Pathophysiology and Treatment of Severe Traumatic Brain Injuries in Children

CE 3.0 ANCC Contact Hours

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ABSTRACT

Traumatic brain injuries (TBIs) in children are a major cause of morbidity and mortality worldwide. Severe TBIs account for 15,000 admissions annually and a mortality rate of 24% in children in the United States. The purpose of this article is to explore pathophysiologic events, examine monitoring techniques, and explain current treatment modalities and nursing care related to caring for children with severe TBI. The primary injury of a TBI is because of direct trauma from an external force, a penetrating object, blast waves, or a jolt to the head. Secondary injury occurs because of alterations in cerebral blood flow, and the development of cerebral edema leads to necrotic and apoptotic cellular death after TBI. Monitoring focuses on intracranial pressure, cerebral oxygenation, cerebral edema, and cerebrovascular injuries. If abnormalities are identified, treatments are available to manage the negative effects caused to the cerebral tissue. The mainstay treatments are hyperosmolar therapy; temperature control; cerebrospinal fluid drainage; barbiturate therapy; decompressive craniectomy; analgesia, sedation, and neuromuscular blockade; and antiseizure prophylaxis.

Keywords: antiseizure prophylaxis, barbiturate therapy, child, hyperosmolar therapy, intracranial pressure, nursing care, pathophysiology, primary injury, secondary injury, trauma, traumatic brain injury, treatment

Traumatic brain injuries (TBIs) in children are a major cause of morbidity and mortality worldwide (Bramlett & Dietrich, 2004; Centers for Disease Control and Prevention, 2010; Feigin et al., 2013; Shao et al., 2012). TBIs are an insult to the brain because of direct trauma from an external force, a penetrating object, blast waves, or a jolt to the head (Faul, Xu, Wald, & Coronado, 2010; Klimo, Ragel, Scott, & McCafferty, 2010). In the United States, motor vehicle collisions and falls are the most frequent precipitating events resulting in over 500,000 pediatric TBIs that lead to hospitalization and deaths each year (Faul et al., 2010). Severe TBIs account for 15,000 admissions annually and a mortality rate of 24% in children in the United States (Piatt & Neff, 2012). Severe pediatric TBIs are defined as children less than 18 years old with a Glasgow Coma Scale (GCS) score of 3–8 (Adelson et al., 2003). An appreciation of the pathophysiologic effects of this injury allows nurses to understand the role of key therapeutic management principles. The purpose of this article is to explore pathophysiologic events, examine monitoring techniques, and explain current treatment modalities and management related to caring for children with severe TBI.

Pathophysiology

By understanding the pathophysiology associated with a TBI, pediatric nurses may be better able to manage and prevent injury progression. Injury progression may be subdivided into primary and secondary injuries, which are associated with major pathways of dysfunction in TBI.

Primary Injury

Primary injury comes about because of linear and rotational forces to brain tissue at the time of impact. Linear forces from acceleration trauma that transverse the skull result in coup and contracoup contusions (Hirsch & Kaufman, 1975). Coup contusions occur when the brain impacts the side of the skull, whereas contracoup contusions occur when the brain hits the side of the skull and then bounces back to the other side of the skull (Stewart, 1944). The two most common types of hemorrhages after TBI in children are epidural and subdural (Case, 2008; Jamous et al., 2009). Epidural hemorrhages are often associated with skull fractures because the skull fragments cause laceration of an artery leading to accumulation of blood between the skull and the dura (Black, 1956). Subdural hemorrhages are a result from the rupture of one or more of the bridging veins within the space between the dura mater and arachnoid membrane. The clinical presentation evolves over days to weeks both

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The author declares no conflict of interest.

DOI: 10.1097/JNN.0000000000000176

in the physical skills and electroencephalographic (EEG) changes (Cohn, 1948).

Rotational forces are a result of the brain moving at a different angular velocity than the skull because the head is not held in a fixed position during the injury (Holbourn & Edin, 1943). Rotational forces can lead to shearing and twisting injury in critical areas of the brain and are often manifested clinically as concussions and diffuse axonal injury (Blumbergs et al., 1994; Mihalik et al., 2010; Moen et al., 2012). Concussions are a traumatic injury to the head that can result in a loss of consciousness and traumatic amnesia (Ward, 1964). However, diagnostic imaging does not show any damage to the brain despite symptom continuation for several weeks postinjury (Eisenberg, Meehan, & Mannix, 2014). Diffuse axonal injury is characterized by widespread damage to axons from the force and shearing sustained during the primary injury (Adams, Graham, Scott, Parker, & Doyle, 1980). These injuries and outcomes vary from child to child based on several factors because the primary injury plays a major part in the extent of the injury (Kondo et al., 2010; Moen et al., 2012).

