Traumatic Brain Injury: Initial Management

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Henry Ford Hospital
September 30th, 2016
Financial Disclosures

- None to report
Objectives

- Discuss the neurologic implications of TBI and identify early interventions known to improve outcome in TBI.
- Describe some of the available Clinical Practice Guidelines for management of TBI.
- Demonstrate the importance of collaboration in providing quality care.
Traumatic Brain Injury

“Traumatic brain injury (TBI) occurs when a sudden trauma, often a blow or jolt to the head, causes damage to the brain.”

- Brain Trauma Foundation

https://www.braintrauma.org/tbi-faqs/
# Classification

## Mild
- **Primary damage/MOI:**
  - Low-velocity acceleration/deceleration
  - Non-penetrating
- **Loss/alteration in consciousness:**
  - < 30 minutes
- **Amnesia:**
  - < 24 hours
- **GCS:**
  - 13-15
- **Imaging:**
  - Negative
- **Comorbidity:**
  - PTSD, overlapping symptoms
- **Outcome:**
  - Transient neuropsychiatric disorders, mostly full recovers, long-term neuropsych after repeated injuries are frequent

## Moderate
- **Primary damage/MOI:**
  - Frequently mixed
  - Blast
  - Acceleration/deceleration
  - Non-penetrating
- **Loss/alteration in consciousness:**
  - > 30 minutes, 24 hours
- **Amnesia:**
  - > 24 hours, > 7 days
- **GCS:**
  - 9-12
- **Imaging:**
  - Transient changes
- **Comorbidity:**
  - PTSD, other injuries
- **Outcome:**
  - Mild-to-moderate, typically chronic, neurological and neuropsych abnormalities

## Severe
- **Primary damage/MOI:**
  - Complex
  - Blast + acceleration/deceleration + penetration
- **Loss/alteration in consciousness:**
  - > 24 hours
- **Amnesia:**
  - > 7 days
- **GCS:**
  - < 9
- **Imaging:**
  - Positive, lasting abnormalities
- **Comorbidity:**
  - Polytrauma, multisystem injuries
- **Outcome:**
  - Death, significant, neurological and neuropsych deficits, severe, chronic physical and neuropsych disabilities
Epidemiology: TBI in the US

- 2.5 million ED visits
- 87% treated and released
- 11% hospitalized and discharged
- 2% died (52,844)
MTQIP Mortality Severe TBI
Twice as likely to survive if TBI guidelines are followed.

“Good” outcomes rose from 35% to 66%.

“Poor” outcomes fell from 34% to 19%.

Potential savings of $3.8 billion.
Henry Ford TBI

- 2015
  - 311 TBI admissions
  - 24 Severe (GCS 3-8)
Guidelines for the Management of Severe Traumatic Brain Injury
3rd Edition

Guidelines for the Management of Severe Traumatic Brain Injury
4th Edition

ACS TQIP
BEST PRACTICES IN THE MANAGEMENT OF TRAUMATIC BRAIN INJURY
Primary and Secondary Injury

- Primary Injury
  - Result of the initial injury

- Secondary Injury
  - Subsequent cerebral ischemia
Secondary Injury

Cerebral Perfusion

- Intracranial Hypertension
- Delayed ICH
- Edema
- Hyperemia
- Carotid dissection
- Seizures
- Vasospasm

Secondary Injury Components:
- Hypoxia
- Hypotension
- Electrolyte imbalance
- Other:
  - Anemia
  - Hyperthermia
  - Hypercarbia
  - Hypoglycemia
Patient 1

- 91 year-old lady fell in her kitchen and struck her face.
- Coumadin and aspirin.
- 5 hours later complains of headache.
- Exam: Alert, recognizes her kids
- 153/90 HR 80 RR 14
Patient 1

- Neuro exam is then recorded as deteriorating to a GCS of 12 and then to a 7.
- She is given etomidate and rocuronium for intubation.
- 3% NaCl 250 ml and Vitamin K 10 mg IV.
- Transferred to main campus.
Patient 1

- Arrives around 8 pm.
- Anisocoria is documented.
- Right sided hemiparesis.
- K centra and platelets.
- Lasix and mannitol.
- Taken to OR with Neurosurgery at 10:30 pm.
Patient 1

VTE prophylaxis
subcutaneous heparin at 48 hours
Neurologic Evaluation

- Rapid neurologic exam at the end of the primary survey to assess:
  - GCS
  - Pupillary size/reaction
  - Lateralizing signs
  - Spinal cord injury/level
Glasgow Coma Scale

