REVIEW ARTICLE

Management of Raised Intracranial Pressure in Head Injury

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Craniocerebral trauma is one of the causes of raised intracranial pressure (ICP) the control and course of which have a critical bearing on morbidity and mortality in headinjured patients. At least 50 percent of patients who die from head injury do so from uncontrollable raised ICP (1).

PATHOPHYSIOLOGICAL CONSIDERATIONS

The skull is a rigid container which holds a fixed volume of brain matter, cerebrospinal fluid (CSF) and circulating blood. Neurones and glia make up 1200 to 1600 ml of the volume of intracranial content, with CSF and circulating blood each accounting for about 100 to 150 ml; there is a small amount, about 50 ml, of extra-cellular fluid in the intracranial cavity (2).

The brain, CSF and blood maintain a dynamic equilibrium, such that a change in the volume of any of them is offset by a reciprocal alteration in the amounts of the other elements such that ICP remains within normal (3). In head injury, it is the brain volume which increases, due either to the development of an intracranial haematoma or of brain oedema or a combination of both. The brain oedema is usually vasogenic in origin, due to disruption of the blood-brain barrier and characterised by protein-rich fluid. It begins in the grey matter where most of the blood vessels lie, and then spreads to the white matter which, because of its greater tissue compliance, ultimately accommodates more water than the grey matter (4).

Compensatory mechanisms come into play in the early stages of head injury to keep the ICP within normal limits (between -3 and +15 mmhg or -5 and +25 cm H20). As brain volume increases, CSF is reduced either by reduced production or increased absorption and cerebral blood flow diminishes. These changes are dependent on the elastance or stiffness of the brain. If brain volume continues to rise unchecked, these compensatory mechanisms break down and raised ICP becomes established.

MEASUREMENT OF ICP

The classical features of intracranial hypertension are often unreliable in head injury, which makes the objective measurement of ICP necessary in its management. Headache is not common in the early stages of head injury and it rarely occurs in the first few days of moderate to severe brain injury even when the patient can complain. Vomiting occurs in head injuries with or without raised ICP, and papilloedema takes a few days to develop (5).

Lumbar puncture offers a simple method of measuring ICP by recording the pressure in the lumbar subarachnoid space using a manometer. There are, however, limitations and dangers. it gives a single reading which can be misleading. It reflects the ICP only when there is free communication between the lumbar and intracranial subarachnoid spaces. The procedure can cause tentorial or cerebellar tonsillar herniation, particularly when there is posterior fossa lesion, and without reflection of the supratentorial ICP. Lumbar puncture is therefore contraindicated in head injury; *it should be done as a diagnostic procedure when there is a suspicion of meningitis.*

The basic requirements for *continuous ICP measurement*, the development of the technique, its clinical applications and the merits and demerits of the various methods used have been extensively reviewed (6, 7). The three standard methods used for continuous measurement are *extradural, subarachnoid/subdural and intraventricular.*

In the extradural monitoring, which is exemplified by the Ladd equipment, the sensor is usually positioned in direct contact with the dura (8); in infants, the Ladd sensor occasionally changes position especially if the extradural space is capacious and it tends to give higher ICP readings than the other two methods. The extradural technique, on the other hand, has the advantages of ease of insertion, simplicity of management and freedom from causing intracranial infection.

Catheters or screws are used for subarachnoid and subdural ICP measurement (10) . The catheters cause leakage of CSF and may provoke infection. The screws (Leeds or Richmond type) are easily positioned through burr holes without puncturing the brain; however, they tend to underread ICP at pressures above 20 mmhg (11).

Intraventricular pressure recording, the original method used by Lundberg (12) one of the pioneers of ICP measurement, is generally accepted to be the most accurate method of measurement. It allows repeated withdrawal and examination of CSF, if required. Its disadvantages include the difficulty of positioning the catheter if the ventricle is displaced or compressed, as may happen in severe head injuries with brain oedema, and the risks of intracranial infection and haemorrhage. Recently a bedside percutaneous technique of intraventricular ICP monitoring in children has been introduced in Glasgow, Scotland (13).

