

ADULT TRAUMA CLINICAL PRACTICE GUIDELINES

# Initial Management of Closed Head Injury in Adults

2nd Edition



Health

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# Contents

|  |           |   |            |
|--|-----------|---|------------|
| <b>Acknowledgements.....</b>   | <b>ii</b> | <b>Transfer to neurosurgical facility .....</b>   | <b>45</b>  |
| <b>Introduction .....</b>  | <b>3</b>  | 9. When should patients with closed head injury be transferred to hospitals with neurosurgical facilities?..... | 45         |
| <b>Changes from 2007 edition.....</b>  | <b>6</b>  | <b>Analgesia .....</b>  | <b>47</b>  |
| <b>Algorithm 1: Initial Management of Adult Closed Head Injury .....</b>   | <b>8</b>  | 10. What analgesia should patients with closed head injury receive? .....                                       | 47         |
| <b>Algorithm 2: Initial Management of Adult Mild Closed Head Injury.....</b>                                       | <b>9</b>  | <b>Anti-convulsants.....</b>  | <b>50</b>  |
| <b>Understanding the grades of recommendation .....</b>  | <b>10</b> | 11. Which patients with closed head injury should receive anti-convulsants? .....                               | 50         |
| <b>Mild Head Injury .....</b>  | <b>12</b> | <b>Evidence Tables .....</b>  | <b>53</b>  |
| 1. What is the definition of a mild head injury? .....   | 12        | <b>Appendices .....</b>   | <b>83</b>  |
| 2. What are the clinically important complications of mild head injury?.....                                       | 14        | Appendix 1: Definition of mild head injury.....   | 83         |
| 3. How should patients with mild head injury be assessed? .....  | 18        | Appendix 2: Initial GCS versus abnormal CT/Neurosurgery.....  | 87         |
| 4. Which patients with mild head injury require a CT scan?.....  | 27        | Appendix 3: Westmead PTA Scale .....  | 89         |
| 5. What should be done when patients with mild head injury deteriorate? .....                                      | 38        | Appendix 4: Abbreviated Westmead PTA Scale.....   | 91         |
| 6. When can patients with mild head injury be safely discharged and what discharge advice should be provided?..... | 39        | Appendix 5: The Glasgow Coma Scale – a practical implementation guide .....                                     | 94         |
| <b>Moderate head injury .....</b>  | <b>42</b> | Appendix 6: Mild head injury discharge advice.....  | 101        |
| 7. What are the proven treatments for patients with moderate head injury? .....                                    | 42        | Appendix 7: NSW Brain Injury Rehabilitation Program .....   | 103        |
| <b>Severe head injury.....</b>   | <b>43</b> | Appendix 8: Methodology .....   | 104        |
| 8. What are the proven treatments for patients with severe head injury? .....                                      | 43        | Appendix 9: Search Strategies .....   | 108        |
|  |           | <b>References.....</b>  | <b>116</b> |

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# Introduction

Trauma is the leading cause of death and disability in children and young adults in New South Wales and closed head injuries cause a significant proportion of this burden.<sup>1, 2</sup> Closed head injury may result in lifelong physical, cognitive, behavioural and social dysfunction for patients which in turn may place major social and financial burdens on their families and society.<sup>3</sup> Recent Australian figures indicate there are approximately 150 patients per 100,000 population admitted to hospital each year with closed head injuries.<sup>3-5</sup> Worldwide figures suggest an incidence range of 200-350 per 100,000 population per year for patients with closed head injury with mild head injury accounting for 80%.<sup>6</sup> Despite the fact that closed head injuries are common, the classification and management of closed head injuries remains surprisingly controversial and subject to variation in clinical practice.<sup>6-10</sup> Due to the large numbers of patients involved it has been estimated that even small improvements in closed head injury management could have significant impact.<sup>11</sup> Furthermore, it has been suggested that the greatest improvements can be made in the better management of those patients with mild to moderate head injury rather than those with severe head injury.<sup>12</sup>

Much of the controversy that exists about closed head injury management stems from the combination of a lack of uniformity in definitions with a paucity of large well designed studies in the area.<sup>11, 13, 14</sup> 'Head injury' is typically used to describe the initial clinical presentation whilst 'traumatic brain injury' or "concussion" are used to describe the subsequent functional outcome. The terms "mild head injury", "mild traumatic brain injury" and "concussion" are largely interchangeable and which term is used depends on whether you are examining emergency medicine, trauma, rehabilitation or sports medicine literature. It is difficult to find two studies that define mild head injury in exactly the same way so comparison of data can be difficult.<sup>6, 8-10, 13</sup>

Similarly, comparison of data in moderate to severe head injury studies is made difficult because controversy exists about how and when best to apply Glasgow Coma Scale (GCS) to sedated or intubated patients.<sup>15</sup> Perhaps most

significantly there have been very few large prospective randomised controlled trials of sufficient power and quality to guide management.<sup>11, 13, 14</sup> However, in the past few years there has been some progress in working toward uniform definitions and some better quality trials and meta-analyses have been published.<sup>6, 8-10, 15-35</sup>

The variety of clinical practice observed worldwide cannot be explained solely by the lack of uniformity of definitions and good quality studies. Much of the variation in management strategies between the USA, Canada, Europe and Australasia is driven by local issues such as the availability of resources, the medico-legal environment and in recent years the concerns about the potential harm from CT radiation.<sup>6, 36, 37</sup> Thus the USA has higher rates of CT scanning for mild head injuries compared to Canada, Europe and the UK. Even within countries and within institutions, considerable variation in practice has been shown to exist.<sup>7, 12, 35, 38</sup> Whilst some variation in clinical practice is to be expected, the introduction of clinical practice guidelines can potentially improve care and ensure adequate access to resources for more isolated areas.<sup>6, 35</sup> Furthermore, clinical guidelines can potentially reduce unnecessary tests and hospital admissions for mild head injury patients by identifying those patients at low risk of neurosurgically significant lesions.<sup>6, 13, 33-35</sup>

## Scope of the guideline

The guideline is intended for use by clinicians managing patients with closed head injury in major and regional trauma services, and urban and rural hospitals. The guideline is concerned with the initial care of the mild, moderate and severely head injured patient. The guideline will make evidence based recommendations on the diagnosis, resuscitation, and disposal of patients with closed head injuries.

The initial management plan for adults is based upon recommendations to be followed subject to the clinician's judgement in each case.

The recommendations however, are not prescriptive nor are they rigid procedural paths. It is recognised that the recommendations may not suit all patients in all clinical situations. They are intended to provide a clinically practical approach to the initial management of closed head injuries based on the current best available evidence. However, as with all guidelines, it should be remembered that they are a clinical tool and should not replace clinical judgement. The guideline relies on individual clinicians to decipher the needs of individual patients.

All recommendations regarding pre-hospital care should be read and considered in conjunction with the Ambulance Service of NSW.

Guidelines for the initial management of head injury in children can be found at [http://www.health.nsw.gov.au/policies/pd/2011/pdf/PD2011\\_024.pdf](http://www.health.nsw.gov.au/policies/pd/2011/pdf/PD2011_024.pdf)

## Aims and objectives

The guideline is intended to assist clinicians throughout NSW in delivering optimal care to patients with closed head injury. It aims to provide information to support clinical decision making, rather than dictate what decisions should be made.

The broad objectives of the guideline are to reduce morbidity and mortality in adult patients with closed head injury by providing clinicians with practical evidence based recommendations to assist them in managing such patients. It is also hoped that the guidelines may prevent unnecessary diagnostic tests and hospital admissions especially in the mild head injury group.

The process of constructing the guideline began with the clinicians on the Trauma Clinical Guidelines Committee posing a series of questions about the initial management of closed head injuries. The final questions were derived from the guideline priority areas identified by the committee; that is, the management of mild head injuries and the timing of transfer of patients with closed head injury from centres with limited resources. The initial management of patients with moderate to severe head injury was felt to be less controversial. This edition also includes recommendations in relation to the use of analgesia and anti-convulsants.

An extensive description of the methodology used for this guideline can be found at Appendix 8, together with the search terms used at Appendix 9.

### The clinical questions addressed:

- 1 What is the definition of a mild head injury?
- 2 What are the clinically important complications of mild head injury?
- 3 How should patients with mild head injury be assessed?
- 4 Which patients with mild head injury require a CT scan?
- 5 What should be done with high risk mild head injury patients when CT scan is unavailable?
- 6 What should be done when patients with mild head injury deteriorate?
- 7 When can patients with mild head injury be safely discharged?
- 8 What discharge advice should be provided?
- 9 What are the proven treatments for patients with moderate head injury?
- 10 What are the proven treatments for patients with severe head injury?
- 11 When should patients with closed head injury be transferred to hospitals with neurosurgical facilities?
- 12 What analgesia should patients with closed head injury receive?
- 13 Which patients with closed head injury should receive anti-convulsants?

## Defining closed head injury

This guideline uses the terms ‘closed head injury’ and ‘mild, moderate or severe head injury’ to identify and classify patients on arrival to hospital. The outcome following presentation with a ‘closed head injury’ will vary from rapid complete recovery to a mixture of structural lesions and functional deficits ranging from trivial to life threatening. The terms “concussion” and “traumatic brain injury” refer to the patient outcome following their initial presentation with a “closed head injury” and are retrospective diagnoses. Important functional deficits following ‘closed head injury’ range from post concussion symptoms and post traumatic amnesia to a variety of disabling persistent physical-cognitive-behavioural-social sequelae.

Many patients who suffer a “mild head injury” will have “mild concussion symptoms” or “mild traumatic brain injury symptoms”. If these acute “concussion” symptoms persist beyond the first few hours they are usually referred to as “post concussion symptoms”. The term “post concussion symptoms” is used to describe the clinical symptoms of mild brain injury that mild head injury patients may suffer for a few days to weeks following their injury. In the

situation where multiple post concussion symptoms persist for several months they are called a “post concussion syndrome”

As this guideline concentrates on the initial management of the patients presenting to hospital, it was felt that the term ‘head injury’ was more relevant to the initial clinical presentation than the term ‘traumatic brain injury’ that essentially refers to the subsequent functional outcome. It was also felt that the clinicians at whom this guideline is aimed would be far more familiar and comfortable with using the term ‘head injury.’ The definition of closed head injury is further discussed in Question 1.

## Classification of closed head injury

This guideline has classified patients with **initial GCS 14-15** on admission as **mild head injury**. This system classifies patients with initial GCS score of 13 in the moderate head injury group due to the patients having similarly patterns of intracranial injury and cognitive behavioural sequelae. The following table gives a rough guide to classification and outcome.<sup>15, 39-43</sup>

Table 1. Summary of closed head injury classification and outcome

|   | Mild Head Injury | Moderate Head Injury | Severe Head Injury |
|---|------------------|----------------------|--------------------|
| Initial GCS   | 14-15            | 9-13                 | 3-8                |
| % of Total  | 80               | 10                   | 10                 |
| Abnormal CT Scan (%) <sup>1,2</sup>   | 5-15             | 30 - 50              | 60 - 90            |
| Neurosurgical Intervention (%) <sup>1,2</sup><br>(excluding ICP monitoring) | 1-3              | 5-30                 | 30-50              |
| Mortality (%) <sup>1</sup>  | <1               | 10-15                | 30-50              |
| Good Functional Outcome (%) <sup>1,2,3</sup>                                | >90              | 20-90                | <20                |

Notes:

1. Generally the lower the GCS the worse the prognosis or the higher the rate of complications
2. Outcome deteriorates with increasing age - “children do better and elderly do worse”
3. Good functional outcome being return to independent ADL and to work or school at 6 months

# Changes from 2007 edition

## Background

The first edition of this guideline was written in 2005 using evidence available until December 2004. The aim of this new edition is to review the evidence published since December 2004 and to provide some additional information on specific topics including the role of anticonvulsants and analgesics in the management of closed head injury.

The aim of the original guideline was to provide a clinically practical evidence based guideline that summarised the initial management of adult closed head injury. It was piloted by the NSW Institute of Trauma and Injury Management (ITIM) and then formally adopted and published by NSW Health in January 2007. There was a conscious effort by the initial guideline team to provide a clinically practical document with clinically useful resources such as algorithms, summaries and discharge advice sheets backed up by a detailed evidence review. The guideline team has continued the same principles for this update, incorporating feedback from clinicians to improve the guideline. The algorithms and mild head injury discharge sheets have been revised to reflect the changes in the body of the guideline and the feedback received.

The guideline team would emphasise that this guideline is a clinical tool designed to assist clinicians and should be used to assist rather than replace the clinical judgement of an experienced clinician caring for an individual patient.

The information provided is based on the best available information at the time of writing, which is May 2010. These guidelines will be updated every five years and consider new evidence as it becomes available.

## New evidence

Since 2004 there have been many new studies and guidelines published about the management of closed head injury. There have been some advances in our understanding of the assessment and treatment of closed head injury but these have been incremental and

evolutionary rather than revolutionary. The basic principles of management of closed head injury remain the same in 2010 as they were five years ago.

The following section briefly outlines the most significant advances in knowledge from the recent literature incorporated in this update.

### Definition of mild head injury

- Recent literature emphasises that significant intracranial injury may occur without loss of consciousness or amnesia
- Patients with initial GCS 13 have a significantly higher rate of intracranial injury and should not be considered as having mild head injury

### Clinically important complications of mild head injury

- Recent literature emphasises that mild post concussion symptoms are common and that patients should receive appropriate discharge advice to assist recovery
- Acute neurosurgical complications are uncommon but important to identify

### Assessment of patients with mild head injury

- Recent literature emphasises that if structured clinical assessment indicates the risk of intracranial injury is low, the routine use of CT scanning is not warranted and is potentially harmful.
- Structured clinical assessment should include initial clinical history and examination, serial clinical observations and clinical risk factor assessment to determine the need for CT scanning
- A variety of clinical decision rules have been developed to determine which patients are at higher risk of intracranial injury and require CT scanning. However, they all require that the clinician is familiar with their inclusion / exclusion criteria and should be used as tools to support clinical decision making, rather than dictate management
- Post traumatic amnesia testing in the emergency



department, eg Abbreviated Westmead PTA Scale (A-WPTAS) can be useful in identifying patients with cognitive impairment at increased risk of structural lesions and post concussion symptoms.

#### Indications for CT scan for mild head injury

- Recent literature emphasises that patients can be risk stratified according to clinical risk factors and clinical decision rules. Patients who are classified as high risk should have CT scans to exclude clinically important intracranial lesions
- Significant head injuries can occur without loss of consciousness or amnesia and that the absence of these features should not be used to determine the need for CT scanning.
- Persistent abnormal mental status manifested by either abnormal GCS or abnormal alertness, behaviour or cognition is a strong indication for CT scanning
- Known coagulopathy and particularly supra-therapeutic anticoagulation are significant risk factors for intracranial injury and that these patients should have early CT scans and be considered for reversal of anticoagulation
- There have been several very large studies addressing this issue in the paediatric literature that have come up with very similar risk factors to the adult literature and have also confirmed that it is safe to discharge low risk patients without CT scanning.

#### Acute neurological deterioration

- Recommendations essentially unchanged
- Previously covered within guideline but now given separate question

#### Discharge of patients with mild head injury

- Recent literature emphasises that patients can be safely discharged for home observation if structured clinical assessment reveals no clinical risk factors indicating the need for CT scanning or following a normal CT scan if indicated.
- Deterioration of mild head injury patients following a normal CT scan is rare. Caution is advised for patients with known coagulopathy and elderly patients where the risk of a delayed subdural haemorrhage is increased.

#### Discharge advice for patients with mild head injury

- New section to emphasise importance of discharge advice
- Recent literature emphasises that all patients with mild head injury should be given both verbal and written discharge advice covering symptoms and signs of acute deterioration, when to seek urgent medical attention, lifestyle advice to assist recovery, information about typical post concussion symptoms and reasons for seeking further medical follow up. As with all discharge advice this should be time specific and action specific.
- An improved version of the original mild head injury advice sheet associated with this guideline has been developed and is now available in several languages.

#### Initial management of moderate head injury (GCS 9-13)

- Recommendations essentially unchanged

#### Initial management of severe head injury (GCS 3-8)

- Recommendations essentially unchanged

#### Transfer of patients with closed head injury to hospitals with neurosurgical facilities

- Recommendations essentially unchanged

#### Analgesia for closed head injury

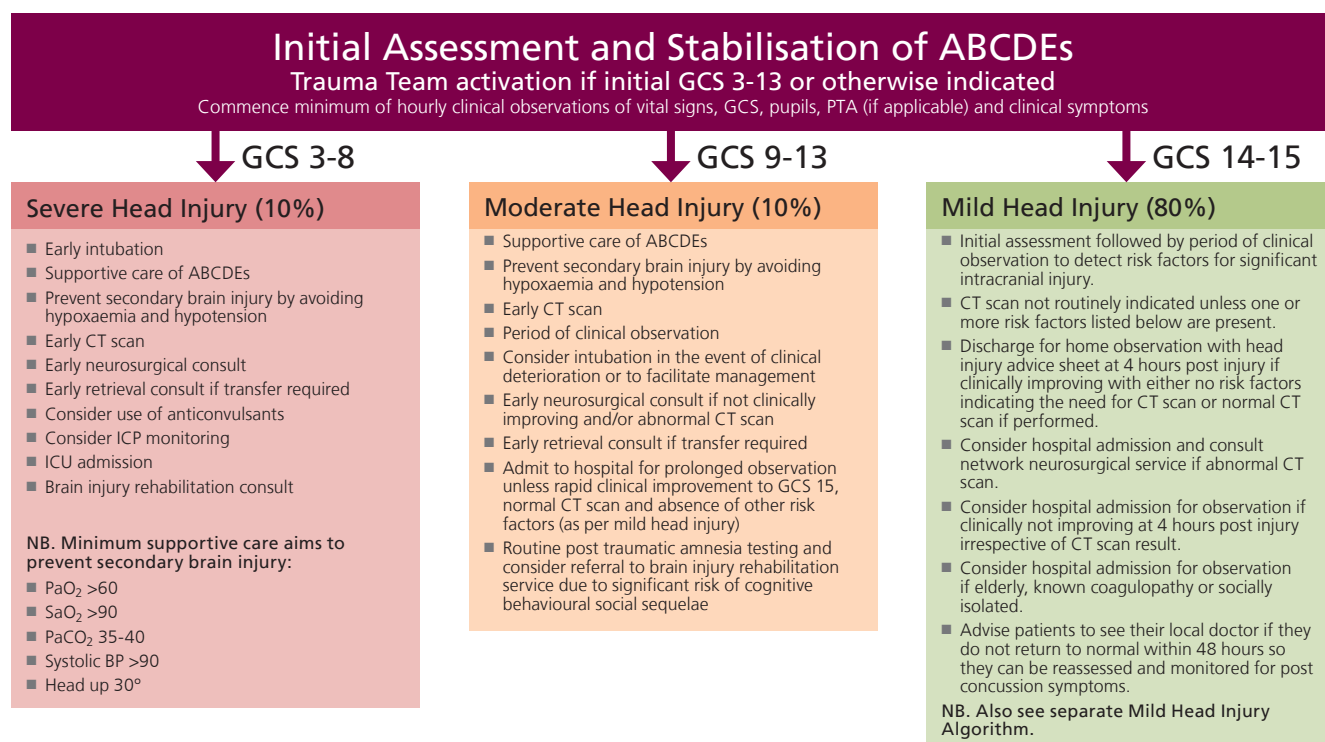
- New section

#### Anticonvulsants for closed head injury

- New section

## Algorithm 1:

# Initial Management of Adult Closed Head Injury



| Risk factors indicating potentially significant mild head injury  |  |  |
|---|--|--|
| <ul style="list-style-type: none"> <li>GCS &lt;15 at 2 hours post injury</li> <li>Deterioration in GCS</li> <li>Focal neurological deficit</li> <li>Clinical suspicion of skull fracture</li> <li>Vomiting (especially if recurrent)</li> <li>Known coagulopathy / bleeding disorder</li> </ul> | <ul style="list-style-type: none"> <li>Age &gt;65 years</li> <li>Post traumatic seizure</li> <li>Prolonged loss of consciousness (&gt;5 min).</li> <li>Persistent post traumatic amnesia (AWPTAS &lt;18/18)*</li> <li>Persistent abnormal alertness / behaviour / cognition*</li> <li>Persistent severe headache*</li> </ul> | <ul style="list-style-type: none"> <li>Large scalp haematoma or laceration.**</li> <li>Multi-system trauma**</li> <li>Dangerous mechanism**</li> <li>Known neurosurgery / neurological deficit.**</li> <li>Delayed presentation or representation**</li> </ul> |
| <p>* particularly if persists at 4 hours post time of injury</p> <p>**clinical judgement required</p>   |  |  |

