



Head Injury

Version No: 3.4

Effective date: 13/11/2023

APPROVALS

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HISTORY

Effective Date	Version No.	Summary of Amendment
Nov 2012	3.0	Reformatted
Mar 2015	3.1	reviewed
Feb 2016	3.2	Reviewed
March 2021	3.3	Major review to incorporate CRASH-3 trial and 2019 AAGBI guidance Removal of adult GCS from appendix A Change of childrens GCS to JRCALC version in appendix A
November 2023	3.4	Minor review to incorporate NICE head injury guideline 2023

REFERENCES

Document Reference Number	Document Title
Appendix A	Glasgow Coma Score – under 4 years old
Appendix B	Head Injury Advice Sheet



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1. Purpose

Traumatic brain injury (TBI) is a major cause of mortality and morbidity. In England and Wales, approximately 1.4 million patients per year attend hospital following head injury and it is the most common cause of death under the age of 40 years. The commonest mechanisms of injury are falls in the elderly and road traffic collisions in the young (1).

HEMS crews are often selectively tasked to or requested to assist crews in managing these patients, particularly to ensure a safe transfer to a major trauma centre.

This CSOP provides guidance on the assessment and management of head injured patients, ranging from those with critical injuries, to those who might be suitable for discharge from scene.

2. Definitions/Acronyms:

Abbreviations	Definitions
GCS	Glasgow Coma Scale
ECG	Electrocardiogram
RSI	Rapid Sequence Induction
PHEA	Pre hospital emergency anaesthesia
IV	Intravenous
ET	Endotracheal
NIBP	Non-Invasive Blood Pressure
CT	Computerised Tomography
ICP	Intra Cranial Pressure
PEEP	Positive-End Expiratory Pressure
SBP	Systolic Blood Pressure
MAP	Mean arterial pressure
CPP	Cerebral perfusion pressure = MAP - ICP

3. Introduction

The principles of head injury management are to prevent secondary neurological injury through the provision of adequate oxygenation, adequate cerebral perfusion and the treatment of any other significant injuries, with rapid transfer to a neurosurgical centre. Even head injured patients who do not require surgery have better outcomes when managed in neurosciences centres.



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Attention should be given to the following markers, which can cause secondary brain injury:

- Hypoxaemia
- Hypo- and hypercarbia
- Hypotension
- Raised intracranial pressure
- Hypo- and hyperglycaemia
- Hyperpyrexia

Furthermore, the results of the CRASH-3 trial of tranexamic acid in traumatic brain injury suggest benefit in certain groups of patients, which will be dealt with below.

An important prognostic factor of traumatic brain injury is the GCS. The presenting GCS must be recorded accurately as it is a useful indicator of severity used in on-going hospital care. Changes over time are more useful than a single assessment. Even patients with small reductions in GCS have a significant incidence of morbidity following their injury. A cohort study from a UK HEMS service showed that in patients with a GCS of 13-15 who were RSI'd, were significantly more likely to have both an abnormal CT head ($p=0.01$) and a clinically important brain injury ($p=0.03$) (2)

4. Pre-hospital assessment of the patient with a head injury

Patients meeting any of the criteria below should be assessed in hospital for their head injury (NICE 2023) (3):

- a [Glasgow Coma Scale](#) (GCS) score of less than 15 on initial assessment
- any loss of consciousness because of the injury
- any [focal neurological deficit](#) since the injury
- any suspicion of a [complex skull fracture or penetrating head injury](#) since the injury
- amnesia for events before or after the injury (it will not be possible to assess amnesia in children who are preverbal and is unlikely to be possible in children under 5)
- a persistent headache since the injury
- any vomiting episodes since the injury (use clinical judgement about the cause of vomiting in children 12 years or under and the need for referral)
- any seizure since the injury
- any previous brain surgery
- a [high-energy head injury](#)



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- any history of bleeding or clotting disorders
- current anticoagulant or antiplatelet (except aspirin monotherapy) treatment
- current drug or alcohol intoxication
- any safeguarding concerns (for example, possible non-accidental injury or a vulnerable person is affected)
- continuing concern by the professional about the diagnosis.

Other key elements of pre-hospital examination and investigations:

- Respiratory rate
- SpO₂
- ECG
- NIBP
- GCS – communicated as E V M
- Pupils
- Blood sugar
- Consideration of co-existing neck injury, although the TARN database has not demonstrated a link between the two
- Limb movements – essential to record prior to PHEA

5. General management principles in head injured patients

Airway

Meticulous attention should be paid to maintaining an unobstructed airway and administering oxygen early.

Nasopharyngeal (NP) and oropharyngeal (OP) airways may be used in combination to maintain a patent airway and allow oxygenation. Securing a patent airway takes precedence over any theoretical concerns of intracranial placement of an NP airway with suspected basal skull fracture, but insert these gently with lubrication to avoid soft tissue trauma and risks of intracranial placement.



