# HEAD INJURY

- Any degree of injury to the head ranging from scalp laceration to LOC to focal neurological deficits



# Traumatic brain injury (TBI)

Traumatic brain injury (TBI) is a nondegenerative, noncongenital insult to the brain from an external mechanical force, possibly leading to permanent or temporary impairment of cognitive, physical, and psychosocial functions, with an associated diminished or altered state of consciousness

# ETIOLOGY

- Motor vehicle accidents
- Falls
- Assaults
- Sports-related injuries
- Firearm-related injuries



Highest among ***adolescents***, young adults, and those older than 75

50% of major trauma deaths are due to TBI

***Vehicle crashes*** are the leading cause of brain injury. ***Falls*** are the second leading cause

***Motor Vehicle*** Crashes - **44%**

***Falls*** - **26%**

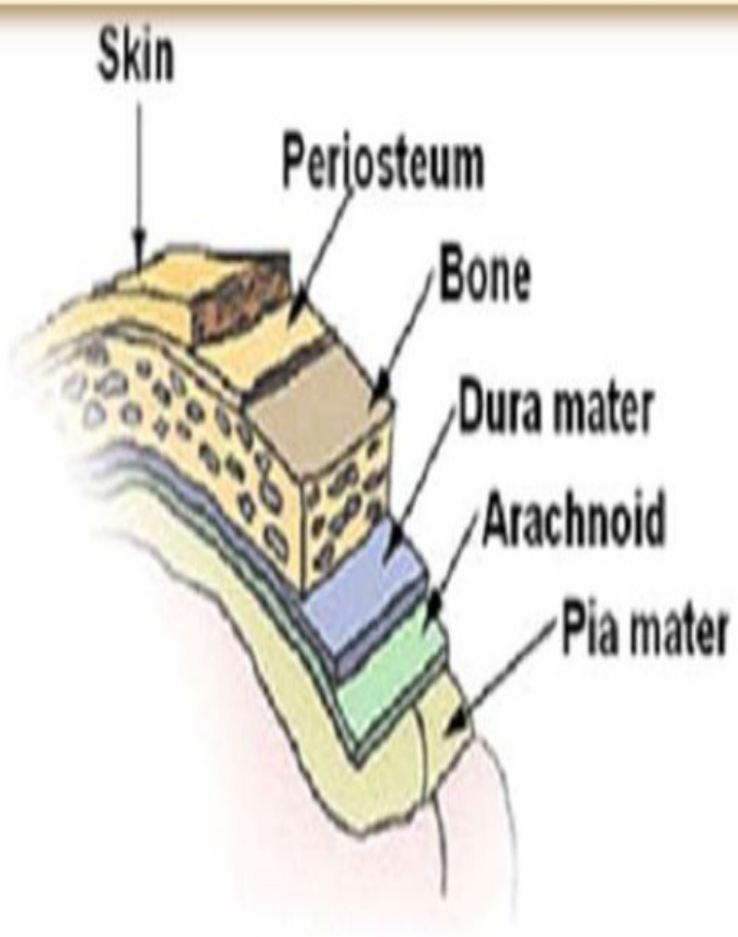
Other/Unknown - **13%**

Assaults- **9%**

Firearms- **8%**

- High potential for poor outcome
- Deaths occur at three points in time after injury:
  - Immediately after the injury
  - Within 2 hours after injury
  - 3 weeks after injury