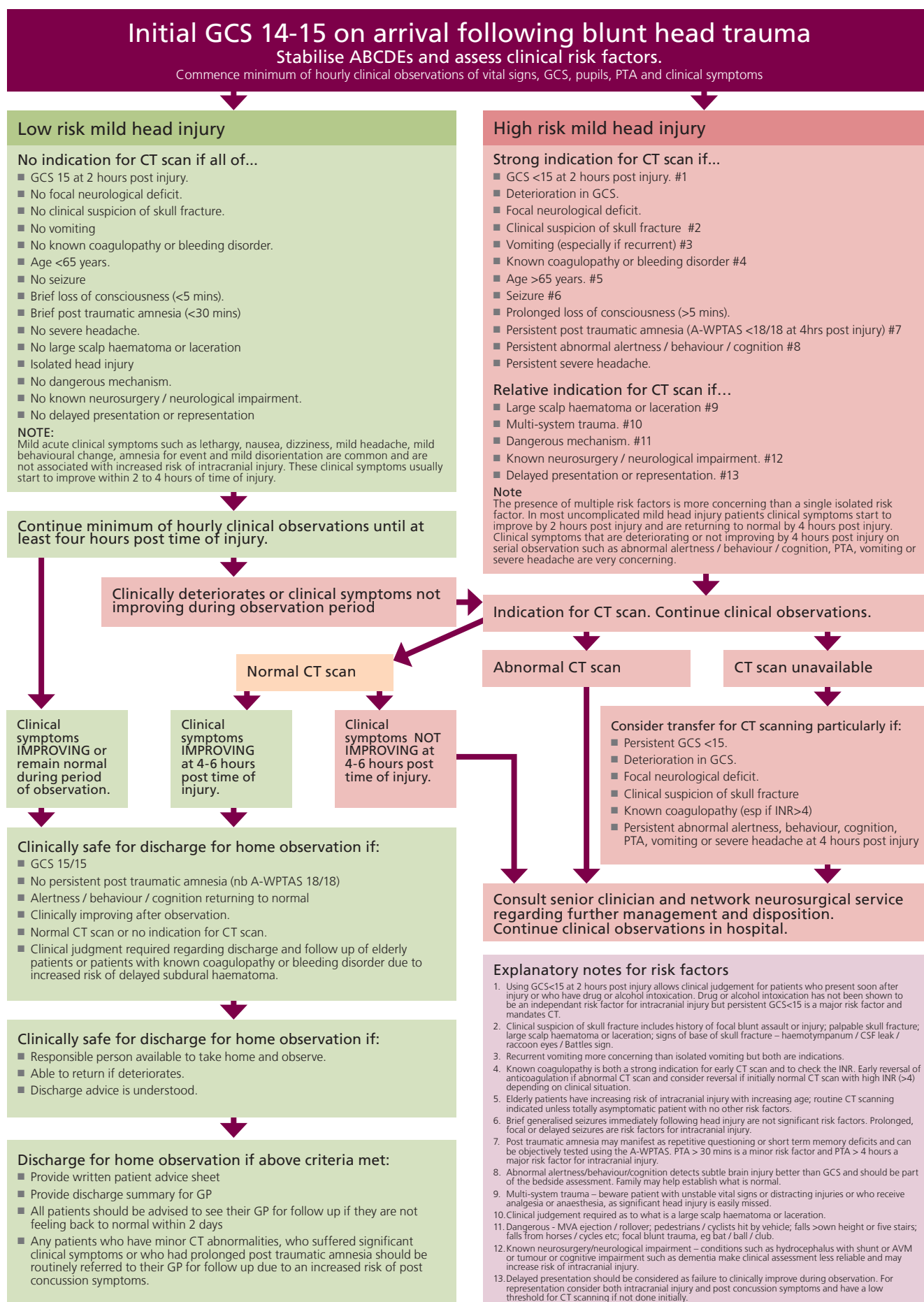
| What should be done when patients with closed head injury acutely deteriorate?   |   |
|--|---|
| <p><b>Early signs of deterioration</b></p> <ul style="list-style-type: none"> <li>Confusion</li> <li>Agitation</li> <li>Drowsiness</li> <li>Vomiting</li> <li>Severe headache</li> </ul> <p><b>Late signs of deterioration</b></p> <ul style="list-style-type: none"> <li>Decrease in GCS by two or more points</li> <li>Dilated pupil(s)</li> <li>Focal neurological deficit</li> <li>Seizure</li> <li>Cushing's response – bradycardia and hypertension</li> </ul> | <p><b>Clinical approach</b></p> <ul style="list-style-type: none"> <li>Resuscitate ABCDEs and exclude non head injury cause</li> <li>Supportive care of ABCDEs</li> <li>Early intubation if indicated</li> <li>Immediate CT scan</li> <li>If clinical or CT evidence of raised ICP/mass effect consult with network neurosurgical and retrieval services re;               <ul style="list-style-type: none"> <li>short term hyperventilation to PaCO<sub>2</sub> 30-35</li> <li>bolus of mannitol (1g/kg)</li> <li>local burr holes/craniectomy when more than 2 hours from neurosurgical care</li> <li>prophylactic anti-convulsants</li> </ul> </li> </ul> |

| When should patients with closed head injury be transferred to hospitals with neurosurgical facilities?  |  |
|--|--|
| <p><b>Potential indications</b></p> <p>Patient with severe head injury</p> <p>Patient with moderate head injury if:</p> <ul style="list-style-type: none"> <li>clinical deterioration</li> <li>abnormal CT scan</li> <li>normal CT scan but not clinically improving</li> <li>CT scan unavailable.</li> </ul> <p>Patient with mild head injury if:</p> <ul style="list-style-type: none"> <li>clinical deterioration</li> <li>abnormal CT scan</li> <li>normal CT scan but not clinically improving within 4-6 hours post injury</li> <li>mild head injury with CT scan unavailable, particularly if:               <ul style="list-style-type: none"> <li>Persistent GCS&lt;15</li> <li>Deterioration in GCS</li> <li>Focal neurological deficit</li> <li>Clinical suspicion of skull fracture</li> <li>Persistent abnormal mental status</li> <li>Persistent vomiting</li> <li>Persistent severe headache</li> <li>Known coagulopathy (particularly if age &gt;65 or INR &gt;4)</li> </ul> </li> </ul> | <p><b>Clinical approach</b></p> <ul style="list-style-type: none"> <li>When in doubt consult your network neurosurgical service.</li> <li>Patients with closed head injuries should be observed in facilities that can manage any complications that are likely to arise. Clinical judgment regarding risk of deterioration is required and neurosurgical consultation may be appropriate.</li> <li>Patients with closed head injuries should be transferred to the nearest appropriate hospital with neurosurgical facilities if there is significant risk of intracranial injury. The transfer of patients to hospitals with CT scan facilities but without neurosurgical services should be avoided.</li> </ul> |

|                               |                     |
|-------------------------------|---------------------|
| <b>AMRS (adult)</b>           | <b>1800 650 004</b> |
| 'formerly the MRU'            |                     |
| <b>NETS (children)</b>        | <b>1300 362 500</b> |
| Network neurosurgical service |                     |

## Algorithm 2:

### Initial Management of Adult Mild Closed Head Injury



# Understanding the grades of recommendation

## Strength of recommendations

This guideline uses the National Health and Medical Research Council's (NHMRC) overall grades of recommendation to indicate the strength of the body of evidence underpinning each recommendation. The body of evidence reflects the evidence components of all the studies relevant to each recommendation. The evidence

components are assessed according to the NHMRC body of evidence matrix (Table 2). The overall grade of the recommendation is determined based on a summation of the rating for each individual component of the body of evidence. Please note that a recommendation cannot be graded A or B unless the evidence base and consistency of the evidence are both rated A or B.<sup>44</sup>

Table 2: Body of evidence matrix<sup>44</sup>

| Components       | A   | B   | C  | D  |
|------------------|---|---|--|--|
|                  | Excellent   | Good  | Satisfactory   | Poor   |
| Evidence base    | one or more level I studies with a low risk of bias or several level II studies with a low risk of bias | one or two level II studies with a low risk of bias or a SR/several level III studies with a low risk of bias | one or two level III studies with a low risk of bias, or level I or II studies with a moderate risk of bias  | level IV studies, or level I to III studies/SRs with a high risk of bias   |
| Consistency      | all studies consistent  | most studies consistent and inconsistency may be explained  | some inconsistency reflecting genuine uncertainty around clinical question   | evidence is inconsistent   |
| Clinical Impact  | very large  | substantial   | moderate   | slight or restricted   |
| Generalisability | population/s studied in body of evidence are the same as the target population for the guideline        | population/s studied in the body of evidence are similar to the target population for the guideline           | population/s studied in body of evidence differ to target population for guideline but it is clinically sensible to apply this evidence to target population | population/s studied in body of evidence differ to target population and hard to judge whether it is sensible to generalise to target population |
| Applicability    | directly applicable to Australian healthcare context  | applicable to Australian healthcare context with few caveats  | probably applicable to Australian healthcare context with some caveats   | not applicable to Australian healthcare context  |

Overall grade A or B recommendations are generally based on a body of evidence that can be trusted to guide clinical practice, whereas Grades C or D recommendations must be applied carefully to individual clinical and organisational circumstances and should be interpreted with care (see table below).<sup>44</sup> This guideline also utilises an additional grade of “Consensus” where appropriate.

The recommendation boxes of each clinical question addressed in this guideline contain clear recommendations with an associated strength of recommendation grade as detailed below. Where appropriate, the author has also added relevant clinical points which support the given recommendation.

| Grade of recommendation | Description   |
|-------------------------|---|
| A                       | Body of evidence can be trusted to guide practice   |
| B                       | Body of evidence can be trusted to guide practice in most situations  |
| C                       | Body of evidence provides some support for recommendation(s) but care should be taken in its application  |
| D                       | Body of evidence is weak and recommendation must be applied with caution  |
| Consensus               | When limited literature was available, the author and editorial group utilised the best available clinical expertise, practices and accepted teachings to reach a consensus on the recommendation |

## Level of evidence

‘Level of Evidence’, applied to individual articles, refers to the study design used to minimise bias. Each article is classified according to their general purpose and study type

in accordance with the NHMRC publication: A guide to the development, evaluation and implementation of clinical practice guidelines.<sup>45</sup> From this, each article was allocated a level of evidence as follows:

|             |   |
|-------------|---|
| Level I     | Evidence obtained from a systematic review of all relevant randomised control trials  |
| Level II    | Evidence obtained from at least one properly-designed randomised control trial  |
| Level III-1 | Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method)  |
| Level III-2 | Evidence obtained from comparative studies (including systematic reviews of such studies) with concurrent controls and allocation not randomised, cohort studies, case-control studies, or interrupted time series with a control group |
| Level III-3 | Evidence obtained from comparative studies with historical control, two or more single arm studies or interrupted time series without a parallel control group  |
| Level IV    | Evidence obtained from a case-series, either post-test or pre-test/post-test  |

For more information on the methodology please see Appendix 8.

# Mild head injury

## 1. What is the definition of a mild head injury?

| Mild Head Injury Definition   | Strength of recommendation |
|---|----------------------------|
| <p>A patient with an initial GCS score of 14-15 on arrival at hospital following acute blunt head trauma with or without a definite history of loss of consciousness or post traumatic amnesia.</p> <p><b>Typical characteristics</b></p> <ul style="list-style-type: none"><li>■ Direct blow to the head or acceleration / deceleration injury.</li><li>■ Transient loss of consciousness or brief post traumatic amnesia.</li><li>■ Transient abnormal alertness, behaviour or cognition.</li><li>■ Rapid clinical improvement</li><li>■ Neurosurgical intervention rare (1-3%)</li><li>■ Abnormality on CT scan relatively uncommon (5-15%)</li><li>■ Post concussion symptoms common.</li><li>■ Long term functional outcome good.</li></ul> <p><b>Specific exclusions:</b></p> <ul style="list-style-type: none"><li>■ Clinically obvious penetrating head injury.</li><li>■ Non-traumatic brain injury.</li></ul> <p><b>Risk Stratification</b></p> <p>Patients may be classified into “high” and “low” risk groups based on the risk of suffering complications of their mild head injury. This risk stratification can be used to assist clinical judgement in determining the need for further assessment (eg CT scan), management and follow up. Stratification into “high” and “low” risk groups is based on the presence or absence of specified clinical risk factors identified by:</p> <ul style="list-style-type: none"><li>■ initial clinical history</li><li>■ initial clinical examination</li><li>■ serial clinical observation</li></ul> <p><b>“Complicated” Mild Head Injury</b></p> <p>A “complicated” mild head injury is a mild head injury resulting in one of the following:</p> <ul style="list-style-type: none"><li>■ significant structural lesion on CT scan</li><li>■ significant acute clinical symptoms</li><li>■ significant post concussion symptoms</li></ul> | CONSENSUS                  |

Recently published studies and guidelines use a variety of criteria to define mild head injury, which is variably referred to as either mild head injury or mild traumatic brain injury.<sup>6, 8-10, 13, 31-35, 46-48</sup> The most common variations concern the initial classification according to GCS and different requirements for loss of consciousness and post traumatic amnesia (summarised in Appendix 1). This variation in the literature makes comparison between studies difficult.

The main reason for this variability is a uniform desire to identify those patients at higher risk of intracranial injury in what is a heterogeneous but essentially low risk group. There is ample evidence to suggest that patients with an initial GCS of 13 should be considered as part of the moderate head injury group due to the frequency of intracranial lesions (25-38%) and cognitive-behavioural-social sequelae (see Evidence Table 1 and Appendix 2).<sup>9, 36, 43, 49-56</sup>

Since 2004 the adult literature has clearly identified that patients may sustain significant head injuries without loss of consciousness or post traumatic amnesia.<sup>9, 33, 43, 47, 57-59</sup> Therefore, the presence of loss of consciousness or post traumatic amnesia should not be used to define mild head injury or guide management. In 2008 Jagoda et al, representing the American College of Emergency Physicians / Centre for Disease Control, updated their definition of mild head injury to reflect the change in the evidence and now include patients with GCS 14 on initial assessment and have eliminated loss of consciousness or post traumatic amnesia as necessary inclusion criteria.<sup>9</sup>

Further risk stratification of mild head injury is then dependent on the presence of associated risk factors and different authors have different approaches. The approaches of some of the best quality studies and guidelines are summarised in Appendix 1. It is interesting to note that when all the initial GCS criteria, inclusion/exclusion criteria and sub-classification systems are all taken into account, that the findings are very similar. These findings are that mild head injury is a heterogeneous group with patients at higher risk of increased intracranial injury identified by persistently abnormal GCS and certain other risk factors.<sup>1, 6, 8-10, 13, 31-36, 47-54, 56-81</sup>

It is important to recognise that these risk factors for intracranial injury do not necessarily predict the risk of post concussive symptoms which are the more common complication of mild head injury. It is important that doctors, patients and their families understand that the absence of a structural lesion on CT scan following a mild

head injury does not exclude the possibility of significant cognitive-behavioural-social sequelae.<sup>9, 82</sup>

The recent paediatric literature has come up with similar definitions for mild head injury to the adult literature and identified persistently abnormal GCS or mental status and other specified risk factors as the major indicators of intracranial injury.<sup>83-87</sup>

## 2. What are the clinically important complications of mild head injury?

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| <p>The clinically important complications of mild head injury are:</p> <ul style="list-style-type: none"> <li>■ structural lesions on CT scan requiring acute neurosurgical intervention</li> <li>■ structural lesions on CT scan requiring hospital admission and/or neurosurgical consultation</li> <li>■ acute clinical symptoms requiring acute hospital admission</li> <li>■ post concussion symptoms causing disabling cognitive behavioural social sequelae</li> </ul>   | CONSENSUS                  |
| <p>Structural lesions on CT scan requiring acute neurosurgical intervention are rare (1-3%).</p> <p>Typical lesions include:</p> <ul style="list-style-type: none"> <li>■ acute extradural haematoma</li> <li>■ acute subdural haematoma</li> <li>■ depressed skull fractures</li> </ul> <p>Structural lesions on CT scan requiring hospital admission and/or neurosurgical consultation are relatively uncommon (5-15%). Not all of these lesions will require hospital admission.</p> <p>Typical lesions include:</p> <ul style="list-style-type: none"> <li>■ small intracranial haematomas/haemorrhages</li> <li>■ minor skull fractures</li> </ul>   | A                          |
| <p>Clinicians and patients should be aware that the absence of a structural lesion on CT scan following mild head injury does not exclude the possibility of significant acute clinical symptoms or significant post concussion symptoms.</p> <p>Acute clinical symptoms are common immediately following mild head injury but should be starting to improve in most patients within two to four hours of time of injury.</p> <p><b>Common acute clinical symptoms include:</b></p> <ul style="list-style-type: none"> <li>■ post traumatic amnesia</li> <li>■ disorientation</li> <li>■ confusion</li> <li>■ drowsiness</li> <li>■ dizziness</li> <li>■ nausea</li> <li>■ vomiting</li> <li>■ headache</li> </ul> <p>Patients with persistent acute clinical symptoms at four hours post time of injury require prolonged clinical observation and a CT scan should be performed (if not already done) to exclude a structural lesion.</p> | A                          |
| <p>Patients with persistent post traumatic amnesia and/or other persistent significant acute clinical symptoms that are not improving require prolonged clinical observation and should be admitted to hospital even if their initial CT scan is normal.</p>  | CONSENSUS                  |



| RECOMMENDATION   | Strength of recommendation |
|--|----------------------------|
| <p>Post concussion symptoms are relatively common following mild head injury and may have significant cognitive-behavioural-social impact on patients and their families.</p> <p>Many patients will have minor post concussion symptoms that will resolve within a few days while some patients will have more significant <b>post concussion symptoms</b> that will take a <b>few weeks to resolve</b>. A small number of patients with mild head injury will have persistent disabling post concussion symptoms after 3 months and will require referral for brain injury rehabilitation assessment. Most of these patients will improve by 12 months.</p> <p>Mild head injury patients with structural lesions on CT scan, a history of significant acute clinical symptoms or documented persistent post traumatic amnesia are at increased risk of post concussion symptoms but post concussion symptoms can occur in the absence of these features.</p> <p>The only interventions that have been shown to be beneficial for post concussion symptoms are education, reassurance and time. Therefore, it is important to provide education about post concussion symptoms to all mild head injury patients. All patients should be given written advice and advised to see a doctor if they are not feeling better within a few days of injury.</p> <p><b>Typical post concussion symptoms include:</b></p> <ul style="list-style-type: none"> <li>■ headaches</li> <li>■ dizziness</li> <li>■ fatigue</li> <li>■ memory impairment</li> <li>■ poor concentration</li> <li>■ mood swings</li> <li>■ behavioural changes</li> <li>■ sleep disturbance</li> <li>■ social dysfunction</li> </ul> | B                          |

Clinically important complications of mild head injury include both structural lesions and functional deficits. The most important structural lesions to identify are those requiring acute neurosurgical intervention. However, functional deficits resulting in cognitive-behavioural-social sequelae are far more common and may have significant impact on patients and their families. It is important that doctors, patients and their families understand that the absence of a structural lesion on CT scan following a mild head injury does not exclude the possibility of significant cognitive-behavioural-social sequelae.

Acute intracranial haematomas requiring acute neurosurgical intervention are the most dramatic and life threatening complications of mild head injury. The identification of structural lesions requiring acute neurosurgical intervention is the most important function of CT scanning because the presence or absence of

other structural lesions does not usually significantly alter outcome. However, multiple studies have shown that these neurosurgically significant lesions are relatively uncommon with incidences of 0.1-3.2% for GCS 15 and 0.5-6.5% for GCS 14 with most of the larger studies finding that acute neurosurgical intervention is required in less than 1% of mild head injury patients (see Evidence Table 1 and Appendix 2 for more detail).<sup>32, 33, 35, 36, 47, 49-53, 68, 74, 77, 88-90</sup> Other intracranial injuries and skull fractures are more frequently noted on CT scans but are usually only clinically important as indicators of the potential for clinical complications such as delayed intracranial haematomas, post traumatic seizures or post concussion symptoms.<sup>52, 76</sup> Delayed acute intracranial haematomas requiring neurosurgical intervention are uncommon following normal CT scans (range < 0.1%).<sup>13, 14, 31, 39, 91, 92</sup>

There has been much debate in the literature about the

importance of identifying abnormalities on CT scan that do not require clinical intervention, such as small intracranial haematomas and small non-depressed skull fractures. Clearly it is important to identify intracranial lesions that require neurosurgical intervention but is it beneficial to identify abnormalities on CT scan that do not require intervention? Concerns about radiation exposure and resource utilisation have influenced this debate. The trend in the literature is to develop strategies to identify clinically important lesions while minimising the number of CT scans performed. The outcome of this strategy is that a small number of minor abnormalities on CT will be missed. Therefore, not all abnormalities detected on CT scan should be regarded as clinically important.

Acute clinical symptoms associated with mild head injury are common and are sometimes referred to as concussion symptoms. These include abnormal mental status (alertness/behaviour/cognition), post traumatic amnesia, vomiting, headache, dizziness and lethargy. In the majority of mild head injury patients, their acute clinical symptoms will rapidly improve and they may be left with mild post concussion symptoms or return to completely normal. In most patients these symptoms start to improve within a couple of hours of injury and it is unusual for significant symptoms to persist for more than 4 hours post time of injury. Persistent acute clinical symptoms indicate a significant functional injury and an underlying structural lesion should be ruled out with a CT scan. Patients with persistent acute clinical symptoms with a normal CT scan should be admitted to hospital for prolonged observation until their symptoms start to improve. They should have continued neurological observations and post traumatic amnesia (PTA) testing.

Post concussion symptoms are relatively common following mild head injury and may have significant cognitive-behavioural-social impact on patients and their families.<sup>3, 8-10, 76, 93-99</sup> Post concussion symptoms include headaches, dizziness, fatigue, memory problems and other cognitive, behavioural and social dysfunction. Post concussion symptoms have been shown in some studies to occur in up to 25 - 50% of patients with mild head injury,<sup>3, 8, 10, 76, 94-96, 98, 99</sup> but in about 10% of cases they may persist with at times significant psychological overlay as post concussion syndrome.<sup>3, 8, 10, 76, 94, 98, 99</sup> In an Australian study Faux et al<sup>100</sup> found that 15% of patients with mild traumatic brain injury continued to complain of post traumatic headache at 3 months compared to 2% of controls. These symptoms usually resolve within three months. The cognitive-

behavioural-social dysfunction caused by mild head injury can be quite disabling, and some researchers have suggested that the severity of impact on lifestyle makes the term 'mild' inappropriate for some patients.<sup>3, 98, 99</sup> Patients with significant persistent post concussive symptoms should be referred to a brain injury rehabilitation service or neurologist by their GP (see Appendix 7).

Most of the studies looking at post concussion symptoms included patients with initial GCS 13-15 with either transient confusion or disorientation or loss of consciousness (<30 min) or PTA (<24 hours) who did not require neurosurgery. Therefore, they tended to exclude lower risk patients without loss of consciousness or amnesia and include higher risk patients with initial GCS 13 when compared to the definition of mild head injury used in this guideline. The inclusion of patients without loss of consciousness or amnesia and the classification of patients with initial GCS 13 as moderate head injury means that the incidence of post concussion symptoms may be less common in the patients classified as mild head injury in this guideline. However, Lannsjö et al<sup>96</sup> in a population based study of patients with initial GCS 15 found that about 34% of patients reported multiple (3 or more on the Rivermead Questionnaire) significant ongoing post concussion symptoms at three months. Kraus et al<sup>95</sup> found about 30% of their patients (GCS 13-15) had a similar frequency of multiple symptoms although it is interesting to note that about 20% of their control group of patients attending ED for other problems reported multiple symptoms. Kraus et al<sup>95</sup> found that headaches, dizziness, forgetfulness and frustration were the Rivermead symptoms that best identified mild head injury patients from the controls. Clearly, post concussion symptoms occur in many mild head injury patients but it is difficult to define which patients will get multiple persistent symptoms due to the mild head injury as many symptoms are common to other conditions, as well as the general population. The findings of these recent studies again emphasised the importance of providing education and follow up information regarding post concussion symptoms to all patients with mild head injury as a significant minority may have persistent symptoms.

Post concussion symptoms are relatively common following mild head injury and may have significant cognitive-behavioural-social impact on patients and their families. Mild head injury patients with structural lesions on CT scan, significant acute clinical symptoms or significant PTA are at increased risk of post concussion symptoms but post

concussion symptoms can occur in the absence of these features. The only interventions that have been shown to be beneficial for post concussion symptoms are education, reassurance and time.<sup>95, 101, 102</sup> Therefore, it is important to provide education about post concussion symptoms to all mild head injury patients.

brain injury rehabilitation can be found in the Motor Accidents Authority of NSW 'Guidelines for mild traumatic brain injury following a closed head injury'<sup>103</sup> and Evidence Table 2.

Further information on post concussion symptoms and

The clinically important complications of mild head injury suggest that the management priorities should be:

- The identification of patients requiring early acute neurosurgical intervention.
- The identification of patients requiring admission to hospital due to the increased risk of deterioration from complications.
- The identification of patients who can be safely discharged for home observation.
- The provision of discharge advice to allow the identification and early return of patients with unexpected deterioration.
- The provision of discharge advice to allow the identification, treatment and follow-up of patients who develop significant post concussion symptoms.

### 3. How should patients with mild head injury be assessed?

| RECOMMENDATION   | Strength of recommendation |
|--|----------------------------|
| <p>Mild head injury patients should be assessed by a process of structured clinical assessment involving a combination of:</p> <ul style="list-style-type: none"> <li>■ initial clinical history and examination</li> <li>■ serial clinical observations</li> <li>■ CT scanning if clinically indicated by risk factors identified on initial or serial assessment</li> </ul>  | A                          |
| <p>Serial clinical observation should include minimum hourly observations of :</p> <ul style="list-style-type: none"> <li>■ vital signs.</li> <li>■ pupillary reactions</li> <li>■ GCS</li> <li>■ alertness / behaviour / cognition</li> <li>■ post traumatic amnesia (PTA) (eg A-WPTAS)</li> </ul> <p>If patients have no significant risk factors for complications of mild head injury and are clinically judged to be “low risk” then they should be observed until at least four hours post time of injury.</p> <p>If patients have any significant risk factors for complications of mild head injury then they should continue to be clinically observed while further assessment is performed.</p> <p>Serial clinical observations should be continued on any mild head injury patients who fail to clinically improve at four hours post injury or who are found to have structural lesions on CT scan.</p> | B                          |
| <p>Assessment for PTA should be performed on all mild head injury patients in the emergency department. Mild head injury patients who are admitted to hospital because they have structural lesions, persistent PTA or clinical symptoms should have daily PTA testing until they are shown to be out of PTA.</p>  | C                          |
| <p>Clinical assessment using clinical risk factors or clinical decision rules can identify those patients at increased risk of intracranial injury requiring further investigation.</p>  | A                          |
| <p>CT scanning is indicated for those mild head injured patients identified by structured clinical assessment as being at increased risk of intracranial injury.</p>   | A                          |
| <p>CT scanning is the most appropriate investigation for the exclusion of neurosurgically significant lesions in mild head injured patients</p>  | A                          |
| <p>If structured clinical assessment indicates the risk of intracranial injury is low, the routine use of CT scanning is neither clinically beneficial nor cost effective.</p>   | B                          |
| <p>Skull x-rays are not sufficiently sensitive to be used as a routine screening investigation to identify significant intracranial lesions.</p>   | A                          |

Clinical history and examination remain the basis for the initial assessment of patients with closed head injury. However, additional clinical tools are available to assist in assessment and management. These clinical tools include Glasgow Coma Scale (GCS), initial serial neurological observation, post traumatic amnesia (PTA) testing, clinical decision rules, CT scanning and prolonged clinical observation in hospital.

