Update in Anaesthesia

Head injury in paediatrics

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SUMMARY

Trauma is one of the leading causes of death and disability in children. Children with serious head trauma often have multiple injuries. Early identification and proper management of these patients greatly affects the outcome.

This article covers pathophysiology of head injury in children; assessment and immediate management; anaesthesia for neurosurgical management; and principles of postoperative care in PICU.

INTRODUCTION

Trauma is one of the leading causes of death and disability in children. The aetiology of injury varies with age and children with serious head trauma often have multiple injuries. The most common mechanism for head injuries globally is from motor vehicle collisions, and in the developing world the incidence is increasing dramatically. The presentation of head injury varies with the severity of the insult ranging from an altered level of consciousness to deep coma. Early identification and proper management of these patients greatly affects the outcome. Survivors of severe traumatic brain injury in childhood unfortunately are frequently left with significant behavioural, cognitive, emotional and physical challenges.

PATHOPHYSIOLOGY

Anatomy

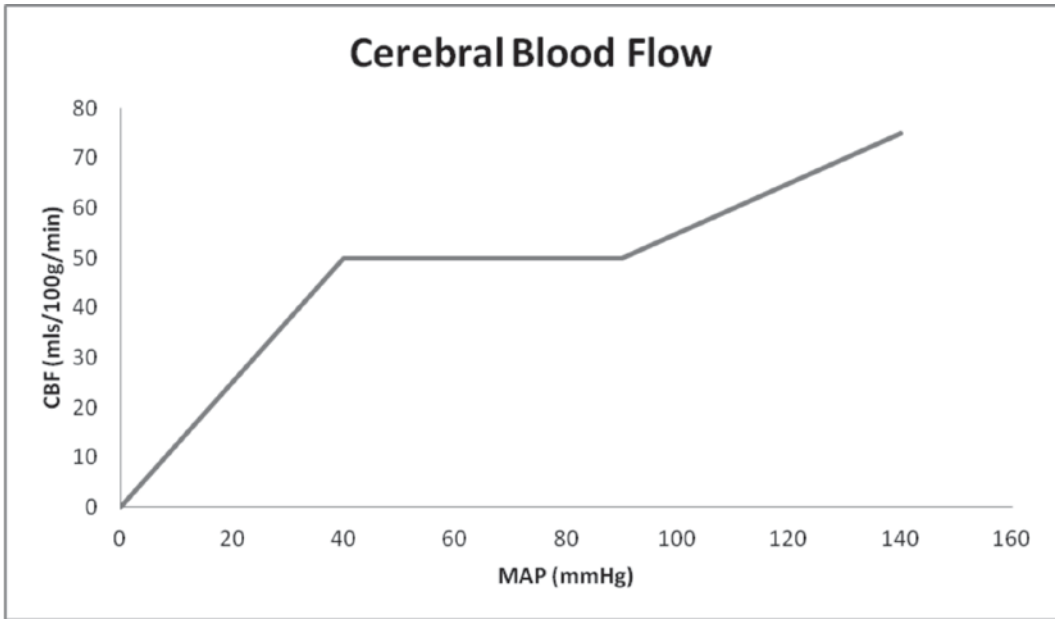
Children have a disproportionately larger and heavier head and relatively weak neck muscles, this makes them vulnerable to head injury following trauma. The open fontanelles and sutures also predispose infants to a higher incidence of subdural haematoma. Primarily for social reasons the causation varies with age; toddlers frequently suffer head injuries from falls, while older children suffer head injuries from road traffic collisions and sports related injuries. Non-accidental injury must always be considered, particularly in infants.

Cerebral blood flow

Normally, cerebral blood flow is maintained at a constant level to meet the metabolic demands of the brain over a wide range of blood pressure by the process of autoregulation. The autoregulation range is not known in infants and children, but is likely to be around 40-90mmHg.

Cerebral autoregulation is impaired by acute brain injury; in this situation, cerebral blood flow follows cerebral perfusion pressure passively.

Figure 1. *Cerebral blood flow*



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As cerebral perfusion pressure is an easier figure to derive clinically, it is this parameter that is usually used as a treatment target.