# LAYERS

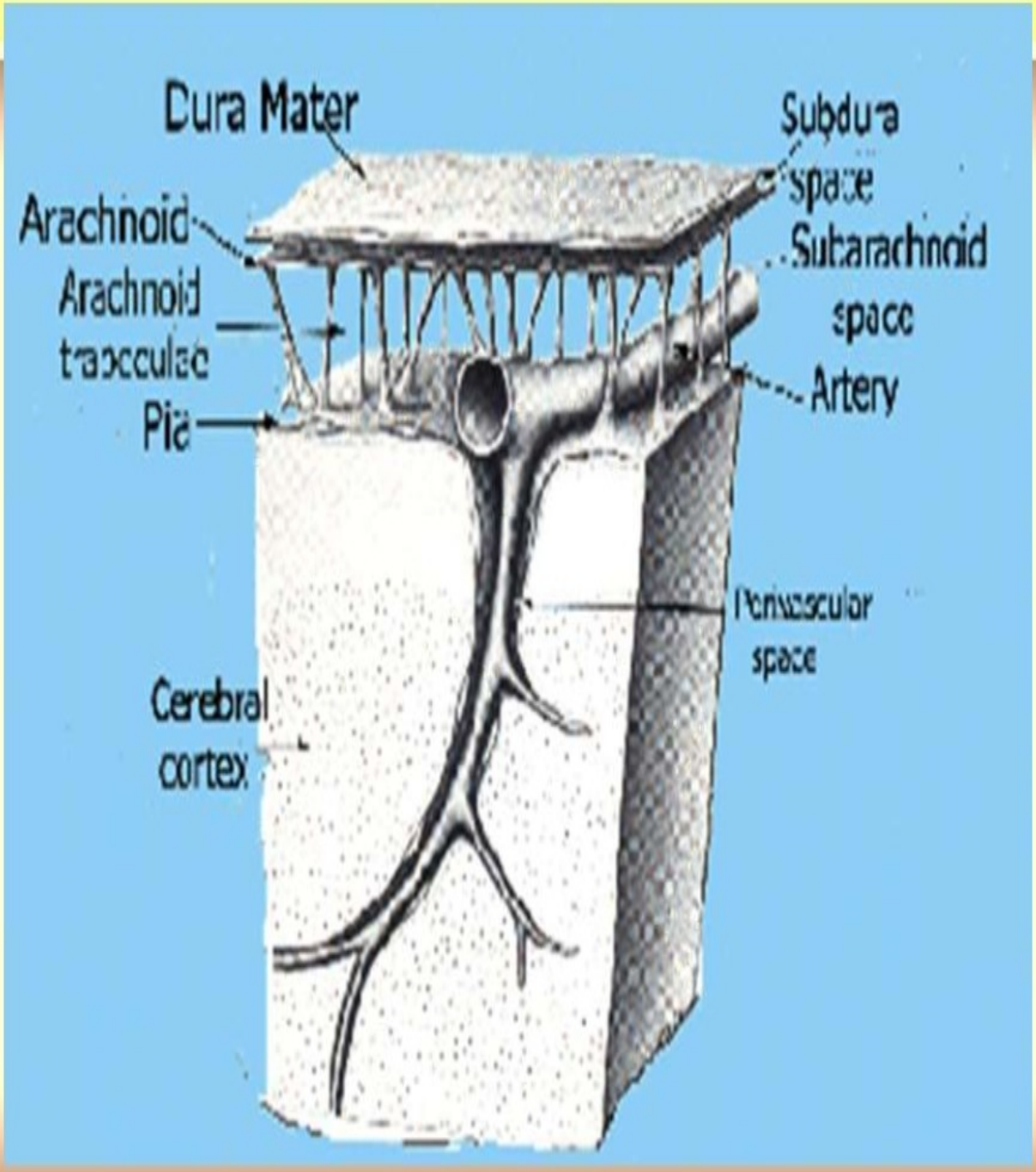



Dura mater -- outer layer lining skull

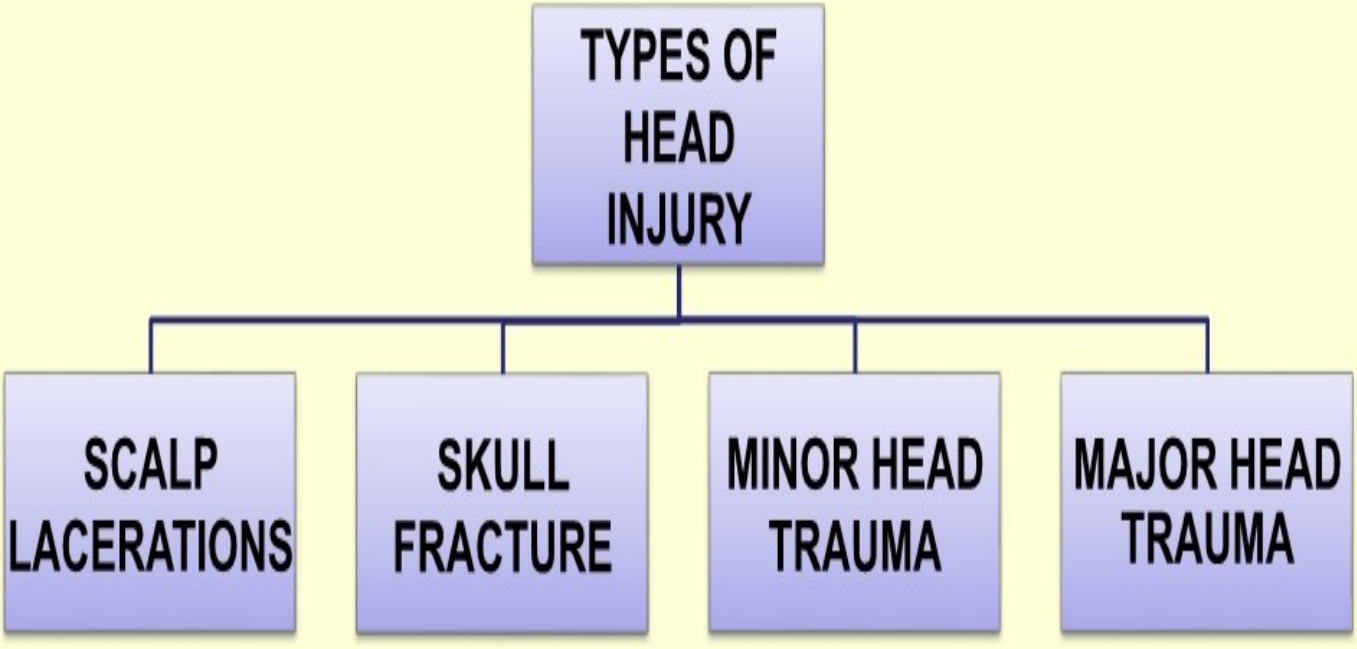
Arachnoid (mater) -- contains blood vessels