## Glasgow coma scale

The Glasgow Coma Scale (Appendix 5) was originally developed by Teasdale and Jennett<sup>104</sup> for the neurological observation of patients with prolonged coma. It was intended to ensure inter-observer reliability and to identify deterioration of patients over time. Since its original introduction its use has been extended such that it is now the standard tool for assessment of level of consciousness in many clinical settings.

GCS is used both for the initial assessment and classification of closed head injuries and for serial assessment of closed head injuries. Initial GCS on admission to hospital is used to classify head injuries into the broad prognostic groups of mild (GCS 14-15), moderate (GCS 9-13) and severe (GCS 3-8). The Brain Trauma Foundation concluded that there is good quality evidence to relate initial GCS score to outcome.<sup>15</sup> However, it must be noted that these are broad outcome measures and initial GCS is only about 75% accurate so treatment should be individualised.<sup>15</sup>

GCS is unreliable if measured before initial resuscitation and stabilisation of the ABCDE's has been completed.<sup>15</sup> Fearnside et al<sup>105</sup> identified that both intubation and sedation interfered with accurate assessment of initial GCS in more severely injured patients and there is lack of uniformity of approach to classifying GCS in these situations.

The other significant area of controversy relates to timing of initial GCS. In unstable patients requiring resuscitation, the optimal time to record initial GCS remains controversial.<sup>15</sup> <sup>105</sup> Similarly, in mild head injury patients the time of presentation related to time of injury will influence the initial GCS and therefore potentially influence clinical decision making in relation to CT scanning.<sup>6, 10, 31, 35</sup> Perhaps the most crucial point to note about initial GCS is that it cannot predict individual outcome for patients with similar GCS scores. Thus, an individual patient with an initial GCS of 14-15 may have a variety of outcomes including

no significant injury, long-term cognitive-behavioural dysfunction or a life threatening extradural haematoma.

This is particularly important for mild head injury patients because GCS primarily assesses the risk of structural lesions. Subtle cognitive changes are not well discriminated within the mild head injury group. This led to the development of the extended Glasgow Coma Scale and the Abbreviated Westmead PTA Scale that assess the duration of post traumatic amnesia as a means of identifying patients at increased risk of cognitive problems.<sup>106-109</sup> Despite these limitations initial GCS on admission remains the standard method for initial classification of head injuries.

When assessing initial GCS in patients with head injury it is worthwhile considering time of injury. Clearly initial GCS for a given patient may vary depending on time of presentation to hospital. Few studies have related GCS to the time of injury with the exception of Stiell et al<sup>35</sup> who found GCS <15 at two hour post injury was a significant predictor of intracranial injury for mild head injury patients. Most recent studies that have reported time of presentation have shown that mild head injuries present around 60 to 90 minutes post injury.<sup>31, 43, 54</sup> An abnormal initial GCS taken within one hour of injury is therefore likely to overestimate the risk of intracranial injury in mild head injury patients who present early.

Prehospital GCS, motor score and return of orientation are other factors to consider when assessing initial GCS. Prehospital GCS was felt to be unreliable,<sup>110</sup> but with more organised prehospital systems, it is gaining further attention.<sup>15, 111</sup> Motor score is the best predictor of outcome of the GCS components.<sup>15</sup> Orientation returns most commonly in the sequence of person, place then time.<sup>112</sup>

The Glasgow Coma Scale is used as one of the parameters in serial observation of head injury patients. This is what it was originally designed for and what it is most useful for. Both the original studies and subsequent studies have validated its use in this fashion and, prior to the advent of CT scanning, alteration in GCS was the most useful tool in predicting intracranial injury.<sup>9, 104</sup> Borg et al<sup>101</sup> in a meta analysis for the World Health Organisation on mild traumatic brain injury concluded that in the absence of CT scanning that hospital observation for at least 24 hours for patients with GCS 15 and other risk factors was a reasonable strategy. Currently, serial GCS remains a standard tool in the monitoring of head injuries when CT scanning is unavailable or when clinical symptoms persist

despite normal CT scanning.

### Summary

- Initial GCS scores are useful to classify closed head injuries into broad prognostic groups requiring further assessment and serial observation.
- Serial GCS scores are useful to observe the trend in clinical condition over time for all patients
- Serial GCS scores may be used as alternative to CT scanning in patients with mild head injury and GCS 15 when CT scanning is not available
- Serial GCS scores are useful for identifying patients with significant risk of intracranial injury in:
  - patients with an initial abnormal GCS score who fail to improve
  - patients whose initial GCS score deteriorates.
- Initial and serial GCS scores are not as valuable in excluding significant injury in:
  - patients with an initial normal GCS score who remain normal
  - patients with an initial abnormal GCS score who improve.
- Initial GCS should only be used for prognostic purposes after initial resuscitation and stabilisation of ABCDE's.
- The motor component of the GCS is the best predictor of outcome
- The extended GCS or Abbreviated Westmead PTA Scale which assess both GCS and duration of PTA are designed to help identify mild head injury patients at increased risk of post concussion symptoms by identifying more subtle cognitive changes.

### Serial clinical neurological observation

Serial clinical neurological observations remain a standard tool for assessing mild head injury patients despite the advent of CT scanning. CT scanning is primarily used to identify structural abnormalities at a given point in time while serial neurological observations are used to monitor clinical condition over a longer period of time. Serial neurological observation typically consists of at least hourly pupillary reactions and GCS assessment in conjunction with vital signs. The symmetry of motor responses and a standardised orientation assessment should be routinely noted as part of the GCS. Neurological assessment should also include qualitative assessment of alertness, behaviour and cognition as this may identify more subtle neurological impairment.

In mild head injury patients the primary aims of serial

neurological observation are the early identification of acute neurological deterioration and the identification and monitoring of persistent mild neurological deficits. Serial neurological observations remain the basic standard of care for the initial management of mild head injury patients and should be used in conjunction with clinical decision rules to determine the need for CT scanning and / or prolonged observation. The qualitative aspects of serial neurological observation may assist in identifying acute deterioration or subtle functional abnormality before there is a change in GCS. Subtle drowsiness, mild disorientation to time, inattention and mildly disorganised thought processes with subtle post traumatic amnesia can easily be missed by over reliance on GCS in a patient who is awake and orientated in person and place.

Controversy exists over the appropriate duration of serial neurological observation for both mild head injury patients who are improving and those who have persistent clinical symptoms or abnormalities on CT scan. Although four hours of initial neurological observations post injury are fairly standard following mild head injury, there is little evidence to support this. There is also some debate as to whether the initial period of observation should be until four hours post injury or for four hours following arrival at hospital. Studies that reported time of presentation showed that most patients present at one to two hours post injury. Clinically most uncomplicated mild head injury patients will start to improve within two to four hours post time of injury. Stiell et al<sup>35</sup> demonstrated that GCS<15 at two hours post injury was one of the most significant risk factors for intracranial injury. Since this guideline has used these clinically important criteria, it was felt that the initial clinical observation period should also be based on time post injury rather than four hours observation from the time of arrival at hospital. It is important that patients who present one hour post injury are not treated the same as those who present at four hours post injury with "routine observations" for four hours. If the patient who presents at four hours post injury is not clinically improving then they are at increased risk of intracranial injury and warrant CT scanning whereas the same patient at one hour post injury probably only requires observation unless there is another strong indication for CT scanning eg suspected skull fracture, seizure, warfarin etc. The initial period of neurological observation should therefore be until at least four hours post time of head injury at which point clinical decisions should be made if they are not clinically improving.

If the serial clinical neurological observations of patients with an initial GCS 15 at 2 hours post injury are improving or have returned to normal at four hours post injury then the evidence would suggest it is safe to discharge them for home observation if they have no risk factors indicating the need for CT scan or if they have an initial normal CT scan. There is no strong evidence to support a specific duration of time of observation. Observation until 4 hours post time of injury should be used as an initial guide. Patients with trivial injuries could be considered for earlier discharge by an experienced senior clinician and patients with high risk factors should be considered for longer periods of observation.

If serial neurological observations are not improving at four hours post injury then serial neurological observations should be continued and clinical decisions need to be made about the need for CT scanning and/or admission to hospital for prolonged observation. The period of admission to hospital for prolonged observation is also controversial as there is little evidence to support the general recommendation of twenty four hours. This period of observation is derived from studies that show that clinical deterioration is unusual in mild head injury patients after twenty four hours. Indeed, prior to the advent of CT scanning serial clinical observation was the standard of care and the need for neurosurgical intervention in mild head injury patients was largely determined on the basis of clinical deterioration. The best location for prolonged neurological observation for lower risk patients is also debated because some studies have shown that admission to hospital does not guarantee that regular neurological observation will occur.<sup>53</sup>

If patients are not clinically improving or have abnormalities on CT scan that warrant hospital admission, then serial clinical observations should be continued. The duration of this time of observation remains poorly defined. Basically, clinical observation should be continued until clinical symptoms improve or until it is felt there is little risk of deterioration. This needs to be individualised but a minimum of 24 hours is normally recommended.<sup>101</sup>

### Summary

- Serial neurological observation is a useful tool for the early identification of acute neurological deterioration and the identification and monitoring of persistent mild neurological deficits.
- Serial neurological observations should include a minimum of hourly GCS assessment, pupillary reactions, PTA and vital signs. Neurological observations should also include qualitative assessment of alertness, behaviour and cognition to detect subtle changes in mental status not assessed by the Glasgow Coma Scale.
- Mild head injury patients should have initial serial neurological observations until at least four hours post time of injury at which point decisions about further management should be made.
- Serial neurological observations should be continued on patients who are admitted to hospital for at least 24 hours and until patients clinically improve and are discharged home.

### Post traumatic amnesia (PTA) testing

Amnesia for the event, short-term memory loss, anterograde amnesia and PTA are all terms used to describe the disruption of memory that typically results from a traumatic head injury. The different terms describe a continuum of memory disruption, and the use of the individual terms depends on the duration of memory loss and individual preference. Amnesia for the event is common and of little clinical significance. Retrograde and anterograde amnesia are typically used to refer to the duration of loss of memory for events preceding or following an injury. PTA is the period of time during which a person is unable to lay down new memories following an injury. PTA and anterograde amnesia essentially refer to the same phenomena but some patients may have memory for events yet still be unable to lay down new memories – the so called islands of memory. Stiell et al<sup>56</sup> identified a duration of greater than 30 minutes of retrograde amnesia as being a significant risk factor for intracranial injury. Smits et al<sup>47</sup> identified a duration of PTA of greater than 2 hours as being a minor risk factor and greater than 4 hours as being a major risk factor for intracranial injury. Many studies have shown that persistent PTA is a significant risk factor for poor functional outcome in all grades of head injury. Any persistent inability to lay down new memories following blunt trauma is perhaps most simply referred to as PTA and the duration of PTA may be used to predict the risk of intracranial injury and the risk of persistent post concussion symptoms. PTA is the term that will be predominantly used in this guideline.



While it has been identified that prolonged PTA is a risk factor for significant post concussion symptoms,<sup>3, 109</sup> the standard tool for identifying PTA in NSW, the Westmead PTA Scale (Appendix 3), is designed to be used over several days. There has been considerable interest in developing bedside PTA screening tools to allow early prediction of which patients are at greatest risk of developing post concussion symptoms. A simple bedside test that can determine whether the patient is out of post traumatic amnesia at the time of the test would be beneficial for both emergency department testing and ward based testing. The Extended Glasgow Coma Scale and the Abbreviated Westmead PTA Scale are examples of tools developed to try to identify persistent PTA in mild head injury patients as a marker for increased risk of post concussion symptoms.<sup>106, 109</sup> Recent Australian studies have looked at using specific bedside tools such as the Revised Westmead Post Traumatic Amnesia Scale in the emergency department to better identify patients with persistent PTA so that these patients can be more closely followed up as they are more likely to suffer significant post concussion symptoms.<sup>106-108</sup> The Abbreviated Westmead PTA Scale (A-WPTAS), developed and validated<sup>113</sup> from the previously validated Revised Westmead PTA Scale, has been successfully trialled in various New South Wales emergency departments (Appendix 4).

From a practical point of view, all patients with mild head injury should be assessed for post traumatic amnesia. It is useful to assess the patient's recall of events following their injury by asking specific questions such as what is their first clear memory, who helped them at the scene and how they got to hospital. This can be used to estimate the period of post traumatic amnesia (anterograde amnesia). Clinicians should also look for symptoms of post traumatic amnesia such as repetitive questioning, failure to remember clinical staff and inability to remember events during their hospital stay. A formal assessment tool like the A-WPTAS or a simple memory assessment technique such as three object recall can be used as a bedside screening test for post traumatic amnesia, to supplement the 'history' of amnesia for events. Patients who have persistent post traumatic amnesia should be considered for CT scanning, prolonged observation and close follow up. **Mild head injury patients who are admitted to hospital because they have structural lesions, persistent PTA or clinical symptoms should have daily PTA testing until they are shown to be out of PTA.**

## Summary

- The identification of persistent PTA in mild head injury patients is a potentially useful marker for the risk of intracranial injury and the risk of developing post concussive symptoms.
- PTA testing should be performed on any patient presenting to hospital following mild head injury.
- The Revised and Abbreviated Westmead PTA Scales are useful bedside tools for assessing PTA in the emergency department

## Clinical decision rules

Clinical decision rules are increasingly being used to assist clinicians in determining the need for particular investigations or management. By identifying individual risk factors and combining them to establish clinical decision rules, which are then prospectively validated, useful evidence based diagnostic tools to assist management can be developed. Well established clinical decision rules also include the NEXUS criteria for cervical spine assessment and the Ottawa Ankle Rules.<sup>114, 115</sup>

Although clinical decision rules are potentially very useful, clinicians need to be aware of the specific inclusion/exclusion criteria used to develop them and the overall quality of the original studies before applying them to their patients.<sup>9, 48</sup> The other important point for clinicians to consider is what level of risk they are prepared to accept. No clinical decision rule can entirely rule out a significant finding for an individual patient, and different clinical decision rules will have different levels of risk. In patients with mild head injury it is important to have high negative predictive value for ruling out significant intracranial injuries but this comes at a cost of lower specificity and therefore the need for more CT scanning. While all the clinical decision rules aim to rule out significant neurosurgical lesions, they have different approaches to the value of identifying intracranial lesions on CT scan that do not require intervention. Clearly it is important to identify intracranial lesions that require neurosurgical intervention, but is it beneficial to identify abnormalities on CT scan that do not require intervention? Concerns about radiation exposure and resource utilisation have influenced this debate. The trend in the literature is to develop strategies to identify clinically important lesions while minimising the number of CT scans performed. Essentially, if you want to do less CT scans you have to accept that you will miss some intracranial abnormalities that are unlikely to require intervention.



Clinical decision rules for adult patients with mild head injury have been developed through studies of large cohorts of patients and prospectively applied to patients to determine their sensitivity, specificity and negative predictive value.<sup>31, 32, 35, 47, 48, 56, 58, 116</sup> These studies aim to identify those patients at increased risk of intracranial injury and develop clinical decision rules about which patients require CT scanning or prolonged observation. The best studies should be well designed and prospectively internally validated in the original population and then externally validated by other groups in other populations. The main design features of these trials are summarised in Evidence Table 1 and Appendix 1.

The most consistent findings of these studies are that abnormal GCS or mental status, clinical suspicion of skull fracture, focal neurological deficit, vomiting, coagulopathy and age > 65 are the best predictors of risk of intracranial injury. Other relevant predictors of risk include mechanism of injury, prolonged loss of consciousness, post traumatic amnesia, severe headache and seizure. Depending on their inclusion / exclusion criteria the authors used combinations of these risk factors to derive clinical decision rules of varying sensitivity and specificity (Evidence Table 1 and Appendix 1). At present, the findings of both Haydel et al<sup>32</sup> and Stiell et al<sup>35</sup> have been adopted as policy by the American College of Emergency Physicians (USA),<sup>9</sup> whilst the findings of Stiell et al<sup>35</sup> have been adapted by the NICE guidelines (UK).<sup>117</sup> The other widely known clinical prediction rule is NEXUS II.<sup>58</sup>

Ibanez et al<sup>33</sup> in 2004 attempted to prospectively identify clinical risk factors predicting intracranial injury and to assess the reliability of previously published clinical guidelines. They found that while clinical risk factors could not detect all intracranial injuries they could be used to detect clinically relevant lesions with a negative predictive value approaching 99%. They also concluded clinicians should be aware of the limitations of clinical decision rules when using clinical guidelines. An Australian study in 2004 by Rosengren et al<sup>118</sup> looked at applying the clinical decision rules developed by Haydel et al<sup>32</sup> and Stiell et al<sup>35</sup> to Australian practice and concluded that both had limitations. More recently an Australian study by Fong et al<sup>57</sup> developed a local guideline by adapting the findings of Haydel et al<sup>32</sup> and Stiell et al<sup>35</sup> and applying the resultant guideline to an Australian population with satisfactory results. The authors stressed the point that no clinical decision rule is infallible and that appropriate explanation and discharge advice was important.

A recent review by Stein et al in 2009 compared most of the best known clinical decision rules for mild head injury by applying them retrospectively to a prospectively collected data base and found that they all performed well for identifying acute neurosurgical lesions but differed in their ability to identify other lesions and their predicted CT scan ordering rates. The authors concluded that NEXUS II probably performed the best but the accompanying editorial commented that it would be reasonable to use any of the clinical decision rules.<sup>48</sup>

The Canadian CT Head Rules are the most widely studied and have been externally validated several times. However, they were applied to GCS 13-15 patients, require loss of consciousness or amnesia and excluded unstable multi-system trauma, coagulopathy, pre hospital seizure and neurological deficit. The Canadian CT Head rule therefore needs to be adapted for application to the broader population or clinicians need to be fully aware of these limitations when using the original rule. The NEXUS II clinical decision rule has not been as extensively externally validated but has the advantage of being relatively simple to use and can be used for all patients with head injury as there were no exclusion criteria other than delayed presentation and both adult and paediatric populations were studied. From a clinically practical point of view the mnemonic "BEAN BASH" can be used to remember the NEXUS II indications for CT scanning patients with head injury at the bedside:

B – behaviour abnormal;  
E – emesis;  
A – alertness abnormal;  
N – neurological deficit;  
B – bleeding disorder;  
A – age>65;  
S – skull fracture suspected;  
H – haematoma scalp.

The most important point for any clinician to recognise is that clinical decision rules should be used as tools to support clinical decisions and should not override clinical judgement. If clinicians choose to use a clinical decision rule, they should be aware of the limitations and inclusion / exclusion criteria of whichever clinical decision rule they decide to use.

## Summary

- Clinical decision rules provide useful adjuncts to the assessment and management of mild head injury patients.

- Studies by Haydel et al<sup>32</sup> (New Orleans Criteria), Stiell et al<sup>35</sup> (Canadian CT Head Rules) and Mower et al<sup>58</sup> (NEXUS II) have provided externally validated evidence based clinical decision rules for mild head injury in adults. The Canadian CT Head Rules are the most extensively studied of the clinical decision rules.
- Clinical decision rules should be used as tools to support clinical decisions and should not override clinical judgement
- If clinicians choose to use a clinical decision rule, they should be aware of the limitations and inclusion / exclusion criteria of whichever clinical decision rule they decide to use.

## Skull x-rays

The literature clearly identifies that both the clinical suspicion of skull fractures and the radiological evidence of skull fracture are significant risk factors for the presence of an intracranial lesion requiring neurosurgical intervention.<sup>6, 31, 32, 35, 66, 74</sup>

If CT scanning is available, the current indications for skull x-rays are few. However, if CT scanning is unavailable, the role of skull x-rays as a screening test is less clear. A detailed meta analysis by Hofman et al<sup>66</sup> concluded that whilst the presence of skull fracture greatly increased the risk of intracranial injury, the absence of a skull fracture did not rule it out (calculated sensitivity 38% calculated specificity 95%). In subsequent clinical guidelines, authors have differed as to whether skull x-rays should be used to detect patients at higher risk of intracranial injury. Jagoda et al<sup>9</sup> argued that the sensitivity of skull x-ray is not sufficient to be used as a screening test. Vos et al<sup>10</sup> and Servadei et al<sup>6</sup> felt that in the absence of CT scanning, a positive skull x-ray can be useful to help allocate patients into higher risk groups for management purposes. On existing evidence, both approaches seem reasonable depending on local management guidelines.

### Summary

- Clinical evidence or suspicion of skull fracture is associated with increased risk of intracranial injury.
- Skull x-rays are not sufficiently sensitive to be used as a routine screening test to identify patients at increased risk of intracranial injury.
- Where CT scanning is unavailable, skull x-ray may be used as an adjunct to identify patients with skull fractures who are at greater risk of intracranial injury (but not to exclude intracranial injury).

## CT scanning

The widespread availability of CT scanning has greatly assisted the management of patients with head injuries. CT scanning has been particularly useful in identifying focal injuries in patients with altered level of consciousness or other risk factors. CT scanning is regarded as mandatory for all head injury patients with a persistent altered level of consciousness. However, the role of the CT scanning in a patient with mild head injury with a normal level of consciousness remains controversial. Multiple clinical decision rules have been developed to try to identify which patients should have a CT scan because of concerns about the routine use of CT scanning for all patients. Particular concerns about the routine use of CT scanning for mild head injury include the financial-resource burden, the potential hazards of radiation and the potential pitfalls of reliance on technology at the expense of clinical assessment.<sup>37, 71, 88, 119-123</sup> Furthermore, CT scans do not identify patients who have cognitive dysfunction which is the most significant complication for most patients. The various pros and cons of CT scanning are summarised below:

### Pros

- Early identification of patients with intracranial injuries requiring acute neurosurgical intervention
- Early identification of patients with other intracranial injuries requiring admission to hospital due to risk of deterioration
- Identification of patients at low risk of deterioration and suitable for discharge
- Identification of patients with structural lesions indicating increased risk of post concussive symptoms.<sup>76</sup>
- Potential cost benefit due to early CT scanning allowing discharge home rather than hospital admission in some patients.<sup>119</sup>

### Cons

- Routine use of CT scanning for mild head injury potentially has a huge financial and resource impact given that more than 90% of scans are negative and less than 1% of scans indicate the need for neurosurgical intervention.<sup>13, 35, 124</sup>
- Some patients, particularly the elderly or those with a known coagulopathy, may develop delayed focal neurosurgical lesions (especially subdural lesions) despite initial normal CT scanning.<sup>69, 125, 126</sup>

- Early CT scans may not demonstrate intra-cerebral contusions which take time to become apparent on CT scanning.
- CT scanning will not demonstrate diffuse axonal injury in most patients.<sup>3</sup>
- Patients may suffer significant post concussive symptoms despite an initial normal CT scan.<sup>3, 94, 98, 99, 110</sup>
- Routine use of CT scanning does not guarantee better identification of significant intracranial injuries if different institutions are compared.<sup>12, 37</sup>
- The risk of cumulative radiation exposure especially among children is of concern.<sup>120-123</sup>
- May delay definitive management of more significant injuries in multi system trauma patients.

