Increased Intracranial Pressure Management

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Causes of Increases ICP

- Intracranial hemorrhage
  - Traumatic brain injury
  - Ruptured aneurysm
  - Arteriovenous malformation
  - Other vascular anomalies
- Central nervous system infections
- Neoplasm
- Vasculitis
- Ischemic infarcts
- Hydrocephalus
- Pseudotumor cerebri
Physiology: Monro-Kellie Doctrine

- Brain parenchyma—80%
- Cerebrospinal fluid—10%
- Blood—10%
- Monro-Kellie doctrine: Because the overall volume of the cranial vault cannot change, an increase in the volume of one component, or the presence of pathologic components, necessitates the displacement of other structures, an increase in ICP, or both
Intracranial Compensation

Intracranial compensation for an expanding mass lesion
Cerebral Edema: Cytotoxic

- Cytotoxic edema is caused by intracellular swelling secondary to direct cell injury.
- Cytotoxic edema is common in patients who have severe cerebral injuries such as traumatic brain injury, diffuse axonal injury, or hypoxic-ischemic injury.
  - These injuries can range from reversibly to irreversibly injured.
  - In contrast, reversible cytotoxic edema can occur with water intoxication.
Cerebral Edema: Vasogenic

- Vasogenic edema results when increased permeability of capillary endothelial cells permits fluid to escape into the extracellular space
- Neurons are not primarily injured
- Vasogenic edema is seen with tumors, intracranial hematomas, infarcts, abscesses, and central nervous system infections
- Therapy to decrease the edema may prevent secondary ischemic injury to surrounding brain tissue since neurons are not primarily injured
  - Steroid therapy may be beneficial for vasogenic edema that occurs in the setting of mass lesions
Cerebral Edema: Interstitial

• Interstitial edema is characterized by increased fluid in the periventricular white matter
• Increased CSF hydrostatic pressure, as occurs with hydrocephalus, is the most common cause
• Interstitial edema responds to therapy to reduce CSF pressure
Effects of trauma

- Increase in volume of any or all of the intracranial components
- Uncoupling of CBF and metabolic activity (loss of autoregulation) which can lead to excessive CBF
- Increased CSF production in response to cerebral hyperemia
- Hypercapnia or hypoxia, which may cause vasodilation and increase CBF
- Herniation, brain swelling, or subarachnoid hemorrhage, which may obstruct the flow of CSF
Effects of trauma

- Combination of these changes can rapidly exceed the limits of intracranial compensation
  - Leading to an increase in ICP and subsequent herniation or ischemia (focal or global)
- Intracranial hypertension may manifest immediately, but more often occurs in the first few days and peaks at day three to five after trauma
- The elevation of ICP may fluctuate in waves that can be triggered by blood pressure changes, hypoventilation, hypoxia, change of head position, hyperthermia, seizures, or simply cerebrovascular instability
Herniation Syndromes

- Herniation of brain tissue can cause injury by compression or traction on neural and vascular structures.
- Herniation results when there is a pressure differential between the intracranial compartments, and can occur in four areas of the cranial cavity:
  - Transtentorial (2)
  - Subfalcian (1)
  - Foramen magnum (3)
  - Retroalar
Herniation Syndromes: Transtentorial

- Most common type
- Results from downward displacement of supratentorial brain tissue into the infratentorial compartment, and can be caused by supratentorial mass lesions, diffuse brain swelling, focal edema, or acute hydrocephalus
- Can cause compression of the third cranial nerve, the upper brainstem, and the cerebral peduncles, as well as distortion or traction of the superior portion of the basilar artery
Herniation Syndromes: Subfalcian

• Occurs when increased pressure in one hemisphere displaces brain tissue under the falx cerebri

• Can cause compression of the anterior cerebral artery and extensive infarction of the frontal and parietal lobes
Herniation Syndromes: Foramen Magnum

• Occurs when downward pressure forces the cerebellar tonsils into the foramen magnum, where they compress the medulla oblongata and upper cervical spinal cord
Herniation Syndromes: Retroalar

- Occurs when increased pressure in the frontal lobes causes posterior displacement over the lesser wing of the sphenoid bone
- Can cause carotid artery compression with anterior and middle cerebral artery infarction
Presentation: Symptoms

- Global symptoms of elevated ICP
  - Headache
    - Probably mediated via the pain fibers of cranial nerve (CN) V in the dura and blood vessels
  - Depressed global consciousness
    - Due to either the local effect of mass lesions or pressure on the midbrain reticular formation
  - Vomiting
- Focal symptoms
  - May be caused by local effects in patients with mass lesions or herniation syndromes
Presentation: Symptoms

- Additional features of traumatic head injury
  - Decreased level of consciousness
  - Pain coming in waves
  - Visual changes
  - Alterations in vital signs
- Infants may present with less specific symptoms
  - Irritability
  - Bulging fontanel
  - Lethargy
  - Flat affect
  - Poor feeding
Presentation: Symptoms