Subarachnoid space -- filled with CSF

Pia mater -- covers brain


Dura Mater
Subdura
space
Arachnoid
Subarachnoid
space
Arachnoid
trabeculae
Artery
Pia
Perivascular
space
Cerebral
cortex

# TYPES


TYPES OF HEAD INJURY
SCALP LACERATIONS
SKULL FRACTURE
MINOR HEAD TRAUMA
MAJOR HEAD TRAUMA

# LACERATIONS

- Easily recognized
  - The most minor type of head trauma
  - Scalp is highly vascular → profuse bleeding
  - Major complication is infection



# SKULL FRACTURES

- LINEAR

break in the continuity of bone without alteration of relationship of parts

cause- Low velocity injuries

- DEPRESSED

Inward indentation of skull

cause- powerful blow



- Comminuted

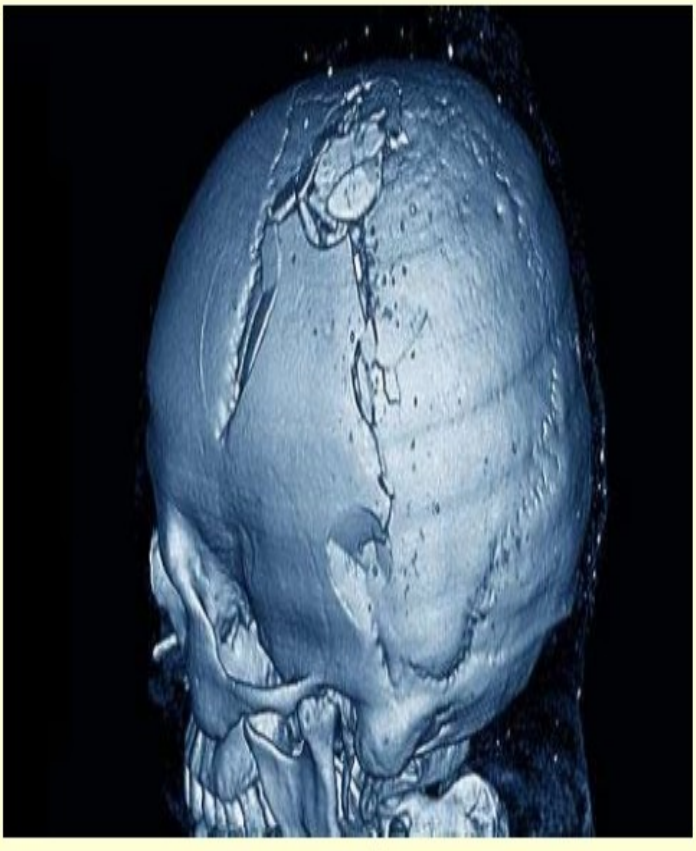
  multiple linear fractures with fragmentation of bones into pieces

- Compound

  Depressed skull fractures and scalp laceration communicating intracranial cavity

## COMMINUTED FRACTURE





compound fracture

# ACCORDING TO LOCATION

- Frontal fracture
- Temporal fracture
- Parietal fracture
- Posterior fossa fracture
- Orbital fracture
- Basilar skull fracture

