

Raised Intracranial Pressure and its Management

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One of the commonest pathological phenomena encountered by the neurosurgeon is that of increased intracranial pressure (ICP) and it has profound influence on the outcome of many intracranial problems. If raised intracranial pressure is not recognised promptly and managed appropriately, there is always a considerable risk in all such patients of secondary brain damage and long term severe disability.

Physiology

The cranium can be thought of as a hollow, rigid sphere of constant volume. There are three main components within the intracranial space : brain (1400 ml), cerebrospinal fluid (CSF) (75 ml) and blood (app. 75 ml) [15]. All these components are essentially non-compressible. The rigid cranial sphere provides the premise of the Monro-Kellie

Common causes of increased ICP

Head Injury	:	Intracranial haematoma (EDH, SDH, intracerebral) Diffuse brain swelling, Contusion.
Cerebrovascular	:	Subarachnoid haemorrhage, Cerebral venous thrombosis, Major cerebral infarcts, Hypertensive encephalopathy.
Hydrocephalus	:	Congenital or acquired (Obstructive or communicating).
Cranio cerebral disproportion		
Brain Tumour	:	(Cysts ; benign or malignant tumour) Secondary Hydrocephalus Mass effect Oedema
Benign Intracranial Hypertension		
CNS Infection	:	Meningitis, Encephalitis, Abscess.
Metabolic Encephalopathy	:	Hypoxic-ischaemic, Reyes syndrome, Hepatic coma, Renal failure, Diabetic ketoacidosis, Hyponatraemia, Burns, Near drowning.
Status epilepticus		

Adapted from references 1, 2, 3, 4.

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doctrine which states that a change in the volume of brain causes a reciprocal change in the volume of one of the intracranial components i. e. either blood or CSF [12].

Normal ICP is between 0-10 mm Hg with 15 mm being the upper limit of normal. Resting ICP represents that equilibrium pressure at which CSF production and absorption are in balance and is associated with an equivalent equilibrium volume of CSF. CSF is actively secreted by choroid plexus at about 0.35 ml/minute and production remains constant provided cerebral perfusion pressure is adequate.

CPP is commonly defined as : $CPP = MAP - ICP$ [MAP : Mean arterial pressure, CPP : Cerebral perfusion pressure].

Spontaneous waves of ICP [13] are :

B Wave is related to various types of periodic breathing with a frequency of 1/2 to 2 per minute.

C Wave is related to Traube-Hering-Mayer waves of systemic blood pressure and has a frequency of 6 per minute.

Both B and C waves are of low amplitude and are not harmful.

Type A wave or Plateau wave is an acute elevation in ICP lasting from 5 to 20 minutes followed by a rapid fall in pressure to former resting level. The amplitude is variable but may reach very high levels (50 to 100 mm Hg) and is associated with clinical signs of **acute brain stem dysfunction**.

Mechanisms involved in raised ICP

A	Mass Lesions	Haematoma, abscess, tumour.
B	CSF Accumulation	Hydrocephalus (obstructive and communicating) and including contralateral ventricular dilatation from supratentorial brain shift.
C	Cerebral Oedema	Increase in brain volume as a result of increased water content : <ol style="list-style-type: none"> 1. Vasogenic—Vessel damage (tumour, abscess, contusion). 2. Cytotoxic—cell membrane pump failure (hypoxaemia, ischaemia, toxins). 3. Hydrostatic—high vascular transmural pressure (loss of autoregulation, post intracranial decompression) 4. Hypo-osmolar-hyponatraemia. 5. Interstitial—high CSF pressure (hydrocephalus).
D	Vascular (congestive) brain swelling	Increased cerebral blood volume. —arterial vasodilatation (active, passive). —venous congestion/obstruction.

~~—venous congestion/obstruction.~~

Monitoring techniques

(A) Clinical features : In the non-trauma patient, there may or may not be a clear history of headache, vomiting and visual disturbances suggestive of papilloedema or a VI nerve palsy. The absence of papilloedema does not exclude raised ICP in patients with acute or chronic problems.

