Traumatic brain injury in children

This article provides a clinical overview of traumatic brain injury in children. It concentrates on the current management guidelines from initial assessment in the accident and emergency department through to specialist critical care.

Injury is the main cause of morbidity and mortality in children around the world (Segui-Gomez and Mackenzie, 2003). In the UK, more than 500 000 children are brought to accident and emergency departments every year with head trauma; of these children 10% are subsequently admitted to hospital (Marcovich, 2006). The death rate is 5.3 per 100 000 children (Jennett, 1998), with mortality as high as 23% in more severe cases of traumatic brain injury (Parslow et al, 2005). Even mild traumatic brain injury can lead to long-term sequelae which may place significant strain on the families involved (Hawley et al, 2003). The most common cause of traumatic brain injury in children is road traffic accidents, followed by falls, inflicted injury and sports-related trauma (Broman and Michel, 1995).

Inflicted trauma or non-accidental injury accounts for between 7 and 10% of head injury in children under the age of 2 years. Violent shaking has been the proposed mechanism of injury to the brain and cranial vault giving rise to the term 'shaken baby syndrome'. It is vital for clinicians to have a high index of suspicion when dealing with such cases. Clinical signs (Tung et al, 2006) that point towards inflicted head trauma in children include:

1. Interhemispheric subdural haematoma
2. Sheer injuries
3. Diffuse axonal injury
4. Contusional white matter tears
5. Retinal detachment.

Pathology of traumatic brain injury in children

Traumatic brain injury is defined as a blow or jolt to the head or a penetrating head injury that disrupts the normal function of the brain. This includes concussion (*Table 1*), cerebral contusion (bruising of the cortical tissue) and diffuse axonal injury (widespread shearing injury of the white matter). The underlying mechanism of traumatic brain injury is classified as either primary or secondary.

Dr AAB Jamjoom *is Foundation Year 1 Doctor, Lincoln County Hospital, Lincoln LN2 5QY,* **Dr RK Gupta** *is Foundation Year 1 Doctor, University College London Hospital, London, and* **Professor UP Devkota** *is Professor of Neurosurgery in the National Institute of Neurological and Allied Sciences, Kathmandu, Nepal*

Correspondence to: Dr AAB Jamjoom

Primary brain injury

This is the injury resulting from the traumatic event. There are two reasons that the paediatric brain is more susceptible to injury than the adult brain. Children have a higher head:torso ratio which amplifies biomechanical forces during injury, and also the paediatric brain has a higher water content and is less myelinated leading to a more diffuse axonal injury compared to adults. Prevention of primary brain injury is best achieved through aggressive road safety education campaigns.

Secondary brain injury

These are physiological and biochemical events that follow primary brain injury (Mazzola and Adelson, 2002). These secondary events may or may not be complicated by second insults which exacerbate secondary brain injury and worsen prognosis. Such second insults include:

Reduced cerebral perfusion

Cerebral perfusion pressure is dependent on mean arterial pressure and intracranial pressure and is calculated as mean arterial pressure – intracranial pressure. Therefore, anything that reduces mean arterial pressure (e.g. hypotension) or increases intracranial pressure (e.g. brain oedema or intracranial haemorrhage) will reduce cerebral perfusion and worsen secondary brain injury.

Hypoxia and hypercapnia

A partial pressure of oxygen of less than 8 kPa impairs brain oxygenation (Ganong, 1993). Hypoxia following head injury may be caused by airway obstruction or a reduction in respiratory drive caused by central nervous

Table 1. Symptoms and signs of concussion

Headache
Dizziness
Confusion
Nausea and vomiting
Noise and light sensitivity
Lethargy
Irritability
Anxiety
Difficulty concentrating
Depression

From Atabaki (2007)

damage. Hypercapnia has a vasodilatory effect on cerebral arterioles which increases the intracranial pressure and in turn reduces the cerebral perfusion pressure. Both hypoxia and hypercapnia exacerbate secondary brain injury.

Pyrexia

There is a 5% increase in cerebral metabolism for every 1°C increase in body temperature (Bissonnette, 1994). This causes a relative hypoxia and increase in intracranial pressure, thus worsening secondary brain injury.

