

# HEAD INJURIES (2)

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# ***SECONDARY EVENTS***

- HYPOXIA
- ISCHEMIA
- EDEMA

# ***SECONDARY EVENTS***

## **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.

# ***SECONDARY EVENTS***

## **ISCHAEMIA**

Poor tissue perfusion with impaired cellular metabolism. This is usually due to blood loss.

Hypovolemic shock in neurosurgery is rare in adults except with severe scalp laceration or associated other system injury.

However; it could happen in infants in subgaleal hematoma or subdural hematoma due to the small volume of their blood

# ***SECONDARY EVENTS***

## **BRAIN EDEMA**

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

# ***SECONDARY EVENTS***

## **VASOGENIC EDEMA**

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

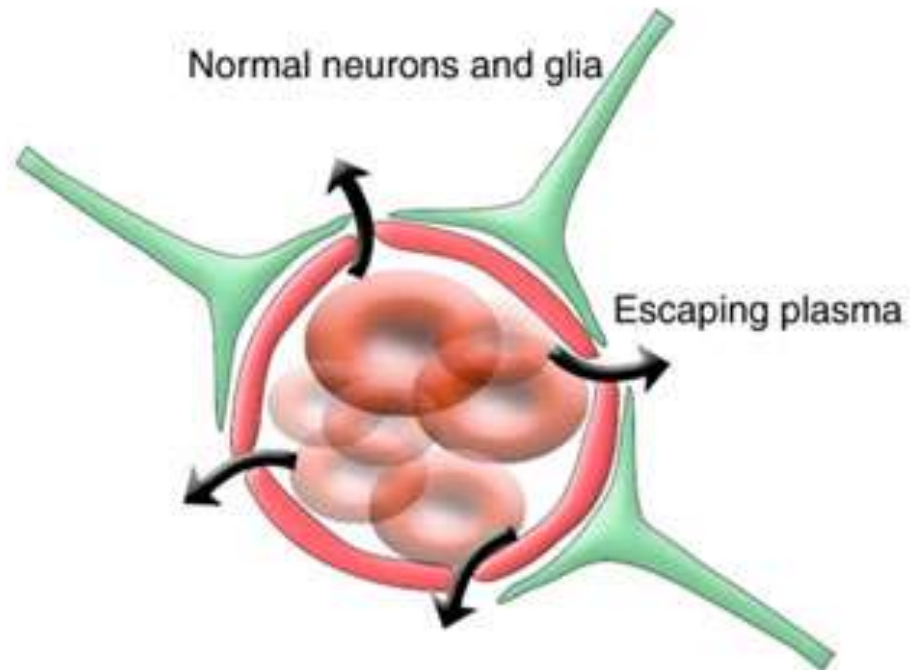
This allows normally excluded intravascular proteins and fluid to penetrate into cerebral parenchymal extracellular space.

Once plasma constituents cross the BBB, the edema spreads; this may be quite fast and widespread.

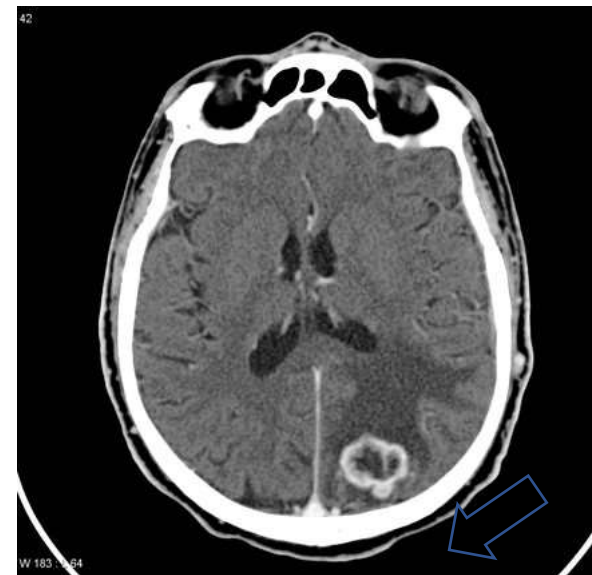
As water enters white matter it moves extracellularly along fiber tracts and can also affect the gray matter.

# ***SECONDARY EVENTS***

## ***VASOGENIC EDEMA***



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



# ***SECONDARY EVENTS***

## **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.

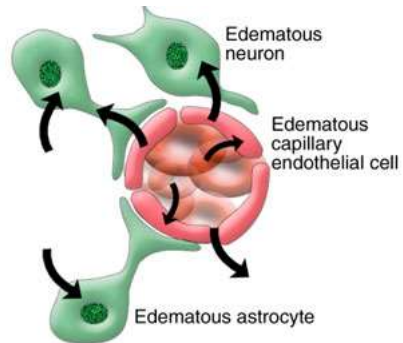
# ***SECONDARY EVENTS***

## **CYTOTOXIC EDEMA**

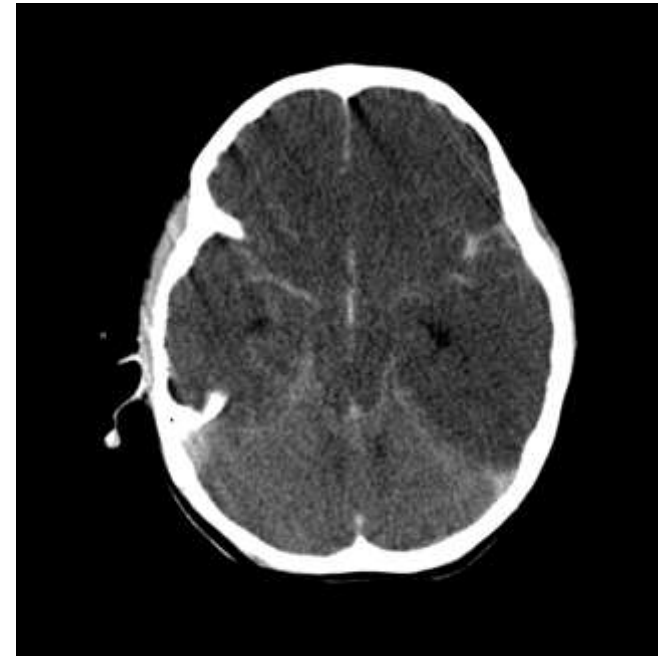
Cytotoxic edema is seen with various intoxications (dinitrophenol, triethylin, hexachlorophene, isoniazid), in Reye's syndrome, severe hypothermia, early ischemia, encephalopathy, early stroke or hypoxia, cardiac arrest, pseudotumor cerebri, and cerebral toxins.

