

Raised Intracranial Pressure (ICP): Management in Emergency Department

Author: Dr. Shruti Sangani*, Dr. Samira Parikh**

INTRODUCTION:

Elevated intracranial pressure (ICP) is a potentially devastating complication which requires prompt recognition, the judicious use of invasive monitoring, and therapy directed at both reducing ICP and reversing its underlying cause. The evaluation and management of adult patients with elevated ICP is reviewed here.

PHYSIOLOGY:

Intracranial pressure (ICP) is the pressure inside the skull and thus in the brain tissue and cerebrospinal fluid (CSF). ICP is measured in millimeters of mercury (mmHg) and, at rest, is normally 7–15 mmHg for a supine adult, and becomes negative (averaging –10 mmHg) in the vertical position.[1] Pathologic intracranial hypertension (ICH) is present at pressures ≥ 20 mmHg. Changes in ICP are attributed to volume changes in one or more of the constituents contained in the cranium.

CSF is produced by the choroid plexus in the central nervous system (CNS) at a rate of approximately 20 mL/h (500 mL/day).[2] CSF is normally resorbed via the arachnoid granulations into the venous system. Occasional transient elevations in ICP can be associated with physiologic events, including sneezing, coughing, or Valsalva maneuvers. The skull is a rigid structure with a fixed internal volume of 1400 to 1700 mL. Under physiologic conditions, the intracranial contents include (by volume) brain parenchyma (80 %), cerebrospinal fluid (10 %) and blood (10 %).[3]

The pressure-volume relationship between ICP, volume of CSF, blood, and brain tissue, and cerebral perfusion pressure (CPP) is known as the Monro-Kellie doctrine or the Monro-Kellie hypothesis. It states that the cranial compartment is incompressible, and the volume inside the cranium is a fixed volume. The cranium and its constituents (blood, CSF, and brain tissue) create a state of volume equilibrium, such that any increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another.[4]

The principal buffers for increased volumes include CSF and, to a lesser extent, blood volume. If there is an increase in the volume of either the brain or blood, the normal initial response is a reduction in CSF volume within the skull. If the pathological process progresses with further increase in volume, venous blood and more CSF is forced out of the skull. These compensatory mechanisms are able to maintain a normal ICP for any change in volume less than approximately 100–120 mL. Ultimately this process becomes exhausted, when the venous sinuses are flattened and there is little or no CSF remaining in the head. Any further increase in brain volume then causes a rapid increase in ICP.

ETIOLOGY:The major causes of increased intracranial pressure include:

- Brain Tissue, Blood, CSF
- Tumours
- Cerebral oedema secondary to trauma, infection, infarction, hyponatraemia, hypertensive encephalopathy, acute liver failure, Reye's syndrome
- Cerebral abscesses
- Cerebral contusions- Intracerebral, subarachnoid, subdural, extradural haematomas
- Arteriolar dilatation secondary to hypoxaemia, hypercarbia, anaesthetic drugs, hyperthermia, seizures, hypotension
- Venous dilatation secondary to venous obstruction from high PEEP, coughing, straining, heart failure, venous sinus thrombosis, head-down tilt, tight neck ties
- Hydrocephalus
- Meningeal diseases
- Choroid plexus tumours

The magnitude of the change in volume of an individual structure determines its effect on ICP. In addition, the rate of change in the volume of the intracranial contents influences ICP. Changes that occur slowly produce less of an effect than those that are rapid.

Cerebral blood flow — Following a significant increase in ICP, brain injury can result from brainstem compression and/or a reduction in cerebral blood flow (CBF).

Autoregulation — CBF is normally maintained at a relatively constant level by cerebrovascular autoregulation of cerebrovascular resistance (CVR) over a wide range of cerebral perfusion pressure (CPP) (50 to 100 mmHg).[5,6]

Cerebral perfusion pressure — Conditions associated with elevated ICP, can be associated with a reduction in CPP. This can result in devastating focal or global ischemia. On the other hand, excessive elevation of CPP can lead to hypertensive encephalopathy and cerebral edema due to the eventual breakdown of autoregulation, particularly if the CPP is >120 mmHg.[5,7,8]

Ultimately, global or local reductions in CBF are responsible for the clinical manifestations of elevated ICP.