### CT scanning and radiation

There is increasing concern about the potential harm from radiation associated with CT scanning.<sup>122, 123, 127-130</sup> The main risk associated with radiation exposure from CT scanning is an increased lifetime risk of fatal cancer.<sup>122, 123</sup> There have also been concerns about the effects of radiation on cognitive development in children. Hall et al<sup>120</sup> showed a significant reduction in educational performance in young children who received the equivalent dose of radiation of one or two CT heads for childhood haemangiomas. The risk posed by radiation is greatest in young children due to the relatively increased dose on more sensitive tissue and the increased time for a cancer to develop. Thus a person who has a CT scan as a child is at much greater risk than a person who has their first CT scan age 65 where the risk is very small. The risk posed by CT scan radiation is cumulative and is greatest in patients with chronic disease who have multiple scans. In patients with moderate to severe head injury there is a clear benefit from performing a CT scan because the probability of identifying a life threatening injury clearly outweighs the small increase in the lifetime risk of cancer. In the case of patients with mild head injury the risk of harm from radiation needs to be weighed against the relatively low probability of identifying a neurosurgically significant lesion on CT scan. This is of most concern in younger patients where the effective radiation exposure is higher, the time available for developing a cancer greater and the chance of cumulative lifetime exposure greater. Hence the interest in developing clinical decision rules for mild head injury patients to determine who really needs a CT scan.

The risk from radiation can either be expressed as an estimated lifetime risk of fatal cancer or as an estimated equivalent dose of radiation which takes into account organ doses and their relative radiosensitivity.<sup>122, 123, 127, 128, 130</sup> It is important to recognise that these risks are all estimates based on epidemiological studies of atomic bomb survivors extrapolated to current estimated doses of radiation delivered by CT scans. Perhaps the best way to put the risk of CT scanning into perspective is to compare the estimated risks with more common everyday risks.<sup>129</sup>

Put into context the risk of radiation from an individual head CT scan is very low.<sup>122, 123, 127-130</sup>

- Estimated overall lifetime risk of fatal cancer (1 in 3)
- Estimated risk of clinically important lesion on CT in mild head injury (1 in 100)
- Estimated additional lifetime risk of fatal cancer from adult trauma panscan (1 in 1,000)
- Estimated additional lifetime risk of fatal cancer from single child CT head (1 in 5,000)
- Estimated additional lifetime risk of fatal cancer from single adult CT head (1 in 10,000)
- Estimated additional lifetime risk of fatal cancer from trauma series x-rays (1 in 20,000)
- Estimated additional lifetime risk of fatal cancer from single chest x-ray (1 in 1,000,000)
- Estimated equivalent dose of radiation from a chest x-ray (0.02 mSv per CXR)
- Estimated equivalent dose of radiation from adult CT head (2 mSv per scan)
- Estimated equivalent dose of radiation from annual background radiation (2 mSv per year)
- Estimated equivalent dose of radiation from adult CT trauma panscan (20 mSv per scan)
- Annual safety limit for radiation exposure for radiation workers (.20 mSv per year)
- Estimated mean equivalent dose of radiation exposure for atomic bomb survivors linked to increased rates of fatal cancer (40 mSv dose)

It is important to recognise that the absolute risk to an individual is relatively small particularly with advancing age. More caution is recommended in children due to the concerns on cognitive development as well as lifetime risk of cancer. The lifetime risk of cancer for a young child receiving a CT head would be roughly double that of an adult (1 in 5000).

The following points try to put the risk into context and are adapted and simplified from the original references: <sup>121-123, 127-130</sup>

- Single adult CT head equivalent risk to having 100 chest x-rays
- Single adult CT head equivalent risk to smoking 1000 cigarettes
- Single adult CT head equivalent risk to driving 5000 km on a highway
- Single adult CT head equivalent risk to one year of background radiation
- Single adult CT head increases lifetime risk of fatal cancer from 30% to 30.01%
- Single young child CT head increases lifetime risk of fatal cancer from 30% to 30.02%
- Single adult CT trauma panscan increases lifetime risk of fatal cancer from 30% to 30.1%

### Timing of CT scanning

There is no direct evidence to confirm what the best time to perform CT scanning in relation to time of injury is. The primary role of early CT scanning in mild head injury is early recognition of extradural or subdural haematomas prior to clinical deterioration.<sup>69</sup> Early neurosurgical intervention prior to clinical deterioration is associated with improved outcome. However, early CT scan may potentially miss other intracranial injuries such as delayed subdural haematomas or contusions which are slower to become evident.<sup>69</sup> Fortunately, most studies have shown that an initial normal CT scan allows safe discharge and that the few patients who deteriorate usually have good outcome.<sup>9, 53, 89, 130</sup> Therefore, it is reasonable to suggest that CT scans should be performed shortly after a decision is made that one is necessary.

### Adjunctive CT scanning

Some mild head injury patients will require CT scanning for other reasons such as cervical spine clearance in the elderly. Clearly, in these circumstances, clinicians should have a lower threshold for performing head CT scans at the same time for ease of management.

### Repeat CT scanning

There has been some debate in the literature about whether mild head injury patients who have initially abnormal CT scans and require admission for hospital observation should have a routine repeat CT scan.<sup>132-136</sup> The evidence from most of these small studies suggests that

most mild head injury patients with minor abnormalities on CT scan do not require routine repeat CT scanning if they are clinically improving with a normal GCS and no neurological deficit unless they are anti-coagulated.

The other question that is often asked is should elderly anti-coagulated patients with normal initial CT scans have routine repeat CT scans and if so when should they be performed and should the patient be admitted for observation. There is little evidence to guide management in these situations. The consensus appears to be that the older the patient, and the more the patient is anti-coagulated (higher INR), the greater the risk of delayed bleeding. However, how to manage that risk remains unclear.

### Summary

- CT scanning is the best investigation for the early identification of neurosurgically significant focal intracranial lesions following mild head injury.
- CT scanning should be used as an adjunct to clinical assessment.
- Where structured clinical assessment indicates the risk of significant intracranial lesion is low, the routine use of CT scanning is unlikely to be of benefit.
- CT scanning does not accurately predict the risk of post concussion symptoms in mild head injury patients.
- Early CT scanning may theoretically not demonstrate some subdural haematomas and cerebral contusions but there is little evidence to suggest that this is clinically relevant in most patients.
- There is little evidence to guide the management of anti-coagulated elderly mild head injury patients with initially normal CT scans who are potentially at risk of delayed bleeds, especially subdural haematomas. There is increased risk with increased age and degree of coagulopathy but how to manage that risk remains unclear.
- Routine repeat CT scanning is not indicated for most clinically improving mild head injury patients with minor abnormalities on initial CT scan.

#### 4. Which patients with mild head injury require a CT scan?

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| <p><b>'High risk' mild head injury requiring CT scan</b></p> <p>The following risk factors identify patients with mild head injury (initial GCS 14-15) at <b>increased risk of clinically significant lesions requiring acute neurosurgical intervention or prolonged observation in hospital</b>. These patients <b>should have early CT scanning</b> if available, if they have any of the following features:</p> <p><b>On initial assessment</b></p> <ul style="list-style-type: none"> <li>■ GCS&lt;15 at two hours post injury**</li> <li>■ Focal neurological deficit</li> <li>■ Clinical suspicion of skull fracture</li> <li>■ Vomiting</li> <li>■ Known coagulopathy or bleeding disorder</li> <li>■ Age &gt;65</li> <li>■ Witnessed seizure</li> <li>■ Prolonged loss of consciousness (&gt;5min)</li> </ul> <p><b>On serial assessment</b></p> <ul style="list-style-type: none"> <li>■ Decrease in GCS</li> <li>■ Persistent GCS&lt;15 at two hours post injury</li> <li>■ Persistent abnormal alertness/behaviour/cognition</li> <li>■ Persistent post traumatic amnesia (A-WPTAS&lt;18/18 at 4hrs post injury)</li> <li>■ Persistent vomiting (≥ 2 occasions)</li> <li>■ Persistent severe headache</li> <li>■ Post traumatic seizure</li> </ul> <p><b>Clinical judgement required if</b></p> <ul style="list-style-type: none"> <li>■ Initial GCS 14 within two hours of injury**</li> <li>■ Large scalp haematoma or laceration</li> <li>■ Associated multi-system injuries</li> <li>■ Dangerous mechanism</li> <li>■ Known neurosurgery/neurological impairment</li> <li>■ Delayed presentation or representation</li> </ul> <p><small>** NOTE: Includes patients with abnormal GCS due to drug or alcohol ingestion.</small></p> | A                          |
| <p><b>If CT scanning is unavailable</b></p> <p>"High risk" mild head injury patients should be closely observed and be considered for transfer to a hospital with neurosurgical and CT scan facilities when CT scan is unavailable.</p> <p>A clear decision about the need for transfer for CT scanning for "high risk" patients should be made at the time of initial assessment or after a brief period of observation. A local senior clinician should be consulted and the patient discussed with the network neurosurgical service.</p> <p>The clinical symptoms of patients with mild head injury typically improve within two to four hours post time of injury. Patients with persistently abnormal or worsening clinical symptoms are at "high risk" of intracranial injury. A clear decision about the need for transfer for CT scanning should be made no later than 4 hours post time of injury.</p>  | CONSENSUS                  |

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| <p>Patients at “highest risk” of intracranial injury who should be discussed with the network neurosurgical service regarding urgent transfer for CT scanning include those with:</p> <ul style="list-style-type: none"> <li>■ Persistent GCS&lt;15 at two hours post injury</li> <li>■ Focal neurological deficit</li> <li>■ Clinical suspicion of skull fracture</li> <li>■ Any deterioration in GCS</li> <li>■ Post traumatic seizure in ED</li> <li>■ Known coagulopathy (particularly if age &gt;65 or INR &gt;4)</li> <li>■ Persistent vomiting or severe headache</li> <li>■ Persistent abnormal alertness, behaviour, cognition or PTA at 4 hours post injury.</li> </ul>   | A                          |
| <p>If it is decided, after consultation with a network neurosurgical service, that a “high risk” patient does not require urgent transfer for CT scanning, then that patient should have close clinical observation in hospital for at least 24 hours and until clinically improving. If there are any signs of deterioration or no improvement, the network neurosurgical service should again be consulted. Rapid transfer to a neurosurgical centre in the event of deterioration must be available if this strategy is to be used.</p>  | CONSENSUS                  |
| <p>If patients are transferred for CT scanning they should ideally be transferred to a hospital with neurosurgical facilities to avoid secondary transfer.</p>  | CONSENSUS                  |
| <p>A skull x-ray may be useful to confirm the presence of a skull fracture that mandates an early CT scan due to the increased risk of deterioration.</p>   | B                          |
| <p><b>‘Low risk’ mild head injury not requiring CT scan</b></p> <p>The following features indicate patients with mild head injury (initial GCS 14-15) at <b>low risk of having clinically significant lesions requiring acute neurosurgical intervention or prolonged observation in hospital</b>. These patients <b>should not routinely have CT scanning</b> if they have all of the following features:</p> <p><b>On initial assessment</b></p> <ul style="list-style-type: none"> <li>■ GCS 15 at two hours post injury.</li> <li>■ No focal neurological deficit.</li> <li>■ No clinical suspicion of skull fracture.</li> <li>■ No vomiting.</li> <li>■ No known coagulopathy or bleeding disorder.</li> <li>■ Age &lt;65 years.</li> <li>■ No post traumatic seizure</li> <li>■ Nil or brief loss of consciousness (&lt;5min).</li> <li>■ Nil or brief post traumatic amnesia (&lt;30min)</li> <li>■ No severe headache.</li> <li>■ No large scalp haematoma</li> <li>■ Isolated head injury</li> <li>■ No dangerous mechanism</li> <li>■ No known neurosurgery / neurological impairment</li> <li>■ No delayed presentation or representation.</li> </ul> <p><b>After a period of observation (until at least four hours post time of injury)</b></p> <ul style="list-style-type: none"> <li>■ GCS 15/15</li> <li>■ No post traumatic amnesia (A-WPTAS 18/18)</li> <li>■ Normal mental status including alertness, behaviour and cognition.</li> <li>■ No clinical deterioration during observation.</li> <li>■ Clinically returning to normal</li> </ul> | A                          |

Mild head injury patients (initial GCS 14-15) require a CT scan if they are clinically assessed as being at significant risk of acute deterioration from an underlying intracranial injury. The WHO Taskforce on MTBI (Borg et al 2004<sup>101</sup>) concluded that on the basis of their meta-analysis that “there is strong evidence that clinical factors can predict CT scan abnormalities and the need for (neurosurgical) intervention in adults.” There have been many recent studies looking at which risk factors predict intracranial injury and the need for CT scan and a variety of clinical decision rules have been developed. These studies are mostly based on large prospectively collected data bases in the USA, Canada, and Europe although their findings have been reproduced in smaller studies throughout the world. Haydel et al<sup>32</sup> (2000 – New Orleans Criteria), Stiell et al<sup>35</sup> (2001 – Canadian CT Head Rules), Mower et al<sup>58</sup> (2005 – NEXUS II) and Smits et al<sup>47</sup> (2007 – CHIP Rule) have all derived clinical decision rules with slightly varying inclusion and exclusion criteria and identified risk factors. Ibanez et al<sup>33</sup> (2004), Fabbri et al<sup>125</sup> (2004) and Stein et al<sup>48</sup> (2009) have also done large studies looking at clinical risk factors and assessing the validity of the various clinical decision rules and guidelines while Rosengren et al<sup>118</sup> (2004) and Fong et al<sup>57</sup> (2008) have performed similar smaller studies on Australian populations. Ono et al<sup>137</sup> (2007), Tureli et al<sup>81</sup> (2008) and Saboori et al<sup>80</sup> (2007) have published recent studies on Japanese, Turkish and Iranian populations. Kupperman et al<sup>85</sup> (2009) and Dunning et al<sup>138</sup> (2006) have recently published large studies in the paediatric population in which they identified clinical risk factors and developed clinical decision rules that are similar to those developed for adults. There is now a large body of evidence suggesting that mild head injury patients can be risk stratified based on clinical assessment. The clinical risk factors that indicate patients are at increased risk of intracranial injury are discussed in the following text, and most of the relevant studies summarised in the evidentiary tables.

Mild head injury patients can be risk stratified into “low” or “high” risk groups based on the presence or absence of identified clinical risk factors. The absence of clinical risk factors on initial assessment combined with a period of observation during which the patient clinically improves makes the probability of a significant intracranial injury extremely unlikely. These low risk mild head injury patients can be discharged for home observation without CT scanning. Among the patients with identified risk factors, there are those who clearly require CT scanning and those in whom clinical judgement may be required. In the high risk group, which includes initially low risk patients who

fail to clinically improve, both CT scanning and prolonged clinical observation are indicated. In the patients where clinical judgement is required this should be based on the clinician’s experience, the number of identified risk factors and clinical observation of the patient.

Early CT scanning allows identification of acute extradural or subdural haematomas or depressed skull fractures requiring neurosurgical intervention and other structural lesions such as intracerebral haematomas or minor skull fractures that put the patient at increased risk of deterioration or complications such as post traumatic seizures, and disabling post concussive symptoms. However, it is important to recognise that the absence of a structural lesion on CT scan does not exclude the possibility of deterioration, post traumatic seizures or significant post concussion symptoms.

Patients with an abnormal CT scan should be discussed with a neurosurgical service and considered for prolonged observation in hospital. Conversely, a normal CT scan makes acute clinical deterioration highly unlikely and allows safe discharge for home observation as long as the patient is clinically improving and does not have a known coagulopathy. A normal CT scan is useful to rule out structural lesions but does not exclude the possibility of significant post concussion symptoms. It is important to stress that CT scanning should be used as a clinical tool in conjunction with clinical assessment and observation as part of an overall management strategy for mild head injury patterns.

### **Individual factors predicting risk of intracranial injury and therefore the need for CT scanning in patients with mild head injury:**

#### ***Initial GCS***

A persistent GCS <15 at two hours post injury is a strong indication for CT scanning. An initial GCS 14 on admission is a relative indication for CT scanning.<sup>31, 33, 35, 36, 47, 48, 50-53, 56, 58, 62, 70, 74, 139</sup>

Several studies have noted the heterogeneity of the original GCS 13-15 mild head injury classification and these findings are summarised in Appendix 1. Patients with an initial GCS 13 have been shown to have similar rates of intracranial injury to those with initial GCS 9-12.<sup>53, 105</sup> Recent studies on mild head injury that published data on patients with



GCS 13 have confirmed that an initial GCS 13 has a much higher rate of intracranial injury.<sup>35, 47, 56</sup> Many other recent studies have used the definition of mild head injury as GCS 14 or 15 on arrival and excluded patients with an initial GCS 13. Although patients with an initial GCS 14-15 have lower rates of intracranial injury as a group, the more recent studies have confirmed a higher risk of intracranial injury for an initial GCS 14.<sup>31, 32, 35, 47, 48, 56, 58</sup> A number of the recent, well designed studies have attempted to identify risk factors which can predict which patients with an initial GCS 14-15 are at highest risk.<sup>31, 32, 35, 47, 48, 56, 58</sup> This evidence confirms that other risk factors can be used to successfully stratify risk within the initial GCS 14-15 group.

One strategy is to relate the significance of the GCS to the time of injury. In most of the studies on GCS 14-15, the reported higher risk of intracranial injury for an initial GCS 14 does not take into account the time of injury. Recent studies that have reported time of presentation (Fabbri et al,<sup>31</sup> Smits et al<sup>54</sup> and Styrke et al<sup>43</sup>) have shown that mild head injuries present around 60 to 90 minutes post injury. An abnormal initial GCS taken within one hour of injury is therefore likely to overestimate the risk of intracranial injury in mild head injury patients who present early. One of the most relevant findings to clinical practice is that of Stiell et al<sup>35</sup> who showed that for patients presenting with an initial GCS 13-15 that GCS <15 at two hours post injury was a useful predictor of risk of intracranial injury. By applying this criteria, both time of injury and GCS are usefully combined in a clinically practical assessment tool. Using GCS <15 at two hours post injury allows for clinical judgement regarding patients who present immediately following injury or with drug or alcohol ingestion, allows for lack of inter observer reliability and stresses the importance of relating persistently abnormal GCS to time of injury. It also emphasises the significance of a patient presenting with a GCS of less than 15 or other signs of abnormal mental status at more than two hours after injury.

### ***Focal neurological deficits***

Focal neurological deficit is a strong indication for CT scanning.<sup>31-35, 47, 48, 53, 58, 62, 68-70, 139-141</sup>

All the major clinical decision rules use neurological deficit as an indication for CT scan or list it as an exclusion criteria. Focal neurological deficits have been shown to significantly increase the risk of intracranial injury.<sup>31, 34, 47-49, 53, 58</sup> Both Haydel et al<sup>32</sup> and Stiell et al<sup>35, 56</sup> excluded focal neurological deficits in their studies due to the previously proven nature of risk. Conversely, Vilke et al<sup>75</sup> showed that a normal neurological examination does not rule out underlying brain injury in mild head injuries.

### ***Skull fractures***

Clinical suspicion or evidence of skull fracture is a strong indication for CT scanning.<sup>32-35, 47-49, 56, 58, 62, 66, 68, 70, 71, 74, 88, 140</sup>

Clinical suspicion or evidence of skull fracture has been shown to be a significant risk factor for intracranial injury. The meta analyses by Hofman et al<sup>66</sup> showed that the x-ray presence of skull fracture had a specificity of 95% for intracranial injury. Clinical suspicion or evidence of skull fracture has been shown by several authors including Stiell et al,<sup>35</sup> Haydel et al<sup>32</sup> and Palchek et al<sup>34</sup> to be a major risk factor for the presence of intracranial injury. This has been supported in subsequent studies by Stiell et al,<sup>56</sup> Mower et al,<sup>58</sup> Smits et al<sup>47</sup> and Stein et al.<sup>48</sup> Clinical suspicion of open, depressed or base of skull fractures is based on the presence of large scalp lacerations or haematomas (especially in children <2 years), obvious skull depression, and base of skull signs such as raccoon eyes, haemotympanum, Battles sign, or CSF leak. Skull fracture should also be suspected on the basis of the mechanism of injury with a significant focal blunt force to the skull such as a bat, ball, bar, boot or club. The presence of significant facial fractures may also indicate the possibility of skull fracture.

### ***Loss of consciousness***

The absence of loss of consciousness does not rule out intracranial injury. Brief loss of consciousness (<5 minutes) slightly increases risk of intracranial injury but should not be considered a routine indication for CT scan in the absence of other risk factors. Prolonged loss of consciousness (>5 minutes) should be considered a strong indication for CT scanning.<sup>1, 6, 9, 31-34, 47, 54, 57-59, 61, 63, 65, 67, 72-74, 141</sup>



Loss of consciousness increases the risk of intracranial injury and is used by many authorities to define mild head injury (Evidence Table 1). However, the absolute risk associated with loss of consciousness is small.<sup>6, 74</sup>

The absence of loss of consciousness or amnesia has been used to classify patients as 'minimal risk'.<sup>6, 8-10</sup> However, more recent studies have shown that the absence of loss of consciousness does not rule out intracranial lesions in either adults or children.<sup>9, 33, 34, 47, 54, 57-59, 65, 85</sup> Similarly, adult studies have shown that transient LOC does not accurately predict the risk of intracranial injury.<sup>33, 61, 67, 73, 141, 142</sup>

Duration of loss of consciousness is also controversial. 'Brief' loss of consciousness in mild head injury patients is usually associated with good functional outcome while a specific time for 'prolonged' loss of consciousness is not clearly associated with poorer outcome.<sup>10, 63, 72, 74</sup> The exact definitions of what should be considered a low-risk duration vary greatly from momentary to five minutes, to 20 minutes to 30 minutes.<sup>6, 8-10</sup> In the NEXUS II study, prolonged loss of consciousness > 5 minutes was found to increase risk of intracranial injury but was not a sufficiently useful discriminator to include in their clinical decision rule derivation.<sup>58</sup> The clinical recommendation of the neurosurgical committee of the Royal Australasian College of Surgeons (RACS) is that loss of consciousness should be considered brief if less than five minutes.<sup>1</sup>

From a practical viewpoint, obtaining a definite history and duration of loss of consciousness is often difficult.<sup>47, 143</sup> In a recent review, Ruff et al<sup>143</sup> detailed the difficulties in getting a reliable history of loss of consciousness. Head injuries are frequently unwitnessed, observers unreliable and patients often affected by alcohol or post traumatic amnesia. It is perhaps simplest to consider loss of consciousness in terms of no loss of consciousness, brief loss of consciousness less than five minutes or prolonged loss of consciousness greater than five minutes. Most patients with a witnessed prolonged loss of consciousness are likely to have other indications for CT scan but in the unlikely event that none were present then a CT scan should be performed.

### ***Post traumatic amnesia***

Persistent post traumatic amnesia is a strong indication for CT scanning. Amnesia for the event does not warrant CT scanning.<sup>31, 32, 35, 47, 56, 74</sup>

Several studies have noted that prolonged anterograde or retrograde post traumatic amnesia are risk factors for intracranial injury and the recent study by Smits et al<sup>47</sup> identified persistent post traumatic amnesia (PTA) as a significant independent risk factor. Amnesia for the event implies transient neurological dysfunction and indicates mildly increased risk of intracranial injury although the absolute risk remains small.<sup>74</sup> Anterograde amnesia (typically defined as the period of loss of short term memory for events following the head injury) and post traumatic amnesia (typically defined as the period of inability to lay down new memories following a head injury) are essentially a continuum. A prolonged duration of both anterograde amnesia and post traumatic amnesia have both been shown to be associated with risk of intracranial injury.<sup>32, 35, 47, 56</sup> Haydel et al<sup>32</sup> identified short term memory deficit as a significant risk factor for intracranial injury. Stiell et al<sup>35</sup> also identified anterograde amnesia of more than 30 minutes as a risk factor but did not include it in their clinical decision rule. Mower et al<sup>58</sup> found that abnormal alertness, in which they included short term memory deficits and perseverating speech, was a significant risk factor for intracranial injury. Retrograde amnesia (defined as the period of loss of short term memory for events prior to the head injury) has been shown by Stiell et al<sup>35</sup> to be of significance if greater than 30 minutes duration.

