

End Organ Perfusion Targeted Goal Directed Management of a Severe TBI Patient



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Introduction: Traumatic brain injuries (TBI) are a leading cause of death among trauma patients and when the outcome isn't faid they are associated with a decrease in a decrease in the decre meaningful recovery and typically multiple morbidities (1.2). Treatment of severe TBI patients is focused towards altempting to ameliorate the devastaling progressive effects of secondary brain injury that develop due to inflammation, ischemia, and oxidative stress that lead to neuronal damage in the setting of increased intracranial pressure (ICP) (1,2)

First tier treatments in these patients include:

- Elevation of the head of bed
- Adequate analgesia and sedation
- Hyperventilation
- Ventriculostomy and ICP monitoring
- Often osmotic therapy with hypertonic saline and/or mannitol (2)

Patients may develop refract ory intracranial hypertension (RICH) being ICP>20mmHg for more than 15 minutes all occurring within 1 hour of best management with first tier therapies (2). These situations require second tier therapies including:

- Pentobarbital coma (PBC)
- Decompressive craniectomy (DC) (2)

It is accepted that management of patients with RICH should be focused towards maintaining adequate cerebral perfusion pressure (CPP) as well as the avoidance of hypotension and hypoxia, how ever, no universal protocol exists regarding how to best accomplish these endpoints (4)

Case Presentation: 19 year old male trauma alert based on the prehospital report of being found unresponsive underneath an ATV. He was intubated on scene and initial GCS was 3T. He was hemodynamically stable on presentation.

On evaluation of this patient, he was found with

- Left temporal bone fracture
- Left sided subdural hematoma
- Extensive left sided subarachnoid hemorrhage associated with a left to right midline shift of 5mm

He was taken emergently to the operating room with neurosurgery where right sided Licox and Camino monitors were placed. At time of insertion, the patient's initial ICP ranged from 24-28mmHg and first tier therapies were initiated.

Maximal first tier therapies did not yield adequate ICP control and 9 hours after presentation, the patient required a decompressive craniectomy. After the procedure, the patient's ICPs remained in the 20s, but progressively increased to as high as 40mmHg.

Pent obarbit al therapy was initiated, with a response maintaining ICPs within the mid 20s. These second tier therapies were initiated in the first 24 hours of presentation.

The pent obarbit al therapy was discontinued on post trauma day (PTD) 11 along with the Licox and Camino monitoring. The patient's partial pressure of oxygen in brain tissue (PbtO2) remained >20mmHathroughout his ICU course. He was extubated on PTD 15 and best GCS was 14 (M 6 V4 E4). The patient progressed well and was discharged to a rehabilitation center on PTD 18.



Patient's appearance prior to cranioplasty and insertion of scdp tissue expander (1). Intraoperative appearance after tissue expander was placed for 1 month (2). Appearance after cranioplasty (3).

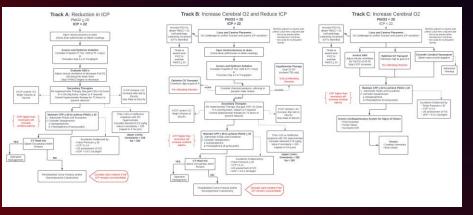
The patient was admitted to inpatient rehabilitation for 15 days and was ultimately discharged home. Two months after his injury, the patient underwent placement of tissue expander of the scalp and three months after his injury, underwent definitive cranioplasty. The patient did experience one seizure 10 months post trauma, which was attributed to THC ingestion and on further follow up has not experienced any further seizure like activity.

Conclusion:

- Current literature supports pent obarbital in setting of RICH
- Decompressive craniectomy decreases length of ICU stay and ICP, but increased the likelihood of poor long term out comes.
- We advocate for early, aggressive end organ perfusion targeted goal directed management of severe TBI patients. Our TBI protocol is targeted specifically towards maintaining a Pbt O2 of greater than or equal to 20mmHg and ICP less than 20mmHg.

Protocolized Management on 1 of 3 Tracks:

Patient responses to initiated therapies are assessed every 30 minutes with subsequent escalations in therapies. Given the current lack of universally accepted protocol for achieving optimal brain oxygenation and reduction of intracranial pressure, we propose early and aggressive optimization of these measures directed by intracranial monitoring as exemplified by our institution's TBI protocol.



¹Marshall GT et al. Pentobarbit al Coma For Refract ory Intra-Cranial ³Cooper JC et al. Decompressive Craniect omy in Diffuse Traumatic Hypert ension After Severe Traumatic Brain Injury: Mortality Predictions Brain Injury. The New England Journal of Medicine 2011;364:1493-1502 and One-Year Outcomes in 55 Patients. J Trauma Inj 2010 Aug;69(2):275-283

²Bernstein JE et al. Pentobarbital ComaWith Therapeutic Hypothermia for Treatment of Refractory Intracranial Hypertension in Traumatic Brain Injury Patients: A Single Institution Experience. Cureus 2020 Sep;12(9):e10591 PubM ed PM ID:33110727

⁴Carney N et al. Guidelines for the Management of Severe Traumatic Brain Injury, 4th Edition. Brain Trauma Foundation 2016 Sep

Resources