CLINICAL MANIFESTATIONS: Symptoms of elevated ICP can be headache (probably mediated via the pain fibers of 5th cranial nerve in the dura and blood vessels), depressed level of consciousness (due to either the local effect of mass lesions or pressure on the midbrain reticular formation) and vomiting. Focal symptoms may be caused by local effects of mass lesions or by herniation syndromes. The most common anatomical locations affected by herniation syndromes include subfalcine, central transtentorial, uncal transtentorial, upward cerebellar, cerebellar tonsillar/foramen magnum, and transcalvarial.[2,9] One notable false localizing syndrome seen following neurologic injury, referred to as Kernohan's notch phenomenon, consists of the combination of contralateral pupillary dilatation and ipsilateral

weakness.[10,11] Signs of raised ICP are Cushing's triad of bradycardia, respiratory depression, and hypertension [2] , papilledema , cranial nerve 6 palsy and spontaneous periorbital bruising.[12]

ICP MONITORING:The purpose of monitoring ICP is to improve the clinician's ability to maintain adequate CPP and oxygenation. The only way to reliably determine CPP (defined as the difference between MAP and ICP) is to continuously monitor both ICP and blood pressure (BP). In general, these patients are managed in intensive care units (ICUs) with an ICP monitor and arterial line. Indications for ICP monitoring are closed head injury, suspected risk for elevated ICP and comatose patients (Glasgow Coma Scale <8).

Types of monitors — There are four main anatomical sites used in the clinical measurement of ICP: intraventricular, intraparenchymal, subarachnoid, and epidural. Intraventricular monitors are considered the "gold standard" of ICP monitoring catheters. However all four techniques need surgical intervention which might not be possible in ED.

In ED, Ocular sonography can provide a noninvasive measure of optic nerve sheath diameter, which has been found to correlate with intracranial pressure. A number of studies have found that diameters of 5 to 6 mm have the ability to discriminate between normal and elevated ICP in patients with intracranial hemorrhage and traumatic brain injury. [13,14]

GENERAL MANAGEMENT: The best therapy for intracranial hypertension (ICH) is resolution of the cause of elevated ICP. Regardless of the cause, ICH is a medical emergency, and treatment should be undertaken as fast as possible. In addition to definitive therapy, there are maneuvers that can be employed to reduce ICP acutely. Urgent assessment and support of oxygenation, blood pressure, and end-organ perfusion are of key importance. If elevated ICP is suspected, care should be taken to minimize further elevations in ICP during intubation through careful positioning, appropriate choice of paralytic agents (if required), and adequate sedation.

Large shifts in blood pressure should be minimized, with particular care taken to avoid hypotension. Hypotension, especially in conjunction with hypoxemia, can induce reactive vasodilation and elevations in ICP. Vasopressors have been shown to be safe for use in most patients with intracranial hypertension, and may be required to maintain CPP >60 mmHg. [15] Standard resuscitation techniques should be instituted as soon as possible like head elevation (30 degree maximum), hyperventilation to a PCO₂ of 26 to 30 mmHg and intravenous mannitol (1 to 1.5 g/kg). Along with these measures, patient should be evaluated aggressively to find out the underlying cause with help of detailed neurological examination and neuroimaging. If a diagnosis of elevated ICP is suspected and an immediately treatable proximate cause is not present, then ICP monitoring should be instituted. The goal of ICP monitoring and treatment should be to keep ICP <20 mmHg.[16] Interventions should be utilized only when ICP is elevated above 20 mmHg for >5 to 10 minutes.

Fluid management — In general, patients with elevated ICP do not need to be severely fluid restricted.[17] Patients should be kept euvolemic and normo- to hyperosmolar. This can be achieved by avoiding all free water (including D5W), 0.45 percent (half normal) saline, and enteral free water and employing only isotonic fluids (such as 0.9 percent (normal) saline). Serum osmolality should be kept >280 mOsm/L, and often is kept in the 295 to 305 mOsm/L range. Hyponatremia is common in the setting of elevated ICP, particularly in conjunction with subarachnoid hemorrhage.

Sedation — Keeping patients appropriately sedated can decrease ICP by reducing metabolic demand, ventilator asynchrony, venous congestion, and the sympathetic responses of hypertension and tachycardia.[18] Propofol and fentanyl can be used for sedation as dosage can be titrated easily and they have a short half-life, thus permitting frequent neurologic reassessment.

Blood pressure control — BP should be sufficient to maintain CPP >60 mmHg. Hypertension should generally only be treated when CPP >120 mmHg and ICP >20 mmHg. Caution should be taken to avoid CPP <50 mmHg or, as noted above, normalization of blood pressure in patients with chronic hypertension in whom the autoregulatory curve has shifted to the right. Mean arterial blood pressure should be maintained on a higher side so as to maintain CPP above 60 mmHg.

Position — Patients with elevated ICP should be positioned to maximize venous outflow from the head. Important maneuvers include reducing excessive flexion or rotation of the neck, avoiding restrictive neck taping, and minimizing stimuli that could induce Valsalva responses, such as endotracheal suctioning. Patients with elevated ICP can be positioned with the head elevated above the heart (usually 30 degrees) to increase venous outflow.

