Emergency Neurological Life Support® Intracranial Hypertension and Herniation Protocol Version 6.0

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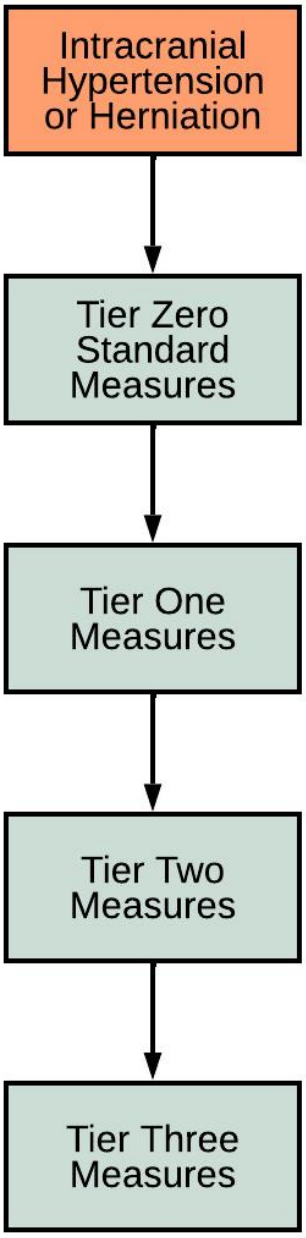
Last updated: September 2024

INTRACRANIAL HYPERTENSION AND HERNIATION ALGORITHM



- Assess CAB's and consider intubation, Sp02>94%
- Consider end tidal C02 monitoring
- Obtain non-contrasted head CT
- Serial neurological assessment including pupillary reactivity
- MInimize noxious stimuli
- HOB 30-45 degrees
- Neck midline
- Analgesia/sedation to patient comfort
- Normothermia
- Avoid and correct hyponatremia
- Steroids in select conditions (brain tumors, abscess, meningitis, noninfectious neuroinflammatory conditions)

- Hyperosmolar Therapy (mannitol or hypertonic saline)
- ICP monitoring and CSF drainage
- Maintain CPP 60-70 mmHg
- Increase sedation and analgesia
- PaC02 at low end of normal 35-38 mmHg
- Consider EEG monitoring
- Repeat head CT
- Consider surgical options

- Optimize sedation and analgesia
- Consider neuromuscular paralysis
- Mild hypocapnia (32-35 mmHg range)
- Perform MAP Challenge to test autoregulation and increase CPP is appropriate

- Sedation tirated to ICP goal or burst suppression on cEEG
- Mild hypothermia 32ºC-36ºC (TBI 35º-36ºC only)
- Decompressive hemicraniectomy in some cases as a lifesaving intervention

COMMUNICATION

- ☐ Age
- ☐ Injury mechanism (e.g., intracerebral hemorrhage, trauma, acute ischemic stroke, etc.)
- ☐ Relevant known medical history
- ☐ Comorbid or complicating conditions
- ☐ Suspected etiology for elevated intracranial pressure (ICP) / herniation (e.g., diffuse edema after trauma)
- ☐ Clinical neurological examination (worst, best, and current exam seen during your care)
- ☐ Steps taken to manage ICP and patient's response to intervention
- ☐ Anticipated next steps (e.g., continue 23.4% for sodium goal > 145 mEq/L with q4 h checks)
- ☐ Does the patient have an external ventricular drain (EVD) or other ICP monitor?
- ☐ What is the patient's vascular access?

INTRACRANIAL HYPERTENSION AND/OR HERNIATION

ICP > 22, > 20 mmHg in Pediatrics, or Clinical Signs of Herniation

Sustained intracranial hypertension and acute brain herniation signify catastrophic neurological events that require immediate recognition and treatment to prevent irreversible injury and death and are associated with many neurological emergency disease states. Increased intracranial pressure and herniation syndromes (ICP crises) mandate the organized implementation of a stepwise management algorithm for maintenance of cerebral blood flow. The goal of this Emergency Neurological Life Support® (ENLS) module is to detail an evidence-based, standardized approach to the evaluation and management of patients with ICP crisis.

Although frequently linked, elevations of ICP and brain herniation can occur independently.