Coma Score (GCS) of 3 to 5; in those with GCS of 6 to 8 with abnormal computed tomography (CT) scans and others with moderately severe injuries with neurological deficits or deterioration (14). It is also indicated in patients with initially normal CT scan but who are considered to be at risk of developing raised ICP. These high risk patients include those aged over 40 years; patients with systolic blood pressure of under 90 mmhg and those with motor posturing (15)

ICP is useful in prognosticating in head injury; patients

with ICP sustained over 40 mmhg rarely survive. It is also useful in controlling diuretic therapy and it may warn the clinician of an incipient expanding intracranial lesion.

TREATMENT OF RAISED ICP

A. SPECIFIC MEASURES. Some simple well known measures can correct raised ICP. Ensuring that the patient has a clear air-way and that there is no kinking of the neck with consequent jugular vein compression can relieve raised ICP.

A known cause of elevated ICP in head injury is intracranial haematoma; surgical evacuation of the latter usually corrects the ICP.

Often in head injury, a specific cause of the elevated ICP is not found, or if found it may coexist with significant brain oedema. The management of postraumatic brain oedema is summarised as follows:

B. CARE OF BRAIN OEDEMA

1. READ ELEVATION

The head-end of the bed is raised to about 30 degrees, taking care not to kink the neck or compress the jugular vein. This position promotes cerebral venous drainage and decreases intracranial venous pressure and ICP. The measure should not be taken if there is peripheral circulatory failure.

2. HYPERVENTILATION

Controlled ventilation is one of the most effective measures of reducing pain oedema and hence of elevated ICP. The patient is sedated and placed on analgesia and muscle relaxant and connected to an automatic ventilator which is adjusted to keep the pCO2 at about 25 to 30 mmHg, (3.3-4kPa).

Mechanism. Hyperventilation lowers arterial pCO2 and causes cerebral vasoconstriction leading to diminished cerebral blood flow and reduced ICP.

Hyperventilation may reduce the rate of CSF formation (16). It may also promote a shift of water between the intracranial compartments described above and lead to excretion of water by the CSF (5).

Clinical Benefits. The beneficial effect of hyperventilation is almost immediate and most marked in the acute stage of head injury. As a rule, there is noticeable reduction in ICP within about 10 minutes of initiating hyperventilation, so that the earlier it is started in severe head injury, the better the outcome.

Limitations. The vasoconstrictive effect of low pCO2 occurs in areas of the brain with normal blood vessels and intact autoregulation mechanisms; damaged vessels do not respond as well.

If the arterial pCO2 is too low, say below 20 mmHg (2.7 kPa) for a long time, hypoxia may develop.

With prolonged hyperventilation, the cerebral blood flow, which was initially reduced, tends to return to normal; alveolar collapse in the lung can also occur. Hyperventilation need not be maintained longer than a week to 10 days; Crockard et al (17) found that if there was no improvement in the level of consciousness of the patient after that period, further hyperventilation produced patients who survived to lead a vegetative existence. If ICP remains elevated and hyperventilation is continued, the ICP may actually rise further.

3. DIURETIC THERAPY

Osmotherapy. Mannitol, with a molecular weight of 180 is the most widely used osmotic diuretic. If hyperventilation fails to bring the ICP down to 25 mmhg or 35 cm H20, mannitol should be started without delay.

It is given intravenously in a 20 percent solution, the dosage being between 1 and 3 grams per kilogram body weight, in a bolus over 10 to 15 minutes. It can be repeated in smaller dosages, the total amount given in a period of about one hour. Strict fluid and electrolyte balance must be maintained, keeping the serum osmolality below 320 m.mol/l.

Mode of Action. Mannitol establishes an osmotic gradient between plasma and the brain parenchyma to the blood. As a result, there is an over-all decrease in the volume of normal brain water, leading to a reduction of intracranial pressure.

Mannitol is particularly active in parts of the brain where the blood-brain barrier is intact; in damaged areas of the brain with deranged autoregulation, mannitol is ineffective and passes into the extravascular space (18).

