I_❤_NEUROSURGERY INITIATIVE

CRANIO-CEREBRAL INJURIES (2)

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SECONDARY EVENTS AND COMPLICATIONS OF HEAD INJURIES

SECONDARY EVENTS

- HYPOXIA
- ISCHEMIA
- EDEMA

HYPOXIA

- It is a decrease in oxygen supply to the brain tissue.
- One of the most common and most preventable causes of patient deterioration after primary injury.
- Can be caused by:

1. Presence of airway obstruction.
2. Acute respiratory distress syndrome.
3. Central respiratory depression.
4. Chest injury as a part of multi-trauma.
5. Unobserved epileptic attack.
6. Shock state.

ISCHAEMIA

- Poor tissue perfusion with impaired cellular metabolism.
  - Hypovolemic shock; could be either:
    - Bleeding to the outside as in the case of scalp laceration.
    - Intracranial bleeding in infants, because the skull's sutures can expand, while in adults intracranial bleeding will not cause shock because of limited space made by the fused suture

EDEMA

- ***VASOGENIC***: Problem in BBB
- ***CYTOTOXIC***: Problem in Sodium Potassium pump
- ***OSMOTIC***: Problem in blood osmolality
- ***INTERSTITIAL***: Problem in CSF brain Barrier

EDEMA

VASOGENIC EDEMA

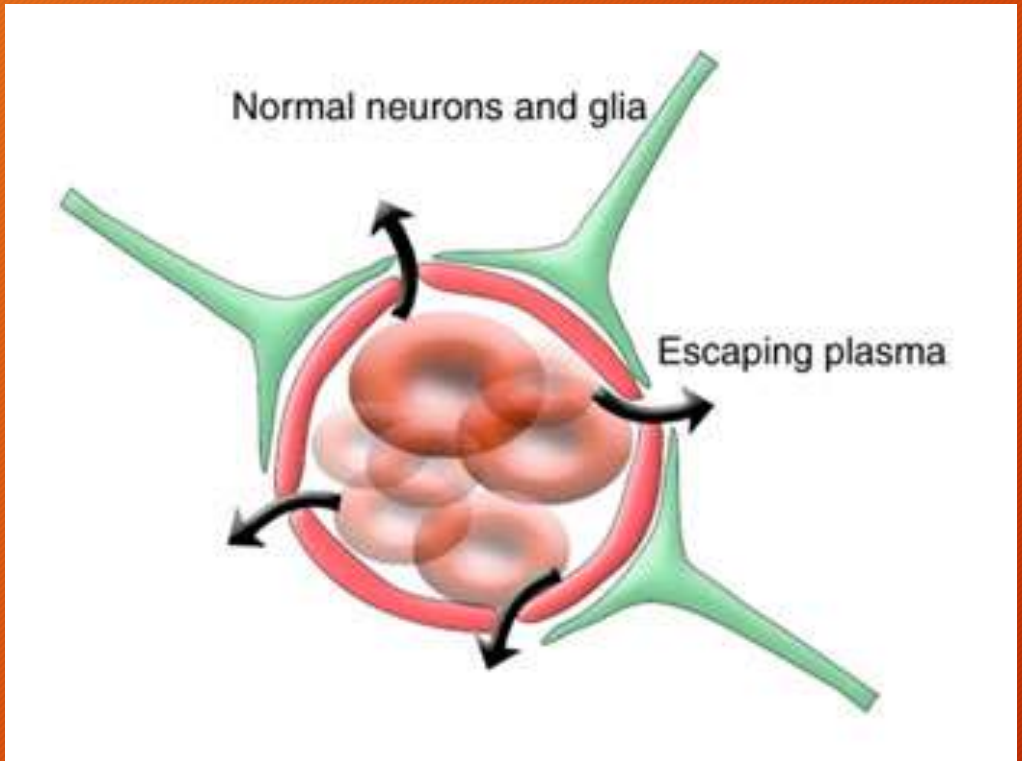
- It occurs due to a breakdown of the tight endothelial junctions which make up the blood–brain barrier (BBB).

- This allows normally excluded intravascular proteins and fluid to penetrate into cerebral parenchymal extracellular space.

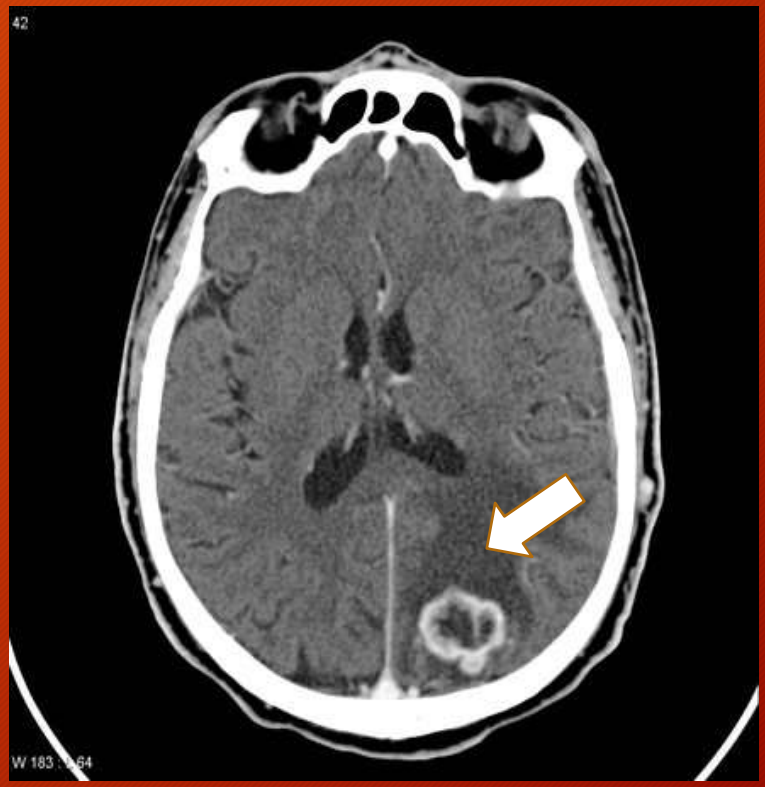
Vasogenic edema

- Once plasma constituents cross the BBB, the edema spreads; this may be quite fast and widespread.
- As water enters white matter it moves extracellularly along fiber tracts and can also affect the gray matter.

Vasogenic edema



CT with contrast: Metastatic lesion with surrounding edema (arrow).



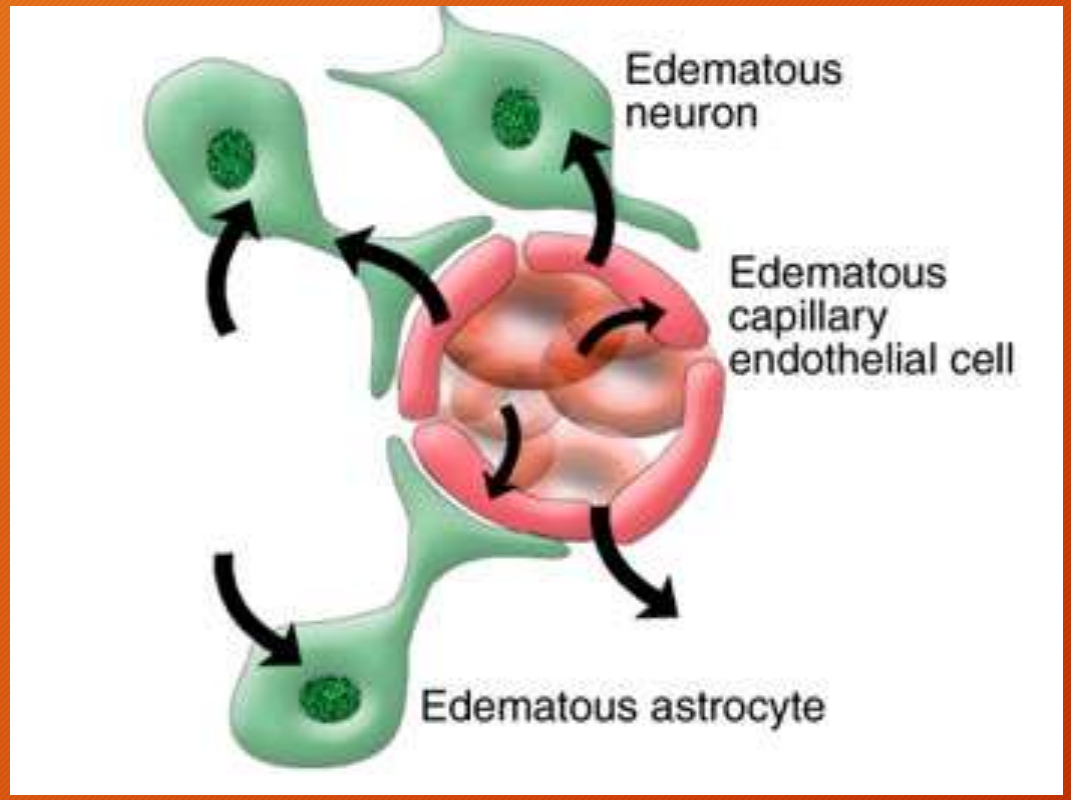
Cytotoxic edema

- In this type of edema the BBB remains intact.
- This edema is due to the derangement in cellular metabolism resulting in inadequate functioning of the sodium / potassium pump in the glial cell membrane.
- As a result there is cellular retention of sodium and water.

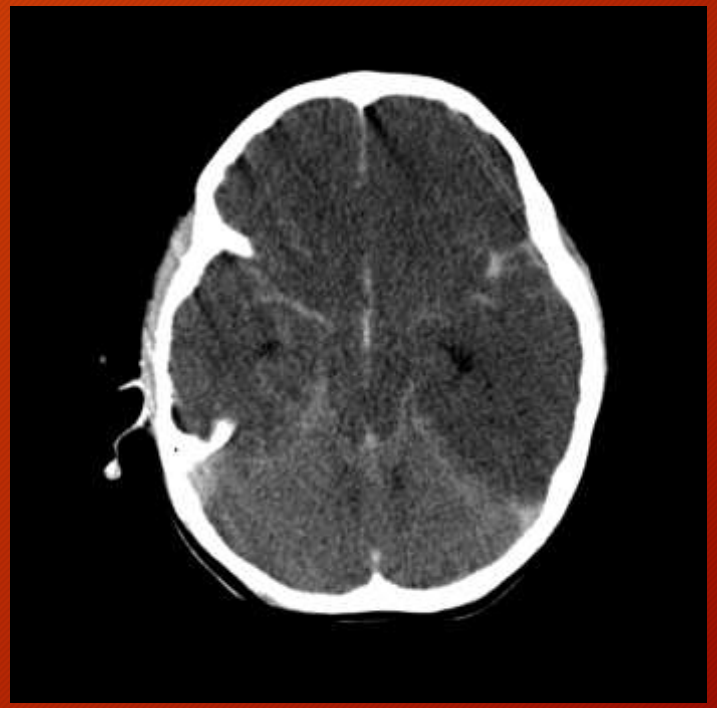
Cytotoxic edema

- Cytotoxic edema is seen with various intoxications (dinitrophenol, triethyltin, hexachlorophene, isoniazid), in Reye's syndrome, severe hypothermia, early ischemia, encephalopathy, early stroke or hypoxia, cardiac arrest, pseudotumor cerebri, and cerebral toxins.

