Abstract

Objective: to review the current therapeutic approach of intracranial hypertension in pediatric patients admitted to intensive care unit.

Sources of data: bibliographic review of the subject based on Medline.

Summary of the findings: the authors noticed that some measures to control intracranial hypertension are consensual, and others remain controversial.

Conclusions: the goals of management of pediatric patients with intracranial hypertension include: normalizing the intracranial pressure, optimizing cerebral blood flow and cerebral perfusion pressure, preventing second insults that exacerbate secondary injury, and avoiding complications associated with the various treatment modalities employed.

Introduction

Intracranial hypertension (ICH) is a condition which affects many patients in intensive care units (ICU) and which has many different origins, both of the central nervous system and of a systemic nature. Intracranial hypertension is one of the most common causes of secondary cerebral lesions in children. The correlation between ICH and morbidity and mortality in pediatric patients justifies the search for an improved understanding of the pathophysiology, leading, consequentially, to more appropriate treatment.

In order to correctly treat children with ICH, continuous monitoring of cerebral function is necessary by means of clinical parameters and technological resources. Clinical examinations do not always provide sufficient information to measure the degree of ICH and some of the methods available for this evaluation require the professional to exercise caution in their use. Intracranial pressure monitoring (ICP) is the only method which is accepted indiscriminately as a safe form of increased intracranial pressure diagnosis, and also for the treatment of ICH in certain clinical situations.
Certain ICH treatment concepts are already very well defined while other aspects remain controversial.8,9

Pathophysiology

Primary cerebral lesions in the post-traumatic patient are the result of direct impacts against the cerebral tissues. A primary cerebral lesion can also occur when a patient is admitted to the ICU after a hypoxic-ischaemic event. This damage can vary from minimal to the irreparable. Secondary cerebral lesions are the result of the biochemical and cellular response to the initial insult. A chain of events occurs within the brain which contributes to the appearance of diffuse cerebral edema with lesions and cellular loss. This damage may include the loss of cerebral blood-flow auto-regulation, rupture of the blood-brain barrier, intracellular (cytotoxic) and extracellular (vasogenic) edema and ischaemic cerebral lesions. Secondary cerebral lesions worsen with the passage of time reaching their peak after around 3 to 5 days. The patient’s prognosis depends upon the severity of each phase of the injury.1

A “second insult” is different from a secondary cerebral lesion. Second insults are events (e.g. hypotension, hypoxia) to which the patient may be subjected after the primary lesion and which will determine the increase in severity of the secondary cerebral lesion and be responsible for worsened prognosis.10 There are reports of the additional effects of hypotension and hypoxia almost doubling the mortality rates in pediatric patients with Glasgow coma score of 3, after cranial-encephalic trauma.1

Intracranial pressure

The intracranial content consists of cerebral tissue (80%), cerebrospinal fluid (10%) and blood (10%).2 Maintenance of ICP at normal values depends upon the preservation of intracranial volume.11,12 Any event which provokes an increase in one of the intracranial components obliges a reduction of the others for ICP not to increase. The compensation process frequently occurs at the cost of a reduction in the volume of cerebrospinal fluid and blood, since cerebral mass is less compressible. Around 30% of the capacity to reduce intracranial volume is provided by the cerebrospinal fluid which may be dislocated to the spinal subarachnoid space or absorbed by arachnoid granulations. When compensation mechanisms have been used to capacity, an increase in ICP occurs as a result.1,11 The increase in ICP, in turn, can provoke a reduction in tissue perfusion, leading to aggravation of the cellular damage by ischaemia and resulting in brain death.1,11 One further ICP increase control mechanism in infants is cephalic perimeter growth, which does not protect them from acute ICH development.11

Normal ICP upper limit values for children are still the subject of disagreements within the literature, in contrast to those for adults which are well established. Intracranial pressure varies with age and 8 to 10 mm Hg are considered normal values for infants and ICP values below 15 mm Hg are considered normal for older children and adults.1,13 Intracranial hypertension has been defined as ICP above 20 mm Hg persisting for more than 20 minutes in adults.1

Cerebral blood flow

Alterations to cerebral blood flow (CBF) are significant to the pathophysiology of ICH, fundamentally to brain damage due to head trauma.14,15 Cerebral blood flow is reduced soon after severe post-trauma brain damage and provides a strategic focus for therapy.15

