Review Article

Management of Severe Traumatic Brain Injury in Adults

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Abstract

Head injury is a major cause of mortality and morbidity in India and responsible for two lakh deaths each year. In addition, over ten lakh people require rehabilitation services each year. The management of head injury has been revolutionised through the introduction of evidence based recommendations by the Brain Trauma Foundation. This article is broadly based around these recommendations and deals with the emergency room, anaesthetic and intensive care management of head injured patients. It recommends the introduction of protocols by each and every institution dealing with head injured patients in order to streamline their management.

Key words : Head injury, Intracranial haematoma, Glasgow coma scale, Intracranial pressure.

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Introduction

India has unenviable distinction of having the highest rate of Traumatic Brain Injury (TBI) in Asia from road traffic accidents and falls[1]. Approximately ten lakh people suffer from severe TBI out of which nearly 2 lakh die. Over ten lakh people require rehabilitation services each year. 60% of TBI are due to road traffic accidents. Falls are responsible for 20 – 25% of cases and occur predominantly in children and elderly. Violence results in 10 % of cases. Alcoholic intoxication is present in 15 – 20% of patients suffering from TBI. 40% of cases are seen in the age group of 21 – 35 years, 20% under 15 years of age and 5% over 65 years. 80% of cases are seen in males. 66% of cases occur in the evening and nights. 71% of cases of TBI are mild, 15% of cases are moderate and 13% of cases are severe. The major behavioural factors responsible for TBI are non-usage of helmets, alcohol influence, over-speeding, dangerous overtaking and careless crossing of roads[2].

Pathology of TBI

Primary injury is the damage produced by the direct mechanical impact and acceleration deceleration stress on the skull and brain tissue. Skull fractures occur over the cranial vault and may be depressed fractures compressing the underlying brain. Fractures involving the skull base result in blood and cerebrospinal fluid leakage into the nose, pharynx or external auditory meatus. Periorbital haematomas (racoon / panda sign) and retro-auricular haematomas (battle sign) are also seen in base of skull fractures.

Intracranial injuries may be classified into diffuse and focal injury. Diffuse brain injury consists of brain concussion or diffuse axonal injury. Concussion is definite retrograde and post traumatic amnesia, even if it is for a few minutes. Diffuse axonal injury is diagnosed on the basis of radiological imaging and should be suspected in coma lasting for more than 6 hours. Focal brain injury consists of brain contusion (coup or contre coup injuries), extradural haematoma, subdural haematoma and intracerebral haematoma (usually located in the frontal or temporal lobes). Subarachnoid haemorrhage is also seen in many cases. (Fig 1 - 4).

