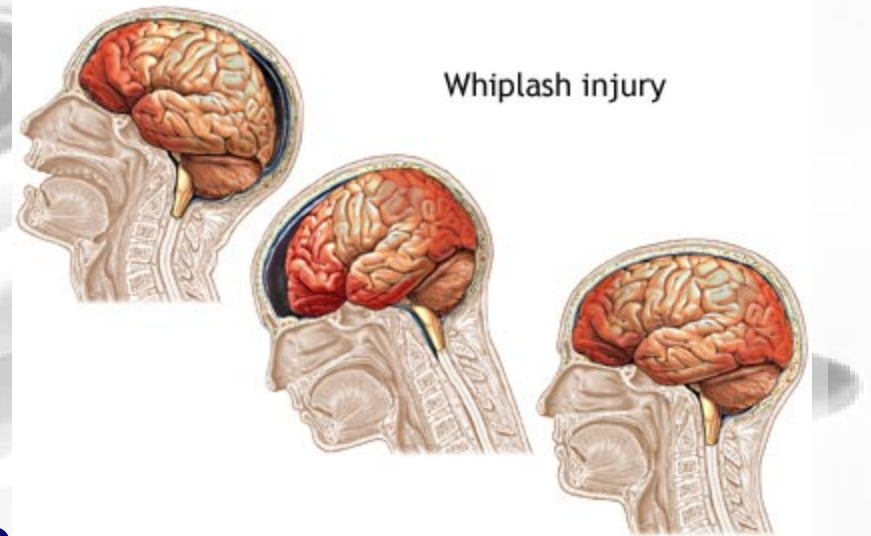
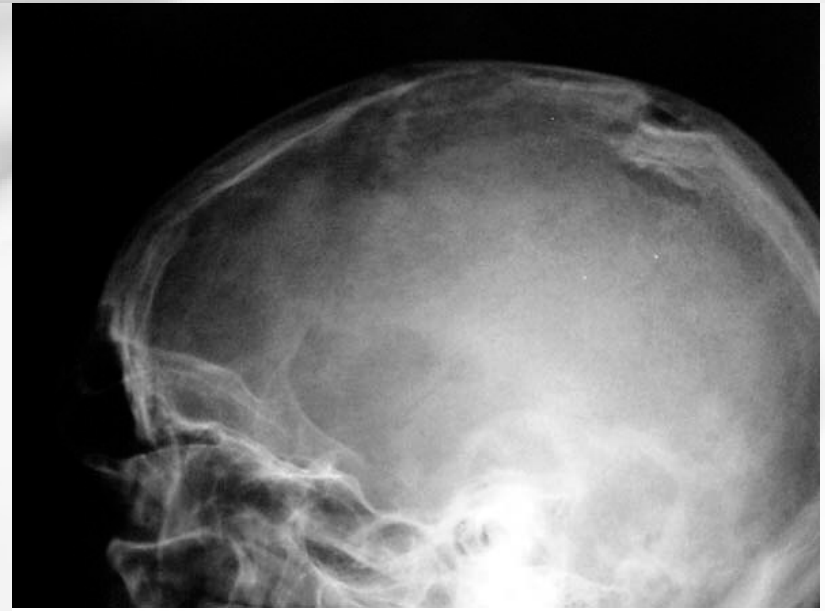


Acute Head Injury



ADAM.

Presented by Omar AL-Rawajfah, RN, PhD

Lecture Outlines

- Etiology and pathophysiology
- Systemic impact of acute brain injury
- Assessment
- Nursing diagnoses
- Collaborative management
- Brain death

Acute Head Injury

- Acute head injury: terms used in discussions about an injury to the head and its structures
- Brain injury: refers to injuries of the brain itself
- Traumatic Brain Injury (TBI): brain injuries results from a traumatic event and may result from a blunt or a penetrating injury
- Closed head injury: a blunt injury to the brain that does not result in an open skull fracture
- Penetrating injury: result from a missile, much as a knife, gun, hammer, etc

Head Trauma

Open

- Skull compromised and brain exposed



Closed

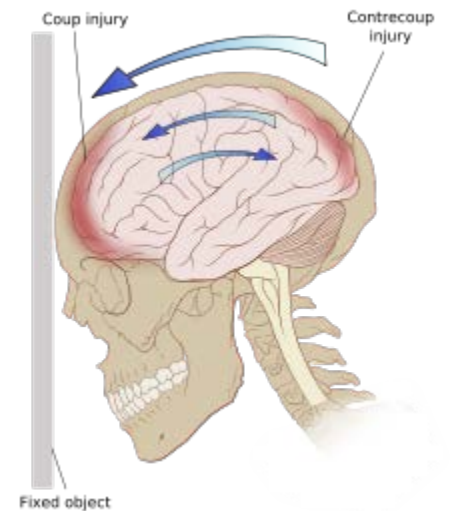
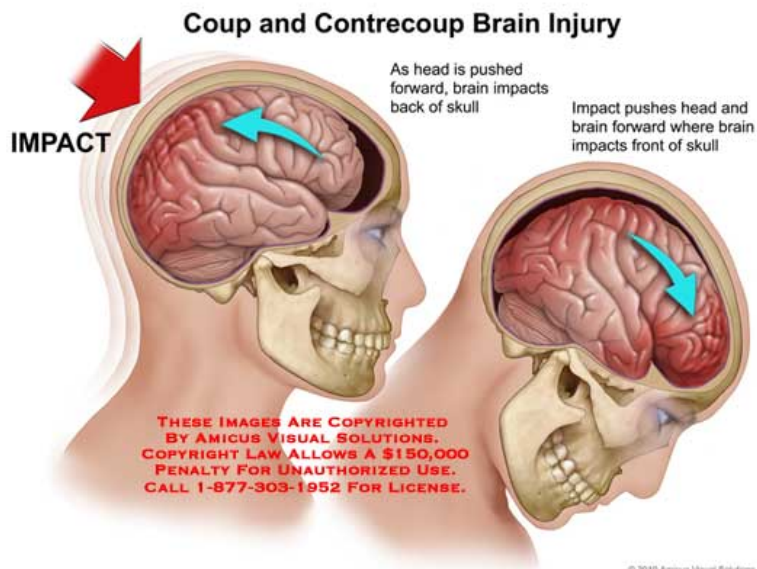
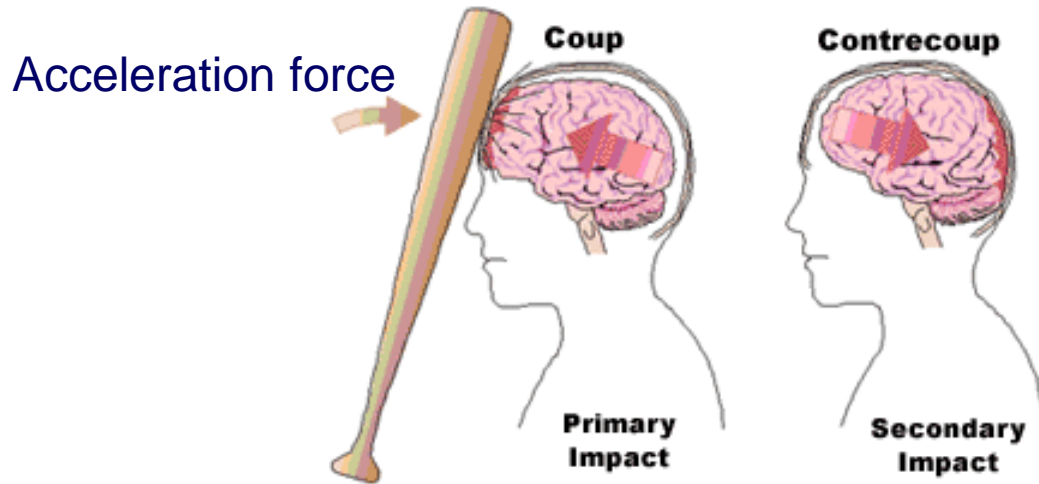
- Skull not compromised and brain not exposed



