Intracranial hypertension treatment

- Utilizing intravascular ultrasound for optimizing patient selection in venous sinus stenting: A pilot study
- Myelin oligodendrocyte glycoprotein antibody-associated disease with aseptic meningitis-like presentation in a paediatric patient
- Cerebral small vessel disease and effects of intensive versus standard blood pressure treatment on cardiovascular outcomes and adverse events
- The CXCL16/CXCR6 axis is linked to immune effector cell-associated neurotoxicity in chimeric antigen receptor (CAR) T cell therapy
- Feasibility and Efficacy of Venous Sinus Stenting for Idiopathic Intracranial Hypertension in a non-Western Country
- Acute Compartment Syndrome and Intra-Abdominal Hypertension, Decompression, Current Pharmacotherapy, and Stable Gastric Pentadecapeptide BPC 157 Solution
- Headache in the paediatric population: the role of the ophthalmologist
- Signs of Intracranial Hypertension in Chronic Inflammatory Polyradiculoneuropathies-A Cross Sectional Cohort Study

---

see also Idiopathic intracranial hypertension treatment.

Intracranial hypertension is usually treated with general maneuvers (normothermia, sedation, etc.) and a set of first-line therapeutic measures (moderate hypocapnia, mannitol, etc.). When these measures fail, second-line therapies are initiated, which include: barbiturates, hyperventilation, moderate hypothermia, or removal of a variable amount of skull bone (secondary decompressive craniectomy).

ICP-lowering therapies are usually administered in a stepwise manner, starting with safer first-line interventions, while reserving higher-risk options for patients with intractable intracranial hypertension.

Measures to lower ICP

General measures that should be routine

1. positioning:

a) elevate HOB 30–45°

b) keep head midline(to prevent kinking jugular veins)

2. light sedation: codeine 30–60 mg IM q 4 hrs PRN, or lorazepam (Ativan®) 1–2 mg IV q 4–6 hrs PRN

3. avoid hypotension (SBP < 90 mm Hg): normalize intravascular volume, support with pressors if needed

4. control HTN; in ICH, aim for patient's baseline,

5. prevent hyperglycemia: (aggravates cerebral edema) usually present in head injury, maybe exacerbated by steroids

6. intubation: for GCS ≤ 8 or respiratory distress. Give IV lidocaine first and antibiotics

7. avoid hyperventilation: keep PaCO2 at the low end of eucapnia (35 mm Hg)

8. prophylactic hypothermia: non-statistically significant trend suggests reduced mortality.

Maintain target temperature for > 48 hours

Measures to use for documented IC-HTN

First, check General measures that should be routine above. Proceed to each step if IC-HTN persists.

1. heavy sedation and/or paralysis when necessary (also assists treatment of HTN) e.g. when patient is agitated, or blunt the elevation of ICP that occurs with certain maneuvers such as moving the patient to CT table. Caution: with heavy sedation or paralysis, the ability to follow the neurologic exam is lost (follow ICPs)

a) for heavy sedation(intubation recommended to avoid respiratory depression→elevation of PaCO2 → ↑ ICP): e.g. one of the following:

● morphine (MSO4): ℞ 2–4 mg/hr IV drip

● fentanyl: ℞ 1–2 ml IV q 1 hr (or 2–5 mcg/kg/hr IV drip)

● sufentanil: ℞ 10–30 mcg test dose, then 0.05 -2 mcg/kg/hr IV drip

● midazolam (Versed®): ℞ 2 mg test dose, then 2–4 mg/hr IV drip

● propofol drip : 0.5 mg/kg test dose, then 20–75 mcg/kg/min IV drip ✖ avoid high-dose propofol (do not exceed 83 mcg/kg/min)

● "low dose" pentobarbital (adult: 100 mg IV q 4 hrs; peds: 2–5 mg/kg IV q 4 hrs)

b) paralysis(intubation mandatory):e.g.vecuronium 8–10mg IV q2–3 hrs

2. CSF drainage (when IVC is being utilized to measure ICP): 3–5 ml of CSF should be drained with the drip chamber at ≤ 10 cm above EAC. Works immediately by removal of CSF (reducing intracranial volume) and possibly by allowing edema fluid to drain into ventricles (latter point is controversial)

