Current Management of Severe Traumatic Brain Injury

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Head Injury in Central Oregon

- Think First
- Impact
- Severe TBI

Brain Trauma Research

- Brain Trauma Foundation
- Charles A. Dana Foundation
- NIH

Introduction

- Traumatic brain injury (TBI) is common - NHIF
- Primary and secondary injuries
- CBF and metabolism
- Cerebral ischemia
- Intensive Care protocols
Metabolic Autoregulation

- Energy requirements
- Oxidative metabolism of glucose
- No intrinsic storage capacity
- Primary function of the cerebral circulation is to provide adequate glucose and oxygen
- Tight coupling between CBF and cerebral metabolism
- Normal resting values

Pressure Autoregulation

- Cerebral circulation - maintain constant CBF despite changes in CPP

\[ CBF = \frac{CPP}{CVR} \]

\[ CPP = MAP - ICP \]

\[ CVR = \frac{L \eta}{\pi^4} \]

Relationship Between CBF and Oxygen Metabolism

\[ CaO_2 = 1.34 \times [hgb] \times O_2\ saturation + (0.003 \times PaO_2) \]

\[ 30\% \]

\[ A-VDO_2 \]

\[ OEF \]

\[ CvO_2 \]
**Current Management of Severe Traumatic Brain Injury**

The main objective of intensive care management is to maintain adequate cerebral perfusion and oxygenation, and avoid medical and surgical complications while the brain recovers.

**Guidelines for the Management of Severe TBI (BTF, AANS,CNS)**

- **Standards**: accepted principles of patient management that reflect a high degree of clinical certainty (PRCT)
- **Guidelines**: particular strategy or range of management strategies that reflect a moderate clinical certainty
- **Options**: remaining strategies which there is unclear clinical certainty
Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Points</th>
<th>Best Eye</th>
<th>Best Verbal</th>
<th>Best Motor</th>
</tr>
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<tbody>
<tr>
<td>6</td>
<td></td>
<td>Obeys</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Oriented</td>
<td>Localizes</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Spontaneous</td>
<td>Confused</td>
<td>Withdraws</td>
</tr>
<tr>
<td>3</td>
<td>To speech</td>
<td>Inappropriate</td>
<td>Decorticate</td>
</tr>
<tr>
<td>2</td>
<td>To Voice</td>
<td>Incomprehensible</td>
<td>Decerebrate</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>None</td>
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Severe TBI Management

- Early intubation
- Rapid transportation to appropriate trauma facility
- Prompt resuscitation
- Early CT scanning
- Immediate evacuation of intracranial mass lesions

ICU Protocol

- Intracranial pressure monitoring
  - GCS 3-8
  - Ventriculostomy vs bolt
  - All pts with ICP monitor receive arterial and central lines

- Sedation and analgesia
  - Propofol and ativan gtt
  - Morphine and fentanyl
- Patient positioning
- Avoid hyperthermia
• Hypertonic saline (goal Na 150-155)
  – IVF D5 1.5%NS with 20meq KCL
  – Increase Sosm (goal 320)
    • Start early prior to elevated ICP
    • Decrease ICP
    • Decrease mannitol use
    • Not harmful, pt euvoletic

• Maintain CPP >70 mmHg
  – CPP < 70
    • Assure euvoletic by keeping CVP 3-8
    • 250cc 5% albumin
    • 500cc bolus NS
  – CPP < 70 despite volume challenge
    • Titrate continuous infusions
      – Neosynephrine @ 30 –100 mcg/min to maintain CPP>70
      – Inadequate response add Norepinephrine 4mcg/min

• Treat ICP > 20
  – Drain CSF for sustained ICP > 20
  – No response:
    • Load mannitol 0.5 gms/kg IV bolus
    • Then mannitol 0.25 gms/kg IV bolus q 2hr ICP>20
  – ICP remains >20 start paralytic (Zemuron)
  – Continued elevated ICP
    • Consider barbiturate coma vs decompressive craniotomy

Conclusion
Indications for Intracranial Pressure Monitoring

- **Standards** - Insufficient data: PRCT- unethical
- **Guidelines**
  - ICP monitoring is appropriate in patients with severe TBI with an abnormal admission CT scan. Severe TBI is defined as a GCS score of 3-8 after cardiopulmonary resuscitation. An abnormal CT scan of the head is one that reveals Hematomas, contusions, edema, or compressed basal cisterns (abnl 53-63%; nl 13%).
  - ICP Monitoring is appropriate in patients with severe TBI with a normal CT scan if 2 or more of the following features are noted at admission: age over 40 years, unilateral or bilateral motor posturing, systolic blood pressure < 90mm Hg (53-63%)
  - ICP monitoring is not routinely indicated in patients with mild or moderate head injury. However, a physician may choose to monitor ICP in certain conscious patients with traumatic mass lesions (3% & 10-20% deteriorate to coma)

