Paediatric Head Injuries – a Review

Severe traumatic brain injury (TBI) in children is a significant cause of morbidity and mortality worldwide. It involves a specialised area of care, in specialised centres, led by a dedicated multidisciplinary team with adequate clinical expertise. The application of protocol-based guidelines designed primarily for adults to the field of paediatrics, remains challenging. In fact, children have been largely excluded from the ongoing, international, randomised control trials, including the RESCUEicp trial, the DECRA trial and the Eurotherm3235 trial, with the lower age limits for inclusion being 10, 15 and 16 years respectively. The NICE guidelines for the triage, assessment, investigation and early management of head injury include infants (<1 year) and children (1-15 years). This paper describes the surgical and non-surgical treatment options applied to the paediatric population in the management of traumatic brain injury.

Epidemiology

Around 1 million patients or 0.3% of the population seen in emergency departments in the UK with head injuries per year. Almost half are children under 16 years of age, who account for 30% of head injury admissions, with the majority (80%) being treated on a general paediatric ward. The mortality rate is 25 per 100,000 in North America and 9 per 100,000 in the UK, accounting for 1% of all deaths, but up to 15-20% of deaths between the ages of 5 and 35 years. Incidence and mortality rates vary with age and gender with peaks occurring in children at the age of school entry. The two age groups at highest risk for TBI are the 0 to 4 year olds and 15 to 19 year olds.

Falls are the most commonly observed cause of minor head injuries in children and adolescents, followed by motor vehicle accidents, pedestrian and bicycle accidents, sports related trauma and child abuse.

Types of head injury

A force applied to the skull may be distributed evenly throughout the skull without causing a skull fracture (closed head injury) but damaging the less rigid brain tissue. When the skull vault is fractured it is described as an open head injury. Open head injuries are much less common in children. Focal injury refers to localised contusions or haematomas as opposed to diffuse axonal injury and hypoxic-ischaemic injury. Pathology following paediatric traumatic brain injury is age-dependent with subdural haematomas and diffuse axonal injury being more common than focal injuries in children and young adults.

Primary brain injury is inflicted at the time of trauma. Contusions may occur at (coup) or opposite (contre coup) the site of impact, the latter typically involving the frontal or temporal lobes. Contusions may bleed, giving rise to an intracerebral and/or subarachnoid haemorrhage. Sudden minor blows (classically antero-posterior) may tear the superior cerebral veins as they enter the superior sagittal sinus resulting in a subdural haematoma. In infants, subdural haematoma is usually widespread, bilateral and thin unlike the more localised subdural in adults.

Brain injury is followed by evolution of the injury when the decreased cerebral blood flow is insufficient to sustain normal neurological functioning. Secondary insults such as elevated intracranial pressure, hypovolaemia, hypoxia and hyperthermia may lead to secondary brain injury. The management of traumatic brain injury is aimed primarily at preventing these potential secondary insults.

Pathophysiology

The Mono-sailure doctrine describes the skull as a rigid structure with a fixed capacity. A rise in intracranial pressure (ICP) is initially compensated for by cerebral autoregulatory mechanisms. When this mechanism is overwhelmed, cerebral hypopfusion triggers a cascade of mechanisms leading to aggravation of the initial injury with worsening oedema and culminating in cerebral herniation. Following the primary brain injury, axons and glial cells, in particular those of the corpus callosum, basal ganglia and periventricular grey...
Management of paediatric head trauma:

Initial management

Stabilisation of the airway, breathing and circulation together with three-point cervical spine immobilisation should be instituted in any child following traumatic brain injury (TBI). The risk of a clinically significant brain or cervical spine injury should then be assessed, using a modified paediatric Glasgow Coma Scale (GCS) in the non-verbal child (Table 1). Assessment should establish the need for CT scanning of the brain and/or cervical spine in a child ≤16 years of age.

Fluid resuscitation and maintenance of blood pressure is essential to ensure cerebral perfusion and resolve shock. Fluid resuscitation should be initiated in all cases of TBI using 10mls/kg boluses of 0.9% saline followed by re-assessment.