- Nontraumatic
  - Headache
    - Nocturnal awakening
    - Worsening by cough, micturition, or defecation
    - Recurrent and localized
    - Progressive increase in frequency or severity
  - Growth abnormalities
  - Nuchal rigidity
  - Focal neurologic deficit
  - Persistent vomiting
  - Known risk factor for intracranial pathology
    - (eg, neurocutaneous syndrome, macrocephaly, hormonal abnormalities)
  - Lethargy
  - Personality change
Presentation: Signs

• Papilledema
  – If present can confirm the diagnosis
  – Papilledema may be absent in acute ICP elevations because it takes several days to become apparent
  – Is not invariably present in patients with intracranial hypertension

Papilledema Papilledema, characterized by blurring of the optic disc margins, loss of physiologic cupping, hyperemia, and fullness of the veins, in a five year old girl with intracranial hypertension due to vitamin A intoxication. Courtesy of Gerald Striph, MD.
Presentation: Signs

• Retinal hemorrhages
  – may be present in patients with increased intracranial pressure, and should raise the suspicion of nonaccidental head trauma
Presentation: Signs

- Infants may develop
  - Macrocephaly
  - Split sutures
  - Bulging fontanel
- Hydrocephalus
  - "Sun setting" appearance of the eyes may appear
Presentation: Signs

• Dilated pupil
  – Usually on the side of the lesion

• Cranial nerve palsies of the third, fourth, and sixth cranial nerves can occur
  – 3rd nerve palsy most common
  – May cause double vision or abnormal head posture
Presentation: Signs

• Level of consciousness
  – Can range from irritability to obtundation or coma

• Hemiparesis, hyperreflexia, and hypertonia
  – Are late signs

• Cushing triad
  – Systemic hypertension, bradycardia, and respiratory depression
    • Another late sign, and may be a preterminal event
Presentation: Herniation

- Earliest clinical signs of transtentorial herniation
  - Headache and altered level of consciousness
    - Followed by pupillary changes
  - Bradycardia is another early sign in children
Presentation: Herniation

- Foramen magnum herniation
  - May have downbeat nystagmus, bradycardia, bradypnea, and hypertension
    - These findings may be exacerbated by neck flexion and improve with neck extension

- Subfalcian herniation
  - Unilateral or bilateral weakness, loss of bladder control, and coma

- Retroalar herniation
  - Results in hemiplegia, coma, and death due to compression of the anterior and middle cerebral arteries
Initial Stabilization

• The treatment of intracranial hypertension depends upon the condition of the child and the etiology of the hypertension
  – First goal is stabilization of the cardiopulmonary status according to standard PALS
  – Once the child is stable, head computed tomography (CT) scan without contrast should be performed
Initial Stabilization

• Maintenance of adequate ventilation and blood pressure are the cornerstones of management of elevated intracranial pressure
  – Adequate ventilation prevents the vasodilation that occurs in response to hypercapnea
  – Maintenance of blood pressure is necessary to prevent cerebral ischemia
• CPP is the difference between MAP and ICP
Initial Stabilization: Airway

• A definitive airway must be established
• Indications for endotracheal intubation in children with elevated ICP include:
  – Refractory hypoxia
  – Hypoventilation
  – Glasgow coma score of 8
  – Loss of airway protective reflexes
  – Acute herniation requiring controlled hyperventilation
  – Need for endotracheal administration of resuscitation medications
Initial Stabilization: Airway

• Considerations during RSI
  – **Lidocaine** may be used intravenously, or as a local anesthetic, to prevent ICP surges
  – **Etomidate** is generally favored as a sedative because of its rapid onset of action and minimal side effects, particularly in the multitrauma patient with hemodynamic instability
  – **Thiopental** is classically recommended for patients with elevated ICP who are hemodynamically stable
    • Can cause cardiac suppression and vasodilatation, leading to decreased MAP and should be used with caution for patients who may develop hemodynamic instability.
  – **Midazolam** provides some cerebral protective effects, but it can also cause hypotension in the dose required for RSI. In addition, its onset of action is slower and less reliable than thiopental.
  – **Ketamine** is contraindicated because it can increase MAP and ICP.
  – **Rocuronium** is preferred for paralysis because succinylcholine may cause increases in ICP.
Initial Stabilization: Breathing

• Ventilation should be provided as necessary to maintain a PaCO2 in the low- to mid-30s
  – Mild hyperventilation causes hypocapnia
    • Results in cerebral vasoconstriction and reduced CBF
    • Decrease in CBF is accompanied by a decrease in cerebral blood volume, which in turn decreases ICP
Initial Stabilization: Breathing

