Management of Severe Traumatic Brain Injury (TBI) in a Prehospital Setting 4

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Abstract

Prehospital emergency care has an important role in the management of both moderate and severe TBI. The aim of the treatment and care is to prevent and decrease secondary injuries. Securing the airway, maintaining adequate blood pressure, oxygenation and ventilation are the key factors. Additionally, a rapid transport to a hospital with neurosurgical facilities is crucial.

Key words: traumatic brain injury, intubation, blood pressure, oxygenation, Glasgow coma scale

Recommendations

Level I

There are insufficient data to support a Level I recommendation for this topic.

Level II

Hypoxemia and hypotension increase morbidity and mortality.

Level III

There are insufficient data to support a Level III recommendation for this topic.

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4.1 Overview

Traumatic brain injury (TBI) is a worldwide health problem with an incidence rate of 262 per 100 000 / year. Most TBIs are mild, approximately 70-80%, with the rest being classified as moderate and severe TBIs. Severe TBI is associated with a high mortality, 30-40% (1).
TBI is a dynamic process where the primary injury occurs at the time of insult. Secondary injuries start to develop immediately after the insult and may continue to progress even days or weeks after the insult. The aim of treatment and care is to prevent and decrease secondary injuries. Quick and correct emergency treatment is therefore a corner stone of TBI care. Severe TBI patients should be transferred to a hospital with neurosurgical facilities as quickly as possible.

Tips, Tricks, and Pitfalls

- Secondary injuries start to develop within minutes of the insult
- Hypotension and hypoxia increase morbidity and mortality
- Hyper- and hypoventilation increase mortality
- Repeat neurological assessment often: GCS and pupils
- Continuous monitoring: ECG, SpO_2, blood pressure
- GCS < 9 patients need to be intubated
- Unexperienced and untrained personnel should not intubate TBI patients
- Severe TBI patients should be transported directly to level I or level II trauma centers

Summary of Prehospital Treatment

Hypoxemia (SpO_2 <90%) and hypotension (RRsys < 120 mmHg) increase morbidity and mortality. They must be avoided and promptly treated. The rule of ABC (ensure protected airway, provide adequate oxygenation and ventilation (breathing) and circulation) applies to the emergency care of TBI. Patients who have impaired consciousness (Glasgow Coma Scale ≤ 8) are unable to maintain their airway, may have impaired respiratory drive and are at risk of aspiration. Endotracheal intubation and controlled ventilation are therefore needed. Intubation is also needed if the patient has better consciousness than GCS of 9 but has concomitant multitrauma and supplemental oxygen does not correct hypoxemia. One must always assume that head injured patients have also an injured neck, until otherwise proven. Normoventilation is recommended ($etCO_2$ 35-40mmHg, 4.5-5.5 kPa) as both hyper- and hypoventilation increases mortality. However, if there are signs of cerebral herniation (i.e. pupil dilatation, Cushing's triad), short lasting hyperventilation may be considered. Hypertonic saline or mannitol may be used to decrease increased intracranial pressure (ICP) and in situations with cerebral herniation. Hypotension is treated with isotonic or hypertonic crystalloids and vasopressors such as noradrenaline or phenylephrine.