Post traumatic amnesia that persists for more than 24 hours has been shown to be a significant risk factor for persistent cognitive-behavioural-social dysfunction and is a clinical indicator of moderate traumatic brain injury.<sup>3</sup> Recent Australian studies have looked at using specific bedside tools in the emergency department to better identify patients with persistent PTA so that these patients can be more closely followed up as they are more likely to suffer significant post concussion symptoms.<sup>3, 106-108</sup> From a practical point of view, all patients with mild head injury should be assessed for post traumatic amnesia. As Ruff et al<sup>143</sup> pointed out, it may be difficult to establish an accurate assessment of the period of PTA due to the patient being told what happened by others, the influence of drugs or alcohol or psychological stress and the limitations of clinical assessment. It is useful to assess the patient's recall of events following their injury by asking specific questions such as what is their first clear memory, who helped them at the scene and how they got to hospital. This can be used to estimate the period of post traumatic amnesia. Clinicians should also look for symptoms of post traumatic amnesia such as repetitive questioning, failure to remember clinical staff and inability to remember events

during their hospital stay. A formal assessment tool like the A-WPTAS (see Appendix 4) or a simple memory assessment technique such as three object recall can be used as an objective bedside screening test for post traumatic amnesia, to supplement the 'history' of post traumatic amnesia for events. Patients who have evidence of prolonged post traumatic amnesia and particularly those who have persistent post traumatic amnesia at four hours post injury should be considered for CT scanning.

## **Post traumatic seizure**

Post traumatic seizures have not been shown to be a major risk factor for intracranial injury but clinical considerations make them a strong indication for CT scanning.<sup>74, 133, 162</sup>

Post traumatic seizures are normally classified as immediate, early (<7days) or delayed (>7days).<sup>144</sup> Brief generalised post traumatic seizures immediately following mild head injury are relatively common and are not usually associated with poor outcome. They are frequently seen on sporting fields and in young children. It has been proposed that these immediate seizures be called "concussive convulsions" and it has been suggested that they are not an epileptic phenomena.<sup>64, 107, 134</sup> McCrory et al<sup>145</sup> demonstrated that outcome was universally good for these "concussive convulsions" in elite Australian sportsmen. Prolonged or focal post traumatic seizures are more likely to be associated with significant intracranial injury. Some patients who have seizures associated with trauma may have pre-existing epilepsy which may either have caused the seizure or resulted in a lower seizure threshold.<sup>146</sup> However, most mild head injury studies do not differentiate between types of seizures when assessing risk factors for intracranial injury.<sup>74, 132</sup> The literature is somewhat contradictory about the risk of seizures being associated with intracranial injury. Many of the larger studies found that post traumatic seizures were not significantly associated with intracranial injury.<sup>85, 115, 168, 172, 174</sup> Haydel et al,<sup>32</sup> however, found that they were significant and Smits et al<sup>47</sup> found that they were not statistically significant but included them in their decision rule because of their perceived clinical importance. Neidlinger et al<sup>146</sup> found that the "yield of unsuspected major intracranial abnormality on CT scan justifies a policy of its routine use in trauma patients with seizure or who are post ictal, regardless of prior seizure history."

From a practical viewpoint, any patient who has a definite

pre or post traumatic seizure witnessed by a reliable observer probably warrants a CT scan to exclude significant underlying pathology even though the yield is likely to be low. A patient who has an early post traumatic seizure while in the emergency department would be considered to have deteriorated and would warrant a CT scan. Patients who have early post traumatic seizures with structural lesions on CT scan are at increased risk of further seizures and developing post traumatic epilepsy and need to be considered for prophylactic anti-convulsants and given appropriate lifestyle advice.

## **Vomiting**

Persistent or recurrent vomiting is a strong indication for CT scanning. Any vomiting is a relative indication for CT scanning.<sup>38, 49, 53, 60, 74, 85, 94, 97, 108, 109, 115, 131, 133, 150, 162, 168, 172, 174, 184</sup>

Vomiting has been identified as a significant risk factor for intracranial injury in many studies.<sup>38, 49, 60, 74, 94, 97, 108, 115, 133, 162, 168, 172, 174</sup> There has been some debate whether persistent vomiting is more relevant than isolated vomiting. Stiell et al<sup>35</sup> identified repeated vomiting (more than one occasion) as being a significant risk factor for intracranial injury. All the major adult clinical decision rules have either vomiting or recurrent vomiting as a major risk factor for intracranial injury. In the paediatric literature recurrent vomiting is also considered a significant risk factor although it is noted that isolated vomiting is more common in younger children. Clement et al<sup>77</sup> identified any vomiting as a significant risk factor for neurosurgical intervention in patients with initial GCS 15.

## **Headache**

Persistent severe headache is a strong indication for CT scanning.<sup>38, 53, 74, 85, 97, 108, 109, 115, 133, 168</sup>

The literature is somewhat contradictory about the significance of headache. Mild headache is a common symptom of mild head injury but severe headache appears to be a significant risk factor for intracranial injury. Many studies have identified either headache or more commonly severe headache as a significant risk factor for intracranial injury.<sup>38, 74, 85, 97, 108, 133, 168</sup> However, Stiell et al<sup>35</sup> and Smits et al<sup>47</sup> both found that headache was not a good discriminator compared to other risk factors and did not

include it in their clinical decision rules. Mower et al<sup>58</sup> found that severe headache was a risk factor but did not include it in their clinical decision rule. Interestingly, in a later study, Clement et al<sup>77</sup> found that severe headache was a significant risk factor for those patients with GCS 15 who required neurosurgical intervention.

The general trend of the literature would suggest that mild headache is not a significant concern but persistent severe headache should be considered a significant risk factor for intracranial injury.

### ***Coagulopathy or bleeding disorder***

Known coagulopathy or bleeding disorder is both a strong indication for early CT scan and also an indication to check the INR and to consider reversal of anticoagulation. Anticoagulated patients with any evidence of haemorrhage on CT scan should have early rapid reversal of anticoagulation. Patients with a supra-therapeutic INR (>4) should be considered for either partial or full reversal and admitted to hospital for prolonged observation. Prolonged observation and follow up repeat CT scan should be considered for any anticoagulated patients or patients with bleeding disorders.<sup>39, 42, 53, 54, 63, 74, 85-87, 110, 115, 153, 162, 172, 174, 187</sup>

Most guidelines and studies on mild head injury mention coagulopathy or bleeding disorder either as a significant risk factor or an exclusion criteria in the assessment of risk of intracranial injury.<sup>42, 53, 74, 85, 87, 115, 153, 162, 172, 174, 187</sup> However, there was no compelling evidence to either support or refute this reasonable assertion in the mild head injury patient group until recently. Mina et al<sup>147</sup> demonstrated that pre-existing anticoagulation significantly increased the risk of death from intracranial injury in trauma patients with head injury. However, this was a heterogeneous patient group with significantly abnormal ISS (mean 17.0 +/- 7.8) and GCS (mean 11.8 +/- 4.0). Subsequent small studies by Franko et al,<sup>78</sup> Ivascu et al,<sup>79</sup> Cohen et al,<sup>148</sup> Fabbri et al<sup>41</sup> and Allard et al<sup>149</sup> have all demonstrated significant risk of both intracranial haemorrhage and mortality in anti-coagulated head injury patients. Cohen et al<sup>148</sup> and Ivascu et al<sup>79</sup> both found that patients with mild head injury were at significant risk of intracranial injury and acute deterioration particularly if they had an increased INR. An initial normal CT scan did not exclude the possibility of deterioration. Franko et al<sup>78</sup>

also demonstrated a link between increased mortality and increased INR (>4). Ivascu et al<sup>79</sup> demonstrated that early rapid reversal of warfarin in patients with intracranial haemorrhage significantly improved mortality.

**Mild head injury patients who are warfarinised are at significantly increased risk of traumatic intracranial haemorrhage particularly if they are elderly or over-warfarinised.** Note that this increased risk applies to asymptomatic patients. They should all receive an urgent CT scan and have an early INR checked. Patients who have a traumatic injury on CT scan or who have a supratherapeutic INR (>4) should be admitted for observation and should be strongly considered for short term reversal of their anti-coagulation as they are at high risk of acute deterioration and death. A routine repeat CT scan within 24 hours or an urgent repeat CT scan if there are any signs of deterioration is recommended for these patients. Clinical judgement is required about the disposition of patients with an initial normal CT scan and normal INR. Prolonged observation and close follow up either in hospital or in the community is reasonable until further evidence is available to guide management.

The evidence is less clear about the risk of traumatic ICH associated with anti-platelet agents or bleeding disorders. There are very few studies specifically addressing the issue and those that have been done provide contradictory findings.<sup>9, 89, 129, 164</sup> There is a clear trend to suggest patients on anti-platelets have an increased risk of bleeding following intracranial haemorrhage but limited evidence to prove that anti-platelets independently increase the risk of intracranial haemorrhage for mild head injury patients. Extrapolating from existing anti-coagulation protocols, population studies on stroke and other studies on mild head injury, it would be reasonable to postulate that increasing age and the presence of more than one anti-platelet agent would increase the risk of bleeding. Since most patients on anti-platelet agents are elderly and elderly patients are recommended to have routine CT scans, the remaining clinical dilemma is what is the risk of delayed bleeding. At present this remains unknown and prolonged observation and close follow up in the community is probably prudent.

### ***Age***

Patient age >65 years is a strong indication for CT scanning.<sup>20, 38, 47, 53, 60, 74, 85, 88, 115, 153, 162, 172, 174</sup>

The literature has shown that there is an increased risk of intracranial injury for patients aged over 60-65 years with mild head injury.<sup>20, 38, 47, 53, 60, 74, 85, 88, 101, 115, 162, 172, 174</sup> All the recently published clinical decision rules have included age over 60 or 65 years as a major risk factor. Mack et al<sup>150</sup> recommended routine head CT for elderly patients suffering mild head injury as they could not identify any useful clinical predictors of intracranial injury in the elderly. Similarly, Rathlev et al<sup>151</sup> analysed the elderly patients from the NEXUS II trial and found that there was an increased risk of intracranial risk with age and that occult presentation was more common. The Brain Trauma Foundation<sup>15</sup> concluded that 'increasing age is a strong independent factor in prognosis, with significant increase in poor outcome above 60 years of age' for patients with severe head injuries. Similarly, Williams et al<sup>76</sup> demonstrated that elderly patients were more likely to sustain complications of mild head injury. Servadei et al<sup>6</sup> have pointed out it is unlikely there is a specific age at which risk of intracranial injury dramatically increases. Fabbri et al<sup>31</sup> found that using age >60 years alone to predict the need for CT scanning in patients with mild head injury was impractical from a cost-resource consideration during a study to validate a set of guidelines. Interestingly, of the 705 patients meeting guidelines criteria for CT scanning based on age >60 years alone who did not have CT scans, Fabbri et al<sup>31</sup> found that only one patient deteriorated within 48 hours. It is worth noting that the NSW Institute of Trauma and Injury Management Trauma Death Review Committee has identified that in 2003 / 2004 elderly NSW patients with head injuries represented a significant number of potentially preventable deaths.<sup>152</sup>

Patients aged >65 with a mild head injury should have a CT scan due to the increased risk of intracranial injury. If CT scan is not available and the patient has no other identified risk factors then the absolute risk is probably small and clinical judgement can be used to justify prolonged observation rather than transfer for CT scan. Prolonged observation in hospital or at home should be considered even if an initial CT scan is normal due to the increased risk of delayed complications.

### ***Abnormal alertness, behaviour or cognition***

Persistent abnormal mental status manifested by abnormal alertness, abnormal behaviour or cognitive impairment is a strong indication for CT scanning.<sup>38, 49, 55, 58, 74, 94, 97, 103, 115, 133, 174</sup>

Persistent abnormal mental status manifested by abnormal

alertness, abnormal behaviour or cognitive impairment has been shown to be strongly associated with intracranial injury.<sup>38, 74, 115, 174</sup> Mower et al<sup>58</sup> used abnormal alertness or behaviour as major criteria in their clinical decision rule and Clement et al<sup>77</sup> found confusion, restlessness and abnormal 3 object recall as significant risks. Similarly, in the paediatric literature, abnormal mental status has been identified as a major risk factor.<sup>49, 74, 94</sup> Although initial GCS partially assesses mental status, it primarily addresses level of consciousness. Clinical observation of alertness, behaviour and cognition will detect more subtle changes in mental status than GCS and should be part of routine neurological observation. Persistent abnormal PTA testing is also an indication of abnormal mental status. Detection of abnormal alertness or behaviour and cognitive impairment requires careful observation by staff and relatives and is particularly important for adults with pre-existing neurological impairment. In patients with pre-existing neurological conditions, a lower threshold for CT scanning is appropriate.<sup>6, 33</sup>

Most studies do not identify at what time post injury that abnormal mental status becomes a definite indication for CT scanning. Extrapolating from the GCS <15 at two hours post injury risk factor, it would be reasonable to identify abnormal mental status as a risk factor at two hours post injury and certainly by four hours post injury. Clinical judgement is required depending on the degree of abnormality as to when to order a CT scan.

### ***Large scalp haematoma or laceration***

Large scalp haematomas or lacerations are relative indications for CT scanning in adults. Large non frontal scalp lacerations have been identified as significant risk factors in young children.<sup>14, 49, 74, 94, 115, 123, 130, 162, 174</sup>

The literature is somewhat unclear about the importance of scalp haematomas and lacerations as clinical risk factors. Haydel et al<sup>32</sup> identified "visible trauma above the clavicles" as a major risk factor. Mower et al<sup>58</sup> identified scalp haematoma as a major risk factor and included it in their clinical decision rule. Smits et al<sup>47</sup> included "contusion to the skull" as a minor risk factor in their CHIP rule. In the other major adult clinical decision rules and studies, scalp haematoma was not identified as a major risk factor but was potentially implied under the broad term "suspected skull fracture." In the paediatric literature, non frontal

scalp haematoma is included in most clinical decision rules particularly among young children.<sup>14, 49, 94, 123, 130, 174</sup> There is no clear guidance in the literature as to what should be considered a significant scalp haematoma or laceration other than a trend to “large” and “non frontal”. There is no evidence for the use of descriptors such as “boggy” or “tense” to help discriminate between significant and non significant haematomas.

### ***Drug or alcohol intoxication***

Drug or alcohol ingestion with a normal mental state is not an indication for CT scanning. Drug or alcohol intoxication resulting in an abnormal mental state is an indication for CT scanning.<sup>22, 38, 40, 53, 74, 85, 115, 162, 168, 172, 174</sup>

Drug or alcohol intoxication is frequently present in patients with head injury and makes patients difficult to assess and manage. Cook et al,<sup>60</sup> in a study of alcohol intoxicated patients, found that clinical examination could not predict which alcohol intoxicated patients had abnormal CT scans. However, they observed that the rate of abnormal CT scan and neurosurgical intervention was similar to that of the non-intoxicated mild head injury population. Several studies have suggested that drug or alcohol intoxication is a risk factor for intracranial injury but the exact definition of intoxication remains vague.<sup>31, 32</sup>

Since 2004 there have been several large well designed studies and clinical decision rules published that have found that alcohol is not an independent risk factor for intracranial injury in patients with mild head injury.<sup>22, 38, 85, 115, 162, 168, 172</sup> Bracken et al<sup>153</sup> looked at the intoxicated patients (3356 patients) in the NEXUS II database who had received CT scans and found that the rate of intracranial injury was lower than the non-intoxicated group. Although it would appear that alcohol intoxication is not an independent risk factor for intracranial injury it remains a common clinical dilemma to decide what to do with a head injured patient with an abnormal mental status when it is not clear whether it is the alcohol or the head injury that is causing the abnormality.

Stiell et al<sup>35</sup> took a slightly different approach and used GCS <15 at two hours post injury as their variable to predict abnormal mental status for whatever reason. They argued that drug or alcohol intoxication was not an independent predictor of intracranial injury if the patient had a normal

mental status at two hours post injury.

From a practical viewpoint, clinically obvious drug or alcohol intoxication should be treated as a risk factor for intracranial injury because it manifests as abnormal mental status which impairs clinical assessment and must be assumed to be due to intracranial injury. It seems reasonable to use the approach of Stiell et al<sup>35</sup> in determining when to order a CT scan in the absence of other risk factors.

### ***Dangerous mechanisms of injury***

Specifically identified high risk dangerous mechanisms that are a strong indication for CT scanning include pedestrian/cyclist struck by vehicle, ejection from vehicle, falls (>1m) and focal blunt trauma to the head (bat, ball, foot). In the absence of these specified risk factors or other risk factors, dangerous mechanism is a relative indication for CT scan in mild head injury patients. Clinical judgment is required.<sup>20, 38, 60, 72, 88, 115, 162, 172, 174</sup>

Epidemiological studies have generally identified motor vehicle accidents, falls and assaults as the commonest causes of head injuries. In studies on patients with mild head injuries, specific high risk factors for intracranial injury that have been identified include focal blunt trauma to the head,<sup>38, 60, 72, 88, 115</sup> pedestrians or cyclists struck by motor vehicles,<sup>20, 60, 88, 172, 174</sup> ejection from motor vehicle,<sup>172, 174</sup> and falls with variable heights specified.<sup>38, 172, 174</sup>

### ***Multi-system trauma***

In mild head injury patients with multi-system trauma, clinical judgement is required regarding the need for CT scanning, particularly in the presence of unstable vital signs or associated injuries requiring significant amounts of analgesia, procedural sedation or general anaesthesia.<sup>162, 172, 174</sup>

Most guidelines and studies on mild head injury have specifically excluded patients with multi-system trauma or unstable vital signs.<sup>162, 172, 174</sup> It is therefore difficult to make evidence based recommendations and clinical judgment is required. A low threshold for performing head CT scan in multi-system trauma patients should be used as subtle neurological signs are easily missed in the presence of distracting injuries. Similarly, clinicians should have a

low threshold for performing CT scans where associated injuries require significant amounts of analgesia, procedural sedation or general anaesthesia.

### ***Pre-existing neurosurgery/neurological impairment***

Pre-existing neurosurgery or neurological impairment is a relative indication for CT scanning.<sup>85, 153, 168</sup>

Pre-existing neurosurgery has been suggested as an indication for CT scanning particularly in the presence of hydrocephalus and shunt placement.<sup>6, 33</sup> Sevadei et al<sup>6</sup> recommended routine CT scanning for patients with either previous neurosurgery or epilepsy. From a practical viewpoint, any pre-existing medical condition resulting in neurological impairment (eg stroke, dementia, and developmental delay) may make clinical assessment difficult. More recent studies have been unable to enrol sufficient numbers of patients to give clear guidance about these risk factors.<sup>48</sup>

### ***Delayed presentation or representation***

Delayed presentation or representation are relative indications for CT scanning. Clinical judgement is required. Patients who present more than four hours post injury with persistent or new clinical symptoms should be regarded as being at relatively high risk for intracranial injury.<sup>4, 6, 44, 53, 114, 119</sup>

Although mild head injuries are very common it is thought that the majority do not present to hospital.<sup>114, 119</sup> Therefore, those that do present to hospital are already a group at slightly increased risk. Of particular concern are those who have a delayed presentation due to persistence of symptoms or those who represent because of ongoing or new symptoms. Most of the larger studies have excluded patients who presented more than 24 hours post injury or who represented, so there is a lack of evidence about these patients. However, it is clear from most of the studies that patient's who have persistent or new abnormal mental status, or persistent or new clinical symptoms such as vomiting or severe headache, have increased risk of intracranial injury.

Delayed presentation due to ongoing symptoms should be regarded as being the same as failing to return to normal after clinical observation. Representation due to persistent

or worsening symptoms should be regarded as the same as clinical deterioration during clinical observation. Therefore, both patients who have a delayed presentation or who represent with new or worsening symptoms should be regarded as being at relatively high risk of intracranial injury.

However, the overall risk of intracranial injury in patients who represent after mild head injuries is low if their initial risk was low and particularly if they had an initial normal CT scan.<sup>4, 6, 44, 53</sup>

### ***Unwitnessed event/unreliable history***

A good history of injury may help predict risk of intracranial injury by identifying dangerous mechanism of injury or significant features such as prolonged loss of consciousness or seizures. This is of particular importance in children. However, in the absence of other significant risk factors there are few studies identifying unwitnessed event or unreliable history as a significant independent risk factor.<sup>140</sup>

### ***Paediatrics***

Although paediatrics were excluded from the search strategy, the author believes that the evidence in the paediatric literature discussed below may assist clinicians in their decision making in the management of adults with closed head injury. The evidence was not identified in an exhaustive systematic literature search but represents the most relevant studies identified by the author.

In the past few years there have been several large studies looking at paediatric head injuries that have attempted to identify clinical risk factors indicating the need for CT scan and use these risk factors to develop clinical decision rules. Clinical decision rules have been developed for children by Palchak et al<sup>34</sup> (2003), Haydel et al<sup>65</sup> (2003 – New Orleans group), Oman et al<sup>87</sup> (2006 – NEXUS group), Dunning et al<sup>138</sup> (2006 – CHALICE group), Atakabi et al<sup>83</sup> (2008 – Canadian CT Head group) and most recently Kupperman et al<sup>85</sup> (2009 – PECARN group). Maguire et al<sup>86</sup> published a systematic view of paediatric clinical prediction rules in 2009 that assessed all the major studies except the Kupperman study. The studies by Dunning et al enrolled 22,772 patients and Kupperman et al enrolled 42,412 patients and these are the two largest well designed studies yet performed on either adult or paediatric patients with head injuries.

The best predictors of the need for CT scan were similar in all the studies and similar to the findings of the adult literature. The best predictors present in nearly all prediction



rules were abnormal mental status or behaviour (including GCS <15 or qualitative assessment), focal neurological deficit, clinically suspected skull fracture (non frontal scalp haematoma), vomiting and dangerous mechanism. Loss of consciousness was significant if “definite” in some and if “prolonged” in others. Severe headache and seizures were identified as significant in several.

Kupperman et al<sup>85</sup> took the clinically practical step of identifying those risk factors which strongly suggested the need for CT scan and differentiating them from those that required clinical judgement to decide whether to observe the child or proceed to CT scan. They made the practical point that where clinical judgement was used it should be based on experience, number of risk factors and a period of observation. In their summary of their clinical decision rule, they included their figures on the percentage of the population likely to be in each group, and the risk of clinically important brain injury in each group. This information could be used to help clinicians and parents to decide how to proceed for an individual child.

In summary, the indications for CT scan and the clinical prediction rules in the paediatric literature are similar to those in the adult literature. There have been large well designed studies that support the use of structured clinical assessment to identify which patients should have a CT scan. Refer to NSW Health Infants & Children: Acute Management of Head Injury, second edition (PD2011\_024) for management of head injury children.

## **What should be done with high risk mild head injury patients when CT scan is unavailable?**

In patients with high risk mild head injury, a normal CT scan combined with clinical assessment will allow the patient to be safely discharged for home observation. If CT scan is unavailable then the patient will require either admission for prolonged observation or early transfer for CT scanning depending on clinical assessment of risk. Prolonged clinical observation for at least 24 hours, associated with clinical improvement, has been shown to make a significant injury unlikely in the majority of mild head injury patients.<sup>91</sup> However, those patients at highest risk for an intracranial injury identified by persistently abnormal GCS or clinical symptoms, deterioration in GCS, focal neurological deficit, or significant clinical suspicion of skull fracture should be transferred for CT scan to allow the early identification of potentially neurosurgically significant injury.

It is reasonable to admit some mild head injury patients to hospital for serial observation if CT scan is unavailable and urgent transfer to neurosurgical care impractical. Prior to the widespread availability of CT scanning, admission for serial assessment of GCS was the standard treatment for patients with head injury. Borg et al<sup>101</sup> (2004 diagnostic procedures), in a meta analysis for the World Health Organisation on mild traumatic brain injury, concluded that in the absence of CT scanning, that hospital observation for at least 24 hours for patients with GCS 15 and other risk factors was a reasonable strategy. Similarly, af Geijerstam et al<sup>91</sup> found in a large population based study on mild head injury patients with GCS 15 that the outcome for a serial observation in hospital strategy was similar to the outcome for an immediate CT strategy at 3 month follow up. This presumed that appropriate care could be delivered in the event of deterioration. Clinical judgement is clearly required where patients have GCS 15 but clinical symptoms that fail to improve with observation. The primary advantage of performing an early CT scan is to be able to safely discharge a patient if the CT scan is normal. Thus, when CT is unavailable, serial clinical observation is a reasonable strategy for mild head injury patients with GCS 15 as long as patients can be transferred to neurosurgical care in a timely fashion in case of deterioration.