Anatomic considerations confound the primary injury process for young children with TBI when compared with adults. Compared with adults, young children have proportionately large, heavy heads; weaker cervical ligaments and muscles; and thin, pliable skulls; all of which can result in increased severity of injury (Calder, Hill, & Scholtz, 1984). When young children sustain a blunt injury to the head, their large, heavy heads and weaker cervical ligaments and muscles cause the mechanical force to be transferred to the cervical spine (C-spine) region, which can result in cervical spinal fractures from the occiput to C2 and cervical cord damage (Nitecki & Moir, 1994). In children aged <8 years, the fulcrum of movement is located at C2–C3 compared with C5–C6 in adults, which may explain why children experience cervical fractures in different locations than adults (Finch & Barnes, 1998). The thin, pliable skull provides less protection to the underlying brain, leaving it more vulnerable to shearing from linear and rotational forces (Leventhal, 1960). The long-term clinical impact of the initial structural changes in the brain from the primary injury is difficult to discern because the early effects of the primary injury are quickly compounded by the secondary injury, which activates complex biomolecular and physiologic reactions.

Secondary Injury

Secondary injury occurs because of alterations in cerebral blood flow (CBF) and the development of cerebral edema, which may lead to neuronal cellular death. If the CBF and cerebral metabolism continues to be compromised (Kilbaugh et al., 2011; Mandera, Larysz, & Wojtacha, 2002; Stiefel, Tomita, & Marmarou, 2005) additional cellular damage and death will occur and more toxic substances will be released (Pasvogel, Miketova, & Moore, 2010; Pun, Lu, & Moochhala, 2009). The longer the secondary injury phase continues, the more likely the child will sustain long-term disabilities (Casey, McKenna, Fiskum, Saraswati, & Robertson, 2008).

The authors provide an excellent review of the pathophysiology supporting ICP treatment in children.

Cerebral Blood Flow

CBF is the mechanism that supplies the cerebral tissue with oxygenated blood. To maintain a consistent cerebral flood flow, cerebral arteries and arterioles dilate and contract in response to hemodynamic changes called cerebral autoregulation (Hekmatpanah, 1970). During the first 0–9 days, cerebral autoregulation is often impaired after a TBI; this results in CBF becoming dependent on cerebral perfusion pressures (CPPs) and intracranial pressure (ICP; Tontisirin et al., 2007). An increased risk for impaired autoregulation has been associated with increased ICP, hyperemia, decreased hematocrit, decreased arterial oxygen (PaO_2), increased cerebral lesion size, and age of less than 4 years (Freeman, Udomphorn, Armstead, Fisk, & Vavilala, 2008; Tontisirin et al., 2007). Clinically, children with impaired cerebral autoregulation have increased odds of poor outcomes (e.g., functional impairment, vegetative state, or death; Tontisirin et al., 2007).

Cerebral Metabolism

Cerebral metabolism is affected after a severe TBI because the tissue is deprived of glucose and oxygen forcing cells into anaerobic metabolism (Holbach, Schröder, & Köster, 1972). Anaerobic metabolism produces less energy and increases lactate production, ultimately making it difficult for cells to maintain normal functional processes. Failure to maintain cellular functioning because of lack of energy creates an acidotic environment, which leads to serious consequences (Bouzat et al., 2014). When the sodium/potassium (Na/K) pumps fail, an excessive influx of the positively charged sodium ions precipitate massive depolarization of neurons. This depolarization leads to the release of glutamate, an

excitatory neurotransmitter that leads to excitotoxicity, as well causing more damage (Chamoun, Suki, Gopinath, Goodman, & Robertson, 2010).

The excess extracellular glutamate activates cell receptors that allow a massive influx of calcium and sodium into the cells, leading to clinically significant consequences. The increased intracellular calcium leads to (a) activation of the nitric oxide synthetase (NO) leading to cerebral edema and ischemia; (b) activation of casplases (destructive enzymes) that damage the DNA of the cell, leading to apoptosis; (c) activation of the proteases family of calpains, which cause the breakdown of the neuronal cytoskeleton leading to the loss of structural integrity of the neuron; (d) damage to the Na/K pump, leading to an influx of additional sodium followed by water with resultant cerebral edema; and (e) mitochondrial injury leading to reactive oxygen species generation, microvascular damage, and cerebral edema (Huh, Franklin, Widing, & Raghupathi, 2006; Robertson, Bucci, & Fiskum, 2004; Robertson, Saraswati, & Fiskum, 2007). The influx of sodium can lead to increased permeability of the calcium ion channels resulting in cell swelling and/or neuronal death (Staal et al., 2010). Glutamate, calcium, and sodium can cause significant damage to the structures and functions throughout the nervous system.

If cerebral blood flow has been disrupted for a period, reperfusion injury may occur when adequate blood flow is restored. The areas of ischemia will likely show the sequelae associated with poor cerebral blood flow (Robertson, Scafidi, McKenna, & Fiskum, 2009). Upon restoration of perfusion, leukocytes attempt to repair the ischemic tissue, which leads to further generation of free radicals. The free radicals likely play a role in cellular death during reperfusion injury (Reddy & Labhasetwar, 2009).

Blood–Brain Barrier and Edema

The blood–brain barrier (BBB) is responsible for restricting movement of certain molecules, including medications and white blood cells, into the central nervous system. This barrier consists of astrocyte endfeet, brain microvascular endothelial cells, and capillary basement membrane. Shearing damage sustained in the primary injury can cause structural damage to the BBB increasing the membrane permeability. During the secondary injury phase, research suggests that excitotoxicity from the excess glutamate leads to an influx of calcium and sodium into the cells (Pun et al., 2009).