Hyperglycaemia

Hyperglycaemia can induce lactic acidosis which has a negative effect on the injured brain and can lead to a worse outcome (Lam et al, 1991).

Hyponatraemia

This is caused by inappropriate antidiuretic hormone secretion or by cerebral salt wasting in traumatic brain injury. This electrolyte imbalance leads to cerebral swelling which in turns raises the intracranial pressure and reduces cerebral perfusion.

Associated head injuries with traumatic brain injury

The term head injury usually refers to traumatic brain injury. However, it also encompasses associated injuries to other structures (such as the scalp and skull) as any transfer of energy to the head can lead to damage to surrounding tissues. It is vital to keep the anatomy of the head in mind when classifying these injuries.

Skin lesions

Trauma to the head can lead to skin lacerations or abrasions. It is important that scalp wounds are debrided and that any underlying skull pathology is sought. Owing to the relatively larger scalp surface area in children, significant blood loss from scalp lesions can lead to shock, but other causes of internal bleeding must first be excluded in such cases.

Skull fractures

There are three main types of skull fractures:

1. Linear skull fractures account for about 75% of all skull fractures (Atabaki, 2007). These fractures can be managed with pain relief and outpatient observation if uncomplicated
2. Depressed skull fractures are brought about by higher energy impacts and tend to require operative repair
3. Basal skull fractures. A number of clinical signs may be apparent in the presence of basilar skull fractures. These may include Battle's sign (bruising behind the ear), panda sign (periorbital bruising), CSF leakage from the ear or nose and haemotympanum (accumulation of blood behind the tympanic membrane). This type of fracture requires close hospital observation.

Intracranial haemorrhage

Haemorrhage into the cranial vault can either be extradural, subdural, subarachnoid or intraparenchymal. Subdural haematomas are most common in children experiencing traumatic brain injury. Such bleeding arises from bridging vessels between the brain cortex and venous sinuses. Classically, these haematomas have a concave appearance on computed tomography scan and require neurosurgical evacuation. Extradural haemorrhage typically arises as a result of fracture of the temporal bone causing laceration of the middle meningeal artery or vein. It is often associated with a 'lucid period' after which the patient deteriorates rapidly. It is therefore vital to observe these patients and to make an immediate neurosurgical referral.

Assessment of a head-injured child in the emergency department

Clinical assessment

Initial management of the child with a traumatic brain injury involves stabilization of the airway and cervical spine, breathing and circulation via the ABCDE approach as dictated by a standard practice such as Advanced Trauma Life Support protocols. Once the patient is stable the following clinical assessments should be undertaken:

- Paediatric Glasgow Coma Scale (*Table 2*)
- Pupillary diameter and reactivity
- Brainstem reflexes

Table 2. Paediatric Glasgow Coma Scale

	Score	Infant/non-verbal child		Verbal child
Eye opening	4	Spontaneously		Spontaneously
	3	To speech		To verbal command
	2	To pain		To pain
	1	No response		No response
Motor response	6	Normal spontaneous movement		Obeys command
	5	Withdraws to touch		Localizes to pain
	4	Withdraws to pain		Flexion withdrawal
	3	Abnormal flexion		Abnormal flexion
	2	Extension		Extension
	1	No response		No response
		<2 years	2–5 years	> 5 years
Verbal response	5	Cries appropriately/coos	Appropriate words	Orientated
	4	Irritable crying	Inappropriate words	Confused
	3	Inappropriate screaming/ crying	Screams	Inappropriate words
	2	Grunts	Grunts	Incomprehensible words
	1	No response	No response	No response

From Marcoux (2005)

- Neurological examination looking for focal deficits and signs of raised intracranial pressure (*Table 3*).

The Glasgow Coma Scale is an important aspect of the assessment. Traumatic brain injury may be classified based on the Glasgow Coma Scale as mild (Glasgow Coma Scale 13–15), moderate (Glasgow Coma Scale 9–12) and severe (Glasgow Coma Scale ≤8) following cardiorespiratory resuscitation. Owing to the complexity of applying the Glasgow Coma Scale to children a study suggested that using the Glasgow Coma Scale motor subscore had at least the same predictive ability as the total Glasgow Coma Scale (Van der Voorde, 2008).