Mechanical ventilation with endotracheal intubation should be instituted in any patient with a GCS ≤8 or when there is a strong suspicion of injury despite normal plain films (AP and lateral views for children <10 years of age, without an A/P peg view) or if plain films are inadequate, should have CT scanning of the cervical spine within an hour of presentation or when sufficiently stable.

Elevating the head to 30º in midline position decreases venous obstruction and may help to control ICP. Sedation and analgesia are also important adjuncts to minimize increases in ICP. Pain and stress increase metabolic demands and increase blood pressure and ICP. Analgesics and sedatives such as fentanyl and midazolam are commonly used. Neuro-muscular blockade is useful to avoid shivering with cooling and allows better control of ventilation by preventing patient-ventilator asynchrony.

Cerebral perfusion pressure (CPP)

Compared to children without TBI, children with TBI have lower cerebral blood flow (CBF) and cerebral hypoperfusion (CBF < 20-25ml/100g/min) is the dominant derangement. In addition, there is impairment of cerebral maturation, with a disruption of their cytoskeletal proteins resulting in failure of axonal transport. Surrounding this area of primary injury is a labile area – a penumbra, where brain tissue is potentially salvageable but prone to secondary insults. A large amount of cytokines, including interleukin-6, IL-10 and soluble adhesion molecules, is released following the primary injury.

Higher concentrations of IL-10 were observed in children younger than four years following traumatic brain injury. The initial high concentrations are probably detrimental to the penumbra zone, but might have a neuroregenerative effect in the long term and at lower concentrations.

High concentrations of glutamate are found in synapses following brain injury. Sodium and calcium channels are activated leading to an accumulation of sodium and water intracellularly. Hypoxia leads to energy depletion thus disabling the sodium-potassium ATPase pump and reducing calcium exchange. The sodium-potassium ATPase pump is required for normal neuronal function and vital for life. This process is accelerated by leakage of calcium from the endoplasmic reticulum and mitochondria following disruption of the cell membrane after injury. This excitotoxic effect culminates in necrosis or apoptosis, depending on which receptors are activated – NMDAR in necrosis and non-NMDAR in apoptosis. Animal studies have shown that immature neurons are more vulnerable to these effects suggesting that the response to excitotoxicity and apoptosis appears to be age-dependent.

Table 1: The Paediatric Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Category</th>
<th>Best Response</th>
</tr>
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<tbody>
<tr>
<td>Eye opening</td>
<td>4</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>3</td>
</tr>
<tr>
<td>To speech</td>
<td>2</td>
</tr>
<tr>
<td>To pain</td>
<td>1</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>

| Motor | 6 |
| Follows commands | 5 |
| Localises pain | 4 |
| Withdraws to pain | 3 |
| Flexes to pain | 2 |
| Extends to pain | 1 |

| Glasgow Coma Scale | |
| Best possible score | 15 |
| Worse possible score | 3 |

| If tracheal intubation then verbal | |
| Best possible score while intubated | 10T |
| Worse possible score while intubated | 2T |

Table 2: Indications for CT scanning of the head in children (1-16 years of age) NICE guidelines

<table>
<thead>
<tr>
<th>Indication</th>
<th>Example</th>
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<tbody>
<tr>
<td>Witnessed loss of consciousness lasting &gt;5 minutes</td>
<td></td>
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<tr>
<td>Amnesia (antegrade or retrograde) lasting &gt;5 minutes</td>
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<tr>
<td>Abnormal drowsiness</td>
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<tr>
<td>Three or more discrete episodes of vomiting</td>
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<tr>
<td>Clinical suspicion of non-accidental injury</td>
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<tr>
<td>Neurological deficit</td>
<td></td>
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<tr>
<td>Focal neurological deficit</td>
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<tr>
<td>Age ≥1 year: presence of bruise, swelling or laceration &gt;5cm on the head</td>
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<tr>
<td>Dangerous mechanism of injury (high-speed road traffic accident either as a pedestrian, cyclist or vehicle occupant, fall from &gt;3m, high-speed injury from a projectile or an object)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1: Pathophysiology of brain swelling

Figure 2: Protocol-driven therapies
autoregulation following traumatic brain injury in children. Maintaining optimal, age-based cerebral perfusion pressure (CPP) has been demonstrated to improve outcome. [CPP = MAP (mean arterial pressure) − ICP]. In infants and children lower levels of CPP are accepted (40-70mmHg) with CPP values outside this range associated with an unfavourable outcome. Aggressive management of CPP can lead to good neurological outcomes despite extremely high ICP. Normovolaemia with augmentation of the CVP to 8 to 10cm should be ensured prior to commencing inotropes. Dopamine has been associated with an increase in cerebral oedema and suppression of most anterior pituitary-dependant hormones and norepinephrine may be the most suitable catecholamine to maintain or restore adequate cerebral perfusion.