Brain edema is the increase of fluid in the brain, which is likely a result of changes in the cellular, molecular, structural, and functional properties of the BBB that usually prevent excess fluid from entering the brain (Unterberg, Stover, Kress, & Kiening, 2004). There are two types of cerebral edema after TBI: vasogenic (interstitial) and cytotoxic (intracellular; Greve & Zink, 2009). Vasogenic cerebral edema is a result of the increased permeability of the BBB because of necrosis of capillary endothelial cells (Unterberg et al., 2004). The damage to capillary endothelial cells allows the leakage of proteins into the interstitial fluid creating an osmotic gradient. This osmotic gradient allows excess water and proteins to accumulate in the interstitial space of the brain (Donkin & Vink, 2010).

Cytotoxic edema occurs because of energy failure and the inability to maintain the Na/K pump. The influx of sodium ions into the intracellular space is followed by an influx of water from the extracellular space in an attempt to create homeostasis (Donkin & Vink, 2010). The interstitial space within the brain is reduced in cytotoxic edema because the cells swell (Unterberg et al., 2004).

Importantly, both types of edema may lead to increased ICP because of the brain's inability to compensate for the increased volume of the brain tissues or increased volume in the interstitial space of the brain. On the basis of the Monro–Kellie hypothesis, the brain will initially attempt to compensate for the edema by displacing cerebrospinal fluid (CSF) into the spinal canal and venous blood into the jugular veins; however, if these mechanisms fail to decrease the volume within the cranial cavity, the ICP will increase (Weed, 1929). The cerebral edema can be monitored clinically through ICPs, visualized on radiographic images, and observed through changes in the child's behavior.

Necrosis and Apoptosis

Primary and secondary injuries can lead to two types of cellular death: necrosis and apoptosis. Necrosis is cellular death that occurs in conditions of very severe hypoxia and ischemia because of lack of oxygen (Johnston et al., 2009). Pathologically, the cell membrane ruptures, the mitochondria are severely damaged, and all the toxic contents (e.g., phospholipases, proteases, and lipid peroxidase) are leaked into the extracellular space leading to inflammation (Miñambres et al., 2008). The leakage from the cells can activate the inflammatory cascade and lead to additional consequences. This is why apoptosis would be the preferred method of cellular death.

If the insult is less severe, the cells may recover or may progress to programmed cell death, known as apoptosis (Johnston et al., 2009). Apoptosis is a caspase-dependent pathway that ultimately results in cell shrinkage and general preservation of the cellular

membranes without associated inflammation that may begin on the first day of injury (Miñambres et al., 2008; Pasvogel et al., 2010). Both necrosis and apoptosis can lead to clinical changes such as decreased cognitive and physical disabilities because of losses in neurons through cellular death (Brown, Elovic, Kothari, Flanagan, & Kwasnica, 2008). In an attempt to mitigate the damage sustained after severe TBI, advanced monitoring techniques are utilized to monitor the progression of the secondary injury or to evaluate the responsiveness to treatments.

Assessing and Monitoring Children With Severe TBI

Initial Assessment and Stabilization

In the emergency department, the specially trained trauma team is responsible for the initial assessment and stabilization of a child with a severe TBI. The primary assessment begins with addressing all potential life-threatening injuries (e.g., tension pneumothorax) including the head injury. The team must determine whether the child has a patent airway, can maintain an adequate respiratory drive (breathing), and has adequate circulation and initial neurologic status (disability). This primary assessment is often referred to as the ABCDs (Emergency Nurses Association, 2007).

The assessment of the airway is used to determine if air can pass through without obstruction to the lungs (McFadyen, Ramaiah, & Bhananker, 2012). The airway must be cleared of any debris by suctioning or other methods, if obstructed. The team may choose to elicit a gag reflex with the suction catheter while suctioning the child to determine if the child is able to manage his or her oral secretions and protect his or her airway from potential aspiration of stomach contents (Marcoux, 2005). If the child has a depressed level of consciousness, the child may be unable to protect his or her airway, and endotracheal intubation may be necessary, while maintaining inline stabilization of the C-spine (McFadyen et al., 2012).

The assessment of breathing is used to determine which interventions are necessary to help the child maintain adequate oxygenation and ventilation (McFadyen et al., 2012). Children who present with abnormal breathing patterns (e.g., central neurogenic hyperventilation, cluster breathing, ataxic breathing, and apnea) may have sustained a brainstem lesion/injury or a C-spine injury with resultant phrenic nerve failure requiring initial respiratory support (e.g., endotracheal intubation and placement on a ventilator; Easter, Barkin, Rosen, & Ban, 2011; Marcoux, 2005). C-spine precautions (e.g., rigid cervical collar, log roll turning method) should be maintained because head injuries are associated with spinal cord injuries (Easter et al., 2011). Maintaining systemic oxygenation and ventilation parameters of PaO_2 301–500 mm Hg (40–67 kPa) and $PaCO_2$ of 36–45 mm Hg (5–6 kPa) are associated with higher rates of survival for children (Ramaiah et al., 2013).

