Traumatic Brain Injury in Children: Acute Care Management

Kristen Geyer, Karen Meller, Carol Kulpan, and Bernice D. Mowery

Trauma remains the leading cause of preventable death in children. Traumatic brain injury (TBI) accounts for a large percentage of trauma-related deaths and complex morbidities. According to the Centers for Disease Control and Prevention (CDC) National Center for Injury Prevention and Control 2010 report, an average of 1.7 million people annually suffer from TBI, accounting for 30% of all traumatic deaths. TBI remains the leading cause of death and disability in pediatric patients from birth to 19 years of age. Each year, 500,000 children are seen in Emergency Departments, and pediatric deaths from TBI are estimated to be 2,500, annually (CDC National Center for Injury Prevention and Control, 2010).

Traumatic brain injuries are predominated in two distinct pediatric age groups: toddlers and adolescents. Males of all ages have a higher incidence of TBI than females. Child abuse, specifically shaken baby syndrome, is the leading cause of TBI in children less than one year of age. Falls are the leading cause of nonfatal brain injury in the toddler age group. Falls from windows or objects, such as televisions falling onto the child's head, are also common factors in this age group (Gill & Kelly, 2013). Motor vehicle crashes, whether driver, passenger, or pedestrian, are the overwhelming cause of TBI in the adolescent age group (CDC National Center for Injury Prevention and Control, 2010). Sports-related head injuries have been given a great deal of press in the past few years due in part to the recent studies of post-concussive injuries occurring at the professional level and in school-age children.

TBIs are categorized as mild, moderate, and severe. Children with mild brain injuries achieve a Glasgow Coma Scale (GCS) of 13 or higher. Moderate TBI is characterized by a loss of consciousness and physical and/or cognitive impairments with a GCS of 9 to 12. Children with severe TBI have GCS less than 8 and require airway and hemodynamic support (Tume, Thorburn, & Sinha, 2008).

Young children are more susceptible to head injury due to part to poor head control and a head size disproportionate to their body. The type of TBI that occurs in children is varied. Hematomas with or without skull fractures are common. Subdural hematomas are venous in origin and may be acute or chronic in presentation. An epidural hematoma is usually arterial in origin and can develop rapidly, requiring early surgical intervention. Concussions are the result of a blow to the head or after rapid deceleration, as seen in motor-pedestrian events. Diffuse axonal injuries are characterized by loss of grey/white matter differentiation, with poor outcomes. Diffuse cerebral edema is most commonly seen in severe TBI.

Pathophysiology

The skull is a rigid compartment and composed of three components: the bone (about 80%), blood (about 10%), and cerebrospinal fluid (about 10%) (Kumar, Singhi, & Singhi, 2012). These components are normally in a state of equilibrium. Increases in the volume of one component are compensated by a decrease in volume in the remaining compartments. In a patient suffering from head injury, normal compensation mechanisms can fail, resulting in increased intracranial pressure (ICP).

Brain injury can be divided into primary and secondary injury. The primary injury is caused by the initial trauma. Physical forces, such as acceleration, deceleration, or rotational forces, have an impact on the tissue and may result in skull fractures, brain contusion, intracranial hematoma, or diffuse axonal injury. Secondary injury is a result of the cascade of events that occurs after the initial injury, including edema, capillary leak, and activation of the secondary inflammatory response. The goal of care for patients with TBI is to aid in the com-

Kristen Geyer, BSN, RN, CCRN, is a Pediatric ICU RN3, Inova Hospital for Children, Falls Church, VA.
Karen Meller, MA, RN, CCRN, is a Pediatric ICU RN, Inova Hospital for Children, Falls Church, VA.
Carol Kulpan, BSN, RN, CCRN, is a Pediatric ICU RN, Inova Hospital for Children, Falls Church, VA.
Bernice D. Mowery, PhD, PNP, RN, is an Education Coordinator, Inova Learning Network, Falls Church, VA, and a member of the Pediatric Nursing Editorial Board.
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Intracranial Pressure