Intracranial pressure (ICP) control and cerebral perfusion pressure (CPP) manipulation have significantly reduced the mortality but not the morbidity rate. The prevention and aggressive treatment of cerebral hypoxoxygenation and control of ICP with a PbtO2-directed protocol (>25mmHg) has been shown to reduced the mortality rate after TBI in major trauma but more importantly, resulted in improved 6-month clinical outcomes over the standard ICP/CPP-directed therapy.

**Hypoxia and hyperventilation**

The association between observed early hypoxia (SpO2 <90% or <7.9 kPa (60mmHg)) and poor outcome is well documented. This association is not as strong as hypotension with mortality only slightly increased in children with both hypoxia and hypotension over those with hypotension alone. Hypoxia should be avoided by maintaining PaO2 >12KPa mmHg and SaO2 ≥98%. PEEP should be limited since increases in intrathoracic pressure may impede jugular venous drainage and diminish compensatory mechanisms based on the Munro–Kellie doctrine. Hyperventilation leads to decreased cerebral blood flow with a 1mmHg change in the [PaCO2] decreasing CBF by 3% in regions of intact autoregulation. The decreased cerebral blood volume lowers the ICP Profound hyperventilation with PaCO2 <35mmHg may be instituted as second tier therapy in the case of refractory intracranial hypertension after a cerebral CT scan. However, routine, severe hyperventilation presents a significant risk for brain hypoxia. Targeting brain tissue oxygen tension (PbtO2) to >25mmHg has shown to improve both the mortality and morbidity after TBI.33 Similarly, if jugular venous oxygen tension (SvO2) is reduced below 60%, a state of relative ischaemia exists, cautioning against any further reduction of PaCO2. There may also be an association between mortality and a lower mean PbtO2, and hence compromised cerebral oxygenation, in patients following subarachnoid haemorrhage (SAH).

**Hyperosmolar therapies**

Hyperosmolar therapies include intravenous mannitol (2.5-5mls/kg of 20% mannitol over 20mins) and hypertonic saline (3-5mls/kg of 3% saline). Both induce a shift of fluid from the intracellular to the extracellular space across an osmotic gradient leading to decreased cerebral oedema, relative brain dehydration and decreased ICP. Hypertonic saline (HS) is as effective as mannitol for the treatment of raised intracranial pressure in traumatic brain injury in children. However, HS may produce less “rebound” intracranial hypertension when compared to mannitol as it cannot be easily removed from intracellular space. HS does not cause obligatory osmotic diuresis and hence is likely to preserve or augment plasma volume rather than deplete it. Mannitol may precipitate acute renal failure at extremes of serum osmolality (limiting multiple doses) and may not be excreted in oligo-anuria whereas HS is renoprotective.

HS directly increases plasma Na+, measurable changes in blood osmolality can be easily monitored by measuring plasma Na+. Plasma sodium levels of >150mmol/L up to 170 mmol/L have been targeted to control ICP (<20-25mmHg) although levels >160mmol/L (with serum osmolalities >320mosm/L) have been associated with reversible renal insufficiency. 3mls/kg of 3% saline may increase plasma Na+ by approximately 2-3 mmol/L. A greater increase may occur if a large diuresis occurs. Conversely, the effect of mannitol on plasma osmolality can only be estimated using an osmole gap.