Fifty percent of head injury patients who have raised ICP on monitoring will exhibit optic disc swelling in only 4% cases. "So fundoscopy may not be of much use in acute head injuries to detect raised ICP"

(B) CT Scanning : It may show a mass lesion, hydrocephalus or diffuse odema.

(C) Invasive methods of ICP monitoring :

(1) Extradural sensor.

(2) Subdural fluid filled catheters are reasonably accurate below 30 mm. Hg.

(3) Catheter tip transducers for ICP monitoring (Camino-Codman types) are of intraparenchymatous and intraventricular types and these are very useful for wave form analysis. Ventricular catheter permits the therapeutic drainage of CSF in case of intraventricular bleeds, ventricular dilatation etc.

(D) Non-invasive ICP monitoring :

(i) Transcranial Doppler : This measures flow velocity in branches of the Circle of Willis, most commonly the middle cerebral artery (MCA).

Indications for ICP monitoring

HEAD INJURY

(a) being artificially ventilated :

- Coma with compression of 3rd ventricle and/or reduction in perimesencephalic cistern on CT
- Coma following removal of intracranial haematoma.
- Coma with decorticate/decerebrate motor response.
- Coma with mid line shift/unilateral ventricular dilatation.
- Early seizures not easily controlled.
- Refractory hyperpyrexia.

(b) Uncertainty over surgery for small haematoma/multiple lesions.

INTRACEREBRAL AND SUBARACHONOID HAEMORRHAGE

- coma.
- postoperatively following intra operative complications.
- hydrocephalus.

COMA WITH BRAIN SWELLING

- metabolic.
- hypoxic/ischaemic.
- infective.

Adapted from references 1, 3, 6



- (ii) Tympanic Membrane Displacement : ICP is transmitted via the cochlear aqueduct to the perilymph of the cochlea provided aqueduct is patent. Perilymphatic pressure may be assessed indirectly by recording displacement of the tympanic membrane during stapedia reflex contraction elicited by loud sound [5].
- (iii) Cerebral electric activity : Electroencephalography (EEG) is helpful in deciding whether cerebral metabolic depressants may be indicated in treatment of raised ICP.

MANAGEMENT STRATEGIES

(A) Emergency resuscitation and diagnosis

Patients who are rapidly deteriorating or already in coma, require immediate resuscitation with intubation and ventilation followed by

a diagnostic CT scan. An intravenous bolus of mannitol (0.5 gms/Kg over 15 minutes may be required if there is evidence of coning such as pupillary dilatation). Acute hydrocephalus demands immediate ventricular drainage. Surgical clots require removal and abscesses tapping.

(B) Post-emergency resuscitation Management

Many neuro surgical units still manage patients without ICP monitoring. ICP monitoring should be selective, based in part, on the initial CT scan. Such monitoring is very educational and greatly assists general nursing and medical care.

To treat raised ICP :

- (a) It must first be identified, avoidable factors prevented or treated and finally active treatment started.

Potential Problems Exacerbating Raised ICP

- (1) Calibration of ICP transducers and monitors particularly to check the zero reference point.
- (2) Neck vein obstruction :
 - Inappropriate position of head and neck – avoid constricting tape around neck.
- (3) Airway obstruction :
 - Inappropriate PEEP, secretions, bronchospasm etc.
- (4) Inadequate muscle relaxant :
 - Breathing against ventilation.
 - Muscle spasms.
- (5) Hypoxia/hypercapnia
- (6) Further mass lesion – rescan.
- (7) Incomplete analgesia, incomplete sedation and anaesthesia.
- (8) Seizures.
- (9) Pyrexia.
- (10) Hyponatraemia.
- (11) Hypovolaemia.



