INTRODUCTION

Head injury is estimated to be the cause of 1,000,000 hospital presentations each year in the UK, with an incidence of severe brain injury of between 10 and 15 per 100,000 population.[1] It may be an isolated injury or be part of multi-system traumatic injury. There is a significant association with cervical spinal injury in those with a depressed level of consciousness.[2]

Little can be done for primary brain injury, i.e. damage that occurs to the brain at the time of injury. Injury prevention strategies such as the wearing of motorcycle helmets or use of vehicle restraint systems (e.g. seatbelts and airbags) are the only viable means of reducing these injuries.[3,4]

Secondary brain injury is that which occurs following the primary event as a result of hypoxia, hypercarbia and hypoperfusion. The reduced level of consciousness may lead to airway obstruction or inadequate ventilation with consequent decreased oxygenation and increased levels of carbon dioxide and a metabolic acidosis. Blood loss from other sources in a multi-system trauma may lead to hypovolaemia and a fall in the cerebral perfusion pressure.

NEVER presume decreased conscious level is solely due to alcohol. Intoxicated patients commonly sustain head injuries as well.

HISTORY

Mechanism of injury

In a person with altered level of consciousness, at risk of intracranial head injury, an appreciation of the forces that were involved in causing the injury is helpful. With scene indicators such as a "bulls-eye" of the windscreen or blood staining of the dashboard or steering wheel in a motor vehicle collision, or significant scratch or fracture damage to a protective helmet, there should be suspicion of significant injury. The identification of a weapon that might have been used in an assault, or blood staining on objects immediately adjacent to the casualty with a bleeding head wound following a fall may be extremely helpful.

A history of a period of loss of consciousness raises the risk of significant injury.[5] The duration and depth of unconsciousness are of great value, as are changes over time. For example, a period of lucidity followed by decreasing consciousness would suggest the development of an extra-dural haematoma.

Amnesia is difficult to quantify. National Institute for Clinical Excellence guidelines state that retrograde amnesia (amnesia for the events before injury) of greater than 30 minutes indicates major injury. Post-traumatic amnesia is less predictive, but also suggests significant injury.

The association of head injury with cervical spine trauma is significant and movement of the limbs should be noted, together with other symptoms suggesting spinal cord injury (***refer to neck and back guideline***).

In the presence of a decreased level of consciousness, other medical causes should be sought. A history of epilepsy might suggest a convulsion or a history of diabetes might suggest hypoglycaemia as a cause for the reduced level of consciousness.

Consideration should also be given to the role of alcohol or recreational and other drugs as a cause.

ASSESSMENT

Assessment of the neurological state takes place after the ABCs have been adequately addressed.

A patient with a depressed level of consciousness is less capable of protecting their airway. Loss of a gag reflex increases the risk of aspiration. The airway should be cleared and maintained, with an airway adjunct if necessary. Manual in-line immobilisation of the cervical spine, with due regard to potential injury during airway manoeuvres must be maintained.

Assessment of the adequacy of breathing, particularly the respiratory rate and the depth of breathing is needed. A respiratory rate of between 10 and 30 breaths per minute with visible and palpable good chest excursion is ideal. Formal measurement of breathing is impractical in the pre-hospital environment during the assessment and resuscitation phase.

Maintenance of the circulation has relevance for maintaining cerebral perfusion. External haemorrhage control and fluid replacement to raise and maintain the blood pressure are important.

A simple **AVPU** score, together with pupillary size and reactivity and the noting of spontaneous movements in particular limbs is sufficient for the primary survey neurological assessment. A formal **Glasgow Coma Scale**[6] **(GCS)** assessment can be done but needs to be thoroughly reliable and reproducible. If done hurriedly or incompletely, it can produce a misleading clinical picture and result in inappropriate on-going care following arrival at the Emergency Department (ED).

Other clear indicators of head injury such as a boggy swelling or laceration of the scalp should be noted and reported, together with the leakage of cerebrospinal fluid (CSF) from the ears and/or nose or blood from the ears. Brain matter coming from a wound should be covered with a light dressing. A finger should not be inserted to feel for a fracture or check the origin of injury.

MANAGEMENT

The aim of pre-hospital treatment is to deliver adequate oxygen to the brain by:

- optimising oxygenation of the blood
- maintenance of the cerebral perfusion pressure.

Administer high concentration oxygen via a non-rebreathing mask, using the stoma in laryngectomee and other neck breathing patients to ensure an oxygen saturation (SpO_2) of >95%, except for patients with chronic obstructive pulmonary disease (COPD) (***refer to COPD guideline***).

Cervical spinal injury should be assumed to be present and managed appropriately (***refer to neck and back trauma guideline***). The patient needs to be delivered to the most appropriate facility for management of the injury. Ideally this would be a centre with neurosurgery, but in many cases a rapidly accessible ED capable of supplementing the pre-hospital management and identifying the extent of injury before promptly referring on is optimal.