When evaluating the potential side effects of continuous hypertonic 3% saline (CHS) as maintenance fluid in patients with brain injury the incidence of moderate hypernatraemia (Na+ >155 mmol/L) and severe hypernatraemia (Na+ >160 mmol/L) was found to be significantly higher in the CHS therapy group than in the normal saline group and moderate and severe hypernatraemia was associated with a higher risk of elevated blood urea nitrogen and creatinine levels. However, CHS therapy was not associated with an increased rate of infection, deep vein thrombosis, or renal failure. Therefore CHS administration in patients with severe injuries was considered to be safe as long as sodium levels are carefully monitored.
Induced hypothermia

Oxidative stress contributes to secondary damage after traumatic brain injury. With normothermia (36.5-37.5°C), the mechanisms of secondary injury are lessened with decreased cerebral metabolism, inflammation, lipid peroxidation, axonal injury, excitotoxicity, cell death, and acute seizures. Hypothermia should be avoided in the acute period following paediatric severe TBI.

Hypothermia attenuates oxidative stress after severe TBI in infants and children. Moderate hypothermia (32-33°C) initiated within the first 24 hours after severe TBI and maintained for 48 hours will have a protective effect on the paediatric brain and can be done safely although arrhythmias and rebound elevated ICP with rewarming have been observed. In a separate larger randomised control trial in children following severe traumatic brain injury, hypothermia therapy initiated within eight hours after injury and continued for 24 hours did not improve outcome and may even cause harm. The value of early induced hypothermia (32-33°C) started early after traumatic brain injury (≤72 hours) is currently being assessed in the Eurotherm3235 trial. Although the minimum recruitment age for this trial is 16 years, the same protocol could be applied to children if modified. Current recommendations are to induce hypothermia in the delayed phase as a rescue therapy for refractory intracranial hypertension rather than early after TBI as a neuroprotectant.

Surgical management

The aim of surgery is to reduce the intracranial pressure either by reducing the volume of the skull contents or by modifying the skull itself in order to prevent further damage and promote healing of the peneumbra. Indications for surgery are both clinical and radiological. A subdural haematoma thicker than 10mm on CT scan, or midline shift greater than 5mm, should be considered for surgery. Asymmetrical or fixed, dilated pupils in a comatose (GCS≤5) patient more than two points between injury and admission, or an ICP >20mmHg despite a smaller subdural as well as a midline shift should also be considered for surgical intervention. Contusions alert the surgeon to the possibility of mass expansion. The site of the contusion is also significant as temporal lobe lesions are more likely to cause uncal herniation with compression of the midbrain.

Barbiturate coma

Barbiturates should only be considered as second line therapy in cases of refractory elevated ICP. Thiopentone produces a dose dependent reduction in CBF and cerebral metabolic rate until the EEG becomes isoelectric (flat) or shows burst suppression, at which point no further reduction occurs despite an increase in barbiturate dose. When comparing pentobarbital versus thiopental in the treatment of refractory intracranial hypertension in patients with traumatic brain injury, ICP was controlled in 18% and 50% of patients, respectively without any statistically significant difference between groups in the rate of infectious complications or hemodynamic compromise. High-dose barbiturate therapy may be considered in haemodynamically stable patients with salvageable severe head injury and refractory intracranial hypertension. Thiopentone appears to be more effective than pentobarbital in controlling intracranial hypertension refractory to first-line measures.

ICP monitoring and CSF drainage

ICP monitoring is an essential tool in the paediatric intensive care unit. With ICP monitoring, timely intervention can be applied to maintain an adequate CPP based on MAP and ICP values. ICP can be measured directly in the subdural space via a ventriculostomy drain but may be difficult to insert in view of slit-like ventricles seen in diffuse cerebral swelling and can be a source of infection despite giving precise readings. An external ventricular drain (EVD) allows removal of CSF as a means of reducing ICP in patients with intracranial hypertension. Indications for surgery are both clinical and radiological. A subdural haematoma thicker than 10mm on CT scan, or midline shift greater than 5mm, should be considered for surgery. Asymmetrical or fixed, dilated pupils in a comatose (GCS≤5) patient more than two points between injury and admission, or an ICP >20mmHg despite a smaller subdural as well as a midline shift should also be considered for surgical intervention. Contusions alert the surgeon to the possibility of mass expansion. The site of the contusion is also significant as temporal lobe lesions are more likely to cause uncal herniation with compression of the midbrain.