Cytotoxic edema



CT without contrast showing edema of both territories supplied by the internal carotid arteries



Osmotic Edema

- Usually the osmolality of the brain is lower than that of the blood, so ECF flows from brain to the intravascular compartment
- If the osmolality of the blood decreases, so that of the brain becomes relatively higher, then ECF flows from the intravascular compartment to the brain.
- As a result of overhydration.

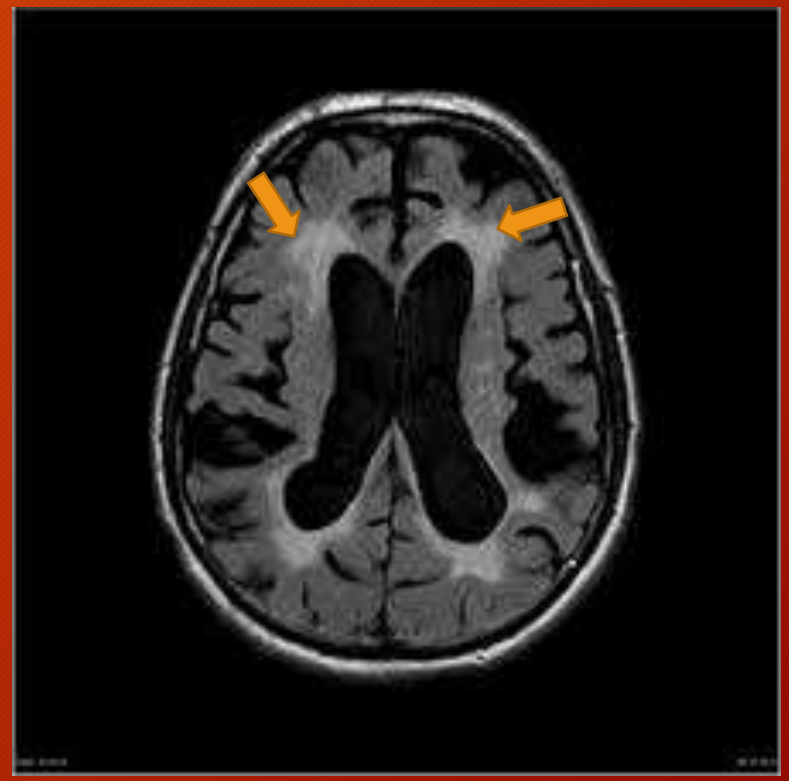
Osmotic Edema

When plasma is diluted by excessive water intake (or hyponatremia), syndrome of inappropriate antidiuretic hormone secretion (SIADH), hemodialysis, or rapid reduction of blood glucose in hyperosmolar hyperglycemic state (HHS), formerly hyperosmolar non-ketotic acidosis (HONK), the brain osmolality will then exceed the serum osmolality creating an abnormal pressure gradient down which water will flow into the brain causing edema

Interstitial Edema

- Here the problem is in the CSF Brain Barrier which is the ependymal lining of the ventricles. It occurs in hydrocephalus
- CSF seeps outside of the ventricle and remains close to it. It has no proteins in it.

MRI FLAIR sequence showing edema as periventricular hyperintense signal (arrow)



COMPLICATIONS OF HEAD INJURIES

- **EARLY**
  - HYPONATRAEMIA
  - INTRACRANIAL HEMORRHAGE/HEMATOMA
  - CSF LEAKS
  - EPILEPSY
- **LATE**
  - HYDROCEPHALUS
  - CHRONIC SUBDURAL HEMATOMA
  - EPILEPSY
  - POST TRAUMATIC SYNDROMES

EARLY COMPLICATIONS:
ELECTROLYTE DISTURBANCE: HYPONATRAEMIA

- *DUE TO LOW SODIUM LEVELS AS A CONSEQUENCE OF ADH SECRETION IN RESPOSE TO TRAUMA*
- *MAY LEAD TO CONFUSION NAUSEA AND LETHARGY. IF LEVEL BELOW 120 mmol/l LEADS TO SEIZURES/ IF BELOW 105 THEN STATUS EPILEPTICUS*
- *TREATED BY SLOW INFUSION OF NORMAL SALINE AT A RATE OF 5 mmol/hour OTHERWISE PONTINE MYLINOLYSIS DEVELOPS. IN STATUS YOU CAN INFUSE FASTER.*

EARLY COMPLICATIONS:
CEREBRO-SPINAL FLUID LEAKS

- *Requires a fracture and a dural tear*
- *Types*
  - *CSF Rhinorrhea if from the nose*
  - *CSF Otorrhea if from the ear*
- *Diagnosed and suspected by:*
  - *Leaking fluid confirmed by beta 2 transferrin*
  - *Presence of pneumocele*
  - *Development of meningitis*

CEREBRO-SPINAL FLUID LEAKS

- ***CSF RHINORRHEA***
  - *Occurs in 25% of patients with anterior basilar fractures*
  - *In 60% of cases occurs in the first week*
  - *May be missed due to swallowing*
  - *80% of cases stop spontaneously within 2 weeks*
  - *17% of cases develops infection*

CEREBRO-SPINAL FLUID LEAKS

- **CSF OTORRHEA**
  - *Occurs in 2% of patients with basilar temporal fractures*
  - *Leakage may be profuse*
  - *95% usually dries up in 2 weeks*
  - *There is 4% risk of infection*

CEREBRO-SPINAL FLUID LEAKS

- **MANAGEMENT**
  - *Admit to hospital*
  - *Prevent examination of nose or ear for fear of introducing infection*
  - *Prevent poking of nose and ear cotton buds.*
  - *Give broad spectrum antibiotics*
  - *External sterile gauze dressing*
  - *Wait for closure*

CEREBRO-SPINAL FLUID LEAKS

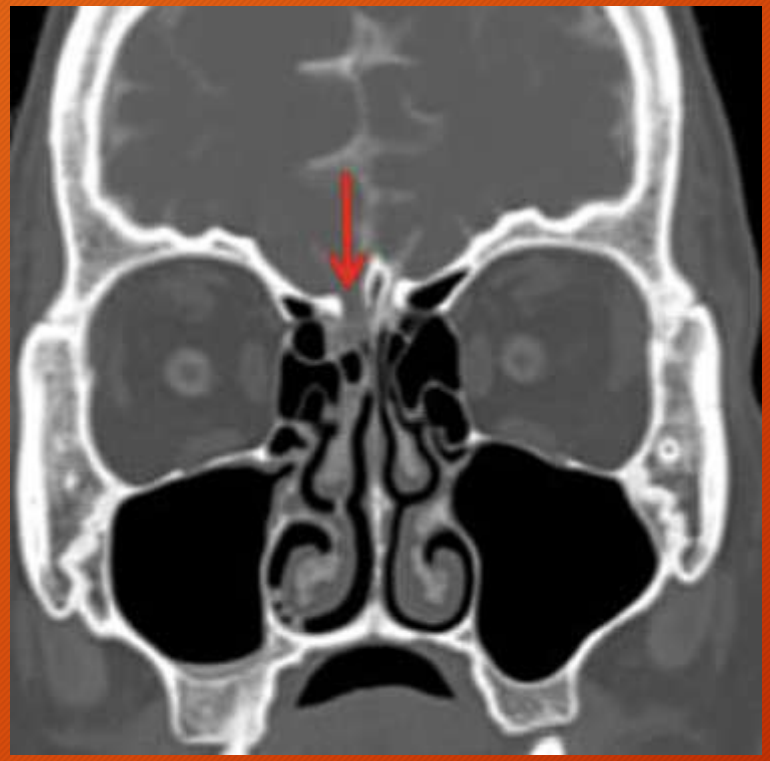
- ***MANAGEMENT***
  - *If it does not close in 2 weeks.*
  - *Insert a external lumbar CSF drainage system.*
  - *Continue antibiotics*
  - *Wait for 2 weeks*
  - *On day 29, challenge the fistula by closing the drainage tube for 24 hours and see what happens.*

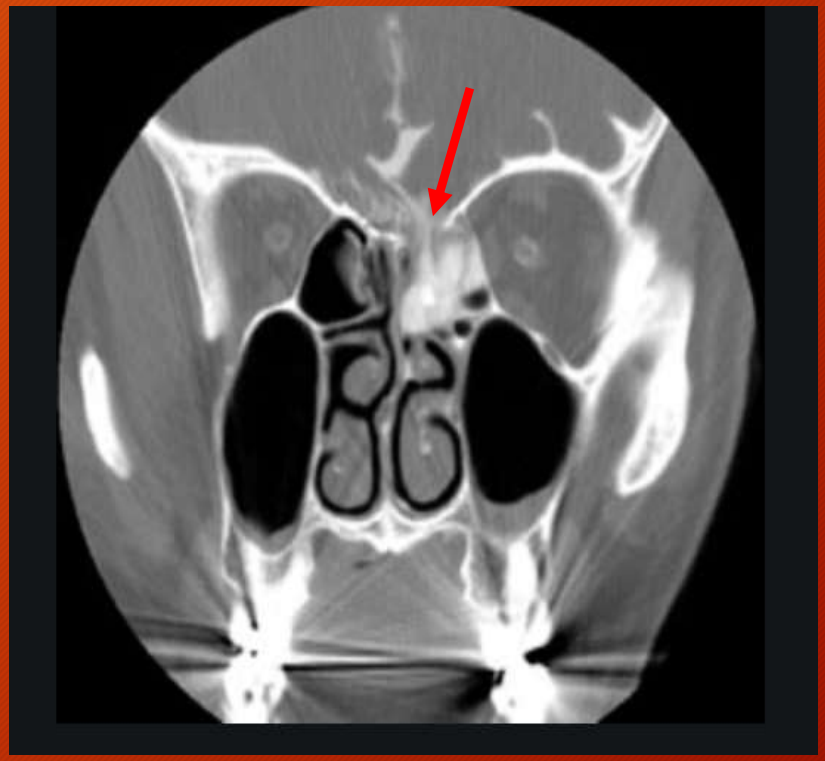
CEREBRO-SPINAL FLUID LEAKS

- ***MANAGEMENT***
  - *If after closing the drain (challenging the fistula), no CSF leaks, then all is good and the fistula had closed. The patient could be discharged.*
  - *If CSF continues to leak, then we need to do cisternography to locate the site of leak prior to surgery which is craniotomy and duroplasty*
  - *We inject an Iodine dye (Metrizamide) into the CSF via the catheter and then we perform a coronal and Sagittal high resolution CT.*

CEREBRO-SPINAL FLUID LEAKS

High resolution coronal CT with Metrizamide showing the site of the CSF leak.