# ***SECONDARY EVENTS***

## **CYTOTOXIC EDEMA**



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



# ***SECONDARY EVENTS***

## **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.

# ***SECONDARY EVENTS***

## **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

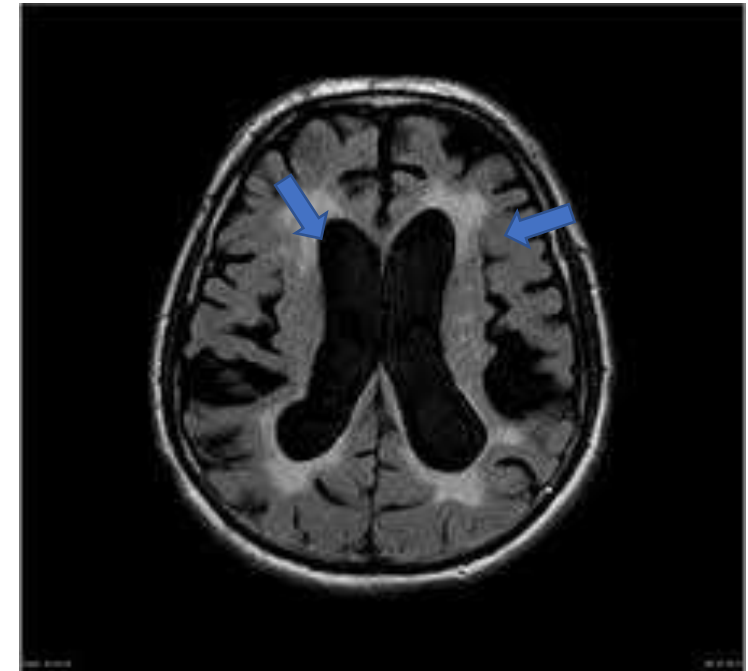
# ***SECONDARY EVENTS***

## **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 response to trauma

May lead to confusion, nausea and lethargy. If the levels drop to 120 mmol/l it will lead to seizures, and if below 105 mmol/l to status epilepticus.

Treated by slow infusion of normal saline or occasionally hypertonic saline depending on the sodium level, the patient age and weight, otherwise; pontine myelinolysis develops.

# ***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

# ***EARLY COMPLICATIONS***

## **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

# ***EARLY COMPLICATIONS***

## **CEREBRO-SPINAL FLUID LEAKS:**

### **CSF OTORRHEA**

- Occurs in 2% of patients with basilar temporal fractures
- 95% usually dries up in 2 weeks
- There is 4% risk of infection

# ***EARLY COMPLICATIONS***

## **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

# ***EARLY COMPLICATIONS***

## **CEREBRO-SPINAL FLUID LEAKS:**

### **MANAGEMENT**

- If it does not close in 2 weeks.
- Insert an 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.

# ***EARLY COMPLICATIONS***

## **CEREBRO-SPINAL FLUID LEAKS:**

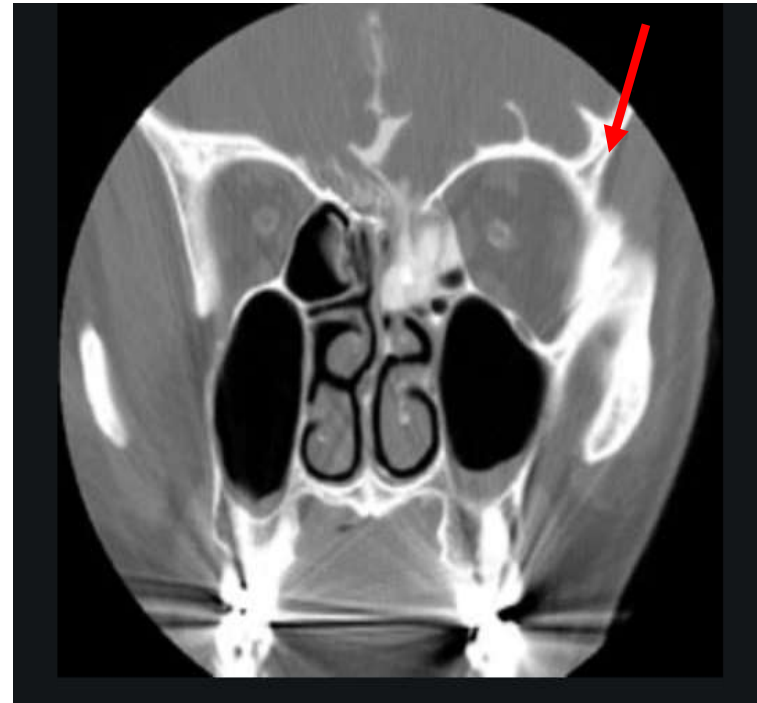
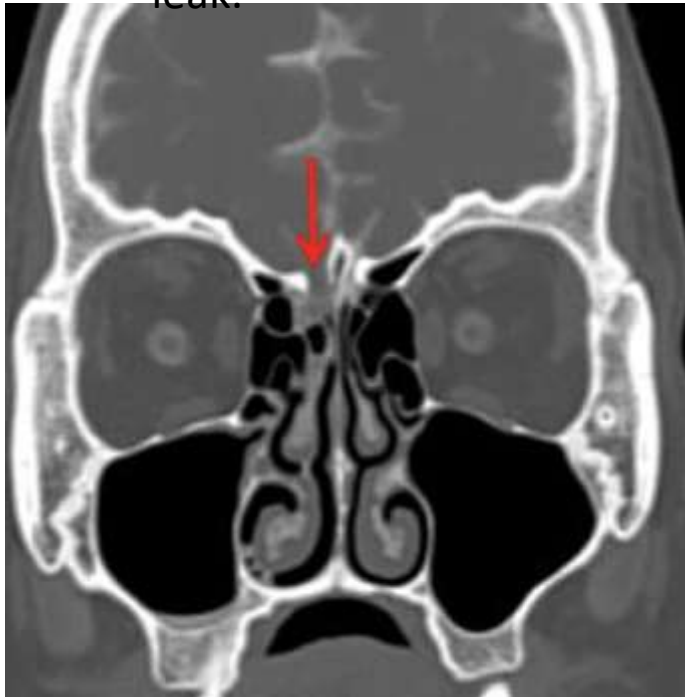
### **MANAGEMENT**

- 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.

