HHS Public Access

Author manuscript

J Pediatr Health Care. Author manuscript; available in PMC 2016 May 01.

Published in final edited form as:

J Pediatr Health Care. 2015 ; 29(3): e1–e7. doi:10.1016/j.pedhc.2014.09.003.

Moderate-to-Severe Traumatic Brain Injury in Children: Complications and Rehabilitation Strategies

Myra L. Popernack, MSN, RN, CPNP,

Pediatric Nurse Practitioner, Department of Pediatric Rehabilitation & Development, Penn State Hershey Children's Hospital, Hershey, PA.

Nicola Gray, BSN, RN, and

Student, Duke University School of Nursing, Durham, NC.

Karin Reuter-Rice, PhD, CPNP-AC, FCCM [Assistant Professor]

Robert Wood Johnson Scholar 2013-16, Duke University Schools of Nursing and Medicine, and Department of Pediatrics, Duke Institute for Brain Sciences, Durham, NC.

Abstract

Traumatic brain injury (TBI) is the leading cause of death in children in the United States. Each year 37,200 children sustain a severe TBI, with up to 1.3 million life-years potentially adversely affected. Severe pediatric TBI is associated with significant mortality and morbidity. Of the children who survive their injury, more than 50% experience unfavorable outcomes 6 months after the injury. Although TBI-associated death rates decreased between 1997–2007, disabilities for TBI survivors continue to have both a direct and indirect impact on the economic and human integrity of our society. The degree of disability varies with the severity and mechanism of the injury, but a realm of physical and emotional deficits may be evident for years after the injury occurs. This article describes the pathophysiology of moderate to severe TBI, its associated complications, and opportunities to improve patient outcomes through use of acute management and rehabilitation strategies. To address the many challenges for TBI survivors and their families, including significant financial and emotional burdens, a collaborative effort is necessary to help affected children transition seamlessly from acute care through long-term rehabilitation.

Keywords

Traumatic brain injury; pediatric; Rancho; Glasgow Coma Score; outcome; cognitive function

Traumatic brain injury (TBI) is the leading cause of death in children in the United States. In 2010, 2.5 million Americans sustained a TBI ranging from mild to severe (Centers for Disease Control and Prevention [CDC], 2014). Each year 37,200 children sustain a severe TBI, with up to 1.3 million life-years potentially adversely affected. According to the CDC, 7440 children died of TBI in 2005, and the full extent of the injury is likely underestimated

Correspondence: Myra L. Popernack, MSN, RN, CPNP, Penn State Hershey Children's Hospital, 500 University Dr, Hershey, PA 17033; mpopernack@hmc.psu.edu..

Conflicts of interest: None to report.

(CDC, 2014). Based on the current best estimates, severe pediatric TBI has a 20% mortality, with a 50.6% unfavorable 6- month outcome. Although TBI-associated death rates have decreased between 1997–2007, disabilities for TBI survivors continue to have both a direct and indirect impact on the economic and human integrity of our society (Aitken et al., 2009; Coronado et al., 2011). The degree of disability varies with the severity and mechanism of the injury, but physical, cognitive, emotional, and behavioral deficits may be evident for years after the injury. This article describes the pathophysiology of moderate to severe TBI, its associated complications, and opportunities to improve patient outcomes using acute management and rehabilitation strategies. To address the many challenges for TBI survivors and their families, including significant financial and emotional burdens, a collaborative effort is necessary to help affected children and their families transition seamlessly from acute care through long-term rehabilitation.

DEFINITION AND EPIDEMIOLOGY OF TBI

The CDC defines an acquired brain injury as being caused by “a bump, blow or jolt to the head or a penetrating head injury that disrupts the normal function of the brain” (CDC, 2014). The injury can be either diffuse, such as a closed head injury from head-to-head contact in football, or a focal penetrating injury from a bullet or sharp object. Acquired brain injuries may result from strokes or other embolic events, infectious diseases, hypoxia/anoxia events, or as postsurgical sequelae.