Cerebral blood flow maintains a relationship with cerebral perfusion pressure and responds to variations in average arterial pressure (AAP), arterial carbon-dioxide partial pressure (pCO2) and arterial oxygen partial pressure (pO2).12 A fall in pO2 provokes progressive vasodilation with an increase of up to 300% in CBF being possible when pO2 reaches 25 mm Hg. Carbon-dioxide provokes cerebral vasodilation. For each mm Hg that pCO2 reduces there is a corresponding reduction of 3% of CBF. Responses to alterations in pCO2 are rapid and equilibrium in reached in a matter of minutes. Cerebral blood flow is also controlled by local metabolism, with the local CBF being higher, the greater the metabolism of a specific area.12

Cerebral perfusion pressure

Cerebral perfusion pressure (CPP) is equal to the difference between AAP and ICP. The recommended CPP value to maintain an adequate cerebral blood flow level is 50 mm Hg. Cerebral perfusion pressure values below 50 mm Hg will lead to a proportional decrease in CBF.2,6

It is believed that young children can withstand CPP values below 50 mm Hg without developing ischaemia. Within certain limits it is possible to maintain CBF independently of AAP: a phenomenon known as autoregulation.11,14 Cerebral insults can compromise CBF autoregulation mechanisms.1,16,17

Traumatic cerebral lesion mechanics

The forces involved in CET of adults and children include contact and inertial forces. As the head-trunk ratio is much greater in children, the angular biomechanical forces which cause accelerations and decelerations are amplified in pediatric victims. As a result there is a greater incidence of diffuse brain damage within this age group.1

Clinical status

During the initial physical examination the focus is on a brief evaluation of neurological status, including level of consciousness and an examination of pupils. There is always a suspicion of cervical trauma with severe CET.
Clinical findings with ICH patients vary from situations in which the neurological examination is normal to those in which there are unmistakable signs of CNS compromise. In infants with ICH, a progressive increase in cephalic perimeter may be the only finding. Clinical status depends upon the speed with which hypertension builds up and the capacity to accommodate the intracranial volume within the calvarium.\textsuperscript{2,3} With children who are conscious, complaints such as headaches, vomiting, double vision, episodes of blindness and intermittent erratic movements may occur. Cushing triage, characterized by bradycardia, bradypnea and arterial hypertension may not be present with children.\textsuperscript{2} The Glasgow coma scale provides a guide for the assessment of such patients (Table 1).

If there is an increase in pressure in one of the hemispheres of the brain, uncal (transtentorial) herniation may occur; if pressure increases in both hemispheres the consequence will be central diencephalic herniation.\textsuperscript{2}

History is an important part of the initial assessment of the severity of head traumas in children. The age of the patient, determination of the height of a fall, the mechanics of impact and also the evolution since the incident of symptoms such as level of consciousness (somnolence, lethargy, coma), the presence of focal neurological symptoms, the occurrence of convulsive crises and headaches are of help in the determination of the risk of CET. Loss of consciousness, in isolation, is a poor indication of prognosis.\textsuperscript{10}

**Imaging exams**

Any child with a suspicion of ICH and who presents an altered level of consciousness, focal neurological deficit or physical signs of head trauma should undergo computerized tomography (CT) of the cranium for etiologic and topographic diagnosis of the resulting lesions\textsuperscript{10} (Figure 1). The presence of increased encephalic volume, indicated by compression or obliteration of the mesencephalic cisterns, is strongly associated with subsequent development of ICH. Other findings which are suggestive of cerebral edema are; staining of white and gray cerebral matter, loss of the subarachnoid space and ventricular compression.

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**Table 1 - Glasgow coma scale**

<table>
<thead>
<tr>
<th>Score</th>
<th>Response</th>
<th>Eyes open</th>
<th>Modified response for infants</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Spontaneous</td>
<td>Spontaneous</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>To Speech</td>
<td>To Speech</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>To Pain</td>
<td>To Pain</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Absent</td>
<td>Absent</td>
<td></td>
</tr>
</tbody>
</table>

**Best motor response**

| 6     | Obey commands | Moves spontaneously |                               |
| 5     | Localizing pain | Localizing pain (withdraws to touch) |                               |
| 4     | Withdrawal from pain | Withdraws in response to pain |                               |
| 3     | Flexion to pain (decorticate posturing) | Flexion to pain (decorticate posturing) |                               |
| 2     | Extension to pain (decerebrate posturing) | Extension to pain (decerebrate posturing) |                               |
| 1     | Absent | Absent |                               |