Management of problems identified by the primary survey should occur as they are identified and are considered in more detail.

Airway management with cervical immobilisation

Hypoxia is the major danger for head injury care and there is clear evidence that it worsens the prognosis[7]. An obstructed airway may occur through loss of muscle tone of the oropharyngeal structures and physical obstruction by the tongue falling backwards or by the accumulation of secretions or blood in the pharynx.

The airway should be inspected for foreign bodies, being aware that with the patient lying supine, any object is likely to have fallen to the back of the mouth. Simple airway manoeuvres to draw the tongue forward should be applied, but because of the need to protect the cervical spine, only jaw thrust and chin lift are acceptable. Head tilting should be a last resort for the airway that cannot be adequately opened and the risk/benefit balance should be considered.

Suction should be used to clear any fluid obstruction but only under direct vision. Stimulation of the pharynx by the suction catheter can raise the intracranial pressure (ICP).

In a patient with some tongue obstruction of the airway, an oropharyngeal airway should be considered. If the patient retains a gag reflex or there is retching then vomiting may be induced and the airway should be removed. Any of these physical responses to insertion will raise the ICP.

If the patient will not tolerate an oral airway, or in some head injured patients where there is jaw clenching, or trismus, it may be necessary to use a nasopharyngeal airway. If the airway is obstructed and the jaw is clenched then the risks of inserting a nasopharyngeal airway are less than the theoretical risk of the airway passing through a basal skull fracture. Unless carefully inserted, this device may exacerbate an airway problem by inducing an epistaxis. The airway should be inserted across the floor of the nose below the inferior turbinate and, unless this is clearly identified by lifting the nostril and advancing posteriorly, there is potential for misplacement and a risk that the airway will cross the cribriform plate and enter the brain. This is particularly a risk if there are facial fractures.

Endotracheal intubation will secure an airway against the risk of aspiration and allow optimal ventilation. Insertion, however, is technically more difficult than simple airway insertion and requires the patient to have lost oropharyngeal reflexes.

If a patient's conscious level is sufficiently depressed that an endotracheal tube can be inserted without the use of sedating and paralysing drugs then the outcome is bleak. UK Paramedics are currently not authorised to administer anaesthetic drugs. It may be possible to intubate if the patient has taken a large amount of alcohol or illegal drugs before injury.

Any instrumentation of the upper airway will raise the ICP and exacerbate the secondary brain injury.[8] The use of force to overcome the resistance is likely to be deleterious to the patient outcome and should not be used. A balance is required between the clear benefits of airway manoeuvres and the danger of raising ICP and increasing the level of injury. An airway escalator is illustrated in **Figure 1**.

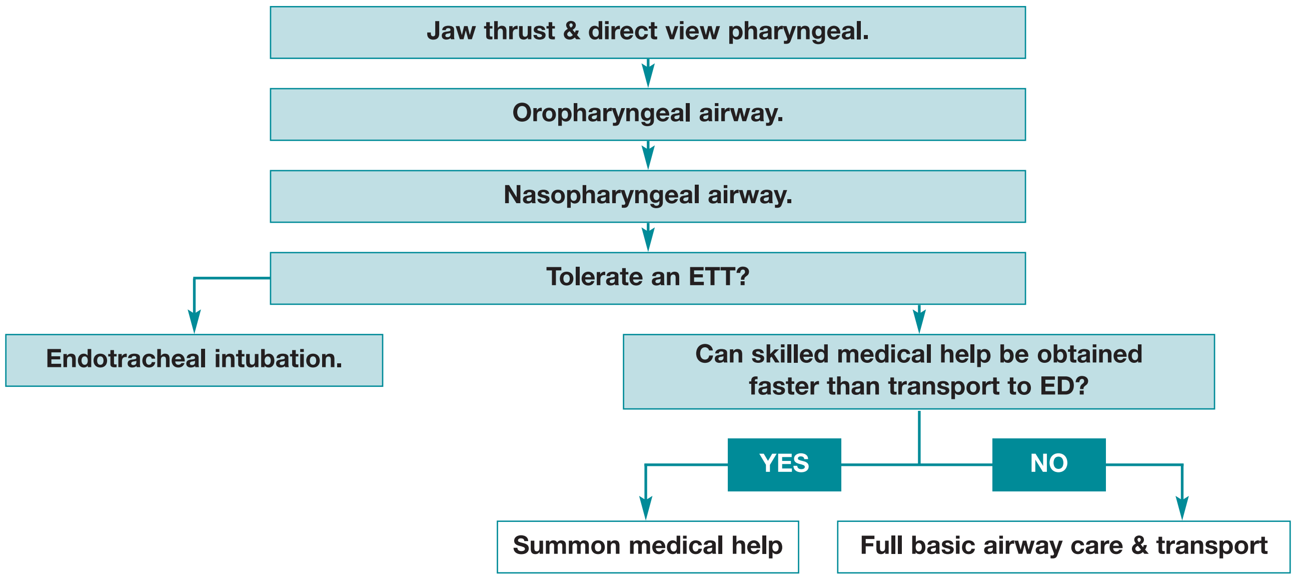

Figure 1- Management of an obstructed airway in head injury.

