Neuroprotection in traumatic brain injury

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Abstract

Traumatic brain injury is the leading cause of trauma-related death and disability in children worldwide. To improve neuro-outcomes after injury, neuroprotective measures are implemented to limit secondary brain injury by providing adequate cerebral perfusion and oxygenation. In this article we review the general supportive and targeted neuroprotective measures that are outlined in the international paediatric guidelines and the physiological basis for these recommendations based on the underlying pathology. We also discuss multimodal neuromonitoring in the PICU. We aim to provide a practical approach on how to respond to deteriorating patients, and manage complications arising during the course of their treatment.

Keywords Cerebral perfusion; critical care; intracranial pressure; neuroprotection; paediatric traumatic brain injury

Introduction

Traumatic brain injury (TBI) is a major cause of mortality and long-term disability in children worldwide. Children affected by TBI are different to adults as their brains are still developing, and functional deficits may become apparent only over time. Long-term cognitive decline and expensive medical care add to physical burden.

Infants and adolescents are most commonly affected, with falls and non-accidental injuries common in younger age-groups, and road traffic accidents commoner in teenagers. The severity of TBI is clinically classified by the Glasgow Coma Scale (GCS) into mild (13–15), moderate (9–12) or severe (≤8).

Shear forces at the time of impact cause primary injury to the brain parenchyma. Blunt trauma on the stationary head damages the brain directly underneath the point of impact (coup injury), and when the head is free to move, it results in an injury opposite to the site of impact (contrecoup injury). These acceleration-deceleration forces can result in disruption of blood vessels, neural and glial tissue leading to irreversible cell damage (Figure 1). Physiological responses to the primary brain injury result in secondary brain injury (SBI), which evolves over hours to days, and provides a window for therapeutic interventions. Inflammatory and neurotoxic cascades are activated, which cause brain swelling, hypoperfusion, hypoxia, temperature dysregulation, loss of autoregulation, and seizures, resulting in SBI.

The two main components of SBI are associated with pressure and perfusion related injuries. As the skull is a rigid bony compartment where the brain parenchyma, blood and cerebrospinal fluid (CSF) exist in equilibrium, an increase in the volume of any one of these intracranial contents will raise intracranial pressure (ICP), unless compensated by a reduction in volume of one or both of the other components. CSF is the main compensatory mechanism, followed by increased cerebral venous drainage in later stages. Beyond limits of compensation, the ICP rises sharply for even a small increase in volume, resulting in intracranial hypertension (ICH). The cerebral blood flow (CBF) is maintained relatively constant across a range of systemic blood pressures with complex autoregulatory mechanisms to sustain the high metabolic demands of the brain. As CBF is difficult to measure bedside, cerebral perfusion pressure (CPP) is used clinically as the driving pressure for blood flow into the brain and is calculated as a difference between mean arterial pressure (MAP) and ICP (MAP − ICP). Severe systemic hypotension and ICH can compromise cerebral perfusion. The main clinical focus of neuroprotective care is stabilizing ICP and optimizing CPP.

In this article, we discuss management priorities in children with moderate to severe TBI (sTBI) from the time of injury to Paediatric Intensive Care Unit (PICU) admission. We discuss the monitoring, set targets, and various interventions used in PICU from baseline care to implementation of first and second tier therapies based on International Paediatric TBI guidelines from 2019.[1]

Stabilization before PICU

Early stabilization is based on rapid assessment and aggressive management of life threatening injuries following the ATLS/APLS guidelines (Box 1). Adequate oxygen and blood supply to the brain are the most important determinants of outcome, as systemic hypoxia and hypotension are detrimental and should be avoided at all costs. Evidence from adult CRASH-3 trial in TBI showed that tranexamic acid (TXA) when used within 3 hours of mild to moderate TBI is safe, and can reduce mortality. Extrapolating this evidence to children, the Royal College of Paediatrics and Child Health (RCPCH) has produced an evidence statement for the use of TXA in paediatric patients with major trauma including TBI (loading dose at 15 mg/kg, max 1 g, and maintenance infusion at 2 mg/kg/hour for 8 hrs or until bleeding stops).