3. "osmotic therapy" when there is evidence of IC-HTN:

a) mannitol 0.25–1 gm/kg bolus (over < 20 mins) followed by 0.25 gm/kg IVP (over 20 min) q 6 hrs PRN ICP >

Literature suggests that 1.4 gm/kg initial dose is more effective. May "alternate" with: furosemide (Lasix®): adult 10–20 mg IV q 6 hrs PRN ICP > 22. Peds: 1 mg/kg, 6mg max IV q 6 hrs PRN ICP>22

b) keep patient euvolemic to slightly hypervolemic

c) if IC-HTN persists and serum osmolarity is < 320 mOsm/L, increase mannitol up to 1 gm/kg, and shorten the dosing interval

d) if ICP remains refractory to mannitol, consider hypertonic saline,either continuous 3% saline infusion or as bolus of 10–20 ml of 23.4% saline

(D/C after ≈ 72 hours to avoid rebound edema)

e) hold osmotic therapy if serum osmolarity is≥320mOsm/L(highertonicity may have no advantage and risks renal dysfunction; see below) or SBP < 100

4. hyperventilation (HPV) to PaCO2 = 30–35 mm Hg

a) ✖do not use prophylactically

b) ✖avoidaggressive HPV(PaCO2≤25mmHg)at all times

c) use only for

● short periods for acute neurologic deterioration

● or chronically for documented IC-HTN unresponsive to sedation, paralytics, CSF drainage, and osmotic therapy

d) avoid HPV during the first 24hrs after injury if possible

5. ✖ steroids: the routine use of glucocorticoids is not recommended for treatment of patients with head injuries

Hyperosmolar therapy for intracranial hypertension

see Hyperosmolar therapy for intracranial hypertension.

Prophylactic Hypothermia for severe traumatic brain injury

see Prophylactic Hypothermia for severe traumatic brain injury.

Decompressive craniectomy

see Decompressive craniectomy for intracranial hypertension treatment.

Hypocapnia

Current guidelines suggests a target of partial pressure of carbon dioxide (PaCO2) of 32-35 mmHg (mild hypocapnia) as tier 2 for the intracranial hypertension management. However, the effects of mild hyperventilation on cerebrovascular dynamics are not completely elucidated. This study aims to evaluate the changes in intracranial pressure (ICP), cerebral autoregulation (measured through pressure reactivity index, PRx), and regional cerebral oxygenation (rSO2) parameters before and after induction of mild hyperventilation. A single-center, observational study including patients with acute brain injury (ABI) admitted to the intensive care unit undergoing multimodal neuromonitoring and requiring titration of PaCO2 values to mild hypocapnia as tier 2 for the management of intracranial hypertension. Twenty-five patients were included in this study (40% female), with a median age of 64.7 years (Interquartile Range, IQR = 45.9-73.2). Median Glasgow Coma Scale was 6 (IQR = 3-11). After mild hyperventilation, PaCO2 values decreased (from 42 (39-44) to 34 (32-34) mmHg, $p < 0.0001$), ICP and PRx significantly decreased (from 25.4 (24.1-26.4) to 17.5 (16-21.2) mmHg, $p < 0.0001$, and from 0.32 (0.1-0.52) to 0.12 (-0.03-0.23), $p < 0.0001$). rSO2 was statistically but not clinically significantly reduced (from 60% (56-64) to 59% (54-61), $p < 0.0001$), but the arterial component of rSO2 (ΔO2Hbi, changes in concentration of oxygenated hemoglobin of the total rSO2) decreased from 3.83 (3-6.2) µM.cm to 1.6 (0.5-3.1) µM.cm, $p = 0.0001$. Mild hyperventilation can reduce ICP and improve cerebral autoregulation, with minimal clinical effects on cerebral oxygenation. However, the arterial component of rSO2 was importantly reduced. Multimodal neuromonitoring is essential when titrating PaCO2 values for ICP management [1)].

1)
Cardim D, Giardina A, Ciliberti P, Battaglini D, Berardino A, Uccelli A, Czosnyka M, Roccatagliata L, Matta B, Patroniti N, Rocco PRM, Robba C. Short-term mild hyperventilation on intracranial pressure, cerebral autoregulation, and oxygenation in acute brain injury patients: a prospective observational study. J Clin Monit Comput. 2024 Feb 4. doi: 10.1007/s10877-023-01121-2. Epub ahead of print. PMID: 38310592.