

Emergency Management of Increased Intracranial Pressure

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Abstract: Primary neurological injury in children can be induced by diverse intrinsic and extrinsic factors including brain trauma, tumors, and intracranial infections. Regardless of etiology, increased intracranial pressure (ICP) as a result of the primary injury or delays in treatment may lead to secondary (preventable) brain injury. Therefore, early diagnosis and aggressive treatment of increased ICP is vital in preventing or limiting secondary brain injury in children with a neurological insult. Present management strategies to improve survival and neurological outcome focus on reducing ICP while optimizing cerebral perfusion and meeting cerebral metabolic demands. Targeted therapies for increased ICP must be considered and implemented as early as possible during and after the initial stabilization of the child. Thus, the emergency physician has a critical role to play in early identification and treatment of increased ICP. This article intends to identify those patients at risk of intracranial hypertension and present a framework for the emergency department investigation and treatment, in keeping with contemporary guidelines. Intensive care management and the treatment of refractory increases in ICP are also outlined.

Key Words: intracranial hypertension, neurotrauma, head injury, ICP management

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TARGET AUDIENCE

This article is intended for clinicians caring for acutely ill children with increased ICP, including emergency physicians, pediatricians, pediatric emergency physicians, pediatric critical care physicians, anesthesiologists, and medical trainees.

LEARNING OBJECTIVE

After completion of this CME article, the reader should be better able to:

1. Assess situations requiring acute management of increased ICP.

Patients presenting to the emergency with symptomatic increases in intracranial pressure (ICP) constitute a medical emergency. Rapid deployment of therapies to maintain cerebral perfusion is critical to patient outcome. An understanding of the physiology of increased ICP allows the clinician to pursue

appropriate therapies that improve both survival and neurological outcome. The mainstay of therapy for patients with increased ICP is the maintenance of optimal hemodynamic and respiratory support to ensure adequate oxygenation and perfusion of the brain. A rapid clinical assessment facilitates recognition of elevated ICP and prompt initiation of targeted therapies against increased ICP. Treatment of increased ICP should follow a stepwise approach, such that highly effective, low-risk interventions precede those with higher-risks and dubious efficacy.

Physiology of Increased ICP

Normal values for ICP increase with age, from approximately 6 mm Hg in infants to 10 to 15 mm Hg in adults.¹ Intracranial pressure is determined by the total force exerted by the brain, blood, and cerebrospinal fluid contained within the fixed volume of the skull. Compensatory mechanisms exist whereby an increase in the volume of 1 intracranial component will cause a decrease in the volume of the remaining 2 to maintain a normal ICP (the Monro-Kellie doctrine). Once the limits of compensation are exceeded, ICP rises.² An open fontanelle and sutures is not wholly protective and may be associated with pathological increases in ICP. Increased ICP may cause cerebral ischemia by directly decreasing cerebral perfusion pressure and hence cerebral blood flow (CBF) to critical levels in keeping with the equation:

$$\text{Cerebral Perfusion Pressure} = \text{Mean Arterial Pressure} - \text{Intracranial Pressure}$$

Therefore, an increase in ICP will cause a decrease in CBF unless there is a corresponding increase in mean arterial blood pressure. When ICP increases or arterial blood pressure decreases, cerebral perfusion pressure falls below a critical value resulting in compromised CBF and brain ischemia (Fig. 1), leading to a cascade of deleterious events. These events are forestalled by cerebral autoregulation, a protective mechanism that normally maintains CBF over a mean arterial pressure range of 50 to 150 mm Hg in response to changes in ICP. Impairment of cerebral autoregulation exacerbates cerebral ischemia and hypoxia during periods of increased ICP associated with hypotension.^{3,4} Secondary brain injury occurs as a result of an endogenous cascade of inflammatory and excitotoxic mediators and enhanced vulnerability of the injured brain to physiologic changes.⁵ It is therefore important to maintain adequate CBF to satisfy metabolic demands. Reduction of ICP is a valuable strategy available to the clinician because manipulation of mean arterial pressure is only partially effective in the presence of increased ICP.