RELEVANT PRACTICE POINTS

1. Raise head end of bed to 30 degrees provided there is no circulatory failure.
2. Intubate and hyperventilate to an arterial carbon dioxide tension of 25 - 30 mmHg (3.3-4kPa).
3. Give mannitol as a drip (20% solution) at 1 - 3g/kg body weight in patients with a lateralising pupil or motor response.

Limitation. Osmotherapy with mannitol has its drawbacks. The effect is short, lasting about 3 to 4 hours. When it is discontinued, a rebound rise in ICP may occur. Repeated doses become less effective and may cause extreme diuresis, severe dehydration, hypotension, tachycardia, and in some cases acidosis and renal failure (19).

Accumulation of mannitol in the damaged areas of the brain often worsens the brain oedema; excessive dosages have, as a result, been known to cause convulsions (5).

Loop Diuretic. Furosemide (Lasix) is most commonly used. It is a sulphonamide diuretic which inhibits distal tubular reabsorption of electrolytes. Its beneficial effect on raised ICP is exerted firstly by reduction of sodium and water migration across the blood-brain barrier, and secondly, by reduced formation of CSF through carbonic anhydrase inhibition in the choroid plexus.

The advantages of furosemide over mannitol include lack of rebound rise of ICP after its use and less changes in electrolytes and osmolality. It is given either alone or in addition to mannitol in a dose of 1 mg per kilogram body weight.

Other Agents Used

Urea was the first effective hyperosmotic diuretic used, given in a dosage of 1 to 1.5 gm per kilogram body weight, but has now been replaced by mannitol in clinical practice. Like mannitol, it is excreted unchanged in the urine and has a rebound effect on ICP. It also increases blood urea nitrogen and arterial blood pressure.

Glycerol, a trivalent alcohol may be given intravenously in a 10 percent solution at a dosage of 1 gm per kilogram body weight, infused at the rate of 5 ml per minute. It may also be given by mouth or through a nasogastric tube.

There is comparatively little experience with its use in clinical practice. The beneficial effect on reducing ICP lasts about 12 hours; its side effects include depletion of electrolytes and blood cells, and causing thrombosis in the vessels used for its intravenous infusion (5).

50 percent Dextrose. It has been found that some patients with severe brain oedema who are refractory to mannitol respond to intravenous administration of 50 percent dextrose (14). The pressure lowering effect of the latter is, however, shortlasting.

4. CSF DRAINAGE

This is a simple and effective method of reducing ICP. In head injured patient whose ICP is monitored through an indwelling intraventricular catheter, removal of a small amount of CSF can promptly and dramatically lower the ICP.

CSF withdrawal establishes a beneficial pressure gradient from the swollen brain to the ventricles, leading to movement of water to the ventricles and thus to the resolution of the oedema of the brain (18).

The technique has its limitations. Thus, its benefits are transient, so that repeated or continuous withdrawal has to be carried out with the attendant risk of infection. Also, the ventricular wall may collapse and consequently kink or block the catheter. The latter contingency can be prevented if the CSF drainage is carried out at an intracranial pressure level of not less than 10 to 15 mmHg.

CSF drainage is contraindicated in the presence of unilateral hydrocephalus as the procedure may worsen any existing midline shift.

5. CORTICOSTEROIDS

About three decades ago, it was observed that corticosteroids effectively reduced the brain oedema around intracranial tumours and brain abscesses and that following craniotomy (20). Thereafter, their usage was extended to the management of the brain oedema which occurs in head injury. How corticosteroids reduce brain oedema is not known exactly, but it is ascribed to preservation of cell membrane integrity and the blood-brain barrier through stabilisation of lyzosomal enzymes.

The universally employed drug is *dexamethasone* which is more potent than other steroids and relatively free of provoking salt and water retention. The initial adult dosage is 10 mg given intravenously and followed by 5 mg 6 hourly and subsequently in diminishing doses for about a week or more until there is evidence of ICP reduction. This regime is still used in many trauma centres, mostly by doctors other than neurosurgeons, with simultaneous administration of antacids to counteract that most dreaded complication of steroid therapy, namely, gastric haemorrhage.