- Quick, simple method to evaluate LOC
- Initial motor score is predictive of patient outcome
- Can be subjective, repeated exams can be beneficial
# GCS

<table>
<thead>
<tr>
<th>Eye opening (E)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>To pressure</td>
<td>2</td>
</tr>
<tr>
<td>To sound</td>
<td>3</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>Untestable</td>
<td>Reason:</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Verbal response (V)</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
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<tr>
<td>Sounds</td>
<td>2</td>
</tr>
<tr>
<td>Words</td>
<td>3</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
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<tr>
<td>Untestable</td>
<td>Reason:</td>
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</table>

<table>
<thead>
<tr>
<th>Motor response (M)</th>
<th></th>
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<tbody>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Extension</td>
<td>2</td>
</tr>
<tr>
<td>Abnormal flexion</td>
<td>3</td>
</tr>
<tr>
<td>Normal flexion</td>
<td>4</td>
</tr>
<tr>
<td>Localizing</td>
<td>5</td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Untestable</td>
<td>Reason:</td>
</tr>
</tbody>
</table>

ACS TQIP
BEST PRACTICES IN THE MANAGEMENT OF TRAUMATIC BRAIN INJURY
Goals of Treatment

Table 2. Goals of Treatment

<table>
<thead>
<tr>
<th>Goal</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse Oximetry</td>
<td>≥ 95%</td>
</tr>
<tr>
<td>ICP</td>
<td>20 - 25 mmHg</td>
</tr>
<tr>
<td>Serum sodium</td>
<td>135-145 mmHg</td>
</tr>
<tr>
<td>PaO₂ ≥ 100 mmHg</td>
<td></td>
</tr>
<tr>
<td>PbtO₂ ≥ 15 mmHg</td>
<td></td>
</tr>
<tr>
<td>INR ≤ 1.4</td>
<td></td>
</tr>
<tr>
<td>PaCO₂ 35-45 mmHg</td>
<td></td>
</tr>
<tr>
<td>CPP ≥ 60 mmHg *</td>
<td></td>
</tr>
<tr>
<td>Platelets ≥ 75 x 10³ / mm³</td>
<td></td>
</tr>
<tr>
<td>SBP ≥ 100 mmHg</td>
<td></td>
</tr>
<tr>
<td>Temperature 36.0-38°C</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin ≥ 7 g/dl</td>
<td></td>
</tr>
<tr>
<td>PH 7.35-7.45</td>
<td></td>
</tr>
<tr>
<td>Glucose 80-180 mg/dL</td>
<td></td>
</tr>
</tbody>
</table>
Reversal of Anticoagulation

Table 1. Prothrombin Complex Concentrate Dosing Recommendations

<table>
<thead>
<tr>
<th>Pre-treatment INR</th>
<th>Less than 4</th>
<th>4 – 6</th>
<th>Greater than 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose of Kcentra #</td>
<td>25 units/kg</td>
<td>35 units/kg</td>
<td>50 units/kg</td>
</tr>
<tr>
<td>Maximum Dose $^2$</td>
<td>2500 units</td>
<td>3500 units</td>
<td>5000 units</td>
</tr>
</tbody>
</table>

# Round calculated doses to nearest vial size; $^2$Do not exceed stated maximum for patients greater than 100kg

- Prothrombin complex concentrate (Kcentra) will decrease INR in 15 to 30 minutes.
- Prothrombin complex concentrate should only be used when rapid, urgent reversal is initiated in the setting of MOST patients. A typical time frame is 36 hours.

PATIENTS ON ANTIPLATELET THERAPY WITH INTRACRANIAL HEMORRHAGE

- For patients with intracranial hemorrhage who are also on Plavix (clopidogrel) one “6 pack” of platelets should be ordered. However, intracranial hemorrhage in most patients does not warrant use of platelets.

Policy Name/Subject: Tier 1: Anticoagulation Reversal Guidelines
Policy No: MMC-52
Type of Document: Policy
Applies to: Tier 1 (All HFHS)
Category: Medication Management
Owner: HFHS Anticoagulation Subcommittee
Approver (Title): HFHS Medication Management Committee

INR 30 minutes after blood factor or FFP administration, then every 6 hours.