Initial assessment and stabilization of circulation begins with checking for a pulse and beginning cardiopulmonary resuscitation, if necessary (Chameides & Ralston, 2011). Stopping any external hemorrhage is also critical to minimize blood loss. Maintaining adequate circulation in children with head injuries is important because CPP depends on the ICP and mean arterial pressure (MAP; Hekmatpanah, 1970). The MAP is a reflection of the average arterial system pressure during a cardiac cycle and can be calculated based on this equation: MAP = (1/3 systolic BP) + (2/3 diastolic BP). To ensure adequate perfusion, in the guidelines from Pediatric Advanced Life Support, the recommendation is to maintain a pediatric (1–10 years old) systolic blood pressure > 70 mm Hg + (2 × age in years) to avoid a hypotensive state (Kleinman et al., 2010; see Table 1 for recommended MAPS based on age; Hazinski, 2013; Top et al., 2011).

The initial neurological status (disability) is assessed through the pediatric GCS, pupil response, and overall responsiveness (Emergency Nurses Association, 2007). The pediatric GCS is a modified version of the adult GCS that accounts for the changing developmental status of children (scored 3–15). If the child has an initial pediatric GCS < 8 (classified as a severe head injury) or is unarousable, the trauma/emergency team considers emergent endotracheal intubation and placement on a ventilator (Falk, 2012; Marcoux, 2005). The initial assessment and stabilization should take less than 10 minutes.

Neurological Assessment

Assessing Cerebral Edema and Posttraumatic Cerebrovascular Injuries

Cranial imaging through computed tomography (CT) and magnetic resonance imaging (MRI) are used to

TABLE 1. Recommended Average Mean Arterial Pressures (MAPs) by Age

Age in Years	MAP (mm Hg)
1–2	50–70
3–4	60–75
5–6	65–75
7–8	70–75
9–10	70–75
11–12	70–80
13–14	80–90

Hazinski, 2013; Top, Tasker, & Ince, 2011.

identify cerebral edema and cerebrovascular injuries. This information is used to determine what neurosurgical interventions are necessary and to provide prognostic information about long-term developmental outcomes (Beauchamp et al., 2011; Suskauer & Huisman, 2009). Cranial CT scans are useful in identifying large hematomas, midline shift, skull fractures, excess CSF accumulation in the ventricles, cerebral edema, and brain herniation (Steinborn et al., 2010; Suskauer & Huisman, 2009). The results of the initial CT scans are used to determine if emergent neurosurgical interventions (e.g., decompression craniotomy) are needed (Suskauer & Huisman, 2009). The major advantage of using CT scan is that the scan takes less than 10 minutes to perform and the child is easily accessible to the nurse throughout the scan. The major disadvantage of using CT is the potential for exposing the child to ionizing radiation (Duhaime, Holshouser, Hunter, & Tong, 2012).

Empirical evidence suggests that diagnostic radiation both in utero and in childhood is associated with an increased risk of childhood cancer (International Agency for Research on Cancer, 2012). The overall cancer incidence is 24% greater for those exposed to CT scans than for those who are not exposed in childhood (Mathews et al., 2013). Children needing a CT scan of the brain are at a greater risk of exposure because the skull is less dense than adult skulls and offer less protection to the underlying cerebral tissue (Morton et al., 2013). The American College of Radiology and the Image Gently campaign have made specific suggestions for radiologists, radiologic technologists, and medical physicist to lower CT doses of radiation and use alternative testing when possible (Strauss et al., 2010). Nurses can assist the clinicians in meeting these new goals by ensuring that only areas that need to be scanned are scanned.

MRI scans are helpful in identifying extra-axial hemorrhage (e.g., subdural hemorrhage) and early areas of ischemia and detecting edema in subacute moderate-to-severe injury (Bigler et al., 2013; Hunter, Wilde, Tong, & Holshouser, 2012). The major disadvantage of using MRI scan is that it takes longer than a CT scan and the equipment taken into the room to monitor the child must be MRI safe. The nurse needs to review MRI safety with parents who may accompany the child into the scan, and the child or parent does not have any MR-contraindicated implantable devices (e.g., deep brain stimulators; Duhaime et al., 2012).

Assessing ICPs

The pressure in the cranial vault is made up of the CSF, blood, and brain. According to the Monro–Kellie hypothesis, ICP is relatively constant because the cranial cavity represents a fixed volume (Weed, 1929). If an increase in the volume of CSF, blood, or brain compartments occurs, the brain will compensate initially by displacing one of the two other compartments. However, when displacement does not decrease the pressure within the cranial cavity, the ICP increases. In the case of TBI, one or more of these components can become increased, leading to increased ICP (Weed, 1929).

