Emergency Neurological Life Support Intracranial Hypertension and Herniation Protocol Version 5.0

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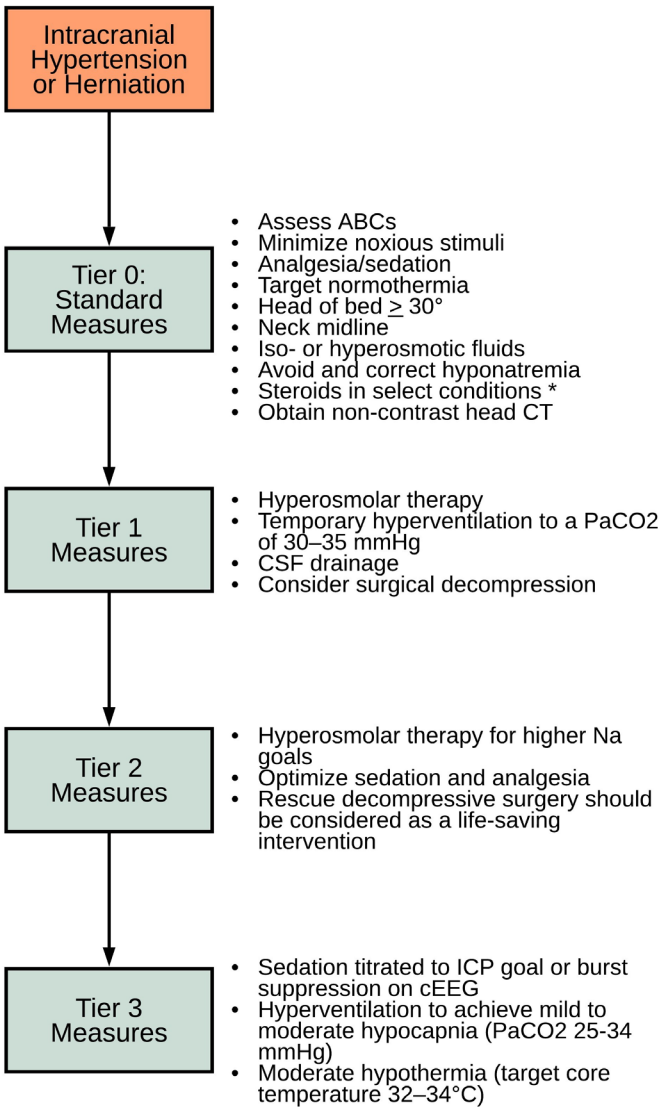
ENLS

EMERGENCY NEUROLOGICAL LIFE SUPPORT

Intracranial Hypertension and Herniation Algorithm



* brain tumors, abscess, non-infectious neuroinflammatory conditions

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 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 EVD or other ICP monitor? □What is the patient's vascular access?

□**Sample sign-off narrative:**
56-year-old male who is status post motor vehicle collision in which he was an unrestrained driver.
The patient's primary survey was significant for failure to protect his airway, due to his mental status, requiring endotracheal intubation—which was performed without complication.
Breathing and circulation were intact; his GCS (E2V3M3) was 8 before being intubated with a left pupil 7 mm and nonreactive and 4 mm reactive pupil on the right.
He was given 150 ml of 3% HTS for his dilated, nonreactive, pupil and was hyperventilated to an EtCO2 of 30. His left pupillary dilation was reduced after these interventions.
His CT head revealed a large subdural hematoma with 6 mm of midline shift.
The patient is going directly to the operating room for hematoma evacuation.
He has no ICP monitor at this time and has 3 16G peripheral IVs.

Intracranial Hypertension and/or Herniation

ICP > 20 mmHg or Clinical Signs

Sustained intracranial hypertension and acute brain herniation are “brain codes,” signifying catastrophic neurological events that require immediate recognition and treatment to prevent irreversible injury and death. As in cardiac arrest, a brain code mandates the organized implementation of a stepwise management algorithm. The goal of this Emergency Neurological Life Support protocol is to detail an evidence-based, standardized approach to the evaluation and management of patients with intracranial hypertension and/or herniation.