# ***EARLY COMPLICATIONS***

## ***CEREBRO-SPINAL FLUID LEAKS:***

High resolution coronal CT with contrast (Cisternogram) 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

# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: EXTRADURAL HEMATOMA**

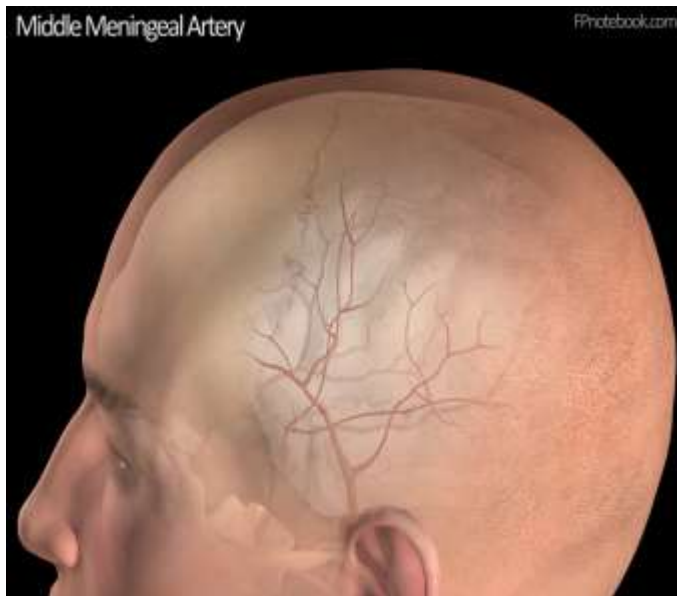
Between dura and skull bone

- 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)

# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: EXTRADURAL HEMATOMA**

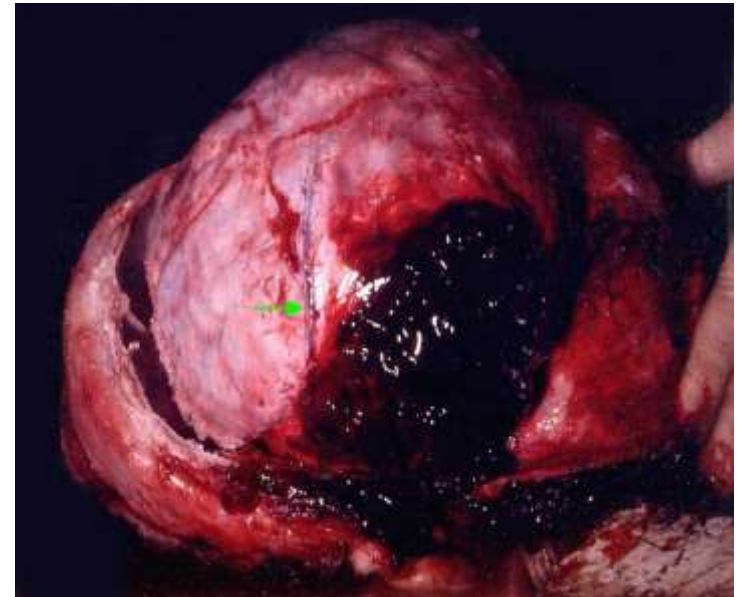
Surface anatomy of MMA.



Linear fractures of the skull.



Postmortem of an EDH



# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: EXTRADURAL HEMATOMA**

### **Two clinical presentations:**

1. Classical 25%
2. Non classical 75%

# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: 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