## 5. What should be done when patients with mild head injury deteriorate?

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| <p>Early signs of deterioration:</p> <ul style="list-style-type: none"> <li>■ Confusion</li> <li>■ Agitation</li> <li>■ Drowsiness</li> <li>■ Vomiting</li> <li>■ Severe headache</li> </ul>  | B                          |
| <p>Late signs of deterioration:</p> <ul style="list-style-type: none"> <li>■ Decrease in GCS by two or more points</li> <li>■ Dilated pupil</li> <li>■ Focal neurological deficit</li> <li>■ Seizure</li> <li>■ Cushing's response – bradycardia and hypertension</li> </ul>  | A                          |
| <p>Clinical approach to neurological deterioration:</p> <ul style="list-style-type: none"> <li>■ Resuscitation and stabilisation of ABCDEs to exclude non head injury cause</li> <li>■ Supportive care of ABCDEs</li> <li>■ Early intubation if indicated</li> <li>■ Immediate CT scan if available</li> <li>■ Early neurosurgical consult</li> <li>■ Early retrieval consult</li> <li>■ If clinical or CT evidence of raised ICP/mass effect consider in consultation with network neurosurgical service: <ul style="list-style-type: none"> <li>- short term hyperventilation to PaCO<sub>2</sub> 30-35</li> <li>- bolus of mannitol (1g/kg)</li> <li>- surgical decompression if more than 2 hours from neurosurgical care</li> <li>- prophylactic anti-convulsants</li> </ul> </li> </ul> | B                          |

Acute neurological deterioration is uncommon in patients with mild head injury. As discussed in Question 3, the recent literature would suggest that acute neurosurgical intervention is required in 1-3% of patients with mild head injury. Patients at highest risk of deterioration can be identified using clinical risk factors and should have a CT scan. It is very uncommon for patients to deteriorate if they have had a normal CT scan. The exception would be elderly patients who are anticoagulated who are at risk of delayed subdural haemorrhage. The other situation in which unexpected deterioration may occur is when an injury present on CT scan has been missed.

The clinical signs vary from early subtle signs of deterioration to more obvious late signs including reduction in GCS or signs of raised intracranial pressure. Clement et al<sup>77</sup> looked at those patients in the Canadian CT Head Rules database with initial GCS 15 who deteriorated and required neurosurgery to try to identify risk factors for deterioration. They found that the development of confusion, agitation,

drowsiness, vomiting or severe headache were potential early signs of deterioration in this group.

Once a mild head injury patient deteriorates then the priorities are exclusion of other injuries, supportive care of the ABCDEs and early CT scan to identify a neurosurgically significant lesion. If a neurosurgically significant lesion is identified, further management should be discussed with a neurosurgical service including measures to reduce intracranial pressure and prevent seizures.

There is good evidence to support the systematic resuscitation of ABCDEs with prevention of hypoxia and hypotension and the early identification and decompression of acute neurosurgical lesions.<sup>25,103</sup> There is some evidence to support short-term hyperventilation and mannitol while awaiting definitive surgery.<sup>2, 23, 102, 144, 188</sup> There is some evidence to support the prophylactic use of anticonvulsants to prevent early seizures.<sup>2, 23, 102, 151</sup>



## 6. When can patients with mild head injury be safely discharged and what discharge advice should be provided?

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| Mild head injury patients can be safely discharged for home observation after an initial period of in-hospital observation if they meet the following clinical, social and discharge advice criteria:   | CONSENSUS                  |
| <b>1. Clinical criteria:</b> <ul style="list-style-type: none"> <li>■ Normal mental status (alertness / behaviour / cognition) with clinically improving minor post concussion symptoms after observation until at least four hours post injury.</li> <li>■ No clinical risk factors indicating the need for CT scanning or normal CT scan if performed due to risk factors being present.</li> <li>■ No clinical indicators for prolonged hospital observation (irrespective of CT scan result) such as: <ul style="list-style-type: none"> <li>- clinical deterioration</li> <li>- persistent abnormal GCS or focal neurological deficit</li> <li>- persistent abnormal mental status</li> <li>- persistent severe clinical symptoms (vomiting / severe headache)</li> <li>- presence of known coagulopathy (clinical judgement required)</li> <li>- persistent drug or alcohol intoxication (clinical judgement required)</li> <li>- presence of multi-system injuries (clinical judgement required)</li> <li>- presence of concurrent medical problems (clinical judgement required)</li> <li>- age &gt;65 (clinical judgement required)</li> </ul> </li> </ul> | A                          |
| <b>2. Social criteria:</b> <ul style="list-style-type: none"> <li>■ Responsible person available to take patient home.</li> <li>■ Responsible person available for home observation.</li> <li>■ Patient able to return easily in case of deterioration.</li> <li>■ Written and verbal discharge advice able to be understood.</li> </ul>  | CONSENSUS                  |
| <b>3. Discharge advice criteria:</b> <ul style="list-style-type: none"> <li>■ Discharge summary for local doctor.</li> <li>■ Written and verbal head injury advice given to patient and nominated responsible person covering: <ul style="list-style-type: none"> <li>- symptoms and signs of acute deterioration</li> <li>- reasons for seeking urgent medical attention</li> <li>- typical post concussion symptoms</li> <li>- reasons for seeking routine follow up.</li> </ul> </li> </ul>  | CONSENSUS                  |
| Written and verbal head injury discharge advice should be given to the patient and a nominated responsible person covering: <ul style="list-style-type: none"> <li>■ symptoms and signs of acute deterioration</li> <li>■ reasons for seeking urgent medical attention</li> <li>■ lifestyle advice to assist recovery</li> <li>■ typical post concussion symptoms</li> <li>■ reasons for seeking further medical follow up.</li> </ul>  | A                          |

## Discharge criteria

Mild head injury patients can be discharged for home observation after initial period of in-hospital observation if they meet clinical, social and discharge advice criteria.<sup>4, 6, 15, 44, 47, 49, 52, 56, 60, 65, 74, 81, 87, 94, 97, 99, 115, 118, 154, 158, 169, 170, 174, 178, 181, 184, 191</sup> Mild head injury patients can be safely discharged from hospital for home observation when the risk of acute deterioration from an underlying intracranial injury is assessed as being low. Safe discharge also requires that the patient has adequate social supports and appropriate advice on when to return to hospital.

The duration of in-hospital observation required will be determined by clinical assessment combined with selective use of imaging. Deterioration following mild head injury may occur due to missed or delayed intracranial haematomas or other complications such as SIADH, post traumatic seizures or severe post concussive symptoms. Although clinical assessment and observation combined with appropriate imaging will identify most at risk patients, the risk of deterioration is never zero. Although uncommon, deterioration may occur even after prolonged periods of observation and / or following normal CT scanning. The challenge of managing mild head injuries is to identify what is reasonable risk and to ensure that the patient is aware of the potential for delayed deterioration.

All the recent clinical decision rules have shown that safe discharge is possible after clinical assessment and/or CT scan. In both a series of meta analyses and a large population study, af Geijerstam et al<sup>3, 4, 6</sup> have shown that the risk of deterioration following mild head injury is low and the risk of deterioration following normal CT scans is very low. De Broussard et al<sup>92</sup> also found that the risk of delayed intracranial complications following mild head injury were very low in a large population study.

The Initial Management of Adult Mild Head Injury algorithm summaries the key points in management relating to safe discharge and some of the significant studies relating to safe discharge are presented in Evidence Table 6. Mild head injury patients should essentially be divided into low and high risk groups based on clinical assessment. Low risk mild head injury patients can be discharged for home observation after a short period of observation in hospital if clinically improving. High risk patients require CT scanning and/or prolonged observation. High risk patients with clinically important abnormalities on CT scan require admission for prolonged observation. High risk patients

with normal CT scanning should also be admitted for prolonged observation unless rapid clinical improvement occurs. In both high and low risk mild head injury patients, potential clinical indications for admission such as intercurrent medical problems and injuries need to be considered. Whatever the period of observation selected, the provision of safe discharge advice and assessment of the patient's social situation is mandatory because occasional cases of deterioration following discharge are unavoidable. An example of a suitable head injury discharge advice sheet is attached at Appendix 6.

## Discharge advice

All patients with mild head injury should be given both verbal and written discharge advice covering signs and symptoms of acute deterioration, when to seek urgent medical attention, lifestyle advice to assist recovery, information about typical post concussion symptoms and reasons for seeking further medical follow up. As with all discharge advice this should be time specific and action specific.

There have been multiple studies that have shown that the risk of acute deterioration following mild head injury is very small particularly if the patient has been assessed as being low risk clinically or has been assessed as high risk and has had a normal CT scan. However, all the authors of major guidelines and clinical prediction rules consistently stress the point that there is a very small risk of deterioration for an individual patient, not to mention the possibility of medical error, which is why all patients should be advised about symptoms and signs of deterioration and when to seek urgent medical attention.

The most important complications of mild head injury to identify are those requiring acute neurosurgical intervention. However, functional deficits resulting in cognitive-behavioural-social sequelae are far more common and may have significant impact on patients and their families. It is important that doctors, patients and their families understand that the absence of a structural lesion on CT scan following a mild head injury does not exclude the possibility of significant cognitive-behavioural-social sequelae. Mild head injury discharge advice should include information about post concussion symptoms including what they are, how to minimise them and when to seek further medical attention if they persist. It is very important that patients are informed about the potential that they will have post concussion symptoms so that they are able

to adjust their lifestyle if required. The best advice to give a patient who suffers from post concussion symptoms is that the symptoms will resolve with time and that they should take a stepwise graded return to sport, work and study. The sports medicine approach to concussion of graded return to play translates well to all mild head injury patients.<sup>153</sup> However, there has been a tendency in the past not to mention post concussion symptoms in discharge advice which may lead to unnecessary distress and confusion for patients and their relatives and hinder their recovery. As Jagoda et al<sup>9</sup> for the American College of Emergency Physicians pointed out, "a glaring omission from most mild TBI discharge instructions is the lack of any mention of the possibility of the patient developing post-concussive symptoms." Holm et al<sup>102</sup> for the WHO Collaborating Taskforce on Mild Traumatic Brain Injury concluded that the only interventions that have been shown to be beneficial for post concussion symptoms are education, reassurance and time. Therefore, it is important to provide education about post concussion symptoms to all mild head injury patients. All patients should be given written advice and advised to see a doctor if they are not feeling better within a few days of injury.

Both Fung et al<sup>155</sup> and Bazarian et al<sup>82</sup> found that most head injury discharge advice sheets in a selection of US emergency departments were either not routinely provided or were difficult to understand or did not include sufficient information. Similarly, Yates et al<sup>156</sup> in a New Zealand study found that a head injury discharge sheet was better understood when written in a simplified form using less complex language.

The mild head injury advice sheet developed for the original version of these guidelines included most of the relevant information suggested by the literature and was well received during the implementation trials and after publication. It has since been further modified by the Motor Accidents Authority (MAA) NSW<sup>103</sup> after community trials to simplify the language and format in line with the recommendations of Fung et al.<sup>155</sup> This updated mild head injury discharge advice sheet is included in this guideline and is available in several languages through the MAA website.

All patients with mild head injury should be advised to follow up with their local doctor if they are not feeling better within a few days. Patients at higher risk of post concussions symptoms should be advised to routinely follow up with their local doctor within two or three days of

discharge from hospital. These patients would include those admitted to hospital for observation, those with minor structural lesions on CT scan, those with significant acute clinical symptoms in the emergency department and those with documented post traumatic amnesia in the emergency department. Elderly patients and those on anticoagulants should also be advised to have routine follow up organised due to the increased risk of complications.

It is important that patients with mild head injury are able to access appropriate follow up from their local doctor following discharge from hospital, particularly if they develop significant post concussive symptoms. The MAA NSW Guidelines for mild traumatic brain injury following closed head injury<sup>103</sup> (2008) were developed to assist prehospital clinicians, emergency department clinicians and general practitioners with the management of patients suffering from persistent brain injury symptoms following closed head injury. The MAA NSW MTBI guideline complements this guideline and provides detailed information and evidence about the recovery and rehabilitation of patients with mild brain injury following closed head injury.

# Moderate head injury

## 7. What are the proven treatments for patients with moderate head injury?

| RECOMMENDATION   | Strength of recommendation |
|--|----------------------------|
| <p><b>Moderate head injury</b></p> <p><b>Standard care:</b></p> <ul style="list-style-type: none"> <li>■ Initial systematic assessment and resuscitation of ABCDEs</li> <li>■ Supportive care of ABCDEs with appropriate attention to positioning (30° head up), basic nursing care and avoidance of hyperventilation or hypoventilation.</li> <li>■ Prevention of secondary brain injury by avoiding hypoxaemia (O2 saturation &lt;90%) and hypotension (systolic BP &lt;90)</li> <li>■ Early CT scan to identify acute neurosurgical lesions</li> <li>■ Period of clinical observation</li> <li>■ Consider intubation in the event of clinical deterioration to facilitate resuscitation of ABCDEs or to facilitate management of agitated patients</li> <li>■ Early neurosurgical consult if not clinically improving and/or abnormal CT scan</li> <li>■ Early retrieval consult if transfer required</li> <li>■ Admit to hospital unless rapid clinical improvement to GCS 15, normal CT scan and absence of other risk factors (as per mild head injury)</li> <li>■ Repeat CT scan at 24 hours if not clinically improving or abnormal initial CT scan</li> <li>■ Routine post traumatic amnesia testing and consider referral to brain injury rehabilitation service.</li> <li>■ If clinical or CT evidence of raised ICP/mass effect consider in consultation with network neurosurgical service: <ul style="list-style-type: none"> <li>- short term hyperventilation to PaCO<sub>2</sub> 30-35</li> <li>- bolus of mannitol (1g/kg)</li> <li>- surgical decompression if more than 2 hours from neurosurgical care</li> <li>- prophylactic anti-convulsants</li> </ul> </li> </ul> <p><b>Outcome:</b></p> <ul style="list-style-type: none"> <li>■ Approximately 80-90% of moderate head injury patients improve and should be managed as complicated mild head injury while 10-20% deteriorate and require management as per severe head injury..</li> <li>■ The majority of patients who suffer moderate head injuries will have some degree of cognitive behavioural social sequelae and should be considered for routine follow up with a brain injury rehabilitation service or a neurologist (see Appendix 7).</li> </ul> | <p><b>B</b></p>            |

The majority of studies in the literature tend to focus on the management of either severe head injuries or mild head injuries. The natural history of patients with moderate head injuries is that they tend to either deteriorate (10-20%) and should then be managed as severe head injuries or improve (80-90%) and can be managed as "complicated" mild head injuries.<sup>40</sup> Patients who present initially with moderate head injuries should all have an early CT scan

and close clinical observation. Patients with moderate head injury have higher rates of intracranial lesions and cognitive behavioural social sequelae. They should be admitted to hospital for at least 24 hours observation unless they rapidly return to normal, have a normal CT scan and absence of other clinical risk factors. All moderate head injury patients should be routinely followed up for evidence of cognitive behavioural social sequelae.

# Severe head injury

## 8. What are the proven treatments for patients with severe head injury?

| RECOMMENDATION   | Strength of recommendation |
|--|----------------------------|
| <p><b>Severe head injury</b></p> <p><b>Standard care:</b></p> <ul style="list-style-type: none"> <li>■ Initial systematic assessment and resuscitation of ABCDEs</li> <li>■ Early intubation</li> <li>■ Supportive care of ABCDEs with appropriate attention to positioning (30° head up), basic nursing care and avoidance of hyperventilation or hypoventilation.</li> <li>■ Prevention of secondary brain injury by avoiding hypoxaemia (O<sub>2</sub> saturation &lt;90%) and hypotension (systolic BP&lt;90)</li> <li>■ Early CT scan to identify acute neurosurgical lesions</li> <li>■ Early neurosurgical consult</li> <li>■ Early retrieval consult if transfer required</li> <li>■ Consider use of anticonvulsants to prevent early post traumatic seizures</li> <li>■ Consider ICP monitoring to guide management of cerebral perfusion pressure.</li> <li>■ Low threshold to repeat CT scan if patient condition changes</li> <li>■ ICU admission</li> <li>■ Routine repeat CT scan at 24 hours</li> <li>■ Brain injury rehabilitation consult</li> <li>■ If clinical or CT evidence of raised ICP/mass effect consider in consultation with network neurosurgical service:               <ul style="list-style-type: none"> <li>- short term hyperventilation to PaCO<sub>2</sub> 30-35</li> <li>- bolus of mannitol (1g/kg)</li> <li>- surgical decompression if more than 2 hours from neurosurgical care</li> <li>- prophylactic anti-convulsants</li> </ul> </li> </ul> <p><b>Minimum supportive care aims:</b></p> <ul style="list-style-type: none"> <li>■ PaO<sub>2</sub> &gt; 60</li> <li>■ SaO<sub>2</sub> &gt; 90</li> <li>■ PaCO<sub>2</sub> 35-40</li> <li>■ Systolic BP &gt; 90</li> <li>■ Head up 30°</li> </ul> <p><b>Poor prognostic indicators:</b></p> <ul style="list-style-type: none"> <li>■ Low GCS (especially motor component).</li> <li>■ Age &gt;60 years (prognosis deteriorates with increasing age).</li> <li>■ Absent pupillary reflexes (after systemic resuscitation).</li> <li>■ Hypotension (systolic BP &lt;90).</li> <li>■ Hypoxaemia (oxygen saturation &lt;90%).</li> </ul> | <p>A</p>                   |

Recent exhaustive reviews by the Brain Trauma Foundation<sup>15</sup> and the Cochrane Review Group<sup>16-30</sup> have looked at the management of severe head injuries. The findings of these detailed reviews are summarised in Evidence Table 4. This guideline summarises the generally accepted initial management steps for severe head injury including those recommended by the current Advanced Trauma Life Support course and the Brain Trauma Foundation.<sup>15, 40</sup> The network neurosurgical service should be consulted about further management of patients with severe head injury as soon as practical after the initial primary survey and resuscitation is completed.

It is important to recognise that for the majority of severe head injury patients the most important aspect of care is systematic resuscitation of the ABCDEs with prevention of secondary brain injury as per current ATLS guidelines.<sup>40</sup> Resuscitation of the ABCDEs with adequate oxygenation and fluid resuscitation and the treatment of other immediately life threatening injuries should be the priority for patients with severe head injury followed by the CT identification of focal intracranial lesions requiring acute neurosurgical intervention.<sup>40, 157</sup> Early intubation to prevent hypoxaemia and facilitate management is recommended.<sup>40</sup> Hyperventilation should be avoided and patients should normally be ventilated to maintain normocarbia.<sup>24, 158</sup> Fluid resuscitation with normal saline or Hartmanns followed by blood products to maintain normovolaemia and mean arterial pressure is recommended.<sup>157</sup> Anticonvulsants are usually recommended to prevent early post traumatic seizures particularly if there is an abnormal CT scan or a history of a witnessed seizure.<sup>17, 29, 151</sup> Blood glucose and temperature should be monitored and maintained in a normal range.<sup>159, 160</sup> Thus, good supportive care of the ABCDEs, with the prevention of hypoxaemia and hypotension, remain the cornerstone of initial management.<sup>40</sup>

The network neurological service should be urgently consulted about any patient with an acute neurological deterioration suggestive of acutely raised intracranial pressure. In the event of acute deterioration, it is important to remember that hyperventilation<sup>24, 158</sup> and intravenous mannitol boluses<sup>161,162</sup> are short-term measures to reduce intracranial pressure whilst the patient is urgently assessed for the need for acute neurosurgical intervention. If an acutely deteriorating patient with a proven extradural or subdural haematoma cannot be transferred to a neurosurgical service within two hours, then the option of local surgical decompression should be discussed with the neurosurgical service.

If the referring clinician is unsure about the need for a particular therapy, such as prophylactic anticonvulsants or antibiotics, then the network neurosurgical service should be consulted. There are many other promising areas of treatment such as induced hypothermia<sup>159, 160</sup> and hypertonic saline.<sup>161</sup> However, there is currently insufficient evidence to recommend these as first line therapy. Corticosteroids have been shown to worsen the patient outcome and are not recommended for the initial management of closed head injury.<sup>163, 164</sup>

Detailed evaluation of subsequent management of severe head injuries by the neurosurgical services are beyond the scope of these guidelines. There is limited high quality evidence to guide management and the relative merits of different strategies are hotly debated in the literature. The Brain Trauma Foundation review, the Cochrane Group reviews, the BMJ Clinical Evidence review<sup>165</sup> and recent Australian reviews<sup>2, 117</sup> all agree that there is no class 1 evidence to guide management of severe head injury patients. However, there is broad consensus agreement that strategies to control cerebral perfusion using ICP monitoring,<sup>27, 28, 61</sup> mild hypothermia<sup>26, 66, 135</sup> and decompressive craniectomy<sup>166</sup> are promising strategies that are being widely used and require further study.

Predicting outcome following closed head injury is difficult. There have been many attempts to provide scoring systems to predict outcome with one of the best known being that provided by the CRASH Investigators<sup>167</sup>. It is perhaps prudent to err on the side of caution in the initial management setting as at least 24-48 hours of investigation and management are required before offering any sort of prognosis.

# Transfer to neurosurgical facility

## 9. When should patients with closed head injury be transferred to hospitals with neurosurgical facilities?

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| <p>A clear decision about the potential need for transfer should be made at the time of initial assessment or after a brief period of observation. A <b>senior clinician</b> should be consulted.</p> <p>The network neurosurgical and retrieval services should be consulted as soon as possible to facilitate early transfer. The following patients should be considered for transfer and discussed with the network neurosurgical service.</p>  | CONSENSUS                  |
| <b>All patients with severe head injury (GCS 3-8)</b>   | A                          |
| <p><b>Patients with moderate head injury (GCS 9-13) if:</b></p> <ul style="list-style-type: none"> <li>■ clinical deterioration</li> <li>■ abnormal CT scan</li> <li>■ normal CT scan but not clinically improving</li> <li>■ CT scan unavailable.</li> </ul>   | CONSENSUS                  |
| <p><b>Patients with mild head injury (GCS 14-15) if:</b></p> <ul style="list-style-type: none"> <li>■ clinical deterioration</li> <li>■ abnormal CT scan</li> <li>■ normal CT scan but not clinically improving at 4-6 hours post injury</li> <li>■ high risk mild head injury with CT scan unavailable if:                             <ul style="list-style-type: none"> <li>- Persistent GCS&lt;15 at two hours post injury</li> <li>- Focal neurological deficit</li> <li>- Clinical suspicion of skull fracture</li> <li>- Persistent abnormal mental status</li> <li>- Persistent vomiting</li> <li>- Persistent severe headache</li> <li>- Any deterioration in GCS</li> <li>- Post traumatic seizure in ED</li> <li>- Known coagulopathy (particularly if age &gt;65 or INR &gt;4)</li> </ul> </li> </ul> | CONSENSUS                  |
| <p>Note – the Ambulance Service of NSW Pre Hospital Major Trauma Triage Protocol (T1), attempts to ensure that, wherever possible, trauma patients with moderate to severe head injury are transferred directly from the pre-hospital setting to a Tertiary Trauma Centre.</p>  |                            |

Patients with closed head injury should be observed in facilities that can manage any complications that are likely to arise. Clinical judgment regarding risk of deterioration is required and early neurosurgical consultation is advisable.

Patients with closed head injury assessed at hospitals without CT scanning facilities should be transferred to the nearest appropriate hospital if there is significant risk of intracranial injury. Transfer of patients to a hospital with CT scanning facilities but without neurosurgical services should be avoided wherever possible.