**Best verbal response**

| 5     | Orientated | Coos and babbles |                               |
| 4     | Confused | Irritable cries |                               |
| 3     | Inappropriate words | Cries to pain |                               |
| 2     | Incomprehensible sounds | Moans to pain |                               |
| 1     | Absent | Absent |                               |

Severe CET (Glasgow score: 3-8); moderate CET (Glasgow score: 9-12); mild CET (Glasgow score: 13-15).
Studies have demonstrated that children less than 2 years old, and particularly less than twelve months, may present normal clinical neurological examinations even when there are tomographic alterations which require surgery.18

Cerebral magnetic resonance imaging (MR) does not provide further information to indicate surgery; however it does provide significant correlation between the extent of lesions and cognitive prognosis, and, is frequently indicated to assess the extent of cerebral and brain stem lesions.10

Invasive intracranial pressure monitoring

Monitoring allows for the correct assessment of ICP and cerebral perfusion pressure, permitting customization of therapy. Continuous monitoring of intracranial pressure is indicated for patients at Glasgow ≤ 8, since increases in intracranial pressure and reductions in cerebral perfusion pressure contribute to secondary lesions. This is performed by means of the insertion of an intraventricular catheter that permits monitoring and drainage of cerebrospinal fluid (when this becomes necessary). Cerebral perfusion pressure should be maintained within normal limits, guaranteeing sufficient oxygen supply to the brain. The observed survival rate from severe CET is 94% when ICP is maintained below 20 mmHg.10

Intracranial pressure monitoring has been most widely studied and its indication most fully established for patients with severe cranial-encephalic trauma. There is a significant reduction in mortality, from 50% to 36%, as result of the employment of intensive care protocols including ICP monitoring.19 Notwithstanding, ICP monitoring can be useful in other situations, despite the absence of standardized indications, such as, for example, during the post-operative period of spontaneous hematomas and tumors and in patients with encephalitis and ischaemic vascular accidents.

The indications for ICP monitoring of patients with cranial-encephalic trauma, according to the recommendations of the Brain Trauma Foundation (2000) are: patients with severe CET and abnormal Computerized Tomography (CT) of the cranium Severe CET is defined as a Glasgow scale score of 3 to 8 after cardiopulmonary resuscitation and CT abnormalities include hematoma, contusions, edema or compromised basal cisterns.20 With adults ICP monitoring is also indicated in the presence of normal cranium CT results when two of the following factors are present: age over 40, systolic arterial pressure < 90 mmHg and abnormal motor posture (decerebrate or decorticate). For children there are no specific recommendations in cases of severe CET when cranium CT results are normal.

Types of monitoring systems

Recommendations exist for the choice of intracranial pressure monitoring system. The ideal monitoring apparatus is that which is reliable, precise, of low cost and which causes minimum morbidity to the patient.20

Currently available monitors permit pressure to be recorded by means of a ventricular drain coupled to an external transducer (e.g. any invasive pressure monitor), a transducer at the tip of an electrode (e.g. CODMAN™) or by means of fiber optic technology (e.g. CAMINO™) (Figure 2).

Ventricular drain (catheter) coupled to an external transducer - These are invasive pressure transducers coupled to the external ventricular drain, and in contact with the liquid column (cerebrospinal fluid). They can be recalibrated at any time. An obstruction of the drain makes measurement imprecise or impossible. An external transducer must be maintained in a fixed position in relation to the patient’s head to avoid measurement errors.

Transducer or fiber optic catheter at the tip of an electrode - The transducer is positioned inside the cranium. They are calibrated before insertion and cannot be recalibrated (without an associated ventricular catheter). As a consequence there is a risk of measurement errors occurring (drift), particularly if monitoring is continued for a number of days. Both systems may present measurement errors, however it appears that the electrode with a transducer at the tip is less likely to suffer from this problem over a 5 day period. The precision of these gauges can be verified by

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Figure 1 - CT of the cranium showing ICP monitoring through ventricular drainage catheter coupled to an external transducer in its tip, in patient with severe CET, multiple head injuries and frontal contusions
using the transducer electrode or fiber optic catheter together with a ventricular drain.