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.
- 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 ABCs – assess airway patency, ventilation, and adequate circulation
- Minimize noxious stimuli such as tracheal suctioning that may elevate ICP
- Adequate analgesia/sedation to reduce increases in ICP while attempting to not oversedate to allow for least influenced clinical neurologic examination
- Target normothermia. If hyperthermia is present, measures should be taken using targeted temperature management (TTM) to normalize body temperature to 36–37.4 °C. Implementation of TTM with a temperature feedback device can improve efficacy
- The head of the bed should be elevated to > 30°, and the head is kept midline to facilitate cerebral venous drainage
- Only iso- or hyperosmotic fluids should be used as intravenous (IV) solutions
- Avoid and correct hyponatremia (serum Na < 135 mEq/L)
- High-dose corticosteroid therapy is initiated for vasogenic edema resulting from brain tumors, abscesses, or non-infectious neuroinflammatory conditions but should otherwise be avoided
- If the brain has not yet been imaged, a non-contrast head CT scan should be performed when the patient can be positioned safely for diagnostic imaging

Tier One

Hyperosmolar therapy, hyperventilation, 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.
- No therapeutic benefit is appreciable with osmolar gap > 20 mOsm/kg.
- HTS is available in concentrations from 2 to 23.4% and can be administered as a bolus alone or in addition to mannitol.
- HTS concentrations boluses ≥ 7.5% should be given via a central venous catheter; when using concentrations lower than this, peripheral lines may be used, but the infusion should be in a large vessel, and the IV site should be carefully monitored for infiltration.
- When infusing HTS, serum sodium concentration levels should be checked every 4–6 h, and serum sodium concentrations should be kept < 160 mEq/L.
- Administration of HTS through intraosseous (IO) access should be done with caution and only with concentrations of 7.5% or less due to uncertain risk of myonecrosis.

Temporary hyperventilation to a PaCO2 of 30–35 mmHg

- A brief course (< 2 h) of hyperventilation to a PaCO2 of 30–35 mmHg may be considered, while definitive treatment is provided. Prolonged hyperventilation can result in cerebral ischemia, thus limiting the duration is imperative.

CSF drainage

- Acute obstructive hydrocephalus, as determined by neuroimaging, should be emergently managed with an external ventricular drainage (EVD) system. If an EVD system is already in place, drain 5–10 ml of CSF for acute rises in ICP [22].

Consider surgical decompression

- If ICP is not controlled, and/or clinical signs of herniation do not resolve with Tier One interventions, decompressive 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.

Hyperosmolar therapy for higher Na goals
If hyperosmolar therapy with HTS has been administered, serum sodium goals may be increased if they are not yet at a maximal concentration.

- In practice, serum sodium concentration > 160 mEq/L is unlikely to provide significant additional benefit.
- Once the ICP has stabilized, sodium concentration should be maintained at the current concentration until the brain edema has improved. This is often achieved with intermittent boluses of 3% NaCl during which serum sodium levels are monitored every 6 h.
- It is controversial whether continuous infusion of 3% NaCl is beneficial for ICP control.

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 > 70 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.

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

Tier Three

No longer a surgical candidate

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 cEEG

- This tier includes administration of pentobarbital (bolus 5–15 mg/kg over 30 min—2 h, then maintenance infusion of 1–4 mg/kg/h) titrated to ICP goal or burst suppression on continuous electroencephalogram (EEG).
- 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.

Hyperventilation to achieve mild to moderate hypocapnia (PaCO2 25-34 mmHg)

- Hyperventilation to achieve mild-to-moderate hypocapnia (PaCO2 25–34 mmHg) may be considered in selected patients who have failed other management in the acute period.
- Prolonged hyperventilation, for more than 6 h, 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 a cerebral oxygen monitoring (e.g., jugular venous oximetry, brain tissue oxygen monitoring) to detect cerebral ischemia.

Moderate hypothermia (target core temperature 32–34°C)

- TTM for mild hypothermia (target core temperature 32–34 °C) may be associated with a reduction in ICP but has not been shown to result in improved outcomes
- TTM for mild hypothermia may be induced with external surface cooling devices, IV infusion of cooled fluids, or intravascular or esophageal cooling devices.
- Implementation of TTM with a temperature feedback device can improve efficacy

- 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.