- Hold warfarin in all scenarios. *Fresh Frozen Plasma (FFP) 15 mL/kg may be considered, if INR response to PCC is inadequate. See Appendix A for conversion of FFP volume to units. PCC = Prothrombin Complex Concentrate. *Note: Kcentra contains heparin and is contraindicated in patients with active heparin-induced thrombocytopenia (HIT). FFP should be used for reversal in the setting of active HIT.
Uncal Herniation

Early:
- Altered level of consciousness
- Ipsilateral pupil dilation
- Contralateral motor weakness

Late:
- Central neurogenic hyperventilation or Cheyne-Stokes respiration
- Ipsilateral fixed & dilated pupil
- Contralateral decortication or decerebration

Uncus of temporal lobe herniates through tentorium from expanding mass lesion compressing cerebral peduncle and CN 3.
Empiric Treatment of Intracranial Hypertension

d. Mannitol/hypertonic saline usage: use in patients without an intracranial monitor only with signs of herniation and no hypovolemia; use in monitored patients with ICP > 20 mm Hg (dosage 0.5 – 1.0 mg/ kg every 6 hours); Mannitol is useful because of its osmotic characteristics, volume expansion properties, and free radical scavenging capability
   i. Lasix should not be used in these patients
   ii. Hypertonic saline may be used as an alternative especially in the multisystem injured patient
Patient 2

- 26 year old woman arrived just before 3 am
- Front passenger T boned on her side
- EMS transport to main campus
- 140/108  HR 95  RR 18
- GCS E2 V2 M4 = 8
- Pupillary light reflex intact
Patient 2

- Intubated with Succinyl choline and etomidate.
- X rays done of the lower extremities and scalp laceration stapled.
- Rocuronium and Versed given for CT.
Patient 2
Patient 2

- ICP monitor inserted with initial pressure of 9
- OR for external fixation.
- OR for Intramedullary nails and SI fixation.
- IVC filter day 5
- Lovenox on day 8
ICP Monitoring

- Does not replace neurological and radiographic examination.
- Indicated in patients (GCS ≤ 8) if there is evidence of structural brain damage on initial CT.
- Recommended to reduce in-hospital and 2-week post-injury mortality.
ICP Monitoring

- High risk for progression (large/multiple contusions, coagulopathy).
- Urgent surgery for extracranial injuries.
ICP Treatment Threshold

- **Level II B**
  - Treating ICP above 22 mm Hg is recommended because values above this level are associated with increased mortality.

- **Level III**
  - A combination of ICP values and clinical and brain CT findings may be used to make management decisions.
CPP Monitoring

- Level II B
- Management of severe TBI patients using guidelines-based recommendations for CPP monitoring is recommended to decrease 2-week mortality
CPP Thresholds

- **Level II B**
  - The recommended target CPP 60 - 70 mm Hg.

- **Level III**
  - Avoid aggressive attempts to maintain CPP above 70 mm Hg with fluids and pressors.
THREE-TIERED MANAGEMENT OF INTRACRANIAL PRESSURE

TIER 1

- Head of bed elevated at 30 degrees (reverse Trendelenburg) to improve cerebral venous outflow
- Sedation and analgesia using recommended short-acting agents (for example, propofol, fentanyl, midazolam) in intubated patients
- Ventricular drainage performed intermittently. Continuous drainage is not recommended unless an additional ICP monitor is placed, as when the drain is open, it does not accurately reflect the true ICP
- Repeat CT imaging and neurological examination should be considered to rule out the development of a surgical mass lesion and guide treatment

*If ICP remains ≥ 20 - 25 mmHg proceed to Tier 2*
TIER 2

- In patients with a parenchymal ICP monitor an EVD should be considered to allow for intermittent CSF drainage
- Hyperosmolar therapy should be given intermittently as needed for ICP elevation and not on a routine schedule

- PaCO2 goal of 30 - 35 mmHg should be maintained, as long as brain hypoxia is not encountered. Additional neuromonitoring (e.g., PbtO2, SjvO2, CBF) may help determine optimal PaCO2
- Repeat CT imaging and neurological examination should be considered to rule out development of a surgical mass lesion and guide treatment
- Neuromuscular paralysis achieved with a bolus “test dose” of a neuromuscular blocking agent should be considered if the above measures fail to adequately lower ICP and restore CPP. If there is a positive response, continuous infusion of a neuromuscular blocking agent should be employed (Tier 3)
TIER 3

(includes potential salvage therapies)