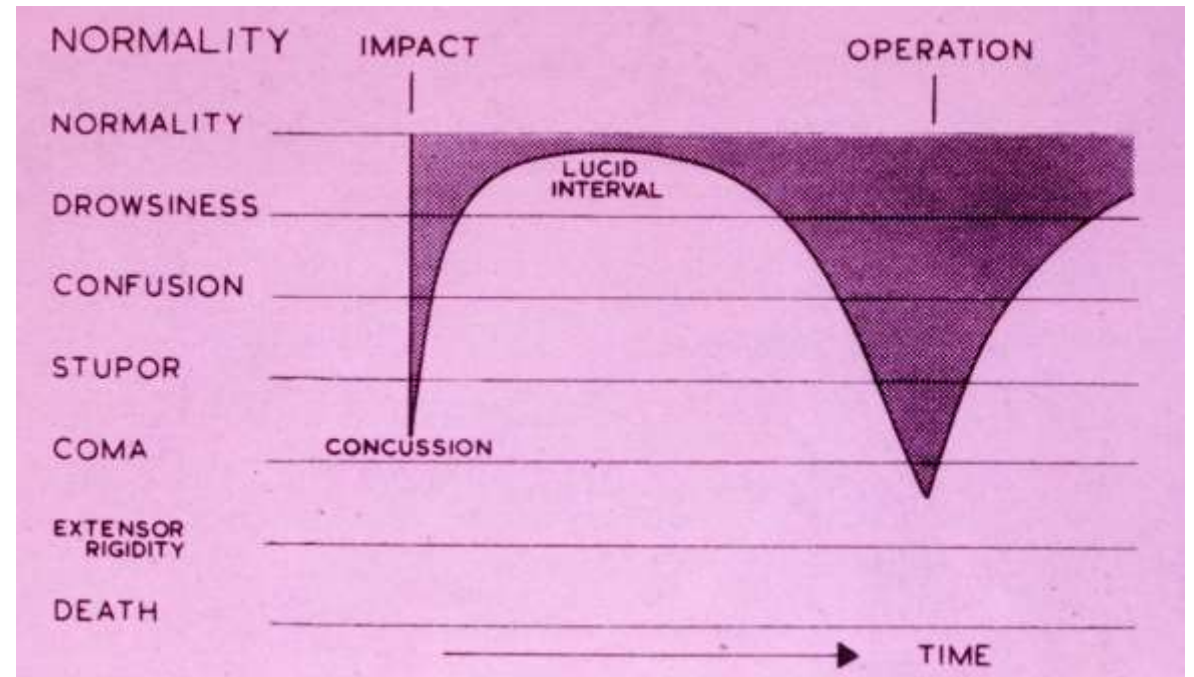
#### Treatment

- Burr-holes
- Craniotomy or Craniectomy

# ***EXTRADURAL HEMATOMA***

INTRACRANIAL HEMATOMAS: EXTRADURAL HEMATOMA

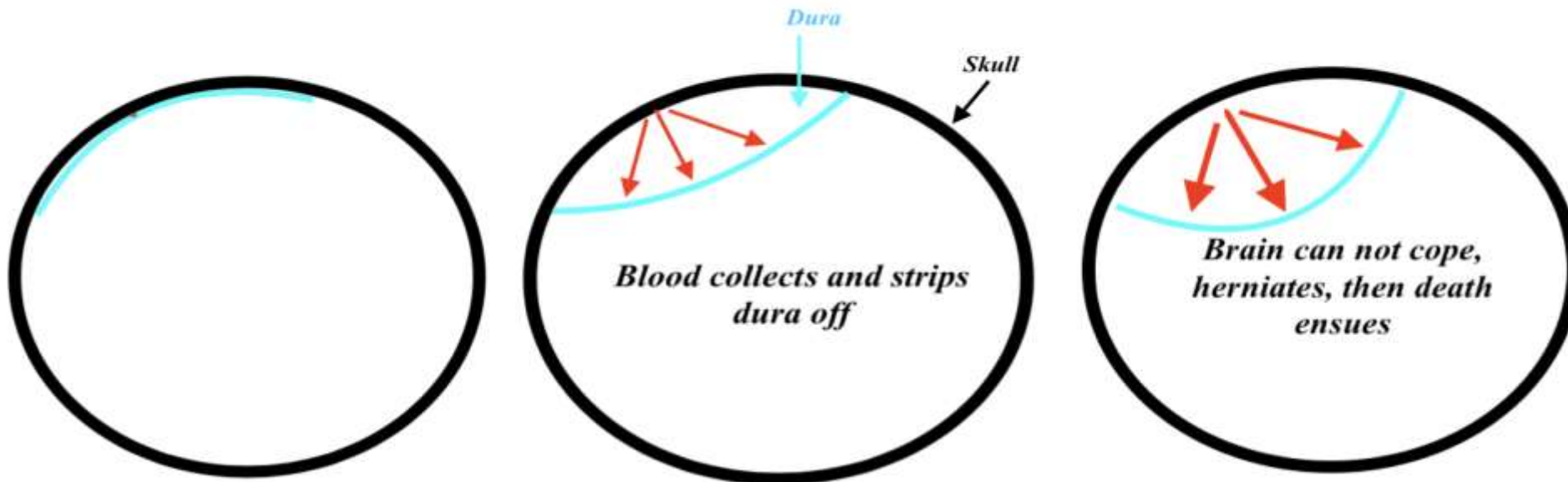
CLASSICAL PRESENTATION



# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: 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



# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: EXTRADURAL HEMATOMA**

### **NON-CLASSICAL PRESENTATION 75%**

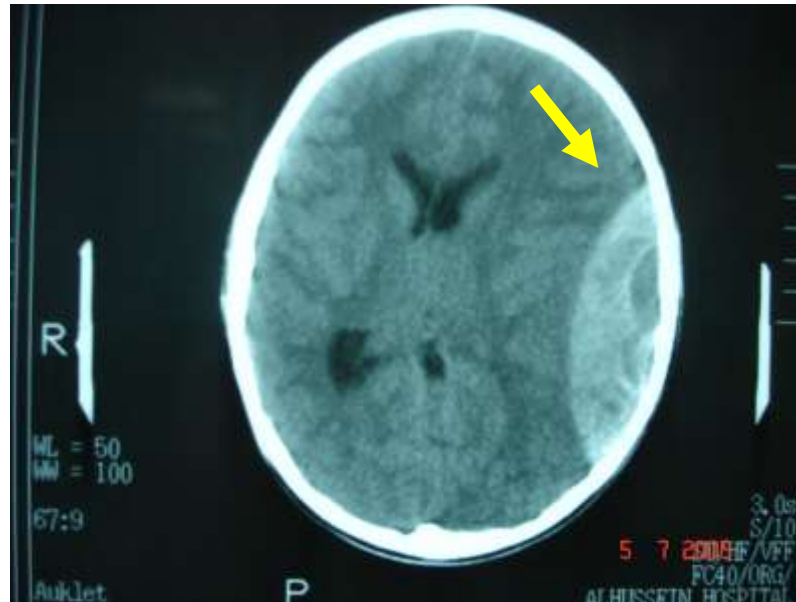
1. Unconscious, remains unconscious, CT shows EDH
2. Conscious, remains conscious, CT shows EDH
3. Conscious, CT shows nothing, admitted, deteriorates, CT shows EDH

# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: EXTRADURAL HEMATOMA**

### **IMAGING**

ACUTE EXTRADURAL HEMATOMA AND SKULL FRACTURE



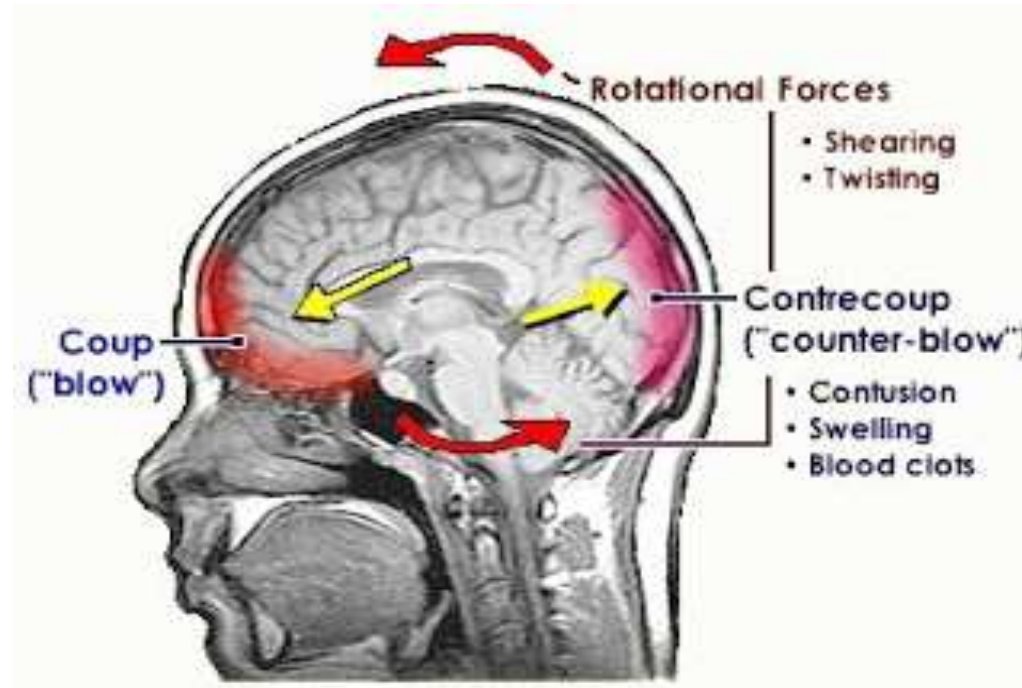
# ***EARLY COMPLICATIONS***

## INTRACRANIAL HEMATOMAS: 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

# ***EARLY COMPLICATIONS***

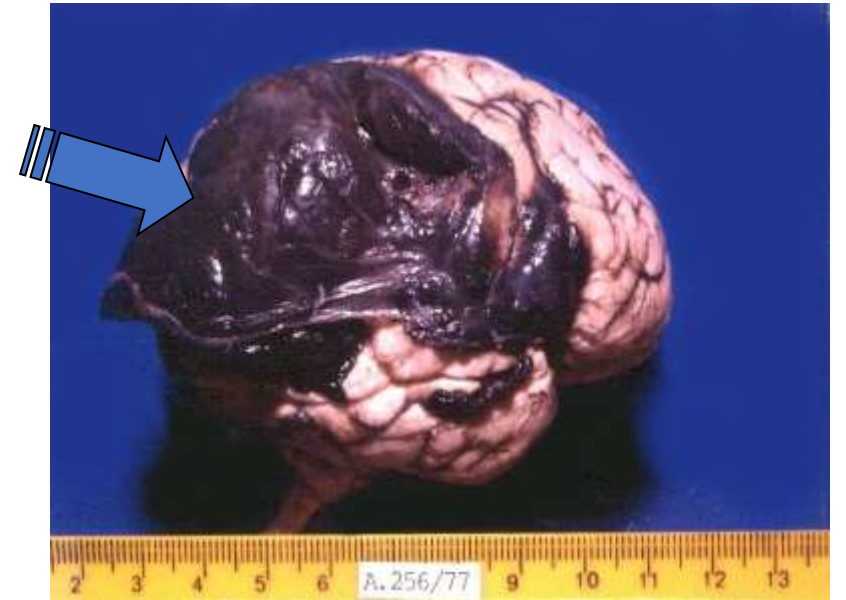
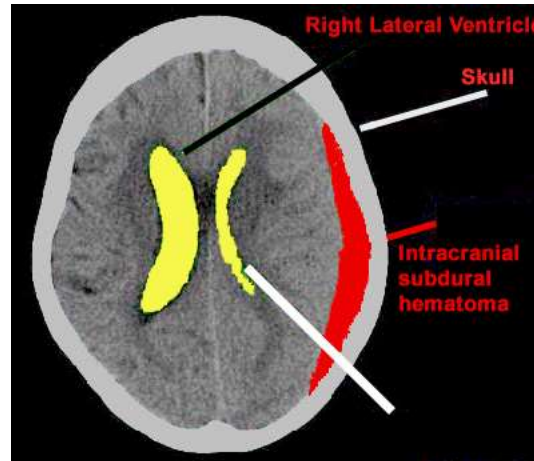
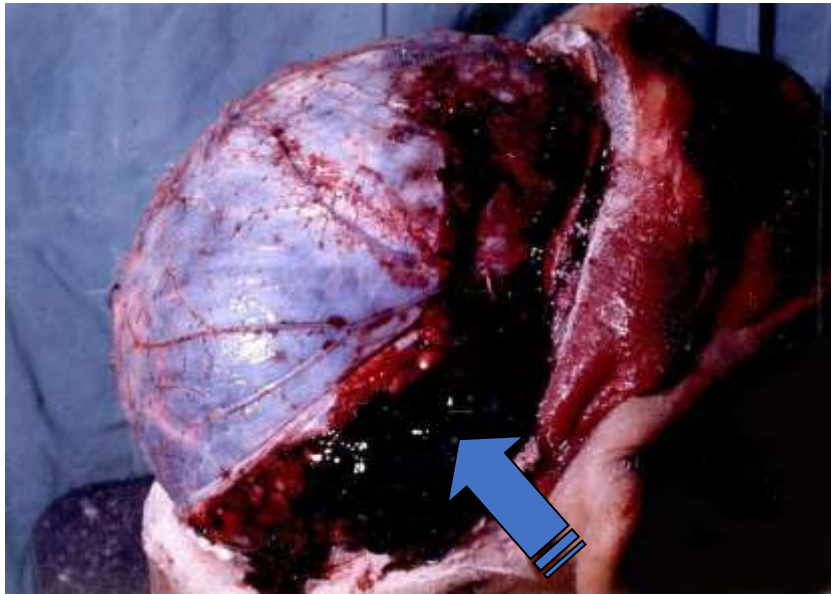
## ***INTRACRANIAL HEMATOMAS: ACUTE SUBDURAL HEMATOMA***



## ***ACCELERATION – DECELERATION INJURY***

# ***EARLY COMPLICATIONS***

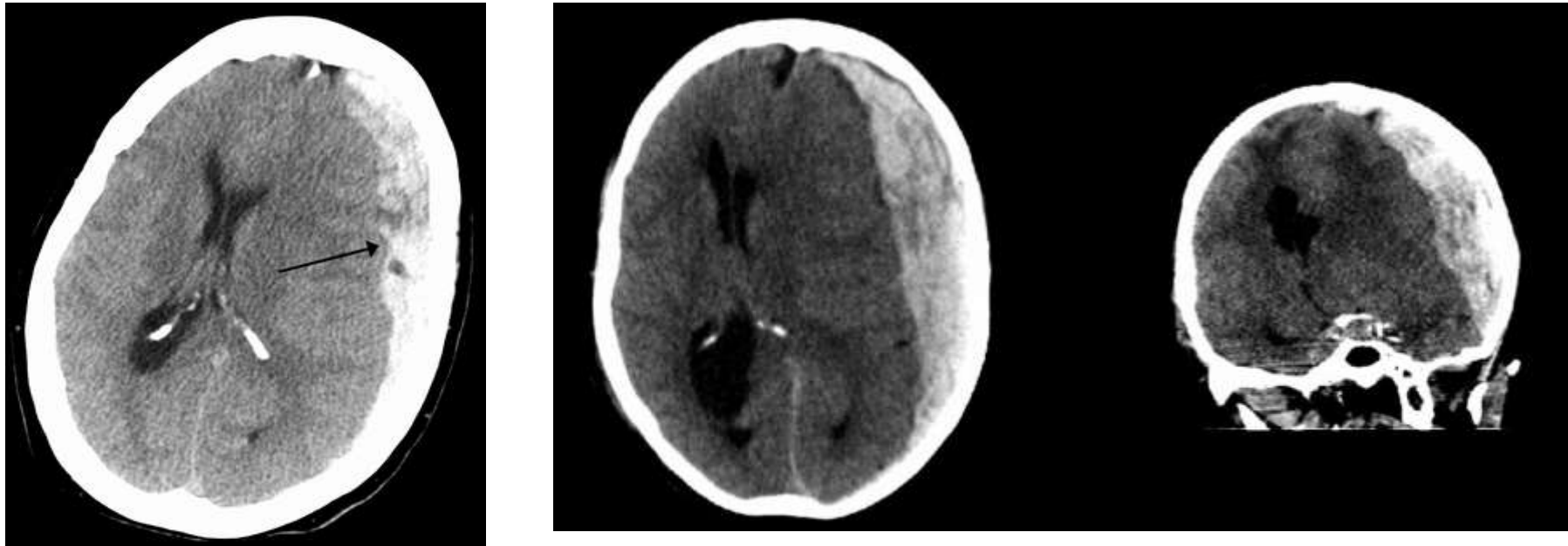
## **INTRACRANIAL HEMATOMAS: ACUTE SUBDURAL HEMATOMA**



# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: ACUTE SUBDURAL HEMATOMA**

**Imaging:** Confirmed by a non contrasted CT



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

# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: ACUTE SUBDURAL HEMATOMA SO; WHAT ARE THE DIFFERENCES?**

### **EXTRADURAL**

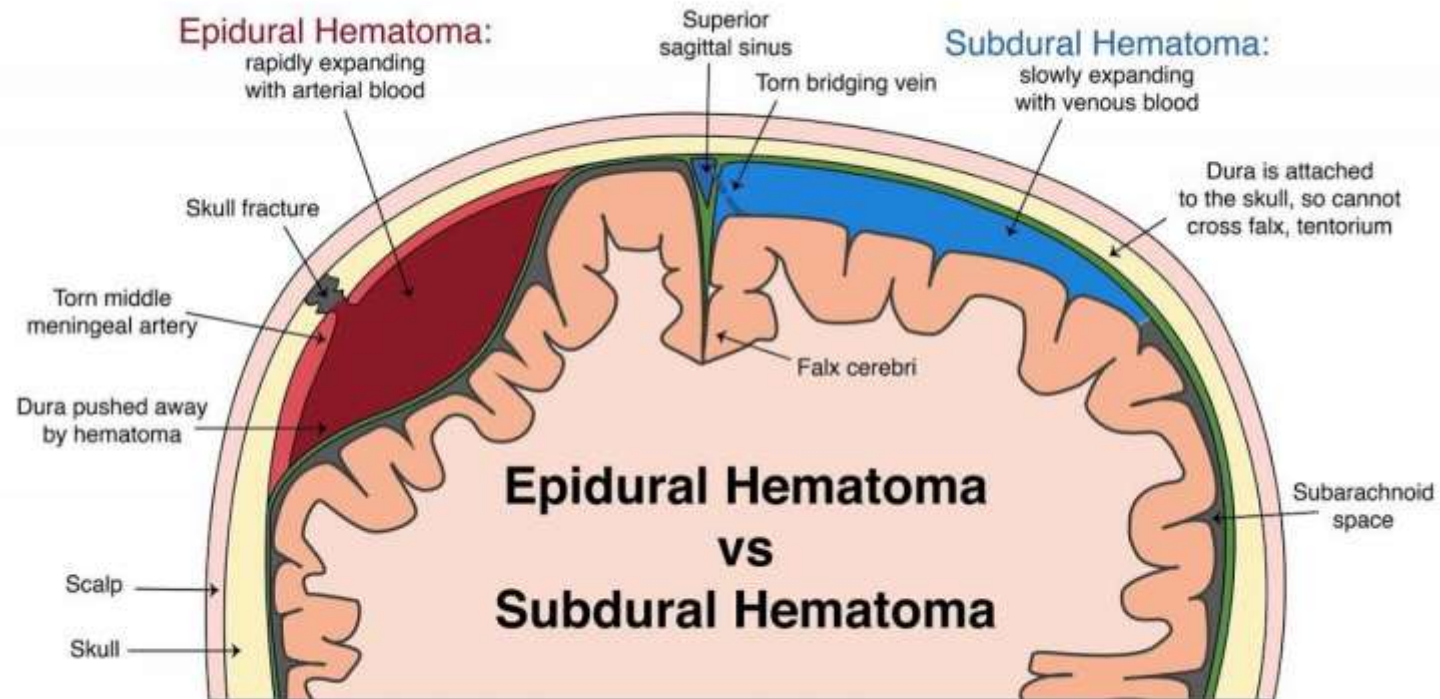
- BETWEEN SKULL AND DURA
- ARTERIAL BLOOD
- FROM MIDDLE MENINGIAL ARTERY
- LOCALIZED
- **USUALLY NO BRAIN INJURY**
- OCCASIONALLY WITH LUCID INTERVAL (25%)
- 25% OF CASES HAVE NO FRACTURES.

### **SUBDURAL**

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

# ***EARLY COMPLICATIONS***

## INTRACRANIAL HEMATOMAS: ACUTE SUBDURAL HEMATOMA EXTRA Vs SUBDURAL HEMATOMA



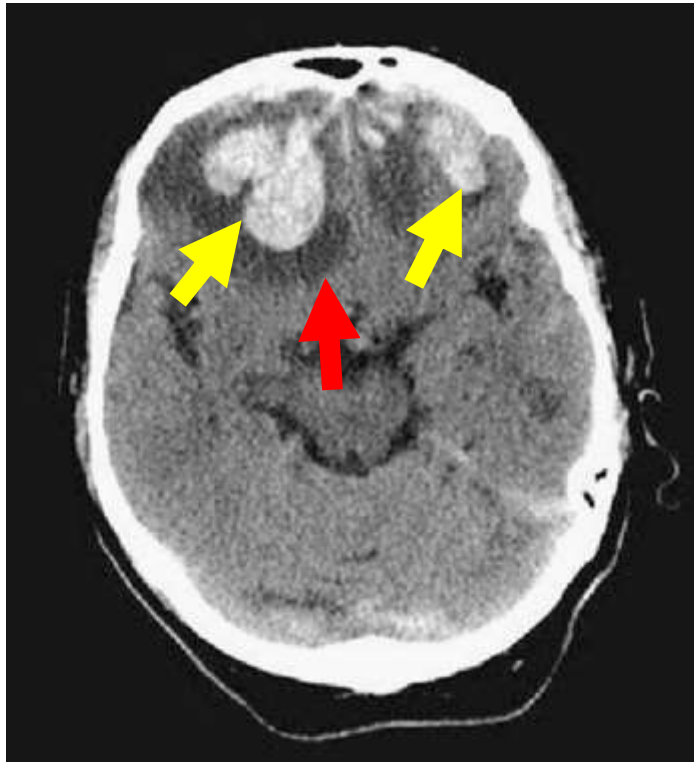
# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: 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.