Assessment of ICP

The history and physical examination are crucial to the assessment of a patient with suspected elevated ICP. Victims of traumatic brain injury have an obvious history suggestive of elevated ICP, whereas in nontraumatic causes, such as shunt blockage, the history may be vague. Hence, the signs of increased ICP can range from subtle to obvious depending on etiological factors and whether it is acutely or chronically elevated. Although

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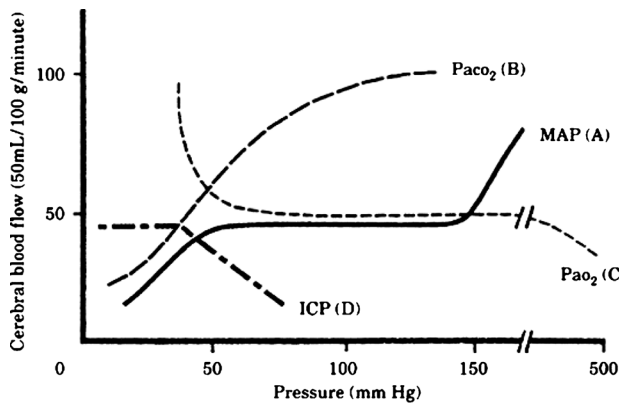


FIGURE 1. The relationships between CBF and mean arterial blood pressure (A), PaCO₂ (B), PaO₂ (C), and ICP (D). Available at: <http://www.neuroicu.info/supratentorialtumors.htm>.

relatively small acute increases in ICP frequently cause obvious symptoms, large chronic increases can present with vague symptoms. The symptoms and signs provide evidence about the adequacy of compensatory mechanisms and how well the increased ICP is being tolerated. Historical features, symptoms, and signs suggestive of increased ICP are listed in Table 1. A suspicion of increased ICP requires urgent head imaging with computed tomography (CT). However, initial stabilization supersedes any investigation and the presence of herniation syndromes (Table 2) should prompt treatment of increased ICP before radiographic investigations.

ACUTE MANAGEMENT OF PATIENTS WITH SUSPECTED OR ACTUAL INCREASED ICP

Initial Resuscitation

A primary assessment of airway, breathing, and circulation must be completed for all patients presenting with potential neurological insults. It is imperative that prompt treatment of the major aggravators of secondary injury, particularly hypotension and hypoxemia, be administered.

Airway protection must be ensured for patients with an altered level of consciousness. Patients with a Glasgow Coma Scale (GCS) score less than 8 require invasive airway support. Normal oxygenation and ventilation must be maintained (oxygen saturation, >95%; PaCO₂, 35–40 mm Hg). Poor ventilation resulting in hypercarbia (PaCO₂, >45 mm Hg) causes cerebral vasodilation and a secondary increase in ICP.¹ Conversely, a recent retrospective review showed that severe hypocarbia (PaCO₂, <30 mm Hg) occurs more frequently during the initial

TABLE 1. Historical Features, Symptoms, and Signs of Increased ICP

Historical Features	Symptoms	Signs
History of trauma	Headache	Ataxia
Previous VP shunt	Diplopia	Seizures
Bleeding diathesis	Nausea	Pupillary asymmetry
Morning vomiting		Decreased LOC
Nocturnal headache		
Developmental regression		

LOC indicates level of consciousness.

TABLE 2. Herniation Syndromes

Herniation Syndrome	Pathophysiology	Clinical Signs
Transtentorial herniation	Brain traverses the tentorium at the level of the incisura	Ipsilateral papillary dilation ± Contralateral hemiparesis
Foramen magnum herniation	Extracranial herniation of brain (cerebellar tonsils, brainstem) through the foramen magnum	Depressed LOC Cushing triad (hypertension, bradycardia, irregular respirations)

LOC indicates level of consciousness.

resuscitation and has been independently predictive of mortality, likely as a result of severe vasoconstriction and the inability of CBF to meet metabolic needs.⁶

Maintenance of a systolic blood pressure greater than the fifth percentile for age is essential. Hypotension during the initial resuscitation is the most critical factor influencing survival for patients with traumatic brain injury. Hypotension requires aggressive fluid resuscitation with crystalloids because there is a greater association between poor neurological outcome and hypotension occurring in the first 6 hours after traumatic brain injury than with hypotension at any other time during recovery.⁷

Seizure Control

Both convulsive and nonconvulsive seizures increase cerebral metabolic rate, thereby precipitating increased CBF and increased ICP. Prompt recognition and treatment of seizures is important in preventing secondary injury. Acute seizure activity is managed in the routine fashion, paying close attention to the possible adverse hemodynamic and ventilatory effects of anti-seizure medications. Adult evidence supports the prophylactic use of phenytoin to decrease the incidence of early posttraumatic seizures.⁸ There is no current evidence to support prophylactic use of antiseizure medication in children⁹; however, it may be prudent to administer a loading dose of long-acting anticonvulsants if transport or delayed definitive care is anticipated.