# Temporal bone fracture

- Boggy temporal muscle because extravasation of blood
- Oval shaped bruise behind the ear in mastoid region (battle sign)
- Otorrhoea



# Parietal bone fracture

- Deafness
- CSF otorrhoea
- Bulging of tympanic membrane by blood or CSF
- Facial paralysis

# Orbital fracture

- Periorbital ecchymosis(RACCOON EYES)
- Optic nerve injury





# Basilar skull fracture

- Otorrhoea, rhinorrhoea
- Bulging of tympanic membrane
- Battle's sign
- Facial paralysis
- Tinnittis , vertigo

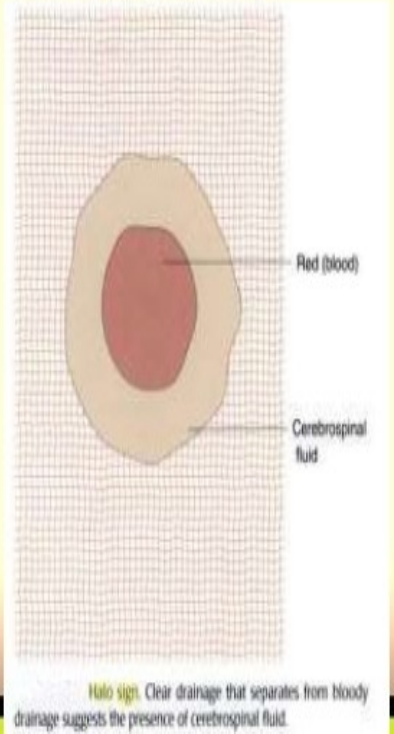
# Test to determine CSF leakage

Method 1

- Check for presence of glucose
- Dextrostrip/ Tes-Tape strip
- If blood is present in the fluid
- The test become unreliable
- Go for 2nd method

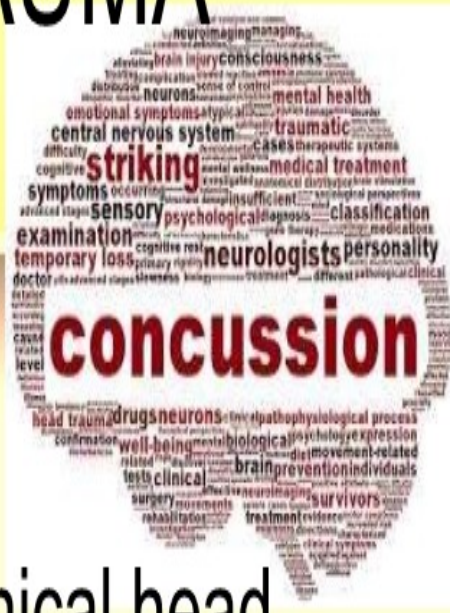
# Method 2( halo ring sign)

- Allow leaking fluid drip onto a white pad/towel
- Observes the drainage
- Within a few minutes the blood coalesces into center and a yellowish ring encircles the blood



Halo sign. Clear drainage that separates from bloody drainage suggests the presence of cerebrospinal fluid.

# MINOR HEAD TRAUMA




- CONCUSSION

A sudden transient mechanical head injury with disruption of neuronal activity and a change in the LOC

It occurs When the brain suddenly shifts inside the skull and knocks against the skulls bony surface

# TYPICAL SIGNS

- Brief disruption of LOC

Concussions can last from a few moments, to an unconscious state for over 3 min

- Amnesia regarding event
- Headache



# MAJOR HEAD TRAUMA

## CONTUSION

It is the bruising of the brain tissue within a focal area

- It is usually associated with a closed head injury

- COUP-COTRECOUP IS OFTEN NOTED





- In this type of injury contusion occur both at the site of direct impact of the brain on the skull( *coup*) and at the a secondary area of damage on the opposite side away from injury ( *contrecoup*) leading to multiple contusion areas

- LACERATIONS

It involve actual tearing of brain tissue and often occur in association with depressed ,open fractures and penetrating injuries