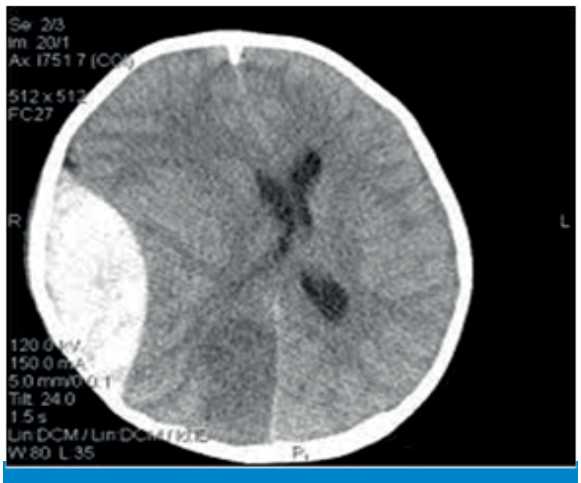

Fig 1 - Acute Extradural Haematoma

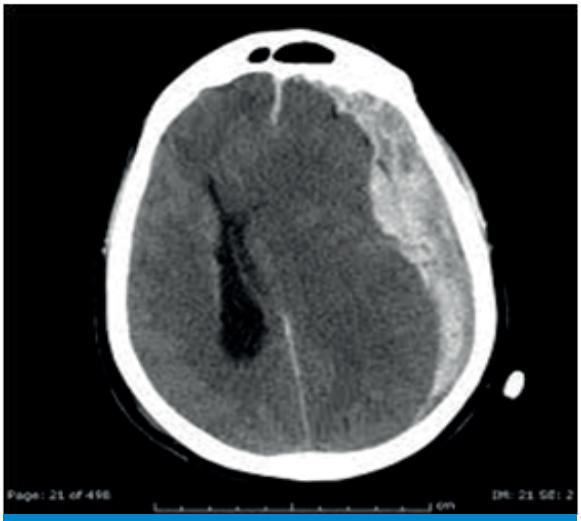

Fig 2 - Acute Subdural Haematoma

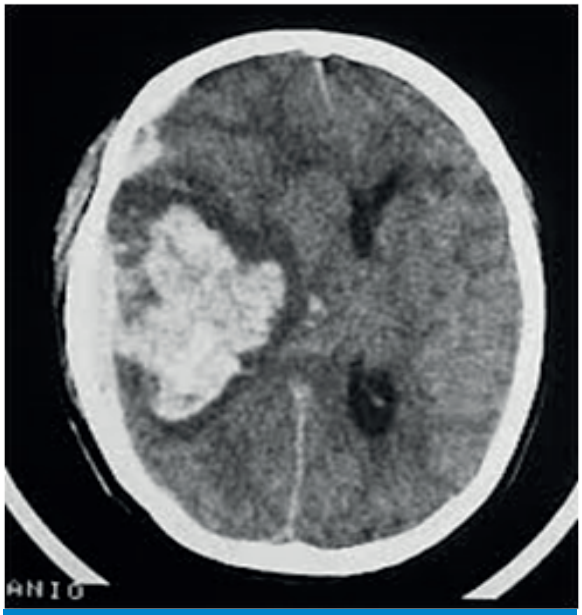

Fig 3 - Intra Cerebral Haematoma

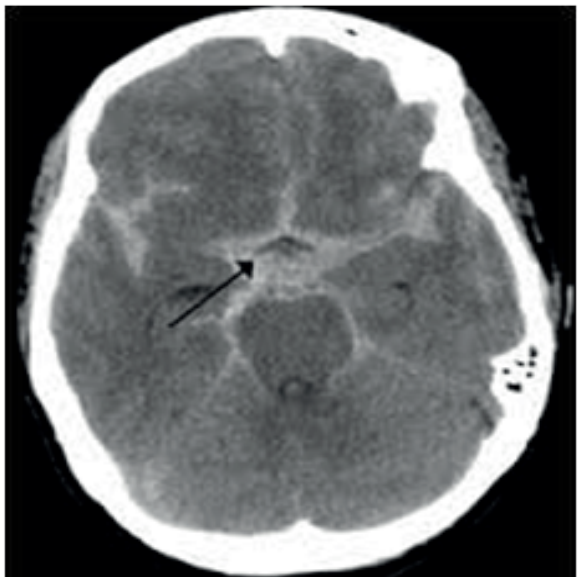

Fig 4 - Sub Arachnoid Haemorrhage

In gunshot injuries to the head, the predominant components are subdural and intra cerebral haematoma and cerebral oedema. High velocity injuries are more difficult to treat due to the cavitation effect of the bullet compared to low velocity injuries (bullet velocity less than 360 metres sec^{-1}) which inflict less damage on the parenchyma.

A lucid interval may occur in extradural and subdural haematomas. An acute subdural haematoma is usually associated with raised intracranial pressure (ICP). A subdural haematoma is acute if the patient becomes symptomatic within 72 hours, sub-acute if between 3 – 15 days and chronic after 2 weeks.

In secondary injury, cerebral hypoxia induced by the primary injury leads to brain swelling which causes the ICP to rise and further worsen the cerebral hypoxia. This vicious cycle is exacerbated by a number of factors whose avoidance will result in a more favourable outcome (Table – 1). All these factors should be diagnosed and treated on an emergency basis in order to limit or prevent secondary brain injury.

Table 1: Factors aggravating secondary brain injury

Extracranial Factors	Intracranial Factors
Airway obstruction, Hypoxia, Anaemia, Hypercarbia, Hypocarbia, Hypotension, Venous congestion, Hypoglycaemia, Hyperglycaemia, Hyponatremia,Pyrexia, Sepsis, Volatile anaesthetic agents.	Haematoma, Raised intracranial pressure, Seizures, Vasospasm, Infection, Cerebral oedema, Herniation, Hydrocephalus, Cerebral ischaemia.

Pre-Hospital management of TBI

The main goals of emergency therapy in the field and emergency department are to prevent and treat all systemic (extracranial) and intracranial insults that cause secondary neuronal injury and ultimately improve outcome in patients with severe TBI. The severely brain injured patient (GCS of 8 or less) should be immediately transported to a centre with round the clock CT scanning facility, operation theatre facility, neurosurgical care and the ability to monitor ICP and treat intracranial hypertension in an intensive care setup. Earlier evacuation of intracranial haematomas when and if indicated has a more favourable outcome. If the airway is compromised, it should be secured at the scene of accident itself. Controlled trials are underway in various developed countries to determine whether the presence of a physician at the scene of accident will decrease morbidity and mortality from TBI.

Emergency department management of TBI

Neurological assessment should ideally be done before endotracheal intubation. The Glasgow Coma Scale (Table – 2) was proposed by Teasdale and Jennet in 1974[3]. The aim was to allow comparisons between various head injury units. The responses are associated with a points system which gives a classification of the degree of impairment. The lower the score, the more severe the impairment. The minimum score is 3 and the maximum score is 15.

Table 2: Glasgow Coma Scale (Adult)

Feature	Parameter	Score
Eye opening	Spontaneous	4
	To verbal command	3
	To pain	2
	None	1
Best verbal response	Oriented, conversing	5
	Disoriented, conversing	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No verbal response	1
Best motor response	Obeys verbal commands	6
	Localizes to pain	5
	Withdrawal	4
	Abnormal flexion (Decorticate)	3
	Extension (Decerebrate)	2
	No motor response (Flaccid)	1

Mild TBI – GCS of 14 to 15. Loss of consciousness if present is less than 5 minutes. These cases require close neurological monitoring but not intensive care.

Moderate TBI – GCS of 9 – 13. These patients have a mixed prognosis.

Severe TBI – GCS of 8 or less persisting for 6 hours or more. Virtually all these patients require ICU admission and ICP monitoring.

Pupillary size, any inequalities, reflex response to light and accommodation should also be assessed along with symmetry of motor function in the extremities. Injuries to other organ systems should then be assessed. Intra-thoracic and intra-peritoneal injuries should be treated without delay. Management of bleeding causing haemorrhagic shock takes precedence over neurosurgical procedures.

Brain herniation syndromes are due to mechanical compression by an accumulating mass or a diffusely increased ICP. Almost all cases present with progressive somnolescence and the Cushing's reflex (hypertension & bradycardia). The following herniation syndromes are described (Figure-5):

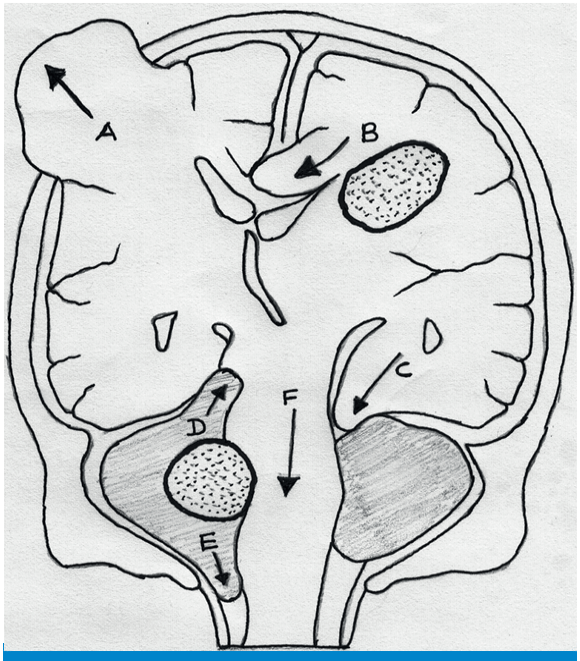

Fig 5 - Brain Herniation Syndromes

A. In Trans-calvarial herniation, the brain protrudes through a defect in the skull. This is often worsened by raised ICP.