Further, the mechanism of injury should be established in order to aid the identification of possible 'talk and die' cases. These patients represent an important population who initially present alert and able to talk following traumatic brain injury before subsequently deteriorating and dying as a result of preventable intracranial pathology. More severe traumatic brain injury (Glasgow Coma Scale score ≤8) necessitates the involvement of an anaesthetist or critical care physician early in the management of the patient (National Institute for Health and Clinical Excellence, 2007).

Table 3. Symptoms and signs of raised intracranial pressure

Headache
Vomiting
Drowsiness
Cushing response – hypertension and bradycardia
Abnormal pupil responses – constriction followed by fixed dilatation
Papilloedema

Table 4. Criteria for selecting children (<16 years) for computed tomography scanning of the head

Witnessed loss of consciousness lasting >5 minutes
Amnesia (antegrade or retrograde) lasting >5 minutes
Abnormal drowsiness
3 or more discrete episodes of vomiting
Clinical suspicion of non-accidental injury
Post-traumatic seizure but no history of epilepsy
Age > 1 year: Glasgow Coma Scale <14 on assessment in the emergency department
Age < 1 year: Glasgow Coma Scale (paediatric) < 15 in the emergency department
Suspicion of open or depressed skull injury or tense fontanelle
Any sign of basal skull fracture (haemotympanum, 'panda' eyes, CSF leakage from ears or nose, Battle's sign)
Focal neurological deficit
Age > 1 year: presence of bruise, swelling or laceration > 5 cm on the head
Dangerous mechanism of injury (high-speed road traffic accident either as pedestrian, cyclist or vehicle occupant, fall from >3 m, high speed injury from a projectile or an object)

From National Institute for Health and Clinical Excellence (2007)

Radiological studies

Computed tomography scanning is the investigation of choice in traumatic brain injury (Marcoux, 2005). Within the UK, there are set criteria for selecting children for computed tomography scanning of the head (*Table 4*). Computed tomography scans can demonstrate skull fractures, contusions and intracranial haematomas which may require involvement of the neurosurgeons (*Table 5*). When computed tomography scanning is not indicated, a plain X-ray trauma series may be undertaken which includes a lateral cervical spine, chest and pelvic X-ray. Magnetic resonance imaging does not add to the diagnostic work up of traumatic brain injury, but it does have prognostic value with regard to cognitive outcome in traumatic brain injury (Suh et al, 2001).

Management of significant traumatic brain injury in children

The principal aim of management in children with significant traumatic brain injury is the prevention of second insults in order to minimize secondary brain injury. As discussed, these second insults include hypoxia, hypercapnia, reduced cerebral perfusion, pyrexia, hyperglycaemia and hyponatraemia. Prevention of such second insults involves the use of general measures, monitoring techniques, surgery and intracranial pressure-specific therapies.

General measures

Cardiorespiratory resuscitation

Cardiorespiratory resuscitation aims to prevent or treat hypotension, hypoxia and hypercapnia in order to optimize cerebral perfusion pressure and oxygen delivery to the brain. In the absence of impending transtentorial herniation, this should form the cornerstone of severe traumatic brain injury management and should be carried out early and aggressively in both the pre-hospital and hospital settings. Hypotension in children may be

Table 5. When to involve neurosurgeons

Surgically significant abnormality on imaging, e.g. subdural haematoma
Persisting coma (Glasgow Coma Scale <8) after initial resuscitation
Unexplained confusion for more than 4 hours
Deterioration in Glasgow Coma Scale after admission (pay greater attention to motor response deterioration)
Progressive focal neurological signs
Seizure without full recovery
Definite or suspected penetrating injury
CSF leak

defined as systolic blood pressure below the 5th centile for age (estimated by 70 mmHg + (2 x age)). However, since hypotension may occur late in children, patients should be examined thoroughly for signs of hypovolaemia. Treatment involves the use of intravenous fluids and, if necessary, vasopressors. Children with Glasgow Coma Scale below 9 should be intubated and ventilated with initial 100% oxygen in order to secure the airway and prevent hypoxia and hypercapnia.