Cerebral perfusion pressure

Cerebral perfusion pressure (CPP) is the blood pressure that perfuses the brain and is defined as the difference between the mean arterial pressure (MAP) and the intra-cranial pressure (ICP). The central venous pressure (CVP) also contributes negatively to the perfusion pressure:

CPP = MAP – (ICP + CVP)

In head injury patients the intracranial pressure, in the absence of invasive monitoring, is often assumed to be 20mmHg, therefore a MAP 70-90mmHg is targeted to achieve a cerebral perfusion pressure of 50-70mmHg.

Mean arterial pressure (MAP)

The MAP may be calculated from systolic (SBP) and diastolic (DBP) pressures:

MAP = DBP + (SBP-DBP/3)

Cerebral perfusion pressure less than 50mmHg has been demonstrated to be a predictor of poor outcome in severe traumatic brain injury in children and adults. Extreme hypertension should also be avoided, as it will result in increased cerebral blood flow and cerebral oedema.

The MAP and therefore CPP vary with age, as does the ICP. There is a wide discrepancy between different organisations as to the acceptable blood pressures in paediatric traumatic head injury. In one of the few evidence based papers Haque and Zaritsky suggested in children aged one to eighteen years the MAP target should be calculated as follows:

MAP target = 1.5 x (age in years) + 55

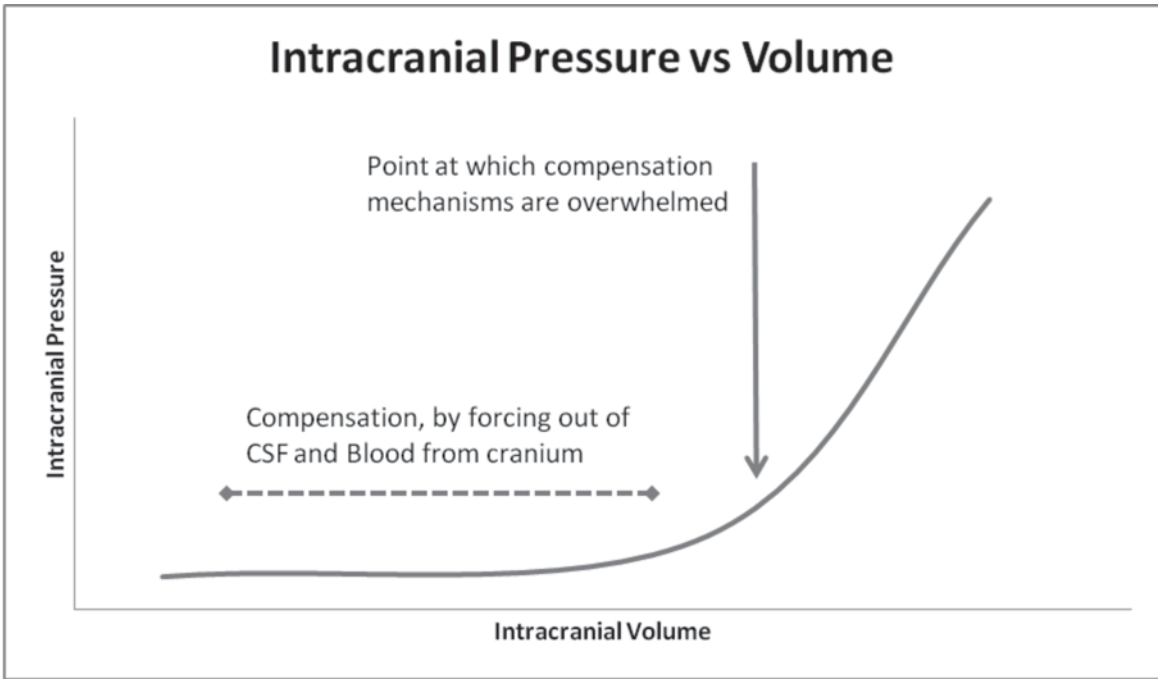
Cautious fluid resuscitation and infusion of vasoconstrictors such as noradrenaline may be required to maintain achieve the target MAP in a child with head injury.

Intracranial pressure

The intracranial pressure (ICP) is determined by the volume of the brain tissue, the cerebrospinal fluid (CSF) volume, and the cerebral blood volume. An increase in any of these volumes increases the ICP. For example an increase in brain tissue may result from swelling of the brain parenchyma, which is referred to as brain oedema.