Intracranial pressure (ICP) monitoring provides objective data necessary for the care of patients with a TBI. Although more research is being conducted on TBI in children, data from adult studies continue to guide management of pediatric patients. In adults, management protocols that include ICP monitoring have been associated with lower mortality rates (Kochanek et al., 2012). ICP monitoring can be done through an intraparenchymal device, also called a bolt, or an intraventricular device connected to an external drain, also called an extraventricular drain (EVD). Data from the two devices have been shown to correlate well. The bolt may cause less local tissue damage; however, an EVD allows for drainage of cerebral spinal fluid (CSF) and is the recommended device in adult TBI guidelines (Bhalla, Dewhirst, Sawardekar, Dairo, & Tobias, 2012). The goal for ICP is less than 20 mmHg for all ages.

Cerebral Perfusion Pressure

Cerebral perfusion pressure (CPP) is the pressure gradient that causes blood flow to the brain. CPP that is too low could cause ischemia and if too high could increase ICP. It is easily calculated as the difference between mean arterial pressure (MAP) and ICP (CPP = MAP - ICP). Normal CPP values are not clearly established for children. However, it is widely accepted that values vary with age, and the minimal CPP necessary to prevent ischemia for infants and toddlers is greater than 40 to 50 mmHg and for children greater than 50 to 60 mmHg. A CPP of less than 40 mmHg is a significant predictor of mortality in children with TBI (Kumar et al., 2012).

Management And Nursing Care

Management of TBI is divided into first-, second-, and third-tier therapy. The goal of all therapies is to maintain a low and stable ICP and adequate blood pressure, preventing any critical drops in CPP. Of the three components in the skull, the brain substance remains fairly constant; therefore, treatment initially focuses on a shift in CSF or blood from the intracranial space. Surgical interventions may be required if medical therapies in the first and second tiers are unsuccessful.

General Measures And First-Tier Therapies

Adequate blood pressure, oxygenation and ventilation. Hypotension and hypoxia are the most common contributors to secondary brain injury and poor outcome (Emeriaud, Pettersen, & Ozanne, 2011). Data suggest that even one episode of hypotension can have a negative impact on outcomes (Bhalla et al., 2012). Correction of hypotension should begin with fluid boluses, but many patients require vasoactive agents to ensure that an adequate blood pressure is maintained.

Patients with a severe TBI require intubation and mechanical ventilation. Hypocapnia-induced cerebral vasoconstriction reduces cerebral blood volume, resulting in a decreased ICP; however, extreme hyperventilation may decrease cerebral oxygenation leading to brain ischemia. Therefore, current recommendations include mild hyperventilation with PaCO2 35 to 38 mmHg while maintaining PaO2 greater than 60 mmHg (Tume et al., 2008). In patients with lower oxygenation, increasing inspiratory time, FiO2, and peak inflating pressures are the preferred treatment over positive end expiratory pressure (PEEP), which is transmitted to the intracranial vault, increasing ICP (Bhalla et al., 2012).

CSF drainage and the external ventricular drainage system. Draining CSF through the use of an external ventricular drain reduces the fluid volume in the skull, decreasing ICP. Drainage can be done intermittently with ICP spikes of greater than 20 mmHg or continuously in those patients with hard-to-control ICP. The ventricular catheter is the preferred device for monitoring ICP. Advantages include low cost and the ability to drain cerebral spinal fluid CSF to reduce ICP. Disadvantages are risks of infection, hemorrhage due to damage of blood vessels as the catheter is placed, and removal of too much CSF, resulting in ventricular collapse. Further, the catheter can become displaced or occluded with blood or tissue, making it nonfunctional (Milonovich & Eichler, 2013).