Etiology and Pathophysiology

- Mechanism:
 - Primary injury: when kinetic force is applied to the cranium and brain
 - Secondary injury: biophysical and biochemical changes occur that alter perfusion, leading to neuronal dysfunction and death
- Five types of kinetic forces:
 - Acceleration force: a moving object hits stationary head (e.g., ball, hummer)
 - Deceleration force: a moving head hits stationary object (e.g., wall, ground)
 - Acceleration-deceleration forces: occurs during high-speed motor vehicle crashes and care versus pedestrian crashes
 - Coup-contrecoup head injury: result of movement of intracranial contents within the cranium
 - Rotational injury: twist within the skull resulting in stretching or tearing of neuron and or cerebral blood vessels

Etiology and Pathophysiology



Deceleration force

Primary injury

- Severity of head injury
 - Mild → GCS 13-15
 - Moderate → 9 – 12
 - Sever → less than 8
- Primary injury divided into:
- Focal injury
 - Scalp lacerations
 - Skull fractures
 - Lacerations and contusions
 - Hematomas
- Diffuse injury
 - Cerebral concussion
 - Diffused Axonal injury (DAI)

Primary injury

- Scalp lacerations
 - Disruption of the outmost protective layer of the brain that is very vascular and may bleed profusely
 - Wound should be cleaned using sterile dressing



Primary injury

- Skull fractures
 - A. Linear fracture
 - Crack in the skull
 - Usually need no surgical intervention
 - B. Depressed skull
 - Displace the bone with or without laceration of the dura or brain tissue
 - Need surgical elevation
 - C. Basilar skull
 - Linear fractures in at the base of the skull
 - Basilar fracture in the anterior fossa may result in periorbital ecchymosis (raccoon or panda eyes)
 - Blood or CSF may drain from the nose or the ears
 - Rhinorrhea: leakage of CSF from the nose
 - Otorrhea: leakage of CSF from the ear
 - Nose or ear should not be packed when CSF leak
 - Obstruction of CSF flow leads to infection



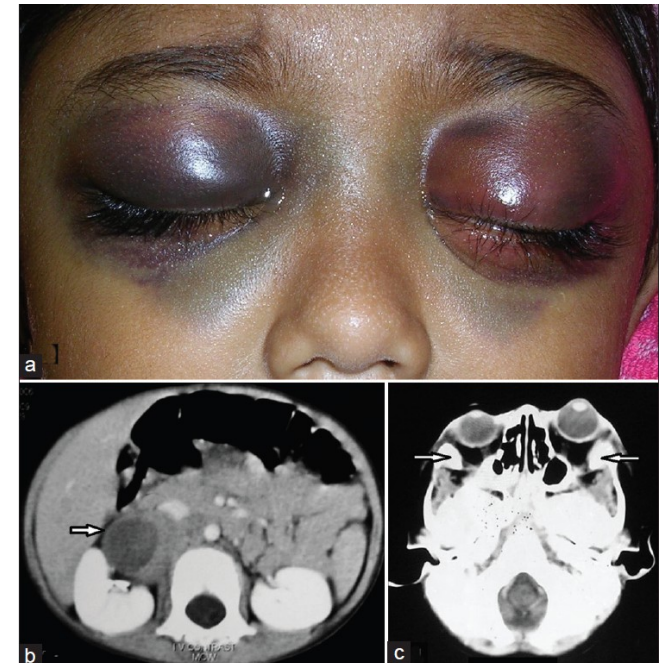
- **Battle's Sign:** Ecchymosis of the mastoid process of the temporal bone.



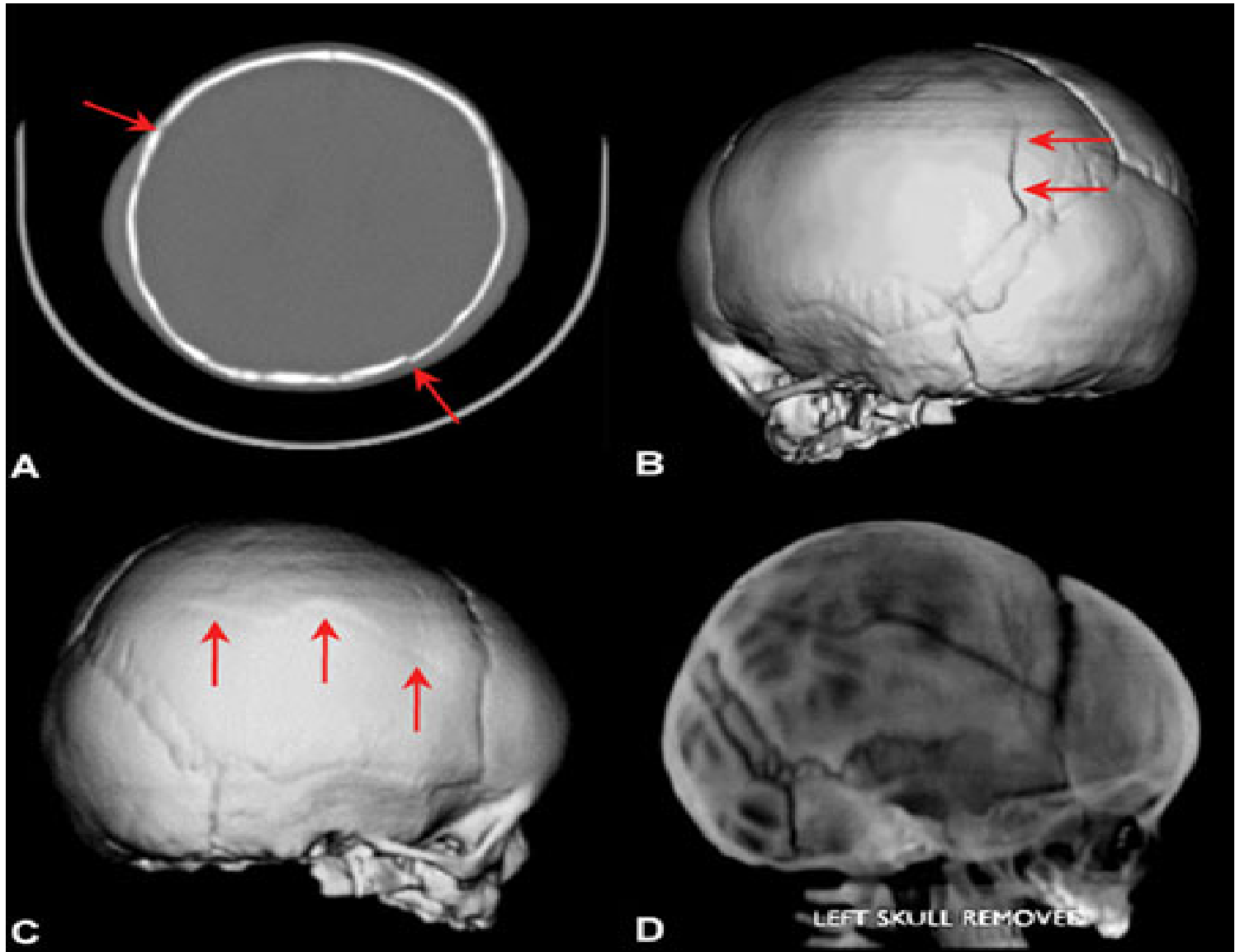
- **Hemotympanum**

Recognizing raccoon eyes

It's usually easy to differentiate raccoon eyes from the "black eye" associated with facial trauma. Raccoon eyes (shown at right) are always bilateral. They develop 2 to 3 days after a closed-head injury that results in a basilar skull fracture. In contrast, the periorbital ecchymosis that occurs with facial trauma can affect one eye or both. It usually develops within hours of injury.