Small increases in ICP can be compensated by an increase in CSF absorption and reduction in intracranial blood volume. Infants can further compensate due to their open fontanelles and suture lines. Sudden acute changes in intracranial pressure are not well tolerated at any age. If compensatory mechanisms are overwhelmed, intracranial pressure will increase rapidly and the brain will herniate through the structures within the skull or the foramen magnum (coning) to cause coma and death.

Figure 2. *Intracranial pressure vs volume*



Typical intracranial pressures are lower in children compared to adults (2–10mmHg vs. 8–18mmHg respectively). In health the central venous pressure does not impact upon the CPP, however in polytrauma, thoracic injuries (e.g. tension pneumothorax or cardiac tamponade) may increase the CVP to such an extent that ICP is impaired.

An increase in the cerebral blood volume could result from the following causes of intracranial bleeding:

- Epidural haemorrhage usually results from rupture of the meningeal arteries between the dura mater and the skull. It occurs quickly and may lead to death if not identified and quickly treated. Treatment is usually surgical drainage through a burr hole or craniotomy.
- Subdural haemorrhage usually results from rupture of the bridging veins between the dura and the arachnoid mater. It can be self limiting but if large can raise the intracranial pressure and will need to be evacuated.
- Intracerebral haematoma arise when there is bleeding from the blood vessels within the brain tissue. The figure below shows the three types of intracranial haemorrhage as would be seen on CT scans (note CT scans are usually shown as if looking up from the feet). (See Figure 3).
- Traumatic subarachnoid haemorrhage occurs when blood accumulates below the arachnoid mater where cerebral spinal fluid normally resides. This type of bleed is more classically associated with spontaneous rupture of cerebral aneurysms but is also common in head injury where it frequently communicates with intraventricular blood.

ASSESSMENT AND IMMEDIATE MANAGEMENT OF CHILDREN WITH HEAD INJURY

Assess the conscious level and pupil responses at the same time as attending to 'ABCDE' as follows:

- Airway with cervical spine immobilisation
- Breathing and ventilatory control
- Circulation and control of obvious external bleeding
- Disability and neurological status, including pupil responses
- Exposure - secondary survey with top to toe examination to detect associate injuries (consider non-accidental injury).

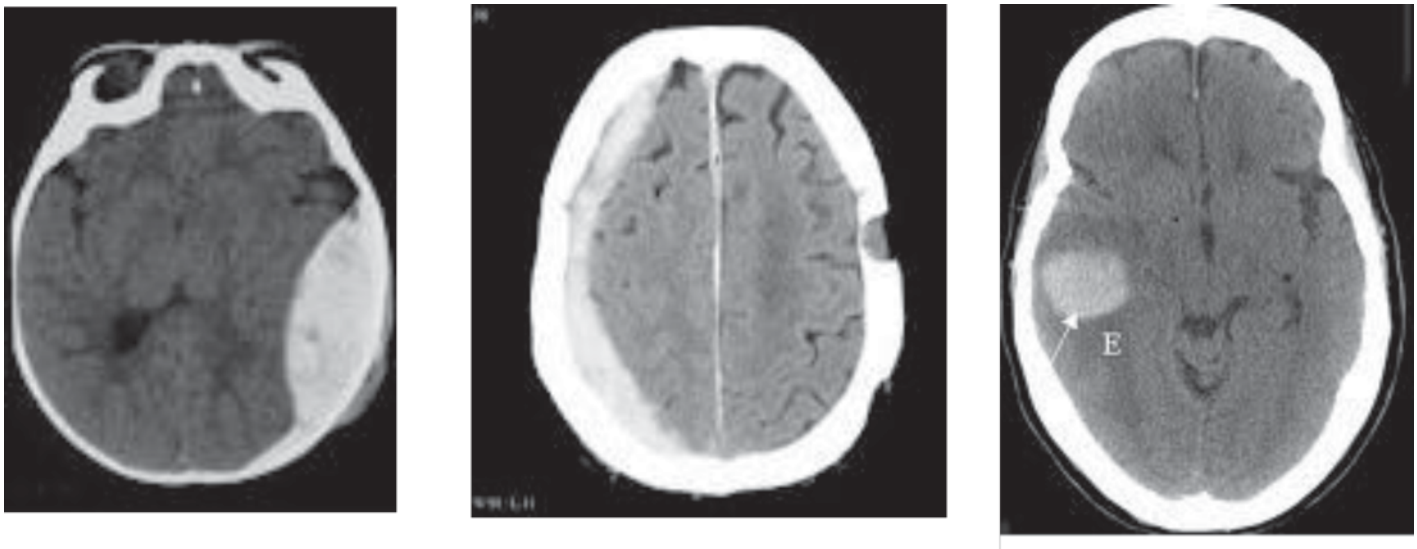
Airway and cervical spine control and immobilisation

