Management of Traumatic Brain Injury

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Traumatic Brain Injury (TBI)

• A “bump, blow or jolt to the head that disrupts the normal function of the brain”¹
  • Mild: Brief change in mental status or consciousness¹
  • Severe: Extended period of unconsciousness or memory loss¹

• Most TBIs that occur each year are mild, commonly called concussions
TBI: Quick Facts

• In 2013, 2.8 million TBI-related emergency department (ED) visits, hospitalizations, and deaths occurred in the US.
  • Contributed to the deaths of nearly 60,000 people.
    • 70% of deaths occurred at the scene or in the ED
    • 30% of deaths occurred later (Mostly due to secondary brain injury) ¹

• In 2012, an estimated 329,290 children (age 18 or younger) were treated in U.S. EDs for sports and recreation-related injuries that included a diagnosis of concussion or mild TBI²
TBI: Causes

• Falls: Leading cause of TBI.
  • In 2013, falls accounted for 47% of all TBI-related ED visits, hospitalizations, and deaths in the US.
  • 54% of TBI-related ED visits hospitalizations, and deaths among children ages 0-14.
  • 79% of TBI-related ED visits, hospitalizations, and deaths in adults aged 65 and older.¹

• Being struck by/against an object was the second overall leading cause of TBI.
  • 15% of all TBI-related ED visits, hospitalizations, and deaths in the US in 2013.
  • 22% of TBI-related ED visits, hospitalizations, and deaths in children ages 0-14.¹

• Motor vehicle accidents were the third overall leading cause of TBI.
  • 14% of all TBI-related ED visits in the US in 2013.
  • 19% of all TBI-related deaths in the US in 2013. (Third-leading cause)¹

• Intentional self-harm was the second leading cause of TBI-related deaths (33%) in 2013.¹
TBI-related deaths: Age groups (2013)

• Highest among persons 75 years of age and older.¹

• Causes of TBI-related death by age group.
  • Falls were the leading cause of death for persons 65 years of age or older.
  • Intentional self-harm was the leading cause of death for persons 25-64 years of age.
  • Motor vehicle crashes were the leading cause of death for persons 5-24 years of age.
  • Assaults were the leading cause of death for children ages 0-4 years.¹
Non-fatal TBI-related injuries (2013)

• Hospitalization rates: Highest among persons 75 years of age and older.

• ED visits: Bimodal distribution
  • Adults 75 years of age and older and children 0-4 years of age.\(^1\)
TBI in sports

• In 2012, 329,290 ED visits (<19 yrs old) for sports and recreation-related injuries that included a diagnosis of concussion or TBI.²
  • Greater than a 100% increase in rate of ED visits (<19 yrs old) between 2001 and 2012.²
Traumatic Brain Injury: Classification

• Traumatic Brain Injury may be classified based on the mechanism (closed vs penetrating) or clinical severity.

  • Severity Classification is based on the presenting Glasgow Coma Scale (GCS):
    • GCS 13 - 15: Mild
    • GCS 9 – 12: Moderate
    • GCS 3 – 8: Severe

<table>
<thead>
<tr>
<th>Eye Opening Response</th>
<th>Verbal Response</th>
<th>Motor Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 = Spontaneous</td>
<td>5 = Oriented</td>
<td>6 = Obey commands</td>
</tr>
<tr>
<td>3 = To verbal stimuli</td>
<td>4 = Confused</td>
<td>5 = Localizes pain</td>
</tr>
<tr>
<td>2 = To pain</td>
<td>3 = Inappropriate words</td>
<td>4 = Withdraws from pain</td>
</tr>
<tr>
<td>1 = None</td>
<td>2 = Incoherent</td>
<td>3 = Flexion to pain or decorticate</td>
</tr>
<tr>
<td></td>
<td>1 = None</td>
<td>2 = Extension to pain or decerebrate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 = None</td>
</tr>
</tbody>
</table>
Traumatic Brain Injury: Classification

• Mild TBI: Most commonly seen as concussions

• Moderate TBI: Associated with prolonged loss of consciousness, abnormal neuro-imaging and focal neurological deficits
  • Patients usually require Neurosurgical evaluation