Fabbri et al<sup>41</sup> recently published a study in which they compared the outcome for mild to moderate head injury patients and initial non-neurosurgical lesions on CT scan when managed in neurosurgical units versus peripheral hospitals. Their system allowed for rapid transfer of patients to the tertiary centre in the event of deterioration and used a teleradiology link to review CT scans. The outcome for patients was not shown to be significantly different. Huynh et al<sup>168</sup> also showed in a retrospective study that trauma patients with GCS 15 and an abnormal CT scan could be safely managed without neurosurgical consultation. Both these studies would support the current NSW practice of managing some head injury patients in non-neurosurgical centres following consultation with a network neurosurgical centre.

The current guideline aims to address the question of when to transfer patients from a non neurosurgical facility to a neurosurgical facility. However, there has been increasing interest in the issue of pre-hospital bypass of trauma patients to ensure that they go directly to a tertiary hospital with neurosurgical facilities. The primary benefit proposed by this strategy is to avoid undue delay in transfer to definitive care. There have been many studies such as the one by Hartl et al<sup>169</sup> that have shown reduced mortality using such strategies. The Ambulance Service of NSW Pre Hospital Major Trauma Triage Protocol (T1)<sup>170</sup> has adopted such a pre-hospital strategy for transferring all major trauma patients directly to a major tertiary trauma hospital or neurosurgical facility wherever possible.



# Analgesia

## 10. What analgesia should patients with closed head injury receive?

| RECOMMENDATION  | Strength of recommendation |
|---|----------------------------|
| <b>Analgesia in isolated mild head injury</b> <ul style="list-style-type: none"> <li>■ Persistent severe headache or worsening severe headache is an indication for a CT scan to exclude a significant intracranial lesion</li> <li>■ Most headaches associated with isolated mild head injury will respond to simple analgesia such as paracetamol.</li> <li>■ Isolated mild head injury patients who require more than paracetamol for headache should be considered for a CT scan to exclude a significant intracranial injury</li> </ul>  | CONSENSUS                  |
| <b>Analgesia guide for isolated mild head injury:</b> <ul style="list-style-type: none"> <li>■ Paracetamol, 1g, q 4-6 hours, maximum 4g/24 hours*</li> </ul> <p>If paracetamol is ineffective as a sole agent then stronger analgesia such as oral opioids or parenteral opioids should not be prescribed to patients with isolated mild head injury unless the need for an initial or repeat CT scan to exclude clinically important intracranial lesions has been considered and a senior clinician has been consulted. After further clinical assessment consider adding;</p> <ul style="list-style-type: none"> <li>■ Codeine Phosphate, 30-60mg, q 4-6 hours*<br/>or</li> <li>■ Oxycodone (immediate release), 5-10mg q 4-6 hours*</li> </ul> <p><i>NB Avoid the use of aspirin / NSAIDS due to increased risk of bleeding</i><br/>* See standard texts for detailed prescribing information</p> | CONSENSUS                  |
| <b>Analgesia guide for mild head injury with associated systemic injuries:</b> <ul style="list-style-type: none"> <li>■ More likely to need titrated intravenous opioids, procedural sedation or general anaesthesia for their associated injuries.</li> <li>■ Have a lower threshold for performing CT scans.</li> <li>■ Require close clinical assessment and observation.</li> <li>■ Appropriate pain relief should not be withheld due to concerns of masking head injury symptoms and signs</li> <li>■ Analgesia needs to be individualised under the supervision of a senior clinician.</li> </ul>  | CONSENSUS                  |
| <b>Analgesia in moderate to severe head injury</b> <ul style="list-style-type: none"> <li>■ Likely to require titrated intravenous analgesia and sedation for associated injuries, clinical management or intubation.</li> <li>■ Will require close clinical observation in a high dependency area following initial clinical assessment and CT scanning.</li> <li>■ Analgesia needs to be individualised under the supervision of a senior clinician.</li> </ul>   | CONSENSUS                  |
| <b>Clinical approach to pain management in closed head injury (all severities)</b> <ul style="list-style-type: none"> <li>■ Consult a senior clinician if any significant change in the patient's condition</li> <li>■ Clinically re-assess if: <ul style="list-style-type: none"> <li>- inadequate analgesia or worsening headache</li> <li>- excessive drowsiness, or other clinical deterioration</li> </ul> </li> <li>■ <b>Before using stronger analgesia:</b> <ul style="list-style-type: none"> <li>- clinically re-assess patient</li> <li>- consider need for CT scan</li> <li>- consult senior clinician</li> </ul> </li> </ul>   | CONSENSUS                  |

No prospective randomised multi-centre trials specifically addressing the issue of analgesia in patients with closed head injury could be identified. Interestingly, Bazarian et al<sup>82</sup> in a review of management of mild head injury patients in selected emergency departments in the USA found that nearly 50% of patients did not have assessment of pain documented and 50% of those received no analgesia. There are many studies addressing the issue of analgesia in the broader patient population. The following recommendations are based on existing analgesia clinical practice guidelines including those available via the NSW Health Clinical Information Access Program (CIAP) such as Therapeutic Guidelines.<sup>171, 172</sup>

## Mild head injury

### Isolated mild head injury patients

- Most headaches associated with isolated mild head injury will respond to simple analgesia such as paracetamol.
- If paracetamol (1g q 4-6 hr max 4g/day) alone is ineffective then codeine phosphate (60mg q 4-6hr max 240mg/daily) or low dose immediate release oxycodone (5-10mg q 4-6hr) can be added after the need for an initial or repeat CT scan to exclude a clinically significant intracranial lesion has been considered.
- The aim of analgesia in patients with isolated mild head injury with associated headache should be to relieve pain without causing excessive drowsiness. Patients should always remain easy to rouse.
- Analgesics containing aspirin or NSAIDs should not be used due to the increased risk of bleeding from platelet dysfunction.
- Patients with persistent or worsening severe headaches requesting increasing analgesia should be clinically reassessed and the need for a CT scan to exclude a clinically significant intracranial lesion considered.
- Patients who are already drowsy or difficult to rouse should not be given additional opioids due to the risk of respiratory depression. Patients with persistent or worsening drowsiness should be clinically reassessed.
- If a headache has not responded to simple analgesics or a patient is abnormally drowsy following analgesia then the patient should be clinically re-assessed for potential complications. Specific questions that should be considered include:

- 1) Are there other signs of clinical deterioration such as persistently abnormal or worsening mental status, behaviour, drowsiness or vomiting?
- 2) Does the patient require an initial CT scan or a repeat CT scan to exclude a clinically important cranial lesion?
- 3) Has a senior clinician been notified/consulted about the change in the patient's condition?

- Stronger analgesia such as increased dosage or oral opioid or parenteral opioid should not be prescribed to patients with isolated mild head injury unless a senior clinician has been consulted and clinically important complications considered.

#### Notes:

- see standard texts for precautions and toxicity eg, Therapeutic Guidelines / MIMS
- avoid aspirin / NSAIDs due to risk of bleeding
- consult a senior clinician if any significant change in the patient's condition
- clinically re-assess if:
  - inadequate analgesia or worsening headache
  - excessive drowsiness, or other clinical deterioration
- before using stronger analgesia:
  - clinically re-assess patient
  - consider need for CT scan
  - consult senior clinician

### Mild head injury patients with other associated injuries

- The same general principles outlined for isolated mild head injury patients apply. However, mild head injury patients with other associated injuries are more likely to need titrated intravenous opioids, procedural sedation or general anaesthesia for their associated injuries.
- Clearly, this has the potential to mask signs of worsening head injury. Therefore, these patients require close clinical observation and clinicians should have a low threshold for performing CT scans on mild head injury patients requiring intravenous opioids,

procedural sedation or general anaesthesia for associated injuries.

- Appropriate pain relief should not be withheld due to concerns of masking head injury symptoms and signs as the patient's pain is likely to make clinical assessment more difficult and mask other signs of injury and the patient should not be allowed to suffer unnecessarily.

## **Moderate to severe head injury**

- Isolated moderate head injury patients who rapidly clinically improve can be treated in a similar way to mild head injury patients. They all require an initial CT scan.
- However, most moderate head injury patients and nearly all severe head injury patients will require titrated intravenous analgesia and sedation for associated injuries, clinical management or intubation. These patients will all require close clinical observation in a high dependency area following initial clinical assessment and CT scanning. Analgesia needs to be individualised under the supervision of senior clinicians.
- Routine analgesia with intravenous opioids is recommended for most intubated patients as pain may cause adverse effects on blood pressure and intracranial pressure.

# Anti-convulsants

## 11. Which patients with closed head injury should receive anti-convulsants?

| RECOMMENDATION   | Strength of recommendation |
|--|----------------------------|
| Consult a senior clinician or your network neurosurgical service before commencing prophylactic anti-convulsants in patients with acute closed head injury   | CONSENSUS                  |
| Prophylactic anti-convulsants are not indicated for patients with uncomplicated mild head injury   | B                          |
| Prophylactic anti-convulsants should be considered in patients with complicated mild head injury or moderate to severe head injury.  | B                          |
| Specific indications to consider prophylactic anti-convulsants in the first week following a head injury include: <ul style="list-style-type: none"> <li>■ Extradural, subdural or intracerebral haematoma on CT</li> <li>■ Depressed skull fracture on CT</li> <li>■ Early post traumatic seizure in hospital (especially if focal or prolonged)</li> <li>■ Severity of head injury (low initial GCS / prolonged coma / prolonged PTA)</li> <li>■ Any suspicion of penetrating injury</li> </ul>  | B                          |
| Prophylactic anti-convulsants decrease the incidence of early post traumatic seizures within seven days of closed head injury.   | B                          |
| Early post traumatic seizures have not been shown to be associated with worse patient outcomes in large population studies.  | B                          |
| Clinical judgment is required on whether to prescribe anti-convulsants for individual patients.  | CONSENSUS                  |
| <b>Indications for anti-convulsants by post traumatic seizure type</b><br>Immediate post traumatic seizures (at time of injury) <ul style="list-style-type: none"> <li>■ Anti-convulsants not warranted unless specific indication present (see above)</li> </ul> Early post traumatic seizures ( up to 7 days post injury) <ul style="list-style-type: none"> <li>■ Anti-convulsants should be considered especially if any of the other specific indications are also present (see above)</li> </ul> Late post traumatic seizures (more than 7 days post injury) <ul style="list-style-type: none"> <li>■ Long term anti-convulsants should be considered after the first late post traumatic seizure due to the increased risk of developing post traumatic epilepsy</li> <li>■ There is no evidence that the routine use of anti-convulsants following closed head injury reduces the risk of late post traumatic seizures.</li> </ul> | B                          |
| <b>Recommended drugs and loading doses*</b><br><b>Standard therapy:</b><br><b>Phenytoin:</b> <ul style="list-style-type: none"> <li>■ Intravenous loading dose: 20 mg/kg in NS (&lt;6.7mg/ml) no faster than 50mg/min</li> </ul> Standard adult IVI loading dose: 1000mg phenytoin diluted in 150ml normal saline over 60 mins with in line micron filter  | B                          |
| <b>Alternative therapies:</b><br><b>Levetiracetam:</b> <ul style="list-style-type: none"> <li>■ Intravenous loading dose: 10mg /kg (max 1000mg)</li> <li>■ Standard adult IVI loading dose: 1000mg levetiracetam in 100ml normal saline over 15 mins</li> </ul> <b>Sodium Valproate:</b> <ul style="list-style-type: none"> <li>■ Intravenous loading dose: 10mg /kg (max 800mg)</li> <li>■ Standard adult IVI loading dose: 800mg in 100ml normal saline over 15 mins</li> </ul> <p>* See standard texts for detailed prescribing information</p>   | C                          |

Post traumatic seizures are a recognised complication of closed head injuries with incidence depending largely on severity of injury. Penetrating injuries have a much higher incidence of post traumatic seizures. Post traumatic seizures are classified as either immediate, early (0-7 days) or late/delayed (>7 days).<sup>144</sup> Immediate and early post traumatic seizures are thought to be associated with the acute injury and are not significantly associated with the development of post traumatic epilepsy. Late post traumatic seizures are less likely to be related to the acute injury and are more likely to be associated with the development of post traumatic epilepsy.<sup>144</sup>

Brief generalised post traumatic seizures immediately following mild head injury are not usually associated with poor outcome or intracranial injury. These immediate seizures are frequently seen on sporting fields and in young children. It has been proposed that these immediate seizures be called "concussive convulsions" and it has been suggested that they are not an epileptic phenomena.<sup>64, 107, 134</sup> McCrory et al<sup>145</sup> demonstrated that outcome was universally good for these "concussive convulsions" in elite Australian sportsmen.

Immediate and early post traumatic seizures are relatively common in patients with mild closed head injury with a reported incidence of up to 5%.<sup>144, 173</sup> The literature is somewhat contradictory about the risk of seizures being associated with intracranial injury. Many of the larger studies found that post traumatic seizures were not significantly associated with intracranial injury.<sup>85, 115, 168, 172, 174</sup> However, Haydel et al<sup>32</sup> found that they were significant and Smits et al<sup>47</sup> found that while they were not statistically significant, included them in their decision rule because of their perceived clinical importance. Neidlinger et al<sup>146</sup> found that the "yield of unsuspected major intracranial abnormality on CT scan justifies a policy of its routine use in trauma patients with seizure or who are post ictal, regardless of prior seizure history."

Immediate or early post traumatic seizures are more common (up to 30%) in patients with moderate to severe closed head injury, and are more likely to be associated with significant intracranial injury.<sup>144, 173</sup> There is an association with underlying structural lesions and the potential for secondary brain injury especially with prolonged, focal or delayed seizures. Any moderate to severe head injury patient who has a post traumatic seizure warrants a CT scan to exclude significant underlying pathology and then the need for prophylactic anti-convulsants should be considered.

The major risk factors for developing early post traumatic seizures include lower initial GCS, depressed skull fracture, penetrating injury, extradural/subdural/intracerebral haematomas and young age.<sup>12, 17, 29, 64, 182</sup> The risk is therefore related to the amount of structural damage. Penetrating injury provides the greatest risk. The risk posed by an intracranial bleed is proportional to the amount of blood.

Delayed or late post traumatic seizures (incidence range 1-15%) that occur more than seven days after injury are associated with the development of post traumatic epilepsy. Risk factors for late post traumatic seizures include lower initial GCS, depressed skull fracture, penetrating injury, extradural/subdural/intracerebral haematomas, elderly (age >65), neurosurgical intervention and early post traumatic seizures.<sup>12, 17, 29, 64, 182</sup>

Acute post traumatic seizures require systematic reassessment of the ABCDEs to exclude systemic causes and termination with benzodiazepines if required. Underlying structural lesions should be excluded with CT scan and then the need for prophylactic anti-convulsants considered.

If prophylactic anti-convulsants are recommended then phenytoin (dilantin) is normally given as there has been extensive experience with its use and it can be given as either an oral or an intravenous loading dose.<sup>11, 17, 29, 69, 151</sup> Alternatives include sodium valproate (epilim) and levetiracetam (keppra). Levetiracetam is being increasingly used in both non traumatic and traumatic epilepsy due to its better side effect profile and may become first line therapy in the future.<sup>174, 175</sup> However, there is limited experience with its use in the trauma setting and phenytoin remains the first line therapy at present.

- Current evidence suggests that anti-convulsants decrease the incidence of early post traumatic seizures within seven days of closed head injury.<sup>17, 29, 151</sup>
- Early post traumatic seizures have not been shown to be clearly associated with worse patient outcome.<sup>17, 29, 151</sup>
- There is no evidence that prophylactic anti-convulsants following closed head injury reduce the risk of late post traumatic seizures.<sup>17, 29, 151</sup>
- Anti-convulsants as a group have many potential side effects and are relatively poorly tolerated by patients long term.<sup>17, 29, 151</sup>

- Clinical judgement is therefore required to balance the potential benefits versus potential harm of anti-convulsants for individual patients with closed head injury.
- Anti-convulsants are therefore only indicated in the first week following closed head injury to reduce the risk of complications from early post traumatic seizures. They should not be routinely continued long term.
- In most patients with mild head injury prophylactic anti-convulsants are not indicated.
- Specific indications to consider prophylactic anti-convulsants in the first week following a closed head injury include:
  - Extradural, subdural or intracerebral haematoma on CT
  - Depressed skull fracture on CT
  - Early post traumatic seizure in hospital
  - Severity of injury (low GCS / duration of coma / duration of PTA)
  - Any penetrating injury
- Long term anti-convulsants should be considered after the first or second late post traumatic seizure (ie after the diagnosis of post traumatic epilepsy).<sup>17, 29, 50, 132</sup>

## Mild head injury

In most patients with mild head injury prophylactic anti-convulsants are not indicated. The risk of seizures is low and the risk of secondary brain injury is low. Mild head injury patients who have had an early post traumatic seizure in hospital and those with structural lesions such as depressed skull fractures or focal intracranial haematomas on CT scan should be considered for anti-convulsants and discussed with a neurosurgical service.

## Moderate to severe head injury

In moderate and severe head injury patients there is a stronger case to consider prophylactic anti-convulsants especially in those with structural lesions on CT scan. The risk of early post traumatic seizures is greater and the potential for secondary brain injury from these seizures is increased. Prolonged post traumatic seizures are of most concern and may be difficult to recognise in intubated patients. Therefore, prophylactic anti-convulsants are more likely to be recommended in these patients. It should be noted that most intubated patients in NSW receive analgesia and sedation with morphine and midazolam infusions and so are already receiving a benzodiazepine anti-convulsant. The decision to use anti-convulsants should be discussed with the relevant neurosurgical service.

## Recommended anti-convulsant doses\*

### *Standard therapy:*

#### Phenytoin:

- Intravenous loading dose: 20 mg/kg in NS (<6.7mg/ml) no faster than 50mg/min
- Standard adult IVI loading dose: 1000mg phenytoin diluted in 150ml normal saline over 60 mins with in line micron filter

### *Alternative therapies:*

#### Levetiracetam:

- Intravenous loading dose: 10mg /kg (max 1000mg)
- Standard adult IVI loading dose: 1000mg levetiracetam in 100ml normal saline over 15 mins

#### Sodium Valproate:

- Intravenous loading dose: 10mg /kg (max 800mg)
- Standard adult IVI loading dose: 800mg in 100ml normal saline over 15 mins

\* See standard texts for detailed prescribing information

# Evidence Tables

1. Studies examining the definition of mild head injury and the assessment of mild head injury patients (including CT scanning)
2. Complications of mild closed head injury
3. Optimal management strategy for high-risk mild head injury patients when CT scanning is not available
4. Proven treatments for moderate to severe head injury
5. Transfer of patients with a closed head injury
6. Discharge of patients with a mild head injury

## Evidence Tables

**Evidence Table 1: Studies examining the definition of MHI and the assessment of MHI patients (including CT scanning)**

| Author and year                                | Study type & LOE   | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments  |
|--|--|---------------|--|--|---|
| Af Geijerstam <i>et al</i> <sup>46</sup> 2003  | Meta-analysis of comparative studies & case series<br>n=24249<br>LOE IV  | fair          | Estimate of the incidence of complications, mortality and pathological CT findings in MHI pts  | For patients with blunt head trauma with LOC and a GCS of 15 on admission, mortality was estimated at 0.1%.  |   |
| Af Geijerstam <i>et al</i> <sup>119</sup> 2004 | Systematic review of cohort / economic modelling studies<br>LOE IV       | fair          | Comparison of the cost of triage CT & discharge vs admission & observation for MHI pts in Sweden   | On average costs were 1/3 less for the triage CT clearance & discharge strategy compared to the admission & observation strategy.  | MHI = GCS15 on admission<br>No studies were found directly comparing costs in actual pts. Authors used 4 studies that used economic modelling for CT strategy, pt data for admission & observation arm. |
| Af Geijerstam <i>et al</i> <sup>39</sup> 2005  | Systematic review of cohort studies and case series<br>n~65000<br>LOE IV | fair          | What is the incidence of adverse outcomes for MHI pts with normal CT findings on admission?  | Only 3 MHI pts who had no abnormalities detected on admission CT experience adverse outcomes within two days post-injury<br>CT is a safe method of early triage for all MHI pts                  | MHI= GCS15 on admission<br>Short follow up time (≤2days)  |
| Af Geijerstam <i>et al</i> <sup>91</sup> 2006  | Multicentre RCT<br>n=2602<br>LOE II                                      | good          | Comparison of triage CT & discharge vs admission & observation for MHI pts. Powered for non-inferiority of triage CT & discharge strategy  | No difference in self-reported adverse outcomes at 3 months post injury.   | MHI = GCS15 on admission  |
| Akopian <i>et al</i> <sup>176</sup> 2007       | Retrospective cohort<br>n=144<br>LOE III-2                               | fair          | To identify factors that predicted poor outcome after blunt head trauma from a cohort of 144 CHI pts admitted to one ICU over a 5yr period | Older age, higher injury severity score and lower GCS were independent predictors of poor outcome.<br>Mortality rate for pts with GCS≥8: 8%<br>Mortality rate for pts with GCS<8: 33% (p<0.0001) |   |



| Author and year                                   | Study type & LOE   | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments  |
|---|--|---------------|---|--|---|
| Bee <i>et al</i> <sup>132</sup><br>2009           | Prospective case series<br>n=207<br>LOE IV                                 | fair          | Should pts with MHI (GCS 14 – 15) PLUS abnormal findings on initial CT scan and no clinical deterioration routinely be admitted to ICU and undergo repeat CT scan within 24hrs? | 28% of MHI pts in this series developed worsening findings either on follow-up CT or on observation in ICU<br>MHI pts with abnormal findings on CT should be admitted to ICU and undergo routine repeat CT scanning within 24hrs                                       |   |
| Biberthaler <i>et al</i> <sup>177</sup><br>2006   | Prospective cohort<br>n=1309<br>LOE III-3                                  | fair          | Would the addition of serum S100-B levels to the current clinical decision rule used in this MHI cohort reduce the number of pts requiring a CT scan                            | S100-B levels identified 92/93 CT+ pts (99% sensitivity), with 30% specificity.<br>The addition of S100-B levels would have reduced the number of CT scans in this MHI cohort by 30%.  | MHI = GCS 13-15 plus one specified 'risk' factor  |
| Borczuk <sup>49</sup><br>1995                     | Retrospective cohort<br>n=1228<br>LOE III-2                                | poor          | What were the clinical predictors of an abnormal CT finding in this cohort of MHI pts?  | Age >60yrs, Skull fracture, neurological deficit or cranial soft tissue injury were associated with an abnormal CT   | MHI = GCS 13-15<br>Absence of LOC/amnesia did not exclude significant injury  |
| Borg <i>et al</i> <sup>101</sup><br>2004<br>(WHO) | Systematic review of 73 comparative studies, no meta-analysis<br>LOE III-3 | good          | What are the clinical indications for MHI pts at high risk of an intracranial lesion?   | MHI pts with a GCS of 15 plus any ONE of the following should undergo CT scanning: Age >60yrs, dangerous mechanism of injury, suspected skull fracture, signs of supra-clavicular trauma, anterograde amnesia, emesis, headache, seizure, drug or alcohol intoxication |   |
| Bracken <i>et al</i> <sup>153</sup><br>2007       | Retrospective data analysis<br>n=13728<br>LOE III-3                        | good          | Does intoxication with alcohol predict intracranial injury, or just interfere with assessment?  | Intracranial injury was detected in 6.9% pts identified by clinicians as intoxicated vs 8.1% of non-intoxicated pts. Intoxication was not an independent predictor of intracranial injury.   | The lower incidence of intracranial injury findings in the intoxicated group was a result of the more liberal use of CT scanning for this group, due to heightened concern of clinicians. |
| Clement <i>et al</i> <sup>77</sup><br>2006        | Retrospective cohort<br>n=4551<br>LOE III-2                                | good          | How accurate is the Canadian CT Head Rule (CCHR) in predicting clinically important brain injury in CHI pts with a GCS of 15?   | CCHR identified 100% of the 26 GCS 15 pts requiring neurosurgical intervention.  | Listed clinical features associated with patients GCS 15 who deteriorated.  |

