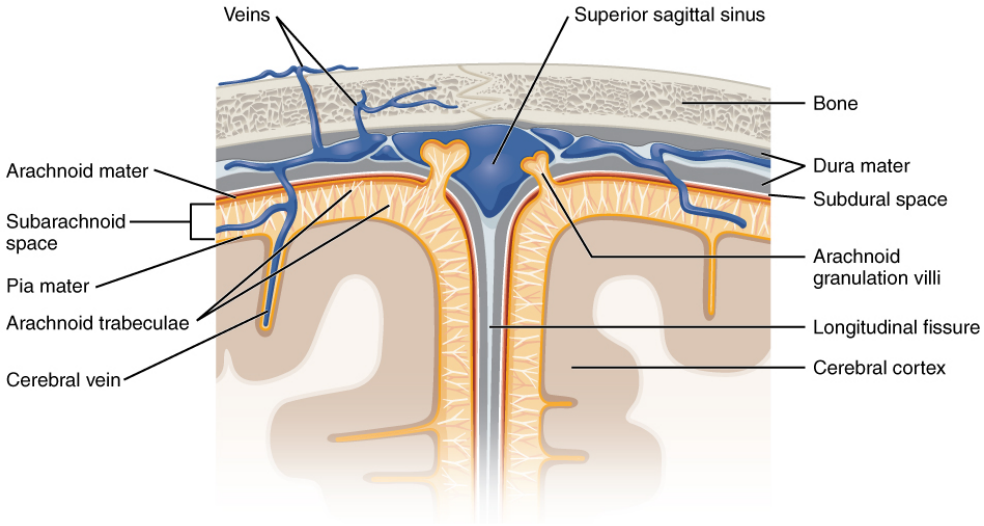
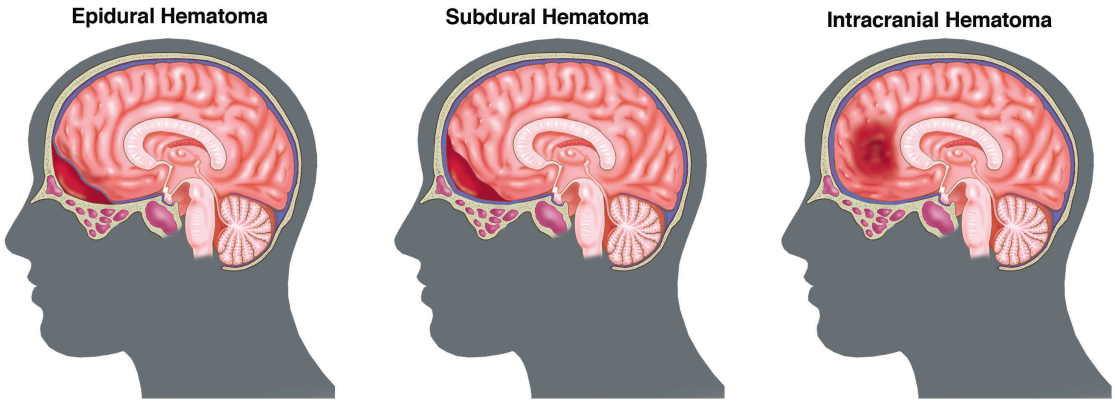
Traumatic Brain Injury (TBI)

I. Background
   a. Estimated 10 million cases leading to hospitalization or death each year[1]
   b. Top three causes of TBI; car accident, firearms, and falls.

II. Pathophysiology
   a. Normal ICP=15 mmHg
      i. Transient increases are due to coughing or sneezing
      ii. The total volume within the skull remains constant and is determined by the sum of the CSF, blood and Blood flow
      iii. Layers of the intracranial space
         1. Skin
         2. Bone
         3. Dura Mater
         4. Subdural space



   b. Abnormal is ICP=20 mmHg
      i. Increased pressure due to increased volume may cause displacement of CSF to the spinal subarachnoid space and compression of the cerebral venous bed.
      ii. The increase in volume and intracranial pressure is exponential, and may present with rapid deterioration when the compensatory mechanism are exceeded.

