Traumatic Brain Injury

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75% of all pediatric trauma hospitalizations are due to head injury
80% of all pediatric trauma deaths are associated with significant neurologic injury
Trauma is the leading cause of death in children > 1 year old.
Mortality rate in severely head injured patients 9 - 35%
Approx. 20% of non-accidental trauma includes head injury (mostly < 2 y/o)
Introduction (con’t)

• Incidence of 200-300/100,000 per year
• Cost $7.5 billion/year in USA
• Multiple etiologies
  – motor vehicle accident (most common)
  – non-accidental trauma
  – falls (2nd most common)
  – flying objects (includes bullets)
Differences in Pediatric vs. Adult Population

- Child’s brain has more water content
- Head makes up higher percentage of body weight
- More prone to hyperemia and edema formation
- Children have higher cerebral metabolic rate
  - more susceptible to hypoxia/secondary injury
- more vasoreactivity
- Brain has more gelatinous consistency and skull more malleable
  - allows more considerable movement and distortion of internal contents
Differences in Pediatric vs. Adult Population II

• Younger children (< 2 y/o) tend to suffer greater damage than older children or adults from similar injuries
• Children < 1 year old have higher morbidity and mortality
Classification of Injuries

- Open or Closed
- Skull fracture
  - linear, comminuted, depressed vs. non-depressed, diastatic fracture, basilar skull fracture, sinus fracture, orbital fracture, laforte type fractures
- Direct vascular trauma
  - large vessel dissection and subsequent thrombosis
- Pure hypoxic injury (s/p cardiorespiratory arrest)
Intracranial Hemorrhage and Mass Lesions

- Subdural hematoma
  - Common
  - usually associated with mild to severe diffuse parenchymal injury
  - tearing/avulsion of bridging veins
  - Lacks lucid interval
  - Prognosis may be worse than epidural
  - may require surgical intervention
Subdural hematoma

- is venous in origin (bridging veins)
- may be associated with a reasonable outcome if removed early
Subdural hematoma

- usually arise from the bridging veins
- bridging veins are more susceptible to tearing when there is cortical atrophy
Subdural Hematoma
Intracranial Hemorrhage and Mass Lesions

- Epidural hematoma
  - usually associated with skull fracture and laceration of a dural artery
  - may have lucid interval followed by rapid deterioration
  - mechanism of injury may not seem severe
  - may require urgent surgical intervention to prevent herniation/death
  - prognosis generally good if appropriately evacuated
Epidural hematoma

- is arterial in origin
- middle meningeal artery is torn
- often is a true neurosurgical emergency
Epidural Hematoma
Subdural vs. epidural
Intracranial Hemorrhage and Mass Lesions

• Intracerebral hematoma
  – represents a vascular injury within the parenchyma
  – may be single, but usually multiple
  – commonly associated with significant parenchymal damage/injury
  – usually small; do not require surgical intervention
  – may be described as “punctate hemorrhages” on CT scan
Intracerebral Hemorrhage
Intracranial Hemorrhage and Mass Lesions

- Subarachnoid hemorrhage
  - also a result of vascular injury
  - may see subarachnoid or intraventricular blood
  - rarely require surgical intervention
  - may result in hydrocephalus (early or late) that requires ventricular drainage
Intracranial Hemorrhage and Mass Lesions

- **Diffuse Axonal Injury (DAI)**
  - pathologic term used to describe widespread cerebral damage at time of impact
  - result of laceration, compression, or stretching and shearing of axons
  - acceleration/deceleration type of injury
  - common in MVA; uncommon in falls
  - results in significant white matter damage
Coup - contracoup injury

- a fall backwards resulted in bilateral injury
- inferior frontal and temporal lobes
Coup - contracoup injury
Definitions

• Primary brain injury - occurs at time of initial impact
• Secondary brain injury - result of blood supply inadequate to meet cerebral metabolic demands
• All therapy aimed at preventing and minimizing secondary brain injury
Systemic Effects of Brain Injury

- Marked ↑catecholamine release from CNS
- Unstable cardiovascular status and possible myocardial injury
- SIADH or DI
- Neurogenic Pulmonary edema - uncommon
- DIC - release of brain stores of thromboplastin
  - associated with increased mortality
Brain’s Response to Injury