ICP can be assessed through clinical observations (e.g., headache, irritability), noninvasive examinations (e.g., transcranial Doppler [TCD]), and direct invasive measures (e.g., intraparenchymal [IP] monitors). Careful and frequent assessment of children with severe TBI may allow the clinician to observe and manage the signs and symptoms of early increased ICP before the child progresses to the late signs. Early signs of increased ICP may be subtle such as irritability and cranial nerve dysfunction. The most ominous late sign of increased ICP is Cushing's triad. Cushing's triad is the result of cerebral ischemia. Clinically, it manifests as increased systolic blood pressure (also widened pulse pressure) to increase cerebral perfusion, bradycardia because of a vagal response triggered by the cardiac baroreceptors, and abnormal or irregular respirations (Cushing, 1903; see Table 2 for signs and symptoms of increasing ICP).

TCD is a noninvasive examination of the blood flow velocity of the middle cerebral arteries through the transtemporal window (temple anterior to the ear) to determine cerebral hemodynamics and indirectly assess intracranial hypertension (Trabold, Meyer, Blanot, Carli, & Orliaguet, 2004; Verlhac, 2011). The use of TCD to evaluate increased ICP is limited in children (Melo et al., 2011; Meyer et al., 2005) and has been used to evaluate cerebral vasospasms (Trabold et al., 2004) and cerebral autoregulation (Vavilala et al., 2004). This noninvasive method may be useful as screening tool in early resuscitation because it can be used in children with coagulation disorders and those with critical injuries before CT scan (Melo et al., 2011). Nursing responsibilities for the procedure include positioning the child supine throughout the procedure.

The two most common types of direct invasive intracranial monitoring devices utilized for monitoring in pediatric TBI are the external ventricular device (EVD) and IP monitors (Wiegand & Richards, 2007). The EVD is the gold standard for measuring ICP. An EVD is a fluid-coupled device with the catheter placed directly in the lateral ventricles that allows access to CSF, making it ideal for drainage of CSF for increased ICP and sampling of CSF (Padayachy, Figaji, & Bullock, 2010). Nursing considerations when caring for a child with an EVD involve assessing for complications;

TABLE 2. Signs and Symptoms of Increasing Intracranial Pressure

Early Signs and Symptoms
Headache
Vomiting
Change in alertness
Irritability
Decreased eye contact
Cranial nerve dysfunction
Seizures
Decrease in Glasgow Coma Scale score
Late Signs and Symptoms
Further deterioration of consciousness
Bulging fontanel
Decreased spontaneous movement
Posturing
Papilledema
Pupil dilation with decreased or no response to light
Increased blood pressure
Irregular respirations
Cushing's triad

Marcoux, 2005.

monitoring for infection including systemic temperature (Geyer, Meller, Kulpan, & Mowery, 2013); assessing the dressing for drainage (Ngo et al., 2009); assessing CSF drainage for color, clarity, and amount (Geyer et al., 2013); and monitoring for signs and symptoms of overdrainage of CSF (e.g., dehydration, hyponatremia) and underdrainage of CSF (Ngo et al., 2009). Nurses must also ensure that, each time the child is repositioned, the EVD is leveled with the foramen of Monro (external auditory meatus) at the prescribed level (Geyer et al., 2013).

IP devices such as the Codman microsensor (Codman, Raynham, MA) and the Camino (Integra Neurosciences, Plansboro, NJ) are also utilized to directly monitor ICP. Generally, the transducer/catheter is placed within the subarachnoid space and then is secured to the scalp (American Association of Neuroscience Nurses, 2011). During the insertion process, the ICP monitor and the intracranial transducers are zeroed and do not require further zeroing (Marcoux, 2005). The extracranial transducers are recalibrated per institutional protocol (usually every 12 hours). The nursing responsibilities involve monitoring for complications associated with IP devices including assessing for hemorrhage and fractures and monitoring for infection (Anderson et al., 2004). With both the EVD and IP monitor, the nurse must also try to keep the child from removing the device and pulling the equipment.

When ICP monitors are available, CPP can be calculated by the following equation: CPP = MAP – ICP. CPP represents an indirect measure of cerebral blood flow. The ICP should be treated to maintain adequate CPP between 40 and 50 mm Hg for children (Kochanek et al., 2012).

Assessing Cerebral Oxygenation

The main methods of measuring alterations in cerebral oxygenation are jugular venous oxygen saturation ($SjvO_2$), brain tissue oxygen partial pressure ($PbtO_2$), and near-infrared spectroscopy (NIRS). $SjvO_2$ measures the balance of global cerebral oxygen delivery and the rate of cerebral metabolic oxygen consumption (Pérez et al., 2003). The catheter to measure $SjvO_2$ is positioned in the bulb of the internal jugular vein (Blissitt, 2009) with the tip of the catheter above the level of C1/C2 disk to minimize contamination from the facial vein (Rohlwink & Figaji, 2010). Generally, normal values of $SjvO_2$ range from 55% to 75% (Pérez et al., 2003). Decreased $SjvO_2$ can occur with decreased systemic oxygen supply, local or systemic hypoperfusion, and increased cerebral metabolism or oxygen extraction (e.g., pyrexia and seizures). Children with severe TBI who experienced two or more $SjvO_2$ measurements $\leq$ 55% had an increased risk of poor neurologic outcome (severe disability, coma or persistent vegetative state, or brain death; Pérez et al., 2003). Increased $SjvO_2$ may occur with decreased cerebral metabolism, increased systemic oxygen supply, restricted oxygen diffusion, and hyperemia (Rohlwink & Figaji, 2010). The limitation to monitoring $SjvO_2$ is the inability to measure focal ischemia because the measurement only provides an assessment of global cerebral oxygenation (Rohlwink & Figaji, 2010).