- (b) It should be treated before herniation occurs.
- (c) Knowledge of ICP may help in prognosis and counselling of relatives. In one series of diffuse head injuries where ICP persistently exceeded 20 mm Hg, almost all patients died compared with mortality rate of 20% in those where ICP could be kept below 20 mm Hg with treatment [7].

(1) Prevention of intracranial hypertension

General medical and nursing care—**avoidable factors** :

- (a) **The position of patient** should minimize any obstruction to cerebral venous drainage by head-up tilt while avoiding any fall in cardiac output. Direct measurement of global CBF (cerebral blood flow) and CPP (cerebral perfusion pressure) suggests that head-up tilt of up to 30° is safe.
- (b) **Hypovolaemia** should be avoided especially in subarachnoid haemorrhage (SAH). Dehydration, when coupled with hyponatraemia, increases risk of cerebral infarction.
- (c) **A stable circulation** must be maintained if necessary with colloids and inotropes (dobutamine or dopamine for its renal sparing action).
- (d) **Systemic hypertension**, if seen, in craniocerebral trauma, should not be treated directly with agents such as sodium nitroprusside. This drug impairs auto-regulation and increases risk of boundary zone infarction [8]. The cause of hypertension like pain or retention of urine should be looked for.
- (e) Majority of neurosurgical patients with hyponatraemia don't have syndrome of

inappropriate secretion of antidiuretic hormone (SIADH) and it is unwise to use fluid restriction to treat these.

- (f) **Seizures** must be recognized in patients who are paralysed and on ventilators. Episodes of pupillary dilatation with increases in arterial blood pressure and ICP are suggestive.
- (g) **Pyrexia** not only increases cerebral metabolism and cerebral vasodilatation but also cerebral edema. Severe hypothermia was used historically, to treat raised ICP. But now it is not used, as mild hypothermia of few °C only reduces cerebral ischaemia because of reasons that are still unclear [11].
- (h) **Hyperglycaemia** should be avoided. There is considerable evidence that cerebral ischaemia and infarction is made worse by hyperglycaemia. Use of high glucose solutions is contra indicated unless it is hypoglycaemic encephalopathy [9].

(2) **Osmotic diuretics** : Intravenous mannitol is invaluable as a first aid measure in a patient with brain herniation as a result of raised ICP. :

- (a) Mannitol removes water from both normal and edematous brain. In patients with peritumoral oedema, mannitol causes withdrawal of water mainly from brain areas having impaired blood-brain barrier as judged by T₁-MRI studies.
- (b) Mannitol administration also leads to decrease in blood viscosity and.
- (c) an increase in brain compliance.
- (d) Mannitol increases cerebral blood flow and cerebral O₂ consumption. It opens blood-brain barrier by dehydrating endothelial cells and thus causing separation of tight junctions and increase capillary diameter and blood flow.

- (e) helps in scavenging free oxygen radicals.
- (f) reduces CSF production.

In practice, mannitol tends to be given as intermittent bolus whenever patient's ICP rises above threshold of 25-30 mm Hg. The effects of mannitol may be potentiated by frusemide, the best synergistic effect being obtained by giving frusemide after 20 minutes, when mannitol bolus is finished.

It is crucial to avoid dehydration and latent hypotension with careful attention to fluid balance. For prolonged osmotherapy, some people continue to recommend glycerol [10].

(3) Hyperventilation

Hyperventilation is one of the most effective means of controlling increased ICP [14]. It achieves its effect by reducing cerebral blood flow and cerebral blood volume. CBF changes approximately 2% per mm Hg. change in PaCO₂. In normal patient, hypercapnia produces cerebral vasodilation without a rise in ICP, but when all compensatory mechanisms regarding ICP control are exhausted, even the mildest respiratory insufficiency with hypercarbia can produce severely increased ICP. Many patients with increased pressure and healthy lungs and systemic circulation often hyperventilate spontaneously down to a PaCO₂ of 30 mm Hg. when intubation is necessary, it should be done most expertly, as straining during intubation can cause a marked elevation in ICP (upto 10 mm Hg—a finding often seen during ICP monitoring).