Positioning
Patients should be positioned with 30° head elevation in the neutral position to reduce intracranial pressure while maintaining mean arterial pressure (Feldman et al, 1992). Jugular venous outflow obstruction, for instance by tight endotracheal ties, should be avoided as this leads to an increase in intracranial pressure (Mazzola and Adelson, 2002).

Fever control
Since hyperthermia (temperature ≥38.5°C) increases risk of secondary brain injury as described earlier, fever must be adequately controlled in patients with severe traumatic brain injury. Febrile patients also require frequent septic screening and there should be a high index of suspicion for meningitis in cases of CSF leak or penetrating injury.

Sedation and analgesia
Opioids and benzodiazepines are frequently used to provide sedation and analgesia. The aim is to reduce anxiety and pain, which are associated with increased intracranial pressure, while also facilitating mechanical ventilation (Orliaguet et al, 2008). Long-term sedatives, which prolong intervals between useful neurological examinations, and prolonged use of propofol, which may lead to propofol infusion syndrome, should be avoided (Sabsovich et al, 2007).

Anticonvulsant therapy
Anticonvulsants may be used in the prevention of early post-traumatic seizures (within 7 days), particularly in high-risk groups such as infants. Seizures should be treated promptly because of the associated increase in intracranial pressure (Temkin et al, 1991).

Monitoring techniques

Intracranial pressure monitoring
Intracranial pressure should be monitored in all children with severe traumatic brain injury. This may be achieved through a variety of methods, but the placement of a ventricular catheter is currently thought to be the most reliable (Adelson et al, 2003). As well as its diagnostic use, this method also allows the drainage of CSF in cases of raised intracranial pressure. Complications include the technical difficulty of insertion, particularly in cases of profound cerebral oedema, and risk of infection, which is proportional to length of time in situ (Orliaguet et al, 2008).

Transcranial Doppler ultrasonography
Transcranial Doppler ultrasonography is a non-invasive method of assessing cerebral blood flow and haemodynamics. This is usually performed by taking measurements from the extracranial internal carotid and middle cerebral arteries. Obtained signals give information about the systolic, diastolic and mean blood flow velocity and are useful in the detection of vasospasm, hyperaemia or oligaemia. This may be particularly useful during the time period before invasive cerebral monitoring is available (Bell and Kochanek, 2008).

Jugular vein oximetry
This is an invasive monitoring technique that provides an indirect assessment of cerebral oxygen use.

Surgery

Surgical interventions may include elevation of depressed skull fractures, evacuation of extradural and subdural haematomas with significant mass effects, and evacuation of intraparenchymal lesions if medical treatment fails (Mazzola and Adelson, 2002). The role of decompressive craniectomy is discussed below.

Intracranial pressure-specific therapies

Raised intracranial pressure and decreased cerebral perfusion pressure following severe traumatic brain injury increases morbidity and mortality. Intracranial pressure-lowering therapy is indicated if a threshold of 20 mmHg is reached, although lower treatment thresholds have been suggested in younger children or if clinical signs of raised intracranial pressure are present (Adelson et al, 2003). Optimal cerebral perfusion pressure treatment thresholds range from 40–65 mmHg in an age-related continuum (Chambers et al, 2006). Guidelines for the management of severe traumatic brain injury in children have suggested first- and second-tier intracranial pressure-specific therapies.

First tier
CSF drainage: This provides an immediate but transient decrease in intracranial pressure and may be achieved using a ventriculostomy catheter, with or without the addition of a lumbar drain (Adelson et al, 2003).
Osmotic therapy: Use of mannitol (0.25–1 g/kg every 2–8 hours) reduces blood viscosity and cerebral oedema, thus decreasing intracranial pressure, and increases intravascular volume, thereby increasing cerebral blood flow. Alternatively, 3% hypertonic saline (0.1–1 mg/kg/hour) increases serum sodium levels, decreases intracranial pressure and increases cerebral perfusion pressure (Khanna et al, 2000).
Hyperventilation: Hypocapnia (partial pressure of carbon dioxide <35 mmHg/4.7 kPa) is associated with a reduction in cerebral blood flow and relative hypoxia in children (Skippen et al, 1997). Prophylactic hyperventilation should therefore be avoided. However, mild hyper-

ventilation may be used as first-tier therapy in refractory intracranial hypertension (Adelson et al, 2003).