EARLY COMPLICATIONS
INTRACRANIAL HEMATOMAS

There are 4 types:

1. *Extradural hematoma*
2. *Subdural hematoma*
3. *Subarachnoid hemorrhage*
4. *Intracerebral hematoma*

EXTRADURAL HEMATOMA

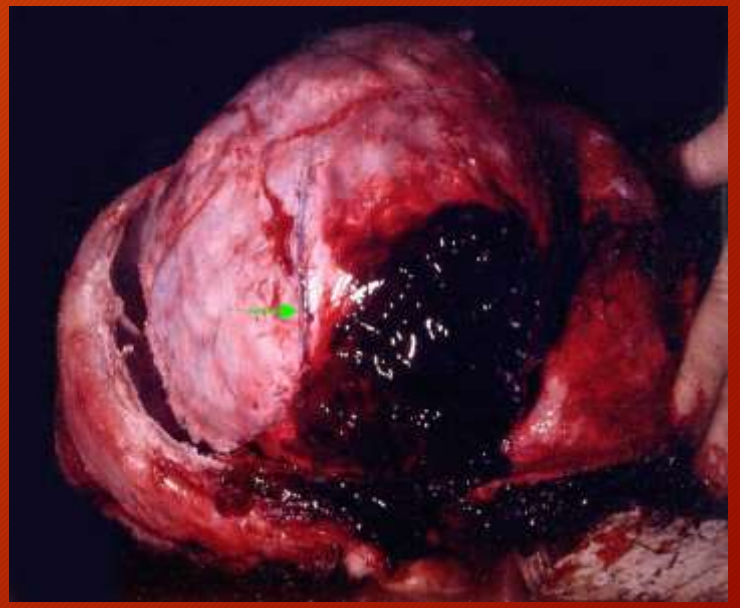
- *Between dura and skull bone*
- *Arterial mainly MMA*
- *In adults 90% associated with fractures*
- *Only 25% of children have fractures*
- *Mostly within 6 hours of the injury (stem of MMA)*
- *6-24 hours (from anterior branch of MMA)*
- *24-36 hours (from posterior branch of MMA)*

EXTRADURAL HEMATOMA

Surface anatomy of MMA.

Linear fractures of the skull.

Post mortem of an EDH



EXTRADURAL HEMATOMA

Two clinical presentations:

1. *Classical 25%*
2. *Non classical 75%*

EXTRADURAL HEMATOMA

CLASSICAL PRESENTATION

Trauma → LOC (concussion) → Wake up
(lucid interval) → LOC (herniation)
Leads to ↑ ICP and neurological damage

Investigations

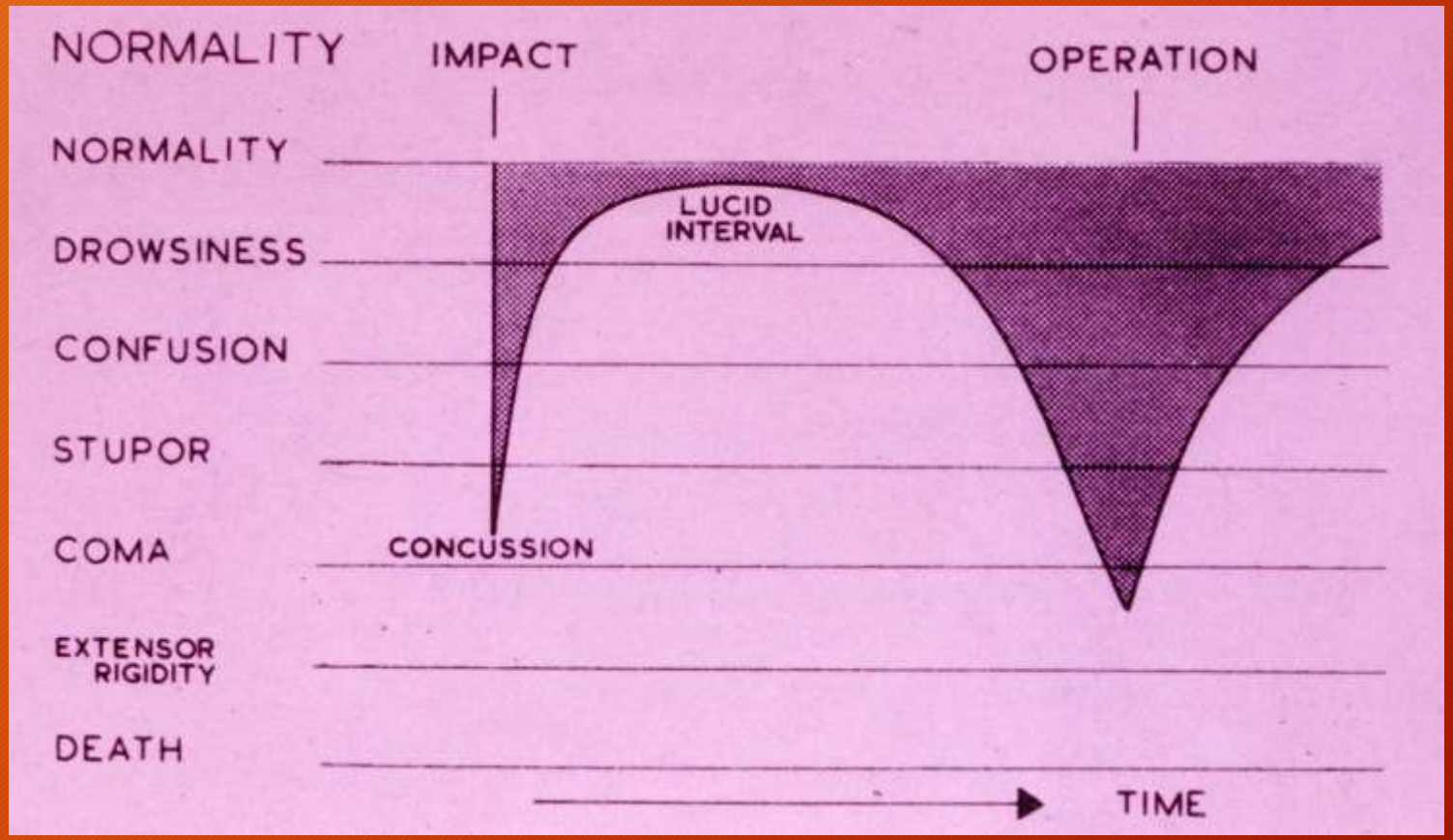
- *IF THERE IS TIME DO CT*
- *IF NO TIME DO SURGERY*

Treatment

- *BURR-HOLES*
- *CRANIOTOMY OR*
- *CRANIECTOMY*

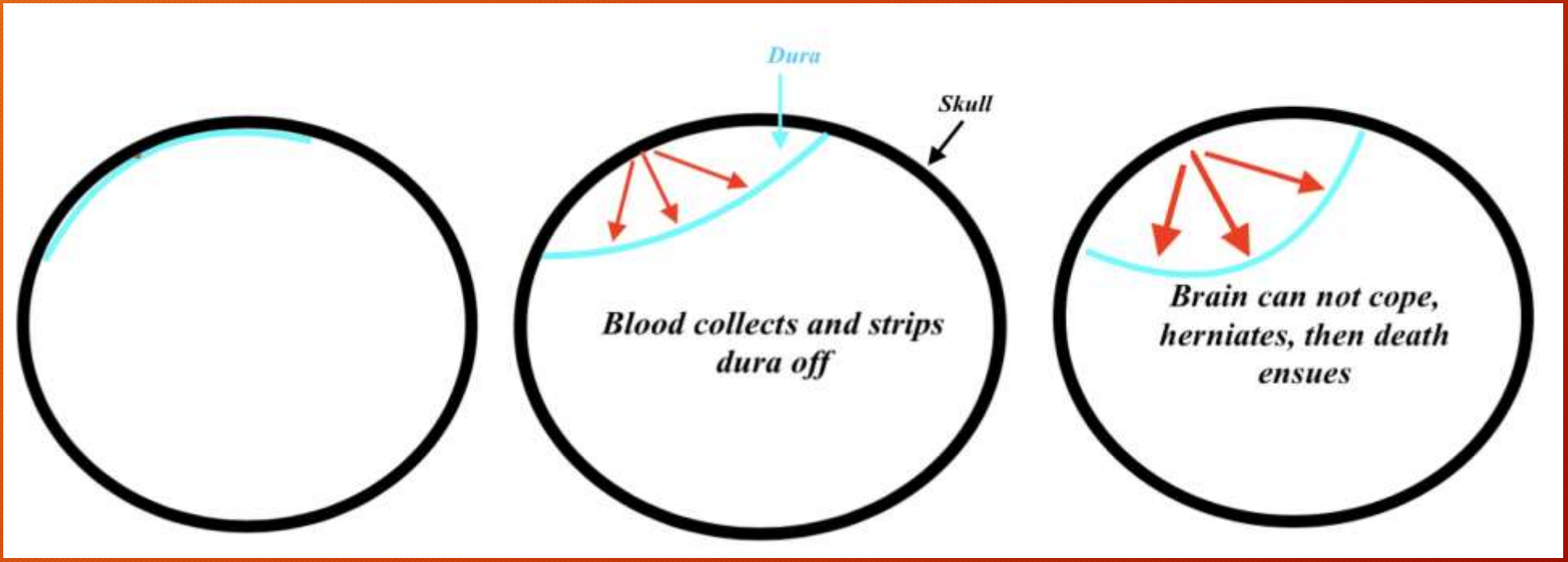
EXTRADURAL HEMATOMA

CLASSICAL PRESENTATION



EXTRADURAL HEMATOMA

Why is the delay in presentation? It is to give time for the collecting blood to strip the dura off the inside of the skull



EXTRADURAL HEMATOMA

- The first loss of consciousness was due to the concussion of the brain
- Concussion does not leave structural changes in the brain
- The patients wakes up and enjoys a “lucid interval”
- Then as Intra cranial pressure (ICP) rises and herniation occurs, the patient loses consciousness

EXTRADURAL HEMATOMA

NON-CLASSICAL PRESENTATION 75%

1. *Unconscious, remains unconscious, CT shows EDH*
2. *Conscious, remains conscious, CT shows EDH*
3. *Conscious, CT shows nothing, admitted, deteriorates, CT shows EDH*

EXTRADURAL HEMATOMA
NON CLASSICAL PRESENTATION

Sport › Boxing › Boxing

Scott Westgarth dead: Boxer dies after being rushed to hospital when he collapsed after light heavyweight fight victory

Westgarth was taken ill shortly after his fight against Dec Spelman at Doncaster Dome on Saturday night and it was announced he had died today