- Development of edema
  - cytotoxic vs. vasogenic
- loss of autoregulation (vasospasm or hyperemia)
- Increase in ICP evolves over hours to days
  - usually peaks at 24 - 96 hours post injury, but may last 3 - 10 days
- If secondary brain injury not prevented, a vicious cycle of deterioration ensues
If cycle not broken......
General Principles

• Must maintain adequate cerebral blood flow (CBF) and cerebral perfusion pressure (CPP)
• CPP = MAP - ICP
• CBF normally constant between MAP 40 -140 mmHg (autoregulation)
• There is variable loss of autoregulation with head trauma
• Generally, maintain ICP < 20 and CPP ≥ 55
• MAP and blood viscosity - 2 most important factors to maintain CBF with impaired autoregulation
General Principles (con’t)

• PREVENT SECONDARY INJURY
  – secondary injury related to cerebral ischemia
  – *early* recognition and treatment of non-neurologic injuries may affect outcome
  – AVOID hypoxia, hypercarbia, hypovolemia, hypotension
Components of Intracranial Space

- 3 non-compressible substances
  - brain 80%
  - blood 10%
  - CSF 10%

- Total intracranial volume is constant
  - any increase in the volume of one component must cause a decrease in the volume of another
Monroe-Kellie

ICP

20

Intracranial Volume
Initial Assessment

- History from witnesses/EMT’s
  - mechanism of injury, LOC, neurologic changes, GCS
- ABC’s with mild hyperventilation
- C-spine immobilization
- After CV and Resp status are stabilized, complete trauma related survey
  - identify obvious injuries
  - look for: hemotympanum, oto/rhinorhea, scalp/facial wounds, periorbital changes, palpation of fontanelle
## Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eye Opening</th>
<th>Verbal</th>
<th>Motor Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>Oriented</td>
<td>Obeys Commands</td>
</tr>
<tr>
<td>To Speech</td>
<td>Confused Conversation</td>
<td>Localizes Pain</td>
</tr>
<tr>
<td>To Pain</td>
<td>Inappropriate Words</td>
<td>Withdraws to Pain</td>
</tr>
<tr>
<td>None</td>
<td>Incomprehensible Sounds</td>
<td>Abnormal flexion</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>Abnormal extension</td>
</tr>
</tbody>
</table>

Total Score = 3 - 15
Glasgow coma scale (modified for young children)

- best verbal response (1-5)

  1 none
  2 restless, agitated
  3 persistently irritable
  4 consolable crying
  5 appropriate words, smiles, fixes + follows
# Children’s Coma Score

<table>
<thead>
<tr>
<th>Ocular Response</th>
<th>Motor Response</th>
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</thead>
<tbody>
<tr>
<td>Pursuit</td>
<td>Flexes and extends</td>
</tr>
<tr>
<td>EOM intact, reactive pupils</td>
<td>4</td>
</tr>
<tr>
<td>Fixed pupils, EOM impaires</td>
<td>Withdraws</td>
</tr>
<tr>
<td>Fixed pupils, EOM paralyzed</td>
<td>Hypertonic</td>
</tr>
<tr>
<td></td>
<td>Flaccid</td>
</tr>
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<td></td>
<td>1</td>
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<table>
<thead>
<tr>
<th>Verbal Response</th>
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<tbody>
<tr>
<td>Cries</td>
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<tr>
<td>Spontaneous respirations</td>
</tr>
<tr>
<td>Apneic</td>
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<tr>
<td></td>
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“Mini” Neurological Exam

- Level of consciousness
- Pupils
- EOM
- Fundus exam
- Extremity movement
- Response to pain
- DTR’s
- Plantar reflexes
- Brainstem reflexes
- GCS

*Should be done ASAP after ABC’s and take only a few minutes.*
Radiographic Studies and Lab Tests

- Plain Skull films
- C-spine series
- Head CT
  - indication: altered LOC, focal deficit, persistent headache or emesis, penetrating injury, seizure, history of LOC
  - many lesions may not be seen for 24-48 hours
- Cerebral angiography and MRI - usually not indicated
- Type and Crossmatch blood - only essential lab test
- Ultrasound/Doppler Flows of Carotid Arteries
Goals of Monitoring and Treating ↑ICP