- Intracranial hypertension is defined as a sustained (> 5 min) elevation of ICP to > 22 mmHg or > 20 mmHg in pediatrics.
- Detection requires invasive monitoring, but certain clinical and physiological signs may suggest elevated ICP prior to instrumentation.
- Herniation syndromes result from intracranial compartmental pressure gradients leading to parenchymal tissue shifts that compress or displace the brainstem, cranial nerves or cerebral vasculature.
- Ischemia or infarction during such vascular compression may cause edema and further aggravate a deterioration in compliance.

Diagnosis

- Clinically, symptoms of increased ICP include headache, nausea and vomiting, pupillary changes and/or altered mental status.
- Patients with increased ICP may demonstrate physical signs of hypertension, bradycardia and irregular respirations or apnea (Cushing's triad), although the concurrence of all three signs is an uncommon and often late finding.
- Common sites for herniation are the cingulum of the medial frontal lobe (subfalcine herniation), medial temporal lobe (uncal herniation) and inferior cerebellum (tonsillar herniation).
- The cardinal signs of transtentorial (uncal) herniation are an acute change in consciousness associated with ipsilateral pupillary dilation and contralateral hemiparesis, resulting, respectively, from compression or displacement of ascending arousal pathways, oculomotor nerve (III) and corticospinal tract.

TIER ZERO

Standard issues to prevent herniation

It is important to stress that any patient who is at risk for elevated ICP should have the Tier Zero interventions in place.

- Assess CABs – assess airway patency, ventilation, and adequate circulation. Keep SpO2 > 94%
- Minimize noxious stimuli that may elevate ICP.
- The head of the bed should be elevated to > 30°, and the head is kept midline to facilitate cerebral venous drainage.
- Administer adequate analgesia/sedation to allow for patient comfort and ventilator synchrony.
- Target normothermia. If hyperthermia is present, measures should be taken using targeted temperature management (TTM) to normalize body temperature.
- Avoid and correct hyponatremia (serum Na < 135 mEq/L).
- High-dose corticosteroid therapy is initiated for vasogenic edema resulting from brain tumors, abscesses, bacterial or tuberculosis meningitis, or non-infectious neuroinflammatory conditions but should otherwise be avoided.
- If the brain has not yet been imaged, a noncontrast head computed tomography (CT) scan should be performed when the patient can be positioned safely for diagnostic imaging.

TIER ONE

Hyperosmolar therapy, CSF drainage, Surgery

Hyperosmolar therapy

- Mannitol or hypertonic saline (HTS) have shown equivalent efficacy in lowering of ICP – see ENLS *Pharmacotherapy* module.
- To be effective, intact blood-brain barrier and osmotic/sodium gradient between brain and serum are required to promote the egress of water from the brain.
- Mannitol is administered as 0.5-1 g/kg IV bolus through a peripheral IV line over 5-15 min and may be repeated every 4-6 h. Repeat dosing of mannitol can be determined based on the osmolar gap, which is derived as the difference between measured and calculated osmolality.
- Clinicians may use osmotic gap goals of 20- 55 mOsm/kg although therapeutic benefit at the high end of the range is unlikely to add more benefit.
- HTS is available in concentrations from 2% to 23.4% and can be administered as a bolus alone or in addition to mannitol.
- When infusing HTS, serum sodium concentration levels should be checked every 4-6 h, and serum sodium concentrations should be kept < 160 mEq/L.

CSF drainage

- Acute obstructive hydrocephalus, as determined by neuroimaging, should be emergently managed with an EVD system. If an EVD system is already in place, drain 5-10 ml of cerebrospinal fluid (CSF) for acute rises in ICP.
- Brain perfusion must be maintained, and a cerebral perfusion pressure (CPP) of 60-70 is recommended.

Consider surgical decompression

If ICP is not controlled, and/or clinical signs of herniation do not resolve with Tier One interventions, surgical options (e.g., evacuation of hemorrhagic contusion) should be considered.

If surgery is not appropriate or not undertaken, Tier Two interventions should be implemented.

If ICP is controlled with Tier One interventions, consider repeating the head CT scan to rule out new processes.

TIER TWO

Hypertonic saline and sedation

If Tier One interventions have failed to control ICP, Tier Two should be engaged.

Optimize sedation and analgesia

Sedation may be increased to aid in ICP management.