Recent data from the CDC have shown that about one-half million of the 1.7 million civilian Americans who sustain a TBI are children ages 14 years and younger. One study estimated that at least 145,000 children were living with a TBI-related disability in 2005 and that the overall total life costs (medical costs and productivity losses) of injuries for children younger than 14 years was $60.4 billion (Corso, Finkelstein, Miller, Fiebelkorn, & Zaloshnja, 2006; Zaloshnja, Miller, Langlois, Selassie, 2008). Causes include falls, motor vehicle crashes, being struck in the head by a moving object or against a stationary one, and assault.

Falls account for about one third of the moderate to severe TBI in children 0–14 years of age, the majority of which occur on the playground for children younger than 10 years. Motor vehicle crashes remain the leading mechanism of injury for death and disability in pediatric TBI. Males are more likely to be injured than females and also have a greater likelihood of fatality as a result of higher injury acuity (CDC, 2014). The injured often have increased risk factors associated with preexisting co-morbidities, such as risk-taking behaviors, learning disabilities, or mental health disorders.

For children who survive brain injury, the mechanism, location, and extent of injury and the immediate interventions used to minimize secondary brain injury significantly influence neurocognitive and motor recovery necessary for independent functioning. Frontal lobe dysfunction is frequently exhibited and results in executive functioning deficits. Executive function involves interrelated skills that are needed to achieve goal-directed behaviors, such as inhibition, attentional control, and a working memory for appropriate planning, problem solving, and processing information. These skills are necessary for success in advancing to

functional independence. Impulsivity and perseveration are commonly associated with frontal lobe dysfunction. In addition to poor focus and attention, short- or long-term memory deficits impair new learning or retrieval of previously learned information (Catroppa & Anderson, 2006). Motor impairment may include paresis, flaccidity, tremors, or spasticity, which impedes the ability to perform activities of daily living. Coordination and proprioceptive or balance issues result in safety concerns. Alterations in sensation may yield auditory or visual impairment, as well as altered perception of touch, with neuropathies and paresthesias resulting in pain or discomfort. Emotional lability may be exhibited as depression, anxiety, irritability, and even aggression, leading to safety concerns for the child and others around him or her.

PHYSIOLOGIC MECHANISMS

When a TBI is sustained, the damage to the brain can be separated into primary injury and secondary injury. Primary injury is the instantaneous damage to the intracranial contents resulting from mechanical forces. Secondary injury is the subsequent damage that occurs over hours to days as a result of altered cerebral blood flow and inflammatory processes. The initial stabilization of the injured child in the prehospital and emergency department setting is focused on minimizing sequelae of the primary injury. Prevention of secondary injury and maximizing positive outcomes is ongoing throughout the intensive care and rehabilitation settings.

Primary injury occurs at the time of impact and is a direct effect of kinetic energy transferred from the mechanical force to the parenchyma. Some common mechanisms of injury include direct impact from an object, rapid acceleration/deceleration as seen in motor vehicle accidents, penetrating injury, and blast waves, which can be the result of an explosion. The acute injury of the parenchyma can be manifested as contusions, hematomas, shearing of white matter tracts, and cerebral edema.

Secondary injury occurs over time as a result of repercussions of the acute injury. Cerebral blood flow is often altered as a result of vasospasm, focal microvascular occlusion, and vascular injury. This secondary ischemia can lead to hypoxia, altered electrolytes and cell metabolism, and neuronal cell death. Cerebral edema of either cytotoxic or vasogenic origin is also a major contributor to secondary injury and often results in increased intracranial pressure and further compromise to cerebral blood flow (Greve & Zink, 2009).

OUTCOME SCORING INSTRUMENTS

The Glasgow Coma Score (GCS) is the most commonly used measure of primary injury in the prehospital and acute care setting for grading TBI severity. The GCS is a standardized 15-point scale with three dimensions: eye opening, best verbal response, and best motor response. The resulting score reflects the primary event of direct brain injury to the parenchyma. A GCS of 13 to 15 indicates a mild brain injury, a GCS of 9 to 12 indicates a moderate brain injury, and a GCS of 3 to 8 is indicative of a severe brain injury (O'Brien, 2012). Recent studies have investigated the predictive value of the initial GCS for morbidity and long-term injury outcomes after TBI. Although there appeared to be a correlation between score and outcomes, other factors influenced outcomes, such as injury severity

scores, associated hypoxicischemic injury, and provision of emergency care provided at a trauma versus nontrauma center (Cicero & Cross, 2013; Nesiama, Pirallo, Lerner, & Hennes, 2012).