**Ideal intracranial location for monitoring**

The measurement of intraventricular pressure is the reference standard for ICP monitoring.

The measurement of ICP with an intraparenchymal or subdural transducer electrode with a catheter coupled to a liquid column is considered to be similar to intraventricular pressure.

The values obtained for pressure using a fiber optic catheter placed within the parenchyma or in a subdural position does not always correlate with intraventricular pressure. On the other hand, measurements taken with transducers at the tips of electrodes placed within the subdural space present large differences to those taken from the interior of the parenchyma.

Measurement by means of an epidural catheter coupled to a liquid column or with a subarachnoid bolt is less precise than intraventricular monitoring.

**Monitoring complications**

Clinically significant intracranial infection associated with ICP monitoring systems is rare. Colonization of systems increases significantly 5 days after implantation and when this is detected the removal of the system is indicated. Irrigation of systems coupled to liquid columns increases the risk of infection (from 6% to 19%). Reductions in infection rates have been reported in observational studies through the use of modified insertion techniques or prophylactic antibiotics.¹⁰

Hemorrhage occurs in around 1.4% of cases, with only 0.5% requiring surgery to drain the hematoma.²⁰

Replacement of ventricular catheters due to malfunction or obstruction occurs in only 3% of cases. In cases where ICP > 50 mmHg there is an increased risk of obstruction or loss of signal. With fiber optic catheters in ventricular or intraparenchymal positions, cases in which replacement due to malfunction is necessary vary from 9% to 40%.²⁰

Systems with catheters coupled to liquid columns and external transducers are the least expensive.

In conclusion, the external ventricular drainage catheter coupled to a pressure transducer is the most precise, reliable and low cost method of monitoring ICP. It offers the treatment option of cerebrospinal fluid drainage. The use of fiber optic catheters (e.g. CAMINO™) or of electrodes with pressure transducers at the tip (e.g. CODMAN™) positioned inside the ventricular drain offers similar benefits, but at an increased cost.
Intracranial pressure monitoring with fiber optic catheters or with electrodes with pressure transducers at the tip located within the parenchyma (intraparenchymal) is similar to intraventricular monitoring, however there is a possibility of measurement errors (drift). Epidural, subdural or subarachnoid monitoring are all less precise.

**Intracranial pressure monitoring technology rankings**
- Ventricular catheter coupled to an external transducer or to an electrode with a transducer at the tip or to a fiber optic catheter.
- Intraparenchymal transducers (electrode or fiber optical).
- Subdural: catheter coupled to a liquid column and external transducer or an electrode with a transducer at the tip.
- Subarachnoid: Bolt coupled to a liquid column and an external transducer.
- Epidural.

Currently available studies do not provide for standardized recommendations with respect of the use of prophylactic antibiotics, surgical technique and time of removal of ICP monitoring. Nevertheless it is the practice of the authors to use antibiotics throughout the monitoring period. Removal of monitoring equipment is performed between 24 and 48 hours after ICP has normalized.

**Electrophysiological monitoring**

Neurophysiological monitoring is a significant additional method for monitoring the neurological function of patients with CET.

Continuous or serial monitoring of somatosensory evoked potential and of electroencephalogram are still controversial, but have been used for early detection of sudden changes in cerebral function (e.g. the evolution of a hematoma).

Electroencephalographic studies show that a subtle slowing of the trace may be associated with a good prognosis and that the absence of variations and reactivity correlate with poor evolution.21

Some studies have shown that a bilateral absence of cortical peaks in somatosensory evoked potential reading is a strong predictor of poor functional evolution in children.22

**Treatment**

Treatment objectives with children with ICH are aimed at the prevention of a second insult which would exacerbate neural damage and accentuate secondary cerebral lesions. The effort to achieve these objectives consists of interventions aimed at reducing intracranial pressure and at maximizing cerebral perfusion pressure and the oxygen supply to the brain. Cerebral perfusion pressure and oxygen supply depend upon adequate ventilation, cardiac function and systemic perfusion.

Immediate care resulting in a correct diagnosis and stabilization of the patient is essential. The treatment routine should include measures to achieve global stabilization of the patient and also specific measures for the control of intracranial pressure.