Sedation and Analgesia

Judicious use of sedatives and analgesics prevents transient increases of ICP caused by pain and agitation. The level of sedation must be balanced to maintain blood pressure and, if possible, facilitate assessment of neurological status. Neuromuscular blockade may be necessary to minimize metabolic demand.

ICP Specific Therapies

Hyperventilation

Although hyperventilation is not routinely recommended, its short-term use for patients with active evidence of cerebral herniation is considered acceptable emergency management. End-tidal CO₂ targets should be maintained between 25 and 30. An immediate neurosurgical consultation must be obtained.

Hyperosmolar Therapies

Both mannitol and hypertonic saline can be given as a bolus if signs of acute neurologic deterioration or cerebral herniation are present. Prophylactic use of hyperosmolar therapy is not recommended and, in the emergency situation, should only be

administered after consultation with neurosurgical or critical care services.

Mannitol results in an early effect of cerebral vasoconstriction (caused by decreased blood viscosity) and a more sustained reduction in cerebral edema through induction of an osmotic gradient.¹⁰ The typical dosage is 0.25 to 1 g/kg of 20% mannitol given over 10 to 20 minutes. Caution must be exercised particularly in polytrauma patients, where the ensuing osmotic diuresis can rapidly cause hypovolemia and hypotension. Diuresis-induced hypovolemia should be corrected by rapid administration of normal saline boluses.

Hypertonic saline causes similar vascular changes to mannitol, and has the added benefit of expanding intravascular volume, which may supplement CPP.¹¹ The typical dosage is 5 to 10 mL/kg of 3% hypertonic saline solution given over 5 to 10 minutes.

Initial Imaging

After initial stabilization, imaging should aim to identify intracranial pathology that requires emergency surgery. An unenhanced head CT is the test of choice in the emergency evaluation of increased ICP to identify signs of hemorrhage, mass effect, and bony injury. Decision algorithms exist to guide the need for CT imaging.¹² In patients with a low GCS, the absence of any discernable abnormality on initial head CT does not rule out increased ICP, and diffuse axonal injury must be considered. Further imaging studies may be necessary depending on patient's stability and changing neurological status. Magnetic resonance imaging (MRI) may be indicated if CT does not explain neurological findings. Magnetic resonance imaging is often the preferred imaging modality for subacute neurological insults.¹³

Transcranial Doppler is an emerging noninvasive tool that allows the early detection of increased ICP by measuring blood flow velocity in both middle cerebral arteries. Multiple studies have shown its efficacy in adults, and most recently a study in children showed a 94% sensitivity to identify increased ICP at admission.¹⁴ However, its role in the emergency evaluation of increased ICP remains undetermined.

POST-RESUSCITATION MANAGEMENT OF SUSPECTED OR ACTUAL INCREASED ICP

Patients with significantly increased ICP require close monitoring and rapid treatment and should be referred for intensive care services. Intensive care management follows the same general guidelines as the emergency department management; however, invasive monitoring is often available. Specifically, the use of ICP monitors allows for targeted therapy against increased ICP and should be considered in all patients with severe intracranial pathology.

Maintenance of Normoglycemia and Normonatremia

Maintaining blood glucose within normal levels prevents secondary brain injury. Hypoglycemia impairs the energy supply to disrupted neurons, whereas hyperglycemia increases oxidative stress. Studies in children are lacking; however, a recent adult study has shown that a blood glucose level between 6 and 9 mmol/L is optimal to achieve appropriate cerebral metabolic function.¹⁵

Hyponatremia creates an osmotic gradient resulting in intracellular expansion. Maintaining normal blood sodium minimizes cerebral edema and fluctuations in ICP. Resuscitation with isotonic fluids is critically important, and hypertonic saline should be used to treat low or falling serum sodium levels. Al-

though no specific targets are established, serum sodium should be maintained at least above 135 mmol/L. Nonosmotic antidiuretic hormone secretion and cerebral salt wasting are important causes of hyponatremia after neurological insults and should be treated aggressively and promptly.¹⁶