The Glasgow Outcome Scale (GOS) score has been used to define neurologic outcomes after TBI. The GOS score ranges from 1 (death), 2 (vegetative), 3 (severely disabled) to the more positive outcomes of 4 (moderately disabled) and 5 (good recovery). Immediate and aggressive interventions to address associated hypoxia and hypotension have been shown to prevent secondary brain insult and therefore positively affect the GOS (Zebrack et al., 2009).

The Rancho Los Amigos Scale, also known as Rancho Levels of Cognitive Functioning, includes an eight-level behavior/response scale developed in 1972 at the Rancho Los Amigos Hospital (Hagen, Malkmus, & Durham, 1972). The scale evaluates the patient's interaction with environmental stimulation as an indication of the stage of recovery from injury. Many of the responses are associated with executive functioning, such as judgment, reasoning, attention and focus, memory, orientation to environment, and appropriateness of verbalizations and actions. Also evaluated is the consistency of responses such as nonpurposeful behavior, agitation, or following commands to various stimuli. The scale ranges from level I (unresponsive to stimuli) to level VIII (purposeful and appropriate response to stimuli).

As injured children move to higher levels of recovery, there are recommended strategies to suggest for families to assist and support the child, to promote comfort, and to provide orientation to the environment. Even at level VIII of the Rancho Levels of Cognitive Functioning, children may continue to have difficulty in new situations. They may become stressed or easily overwhelmed when challenged, have difficulty with problem solving, and/or need guidance in decision making. The variability in agitation level at level IV presents a particularly difficult stage of recovery, not only in terms of safety but also of contributing to the family's anxiety at seeing the child appear uncomfortable and difficult to calm. Because the child may advance to this stage while in the intensive care setting, it can be challenging for care-givers to differentiate agitation as a stage of neurocognitive recovery from withdrawal from narcotics or sedatives. One can reassure the family that once a child reaches this level, progress generally advances forward to additional stages of cognitive recovery.

ACUTE AND REHABILITATIVE MANAGEMENT GOALS FOR TBI

In 2012, the Society of Critical Care Medicine released updated Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents-Second Edition (Kochanek et al., 2012) These guidelines reflected the latest research and best practice recommendations to optimize recovery and outcomes. Clinicians now have a nationally standardized, evidence-based, goal-directed approach to care.

The first acute injury management goal is to promote neuroprotection and cerebral perfusion. Medical modalities are focused on the prevention of intracranial hypertension, systemic hypotension, hypoxemia, hypocarbia or hypercarbia, and hypoglycemia.

Additionally, therapies that promote normothermia and prevent of seizures are used, and devices that monitor and/or reduce intracranial pressure are standard of care. When intracranial pressure is refractory to medical management or the risk of uncal herniation is eminent, surgical intervention by a decompressive craniectomy may be required. By resecting a segment of cranial bone in the parietotemporal area and by opening the dura mater, alleviation of pressure and evacuation of hematomas is possible. A decompressive craniectomy may be performed unilaterally or bilaterally. The dura mater is then closed with synthetic material and the resected bone segment is cultured and placed in sterile frozen storage for a future cranioplasty. Complications associated with craniectomy are hygromas, infection, hydrocephalus, or paradoxical brain herniation. When the brain edema is resolved as indicated by computerized tomography, the harvested bone segment is surgically replaced at about 6 to 12 weeks, based on the neurosurgeon's assessment. If the autologous bone flap is unable to be used, alternative implant materials such as titanium may be used (Beauchamp et al., 2010; Suarez et al., 2011) Bone flap replacement is preferred as soon as deemed possible by the neurosurgical and rehabilitative team. The wearing of protective head gear until replacement is based on physician preference and patient safety needs. Prior to cranioplasty, the site of absent bone flap must be monitored for fluctuations in fluid levels, which may indicate complications as previously listed. The absent bone flap and subsequent replacement of the bone segment may produce significant anxiety for families, requiring both reassurance and education about the procedure and process for reconstruction.

