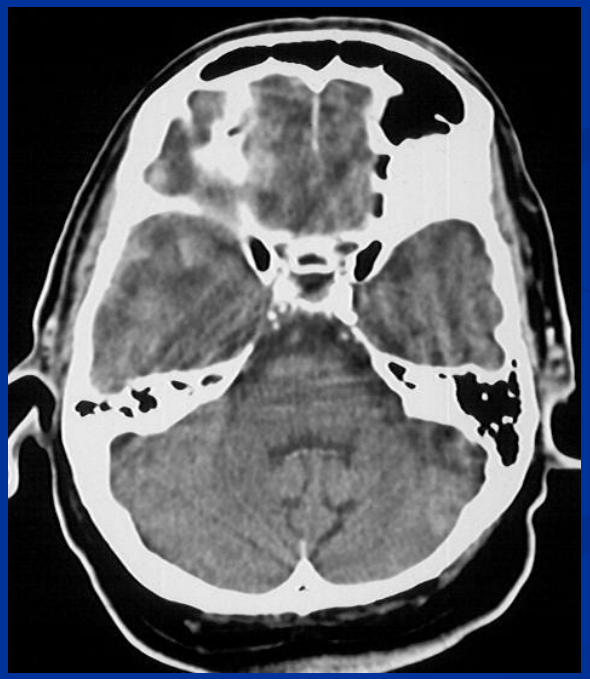
Brain oedema
Fracture of skull
Epidural haematoma
Subdural haematoma
Intracerebral haematoma
Subarachnoideal bleeding

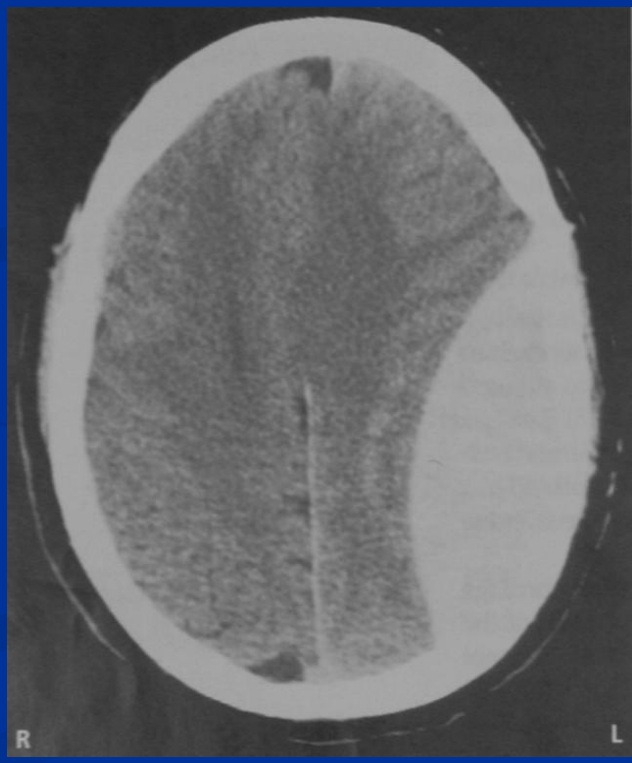


TBI

- Focal damage (the area of the force)



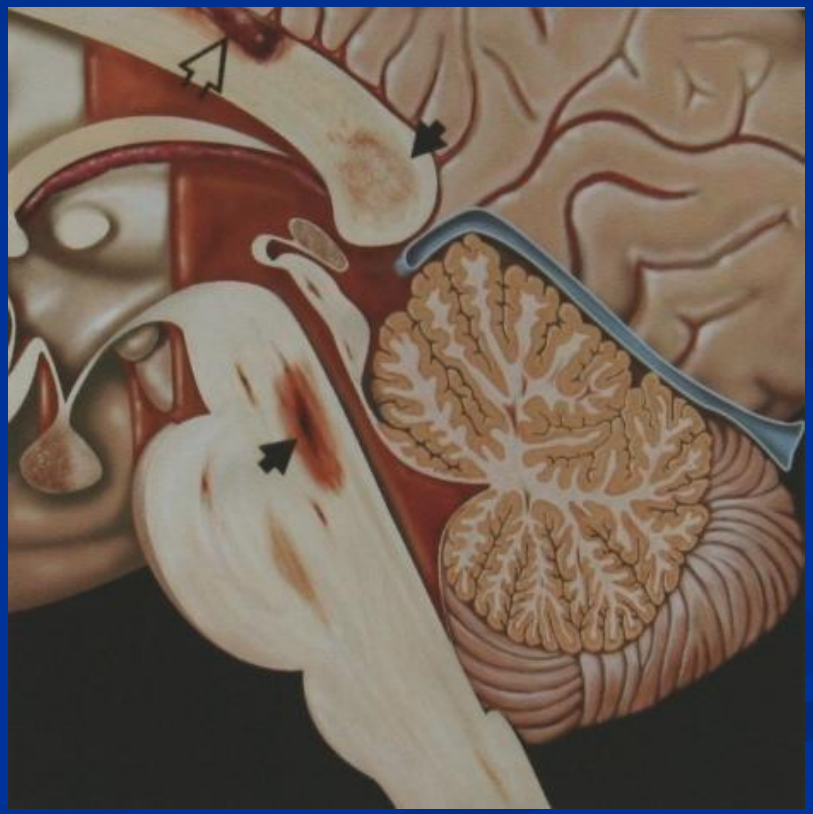




TBI

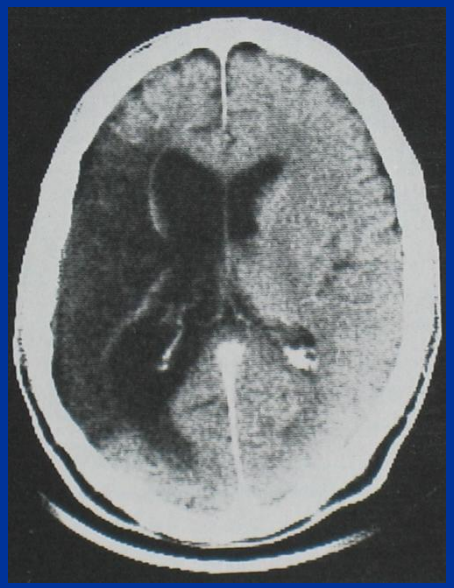
- Diffuse axonal injuries
  - Damage to the pathways (axons) that connect the different areas of the brain. This occurs when there is twisting and turning of the brain tissue secondary to unrestricted head movement at the time of blast.
  - Affects white matter of the cerebrum, corpus callosum, deep grey matter, internal capsule, upper brainstem and the cortico-medullary junctions of cerebral cortex
  - Damage to rats axonal cytoskeleton results on loss of their elasticity and impaired transport and accumulation of axonal transport proteins within axonal swellings.
  - Axonal injuries cause consciousness.

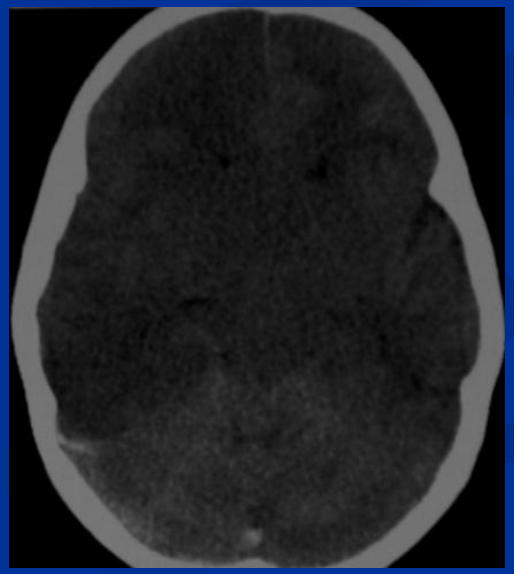
Diffuse axonal injuries



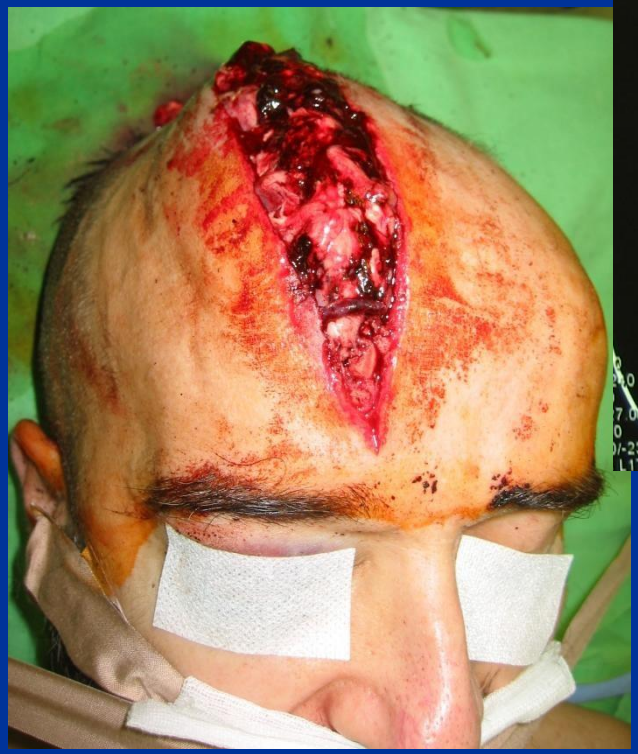
Closed skull-brain injury

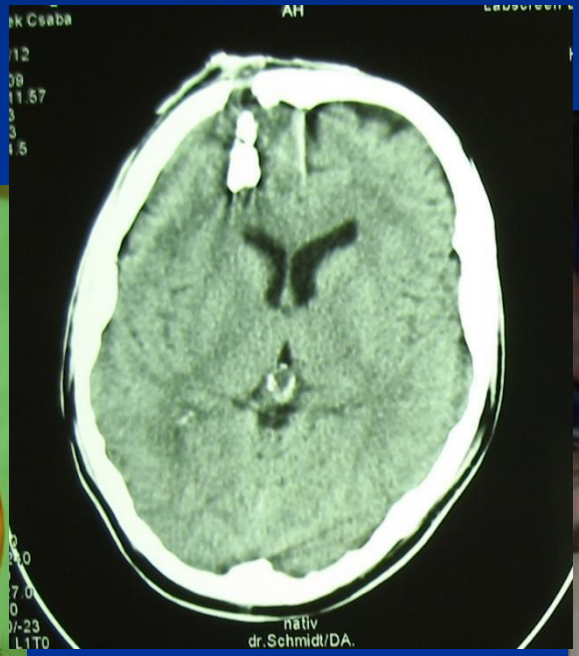
Hypoxia, ischaemia (penumbra)
Diffuse microvascular damage (damage of autoregulation , CO_2 reactivity) - oedema

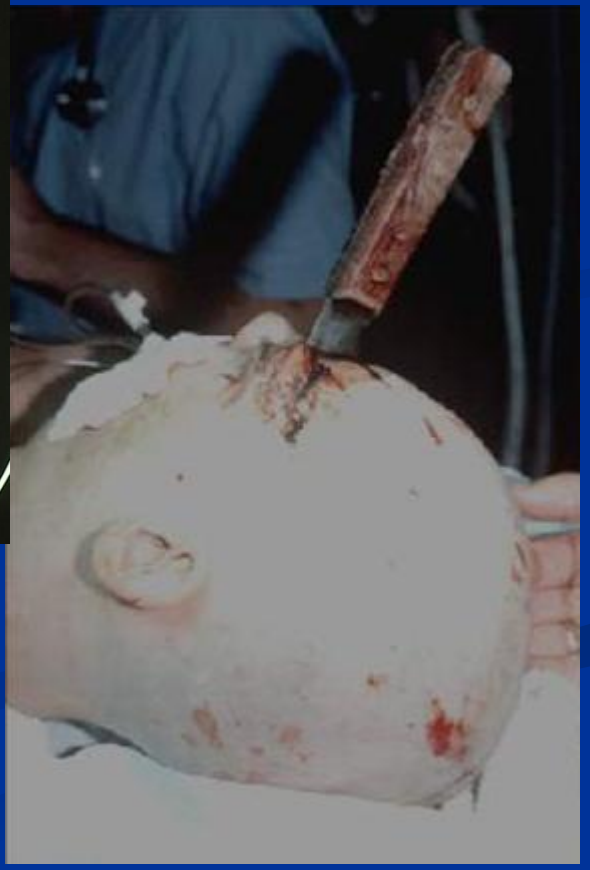


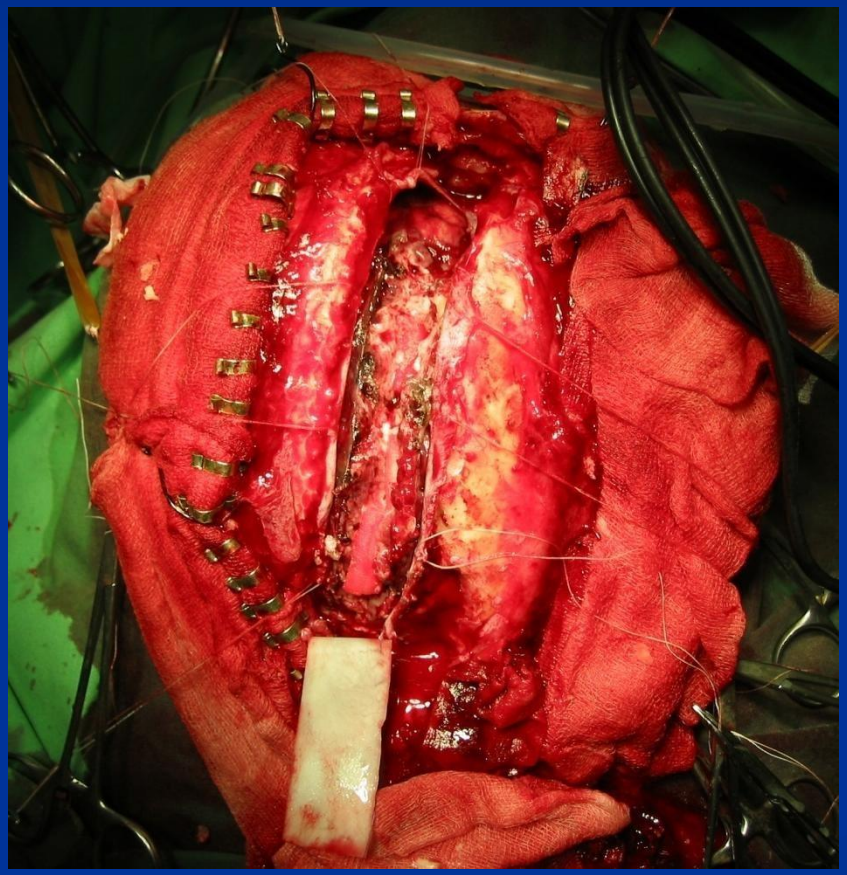


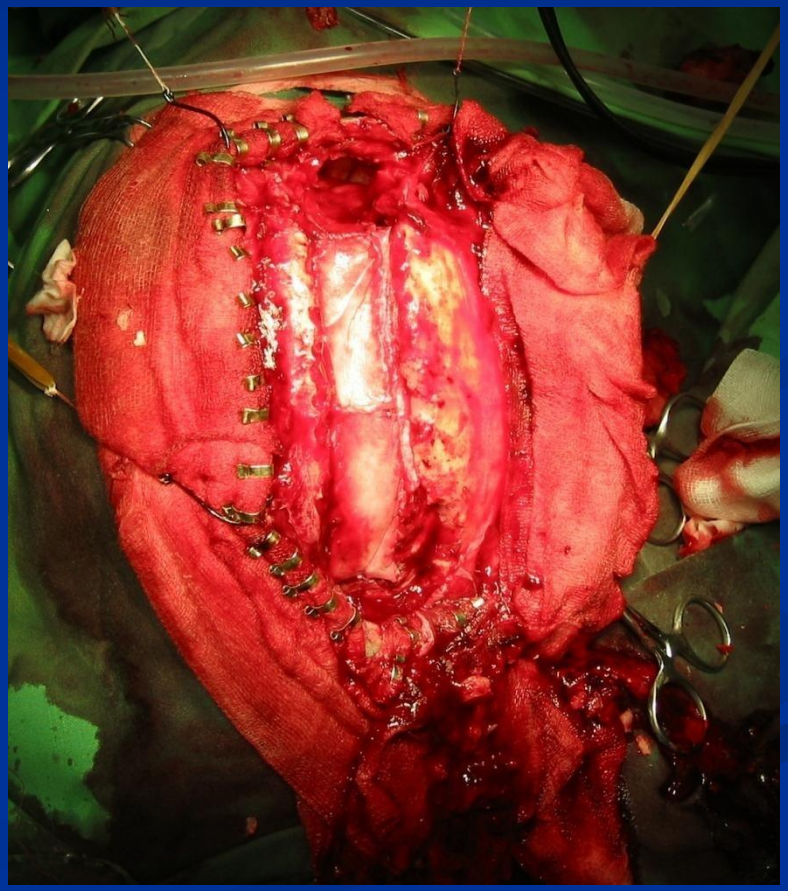
Open skull-brain injury (foreign body)