Second tier

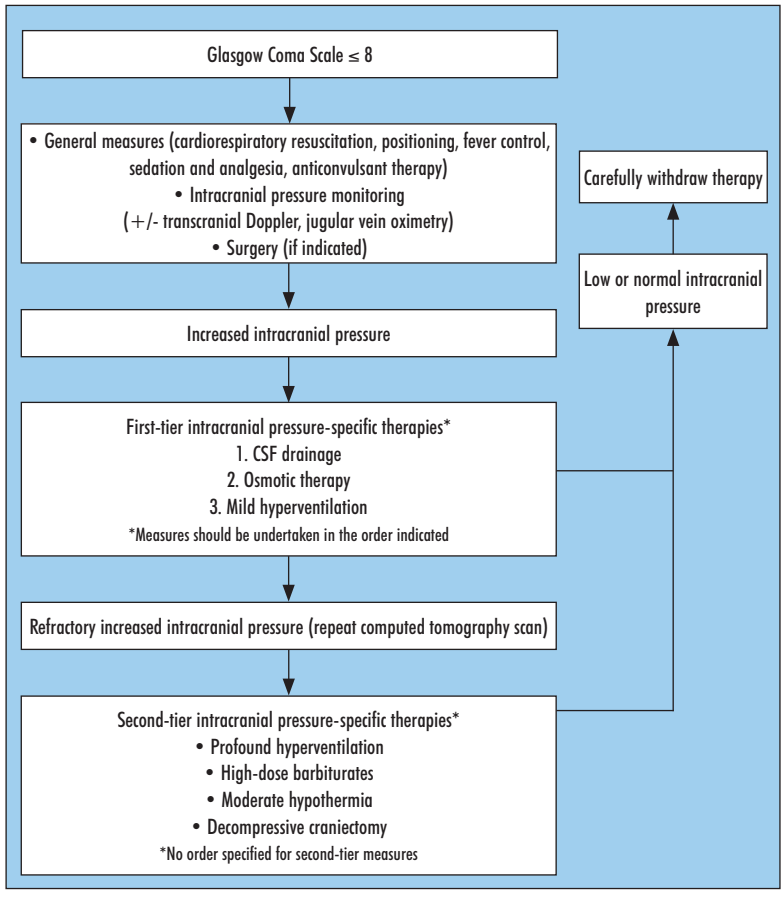
Profound hyperventilation: Profound hyperventilation (partial pressure of carbon dioxide <30 mmHg) may be used as a second-tier method of treating refractory intracranial hypertension although monitoring to detect cerebral ischaemia (cerebral blood flow, jugular venous oxygen saturation or brain tissue oxygen) is recommended (Adelson et al, 2003).

High-dose barbiturates: Barbiturates reduce intracranial pressure by altering cerebrovascular tone and reducing metabolic demands. Their use requires haemodynamic monitoring and cardiovascular support.

Moderate hypothermia: It had been suggested that hypothermia may result in a reduction in cerebral metabolic rate. However, a study indicated that hypothermia therapy does not improve neurological outcome in children with traumatic brain injury (Hutchinson et al, 2008).

Decompressive craniectomy: This involves the temporary surgical removal of a segment of skull in order to relieve intracranial hypertension. Early decompressive craniectomy shows promise in the management of intracranial hypertension in children, although this is an area where more research is required (Taylor et al, 2001; Jaeger et al, 2003).

Figure 2. Flow chart demonstrating management of children with severe traumatic brain injury. Adapted from Mazzola and Adelson (2002).