Linear Fracture



Depressed skull



Primary injury

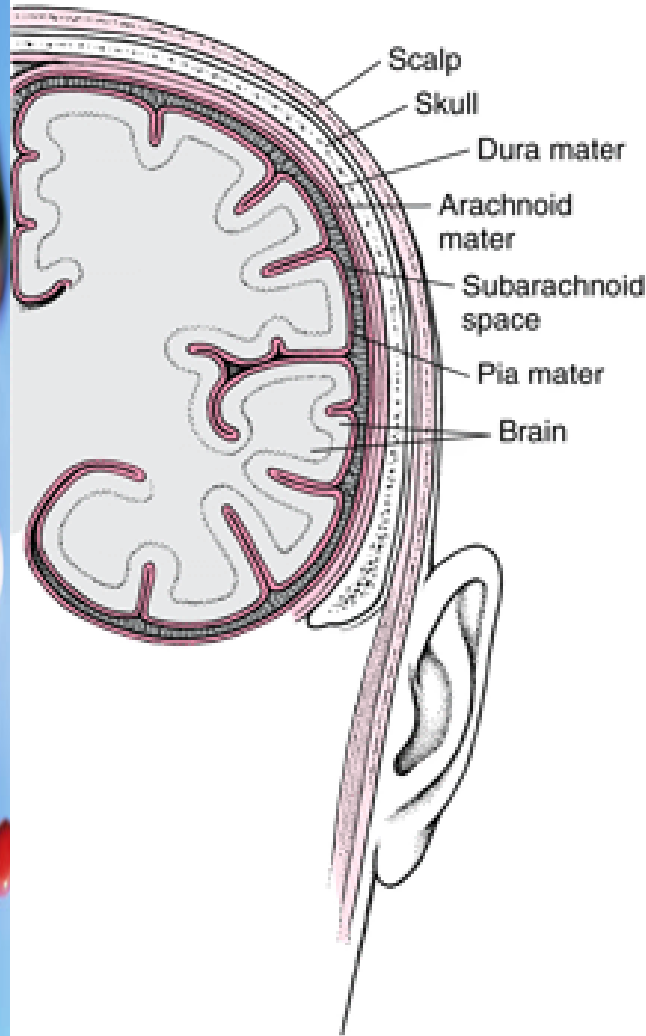
- Laceration & Contusion
 - Can be focal or multiple lesions
 - CT scan is used to locate the lesions
 - Usually in the frontal or temporal lobes
 - It is tearing of brain tissue beneath a depressed skull
 - Mortality associated with cerebral contusion may be related to development of cerebral edema
 - Cerebral edema is peaked within 24-72 hrs
 - Patient may experience agitation, motor dysfunction, coma, short memory loss
 - Sometimes surgical debridement of necrotic tissue is performed

Primary injury

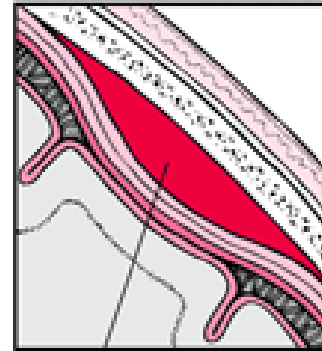
- Hematomas
 - Epidural hematomas
 - Lies outside the dura between the dura and cranium
 - Result in displacing the brain
 - The death rate become high when the hematoma result from arterial bleeding
 - Treatment usually surgical removal of the clot
 - Early treatment result in good prognosis
 - Subdural hematomas (SDH)
 - Accumulation of blood below the dura and above the arachnoid
 - Usually result from venous bleeding from the cortex or bridging veins between the dura and brain surface
 - More common with alcoholic elderly
 - Acute SDH:
 - Symptoms appear within the first 24-72hr
 - Need close neurological monitoring including ICP
 - Immediate surgical evacuation of the clot usually required

Skull and Brain Layers

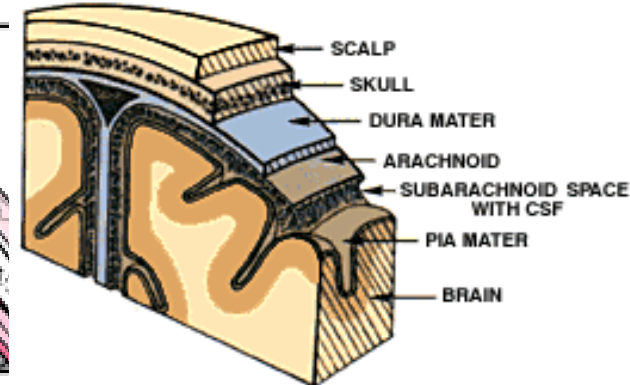
Cross Section of the Brain



Epidural Hematoma

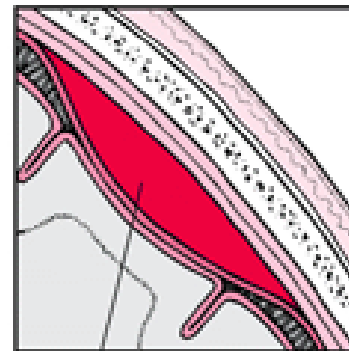


Bleeding between the dura mater and the skull.