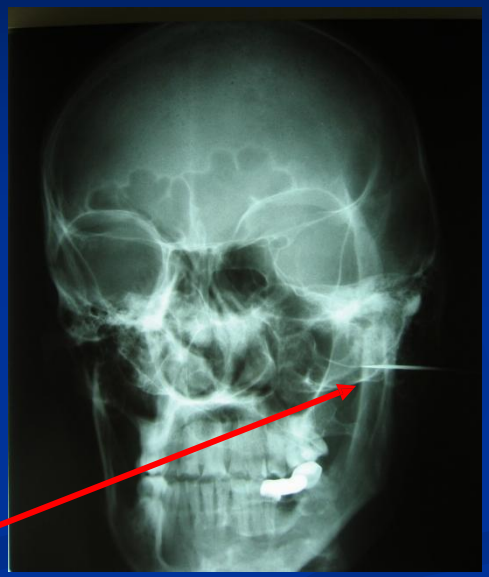


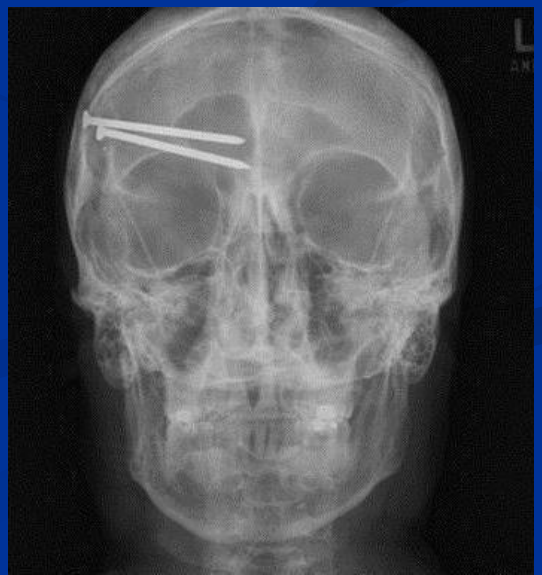


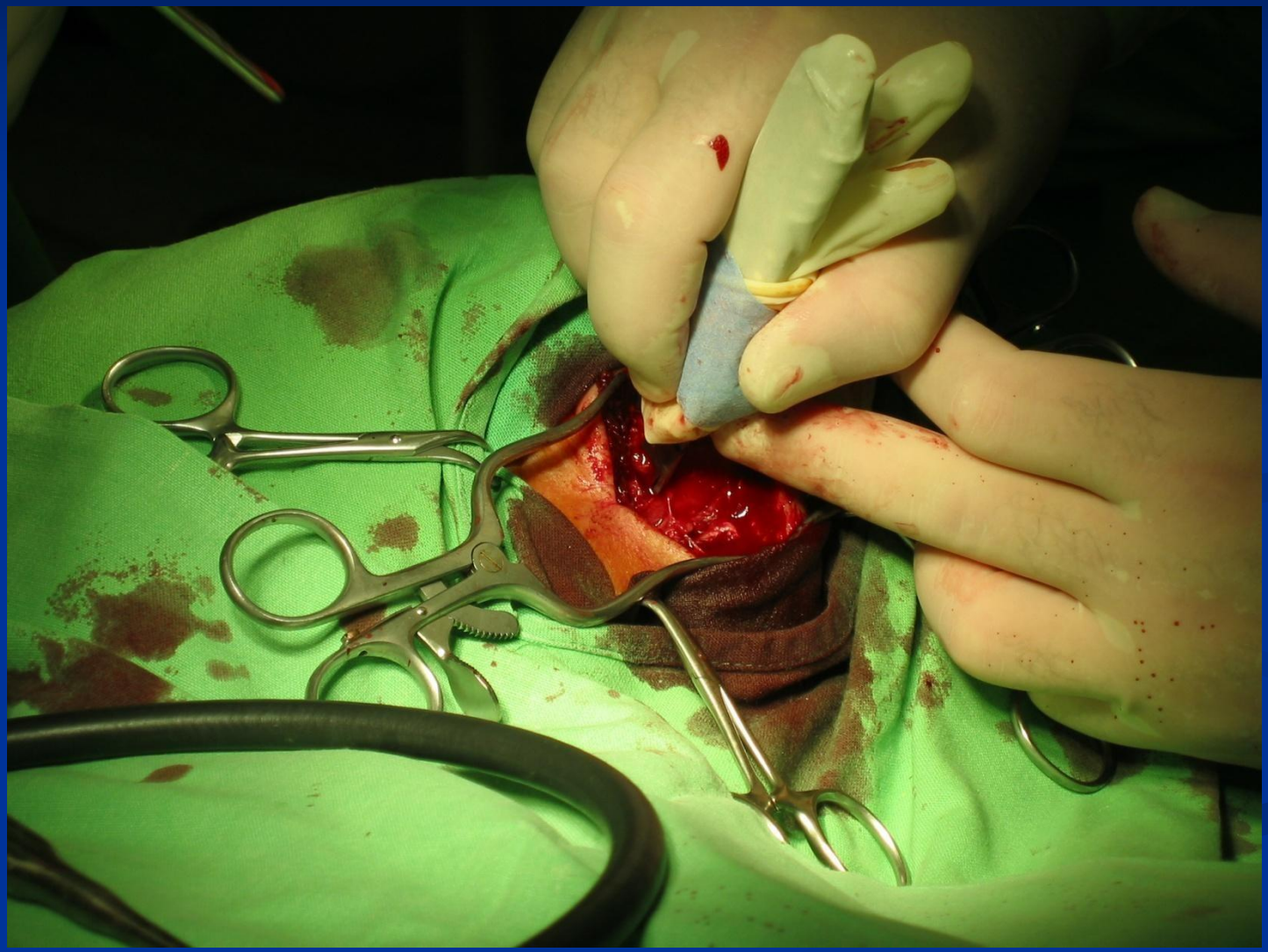




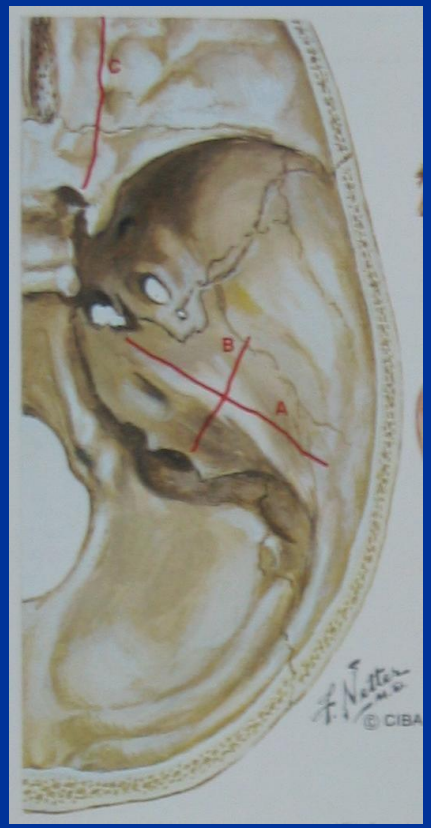
The seriousness of injury:

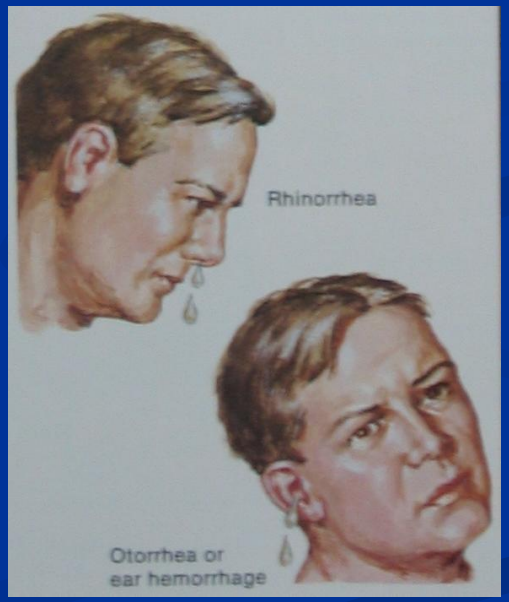






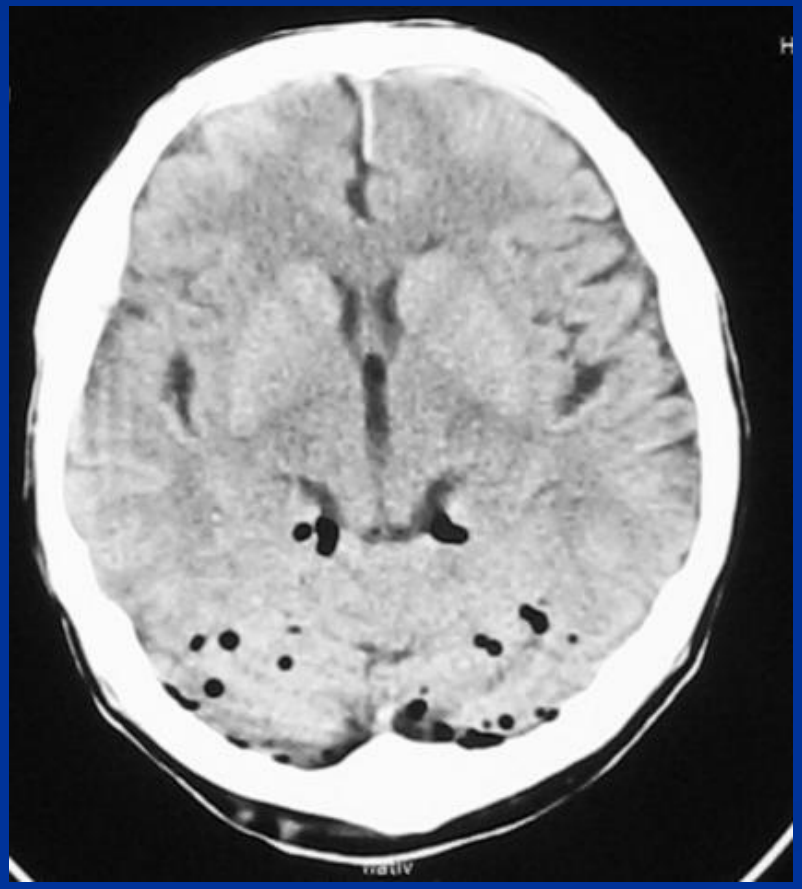
- Skull base fractures (frontale, pyramis)

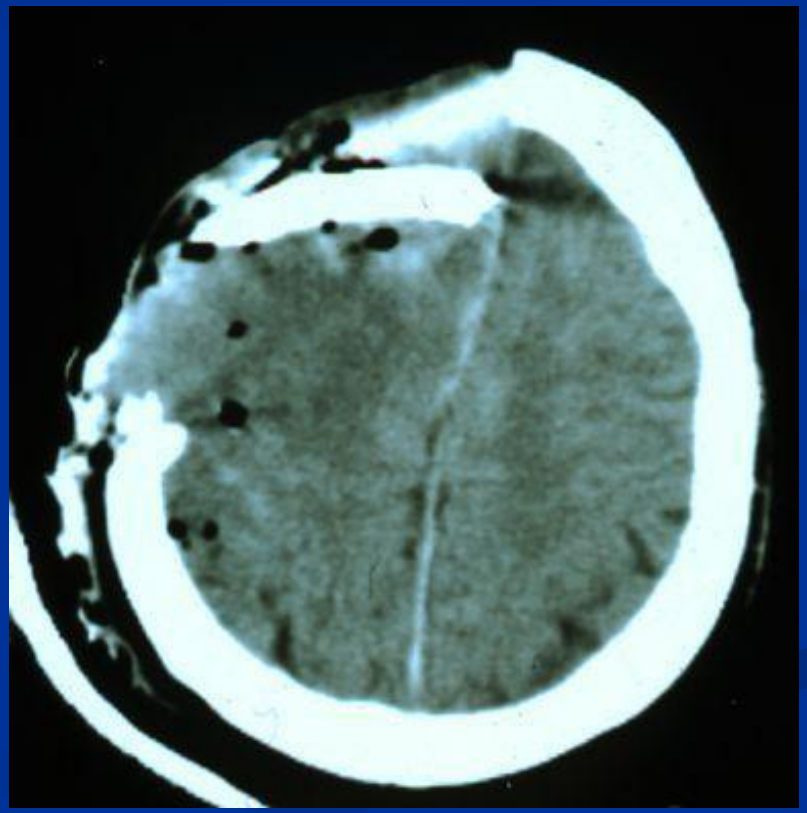




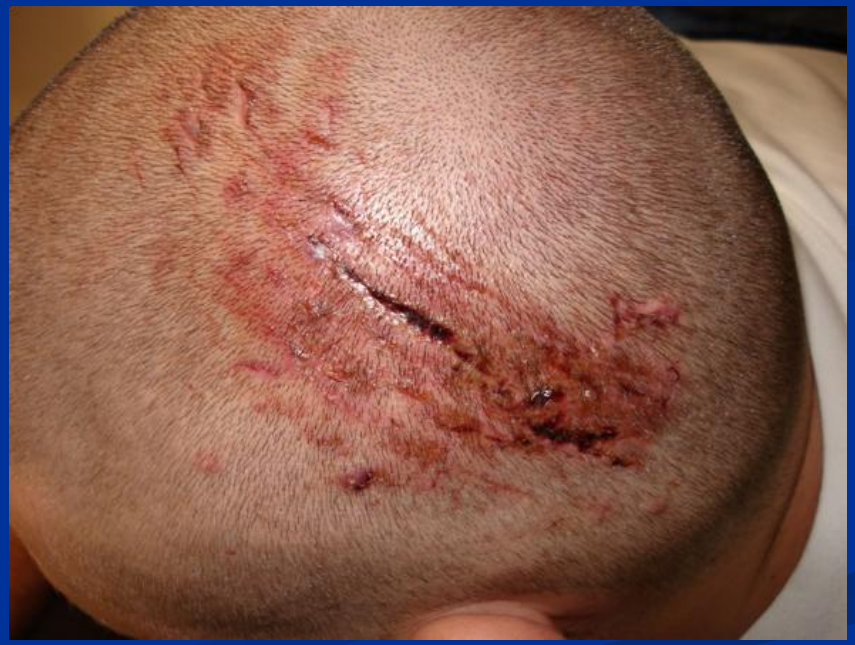
Open skull-brain injury

Pneumocephalus: intracranial air





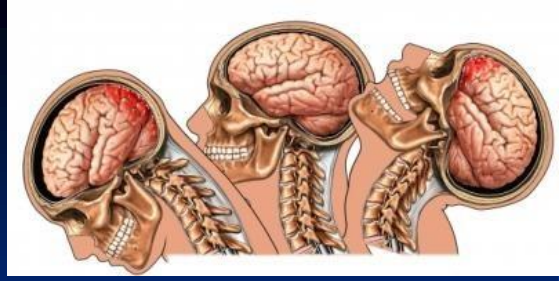
Head injury



Commotio cerebri

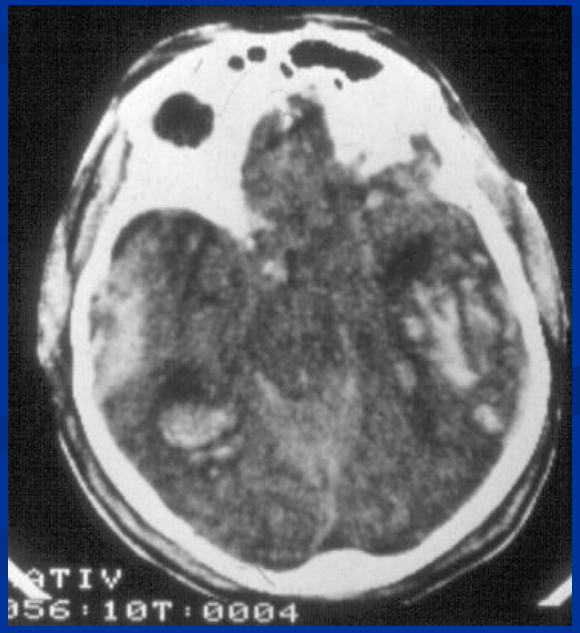
- Lost of consciousness (2-10 min)
- Apnoe (6-12 sec)
- Bradycardia (1-2 min)
- Amnesia: congrad,retrograd, anterograd
- CT negative
- Treatment: rest in bed, observatio, dehydration?

Contusio cerebri



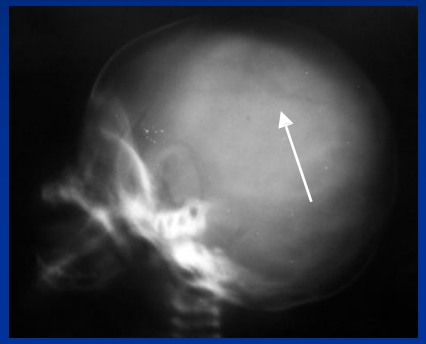
- Lost of consciousness
- Other neur. deficit
- Damage of tissue of the brain haematoma, or oedema

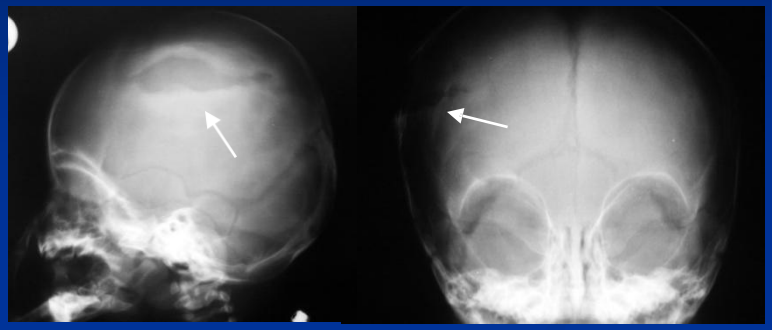
- Treatment: oedema ↓ if GCS <8 measure of ICP clin. and radiol. observation haematoma evacuatio?