Prognosis and long-term consequences

There has been a significant improvement in the morbidity and mortality of head injury in children over the last two decades. The Glasgow Coma Scale is one of the best prognostic predictors following head injury (Keskil et al, 1995). Outcomes are also worse in infants and older adolescents (Luerssen et al, 1988; Levi et al, 1991) and in cases of raised intracranial pressure and decreased cerebral perfusion pressure (Carter et al, 2008). Furthermore, paediatric head injury has long-term neurodevelopmental consequences including difficulties with memory, anxiety, headaches and behavioural problems (Atabaki, 2007). Attention to the impact of such consequences is required if the individual, long-term needs of children with traumatic brain injury are to be addressed.

Conclusions

Head trauma is an important cause of morbidity and mortality in children. Prompt clinical and radiological assessment is vital in management of these patients although it is also imperative that any co-existing extracranial injuries are not neglected. A study by the UK Paediatric Traumatic Brain Injury Study Group demonstrated wide inter-centre variation in the management of children with significant head injuries (Morris et al, 2006), so it is hoped that guidelines published by Adelson et al (2003) will now have lead to greater standardization of practice. More research is required to explore whether this has been the case, and to fully establish the indications for intracranial pressure-reducing therapies such as decompressive craniectomy and therapeutic moderate hypothermia in the management of children with traumatic brain injury. BJHM

The authors would like to thank Professor AB Jamjoom for his kind and expert advice on writing this manuscript.

Conflict of interest: none.

Adelson PD, Bratton SL, Carney NA et al (2003) Guidelines for the acute medical management of severe traumatic brain injury in infants, children and adolescents. *Paeditr Crit Care Med* **4**: S1–S75

Atabaki SM (2007) Pediatric head injury. *Pediatr Rev* **28**(6): 215–24

Bell MJ, Kochanek PM (2008) Traumatic Brain Injury in children: recent advances in management. *Indian J Pediatr* **75**(11): 1159–65

Bissonnette B (1994) Anesthesia for neurosurgical procedures. In: Gregory GA, ed. *Pediatric Anesthesia*. 3rd edn. Churchill Livingstone, Edinburgh: 375–419

Broman SH, Michel ME (1995) *Traumatic head injury in children.* Oxford University Press, New York, US

Carter BG, Butt W, Taylor A (2008) ICP and CPP: excellent predictors of long term outcome in severely brain injured children. *Childs Nerv Syst* **24**(2): 245–51

Chambers IR, Jones PA, Lo TY et al (2006) Critical thresholds of intracranial pressure and cerebral perfusion pressure related to age in paediatric head injury. *J Neurol Neurosurg Psychiatry* **77**(2): 234–40

Feldman Z, Kanter MJ, Robertson CS et al (1992) Effect of head elevation on intracranial pressure, cerebral perfusion pressure, and cerebral blood flow in head-injured patients. *J Neurosurg* **76**(2): 207–11