Rehabilitation goals are established upon admission and re-evaluated throughout recovery. The long-term goal is to maximize the child's functional independence and neurocognitive abilities in developmental age-appropriate activities of daily living. Strategies are used to meet these goals within physical and cognitive limitations while utilizing the child's strengths. Two theories for the mechanism of recovery from TBI have been suggested: restitution and substitution. Restitution reflects the early postinjury natural course of physiologic healing and recovery that occurs with reactivation of neural pathways and restoration of function. Substitution reflects the transmission of neural function from injured to noninjured brain tissue so that structural reorganization and compensation occur. Although an overlap of these two mechanisms occurs in the acute phases, substitution is thought to be the predominant mechanism after 6 months when new learning occurs (Catroppa & Anderson, 2006). Interesting research has been done regarding time to follow commands and duration of posttraumatic amnesia as predictors of overall functional outcomes as measured by WeeFIMII scores (Suskauer et al., 2009).

Rehabilitation is focused on using alternative strategies to compensate for cognitive deficits, facilitate neurocognitive recovery and motor skill development, manage comorbidities and minimize complications, and maximize potential for functional independence at the level of impairment. Reintegration into the child's home, school, and community is a common goal, which is influenced by parental/caregiver education as well as available online resources (Table). Reintegration into the child's previous school system begins in acute care with initiation of contact with educators to gather information about the child's previous level of functioning. This information will be very useful to provide the proper support with consideration of prior learning needs. Discharge planning begins upon admission as well to establish a medical home and ongoing outpatient care in the child's local area.

Once the child achieves hemodynamic stability and remains stable in the acute care unit, initial rehabilitation needs are addressed by the acute care therapists. Upon admission to inpatient rehabilitation, evaluation of the child's functional independence is made using the WeeFIM II System (Uniform Data System of Medical Rehabilitation, Amherst, NY). This 18-item performance-based instrument assesses the child's mobility, self-care, and cognitive abilities compared with peers of similar age for ages 6 months to 7 years of age. The instrument may be used as well for patients up to 21 years of age who have delayed development of functional abilities. Scores range from level 1 (complete dependence for skills) up to level 7 (complete independence). Items include self-care, sphincter control, transfers, locomotion, communication, and social cognition. A baseline evaluation is performed every 8 hours of the first 24 hours (three assessments) to allow for variation in fatigue or participation. The lowest score is used to compare with a single measurement taken at discharge. The difference between scores reflects total functional gain achieved during the rehabilitation stay. The length of hospital stay is also documented to interpret functional gains per patient days. These data are tabulated by the Uniform Data System of Medical Rehabilitation (UDS) for use in comparing rehabilitation facilities nationwide for the purposes of benchmarking (UDS, 2006).

COMMON PROBLEMS IN TBI MANAGEMENT

Dysautonomia is estimated to affect about one third of patients after moderate to severe TBI in the first few weeks after brain injury. The constellation of symptoms includes tachycardia, hyperthermia, diaphoresis, muscle over-reactivity, increased respiratory effort or rate, hypertension, and pupillary dilatation. This response is commonly referred to by numerous names such as brain or thalamic storming, autonomic dysreflexia, and, more recently, paroxysmal sympathetic hyperactivity (Perkes, Baguley, Nott, & Menon, 2010). These symptoms may be incorrectly identified as narcotic withdrawal. The incidence of a triad of symptoms, namely hypertension, dysto nia, and diaphoresis, has been studied and found to be associated with poorer outcomes, possibly from secondary brain injury from the hypermetabolic state or elevated sympathetic tone. These symptoms reflect increased sympathetic nervous system activity resulting from hypothalamic-pituitary dysfunction and are more likely to occur after hypoxic/ischemic events. Compared with injured children who do not have dysautonomia, children with dysautonomia required longer rehabilitation and had less improvement in Wee-FIM motor skills scores (Kirk et al., 2012). Therefore, effective management of dysautonomia is especially critical. In addition to strategies such as environmental controls to reduce noxious stimuli, pharmacologic management includes the use of benzodiazepines, opioid antagonists, beta-blockers (e.g., propranolol), central dopamine agonists (e.g., bromocriptine), dopa-mine antagonists (e.g., chlorpromazine), and alpha-agonists (e.g., clonidine; Kirk et al., 2012; Perkes et al., 2010). Eventual resolution of the symptoms typically occurs, and medications are tapered as tolerated.

