Purpose

This procedure outlines the principles of management of patients suffering prehospital TBI.

Procedure

Pre-Hospital Traumatic Brain Injury

For Review

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1. Overview

Traumatic brain injury (TBI) is a leading cause of morbidity and mortality in civilian trauma victims[1]. GSA-HEMS regularly attends patients with TBI (often with other associated major traumatic injuries). Transport directly to a Major Trauma Centre whilst focussing on the prevention of secondary injury and further physiological insults are the keys to maximising the chances of a good neurologic outcome in these patients.

2.2 Airway and Ventilation

2.2.1 Patients suffering suspected TBI need rapid assessment and management of potentially compromised airway and ventilation, which may be compounded by agitation, concomitant facial injury, and the need for cervical spine immobilisation.

2.2.2 The initial priority is to establish a patent airway and ensure oxygenation using basic manoeuvres and airway adjuncts.

2.2.3 A low threshold should be maintained for emergency pre-hospital anaesthesia of patients suffering TBI to prevent secondary brain injury.

2.2.4. Ketamine is the preferred induction agent for patients with TBI as it has a safer haemodynamic profile and can be used as an effective pre-anaesthetic and post-sedation agent.[4] Refer to GSA-HEMS RSI Manual.

2.2.5 Prior to intubation a focussed examination should be performed with documentation of GCS and its constituent parts, pupillary size and reactivity and the presence of signs of spinal cord injury.

2.2.6 Routine hyperventilation should be avoided as it has been shown to be associated with increased mortality in TBI patients intubated in the field.[1,2]

2.2.7 An intubated patient with TBI should be placed on a ventilator as soon as practicable rather than hand bagged, as this allows for greater control of ventilation parameters and avoids breath-to-breath variation in ventilation and PaCO2.

2.3 Endpoints of Resuscitation

2.3.1 Early neurosurgical intervention may be necessary in a small number of patients, particularly those who “talk and then deteriorate” and scene time should be strictly minimised for such patients. A much larger proportion of patients will need meticulous critical care focused on maintenance of normal physiological parameters and avoidance of hypoxia, hyper- or hypo-carbia, and aggravation of elevated intracranial pressure (ICP).

2.3.2 Mortality after severe TBI has been shown to be highly associated with episodes of hypoxia and/or hypotension.

2.3.3 During resuscitation the following physiologic parameters should be maintained :

- Normoxia oxygen saturation: Sa02 90-98%[1]
- Normotension: SBP>90mmHg[1]
- Normocapnoea: ETCO2 30-35mmHg [1,2]

2.3.4 Remember that PaCO2 will be higher than ETCO2 and this discrepancy may be higher in patients with chest injuries or hypovolaemia.[3]

2.4 Minimising Raised ICP

2.4.1 As cerebral perfusion pressure (CPP) is the difference between mean arterial pressure (MAP) and ICP, measures to minimise the latter are reasonable therapeutic strategies in all patients with TBI[1].

- Adequate sedation with opioid (morphine or fentanyl) and sedative (midazolam or ketamine) followed by neuromuscular relaxation once well sedated.
- Minimising excessive PEEP settings (<=5cm H2O).
- Tracheal tubes secured without impeding jugular venous return.
- Patient transported head up 30 deg where possible (whilst maintaining spinal precautions where indicated).

2.4.2 In the pre-hospital environment we must rely on clinical signs of raised ICP which may be unreliable. These include:

- deteriorating GCS
- progressive focal deficits
- anisocoria - dilated, unresponsive pupil(s)
- Cushing's Response (hypertension and bradycardia)
- pre-terminal cardiovascular collapse

2.4.3 In the event of suspected raised ICP, then further strategies to temporarily reduce ICP may be considered.

- Hypertonic Saline: Patients with anisocoria, or deteriorating GCS with suspected raised ICP should be administered hypertonic saline (7.5%) at a dose of 5ml/kg (max. 250ml)[5] If there is no improvement a brief period of mild hyperventilation to an ETCO2 20-25mmHg (PaCO2 25-30mmHg) may be considered.
- Pre-hospital Activation of Operating Theatres: direct communication with the receiving trauma centre to activate neurosurgical team prior to arrival.

3. Documentation

In addition to routine observations including pre-RSI GCS and bilateral limb movements, pupillary responses should be documented regularly for all patients.

4. References

1. Brain Trauma Foundation 2007: Guidelines for the Management of Severe traumatic Brain Injury (3rd ed) Journal of Neurotrauma, vol 24, supp 1.

2. Coles P et al Hyperventilation following head injury: Effect on ischaemic burden and cerebral oxidative metabolism. Crit Care Med 2007 Vol35(2) 568-78

3. Warner K et al The Utility of Early End-Tidal Capnography in Monitoring Ventilation Status after Severe Injury. J Trauma. 2009 Jan;66(1):26-31

4. Morris, C 2009:Anaesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent? Anaesthesia. 64(5):532-9

5. Bulger, EM et al 2010: Out-of-Hospital Hypertonic Resuscitation Following Severe Traumatic Brain Injury. JAMA 2010;304(13):1455-1464.