Establish a patent airway with jaw thrust, making sure to keep the cervical spine immobilised. Suspect cervical spine injuries whenever there is high-energy mechanism of injury, reduced level of consciousness, and tenderness or bruising of the cervical spine. Foreign objects in the mouth and pharynx should be scooped out with a finger and secretions gently suctioned. Immediately provide oxygen by mask as soon as the airway is patent.

Breathing and ventilatory control

Assess the respiratory rate. Look for chest expansion while assessing symmetry and adequacy. Listen for presence/absence of breath sounds. Palpate for emphysema and broken ribs. Assess the percussion note. Pathologies to look out for include pneumo-haemothoraces, flail chest, surgical emphysema. If

Figure 3. *CT scans of different types of intracranial haemorrhage*



Left extradural haematoma **Right subdural haematoma** **Right intracerebral haematoma**

any of these are present they should be managed immediately:

- Give rescue breaths if the child is not breathing
- Decompress a tension pneumothorax by a large bore cannula in the second intercostal space midclavicular line on the affected side
- Chest drain insertion for a haemothorax and also as definitive management for the tension pneumothorax.

Consider advanced airway management in unconscious patients who cannot protect their airway and also in children with hypo- or hyper- ventilation and significant injuries to the head, neck and thorax.

Use arterial blood gas analysis to assess the oxygenation, ventilation and acid base, and electrolyte status.

Circulation and control of obvious external bleeding

Assess the haemodynamic status of the child first by feeling for arterial pulsation. Use the brachial artery in small children and the carotid in older children. Avoid palpating both carotid arteries at once as this may cause cerebral hypoperfusion.

Treat hypotension actively, obtain venous access as an early priority and collect blood samples for full blood count, electrolytes and group and save. Rule out any potential sources of external bleeding - an isolated head injury will not cause hypotension.

As discussed above, a higher than normal blood pressure should be maintained to ensure adequate cerebral perfusion. Use normal saline or Ringers for initial resuscitation. Give an initial bolus of $10ml.kg^{-1}$, repeat if hypotension persists. Avoid over hydration and anaemia. Ensure a haemoglobin $>7g.dl^{-1}$ for adequate oxygen delivery. Look out for active bleeding and prevent any further blood losses.

Disability

Assess the neurological status of the patient quickly using the AVPU scale.

Alert

Responds to Verbal stimuli

Responds to Painful stimuli

Unresponsive

Assess pupil size, equality and responses to light alongside the AVPU as this can give clues that an intracranial haemorrhage is present, and can help guide surgical management.

The pupils should be equal in size and round. Abnormal shapes may indicate cerebral damage; oval shape could indicate intracranial hypertension.

The normal pupil reaction to light is brisk. After removal of the light source, the pupil should return to its original size. There should also be a consensual reaction to the light-source - that is the opposite pupil also constricts when the light source is applied to one eye. Unreactive pupils can be caused by an expanding mass compressing the third cranial nerve. A fixed dilated pupil may be due to herniation of the medial temporal lobe.

It is important to consider factors that may affect the assessment of pupils:

- Any pre-existing irregularity with the pupils, for example cataracts, false eye or previous eye injury
- Any other factors that can cause pupillary dilation, for example medications including atropine and sympathomimetic drugs (adrenaline) and direct trauma (traumatic mydriasis)
- Any pre-existing factors that can cause pupillary constriction, for example medications including narcotics and topical beta-blockers.

The best guide to the severity of head injury is the conscious state and the Glasgow Coma Scale (GCS), which allows the conscious state to be quantified. The score is decided on the patient's best responses. Note that:

- The GCS may be falsely low if one of the following is present: shock, hypoxia, hypothermia, intoxication, post-ictal state or sedative drug administration
- The GCS may be impossible to evaluate accurately if the patient is agitated, uncooperative, dysphasic, intubated or has significant facial or spinal cord injuries
- It should be repeated regularly every 15 minutes, as rapid deterioration may occur
- The GCS is an important tool, but should be used in conjunction with a full neurological assessment to assess the child's neurological state.