**General Measures**

**Head positioning**

The head should be maintained in a neutral position, elevated by thirty degrees in order to optimize venous return. When it becomes necessary to move the patient, the head should be maintained aligned with the spine. Rotational movements to the right may increase ICP more than those to the left. Prone positioning should be avoided as it increases intra-abdominal and intrathoracic pressures with increased ICP as a consequence.

**Body temperature**

The objective in terms of temperature is to maintain the patient at normal body temperature, aggressively avoiding hyperthermia, since this can increase cerebral metabolism.12 Prolonged hypothermia may reduce leukocytes increasing the risk of infection and does not improve morbidity or mortality from many neural insults. Furthermore it may cause disturbances to ventricular conduction and the coagulation cascade.17,23 There are other issues related to this subject which will be further discussed below.

**Hemodynamic monitoring**

The installation of an arterial catheter is recommended to continuously measure AAP and of a central catheter to monitor central venous pressure (CVP). Hypotension should be aggressively treated with vasoactive drugs. Mild systemic arterial hypertension (SAH) should be tolerated, since this can be a compensating factor maintaining CPP. In situations where cerebral autoregulation has been lost, any change in pressure can be transmitted directly to the cerebral veins, with an increased risk of edema or ischaemia. In such cases sodium nitroprusside or beta-blockers can be employed.

**Respiratory management**

Intubation is recommended when the patient shows clinical signs of ICH, Glasgow score less than or equal to 8, respiratory suffering, hypercapnia or refractory hypoxemia.12

The rapid-sequence intubation technique (preparation, pre-oxygenation, sedation, cricoid pressure, neuromuscular blockade and oral endotracheal intubation) has proved to be safer than nasotracheal intubation or oral endotracheal intubation without neuromuscular blocking.24,25

Patient saturation levels should be maintained above 92%. PaCO2 should be maintained at around 35mm Hg (avoiding PaCO2 levels > 38mm Hg). Prophylactic
Hyperventilation should not be employed due to the risk of arterial vasoconstriction and the resultant cerebral ischaemia. A low respiratory frequency is recommended for assisted ventilation, since the increased expiratory period facilitates venous return. The use of positive end-expiratory pressure (PEEP) is not contra-indicated, however its potential for hemodynamic interference should not be ignored.\textsuperscript{12}

Hemoglobin and hematocrit should be maintained above 10 mg/dL and 30\% respectively.\textsuperscript{10}

\textbf{Sedation/Analgesia}

The patient should be maintained without pain or agitation, avoiding stimulation whenever possible. Before aspiration or intubation, the use of intravenous lidocaine 1 mg/Kg is recommended with the objective of avoiding PIC increase.\textsuperscript{12} Commonly used drugs are midazolam, morphine or fentanyl. Propofol infusion should be limited to 12 hours because of the risk of hypertension and metabolic acidosis. The use of ketamine should be avoided as it increases ICP. Sometimes it is necessary to induce muscular paralysis with agents derived from curare. Once paralyzed, the patient requires adequate attention to all corporal pressure points. Patients who have been treated with curare should be monitored continuously with electroencephalography because of the risk of a convulsive crises. Prophylaxis for venous thrombosis should be considered for older children and those requiring high doses of barbiturates or prolonged paralysis.\textsuperscript{10}

\textbf{Control of convulsive crises}

Convulsive crises may lead to hypoxemia and hypercapnia with increases in ICP and CBF. Studies with adults have shown phenytoin to be an effective prophylactic during the first week post-trauma, reducing the number of convulsive crises. For children, the use of anticonvulsants is indicated in cases of: repeated convulsive crises, a history of epilepsy, evidence of severe cortical contusions or evidence of laceration at surgery.\textsuperscript{10}

\textbf{Nutritional support}

Patient nutrition should be started early, preferably enteral. Administration should be around 30 to 60\% of the basic metabolic usage. Patients treated with high doses of barbiturates may require parenteral nutrition because of gastroparesis or protracted ileum.\textsuperscript{12}

Rigorous glycemia control should be promoted, avoiding the infusion of glucose during the first 48 hours, unless there is hypoglycemia (<75 mg/dL), in consideration of the potential risk of lactic acidosis.\textsuperscript{10}

