Head Injury Management Update

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Traumatic brain injury (TBI) is one of the most serious, life-threatening conditions in trauma victims. Prompt and appropriate therapy is necessary to obtain a favorable outcome. Using national data from the Center for Disease Control (CDC), TBIs have the following impact in the United States each year:

1 million people are treated and released from hospital emergency departments;

230,000 people are hospitalized and survive;

50,000 people die each year;

More than 80,000 are discharged from the hospital with TBI-related disabilities.[1]

Head injury occurs most frequently in adolescents, young adults, and people older than 75 years of age. In all age groups, males are affected two times more often than females and are more likely to sustain severe head injury. The leading causes of TBI are motor vehicle crashes, falls and violence. More than 50% of patients with severe head injury have multiple injuries resulting in significant blood loss, systemic hypotension and hypoxia.[2]

Perioperative management of head-injured patients focuses on aggressive stabilization of the patient and avoidance of systemic and intracranial insults that cause secondary neuronal injury. Secondary brain injury may arise directly from activation of biochemical and inflammatory cascades, or indirectly following intracranial hypertension, reduced cerebral blood flow, vasospasm, hypoxemia, or increased cerebral metabolic rate. Secondary brain injury complicates the course of the majority of head-injured patients, adversely influencing outcome. These secondary insults are potentially preventable and treatable.

I. Head Injury Guidelines

In 1995**,** the Brain Trauma Foundation approved guidelines for the initial resuscitation of the severe head injury patient and treatment of intracranial hypertension recognizing the need to standardize care to improve outcome in head-injured patients.[3] A task force was formed in 1998 to review and update the scientific evidence for the guidelines. These evidence-based guidelines for the management of severe TBI were published in 2000 (Table 1).[2] This extensive review of the literature recommends three standards based on Class I evidence and several guidelines based on Class II evidence. An update specifically addressing cerebral perfusion pressure (CPP) management was published on the web in 2003.[4] In March, 2006, new guidelines were published by The Brain Trauma Foundation and The Congress of Neurological Surgeons.[5] This review presents literature-based recommendations for the surgical management of TBI. However, compared with the *Guidelines for the Management of Severe Traumatic Brain Injury,* there are no controlled clinical trials in the literature to support different forms of surgical management or surgical versus conservative therapy. As with the other guidelines in severe TBI, the authors state that "*this is a document in evolution*", and revisions will be made as new knowledge is gained.[5]

Table 1. Recommendations from Guidelines for the Management of Severe Traumatic Brain Injury (TBI)

Standards based on Class I evidence:

If ICP is normal, avoid chronic prolonged hyperventilation therapy ($PaC0_2$ < 25 mm Hg).
Administration of steroids does not improve outcome or reduce ICP.
Prophylactic use of anticonvulsants does not prevent late post traumatic seizures.

Guidelines based on Class II evidence:

All regions should have an organized trauma care system.
Avoid or correct immediately hypotension (systolic blood pressure < 90 mm Hg) and hypoxia ($Sp0_2$ < 90% or $Pa0_2$ < 60 mm Hg).
Indications for intracranial pressure (ICP) monitoring include Glasgow Coma Score 3-8 with abnormal CT scan or 2 or more of the following adverse features: age > 40 yrs, motor posturing, systolic blood pressure < 90 mm Hg.
Initiate treatment for ICP at an upper threshold of 20-25 mm Hg.
Avoid the use of prophylaxtic hyperventilation ($PaC0_2 \leq 35$ mm Hg) therapy during the first 24 hr after severe TBI.
Mannitol is effective for control raised ICP after severe TBI, in doses ranging from 0.25 g/kg body weight to 1 g/kg body weight.
High-dose barbiturate therapy may be considered in hemodynamically stable salvageable severe TBI patients with intracranial hypertension refractory to maximal medical and surgical ICP lowering therapy.
Provide nutritional support (140% of resting energy expenditure in nonparalyzed patients and 100% of resting energy expenditure in paralyzed patients) using enteral or parenteral formulas containing at least 15% of calories as protein by day 7 after injury

Data adapted from Robertson CS.[6]
Data adapted from Bullock RM, et al.[2]

II. Preanesthetic Assessment and Stabilization

Preanesthetic assessment of the head-injured patient includes: airway (cervical spine), breathing (ventilation and oxygenation), circulatory status, associated injuries, neurologic status (Glasgow Coma Scale), preexisting chronic illness, circumstances of the injury (time of injury, duration of unconsciousness, associated alcohol or drug use).

Secondary insults complicate the course of more than 50% of head-injured patients. An outcome study using data from the Traumatic Coma Data Bank revealed that hypotension after head injury is associated with greater than 70% of patients experiencing significant morbidity and mortality.[2] The combination of hypoxia and hypotension is significantly more detrimental (> 90% of patients with severe outcome or death).