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Breathing

Assess and treat any chest injury.

High flow oxygen should be administered early. This can be titrated down to maintain an SPO₂ >95%.

Circulation

After TBI the brain may lose the ability to autoregulate cerebral blood flow and therefore a fall in blood pressure may result in a reduction in cerebral perfusion pressure even if the ICP is normal. There is evidence that depth and duration of out-of-hospital hypotension are associated with increased traumatic brain injury mortality (4)

AAGBI recommendations (5) are to maintain a systolic BP of >110mmHg and a mean arterial blood pressure (MAP) of 90 mmHg. However, there is evidence to suggest that there is a linear relationship between systolic blood pressure and mortality with no inflection point around the 90 systolic mark, hence it may be wrong to over emphasise this as a 'cut off'. It may also be the case that clinically meaningful hypotension may not be as low as current guidelines suggest (6)

In a patient with an apparently isolated head injury, liberal volume resuscitation and vasopressors can be used to optimise cerebral perfusion pressure.

Adopt a standard approach to the polytrauma patient to optimise blood pressure. For patients who have haemorrhagic shock and a traumatic brain injury (NICE 2016) (7):

- if haemorrhagic shock is the dominant condition, continue restrictive volume resuscitation or
- if traumatic brain injury is the dominant condition, use a less restrictive volume resuscitation approach to maintain cerebral perfusion.

Tranexamic acid (TXA)

NICE guidance recommends that for patients with a GCS score of 12 or less who are not thought to have active extracranial bleeding, consider:

- a 2 g intravenous bolus injection of tranexamic acid for people 16 and over
- a 15 mg/kg to 30 mg/kg (up to a maximum of 2 g) intravenous bolus injection of tranexamic acid for people under 16.



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- Give the tranexamic acid as soon as possible within 2 hours of the injury, in the pre-hospital or hospital setting and before imaging.

The CRASH-3 study results (8) can also be interpreted as follows:

- TXA is most effective in patients with mild and moderate head injury. In severe head injury there was no clear evidence of benefit but it is recommended that TXA should still be administered as the potential benefits outweigh the risks and there was no evidence of harm in this group provided TXA was administered within 3 hours.
- Give TXA as soon as possible after injury - early treatment was more effective in patients with mild and moderate head injury, but there was no impact of time to treatment in severe head injury. TXA should be administered as soon as possible after head injury and ideally within one hour.
- TXA is safe in TBI patients - the risk of vascular occlusive events and other complications were similar in the TXA and placebo groups.

Disability

Assess pupils (size and reactivity) and limb movements on initial assessment, prior to and after PHEA.

Check a blood glucose in every head injured patient and if <4 mmol/L cautiously treat using 10% dextrose, closely monitoring the blood glucose as hyperglycaemia is also harmful to the injured brain. This is particularly important in children.

Treat agitation by sedation and PHEA (see below). Agitation and restraint will raise the ICP and the cerebral oxygen demand.

Do not withhold analgesia even if the patient is unable to communicate their pain to you – use titrated morphine, fentanyl or ketamine IV.



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Treat seizures with IV benzodiazepines if they are not self-terminating. A patient who is actively fitting after head injury or is deemed to be in status epilepticus post head injury should have a PHEA.

Signs of rising ICP and actual or impending herniation include:

- GCS <8
- plus unilateral or bilateral pupil dilation, unresponsive to light
- and/or progressive hypertension and bradycardia

These patients should be intubated and ventilated to a target ETCO_2 4.0-4.5 kPa, adequate sedation and paralysis ensured and consideration given to hypertonic saline.

Exposure and packaging

Compression of the jugular veins will reduce venous return from the head and increase ICP. As per TAAS CSOP 009 a cervical collar should not be used to immobilise the cervical spine, but immobilisation should be maintained by head blocks and tape. Endotracheal tube ties can also constrict the neck veins and should be checked and loosened before transport. The preferred approach is to use a commercial ET tube holder (Thomas).

Where possible, the patient should be transported in a 30 degree head up position to facilitate venous drainage. This can be achieved through raising the head end of the scoop on a few blankets.

Extreme G-Forces (braking, acceleration and high-G turns) should be avoided.

Core body temperature should be maintained. Anaesthetised patients should be wrapped in a Blizzard Blanket as per CSOP 012.

Pre Hospital Emergency Anaesthesia for head injuries

TAAS CSOP 13 should be followed for all PHEAs.



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The indications for PHEA for head injured patients include (5)

- GCS ≤ 8
- Significantly deteriorating conscious level (e.g. a fall in GCS of two points or more, or a fall in motor score of one point or more)
- Loss of protective laryngeal reflexes
- Failure to achieve PaO₂ ≥ 13 kPa; (aim for peripheral oxygen saturation $\geq 95\%$)
- Hypercarbia (PaCO₂ > 6 kPa)*
- Spontaneous hyperventilation (PaCO₂ < 4.0 kPa)*
- Bilateral fractured mandible
- Copious bleeding into the mouth (e.g. from skull base fracture)
- Seizures

*remember that ETCO₂ poorly approximates to PaCO₂. The PaCO₂ target is 4.5-5.0kPa.