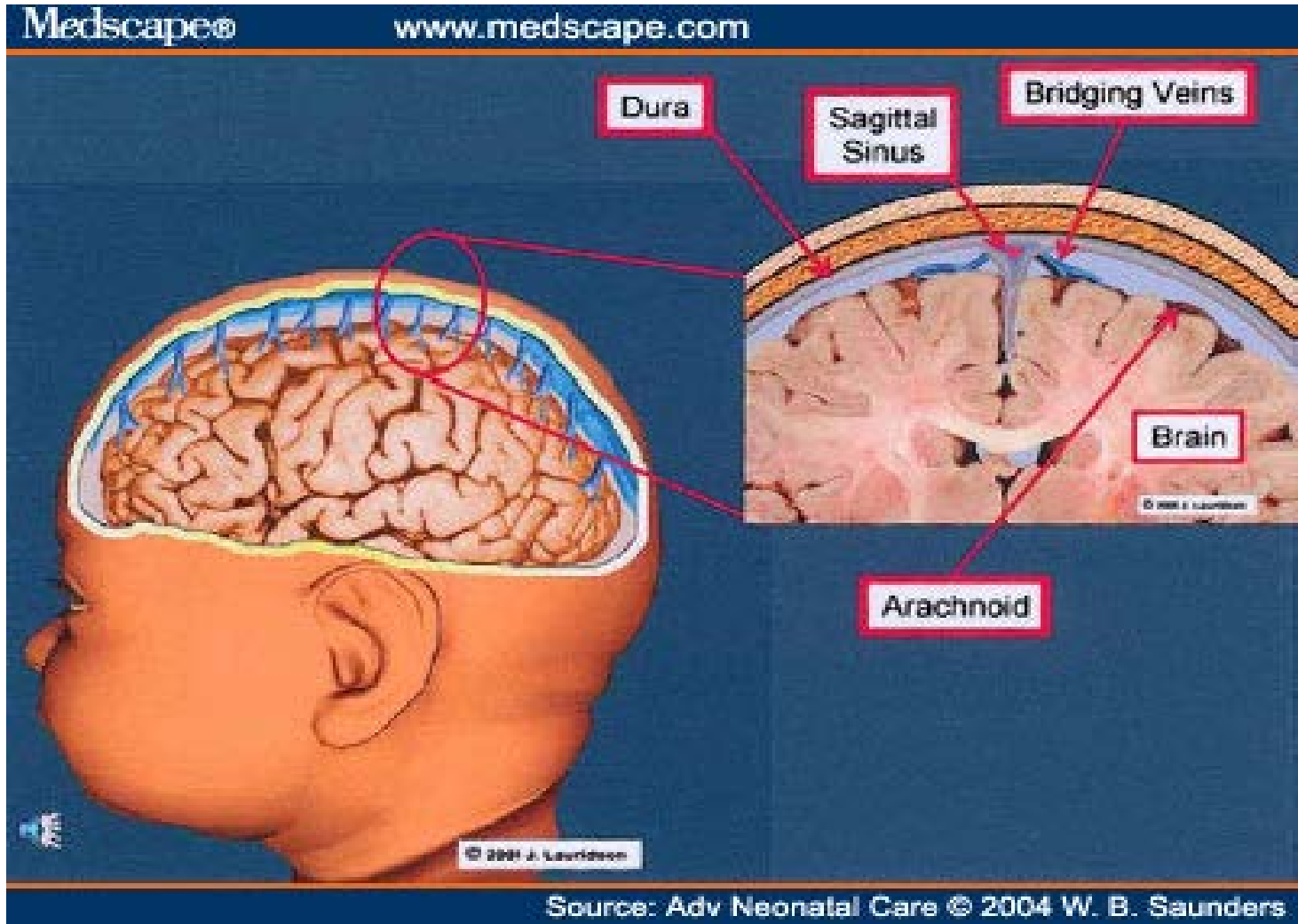
The brain's protective barriers (Section shown is from top, center of head)

Subdural Hematoma

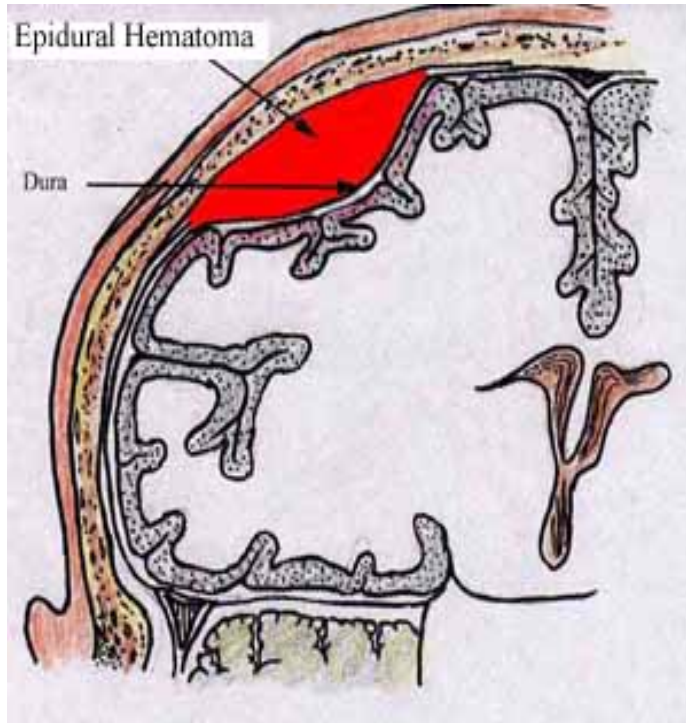


Bleeding between the arachnoid mater and the dura mater

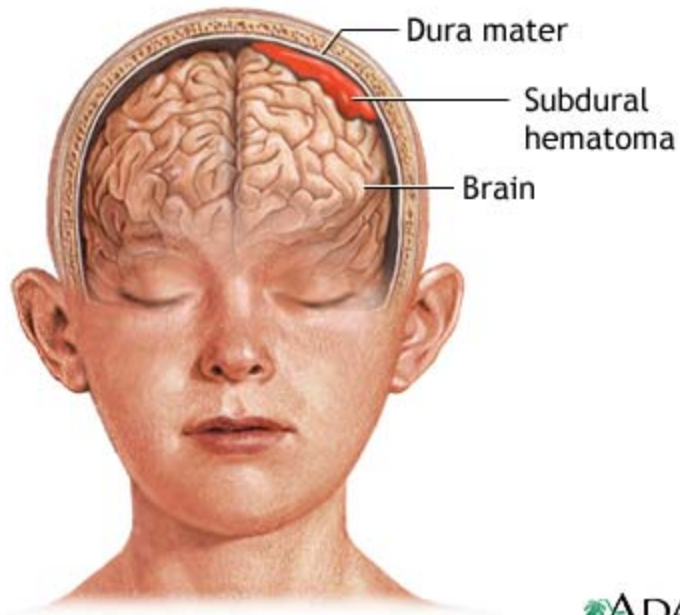
Different Anatomical Locations Of Hematoma



