Management of Increased Intracranial Pressure in the Critically Ill Child With an Acute Neurological Injury

Kelly Keefe Marcoux, MSN, CPNP-AC, CCRN

Children may have increased intracranial pressure (ICP) for a variety of reasons. The most common etiologies of increased ICP in the pediatric intensive care unit (PICU) are due to severe traumatic brain injury (TBI), hydrocephalus, brain tumors, infections (ie, meningitis, encephalitis), metabolic encephalopathy, hypoxic/ischemic brain injury, cerebral infarction, and intraparenchymal blood due to a ruptured arteriovenous malformation or aneurysm. The mechanism by which ICP increases varies depending on the specific etiology (Table 1). The increased ICP can be due to either an increase in tissue volume, cerebral blood volume, or cerebrospinal fluid (CSF) volume. Examples of these are listed in Table 2. One or more of these factors occurring alone or simultaneously can increase ICP. The pathophysiology and management of increased ICP is based on the Monro-Kellie doctrine.

Normal Cerebral Physiology

Intracranial pressure is defined as the total pressure exerted by the brain, blood, and CSF in the intracranial vault. The Monro-Kellie doctrine states that the cranium is a fixed vault made up of 3 components: the brain (≈80%), blood (≈10%), and CSF (≈10%). When there is an increase in any one of these components, one or more of the other components must decrease to keep the total volume the same. The blood and the CSF are the only compartments that can compensate. The CSF compensates by displacing CSF from the ventricles and the cerebral subarachnoid...
Table 1: Etiology of Increased Intracranial Pressure Based on Acute Neurological Insult

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<thead>
<tr>
<th>Acute Neurological Insult</th>
<th>Possible Etiology of Increased Intracranial Pressure</th>
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<tbody>
<tr>
<td>Traumatic brain injury</td>
<td>Hematoma, cerebral edema, cerebral ischemia, intraventricular or intracerebral hemorrhage</td>
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<tr>
<td>Hydrocephalus</td>
<td>Obstruction or impairment of cerebrospinal fluid absorption, cerebral edema</td>
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<tr>
<td>Infections (e.g., meningitis, encephalitis)</td>
<td>Meningeal scarring, inflammatory response, cerebral edema, hydrocephalus, cerebral hyperemia</td>
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<tr>
<td>Brain tumor</td>
<td>Mass effect of tumor, peritumoral edema, hydrocephalus</td>
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<tr>
<td>Intraparenchymal bleed</td>
<td>Mass effect, cerebral edema</td>
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<tr>
<td>Intraventricular bleed</td>
<td>Hydrocephalus</td>
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Space through the foramen magnum to the spinal subarachnoid space. The production of CSF can also be decreased, as well as the absorption increased, to decrease the total CSF. In addition, venous blood compensates by being displaced by the dural venous sinuses. For instance, if there is an intracranial mass (e.g., brain tumor), the brain and the arterial volume will remain static while the CSF and the venous volumes will decrease until they can no longer compensate, at which point the ICP will increase (Figure 1). However, infants and children with open fontanels and sutures (usually less than 18 months old), may be able to compensate longer to chronic changes in the intracranial vault, but will still be susceptible to acute increases in ICP. It is crucial to understand the Monro-Kellie doctrine because management of increased ICP revolves around this basic principle.

Other key concepts of intracranial dynamics include autoregulation, compliance, cerebral blood flow, cerebral metabolic rate, and cerebral perfusion pressure (CPP). Autoregulation is the maintenance of a steady cerebral blood flow (CBF) by vasoconstriction and vasodilatation of the cerebral vessels despite fluctuations in systemic blood pressure. If autoregulation is impaired, CBF and cerebral blood volume (CBV) will become dependent on changes in systemic blood pressure. Compliance is an indicator of the brain’s tolerance to increases in ICP. It is defined as a change in pressure resulting from a change in volume. Each patient has varying degrees of compliance even with similar injuries. The exact factors contributing to this are still unknown. When the patient’s compliance is exhausted, there is a dramatic increase in the pressure/volume curve, leading to a rapid elevation in ICP.

In an uninjured brain, cerebral blood flow is regulated to supply the brain with adequate oxygen and substrates to meet its demands. The main physiologic influences on CBF are the partial pressure of arterial carbon dioxide (PaCO₂), arterial oxygenation, pH, CPP, and cerebral metabolic rate. The PaCO₂ is directly proportional to CBF and is the most potent chemical mediator of CBF. An increase in PaCO₂ will cause vasodilation, which will increase CBF and, thereby, potentially increase ICP. Likewise, acidosis and hypoxemia will also increase CBF by causing vasodilation. CBF in excess of tissue demand will lead to hyperemia. Normal CBF in an adult is approximately 50–70 mL/100 g/min, and CBF of healthy children was found to be as high as 108 mL/100 g/min. CBF < 20 mL/100 g/min is usually indicative of cerebral ischemia and has been associated with a poor outcome in children with TBI.

Seizure activity and fever will increase CBF and cerebral metabolic rate. Methods to decrease the cerebral metabolic rate, such as hypothermia and barbiturates, will also decrease CBF. Cerebral metabolism is
dependent on glucose to meet the body’s energy demands. The brain functions via adenosine triphosphate (ATP) and the Krebs cycle to maintain aerobic metabolism. If there is hypoxia, the Krebs cycle cannot be activated and anaerobic metabolism will ensue. This will lead to the production of pyruvate and lactate, which will decrease the amount of ATP available for the cells and, thereby, decrease the amount of energy available.4

Cerebral perfusion pressure (CPP) is the pressure at which cells are perfused and is an important indicator of CBF. Sufficient CPP is necessary to prevent the development of secondary cerebral ischemia. CPP provides an indirect measurement of CBF and is calculated by measuring the difference between the mean arterial pressure (MAP) and the ICP as demonstrated by the following equation: CPP = MAP – ICP. Normal CPP values for children are not clearly established, but the following values are generally accepted as the minimal pressure necessary to prevent ischemia: adults CPP >70 mm Hg; children CPP >50–60 mm Hg; infants/toddlers CPP >40–50 mm Hg.5 Research has shown that a CPP <40 mm Hg is a significant predictor of mortality in children with TBI.6 Furthermore, in a study of 17 comatose children with severe central nervous system (CNS) infections, a CPP <30 mm Hg was associated with universal mortality.7