Emergency Therapy. The first step is to secure an open airway and insure adequate ventilation to prevent secondary injury from hypoxia and hypercarbia. When a cervical spine fracture has not been excluded by radiographic evaluation, cervical alignment with manual in-line stabilization (MILS) is recommended during emergent intubation. (Please note that a cadaver study suggests that MILS does not limit movement across a complete C_{4-5} fracture dislocation with ligamentous injury). If facial fractures and soft tissue edema prevent direct visualization of the larynx, a fiberoptic intubation or intubation with an illuminated stylet may be attempted. In the presence of severe facial and/or laryngeal injuries, a cricothyrotomy may be required. Nasal intubations are avoided in the presence of a suspected basal skull fracture, severe facial fractures, and bleeding diathesis.

Following control of the airway in the head-injured patient, attention should focus on resuscitation of the cardiovascular system. A major concern during fluid resuscitation is the development of cerebral edema. Based on animal research, it appears that the best way to avoid cerebral edema during fluid resuscitation in the injured brain is to maintain normal serum osmolality and colloid oncotic pressure. Therefore, circulating blood volume should be restored to normovolemia with glucose-free isotonic crystalloids and colloid solutions. Glucose-containing solutions are avoided because of a significant association between elevated plasma glucose levels and worse neurologic outcome.

A full ATLS trauma evaluation is on-going as therapeutic interventions to control intracranial hypertension are instituted. The head is elevated to 15^0 and maintained in a neutral position. Mannitol (0.25 to 1 g/kg) is administered to acutely lower ICP. After tracheal intubation, the patient is given a muscle relaxant and mechanically ventilated to a $PaCO_2$ of 35 mmHg. Hyperventilation to a $PaCO_2$ of less than 30 mm Hg is avoided unless transtentorial herniation is suspected.

III. Intraoperative Management

Anesthetic Management. In some patients, severe intracranial hypertension precipitates reflex arterial hypertension and bradycardia (Cushing's triad). A reduction in systemic blood pressure in these patients can further aggravate cerebral ischemia by reducing **cerebral perfusion pressure** (CPP = MAP - ICP). CPP should be maintained between 60-110 mm Hg. The choice of anesthetic agents depends on the condition of the patient. Please refer to Bendo et al.[7] for a more complete discussion. Anesthetic management is directed at avoidance of secondary brain injury. Intraoperative hypotension secondary to blood loss or precipitated by anesthetic drugs must be avoided by appropriate volume expansion. Maintenance of ventilation ($PaC0_2 \geq 35$ mm Hg) and oxygenation ($Pa0_2 > 60$ mm Hg) is extremely important.

Brain Protection. Reducing the cerebral metabolic requirement for oxygen ($CMR0_2$) is the mainstay of pharmacologic brain protection, and barbiturate administration is the only such intervention that has proven useful in humans. Since many drugs that depress $CMR0_2$ do not protect the brain, additional mechanisms of action have been sought to explain why barbiturates sometimes do protect. In addition to lowering CMR, barbiturates often reduce elevated ICP that is refractory to hyperventilation and mannitol. Experiments in nonhuman primates indicate that some of the protective effects of barbiturates during focal ischemia can be attributed to vasoconstriction in areas of healthy brain that shunts cerebral blood flow to injured areas.

When ischemia reduces supply, hypothermia remains the *sine qua non* for reducing demand. A reduction of body temperature to 33 to 35^o C may confer cerebral protection. Although recent clinical trials of moderate hypothermia after head injury have been encouraging, none has shown statistically significant improvement in outcome. The multi-institutional study of postoperative mild hypothermia in head injury patients was terminated by its Safety Monitoring Board after enrolling 392 patients.[8] The results showed no difference in mortality between hypothermic and normothermic patients, and hypothermic patients experienced more medical complications. Subgroup analysis revealed that younger patients (45 years or younger), who were hypothermic on admission and

assigned to the hypothermic group, tended to have better outcomes than those assigned to the normothermic group. A new study looking at this group with an earlier induction of hypothermia and more consistent critical care has been initiated.

It remains unclear whether there is a therapeutic "window of opportunity" for inducing protective post-injury hypothermia. When induction of hypothermia is elected, it is recommended that meticulous care is necessary to avoid adverse side effects such as hypotension, cardiac arrhythmias, coagulopathies, and infections. Rewarming should be carried out slowly.[9] In this population, there is no doubt that hyperthermia associates strongly with poor outcome.