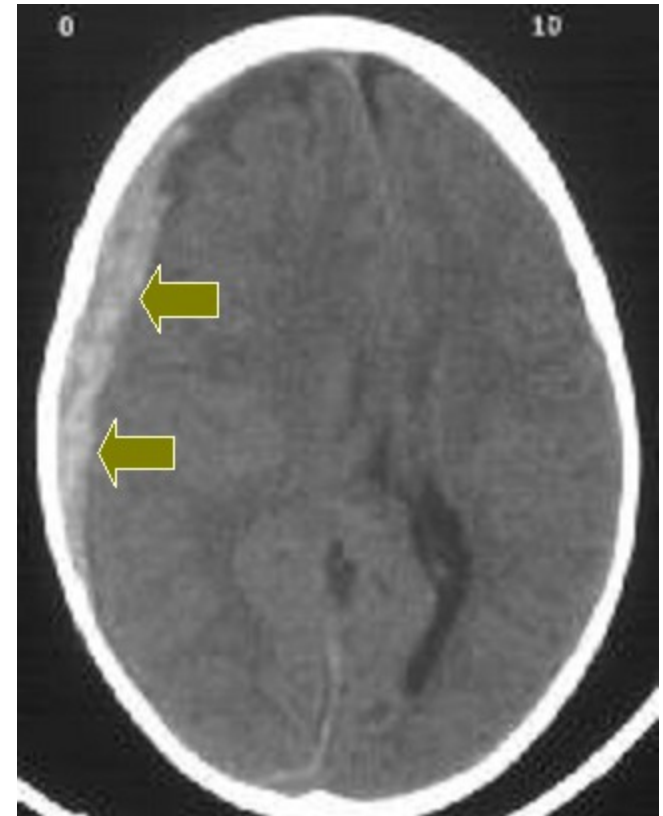
Epidural Hematomas



Subdural Hematomas



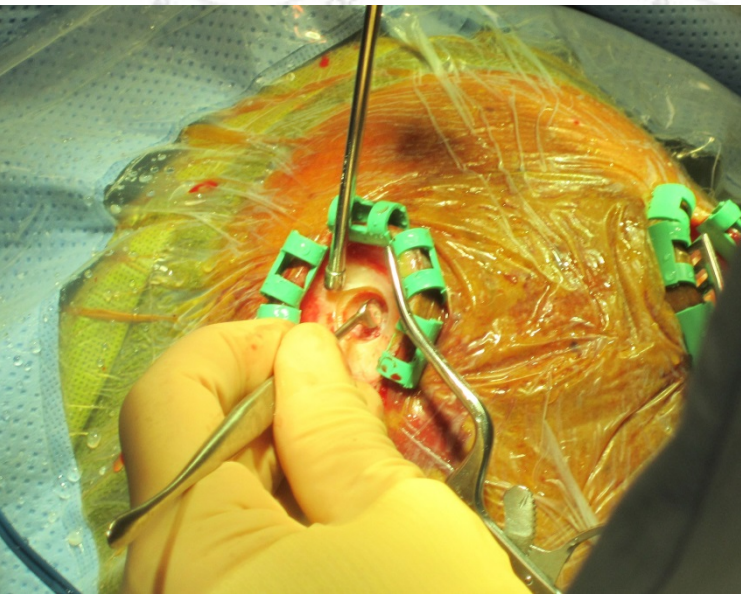
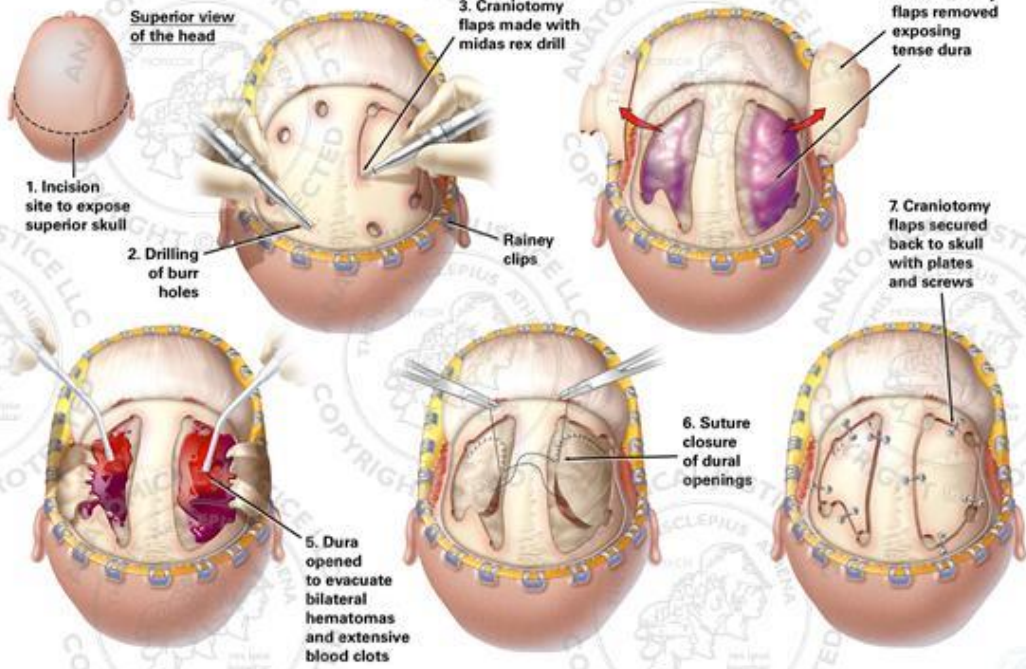
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Primary injury

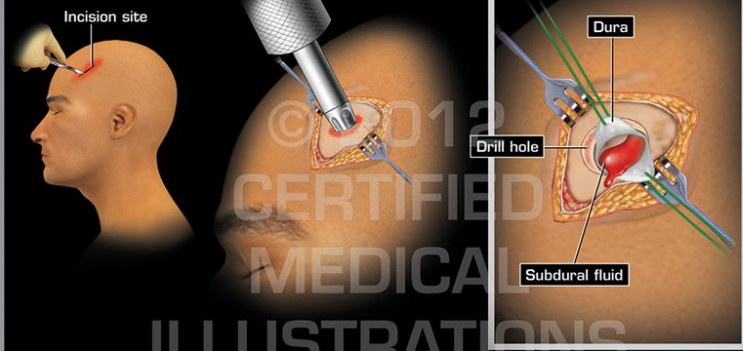
- Hematomas
 - Subdural hematomas (SDH)
 - Chronic SDH (subacute):
 - Symptoms appear after 2 day - 2 weeks of the trauma
 - Usually seen in elderly substance abuse with brain atrophy
 - Bleeding is slow therefore need longer time to experience neurologic depression
 - Treatment include burr holes, irrigation, insertion of catheter to drain the clot
 - Placing patient in supine position
 - Intracerebral Hematoma
 - Bleeding into the parenchyma tissue
 - Associated with depressed skull, penetrating injury, acceleration-deceleration injuries
 - Produce focal neurologic deficits related to their location
 - Surgical removal of the large clot is performed, smaller clots are left to reabsorb on their own