Normal ICP values are estimated at 2–6 mm Hg for infants and 3–7 mm Hg for young children. The normal ICP for older children and adults is 0–10 mm Hg.8 Intracranial hypertension is defined as an ICP >20 mm Hg for more than 5 minutes, although a lower number may be used for infants and young children. One suggestion for pediatric patients has been to maintain the ICP <20 mm Hg for children aged 8 years to adults, <18 mm Hg for children aged 1–8 years, and <15 mm Hg for infants.5

An ICP >20 mm Hg has been found to be a powerful predictor of poor neurological outcome in adults with TBI.9,10 ICP normally increases with activities such as suctioning, painful stimuli, and coughing and does not warrant intervention unless it does not return to baseline within about 5 minutes. However, interventions to minimize the ICP response to these activities, such as lidocaine prior to suctioning, should be instituted. It is important to distinguish “normal” or expected increases in ICP versus intracranial hypertension because the latter requires immediate intervention based on a tiered management approach.

Pathophysiology Related to Increased ICP

In addition to the primary injury caused by TBI, a space-occupying lesion, or an intracerebral bleed, etc., cerebral edema is often a major cause of increased ICP. Cerebral edema can be categorized as vasogenic, cytotoxic, or interstitial. However, rarely does one occur in isolation; rather, the patient with cerebral edema may have a combination of all 3 mechanisms. Vasogenic cerebral edema is due to increased capillary permeability around the area of injury or inflammation. It can be local or diffuse and occurs around mass lesions (eg, brain tumors, abscesses, intracerebral hematomas) and inflammatory processes (eg, meningitis, encephalitis). Cytotoxic cerebral edema is due to cerebral ischemia and hypoxia, causing irreversible cell death. The cells affected may include neurons and astrocytes, and occurs in diffuse axonal injury, cerebral infarction, and near drowning. Interstitial cerebral edema is often seen in patients with obstructive hydrocephalus or excessive CSF production and is due to an increase in the hydrostatic pressure of CSF. Cerebral edema begins to occur immediately after TBI, intracerebral hemorrhage, and other cerebral insults, and increases for 72 hours post-insult.11
Assessment and Monitoring

The initial assessment of the neurologically injured child consists of assessment of ABCs (airway, breathing, and circulation), Glasgow Coma Scale (GCS) score, cranial nerve function, signs and symptoms of increased ICP, temperature, and cardiorespiratory assessment. Astute observation of any abnormal respiratory patterns is crucial, since this may be the first vital sign change indicating neurological dysfunction. Abnormal respiratory patterns that may be detected include Cheyne-Stokes, central neurogenic hyperventilation, apneustic, ataxic, and cluster breathing. All, except for Cheyne-Stokes, indicate brain stem abnormality. Cheyne-Stokes is less specific and indicates a cerebral, cerebellar, or brainstem impairment.

The most ominous cardiorespiratory abnormality is Cushing’s triad. Cushing’s triad occurs when there is cerebral ischemia causing peripheral vasoconstriction. This vasoconstriction leads to increased systolic blood pressure to improve cerebral perfusion. Cardiac baroreceptors sense this increased blood pressure, resulting in a vagal response manifested as bradycardia. Abnormal or irregular respirations are the final component of Cushing’s triad and occur due to brainstem compression. It is important to note earlier signs of increased ICP (see Table 3) because Cushing’s triad is a very late sign in neurologically injured children and usually indicates impending herniation.

Neurological Assessment

A thorough neurological assessment must be performed as soon as the child is clinically stable because this will serve as the baseline for comparison of future examinations. After the cardiorespiratory status has stabilized, the GCS is determined. It is important to utilize a modified GCS for infants and young children in order to obtain the most accurate score (Table 4). In a study of 151 children, a GCS < 8 twenty-four hours after TBI, in addition to factors such as hypoxia and cerebral edema, was associated with a poor outcome. To obtain the best response, it is necessary to give the patient the maximal stimulation—this may include giving painful stimuli such as mandibular pressure, sternal rub, or supraorbital pressure. These tests are optimal because they assess the central nervous system response, whereas fingerbed pressure assesses peripheral pain response.

Cranial nerve (CN) testing is next and begins with an assessment of CN III to determine direct and consensual pupillary response including size, reactivity, and shape of each pupil. It is imperative to be aware of medications that were administered that may affect pupil response (eg, atropine, narcotics). At least 10 seconds should elapse between each eye exam to allow the consensual response to fade. Abnormal pupillary responses include pupils that are unequal, constricted, dilated, nonreactive, or have hippus (Table 5). Hippus is the abnormal dilation and constriction of the pupil in response to bright light. It may indicate pressure on CN III and be associated with transtentorial herniation or it may be insignificant and represent a normal variant.

If the patient is awake, the 6 fields of gaze are assessed to determine extra-ocular movements (EOMs). If the patient is unresponsive, EOMs can be assessed by oculocephalic testing (Doll’s eyes) and oculovestibular testing (cold calorics). However, if the C-spine has not been cleared, the oculocephalic test should be deferred. An abnormal response