# ***EARLY COMPLICATIONS***

## **INTRACRANIAL HEMATOMAS: 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).*

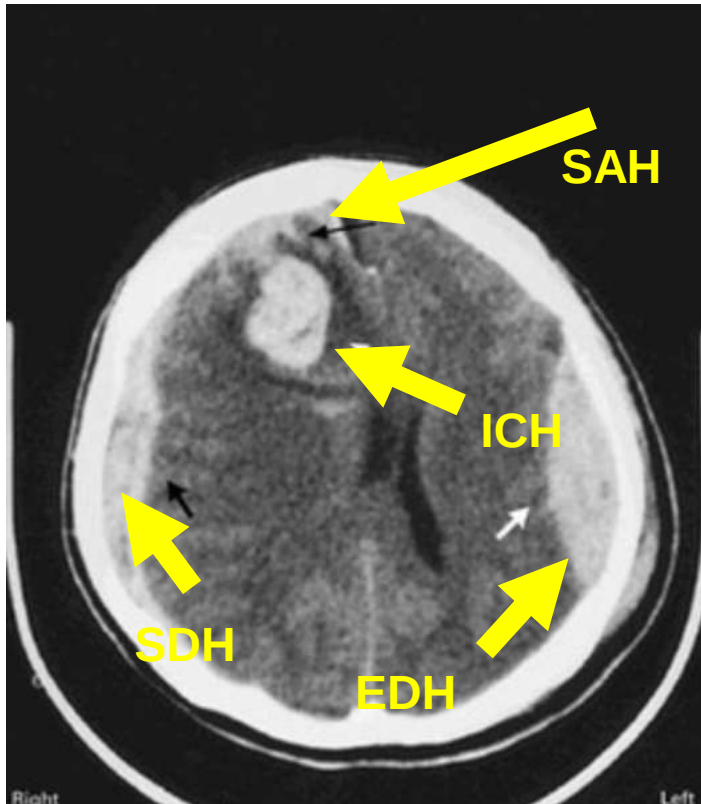
# ***EARLY COMPLICATIONS***

## ***INTRACRANIAL HEMATOMAS: 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

# ***EARLY COMPLICATIONS***

## **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 (THE OSMOTIC THEORY)

# ***LATE COMPLICATIONS***

## **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.

# ***LATE COMPLICATIONS***

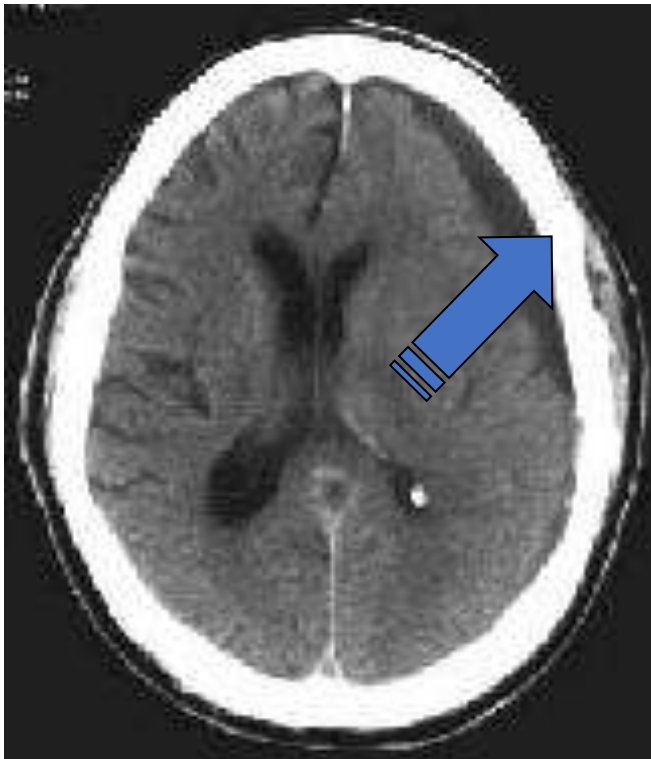
## **CHRONIC SUBDURAL HEMATOMA**



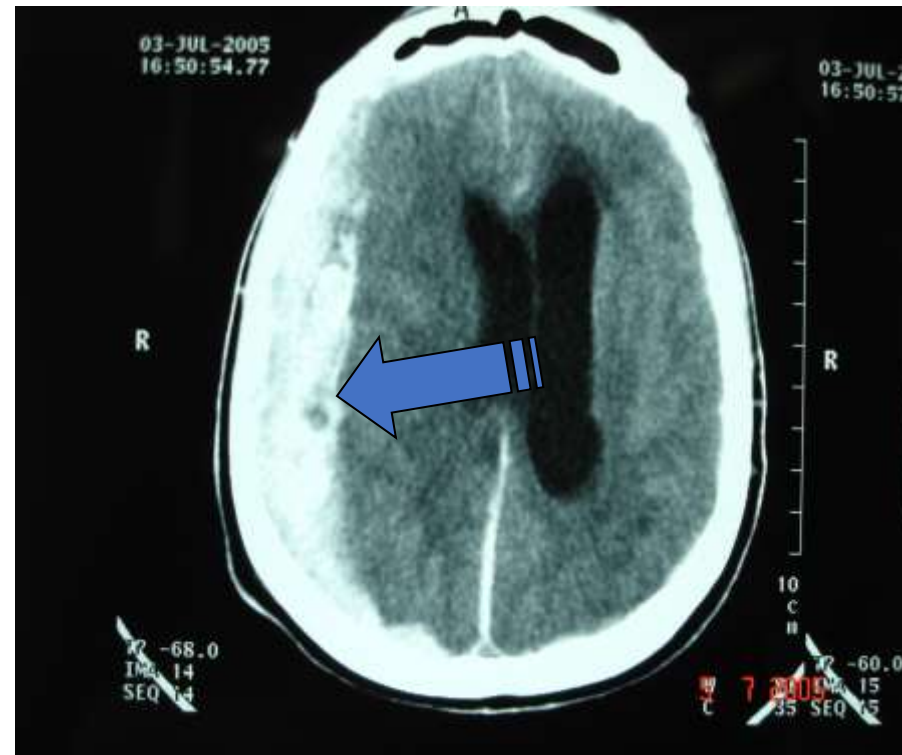
Non contrasted CT showing the semilunar hypodense appearance of the chronic subdural hematoma. Note the ventricular compression, the midline shift and the effacement of sulci; all indicative of high ICP