B. In Subfalcine herniation, the cingulate gyrus of the frontal lobe is pushed under the falx cerebri. Clinical signs include increased tone or paresis in the contralateral leg.

C. In Uncal herniation, there is displacement of the medial edge of the uncus and the hippocampal gyrus medially and over the ipsilateral edge of the tentorial foramen causing compression of the midbrain. The ipsilateral or contralateral occulomotor nerve may be stretched or compressed. Initially there will be asymmetric pupillary dilatation (anisocoria) which will progress to ipsilateral pupillary dilatation and contralateral hemiparesis.

D. Upward cerebellar herniation is uncommon and is usually due to upward herniation of vermis and cerebellar hemispheres through the tentorial foramen due to infratentorial mass lesions. Radiology usually reveals effacement of the superior vermian cistern, compression of the fourth ventricle, upward and forward displacement of the quadrigeminal plate, mesencephalon and cerebral aqueduct causing supratentorial hydrocephalus. If left untreated, there will be medullary compression leading to bradycardia and ventilatory arrest.

E. Tonsillar herniation is due to a supra or infra tentorial mass lesion and is a rapid and often fatal event unless recognized immediately and treated. The cerebellar tonsils descend through the foramen magnum compressing the lower brain stem resulting in ventilatory arrest. Of all the herniation syndromes, this one has the narrowest window of intervention to prevent death.

F. Cerebral transtentorial herniation is characterised by displacement of the cerebral hemispheres and basal ganglia downwards while the diencephalon and adjacent midbrain are pushed through the tentorial notch. This is usually due to lesions occupying the intracranial vertex or frontal-occipital poles. Clinical presentation will be an impairment of vertical gaze and bilateral extensor posturing.

Tracheal Intubation – Tracheal intubation is indicated in all cases with a GCS of 8 or less. All head injured patients should be considered as having a full stomach. Many of them have an associated cervical spine injury. Rapid sequence intubation with manual in line stabilization (MILS) is preferred in haemodynamically stable patients. The patient is pre-oxygenated with 100 % oxygen and anaesthesia is induced with Thiopentone 3 – 4 mg Kg^{-1} or Propofol 1 – 2 mg Kg^{-1}. Succinyl Choline 1.5 mg Kg^{-1} is then administered and the trachea intubated. Applying cricoid pressure if a cervical spine injury is not ruled out is controversial. One recommendation would be to apply cricoid pressure after placing the posterior part of the Philadelphia hard collar.

Routine intravenous induction is carried out in a patient without full stomach and stable haemodynamics by titrating the Thiopentone or Propofol dosage to minimize hypotension. Rocuronium 0.6 - 1.0 mg Kg^{-1} may be used to facilitate endotracheal intubation since

it has no effect on cerebral dynamics. Lignocaine 1.5 mg Kg^{-1} given 90 seconds before laryngoscopy will blunt any haemodynamic response and attenuate any rise in ICP. A small dose of propofol or fentanyl or a short acting beta-blocker like esmolol may also be used to blunt the laryngoscopic response.

Ketamine is contraindicated in the routine management of TBI since it elevates the ICP. However, it may have a small role to play in inducing patients in haemorrhagic shock. Midazolam at 0.2 mg Kg^{-1} may be used for induction. It has minimal haemodynamic effects and hence the cerebral circulation is usually not disturbed. It however interferes with neurological assessments. Fentanyl produces minimal to moderate decreases in MAP and CPP at a dose of 2 – 4 mcg Kg^{-1}. It increases the ICP to a moderate extent[4]. When larger doses are used, measures to maintain systemic BP may be required.

If facial fractures and soft tissue oedema prevent direct visualization of the larynx, a fibre-optic intubation or intubation over an illuminated stylet may be tried. Avoid nasal intubation if a base of skull fracture is suspected to avoid intracranial placement of the endotracheal tube or introduction of contaminated material into the brain. Nasal intubation should also be avoided in the presence of severe facial bone fractures or a bleeding diathesis. Instruments for an emergency tracheostomy should be available. Once the airway is secured, insert an oro-gastric tube to decompress the stomach.

Antibiotics should be administered at the time of intubation to reduce the incidence of pneumonia. However, prophylactic antibiotic use for ventricular catheter placement to reduce infection is not recommended[21].

After securing the airway, mechanical ventilation is initiated and the respiratory rate and tidal volume are adjusted to maintain a $PaCO_2$ of 35 mm Hg. Hyperventilation to a $PaCO_2$ of 30 mm Hg is indicated only if transtentorial herniation is suspected. Prophylactic hyperventilation to a $PaCO_2$ of less than 25 mm Hg is not recommended. Hyperventilation is used only as a temporary measure to reduce ICP and should ideally be avoided during the first 24 hours after injury. If hyperventilation is used, Jugular venous oxygen saturation or brain tissue oxygen tension should be monitored. The Fraction of inspired oxygen (FiO_2) and positive end expiratory pressure (PEEP) are adjusted to maintain a PaO_2 of 100 mm Hg.Hypoxaemia (PaO_2< 60 mm Hg or SpO_2 < 90 %) should be avoided[5]. A PEEP level upto 12 cm H_2O is safe in head injury and does not raise the ICP[6]. Use a fibreoptic bronchoscope to suction out aspirated material from the bronchi.