Airway

Depressed consciousness following TBI compromises protective airway reflexes, increasing risk of aspiration and hypoxemia. Endotracheal intubation is indicated in TBI patients with GCS ≤8, rapidly worsening GCS, loss of protective airway reflexes, significant facial injury, hypoventilation, hypoxia, haemodynamic instability, and seizures. Intubation is classically performed by the most skilled provider, via oral route, with manual in-line stabilization to protect the cervical spine. Rapid sequence induction is the standard of care to reduce the risk of aspiration. All precautions should be taken to avoid hypoxia and blood pressure fluctuations during intubation, as the act of laryngoscopy, cough and gag reflexes can affect ICP. Pre-medication with fentanyl can

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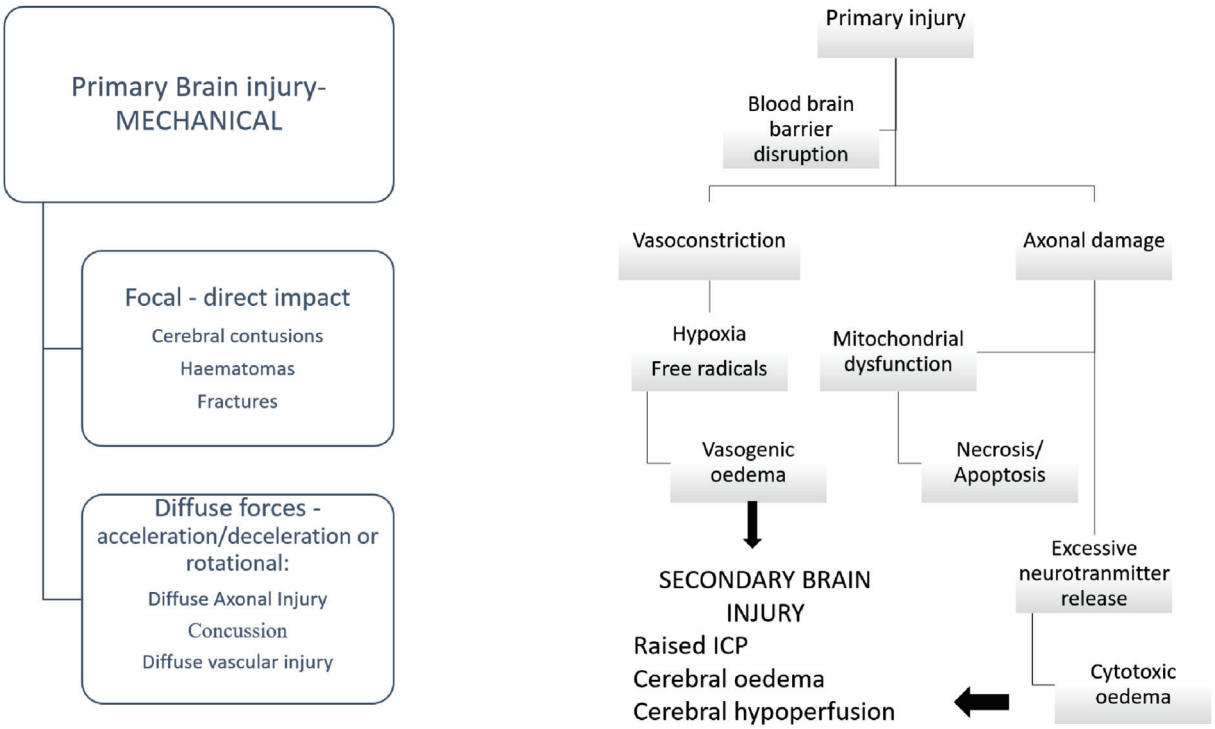

Figure 1 Primary and secondary brain injury.

prevent an exaggerated reflex sympathetic response to laryngoscopy which can raise ICP. Ketamine and rocuronium are the usual agents of choice and work well even in haemodynamically unstable children. In normotensive and hypertensive children cautious doses of propofol or thiopentone may be used.