Careful assessment and management of $SjvO_2$ catheters is necessary to ensure accurate results and prevent complications. When sampling blood from the catheter, withdraw at a rate of 2 milliliters per minute to decrease the risk of extracerebral contamination. Recalibrate in vivo using a blood sample at least every 24 hours. Maintaining a pressurized saline bag with continuous flush may prevent clots and facilitate proper functioning of the catheter (Blissitt, 2009). Nurses need to monitor for complications that can occur during insertion or while the catheter is indwelling: carotid artery puncture, hematoma formation, infection, thrombosis, and raised ICP (Rohlwink & Figaji, 2010).

Brain tissue oxygenation ($PbtO_2$) monitors (e.g., Licox [Integra Neurosciences, Plainsboro, NJ]) provide an estimate of the balance between cellular oxygen consumption and regional oxygen supply

(Purins, Lewén, Hillered, Howells, & Enblad, 2014). $PbtO_2$ is thought to be reflective of CBF, arterial oxygen tension, or a product of CBF and arteriovenous tension difference in oxygen tension (De Georgia, 2014; Rosenthal et al., 2008). Normal parameters for $PbtO_2$ values are >30 mm Hg (Maloney-Wilensky & Le Roux, 2010; Stippler et al., 2012). $PbtO_2$ values < 20 mm Hg are usually considered to be approaching ischemia (De Georgia, 2014). When $PbtO_2$ values are between 0 and 5 mm Hg for greater than 1 hour, there is a high risk of severe disability or death (Figaji et al., 2009; Figaji & Kent, 2010). Leading causes of decreased $PbtO_2$ levels include decreased arterial partial pressure of oxygen, poor cerebral blood flow, tissue barriers to diffusion (e.g., cytotoxic edema), increased arterial CO_2, increased ICP, and low CPP that can decrease $PbtO_2$ and lead to ischemia (Rohlwink et al., 2012).

The Licox system, the major $PbtO_2$ monitor utilized, can also monitor brain temperature and ICP simultaneously by probes inserted through a triple-lumen bolt (Stevens, 2004). The triple-lumen bolt is usually placed in the frontal lobe in noncontused tissue identified on CT scan (Narotam, Burjonrappa, Raynor, Rao, & Taylon, 2006; Stiefel et al., 2006; Ushewokunze & Sgouros, 2009) and at least 14 millimeters from other probes (e.g. EVD; Stevens, 2004). Because of localized microtrauma at the insertion site, the $PbtO_2$ sensor readings are not considered accurate for the first 30–120 minutes after insertion. The current recommendation is to maintain $PbtO_2 \geq 10$ mm Hg (Kochanek et al., 2012). After insertion, the nurse must ensure the Licox cables are secured per institutional protocol, usually at two points of tensions, one directly on the patient's head where the bolt is anchored to the skin and the other at the patient's shoulder (Wilensky et al., 2005). The $PbtO_2$ monitor does not require daily recalibration (Stevens, 2004). The dressing at the site is changed every 48 hours or when saturated and replaced with a dry, sterile, occlusive dressing (Wilensky et al., 2005). The nurse also needs to monitor for possible complications associated with $PbtO_2$ monitors: infection (Stiefel et al., 2006), hemorrhage (Stiefel et al., 2006), and catheter dislodgement (Stevens, 2004).

NIRS is a noninvasive measure of regional oxygen saturation (rSO_2) that provides continuous measurements. This measurement describes the balance of oxygen supply and oxygen demand of the regional cerebral tissue (Drayna, Abramo, & Estrada, 2011). The normal parameter of rSO_2 for children is about 70% (Yaron et al., 2003; Yoxall, Weindling, Dawani, & Peart, 1995). Preliminary data from 30 children suggest that cerebral rSO_2 is impacted by changes in end-tidal CO_2, heart rate, and hematocrit but is not effected by changes in ICP or CPP (Zuluaga, Esch, Cvijanovich, Gupta, & McQuillen, 2010). NIRS has not been used extensively in children with TBI (Amigoni et al., 2011).

Current Treatments for Children With Severe TBI

Many of the treatments for severe TBI are determined based on assessment findings of increased ICP, impaired cerebral oxygenation, cerebral edema, and cerebrovascular injuries. The treatments aim to restore adequate CBF and cerebral metabolism, reduce cerebral edema, and minimize cellular death to ameliorate secondary injury. The "Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents" (Kochanek et al., 2012) provides treatment-recommendation-based levels 1, 2, and 3. The treatment-recommendation-based levels are determined by the strength of the study designs available for each treatment, with level 1 having the strongest evidence and level 3 having the least evidence (Kochanek et al., 2012; see Table 3 for a summary of the evidence for treatment of severe TBI). The main treatment modalities include hyperosmolar therapy, temperature control, consideration of etomidate to control severe increased ICP and thiopental to control ICP, and consideration of prophylactic treatment to reduce incidence of early posttraumatic seizures (PTSs).