Drug assisted intubation is possible in the pre-hospital environment but the research evidence for Paramedics having this skill is currently lacking. A mobile medical team resource may be able to provide this option, however the delay in their arrival on scene and the delays that will then follow while the airway is secured need to be balanced against the benefit of definitive airway control over optimal airway control with simple manoeuvres and rapid transfer.

There are a number of intermediate "rescue" airways available such as the laryngeal mask airway (LMA) and combitube. The insertion of these large devices into the pharynx will raise ICP and they do not secure the airway. While they may allow an airway to be obtained, the case for their use in UK trauma practice is currently not established.

Surgical airways, needle cricothyroidotomy and surgical cricothyroidotomy, are possible in the field but have significant complication rates and are profoundly stimulating to the patient and may increase the ICP.

There is a clear association between head injury and concurrent cervical spine injury. In the presence of an altered level of consciousness or while under the influence of alcohol, a reliable clinical assessment of the cervical spine cannot be made. Cervical spine immobilisation is mandatory. There is some evidence that a firmly fitting cervical collar may raise the ICP [9,10] but immobilisation is required to prevent exacerbation of any spinal cord injury. It is being suggested that a collar should be used during extrication but that once the patient is fully immobilised on a long board with blocks and tape to the head the collar could be loosened. This could not yet be considered the standard of care in the pre-hospital field (***refer to neck and back trauma protocol***).

Breathing

The importance of treating hypoxia and preventing hypercarbia has been described.

In the presence of a depressed level of consciousness, there may be inadequate respiratory effort.

- consider assisted ventilation at a rate of 12–20 respirations per minute if:
  - oxygen saturation (SpO_2) is <90% on high concentration O_2
  - respiratory rate is <10 or >30
  - expansion is inadequate.

A combative patient is unlikely to have inadequate ventilation but could certainly be hypoxic. The less conscious patient group may require support both in terms of rate and chest excursion.

Circulation

Cerebral perfusion pressure (CPP) needs to be maintained for optimal patient benefit. This is determined by the balance between mean arterial pressure (MAP) (mathematically the mean pressure during the cardiac pumping cycle) pushing blood into the brain and the ICP resisting this.

CPP = MAP – ICP

$$\text{MAP} = \text{diastolic pressure} + \frac{(\text{systolic pressure} - \text{diastolic pressure})}{3}$$

The ICP is increased by the presence of anything that occupies space (haematoma, oedema/swelling) or causes vasodilatation (hypoxia, hypercarbia).

In UK practice, it is currently accepted that best care is achieved by maintaining an MAP of >90 mmHg and a systolic of the order of 120mmHg. Pressures below this are related to a poorer neurological outcome. That hypotension worsens outcome is clearly established, but the true minimum pressure is not fully defined.[11,12]

Hypotension is unlikely to be caused by isolated head injury and so would usually indicate the presence of other significant injury causing blood loss.

Fluid Therapy

Obtain IV access.

Current research shows little evidence to support the routine use of IV fluids in adult trauma patients. In circumstances such as penetrating chest and abdominal trauma, survival worsens with the routine use of IV fluids.[13]

Fluids may raise the blood pressure, cool the blood and dilute clotting factors, worsening haemorrhage. Therefore, current thinking is that fluids should only be given when major organ perfusion is impaired.

If there is visible external blood loss greater than 500mls, fluid replacement should be commenced with a 250ml bolus of crystalloid.

Central pulse **ABSENT**, radial pulse **ABSENT** is an absolute indication for urgent fluid. If the patient has a carotid pulse but no radial pulse then other clinical factors should also be considered before decision on fluid administration.

Central pulse **PRESENT**, radial pulse **ABSENT** is a relative indication for urgent fluid depending on other indications including tissue perfusion and blood loss.

Central pulse **PRESENT**, radial pulse **PRESENT DO NOT** commence fluid replacement,[14] (unless there are other signs of poor central tissue perfusion (e.g. altered mental state, cardiac rhythm disturbance).

DO NOT delay at scene for fluid replacement; wherever possible cannulate and give fluid **EN-ROUTE TO HOSPITAL.**

The use of mannitol to reduce brain swelling and size can be used in the presence of focal neurological signs to prevent herniation but is currently limited to use under medical direction. Hypertonic saline is also being investigated, but is not currently usual standard of care in the UK.