• Aggressive hyperventilation with PaCO2 below 30
  – Indicated only if there are clinical signs of acute herniation
  – May prevent herniation by relieving the pressure differential in the intracranial compartments
  – Associated risk of cerebral ischemia with excessive lowering of CBF can be justified only in patients who have signs of ongoing herniation
**Neurologic Effects of Hypocapnia**

- **Excitatory amino acid release**
- **Decreased cerebral blood flow**
- **Low carbon dioxide levels**
- **Increased neuronal excitability**
- **Vasoconstriction**
- **Decreased cerebral blood volume**
- **Decreased oxygen delivery**
- **Increased oxygen consumption**

**Neurotoxicity**
- **Membrane destruction**
- **Excess calcium**
- **Enzymes**

**Cerebrospinal fluid alkalosis**

**Tissue buffering**

**Normalization of partial pressure of arterial carbon dioxide**

**Reperfusion injury**

**Vasodilatation**
Initial Stabilization: Circulation

• Cerebral perfusion must be maintained to prevent secondary ischemic injuries
• Hypovolemia should be treated with hypertonic fluids with a goal of attaining a state of normal volume
• Excess intravascular volume may exacerbate the development of cerebral edema
Evaluation: Neuroimaging

- Head CT may demonstrate:
  - Underlying etiology of elevated ICP (e.g., mass lesion, hemorrhage)
  - Findings consistent with elevated ICP (e.g., midline shift, effacement of the basilar cisterns)
  - And/or effacement of the sulci)
- Patients without these findings on initial CT may have elevated ICP
  - This was demonstrated in a prospective study of 753 patients treated at four major head injury research centers in the United States, in which patients whose initial CT scan did not show a mass lesion, midline shift, or abnormal cisterns had a 10 to 15 percent chance of developing elevated ICP during their hospitalization

Evaluation: Neuroimaging

- Other studies have shown that up to one-third of patients with initially normal scans developed CT scan abnormalities within the first few days after closed head injury.


- Together, these findings demonstrate that ICP can be elevated even in the setting of a normal initial CT, highlighting the role of follow-up imaging in patients who develop clinical evidence of increased ICP during hospitalization.
Evaluation: Lumbar Puncture

• LP, if necessary, should be deferred until after head CT scan in any patient in whom intracranial hypertension is suspected
  – Due to the possibility of precipitating herniation across the tentorial notch or into the foramen magnum by increasing the pressure gradient between compartments

• In patients in whom central nervous system infection is a strong consideration, deferral of lumbar puncture should not delay the initiation of empiric antibiotic therapy
Management

• Goals of therapy
  – Minimize ICP elevation
  – Maintain adequate cerebral perfusion pressure to prevent secondary ischemic injury
    • CPP in adults 60 to 70 mmHg
    • The minimum acceptable CPP in children has not been defined, but is probably lower than that for adults (eg, 50 to 60 mmHg)
    • Extreme elevations in CPP may exceed the capabilities of CBF autoregulation and further increase ICP
Management

• The best therapy for elevated ICP is resolution of the underlying cause
• Regardless of the cause, ICH is a medical emergency, and treatment should be undertaken as expeditiously as possible
• Early neurosurgical consultation should be obtained to assist with management decisions regarding the excision of mass lesions, ICP drainage, and ICP monitoring
Management

• Rapid treatment of hypoxia, hypercarbia, and hypotension

• Fluids (eg, normal saline or 3% NaCl) should be administered to patients to maintain adequate MAP
  – If this fails, infusions of epinephrine can be initiated

• Elevation of the head of the bed from 15 to 30 degrees
  – Mild head elevation can lower ICP without adversely affecting MAP or CPP
  – Elevation greater than 40 degrees may decrease CPP
Management

• Aggressively treating fever with antipyretics and cooling blankets
  – Hyperpyrexia increases cerebral metabolism and increases CBF, further elevating ICP
  – Controlling shivering in intubated patients with muscle relaxants
Management

• Administering prophylactic phenytoin or phenobarbital to patients who are at high risk of developing seizures
  – Breakthrough seizures are best treated with benzodiazepines

• Maintaining adequate analgesia to blunt the response to noxious stimuli
Management: Intubated Patients

• Maintaining the head in a midline position
• Avoiding high positive pressures and end expiratory pressures
  – May increase intrathoracic pressure and impede venous drainage
• Maintaining adequate sedation to permit controlled ventilation
  – Neuromuscular blockade may be required if ICP remains elevated despite adequate sedation
    • Muscle relaxation also can prevent fighting against the ventilator and permit hyperventilation if it is required; short-acting agents are preferred, and can be withheld periodically to permit neurologic evaluation
  – Administration of lidocaine before endotracheal tube suctioning to blunt the gag and cough responses
Management: Mannitol