ICP monitoring is a mainstay in the treatment of severe TBI (Exo et al., 2011), with most guidelines considering values of >20–25 mm Hg requiring treatment (Kochanek et al., 2012). Current research also suggests maintaining $PbtO_2$ at a minimum of 10 mm Hg (Kochanek et al., 2012; Narotam et al., 2006). When the monitoring parameters are not within the acceptable ranges, treatments are necessary to prevent further injury to the cerebral tissue.

Hyperosmolar Therapy

Hyperosmolar therapy creates an increased serum osmolality by pulling fluid from interstitial space back into the cerebral vessel and into systemic circulation, lowering the volume in the cranial vault (Knapp, 2005). Hyperosmolar therapy with 3% hypertonic saline solution should be considered for intracranial hypertension because it alters cerebral volume (Weed, 1929). The effective acute dose of hypertonic saline is 6.5–10 milliliters per kilogram and the continuous dose is 0.1 milliliters to 1.0 milliliters per kilogram per hour to maintain ICP < 20 mm Hg (Kochanek et al., 2012). Nurses need to monitor serum osmolality every 4–6 hours to ensure it is maintained below 360 milliosmole per liter for 3% hypertonic saline (Kochanek et al., 2012).

TABLE 3. Treatments for Children With Severe TBI

Treatment	Level of Evidence	Recommendation
Hyperosmolar therapy	2	Hypertonic saline acute bolus dose of 6.5–10 ml/kg for increased ICP.
	3	Continuous 3% saline dose of 0.1–1.0 ml/kg/hr to maintain ICP < 20 mm Hg and serum osmolarity < 360 mOsm/l.
Temperature control	3	Avoid hyperthermia.
CSF drainage	3	CSF drainage through an EVD may be considered.
Barbiturates	3	High-dose barbiturate therapy in cases of refractory intracranial hypertension.
Decompressive craniectomy	3	Consider in early signs of neurologic deterioration or herniation or are developing intracranial hypertension refractory to medical management during the early stages of treatment.
Analgesics, sedatives, and neuromuscular blockade	3	Etomidate may be considered to control severe intracranial hypertension. Thiopental may be considered to control intracranial hypertension.
Antiseizure prophylaxis	3	Prophylactic treatment with phenytoin may be considered to reduce the incidence of early PTS.

Note. Adapted from Kochanek et al. (2012). TBI = traumatic brain injury; ICP = intracranial pressure; CSF = cerebrospinal fluid; EVD = external ventricular device; mOsm = milliosmole; ml = milliliter; kg = kilogram; hr = hour; l = liter; PTS = posttraumatic seizures.

Temperature Control

Systemic hyperthermia from inflammation after TBI can lead to additional secondary injury because pyrexia exacerbates many of the biochemical reactions (e.g., excitotoxicity, production of reactive oxygen species; Puccio et al., 2009). Animal studies suggest that hyperthermia is associated with poor outcomes and should be avoided, and thus, maintaining normothermia is recommended in severe TBI (Kochanek et al., 2012). Nurses should monitor the child's temperature and adjust the environment to ensure the child is not hyperthermic, also alerting the healthcare team if the child becomes hyperthermic.

CSF Drainage

Drainage of CSF is to reduce the volume of intracranial fluid, which in turn decreases the volume within the cranial vault and thus reduces the ICP. Drainage of CSF can occur through the EVD (Kochanek et al., 2012). The EVD can also be used for monitoring ICP, which is helpful in the management of children with severe TBI. Normal output of CSF is 3–5 milliliters per hour for infants, 5–10 milliliters per hour for children, and 10–15 milliliters per hour for adolescents (Vernon-Levett, 2006).

Barbiturate Therapy

Very limited evidence exists for the use of barbiturates in severe TBI. The use of barbiturates may be necessary, when CSF drainage, hyperosmolar therapy, and sedation and analgesia have not effectively lowered the ICP to maintain an adequate CPP (Marshall et al., 2010). Barbiturates decrease ICP by suppressing metabolism and altering vascular tone (Kochanek et al., 2012). Pentobarbital or thiopental are administered as the medications of choice (Glick, Ksendzovsky, Greesh, & Raksin, 2011; Marshall et al., 2010; Mellion et al., 2013). Nurses generally monitor patients with TBI who receive barbiturates for medically induced comas closely. Vital signs are measured at least hourly, if not more frequently, depending on the child's condition (Mazzola & Adelson, 2002). Continuous arterial blood pressure monitoring and cardiovascular support to maintain adequate CPP is usually required (Kochanek et al., 2012). Nurses also monitor for electrographic burst suppression, which is evaluated through the use of continuous EEG (Mellion et al., 2013). These children require critical care by vigilant specially trained nurses.