Disability

A rapid and mini-neurological assessment is required at the point of first assessment to give a baseline against which improvement or deterioration can be measured. It should consist of:

Conscious level

This is a good indicator of severity of injury and progression over time. The AVPU (alert, voice responsive, pain responsive, unresponsive) scale is quick and reproducible. It is generally acceptable during the early stages of resuscitation.

AVPU scale

A	Alert
V	Voice responsive
P	Pain responsive
U	Unresponsive

The GCS is more sensitive to change but requires careful formal assessment; it should not be approximated. The headline figure falls between 3 and 15 but should be broken down into its three components.

Table 1 – Glasgow Coma Scale[6]

GLASGOW COMA SCALE	
Item	**Score**
Eyes Opening:	
Spontaneously	4
To speech	3
To pain	2
None	1
Motor Response:	
Obeys commands	6
Localises pain	5
Withdraws from pain	4
Abnormal Flexion	3
Extensor response	2
No response to pain	1
Verbal Response:	
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
NO verbal response	1

There are a number of pitfalls in the assessment:

- in the absence of eyelid muscle tone in a deeply unconscious person, the eyes may be open. If there is no response to stimulation, then this is recorded as **"none".**
- if there is severe facial swelling that would prevent eye opening, then this is recorded as such.
- verbal response in small children does not fit with the scale and so a modification is applied:

5	appropriate words, social smile, fixes and follows with eyes
4	cries but consolable
3	persistently irritable
2	restless and agitated
1	none

- deaf patients or those who cannot give a verbal response, such as those with a tracheostomy are recorded as found, but a caveat is included in the assessment.
- during motor assessment, if there is a difference between the two sides of the body, then the better response is recorded.

Pupil response

These should be round and equal in size. They should respond promptly by constriction when a light is shone into them. When light is shone in one, the other pupil should respond appropriately to this too. Any abnormalities should be noted.

Causes for abnormality include local trauma and loss of sight through other eye disease. Many more elderly patients may be taking medication to dilate or constrict the pupil and these can have a long duration of action. Even opiate analgesia may be the cause.

The cause cannot be assumed in the presence of head injury and it may be an indicator of significant brain injury/swelling.

Convulsions

A fit can occur immediately after the blow to the head and have little prognostic significance but may also occur a little later with major significance, indicating significant intracranial pathology. Post-traumatic epilepsy can develop, but could certainly not be determined in the early stages of care.

Management is the same as for any convulsion. Protection from further harm, protection of the airway and oxygenation are the key interventions.

Assess blood glucose en-route to hospital

Secondary Survey

A thorough assessment of an injured patient requires full exposure and a careful stepwise assessment of all areas, both front and back. This is not appropriate in the pre-hospital environment, but it is reasonable to note any other injuries that have been found or are suspected during the pre-hospital phase.

It is important to gather any scene information about the mechanism and potential confounding factors, since the ED will be "blind" to these. Witness accounts are very valuable. The time course of the patient following injury is very valuable; has there been any consciousness, has the patient walked, or even moved limbs, since the injury?

Information on patient identity or clues as to the next of kin will also be invaluable.

Transfer to further care

Victims of significant head injury require optimal oxygenation and tissue perfusion as quickly as possible and then evaluation and appropriate management of any lesion within the cranial vault and optimal management of the pressures, both blood and intracranial.

Early optimisation is probably best achieved by transporting to the nearest ED capable of securing a definitive airway and performing this assessment. More extensive monitoring and evaluation requires a neurosurgical facility at the earliest opportunity.

There is no doubt that early evacuation of an intracranial haematoma is in the patient's best interests and has a significantly improved outcome. Systems need to design a patient care process that ensures that the patient gets to definitive care as soon as possible i.e. directly to an ED with neurosurgery on site, but balances this with the hazard of delay in optimisation through longer pre-hospital transport times.

The process should be predetermined rather than "done on the fly". The solution may be to take to the nearest ED and do a secondary transfer, or bypass facilities and travel directly to the neurosurgical centre. This will vary with the geography and the capacity of the system.

Key Points – Head Trauma

- **NEVER** presume decreased conscious level is solely due to alcohol; intoxicated patients commonly sustain head injuries as well.
- Ascertain duration and depth of unconsciousness, as a period of unconsciousness raises the risk of significant injury.
- Unconscious patients are less capable of protecting their airway; obstruction may occur through loss of muscle tone, physical obstruction, the tongue falling backwards, or by the accumulation of secretions or blood in the pharynx.
- Simple airway manoeuvres to draw the tongue forward should be applied, but because of the need to protect the cervical spine, only jaw thrust and chin lift are acceptable.
- The aim of pre-hospital treatment is to deliver adequate oxygen to the brain.

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METHODOLOGY

Refer to methodology section.