- Prevent secondary injury by maintaining adequate CPP/CBF
- Prevent herniation
- Recognize and treat adverse events quickly
ICP Monitoring

• Indications:
  – GCS ≤ 8
  – Rapid deterioration of neurologic status
  – Unable to assess neurologic status due to necessary sedation or need to go to OR
Types of ICP Monitors

- Intraventricular drain
  - only type that allows CSF drainage
- Epidural (bolt)
- Subarachnoid
- Parenchymal
- External (if open fontanelle)
Intraventricular Monitor
The normal ICP waveform contains three phases:

- **P1** (percussion wave) from arterial pulsations
- **P2** (rebound wave) reflects intracranial compliance
- **P3** (dichrotic wave) represents venous pulsations
Intracranial compliance

- Percussion (arterial) $P_1$
- Tidal (rebound) $P_2$
- Dichrotic $P_3$ (venous)

Low pressure wave, compliant cranium

High pressure wave, non-compliant cranium
B-waves are frequent elevations (up to 50 mm Hg) lasting several seconds, occurring in two minute cycles.

- b-waves are suggestive of poor intracranial compliance
intracranial pressure: 'b' waves

cm H₂O

minutes
ICP: a-waves

A-waves (plateau waves) last 5-20 minutes, and often accompany symptoms of brainstem dysfunction.

• cerebral perfusion pressure may be decreased

• a-waves often herald decompensation
ICP: a-waves II
ICP: a-waves mechanism

A-waves (plateau waves) result when mean systemic blood pressure decreases below threshold.

- cerebral perfusion pressure (CPP) falls below ischemic threshold
- cerebrovasodilation occurs in response
- in a non-compliant cranium, this vasodilation results in greatly increased intracranial pressure
ICP: 'a' waves, terminal plateau, decompensation
Additional Monitoring

• Jugular venous bulb catheter
  – monitor jugular venous oxygen saturation, glucose levels, pH, lactate level
  – may give indication if cerebral metabolic demands are being met
  – has not yet been shown to affect outcome

• Pulmonary Artery Catheter (Swann-Ganz)
  – may be necessary if significant hemodynamic instability or use of barbiturate coma
Treatment of ICP

- In order to ↓ICP and ↑CPP, must ↓volume of 1 of the 3 components of the intracranial vault.
- Begin treatment as ICP approaches 20.
To ↓ CSF Volume

- Drain
  - if ventricular drain present
- Decrease production of CSF
  - acetazolamide
  - minimal effectiveness
To ↓ Brain Volume

- Osmotic diuretics
  - mannitol, glycerol, urea
- Loop diuretics
  - furosemide
- 3% Saline - as bolus or continuous infusion
- Maintain serum osmolarity ~ 320 (or higher?)
- fluid restriction vs. euvolemic state
- If all else fails, consider surgical reduction
To ↓ Cerebral Blood Volume

- MILD hyperventilation
  - PaCO$_2$ 30 - 35 torr
- ↑ Head of bed
- Head midline
- Seizure control
  - consider prophylaxis

- Temperature control
  - avoid fever
- Minimal necessary PEEP
- Minimal adequate CVP
- Sedation
- Barb Coma
  - requires continuous EEG monitoring
Other Miscellaneous Treatments

- Mild hypothermia
  - 33-35°C
  - ↓CMRO₂ → ↓CBV
- Prophylactic Dilantin
- Lidocaine for suctioning
- No IV Dextrose

- Use of pressors
  (sympathetic overdrive)
  - variable response
- Family Voices
- Quiet environment
- Sedation
  - especially if agitated or appear in pain
Related Late Complications

- Herniation
- Vascular Compromise
  - AV fistulae
  - traumatic aneurysms
  - thrombosis
- Hydrocephalus
- Seizures
Outcome after Traumatic Brain Injury

• In general, children can have better outcomes than adults
• Exception is children < 2 y/o
• GCS is poor predictor of outcome
• Absence of SSEP universally associated with poor outcomes
• In general, outcome very hard to predict
  – may have complete recovery, mild or focal deficit, PVS, or death