Ventilation

The aim is to maintain normoxemia and normocarbia to maintain cerebral oxygenation and perfusion. Oxygen saturation should be continuously monitored, hypoxemia should be corrected once identified using appropriate positioning, airway adjuncts and supplemental oxygen to maintain saturation above 90%. Carbon dioxide (CO_2) has profound reversible effects on cerebral vasculature and hence should be tightly controlled. Hypercapnia causes cerebral hyperaemia through cerebral vasodilation which can increase ICP, whereas hypocapnia causes vasoconstriction and reduced CBF with potential for cerebral ischaemia. Intubated patients should have continuous capnography. Initial targets are defined in Box 1. Hyperventilation should be avoided, except transiently to address impending herniation, which is discussed later.

Circulation

Hypotension is a known risk factor for poor neurological outcome post TBI. In children, blood pressure (BP) should be targeted to more than the 75th centile for age, using appropriate sized blood pressure cuffs. The primary aim of prehospital resuscitation is to prevent SBI due to hypotension. Normal saline (0.9%) is used for resuscitation and as initial maintenance fluid (70% of total daily maintenance) aiming for normovolaemia.[1] Early use of vasopressors restores cerebral perfusion pressure by optimizing CBF to meet metabolic demands; where indicated, use of appropriate blood products is favoured over large volume crystalloid resuscitation because of the risk of haemodilution and cerebral hypoxia.

Analgesia, sedation and neuromuscular blockade

Level of consciousness should be assessed using GCS, with specific emphasis on the motor component, prior to administering any medications and regularly thereafter, along with pupillary response. Sedation-analgesia is commonly administered as a continuous infusion, titrated to effect, with close haemodynamic monitoring. A combination of opiates (morphine or fentanyl) and benzodiazepines (midazolam) is commonly used.[1] Propofol is an alternative in haemodynamically stable children, if easier to administer. Neuromuscular blockade (NMBA) should be used as continuous infusion (or boluses if easier) prior to admission to PICU.

Neuro-imaging

Following initial stabilization, early neuro-imaging (CT scan) helps decide management priorities by identifying need for urgent neurosurgical interventions (for e.g., haematoma requiring urgent evacuation) and neuroprotection. NICE guidelines recommend an urgent non-contrast CT scan to detect TBI. While a normal early scan within 6 hours of injury does not guarantee a positive outcome, an abnormal scan helps to define the speed of transfer and interventions required. Repeat neuro-imaging may be required as dictated by regular neurological examination, given that TBI is an evolving disease. Invasive ICP monitoring is useful in children whose clinical deterioration maybe masked by continuous sedation and paralysis.

Transfer

Children with TBI are best managed in centres with expertise where protocolized treatment and early multi-disciplinary input

Management priorities for children with TBI during transfer and before admission to PICU

1. Secure airway
2. Cervical spine protection with blocks and tapes, no ties around the neck
3. Head in midline with end of the bed/trolley elevated to 15—30°
4. Minimum monitoring:
    a. Pulse oximetry (SpO_2)
    b. End tidal CO_2 ($EtCO_2$)
    c. Non-invasive blood pressure (BP), (ideally invasive with arterial line)
    d. Temperature (periodic if continuous not possible)
    e. Glucose
    f. Pupillary size and reaction
5. Optimize ventilation to following targets:
    a. SpO_2 greater than 97% (PaO_2 greater than 11 kPa)
    b. $EtCO_2$ 4.5—5 kPa (ideally titrated to $PaCO_2$)
6. Optimize mean blood pressure to upper limit of normal for adequate cerebral perfusion:
    a. less than 2 years greater than 55 mmHg
    b. 2—6 years greater than 60 mmHg
    c. greater than 6 years greater than 70 mmHg
7. Analgesia, sedation and paralysis:
    a. Midazolam: 50—300 microgram/kg/h
    b. Morphine: 20—80 microgram/kg/h
    c. Vecuronium: 50—100 microgram/kg/h or rocuronium
8. Maintenance fluids 0.9% saline at 70% of total daily requirement (add glucose when blood sugar is less than 4 mmol/l)
9. For transfer:
    a. Experienced medical and nurse escorts
    b. On-going communication with receiving team and appropriate place of transfer (theatres, emergency department, PICU)
    c. Boluses of anaesthetic, hypertonic saline
    d. Ensure adequate monitoring as above
    e. Check blood gas and haemoglobin before transfer
    f. Adequate supply of emergency drugs and equipment