**TABLE 3**  •  Signs and Symptoms of Increasing Intracranial Pressure

<table>
<thead>
<tr>
<th>Early signs and symptoms</th>
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<tr>
<td>- Headache</td>
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<tr>
<td>- Emesis</td>
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<tr>
<td>- Change in level of consciousness</td>
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<tr>
<td>- Decrease in Glasgow Coma Scale score</td>
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<tr>
<td>- Irritability</td>
</tr>
<tr>
<td>- Sunsetting</td>
</tr>
<tr>
<td>- Decreased eye contact (infants)</td>
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<tr>
<td>- Pupil dysfunction</td>
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<tr>
<td>- Cranial nerve dysfunction</td>
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<tr>
<td>- Seizures</td>
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<tr>
<td>Late signs and symptoms</td>
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<tr>
<td>- Further decrease in level of consciousness</td>
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<tr>
<td>- Bulging fontanels</td>
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<tr>
<td>- Decreased spontaneous movements</td>
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<tr>
<td>- Posturing</td>
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<tr>
<td>- Papilledema</td>
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<tr>
<td>- Pupil dilation with decreased or no response to light</td>
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<tr>
<td>- Increased blood pressure</td>
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<tr>
<td>- Irregular respirations</td>
</tr>
<tr>
<td>- Cushing’s triad (late, ominous sign)</td>
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to oculocephalic testing (as you turn the patient’s head side-to-side, the eyes remain fixed and do not rotate) may indicate injury to the midbrain or pons, or that the patient is in a deep coma. An abnormal response to oculovestibular testing (after irrigating each external auditory canal with iced water, the eyes do not deviate toward the side of irrigation with nystagmus) may indicate injury to cranial nerves III, VI, or VIII, or brain stem injury. The fundus is examined for papilledema. Although the presence of papilledema indicates increased ICP, its absence does not indicate the absence of increased ICP. The fundus is also examined for retinal hemorrhages, which may indicate child abuse, sagittal sinus thrombosis, or coagulation abnormalities.

Further cranial nerve testing in the unresponsive or intubated child should include eliciting a gag or cough reflex, and assessment of swallowing. Impairment in CN IX and X can greatly affect the child’s ability to manage their oropharyngeal secretions and protect their airway. Cranial nerves and their functions are summarized in Table 6.

Motor assessment includes recognition of any abnormal posturing (eg, extension, flexion, flaccidity), muscle symmetry, strength, and tone. Flaccidity indicates severe dysfunction of the lower brain stem. Deep tendon reflexes are elicited to assess for central and peripheral nervous system dysfunction.

Abnormalities that are detected in the neurological exam must be correlated with the patient’s previous examination, diagnosis, and radiologic findings. The location of a mass lesion or hemorrhage, or presence of mass effect or hydrocephalus must be assessed immediately to accurately diagnose and respond to impending herniation.

### Radiologic Assessment

The radiologic study of choice for immediate assessment of the child with acute...
neurological impairment or deterioration is a noncontrast CT scan. A contrast CT scan may be necessary for further diagnosis of space occupying and vascular lesions, and infections, but is not warranted in the initial assessment of the child with increased ICP. On a noncontrast CT, highly dense structures, such as blood, will appear white, and low density areas, such as air, CSF, and edema, will appear black. Initial inspection of the CT scan should proceed with observation of any intracranial mass lesion, midline shift, skull fracture, presence of hemorrhage (extra-axial, intraparenchymal, or intraventricular), ventricular size, symmetry, and patency of basal cisterns (Figure 2). CT findings consistent with increased ICP include a mass lesion, intracerebral hemorrhage, midline shift, loss of sulci, ventricular effacement, and cerebral edema (Figure 3).

Neurological Monitoring

ICP monitoring is indicated for children with TBI whose GCS <8 or for children with other acute neurological injuries who have clinical signs of increasing ICP, are status post resection of acute traumatic intracranial hematoma or other major neurosurgical procedures, or have a neurodiagnostic test indicative of a high probability of increased ICP (ie, cerebral edema, midline shift, cisternal compression, intracerebral hemorrhage). Children who do not usually benefit from ICP monitoring are children with hypoxic injuries (ie, near drowning) and some central nervous system infections (ie, encephalitis).

Although ICP monitoring has never been subjected to randomized controlled studies to evaluate its effectiveness, its use has been associated with decreased morbidity and mortality, and improved outcome in patients with TBI, intracerebral hemorrhages, and CNS infections. It is not the monitor itself that improves outcome, but the information gleaned from this modality that guides appropriate interventions. ICP monitoring is crucial to identify rapidly increasing pressure and to institute appropriate therapy to prevent intracerebral herniation and preserve cerebral perfusion. It is also necessary to monitor ICP in order to calculate the CPP.

The current gold standard for ICP monitoring is intraventricular catheter placement, preferably an implantable microtransducer (fiberoptic or strain-gauge). This method allows simultaneous monitoring of ICP and management of increased ICP by CSF drainage. Other methods of monitoring ICP include intraparenchymal, subdural, subarachnoid, and epidural catheter placement—although the latter locations are very rarely used. Depending on the type of catheter, care will vary. ICP monitors with intracranial transducers are zeroed at the time of placement and therefore do not require any further zeroing. However, extracranial transducers require frequent zeroing and leveling because they must be adjusted with changes in patient position and recalibrated to atmospheric pressure.

In the past, children at risk for increased ICP were managed primarily with interventions to decrease their ICP and maintain an adequate CPP. This remains true, but the role of cerebral oxygenation and the ability to monitor it has recently been incorporated into the care of these children. Adjunct monitoring modalities utilized to prevent cerebral ischemia and optimize the patient’s outcome include transcranial doppler (TCD) ultrasonography, jugular venous oxygenation saturation (SjvO2) monitoring, brain tissue...
partial pressure oxygen monitoring (PbtO₂), non-invasive cerebral oxygenation monitoring, and brain metabolism monitoring.

As mentioned previously, CPP monitoring involves calculating the difference between MAP and ICP. CPP can also be assessed via xenon computed tomography (CT) scan, perfusion CT, perfusion magnetic resonance imaging (MRI), and positron emission tomography (PET) scanning. These modalities offer excellent information regarding regional CBF but are limited to one point in time. Continuous monitoring of CBF would be ideal and allow for interventions aimed at optimizing cerebral perfusion. For instance, if CBF were low, interventions would be directed at increasing vasopressor support or fluid therapy, or by decreasing cerebral demand by increasing sedation. If the patient were hyperemic, then methods to decrease the CBF, such as hyperventilation, would be instituted.18

CBF can also be evaluated via TCD ultrasonography, which non-invasively determines CBF velocity in the proximal vessels of the Circle of Willis. This is particularly helpful when assessing for vasospasm and stenosis in a child with a stroke. The adequacy of CBF related to cerebral metabolic demand can be assessed by SjvO₂ and PbtO₂. SjvO₂ is a continuous measurement of the oxygen saturation in the jugular vein after cerebral perfusion has occurred. The jugular mixed venous saturation is compared to the arterial oxygen saturation, and the arteriovenous oxygen content difference (AVDO₂) is then calculated. Normal SjvO₂ is 55% to 70%; <55% indicates cerebral hypoperfusion; <40% indicates cerebral ischemia, whereas >75% indicates luxury perfusion and possible cellular death. Although SjvO₂ is helpful in assessing cerebral oxygenation, a more direct approach is the measurement of partial pressure brain tissue oxygenation.