- Intracerebral hemorrhage commonly associated

# COMPLICATIONS

- INTRACRANIAL HAEMORRHAGES

*Extra- axial hemorrhage*

- Epidural hematoma
- Subdural hematoma-

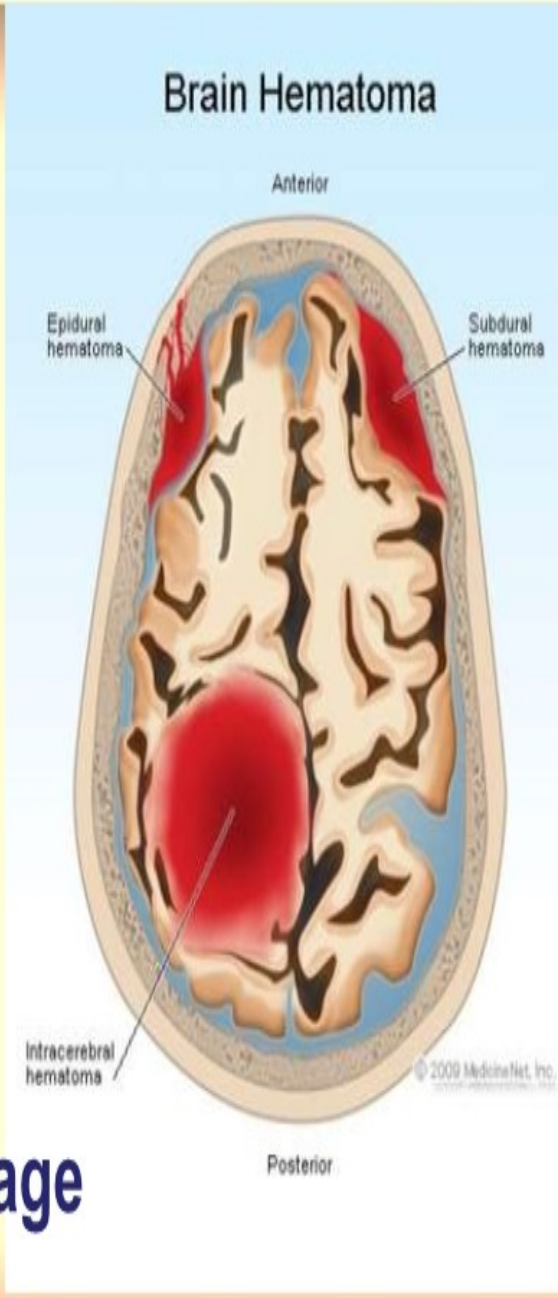
  Acute

  Chronic

- Subarachnoid hemorrhage

*Intra-axial hemorrhage*

- Intra-parenchymal hemorrhage
- Intra-ventricular hemorrhage



# EPIDURAL HEMORRHAGE

- A neurologic emergency
- Most common type of intracranial hemorrhage
- Results from bleeding between the dura and the inner surface of the skull
- Blow to the *temporal, parietal bone*
- Commonly bleeding by arterial origin- breakage to middle meningeal artery
- Venous- dural venous sinus

# Clinical manifestation- EDH

- The patient is initially unconscious after the trauma
- The patient then awakens and has a lucid interval followed by a decrease in LOC
- Headache
- Nausea and vomiting

- On head CT the clot is bright, **biconvex shaped clot** and has a well-defined border that usually respects cranial suture lines



- A rapid Open craniotomy for evacuation of the congealed clot and hemostasis is indicated for EDH
- Prevention of cerebral herniation can dramatically improve outcome

# SUB-DURAL HEMATOMA

- Subdural hematoma occurs from bleeding between the dura matter and the arachnoid layer of the meninges
- Types
  1. acute subdural hematoma
  2. subacute subdural hematoma
  3. Chronic subdural hematoma

- SDH usually results from venous bleeding, usually from tearing of a bridging vein running from the cerebral cortex to the dural sinuses.
- Hematoma may be slower to develop

# Acute subdural hemorrhage

- It develop 24-48 hrs after the severe head trauma
- Commonly related to acceleration-deceleration injury
- Clinical manifestations as same as elevated ICP

- The size of hematoma determines the patient clinical presentation
- Decreasing LOC from drowsy and confused to unconsciousness
- Headache
- Ipsilateral pupil dilation
- Motor signs

On head CT scan, the clot is bright or mixed-density, crescent-shaped (lunate), may have a less distinct border



- Open craniotomy for evacuation of the clot and decompression is indicated for any acute SDH more than 1 cm in thickness, or smaller hematomas that are symptomatic