Ganong WF (1993) Gas transport between lungs and the tissues. In: Ganong WF, ed. *Review of Medical Physiology*. 16th edn. Prentice Hall, International Inc, New York: 604–10
Hawley CA, Ward AB, Magnay AR (2003) Parental stress and burden following traumatic brain injury amongst children and adolescents. *Brain Inj* **17**: 1–23
Hutchinson JS, Ward RE, Lacroix J et al (2008) Hypothermia therapy after traumatic brain injury in children. *N Engl J Med* **358**: 2447–56
Jaeger M, Soehle M, Meixenberger J (2003) Effects of decompressive craniectomy on brain tissue oxygen in patients with intracranial hypertension. *J Neurol Neurosurg Psychiatry* **74**: 513–15
Jennett B (1998) Epidemiology of head injury. *Arch Dis Child* **78**: 403–6
Keskil IS, Baykaner MK, Cevikrt N, Kaymaz M (1995) Assessment of mortality associated with mild head injury in the pediatric age group. *Childs Nerv Syst* **11**: 467–73
Khanna S, Davis D, Peterson B, Fisher B, Tung H, O'Quigley J, Deutsch R (2000) Use of hypertonic saline in the treatment of severe refractory posttraumatic intracranial hypertension in pediatric traumatic brain injury. *Crit Care Med* **28**(4): 1144–51
Lam AM, Winn HR, Cullen F, Sundling N (1991) Hyperglycaemia and neurological outcome in patients with head injury. *J Neurosurg* **75**: 545–51
Levi L, Guillburd JN, Linn S, Feinsod M (1991) The association between skull fracture, intracranial pathology and outcome in pediatric head injury. *Br J Neurosurg* **5**: 617–25
Luerssen TG, Klauber MR, Marshall LF (1988) Outcome of head injury related to patients' age. *J Neurosurg* **68**: 409–16
Marcoux KK (2005) Management of increased intracranial pressure in the critically ill child with an acute neurological injury. *AACN Clin Issues* **16**: 212–31
Marcovich H (2006) Managing minor head injury in children. *BMJ* **333**: 455-6
Mazzola CA, Adelson DP (2002) Critical care management of head trauma in children. *Crit Care Med* **30**(11): S393–S401
Morris KP, Forsyth RJ, Parslow RC, Tasker RC, Hawley CA; UK Paediatric Traumatic Brain Injury Study Group; Paediatric Intensive Care Society Study Group (2006) Intracranial pressure complicating severe traumatic brain injury in children: monitoring and management. *Intensive Care Med* **32**(10): 1606–12
National Institute of Health and Clinical Excellence (2007) *Head Injury.* National Collaborating Centre for Acute Care, London
Orliaguet GA, Meyer PG, Baugnon T (2008) Management of critically ill children with traumatic brain injury. *Ped Anesth* **18**(6): 455–61
Parslow RC, Morris KP, Tasker RC, Forsyth RJ, Hawley CA (2005) Epidemiology of traumatic brain injury in children receiving intensive care in the UK. *Arch Dis Child* **90**: 1182–7
Sabsovich I, Rehman Z, Yunen J, Coritsidis G (2007) Propofol infusion syndrome: A case of increasing morbidity with traumatic brain injury. *Am J Crit Care* **16**: 82–5
Segui-Gomez M, Mackenzie EJ (2003) Measuring the public health impact of injuries. *Epidemiol Rev* **25**: 3–19
Skippen P, Seear M, Poskitt K, Kestle J, Cochrane D, Annich G, Handel J (1997) Effect of hyperventilation on regional cerebral blood flow in head-injured children. *Crit Care Med* **25**(8): 1402–9
Suh DY, Davis PC, Hopkins KL, Fajman NN, Mapstone TB (2001) Nonaccidental pediatric head injury: Diffusion weighted imaging findings. *Neurosurgery* **49**: 309–19
Taylor A, Butt W, Rosenfeld J et al (2001) A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. *Childs Nerv Syst* **17**(3): 154–62
Temkin NR, Dikmen SS, Winn HR (1991) Management of head injury post-traumatic seizures. *Neurosurg Clin N Am* **2**(2): 425–35
Tung GA, Kumar M, Richardson RC, Jenny C, Brown WD (2006) Comparison of accidental and non-accidental traumatic head injury in children on non-contrast computed tomography. *Paediatrics* **118**: 626–33
Van der Voorde P, Sabbe M, Rizopoulos D, Tsonaka R, De Jaeger A, Lesaffre E, Peters M (2008) Assessing the level of consciousness in children: a plea for the Glasgow Coma Motor Subscore. *Resuscitation* **76**(2): 175–9

KEY POINTS

- The paediatric brain is more susceptible to injury than the adult brain.
- Mortality can be as high as 23% in more severe cases of traumatic brain injury in children.
- Initial management of head-injured children involves stabilizing their airway, breathing and circulation.
- Computed tomography scanning is the investigation of choice and has specific indications.
- Critical care for traumatic brain injury in children involves general measures such as cardiorespiratory resuscitation, adequate positioning, fever control, sedation and anticonvulsant therapy.
- Monitoring techniques are vital and include intracranial pressure monitoring and transcranial Doppler ultrasound.
- Surgical intervention is required with depressed skull fractures and for the evacuation of subdural and extradural haematomas.
- Intracranial pressure-specific therapies are divided into first- and second-tier approaches.