PbtO₂ is a more specific method of measuring cerebral oxygenation via a microprobe inserted directly into parenchymal tissue or the penumbra of an intracerebral lesion.
There is controversy regarding the optimal insertion area. Some advocate for placement in uninjured brain tissue to assess global cerebral oxygenation, while others opt for placement in the penumbra of an intracerebral lesion to obtain information of cerebral oxygenation for the area most at risk. Ideally, either method allows for continuous monitoring of cerebral oxygenation and early recognition of cerebral ischemia. The various types of catheters currently available (e.g., Neurotrend [Diametrics Medical Limited, St. Paul, MN], LICOX [Integra Life Sciences, Plainsboro, NJ]) may yield different values, and interventions should be adjusted accordingly. PbtO$_2$ is a more specific method of measuring cerebral oxygenation via a microprobe inserted directly into parenchymal tissue or the penumbra of an intracerebral lesion. There is controversy regarding the optimal insertion area. Some advocate for placement in uninjured brain tissue to assess global cerebral oxygenation, while others opt for placement in the penumbra of an intracerebral lesion to obtain information of cerebral oxygenation for the area most at risk. Ideally, either method allows for continuous monitoring of cerebral oxygenation and early recognition of cerebral ischemia. The various types of catheters currently available (e.g., Neurotrend [Diametrics Medical Limited, St. Paul, MN], LICOX [Integra Life Sciences, Plainsboro, NJ]) may yield different values, and interventions should be adjusted accordingly. Using the LICOX catheter, normal PbtO$_2$ values in uninjured parenchyma are $\geq 20$ mm Hg and interventions to improve cerebral oxygenation are indicated if the value is $< 15$ mm Hg. For non-injured brain tissue, normal values are between 20 to 35 mm Hg. A decrease in partial pressure of brain tissue oxygen can be seen with decreased PaCO$_2$, hypoxemia, increased ICP, and decreased CPP. Most of the available research on the utility of brain

**Figure 3.** Abnormal brain computed tomograph scan. A 16-year-old status/post MVA; note right occipital EDH; mass effect with right to left shift, uncal herniation; multiple parenchymal hemorrhagic contusions. MVA, motor vehicle accident; EDH, epidural hematoma.
tissue oxygenation is from adult TBI patients. There are a few studies from adult patients with brain tumors, arteriovenous malformations, and stroke demonstrating that the additional information obtained from this modality is beneficial. A decrease in partial pressure of brain tissue oxygen can be seen with decreased PaCO₂, hypoxemia, increased ICP, and decreased CPP. Most of the available research on the utility of brain tissue oxygenation is from adult TBI patients. There are a few studies from adult patients with brain tumors, arteriovenous malformations, and stroke demonstrating that the additional information obtained from this modality is beneficial.

Noninvasive measurement of cerebral oxygenation can be done by near-infrared spectroscopy, which measures cerebral oxygen saturation via oximetry. This method has the advantage of being noninvasive and portable, but it is not commonly used in pediatrics.

Brain metabolism monitoring provides an individual assessment of brain chemistry after injury to guide treatment accordingly. Currently, it is not routinely used in most PICUs, but would allow for measurement of neurochemicals (i.e., lactate, pyruvate, glucose, glutamate, urea, and glycerol) via an intraparenchymal microdialysis catheter.

Management of Increased ICP

Despite the etiology of the primary neurological injury, the main focus of management is to prevent and minimize secondary injury. Although primary injury and secondary injury most commonly refer to children with TBI, this terminology can also be applied to children with metabolic or hypoxic-ischemic encephalopathy and children with nontraumatic primary brain lesions, infections, and intracranial hemorrhage. The primary injury refers to the initial insult regardless of the mechanism of injury or illness. The secondary brain injury refers to the processes that occur within hours to days after the primary injury that can be prevented or minimized—such as cerebral ischemia, cerebral edema, and neurochemical alterations. Mitigating factors known to worsen secondary injury are hypoxia and hypotension. Other factors that may worsen secondary brain injury after TBI include the release of excitatory neurotransmitters, the formation of free radicals, and increased levels of intracellular calcium and potassium. The neurological devastation caused by the secondary injury is often worse than the underlying primary disorder. Therefore, management is directed at prevention of secondary injury.

Recently, recommendations on the management of increased ICP in children with TBI have been published. These recommendations are based on pediatric studies and extrapolated from adult TBI research. Since there are limited outcome studies to support the current management of children with increased ICP from etiologies other than TBI, this management is often instituted for these children also. A schematic of increased ICP management is provided in Figure 4. The reader is also referred to the “Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents” for an algorithm detailing escalation of therapy.

ICP management based on the Monro-Kellie doctrine includes interventions to decrease cerebral volume, control CSF volume, control CBV, and decrease cerebral metabolic rate (Table 7). The goal of these therapies is to maintain an age-appropriate CPP and an ICP < 20 mm Hg or as appropriate for age. In adults with TBI, the maintenance of CPP > 70 mm Hg is used in some centers based on research demonstrating that increasing the CPP, rather than decreasing the ICP, improves patient outcomes. However, there are limited data on the benefits of CPP-driven management in pediatrics, and it is not routinely implemented.