After TBI, children are at increased risk for posttraumatic seizures. Early posttraumatic seizures may occur in the first 7 days, whereas late seizures occur after 1 week. Prophylaxis antiepileptic drug (AED) therapy is often prescribed for a minimum of 7 days. In the literature, there appears to be a debate as to which AED is most effective (Bansal, Blalock, Kebede, Dean, & Carpenter, 2014; Chung & O'Brien, 2014; Liesemer, Bratton, Zebrack,

Brockmeyer, & Statler, 2011). Levetiracetam and phenytoin are frequently used for early AED prophylaxis. Levetiracetam is well tolerated and eliminates the need for obtaining serum drug levels, although phenytoin is less costly. More studies are needed to determine the most effective AED for prevention of posttraumatic seizures. Regardless of prophylaxis, monitoring for seizures to prevent secondary injury from further trauma, hypoxemia, or increased metabolic demand is imperative.

Dysphagia related to cerebellar dysfunction may result in impaired oral motor skills of injured children. The four stages of swallowing (oral preparatory, oral, pharyngeal, and esophageal) must work in concert to transition a bolus of food from the anterior to the posterior oropharynx and prevent airway aspiration. Aspiration may be silent or chronic, resulting in pulmonary issues such as pneumonia or wheezing with respiratory compromise. Under the guidance of a speech language pathologist, radiographic imaging via a rehabilitation swallow study should assess oral motor transit of a food bolus, as well as thick to thin fluids. The results of the assessment determine strategies to facilitate advancement in oral feedings. Compensatory strategies are often utilized, such as texture modification of thick liquids or solids with honey or nectar, in addition to maneuvers such as head position or tucking the chin. Research has shown that these functional modifications will improve swallowing biomechanics for the short term (Miller, 2011). In addition to traditional compensatory techniques, recent approaches to treatment of swallow dysfunction have included use of electrical stimulation for modulation of neuronal systems affecting swallowing function. Trained speech language pathologists apply electrodes to the skin at the oropharyngeal neuromuscular junctions to deliver pulses of electrical stimulation to the muscles needed for swallowing in conjunction with ingestion of food or liquids (Doeltgen & Huckabee, 2012; Miller, 2011). Oral motor integrity plays an integral part in the ability to manage oral secretions as well. Until adequate oral nutrition is provided, alternative tube feeding options, such as nasogastric, nasojejunal, or gastrostomy feeding, are maintained to meet nutritional and caloric requirements and provide daily maintenance of fluid volumes.

Deep vein thrombosis, which is less common in the uninjured pediatric population (O'Brien, 2012), is a frequent complication of TBI. Risk increases for multi-trauma patients, who are more likely to have hemodynamic instability resulting in vascular stasis, coagulopathic states, infections, and the use of invasive hemodynamic monitoring catheters such as central venous access. Comorbidities of extremity fractures and prolonged immobilization also contribute to such events. Early detection via clinical examination and venous Doppler ultrasound documentation of perfusion deficits should be followed by initiation of therapeutic anticoagulants as soon as the patient is considered not to be at risk for intracranial bleeding. Ongoing clinical observation is imperative to detect any signs of pulmonary embolism, including dyspnea, anxiety, cough, tachypnea, or tachycardia. Anticoagulant therapy may begin with subcutaneous low-molecular-weight heparin (LMWH) and transition to oral warfarin with bridging doses of LMWH until an international normalized ratio of 2-3 is achieved (O'Brien, 2012). Although LMWH eliminates the need for frequent prothrombin/international normalized ratio monitoring, intermittent laboratory draws are sometimes better tolerated than twice-daily subcutaneous injections for 3 to 6 months. Daily clinical monitoring of the affected extremity is recommended with documentation of initial circumferential measurements, as well as the

presence and location of any pain, edema, or erythematous areas. Repeat imaging is performed to verify resolution and adequate perfusion of the affected limb prior to discontinuation of medication. Placement of a removable inferior vena cava filter may be appropriate as well.

Dyspraxia is a sensory processing disorder of frontoparietal brain injury that results in impaired performance of motor skill tasks. Postural control through the activation of core muscle groups is needed to perform functional motor movements from reintegration of vestibular, visual, and proprioceptive feedback through physical and occupational therapy. Through upright positioning and other neurodevelopmental techniques, therapists deliver specific sensory input for neuromuscular re-education to develop muscle tone, coordination, balance, and motor planning.