The external ventricular drainage (EVD) system kit contains an adjustable collection chamber (buretrol), a stopcock at the zero mark where the transducer will be placed; a leveling device or laser, a stopcock for sampling cerebral spinal fluid (CSF), and a collection bag. Sterile technique, including cap, mask, sterile gown, and gloves, is maintained to prepare the EVD system. The system should be primed with preservative-free sterile saline to displace the air prior to the surgeon attaching it to the intraventricular catheter, and the end should be kept sterile. A catheter is inserted into the ventricle and then connected to an external pressure transducer via a fluid-filled system. The stopcock of the transducer must be placed at a consistent, precise location of reference. Reference points for the lateral ventricles are the external auditory meatus (tragus of the ear) or halfway between the outer canthus of the eye and the external canal of the ear. Every institution should consistently use one reference landmark for external ventricular drains. After the ventricular catheter has been placed, the external transducer should be leveled. To ensure accurate hemodynamic measurements, the patient and transducer must be positioned at the same level before the system is zeroed. It is important to note the reference point (e.g., tragus) so if the patient’s position or height of the bed is changed, the EVD is adjusted to the proper level. “Zeroing” the transducer adjusts the transducer so it reads zero when open to the atmosphere. The system will begin pressure measurements at a neutral pressure point of 0 mmHg (see Figure 1) (Ballestas et al., 2007).

The level of the collection chamber determines the flow of CSF from the ventrices. The pressure gradient between the ventricles and the collection chamber causes the CSF flow. After zeroing the transducer, the collection chamber should be raised to the level ordered by the physician. The physician usually orders this in “cm of H2O” above the reference point (e.g., tragus) for the lateral ventricle. The EVD system has two scales potentially confusing to practitioners: one in cm H2O and the other in mmHg (1.36 cm H2O = 1 mmHg). If the physician orders the EVD to be leveled at 12 cm H2O above the external auditory meatus, but the bedside monitor reads the ICP in mmHg, the ICP will have to be greater than 9 mmHg to drain. Note that 9 mmHg is
just above the 12 cm H2O on the device (see Figure 2). When the bedside monitor ICP reads “9,” drainage should be seen in the collection chamber. Physician orders should also include whether drainage should be intermittent or continuous. The direction of the stopcock will determine the flow of CSF. When the stopcock is off to the transducer, it will continuously drain but not monitor ICP. When the stopcock is off to the drain, it will not drain but continuously read an ICP. When continuously draining, an intermittent ICP reading should be done every hour and as indicated by changes in the patient’s condition (Almond et al., 2013).

Normal ICP range varies with age, and acceptable pediatrics ranges have not been established. Normal values are considered less than 10 to 15 mmHg for adults and older children, and less than 3 to 7 mmHg for young children. ICP values greater than 20 to 25 mmHg require treatment, and greater than 40 mmHg may indicate life-threatening intracranial hypertension (Rangel-Costello, Gopinath, & Robertson, 2008). Increases in ICP or decreases in blood pressure can reduce CPP.

Although intraventricular catheters for the management of CSF drainage to decrease ICP have been considered the gold standard, there are many actions taken in conjunction with this therapy. Other interventions include sedation, analgesia, mild hyperventilation, mild hypothermia, craniectomy, antiseizure prophylaxis, and hyperosmolar therapy.

Nursing management. Accurate monitoring of an EVD is essential to prevent complications and is an important nursing responsibility. Figure 3 summarizes nursing responsibilities when caring for a patient with an EVD. Although the topic of ICP waveform is beyond the scope of this discussion, it is important to note that nurses in critical care areas will need to routinely assess the ICP waveform. To facilitate efficient response to increases in ICP, physician order sets for pediatric patients with severe TBI can be helpful (see Figure 4).