Airway, Breathing, and Circulation

The initial management of the child with suspected increased ICP is assessment of airway, breathing, and circulation (ABCs). Even prior to a thorough neurological exam, if the patient is unarousable or having difficulty maintaining a patent airway, rapid sequence intubation should occur. Endotracheal intubation should also be considered if the child has a GCS < 8, a CT scan consistent with diffuse cerebral edema, neurological injury at risk for decompensation, chest wall...
Figure 4. Increased intracranial pressure (ICP) algorithm. Continual assessment of neurological and cardiorespiratory status is imperative. Obtain head computed tomography (CT) if ICP continues to increase despite escalating therapies. GCS, Glasgow Coma Scale score; HOB, head of bed; NMB, neuromuscular blockade; PaCO₂, partial pressure of arterial carbon dioxide; SjvO₂, jugular venous oxygen saturation; CSF, cerebrospinal fluid; PbtO₂, partial pressure of brain tissue oxygen.
Table 7: Management of Increased Intracranial Pressure

Goals of Management | Interventions
--- | ---
Decrease cerebral volume | • Evacuation of mass lesion
| • Maintain euvolemia
| • Hyperosmolar therapy
Decrease CSF volume | • Ventriculostomy to drain CSF
| • +/- lumbar drain
Decrease cerebral blood volume | • Maintain head midline with head of bed 30°
| • Maintain normocarbia
| • Consider mild hyperventilation if refractory intracranial hypertension
Decrease metabolic rate | • Normothermia
| • Sedation and analgesia
| • Seizure management and prophylaxis
| • For refractory intracranial hypertension
  - barbiturate therapy
  - mild/moderate hypothermia

CSF, cerebrospinal fluid.

Instability, abnormal respiratory pattern, loss of protective airway reflexes, or upper airway obstruction. Intubation should proceed with administration of medications to blunt the ICP during the procedure. Suggested medications to facilitate the intubation without increasing the ICP are thiopental, lidocaine, and a short-acting, nondepolarizing neuromuscular blockade agent (e.g., vecuronium, atracurium).

Adequate oxygenation is necessary to prevent the sequelae of secondary insults and should be maintained with a PaO2 > 60 mm Hg, an oxygen saturation > 90%, and physiologic positive end expiratory pressure (PEEP) of 5 cm H2O. Increasing the PEEP to 10 cmH2O has not been associated with a worse neurological outcome. The deleterious effects of hypoxemia are more significant than the possible venous congestion caused by PEEP > 5 cm H2O.25,26

Age-appropriate blood pressure must also be maintained or restored to ensure adequate CPP and prevent further ischemia. The avoidance of hypotension is crucial since it has been associated with increased mortality in children with TBI.31,27-30 Hypotension in children is defined as systolic blood pressure below the fifth percentile for age or by clinical signs of shock.3 Median systolic blood pressure (50th percentile) can be estimated by using the following formula for children greater than 1 year of age: 90 + (2 x age in years).31 Children in hypovolemic shock can compensate fairly well and may not become hypotensive until they become profoundly hypovolemic. Other signs of decreased perfusion and shock include tachycardia, decreased urine output (<1 mL/kg/hr), weak/thready/absent peripheral pulses, prolonged capillary refill time (>2 seconds), and/or decreased mentation. Fluid boluses are administered as needed to the hypotensive child. The neurologically injured child must be fluid resuscitated the same as any other child presenting in shock. There is no indication for fluid restriction. Vasopressor support is initiated if the child remains hypotensive despite appropriate fluid resuscitation.

Positioning

The patient’s head is positioned midline to encourage jugular venous drainage and the head of the bed is elevated to 15° to 30°. These methods have been found to be effective in decreasing intracranial pressure and optimizing CPP in adult patients with TBI.32-34 Both increasing the head of the bed beyond 30° and decreasing the head of the bed below 15° have been associated with increased ICP and/or decreased CPP.33,35 In another study,36 CPP was found to be optimal with the bed in a flat position; however, ICP increased. A study of adult patients with cerebral lesions (i.e., tumors, infections) recommended supine flat position due to a linear decrease in CBF with head of bed increased to 45°.37 However, if ICP was elevated and CBF was normal or increased, positioning the head of bed 30° was recommended.37

Based on the available literature, the child’s head should be maintained midline to prevent impairment in drainage from the external jugular veins and the head of bed should be maintained at 30° with alterations based on the child’s response. The child must be euvolemic prior to placing in this position to avoid orthostatic hypotension.
Cerebrospinal Fluid Drainage

CSF drainage provides an immediate, but transient, decrease in ICP due to reduction in CSF.\textsuperscript{38} Depending on the patient’s status, it can be done either continuously or intermittently. The optimal method of continuously monitoring ICP and draining CSF simultaneously is via a ventriculostomy catheter. There is limited research on the effect of CSF drainage on ICP, CPP, CBF, and metabolic rate. One pediatric study\textsuperscript{39} revealed that children undergoing ventricular drainage had decreased ICP and improved outcome. Another study\textsuperscript{40} in adults compared ventricular drainage to mannitol and found that both treatments decreased ICP.

For refractory intracranial hypertension, lumbar drainage may be considered if the basal cisterns are open, and there is no evidence of midline shift or a significant mass lesion on neuroimaging.\textsuperscript{41}

Osmotic Therapy

Mannitol has been a cornerstone of intracranial hypertension management for decades, but has not been compared to a placebo in randomized controlled studies. Its efficacy has been evaluated by comparison to other existing therapies for intracranial hypertension. When mannitol was compared to CSF drainage, it was found to be more effective in the management of increased ICP in adults with TBI.\textsuperscript{40} Boluses of mannitol were also found to be more effective than a continuous drip.\textsuperscript{42} Some have argued that administration of mannitol on a standard dosing regimen (eg, every 2 hours), instead of as needed for ICP \textgtr 25 mm Hg, provides better ICP control.\textsuperscript{43}

Mannitol has two mechanisms of action: (1) an osmotic diuretic that produces an osmotic gradient, drawing fluid from the brain tissue into the vascular space to be excreted via kidneys and (2) a rheological effect that decreases blood viscosity and hematocrit and increases CBF and cerebral O\textsubscript{2} delivery.\textsuperscript{22} The rheologic effect occurs immediately and is responsible for decreased ICP within minutes of administration. This occurs due to increased plasma volume, which decreases blood viscosity and hematocrit, and increases CBF and cerebral oxygen delivery that leads to vasoconstriction and decreased ICP. The osmotic diuresis develops more slowly and lasts up to 6 to 8 hours. As such, mannitol is usually given as a bolus every 4 to 6 hours since its osmotic effect occurs 15 to 30 minutes after administration and its duration of action is 2 to 8 hours. Autoregulation of CBF must be intact for the effects of mannitol to work; however, autoregulation is often disrupted in brain injured patients.