The modified paediatric GCS is the standard tool for the assessment of a child's neurological status over time. The trend in the level of the consciousness is more important than a single value. (See Table 1 below). A GCS of 15 indicates no neurological disability and GCS of 3 means a deeply unconscious patient. All children with GCS less than 8 should be intubated and ventilated (see below).

Exposure

Expose the child to allow head to toe and back examination. Do this cautiously to avoid hypothermia, i.e. in a cool environment, keep the child covered when possible. Treat

Table 1: *Modified Glasgow Coma Scale for children*

Score	Score	Infants	Older children
4	*Eye opening*	Spontaneously	Spontaneously
3		To verbal	To verbal
2		To pain	To pain
1		No response	No response
5	*Speech*	Babbles and coos normally	Oriented
4		Spontaneous irritable cries	Confused
3		Cries to pain	Inappropriate words
2		Moans to pain	Incomprehensible sounds
1		No response	No response
6	*Best motor response*	Normal spontaneous movement	Follows commands
5		Withdraws to touch	Localises pain
4		Withdraws to pain	Withdraws from pain
3		Abnormal flexion to pain	Abnormal flexion to
2		Extension to pain	Extension to pain
1		No response	No response

any injuries immediately they are discovered. Note that scalp lacerations may result in significant blood loss. If the child remains cardiovascularly unstable and requires volume resuscitation, consider other sites of blood loss, for instance, chest, abdomen, pelvis or major limb fracture. Specifically look for:

- Lacerations, bruising and deformity of the face and scalp. Be aware that scalp lacerations may result in significant blood loss
- Signs of base of skull fractures which include:
  - Bleeding or leakage of cerebrospinal fluid from ear or haemotympanum
  - Periorbital bruising ("racoon eyes") and bruising around the mastoid (Battle's sign)
  - Rhinorrhea (CSF leak from the nose).

History

It is important to gain as much information as possible regarding the incident and specifically to determine:

- Time, mechanism and circumstances of the injury; speed of the vehicle; any restraints? (seat belt, car seat)
- Loss or impairment of consciousness with duration (may be inconsistent or unreliable)
- Seizures or fits
- Nausea and vomiting (children may vomit 2 or 3 times, even after a minor head injury)
- Clinical course prior to consultation - stable, deteriorating, improving
- Other injuries sustained.

Even in the heat of a major trauma, always seek a history. Especially in children, it could be that the injury might be not the reason for the coma, but vice versa. A child with cerebral malaria or meningitis may become unconscious and then subsequently fall.

Radiological investigations

Consider the following investigations in patients presenting with traumatic head injury (see Table 2):

Skull Xray

This is useful in areas where CT or MRI scans are not available. Lateral and antero-posterior (AP) views of the skull should be taken. Look for depressed fracture, diastasis and other bony abnormalities. Skull Xray is no better than clinical examination at identifying intracerebral pathology.

Computed Tomography (CT) scan

This is the imaging of choice. It reveals bony pathologies, haematoma (appears hyperdense when compared to brain parenchyma), evidence of cerebral oedema (hypodense

compared to normal brain parenchyma and isodense compared to CSF) and also mass effect (midline shift).

Magnetic resonance imaging (MRI)

MRI can be used in the acute setting to determine the presence and extent of injury and to guide surgical planning and minimally invasive interventions. It is also important in the long-term management of traumatic brain injury to identify chronic sequelae, determine prognosis, and guide rehabilitation. It is however expensive and time consuming.

Cervical spine Xray

A lateral view of the cervical spine is indicated to rule out cervical bony injury or dislocation. However, if CT or MRI is available, do a CT/MRI of the cervical spine at the same time as the head scan as these are more precise and save time.

NEUROSURGICAL MANAGEMENT

Neurosurgical management includes operative removal of epidural or subdural haematomas, or insertion of an extraventricular drain (EVD) as soon as possible after the diagnosis is made. An intra-cranial pressure monitor can be placed for ICP and CPP monitoring and further management in the paediatric intensive care unit.