Fever — Elevated metabolic demand in the brain results in increased cerebral blood flow (CBF), and can elevate ICP by increasing the volume of blood in the cranial vault. Therefore, aggressive treatment of fever, including acetaminophen and mechanical cooling, is recommended in patients with increased ICP.

Antiepileptic therapy — Seizures can both complicate and contribute to elevated ICP and hence antiepileptics are recommended.

SPECIFIC THERAPIES: Mannitol — Osmotic diuretics reduce brain volume by drawing free water out of the tissue and into the circulation, where it is excreted by the kidneys, thus dehydrating brain parenchyma.[19,20,21] The most commonly used agent is mannitol. It is prepared as a 20 percent solution, and given as a bolus of 1 g/kg. Repeat dosing can be given at 0.25 to 0.5 g/kg as needed, generally every six to eight hours. Use of any osmotic agent should be carefully evaluated in patients with renal insufficiency. The effects are usually present within minutes, peak at about one hour, and last 4 to 24 hours. In addition, mannitol can lower systemic BP, necessitating careful use if associated with a fall in CPP.

Other diuretics — Furosemide, 0.5 to 1.0 mg/kg intravenously, may be given with mannitol to potentiate its effect. However, this effect can also exacerbate dehydration and hypokalemia.

Hypertonic saline — Mannitol and hypertonic saline have been compared in at least five randomized trials of patients with elevated ICP from a variety of causes (traumatic brain injury, stroke, tumors).[22,23,24] A meta-analysis of these trials found that hypertonic saline appeared to have greater efficacy in managing elevated ICP, but clinical outcomes were not examined.[25]

Glucocorticoids — Glucocorticoids may have a role in case of intracranial hypertension caused by brain tumors and CNS infections but are not useful in the management of cerebral infarction or intracranial hemorrhage.

Hyperventilation — Use of mechanical ventilation to lower PaCO₂ to 26 to 30 mmHg has been shown to rapidly reduce ICP through vasoconstriction and a decrease in the volume of intracranial blood; a 1 mmHg change in PaCO₂ is associated with a 3 percent change in CBF. Hyperventilation also results in respiratory alkalosis, which may buffer post-injury acidosis. The effect of hyperventilation on ICP is short-lived (1 to 24 hours). Following therapeutic hyperventilation, the patient's respiratory rate should be tapered back to normal over several hours to avoid a rebound effect. Therapeutic hyperventilation should be considered as an urgent intervention when elevated ICP complicates cerebral edema, intracranial hemorrhage, and tumor. Hyperventilation should not be used on a chronic basis, regardless of the cause of increased ICP.

Hyperventilation should be minimized in patients with traumatic brain injury or acute stroke. In these settings, vasoconstriction may cause a critical decrease in local cerebral perfusion and worsen neurologic injury.

Barbiturates — The use of barbiturates is predicated on their ability to reduce brain metabolism and cerebral blood flow, thus lowering ICP and exerting a neuroprotective effect. Pentobarbital is generally used, with a loading dose of 5 to 20 mg/kg as a bolus, followed by 1 to 4 mg/kg per hr. Treatment should be assessed based on ICP, CPP, and the presence of unacceptable side effects. Continuous EEG monitoring is generally used; EEG burst suppression is an indication of maximal dosing. Barbiturate therapy can be complicated by hypotension, possibly requiring vasopressor support. The use of barbiturates is also associated with a loss of the neurologic examination, requiring accurate ICP, hemodynamic, and often EEG monitoring to guide therapy.

Therapeutic hypothermia — Hypothermia decreases cerebral metabolism and may reduce CBF and ICP. Hypothermia can be achieved using whole body cooling, including lavage and cooling blankets, to a goal core temperature of 32 to 34°C. The value of therapeutic hypothermia has been best assessed in patients after traumatic brain injury (TBI), but its role has not been well established in that setting. Given the uncertainties surrounding the appropriate use of therapeutic hypothermia in patients with elevated ICP, this treatment should be limited to clinical trials, or to patients with intracranial hypertension refractory to other therapies.

Removal of CSF by ventriculostomy or decompressive craniotomy can also be employed in urgent settings if surgical facilities are available. A lumbar drain is generally contraindicated in the setting of high ICP due to the risk of transtentorial herniation.

SUMMARY: The best therapy for raised intracranial pressure is resolution of the cause. Regardless of the cause, treatment should be undertaken as quickly as possible, and should be based on the principles of resuscitation, reduction of the volume of the intracranial contents, and reassessment.

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