Recently hypertonic saline (HS) administration has been shown to decrease ICP and improve outcome in pediatric TBI patients.\textsuperscript{44-47} Hypertonic solutions have the advantage of decreasing ICP and decreasing cerebral volume by producing an osmotic gradient in the brain, while also supporting intravascular volume. Pediatric studies in children with TBI have found that HS is effective in decreasing ICP and improving CPP with no adverse reactions, including children with refractory intracranial hypertension.\textsuperscript{44-47} Hypertonic saline has also been reviewed in children with diabetic ketoacidosis and presumed cerebral edema. The use of 10 mL/kg bolus dosing of HS was found to correlate with an improvement in the child’s GCS and level of consciousness.\textsuperscript{48} HS is usually administered as a continuous infusion, starting at 0.1 to 1.0 mL/kg/hr. Serum sodium and neurological status must be closely monitored during therapy due to the possibility of osmotic demyelination syndrome (central pontine myelinosis), which can occur with a rapid increase in serum sodium.\textsuperscript{49} This destruction of the white matter, often to the pons, can cause significant neurological devastation due to rapid osmotic shifts. In addition, the formation of organic osmolytes (idiogenic osmoles), which are osmotically active molecules that accumulate intracellularly in the brain during hyperosmolar states to maintain cerebral volume and prevent cellular dehydration, may contribute to cerebral edema upon abrupt discontinuation of hyperosmolar therapy. As such, when HS therapy is no longer indicated, serum sodium should be slowly corrected to normal values (0.5 to 1.0 meq/L/hour) to avoid complications associated with osmotic shifts. Other potential complications of HS therapy include rebound increase in
ICP\textsuperscript{50} and renal insufficiency.\textsuperscript{44} In addition to decreasing cerebral edema, advantages of HS may include improved hemodynamic status by expansion of plasma volume, as well as vasoregulatory effects and immune modulation that may contribute to prevention of secondary injury.\textsuperscript{49}

Regardless of which osmotic therapy is instituted, the patient must be maintained euvoletic with an indwelling catheter measuring urine output throughout therapy. Maintenance and bolus intravenous fluids are administered to maintain euvolemia. Serum osmolality must also be monitored to prevent renal tubular dysfunction. With mannitol, the maximum recommended serum osmolality is 320 mOsm/L. However, a serum osmolality of 365 mOsm/L has been well tolerated in children receiving HS.\textsuperscript{44,45} It is unclear whether this disparity is due to inherent differences between mannitol and HS, current management aimed at euvolemia versus fluid restriction, or differences between adults and children with regard to nephrotoxicity susceptibility.\textsuperscript{53} Recently, some concern was expressed regarding increased renal dysfunction when serum sodium is $>160$ meq/L in euvoletic children with intracranial hypertension;\textsuperscript{51} however, these findings have not been reported in randomized controlled trials.

**Ventilation**

In the past, prophylactic hyperventilation was a mainstay of therapy for increased ICP management. It was thought that children after TBI were hyperemic, which led to cerebral edema and increased ICP and that hyperventilation decreased CBF and improved outcome.\textsuperscript{52} However, the role of hyperemia was challenged when a study\textsuperscript{2} revealed that children normally have higher resting CBF than adults, not just children after TBI. Further studies\textsuperscript{3,53} revealed that children may actually have decreased CBF after TBI.

Hyperventilation was employed to decrease ICP by causing vasoconstriction, which decreased CBF and CBV. However, recent studies have demonstrated that aggressive hyperventilation dramatically decreases CBF, which may give rise to or exacerbate cerebral ischemia, thus enhancing rather than decreasing secondary injury.\textsuperscript{54–57} Patients that have received hyperventilation have also been found to have a worse outcome.\textsuperscript{58,59}

Hyperventilation is currently only recommended in the initial management of increased ICP for acute, significant increases in ICP.\textsuperscript{60,61} It is not recommended for prophylactic treatment of increased ICP because of the potential for worsening cerebral ischemia. Furthermore, it depletes the brain tissue interstitial bicarbonate buffering capacity, which may lead to a loss of local vasoconstrictor effects.\textsuperscript{62} Normally, alkalosis causes arteriolar constriction, but with loss of this buffer system, it can no longer respond appropriately and may not cause vasoconstriction to decrease CBF. Mild hyperventilation ($\text{PaCO}_2$ 30 to 35 mm Hg) may be implemented if increased ICP continues despite CSF drainage, appropriate sedation and analgesia, head position, and osmolar therapy. Significant hyperventilation ($\text{PaCO}_2 < 30$ mm Hg) should be reserved for patients with refractory intracranial hypertension unresponsive to initial therapies.\textsuperscript{58} Intermittent hyperventilation should be instituted for acute, sharp increases in ICP or signs of impending herniation.\textsuperscript{63,64}

**Temperature Regulation**

Maintaining the patient normothermic ($T = 37^\circ\text{C}$) is the goal to prevent complications of both hypothermia ($T < 35^\circ\text{C}$) and hyperthermia ($T > 38.5^\circ\text{C}$). Patients who have acute neurological injury may have temperature fluctuations due to infection, sepsis, intracranial blood, and hypothalamic disturbances. A core temperature greater than 37.5\textdegree C is associated with increased ICP and increased cerebral metabolic rate of oxygen. In experimental models of brain injury, hypothermia has been found to be neuroprotective by decreasing cerebral metabolism, extracellular glutamate release, mobilization of calcium, production of free radicals, and nitric oxide synthesis.\textsuperscript{65} However, in studies of adults with TBI, hypothermia has been associated with increased systemic complications and no improvement in patient outcomes.\textsuperscript{66–68} Potential side effects of hypothermia include increased risk of pneumonia, skin breakdown, electrolyte imbalances, hypotension, coagulopathy, and shivering.