When there is intractable raised ICP, a decompressive craniectomy to remove part of the frontal lobes may be indicated.

Anaesthesia for evacuation of intracranial haematoma

A small percentage of children with head injury may require surgery to evacuate intracranial haematoma. Bear in mind that:

- Volatile anaesthetic agents reduce cerebral metabolic rate but increase cerebral blood flow and ICP. Halothane increases ICP more than isoflurane and should be avoided if possible.
- Intravenous anaesthetic agents reduce the cerebral metabolic rate, and also reduce cerebral blood flow and ICP.
- Use adequate doses of opioids to obtund the reflex cardiovascular responses to intubation.
- Suxamethonium is indicated for rapid sequence induction in head injured patients even though it causes transient increase in ICP.
- Although ketamine has beneficial effects on blood pressure and is an analgesic, it raises ICP when used as a single agent in induction doses so is usually avoided.

Table 2. *Indications for CT or MRI scanning in children*

Age over 1 year: Glasgow coma score <14 on assessment on initial assessment
Age under 1 year: Glasgow coma score paediatric <15 on assessment on initial management
Age under 1 year and presence of bruise, swelling, or laceration (>5 cm) on the head
Dangerous mechanism of injury
Clinical suspicion of non-accidental injury
Loss of consciousness lasting more than five minutes (witnessed)
Post-traumatic seizure but no history of epilepsy
Abnormal drowsiness
Confusion or aggression
Signs of a basal or depressed skull fracture
Worsening level of consciousness
Failure of the mental status to improve over time
Focal neurological findings
Penetrating skull injuries
Amnesia

MANAGEMENT IN THE PAEDIATRIC INTENSIVE CARE UNIT (PICU)

The management is mainly supportive, to contain the primary injury and prevent secondary insult to the brain. It is important to maintain cerebral perfusion pressure by control of ICP and haemodynamic status.

General principles of PICU management

Management of intracranial hypertension and cerebral perfusion pressure (CPP).

Intracranial hypertension is defined as ICP >20mmHg for >5 min. Recommendations for adults include maintaining an ICP of less than 20mmHg and perfusion pressure (CPP) of >60mmHg. These may be applicable for older children. Although no normal data is available through clinical evidence, in infants with severe traumatic brain injury it would be logical to suggest targeting for an ICP of <15mmHg and a CPP of >45 to 50mmHg. Careful use of inotropic agents such as dopamine or noradrenaline may be necessary to maintain these parameters. Induced hypertension with phenylephrine is not recommended since cerebral autoregulation is lost and further increases in cerebral blood flow may exacerbate oedema formation by hydrostatic effect. Some units advocate the insertion of a jugular venous bulb catheter, to allow measurement of the SjO_2. This allows identification of global mismatch between oxygen delivery to the brain and cerebral metabolic rate.

CSF drainage by venticulostomy has been shown to be as effective as mannitol therapy in reducing ICP.

Ventilation

As both low oxygen (hypoxia) and carbon dioxide retention (hypercarbia) cause cerebral vasodilatation and increase cerebral blood flow, it is key to maintain these in the normal range (PaO_2 8-12kPa and $PaCO_2$ 4-5kPa). Over-ventilation to hypocarbia causes damaging cerebral vasoconstriction. Hyperventilation is occasionally employed as part of salvage therapy if the ICP is very high. This may be as a bridge to neurosurgical intervention, or more long term if jugular venous oxygenation is monitored.

It is assumed that all children with GCS less than 8 will not be able to protect their airway or maintain ventilation adequately; intubation and mechanical ventilation is required. Keep the head in the midline and at 30 degrees elevated position. Positive end expiratory pressure (PEEP) in the range of 4 to 5 cm of H_2O has minimal impact on ICP, and should be used to prevent atelectasis.

Patients with severe head injuries often develop neurogenic pulmonary oedema. This may require further elevation of the PEEP if adequate oxygenation cannot be achieved.

Sedation and muscle relaxation

Sedation and initially muscle relaxation are recommended for adequate control of ICP. An opioid such as morphine, midazolam and a non-depolarising neuromuscular blocker are good in combination as long as hypotension can be avoided. Ketamine was previously avoided but new data suggest that it produces minimal effects on ICP when used in combination with midazolam, and it may help to maintain the blood pressure. Pentobarbitone, phenobarbitone, or propofol may be used, subject to availability. In severely raised ICP multiple agents may be required, but it is essential to avoid hypotension.