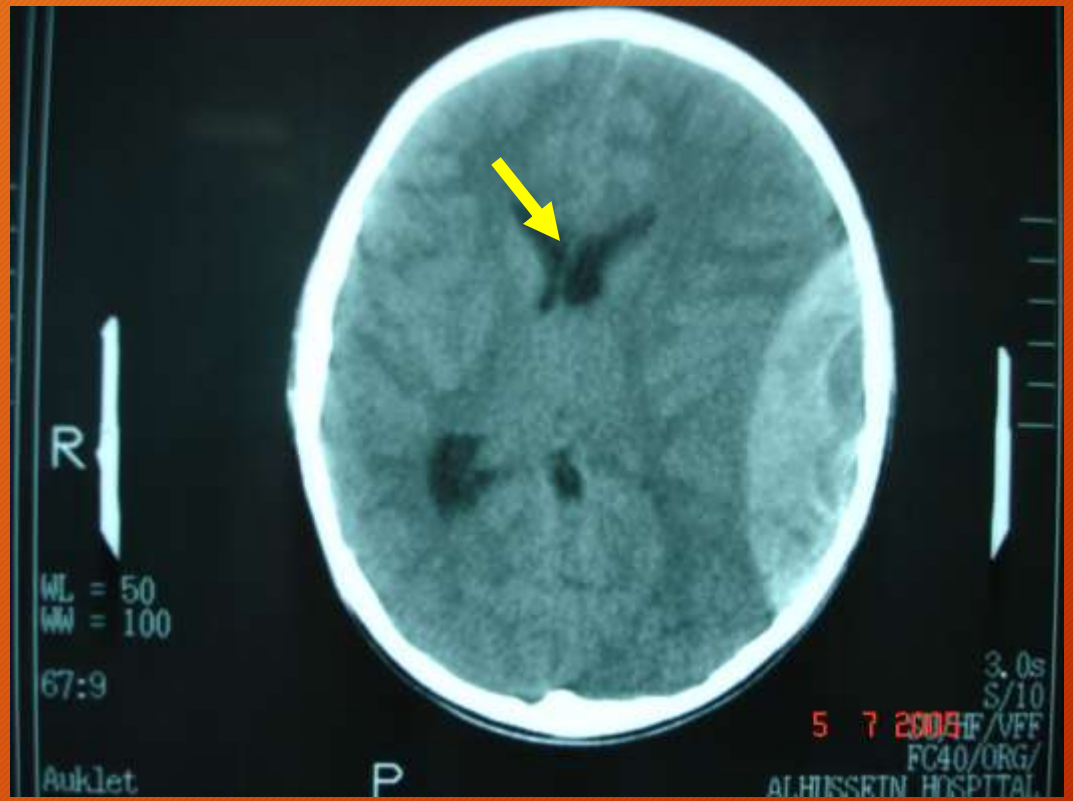
SHARE 11 COMMENTS By Martin Domin 14:05, 27 FEB 2018 UPDATED 14:08, 27 FEB 2018 SPORT

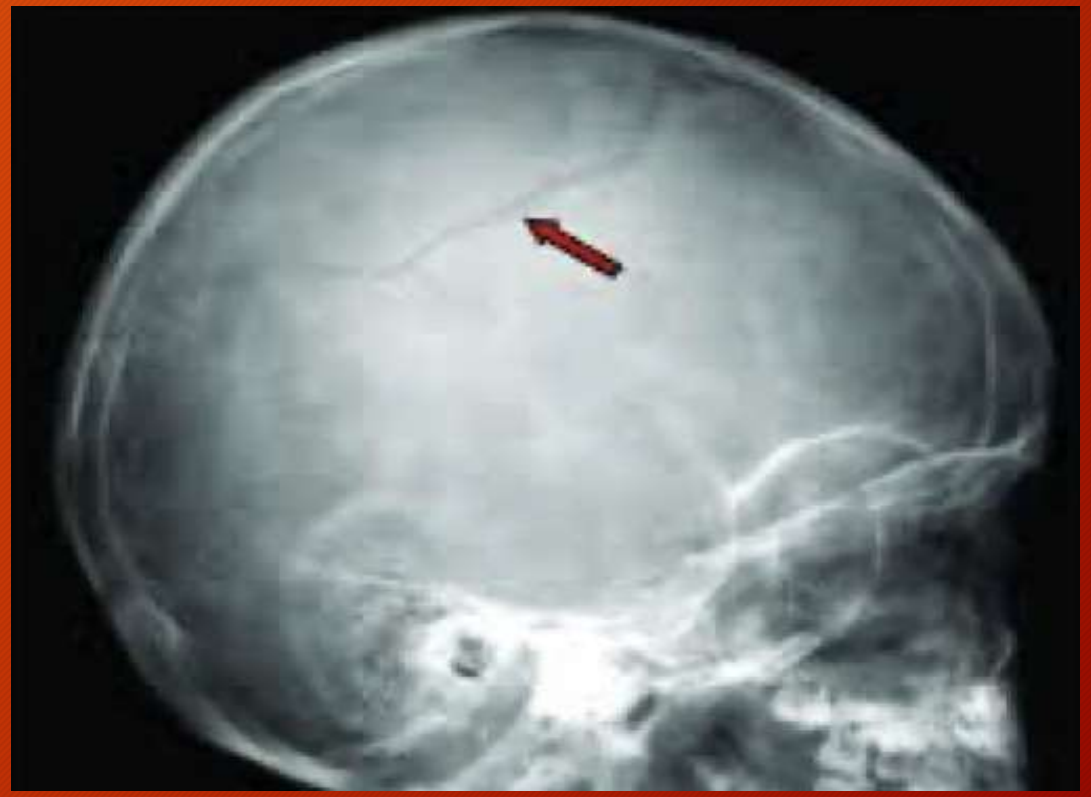
He was seen with his head in his hands as Spelman was interviewed, an image which sparked concern on social media.

Afterwards, Scott, 31, who combined boxing with jobs as a chef, and personal trainer, spoke to doctors in the dressing room who sent him to hospital in an ambulance.

He began to feel sick on the journey, and it is suspected he had a bleed on the brain. Boxing chiefs were informed of his death in Sheffield's Royal Hallamshire Hospital on Sunday morning.

EXTRADURAL HEMATOMA





ACUTE EXTRADURAL HEMATOMA AND SKULL FRACTIURE

EXTRADURAL HEMATOMA

Craniotomy and evacuation of extradural hematoma

Craniotomy for Evacuation of Extra Dural hematoma

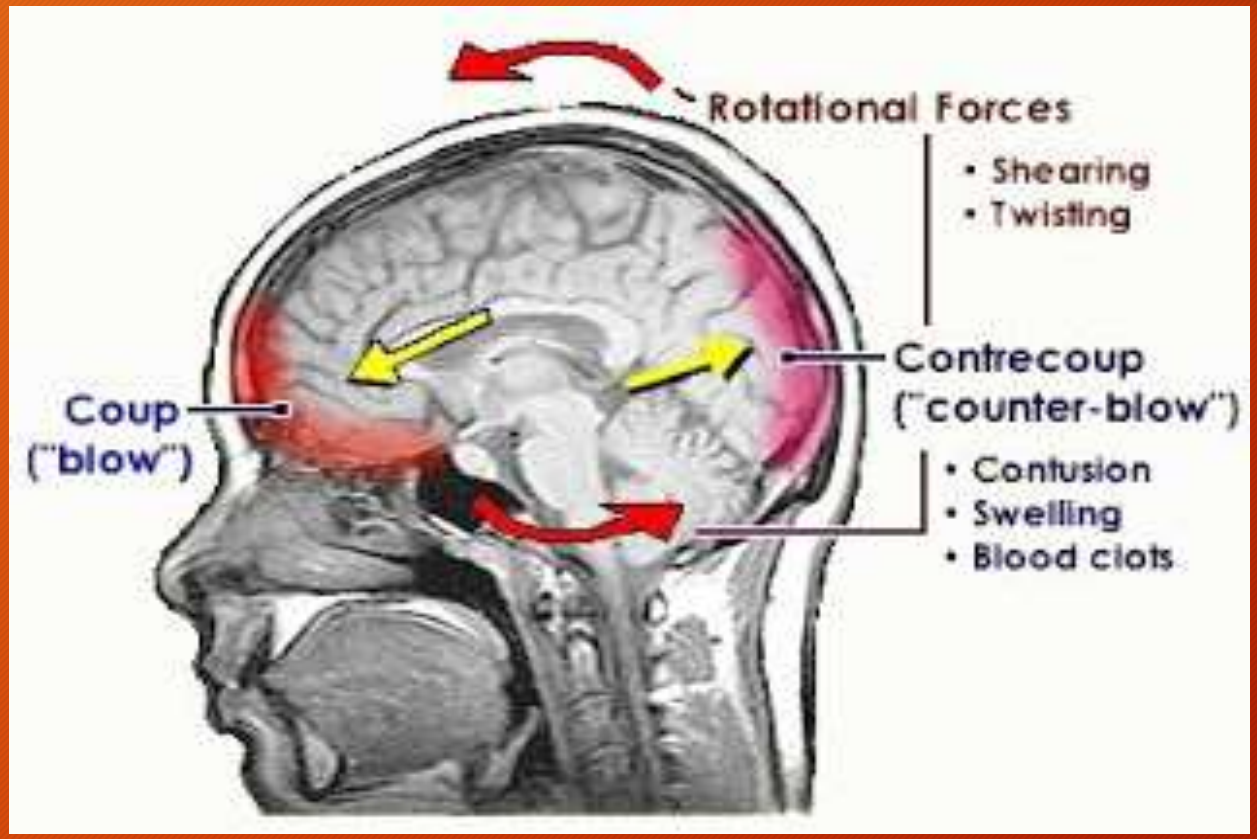
ACUTE SUBDURAL HEMATOMA

- *Between brain and dura*
- *Originates mainly from bridging veins and brain vessels*
- *If small may become chronic*
- ***Usually part of severe brain laceration and injury***

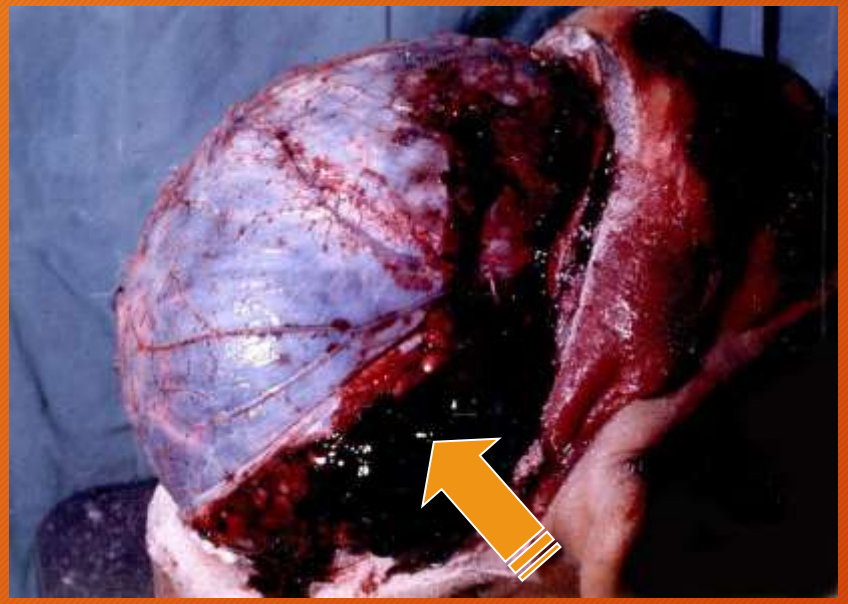
Due to direct trauma, acceleration deceleration