- Decompressive hemi-craniectomy or bilateral be performed if treatments in Tiers 1 and 2 are limited by development of side effects of mec

- Neuromuscular paralysis via continuous infusion of a neuromuscular blocking agent can be employed if there is a positive response to a bolus dose. The infusion should be titrated to maintain at least two twitches (out of a train of four) using a peripheral nerve stimulator. Adequate sedation must be utilized

- Barbiturate or propofol (anesthesia dosage) coma may be induced for those patients who have failed to respond to aggressive measures to control malignant intracranial hypertension, however it should only be instituted if a test dose of barbituate or propofol results in a decrease in ICP, thereby identifying the patient as a “responder.” Hypotension is a frequent side effect of high dose therapy with these agents. Meticulous volume resuscitation should be ensured and infusion of vasopressor/inotropes may be required. Prolonged use or high dose of propofol can lead to propofol infusion syndrome. Continuous EEG may be used to ensure targeting of the infusion to burst suppression

Decompressive Craniectomy in Diffuse Traumatic Brain Injury

D. James Cooper, M.D., Jeffrey V. Rosenthal, M.D., Lynette Murray, B.App.Sci., Yaseen M. Arabi, M.D., Andrew R. Davies, B.M., B.S., Paul D’Urso, Ph.D., Thomas Kessmann, M.D., Jennie Ponsford, Ph.D., Ian Seppelt, M.B., B.S., Peter Reilly, M.D., and Rory Wolfe, Ph.D., for the DECRA Trial Investigators and the Australian and New Zealand Intensive Care Society Clinical Trials Group

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BEST PRACTICES IN THE MANAGEMENT OF TRAUMATIC BRAIN INJURY

Together, We Can
VTE Prophylaxis

- TBI patients are high-risk for VTE
- MOST patients qualify for pharmacologic prophylaxis within 72 hours
- Level III
  - LMWH or SQ heparin chemoprophylaxis may be considered if the brain injury is stable and the benefit is considered to outweigh the risk of increased intracranial hemorrhage.
# VTE Prophylaxis

<table>
<thead>
<tr>
<th>Low risk</th>
<th>Moderate risk</th>
<th>High risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>No moderate or high risk criteria</td>
<td>Subdural or epidural hematoma &gt; 8 mm Contusion or intraventricular hemorrhage &gt; 2 cm Multiple contusions per lobe Subarachnoid hemorrhage with abnormal CT angiogram Evidence of progression at 24 hrs</td>
<td>ICP monitor placement Craniotomy Evidence of progression at 72 hrs</td>
</tr>
</tbody>
</table>

| Initiate pharmacologic prophylaxis if CT stable at 24 hrs | Initiate pharmacologic prophylaxis if CT stable at 72 hrs | Consider placement of an IVC filter* |

*ACS TQIP BEST PRACTICES IN THE MANAGEMENT OF TRAUMATIC BRAIN INJURY*
VTE Prophylaxis

**VENOUS THROMBOEMBOLISM PROPHYLAXIS**

- For patients with intracranial hemorrhage, Lovenox (enoxaparin) 30 mg twice daily should be used for VTE prophylaxis. This should usually be started ONLY 48 hours AFTER time of injury and after at least 1 stable head CT obtained at least 24 hours out from injury, the patient should also have a stable neurologic exam. For hemorrhages of medium to large size, or with questionable neurologic status, the Neurosurgery Senior Staff may elect to hold enoxaparin. Unfractionated heparin can be used for prophylaxis as an alternative but its efficacy in these situations is indeterminate. Enoxaparin should NOT to be used in any patient requiring operative intervention for intracranial hemorrhage.

- For patients with spinal cord injury or spine fracture, Lovenox (enoxaparin) 30 mg twice daily should be used for VTE prophylaxis. Enoxaparin should NOT to be used in the perioperative period for patients requiring operative intervention, or in patients in whom there is concern for spinal cord hemorrhage due to the nature of their injury.
Summary

- Toolbox for the initial management of TBI.
  - TQIP 2015
  - Brain Trauma Foundation
  - HFH Institutional guidelines

- How can I apply some of this?
  - Have a shared goal.
  - Communicate
  - Collaborate.

www.braintrauma.org

Guidelines for the Management of Severe Traumatic Brain Injury
4th Edition

www.facs.org/quality-programs/trauma/tqip/best-practice
Thank you

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  – Trauma program manager