Fluid resuscitation and haemodynamic stabilization – Fluid resuscitation should be guided by systemic blood pressure, urine output and central venous pressure. Sympathetic over activity in patients with severe TBI along with a reflex response to raised ICP often maintains the blood pressure at normal levels. Induction of anaesthesia in these patients often unmasks hypovolaemia and precipitates hypotension.

Total osmolality is the most important factor that determines the formation of cerebral oedema. Normovolaemia should be maintained in these patients using isotonic and hypertonic solutions. Plasma total osmolality should however be maintained at less than 320 mosm Kg^{-1}.When large volumes of crystalloid are required, use an isotonic solution like Normal saline (308 mosm L^{-1}). Hypertonic saline (3 %) can be beneficial in severe TBI. In addition to being a useful resuscitation fluid, hypertonic saline reduces cerebral oedema and ICP. A rebound phenomenon is not seen following the use of this solution. The dose is 125 to 250 ml every 6 hours. It may also be used as a continuous infusion at 0.5 – 1.0 ml Kg^{-1} $hour^{-1}$. Stop hypertonic saline once the serum sodium concentration reaches 160 mEq L^{-1}. Potential adverse effects include central pontine myelinolysis, seizures, congestive heart failure, hypokalemia, hyperchloraemic acidosis and coagulopathy.

Hydroxy Ethyl Starch (HES) and albumin have also been used for resuscitation. The maximum dose of HES is 20 ml / Kg. HES can worsen coagulopathy which is seen in approximately 20 % of patients with TBI. Dextrans should be avoided because of their effect on platelet function.

Blood should be transfused to maintain a haematocrit over 30 % while blood products may be needed to correct coagulation defects such as disseminated intravascular coagulation (DIC) induced by the release of brain thromboplastin.

Ringer's Lactate is hypotonic (273mosmKg^{-1}) and will result in an increase in cerebral oedema and ICP as well as contribute to lactic acidosis in ischaemic areas of the brain. Dextrose containing solutions should be avoided because hyperglycaemia is associated with a poorer neurological outcome. In addition, the free water load left after the glucose is metabolised will cause a reduction in plasma osmolality and worsen cerebral oedema.

If the blood pressure and cardiac output cannot be restored to normal through fluid resuscitation alone, inotropes may have to be administered. An infusion of dopamine or phenylephrine is preferred over other inotropes.

Radiological Evaluation – CT scanning remains the radiological investigation of choice for evaluating TBI. Indications for scanning TBI patients may be according to the Canadian criteria[7] or New Orleans criteria[8] (Table – 3). Brain imaging helps to distinguish cases that require immediate surgical intervention from those that do not and may also identify those whose protracted recovery requires early tracheostomy and feeding gastrostomy.

Indications for early surgical intervention based on CT findings include open depressed skull fractures with dural laceration and various haematomas. An extra dural haematoma with a volume of over 30 ml should be evacuated regardless of GCS. An acute EDH should be evacuated if associated with a GCS of 8 or less and unequal pupils[9].One however has to keep in mind that the acute EDH may be just an incidental finding and not

Table 3: Computed Tomography scanning rules for minor head injury

Canadian Criteria	New Orleans Criteria
High Risk GCS < 15 at 2 hours after injury Suspected open / depressed skull fracture Any sign of base of skull fracture More than one episode of vomiting Age over 64 years **Medium Risk** Amnesia before impact of > 30 minutes High risk mechanism of injury	Persistent anterograde amnesia with GCS of 15 Intoxication (Alcohol / Drugs) Physical evidence of trauma above the clavicles Age > 60 years Seizures (witnessed or suspected) Headache Vomiting Coagulopathy

contributing to the low GCS. The neurosurgeons clinical judgement and wisdom also plays a role before taking up the patient for haematoma evacuation. An acute subdural haematoma of over 10 mm thickness or if associated with a midline shift of over 5 mm should be evacuated. An acute SDH of less than 10 mm thickness with a GCS of less than 9 should have their ICP monitored[9]. A contusion (usually of the temporal lobe and less commonly the frontal lobe) if associated with compression of the basal cisterns should be treated surgically to avoid herniation.

Diffuse Axonal Injury (DAI) is a diffuse nonfocal pattern of injury for which surgical treatment is not indicated unless intractable intracranial hypertension develops. The Marshall classification of initial CT scan appearance categorizes patients with DAI (Table – 4). CT scan appearance in mild DAI includes loss of grey white differentiation, ventricular compression and intra-ventricular blood. Severe high velocity injuries may be associated with multifocal contusions, oedema, effacement of basilar cisterns and brain stem compression. Magnetic Resonance Imaging (MRI) is better than CT scan in revealing the diffuse nature of the injury. Findings include punctate haemorrhages in the peri-ventricular white matter, corpus callosum and brain stem, traumatic sub-arachnoid haemorrhage, intraventricular haemorrhage and tissue tear haemorrhages.

Table 4: Marshall Classification of initial CT scan appearance in patients with Diffuse Axonal Injury

Injury Grade	CT Appearance	Mortality
I	Normal CT scan	9.6 %
II	Cisterns present - shift < 5 mm	13.5 %
III	Cisterns compressed / absent – shift < 5 mm	34 %
IV	Cisterns compressed / absent – shift > 5 mm	56.2 %

Anaesthetic management of TBI

The main goals of anaesthetic management are the optimization of cerebral perfusion and oxygenation, prevent secondary injury and provide good surgical conditions.There is no single way of anaesthetising these patients and the suggested management is what is being generally practiced by the author. General anaesthesia is preferred since it facilitates good control of the ventilatory and circulatory systems. Many patients reach the operation theatre with an endotracheal tube in-situ. In patients who are yet to be intubated, anticipate a full stomach, a contracted intravascular volume due to bleeding elsewhere and a possible cervical spine injury.