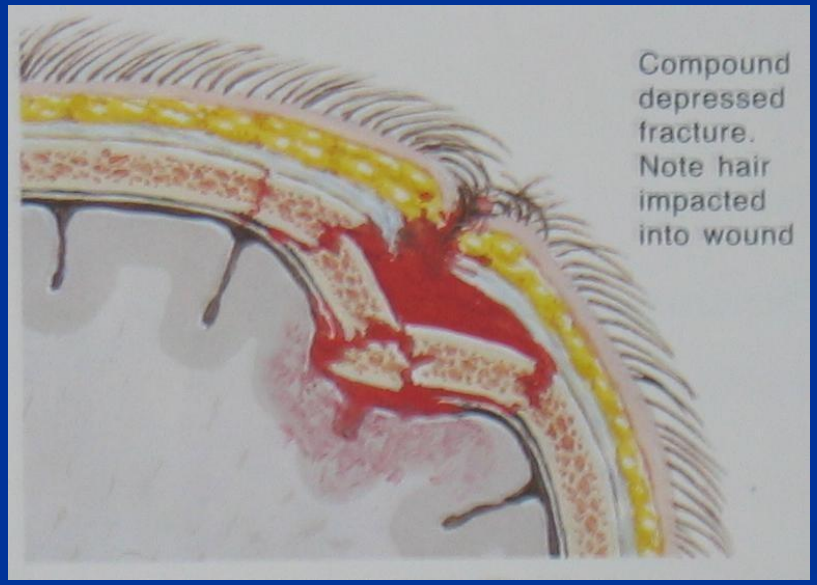


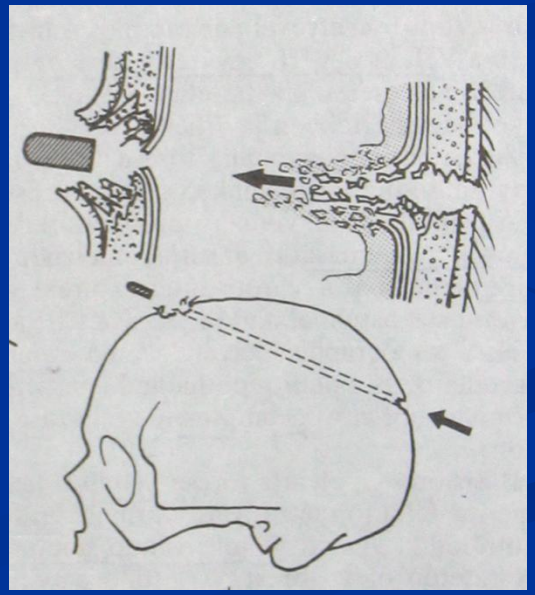
Skull bone fracture

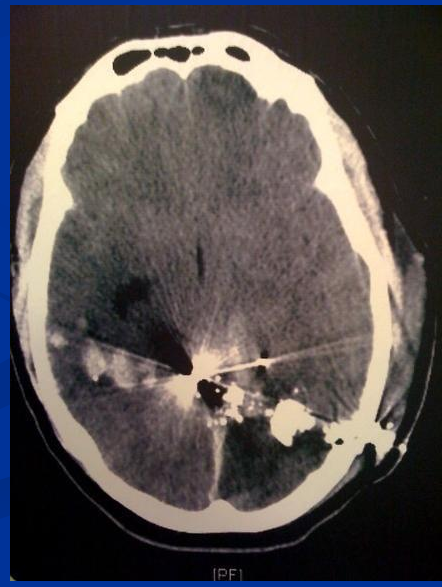
Linear
Impressive
Perforating



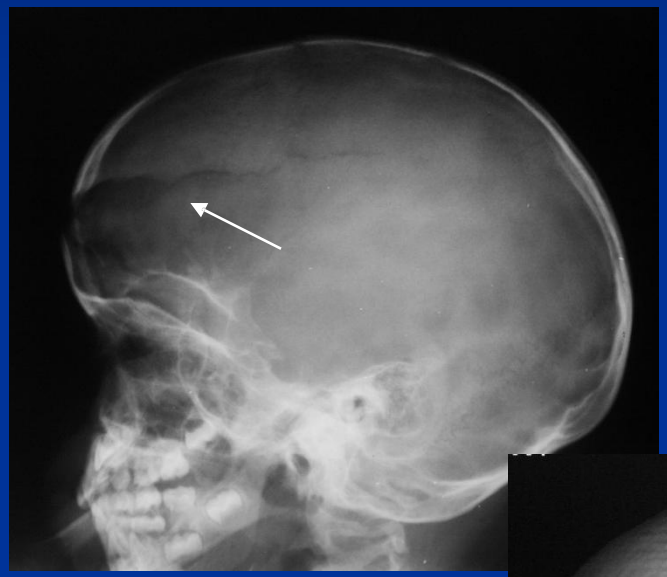


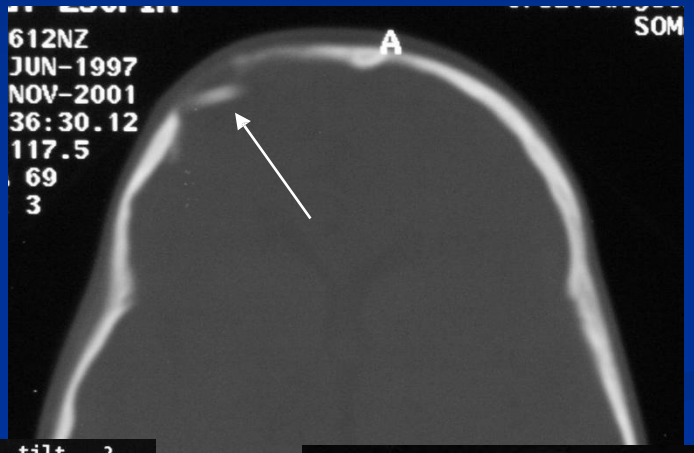


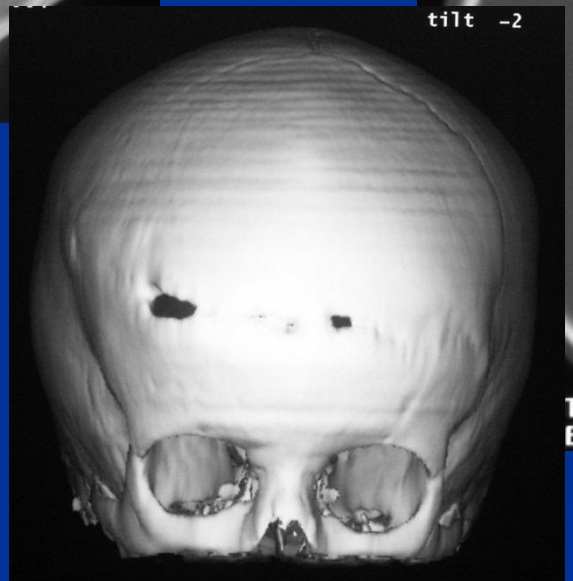


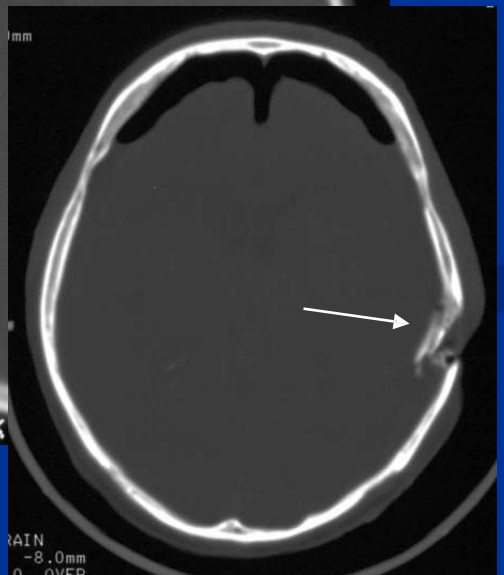


Skull bone fracture

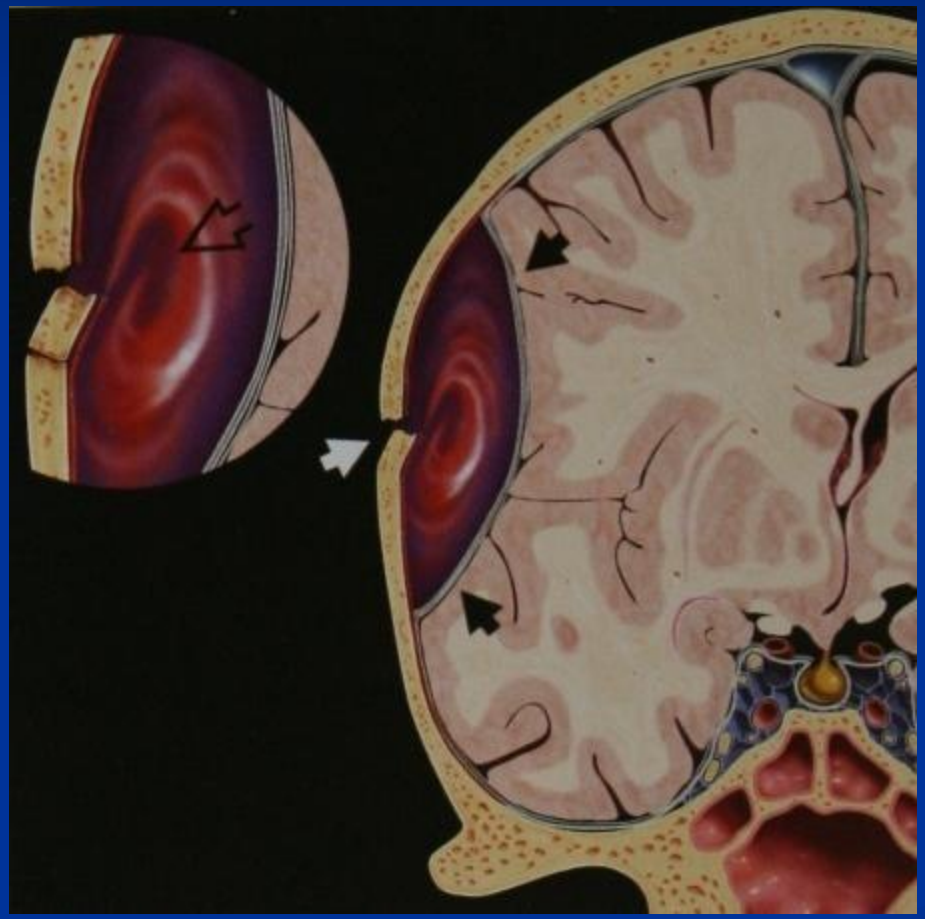


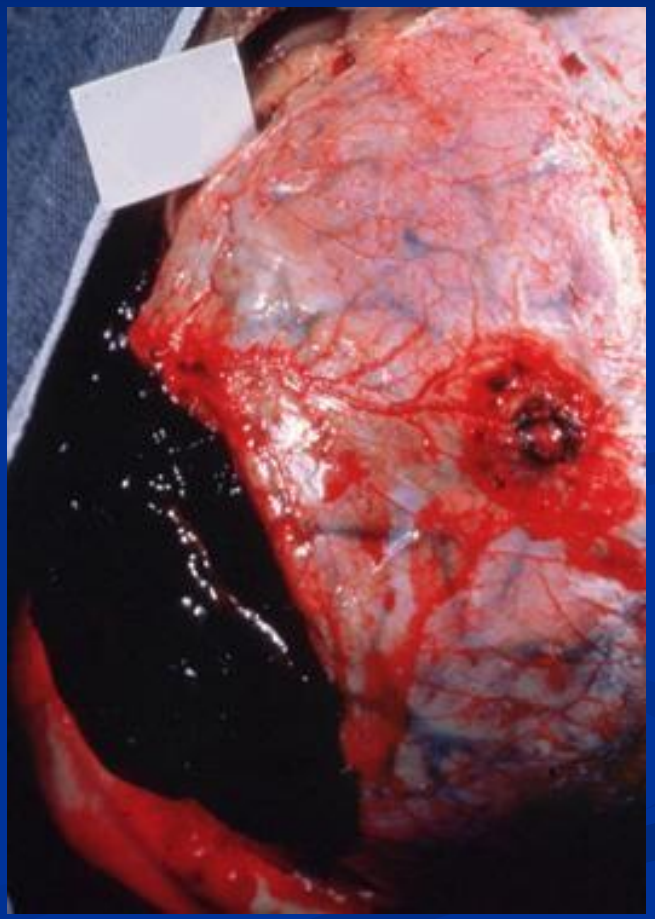






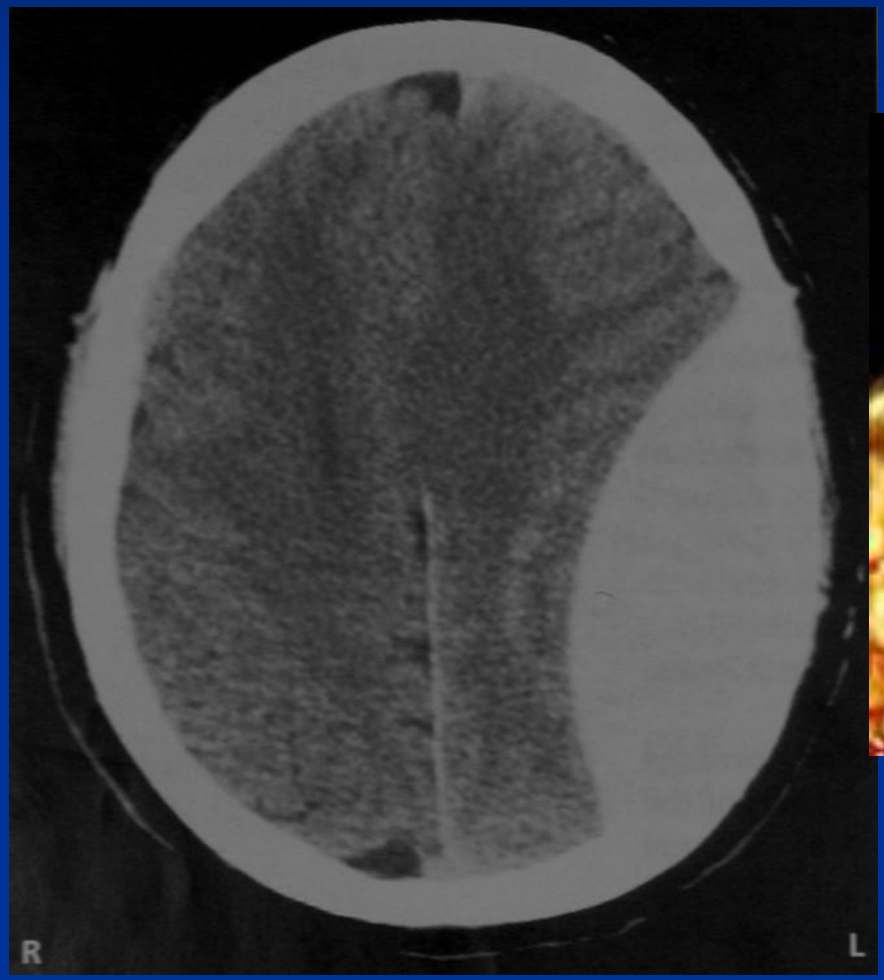
Epidural haematoma

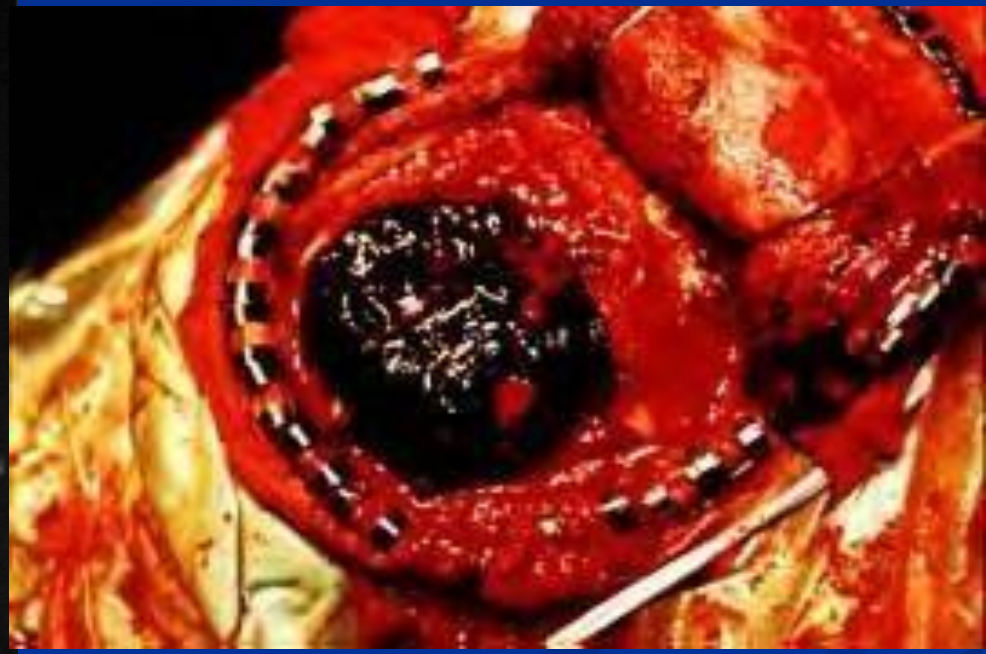




a. meningea media damage, CT: biconvex (lentiform sign)

Epidural haematoma



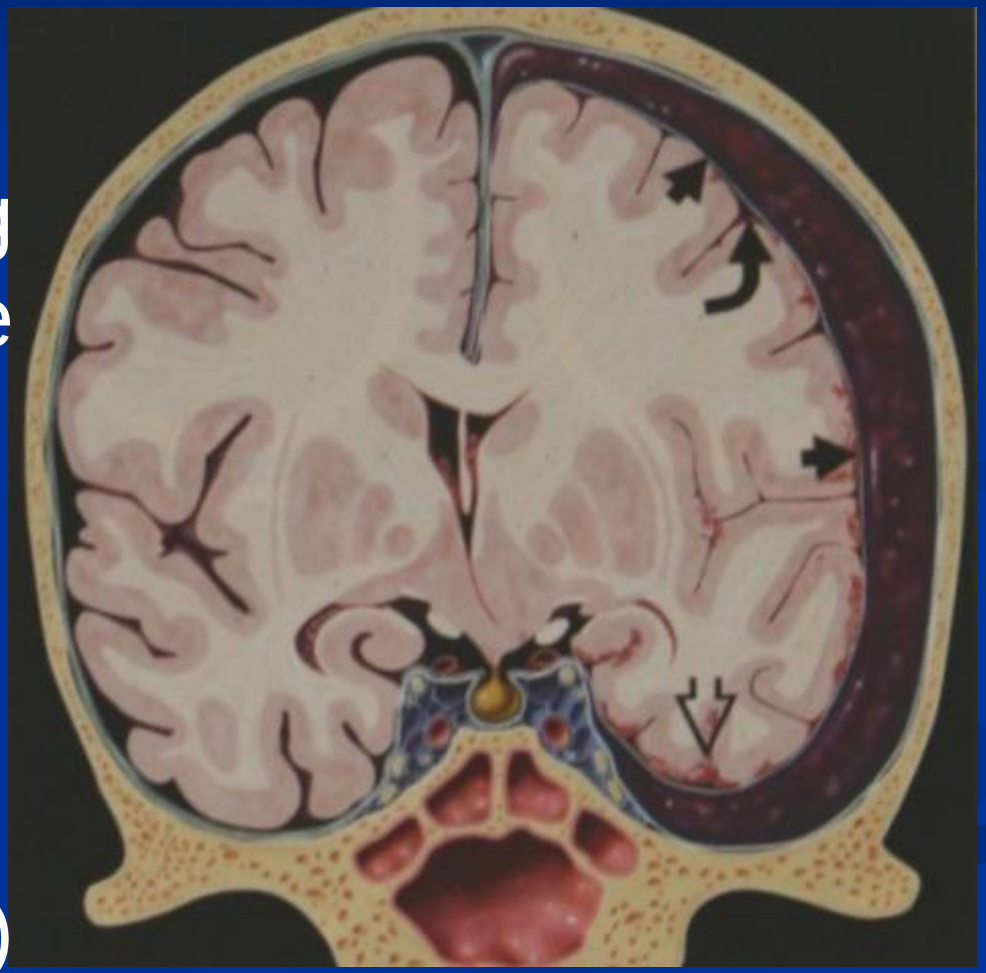


Treatment: haematoma evacuatio, supply of bleeding

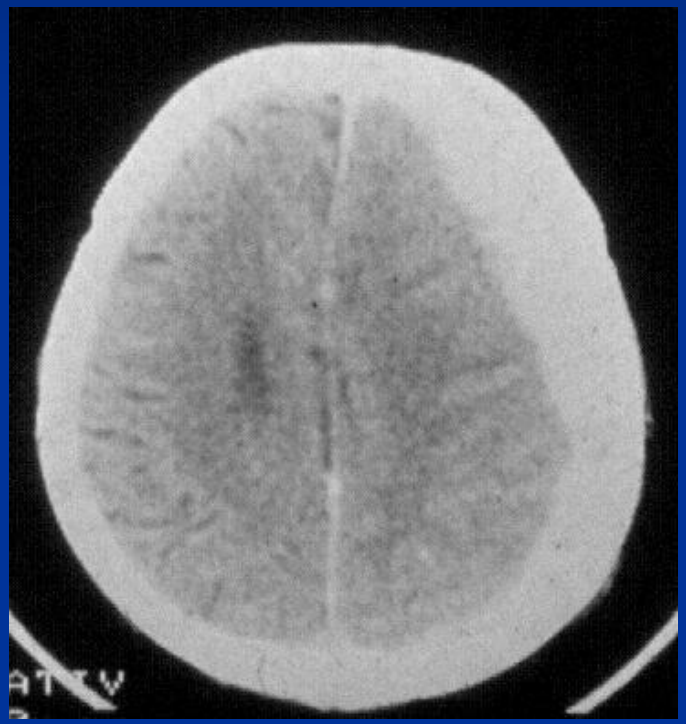
Subdural haematoma

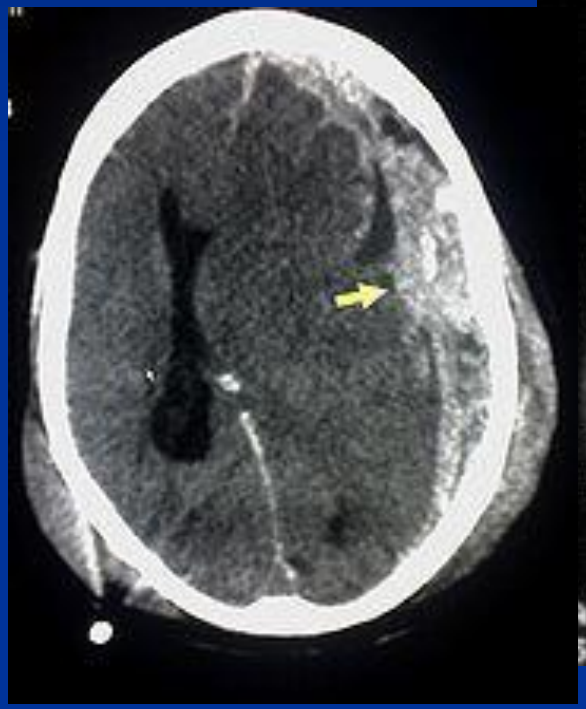
Result from the tearing of bridging veins crossing the subdural space

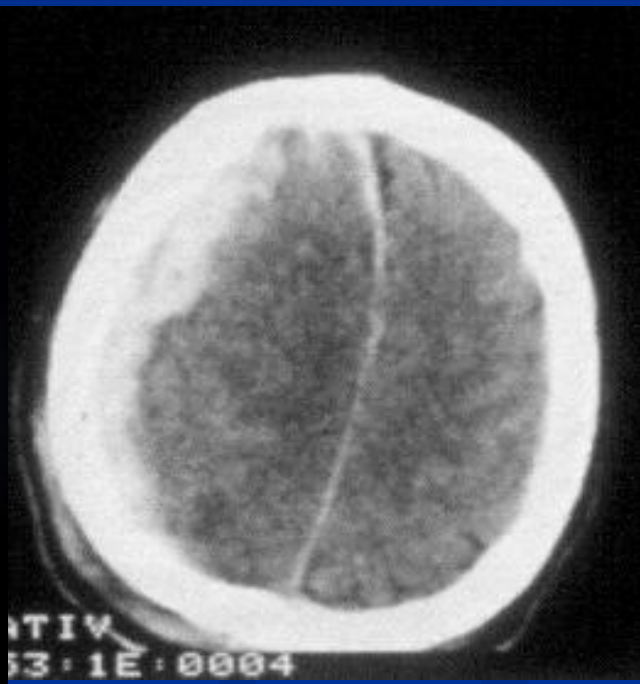
-acut
-subacut
-chronic
(reduced density)



