



Purpose

This procedure outlines the management principles of patients being retrieved with traumatic brain injury (TBI), spontaneous intracranial haemorrhage (including subarachnoid haemorrhage) and patients undergoing management for raised intracranial pressure.

Procedure

Neuroprotection

For Review

Aug 2015

1. Overview

The interfacility transfer of patients with intracranial insults, both traumatic and spontaneous, constitutes a significant component of GSA-HEMS' case load. Prevention of secondary injury and further physiological insults are the keys to maximising the chances of a good neurologic outcome in these patients.

2. Aims

2.2 To outline the management principles of patients being retrieved with traumatic brain injury (TBI), spontaneous intracranial haemorrhage (including subarachnoid haemorrhage) and patients undergoing management for raised intracranial pressure.

3. Interhospital Retrieval of Neurocritical Care Patients

3.1. Common reasons for retrieval of patients with TBI or spontaneous intracranial events include:

- traumatic intracranial bleeding for ongoing critical care +/- neurosurgical intervention at a trauma centre
- spontaneous parenchymal ICH
- subarachnoid haemorrhage for surgery or endoluminal coiling of aneurysm.
- Acute hydrocephalus requiring ventricular drainage

3.2. At the time of tasking it is essential to assess the urgency of the transfer (need for time-critical neurosurgical intervention) as well as any particular requirements of the accepting neurosurgical team.

3.3 If a patient is being transferred for aneurysmal SAH consider the need for future specialist interventions like interventional radiology in order to avoid a subsequent transfer.

3.4. As outlined in the HOP Pre-hospital Traumatic Brain Injury, the main aims are to prevent secondary brain injury, secondary brain insults and avoid further increased ICP¹.

4. Surgical Management¹

4.1. Patients with EDH or acute SDH are likely to benefit from early neurosurgical evacuation. Patients who deteriorate (defined as a decrease of GCS by 2 or more points, or pupillary enlargement) should be retrieved as soon as practicable to the nearest Trauma Centre with neurosurgical capability.



4.1.1. If the transfer is expected to take longer than two hours then it is recommended² that the team discuss surgical options with the receiving neurosurgeon.

4.1.2. Options include:

- On-site burr-hole exploration on-site by the most competent local practitioner, general surgeon or by the retrieval physician in the regional hospital.
- Arranging a neurosurgeon (via MRU) to travel with a retrieval team to perform the operation.

4.2. The decision made with neurosurgical consultation is based on:

- Estimated transfer time
- Clinical state – GCS, pupillary sizes and light reflexes
- Rate of deterioration
- CT scan (if available)
- Level of surgical experience and range of neurosurgical equipment available at the regional hospital

5. Sedation and Paralysis

5.1. *In order to avoid spikes in ICP it is imperative that the patient be adequately analgesed and sedated.*

5.2 Movement between stretchers and changing ventilation circuitry are events which may cause patients to cough, gag or suffer arousal unless very well sedated. The ideal sedation regimen provides adequate analgesia and be easily titratable to effect with minimal haemodynamic responses.

5.2.1. Fentanyl is an effective analgesic with sedative properties and is cardiovascularly stable.

5.2.2. Midazolam has anticonvulsant properties which may be desirable.

5.2.3. Propofol is rapidly titratable, reduces cerebral metabolism and allows for neurologic assessment shortly after weaning. It may cause more cardiovascular depression.

5.2.4. Ketamine provides excellent analgesia as well as dissociative sedation. Historical concerns about its use in patients with raised ICP are unfounded, as it generally supports MAP and hence CPP.

5.3. The use of paralytic agents should be considered in all patients with raised ICP following **adequate analgesia and sedation**. Whilst muscle relaxants can mask clinical signs of seizure activity they are effective in preventing coughing and gagging and patient-ventilator asynchrony which can aggravate raised ICP.

4.4 Blood Pressure Manipulation

4.4.1 General Recommendations

Cerebral autoregulation in the injured brain may be impaired and a target CPP of 50-70mmHg is recommended. However, unless an external ventricular drain (EVD) with pressure monitoring is present it is not possible to determine CPP. Blood pressure targets must therefore be empirically chosen and should be discussed with the receiving neurosurgical team.

4.5 TBI

As discussed in the pre-hospital management of TBI, it is prudent to maintain the SBP over 90mmHg.^{1, 2}



4.6 SAH

4.6.1 In the case of unclipped aneurysmal SAH, the risk of re-bleeding in the first 24hr (7-17%)³ must be balanced against that of subsequent cerebral vasospasm and subsequent ischaemia which peak at 7-10 days³.

4.6.2 Hypertension is commonly present at the time of presentation. The evidence is weak but supports targeting a SBP no higher than 160mmHg.³

4.6.3 Following surgical management or endo-luminal coiling procedures, the emphasis shifts to maintaining cerebral perfusion, and a more permissive upper limit of BP may be acceptable, as with other forms of stroke.

4.6.4 Useful agents to control BP in this setting include:

- optimising analgesia and sedation
- esmolol or metoprolol
- hydralazine
- nimodipine infusion if preferred by neurosurgical team.