Box 1

improves the outcome. Hence, early referral and safe time-critical transfer to a centre with PICU and neurosurgical expertise can provide the required surgical intervention and definitive neuroprotection. It may not always be possible to achieve full stability and monitoring before transferring children with life-threatening emergencies like an expanding haematoma causing brain compression; only early urgent transfer for surgical evacuation will save life and improve outcome. All transfer decisions should be taken in discussion with the tertiary centre.

Critical care and baseline neuroprotection

Positioning

Simple physical interventions like head-of-bed elevation to 30—45° or reverse Trendelenberg position, with head and neck in midline to prevent kinking of the jugular veins facilitates cerebral venous drainage, reducing ICP while not affecting CBF. Head-end elevation beyond 45^0 can reduce MAP and CBF. Tight cervical collars and constricting tracheostomy ties should be avoided.

Ventilation

Titration of ventilation to achieve the targets set for oxygenation and CO_2 is continued after admission to PICU. In patients with associated lung injury who develop acute respiratory distress syndrome, the classical strategies of lung protective ventilation like low tidal volume, permissive hypercapnia, high PEEP and lower oxygenation goals need to be used cautiously in the context of TBI. High PEEP can raise intrathoracic pressure depending on the lung compliance, which impedes cerebral venous drainage, and causes alveolar hyperinflation which can cause hypercapnia. Adjustments in oxygen, PEEP, and minute ventilation should be performed in tandem with ICP monitoring.

Circulation

Ongoing careful fluid and electrolyte management to achieve adequate haemodynamic stability continues on PICU. Isotonic normal saline is preferred over balanced crystalloids in patients with TBI and remains the fluid of choice. Invasive blood pressure monitoring through a secure arterial line is carried out to carefully titrate MAP. Detailed management of CPP will be discussed further under first tier therapies.

Patient comfort

Comfortable environment with reduced noise, lighting and restraints lessens anxiety and agitation related spikes in ICP. Sedoanalgesics provide comfort and exert neuroprotective effects by reducing cellular metabolism. Recent guidelines recommend a more selective use of NMBA as benefits are weighed against risks.[1] The addition of NMBA facilitates mechanical ventilation, prevents shivering, prevents ICP surges related to suctioning or physiotherapy, and reduces overall energy expenditure. These potential benefits have to be weighed against the adverse effects of continuous NMBA infusion which include limitation to clinical assessment, masking of seizures, and critical illness neuromyopathy.

Fluids and nutrition

Children with TBI should be started on early (less than 72 h) enteral nutrition to reduce mortality. Traditionally TBI has been considered a hypermetabolic state, but studies have shown that the neuroprotective bundle including sedation, NMBA and targeted temperature management may in fact result in a state of hypometabolism. It is therefore important to avoid over- or under-feeding to improve outcomes. If enteral route is not tolerated, parenteral nutrition can be considered.