Pain and anxiety increase cerebral metabolic demands and should be treated promptly. Intermittent thiopentone and IV lidocaine are recommended to blunt raised ICP response while suctioning the endotracheal tube. Alternatively instillation of lidocaine into the endotracheal tube may be as effective.

Seizure control

Seizures, both convulsive and non-convulsive, are extremely common after head injury. They increase both cerebral metabolic rate and ICP and must be avoided or treated promptly. Deep sedation should reduce the rate of seizure activity but may not abolish it totally.

Midazolam boluses may be used to control seizures. Propofol or thiopentone may also be used. Treat hypotension associated with the use of these agents with fluid therapy. Start Phenytoinin patients with posttraumatic seizures. Use of prophylactic phenytoin in head injured children without seizures is not supported by clinical evidence.

If available, EEG monitoring is extremely useful in identifying non-convulsive seizures, which should be controlled to reduce the cerebral oxygen consumption.

Fluid therapy

Initial resuscitation should be with normal saline. Hypotonic fluids must be avoided in children with head injury as a fall in plasma sodium will exacerbate cerebral oedema. Ringer's lactate is slightly hypotonic and many authors suggest it should not be used. Maintain plasma sodium and plasma osmolality within the high normal range (aim for plasma sodium 150mmol.l^{-1} in severe head injury). Place a central line to guide fluid therapy by central venous pressure monitoring.

Maintain glucose within normal levels. Due to stress of the head injury, serum glucose is commonly high, so avoid glucose containing fluids initially.

Children with traumatic brain injury are susceptible to a variety of abnormalities of plasma sodium for example:

- Syndrome of inappropriate antidiuretic hormone (SIADH). This is characterised by hyponatraemia,

low plasma osmolality, high urinary osmolality, normo- or hypervolaemia. Patients are at risk of developing cerebral oedema. Manage promptly with fluid restriction if asymptomatic or hypertonic saline if symptomatic (1-2 $ml.kg^{-1}$ 3% saline iv bolus).

- Cerebral salt wasting. This typically presents with hyponatraemia, high urinary osmolality, hypovolaemia and hypotension. Manage with an IV bolus of normal saline.
- Diabetes insipidus. This presents with hypernatraemia, high plasma osmolality, low urinary osmolality, hypovolaemia and hypotension. Diabetes insipidus occurs as a result of failure of blood supply to the posterior pituitary with loss of ADH production in the posterior pituitary. Treat it by administration of DDAVP (Desmopressin). Note that diabetes insipidus is often a late sign in head injury and often heralds brain stem death.

Mannitol

Mannitol reduces ICP by two mechanisms, osmotic and reduction in viscosity. Its osmotic effect is the more effective mechanism. Reduction of viscosity is transient, and depends upon autoregulation being intact.

Mannitol in doses of 0.5 to $1g.kg^{-1}$ may be used intravenously at 6 hourly intervals with monitoring of serum osmolality (aim to keep serum osmolality under $320mOsmol.kg^{-1)}$. Mannitol is contraindicated if any of the following is present:

- Serum osmolality is $>330mOsmol.kg^{-1}$
- The patient is hypotensive
- The patient is known to be in renal failure.

Rapid boluses of mannitol can transiently increase ICP by causing transient systemic hypertension and should be avoided. Mannitol also has a theoretical risk of enlarging a haematoma by rapid shrinkage of brain and tearing of bridging veins.

Temperature control

Body temperature has an important effect on cerebral blood flow. For every 1°C increase in body temperature, there is a 5% increase in cerebral metabolic rate leading to an increase in cerebral blood flow and intracranial pressure. In children with head injury, avoid pyrexia (>37.6°C), and aim for normothermia or moderate hypothermia. Excessive hypothermia (< 33°C) has been shown to increase mortality.
In patients with refractory ICP induced hypothermia may be used as a last resort, however clinical studies have shown this to be of no benefit.

Steroids

There is no clinical evidence supporting the use of steroids in cerebral oedema due to head injury. Reserve steroids for patients with brain tumours.

