Pediatric Brain Injury

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Introduction: Pediatric neurotrauma (traumatic brain injury [TBI]) is the leading cause of death in children over one year of age. TBI should be considered in all children following trauma, particularly those with a suspicious mechanism of injury, loss of consciousness, multiple episodes of emesis, tracheal intubation and extracranial injuries. Children are more susceptible to TBI because they have a larger head to body size ratio, thinner cranial bones providing less protection to the intracranial contents, less myelinated neural tissue which makes them more vulnerable to damage, and a greater incidence of diffuse injury and cerebral edema compared to adults. Children have a higher incidence of increased intracranial pressure (ICP) following TBI than adults (80% vs. 50%). Diffuse TBI is the most common type of injury and results in a range of injury severity. The diagnosis of TBI is primarily made by computed tomography (CT) of the brain and is associated with increased ICP. Patients with diffuse axonal injury may initially have a normal CT scan despite significant neurological findings, and increased ICP; repeat CT scan often shows secondary injury due to cerebral edema. The acute treatment of pediatric TBI is directed at preventing secondary injury from systemic hypotension, hypoxia, hypocarbia, and hyperglycemia.

Physiology and Pathophysiology

*Cerebral Metabolic Rate (CMR), Cerebral Blood Flow (CBF), Cerebral Autoregulation and ICP:* Global CMR for oxygen and glucose is higher in children than in adults (oxygen 5.8 vs. 3.5 mL/100g brain tissue/min and glucose 6.8 vs. 5.5 mL/100 g brain tissue/min respectively). Unlike in adults, CBF changes with age and may be higher in girls compared to boys. Following TBI, CBF and CMRO₂ may not be matched, resulting in either cerebral ischemia or hyperemia but recent work demonstrates that the incidence of cerebral hyperemia is only between 6-10%, and that CMRO₂ may be normal, low or high after TBI. Data suggest that healthy infants may autoregulate CBF as well as older children during low dose sevoflurane anesthesia. Similar to adults, the incidence of impaired cerebral autoregulation is higher following severe compared to mild TBI (42% vs. 17%) and children with impaired cerebral autoregulation early after TBI may have poor long term outcome. One potential explanation for this association may be hypotension, which is common after pediatric TBI, and may lead to cerebral ischemia. In adults, normal intracranial pressure (ICP) is between 5 and 15 mmHg compared to 2-4 mmHg in young children. Unlike the adult with relatively poor cranial compliance, the infant with open fontanelles may be able to accommodate slow and
small increases in intracranial volume by expansion of the skull. However, rapid expansion of intracranial volume, small as it may be, can explain the not uncommonly encountered rapid deterioration in infants following TBI.

Clinical Management
Initial Assessment: The initial approach to the traumatized child involves the primary and secondary surveys, and definitive care of all injuries and the principles outlined in the 2003 Brain Trauma Foundation Guidelines for managing children with severe TBI. The GCS score (modified for children) is the most commonly used neurological assessment.

Airway Management: The most important therapy during the primary survey phase is to establish an adequate airway. The lucid and hemodynamically stable child can be managed conservatively but if the child has altered mental status, attempts should be made to establish the airway by suctioning the pharynx, chin-lift and jaw thrust maneuvers, or insertion of an oral airway. Children with a GCS score < 9 require tracheal intubation for airway protection, and management of increased ICP. However, recent studies demonstrate no survival or functional advantage of pre-hospital tracheal intubation compared to pre-hospital bag-valve-mask ventilation in pediatric TBI. The most common approach to tracheal intubation remains direct laryngoscopy and oral intubation with cricoid pressure after induction of anesthesia, ventilation with 100% oxygen, under in-line stabilization, without traction. Naso-tracheal intubations are contraindicated in patients with basilar skull fractures.

Anesthetic Technique: Most recommendations regarding choice of anesthetic technique and monitoring are extrapolated from data in adults. All intravenous sedative hypnotic induction agents, including barbiturates, etomidate and propofol, that are used to facilitate tracheal intubation are potent cerebral vasoconstrictors, cause coupled reduction in CBF and CMRO₂, and can decrease ICP. Opioids and benzodiazepines can be safely used to facilitate tracheal intubation but should be used in small doses. Ketamine should probably be used with caution in patients with TBI. Lidocaine is commonly used as an anesthetic adjunct to prevent increases in ICP induced by laryngoscopy and tracheal intubation in patients whose hemodynamic instability precludes use of large doses of sedative hypnotic agents. All inhalational agents are cerebral vasodilators but < 1 MAC of sevoflurane does not increase middle cerebral blood flow velocities compared to other agents. Sevoflurane may be the preferred volatile agent. Nitrous oxide can increase ICP. Muscle relaxants have little effect on the cerebral circulation. Succinylcholine can be safely administered without causing increase in ICP with or without a defasiculation dose of nondepolarizing muscle relaxant. Succinylcholine is a better choice than rocuronium if there are any concerns for a
difficult airway. Obtaining vascular access in the traumatized child can be very challenging. A well functioning 20 gauge or larger peripheral intravenous catheter will suffice for induction of anesthesia. Saphenous veins are commonly used. A second intravenous line should be started after induction. In emergent cases, if peripheral access is unsuccessful after 2 attempts, an interosseous line should be placed. Central venous catheters should be inserted by experienced personnel. Children can become hypovolemic from scalp injuries and isolated TBI. Isotonic crystalloid solutions are commonly used during the anesthetic and for cerebral resuscitation. Hypotonic crystalloids should be avoided and the role of colloids is controversial. The use of hydroxyethyl starch is discouraged because of its role in exacerbating coagulopathy. Hypertonic saline 0.1-1.0 ml/kg may be used to lower ICP and improved CPP. Retrospective studies suggest that hyperglycemia (glucose > 200mg/100 mL) is associated with poor outcome.

**Monitoring:** Standard ASA monitors, and invasive arterial blood pressure monitoring is recommended. Central venous pressure monitoring can be useful. The internal jugular line may be safely placed and used without increasing ICP. Retrograde jugular venous saturation monitoring can be useful to guide the degree of hyperventilation in patients with TBI but is not standard of care. ICP monitoring is useful during surgery involving extracranial injuries since cerebral perfusion pressure can be calculated but any preexisting coagulopathy must be treated prior to monitor placement. Urine output must be monitored. It is unclear at this time whether hypothermia affords any benefits in children with TBI but a recently published study by Hutchison and colleagues shows harm. Hourly arterial blood gases, and tests of coagulation need to be examined. ICP monitoring should be used to guide blood pressure management in children with TBI undergoing non-neurosurgical procedures. 

**Hemodynamics (Intracranial Pressure and Blood Pressure):** The presence of the Cushing’s reflex and autonomic dysfunction might be the only indicators of increased ICP. While SBP < 5th percentile defines hypotension, in the absence of ICP monitoring and suspected increased ICP, supranormal systolic blood pressure may be needed to maintain cerebral perfusion pressure (CPP). At a minimum, MAP should not be allowed to decrease below values normal for age by using vasopressors.

**Summary:** Pediatric TBI results in large societal costs. Therefore, efforts to improve outcome are extremely important. Although many general principles of managing pediatric TBI are similar to adults, there are unique anatomic, physiological and patho-physiological features of children with TBI worth recognizing.


5. Pediatric Trauma in Advanced Trauma Life Support Course for Physicians. USA, American College of Surgeons, 1993 pp 261-281.