Nursing interventions and ICP. Endotracheal suctioning and manual ventilation (ETSMV) and log-rolling are reported to significantly increase ICP in pediatric patients older than two years without craniectomy, exceeding 30 mmHg in some patients. Lidocaine should be used five minutes prior to endotracheal suctioning to suppress cough reflex and minimize the Valsalva maneuver, which can raise ICPs. Doses of lidocaine should not be repeated more frequently than every two hours (Kumar et al., 2012). Administering sedatives and analgesics several minutes before suctioning may also be beneficial. No significant increase in ICP has been reported with eye care,
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Figure 4.
Sample from Order Set: Increased Intracranial Pressure (ICP) Management

If ICP is greater than 20 mmHg for more than 5 minutes or greater than 30 mmHg for 1 minute:
- Assure volume and MABP status as outlined per Order Set using volume and pressors.
- Remove external stimuli.
- With extended elevation, open ventriculostomy drain (if present) for 10 minutes.
- With continued elevation, fentanyl bolus and start infusion if not already present.
- With continued elevation, versed bolus and start infusion if not already present.
- With continued elevation, vecuronium bolus.
- Call MD if ICP remains greater than 20 mmHg for further therapy.

Tier 1 – Mannitol, hypertonic (3%) saline, and mild hyperventilation (pCO₂ 30 to 35).
Tier 2 – Thiopental coma, aggressive hyperventilation (pCO₂ less than 30), aggressive hyperosmolar therapy (Na greater than 160), lumbar drain, and mild hypothermia (32 to 34 degrees C).

mouth care, or bathing (Tume, Baines, & Lisboa, 2011).

In addition to draining CSF, other nursing measures to decrease ICP include administering sedatives and analgesia, positioning, and limiting stimulation. In adult patients, neither CSF drainage nor limiting stimulation had significant effects on ICP values at one minute or five minutes after the intervention. Olson, McNett, Lewis, Rieman, and Bautista (2013) found that family members talking in the room and the administration of sedatives and analgesia lowered ICP significantly. Although significant effects of nursing interventions on ICP have been reported, these effects were not consistent in all patients. Further research to evaluate the effects of nursing interventions on ICP in pediatric patients is needed.

Proper patient positioning. The patient’s head should remain elevated 15 to 20 degrees to reduce venous outflow pressure. Elevating higher could lead to a decrease in CPP (Bhalla et al., 2012). The head should also remain midline. Rotation of the head impedes jugular venous flow and raises ICP. The patient may still be wearing a c-spine collar, which can help maintain the head in the midline position; however, care should be taken that the collar not be too tight, which could impede venous drainage.

Sedation and analgesia. Sedatives and analgesics are used to decrease cerebral metabolic demand, thereby reducing cerebral blood volume and ICP. Appropriate pain management is also crucial to facilitate normal blood pressure and ICP. Agents such as midazolam, morphine, fentanyl, and dexmedetomidine can be used, adapting doses to the individual patient. Although recently challenged, it has long been accepted that ketamine increases ICP and should be avoided in patients with TBI (Bhalla et al., 2012).

Seizure prophylaxis. Seizures have been reported in 12% to 40% of patients within the first seven days following moderate to severe TBI (Bhalla et al., 2012). Infants and small children are at a higher risk of post-traumatic seizures because of the increased excitability of the developing brain (Bhalla et al., 2012). Seizures may also occur with electrolyte imbalances. Seizure activity increases the cerebral metabolic rate, which can raise ICP. It may be difficult to detect seizures in the deeply sedated patient with TBI. The use of continuous EEG monitoring may be helpful. Subtle signs, such as sudden changes in pupillary reaction along with tachycardia, with or without desaturation, may be indicative of underlying seizure activity. Propylactic anticonvulsant therapy using phenytoin must be considered during the first week following injury.

Temperature control. Hyperthermia must be avoided because it increases metabolic rate by 10% to 13% per degree Celsius (C) and is a potent cerebral vasodilator (Kumar et al., 2012). First-tier therapy involves keeping the temperature below 38°C using antipyretics and surface cooling as necessary (Kumar et al., 2012). Evaluation of the benefits of therapeutic hypothermia (temperatures less than 35°C) because first-tier therapy has shown conflicting results. More research is needed to determine if lowering temperatures in first tier is neuroprotective, and if so, to establish protocols for effectiveness (Fox et al., 2010; Emeriaud et al., 2011).

