

Intracranial Hypertension

Risk

- Incidence in USA: >50% of pts presenting with head trauma or other intracranial pathology (>600,000/y)
- Gender predominance: Only for certain etiologies (i.e., TBI and males)

Perioperative Risks

- Increased risk of herniation leading to subsequent brain infarction, disability, coma, and death

Worry About

- Controlling ICP and preventing brain ischemia/herniation
- CV and respiratory instability
- Coexisting injuries in trauma pts (occult cervical spine and intra-abdominal injuries)

Overview

- Intracranial compartment has fixed volume with three components (brain = 85%, CSF = 10%, CBV = 5%).
- Increased volume of one component (e.g., tumor, hydrocephalus, or hemorrhage) without compensatory

decrease in another compartment elevates ICP, leading to ICH (ICP >20 mm Hg for >5 min typically, but individual patients' threshold for injury varies).

- ICH reduces CPP (CPP = MAP – ICP), causing brain ischemia and/or infarction.
- ICH causes ICP gradients that may extrude brain parenchyma through dural or bony passages, resulting in herniation. Subfalcine herniation compresses the anterior cerebral artery. Transtentorial herniation compresses the posterior cerebral artery and herniation from craniotomy may compress the middle cerebral artery.
- Some anesthetic agents, hypercapnia, and hypoxemia increase CBF, increasing CBV and ICP. In cases of loss of autoregulation Htn may also increase CBF.

Etiology

- Abnormal increase in volume of parenchymal compartment, CSF or CBV, usually a secondary process accompanying other pathology (e.g., cerebral edema in TBI, cerebral infarct, tumor, inflammation; hemorrhage in TBI, ICH, SAH; hydrocephalus in intraventricular

hemorrhage, compression of ventricles; decreased venous drainage as in cerebral venous thrombosis)

Usual Treatment

- Treatment of primary disease (e.g., removal of tumor, hematoma, or abscess; hemicraniectomy in middle cerebral artery syndrome).
- CSF drainage with either an external ventricular drain or lumbar drain.
- Secure airway if needed and control ventilation, avoid hypoxemia (PaO₂ >90 torr), and avoid hypercapnia and severe hypocapnia (PaCO₂ <30 torr).
- Establish stable hemodynamics (normotension but estimated CPP >60 mm Hg).
- Head elevation above heart and neutral neck position to promote cerebral venous return.
- Osmotic therapy (mannitol or hypertonic saline) to decrease brain parenchyma volume.
- Anesthetic infusion to decrease CMRO₂ after airway is secured.
- Corticosteroids (for vasogenic edema only as in neoplasm or abscess).

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Dysrhythmias, unstable vital signs Inferior wall myocardial ischemia		BP Pulse S ₃ gallop	Tachycardia, bradycardia, prolonged QT interval, ECG, ECHO
RESP	Irregular breathing		Respiratory rate and pattern	
GI	Reduced gut motility	Vomiting		
RENAL	ADH disturbances: SIADH, central DI		Oliguria Polyuria	Urine Na, urine K, urine urea, urine Osm, serum lytes
CNS	Altered function	Headache, vomiting, unconsciousness	Neurologic deficits, papilledema	Direct ICP measurement (ventriculostomy, intracranial bolt)

Key References: Stevens RD, Shoykhet M, Cadena R: Emergency neurological life support: intracranial hypertension and herniation, *Neurocrit Care* 23(Suppl 2):S76–S82, 2015; Stocchetti N, Maas AI: Traumatic intracranial hypertension, *N Engl J Med* 370(22):2121–2130, 2014.

Perioperative Implications

Preoperative Preparation

- Judicious or no preoperative sedation due to risk of depressed ventilatory drive leading to hypoventilation and hypercapnia and increased ICP.
- Assess volume status.

Monitoring

- Consider arterial cath for BP monitoring and for serial ABGs to properly manage mechanical ventilation and control PaCO₂.
- Consider CVP monitor.
- Continue intracerebral multimodal monitoring (i.e., ICP, brain tissue oxygen, microdialysis, jugular venous saturation) if present.

Airway

- Neutral cervical spine position for tracheal intubation if traumatic injury
- Possible aspiration risk (emergency procedure or severe ICH)

Preinduction and Induction

- Neutral neck position and head elevation.

- Deep anesthetic level and complete muscle relaxation with NMBD at time of laryngeal intubation to avoid coughing, sympathetic response, and further increase in ICP.
- Maintain CV stability and CBF with the use of vasopressors as necessary.

Maintenance

- Hypnotic agent (either propofol or volatile anesthetic) and narcotic infusion.
- Maintain volatile anesthetic at less than 1 MAC; N₂O use is controversial.
- Normoventilation (PaCO₂ to 35–40 torr) and use PEEP to maintain FRC and oxygenation; avoid excessive PEEP.
- Maintain MAP such that estimated CPP >60 mm Hg and place transducer at the tragus.

Extubation

- Maintain tracheal intubation if concerns about postoperative respiratory function or persistent ICH; otherwise, prompt extubation for early neurologic evaluation.
- Avoid coughing and bucking on the tube.

Adjuvants

- Benzodiazepines, beta-blockers, and antihypertensives

Postoperative Period

- If ICH persists, continue stepwise approach to decrease ICP and maintain adequate ventilation and/or oxygenation, sedation, NMB, and mild hypothermia as necessary.

Anticipated Problems or Concerns

- Use isotonic crystalloid or colloid IV solutions to minimize cerebral edema.
- Renal dysfunction and severe hypovolemia are possible with mannitol; hypervolemia and acute CHF exacerbation are possible with hypertonic saline.

Acknowledgment

Thank you to Kevin J. Gingrich for his contribution to the previous version of this chapter.