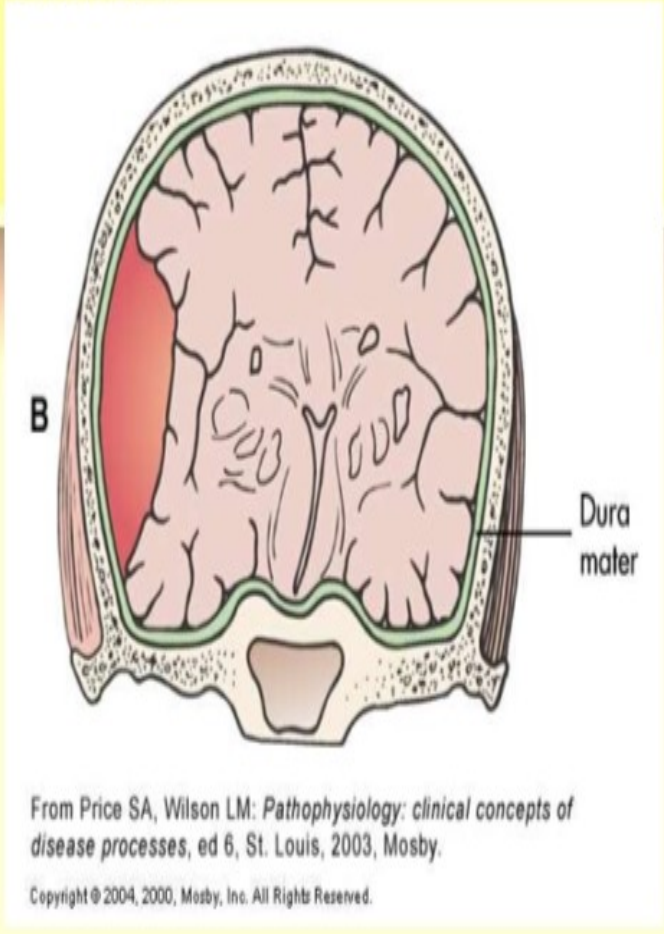
# SUBACUTE SUBDURAL HEMATOMA

- Usually occurs within 2-14 days of the injury
- The alteration in mental status as hematoma develops
- Progression depends on the size and location of hematoma

# CHRONIC SUBDURAL HEMATOMA

- It develops over weeks or months after seemingly minor head injury
- The peak incidence of chronic SDH is in 50-60 Years of age
- Clinical manifestations is progressive alteration in LOC

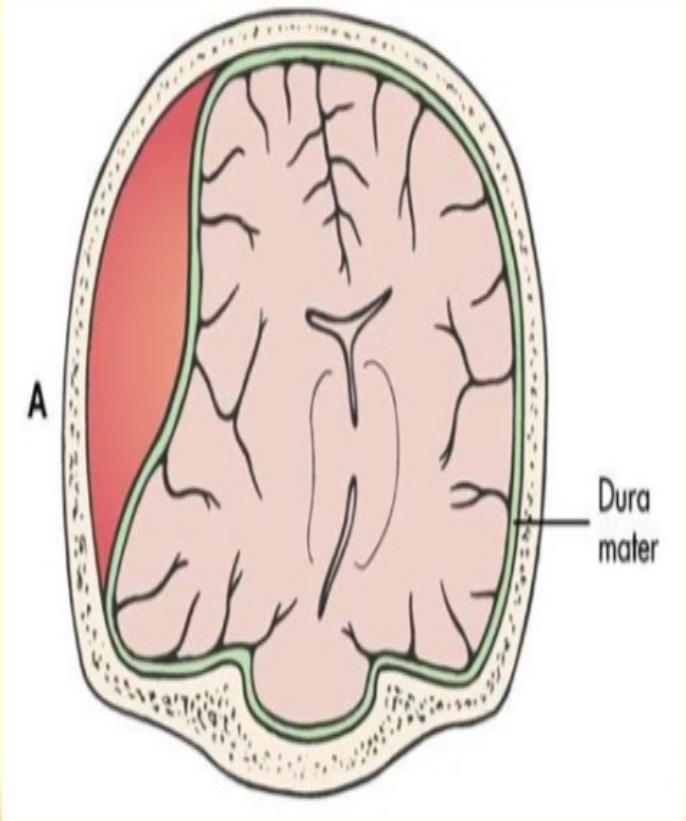
# Epidural and Subdural Hematomas




From Price SA, Wilson LM: *Pathophysiology: clinical concepts of disease processes*, ed 6, St. Louis, 2003, Mosby.

Copyright © 2004, 2000, Mosby, Inc. All Rights Reserved.