Spasticity and elevated muscle tone commonly evolve after brain injury because of dysfunction of sensorimotor control in the upper motor neurons (Kheder & Nair, 2012). Although a child requires a degree of tone for postural control during sitting and transfers, increased flexor tone and spasticity can become disabling and interfere with function. Spastic dystonia leads to discomfort, contractures, or other deformities, such as talipes equinovarus or scoliosis. In rehabilitation, it is prudent for the medical, nursing, and therapy team members to monitor the spasticity for a short interval, such as 2 weeks, for natural improvement, while maximizing physical therapy with therapeutic stretching exercises and medications. Evaluation of pain and spasticity is assessed daily by the rehabilitation team in relation to interference with functional gains, as well as caregiver tasks such as personal hygiene. This information is used to guide medication management by the medical staff. Spasms may interfere with bowel and bladder function and result in alterations in skin integrity from vulnerable tissue pressure or sheering injury. The Modified Ashworth Scale grades degree of spasticity in muscle tone on an ordinal scale (Bohannon & Smith, 1987).

Medications to manage tone are started at “low and slow” doses. Baclofen, a gamma aminobutyric acid B receptor agonist, is most widely used initially and is titrated to tolerance and effect. Associated fatigue may require a larger dose to be given initially at night. Over time the child should accommodate to the medication with fewer signs of fatigue symptoms. Cloni-dine, a central alpha agonist, is also frequently used and may be transitioned from every-8-hour dosing to transdermal delivery with a 24-hour enteral bridge until the patch is effective. This method permits a more even distribution and medication effect without variability in blood pressure levels. Gabapentin, a modulator of excitatory neurotransmitter release, may be useful as well with additional benefit of pain relief. Tizanidine, an alpha-2 receptor agonist, acts as a central muscle relaxant as well; however, tizanidine requires monitoring of liver enzymes for risk of acute hepatitis, which may limit its use in children. Benzodiazepines may be used, but sedative effects may interfere in daytime use, yielding undesired fatigue and a decreased level of alertness. The goal of single or combined medications is to optimize spasm relief and minimize adverse effects, which impede neurocognitive recovery.

If contractures evolve or spasticity is unrelieved after maximizing pharmacologic management and therapeutic exercises, a skilled medical provider may evaluate the child for

focal treatment with botulinum toxin serotype A, which targets a specific skeletal muscle group by binding with acetylcholine receptors. The injections may be performed with use of conscious sedation for comfort if needed and provide temporary focal relief without generalized effects. The effects are evident within 2 weeks and may last up to 3 months. Serial casting or splinting may be implemented in addition to ongoing therapy to maximize the beneficial effect of the botulinum toxin injections (Intiso, 2012; Kheder & Nair, 2012; Lubsch, Habersang, Haase, & Luedtke, 2006; Van Rhijn, Molenaers, & Ceulemans, 2005). If focal treatment is ineffective and more general relief is indicated, insertion of a baclofen pump may be instituted for continuous intrathecal medication infusion. However, the inserted pump may interfere with the ability to ambulate and has inherent risks associated with placement (Gooch, Oberg, Grams, Ward, & Walker, 2004). Surgical interventions such as selective rhizotomy, tenotomy, or myotomy are reserved for failure of pharmacologic and physiotherapy management to achieve patient goals.

CONCLUSION

Despite concerted efforts to prevent pediatric TBI through public education on helmet use and playground, seat belt, and car seat safety, TBI remains a significant cause of morbidity and mortality. Tighter gun control laws and effec tive methods to prevent child abuse may also reduce the incidence of TBI. Health care providers can take an active role in expanding knowledge about evidence-based interventions to prevent secondary injury associated with pediatric TBI and to maximize neurocognitive recovery and functional independence. Health care providers must positively influence outcomes and help children and their families meet the challenges associated with surviving TBI. Health care providers are uniquely positioned to promote prevention, early intervention, appropriate use of severity and functionality scoring, and effective acute and rehabilitation therapies.