Bilateral Craniotomies and Evacuation of Subdural Hematomas

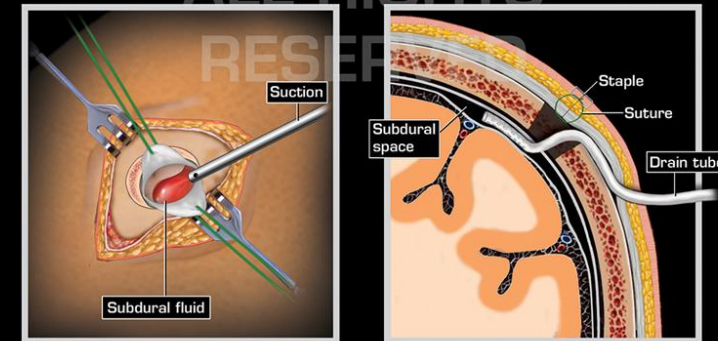


BURR HOLE FOR EVACUATION OF SUBDURAL HEMATOMA

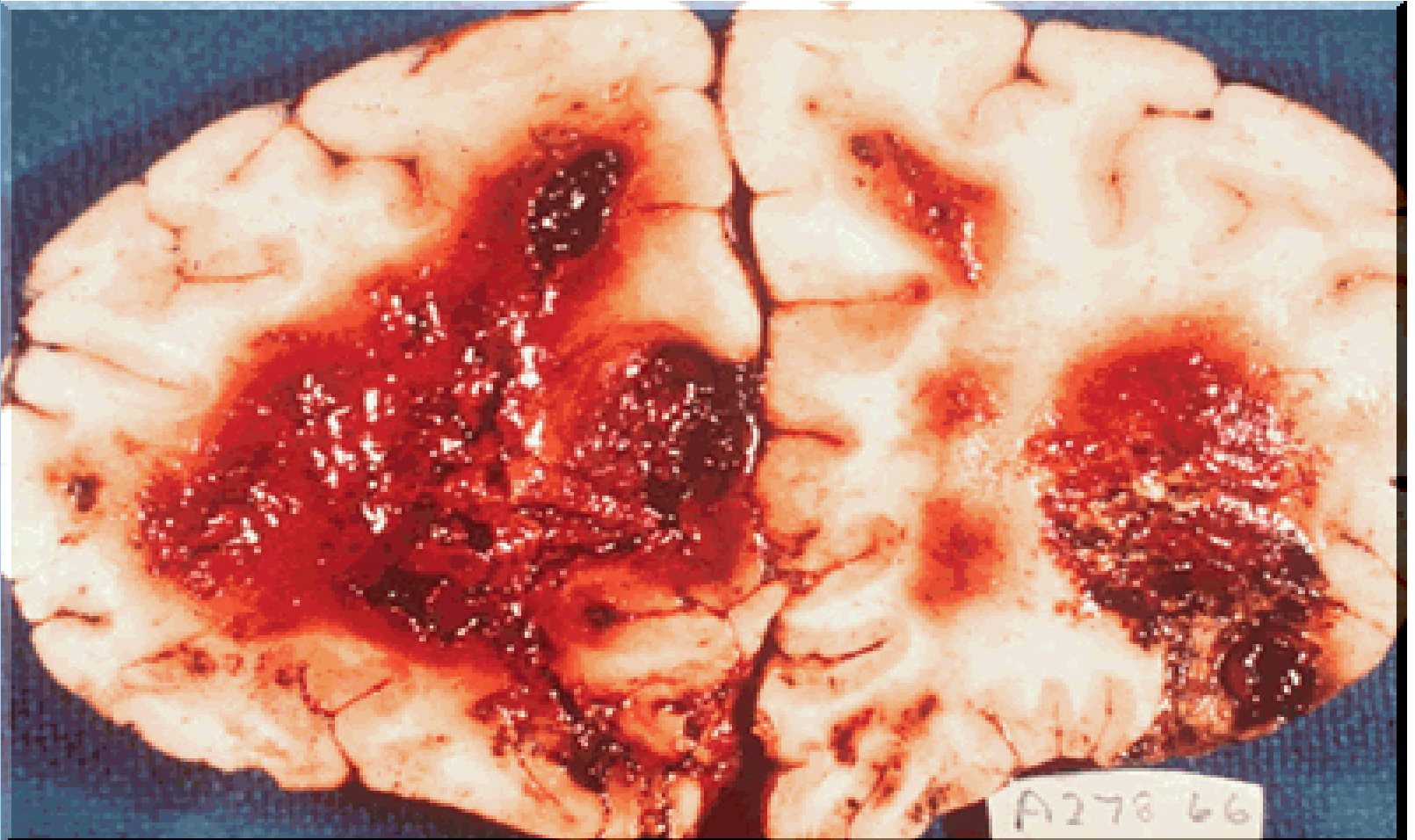
- A. Incision Site Over Left Frontal Area B. Drilling of Hole at Skull C. Opening of Dura



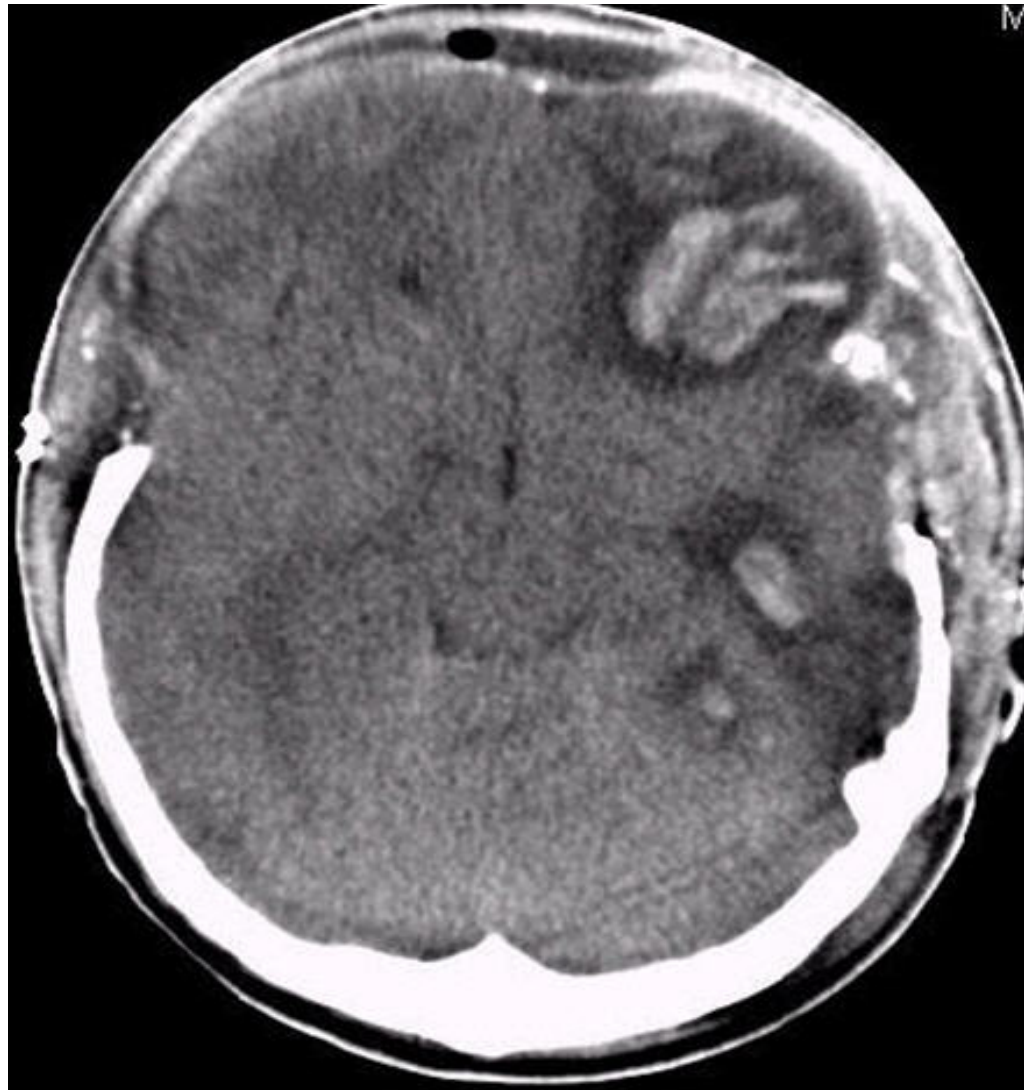
- D. Evacuation of Subdural Fluid E. Placement of Drain Tube