Given the potential benefits of hypothermia, moderate hypothermia ($33^\circ\text{C}$) has been
researched for its role in the management of increased ICP. Adult studies have shown that moderate hypothermia may improve outcome. Recently, very mild hypothermia (35–35.5 °C) was studied in adult head injured patients and was found to decrease intracranial hypertension and maintain sufficient CPP. This study also found that decreasing the body temperature below 35 °C did not improve intracranial hypertension and actually decreased CPP. A recent pediatric study instituted moderate hypothermia within 6 hours of TBI and continued it for 48 hours. This study demonstrated a decrease in the severity of intracranial hypertension and was tolerated without any side effects, although there was no significant difference in mean ICP and CPP.

Children with increased ICP should be normothermic with interventions aimed at prevention of fever and shivering. Ideally, core or brain temperature is measured. Core temperatures can be measured via a Swan Ganz catheter or a bladder or rectal probe. Brain temperature can be monitored via SjvO2 or PbtO2 catheters. Moderate hypothermia is reserved for children with refractory intracranial hypertension not responsive to initial therapies. Upon discontinuation of hypothermia treatment, the patient must be rewarmed slowly to prevent complications, such as electrolyte imbalances (particularly potassium), an increase in cerebral edema, acidosis, and hypotension.

**Sedation, Analgesia, Neuromuscular Blockade**

Children with acute brain injury who are mechanically ventilated should be appropriately sedated and receive adequate pain management to prevent pain and anxiety, which will increase the cerebral metabolic rate and ICP. There are no randomized controlled studies comparing sedation methods in children with acute neurological injury. The most common agents used are opioids, benzodiazepines, and/or barbiturates. Administration of neuromuscular blockade agents (NMB) may be necessary to facilitate mechanical ventilation and control PaCO2, prevent shivering, and prevent movement that may increase ICP. Use of NMB will limit physical examination findings of increased ICP to assessment of the pupillary response; no other assessments will be accurate while the patient is pharmacologically paralyzed. An ICP monitor is usually necessary in these patients. Additional monitoring may include the Bispectral Index (BIS) to provide an objective measure of cerebral electrical activity and assign a numerical value to the level of sedation.

Since the use of NMB will eliminate motor activity associated with seizures, but not brain epileptiform activity, children at high risk for seizures may require continuous electroencephalograph (EEG) monitoring. Indications for NMB must be weighed against obscuring the neurological examination, as well as a possible increase in patient complications. In a study of adults with TBI, the use of NMB was associated with increased intensive care unit (ICU) length of stay and nosocomial pneumonia.

**Seizure Prevention and Management**

Children who have sustained a significant head injury are more likely than adults to have seizures, possibly due to their lower seizure threshold. Patients with GCS < 8 have an increased risk of early seizures after TBI. If the child presents with seizure activity, initial treatment with a benzodiazepine (eg, lorazepam) and/or phenytoin (or fosphenytoin) should be instituted, followed by anti-epileptic medication (eg, phenytoin, fosphenytoin) for at least 2 weeks. The etiology of the seizure (ie, temporal lobe tumor, intraparenchymal bleed) and the patient’s clinical status will ultimately determine the duration of antiepileptic treatment.

Children who have significant parenchymal injury after TBI or are less than one year old are at increased risk of seizure activity and should be considered for seizure prophylaxis to prevent early posttraumatic seizures. In children with TBI, seizures most commonly occur within the first 24 hours after injury. Regardless of the seizure etiology, seizure activity must be terminated immediately in the child with increased ICP. Seizures increase the metabolic rate, which will increase CBF and CBV leading to an increase in ICP. If the metabolic demands exceed the supply, cerebral ischemia will ensue, leading to irreversible neurological damage.

There is limited research on the use of anti-epileptic medications after traumatic
brain injury. Studies in adults have shown that anti-epileptic medications for long-term prophylaxis did not improve outcome and are only effective in the first week after TBI to decrease the incidence of early onset seizures. In children, anti-epileptic medications are recommended for short-term use, unless deemed otherwise by the child’s seizure activity.

**Barbiturate Therapy**

The administration of high dose barbiturates (e.g., pentobarbital) is reserved for intracranial hypertension refractory to the aforementioned interventions. Barbiturates decrease ICP by decreasing cerebral metabolic rate, which decreases glucose utilization and oxygen demand, but may cause significant hemodynamic instability. Although there are limited pediatric studies examining barbiturate effectiveness and role in outcome, some studies have shown that intractable ICP may improve and in some lead to a good outcome. However, many patients require vasopressor support during therapy. Adult studies have also reported pentobarbital as an effective treatment to decrease ICP. However, some have shown no benefit in outcome, while others have shown an association with worse neurological outcome due to jugular venous desaturation.

Monitoring of the child in a barbiturate coma should include burst suppression via EEG, invasive hemodynamic monitoring (e.g., arterial blood pressure, central venous pressure, SvO2), and frequent assessment of oxygenation status. Because of the significant side effects of barbiturate therapy and the limited data supporting its use, barbiturate therapy is only recommended for children with refractory intracranial hypertension.

**Surgical Management**

Initial surgical management includes the immediate evacuation of a mass lesion (e.g., brain tumor, epidural hematoma) and placement of ICP monitor as indicated, preferably a ventriculostomy. Decompressive craniectomy to increase intracranial compliance is reserved for patients with refractory intracranial hypertension, although some studies have advocated its use early after TBI before secondary injury occurs.

**Steroids**

The use of corticosteroids is not indicated in the management of increased ICP after TBI. However, steroids may be indicated in decreasing swelling and stabilizing the cell membrane in patients with increased ICP due to mass lesions, such as brain tumors and abscesses, inflammation, and infections.

**Fluids, Electrolytes, and Nutrition**

The main goals of fluid therapy are to maintain the patient euovolemic, normoglycemic, and prevent hyponatremia. In a recent pediatric study of 170 children with TBI, mean serum glucose >200 mg/dL was associated with poor neurological outcome, and serum glucose >300 mg/dL on admission was associated with 100% mortality. Although the exact mechanism of hyperglycemia after TBI is multifactorial and not completely understood, the detrimental effects of hyperglycemia on the injured brain have been repeatedly demonstrated. Current treatment in adults with TBI includes stringent glycemic control with insulin therapy.