Nutrition

Early institution of enteral feeds is recommended if there is no associated intra-abdominal injury to major organs such as liver, spleen, or duodenal hematoma.

Positioning

Nurse intubated children at 30 degrees head up to reduce ICP and reduce the risk of ventilator-associated pneumonia (VAP). Regular turning and the use of splints will reduce bedsores and contractures.

Rehabilitation

Tracheostomy may be indicated in patients in prolonged comatose state who cannot protect their airway or who require long-term ventilation.

Once the patient has recovered from acute injury in the PICU, early physiotherapy for prevention of deep vein thrombosis and prevention of contractures may be necessary. Arm, leg, hand and feet splints should be used as indicated.

Table 3. *Outcomes after paediatric head injury*

Early complications
• Transient cortical blindness
• Seizures
• Cranial Nerve palsy
• Diabetes insipidus
• Syndrome of inappropriate secretion of ADH
• Cortical venous occlusion
• Hemiparesis
Late complications
• Post traumatic epilepsy
• Post traumatic aneurysm
• Meningitis
• Hydrocephalus
• Memory loss
• Disability
• Muscle contractures

OUTCOMES OF HEAD INJURY IN CHILDREN

Complications of traumatic head injury can be grouped into early and late categories (see Table 3).

Several outcome scores have been described. The King's Outcome Score for Childhood Head Injury, which was derived from the adult Glasgow Outcome Score is described in Table 4.

ICU mortality in paediatric severe traumatic brain injury is slightly lower than in adult practice. Children younger than 4 years of age have been reported to have poor prognosis similar to adults while better outcomes have been reported in 5 to 15 year age group. Unfortunately in many survivors there are significant neurocognitive, educational and social consequences. The GCS at 24hrs remains the strongest predictor of outcome.

CONCLUSION

The improvement in outcomes from traumatic head injury over the past decade has been achieved by strict attention to physiology, a protocolised approach to treatment and underlying improvements in critical care. In well-resourced countries the focus is on centralised care allowing more invasive monitoring particularly of ICP. Unfortunately in less well-resourced countries the burden of road traffic collisions is increasing, but with the application of applied physiology one would still expect to make an impact on outcomes.

REFERENCES

1. Raghavan K, Waddington R. Management of Paediatric traumatic brain injury. *Anaesthesia Tutorial of the Week* 127 (2009). Available at: http://www.wfsahq.org/components/com_virtual_library/media/f1fa2673a7be4eb43f4f859802090b135-4fa20962568cbe51f494b34724e63b32-127---management-of-paediatric-traumatic-brain-injury.pdf

2. Khilnani P. Management of paediatric head injury. *India J Crit Care Med* 2004; **8**: 85-92.

3. Dykes E. Paediatric trauma. *Br J Anaesth* 1999; **83**: 130-8.

4. Haque I, Zaritsky A. *Pediatr Crit Care Med* 2007; **8**:138 –144.

5. Verive M et al. Paediatric head trauma. Medscape 2012 http://emedicine.medscape.com/article/907273-overview (accessed 4th October 2014.

6. Alterman D et al. Considerations in paediatric trauma. Medscape 2011. http://emedicine.medscape.com/article/435031-overview (accessed 4th October 2014).

7. Clayton TJ et al. Reduction in mortality from severe head injury following introduction of a protocol for intensive care management *Br. J. Anaesth*. 2004; **93**: 761-67.

8. Crouchman M et al. A practical outcome scale for paediatric head injury. *Arch Dis Child* 2001; **84**: 120–4.

Table 4. *The King's Outcome Score for Childhood Head Injury*

1	*Death*
2	*Vegetative* The child is breathing spontaneously and may have sleep/wake cycles. He may have non-purposeful or reflex movements of limbs or eyes. There is no evidence of ability to communicate verbally or non-verbally or to respond to commands.
3	*Severe disability* (a) The child is at least intermittently able to move part of the body/eyes to command or make purposeful spontaneous (b) Implies a continuing high level of dependency.
4	*Moderate disability* (a) The child is mostly independent but needs a degree of supervision/actual help for physical or behavioural problems (b) The child is age-appropriately independent but has residual problems with learning/behaviour or neurological sequelae affecting function.
5	*Good recovery* (a) This should only be assigned if the head injury has resulted in a new condition that does not interfere with the child's well being and/or functioning.