Cannulate the radial or dorsalis pedis artery for direct blood pressure monitoring before induction. The arterial BP transducer should be zeroed at the level of the circle of Willis (external auditory meatus). Hypertension should be treated cautiously because it may be a compensatory hyperactivity of the sympathetic nervous system in response to raised ICP (Cushings reflex). Assess adequacy of volume replacement, analgesia, ventilation and oxygenation. If necessary, treat hypertension with a beta-blocker like esmolol which has a minimal effect on cerebral dynamics. The cerebral perfusion pressure (CPP) is maintained over 60 mm Hg. A higher CPP may be required in hypertensives, elderly patients and those with cerebrovascular disease. A head up tilt of 10 – 30 degrees should be used to facilitate venous return and CSF drainage. Any rotation or flexion of the head and neck for surgical positioning should not obstruct venous return from the cranium.

The ideal agent for maintenance of anaesthesia should reduce ICP, protect the brain against any ischaemic or metabolic insults and maintain adequate oxygen supply to the brain tissue. Thiopentone infusions decrease CBF, cerebral blood volume and ICP. They protect against focal brain ischaemia. They can however depress the cardiovascular system which can result in systemic hypotension and worsening of cerebral ischaemia. Prolonged infusions can result in persistent sedation.The cerebral haemodynamic effects of propofol are similar to thiopentone. One advantage of propofol is that emergence from anaesthesia is rapid even after a prolonged infusion. Hypotension during a propofol infusion may be attenuated by correcting any hypovolaemia before starting it. Patients receiving propofol infusions have a reduced incidence of postoperative nausea and vomiting. Prolonged infusions of propofol at high doses can produce significant morbidity[10]. Etomidate is similar to thiopentone in reducing CBF, $CMRO_2$ and ICP with better cardiovascular stability. Prolonged usage may however cause adrenocortical suppression.

Inhalational agents decrease $CMRO_2$ through direct action and increase CBF by causing cerebral vasodilatation. Concentrations of less than 1.0 MAC should be used. It is advisable to avoid volatile agents in patients with raised ICP altogether and use an intravenous technique instead. It is the author's practice to maintain anaesthesia with an infusion of propofol till the dura is opened. Isoflurane is a potent depressant of $CMRO_2$ and increases CBF only over 1.0 MAC concentration. Sevoflurane has a lesser effect on cerebral haemodynamics than isoflurane. Sevoflurane is however metabolized to Compound A on contact with soda lime in the closed circuit and this substance may achieve toxic levels during prolonged anaesthesia with low flows. Desflurane at higher concentrations increases CBF and ICP. Both Sevoflurane and Desflurane are much more expensive than Isoflurane.

Nitrous Oxide also dilates cerebral vessels and increases ICP. In addition, it should be avoided if a pneumocephalus is demonstrated on CT scan of the brain as it diffuses into the air space increasing its volume and pressure.

Muscle relaxants facilitate IPPV and reduce ICP. Coughing and straining, which can increase ICP may be avoided with their use. Vecuronium has no effect on ICP, systemic blood pressure or heart rate. Cis-Atracurium has little effect on ICP and does not depend on the liver or kidneys for elimination. It does not release histamine like atracurium. Rocuronium is useful for intubation because of a rapid onset of action and lack of any effect on the ICP.

Local anaesthetics should be infiltrated at the site of skin incision and skull pin insertion to prevent systemic and intra-cranial hypertension.

Acute brain swelling and protrusion through the craniotomy should be treated by adjusting the ventilation, oxygenation, depth of anaesthesia [Bolus dose of Thiopentone (15 – 30 mg Kg-1)], osmotic diuretics and muscle relaxants. CSF drainage (10 – 20 ml) through an intraventricular catheter may also be used to immediately reduce the ICP.

Monitoring – Routine monitoring includes ECG, noninvasive BP, invasive BP, pulse oximetry, end tidal CO_2, temperature, urine output, central venous pressure, neuro-muscular blockade, arterial blood gases and laboratory investigations like haematocrit, serum electrolytes, plasma glucose and serum osmolality. Some of the methods of monitoring described are not available in the author's institution and are mentioned only for the sake of completion.

ICP Monitoring – The ICP should be monitored as a guide to therapy and also to assess the response to therapy and determine the prognosis. It should ideally be monitored in all salvageable patients with a GCS of 8 or less and an abnormal CT Scan (one that reveals a haematoma, contusion, swelling, herniation or compressed basal cisterns)[11]. The ventricular catheter connected to an external transducer is the most accurate and reliable monitor of ICP and allows easy calibration and drainage of CSF to reduce ICP. CPP can be calculated easily (MAP – ICP). Treatment is initiated if ICP is persistently elevated over 20 mm Hg[12]. ICP can also be measured from epidural, subarachnoid and intraparenchymal locations (Figure-6).

ICP monitoring is usually done from the supra tentorial region of the cranium. Intraventricular catheter placement may be difficult if the ventricle is compressed due to high ICP. They have been associated with a higher incidence of infection and a greater potential to cause brain tissue injury during placement. Monitoring ICP from subarachnoid, subdural and epidural locations are less accurate than intraventricular catheters. Fibreoptic ICP monitoring systems may be placed in the parenchyma, subdural or intraventricular compartment. They are expensive, prone to drift, cannot be calibrated in vivo and fragile. Micro-pressure transducer ICP monitoring systems do not have the disadvantages of the fibreoptic systems.