PaCO₂ should be reduced to 25-30 mm Hg. More enthusiastic hyperventilation may precipitate cerebral ischaemia with EEG slowing and C.S.F. lactic acidosis. Thus cerebral ischaemia produced by increased ICP may be relieved only to be replaced by cerebral ischaemia

caused by cerebral vasoconstriction. When PaCO₂ is reduced by hyperventilation, blood may be shunted from normal brain, where vessels are normally responsive to changes in PaCO₂, to areas of damaged brain where vessels are maximally dilated. This phenomenon is of potential benefit since areas of ischaemia would be better perfused.

The cerebral vasoconstrictor effect of hypocapnia, induced by hyperventilation, does not persist much beyond 24-36 hrs., probably in part because the bicarbonate buffering mechanisms within the brain and cerebrovascular smooth muscle themselves readjust to return extracellular and intracellular pH nearer to the original values. This phenomenon has been confirmed in vivo in normal subjects by magnetic resonance spectroscopy.

(4) Buffer tris hydroxy methyl aminomethane (Buffer THAM)

CSF lactate accumulation and CSF acidosis occurs after head injury. Both severity of injury and the proportion of patients with poor outcome are related to high and increased CSF lactate levels. The deleterious effect of CSF acidosis can be ameliorated by I.V. THAM as suggested by Akiota *et. al.*, following epidural balloon compression of brain in dogs [16]. Evidence is accumulating both experimentally and in humans that THAM is at least as effective as mannitol in reducing experimental oedema in the brain and in lowering ICP. THAM reduces the demand for mannitol and CSF drainage.

(5) Indomethacin

It has been known since 1973 that cerebrovascular CO₂ response is blocked by indomethacin in doses that partly inhibit brain cyclo-oxygenase activity in vivo. Cerebral



venous pressure is very significantly reduced suggesting that ICP is reduced. More trials would establish a concrete role of indomethacin in patients who are having refractory raised ICP not responsive to hyperventilation and barbiturate sedation.

(6) **Continuous CSF drainage and surgical decompression**

External ventricular drainage (EVD) is a rapid procedure in emergency in a patient with acute hydrocephalus. In all cases of external drainage, CSF should be drained gradually against a positive pressure of 15-25 cm H₂O. It is an optimal method of controlling ICP in patients with SAH where cause is disturbed CSF circulation.

Removal of bone flaps or subtemporal decompression are performed much less routinely. Benign intracranial hypertension (BIH) can be treated by optic sheath fenestration and theco-peritoneal shunting.

(7) **Steroids**

The mechanism of remarkable effect of glucocorticoids such as dexamethasone on focal, relatively, chronic cerebral lesions remain incompletely understood. In traumatic cerebral contusions, when patients are put on steroids and ICP monitoring, ICP waves and compliance improve. Brain biopsy for tumour is much safer after at least three days of dexamethasone, (10 -20 mg loading dose, followed by 4 mg 6 hourly). Much controversy has surrounded the use of very high dose steroids in diffuse head injury but careful controlled trials have shown no benefit. One purported mechanism of action for steroids involves lipid peroxidation and free radicals. Cerebro-vascular effects of acute hypertension

and SAH may involve free O₂ radicals which damage endothelium. Non-glucosteroids analogues of methyl prednisolone as well as methyl prednisolone itself weakly inhibit lipid-peroxidation.

(8) **Lazaroids**

The 21-aminosteroids (antioxidant family known as **Lazaroids**) are potent inhibitors of lipid peroxidation and have Vit. E sparing effect. Lazaroid U-74006 F is undergoing large scale clinical trials in head injury and SAH.

(9) **Cerebral metabolic depressants : excitotoxic amino acid antagonists**

Brain energy metabolism is depressed more conveniently by hypnotic agents like barbiturates [6], etomidate, propofol, althesin and gamma hydroxybutyrate rather by deep hypothermia. Unfortunately, all these agents have side effects like systemic hypotension often compounded by dehydration or hypovolaemia. Short-term protection during aneurysmal surgery with barbiturate or propofol is widely used.