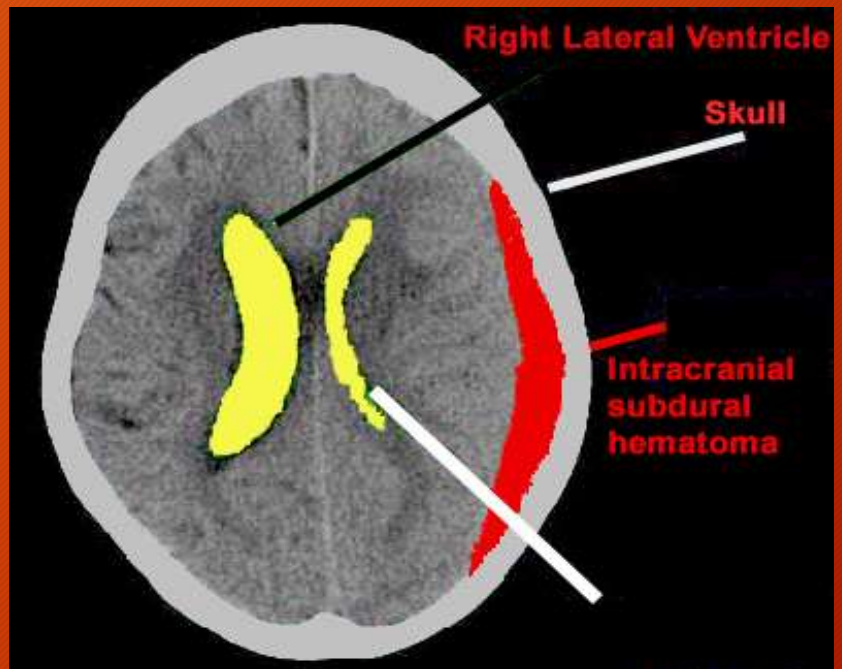
- *Presents like a non-classical EDH but the picture is over shadowed by the TBI*
- *Confirmed by a non contrasted CT*
- *Treatment*
  - *That of the head injury*
  - *Evacuation*

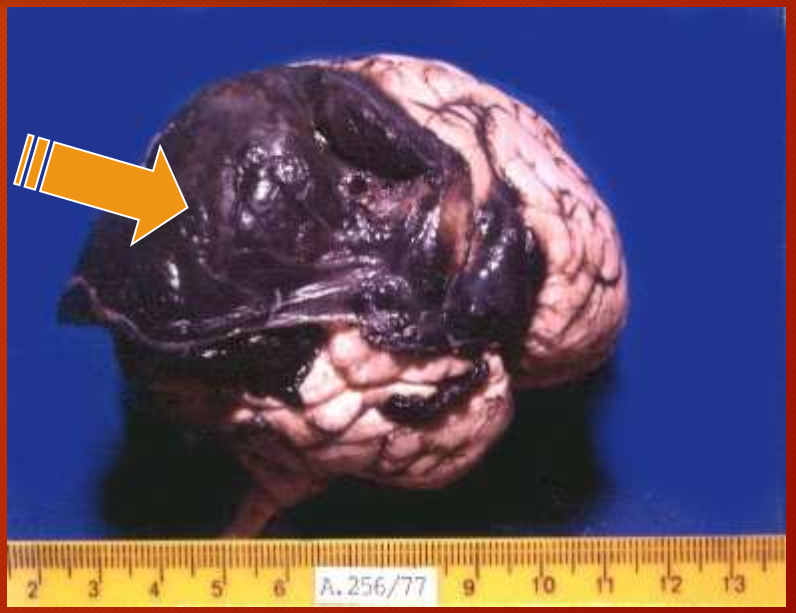
ACUTE SUBDURAL HEMATOMA



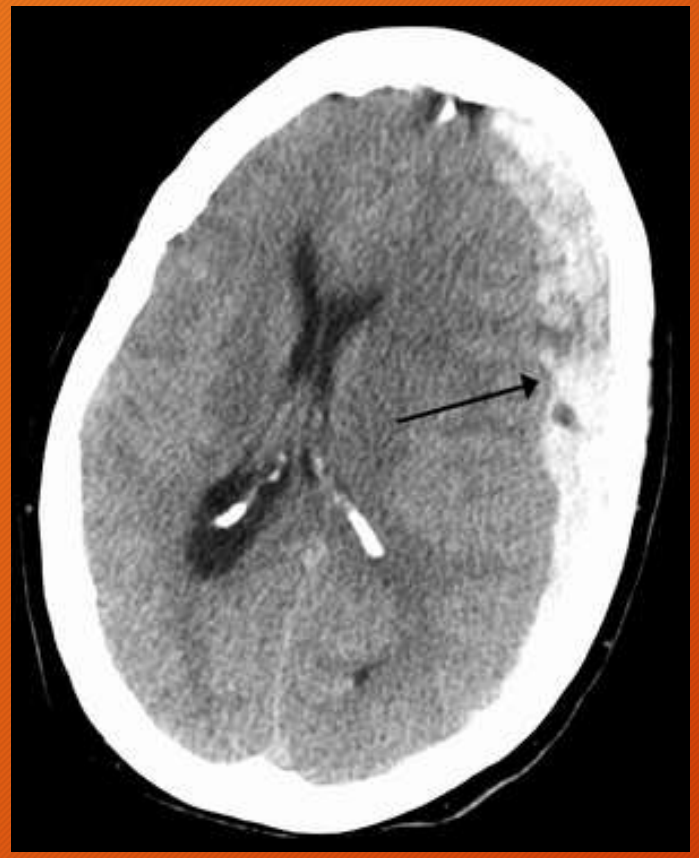
ACUTE SUBDURAL HEMATOMA

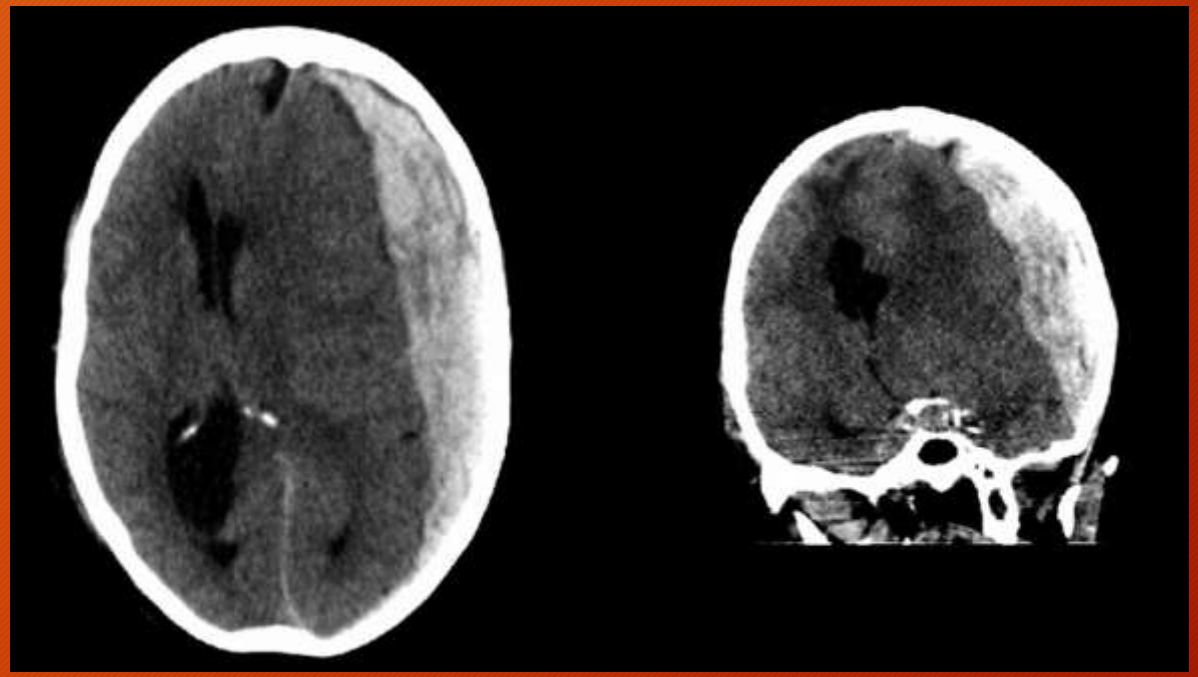






ACUTE SUBDURAL HEMATOMA





Non contrasted CT showing the hyperdense semilunar or cresentic appearance of an acute subdural hematoma

ACUTE SUBDURAL HEMATOMA

Craniotomy and evacuation of acute subdural hematoma

SO WHAT ARE THE DIFFERENCES?