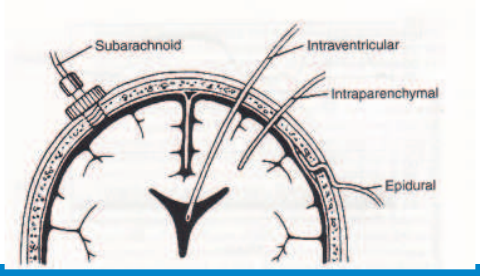

Fig 6 - ICP monitoring from various locations

SjO_2 – Jugular venous oxygen saturation (SjO_2) provides continuous information about the balance between global cerebral oxygen demand and supply. Retrograde cannulation of the internal jugular vein is required. Normal SjO_2 is 60 – 75 % (Table – 5). A reduction in SjO_2 can be caused by hyperventilation, decreased CPP or cerebral vasospasm and a SjO_2 of less than 50 % for over 15 minutes is associated with a poor neurological outcome[13].

Table 5 - Jugular venous oxygen saturation – clinical correlation

SjO_2	Clinical condition
< 55 %	Ischaemic levels of CBF
55 – 60 %	Hypoperfusion
60 – 75 %	Normal range
75 – 90 %	Hypoperfusion
> 90 %	Brain death

Transcranial doppler – High values for flow velocity may indicate vasospasm or hyperaemia. Values over 120 cm sec-1 in the middle cerebral artery correlates with angiographically demonstrated vasospasm and values over 200 cm sec-1 indicates severe vasospasm. TCD also helps to assess CO_2 reactivity, auto-regulation, response to treatment and also helps to estimate CPP.

Brain Tissue Oxygen Tension (BTOT) – This is measured using a probe placed in the cerebral parenchyma. A BTOT of lesser than 15 mm Hg for over 30 minutes is associated with increased mortality[13]. The main disadvantage of BTOT monitoring is that it gives only a focal picture.

Near Infrared Spectrometry – Oxy-haemoglobin and deoxy-haemoglobin are strong absorbers of light in the near infrared spectrum. Differences in their absorbtion spectra allows the measurement of oxygen saturation in cerebral blood under the sensor through the skull bone. This is an indirect indicator of adequacy of CBF.

Monitoring for air embolism – Posterior fossa procedures in the sitting position carry the highest risk of venous air embolism. Methods for the detection of venous air embolism are listed in Table – 6 (from the most sensitive to the least sensitive).

Recovery from Anaesthesia and Postoperative management - Patients who had a good GCS preoperatively and have undergone an uneventful surgery may be awakened and extubated in the operation theatre. This facilitates early neurological assessment. Avoid systemic hypertension and

coughing while emerging from anaesthesia as they are predisposed to cerebral oedema, raised ICP and haematoma formation. If the level of consciousness is depressed preoperatively, ventilate the patient electively in the postoperative period. Poly trauma patients and patients who are hypothermic should also be ventilated electively in the post-operative period. Causes of delayed awakening in the operation theatre include a low preoperative GCS score, elevated ICP, residual drug effects, metabolic and electrolyte disturbances, hypothermia and seizures.

Table 6 - Methods for detecting air embolism

Trans oesophageal Echocardiography (0.02 ml / Kg)
Precordial Doppler (0.05 ml / Kg)
Pulmonary Artery Catheter (0.25 ml / Kg)
Expired CO_2 / N_2 monitoring (0.5 ml / Kg)
Fall in oxygen saturation
Change in BP and heart sounds (mill wheel murmur)

Intensive care management of the TBI patient

The indications for monitoring ICP have been listed in the previous section. An intraventricular catheter is preferred since it provides accurate readings and allows therapeutic drainage of CSF. Loss of autoregulation induced by TBI will result in the CBF becoming dependant on the mean arterial pressure (MAP). Reductions in MAP will then cause cerebral ischaemia while increases in MAP results in hyperaemia, cerebral oedema and rise in ICP. The Lund approach to treat raised ICP works on the assumption that the integrity of the BBB for salts is lost following TBI. The goal of management is to limit the resulting oedema and ICP elevation by using osmotic diuretics and maintaining an ICP of less than 20 mm Hg.

The CPP should be maintained between 50 and 70 mm Hg to maximize brain tissue oxygenation. Arterial hypotension (SBP < 90 mm Hg) should be avoided[12]. The systolic BP should be maintained between 90 and 160 mm Hg. Adequate MAP is maintained by infusing isotonic fluids to maintain euvolaemia or slight hypervolaemia. Intravascular volume status can be assessed by monitoring the CVP and urine output, respiratory variation in the arterial pulse pressure and ultrasonographic assessment of inferior vena cava size changes with different phases of breathing.

Management of raised ICP – CPP is the difference between MAP and ICP and should be maintained over 60 mm Hg. A head up tilt of 10 – 30 degrees should be used to facilitate venous return and CSF drainage.

The head and neck should be maintained in a neutral position so that venous return from the cranium is not obstructed. Tight endotracheal tube ties should not be used for the same reason. Incremental drainage of CSF through an intraventricular catheter (15-20 ml) will bring down the ICP immediately.

Mannitol and Frusemide may be used as diuretics. Mannitol 0.25 – 1.0 gm Kg^{-1} is administered over 10 - 20 minutes and repeated every 3 – 6 hours. A larger dose of 1.0 – 1.5 gm Kg^{-1} is used if transtentorial herniation is suspected. It is recommended to restrict usage of mannitol, prior to monitoring ICP, to patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes[14]. The serum osmolality should be monitored and maintained at less than 320 mosm Kg^{-1}. Frusemide prevents the rebound phenomenon seen with mannitol. Infusions of hypertonic saline and glycerol are also being used in the management of raised ICP.

Mechanical ventilation should be adjusted to maintain a PaCO2 of 35 mm Hg. Lower levels of 30 mm Hg is indicated only if impending herniation is suspected and is used as a bridge to surgical decompression. Levels below 30 mm Hg can produce vasoconstriction and aggravate cerebral ischaemia. Once hyperventilation is instituted, other measures to reduce ICP should be introduced and the PaCO2 allowed to normalize as soon as possible.