\textbf{Fluid management}

Restriction of water intake is indicated in patients with dilutional hyponatremia.\textsuperscript{11} Furthermore electrolytes and osmolarity should be regularly monitored, taking into account the current tendency to maintain patient serum sodium content at higher levels.\textsuperscript{9} Maintain rigorous urinary output control with special attention to patients who have used/are using diuretics to avoid dehydration.\textsuperscript{11}

\textbf{Specific treatment for increased intracranial pressure}

Specific treatment for increased intracranial pressure aims at keeping cerebral perfusion pressure at 40-45 mm Hg with infants and young children and at 50-55 mm Hg with older children and adolescents. Treatment for ICH should be started if ICP: > 15 mmHg with infants, > 18 mmHg with children younger than 8 and > 20 mmHg for older children and adolescents.\textsuperscript{10}

\textbf{Cerebrospinal fluid Drainage}

This strategy is employed to reduce ICP when the patient has an intraventricular catheter. The removal of quantities of between 3 and 5 ml of cerebrospinal fluid at a time with reevaluation of PIC each time is recommended.\textsuperscript{12}

\textbf{Diuretic and osmotic Agents}

The use of osmotics and diuretic agents presupposes that the patient is maintained in euvolemia with hyperosmolarity.

\textbf{Mannitol}: Mannitol will initially cause plasma expansion reducing hematocrit and blood viscosity, increasing blood flow and the provision of oxygen to the brain, reducing ICP within a few minutes. During a second phase, serum osmolarity increases, dehydrating the cerebral parenchyma.\textsuperscript{11,12} When administered in bolus, mannitol will reduce ICP in between 1 and 5 minutes, peaking after between 20 and 60 minutes. However, when an urgent reduction is necessary, the initial dose should be 1 mg/Kg should be administered in approximately 20 minutes.\textsuperscript{12,17} Mannitol is dramatically effective, reversing signs of transtentorial herniation.\textsuperscript{8} Continuing the treatment, 0.25 to 0.5 mg/Kg should be administered every 2 to 4 hours, monitoring plasma osmolarity because of the risk of renal insufficiency. Plasma osmolarity should be maintained at around 320 mOsm/L.\textsuperscript{12} Mannitol penetrates the blood-brain barrier and, if used for prolonged periods, can cause an increase in ICP. Furthermore, doses should be reduced gradually as mannitol can cause ICH rebound.\textsuperscript{9,12}

\textbf{Furosemide}: Can be used at doses of 1 mg/Kg up to every six hours in order to reduce cerebrospinal fluid production, primarily when ICP remains elevated in spite of Mannitol use.\textsuperscript{2,11,12} It should not be used if serum osmolarity is above 320 mOsm/L.\textsuperscript{12} Some authors consider its use unnecessary.\textsuperscript{11}

\textbf{Barbiturates}

Used to control refractory ICH in hemodynamically stable patients. They reduce ICP by reducing cerebral metabolism with reduced CBF as a consequence. The factor...
limiting barbiturate use is related to the decrease in sympathetic tone, leading to peripheral vasodilation which occurs in up to 50% of patients.\(^ {11,12,26}\)

The short action barbiturate, for intravenous use, which is available in our locale is thiopental. We begin with an attack dose of 5mg/Kg over 10 minutes. Continuous infusion is then maintained at a level of between 1 and 5mg/Kg/hour, with boost doses of 2.5mg/Kg, if and when necessary. Therapeutic serum levels should be maintained between 6 and 8.5mg/dl.\(^ {12}\)

Treatment lasts for a minimum of 48 hours after ICP is controlled, and is reduced gradually. If ICP remains elevated despite adequate serum levels of the medication, or if hypotension ensues, the infusion should be suspended.

**Hyperventilation**

Hyperventilation should only be employed in situations in which transtentorial herniation is imminent (with transient dilation of pupils, abnormal posture, inexplicable hypertension or bradycardia) or in cases of refractory ICH, because of the risk of cerebral ischaemia. Reducing PaCO\(_2\) to below 30 mmHg may lead to loss of cerebral autoregulation. When hyperventilation is indicated, the objective should be to maintain PaCO\(_2\) between 30 and 35 mmHg initially and between 25 and 30 mmHg during a second phase. It is important to aggressively avoid PaCO\(_2\) \(\leq 25\) mmHg and, if control over ICP is achieved, the patient should be slowly returned to normal breathing, because of the risk of rebound ICH.\(^ {11,12}\)

**Corticosteroids**

No studies exist which show that any advantage is gained through their use. They are not recommended in clinical practice.