# ***LATE COMPLICATIONS***

## **CHRONIC VERSUS ACUTE SUBDURAL HEMATOMA ON NON-CONTRASTED CT**



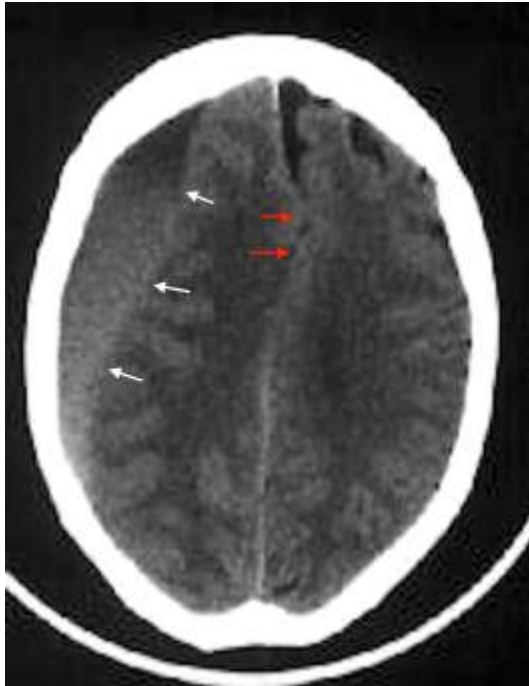
***CHRONIC SUBDURAL HEMATOMA***



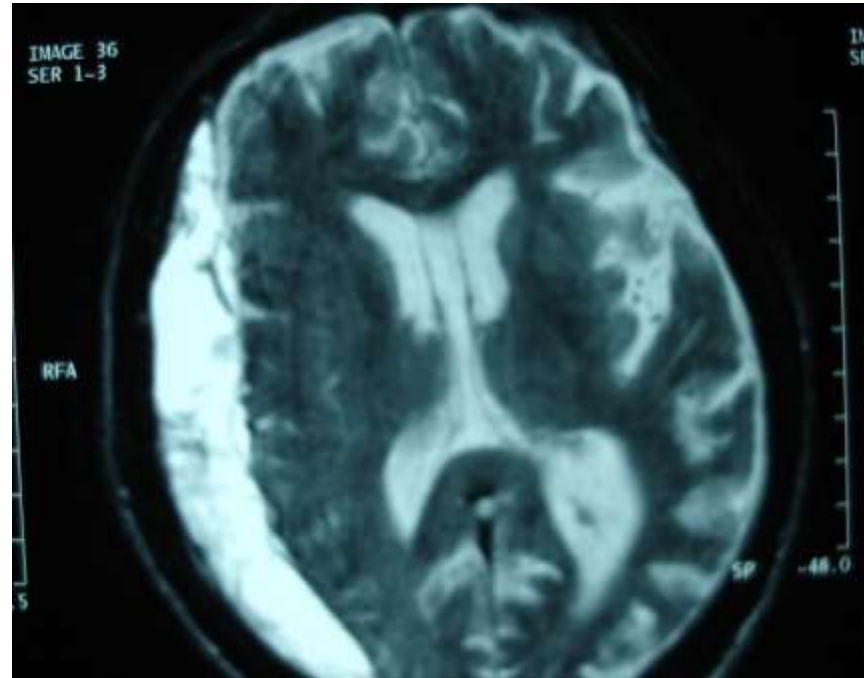
***ACUTE SUB DURAL HEMATOMA***

# ***LATE COMPLICATIONS***

## **CHRONIC SUBDURAL HEMATOMA**



CT



MRI

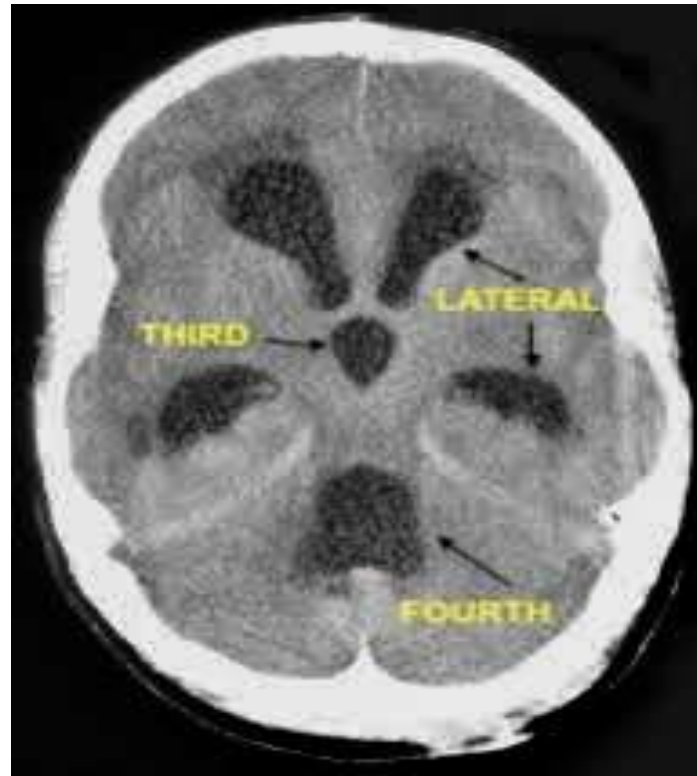
# ***LATE COMPLICATIONS***

## **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

# ***LATE COMPLICATIONS***

## ***HYDROCEPHALUS***



*Non contrasted CT showing communicating 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

# ***LATE COMPLICATIONS***

## **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
    - DIZZINESS
    - VISUAL DISTURBANCES
    - HEARING PROBLEMS
    - BALANCE DIFFICULTIES
  - **COGNITIVE:**
    - CONCENTRATION DIFFICULTY
    - DEMENTIA

# ***LATE COMPLICATIONS***

## **POST CONCUSSION SYNDROME**

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

- ❑ ***TREATMENT***
  - REASSURANCE*
  - SUPPORT*

# *FURTHER READING*

- Neurosurgery Made Easy by Prof. Walid Maani available at [Amazon.com](https://www.amazon.com) in paper and kindle format and at [Lulu.com](https://www.lulu.com) in pdf format
- Introduction to Neuroimaging by Prof. Walid Maani available at [Amazon.com](https://www.amazon.com) in paper and kindle format and at [Lulu.com](https://www.lulu.com) in pdf format