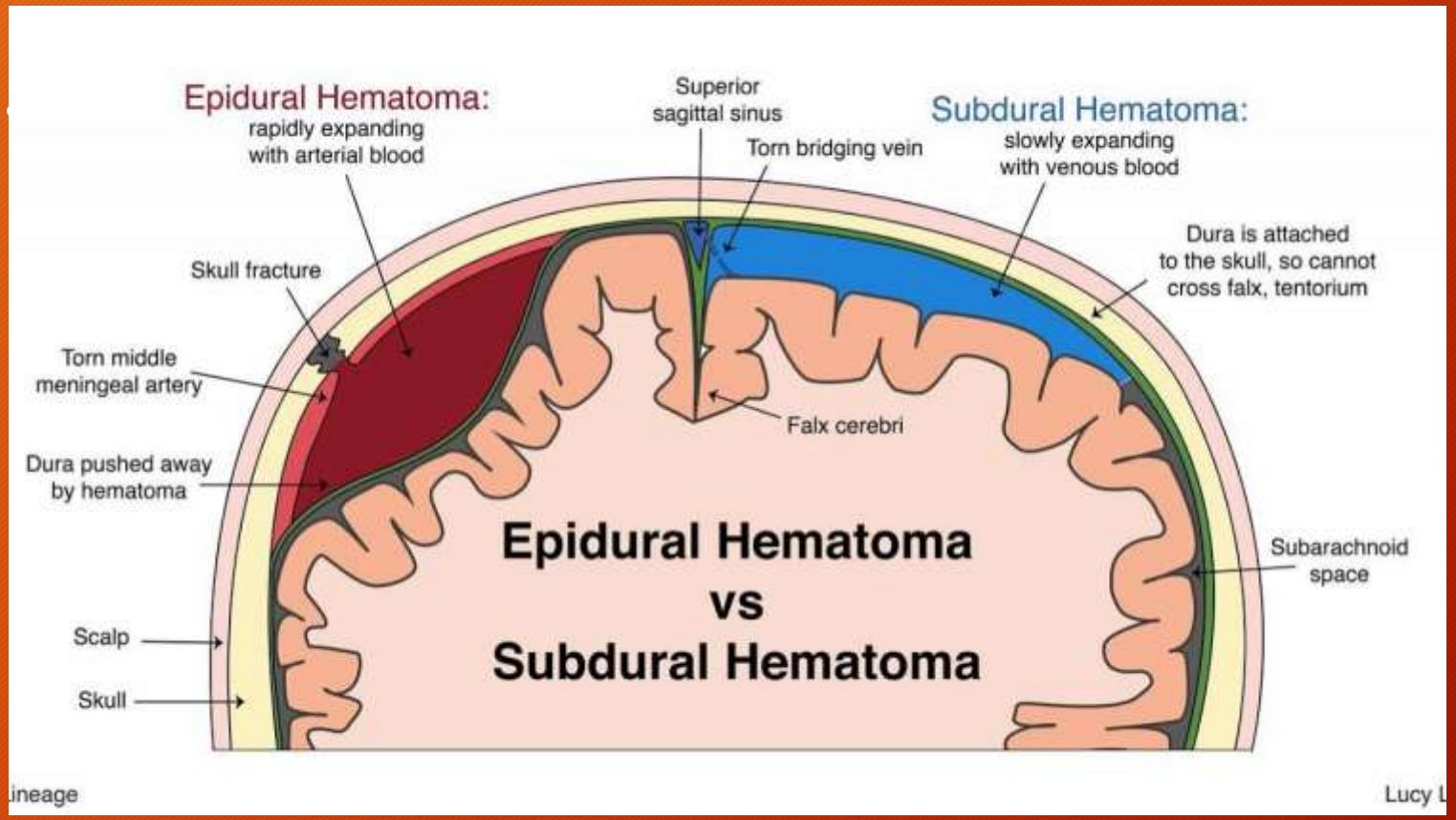
EXTRADURAL

- BETWEEN SKULL AND DURA
- ARTERIAL BLOOD
- FROM MIDDLE MENINGIAL ARTERY
- LOCALIZED
- **USUALLY NO BRAIN INJURY**
- OCCASIONALLY WITH LUCID INTERVAL (25%)
- 25% OF CASES HAVE NO FRACTURES.

SUBDURAL

- BETWEEN DURA AND BRAIN
- VENOUS BLOOD
- FROM BRIDGING VEINS
- WIDE SPREAD
- **USUALLY SEVERE BRAIN INJURY**
- NO LUCID INTERVAL

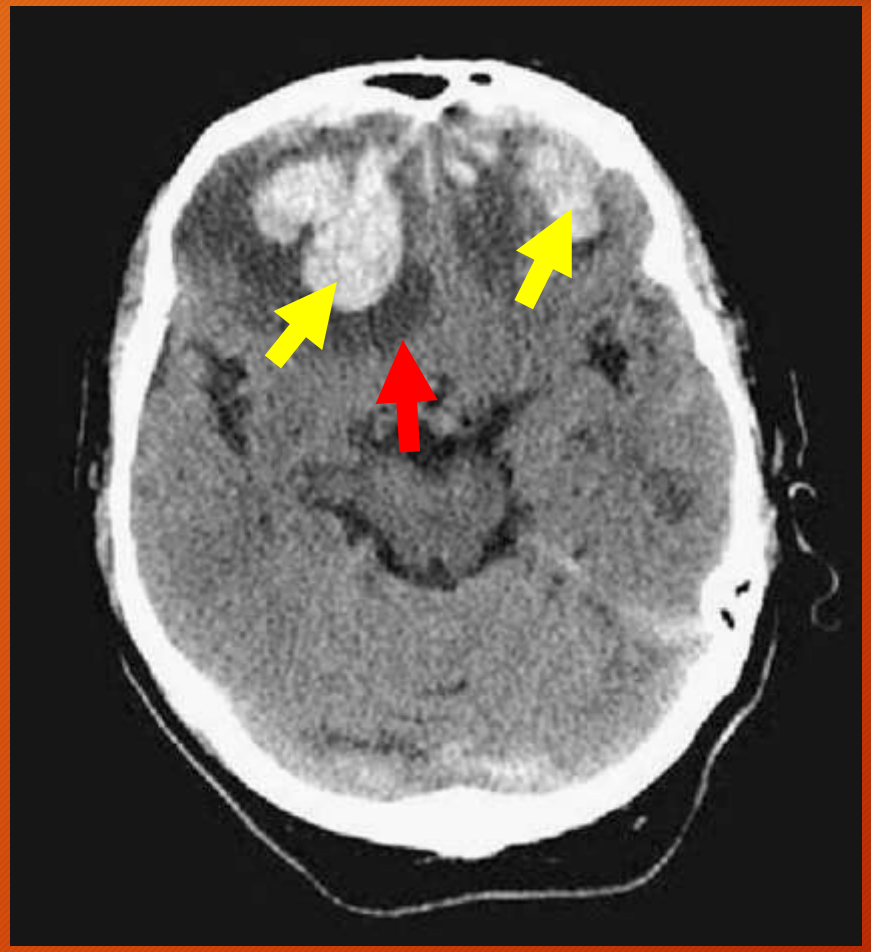
EXTRA Vs SUBDURAL HEMATOMA



INTRACEREBRAL HEMATOMA

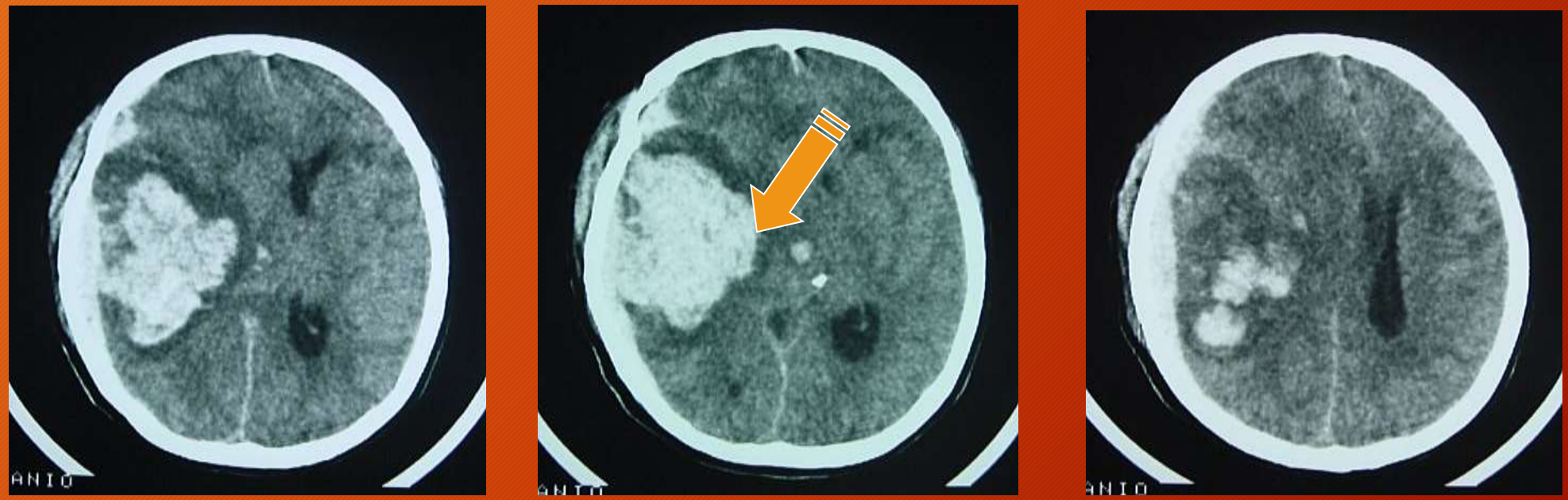
- *Usually due to direct trauma or acceleration deceleration injury*
- *Mainly at poles of lobes and under surface of brain*
- *Could have been a hemorrhagic contusion*
- *Usually part of severe head injury*
- *Diagnosis is by non contrasted CT*
- *Treatment:*
  - *That of the head Injury*
  - *Craniotomy and evacuation if the cause of continued deterioration.*

INTRACEREBRAL HEMATOMA



Non contrasted brain CT showing bifrontal contusions changing to hematomas by increase of the blood inside them (yellow arrows). Note the surrounding edema (red arrow).

INTRACEREBRAL HEMATOMA

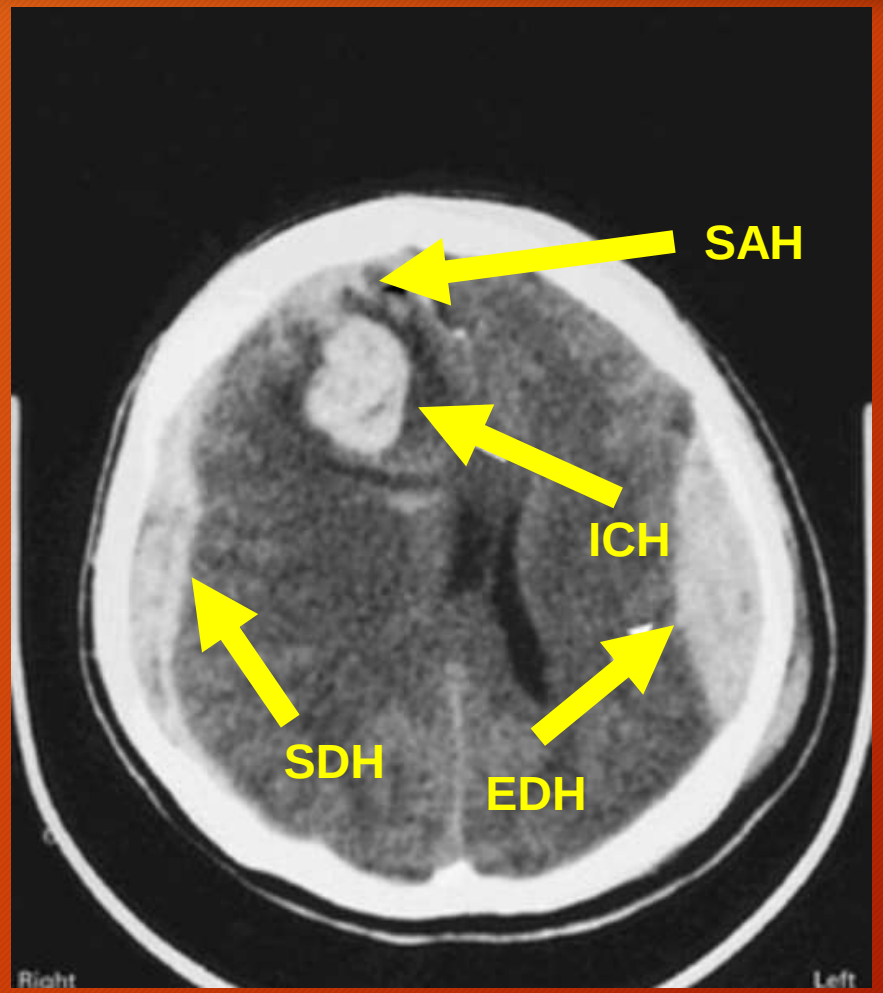


Non contrasted brain CT showing traumatic intra cerebral hematoma

SUBARACHNOID HEMORRHAGE

- *A very common occurrence*
- *May lead to hydrocephalus by blocking the arachnoid granulations and prevent absorption.*
- *Could be seen on CT as a separate entity or accompanying other injuries.*
- *Treatment:*
  - *Analgesia for headache*
  - *Treat the hydrocephalus if it develops*