Hypo-and hyper-glycaemia have negative impact on TBI. Fluids containing glucose are not routinely used due to the anticipated stress hyperglycaemia secondary to TBI. In younger children, 5% dextrose with 0.9% saline can be considered to avoid hypoglycaemia. Regular glucose monitoring is advocated, targeting normoglycaemia. Intensive glucose control carries the risk of hypoglycaemia; insulin is used only for persistently high glucose levels (greater than 180 mg/dL), with vigilant monitoring.[1]

The baseline target for sodium (Na^+) level is greater than 140 mEq/L, avoiding sustained levels more than 160 mEq/L[1]. Higher target levels may become necessary if the clinical condition evolves and complications arise as discussed below.

Temperature control

Patients with TBI develop hyperthermia due to impaired thermoregulation secondary to autonomic instability. Hyperthermia is associated with increased cerebral metabolic demand and neuronal death, leading to poor outcomes. The goal is targeted temperature management to 36–37 °C and avoiding hyperthermia.[1] Surface cooling systems circulate cold air around the patient, additional antipyretics can be used for temperatures ≥38 °C. It is important to prevent shivering and hypotension secondary to cooling. Even though hypothermia (less than 36 °C) can theoretically decrease cerebral metabolic demand, inflammation, excitotoxity, cell death and seizures, its safety and efficacy continues be a subject of ongoing debate with studies showing evidence of neutral to negative impact. However, controlled late application of hypothermia can have a role in treating refractory ICH as discussed below.

Seizure control

Patients with TBI are at risk of early (less than 7 days) or late post-traumatic seizures (PTS) which depends on the severity and type of injury, presence of depressed or penetrating skull fractures or retained fragments, location of lesion, presence of contusion or haemorrhage and age. Children in general have lower seizure threshold, and in those with TBI, electrographic seizures occur more than in adults. Despite absence of strong evidence, current guidelines recommend use of prophylactic antiepileptic drug therapy, with either phenytoin or levetiracetam to reduce early PTS in children with sTBI.[1] Continuous electroencephalography (cEEG) monitoring should be considered especially in children who are muscle-relaxed.

Transfusion

Haemoglobin (Hb) levels should be kept greater than 7 g/dL in children with sTBI to ensure adequate cerebral oxygenation. In selected patients, and depending on clinical and monitored parameters, the target level may be adjusted to achieve higher Hb concentrations.

Coagulation

Coaguloapthy is a common complication of TBI, often hypo- and rarely hyper-coagulability. There is no consensus regarding the optimal treatment targets for coagulation parameters in children with TBI. In addition to conventional tests, thromboelastography is useful in monitoring haemostasis. A goal-directed approach in patients with active bleeding, those who require invasive neuromonitoring or surgical intervention is more appropriate than empiric blood product transfusion. Fresh frozen plasma is most commonly used to correct clinical bleeding.

Venous thromboembolism risk assessment is equally important given the risk of hypercoagulability. Graduated compression stockings or intermittent pneumatic compression should be used in sedated children. Regular thromboprophylaxis should be considered for older children once the risk of bleeding and surgical interventions have been addressed.

General care

General PICU care remains important. Patients require regular repositioning to avoid pressure ulcers, effective eye care to prevent corneal defects, and physiotherapy to mitigate pulmonary and neuromuscular sequelae. Children with sTBI are at an increased risk of stress ulceration due to multiple factors; prophylaxis should be started in all patients and reviewed once on enteral feeds. Laxatives should be used as required. A tertiary trauma survey should be performed, and any additional injuries identified and managed as appropriate.

Monitoring and advanced therapies

The aforementioned strategies are the baseline standard of care for any child with sTBI admitted to PICU. Clinical examination cannot be performed continuously, and is limited by sedation and NMBA. Patients with worsening intracranial disease can only be identified by real-time monitoring for timely intervention to prevent irreversible brain damage. This helps to assess response to treatment, and to modify it when necessary.

Multimodal neuro-monitoring using various non-invasive (continuous and qualitative EEG -cEEG and qEEG, Near Infra-Red Spectroscopy- NIRS, Optic Nerve Sheath Diameter- ONSD, Trans Cranial Doppler- TCD) and invasive techniques (ICP, CBF, brain tissue oxygenation ($PbtO_2$)), along with physiological parameters can aid detection of brain injury.

