Traumatic Brain Injury

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Disease Demographics

- Traumatic brain injury (TBI) is a significant public health issue with over 2.5 million emergency department visits and more than 50,000 deaths annually. The economic impact is estimated to be over $75 million annually in the US and is probably underestimating the impact on loss of productivity and long-term care associated.
- Well- designed, controlled trials are lacking in most areas of care. Therefore groups of clinical and research experts have come together to examine the available data and provide expert opinion. Consensus from groups such as the American College of Surgeon's Trauma Quality Improvement Program (TQIP) and the Brain Trauma Foundation (BTF) have provided practical clinical guidelines supported with the best available evidence for recommendations. References to these two documents are listed here and the recommendations highlighted throughout the document.[1-2]

  https://www.facs.org/-/media/files/quality-programs/trauma/tqip/tbi_guidelines.ashx?la=en

  https://braintrauma.org/uploads/03/12/Guidelines_for_Management_of_Severe_TBI_4th_Edition.pdf

- All phases of care from pre-hospital and small rural facilities to quanternary trauma centers should employ tiered, goal-directed care for TBI based on their capability and capacity. The goals of care are to support to brain tissue by ensuring adequate oxygenation, avoiding hypotension and minimizing metabolic stress.

Tiered Management of Intracranial Injury

Tier I (accomplished in all levels of trauma center and in transport)

- The head of the bed should be elevated using reverse Trendelenburg. The head should be kept midline and cervical collar should be sufficiently loose as to not impede venous return from the head as increased venous pressure leads to increased intracranial pressure.
- Patients with diminished (GCS $\leq$ 8), or declining levels of consciousness should have their airways protected and sedation employed. Sedation with benzodiazepines, propofol, dexmetomindine and ketamine are all useful, depending on the anticipated duration of sedation and the patient's volume status. Ketamine 0.5mg/kg/hr titrated to effect has been shown to be useful for ICU sedation with mechanical ventilation and has excellent hemodynamic parameters.
- Efforts should be directed to maintaining normal blood pressure for age. This is best accomplished by ensuring adequate volume status. Transfusion with plasma increases intra-vascular volume, decreases inflammation and begins to correct coagulopathy. Transfusion with packed red blood cells should be reserved for a hemoglobin level of 7 g/dl or less according to the TQIP best practice recommendations. Thromboelastography may be considered for assessing further coagulopathy.
- Osmolar therapy can begin in the remote care areas with hypertonic saline. 3% Normal saline can be administered in a bolus of 3-5 cc/kg (up to 250 cc) over a 30-minute period.

Mannitol should be reserved in the field or rural stabilizing hospital until volume status is assessed to be adequate.

- Anti-epileptic prophylaxis is controversial with mixed results relating prophylaxis to outcomes. This may represent the heterogeneity in the practice. There appears to be no benefit of levetiracetam over phenytoin in seizure prophylaxis but there is a consensus that phenytoin causes more depression of the CNS. Levetiracetam dosing is 10 mg / kg for pediatric patients and 500 mg for adults. [3-4]
- All patients with TBI on an anticoagulant should have the anticoagulant reversed as soon as practical. Many of the Xa inhibitors can be monitored using anti –factor Xa activity and, if ingested within two hours of presentation, activated charcoal can be used to decrease the effect. KCentra (a four factor PCC) at 2000 units can be considered for decreasing the anticoagulant effect. Coumadin effect can be measured with INR and reversed with vitamin K+ and FFP infusions, as well as KCentra. Other drugs are eliminated partially by hemodialysis.
- Temperature should be maintained in the normal range of 36 – 37.5 degrees Celsius. This is important since increasing temperature increases the cerebral metabolic rate. PaCO2 goals should be 35 – 40mmHg and continuously trended with ETCO2 once correlated with blood gas data. PaCO2 levels in the normal range correlate with "normal" cerebral blood flow.
- Large traumatic hematomas should be evacuated prior to the patient deteriorating when possible. Large hematomas are those that cause midline shift, typically greater than five millimeters or compress the basal cisterns. Patients who arrive in the trauma bay with significant changes in their mental status and large hematomas should be taken urgently to the OR for evacuation. Drainage of these hematomas is performed through a wide craniotomy.
- Operation for depressed skull fractures depends on the location, the relationship to the sinuses and the degree of contamination. Not all depressed skull fractures need operative intervention and those overlying the sagittal sinus should be managed non-operatively. Open fractures of the skull should be treated the same as other open fractures, with first generation cephalosporins. A Cochrane review by Ratilal in 2015 demonstrated no benefit of prophylactic antibiotics for basilar skull fractures.[5]

Tier II (accomplished in tertiary trauma centers)

<u>ICP monitoring</u>[6]

- While data on the relationship between ICP monitoring and outcome is equivocal, the general consensus is that monitoring is appropriate for patients with structural brain injury and a sustained GCS $\leq$ 8 after resuscitation. Monitoring may also be indicated for those with higher GCS but lesions at high risk of progression, or in whom close clinical evaluation is not possible, such as prolonged extracranial operation.
- External ventricular drain (EVD) is the preferred method of monitoring because of its dual therapeutic potential. ICP thresholds between 20 and 25 mmHg are reasonable with targeted cerebral perfusion pressures (CPP) of 60 mmHg and no lower than 50 mm Hg. Additional monitoring may be helpful in identifying times of increased oxidative stress, hypoxia or changes in blood flow to the injured brain.