4.1 Background

4.1.1 Prehospital Assessments

4.1.1.1 Oxygenation and Blood Pressure

Hypoxemia, defined as peripheral oxygen saturation (SpO_2) < 90%, and hypotension, (previously defined as systolic blood pressure (SBP) < 90 mmHg) are both associated with high mortality and morbidity. A recent study including 13151 patients observed that 20.7% and 28.1% patients died if they had only hypotension or hypoxemia, respectively and mortality was 43.9% if both conditions existed (2). Of those patients who did not have hypotension or hypoxemia, only 5.6% died. Updated Brain Trauma Foundation (BTF, www.braintrauma.org) guidelines from 2017 recommends higher systolic blood pressure values than 90 mmHg. They recommend that SBP should be kept at ≥ 100 mmHg for patients 50 to 69 years and at ≥ 110 mmHg or higher for patients 15 to 49 or > 70 years old. In a TBI patient material consisting of over 3800 patients, aged 10 years or older, there was a linear association between systolic blood pressure, ranging from 40 to 119 mmHg, and death. An increase of systolic blood pressure of 10 mmHg was associated with a 18.8% decreased adjusted odds of death (3). A bimodal distribution of death has been observed in TBI patients, optimal SBP being between 120 and 140 mmHg (4).
Cerebral perfusion pressure (CPP) is calculated as: Mean arterial pressure (MAP) – Intracranial pressure (ICP). BTF recommends that CPP should be kept between 60-70 mmHg.
Blood pressure should be monitored frequently if it is measured noninvasively. If possible, an arterial cannulation and continuous blood pressure measurement would be preferable as it is less prone to disturbances during the

transportation and allows simple sampling for blood gas analysis. Electrocardiography and SpO_2 must also be monitored continuously.

4.1.1.2 Glasgow Coma Scale

The most common tool for assessing level of consciousness is the Glasgow Coma Scale (GCS) (Table 1). Chapter 3 will discuss this topic further and www.glasgowcomascale.org demonstrates in detail how it should be performed. GCS is a clinically important tool and it should be performed immediately after the ABC evaluation. When evaluating GCS, hypoxaemia, hypoventilation, hypotension and hypoglycemia must be corrected, otherwise GCS is not reliable. There are also confounding factors that may decrease the summed GCS score, such as. alcohol and narcotic drugs, spinal cord injury, orbital swelling and dysphasia. Repeated and documented GCS are valuable means for monitoring the trend of consciousness and for identifying possible worsening in neurological status. GCS is known to correlate with the severity of TBI. However, GCS at the scene is not such a reliable outcome predictor as GCS obtained upon admission and motor score has the best predictive power (5,6). GCS is performed by evaluating the patient's eye opening (1-4 points), verbal response (1-5 points) and best motor response (1-6 points) to speech or painful stimulus. When testing eye opening to pain, a peripheral stimulus should be used as the grimace associated with central pain may cause eye closure. If patients do not obey commands and motor response to painful stimulus is used, then central stimulus should be used. This is done by pressing the supraorbital notch. However, in cases of facial fractures adjacent to supraorbital notch, a peripheral stimulus is safer and is performed by applying slowly increasing pressure to nail tip. The points of each area are recorded separately, and the summed GCS score must be documented.

Table 1. GCS

GCS	
Eye opening	
Spontaneous	4
Speech	3
Pain	2
None	1
Verbal response	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1
Motor response	
Obey command	6
Localise pain	5
Flexor withdrawal (normal flexion)	4
Abnormal flexion	3
Extension	2
None	1

4.1.1.3 Pupils and their reactivity

Chapter 3 elaborates on this topic in detail. Evaluating and documenting the size of the pupils (in mm) and their reactivity to light are also essential as any changes in them gives valuable information. Normal pupils may have a 1 mm diameter difference. A bright flashlight is used to assess the light reactivity. In a normal situation, the pupil constricts to a light and so does the contralateral pupil (direct and consensual reaction). Both the right and left pupil findings must be documented.

Before pupil evaluation, hypoxemia, hypoventilation and hypotension must be treated. An important sign of uncal herniation is a unilaterally dilated and unreactive (fixed) pupil. In this situation, the ipsilateral anterior temporal lobe is pushed through the tentorium and the uncus of the temporal lobe compresses the oculomotor nerve (III cranial nerve). Damage to the medulla results in bilaterally dilated and fixed pupils. Acute pupil dilation is considered a neurological emergency.

Orbital trauma or a direct trauma to the eye may injure short ciliary nerve and pupillary sphincter muscle, respectively, also resulting in a dilated pupil. Documentation of traumas to the aforementioned areas is therefore important.