Evidence to support ICP Monitoring

- Multiple studies have shown correlation between high ICP and poor outcome in severe TBI – Marmarou TCDB 428p w/STBI and ICPM - most predictive factor for poor outcome ICP>20
- Jennet 1977 reported on 700 patients with severe TBI Tx without ICP monitoring – 50% mortality
- Becker reported 30% mortality using a protocol centered on ICP monitoring
- Many studies today are reporting mortality rates in the 20% range
- Ghajar 1998 reported on 49 consecutive patients in nonrandomized study, 34 received aggressive ICP management – 12% mortality; 15 without ICP monitors – 53%Mortality

Intracranial Pressure Treatment Threshold

- **Guideline:** ICP treatment should be initiated at an upper threshold of 20mm Hg
- Mannitol - osmotic diuretic (Guideline)
  - Limited data suggest that intermittent boluses may be more effective than continues infusion (0.25-1 gm/kg)
- Role of CPP (CPP=MAP-ICP) / loss of pressure autoregulation
  - PRCT in progress

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ACUTE HYPERVENTILATION AND ISCHEMIA FOLLOWING TBI

- Hyperventilation is routinely employed in TBI
- **Standard:** In the absence of increased ICP, chronic prolonged HV therapy (PaCO2 < 25 mm Hg) should be avoided after severe TBI
- **Guideline:** the use of prophylactic HV (PaCO2 <= 35 mm Hg) therapy during the first 24 hours after severe TBI should be avoided because it can compromise cerebral perfusion during a time when CBF is reduced.
- Options:
  - Hyperventilation therapy may be necessary for brief periods when there is acute neurologic deterioration, or for longer periods if there is intracranial hypertension refractory to sedation, paralysis, CSF drainage, and osmotic diuretics.
  - SjvO2 , A-VDO2 , and CBF monitoring may help to identify cerebral ischemia if HV, resulting in PaCO2 values less than 30 mm Hg, is necessary.
Barbiturates in the control of Intracranial Hypertension

- **Guideline**: High-dose barbiturate therapy may be considered in hemodynamically stable salvageable severe head injury patients with Intracranial hypertension refractory to maximal medical and surgical Intracranial pressure lowering therapy.
- **Utilization of barbiturates for the prophylactic treatment of ICP is not indicated**
- Schwartz 1984 PRCT – Barbs compared to mannitol, ICP>25/15 min, in nonoperative group, mortality – mannitol 41%, barbs 77%
- Eisenberg 1988 PRCT – GCS 4-8 with intractable ICP elevations, Barbs vs standard Tx, ICP remained uncontrollable 68% barbs vs 83% standard Tx. If patient responded to barbs then 92% chance of survival, if not 17% survival
- Cochrane database review – compared 7 randomized and quasi randomized studies. Conclusion – no evidence to support barbiturates improve outcome. Significant hypotension observed in 1 out 4 patients.

Seizure prophylaxis

- **Standards**: Prophylactic use of phenytoin, carbamazepine, or phenobarbital is not recommended for preventing late posttraumatic seizures
- **Guidelines**: none
- **Options**: anticonvulsants may be used to prevent early posttraumatic seizures in patients at high risk for seizures following head injury. Phenytoin and carbamazepine have been demonstrated to be effective in preventing early posttraumatic seizures
  - High risk factors: GCS<10, cortical contusion, depressed skull fracture, SDH, EDH, ICH, penetrating head wound, seizure within 24 hours of injury

The role of glucocorticoids in the treatment of TBI

- **Standard**: the use of glucocorticoids is not recommended for improving outcome or reducing ICP in patients with severe TBI
- **Spinal Cord Injury (NASCIS)**
  - Treatment started within 3 hours of injury: 30mg/kg load, 5.4mg/kg/hour for 24 hours
  - Treatment started between 3 and 8 hours post injury: load followed by maintenance dose for 48 hours

Cerebral Pathophysiologic Response to Injury

- Ischemic stroke
- Retraction injury
- TBI
- Cold Injury

Ischemic Infarction

- CBF
- CMRO₂
- OEF

Percent Change

Time