4.7 Spontaneous Intracranial Haemorrhage

Evidence for both the role and subsequent targets for blood pressure control in spontaneous ICH are even less evidence based but guidelines suggest controlling blood pressure where SBP >200mmHg or MAP > 150 by rapid acting titratable intravenous antihypertensives (Class C Recommendation)⁴

4.8 Reversal of Anticoagulation

All patients with intracranial haemorrhage should have consideration of reversal of warfarin or heparin therapy as soon as possible. Prothrombin Complex Concentrate with Vit K +/- Fresh frozen plasma should be administered to warfarinised patients.⁵

4.9 External Ventricular Drains (EVD)⁶

4.9.1 Patient are often retrieved with an EVD in-situ. Management of these drains in transit should be discussed with the neurosurgical team, in particular the need for ongoing monitoring, intermittent versus constant CSF drainage, and the preferred drainage height (pressure).

4.9.2 Pitfalls with EVDs include:

- Prolonged clamping leading to unrecognized rises in ICP or drain blockage.
- Difficulty maintaining the EVD's reference (zero) point at the level of the tragus.
- Over drainage associated with patient movement or vehicle G-forces. This can lead to recurrent haemorrhage.

4.9.3 There are 2 main management strategies:

1. Keep the EVD open throughout the retrieval except during the immediate patient bed/stretchers transfers.
2. Leave EVD closed and intermittently open (every 3- 5 min). This might be preferred in severe turbulence or a very rough road journey.



- 4.9.4 The zero point should be ascertained level with the tragus and the drainage chamber secured to the bridge (or to ceiling in vehicles). (see fig. 1)
- 4.9.5 It is preferable to clamp the drain when sliding or loading the patient and at take-off/landing.
- 4.9.6 The EVD should never be flushed as this can increase ICP.

4.10 Seizure Management

- 4.10.1 Prophylactic phenytoin (loading dose 20mg/kg at 1 mg/kg/min) reduces the chance of early (< 1 week) seizures following TBI but does not alter long term outcome.⁸ After SAH or ICH the routine use of prophylactic anticonvulsants is also controversial. Those patients who have suffered a seizure or who are at high risk (eg high grade SAH with focal signs) should be considered for prophylactic phenytoin.
- 4.10.2 Active seizures (convulsive or non-convulsive) should be managed aggressively and reversible causes excluded (eg hypoxia, hypoglycaemia, hyponatraemia).

4.11 Other Adjuncts

- 4.11.1 Hypothermia- although demonstrated to reduce cerebral metabolism, there is no good evidence supporting the use of therapeutic hypothermia following TBI in adults.⁷
- 4.11.2 IV Nimodipine- there is good evidence that enteral nimodipine commenced within 48hrs of SAH reduces the chance of delayed cerebral vasospasm, which is maximal between day 4 and 10⁸.
- 4.11.3 There is no evidence that IV nimodipine within 24hrs of SAH is helpful particularly as cerebral vasospasm is a delayed event, although many neurosurgical centres commence it. It is reasonable to cease a nimodipine infusion for short transfers if it poses logistic difficulties.⁸
- 4.11.4 There is no role for the use of steroids in TBI or SAH.

5. Documentation

In addition to routine observations, pupillary responses and where feasible GCS and limb motor function should be documented regularly for all patients with neurologic emergencies.

6. Review Date

May 2014

7. References

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5. Bershad EM, Suarez JI Prothrombin Complex Concentrates for oral anticoagulant therapy-related intracranial haemorrhage: a review of the literature. Neurocrit Care 2010 Jun;12(3):403-13.
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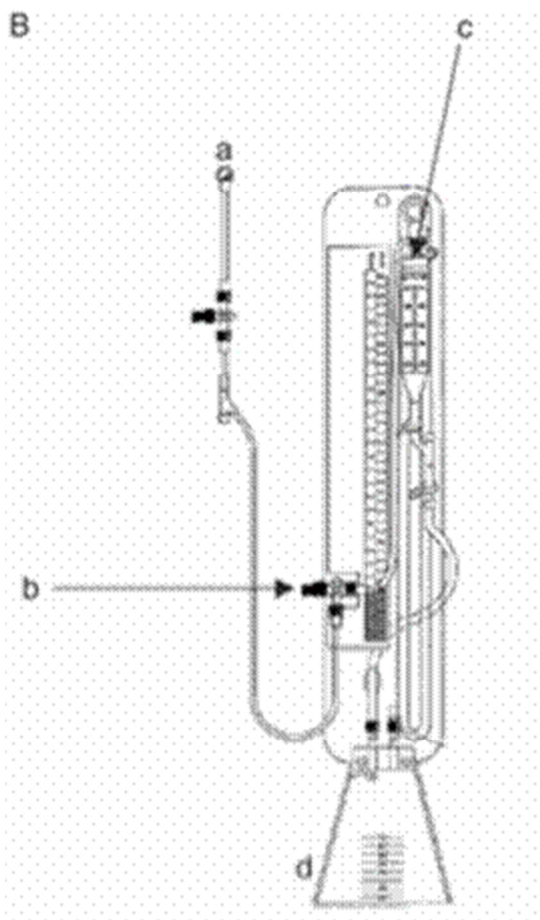


Figure 1⁶

a. Patient connection

b. Three way tap for connection to drain and/or pressure transducer. “Zero point” should be set at the level of the patient’s tragus.

c. Drip chamber set at prescribed distance above “zero point”.

d. Drainage bag.

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