Glucose control. Hyperglycemia occurs in 38% of children after TBI and has been associated with poorer outcomes when compared to children with glucose levels less than 200 mg/ml (Milonovich & Eichler, 2013). Stress-related hyperglycemia from increases in catecholamine release following trauma and the hypermetabolic state is usually seen in the first 18 hours post-injury and peaks in three to four days (de Carvalho & Leite, 2009). However, based on current literature, there is not enough evidence for strict glycemic control in pediatric patients (Kochanek et al., 2012). Infants and young children are notably at greater risk for hypoglycemia (Hazinski, 2013). Hyperglycemia should be treated carefully, avoiding hypoglycemia because brain metabolism depends on a constant supply of glucose (Emeriaud et al., 2011). Monitoring patients for both hypo- and hyperglycemia to provide individual and appropriate treatment is crucial to preventing secondary damage from TBI.

Hyperosmolar therapy: Mannitol and hypertonic saline. The goal of hyperosmolar therapy is to increase the osmotic gradient across the tissue membrane, leading to a shift of water from the tissue cells into the intravascular space, reducing brain water content, and thus, ICP. Treatment options are mannitol or 3% hypertonic saline. Mannitol can decrease ICP by reducing blood viscosity as well as drawing water from the brain parenchyma into the systemic circulation. Mannitol is available in a 20% solution, and dosing regimens vary from 0.25 to 1.0 gram/kg (Bhalla et al., 2012). Another option is a 3% saline solution, which can be administered as a bolus or as a continuous infusion, ranging from 0.1 to 1.0 ml/kg per hour, with the goal of keeping serum sodium between 145 to 160 mEq. The minimal dose needed to keep ICP less than 20 mmHg should be used. The benefit of using hypertonic saline rather than mannitol is its lack of diuretic effects, preserving intravascular volume and reducing the risk of a reduc-
tion in blood pressure and CPP (Tume et al., 2008).

It is important to monitor serum osmolality when using hyperosmolar therapy. Serum osmolality above 320 mOsm should be avoided because acute renal failure can occur, although tolerance of levels as high as 360 mOsm have recently been reported (Kochanek et al., 2012). Electrolytes should also be closely monitored and replaced as needed.

Blood products. Both anemia and coagulopathy are common after TBI. Tissue factor is released following brain injury, which then activates the coagulation cascade, resulting in thrombin formation and the depletion of coagulation factors. Treating anemia has been common following TBI, but current concerns about transfusion side effects and lack of evidence to support an optimal hemoglobin target have resulted in lower acceptable transfusion thresholds (Emeriaud et al., 2011). Blood products should be used to treat coagulation disturbances especially prior to invasive procedures such as EVD or ICP catheter placement (Bhalla et al., 2012).

Second-Tier Therapies

Heavy sedation/barbiturate coma. Some children with severe TBI may develop intracranial hypertension that is resistant to first-tier medical management. These patients may require increased levels of sedation, including barbiturates. High-dose barbiturate therapy lowers ICP by decreasing cerebral metabolism, thus decreasing cerebral blood flow. Continuous infusion rates are titrated by monitoring EEG patterns to achieve burst suppression. Side effects can include hypertension and decreased cardiac output; thus, the use of additional inotropes may be necessary to maintain an adequate blood pressure (Kochanek et al., 2012).

Moderate hypothermia. If first-tier therapy is not successful, therapeutic hypothermia may reduce cerebral metabolic demands, inflammation, lipid peroxidation, excitotoxicity, cell death, and acute seizures. Hypothermia is defined as temperatures less than 35°C. If hypothermia is used, it may be necessary to use a neuromuscular blockade to prevent shivering (Kochanek et al., 2012).