Nevertheless, the beneficial effect of dexamethasone in reducing elevated ICP in severe head injury is now controversial. The initial animal experiments on the place of steroids in head injury treatment gave conflicting and inconsistent results (21). The claim that glucocorticoids reduced CSF production in dogs (22) was disproved by others (23). To be effective, it was suggested that corticosteroids should be given before injury but the benefits were not noticeable until about 19 to 24 hours after administration, implying that steroids cannot be useful for the immediate treatment of acute brain oedema.

The earlier clinical trials showed better clinical outcome in patients with diffuse brain injury treated with dexamethasone than those without it. The results were considered unreliable because those trials were uncontrolled. The first double blind controlled study which compared placebo with low dose and high dose dexamethasone in severe head injury showed reduced morbidity and mortality in the steroid treated patients, especially among those on megadosages (over 100 mg in the first 24 hours after head injury).

These positive claims were not supported by the prospective double blind controlled trials carried out in the last ten years (24, 25). These recent studies showed that steroids increased the susceptibility of the head injured patient to gastric haemorrhage, hyperglycaemia, pulmonary infection and negative nitrogen balance. Some series which had the opportunity of postmortem examination of fatal cases of severe head injury reported that the anatomical damage was considerable enough that at least 90 percent of the deaths could not have been prevented by steroids (26). Jooma in an extensive editorial review concluded that "dexamethasone confers no benefit to the seriously head injured patient and may infact be harmful" (21).

6. BARBITURATES

Pentobarbitone-induced coma may be useful when ICP fails to come down with the five methods described above (27).

How it works is unknown for sure, but the method was suggested by the observation that thiopentone sometimes sharply reduces ICP during anaesthesia (28). What is known is that barbiturate depresses cerebral metabolic activity and hence reduces blood flow to the normal parts of the brain, leading to more blood supply to the damaged areas.

The dosage used is 3 to 5 mg per kilogram body weight, ensuring a blood level of 2. 5 to 3. 5 mg per dl. The use of barbiturate-induced coma in reducing raised ICP is a major management procedure which needs all the sophistication of an up-to-date intensive care unit (3).

7. HYPOTHERMIA

This also acts by reducing cerebral metabolism and biochemical reactions in the brain. James et al (29) found that hypothermia of 27 to 36°C could reduce the ICP by about 50 percent; however, this pressure-lowering effect is not consistent.

In practice, hypothermia is not to be used alone; with barbiturates (400 mg pentobarbitone in adults and 150 mg in children) it has an additive effect in lowering elevated ICP unresponsive to other forms of treatment. Hypothermia is not recommended for longer than five days, otherwise, complications such as pneumonia and gastrointestinal haemorrhage may occur.

8. HYPERBARIC OXYGEN

How hyperbaric oxygenation reduces raised ICP is not known, but experimental and clinical evidence suggests that it may reduce ICP by as much as 30 percent (30). This occurs only in patients with intact cerebral autoregulation mechanisms; the method is effective only when applied within about 30 minutes of the head injury.

There are other limitations. Thus, it is difficult to nurse the head injured patient in a hyperbaric chamber. Once hyperbaric oxygenation is stopped, there may be a rebound rise in intracranial pressure. When the ICP is high, this method is ineffective.

9. SURGICAL DECOMPRESSION

The last resort for dealing with intractably high ICP in severe head injury is surgical decompression of the intracranial compartment by either subtemporal or suboccipital craniotomy or by the more radical surgical removal of parts of the calvarium. These heroic measures have largely fallen into disrepute (5).

SUMMARY

Head injury is one of the important causes of raised intracranial pressure (ICP) provoked by either intracranial haematoma or brain oedema or a combination of both. Continuous measurement of ICP is performed by either the extradural, subdural or the ventricular technique. The usual methods of reducing elevated ICP include the maintenance of an adequate of an adequate airway; prompt removal of any significant intracranial haematoma; head elevation when not contraindicated by shock; controlled ventilation; use of diuretics and sometimes of dexamethasone and where feasible CSF withdrawal by the ventricular route. In resistant cases, aggressive measures by barbiturate induced coma combined with hypothermia, hyperbaric oxygenation and surgical decompression may be used.

(References are available from the author on request)