





Superior sagittal sinus

Superior cerebral veins (penetrating arachnoid and passing through subdural space to enter superior sagittal sinus)

Dura mater —

Superior cerebral veins (beneath arachnoid)



Middle meningeal artery and veins

- Superior anastomot vein (of Trolard)

Inferior anastomoti vein (of Labbé)

Superficial middle cerebral vein

Inferior cerebral veins (beneath arachnoid)











Case 1

Ddx

Epidural Hematoma Subdural Hematoma Concussion

Subdural Hematoma

- Bleeding within meninges
 - Beneath dura mater and/or within subarachnoid space
 - Above pia mater
- Slow bleeding
 - Superior sagital sinus
- Signs progress over several days or weeks
 - Slow deterioration of mentation
- More common than epi's, esp in elderly, alcoholics









Case 2

Ddx

Epidural Hematoma Subdural Hematoma Concussion

Epidural Hematoma





Epidural Hematoma

- Bleeding between dura mater and skull
- Involves arteries
 - Middle meningeal artery most common
- Rapid bleeding & increased pressure = reduction of oxygen to tissues
- Herniates brain toward foramen magnum



Epidural hematoma











Cerebral Cortex/Upper Brain Stem

- BP ↑, HR ↓
- Pupils reactive
- Cheyne-Stokes resps
- First localizes stimuli, then withdraws, then flexion (decorticate)
- Can be reversed at this stage!

Middle Brain Stem

- HR ↓ ↓, BP ↑ ↑
 - Wide PP, bradycardia
- Pupil nonreactive or sluggish
- Extension (decerebrate)
- Seizures
- Good luck. Poor outcome.

Lower BS/Medulla

- Blown pupil
- Ataxic respirations
- Flaccid
- Fluctuating HR
- Decreased, fluctuating BP
- Not survivable

What Kills The Head Injured Patient?

- Unmanaged airways and inadequate ventilation
- Uncontrolled ICP
- Decreased cerebral perfusion

How Can We Decrease Morbidity & Mortality in Head Injured Patients?

- Open & clear Airway. Keep it so.
- Ventilate and Oxygenate
 - Ventilate at 20/min if signs of severe head injury present
- Elevate head to 30°
- Drive real fast to trauma center

Case 3





Diffuse Brain Injury

- Due to stretching forces placed on axons
- Pathology distributed throughout brain
- Types
 - Concussion
 - Moderate Diffuse Axonal Injury
 - Severe Diffuse Axonal Injury



Mild Diffuse Axonal Injury

- Concussion
 - Nerve dysfunction without anatomic damage
- Transient episode of
 - Confusion, disorientation, event amnesia
- Management
 - ABC's, c-spine considerations!
 - ALS!
 - Frequent reassessment of mentation

Moderate Diffuse Axonal

- "Classic Concussion", 45 % of all DAI
- Same mechanism as concussion
 Additional: Minute bruising of brain tissue
- Unconsciousness
 - If cerebral cortex and RAS involved

Signs & Symptoms

- Unconsciousness or persistent confusion
- Loss of concentration, disorientation
- Retrograde/antegrade amnesia
- Visual and sensory disturbances
- Mood or Personality changes
- Management:
 - Same as mild DAI
 - Watch for worsening!

Severe Diffuse Axonal Injury

- Brainstem Injury
- Significant mechanical disruption of axons
 - Cerebral hemispheres and brainstem
- High mortality rate
- Signs & Symptoms
 - Prolonged unconsciousness
 - Cushing's reflex
 - Decorticate or decerebrate posturing

- Management
 - C-spine considerations
 - ABC's
 - Airway a priority!
 - Hyperventilate 20/min, normal TV!

-ALS





Cranial Injury

- Basilar Skull Fracture
 - Tear Dura Mater
 - Permit CSF to drain through an external passageway
 - May mediate rise of ICP
 - Evaluate for "Target" or "Halo" sign









Cranial Injury

- Basal Skull Fracture Signs
 - Battle's Signs
 - Retroauricular Ecchymosis
 - Associated with fracture of auditory canal and lower areas of skull
 - Raccoon Eyes
 - Bilateral Periorbital Ecchymosis
 - Associated with orbital fractures



Retroauricular ecchymosis (Battle's sign).

Bilateral periorbital ecchymosis (racoon eyes).



Management: Skull Fracture

- C-spine precautions
- ABC's
 - OPA, NPA?
 - BVM/ET if necessary
 - Rate? TV?
- ALS
- Do not push on skull!
- Leave impaled objects in place!
 Secure adequately





Brain Injury: Causes

- Direct
 - Primary injury caused by forces of trauma
- Indirect
 - Secondary: caused by edema, hemorrhage, infection, pressure, and inadequate perfusion
 - Tertiary: caused by apnea, hypotension, pulmonary resistance, and dysrhythmia

Mechanism For Brain Injury

- ICP starts to increase
- As ICP increases, cerebral blood flow decreases
 - Swelling compresses arteries = \downarrow CBF = \downarrow O₂
 - Cell death = edema around necrotic tissue
 1 ICP
 - $-CO_2$ accumulation = vasodilation = $\uparrow \uparrow ICP$

Levels of Increasing ICP

- Cerebral cortex/upper brain stem
- Middle brain stem
- Lower brain stem/medulla



Cerebral Cortex/Upper Brain Stem

- BP ↑, HR ↓
- Pupils reactive
- Cheyne-Stokes resps
- First localizes stimuli, then withdraws, then flexion (decorticate)
- Can be reversed at this stage!

Middle Brain Stem

- HR ↓ ↓, BP ↑ ↑
 - Wide PP, bradycardia
- Pupil nonreactive or sluggish
- Extension (decerebrate)
- Seizures
- Good luck. Poor outcome.

Lower BS/Medulla

- Blown pupil
- Ataxic respirations
- Flaccid
- Fluctuating HR
- Decreased, fluctuating BP
- Not survivable

What Kills The Head Injured Patient?

- Unmanaged airways and inadequate ventilation
- Uncontrolled ICP
- Decreased cerebral perfusion

How Can We Decrease Morbidity & Mortality in Head Injured Patients?

- Open & clear Airway. Keep it so.
- Ventilate and Oxygenate
 - Ventilate at 20/min if signs of severe head injury present
- Elevate head to 30°
- Drive real fast to trauma center