Electronic devices (Camino and Codman design) allow analysis of the ICP waveform in addition to ICP monitoring, which provides a useful adjunct to treatment. The ICP waveform is a modified arterial pressure waveform where the P1 peak is at the start of systole, the P2 peak is at the top of diastole, and the P3 peak is at the end of systole. The Lundberg A waveform is a modified arterial pressure waveform where the P1 peak is at the start of systole, the P2 peak is at the top of diastole, and the P3 peak is at the end of systole. The Lundberg A waveform is a modified arterial pressure waveform where the P1 peak is at the start of systole, the P2 peak is at the top of diastole, and the P3 peak is at the end of systole. The Lundberg A waveform is a modified arterial pressure waveform where the P1 peak is at the start of systole, the P2 peak is at the top of diastole, and the P3 peak is at the end of systole. The Lundberg A waveform is a modified arterial pressure waveform where the P1 peak is at the start of systole, the P2 peak is at the top of diastole, and the P3 peak is at the end of systole.
Evacuation of haematomas

Acute intracranial haematoma is best evacuated via a craniotomy with or without a bone flap removal and duroplasty especially when the patient is comatose. This provides access to potential bleeders and the traumatised brain which when severe might require a lobectomy.

Organised haematomas can be evacuated either via twist drill craniostomy or via burr hole trepanation. This can be done in conjunction with a craniectomy. Craniotomy may have a higher rate of recurrence than burr hole trepanation and a craniectomy might be a good therapeutic option for recurrence. Twist drill craniostomy is a quicker procedure with faster regression of the residual subdural effusion but there is no difference in outcome when compared to burr hole trepanation. Double burr hole drainage did not offer any further advantage, unless specifically indicated. In the paediatric population chronic subdural haematomas typically present in patients less than four months of old with a history suggestive of non-accidental injury. External drainage as a treatment option for chronic subdural haematoma is sometimes under taken but this offers no advantage. Burr hole drainage with irrigation of the haematoma cavity and closed-system drainage is currently recommended. Extensive craniotomy with membranectomy should be reserved for those with acute rebleeding with solid haematoma.

Decompressive craniectomy

The standard decompressive craniectomy procedure described in the RESCUicp trial suggests excising a bone flap of at least 12cm diameter (avoiding herniation) and opening the dura and leaving it open (possibly with a duroplasty). When the swelling is predominately unilateral and there is midline shift, the craniectomy should be on the side of the swelling and should decompress the temporal lobe. Bifrontal decompressive craniectomy with bilateral U-shaped opening of the dura is recommended for diffuse brain swelling. Maximum decompression is achieved by ligating and dividing the superior sagittal sinus and the falx (anteriorly). If the frontal sinus is inadvertently opened it should be cranialised by excising the posterior wall, stripping the mucosa and plugging it with pericranium, free muscle and/or tissue glue.

The ICP should be monitored even post-craniectomy (Figure 6) using a burr hole or bolt at least 3cm away from the bony edge of the craniectomy. Tight bandaging or positioning the patient’s head on the craniotomy side after decompression should be avoided. Cranioplasty is recommended within six months following decompressive craniectomy.

Craniectomy can be used alone or in combination with a barbiturate coma. Outcome is improved when decompressive craniectomy is used to treat raised intracranial pressure in children. Conversely according to a nine-year retrospective study from Utah there was a high rate of mortality in those paediatric patients where the craniectomy was done for raised ICP only (compared to raised ICP and evacuation of a mass lesion). The value of a decompressive craniectomy for the management of traumatic brain injury is currently being assessed in the RESCUicp trial in children ≤10 years of age.

Conclusion

Current management of paediatric traumatic brain injury needs to be examined and new trials instituted to include the paediatric population, in order to provide standardised guidelines, which achieve the best possible outcome in terms of morbidity and mortality. Equally important is the environment in which paediatric traumatic brain injury occurs. When dealing with trauma in children, a clinician must be alert to the possibility of non-accidental injury and investigate appropriately with the involvement of a multidisciplinary team. Safeguarding children by preventing accidental and identifying non-accidental injury remains a key factor in management.

REFERENCES