- Hyperosmolar therapy should be instituted with target Na+ goals of 145 – 150mEq/L within the first six hours of ICU stay. This is achieved with hypertonic saline, or hypertonic lactate in intermittent boluses or a continuous drip.
- PaCO2 goals may be decreased to 30-35 mmHg with sustained increased ICP but additional monitoring may be useful in determining the effect of this change on cerebral oxygenation. This maneuver will cause decreased blood flow to the brain thereby decreasing the volume in the fixed space, decreasing pressure. The consequence is that the areas of marginal perfusion may be further compromised. This, therefore, should be a temporizing maneuver.
- Neuromuscular paralysis may be tried as a test dose to see the effect on elevated ICP, once adequate sedation and analgesia is ensured. If paralysis is effective, then a continuous drip may be instituted and continuous EEG monitoring initiated to detect seizure activity. Paralysis should be titrated using neuromuscular twitch monitoring to two of four twitches.
- Persistent elevation of the ICP despite tier II therapies warrants consideration of repeat CT imaging to assess for new or expanding mass effect.

Multimodality Brain Monitoring

- Multimodality therapies are increasingly common in Neurocritical care units to monitor cerebral blood flow and cerebral oxygenation. Transcranial Doppler, thermal diffusion probes and $SjVO_2$ monitoring are all useful adjuncts to ICP monitors and EVD's. Tasneem etal. provide an excellent review of brain physiology and these monitoring adjuncts.[7]

How and When to Stop ICP and Brain Oxygenation Monitoring?

- There are no widely validated guidelines to answer this question. Based on clinical experience, monitoring can be stopped when the causes of the intracranial hypertension have resolved, the patient has been clinically and radiologically stable for at least two days, and there has been no evidence of worsening physiologic markers during gradual tapering of ICP reduction therapies. Prolonged use of ventricular catheters increases the risk of infection and prevents adequate anticoagulation and therefore should be avoided.

Tier III

- Careful discussion needs to take place at this stage between the treating trauma/critical care team and the neurosurgical team. Tier III therapies are considered salvage therapy and need to be tailored to the patient's needs. Options include decompressive craniectomy and chemically induced coma.
- Coma may be induced with either propofol, which has a potential risk of propofol infusion syndrome, or barbiturate, which may cause profound hypotension.
- Treatment with barbiturates often require the concomitant use of inotropic agents. Another consequence of barbiturate use is drug build up and a long elimination time. This may preclude brain death testing if institutional policy requires the drug to be out of the system prior to initiating testing.

- Either propofol or barbiturates should be given first as a test dose and instituted as a continuous drip only if the patient responds favorably to the single dose. The endpoint of chemically induced coma therapy is burst suppression on continuous EEG monitoring.
- Decompressive craniectomy can be performed early in the management, if the initial CT scan demonstrates significant swelling with impending herniation and a neurological exam with some evidence of brain function. In these cases, the ICU management moves directly to tier III and is maintained for a period of three to five days, or until anticipated swelling resolves.
    - All decompressive craniectomies should be performed through a large craniotomy, based either on the side of maximal swelling or in a bifrontal fashion if the swelling is diffuse.
- Interventions performed in the late phase following progression through tiered management have been shown to decrease ICP but not to definitively change outcome.

Special Considerations

Nutritional support

- Enteral nutrition should be started when the patient is hemodynamically stable and ideally within 24 hours of admission to the ICU. Full enteral fees should be accomplished as soon as possible following admission. Care should be taken when initiating or continuing enteral nutrition in patients on vaso-active drugs for increased CPP treatement. A guideline is that if the drug doses needed are realtively low feeds can be started or resumed:
  Epinephrine & Norepi $\leq$ 5 mcg/min, dopamine < 10mcg/kg/min, vasopressin < 0.04units / min and milrinone 0.375mcg/kg/min or less.
- Post-pyloric feeding has the advantage of decreasing the risk of pneumonia. However, post-pyloric feeding is not mandatory and use of the stomach is preferable to parenteral feeds.
- Patients that cannot be fed enterally may be fed parenterally with careful attention paid to maintaining euglycemia.

VTE prophylaxis[8-9]

- The incidence of VTE is as high as 30% for patients with severe TBI. Mechanical prophylaxis should be started on admission to the ICU. Chemical prophylaxis may be safely started within 24 hours of a stable CT in lower risk patients and within 72 hours of a stable CT for patients with moderate risk injuries.
- Chemical prophylaxis can typically be started within 24 hours of removal of an ICP monitor.

Timing of extra-cranial operations[10]

- Controversy exists in the optimal timing of orthopedic procedures in the face of severe TBI. Early operative fixation of skeletal and facial fractures has not been shown to worsen outcomes in patients with severe TBI and has been shown to decrease pulmonary complications in multisystem injury patients. Less urgent operations should be delayed

until the intracranial hypertension has been stable for 24-48 hours. ICP should be monitored during extra-cranial procedures when possible.

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Other Suggested Readings

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