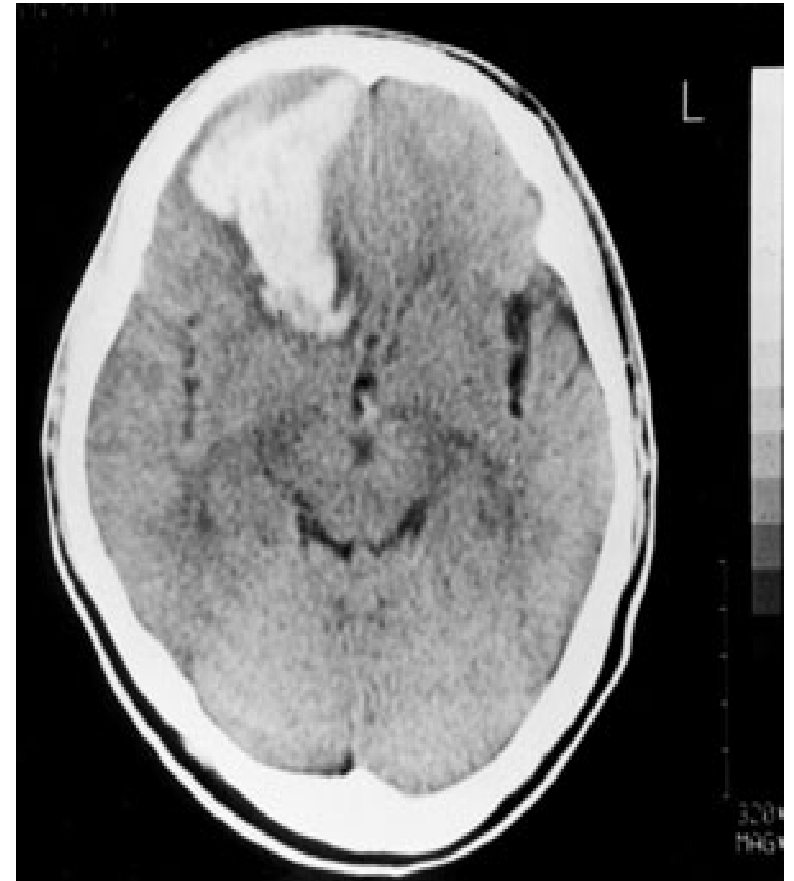
Cerebral Contusions



Cerebral Contusions



Intracerebral hematoma



Primary injury

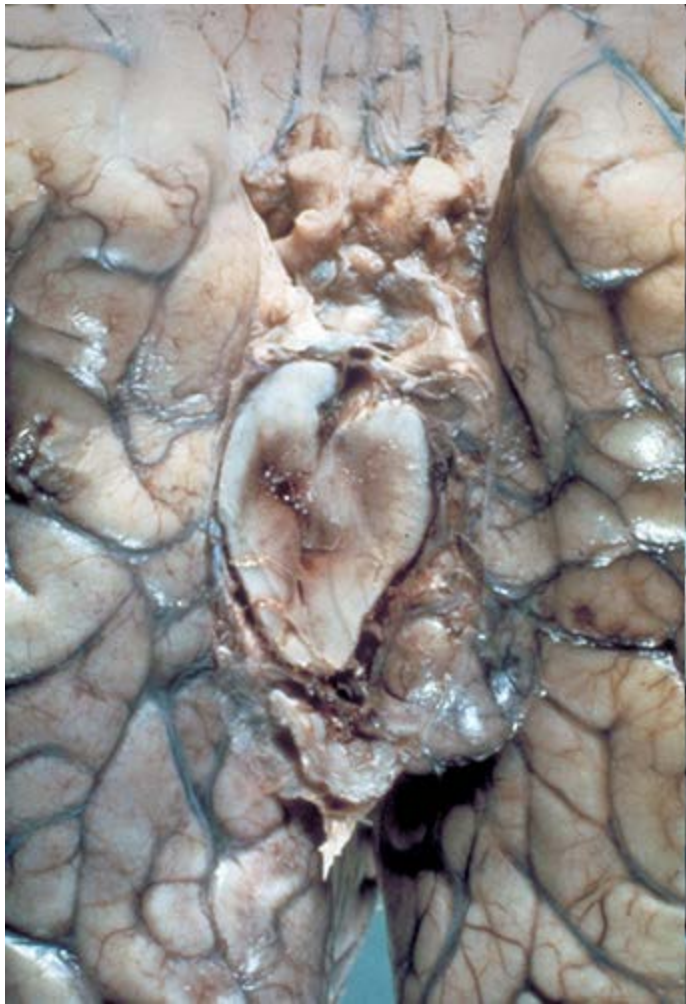
- Penetrating injuries
 - Mainly caused by gunshots
 - Controversy exists about surgical debridement of the wound and removal of the bullet
- Diffuse injuries
 - Cerebral concussion
 - Alteration of mental status as result of head trauma
 - Range from memory disturbances to alteration of level of consciousness
 - Not associated with structural abnormalities or radiographic imaging
 - Diffuse axonal injury (DAI): tearing and fraying of myelinated axons in the deep white matter
 - Mild DAI: coma less than 24 hrs
 - Moderate DAI: more than 24hrs transient flexor or extensor posture
 - Sever DAI: more prolonged with extensor posture
 - No special surgical treatment to be done to repair the injury
 - MRI is helpful
- Traumatic subarachnoid hemorrhages
 - Bleeding into the subarachnoid space resulting in vasospasm
 - Administering Nimodipine, Ca- channels blockers improve the outcomes for these patients

Secondary injury

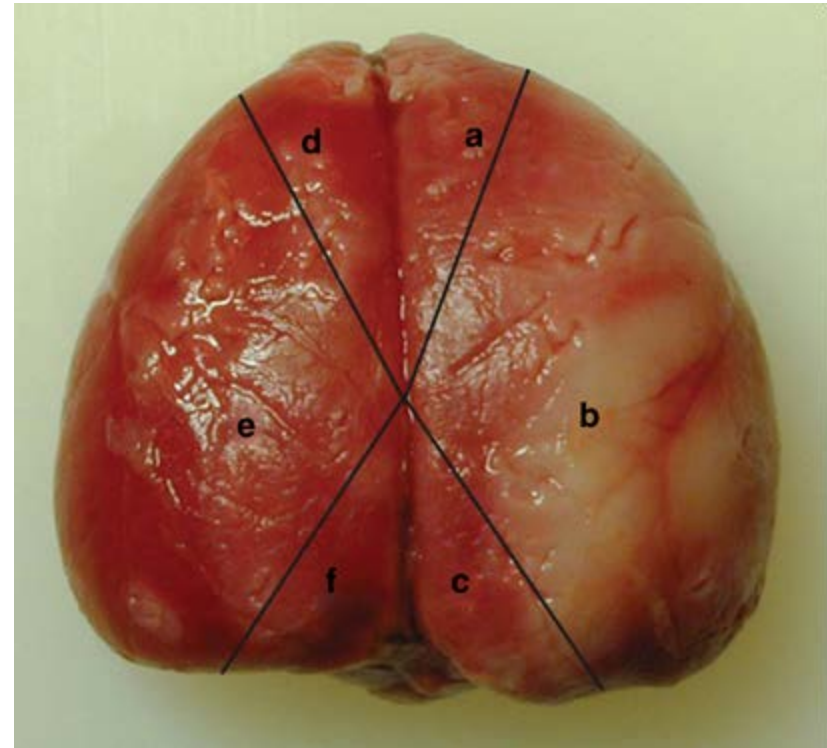
- Conditions that lead to further brain damage after the initial trauma or damage that caused by biophysical and biochemical process that result in alteration of cerebral perfusion
- E.g., uncontrolled increased ICP, cerebral ischemia, systemic hypotension, local or systemic infections
 - Hypotension: leads to decrease cerebral perfusion
 - Hypertension: leads to cerebral edema and increase ICP
 - CO₂ retention and hypoxia result in cerebral vasodilatation
 - Hyperoxygenation result in cerebral vasoconstriction
 - Normal CBF is about 50mL/100g of brain per min
 - Cerebral ischemia result when CBF drops to 10 -15 mL/100g per min
 - Death occurs when CBF drops to 10 -15 mL/100g per min