Magnesium chloride (Rectal administration) which was old fashioned treatment for severe head injuries, is now known to be non-competitive NMDA receptor antagonist.

(10) **Anti convulsants**

Epilepsy has long been known to raise ICP and increase the risk of cerebral ischaemia as a result of massive increase in cerebral electrical activity and oxidative mechanism resulting in jeopardising both metabolic demand and CPP. Seizures must be treated aggressively.

Management in Children

The management of raised ICP in childhood must take into account a number of factors [1].

The critical values for ICP, ABP and CPP are lower, the younger the child. Normal ICP in the new born is probably of the order of 2-4 mm Hg. Arterial blood pressure (ABP) at birth is about 40 mm Hg, 80/50 by one year and 90/60 during early school years.

Hyperaemia plays a greater role as a cause of raised ICP in children after head injury than adults. It is phenomenon of dysautoregulation that is more important in children because of immature blood-brain barrier.

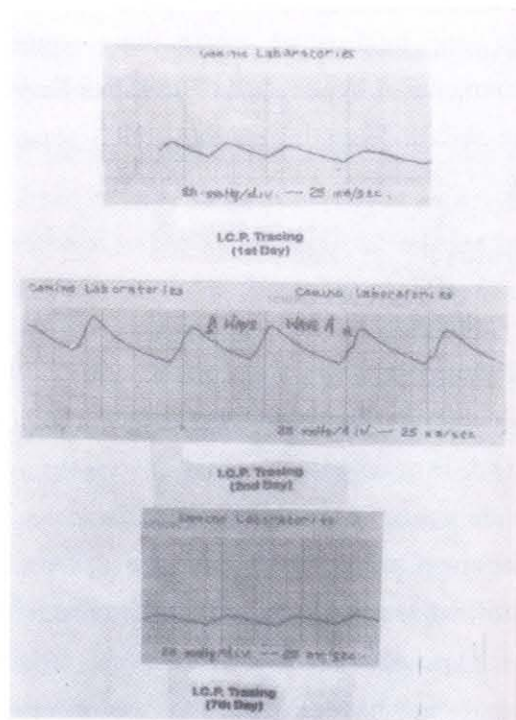
Author's Note

The author, during his tenure as Neurosurgeon in Saudia Arabia (K.S.A.), has the personal experience of using intraparenchymatous and intraventricular types of fibre-optic catheters with pressure-sensors at their tips. The ICP is amplified by an amplifier and directly transmitted to display monitor. Facility for recording of wave forms on ECG paper is also there. He has used these ICP measuring techniques in patients of head injuries having Glassgow coma scale (GCS) between 5-11. These catheters can be kept for 3 to 4 days inside the cranium through non-dominant precoronal burr hole. Zero correction is very important and must be checked every day especially in cases where there are reasons to doubt about the unexpected lows and highs of ICP. Thus, management of ICP can be scientifically carried out, the raised pressure being titrated by changes in ventilator setting and by addition of osmotic diuretics. Simple maneouvers like raising head of the patient by 20°, smooth intubations while anaesthetising cases of raised ICP, use of non-narcotic and narcotic analgesia in

paralysed patients etc. can do wonders in managing raised pressure. ICP monitoring may not be therapeutic, by itself, in head injuries but it definitely helps in management protocol.

Below is the tracing of ICP wave analysis of a young girl who after a fall, had right temporal lobe contusion. ICP monitoring was instituted. Her ICP was between 15-25 cm of H₂O. After 36 hours, suddenly ICP shot up to 35. Her ICP wave form changed and clinically, she had deterioration with right pupillary dilatation. Repeat CT showed increased brain oedema. She was put on hyperventilation-osmotic diuretic regimen and she responded.

ICP TRACING



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