## Epidural Hematoma




## Subdural Hematoma

Fig. 55-15

# Epidural and Subdural Hematomas

| Hematoma type | Epidural | Subdural |
|---|---|---|
| Location | Between the skull and the dura | Between the *dura* and the arachnoid |
| Involved vessel | Temperoparietal (most likely) - Middle meningeal artery<br>Frontal - anterior ethmoidal artery<br>Occipital - transverse or sigmoid sinuses<br>Vertex - superior sagittal sinus | Bridging veins |
| Symptoms | Lucid interval followed by unconsciousness | Gradually increasing headache and confusion |
| CT appearance | Biconvex lens- limited by suture lines | Crescent shaped- crosses suture lines |

Fig. 55-15

# SUB ARACHNOID HEMORRHAGE

- Bleeding occurs between the arachnoid and pia mater

**CAUSES**

- **Rupture of Berry aneurism**
- **Trauma (fracture at the base of the skull leading to internal carotid aneurysm)**

- **Clinical Features:**
- Explosive headache,
  "worst headache of my life",
- nausea and vomiting, decreased LOC or coma.
- Signs of meningeal irritation

- Increased attenuation is seen in the CSF Spaces over the cerebral hemisphere



# Intracerebral Hemorrhage (ICH)

Intraaxial hemorrhage is hemorrhage that occurs within the brain tissue itself

Two main types:

1) *Intraparencymal hemorrahge*- ICH extending into brain parenchyma;
2) *Intra-ventricular hemorrhage*- ICH extending into ventricles;

# CAUSES

Hypertensive vasculopathy (70-80%)

Ruptured Aneurism

Trauma- 16%

## Clinical presentation:

- Rapidly progressive severe headache, building over several minutes, often accompanied by focal neurological deficits, nausea and vomiting, decreased level of consciousness.

# S/S depend site of hemorrhage:

Basal ganglia/internal capsule - hemiparesis,dysphasia

Cerebellum - ataxia, vertigo

Pons - cranial nerve deficits,coma

Cerebral cortex - hemiparesis, hemisensory loss, hemianopsia, dysphasia

# Diagnostic measures

- History collection and physical examination
- Computerised tomography
- Magnetic resonance imaging
- Positron emission tomography
- X-RAY

# Taking a history in head injury

- ■ Mechanism of injury
- ■ Loss of consciousness or amnesia
- ■ Level of consciousness at scene and on transfer
- ■ Evidence of seizures
- ■ History of vomiting
- ■ Pre-existing medical conditions
- ■ Medications (especially anticoagulants)
- ■ Illicit drugs and alcohol

# Physical examination

- ■ Glasgow Coma Score
- ■ Pupil size and response
- ■ Signs of skull fracture

  Bilateral periorbital edema (raccoon eyes)

  Battle's sign (bruising over mastoid)

  Cerebrospinal fluid rhinorrhoea or otorrhoea

  Haemotympanum or bleeding from ear
- ■ Full neurological examination: tone, power, sensation, reflexes

# Computerised tomography

- CT scan is considered the best diagnostic test to evaluate for cranio-cerebral trauma because it allows rapid diagnosis and intervention in the setting
- The National Institute for Health and Clinical Excellence (NICE) has published some guidelines for when to carry out a CT scan in a patient with head injury

# NICE guidelines for (CT) in head injury

- Glasgow Coma Score (GCS) < 13 at any point
- ■ GCS 13 or 14 at 2 hours
- ■ Focal neurological deficit
- ■ Suspected open, depressed or basal skull fracture
- ■ Seizure
- ■ Vomiting > one episode
- Urgent CT head scan if none of the above but:
- ■ Age > 65
- ■ Coagulopathy (e.g. on warfarin)
- ■ Dangerous mechanism of injury (CT within 8 hours)
- ■ Antegrade amnesia > 30 min (CT within 8 hours)

- An MRI scan is more sensitive than CT scan in detecting small lesions
- A cervical spine X-ray indicated to detect any cervical injury
- Transcranial doppler allow the mesurement of CBF

# Management

- Severe head injury is best managed in a neurointensive care setting
- The patient should be positioned with the head up 30 degree
- It is important to ensure that the cervical immobilisation collar does not obstruct venous return from the head

# Airway and ventilation

- patient in traumatic coma is unable to protect their airway and is at risk for aspiration
- Maintain a normocapnia

# Circulation and cerebral perfusion pressure

- Hypotension and hypoxia as a major cause of secondary brain injury.
- A systolic BP < 90 mmHg worse outcome in traumatic coma
- Cerebral perfusion pressure should be maintained at > 65 mmHg in severely head-injured patients.