Subdural haematoma: acut

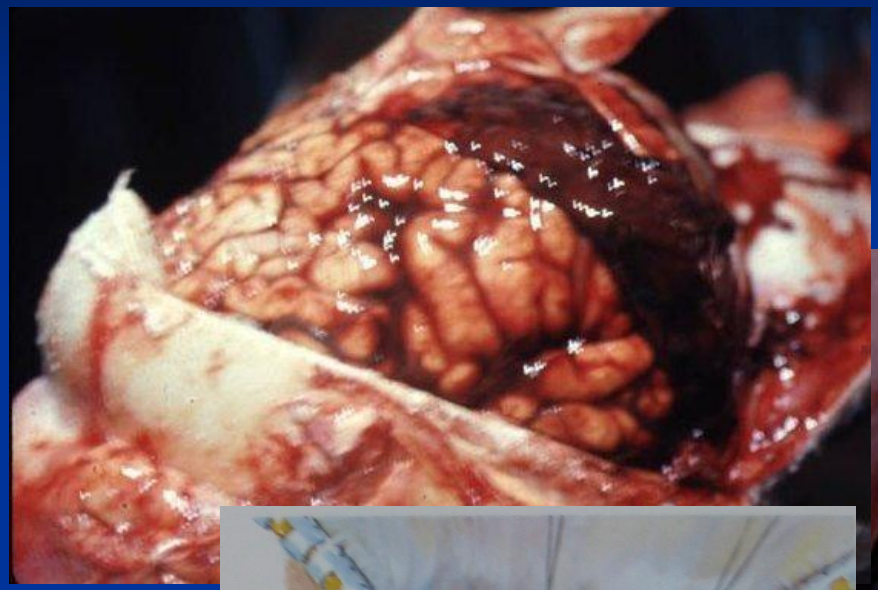


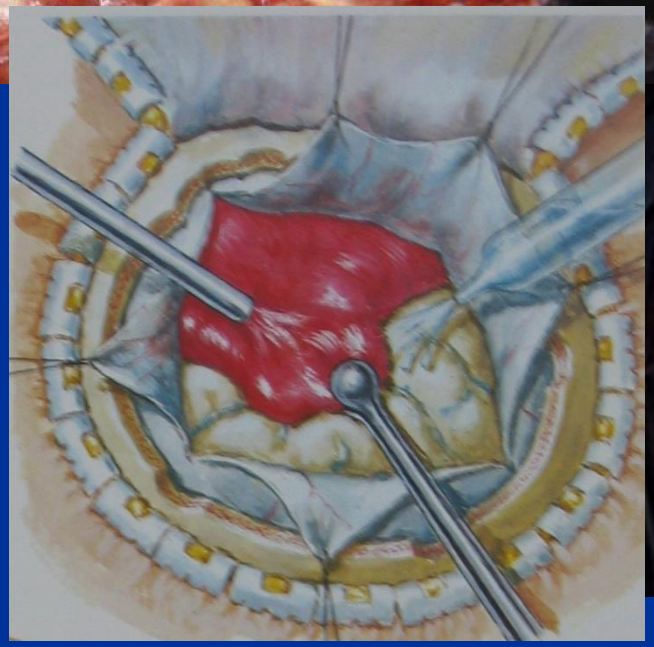


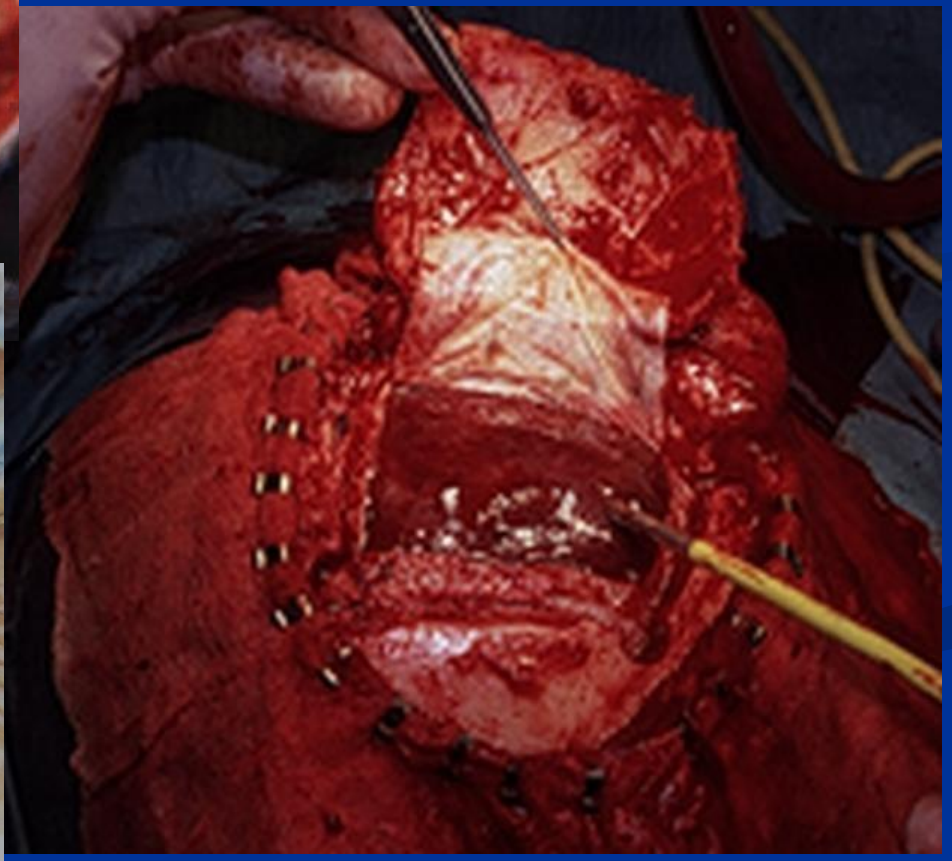


Treatment: haematoma evacuation (craniotomy, burr hole)

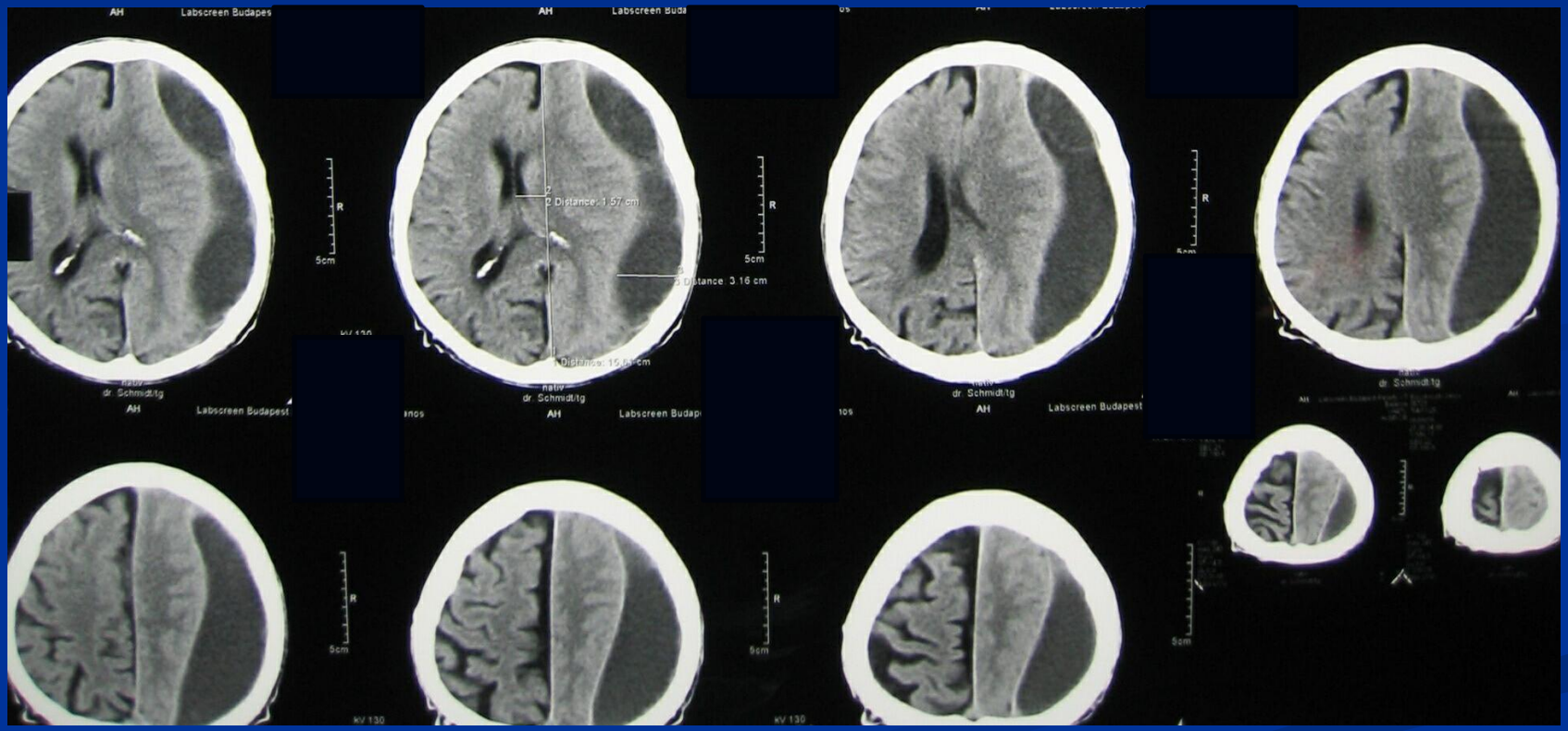
Subdural haematoma





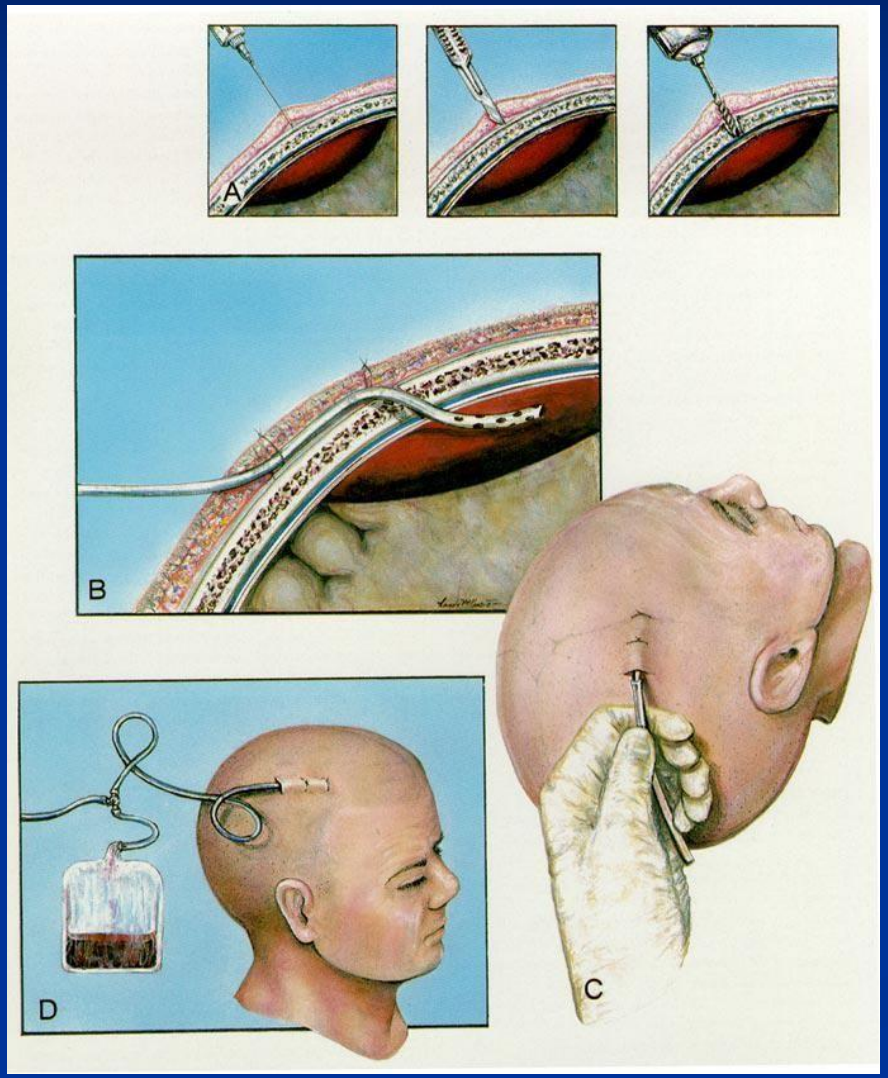


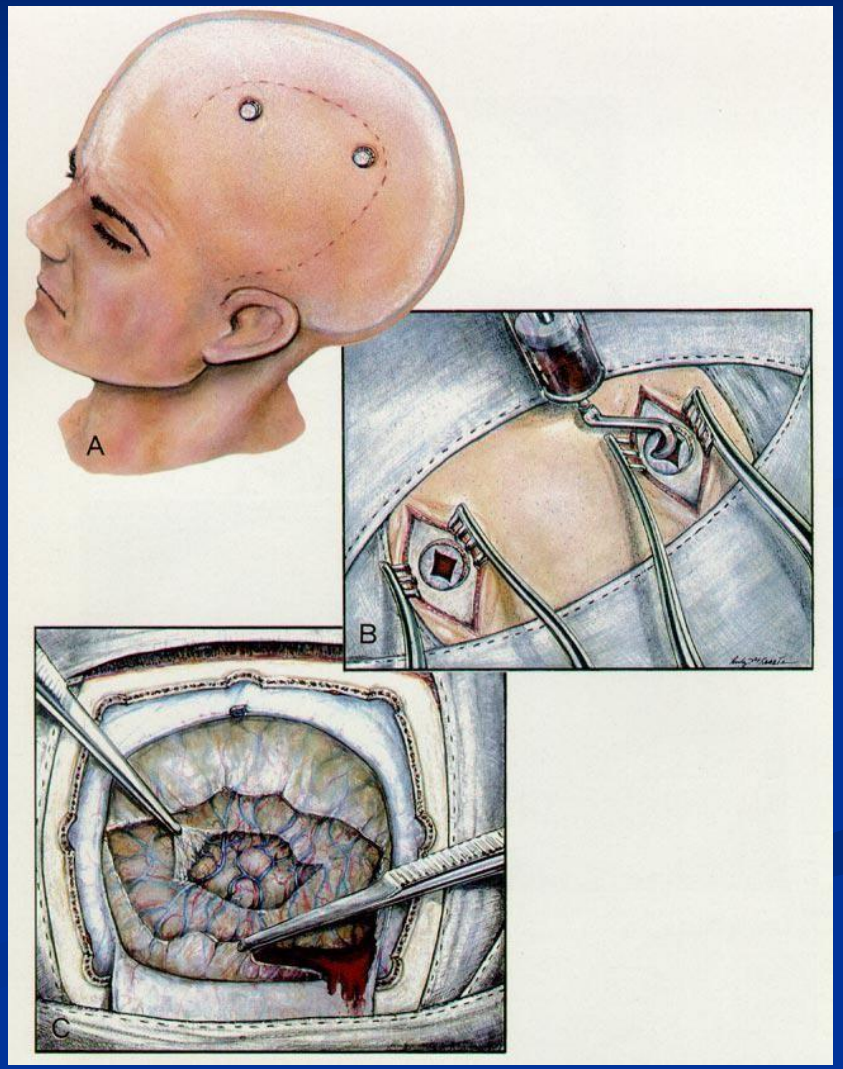
Subdural haematoma: chr.



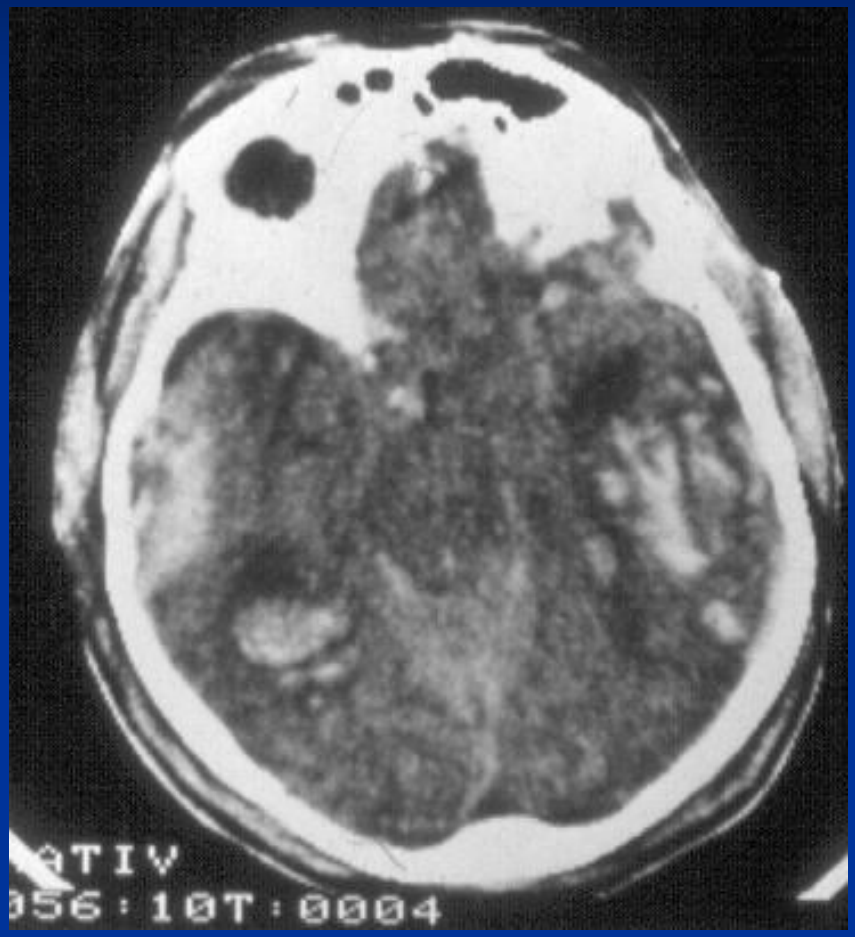
Treatment: haematoma evacuation (burr hole, craniotomy)

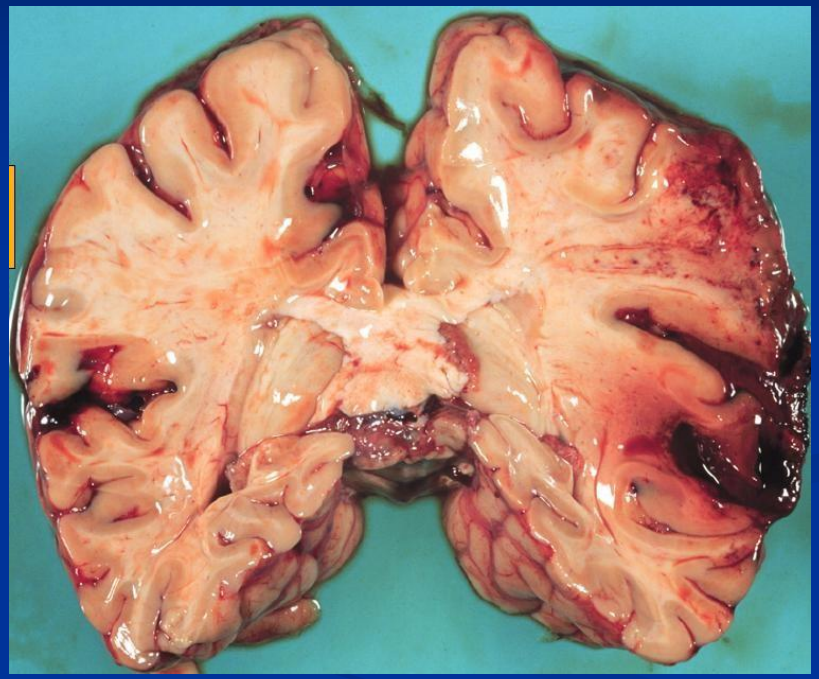
Subduralis haematoma: chr.