Decompressive Craniectomy

Decompressive craniectomy (DC) is a surgical procedure to remove a portion of the skull, debride the necrotic tissue, and allow unimpeded swelling of the brain and improved CBF (Morrow & Pearson, 2010). DC may be considered for children showing early signs of neurologic deterioration or herniation in the early stages of ICP or evaluation of a mass lesion refractory to medical treatment (Bowers, Riva-Cambrin, Hertzler, & Walker, 2013). The outcomes of children who undergo DC are generally positive; however, few studies exist examining pediatric patients (Thomale, Graetz, Vajkoczy, & Sarrafzadeh, 2010; Weintraub, Williams, & Jane, 2012). In studies with children who underwent

DC because of sudden increases in ICP, long-term outcomes showed moderate disability to good recovery of most of the children (Figaji, Fieggen, & Peter, 2003; Josan & Sgouros, 2006; Ruf et al., 2003). Currently, a large international randomized-controlled trial, "Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intra-Cranial Pressure (RESCUEicp)," is underway to better understand if DC is an effective form of treatment for TBIs for individuals aged 10–65 years (Bohman & Schuster, 2013; Hutchinson et al., 2006). Nurses caring for these children need to monitor for signs and symptoms of infection (Hockenberry & Wilson, 2007), maintain precautions to prevent physical pressure around the area without an intact skull, and assess and manage pain (Vernon-Levett, 2006).

Analgesia, Sedation, and Neuromuscular Blockade

The purpose of administering analgesia and sedation medication is to decrease pain and stress, which increases cerebral metabolic demands and increases cerebral blood volume, in turn raising ICP (Kochanek et al., 2012). Different analgesics and sedation medications can affect the cerebral vasculature, metabolism, autoregulation, ICP, and CPP. Unfortunately, because most analgesic and sedation medications have not been evaluated, in regard to the effect on ICP, they cannot be recommended as a treatment for intracranial hypertension. Etomidate and thiopental can be considered to treat intracranial hypertension. If etomidate is utilized, the risks of adrenal suppression must be considered (Kochanek et al., 2012). The use of neuromuscular blockade agents is mentioned in the guidelines as a consideration, but no recommendations are offered. Because of the paucity of evidence, additional medications frequently utilized in the pediatric intensive care are also reviewed. The recommendation is to allow the treating clinician to choose which medication is best for the patient.

Opioids (e.g., morphine, fentanyl), classified as analgesics agents, can directly affect the respiratory centers in the medulla, leading to a decreased respiratory rate and subsequent increased $PaCO_2$ (Kilbaugh, Friess, Raghupathi, & Huh, 2010). The increased $PaCO_2$ can cause vasodilatation of the cerebral vessels, increase ICP, and decrease CPP. The nurse should monitor for common side effects of opioids: constipation, urinary retention, sedation, nausea, vomiting, respiratory depression, bradycardia, hypotension, and pruritis (Kilbaugh et al., 2010).

Analgesia is often administered in conjunction with sedation. The classification of medications generally used for sedation is benzodiazepines (e.g., midazolam, lorazepam, diazepam). Benzodiazepines have several advantages for children with severe TBI: sedation, anxiolysis, muscle relaxation, anterograde amnesia, decreased cerebral metabolic rate, and anticonvulsant properties (Kilbaugh et al., 2010). However, clinically important side effects exist that could be detrimental: decreased blood pressure and depressed ventilation (Meyer et al., 2010). Initially, nurses should monitor vital signs each hour or more frequently depending on the child's condition and continue to monitor the child's pain level (Hockenberry & Wilson, 2007).

Antiseizure Prophylaxis

Prophylactic treatment with phenytoin may be considered to reduce the incidence of early PTSs (Kochanek et al., 2012). Early PTSs are witnessed clinical seizures or abnormal trace recordings on EEG within the first 7 days after the traumatic event (Arango et al., 2012), with about 44% of children having at least one seizure (Arndt et al., 2013). Risk factors associated with any type of PTS are young age, type of trauma (abusive head trauma), epidural hematoma (Arango et al., 2012), and skull fracture (Arndt et al., 2013). Risk factors associated with subclinical PTS are young age, abusive head trauma, subdural hematoma, and intra-axial hemorrhage (Arndt et al., 2013). Additional research is needed to guide the treatment of antiseizure prophylaxis.

Conclusion

The pathophysiologic effects of primary and secondary injuries play a key role in the overall outcomes for children with TBI. Trauma prevention is the only way to combat primary injury. Nurses can help educate parents and children about seat belt safety, falls preventions, and bicycle safety. The secondary injury phase that occurs when a complex biochemical and physiologic process is activated requires vigilant nursing care. Nurses must monitor for alterations in arterial oxygenation, cerebral metabolism, cerebral autoregulation, and cerebral blood flow through changes in the child's behavior and highly technical biometric equipment. The care and monitoring of children with severe TBI continues for days to weeks because the cerebral ischemic leads to increased permeability of the BBB and cellular death. Treatments exist to combat the effects of the secondary injury, but these treatments cannot "cure" TBI. The children who sustain a TBI may have no residual impairments, but others may experience catastrophic social, emotional, cognitive, and functional impairments.

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