Decompressive Craniectomy

Decompressive craniectomy is a controversial line of management and is practiced in certain centres only. Post surgical quality of life should be considered before planning this procedure. Decompressive craniectomy decreases ICP and improves outcome in young patients who deteriorate within 48 hours of TBI. It should be considered prior to initiating barbiturate therapy (< 55 years of age is a relative indication). It should be considered in all cases of non-fatal primary brain injury associated with asymmetric or focal brain swelling on CT scan who have refractory intracranial hypertension which has failed to respond to maximal medical therapy, with or without CSF drainage.

Barbiturates – Barbiturates have cerebral protection and ICP lowering effects and must be considered in severe TBI cases in which raised ICP is refractory to medical and surgical management. Barbiturate therapy should be initiated only after the patient is haemodynamically stable. Clinical trials have however not revealed any clear benefit or improved outcome associated with the use of high dose barbiturate therapy for refractory intracranial hypertension. High dose barbiturate therapy is indicated in hemodynamically stable salvageable patients who have raised ICP refractory to the previous steps. Barbiturates reduce ICP by decreasing CBF in parallel with CMRO2. Thiopentone is administered in a loading dose of 15 mg Kg^{-1} followed by an infusion of 0.2 mg $Kg^{-1}min^{-1}$. Thiopentone infusions are associated with haemodynamic instability, prolonged recovery after stopping the infusion and difficulty in neurological assessment. Prophylactic administration of barbiturates to induce burst suppression in head injured patients is not recommended[15].

Hypothermia – Mild hypothermia (34 – 36 degrees) has been demonstrated to markedly attenuate ischaemic cerebral injury in animal models. The mechanism may be due to a reduction in metabolic demand, free radical formation and oedema formation. Adverse side effects of hypothermia include hypotension, cardiac arrhythmias, coagulopathies and infection. Rewarming

should be carried out gradually. Studies indicate that there is a greater reduction in mortality risk when hypothermia is maintained for more than 48 hours[16]. Hypothermia is not routinely used in clinical practice.

Steroids – The CRASH trial which studied the effect of early administration of methyl prednisolone on outcome after TBI in 10008 patients revealed a higher risk of death within two weeks of injury in the group receiving steroids than in the group receiving placebo[17]. There was also a higher risk of subsequent death or severe disability. The trial investigators concluded that corticosteroids should not be routinely used in the treatment of TBI. Besides steroid administration also causes hyperglycaemia and is associated with an increased risk of gastrointestinal haemorrhage.

Anticonvulsants – Administration of phenytoin to severe TBI patients prevents early onset seizures, but has no effect on late post traumatic seizures. Seizure prophylaxis reduces the incidence of seizures but has not been shown to improve neurological outcome. Phenytoin should be infused at a rate of less than 50 mg min^{-1} in adults to avoid cardiovascular depression. Indications for seizure prophylaxis include a GCS of less than 10, cortical contusions, depressed skull fracture, intracranial haematoma, penetrating head injury and seizures occurring during the first 24 hours after TBI. Seizure prophylaxis is recommended during the first week after TBI. Phenytoin at a loading dose of 15 mg Kg^{-1} over 20 minutes followed by 5 – 7 mg Kg^{-1} day-1 or Fosphenytoin 15 – 20 mg Kg^{-1} loading dose followed by 4 – 6 mg Kg^{-1} day^{-1} is used. A multicentre prospective study has recently concluded that there is no statistically significant difference between levetiracetam and phenytoin in early TBI seizure prophylaxis[18].

Nutrition - Nutritional support should be initiated as soon as possible. Full nutritional support should be achieved by the seventh post injury day. 15 % of total calories should come from proteins. Enteral feeding is preferred and a naso-jejunal tube should be placed to protect against aspiration and gastric intolerance. Stress ulcer prophylaxis is indicated with a H2 inhibitor or proton pump inhibitor (PPI). At least two studies have shown that neurological outcome is poorer if the plasma glucose levels are over 200 mg dl^{-1}[19,20]. Attempts to maintain euglycaemia (80 – 110 mg dl^{-1}) may carry an excessive risk of hypoglycaemia which is not good for the brain. In the absence of clear guidance, maintaining a value between 120 – 160 mg dl^{-1} is probably the safest course of action.

Early tracheostomy should be performed to reduce the duration of mechanical ventilation. However it does not alter mortality or rate of nosocomial pneumonia. Early extubation in qualified patients may be done without increased risk of pneumonia[21].

Deep vein thrombosis (DVT) prophylaxis with TED stockings or pneumatic compression devices should be started as soon as possible. Their use should be continued until patients are ambulatory. Low molecular weight heparin can usually be used in combination with mechanical prophylaxis. There is however an increased risk of expansion of intracranial haemorrhage. There is insufficient evidence to support recommendations regarding the preferred agent, dose or timing of prophylaxis[22]. Any coagulopathy if present should be corrected. Coagulation is impaired in these patients due to release of brain thromboplastin, haemorrhage induced decrease in coagulation factors and increased levels of fibrin degradation products.

Pyrexia (Fever) increases CMRO2 and ICP and may worsen outcome in TBI. It should be treated with cooling blankets and paracetamol. Causes for fever in TBI patients include phenytoin, infection and injury to hypothalamus. The treatment goal should be to maintain normothermia (body temperature of 37°C) in these patients.