In the following sections we discuss ICP monitoring and application of first and second tier therapies when baseline management alone is insufficient to control ICP (Table 1, Figure 2).

Intracranial pressure monitoring[2]

Normal ICP values vary with age and range, from 2 mmHg in neonates to 15 mmHg in older children. ICP monitoring is considered an integral part of managing sTBI despite paucity of controlled trials.[1] Sustained elevation in ICP greater than 20 mmHg for greater than 5 min should be treated.[1] ICH secondary to mass-effect or cerebral oedema can result in cerebral ischaemia, herniation and brain-stem compression. The goal is to maintain ICP less than 20 mmHg in all age groups.

There are many ICP monitoring devices, both invasive and non-invasive. Micro-transducers are used to measure ICP in the brain parenchyma; they are less invasive and easier to insert and are used most frequently in the UK. External ventricular drains (EVD) placed in the lateral ventricle can monitor ICP and also allow CSF drainage if required; they can be technically difficult to insert with cerebral oedema and carry the risk of infections and haemorrhage. Non-invasive neuro-monitoring mentioned previously, along with brain imaging are alternative means of assessing ICP, none of which have proven to be reliable alternatives to invasive ICP monitoring.

First tier therapies

Managing intracranial pressure: tier one therapies reiterate the optimization of standard neurocritical care practices discussed till now. If ICP remains elevated, CSF can be drained if an EVD is present. Hyperosmolar therapy with hypertonic saline or mannitol may be initiated subsequently or concurrently. Upper limit of serum osmolarity with hypertonic saline is 360 mOsm/L, maintaining sodium less than 160 mEq/L. If necessary,

Management of severe TBI (if ICP/CPP targets not met)

	First tier therapies	Second tier therapies
Medical	Hyperosmolar therapy • 3% sodium chloride (2–5 ml/kg, max 100 ml over 15–20 min) • Mannitol (0.25–1.5 g/kg) Optimize • Sedation/NMBA • Ventilation ($PaCO_2$ ≈4.5 kPa) • Blood pressure (minimum CPP 40 mmHg) • Temperature (<38 °C) • Haemoglobin (Hb >7 g/dL) • Antiepileptic drugs (phenytoin/ levetiracetam)	3% sodium chloride infusion (0.1–1 ml/kg/h) Consider • Hyperventilation to reduce $PaCO_2$ (4.0–4.5 kPa) • Induced hypothermia (32–34 °C) • Thiopentone infusion to achieve burst suppression (with continuous EEG monitoring)
Surgical	CSF drainage if EVD present	Haematoma evacuation Decompressive craniectomy

If signs of herniation
- Hyperventilate to reverse pupil change
- FiO_2 1.0
- Bolus hypertonic saline or mannitol
- Drain CSF if EVD
- Urgent CT scan

Table 1

hyperosmolar therapy can be repeated along with additional analgesia and/or sedation and NMBA should be considered.[1]

Managing cerebral perfusion pressure: in a non-injured brain, cerebral autoregulation (CA) maintains a stable CBF over a wide range of blood pressure (50–150 mmHg). When autoregulatory mechanisms are exceeded, CBF becomes dependent on the MAP, and a rise in MAP increases CBF, inadvertently raising ICP. Therapies like hypertonic saline used to reduce ICP can often improve MAP and thereby CPP, whereas sedation/analgesia boluses can drop both ICP and MAP. CPP-targeted therapies aim to maintain CPP of 40 mmHg in younger children, and higher age-specific thresholds up to 60 mmHg for older children and adolescents.[1] CPP targeted therapies should be concurrently