• Severe TBI: Abnormal neuro-imaging which may include cerebral contusions, intracranial hemorrhage or skull fractures
## TBI Severity

<table>
<thead>
<tr>
<th>Severity</th>
<th>GCS</th>
<th>LOC</th>
<th>AOC</th>
<th>PTA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>13-15</td>
<td>&lt;30 minutes</td>
<td>&lt; 24 hours</td>
<td>&lt; 24 hours</td>
</tr>
<tr>
<td>Moderate</td>
<td>9-13</td>
<td>&gt;30 minutes &lt; 24 hours</td>
<td>&gt; 4 hours</td>
<td>&gt;24 hours &lt;7days</td>
</tr>
<tr>
<td>Severe</td>
<td>3-8</td>
<td>&gt;24 hours</td>
<td>&gt;24 hours</td>
<td>&gt;7 days</td>
</tr>
</tbody>
</table>

LOC - Loss of consciousness; AOC - Alternation of consciousness; PTA - Post-traumatic amnesia

Marshall et.al. (2012)
Initial Management: ABCs

• **Airway**

  - Important to help optimize cerebral oxygenation and perfusion
    - Duration and severity of hypoxia and hypotension in the early period has dramatic consequences on the ultimate clinical outcome\(^3\)

• **Breathing**

  • Advanced Trauma Life Support Guidelines:
    - Oxygen saturation greater than 90%
    - Goal is normoxemia to mild hyperoxemia as extreme hyperoxemia is associated with increased risk in severe TBI\(^3\)
TBI: Initial Management

• **Circulation:** - Hemorrhage control and fluid resuscitation with blood products and crystalloids
  - Avoiding hypotension (i.e. keeping SBP >90mmHg)$^3$

• Head Position: Keep in midline position and elevated to 30 degrees
  - Assume an occult cervical spine injury in any TBI patient with altered mental status or blunt injury above the clavicle until rule-out with imaging$^3,5$
Secondary Survey

• Detailed but rapid neurologic or disability examination
  • Diagnosis of TBI is made based on clinical suspicion and confirmed with imaging
  • Altered mental status or obtundation may also be secondary to:
    • Oxygenation or inadequate ventilation
    • Hypoglycemia
    • Medication or toxin exposure
    • Hypoperfusion\(^3,5\)

• Head CT is considered the first-line imaging technique for TBI patients\(^3\)
• Image courtesy of EBM consult (www.ebmconsult.com)
TBI: Common CT findings
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Cerebral Herniation
TBI: Blood pressure and fluid management

• Maintenance of euvolemia and adequate cerebral perfusion is the goal\(^3\)

• **CPP = MAP – ICP**
  - Recommended goals are SBP >90mmHG and CPP >60mmHg\(^3,6\)

• Autoregulation is impaired and regional cerebral blood flow becomes directly dependent on systemic blood pressure\(^3,7\)
  - Resuscitation with crystalloid fluids and blood products is usually necessary to help maintain cerebral perfusion pressure (CPP)
  - Hypotonic fluids (1/2NS and Lactated Ringer’s) can exacerbate cerebral edema and should be avoided\(^3,8\)
TBI: Ventilation and airway management

• Goals are to maintain PaO$_2$ above 60mmHg, Keep Oxygen saturation greater than 90% and avoidance of hyperoxymia (PaO$_2$ > 487mmHg)3

• Indications for artificial airway placement are:
  • GCS of 8 or less
  • Clinical concern of patient’s inability to ventilate or protect his/her airway

• Over-aggressive ventilation should be avoided because of the potential for decreased cerebral perfusion at PCO$_2$ < 25mmHG

Significantly elevated ICP can result in cerebral herniation and ischemia.