Complications of TBI

Neurogenic Pulmonary Oedema –The aetiology of neurogenic pulmonary oedema is massive transient central sympathetic discharge due to raised ICP following severe TBI and is particularly associated with hypothalamic lesions. It may rapidly progress to death or resolve completely within a few hours to days. There is systemic vasoconstriction, redistribution of blood from the systemic to the pulmonary circulation,left ventricular failure, pulmonary venoconstriction and increased pulmonary capillary permeability. The treatment is to decrease ICP, reduce systemic blood pressure using diazoxide (1-3 mg IV) every 5 minutes upto 150 mg or phenoxybenzamine (1 mg Kg^{-1} as an infusion over 2 hours to a maximum of 4 mg Kg^{-1} day-1), IPPV to support ventilation and inotropes to support the circulation if needed.

Syndrome of Inappropriate ADH secretion – SIADH causes water retention along with continued excretion of sodium. This causes dilutional hyponatremia, decreased serum osmolality, increased urine osmolality and decreased urine output. Water retention and serum hypo-osmolality cause nonspecific signs of water intoxication like nausea, vomiting, headache, irritability, disorientation, seizures and coma. Treatment consists of water restriction, loop diuretics and hypertonic saline. Mild cases are treated with water restriction alone (1-1.5 L day^{-1}). More severe cases are treated with water restriction and loop diuretics which impair the ability of the kidneys to concentrate urine (Frusemide 10 – 20 mg every 6 hours). In severe cases where the serum sodium level is less than 125 $mEqL^{-1}$, infuse hypertonic saline at 1-2 ml Kg^{-1} hr^{-1} for 2 to 3 hours. Correct serum sodium at a rate of 0.5 mEq L^{-1} hr^{-1} to avoid central pontine myelinolysis.

Diabetes Insipidus – DI is less commonly seen in TBI. It develops 12-24 hours after injury and lasts for a few days. A decrease in ADH levels results in the excretion of large volumes of dilute urine (4-14 L day^{-1}) that results in dehydration, hypernatremia, increased serum osmolality (over 320 $mosmKg^{-1}$), decreased urine osmolality (less than 200 $mosmKg^{-1}$) and decreased urine specific gravity (< 1.005). Serum sodium levels are often elevated (> 145 $mEqL^{-1}$). Signs of hypernatremia include decreased level of consciousness, muscle weakness, irritability, spasticity, confusion, ataxia, seizures and coma. Treatment of DI is

by calculating the free water deficit using the formula [(serum sodium – 140) X body weight(Kg) X 0.6 / 140] and replace it with a hypotonic solution like 0.45 % saline or Ringers Lactate. Desmopressin (DDAVP) is used to treat DI. The intravenous or subcutaneous dose is 0.3 mcg $Kg^{-1}day^{-1}$ in two divided doses. The oral dose is 0.05-1.2 $mgday^{-1}$ in three divided doses. The intranasal dose is 10-40 mcg day^{-1} in three divided doses. Once the intravascular volume is restored, persistent hypernatremia may be treated using Hydrochlorothiazide 50-100 mg day^{-1} IV.

Cerebral salt wasting – CSW is caused by increased secretion of atrial natriuretic peptide, brain natriuretic peptide and c-type natriuretic peptide. Natriuresis, diuresis and vasodilatation occur due to the suppression of aldosterone synthesis by these peptides.Excessive loss of sodium in urine (150-200 $mEqL^{-1}$) causes hyponatremia. Sodium should be replaced in these cases along with fluid administration. Hypertonic saline may be infused in these patients with close monitoring of serum sodium levels. The rate of correction should not exceed 0.5 mEq L^{-1} hr^{-1}.

Conclusion

The management of head injury is multidisciplinary and is a team effort involving the emergency room physician, the neurosurgeon, the neuro-anaesthetist and the intensivist. Each and every institution dealing with these cases should evolve protocols based on their needs and availability of resources to achieve the best possible results. However it should be understood that these are purely guidelines that may have to be customised to suit the individual patient and the individual consultant at that point in time. This approach has led to an improvement in the management of these cases with a reduction in mortality and morbidity.

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Industrial Toxins and Brain development

Increasing incidence of autism, attention deficit hyperactivity disorder and dyslexia among children has prompted some to claim that toxic chemicals might be triggering a "silent pandemic" of neurodevelopmental disabilities. In a report published online on February 15, 2014 in Lancet Neurology, researchers from Harvard and Mount Sinai have identified six more chemicals as "developmental neurotoxicants: manganese, fluoride, chlorpyrifos and DDT (pesticides), tetrachloroethylene (a solvent), and the polybrominated diphenyl ethers (flame retardants). Manganese is associated with diminished intellectual function and impaired motor skills; solvents are linked to hyperactivity and aggressive behaviour; and certain types of pesticides may cause cognitive delays. Controlling the pandemic is difficult as the usage of most of the industrial chemicals are poorly regulated and requires international cooperation. The authors justly call for an international action. (Philippe Grandjean, Philip Landrigan. Neurobehavioral effects of developmental toxicity. Lancet Neurology, February 2014 DOI: 10.1016/S1474-4422(13)70278-3)

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Reality And Delusion

Most of us humans are not delusional because our brain screens the conclusions we draw from our experiences through "reality testing": we discard those ideas which are not supported by evidence as unreal. For example, when we get headache, we may briefly consider brain tumour as its cause until that idea gets tested as unreal and is rejected. But delusional people cling on to their ideas even when incontrovertible evidence shows their ideas to be unreal. In a new study published in Frontiers in Psychology, Professor Philip Gerrans of University of Adelaide, claims that persistent delusions are the result of problems with "reality testing". People with "reality testing" problems find it difficult to break free from illogical ideas and suffer from severe mental health issues that may prove a threat to themselves or others. Understanding the mechanism of "reality testing" may help in providing relief to these sufferers. It would be interesting to find out if "reality testing" is functional in highly religious people and fan boys! (Philip Gerrans. Pathologies of Hyperfamiliarity in Dreams, Delusions and Déjà Vu. Frontiers in Psychology, 2014 (in press) DOI: 10.3389/fpsyg.2014.00097)

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