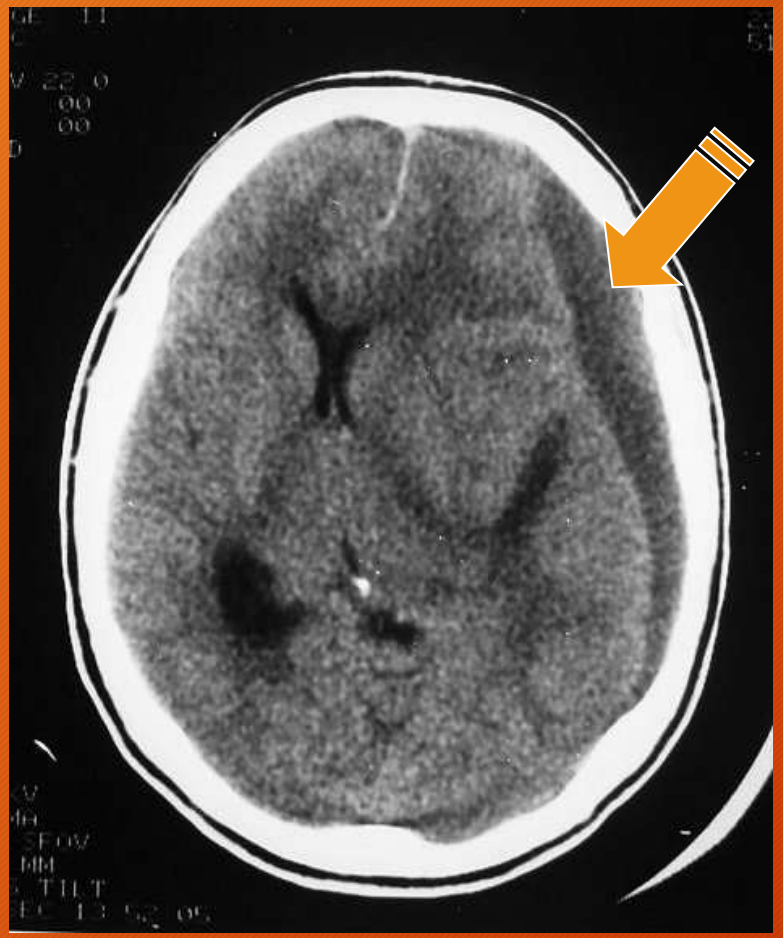
THE 4 TYPES OF INTRACRANIAL HEMORRHAGE IN ONE PATIENT



LATE COMPLICATIONS:
CHRONIC SUBDURAL HEMATOMA

- *There is usually no history of trauma, or a minor trauma 6 weeks earlier.*
- *The patient is usually an elderly person mostly taking anticoagulants*
- *Usually the complaint starts by headache then memory disturbances, unsteadiness. Later neurological deficits may appear and occasionally incontinence of urine.*
- *The reason for delay in presentation is for the clot to enlarge by absorbing CSF into the organized clot which is surrounded by a semipermeable membrane*

CHRONIC SUBDURAL HEMATOMA

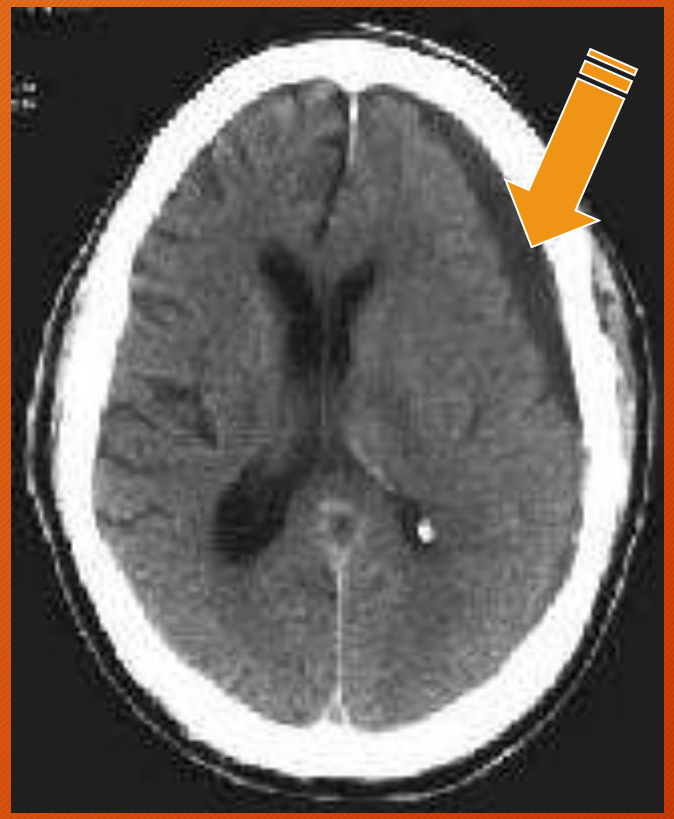


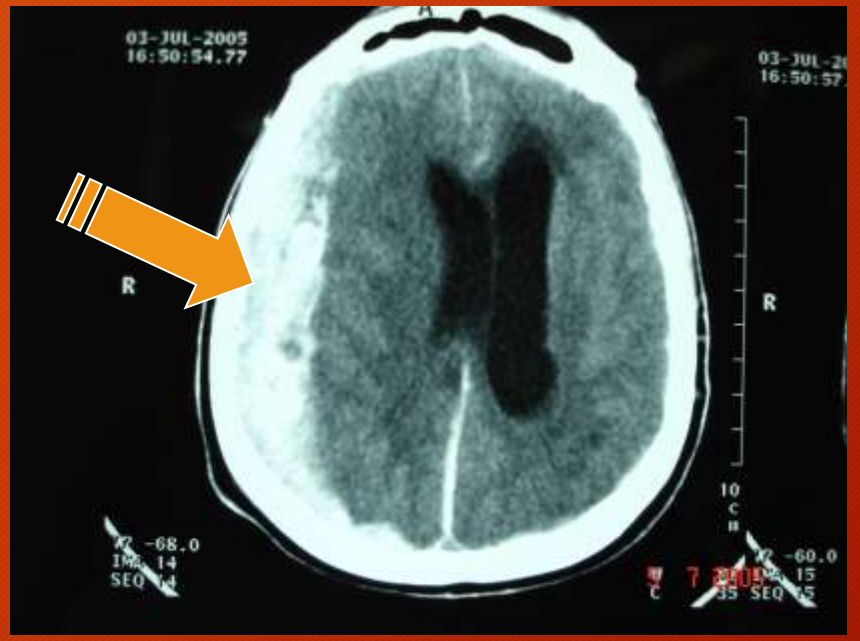
Non contrasted CT showing the semilunar hypodense appearance of the chronic subdural hematoma. Note the ventricular compression, the midline shift and the effacement of sulci; all indicative of high ICP.

CHRONIC SUBDURAL HEMATOMA

- *The hematoma is fluid in nature*
- *The color of the fluid is brown like used motor oil.*
- *Treatment consists of steroids if the hematoma is thin and the symptoms are mild, or burr hole evacuation.*
- *Usually two burr holes are done and the subdural space washed with saline. A catheter may be left for drainage for 48 hours.*