Mild hyperventilation. Hyperventilation causes vasoconstriction and consequent lower ICP, but if the CO₂ is pushed too low, the blood flow may be diminished to the point of ischemic injury. Hyperventilation where PaCO₂ is pushed as low as 30 mmHg is an accepted therapy for severe TBI. Continuous end-tidal CO₂ (etCO₂) monitoring as well as monitoring of brain oxygenation may prevent decreasing CO₂ too low and prevent cerebral ischemia (Emeriaud et al., 2011; Kochanek et al., 2012).

Third-Tier Therapies

Decompressive craniectomy. For patients showing early signs of neurologic deterioration or herniation, or for those not responding to first- or second-tier therapy, surgical treatment involves removing part of the skull. This allows for swelling to occur more freely while limiting secondary injury. Procedures vary and may consist of uni- or bilateral subtemporal decompressions, hemispheric craniotomies, circumferential craniectomy, or bifrontal craniectomy (Kochanek et al., 2012). Once the edema has subsided, a cranioplasty is performed, either placing the patient’s autologous bone flap or a specialized ceramic titanium plate.

Endocrine Dysfunction

Children with TBIs may also suffer from endocrine abnormalities, including syndrome of inappropriate antidiuretic hormone (SIADH), diabetes insipidus (DI), or cerebral salt wasting (CSW) (Roberts, 2013). A discussion of each follows.

SIADH is the result of increased antidiuretic hormone (ADH) secretion. Increased water re-absorption causes increased intravascular volume with decreased urine output. The signs of SIADH in children with TBI include increased central venous pressure, low serum osmolality, low (less than 130 meq/L) serum sodium, and increased sodium in the urine with high urine osmolality. Treatment for SIADH includes fluid restriction and diuretics. A slow correction of serum sodium levels uses an intravenous solution of 3% saline at 2 to 4 cc/kg. Careful monitoring of serum sodium levels and observing the patient for seizures is essential.

Diabetes Insipidus (DI) is the opposite of SIADH. An inadequate supply of ADH, which is often seen at the end stage (brain death) following TBI, causes DI. The lack of ADH prevents water reabsorption, thereby causing significant increases in urine output. Clinical signs of DI include polyuria, dehydration, hypernatremia, and increased serum osmolality. Although the onset of DI is an ominous sign, clinical management is indicated to preserve organ function and hemodynamics in the prospect of organ donation. Treatment includes volume replacement, usually equal to urine output, in addition to vasoressin IV or intranasal/SQ desmopressin.

Cerebral salt wasting (CSW) may be the result of natriuretic peptides, specifically atrial and brain peptides, which are salt-wasting hormones that alter blood. The first signs of CSW present at two to seven days after injury and include low sodium levels (less than 130 meq/L) with high urine output and dehydration. The management of CSW includes volume replacement and sodium correction with 3% saline solution. The use of fludrocortisone, a potent mineralocorticoid, has been shown to stimulate reabsorption of sodium and water in patients with CSW.

Urine output and central venous pressure help differentiate SIADH from CSW. In SIADH, urine output is low with volume overload (increased CVP), whereas CSW usually presents with high urine output and mild to moderate dehydration (decreased CVP). It is important to observe for seizure activity when the patient with SIADH/CSW has low serum sodium levels (Roberts, 2013).

Nutrition

In the healthy child, nutrition is necessary for normal growth and development. On average, the daily energy requirements for a healthy child can be as high as 90 kcal/kg (Martin & Kerr, 2013). A child with a traumatic injury will have an increased caloric need due in part to a hypermetabolic state. The hypermetabolic state is exhibited as fever, increased oxygen consumption, tachycardia, and hyperglycemia (de Carvalho & Leite, 2009). Nutrition in the trauma patient is essential for healing and continued organ function. The lack of adequate nutrition has been shown to delay wound healing, increase the risk of infection, and prolong overall recovery (Thorpe & Eidov, 2003).

