

## Guideline for North Bristol Trust

# Care of Head Injured Patients

**This guideline describes the following:**

- Key Summary of Principles of Management of Head Injured Patients

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## Key Points

1. Effective analgesia is critical for all major trauma patients. All patients with significant pain should receive IV paracetamol if not otherwise contraindicated. Avoidance of sedating narcotics may have significant potential advantages in head injured patients and should be used with caution and titrated to effect.
2. Sedation (for any reason) makes accurate assessment of the GCS impossible and should be used only in order to gain control of an agitated patient in the pre-oxygenation phase of rapid sequence induction of anaesthesia.
3. RSI technique in head injury: Maintain oxygen saturations >94%, minimise pharyngeal and laryngeal stimulation and avoid unplanned hyperventilation; all of which risk poorer outcomes.
4. Following RSI: ventilation, volume management and packaging must be carefully considered but rapidly initiated with specific attention to optimisation of cerebral perfusion pressure.
5. Emergency control of clinically suspected raised ICP or impeding herniation can be attempted with boluses of 3ml/kg of 5% saline.

**Related Guidelines:  
Traumatic Brain Injury**

## Care of Head Injured Patients

### Background

The principles of head injury management are the provision of adequate oxygenation and cerebral perfusion, treatment of other significant injuries and rapid transfer to a neurosurgical service.

Many patients with head injury do not require urgent neurosurgery but, if they do, taking them directly to a neurosurgical centre cuts the time dramatically. Even when surgical intervention is not required patients with head injury do better when managed in neurosurgical centres.

Indications for emergency anaesthesia in patients with head injury are straightforward:

- Unconsciousness
- airway compromise
- ventilatory compromise

We also anaesthetise a number of patients with head injury and a relatively high GCS (9 – 14). Most of these patients have cerebral agitation and we know that patients who have cerebral agitation have a high incidence of intracranial pathology. Anaesthesia in this patient group makes them more manageable and may reduce the severity of secondary injury.

## Key Principles

### 1. Use of Analgesia & Sedation

**Sedation in head injured patients is a high risk procedure and should be performed only in the presence of those with significant experience and/or expertise.**

Effective multimodal analgesia is associated with better outcomes in head injured patients. All patients without contraindications should receive paracetamol (IV) and consideration of non-steroidal and opioid analgesia in the usual fashion.

Oral codeine, where appropriate may achieve significant analgesia with minimal sedation, facilitating more accurate assessment of GCS and clinical condition.

If patients are in severe pain from a head injury alone then this could signify intracranial pathology until proved otherwise. However pain primarily from systemic injury may push patients into the 'agitated' category; thus if effective analgesia cannot be achieved without the use of potentially sedating narcotic analgesia, small doses of fentanyl, morphine or oxycodone should be titrated to effect.

Sedation (for any reason) makes accurate assessment of the GCS impossible and should be used only in order to gain control of an agitated patient in the pre-oxygenation phase of rapid sequence induction of anaesthesia.

**Ketamine:** Concerns relating to its use in un-intubated patients with head injury (due to the possibility that ketamine raises ICP when CO<sub>2</sub> is not controlled) are largely unfounded. Ketamine has the advantage of not impairing respiratory drive and of being haemodynamically stable; its use is increasing in all traumatically injured patient groups.

If being used for induction of anaesthesia, then common practice is to use 10-20% of the intended induction dose as a sedative premedication to facilitate patient positioning and preoxygenation. The subsequent induction dose of ketamine should be reduced.

**Midazolam:** If the patient is agitated or combative, sedate with 1-2mg aliquots of midazolam until control is achieved and then proceed to rapid sequence induction. This also enables effective pre-oxygenation.

**Propofol:** This should be used in caution due to significant risk of apnoea, hypoventilation and loss of systemic vascular resistance. Its only use would be in the context of achieving preoxygenation prior to RSI where propofol is being used as the induction agent (usually, isolated head injury with significant hypertension).

## 2. Rapid Sequence Induction (RSI)

RSI technique in head injury should minimise CO<sub>2</sub> increases and pharyngeal and laryngeal stimulation in an attempt to minimise ICP rises. Meticulous attention to oxygenation is also important as is prevention of hyper- and hypoventilation (which has been associated with poor outcomes).

**This may be achieved by:**

### **Adequate induction agent**

- Use of adequate dose of Fentanyl and Ketamine where allowed by the patient's cardiovascular status.

### **Adequate paralysis:**

- Use 1mg/kg of Rocuronium
- Reparalyse frequently

### **Gentle and minimal laryngoscopy:**

- Avoid touching the posterior pharyngeal wall during intubation

**Minimal tube movement. Hold the tube when the patient is moved.**

## 3. Ventilation

Ventilate to low normocapnia (end-tidal CO<sub>2</sub> of 30 mmHg/4.0KPa). This equates to a PaCO<sub>2</sub> of approximately 4.5KPa in normal individuals. This minimises the risk of cerebral vasodilation (high PaCO<sub>2</sub>) and cerebral vasoconstriction (low PaCO<sub>2</sub>).

High levels of PEEP can increase ICP. Use of more than 5 cmH<sub>2</sub>O of PEEP without well founded clinical reason should be avoided.

## 4. Use of IV Fluids

After significant head trauma, the brain may lose the ability to autoregulate cerebral blood flow. A fall in mean arterial pressure may therefore result in a reduction in cerebral oxygen delivery even if the ICP is normal.

When effective splinting of limbs / pelvis has been maximised, then fluids should be administered to achieve a systolic blood pressure of 100mmHg. This can be increased to 120mmHg in isolated head injury.

## 5. Packaging

Compression of the jugular veins will reduce venous return from the head and neck. This can increase ICP. The cervical collar, if used, should therefore be left slightly loose. Cervical spine immobilisation will be maintained with head blocks and tape on the scoop stretcher. The neck veins can also be constricted by a tight tracheal tube tie – this should be checked and loosened. Tube tapes are a sensible alternative. The patient should be managed in a 20-30 degree head up position to maximise venous drainage. Tilt the whole trolley to achieve this, in adequately resuscitated patients.

## 6. Control of ICP / Impending Herniation

### **Hypertonic Saline (HTS):**

HTS has been shown to lower ICP in severe head injuries and may have other beneficial effects such as increasing circulating volume, minimal alteration to coagulation and anti-inflammatory properties. It is used extensively in ICU to lower refractory ICPs. North Bristol Trust uses sodium chloride 5%. There is no evidence that one formulation of hypertonic saline offers advantages over another. It is available as a 250ml or 500ml infusion bag.

### **Administration Policy:**

3ml / kg (to a maximum of 200ml) of 5% hypertonic saline should be delivered by well secured large bore peripheral (>18 gauge) cannula over 10 minutes in patients with signs of actual or impending herniation resultant from severe head injury:

- Unilateral or bilateral pupil dilation / GCS < 8 (usually 3)
- Progressive hypertension (SBP over 160mmHg) and bradycardia (pulse below 60) / GCS <8 (usually 3).

This dose is given once and given regardless of blood pressure.

In patients with blunt trauma, hypotension and head injury a bolus of HTS as above will help restore circulating volume and may protect against cerebral hypoperfusion and reduce oedema.

## Further Information and References

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### Further Information

NICE CG 44: Management of Head Injury

### References

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