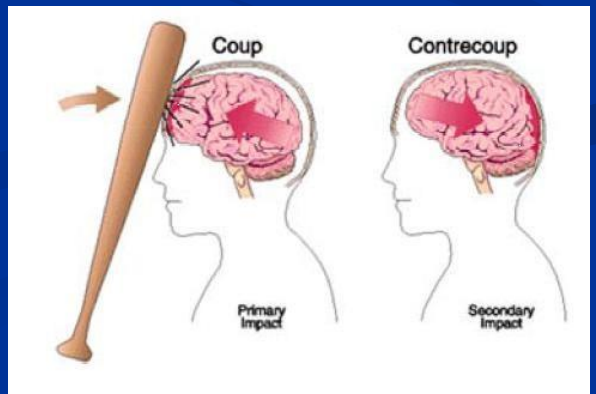




Cerebral haemorrhagic contusion

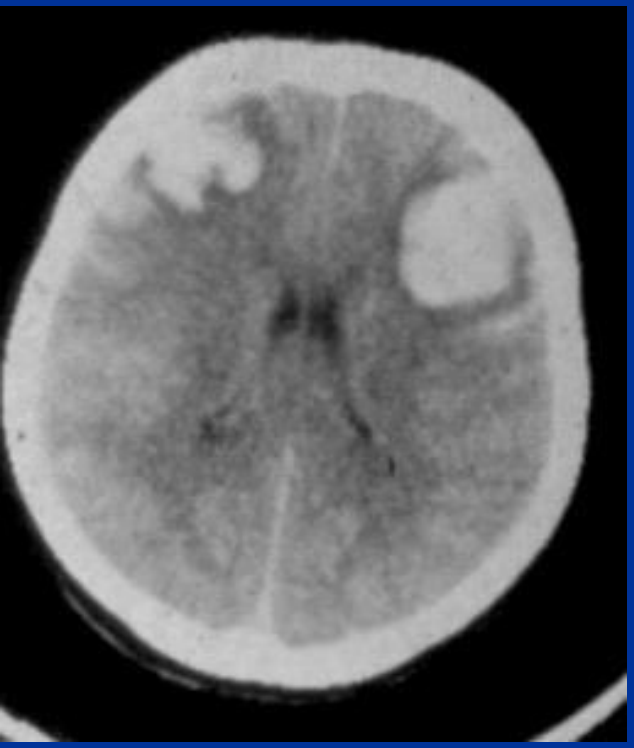


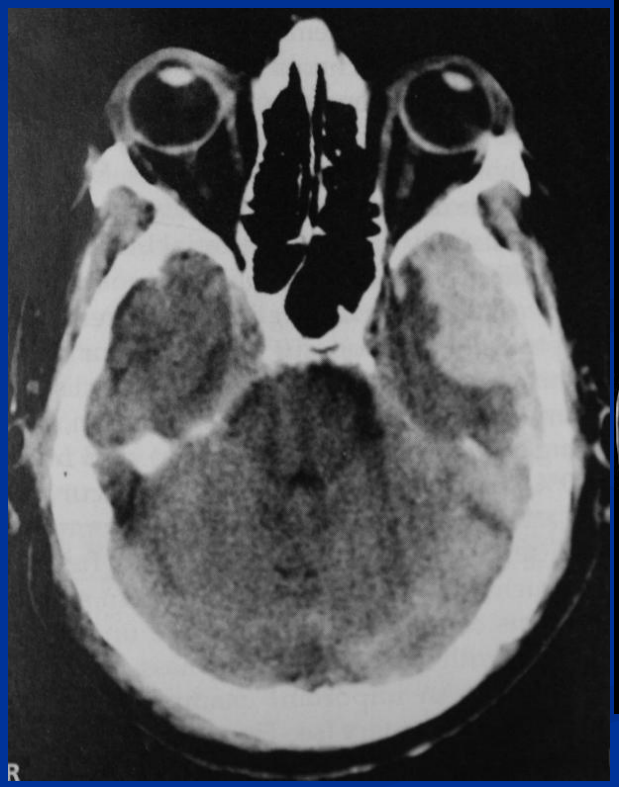


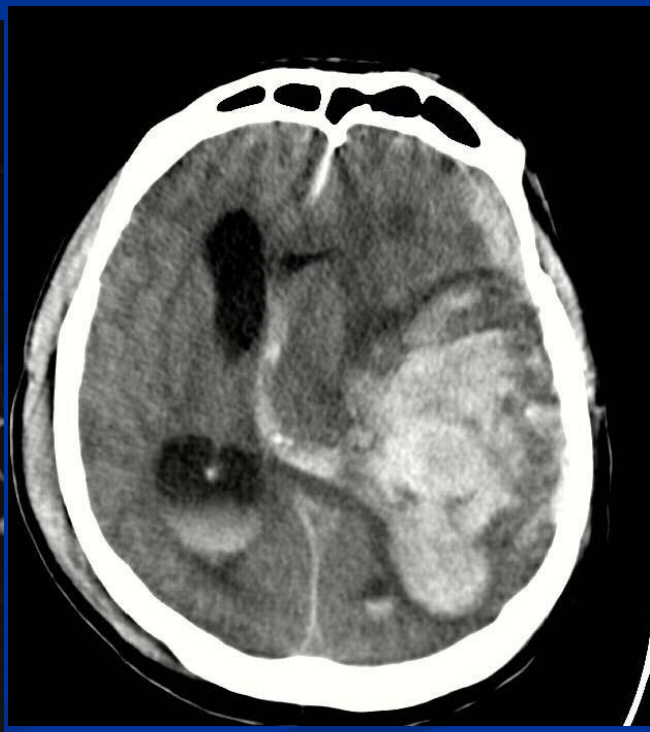


Contre-coup – Most contusions represent the brain coming to a sudden stop against the inner surface of the skull (the opposite side)

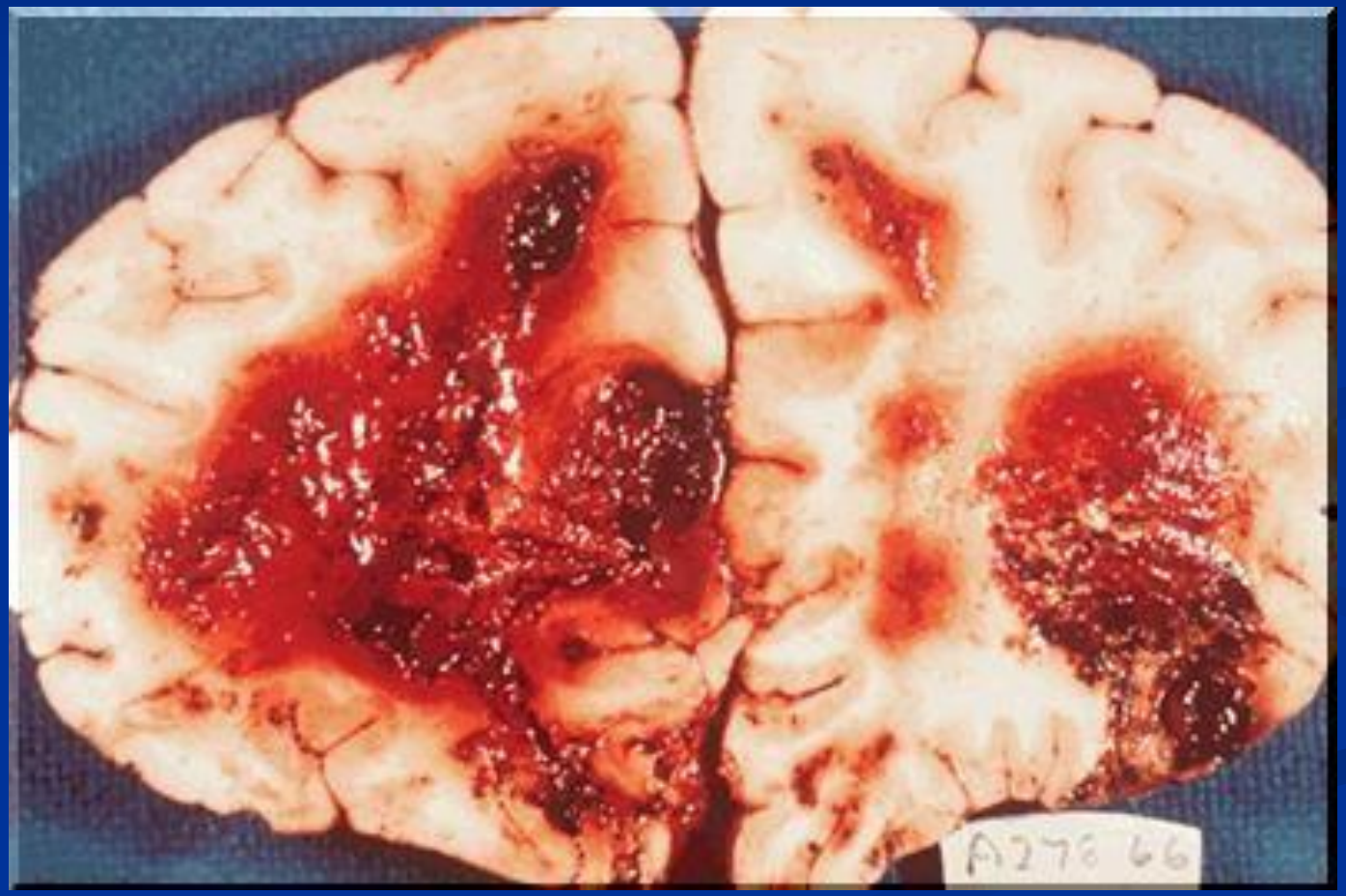
Cerebral haemorrhagic contusion







Cerebral haemorrhagic contusion



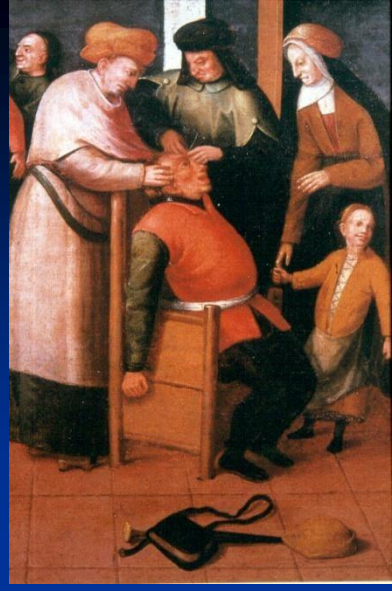
Treatment

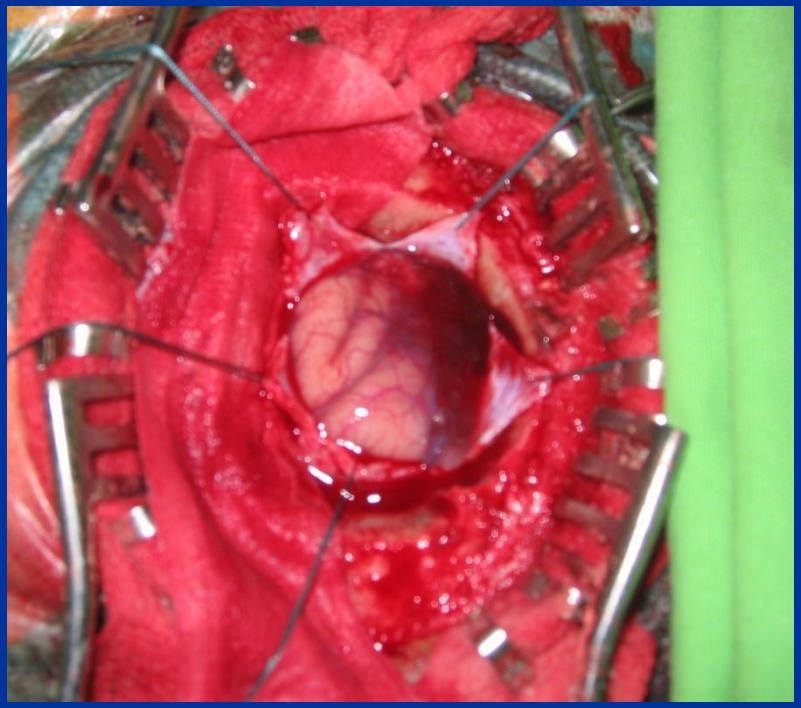
Conservative: monitoring and controlling ICP (decrease of oedema, dehydration)

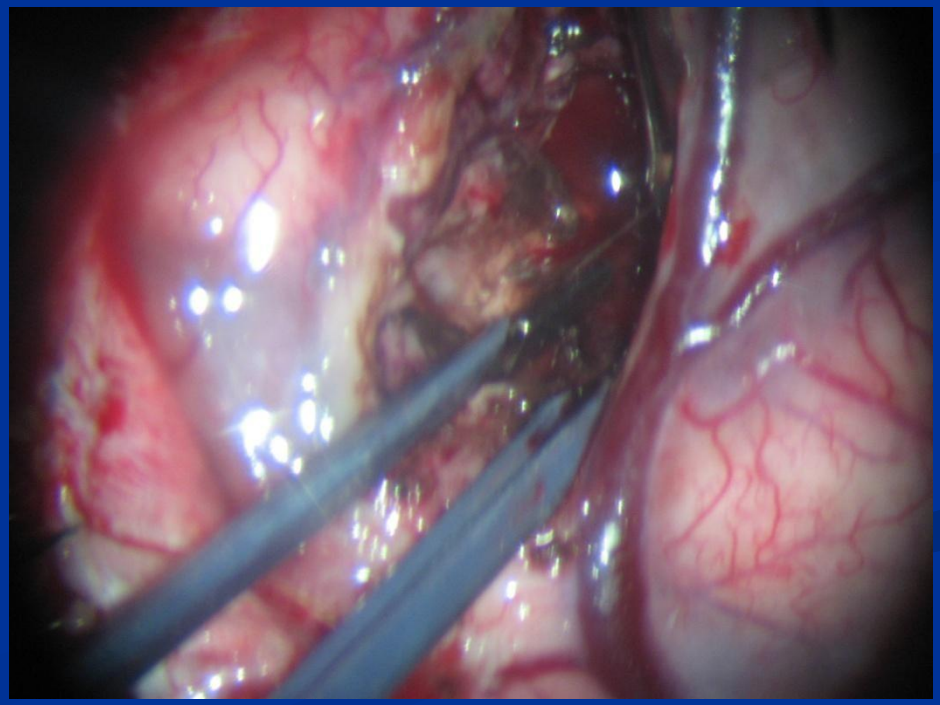
Operation: craniotomy and haematoma evacuation

Rarely: punctio and drainage

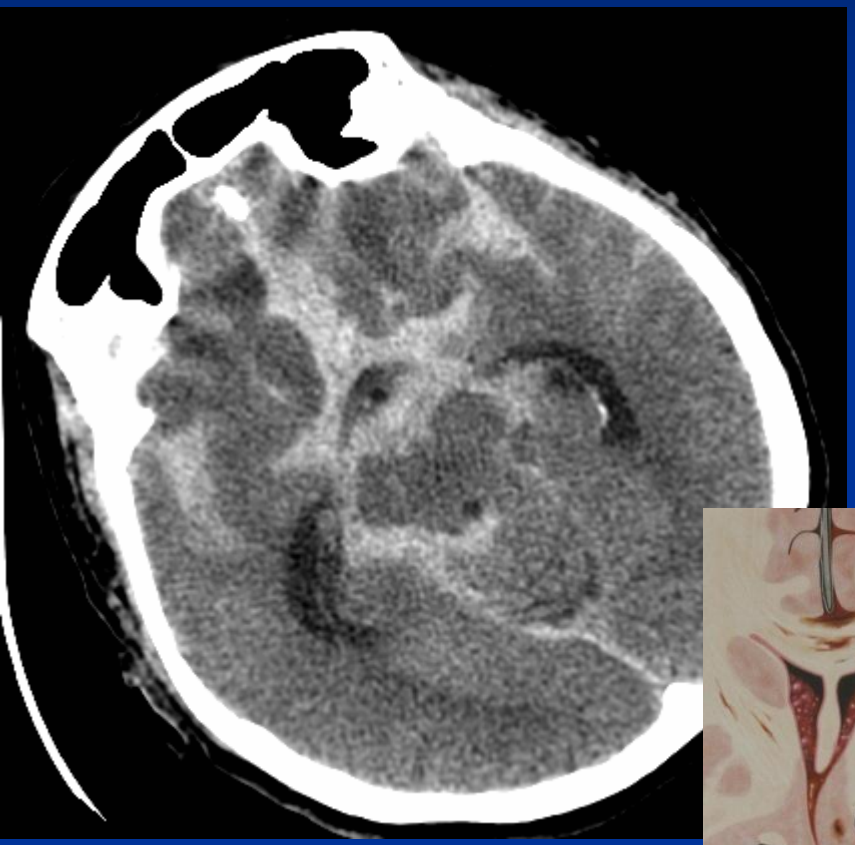
Haematoma evacuatio

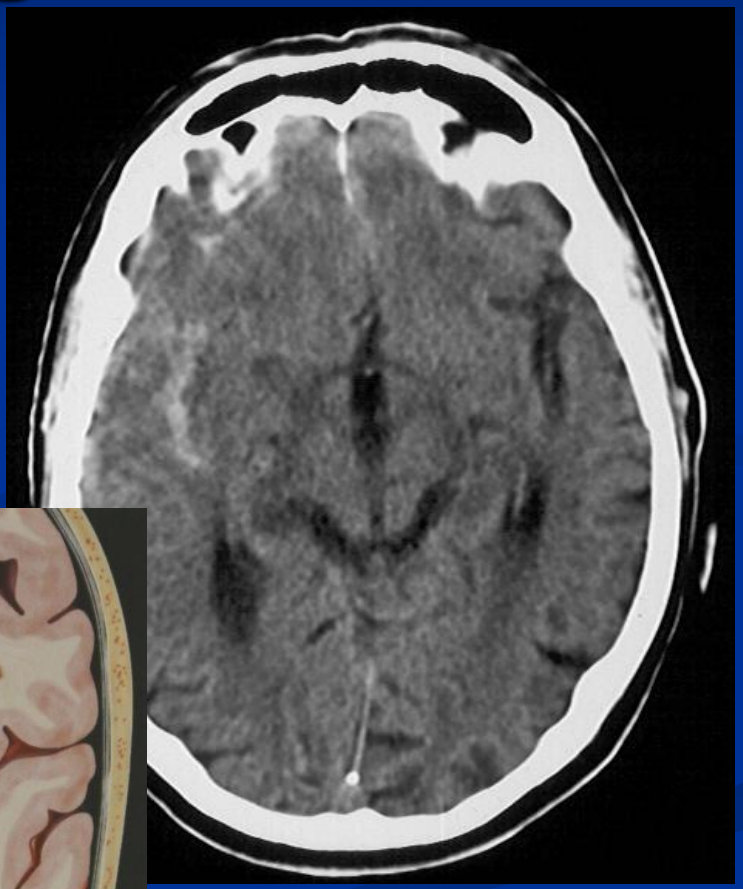


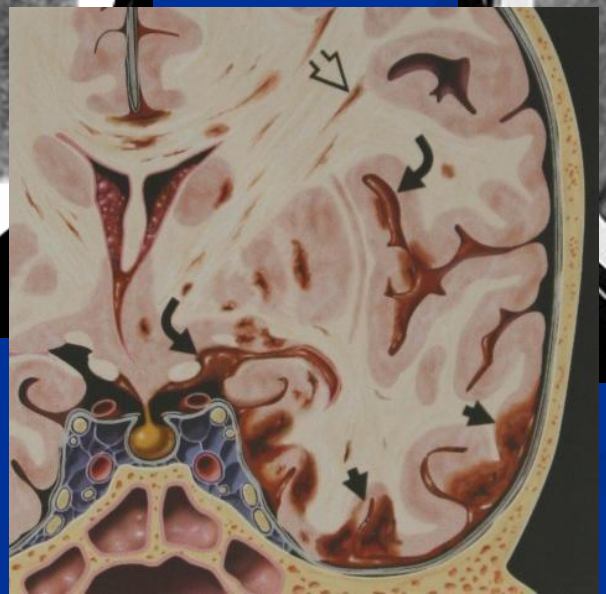




Traumatic subarachnoid haemorrhage







About treatment:

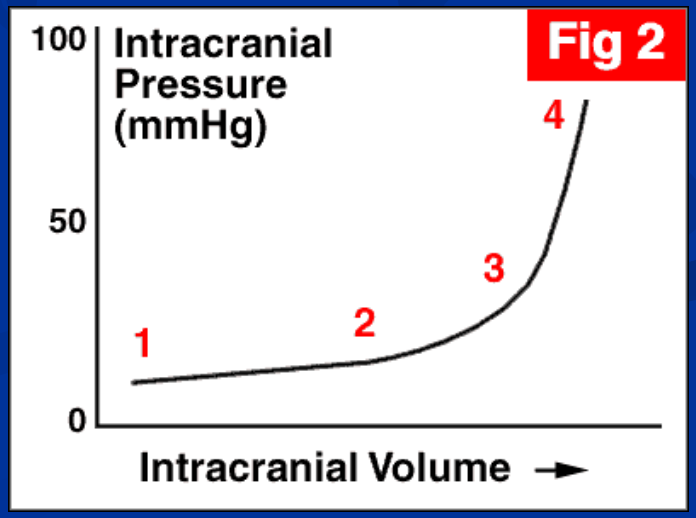
Prevention and treatment of intracranial hypertension

Maintenance of adequate and stable cerebral perfusion pressure

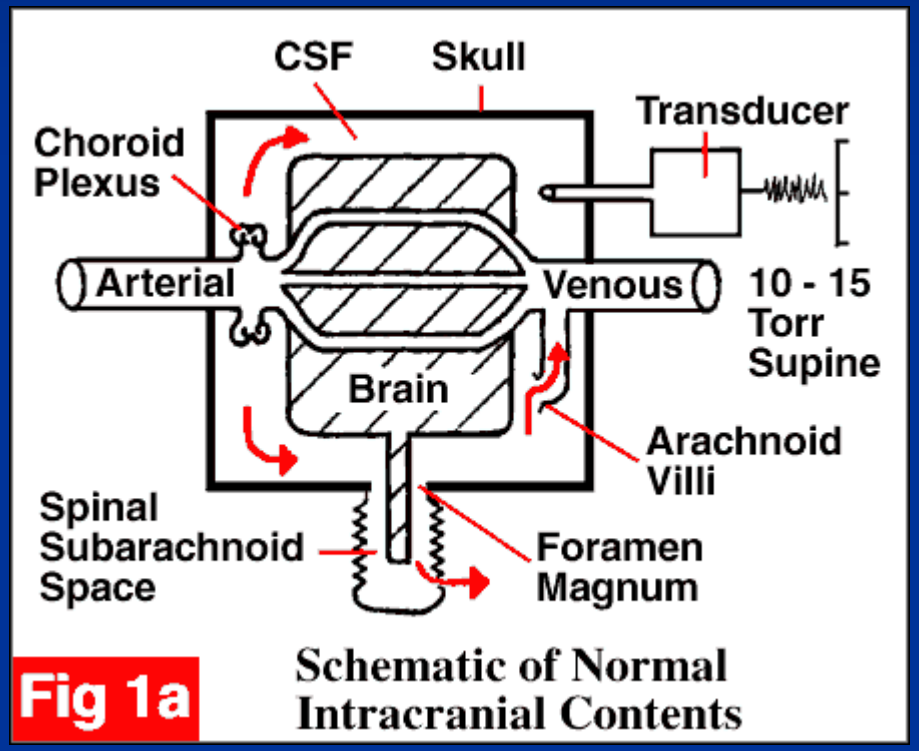
Avoidance of systemic secondary brain insults

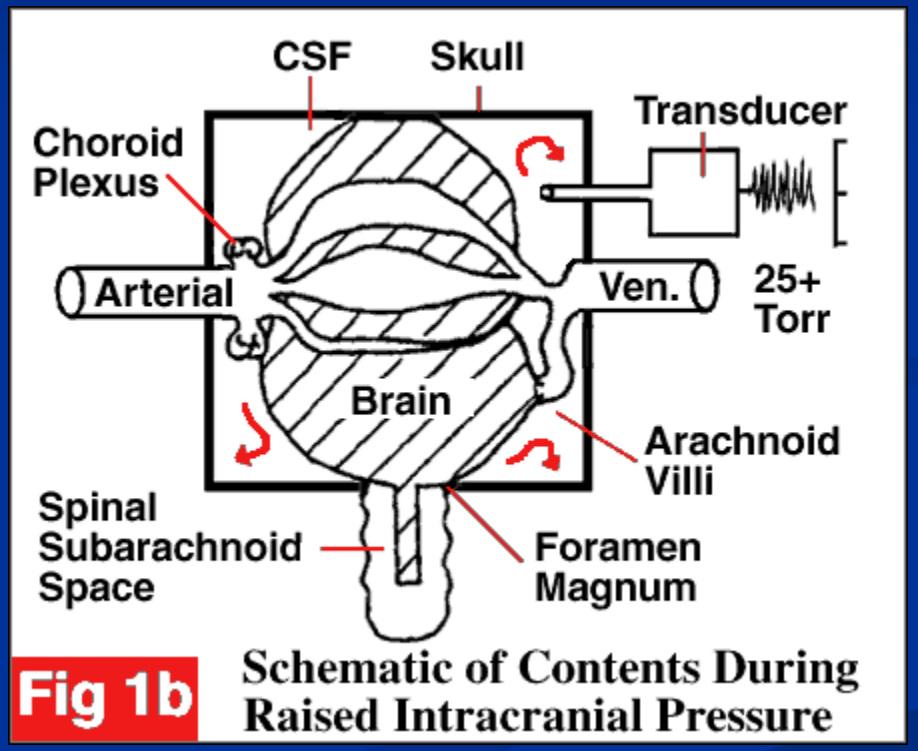
Optimization of cerebral haemodynamics and oxygenation

Norm. ICP < 10 mmHg



Intracranial volume



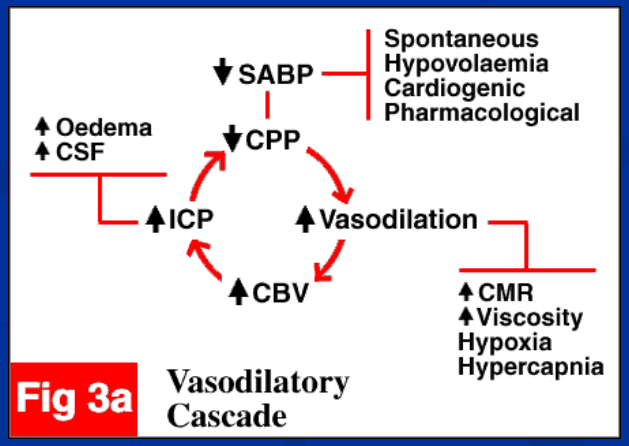


brain (80%), blood (12%) and liquor (8%).

ICP and CPP

Cranial perfusion pressure = mean arterial pressure – intracranial pressure (**CPP = MAP - ICP**)

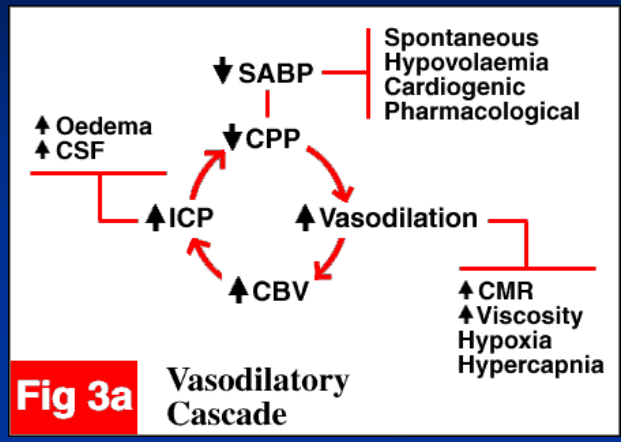
Normal CPP= 80Hgmm
(mild oedmea: (GCS 13-15)
ICP=20 or more Hgmm,
severe oedema,somatosus-soporosus (GCS <8)
Normal ICP= 5-13Hgmm



Pathophysiology

„circulus vitiosus”:

- trauma
- disturbed of microvascularis regulatio ischaemia, hypoxia
- oedema - (cytotoxic - vasogen) ICP increase
- disturbed of brain tissue perfusion , ischaemia



Therapy of oedema

-Hyperosmotic (Mannitol)

-hyperventillation (PCO_2 decrease – vasoconstrictio)

-liquor drainage (ventricular punctio)

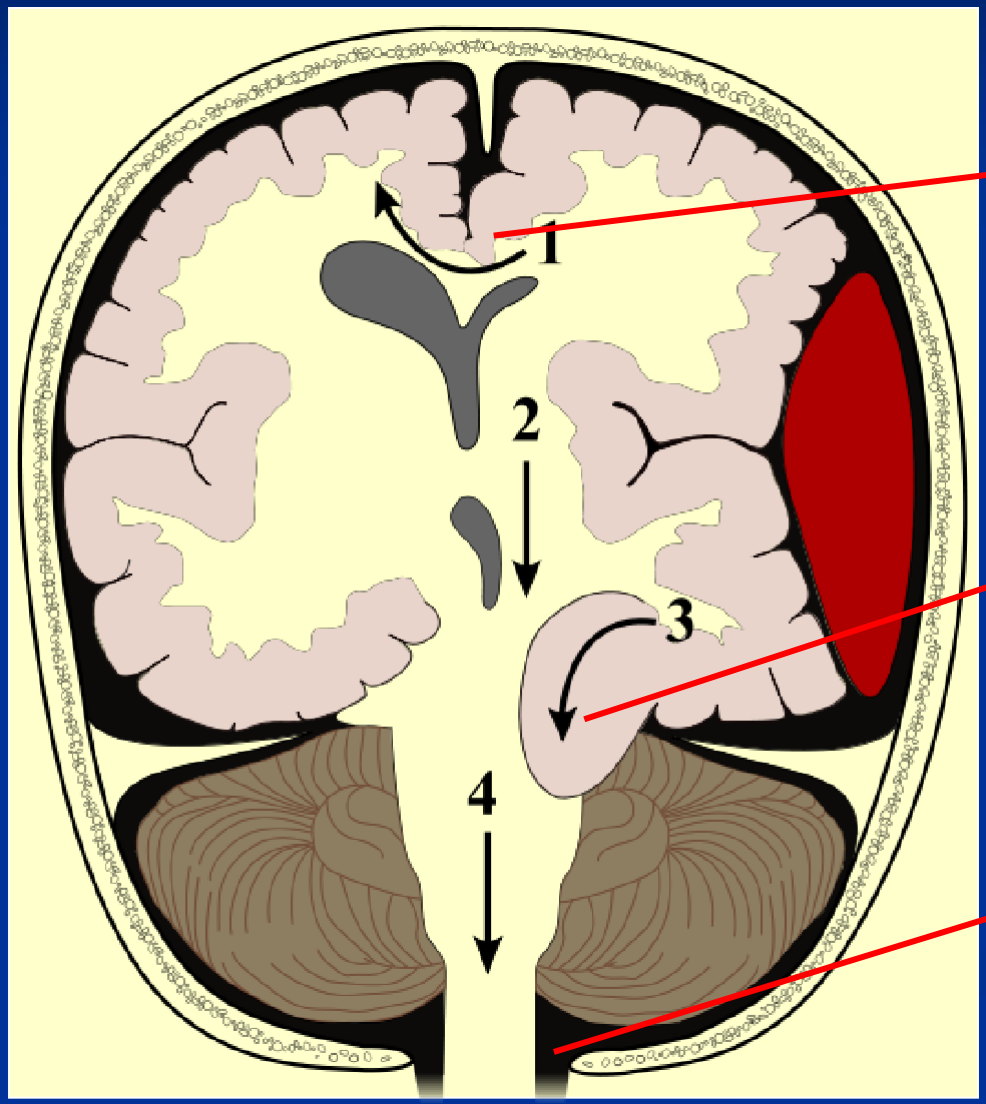
-barbiturat therapy (decrease of brain tissue metabolismus)

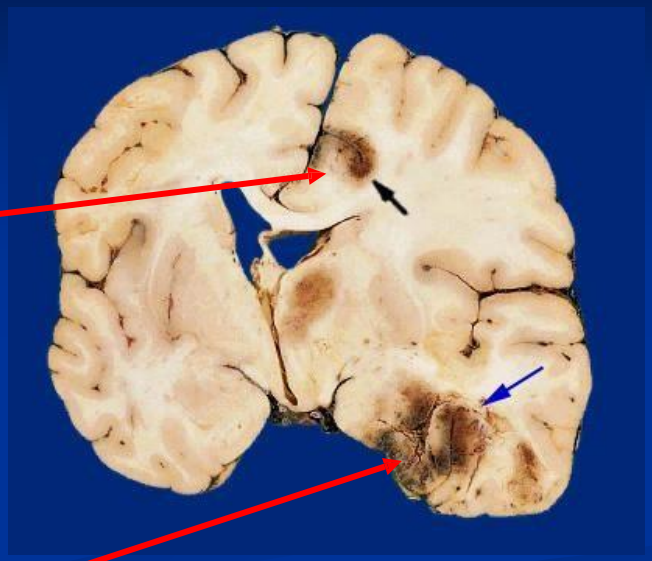
-diureticum (Furosemid)

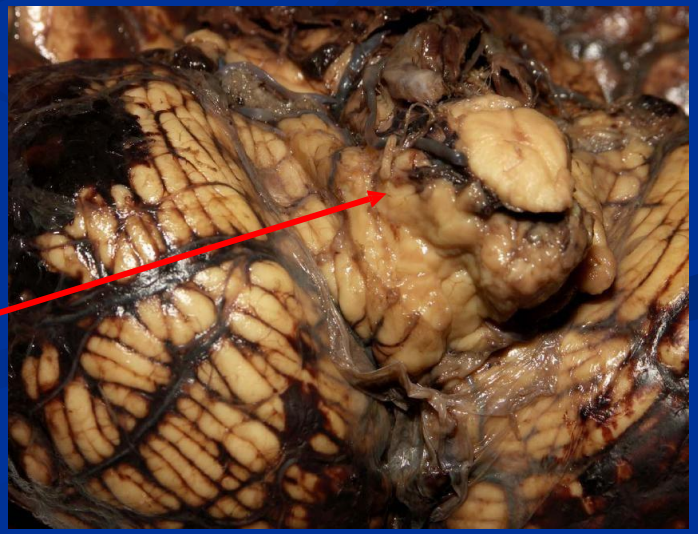
Symptoms of increase ICP

-Headache
-Vomiting
-Papilla oedema, visual disturbances
-Pupilla difference
-Loss of consciousness
-”Brainstem” herniation: bulbus deviation, Wernicke-Mann (flexio-extensio)
-Disturbe of cardiorespiratory system
-Cushing-reflex: increase of blood pressure +bradycardia

Brain herniation







Treatment

subdural, epidural, i.c. haematoma evacuation

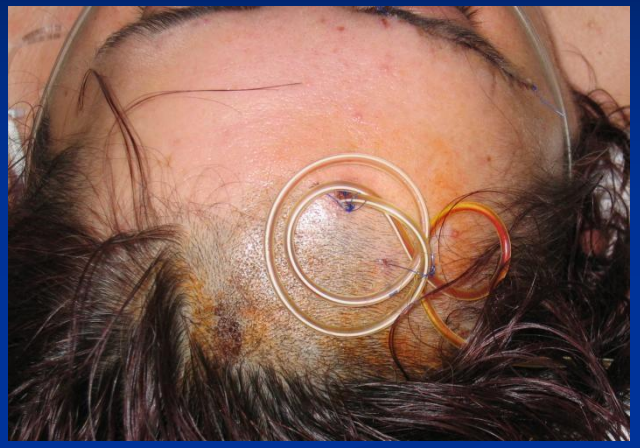
- ICP < 20 Hgmm and CPP > 60 Hgmm
  Diuretic therapy, osmotherapy, liquor drenage,intubation,hyperventilatio

- If in spite of osmotherapy ICP > 20 Hgmm
  barbiturat coma
- If i spite of using osmotherapy and barbiturat ICP > 20 Hgmm