Acknowledgments

Karin Reuter-Rice received support for this work by NIH-NINR 1P30 NR014139-01 Adaptive Leadership for Cognitive Affective Symptom Science (ADAPT), Institute of Nursing Research Center of Excellence (2012-2015) in addition to funding by the Robert Wood Johnson Foundation as a Nurse Faculty Scholar Project #71244 (2013-2016).

REFERENCES

Aitken M, McCarthy M, Slomine B, Ding R, Durbin D, Jaffe K, Mackenzie EJ. Family burden after traumatic brain injury in children. Pediatrics. 2009; 123(1):199–206. [PubMed: 19117883]

Bansal S, Blalock D, Kebede T, Dean N, Carpenter J. Levetiracetam versus (fos)phenytoin for seizure prophylaxis in pediatric patient with intracranial hemorrhage. Journal of Neurosurgery: Pediatrics. 2014; 13(2):209–215. [PubMed: 24286154]

Beauchamp K, Kashuk J, Moore E, Bolles G, Rabb C, Seinfield J, Sauaia A. Cranioplasty after postinjury decompressive craniectomy: Is timing of the essence? The Journal of Trauma, Injury, Infection, and Critical Care. 2010; 69(2):270–274.

Bohannon R, Smith M. Interrater reliability of a modified Ashworth scale of muscle spasticity. Physical Therapy. 1987; 67(2):206. Retrieved from http://www.rehabmeasures.org/PDF%20Library/Modified%20Ashworth%20Scale%20Instructions.pdf. [PubMed: 3809245]

Catroppa C, Anderson V. Planning, problem-solving and organizational abilities in children following traumatic brain injury: Intervention strategies. Pediatric Rehabilitation. 2006; 9(2):89–97. [PubMed: 16449067]

Centers for Disease Control and Prevention. TBI data and statistics. 2014. Retrieved from http://www.cdc.gov/traumaticbraininjury/data/index.html

Chung M, O'Brien N. Incidence of early seizures with levetiracetam prophylaxis in children after traumatic brain injury. Neurology. 2014; 82(10):S60.002.

Cicero M, Cross K. Predictive value of initial Glasgow Coma Scale Score in pediatric trauma patients. Pediatric Emergency Care. 2013; 29(1):43–48. [PubMed: 23283262]

Coronado V, Xu L, Basavaraju S, McGuire L, Wald M, Faul M, Hemphill JD. Surveillance for traumatic brain injury—related deaths—United States, 1997-2007. Morbidity and Mortality Weekly Report: Surveillance Summaries. 2011; 60(5):1–32. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/ss6005a1.htm. [PubMed: 21544045]

Corso P, Finkelstein E, Miller T, Fiebelkorn I, Zaloshnja E. Incidence and lifetime costs of injuries in the United States. Injury Prevention. 2006; 12(4):212–218. [PubMed: 16887941]

Doeltgen S, Huckabee M. Swallowing neurorehabilitation: From the research laboratory to routine clinical application. Archives of Physical Medicine and Rehabilitation. 2012; 93:207–213. [PubMed: 22289228]

Gooch J, Oberg W, Grams G, Ward L, Walker M. Care provider assessment of intrathecal baclofen in children. Developmental Medicine & Child Neurology. 2004; 46:548–552. [PubMed: 15287246]

Greve MW, Zink BJ. Pathophysiology of traumatic brain injury. Mount Sinai Journal of Medicine. 2009; 76:97–104. [PubMed: 19306379]

Hagen, D.; Malkmus, D.; Durhan, P. Level of cognitive functioning. Rancho Los Amigos Hospital; Downey, CA: 1972.

Intiso D. Therapeutic use of botulinum toxin in neurorehabilitation. Journal of Toxicology. 2012; 2012:802893. http://dx.doi.org/10.1155/2012/802893. [PubMed: 21941544]

Kheder A, Nair K. Spasticity: Pathophysiology, evaluation and management. Practical Neurology. 2012; 12:289–298. [PubMed: 22976059]

Kirk K, Shoykhet M, Jeong J, Tyler-Kabara E, Henderson M, Bell M, Fink EL. Dysautonomia after pediatric brain injury. Developmental Medicine & Child Neurology. 2012; 54(8):759–764. [PubMed: 22712762]