Trauma patients should be evaluated for individual nutritional requirements because pre-trauma nutri-
tional status and a child’s current state of injury may require adjustments to caloric and nutrient needs (Whitehurst, 2009). Trauma patients can have protein loss due to drainage of cerebral spinal fluid, and from chest tubes and open wounds; therefore, a nutritional evaluation of protein and amino acids required for healing is essential to maintain nitrogen balance.

Current recommendations advocate full feedings no later than seven days post-injury (Kochanek et al., 2012). Enteral feedings are the preferred method to provide nutrition in the absence of concurrent abdominal injuries. Enteral feedings provided through nasogastric or transpyloric feeding tubes, and have been shown to preserve gut integrity and reduce translocation of bacteria. Studies have shown that earlier enteral feedings can reduce infection, improve healing, and demonstrate better outcomes three months and one year after injury (Cook, Peppard, & Magnunson, 2008; Gathinji & Harris, 2009).

For patients with TBI with concurrent abdominal injuries where enteral feeds are not possible, parental nutrition with intralipids may be required. The administration of parenteral nutrition escalates risks because central venous access is required, which increases chances of associated blood stream infection and possibly also adding to the complication of glycemic control.

Family and Outcomes

Many children and their families who survive the acute care course of TBI will face an uncertain and challenging future. During all phases of care, the family’s need for information remains paramount. Family members are bombarded with treatment terminology: TBI, ICP, CPP, and craniectomy. Families need information they can understand that is consistent, honest, and frequently updated (Keenan & Joseph, 2010). The multitude of consultants who may give conflicting information contributes to an already stressful situation. Open communication and frequent updates help clarify and reinforce information for families. During the course of therapy, families are dealing simultaneously with uncertainty while holding on to hope (Keenan & Joseph, 2010). Uncertainty is seen in the early stages of therapy and is focused on survival and defining the extent of injury. Hope cannot be underestimated nor undervalued by the health care team.

A small sign of progress, such as no ICP spikes in a four-hour period, is often seen as a major positive progression to a vigilant family member.

There is increasing evidence of post-traumatic stress symptoms and disorder in both patients and family members. The financial stress of hospitalization, loss of income, and emotional stress adds complexity to the family dynamics and the need for additional support.

Unfortunately, current methods are unable to predict which patients who survive the initial TBI hospitalization will have detrimental outcomes. An encouraging Glasgow Outcome Score at the time of discharge is a poor indicator for quality of life 12 months post-injury (Winthrop, 2010). The long-term effects of TBI may not be evident until six to 12 months after injury, depending on the age of the child at the time of injury. In some cases, deficits may not present until years later and are often exhibited by declining school grades or changes in behavior.

Sleep disturbances are the most common sequelae reported to affect children after TBI (Wiseman-Hakes, Colantonio, & Gargaro, 2009). Cognitive, behavioral, and emotional deficits have been noted in children of all ages. Cognitive deficits are seen in the form of memory loss, impaired judgment, and concentration. Behavioral and emotional disorders, such as depression, mood swings, and social adjustment, are seen but may not become evident until a child’s return to school (Lewandowski & Frosh, 2003).

Summary

The intensive nursing management of the pediatric patient with severe TBI requires attention to physiological monitoring and intervention, as well as psychosocial support for the child and family. Initial stabilization and the measures to prevent secondary injury after TBI provide complex nursing challenges. Increased study of interventions for TBI in the pediatric population is improving outcomes, but more research is needed to evaluate the effects of nursing interventions on ICP, determine what measures are effective for supporting the patient and family, and develop a model to predict long-term outcomes so that interventions can be initiated earlier.

References


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**Goal**

To provide an overview of traumatic brain injury, treatment modalities, and support for patients and their families.

**Objectives**

2. Discuss the pathophysiology of TBI.
3. Explain the three tiers of therapy for patients with TBI.

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