Decompressiv craniectomy

ICP monitoring

Under GCS 8

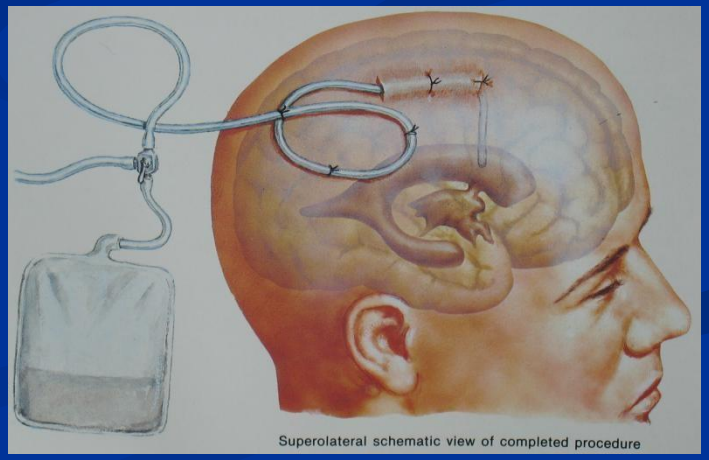


Type of ICP monitoring

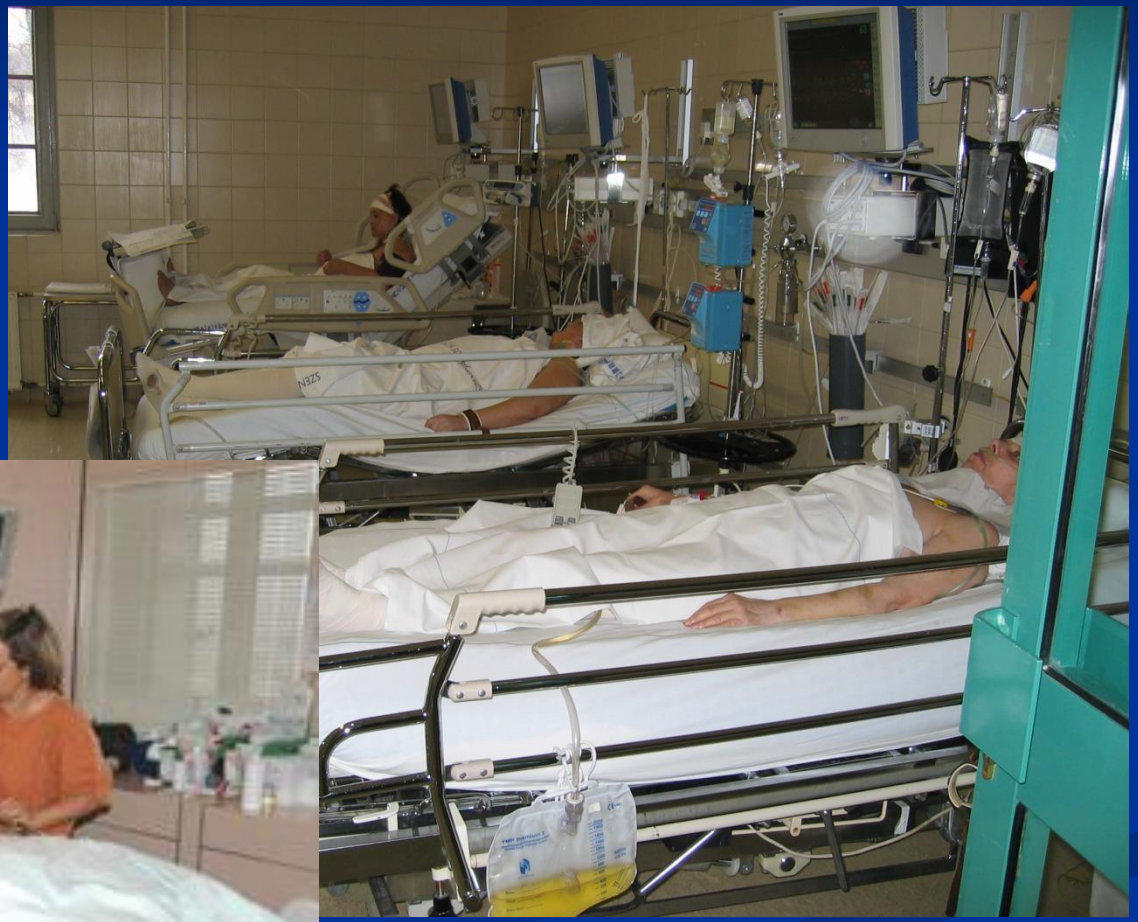
- Epidurális

- Intraparenchymális

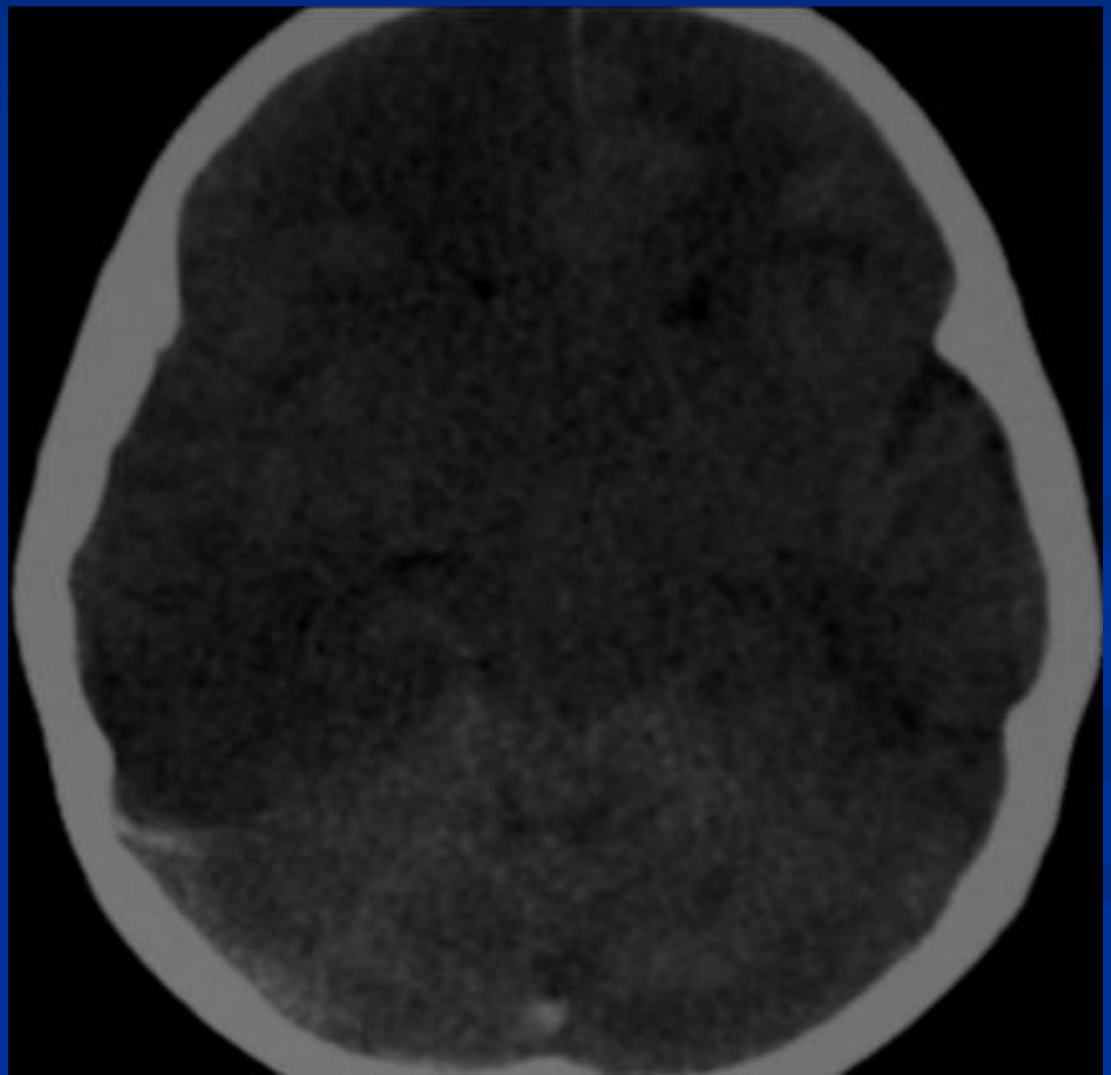
- Intraventriculáris (with liquordrainage)