Patient Position

Maintaining the head midline and elevated at 30 degrees maximizes cerebral venous return, which lowers ICP without lowering cerebral perfusion pressure.¹⁷

Temperature Control

Hyperthermia increases cerebral metabolic demand by 5% for every 1°C elevation in body temperature, which may result in relative tissue hypoxia.¹⁸ Environmental measures such as forced-air cooling blankets and pharmacologic measures may be necessary. Although cooling is presently used almost exclusively in the intensive care unit (ICU), brain cooling is likely to be used more commonly in prehospital and emergency settings in the near future.

Hyperosmolar Therapy

Hyperosmolar therapy with mannitol and/or hypertonic saline is commonly escalated in the ICU. Continuous infusions of hypertonic saline are titrated as needed to maintain ICP less than 20 mm Hg. Ideally, the serum sodium should not exceed 160 mmol/L, however, higher levels may be well tolerated.¹⁹ Mannitol is titrated to maintain ICP less than 20 mm Hg while maintaining serum osmolality no greater than 320 mOsm/L.

CSF Drainage

Drainage of CSF may be considered in patients with ventriculo-peritoneal shunts in the emergency department or the ICU, whereas external ventricular drains are used almost exclusively in the ICU. Drainage of CSF should occur after the other goals outlined above are deemed insufficient to control ICP. CSF is drained in small aliquots as needed to maintain the ICP below the desired target (typically 20–25 mm Hg).

Lumbar drainage may be considered in patients with a functioning ventriculostomy, open basal cisterns, and no evidence of a major mass lesion or shift on imaging studies.⁹

MANAGEMENT OF REFRACTORY INTRACRANIAL HYPERTENSION

Increased ICP persisting despite appropriate deployment of the aforementioned therapies requires consideration of “tier two” therapies. The long-term effects of these therapies are uncertain, despite their obvious effect on the measured ICP. Initiation of these therapies should be considered only after discussion with a critical care physician and a neurosurgeon.

Cooling

Evidence relating hyperthermia to worse outcome in animals and adult humans has led to a longstanding interest in the potential role for therapeutic hypothermia following traumatic brain injury. Trends toward improved outcome have been demonstrated in multiple adult studies,^{20,21} however, pediatric data are lacking.

Hyperventilation

Historically, aggressive hyperventilation has been used in children with increased ICP following traumatic brain injury. More current literature demonstrates that hyperemia is relatively uncommon following traumatic brain injury,²² and that hyperventilation may decrease cerebral perfusion and induce brain ischemia.^{23–26} The exact relationship between hyperventilation

and outcome in patients with increased ICP has not been established. For cases where long-term hyperventilation is being used, intermittent assessment of brain oxygenation using either a jugular venous bulb catheter or xenon CT perfusion scans should be considered.

Decompressive Craniectomy

Despite the fact that decompressive craniectomy clearly lowers ICP in children with medically refractory increased ICP,^{27–29} there is only weak evidence suggesting that the use of decompressive craniectomy improves clinical outcome.^{28–30} Decompressive craniectomy may be considered in cases of refractory increased ICP when: diffuse swelling is evident on CT imaging, the injury is less than 48 hours old, there are no sustained episodes of elevated ICP before surgery, GCS has been greater than 3 on at least one prior assessment, and there is evidence of an evolving cerebral herniation syndrome.

Barbiturate Coma

Barbiturate coma has been shown in small pediatric studies to reduce ICP that remains elevated despite maximal medical and surgical efforts.^{31,32} No studies have adequately evaluated outcome in patients treated with barbiturate coma. Barbiturate coma should generally be avoided, but it may be considered in refractory cases of increased ICP that persist despite all other measures.

SUMMARY

Despite the varied causes of increased ICP, its acute management is largely independent of etiology. Aggressive actions to support airway, breathing, and circulation form the mainstay of therapy. General measures including analgesia, sedation, and use of isotonic resuscitation fluids are standard in the emergency department. ICP-specific therapies should be used for those patients with obvious clinical findings resulting from suspected increased ICP. Cranial imaging is a priority, although those patients with a herniation syndrome should be treated urgently before CT confirmation of brain herniation. Neurosurgical assessment is warranted urgently in all patients with suspected increased ICP.

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