Secondary injury

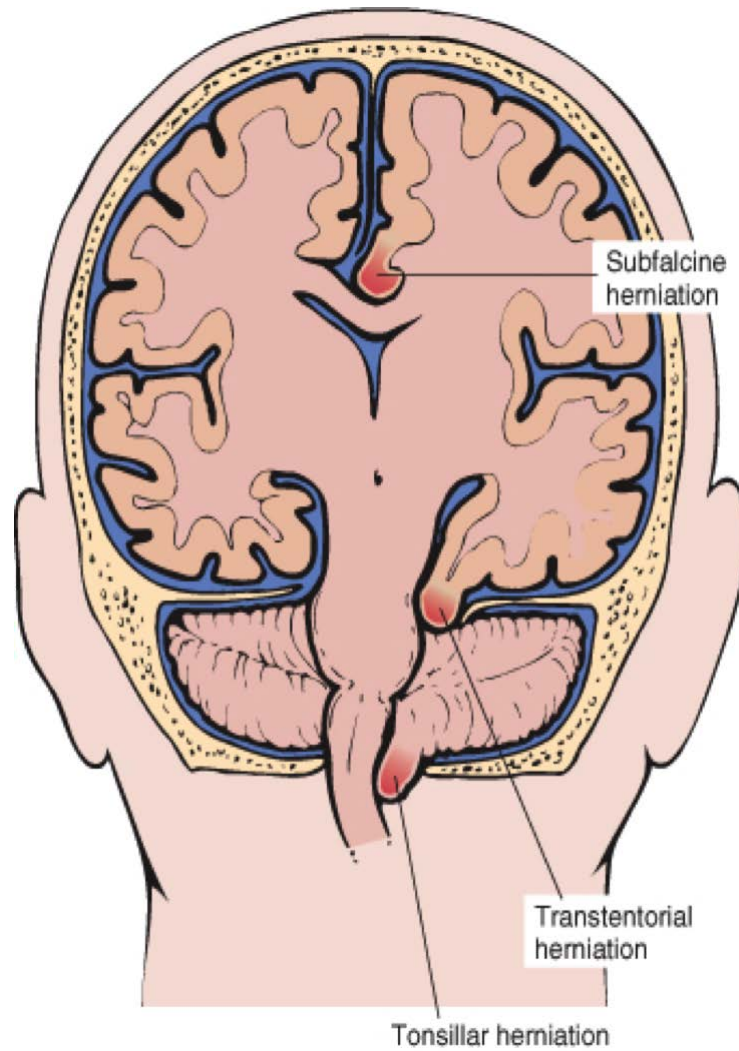
- Cerebral Edema
 - Commonly occurs 24 – 48 hrs after in the injury
 - Untreated edema may result in herniation syndrome
 - Cytotoxic edema: result from disruption of Na-K pump → allowing an influx of Na and water into brain tissue
 - Vasogenic edema: result from disruption of blood brain barrier
- Ischemia
 - Inadequate blood supply to the brain tissue to meet the metabolic demand
 - End result of ischemia is infraction & tissue death
- Herniation Syndrome
 - State in which cerebral structures shift inside the cranium under high pressure
 - Cushing's triad describes the late sings of herniation
 - The condition become the worst when the cerebral tissue shifted downward from foramen magnum (**cerebellar tonsillar herniation**)



Cerebral Herniation Syndrome



Cerebral Edema



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Cerebral Herniation Syndrome

Persistent Vegetative State

- Sleeplike coma followed by a return to the awake state but with a total lack of apparent cognition
- Permanent damage of the higher cortical function of the cerebral hemispheres with intact lower function of the brainstem
- Sleep-wake cycle is exist, open eye spontaneously and in response to verbal stimuli
- 4 weeks are needed after the brain injury to reach to this diagnoses

Assessment

- Signs and symptoms of traumatic head injury
 - Pupillary reactivity
 - GCS
 - Cognitive assessment for mild injury
 - Motor function assessment
- Laboratory and diagnostic tests
 - Skull x-ray: fractures
 - CT scan: mass lesions, edema, swelling, infarction, fractures
 - MRI: unexplained findings from other diagnostic test
 - CSF: chemistry and CBC

Nursing Diagnoses

- Altered cerebral tissue perfusion
- Decreased adaptive capacity, intracranial
- Ineffective airway clearness
- Impaired gas exchange
- Impaired verbal communication
- Impaired physical mobility

Collaborative Management

- **Airway management**
 - Intubation with adequate FIO₂ if needed
 - The goal is to keep PaO₂ = 100 mm Hg, PaCO₂ = 35 -45
 - Hypoventilation should be avoided because it leads to cerebral vasodilatation → increased ICP
 - Hyperventilation should be avoided to prevent cerebral vasoconstriction
 - Hyperoxygenation result in cerebral vasoconstriction → decrease CBF
- **Monitoring and controlling ICP**
 - ICP monitoring for patient with GCS less than 8
 - Allow chance for rapid and proper treatment options

Collaborative Management

- Maintaining Cerebral Perfusion
 - Maintain CPP greater than 60 mmHg
 - Decrease the ICP and improve MAP
- Prevent and Treat Seizures
 - Post-traumatic seizures are commonly occur in the 7-day period
 - Seizures have sever negative effects on ICP and cerebral metabolic demand
 - Phenytoin is commonly used → close monitoring for BP is needed → administered slowly
 - Fever should be avoided as possible
- Monitoring Fluid and Electrolyte Status
 - Hyponatremia is very common because of Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH) and cerebral salt-wasting syndrome
 - Hypotonic solutions should be avoided, they may cause cerebral edema
 - Hypotension should be aggressively treated with IV fluid
 - Glucose solutions should be avoided, they are associated with increased mortality

Collaborative Management

- Monitoring & treating cardiopulmonary complications
 - Hemodynamic monitoring → Systematic arterial blood pressure should not exceeds 130-140 mm Hg
 - Prophylaxis DVT
 - ARDS treatment
 - Prevent aspiration
- Ensuring Optimal Nutrition
 - Head injury cause hypermeabolic and hypercatabolic state
 - Enteral or parenteral feeding should be started within 7 days of the injury
 - 140% of resting energy expenditure is recommended for patient who is not paralyzed and 100% for paralyzed one

Collaborative Management

- Managing Musculoskeletal & Skin Complications
 - Neutral head position or elevation 30 degree
 - Splinting of the hand and feet
 - Physiotherapy and positionig
- Surgical management
 - The goal is to decrease the neurological damaged result from increased ICP
 - Craniectomy: removal of depressed bone to relieve the pressure on the brain tissue
 - Debridement and repair may be required

Brain death

- The brain death examination seeks to confirm the following three cardinal findings
 - Coma or unresponsiveness
 - Deep coma on response to any type of stimulus
 - Absence of brainstem reflexes
 - Doll's eye test
 - Caloric ice water test
 - Protective reflexes (cough, gag, & corneal)
 - Apnea
 - No chest movement when removed from the ventilator
 - PaCO₂ after 8 minutes → if greater than 60 mmHg or more than of 20mmHg of the baseline
- EEG results

Goodbye!



The End



Thank You!