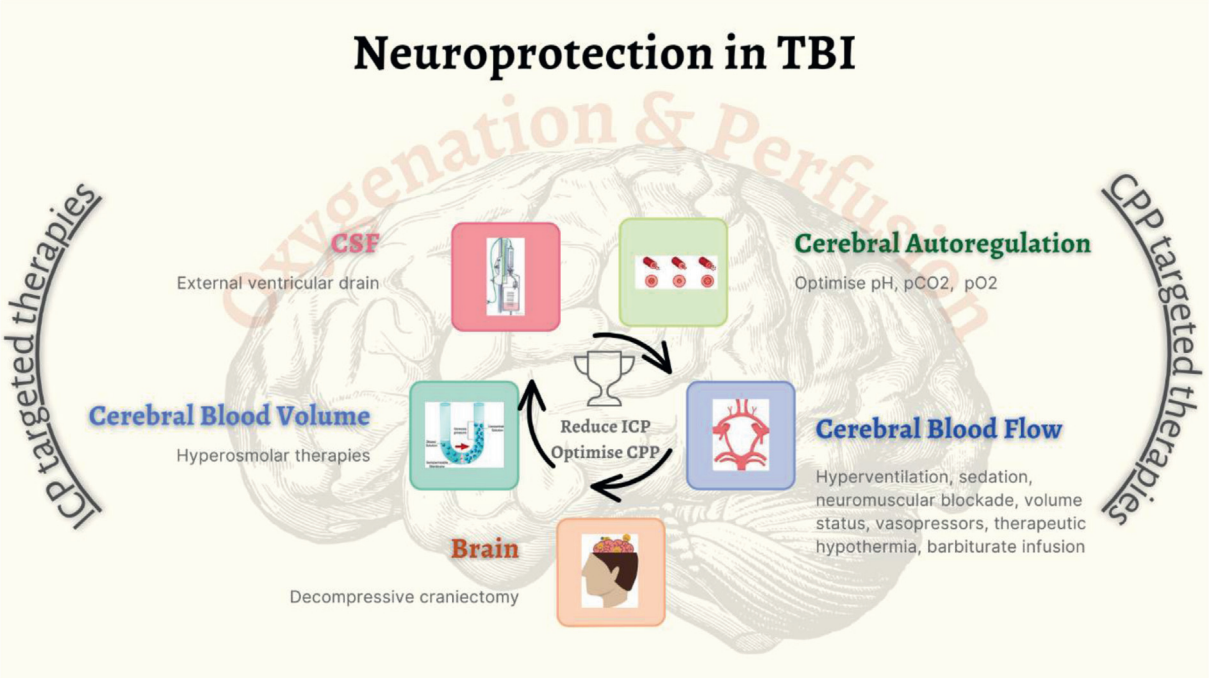

Figure 2 Neuroprotection in TBI.

ICP-directed as CPP can be maintained even in the face of raised ICP resulting in herniation. Therefore, titrating vasopressors to maintain CPP must be carefully balanced in conjunction with other therapeutic interventions including ventilation, sedation and ICP reducing strategies.

Mild degrees of ICH might be tolerated and second tier therapies deferred, provided CPP is maintained.[1] If ICP remains elevated despite first tier interventions, second tier therapies should be used.

Second tier therapies

In TBI patients where increased ICP becomes refractory to first tier therapies, if feasible, a repeat head CT scan should be performed to identify amenable surgical pathology. In addition to potential surgical interventions, we discuss below medical interventions that should be considered for all patients with refractory ICH although the decision to apply them must be individualized for each patient.

Hyperventilation: temporary therapeutic hyperventilation to achieve $PaCO_2$ 3.7–4.5 kPa can be cautiously attempted in refractory ICH, whilst other therapeutic interventions to reduce ICP take effect. The cerebral microvasculature reactivity to CO_2 remains relatively intact even when pressure autoregulation is altered. Hypocapnia induced cerebral vasoconstriction reduces cerebral blood volume and ICP, but with the risk of ischemia secondary to reduced CBF. If hyperventilation is used for longer duration, advanced neuromonitoring, in particular $PbtO_2$, should be considered and maintained to prevent cerebral ischaemia.[1]

Therapeutic hypothermia: while early prophylactic hypothermia is not recommended, late moderate (33–35 °C) hypothermia can be used for refractory ICH control with effect on suppressing cerebral metabolism.[1,3] Rewarming should be done cautiously at a rate of 0.5–1.0 °C or slower, every 12–24 h, to avoid complications.

