Traumatic Head Injuries
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Traumatic Brain Injury (TBI)
- Major contributing cause of trauma deaths
- Many survivors have permanent disability
- Commonly occurs in young adults (mostly males)
- Many deaths can be prevented by early basic airway management

Anatomy

Skull
Dura mater
Arachnoid membrane
Pia mater
The Brain

Pathophysiology

• Primary brain injury
• Secondary brain injury
Secondary Brain Injury

- Hypoxia
  - O2, suction, secure airway
- Hypotension
  - Control bleeding, give fluids
- Increased or decreased CO₂
  - Secure airway, Ventilate appropriately
- Increased or decreased blood glucose
  - Check blood sugar, give NS or LR-not D5

Secondary Brain Injury

- Intracranial causes
  - Seizures
    - Treat with Ativan, valium, versed, phenobarb, phenytoin, protect airway
  - Cerebral edema/increased ICP
    - Treat with Mannitol, surgery, hypertonic saline, hyperventilation?
  - Hematomas
    - Surgery

Skull Fractures

- Simple linear fracture
- Basilar skull fracture
- Open skull fracture
- Depressed fracture
Linear Skull Fracture

- Most common type of skull fracture
- Probably will not see on clinical exam
- Often occur without scalp laceration

Basilar Skull Fracture

- Associated with major impact trauma
- Linear fractures can extend into base of skull becoming basilar fractures
- Clinical findings:
  - Raccoon’s eyes & Battle’s sign
    - Don’t show up for several hours after injury
  - Blood behind tympanic membrane
  - CSF leakage from ears

Basilar Skull Fracture

- Raccoon’s Eyes
  - Result from fracture of the base of the sphenoid sinus

Black eyes that are visible immediately after trauma are more likely the result of direct facial trauma
Battle’s Sign

Ecchymosis over the mastoid process
Caused by fracture of the temporal bone

Open Skull Fracture

High energy transfer causes opening through skull into cerebral contents
High complication rate & mortality
- Infection
- Requires surgical intervention

How will you manage this injury?
Depressed Skull Fracture

- Focused, high energy blow to head
- Commonly have associated scalp laceration with bleeding

Intracranial Hypertension

- Cerebral perfusion pressure
  - CPP = MAP – ICP
  - If ICP increases and MAP is unchanged, then CPP drops
  - Body responds to increased ICP by increasing MAP (Cushing reflex)

Intracranial Hypertension

If ICP is too high (or CPP too low), blood and oxygen can’t get to brain cells
Early Signs of Increased ICP

- Headache, Nausea & vomiting
- Altered LOC (Decreased GCS)

Brain Herniation

Herniation occurs when extremely high ICP “pushes” the brain stem through the opening in the base of the skull.

Signs of Possible Herniation

- Dilated, unreactive or unequal pupils
- Pt. becomes unresponsive (GCS score drops)
Signs of Possible Herniation

• Cushing’s triad: Very bad sign!
  1. Increased systolic BP (with widening pulse pressure)
  2. Decreasing HR
  3. Irregular respirations

• Abnormal posturing

Abnormal Posturing

Decorticate posturing

Decerebrate posturing

Management

• C-Spine immobilization
  – Not necessary for penetrating trauma
• Ensure patent airway
  – Have suction ready
• Oxygen-monitor O2 saturations
  – BVM assist if necessary
• IV if able
• Check blood sugar if able
Management

Management options for increasing ICP:
• Elevate head
• Sedation
• Chemical paralysis
• Osmotherapy (mannitol/hypertonic saline)
• Normal ventilation for most

Avoid over-ventilating patients

Management

• What does hyperventilation do?
  – Cerebral vasoconstriction
  – Effective at decreasing ICP at the expense of cerebral perfusion
• Only appropriate for temporary measure for acute increased ICP (patient herniating)

Two Important Things to Avoid

• Hypoxemia
  – Even one episode of hypoxemia increases mortality
• Hypotension
  – What is ideal BP?
    • Some ongoing studies suggest SBP somewhere around 130 (MAP >65)
Patient #1: Me

Your patient is a 30-year-old softball player who fell over backwards while backpedaling for a fly ball.

Bystanders report a 1-minute loss of consciousness.

Patient #1: Me

He is now awake, restless & complaining of a headache and nausea. He is repeating the same questions over & over.

A - Open
B - Normal
C - Normal
D - GCS score 14 (E-4, V-4, M-6), PERL

Patient #1: Me

Transported to ER
Placed in C-spine immobilization (eventually)
CT scan negative

Classic concussion presentation

Prognosis?
You are called to a local bar where a 22-year-old male (Jimmy) has been in a fight. Bystanders state that your patient was hit on the side of the head with a pool cue. The scene is safe.

The fight was about 1 hr ago. He was briefly knocked out, then woke up and seemed to be fine (acting drunk & agitated), so nobody called an ambulance. Now he is unresponsive with snoring respirations.

The classic epidural hematoma presentation:
- Initial LOC
- Lucid period
- Unresponsive
- A time-critical surgical emergency!

Prognosis? 80-85% survival
**Epidural Hematoma**

Caused by tear of middle meningeal artery

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**Patient #3**

- 83 year old female
- Fell in shower two days ago
- Complaining of increasing headache
- Became nauseated, increasingly confused with decreased LOC

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**Comparison of epidural, subdural and intracerebral hematomas**
Patient #3

- His GCS is 9 (E=2, V=2, M=5)
- HR 96, RR 16, BP 190/80
- Left pupil dilated
- SaO2 97% on NRB mask

Presentation consistent with:
Subdural Hematoma

Patient #4

You are called to the scene of a rollover MVC where a 24-year-old male was ejected from the vehicle. You see the patient lying supine on the ground. His breathing is noisy and slow. He has a large scalp laceration. You identify no hazards.

Primary Survey

A - Snoring, gurgling noises
B - RR slow, irregular and shallow; BS decreased bilaterally
C - Moderate bleeding from scalp; slow, bounding carotid pulse
D - GCS score 5 (E-1, V-1, M-3)

Vitals: RR 8 & irregular; pulse 52; BP, 188/110

Cushing’s Triad
Diffuse Axonal Injury

Axon

A. Trauma causes the axon to twist and tear
B. The result is permanent death of the brain cell

Questions?

Special thanks to Sheila Crow of Stitchin’ Dreams Embroidery

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For providing our Secret Question prize
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