Neurosurgical Aspects of Closed Head Injury

Michael Bruce Horowitz, M.D.
Definition of Closed Head Injury

• Insult rendered to the brain following a traumatic event that does not create an opening in the skull aside from a linear skull fracture

• Typical events include:
  – Motor vehicle accident
  – Fall
  – Assault with a blunt object
  – Domestic abuse including shaken children
  – Sports injuries
Etiology of CHI

• Acceleration-Deceleration is critical to the development of most CHI (no intracranial seatbelts)
• The skull’s interior contour is critical to the development of most CHI (no intracranial air bags)
• The brain’s gelatinous consistency is critical to the development of most CHI
• The axonal structure to neurons and the neurologic system’s architecture of white matter pathways and tracts is critical to the development of most CHI
Epidemiology of **Traumatic Brain Injury**

- Incidence between 100-400/100,000/year
- Male:Female 2:1
- Peak incidence ages 15-35
- In England, most common cause of death in children ages 1-15
- Over 50% of trauma related deaths are associated with TBI
Pathophysiology

• Types of CHI
  – Diffuse Axonal Injury (DAI)
    • DAI is caused by angular accelerations of the head and subsequent rotation and torque of the cerebral hemispheres, brainstem, and their deep fiber tracts and nuclei
    • Small hemorrhages are seen in the corpus callosum, septum pellucidum, deep gray matter, midbrain, pons
    • Axons ultimately degenerate
Diffuse Axonal Shearing Injury
DAI
Pathophysiology

• Types of CHI
  – Focal contusions (bruises) and lacerations (cuts) with subsequent edema
  – *Coup- contra coup* injuries involving frontal lobe and temporal lobe tips, occipital poles
  – Intracranial hematomas
    • Subdural
    • Epidural
    • Intraparenchymal
Linear Skull Fracture
Linear Skull Fracture
Raccoon’s Eyes
Battle's Sign
Epidural Hematoma
Epidural Hematoma
Epidural Hematoma
Anatomic diagram depicts typical locations of contusional traumatic brain injuries.
Contusions
Contusions on MRI
Intraparenchymal Hematoma
Pathophysiology

- Primary brain injury at the time of insult leads to a series of other secondary inflammatory biochemical changes in the brain tissue
- Inflammation period lasts 2-3 weeks
- During this inflammatory period the brain is more susceptible to hypotension, hypoxia, pyrexia
Factors in the Post Injury Period

- Excitotoxins
  - Excitatory amino acids (aspartate and glutamate) released in response to reduction in cerebral blood flow
  - Lead to cell death by activation of N-methyl D-aspartate (NMDA) receptor and the associated Ca ion channel
  - Ca influx into the cell leads to cell membrane destruction and cell death
  - Research into NMDA receptor blockers has not yet proved protective
Factors in the Post Injury Period

• Inflammation
  – After injury intracranial cells increase their production of cytokines which activate inflammatory cascades
  – After injury adhesion molecules increase leading to an influx of leukocytes which can in turn damage brain tissue

• Free Radicals
  – Free radical production increases after injury leading to cell membrane damage

• Hyperglycemia
  – Leads to lactic acid production
Secondary Brain Injury

- Secondary brain injury refers to factors that present subsequent to the initial insult and deleteriously affect brain function thus further compromising activity and recovery.
- Examples are:
  - Pyrexia
  - Secondary brain swelling and herniation
  - Extra-axial masses and increased intracranial pressure or herniation
  - Intracranial hematomas
Secondary Brain Injury

• Each of these secondary injuries further affects brain function through:
  – Hypoxia
  – Hypoperfusion
  – Reduction in cerebral perfusion pressure
  – Acidosis

• Resulting mismatches between cell metabolism and energy supply along with physical distortion of neural architecture leads to cell and tissue death
What is the clinicians role?