For patients with moderate and severe TBI, the following conservative measures should be employed to minimize ICP elevation:

- Raise head of bed to 30 degrees
- Keep the head midline
- Avoid circumferential neck compression
- Avoid placement of central venous lines into the dominant internal jugular vein
- Avoid placing the patient in the Trendelenburg position (even for central line placement)
- Aggressive treatment of fever, seizures, pain and agitation

TBI: Indications for ICP monitoring

- An ICP monitor should be placed if a patient has a GCS of 8 or less (after resuscitation) and an acute abnormality on head CT (cerebral contusions, effacement of basal cisterns, evidence of herniation, hydrocephalus)\textsuperscript{3,6}
- ICP < 20mmH\textsubscript{2}O
- CPP > 60mmHg
Medical Management of Elevated ICP

• Hyperventilation:
  • Recommendation is to strictly avoid $\text{PCO}_2$ levels below 25mmHg
  • Should be used only as an emergent and temporary intervention
    • Prolonged hyperventilation is associated with worsening cerebral edema
  • If hyperventilation is continued for >12 hours, metabolic compensation negates any helpful effects of hyperventilation
  • Reliably lowers $\text{PCO}_2$ and ICP within seconds, during a herniation event $^3,^6$
Medical Management of Elevated ICP

• Hyperosmolar Therapy:
  • Creates an osmotic gradient which causes movement of water from intracellular and extracellular compartments of the brain into the vasculature, thus reducing the volume of the overall cranial compartment\textsuperscript{3,8}

• Mannitol:
  • Administered Intravenously: via central or peripheral like
  • Recommended dose is 0.25 to 1.0 g/kg
  • Serum Osmolality of 320mOsm/L is a generally accepted treatment endpoint\textsuperscript{3,9,10}
Medical Management of Elevated ICP Cont.

• Hypertonic Saline: Common concentrations are 2%, 3%, 7.5% and 23.4%
  • Generally more effective than mannitol at ameliorating elevated ICP
    \(^3\)

  • High concentrations (3% or higher) should be administered through a central venous line over 10-15mins to prevent phlebitis and hypotension
    \(^3\)

  • A bolus dose of 30ml of 23.4% hypertonic saline can reduce ICP by > 8mmHg and can increase CPP by 6mmHg\(^3,11\)

  • A commonly used treatment goal is to achieve serum Na levels of 145 to 155 mEq/L (Serum osmolality of 300 to 320 mOsm/L)\(^3,8\)
Reduction of Cerebral Metabolic Rate of Oxygen (CMRO$_2$)

- Induction of Pharmacologic coma for refractory ICP
  - Reducing cerebral metabolism, resulting in reduction of cerebral blood flow and oxygen demand$^3$

- Pentobarbital: Most commonly used agent for induction of pharmacological coma
  - Loading dose of 10mg/kg over 30 minutes, then 5mg/kg/hr over 3 hours, then maintenance dose of 1mg/kg/hr
    - Goal is burst suppression on EEG or satisfactory reduction in ICP$^3,12$
Reduction of Cerebral Metabolic Rate of Oxygen (CMRO2)

• Propofol: Loading dose of 2mg/kg followed by a titrated infusion of up to 100micrograms/kg/min
  • Goal is burst suppression on EEG or satisfactory reduction in ICP

• Long-term and high dose propofol infusions are associated with the development of propofol infusion syndrome\(^3,13\)
  • Renal failure
  • Rhabdomyolysis
  • Hepatomegaly
  • Hyperkalemia
  • Myocardial failure
  • Metabolic acidosis
  • Hepatomegaly
Potential treatment options

• Induced Hypothermia
  • Risk of coagulopathy and anti-platelet effects should be considered\(^3\)

• Gender-based differences in TBI-related outcomes
  • Progesterone may be neuro-protective against cerebral edema\(^3,14\)
  • ProTECT Trial (Progesterone for the Treatment of Traumatic Brain Injury)
    • Did not show a benefit of progesterone over placebo in the improvement of patient outcomes with acute TBI
  • SynAPSE Trial (Progesterone for Severe Traumatic Brain Injury)
    • No clinical benefit of progesterone in patients with severe TBI\(^15\)
Surgical Treatment: Decompressive Craniectomy

• Operative Video
References:


3. Marshall, SA, Riechers RG. Diagnosis and Management of Moderate and Severe Traumatic Brain Injury Sustained in Combat. 2012; 177, 8:76