- Establishes an osmotic gradient between plasma and parenchymal tissue, resulting in a net reduction in brain water content
- Rapid onset of action and maintains its effect for a period of hours
- Can be used to decrease ICP and improve CPP include acute herniation, acute elevation of ICP, and ICP elevation that does not respond to other therapies
Management: Mannitol

- Recommended dose is 0.25 to 1 g/kg IV bolus
- Repeat doses can be administered every six to eight hours to increase serum osmolarity to 300 to 310 mOsm/L
- Carefully evaluate in patients who have renal insufficiency
Management: Mannitol Controversies

- Mannitol administration has the potential side effects of hyperosmolarity, hypovolemia, electrolyte imbalance, and acute renal failure
- More common with chronic or high-dose administration
- Serum osmolarity, serum electrolytes, and renal function should be measured at least every six to eight hours
- When administered chronically and in high doses, mannitol may cross the injured blood-brain barrier at the site of the cerebral lesion and cause an exacerbation of cerebral edema
Management: Hyperventilation

- Can effectively lower ICP via its effect on PaCO2
  - Low PaCO2 causes cerebral vasoconstriction, decreased CBF, and consequently, decreased cerebral blood volume and ICP
- Aggressive hyperventilation may decrease CBF enough to cause cerebral ischemia and actually increase the extent of brain injury
  - In one study of 21 patients with severe traumatic brain injury, forced hyperventilation to an end-tidal PCO2 of 21 mmHg normalized ICP and CPP, but significantly reduced cerebral oxygenation
  - Reserved for episodes of acute brain herniation or ICP elevation that fail to respond to other therapies
Management: CSF Drainage

• In cases of uncontrolled intracranial hypertension, an intracranial drain can be placed to remove CSF and monitor ICP
  – As the ICP increases, the compliance of the brain decreases, and small changes in volume (eg, the removal of as little as 1 mL of CSF) can significantly reduce ICP
Management: Barbiturate Coma

- Barbiturates are used to treat intracranial hypertension that is refractory to other modalities
  - Pentobarbital is the barbiturate that is best studied and most commonly used
    - Works by decreasing the cerebral metabolic rate, which causes a reduction in CBF and thus, in ICP
    - May also provide some protective effect for the brain tissue during periods of hypoxia or hypoperfusion
    - Ability to control ICP elevations with barbiturates is associated with a decreased mortality rate
Management: Barbiturate Coma

• Barbiturates produce cardiac suppression, which may result in hypotension
  – Should be anticipated and treated promptly with fluids and inotropic support if necessary
• Invasive cardiopulmonary monitoring may be needed
• May also benefit from EEG monitoring to maintain a burst suppression pattern and to monitor for underlying seizures
Management: Hypertonic Saline

• Has been shown to decrease ICP and increase CPP in patients with elevated ICP that is refractory to conventional therapy
  – Acts by establishing an osmotic gradient that reduces brain water content
  – Appears to maintain efficacy with repeat dosing even in patients who have stopped responding to mannitol
    • Unlike mannitol, hypertonic saline does not cause profound osmotic diuresis, and the risk of hypovolemia as a complication is decreased
  – Theoretical complications, such as hyperosmolarity, central pontine myelinolysis, and congestive heart failure, have not been reported
Management: Hypothermia

• Controlled hypothermia has been shown to help reduce ICP in some patients with refractory intracranial hypertension and may improve outcome.
• Most appropriate time and population for this therapy remain to be determined.
Management: Glycemic Control

• Both hyperglycemia and hypoglycemia in children with head injuries have been associated with poor outcomes.

• Many trauma centers aggressively treat hyperglycemia in patients with head injuries with insulin and avoidance of excessive use of dextrose-containing intravenous fluids.
  – Patients also should be monitored for hypoglycemia, which can adversely affect brain tissue, particularly in infants and small children who have smaller glycogen stores.
Approach to Increased ICP in the Neurologically Injured Child

ABCs

Neurological exam
GCS <8 or CT scan findings consistent with increased ICP

Intubate

Operating room if resectable mass lesion
Insertion of ICP monitor +/- ventriculostomy

HOB <30° with head midline
Sedation Analgesia +/- NMB
Normocarbia PaCO₂ ≈ 35
Normovolemia SpO₂ >92%
PaO₂ >60

ICP >20 mm Hg

CSF diversion
Minimize stimulation
Prevent/treat Seizures
Prevent/treat fever

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ICP >20 mm Hg

Repeat head CT
Osmotherapy:
mannitol and/or hypertonic saline
Mild hyperventilation PaCO₂ 30-35 mm Hg
Consider SjvO₂, PbtO₂ monitoring

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ICP >20 mm Hg despite above interventions
Refractory Intracranial Hypertension

Barbiturate therapy
Moderate hypothermia (32–33°C)
Hyperventilation (PaCO₂ <30)
Decompressive craniectomy
Lumbar drain