• We cannot prevent the initial injury
• We strive to recognize and treat secondary injuries as well as try to prevent their development
• These goals define a range of community standards for post injury neurosurgical management
Community Standards

• Physicians deal with two communities
  – Geographic community in which we practice (hospital environment and its capabilities)
  – Neurosurgical community
• Often the ideals of one community do not or cannot meet the expectations of the other
• The reasons for these mismatches need to be taken into consideration when reviewing cases or determining liability and when obtaining physician opinions.
• Not everyone practices in an Ivory Tower
Patient Assessment

• Assess in field and try to get patient to a neurosurgical or trauma center ASAP
• Physical Examination
  – Associated trauma- skull, ears, spine, body, heart, lungs
  – In children- retinal examination
• Neurological Examination
  – Glasgow Coma Score
  – Pupillary examination
• History- mode of injury
• Laboratory tests- blood count, coagulation profile
Indications for Neurosurgical Consultation

- CT scan demonstrates an intracranial mass lesion
- Intracranial lesion is suspected yet a head CT cannot be obtained because of other life threatening injuries (pneumoventriculography possible)
- GCS < 9; External ventricular drain needed
- Confusion for more than 4 hours
- Deterioration in neurological examination
- Focal neurological findings
- Compound depressed skull fracture
- CSF leak
Patient Assessment

• Glasgow Coma Score
  – Eye Opening
    • Spontaneous 4
    • To Speech 3
    • To Pain 2
    • None 1
  – Verbal Response
    • Oriented 5
    • Confused 4
    • Inappropriate 3
    • Incomprehensible (sounds) 2
    • None 1
  – Best Motor
    • Obeys commands 6
    • Localizes to pain 5
    • Withdraws from pain 4
    • Decorticate 3
    • Decerebrate 2
    • None 1

Total Score 3 - 15
### Glasgow Coma Scoring

<table>
<thead>
<tr>
<th>Eye Opening (E)</th>
<th>Motor Response (M)</th>
<th>Verbal Response (V)</th>
</tr>
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<tbody>
<tr>
<td>Spontaneous...4</td>
<td>Obeys...6</td>
<td>Oriented...5</td>
</tr>
<tr>
<td>To speech......3</td>
<td>Localizes...5</td>
<td>Confused conversation...4</td>
</tr>
<tr>
<td>To pain........2</td>
<td>Withdraws...4</td>
<td>Inappropriate words...3</td>
</tr>
<tr>
<td>Nil ............1</td>
<td>Abnormal flexor response...3</td>
<td>Incomprehensible sounds...2</td>
</tr>
<tr>
<td>Open your eyes</td>
<td>Extensor response...2</td>
<td>No response</td>
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<tr>
<td>Response to speech...3</td>
<td>Nil (no response)...1</td>
<td>1969</td>
</tr>
<tr>
<td>Nil (no response)...1</td>
<td>Extensor response...2</td>
<td>1972</td>
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**Coma score (E + M + V) = 3 to 15**
Patient Assessment

• Why is GCS important?
  – Allows healthcare personnel to quickly discuss patients and relate clinical status
  – Allows for accurate serial assessment of patient progress
  – May predict the likelihood of other injuries
  – May help predict clinical outcomes
Patient Assessment

- Risk of an operable hematoma in CHI patient

<table>
<thead>
<tr>
<th>GCS</th>
<th>Risk</th>
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<tbody>
<tr>
<td>15</td>
<td>1 in 3615</td>
</tr>
<tr>
<td>14</td>
<td>1 in 51</td>
</tr>
<tr>
<td>3-8</td>
<td>1 in 7</td>
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</tbody>
</table>
Patient Assessment

• GCS can help predict patient outcomes
• GCS can help predict likelihood of elevated intracranial pressure (ICP)
  – GCS < 9
    • ICP > 10 in 82%
    • ICP > 20 in 44%
    • ICP > 40 in 10%
Patient Assessment

• Imaging Evaluation
  – CT scanning
    • Indicated for any patient who suffers an injury that leads to a GCS < 15
    • Unclear if it is indicated for normal GCS and head injury such as a linear skull fracture
    • CT looks for blood clots (intra and extraaxial), brain swelling, air leaks, bony fractures
    • Findings dictate need for surgical therapy and types of medical therapy
Patient Management

- CT/Xenon Blood flow studies
  - Look at cerebral blood flow which can help determine subsequent management with ventilator and medications
  - Not available at many centers
Patient Assessment

– MRI scanning
  • Usually useful in the subacute or chronic period when looking for more occult brain injuries especially in deeper areas or in the brainstem
  • Not a typical study done in the acute period

– Angiography
  • Done when there is suspicion of a vascular injury
Patient Assessment