IV. Postoperative Care/Critical Care.

In the critical care unit (CCU), the main objectives are to optimize recovery from primary brain injury and prevent secondary injury. This requires provision of optimal systemic support for cerebral energy metabolism and adequate CPP, and normalizing of ICP for injured brain (Table 2). Prompt recognition and treatment of systemic complications that contribute to secondary injury are essential to head injury management. To achieve this, multimodality systemic and cerebral monitoring should be instituted.[10] Monitoring of ICP, CPP and CBF (or TCD, LDF) should be standard practice. Monitors of cerebral oxygenation e.g. jugular bulb oximetry and brain tissue $p0_2$, and brain metabolism can provide more specific information for managing cerebral hypoxia and ischemia. There is controversy concerning the best management protocol for optimizing outcome in TBI patients.[6] A management protocol that uses individualized assessment and a multi-targeted approach to optimize therapy and reduce the risk of iatrogenic injuries is gaining acceptance.[11]

Table 2. Severe Head Injury Patient (GCS 8 or less)

Treatment of Intracranial Hypertension

1. Insert ICP monitor
2. Maintain CPP > 60 mm Hg.

First Tier Therapy:
- Ventricular drainage (if available)
- Mannitol 0.25 - 1 g/kg IV (may repeat if serum osmolarity < 320 mOsM/L and patient euvolemic)
- Hyperventilation to $PaC0_2$ 30-35 mm Hg

Second Tier Therapy:
- Hyperventilation to $PaC0_2$ < 30 mm Hg - ($Sj0_2$ $AVD0_2$ and/or CBF monitoring recommended)
- High dose barbiturate therapy
- Consider hypothermia
- Consider hypertensive therapy
- Consider decompressive craniectomy

Adapted from the Guidelines for the Management of Severe TBI[2] and The Brain Trauma Foundation Update Notice[4]

V. Summary

The major goal of perioperative management of TBI patients is to prevent secondary damage. Therapeutic measures based on established guidelines and recommendations must be instituted promptly throughout the perioperative course. Appropriate selection of anesthetics and meticulous general management of respiration, circulation, metabolism, fluid replacement, and temperature are all essential to improve outcome in this high risk population.

References and Suggested Reading

1. Brain Injury Statistics. CDC programs in brief from the 2000-2004 neurotraumaregistry.com.

2. Bullock RM, Chesnut RM, Clifton GL, et al. Management and prognosis of severe traumatic brain injury. Part I: Guidelines for the management of severe traumatic brain injury. J Neurotrauma. 2000;17:451.

3. Guidelines for the management of severe head injury. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care. J Neurotrauma. 1996;13:641.

4. The Brain Trauma Foundation Update Notice: Guidelines for the management of severe traumatic brain injury: Cerebral perfusion pressure. 2003. Available from http://www2.braintrauma.org/guidelines/.

5. The Brain Trauma Foundation and The Congress of Neurological Surgeons. Guidelines for the Surgical Management of Traumatic Brain Injury. Neurosurgery Supplement. 2006; 58(S2): 1.

6. Robertson CS. Management of cerebral perfusion pressure after traumatic brain injury. Anesthesiology. 2001; 95:1513.

7. For additional references, see Bendo AA, Kass IS, Hartung J, Cottrell JE. Anesthesia for Neurosurgery. In Barash PG, Cullen BF, Stoelting RK (eds): Clinical Anesthesia, 5th ed, Lippincott Williams & Wilkins, Philadelphia, 2006, p 746-789.

8. Clifton GL, Miller ER, Choi SC, et al. Lack of effect of induction of hypothermia after acute brain injury. NEJM. 2001; 344: 556.

9. Polderman KH. Application of therapeutic hypothermia in the ICU: Opportunities and pitfalls of a promising treatment modality. Part 1: Indications and evidence. Intensive Care Med. 2004; 30: 556.

10. Cremer OL, van Dijk GW, Amelink GJ, et al. Cerebral hemodynamic responses to blood pressure manipulation in severely head-injured patients in the presence or absence of intracranial hypertension. Anesth Analg. 2004; 99: 1211.

11. Warner DS, Borel CO. Treatment of traumatic brain injury: One size does not fit all. Anesth Analg. 2004 99:1208.

12. Fritz HG, Bauer R. Secondary injuries in brain trauma: Effect of hypothermia. J Neurosurg Anesthesiol. 2004; 16: 43.

13. Doppenberg EMR, Choi SC, Bullock R. Clinical trials in traumatic brain injury: Lessons for the future. J Neurosurg Anesthesiol. 2004; 16: 87.

14. Edwards P, Arango M, Balica L, et al. Final results of MRC CRASH, a randomized placebo-controlled trial of

intravenous corticosteroid in adults with head injury-outcomes at 6 months. Lancet. 2005; 365: 1957.

15. Roberts I, Yates D. Sandercock P, et al. Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head injury (MRC CRASH trial): randomized placebo-controlled trial. Lancet. 2004; 364:1321.
16. Jeremitsky E, Omert LA, Dunham M, et al. The impact of hyperglycemia on patients with severe brain injury. J Trauma. 2005;58: 47.
17. Cruz J, Minoja G, Okuchi K, Facco E. Successful use of the new high-dose mannitol treatment in patients with Glasgow Coma Scale scores of 3 and bilateral abnormal pupillary widening: A randomized trial. J Neurosurg. 2004;100:376.
18. Marshall LF. High-dose mannitol. J Neurosurg. 2004;100:367.
19. Cooper DJ, Myles PS, McDermott FT, et al. Prehospital hypertonic saline resuscitation of patients with hypotension and severe traumatic brain injury. JAMA.. 2004; 291:1350.