**Other ICH treatment strategies**

**Sodium Chloride at 3%**

Recent studies have returned to look at the use of a hypertonic sodium chloride solution (NaCl 3%) which increases osmolarity, reducing ICP and maintaining intravascular volume. It acts by creating an osmotic gradient at the intact blood-brain barrier, reducing cerebral volume. Research on animals and n children have shown that both Mannitol and NaCl 3% are effective in reducing ICP, but that the effect achieved with a hypertonic saline solution infusion is more accentuated and lasts longer. The use of NaCl 3% is recommended when ICP continues elevated (> 25 mmHg) despite maximum therapy including controlled hyperventilation and barbiturate coma.\(^ {27}\)

The objective is to raise sodium levels to 160 mEq/L and maintain osmolarity at around 330mosm/L. The administration of NaCl 3% should be performed by continuous infusion, with serum sodium control every six hours, respecting a maximum sodium increase of around 15 mEq/L/day. Peterson et al. suggest a continuous infusion of NaCl 3% varying from 0.1 to 1 ml/Kg/h, depending upon the sodium concentration and fluid ration desired. In order that rebound cerebral edema does not occur, a rapid decrease in osmolarity should be avoided. During withdrawal, a maximum decrease in serum sodium concentration of 10 mEq/L/day is recommended. In this way, there is little risk of the patient developing pontine myelinolisis, cerebral hemorrhage or renal insufficiency.\(^ {9,26,28,29}\)

**Decompressive craniectomy**

While there is work extant which proposes decompressive craniectomy in cases of refractory ICH, there is no standardized recommendation for its use. Studies reveal better results when it is employed at an early stage (< 48 hours), in selected cases.\(^ {30}\)

**Mild hypothermia**

Mild hypothermia (32-34 °C) has been shown to be neuroprotective in experiments on animals. It reduces excitatory amino acid levels in the peri-trauma region.

Research performed on adult patients reveal conflicting results.\(^ {31}\) While some published work does not show this therapy to be of advantage, other, more recent, studies suggest benefits from its use. Work is underway with the objective of demonstrating the beneficial potential of this therapeutic method.\(^ {15}\)

**Dexanabinol**

This is a non-psychotropic, synthetic cannabinoid which is non-competitive NMDA receptor antagonist (N-methyl-d-aspartate), which also acts as a toxic radical remover and inhibits the production of tumor necrosis factor. Reductions in ICH and systemic hypotension are observed which, when added to its anti- excitatory, antioxidant and anti-inflammatory properties make dexanabinol a promising drug for the treatment of ICH. It is indicated during the first six hours of severe CET. It is currently at the clinical trial phase.\(^ {15}\)

Figure 3 presents the algorithm of management of intracranial hypertension.

**Morbidity and mortality**

During the last twenty years there has been a significant reduction in morbidity and mortality associated with head trauma in children. However, they remain at higher levels than with adults, peaking among infants.\(^ {10}\)
GLASGOW ≤ 8
Abnormal brain CT scan

ICP monitoring
Maintain CPP > 50 mmHg

Intracranial hypertension (ICP > 20 mmHg)

First line interventions
1. 30° head elevation
2. Sedation
3. PaCO₂ between 35-38
4. O₂ saturation = 92%
5. CSF drainage

High ICP → Consider CT

Second line interventions
1. Mild hyperventilation (PaCO₂ between 30-35 mmHg)
2. Mannitol
3. Barbiturate

High ICP → Consider CT

Third line interventions
1. Hyperosmolar saline solution (NaCl 3%)
2. Mild hyperventilation (PaCO₂ < 30 mmHg)
3. Decompressive craniectomy
4. Mild hypothermia (32-35°C)

High ICP → Consider CT

Figure 3 - Algorithm of management of intracranial hypertension

Conclusions

The objectives of intracranial hypertension management of children include: criteria based monitoring of intracranial pressure, permitting attempts to normalize intracranial pressure, optimize cerebral blood flow and cerebral perfusion pressure, prevent imbalances which exacerbate secondary lesions and avoid complications associated with treatments employed. Despite significant advances in understanding of the mechanisms which cause secondary lesions, long-term prognosis for infants and children who have suffered CET remains poor.


