Abstract:

Traumatic brain injury (TBI) is a major public health problem in the United States. In children, it is a leading cause of morbidity and mortality. The purpose of this article is to review the epidemiology, pathophysiology, and the evidence for management of children with TBI in the emergency department. The main objectives of TBI management are to stabilize and resuscitate and to avoid secondary brain injury (hypoxemia, hypercapnia, hypotension, hyperthermia, electrolyte imbalance, and seizure). This article will also review the evidence for the management of children with mild and moderate TBI.

Keywords:

traumatic brain injury; intracranial pressure; autoregulation; cerebral blood flow

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Acute Management of Children With Traumatic Brain Injury

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raumatic brain injury (TBI) is a major public health issue in the United States. In children, it is a leading cause of morbidity and mortality. Over the last 20 years, the creation of specialized pediatric trauma centers and adult trauma centers with qualifications in pediatrics has improved the outcome of children treated for head injury. In 2003, the first edition of the "Guidelines for the Acute Medical Management of Severe TBI in Infants, Children, and Adolescents, 2" was published and an updated edition of these guidelines was released in 2012.³ Only recently has 1 pediatric study demonstrated an association between adherence to these guidelines and improved outcome.⁴ Indeed, the adherence to the 2003 guidelines within the first 72 hours of medical management of children with severe TBI revealed lower hospital mortality and enhanced neurologic outcome. 4 These important findings underline the importance of following the pediatric TBI guidelines in the emergency department (ED) and pediatric intensive care unit settings. The purpose of this article is to review the epidemiology and pathophysiology of TBI in children and to suggest a stepwise approach to the management of these patients in the ED (Figure 1).

EPIDEMIOLOGY

Unintentional injuries are the leading cause of death in children aged from 1 to 19 years, accounting for 42% of all deaths in this age group. 5 Approximately, 700 000 children with TBI present annually

Initial Resuscitation General Principles C-Spine immobilization Head positioning to facilitate venous Monitoring drainage Normothermia (35.5 to 37°C) Adequate analgesia and sedation Airway Normo or hypernatremia Patency, secretions Avoid hypoglycemia Breathing Avoid anemia O_2 for sat > 95%PaCO₂ 35-45 mmHg Seizure prophylaxis Circulation Assess for necessity of intracranial Avoid hypotension BP > 95th percentile pressure (ICP) Aggressive fluid resuscitation monitoring Prepare for safe intubation Neuroimaging **ICP Crisis** Head CT Hyperosmolar therapy Identify neurosurgical Hyperventilation emergencies Integrity of bone and soft tissue, presence of hemorrhage and edema

Figure 1. Proposed algorithm for management of severe TBI.

to an ED in the United States. 6 Of these, 60 000 children require hospitalization and 6200 children die. Children aged younger than 4 years are highly vulnerable to TBI. Most TBIs in children are secondary to falls (50%), being struck by/against an object (25%), and motor vehicle or traffic injury (6.8%).

The Glascow Coma Scale (GCS) is a clinical classification used to quantify the severity of TBI based on the patient's eye, verbal, and motor examination. A GCS score between 13 and 15 is considered a mild TBI, a score between between 9 and 12 is considered moderate, and a score of 8 and less is viewed to reflect a severe TBI. The severity of the injury, assessed by the GCS score and the nature of the lesion, is closely related to the outcome.⁷ Long-term sequelae may include academic, cognition, and neurobehavioral deficits. Younger children show limited recovery and have worse long-term neurocognitive and psychosocial outcomes in term of attention, speed of information processing, effective executive skills, and social integration.⁷

PATHOPHYSIOLOGY

The primary injury after TBI consists of the mechanical damage sustained at the time of the trauma event. This injury can be classified as follows: skull fracture, soft tissue injury, extraaxial hemorrhage (epidural, subdural, subarachnoid, and intraventricular hemorrhage), intracranial lesions (diffuse axonal injury, cortical contusion, and intraparenchymal hemorrhage), and vascular injury. Secondary brain injuries are the result of events after the initial TBI and include hypoxemia, hypercapnia, hypotension, and metabolic disturbances. These secondary injuries can develop and

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