4.2 Prehospital Treatment

4.2.1. Airway, Ventilation, and Oxygenation

A TBI patient with GCS ≤ 8 must be intubated as they are not able to maintain their airway, may have low respiratory drive and they are at risk of aspiration. Short acting anaesthetic agents should be used for intubation and maintaining sedation during the transport as the neurological status is often reassessed when the patient arrives to the emergency department. Short acting opioids, propofol and short acting benzodiazepines are suitable for intubation. Ketamine has become popular in the emergency medicine due to its stable haemodynamical properties but there are currently not enough data regarding its safety in TBI patients, used as a sole anaesthetic agent. Some data shows that, when it is used in conjunction with other anaesthetics such as midazolam or propofol and patients are already mechanically ventilated, it does not increase ICP (7). In addition, hallucinations associated with ketamine administration may impair reliable neurological assessment upon admission to the hospital. However, in haemodynamically unstable patients with multiple trauma, ketamine is probably a safe choice as propfol is known to decrease blood pressure.

Prehospital intubation of TBI patients has resulted in conflicting results regarding their survival and neurological outcome. These contradictory results seem to arise from the experience and skills of the personnel. Data supports that unexperienced and untrained personnel should not intubate TBI patients (8). The correct endotracheal tube placement must be ensured by pulmonary auscultation and end-tidal CO_2 ($etCO_2$) measurement. Endotracheal tubes should be secured with drapes and the head of the bed should be elevated 15-30 degrees and neck and head positioned in neutral position to ensure cerebral venous return. Of note, semi-rigid collars may also obstruct cerebral venous return.

Normoventilation ($etCO_2$ 35- 40mmHg, 4.5-5.5 kPa) is recommended as both hypoventilation and hyperventilation results in increased mortality (9). Hypoxaemia is corrected by administering supplemental oxygen. SpO_2 is aimed to be maintained > 90%. Hyperoxia may be harmful in critically ill patients and normoxia is suggested in TBI patients, although high quality studies on the topic are still lacking.

4.2.2 Fluid Resuscitation

Isolated head trauma does not lead to hypovolaemia and hypotension. However, if the patient has concomitant high cervical injury, this may cause neurogenic shock with hypotension and bradycardia. In addition, TBI with concomitant multitrauma with hemorrhage may result in hypovolaemia and hypotension. Both result in reduced cerebral perfusion pressure and oxygen delivery, predisposing the already injured and vulnerable brain to secondary injuries. Fluid resuscitation aims to restore oxygen delivery and adequate cerebral perfusion pressure and cerebral blood flow. Isotonic or hypertonic crystalloids (10) are suitable for fluid resuscitation in the TBI patients. Hypertonic crystalloids can also be used as they decrease the ICP but they do not improve the outcome of the patients (11). The use of colloids and albumin are not recommended (12-15) as they may increase the mortality in TBI patients. Glucose containing fluids should be avoided, unless patient has hypoglycemia (B-gluck < 5 mmol/l). If fluid therapy does not increase the blood pressure, vasoactive agents should be used. Noradrenaline and phenylephrine are recommended as continuous infusion.

4.2.3 Cerebral Herniation

Frequent neurological assessment is important as the patient's clinical status can worsen rapidly due to ongoing processes such as mass lesions, cerebral oedema or hydrocephalus. Clinical signs of increased ICP and cerebral herniation are loss of consciousness or progressive neurologic deterioration (at least 2 points decrease in GCS), one or both pupils dilated and unreactive to light, Cushing's triad (hypertension and bradycardia) and extension or no reaction to painful stimulus.

Sedation and analgesia must be sufficiently provided, preferably with a continuous infusion.

Hyperosmolar therapy with either mannitol or hypertonic saline is recommended. They both increase osmotic gradient, which induces water to move from cerebral tissue to the extracellular space, thus reducing cerebral oedema. Mannitol may cause electrolyte disturbances and hypovolemia due to excessive diuresis (16). Hypertonic saline increases cardiac output, blood pressure and cerebral blood flow (16,17). Hypertonic saline may be more effective and have longer lasting property to reduce ICP than mannitol (18,19).