- Propofol has been shown to reduce cerebral metabolic demand (CMRO2) and cerebral blood volume (CBV) and, consequently, ICP.
- Administer propofol as a bolus of 1-2 mg/kg. May be continued as an infusion (titrate to maximum 200 µg/kg/min) in ventilated patients.
- Propofol, especially when given as a bolus dose, is associated with circulatory depression, which should be corrected with IV fluids and/or vasopressors to maintain CPP goals.
- A small subset of patients receiving propofol may develop a propofol infusion syndrome characterized by metabolic acidosis, cardiac dysfunction, rhabdomyolysis, and hypertriglyceridemia, often with a fatal outcome. Young pediatric patients may be at higher risk of propofol infusion syndrome.
- Propofol infusion syndrome is more likely to develop at doses greater than > 80 mcg/kg/min administered for > 48 h. If propofol is infused at these extreme doses (200 µg/kg/min), it should only be done temporarily, while other corrective measures are executed.

Neuromuscular blockade

- Neuromuscular blocking agents (NMBAs) may be considered to prevent coughing, posturing, shivering or ventilator dyssynchrony, thereby facilitating improved venous outflow from the brain.
- Safety considerations include monitoring for pressure wounds, eye lubrication and use of positioning aids for feet and arms.

Hyperventilation to achieve mild hypocapnia (PaCO2 32-35 mmHg)

- Hyperventilation to achieve mild-to-moderate hypocapnia may be considered in selected patients who have failed other management in the acute period.
- Prolonged hyperventilation is unlikely to be beneficial and may cause or exacerbate ischemic injury due to hypocapnia-associated cerebral vasoconstriction. Hence, hyperventilation should ideally be accomplished in conjunction with cerebral oxygen monitoring (e.g., jugular venous oximetry, brain tissue oxygen monitoring) to detect cerebral ischemia.

MAP Challenge to test autoregulation

- Measurement of dynamic changes of cerebral autoregulation can correlate a systemic hemodynamic parameters such as arterial blood pressure with intracranial physiological parameters such as ICP, transcranial Doppler-derived cerebral blood flow (CBF), velocity, or brain tissue ($PbtO_2$).
- Each patient's unique autoregulation curve could be derived from these measurements, and optimized CPP and optimized arterial blood pressure can be individualized specifically for the needs of the patient in real time.

TIER THREE

Tier Three measures represent the most aggressive level of management and also carry the highest risk of adverse effects. Rigorous randomized prospective studies are lacking, and recommendations are driven by consensus.

Sedation titrated to ICP goal or burst suppression on continuous electroencephalogram (cEEG),

Mild hypothermia 32°-36° C (TBI 35-36° C only)

- Decompressive hemicraniectomy in some cases as a lifesaving intervention

Sedation titrated to ICP goal or burst suppression on cEEG

- This tier includes administration of sedation including propofol, benzodiazepines, ketamine, or barbituates titrated to ICP goal or burst suppression cEEG.
- Pentobarbital (bolus 5-15 mg/kg over 30 min-2 h, then maintenance infusion of 1-4 mg/kg/h) is often used.
- Some patients may not tolerate pentobarbital bolus at these doses because of cardiovascular complications, such as hypotension. Often, arterial vasopressors are necessary for hemodynamic support.
- EEG should be continuously monitored, and pentobarbital titrated either to ICP or to EEG burst suppression of 5-20 s or at least 50%.
- The pentobarbital infusion is continued for 24-96 h, while the processes driving ICP are treated.
- Pentobarbital is associated with respiratory depression, cardiovascular instability, immune suppression and paralytic ileus. During treatment, the neurological examination is limited by sedation.
- High-dose pentobarbital can mimic signs of brain death including unreactive pupils even by pupillometry, and caution is to be exercised in prognostication, as pentobarbital plasma clearance may take days after discontinuation of infusion; however, redistribution from the CNS occurs more rapidly.

Moderate hypothermia (target core temperature 32°-36°C/ 35°-36ºC for TBI patients).

- TTM for mild hypothermia (target core temperature 32°-36°C) may be associated with a reduction in ICP but has not been shown to result in improved outcomes.
- In the case of TBI, hypothermia may worsen outcomes. Targeted temperatures should be limited to 35º-36ºC for these patients.
- Implementation of TTM with a temperature feedback device can improve efficacy.
- When using TTM for mild hypothermia, external surface cooling devices or intravascular or esophageal cooling devices may be used.
- Hypothermia may be associated with shivering, cardiac arrhythmias, sepsis and electrolyte disturbances, and protocols for induction, maintenance and rewarming should be used to prevent or treat these complications.

Rescue decompressive surgery should be considered as a life-saving intervention.