ACUTE AND CHRONIC SUBDURAL HEMATOMA

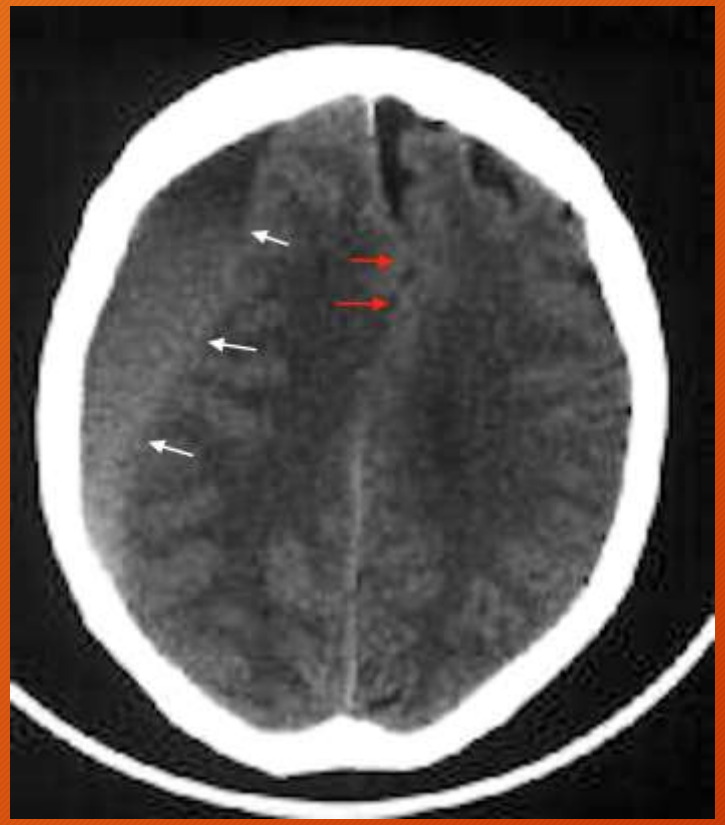




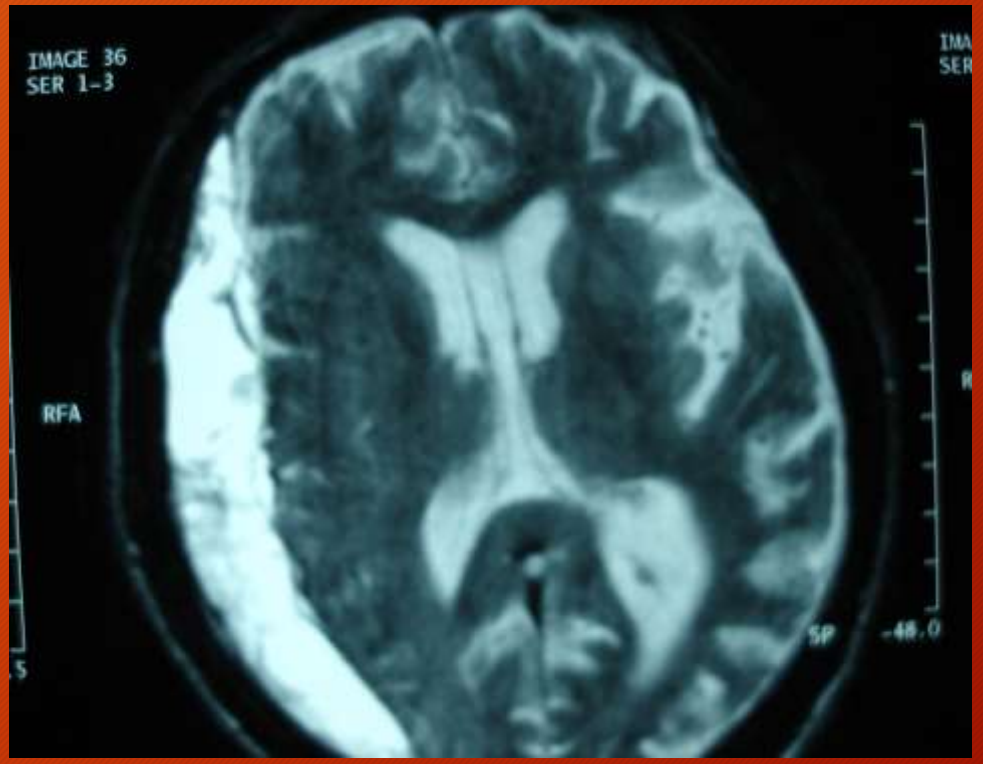
CHRONIC SUBDURAL HEMATOMA

ACUTE SUB DURAL HEMATOMA

CHRONIC SUBDURAL HEMATOMA

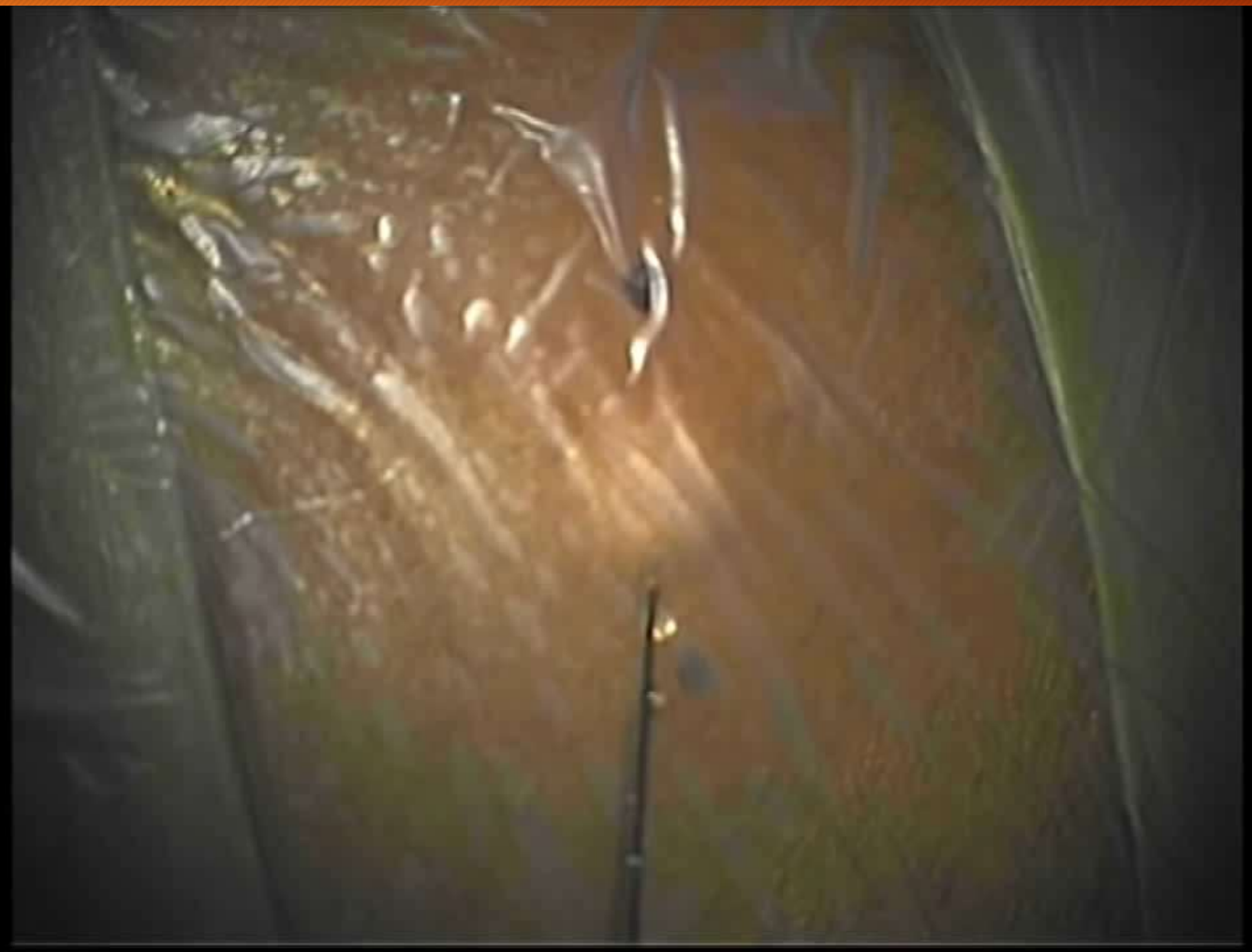


CT



MRI

CHRONIC SUBDURAL HEMATOMA

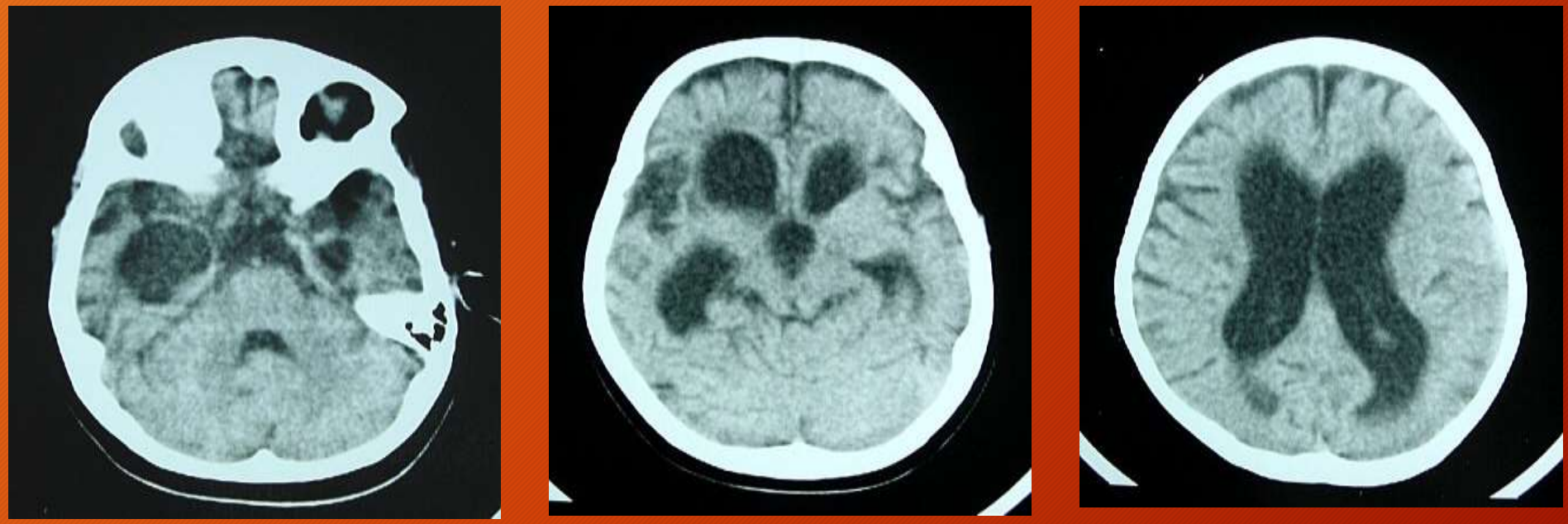


Evacuation of chronic subdural hematoma using two burr holes under local anesthesia

LATE COMPLICATIONS:
HYDROCEPHALUS

- *DUE TO BLOOD IN CSF*
- *USUALLY OF COMMUNICATING TYPE*
- *SHOULD BE SUSPECTED IN DELAYED RECOVERY*
- *MAY LEAD TO:*
  - *HEADACHE*
  - *DETERIORATION IN MENTAL FUNCTION*
  - *ATAXIA*
  - *INCONTINENCE*
- *MAY REQUIRE SHUNTING*

HYDROCEPHALUS



LATE COMPLICATIONS:
EPILEPSY

- *DEPENDS ON LOCATION OF INJURY, EXTENT OF INJURY AND AGE*
- *MAY LEAD TO HYPOXIA AND ↑ ICP*
- *COULD BE PREDICTED AND SCORED*
- *TREATED BY*
  - ❑ *CARBAMAZEPINE (TEGRETOL)*
  - ❑ *PHENYTOIN (EPANUTIN)*
  - ❑ *PHENOBARBITONE*

EPILEPSY

TWO CATEGORIES

- ***EARLY***
  - *WITHIN FIRST WEEK OF INJURY*
  - *5% OF CASES*
  - *10% IN CHILDREN BELOW 5 YEARS*

- ***LATE***
  - *AFTER FIRST WEEK OF INJURY*
  - *5% OF CASES*
  - *50% DEVELOP DURING FIRST YEAR*

LATE COMPLICATIONS:
POST CONCUSSION SYNDROME

- *COLLECTION OF SYMPTOMS DUE TO MINOR HEAD TRAUMA*
- *CONTROVERSIAL WHETHER ORGANIC OR PSYCHOLOGICAL*
- *SYMPTOMS:*
  - *SOMATIC:*
    - *HEADACHE*
    - *DIZINESS*
    - *VISUAL DISTURBANCES*
    - *HEARING PROBLEMS*
    - *BALANCE DIFFICULTIES*
  - *COGNITIVE:*
    - *CONCENTRATION DIFFICULTY*
    - *DEMENTIA*

POST CONCUSSION SYNDROME

- *PSYCHOLOGICAL:*
  - *EASY FATIGABILITY*
  - *LOSS OF LIBIDO*
  - *EMOTIONAL DISTURBANCES*
  - *PERSONALITY CHANGES*
  - *INSOMINIA*
  - *PHOTOPHOBIA*

- *TREATMENT*
  REASSURANCE
  - *SUPPORT*

FURTHER READING

- **Neurosurgery Made Easy** by Walid Maani available at Amazon.com in paper and kindle format and at Lulu.com in pdf format
- **Introduction to Neuroimaging** by Walid Maani available at Amazon.com in paper and kindle format and at Lulu.com in pdf format