Hyperventilation constricts cerebral arterial vessels and reduces cerebral flow (CBF) and ICP. CBF is often reduced in the acute phase of TBI and further decrease in CBF may lead to cerebral ischemia. In a small study of TBI patients, hyperventilation decreased ICP and improved CPP but increased hypoperfused brain volume (20). Hyperventilation worsens the neurological outcome in TBI patients (21). However, short term hyperventilation can be useful in cases of imminent herniation and may outweigh its harmful effects. In a such case, hyperventilation should be titrated to $etCO_2$ of

30-35 mmHg, 3.9-4.6 kPa.
In patients with concomitant multitrauma, increased ICP can also result from increased venous pressure due to pneumothorax or abdominal injury. Pneumothorax must be treated promptly at the scene with chest drain or with thoracosentesis if drainage is not possible.

4.2.4 Seizure treatment

TBI patients are at risk of epileptic seizures, which may cause and worsen secondary injuries. They must be promptly treated. Intravenous propofol or benzodiazepines such as lorazepam or midazolam are first line choices. Of antiepileptic drugs, phosphenytoine and levetiracetam are currently the most widely used. They both seem prevent early seizures in TBI but in long term use phosphenytoine may worsen neurological outcome (22,23).

4.2.5 Hypothermia

Hypothermia has been considered as neuroprotective and several studies have assessed its role in the outcome of TBI patients. The recent POLAR study demonstrated that early prophylactic hypothermia in TBI patients does not improve neurological outcome or reduce mortality (24) and its use is not recommended.

4.3. Transport

TBI patients should be transported to Level I or Level II trauma centers with 24 hours coverage of neurosurgery, anaesthesiology, radiology (CT), traumatology and intensive care. There are no prospective randomized studies comparing "scoop and run" with "stay and play" on the outcome of TBI patients and both approaches are used in Nordic countries (25).
Severe TBI patients seem to benefit from a rapid transfer directly to a neurotrauma center and during transport all efforts must be done to prevent secondary injuries.

4.4. Paediatric TBI

Paediatric patients are not small adults and the etiology and pathology of paediatric TBI differs from that of adults. However, like in adults, hypotension and hypoxia increases mortality in paediatric TBI patients. Accordingly, assessment, treatment approaches and principals, such as avoiding hypoxemia and hypotension, are applied in the same way as in adults.

Paediatric patients should be transferred to Level I or II trauma centers familiar with paediatric TBI patients or to trauma centers familiar with paediatric TBI patients.

4.4.1 Oxygenation and blood pressure

Paediatric SpO_2 target is also > 90% whereas blood pressure targets are age dependent. In the paediatric population, hypotension is defined according to American College of Surgeons as follows:
Age 0-28 days, SBP < 60 mmHg, 1-12 months, SBP < 70 mmHg, 1-10 years, SBP < 70+2 x age in years, > 10 years, SBP < 90 mmHg. Current paediatric TBI guidelines from 2019 state that in children minimal CPP is 40 mmHg and threshold 40-50 mmHg should be considered, with infants at the lower end and adolescents at the upper end (26).
However, it may be that these hypotension and CPP thresholds in paediatric TBI patients are too low (27) and they should be more age specific (28) but data is currently too limited.

4.4.2. Paediatric GCS

Just like in adults, in paediatric patients GCS is assessed after stabilization. In children over 2 years old, adult GCS can be employed whereas the pediatric GCS is used in pre-verbal children.

Table 2. Paediatric GCS

PGCS	
Eye opening	
Spontaneous	4
Speech	3
Pain	2
None	1
Verbal response	
Coos, babbles	5
Irritable cries	4
Cries to pain	3
Moans to pain	2
None	1
Motor response	
Normal spontaneous movement	6
Withdraws to touch	5
Withdraws to pain	4
Abnormal flexion	3
Extension	2
None	1

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