- CT findings may help predict the presence of elevated ICP
- Elevated ICP leads to further brain injury by mechanically disrupting brain tissue and possibly reducing cerebral blood flow
Patient Assessment

- High/low density lesion on CT  53 – 63% chance increased ICP
- Normal CT  13% incidence of elevated ICP

• Factors with a normal CT that increase the incidence of increased ICP
  – Age > 40
  – Systolic BP < 90 mm Hg
  – Abnormal motor posturing
• 2 or more features  60% incidence increased ICP
• 1 or more features  4% incidence increased ICP
Patient Assessment

- Absent or compressed basal cisterns on first CT scan with GCS < 9
  - Absent: 77% mortality
  - Compressed: 39% mortality
  - Normal: 22% mortality

- Absent: 85% poor outcome
  - Compressed: 64% poor outcome
  - Normal: 44% poor outcome

- No survivors when absent cisterns associated with 15 mm shift
  - 74% with absent cisterns had ICP > 30
Basal Cisterns
CT Imaging Diagnostic Categories in CHI and Mortality

- Diffuse Injury I  No visible abnormality  9.6% mort.
- Diffuse Injury II  Cisterns present with 0-5 mm shift; no high density lesion greater than 25cc  13.5% mort.
- Diffuse Injury III  Cisterns compressed or absent; shift 0-5mm; no high density lesion > 25cc (swelling)  34% mort.
- Diffuse Injury IV  Shift > 5 mm; no lesion > 25 cc (shift)  56.2% mort.
- Evac. Mass Lesion  Any lesion surgically evacuated  38.8% mort.
- Nonevac Mass Les Mass > 25cc not evacuated  52.8% mort.
- Brainstem injury  66.7% mort.
Patient Management

• Remove mass lesion if:
  – Patient has a survivable exam
  – Patient is age appropriate
  – It is associated with brain shift (usually > 1 cm)
  – GCS is abnormal
  – It is in a location that portends imminent danger (ie: low middle fossa)
  – Clot size is considered significant (ie: 60cc rule)
Patient Management

• Acute Subdural Hematoma
  – Very common extra-axial mass lesion in CHI
    • Mortality 42 – 90%
  – Usually a marker for more diffuse underlying brain injury from shear forces
    • GCS 3-5  Mortality 76-84%, Functional recovery rate 14%
    • GCS 6-8  Mortality rate 36-48%, Functional recovery rate 25-40%
    • GCS 12-15 Mortality rate 0%, Functional recovery rate 92%
Patient Management

• Acute Subdural Hematoma
  – Conscious at surgery    Mortality rate 6%
  – Unconscious at surgery  Mortality rate 77%
  – Pupils abnormal         Mortality rate 75%
  – Decerebrate             Mortality rate 77%
  – Decerebrate + Unreactive pupils Mortality rate 95%
  – Age >50, time to evacuation > 4 hours, elevated post-op ICP = Poor prognosticators
Patient Management

• Elevated ICP (Usually treat when ICP is greater than 20)
  – ICP monitor placed
  – CSF drained
  – Hyperventilation to reduce CBF - Controversial issue
  – Mannitol/Lasix to reduce extracellular fluid
  – Barbiturates to reduce CBF
  – Reduce body temperature (medications, devices)
  – Surgical removal of brain tissue and/or skull
Patient Management

• Try to reduce incidence of secondary insults
  – Hypoxia
  – Infection
  – Hyperglycemia
  – Fever
  – Deep venous thrombosis
  – Decubitus ulcers
  – Brain swelling
  – Seizures
ICU Goals

• Keep serum sodium $>135-140$ and $<160$. A low Na can lead to increased brain swelling
• Avoid hyperglycemia to avoid lactic acidosis
• Nutritional support within 72 hours of CHI. Metabolic expenditure can be 120-250% of a normal patient. Keep some nutrition in gut.
• Mild head elevation ???
• Physical therapy
• DVT prophylaxis
Rehabilitation

- It takes most adults at least one year to recover maximally from a severe CHI.
- Rehabilitation centers are a key part of this recovery phase.
Outcomes Evaluation

• Glasgow Outcome Score
  – 1  Dead
  – 2  Persistent Vegetative State
  – 3  Severe Disability
  – 4  Moderate Disability
  – 5  Good Recovery
Outcomes

• Other outcomes scales exist yet it is unclear which ones are best.