Other recognised relative indications include:

- Facilitation of safe patient transfer
- Agitation
- Anticipated clinical course

Drugs

Choice of induction agents should be in line with the TAAS PHEA SOP.

Adequate sedation and paralysis is important to minimise further rises in ICP during transport. Aim for a systolic BP of less than 200 mmHg but avoid excessive falls in MAP from baseline (pre induction) which may adversely affect CPP in the injured brain.

Improving outcomes with pre hospital management

During PHEA of the head injured patient the following '**H bombs**' should be avoided to improve outcome (9):

- Hypoxia
- Hypotension



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- Hyperventilation

In practice, this means:

- Pre-oxygenating the patient adequately which may require analgesia or sedation, and aborting intubation attempts if the patient is desaturating.
- NIBP should be recorded every minute in the peri-induction period (5)
- Small titrated doses of a vasopressor may be used to achieve an adequate MAP and hence CPP in patients with isolated TBI.
- Ensure the person ventilating can see EtCO₂ monitoring and adjusts rate accordingly
- Transfer to ventilator early for consistent ventilation
- EtCO₂ should be maintained at 4.5 – 5.0 kPa (35 – 40 mmHg) in every ventilated patient unless there is unequivocal evidence of raised intracranial pressure or coning in which case hyperventilation to an ETCO₂ of 4.0-4.5 is indicated. This minimises the risk of cerebral vasoconstriction at low PaCO₂ or cerebral vasodilation at a high PaCO₂.

PEEP

AAGBI (5) recommend a PEEP of 5cmH₂O to prevent atelectasis and state that up to 10cmH₂O of PEEP will not adversely affect cerebral perfusion, although in practice an initial PEEP of 10cmH₂O is seldom required. As such PEEP should be considered on a case by case basis.

Management of hypertension

This should be managed pre hospital by increasing the sedative agent being used post PHEA. Particular consideration should be given as to whether the hypertension is a sign of awareness.



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Children

The TAAS PHEA SOP should be followed with regards to children aged 8 years or under, who should not be anaesthetised unless the TAAS Doctor is an NHS anaesthetist with paediatric expertise or the clinical condition of the child is such that paediatric PHEA is the only option.

According to AAGBI (5) there may be a role for hypertonic saline before PHEA to help avoid the predictable increase in ICP resulting from apnoea induced hypercapnia at laryngoscopy.

Children are prone to a hypotensive response to acceleration and ICP spikes on deceleration and this should be considered if a land ambulance transfer is being undertaken.

The combative patient

Analgesia and sedation will be required to allow assessment for other injuries, monitoring, oxygenation and safe transfer. The practitioner should be aware that after a certain amount of sedation it is likely the patient will require PHEA due to inadequate airway control and hypoventilation.

The choice of agent used for sedation depends on the rest of the patient's injury pattern and physiology. In the haemodynamically normal patient with an isolated head injury, midazolam is the preferred option; in the shocked patient with multiple injuries, ketamine is more likely to preserve the blood pressure and would therefore be a preferable agent.

In either case, the sedation must be given in small doses titrated to effect.

Hypertonic saline

Indications:

TBI with signs and symptoms suggestive of raised ICP or focal lesion that continue post PHEA and ventilation to normocapnoea, signs would be:

- Pre RSI GCS<8 plus
 - SBP>160



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- Or pulse <60
- Or Uni or bilateral pupil dilatation

Dose and Administration:

5ml/kg 2.7% hypertonic saline to a maximum volume of 350ml, once only. The dose should be administered using a 50 ml syringe and three way tap in the circuit to avoid accidental overdose. The hospital handover must specifically include full details of the hypertonic saline administration.

6. Triage

Transfer all significant head injured patients to a Major Trauma Centre according to the Major Trauma Triage Tool in applicable to the geographical area where the TAAS crew is working.

7. Minor Head Injuries

A head injured patient MUST be referred to hospital if any of the criteria in section 4 above are met.

It may be appropriate to discharge some patients from scene. If doing so, ensure you have fully assessed and cleared the cervical spine and there are no other significant injuries requiring assessment at hospital. The patient should be given clear verbal instructions of the symptoms and signs to look out for and to attend their local emergency department if there are any problems. The patient should avoid alcohol, get plenty of sleep and should be supervised by a responsible adult for the first 24 hours. Simple analgesia e.g. Paracetamol or Ibuprofen can be used for headache. If TAAS staff are discharging a patient from the scene the patient must be given a written head injury advice sheet. Copies of these are in appendix B and they are sourced from NICE with minor adaptations to TAAS work. Verbal advice should include advice on driving a car or motorbike and this should be given on a case by case basis.

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