Hyperosmolar therapy: in refractory ICH, hyperosmolar therapy can be escalated, while monitoring osmolarity and serum sodium levels[1] (Table 1). Hypertonic saline can be used as a continuous infusion if not contraindicated by thrombocytopenia (less than 100×10^9/L), abnormal clotting (INR greater than 1.4) or rise in creatinine more than twice the baseline.

Barbiturate infusion: thiopental or pentobarbital infusion is used as last resort in refractory ICH. Ideally, cEEG should be used to titrate barbiturate therapy to minimize the dose required and hence significant side effects. Barbiturates reduce cerebral metabolism, and thereby CBF and ICP. Close haemodynamic monitoring is essential as barbiturates can cause hypotension, arrhythmias, and respiratory complications apart from predisposition to secondary infections and other organ failures. The infusion is used for the least possible time and is weaned once the ICP remains less than 20 mmHg for 24 hours.[1] The most commonly used agents are thiopental and pentobarbital.

Surgical interventions: emergency neurosurgical intervention is indicated for the removal of a mass lesion causing raised ICP. Decompressive craniectomy is considered for diffuse brain swelling refractory to medical management.[1] It carries significant risk and is only considered in specific cases given lack of sufficient evidence of benefit.[4]

Advanced neuromonitoring: additional advanced monitoring can be considered, especially in children with uncontrolled ICH, depending on the local availability and expertise. The main parameters studied focus on CBF, CA, $PbtO_2$ and metabolism. The main focus is on studying CA and maintenance of CPP. Although there is limited evidence in paediatric TBI, one such parameter increasingly being studied in this population is pressure reactivity index (PRx). PRx is a correlation coefficient between MAP and ICP and relates the slow changes in systemic BP with fluctuations in ICP. In children with continuous BP and ICP monitoring, PRx can be calculated continuously to get real-time information on CA. This information can be used to calculate the "optimum" CPP where CA works best for an individual patient at a specific point in time. Similar indices with blood flow velocities have been calculated by TCD but are difficult to measure in real-time.

In organizations where $PbtO_2$ monitoring is used, a minimum target level of 10 mmHg should be maintained.[1] In general, managing ICP and CPP will improve $PbtO_2$; but specific interventions can increase $PbtO_2$ such as raising FiO_2, MAP and $PaCO_2$, and optimizing the Hb.

Weaning: the duration of neuroprotection varies but is usually continued for a minimum of 72 hours to accommodate for developing cerebral oedema. A period of stability with acceptable ICP and CPP for 24 hours is required before weaning is attempted. The rate of withdrawal depends on the severity of disease and intensity of interventions applied.

Conclusion

Outcomes from sTBI in children can be improved by early and aggressive management. This begins on-scene and continues in the PICU, ideally in a trauma centre with all the required expertise. Hypoxia and hypotension should be aggressively prevented/treated while managing these critically-ill children. Standard neuroprotective strategies follow an ABCDE approach with additional emphasis on analgesia/sedation and temperature. Further management involves invasive ICP monitoring with careful titration of ICP and CPP for best outcomes. Hyperosmolar therapy is the only positive Level II recommendation in TBI. Advanced neuromonitoring techniques are used depending on the availability and local expertise. First and second tier therapies are indicated using a pragmatic approach when ICP remains elevated despite optimal baseline measures. The need for neurosurgical intervention should be reviewed at presentation and in the event of elevated ICP, refractory ICH or concerns of herniation. Outcomes are best with a multidisciplinary team approach with good communication between all team members. ◆

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