# Control of intracranial pressure

- Position head up 30º
- Avoid obstruction of venous drainage from head
- Sedation +/– muscle relaxant
- Normocapnia
- Diuretics: furosemide, mannitol
- Seizure control
- Normothermia
- Barbiturates

# MEDICATIONS

- Osmotic diuretics
- Anticonvulsants
- Barbiturates
- Calcium Channel Blockers

# OSMOTIC DIURETICS

- MANNITOL 25%
- 1.5-2 g/kg IV infused over 30-60 minutes

# ANTICONVULSANTS

## PHENYTOIN

where it may inhibit spread of seizure activity in motor cortex

## DOSAGE-

- Load 10-15 mg/kg THEN
- Maintenance: 100 mg IV/PO q6-8hr PRN

# BARBITURATES

## PENTOBARBITAL

- It will reduce the brain metabolic rate and helps reduce ICP.

Dosage- 100 mg IV OR 150-200 mg IM

# Surgical management

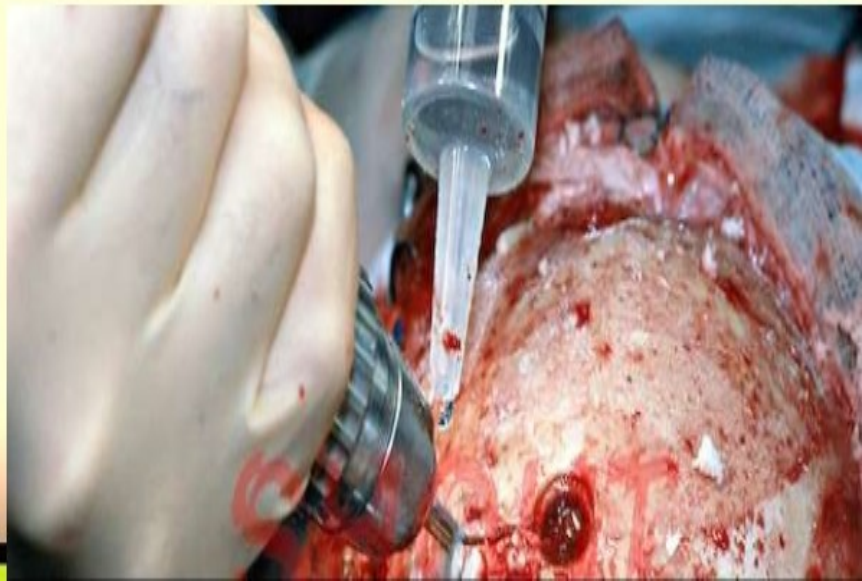
**No surgical intervention if collection <10ml**

***Indication of surgical decompression:***

- The GCS score decreases by 2 or more points between the time of injury and hospital evaluation
- The patient presents with fixed and dilated pupils
- The intracranial pressure (ICP) exceeds 20 mm Hg

# Types:

- **Burr-hole-**
  opening into cranium with a drill
- **Craniotomy-**
  bone flap is temporarily removed from the skull to access the brain



- **Craniectomy –**

  Excision into the cranium to cut away a bone flap

- **Cranioplasty -**

  surgical repair of a defect or deformity of a skull

# Nursing management

- Nursing assessment
  - ABC
  - GCS Score
  - Neurologic examination
  - Signs of elevated ICP
  - Signs of CSF leakage

# Nursing diagnosis

- Ineffective tissue perfusion (cerebral) related to interruption of CBF associated with cerebral hemorrhage and edema
- Acute pain (headache) related to trauma and cerebral edema
- Hyperthermia related to increased metabolism, and loss of cerebral integrative function secondary to possible hypothalamus injury

- Impaired physical mobility related to decreased LOC and treatment –imposed bedrest
- Anxiety related to abrupt change in health status, hospital environment and uncertain future
- Risk for complication related cerebral edema and hemorrhage

# Preventive Measures

## Health Promotion

- Prevent car and motorcycle accidents
- To Wear safety helmets

# Rehabilitation

## Ambulatory and Home Care

- Nutrition
- Bowel and bladder management
- Seizure disorders
- Family participation and education

# Unconscious stages

- **Stupor** is a state of partial or near complete unconsciousness in which the patient is lethargic, immobile, and has a reduced response to stimuli.
- **Coma** is a state in which the patient is totally unconscious and cannot be aroused even with strong stimuli.

# Persistent vegetative state

- It is a condition in which awake patients are unconscious and unaware of their surroundings and the cerebral cortex is not functioning. A vegetative state can result from diffuse injury to the cerebral hemispheres of the brain without damage to the lower brain and brainstem. The vegetative state is considered permanent if it persists for 12 months after TBI