Parenteral dextrose is avoided for at least 48 hours after acute neurological injury due to the increased risk of lactic acidosis, unless the patient is hypoglycemic—which requires prompt treatment. Enteral feedings may and should be instituted within 72 hours after injury. Early enteral feeding in adult patients with TBI has been associated with a dramatic decrease in ICU days and complications. Children with TBI have been found to have caloric needs 30%-60% greater than their basal metabolic expenditure and should be supplemented accordingly with the appropriate formula and rate. Unless contraindicated, the preferred method of delivering nutrition is via the enteral route. Due to potential impairments in the protective airway reflexes, it may be prudent to administer feedings via a postpyloric feeding tube to decrease the risk of aspiration.

Children with increased ICP should receive fluids at a daily maintenance rate, as well as fluid boluses as indicated for hypovolemia, hypotension, or decreased urine output. Since dextrose is usually avoided for the first 48 to 72 hours after injury, maintenance fluids usually consist of normal saline with
the daily requirements of potassium chloride based on body weight. All fluids administered must be isotonic or hypertonic (eg, hypertonic saline) and hypotonic fluids must be avoided. In addition, hyponatremia must be prevented since it will worsen cerebral edema. If hyponatremia occurs, it must be corrected slowly to prevent central pontine myelinosis that can occur with rapid correction of sodium.

Nursing Care

In addition to the nursing and medical interventions outlined above, care of the child and family is an important component in providing optimal patient care. In pediatrics, the child is cared for within the context of the family, and the child with increased ICP is no exception. It is important to encourage the caregivers and siblings (as appropriate) to touch and speak to the child. Studies in children and adults have found that ICP did not increase significantly or actually decreased with family presence or with physical touch. During these interactions, the child’s ICP and hemodynamic status are monitored and care modified accordingly for any significant change.

There is limited research on whether clustering patient care activities (ie, bathing, suctioning, procedures, etc.) or providing rest periods in between activities is more beneficial to the patient. It is recommended to monitor the patients ICP, MAP, heart rate, central venous pressure, and CPP and continue or abort care accordingly. Additional sedation and analgesia should also be provided prior to any procedure that may cause pain, anxiety, or increased ICP, although the research in this area is lacking.

Research on the use of lidocaine prior to endotracheal suctioning to attenuate the ICP response in pediatric patients is scant and dated. Based on the available research, lidocaine (via endotracheal tube or intravenously) is usually recommended prior to suctioning to blunt the ICP response. One study demonstrated a more significant suppression of ICP elevation with 2 mL of 4% lidocaine administered intratracheally due to its anesthetic effect on the tracheobronchial mucosa. Lidocaine 1.5 mg/kg is the recommended dose for IV administration prior to suctioning. Regardless of the method of administration, the patient’s response must be monitored and care adjusted accordingly.

□ Summary

Neuro-critical care of children with increased ICP revolves around the basic tenet of the Monro-Kellie doctrine and aims to decrease one or more of the intracranial compartments. The goal of management is to control CSF volume, brain volume, and blood volume. This control is achieved by

<table>
<thead>
<tr>
<th>TABLE 8</th>
<th>Summary of Intracranial Pressure Management</th>
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<tbody>
<tr>
<td>• Maintain adequate oxygenation: PaO₂ &gt; 60; SpO₂ &gt; 90%</td>
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<tr>
<td>• Maintain adequate systemic perfusion—prevent hypotension and hypoxia</td>
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<tr>
<td>• Maintain age appropriate ICP and CPP; or ICP &lt; 20 and CPP &gt; 50–70</td>
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<tr>
<td>• Drain CSF as indicated</td>
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<tr>
<td>• Positioning: maintain supine with head of bed ~30° and head midline</td>
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<tr>
<td>• Review CT scan and neurodiagnostic results as indicated</td>
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<td>• No indication for prophylactic hyperventilation</td>
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<tr>
<td>• Hyperventilation—only for acute ICP elevations or impending herniation</td>
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<tr>
<td>• Order lidocaine prior to suctioning</td>
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<tr>
<td>• Mannitol q4h for ICP &gt; 20 mm Hg; maintain serum osmo &lt; 320 mOsm</td>
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<tr>
<td>• Consider hypertonic saline; maintain serum osmo &lt; 360 mOsm/L</td>
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<tr>
<td>• Maintain euvoolemia</td>
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<td>• Monitor electrolytes; prevent hyponatremia</td>
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<tr>
<td>• Monitor glucose—prevent hyperglycemia and hypoglycemia</td>
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<tr>
<td>• Maintain normothermia—for every change by 1°C, ICP increases several mm Hg</td>
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<tr>
<td>• Provide adequate sedation and analgesia; consider neuromuscular blockade</td>
<td></td>
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<tr>
<td>• Monitor for seizure activity; order anti-epileptics as indicated</td>
<td></td>
</tr>
<tr>
<td>• Institute enteral feedings within 72 hours after injury</td>
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<tr>
<td>• Frequent neurological assessment: Glasgow Coma Scale, pupil reactivity, extraocular movements, motor function, vital signs</td>
<td></td>
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<tr>
<td>• Minimize auditory and visual stimulation</td>
<td></td>
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<tr>
<td>• Encourage family touch and patient contact</td>
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<tr>
<td>• Provide patient and family support and education</td>
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<tr>
<td>• Consider 2nd-tier therapy for refractory intracranial hypertension</td>
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</tbody>
</table>

PaO₂, continuous arterial oxygen saturation; ICP, intracranial pressure; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; CT, computed tomography.
proper patient positioning, normocarbia, CSF drainage, euvoletic osmolar therapy, sedation and analgesia, optimal cardiorespiratory status, normothermia, and seizure prophylaxis based on the patient’s condition and response (Table 8). Implementation of hyperventilation, moderate hypothermia, and barbiturate therapy is reserved for patients with refractory intracranial hypertension. The best neurological outcome is associated with control of ICP < 20 mmHg (or age appropriate) and a CPP > 50 (or age appropriate), as well as prevention of hypoxia and hypotension. Much research is still needed to optimize management strategies for children with acute increased intracranial hypertension, particularly nursing interventions that may ultimately improve patient outcomes.

References


76. Pittman T, Bucholz R, Williams D. Efficacy of