Kochanek PM, Carney N, Adelson PD, Ashwal S, Bell MJ, Bratton S, Warden CR. Guidelines for the acute medical management of severe traumatic brain injury in infants, children, and adolescents—second edition. Pediatric Critical Care Medicine. 2012; 13(Suppl. 1):S1–S82. [PubMed: 22217782]

Liesemer K, Bratton S, Zebrack M, Brockmeyer D, Statler K. Early post-traumatic seizures in moderate to severe pediatric traumatic brain injury: Rates, risk factors, and clinical features. Journal of Neurotrauma. 2011; 28:755–762. [PubMed: 21381863]

Lubsch L, Habersang R, Haase M, Luedtke S. Oral baclofen and clonidine for treatment of spasticity in children. Journal of Child Neurology. 2006; 21(12):1090–1092. [PubMed: 17156708]

Miller C. Aspiration and swallowing dysfunction in pediatric patients. Infant, Child, & Adolescent Nutrition. 2011; 3(6):336–343.

Nesiama J, Pirallo R, Lerner E, Hennes H. Does a pre-hospital Glasgow Coma Scale Score predict pediatric outcomes? Pediatric Emergency Care. 2012; 28(10):1027–1032. [PubMed: 23023472]

O'Brien, N. Traumatic brain injury.. In: Reuter-Rice, K.; Bolick, B., editors. Pediatric acute care: A guide for interprofessional practice. Jones & Bartlett Learning; Burlington, MA: 2012. p. 1197-1204.

Perkes I, Baguley I, Nott M, Menon D. A review of paroxysmal sympathetic hyperactivity after acquired brain injury. Annals of Neurology. 2010; 68(2):126–135. [PubMed: 20695005]

Rancho Los Amigos Scale. Retrieved from http://www.abiebr.com/set/case-study-2/23-ranchos-los-amigos-scale-cognitive-functioning

Suarez E, Gonzalez A, Diaz C, Salido A, Garde A, Glores J. Decompressive craniectomy in 14 children with severe head injury: Clinical results with long-term follow-up and review of the literature. The Journal of Trauma, Injury, Infection, and Critical Care. 2011; 71(1):133–140.

Suskauer S, Slomine B, Inscore A, Lewelt A, Kirk J, Salorio C. Injury severity variables as predictors of WeeFIM scores in pediatric TBI: Time to follow commands is best. Journal of Pediatric Rehabilitative Medicine. 2009; 2(4):297–307.

Uniform Data System for Medical Rehabilitation. The Wee-FIMÒ clinical guide, version 6.0. Author; Amherst, NY: 2006.

Van Rhijn J, Molenaers G, Ceulemans B. Botulinum toxin type A in the treatment of children and adolescents with an acquired brain injury. Brain Injury. 2005; 19(5):331–335. [PubMed: 16094780]

Zaloshnja E, Miller T, Langlois J, Selassie A. Prevalence of long-term disability from traumatic brain injury in the civilian population of the United States, 2005. Journal of Head Trauma and Rehabilitation. 2008; 23(6):394–400.

Zebrack M, Dandoy C, Hansen K, Scaife E, Mann N, Brat-ton S. Early resuscitation of children with moderate-to-severe traumatic brain injury. Pediatrics. 2009; 124(1):56–64. [PubMed: 19564283]

Dysautonomia is estimated to affect about one third of patients after moderate to severe TBI in the first few weeks after brain injury.

Health care providers can take an active role in expanding knowledge about evidence-based interventions to prevent secondary injury associated with pediatric TBI and to maximize neurocognitive recovery and functional independence.

TABLE

Resources

Organization/scale	Web site
American Association of Neurologic Surgeons	www.aans.org
Brain Injury Association of America	http://www.biausa.org/brain-injury-children.htm
Brain Trauma Foundation	www.braintrauma.org/coma-guidelines
Centers for Disease Control and Prevention	http://www.cdc.gov/TraumaticBrainInjury/data/index.html
National Institute of Neurological Disorders and Stroke	www.ninds.nih.gov
Rancho Los Amigos Scale	http://www.abiebr.com/set/case-study-2/23-ranchos-los-amigos-scale-cognitive-functioning
The National Center on Shaken Baby Syndrome	www.dontshake.org