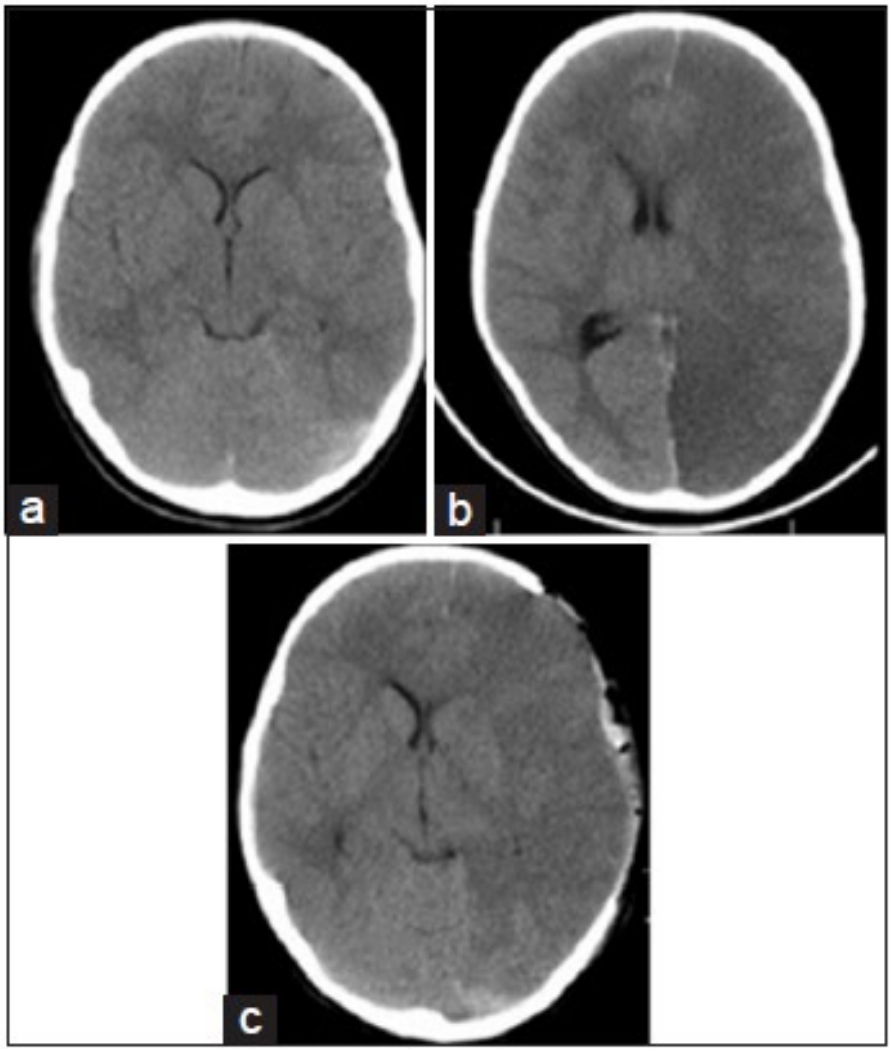


c. Types of injuries
    i. Open head injury
        1. GSW, penetration of the skull
        2. Large focal damage
    ii. Closed head injury
        1. Falls, MVCs
        2. Focal damage with diffuse damage to axons
    iii. Deceleration injuries
        1. Sudden transition in acceleration can result in axonal shearing
        2. Massive axonal shearing and neuron death
    iv. Chemical/ Toxic
    v. Hypoxia
    vi. Tumors
    vii. Infections
    viii. Stroke

III. Assessment
    a. Physical
        i. Symptoms
            1. Mild- Headache: visual disturbances, seizures, N/V
            2. Moderate-severe: sustained LOC, dilation of one or more pupils, inability to wake
    b. Neurological
        i. Glasgow Coma Scale (GCS)
            1. Takes into account motor response, verbal response, eye opening
            2. Scores
                a. 13-15: Mild
                b. 9-12: Moderate (LOC)
                c. 3-8: Severe (Coma)
        ii. Ranchos Los Amigos
            1. Scale 1-8 (with 1= no response)
            2. Not typically used at JHH

IV. Diagnosis
    a. CT scan

i. Increased intracranial pressure may cause distortion of the brain tissue and cause a midline shift or displacement of the brain tissue via herniation
    1. Herniation is a medical emergency!



V. Treatment
    a. Depends on the injury
        i. Intracranial causes (hematomas, contusions) will require surgical evacuation, decompressive craniectomy
        ii. Intracranial edema and increased ICP may be treated with hyperosmolar fluids or craniectomy
        iii. Extracranial causes (airway obstruction, fever, hypertension associated with pain) may be treated with oxygenation, antipyretics, sedation, etc)
    b. Oxygenation
        i. Goal PaO2 >60 mmHg or O2 sat >90%
    c. Blood Pressure
        i. Systolic pressures <90 mmHg should be avoided
    d. Hyperosmolar therapy
        i. Mannitol

1. Mechanism
    a. Immediate plasma expanding effect→ reduces hematocrit→reduces blood viscosity→increases cerebral blood flow→increases O2 delivery
2. Effects: Mannitol causes systemic diuretic effects that pulls fluid into the cerebral vasculature and may decrease the cerebral perfusion pressure (CPP)
3. Doses: 0.25-1 g/kg (per guidelines)

ii. Hypertonic saline
1. Mechanism:
    a. Osmotic mobilization of water across the blood brain barrier→ draws water out→ reduces intracranial pressure
2. Effects: Unlike mannitol, Hypertonic saline does not cause systemic vascular expansion and maintains CPP
3. Place in therapy: The use of 2 to 23.4 % saline has been studied. Hypertonic saline was shown to be superior to mannitol in reducing ICP; however, patients also received hetastarch of dextran with the saline solution. So, the effects are unclear.[2]
4. Dosing:
    a. Bolus- 5ml/kg of 3%
5. Side effects:
    a. Pontine myelinolysis occurs due to the rapid increase in serum sodium levels.
        i. Unlikely if sodium levels increase less than 12 meq/L in 24 hours

iii. Prophylactic Hypothermia
1. Not associated with decreased mortality but may decrease ICP

iv. Infection prophylaxis
1. To prevent infection of the ICP monitors (occurs in <1-27%), not supported by evidence.

v. DVT prophlyaxis
1.