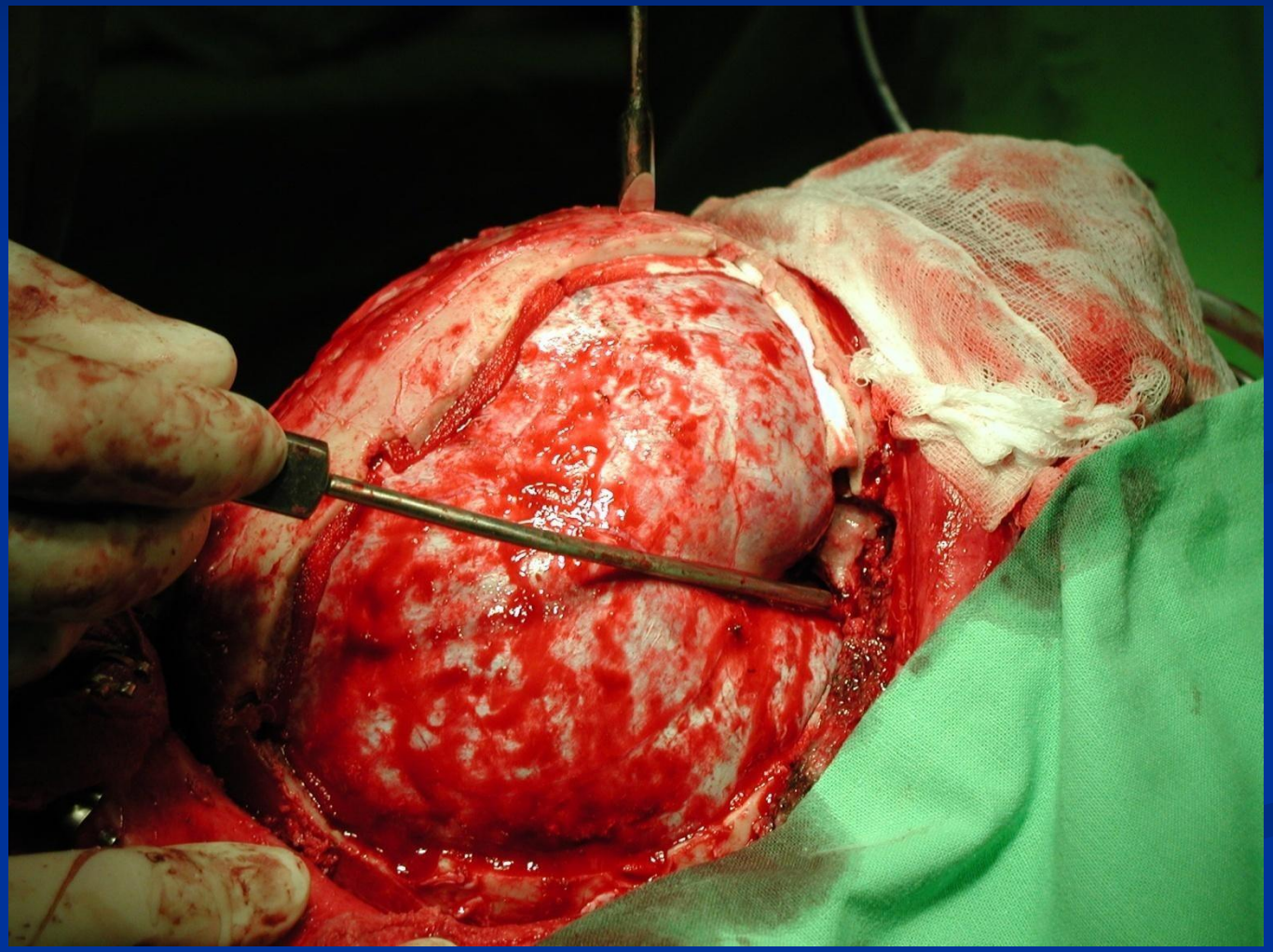
Intensive care unit



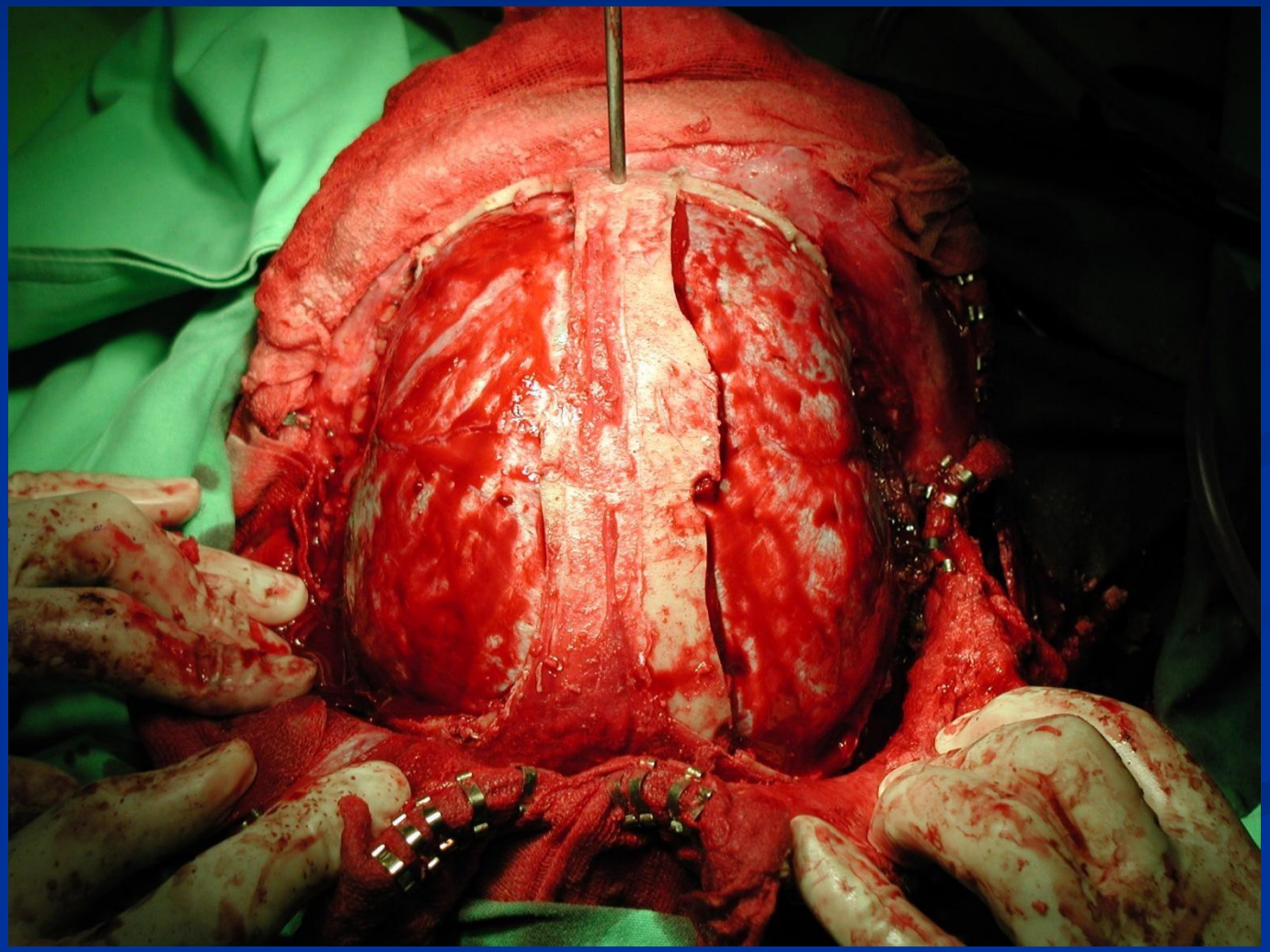
Diffuse brain oedema



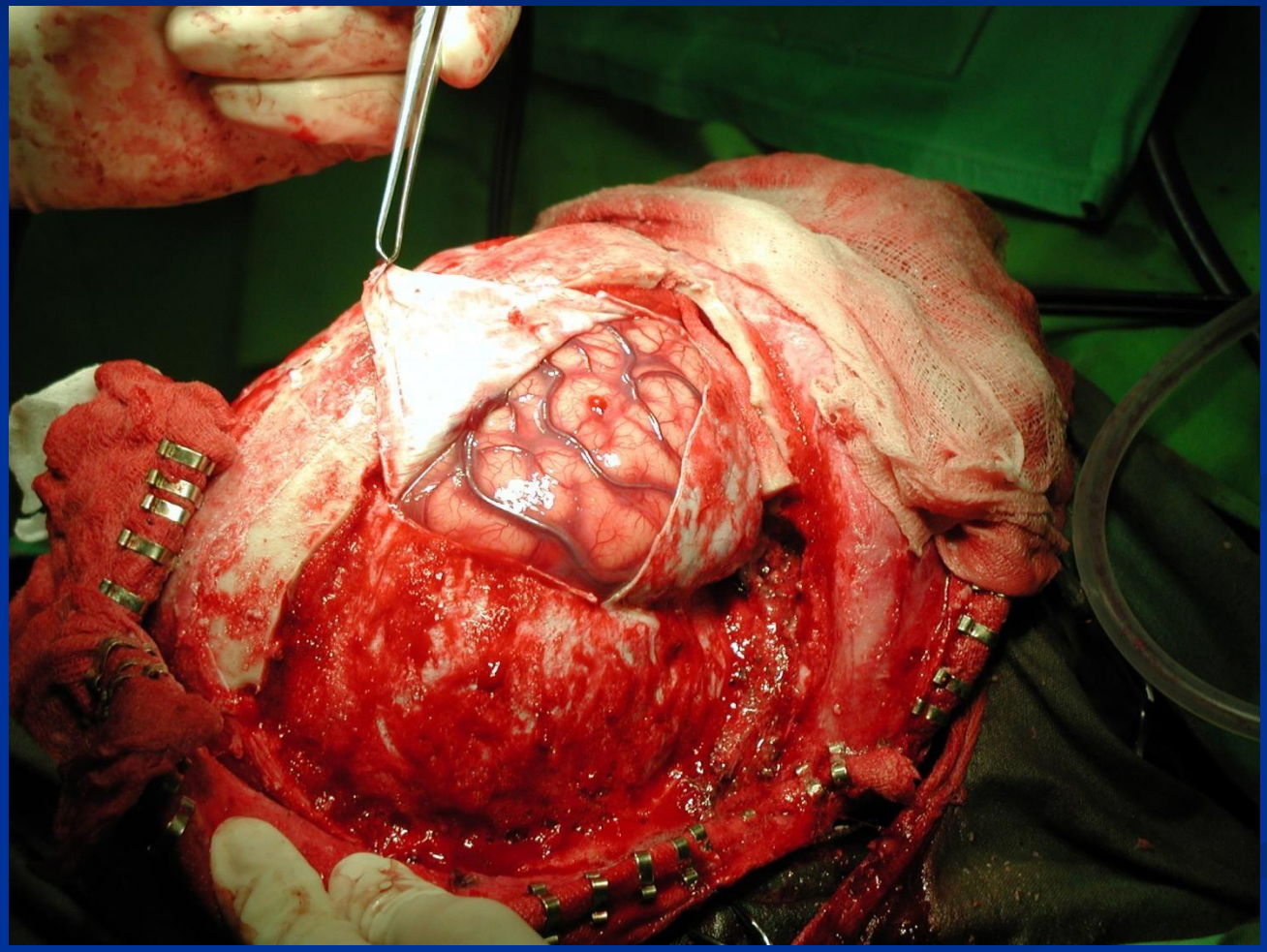
Decompressive craniectomy



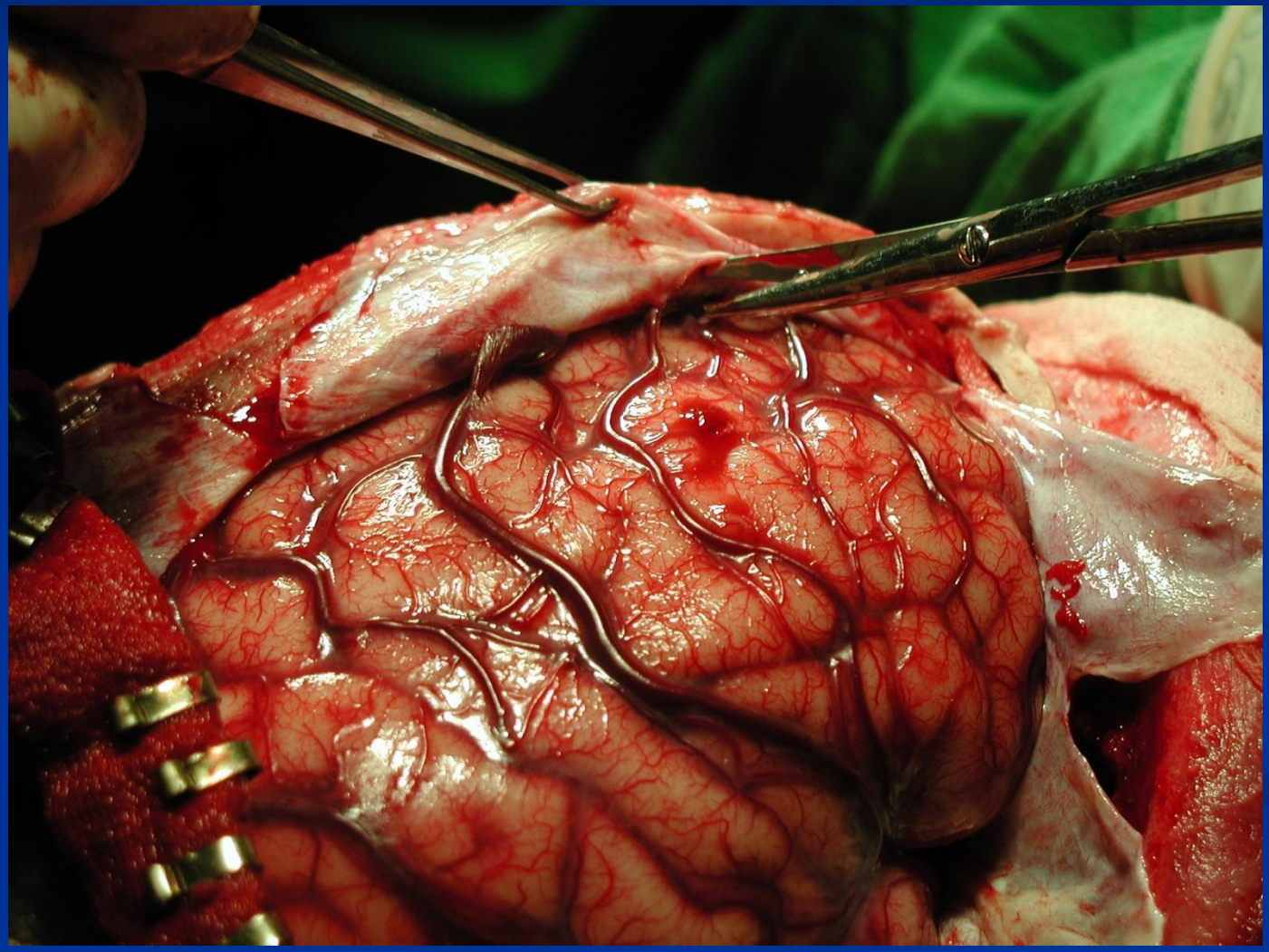
Decompressive craniectomy



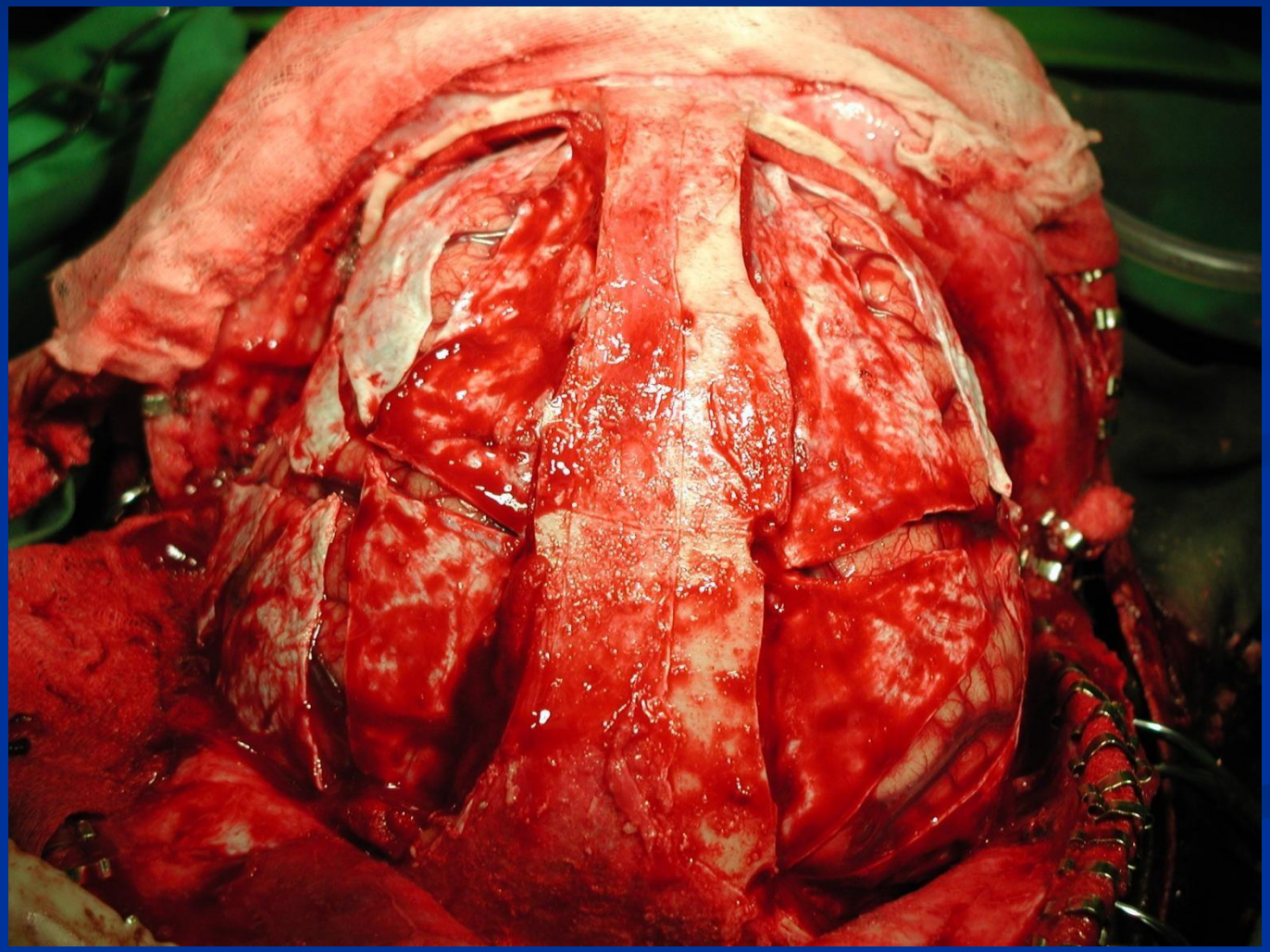
Decompressive craniectomy



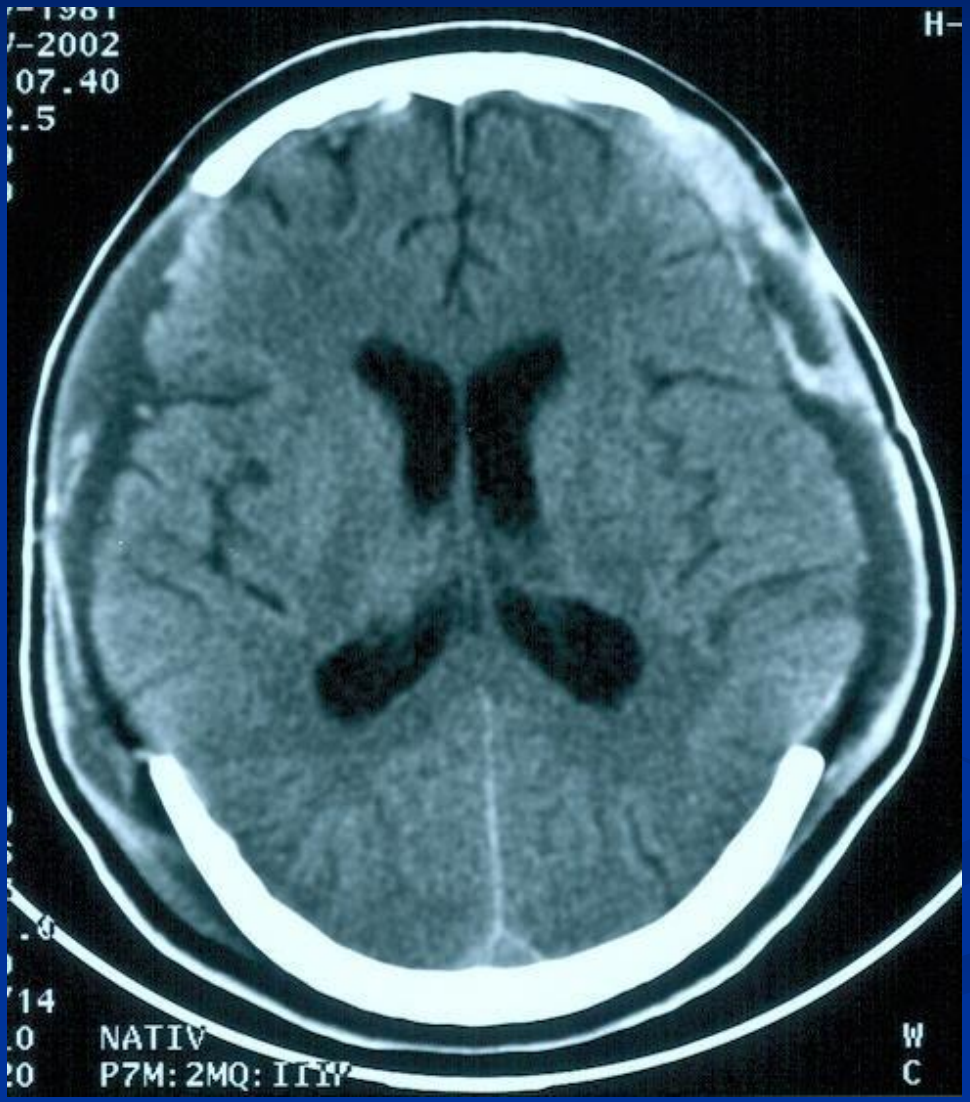
Decompressive craniectomy



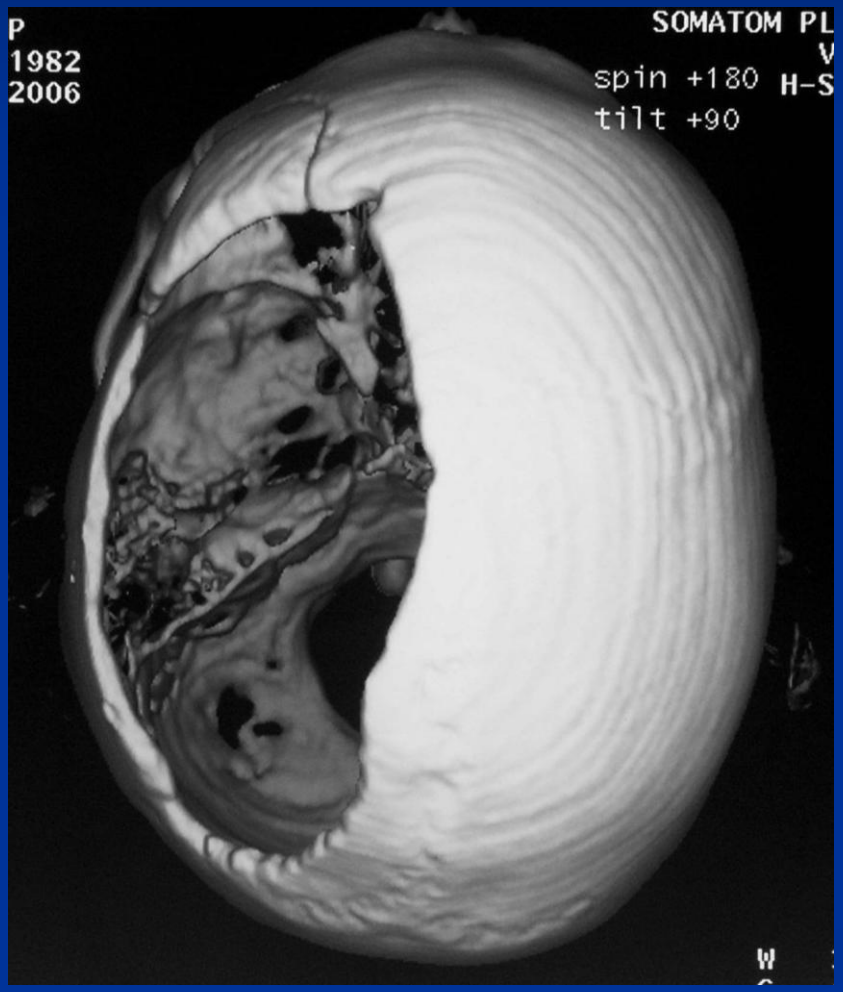
Decompressive craniectomy

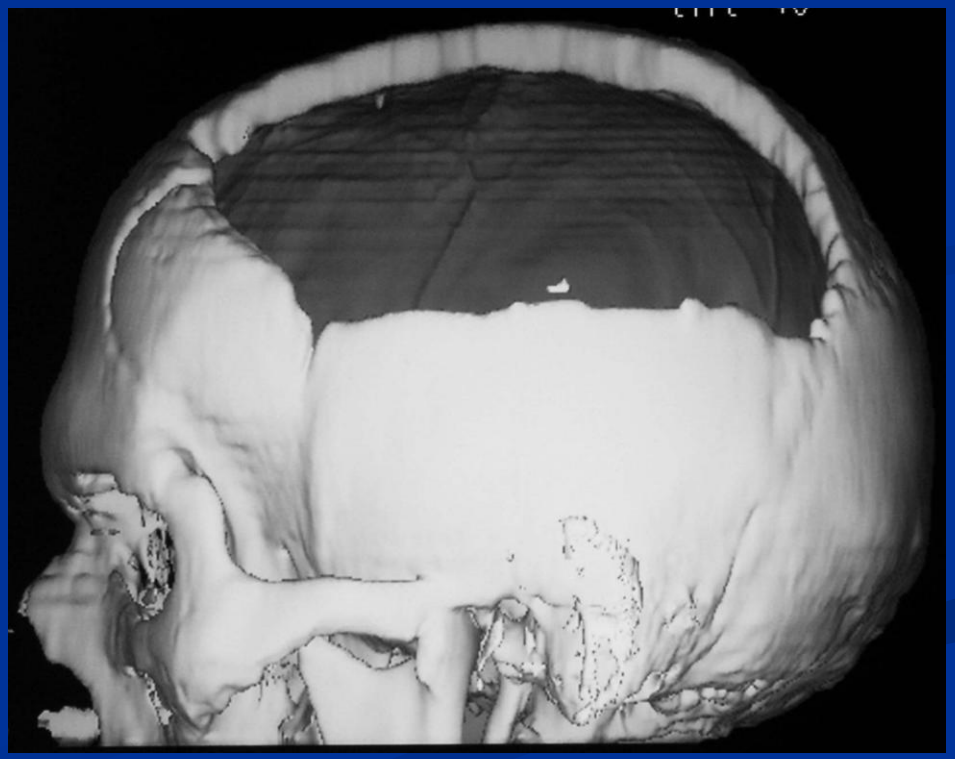


Decompressive craniectomy

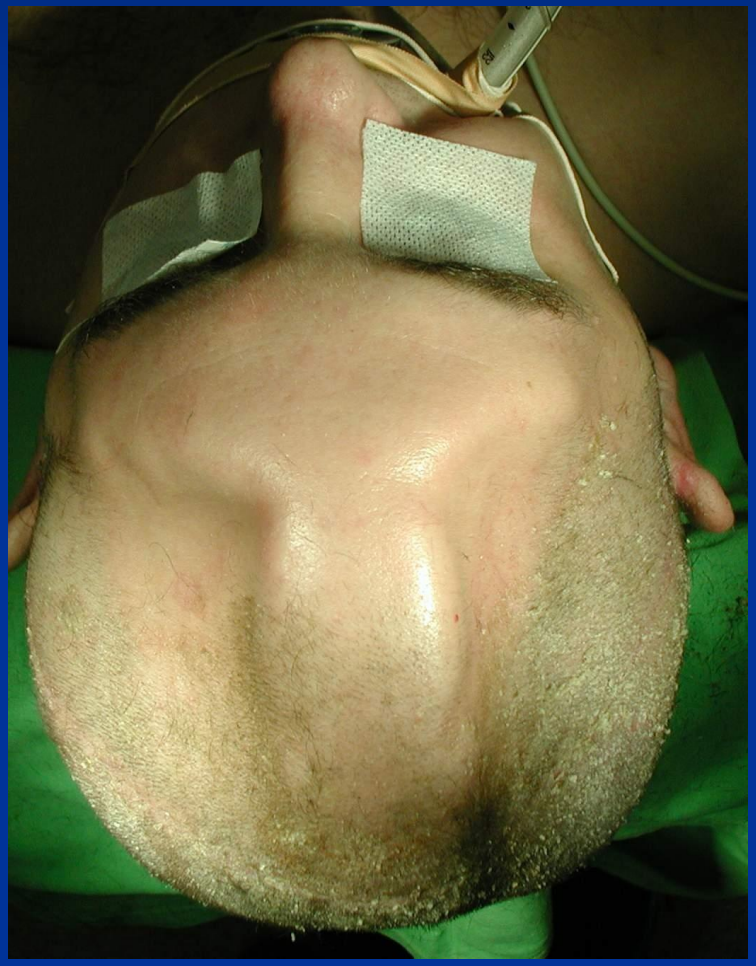


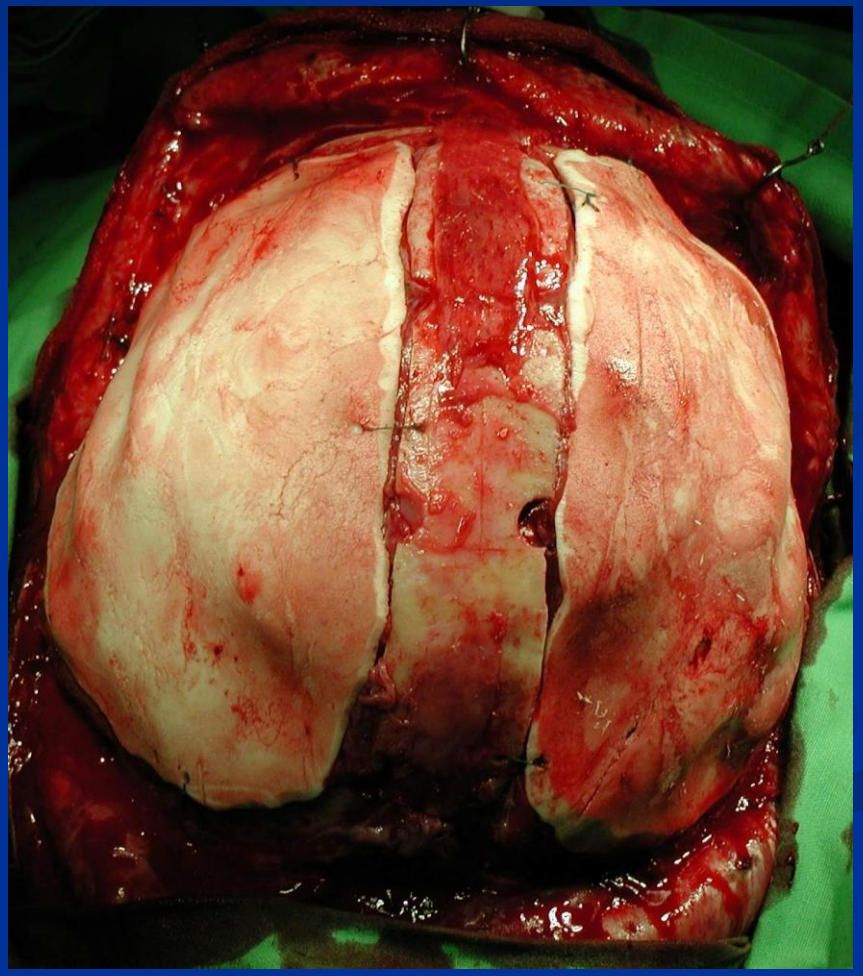
Decompressive craniectomy



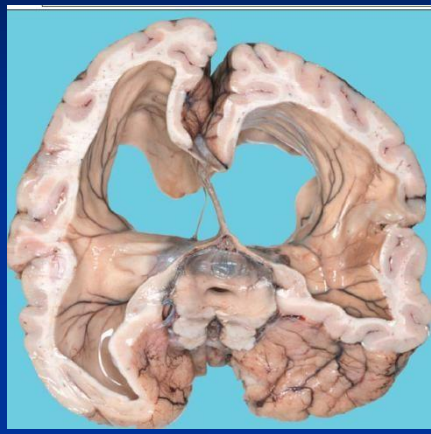


Cranioplastic

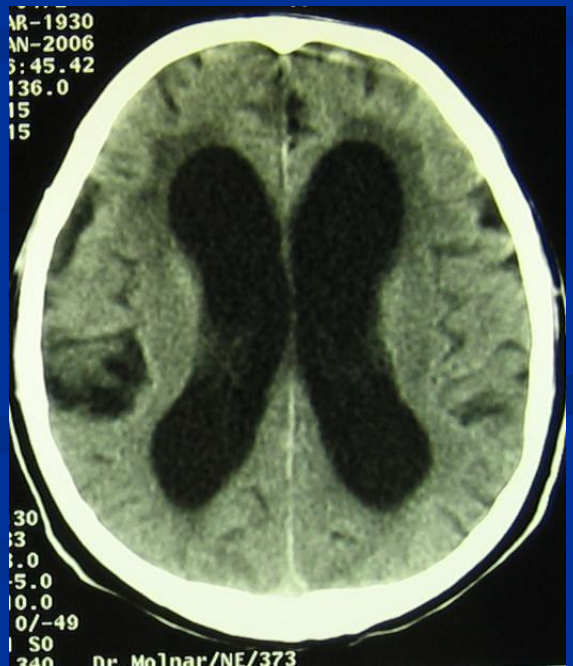




Posttraumatic hydrocephalus



- headache
- dementia
- ataxia (frontalis dysbasia)
- incontinentia urinae



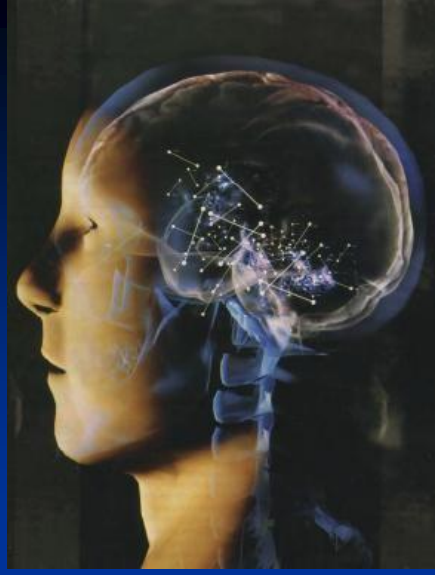
- Treatment: Ventriculo-peritoneal shunt

Posttraumatic epilepsy

- <5% cases without loss of consciousness,
- 10-11% impressed bone fracture
- 10-14% loss of consciousness >24h
- 20-30% haemorrhagic contusion
- 45% IC haematoma

Treatment: carbamazepin, hydantoin

The aim:



management of
secondary brain injury to
decrease of mortality

KEEP CALM AND THANK YOU FOR YOUR ATTENTION