| Author and year  | Study type & LOE   | Study quality | Study question / objective   | Study outcomes/findings relevant to question  | Comments   |
|--|--|---------------|--|---|--|
| Culotta <i>et al</i> <sup>50</sup><br>1996               | Retrospective cohort<br>n=3370<br>LOE III-2                    | fair          | What is the relationship of admission GCS scores to variables indicative of injury severity in this cohort of MHI pts? | An initial GCS <15 is a significant risk factor for both abnormal CT scan findings and neurosurgery.  | There is significant heterogeneity in injury severity between GCS 13-14 and GCS 15 MHI pts.  |
| Dacey <i>et al</i> <sup>51</sup><br>1986                 | Prospective cohort<br>n=610<br>LOE III-2                       | fair          | Identification of factors identifying MHI pts requiring neurosurgery   | Risk factors: Initial GCS <15 or skull fracture   | This study primarily examined the association of the presence of skull fracture with the need for neurosurgery   |
| Dunham <i>et al</i> <sup>36</sup><br>1996                | Prospective cohort<br>n=2587<br>LOE III-2                      | fair          | What were the clinical factors predictive of abnormal findings on CT scanning in this cohort of MHI pts?               | GCS <15, age >60years or cranial soft tissue injury were independently predictive of abnormal CT findings   | MHI = GCS15 PLUS LOC/ amnesia or GCS 13-14<br>Noted that determination of initial GCS may be subject to inter-observer and inter-centre variability  |
| Dunning <i>et al</i> <sup>138</sup><br>2006<br>(CHALICE) | Prospective cohort<br>n= 22772<br>Children only<br>LOE III - 1 | good          | Which clinical criteria predicted the presence of clinically important lesions in children with head injury?           | Clinical criteria suggesting need for CT scan due to risk of clinically important lesion were (summarised from original):<br><br>LOC >5min<br>Amnesia >5min<br>Abnormal drowsiness<br>Vomiting – recurrent<br>Suspicion NAI<br>Seizure<br>GCS<14 (or GCS<15 if <1yr)<br>Suspicion skull fracture<br>Neurodeficit<br>Scalp haematoma/laceration if <1yr<br>Dangerous mechanism | Very large multicentre study with good follow up<br><br>Reported NPV for no clinically significant intracranial injury for patients with GCS 13-15 (22579) was 99.9% with a CT ordering rate of 13.3%<br><br>Important paediatric study with similar findings to adult studies |

| Author and year                              | Study type & LOE                             | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments  |
|--|--|---------------|--|--|---|
| Fabbri <i>et al</i> <sup>31</sup><br>2004    | Prospective cohort<br>n=5578<br>LOE III-1    | good          | Validation of MHI guideline risk factors for post traumatic lesion, neurosurgery or post concussive symptoms at 6 weeks:<br><br>High risk factors:<br>GCS 14 or 15 with any of:<br>Neurodeficit, skull fracture, coagulopathy, age >60yrs, previous neurological symptoms, epilepsy or drug or alcohol intoxication.<br>Medium risk factors:<br>GCS 15 with any of:<br>LOC, amnesia, emesis or diffuse headache<br>Low risk factors:<br>GCS 15 with none of the above. | The best predictors of intracranial injury and neurosurgery were abnormal GCS or skull fracture and to a lesser extent other clinical findings.<br><br>6 week outcome was best predicted by abnormal initial GCS or skull fracture   | Risk of post concussive symptoms were similar in all groups, while the rates of intracranial injury were significantly higher in the high risk group.<br>Neurosurgical intervention occurred in 71 pts, 0 from the low risk group, 5 from the medium risk group and 66 from the high risk group |
| Fabbri <i>et al</i> <sup>116</sup><br>2005   | Prospective cohort<br>n=7955<br>LOE III-2    | good          | Comparison of the predictive accuracy of the NICE vs the NCWFNS criteria for predicting adverse outcomes in MHI pts  | In this cohort, NICE criteria had 93.5% sensitivity, 70% specificity; NCWFNS criteria had 97.8% sensitivity and 45% specificity for predicting adverse events in MHI pts. Authors concluded that NICE criteria were reliable and resource saving, as less pts would undergo CT scanning. | MHI = GCS 14-15, Age ≥10yrs<br><br>6 month follow-up period   |
| Falmirski <i>et al</i> <sup>61</sup><br>2003 | Prospective cohort<br>n=331<br>LOE III-2     | fair          | Was GCS14-15 with LOC predictive of intracranial injury, or did the inclusion of other clinical criteria increase the predictive value?  | 6% of pts with GCS 14-15, LOC but no other clinical criteria showed intracranial injury on CT, but required no intervention<br>23% of pts with GCS 14-15, LOC and at least one of 10 clinical criteria showed intracranial injury on CT  | LOC alone is not predictive of significant head injury; other clinical criteria should be present.  |
| Feuerman <i>et al</i> <sup>139</sup><br>1988 | Retrospective case series<br>n=373<br>LOE IV | fair          | Comparison of skull x-ray, CT scan & observation for predicting subsequent deterioration or presence of an operative haematoma in MHI pts  | A GCS <15, neurological deficit or abnormal state were all predictive of neurological deterioration or haematoma<br>Skull x-ray of no utility  | MHI = GCS 13-15   |

| Author and year                                    | Study type & LOE   | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments   |
|--|--|---------------|--|--|--|
| Gomez <i>et al</i> <sup>62</sup><br>1996           | Retrospective cohort study<br>n= 2484<br>LOE III-2           | fair          | What were predictors of abnormal CT findings in this cohort of MHI pts?  | Advanced age, an initial GCS <15, skull fracture or focal neurological deficit were associated with abnormal CT findings   | MHI = 13 – 15<br>Relatively few CT scans were performed in this cohort   |
| Haydel <i>et al</i> <sup>32</sup><br>2000<br>(NOC) | Prospective cohort study<br>n=909 (age>10yrs)<br>LOE II      | good          | What were the clinical predictors of positive CT findings in this cohort of MHI pts with an initial GCS15 and LOC/amnesia? | All pts with a positive CT scan had at least one of the following criteria [likelihood ratio]:<br>Anterograde amnesia [15.0]<br>Supra-clavicular trauma [11.0]<br>Drug or alcohol intoxication [11.0]<br>Seizure [3.0]<br>Age >60years [3.0]<br>Headache [2.0]<br>Emesis [2.0] | Clinical decision rule of the seven criteria was 100% sensitive and 25% specific with a negative predictive value of 100%.<br>Exclusion criteria included patients without LOC or amnesia, GCS13or14, neurodeficit and did not have enough patients to assess coagulopathy.<br>Supraclavicular trauma was a very broad inclusion criteria. |
| Hofman <i>et al</i> <sup>66</sup><br>2000          | Meta-analysis of cohort studies<br>n=20 studies<br>LOE III-2 | good          | Is the presence of a skull fracture predictive of intracranial haemorrhage in MHI pts (GCS 13-15)?                         | The prevalence of ICH ~ 8/100<br>The sensitivity for a skull x-ray was 39%, specificity 95%  |  |
| Horowitz <i>et al</i> <sup>67</sup><br>2001        | Retrospective case series<br>N=100<br>LOE IV                 | fair          | Was transient LOC predictive of intracranial injury for this series of MHI pts?  | Transient LOC did not predict the need for subsequent neurosurgery   | Skull radiography is not a useful screening tool for intracranial injury in MHI pts. Skull fracture is associated with increased risk of intracranial injury.  |
| Hsiang <i>et al</i> <sup>52</sup><br>1997          | Prospective cohort<br>n=1360<br>LOE III-2                    | fair          | Should the definition of MHI include GCS 13 -15?   | In this cohort, an initial GCS <15 was associated with a higher risk of abnormal CT findings, neurosurgery or poor outcome, compared with an initial GCS of 15.  | Authors state that MHI should be defined as a GCS of 15 without acute radiographic abnormalities, and high-risk MHI should be defined as a GCS of 13 or 14, or a GCS of 15 with acute radiographic abnormalities   |

| Author and year                           | Study type & LOE  | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments   |
|---|---|---------------|--|--|--|
| Huda <i>et al</i> <sup>122</sup><br>2007  | Non comparative bench study                                     | n/a           | Estimation of radiation doses from CT scanning   | The adult pt effective dose of radiation in a single head CT scan is comparable to the annual mean natural radiation exposure in the USA (3mSv/y), and is substantially lower than the reported threshold for the induction of deterministic effects   | Study used mechanistic models of human anatomy   |
| Ibanez <i>et al</i> <sup>33</sup><br>2004 | Prospective cohort<br>n=1101<br>LOE II                          | good          | In this cohort of MHI Pts (GCS 14-15) what risk factors were associated with intracranial lesions on CT scanning?<br><br>Are clinical guidelines useful in identifying clinically important intracranial lesion sin the MHI patient? | 7.5% of this cohort had intracranial lesions on CT scan. The clinical risk factors associated with a lesion were:<br>GCS 14, neurological deficit, LOC, skull fracture, emesis, severe headache, coagulopathies, age >65yrs, significant extracranial lesions, hydrocephalus with shunt.<br>Clinical guidelines: miss some abnormal CT scans, identify clinically important intracranial lesions | Findings consistent with previous studies identifying risk factors. Supports use of clinical decision rules for identifying clinically important lesions. Excellent discussion on the pros & cons of clinical guidelines. Confirmed the absence of LOC as not useful in ruling out intracranial injury |
| Ivascu <i>et al</i> <sup>79</sup><br>2005 | Prospective cohort with historical control<br>n=82<br>LOE III-3 | good          | Comparison of the 'Coumadin protocol' of immediate triage CT and rapid reversal of anticoagulant status of anticoagulated MHI pts with ICH on CT vs delayed triage for anticoagulated MHI pts  | The Coumadin protocol pts had a mean time to reversal of anticoagulant of 1.9hrs vs 4.3 hrs for the historical group. Mortality rate was 10% for the Coumadin protocol group vs 48% for the historical control group.  | 16/19 pts in Coumadin protocol group had a GCS 14 or 15. Coumadin group received FFP and Vitamin K.  |
| Jeret <i>et al</i> <sup>68</sup><br>1993  | Prospective cohort<br>n=712<br>LOE III-2                        | fair          | What were the clinical predictors for abnormalities on CT scanning?  | Increasing age, skull fracture or a dangerous mechanism of injury were associated with the presence of intracranial pathology on CT  | Concluded that no clinical prediction rule could be developed to exclude intracranial injury   |
| Jones <i>et al</i> <sup>78</sup><br>2006  | Retrospective cohort<br>n=1020<br>LOE III-2                     | fair          | Is the use of clopidogrel in CHI pts >50 yrs age associated with increased mortality / morbidity compared to matched pts not on clopidogrel?   | Pts on clopidogrel had an increased risk of re-bleeds, neurosurgery and repeat neurosurgery. Mortality was not significantly different between the two groups.   | Included all CHI   |

| Author and year                               | Study type & LOE   | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments  |
|---|--|---------------|---|--|---|
| Kuppermann <i>et al</i> <sup>85</sup><br>2009 | Prospective cohort<br>n= 42412<br>Children only<br>LOE III-1 | good          | Which clinical criteria predicted the presence of clinically important lesions in children with head injury (GCS14-15)?                                       | <p>Clinical criteria suggesting need for CT scan due to risk of clinically important lesion were (summarised from original) for children 2yrs or older with GCS 14-15:</p> <p>CT recommended (14% of population with 4.3% risk ciTBI) if;<br/>GCS 14 or other signs altered mental state (drowsy/behaviour/repetitive)<br/>Clinical skull fracture</p> <p>CT or observation (27.7% of population with 0.9% risk ciTBI) if;<br/>LOC<br/>Vomiting<br/>Severe headache<br/>Dangerous mechanism</p> <p>Decide CT or observation based on clinical experience/judgment, multiple or isolated findings, worsening symptoms or signs with ED observation &amp; parental preference)</p> <p>CT not recommended (58.3% of population with &lt;0.05 risk ciTBI) if<br/>None of above</p> |   |
| Lee <i>et al</i> <sup>69</sup><br>1995        | Prospective cohort<br>n=1812<br>LOE III-2                    | fair          | What were the clinical predictors of neurological deterioration in this cohort of pts with a GCS of 15 who have sustained a blow to the head, LOC or amnesia? | The risk factors for deterioration were age >60yrs, abnormal mental status (drowsiness), focal neurological deficit, headache or emesis  | 57% of the pts who deteriorated did so in the first 24hrs.<br>23/28 pts who deteriorated required subsequent neurosurgery |
| Mack <i>et al</i> <sup>150</sup><br>2003      | Retrospective cohort<br>n=133<br>LOE III-2                   | fair          | What are the clinical criteria differentiating MHI pts ≥65yrs age with intracranial injury from MHI pts in the same age group without intracranial injury?    | The study examined 13 potential clinical indicators and only one (chronic altered mental status) was significantly associated with intracranial injury.<br>Authors conclude that CT scans are recommended for all MHI pts ≥65  | MHI = GCS 13-15<br><br>Category of head injury pts included in the study were not defined                                 |

| Author and year                           | Study type & LOE                         | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments   |
|---|--|---------------|--|--|--|
| Madden <i>et al</i> <sup>70</sup><br>1995 | Prospective cohort<br>n=273<br>LOE III-2 | fair          | What clinical criteria predict intracranial injury on CT?  | Initial GCS<15, Abnormal mental status, deterioration, LOC, any skull fracture, focal neurological signs (pupils), facial injury                                     | These criteria detected all pts requiring neurosurgery, but missed two 2/273 with abnormal CT scan results   |
| Miller <i>et al</i> <sup>71</sup><br>1996 |  | fair          | What is the clinical utility of routine CT scanning in MHI pts with an initial GCS of 15 and LOC/amnesia?  | 0.2% of this cohort required subsequent neurosurgery. Higher risk if emesis, nausea, headache or a skull fracture was present.                                       | Routine CT scanning not recommended for pts with GCS 15 and LOC/amnesia unless other clinical signs/symptoms of skull fracture or head injury are present  |
| Miller <i>et al</i> <sup>88</sup><br>1997 |  | fair          | What is the predictive value of the presence of severe headache, nausea, emesis or skull fracture for abnormal CT findings in this cohort of MHI pts with a GCS of 15 & LOC? | All four factors were independently associated with the need for subsequent neurosurgery and abnormal CT findings  | The use of these four clinical criteria in this cohort of MHI patients would have resulted in a 61% reduction in the number of head CT scans and still identify all patients who require neurosurgery and 65% of pts with an abnormal CT |
| Mina <i>et al</i> <sup>147</sup><br>2002  |  | fair          | In a cohort of head injured pts who were already on anticoagulant therapy, was there an increased risk of intracranial lesion compared to non-anticoagulated, matched pts?   | Head injured pts on anti-coagulation therapy have a four – fivefold increased risk of mortality compared to matched head-injured pts not on anti-coagulation therapy | This cohort were not isolated MHI pts – average ISS of 17 and average GCS of 11  |



| Author and year  | Study type & LOE                          | Study quality | Study question / objective   | Study outcomes/findings relevant to question  | Comments  |
|--|---|---------------|--|---|---|
| Mower <i>et al</i> <sup>58</sup><br>2005<br>(NEXUS II) | Prospective cohort<br>n=13728<br>LOE II   | good          | What clinical characteristics can reliably identify patients with closed head injury who do not have clinically important intracranial injuries and consequently do not require imaging? (NEXUSII) | Risk factors for intracranial injury (NPV 99.1%, sensitivity 98.3%, specificity 13.7%) identified as;<br>Neurological deficit (included GCS<15)<br>Abnormal alertness<br>Abnormal behaviour<br>Persistent vomiting<br>Skull fracture<br>Scalp haematoma<br>Age > 65<br>Coagulopathy | Clinical decision rule applied to all closed head injury patients. Did not exclude patients without LOC or amnesia. Potentially broad clinical application. Prolonged loss of consciousness (>5min) identified as potentially significant variable but did not improve sensitivity and reduced specificity. Considered seizure and progressive severe headache due to clinical importance but when added to original criteria also did not significantly improve sensitivity and reduced specificity of the clinical decision rule. Concluded that no clinical decision rule is perfect and there is always a trade off between sensitivity and specificity and that clinical judgment is required. |
| Nagy <i>et al</i> <sup>89</sup><br>1999                | Prospective cohort<br>n=1170<br>LOE III-2 | fair          | Can pts with a GCS of 15 plus LOC/ amnesia be safely managed with CT only, observation only or both?   | 39 pts had abnormal findings on CT, 4 required neurosurgery. No pts with negative findings on CT deteriorated during the observation period (24hrs)   | Authors recommend discharge if initial CT is negative. Of note was the finding that in 969 pts the LOC status was unable to be determined, highlighting the difficulties of assessment  |

| Author and year                               | Study type & LOE  | Study quality | Study question / objective   | Study outcomes/findings relevant to question  | Comments   |
|---|---|---------------|--|---|--|
| Ono <i>et al</i> <sup>137</sup><br>2007       | Prospective cohort<br>n=1064<br>LOE III-2               | good          | In MHI pts, what clinical features and risk factors are associated with intracranial lesions?  | Statistically significant associations were found between the presence of intracranial lesions and: age >60yrs, male gender, alcohol consumption, headache, nausea/emesis or LOC/amnesia,   | MHI = GCS 14-15  |
| Palchak <i>et al</i> <sup>34</sup><br>2003    | Prospective cohort<br>n=2043 children only<br>LOE III-1 | good          | What risk factors were predictive of the need for acute intervention (defined by a neurosurgical procedure, antiepileptic medications for > 1 week, persistent neurologic deficits, or hospitalization for ≥2 nights) in this cohort of children with blunt head trauma? | Important factors for identifying children at low risk for traumatic brain injuries after blunt head trauma included the absence of: abnormal mental status, clinical signs of skull fracture, a history of vomiting, scalp hematoma (in children ≤2 years of age), and headache. | Similar findings to adult studies  |
| Rockswald <i>et al</i> <sup>179</sup><br>1987 | Case series<br>n=215<br>LOE IV                          | poor          | Identification of severe head injury pts who are talking at initial presentation and then deteriorate (GCS <8)   | Of the 215 severe head injuries presenting in this series, 33 'talked then deteriorated'  | Confirms need to be aware of seriousness of neurological deterioration as a clinical sign                    |
| Saboori <i>et al</i> <sup>80</sup><br>2007    | Prospective cohort<br>n=682<br>LOE III-2                | fair          | In MHI pts, what clinical features and risk factors are associated with the presence of intracranial lesions?  | Statistically significant associations were found between the presence of an intracranial lesion on CT and; emesis, skull fracture or age>60yrs   | MHI = GCS 15   |
| Shackford <i>et al</i> <sup>53</sup><br>1992  | Retrospective cohort<br>n=2166<br>LOE III-2             | fair          | What is the risk of deterioration in MHI pts (with an isolated head injury) with no abnormal findings on CT and normal neurological examination?   | The sensitivity of the CT scan was 100%, with positive predictive value of 10%, negative predictive value of 100%, and specificity of 51%. GCS13 or neurological deficit were risk factors for deterioration  | MHI = GCS 13-15<br>Reported that "admission to hospital does not guarantee skilled neurological observation" |
| Sharma <i>et al</i> <sup>73</sup>             | Prospective cohort<br>n=100<br>LOE III-2                | fair          | Is a history of LOC predictive of intracranial injury in this series of MHI pts?   | GCS is a predictor of intracranial injury<br>Duration of LOC more useful as a predictor of intracranial injury than simply presence / absence of LOC  | A brief history of LOC is not predictive of intracranial injury  |
| Shores <i>et al</i> <sup>108</sup><br>2008    | Prospective cohort for (diagnostic)<br>n=170<br>III-1   | fair          | Is the R-WPTAS more accurate compared to the GCS for assessing cognitive impairment in mTBI in the ED setting, using neuropsychological testing as the standard?   | The R-WPTAS had 60% sensitivity, 91% specificity; GCS had 13% sensitivity, 98% specificity for identifying cognitive impairment in mTBI pts in the ED department.   |  |

| Author and year                               | Study type & LOE  | Study quality | Study question / objective   | Study outcomes/findings relevant to question  | Comments   |
|---|---|---------------|--|---|--|
| Ono <i>et al</i> <sup>137</sup><br>2007       | Prospective cohort<br>n=1064<br>LOE III-2               | good          | In MHI pts, what clinical features and risk factors are associated with intracranial lesions?  | Statistically significant associations were found between the presence of intracranial lesions and: age >60yrs, male gender, alcohol consumption, headache, nausea/emesis or LOC/amnesia,   | MHI = GCS 14-15  |
| Palchak <i>et al</i> <sup>34</sup><br>2003    | Prospective cohort<br>n=2043 children only<br>LOE III-1 | good          | What risk factors were predictive of the need for acute intervention (defined by a neurosurgical procedure, antiepileptic medications for > 1 week, persistent neurologic deficits, or hospitalization for ≥2 nights) in this cohort of children with blunt head trauma? | Important factors for identifying children at low risk for traumatic brain injuries after blunt head trauma included the absence of: abnormal mental status, clinical signs of skull fracture, a history of vomiting, scalp hematoma (in children ≤2 years of age), and headache. | Similar findings to adult studies  |
| Rockswald <i>et al</i> <sup>179</sup><br>1987 | Case series<br>n=215<br>LOE IV                          | poor          | Identification of severe head injury pts who are talking at initial presentation and then deteriorate (GCS <8)   | Of the 215 severe head injuries presenting in this series, 33 'talked then deteriorated'  | Confirms need to be aware of seriousness of neurological deterioration as a clinical sign                    |
| Saboori <i>et al</i> <sup>80</sup><br>2007    | Prospective cohort<br>n=682<br>LOE III-2                | fair          | In MHI pts, what clinical features and risk factors are associated with the presence of intracranial lesions?  | Statistically significant associations were found between the presence of an intracranial lesion on CT and; emesis, skull fracture or age>60yrs   | MHI = GCS 15   |
| Shackford <i>et al</i> <sup>53</sup><br>1992  | Retrospective cohort<br>n=2166<br>LOE III-2             | fair          | What is the risk of deterioration in MHI pts (with an isolated head injury) with no abnormal findings on CT and normal neurological examination?   | The sensitivity of the CT scan was 100%, with positive predictive value of 10%, negative predictive value of 100%, and specificity of 51%. GCS13 or neurological deficit were risk factors for deterioration  | MHI = GCS 13-15<br>Reported that "admission to hospital does not guarantee skilled neurological observation" |
| Sharma <i>et al</i> <sup>73</sup>             | Prospective cohort<br>n=100<br>LOE III-2                | fair          | Is a history of LOC predictive of intracranial injury in this series of MHI pts?   | GCS is a predictor of intracranial injury<br>Duration of LOC more useful as a predictor of intracranial injury than simply presence / absence of LOC  | A brief history of LOC is not predictive of intracranial injury  |
| Shores <i>et al</i> <sup>108</sup><br>2008    | Prospective cohort for (diagnostic)<br>n=170<br>III-1   | fair          | Is the R-WPTAS more accurate compared to the GCS for assessing cognitive impairment in mTBI in the ED setting, using neuropsychological testing as the standard?   | The R-WPTAS had 60% sensitivity, 91% specificity; GCS had 13% sensitivity, 98% specificity for identifying cognitive impairment in mTBI pts in the ED department.   |  |

| Author and year                           | Study type & LOE                          | Study quality | Study question / objective  | Study outcomes/findings relevant to question  | Comments   |
|---|---|---------------|---|---|--|
| Sifri <i>et al</i> <sup>135</sup><br>2006 | Prospective cohort<br>n=130<br>LOE III-3  | good          | In MHI pts with evidence of an intracranial bleed on CT and no subsequent neurological deterioration, does a routine CT scan within 24hrs change management?  | 99/130 pts had an intracranial bleed identified on initial CT scan with normal subsequent neurological observations. On repeat CT scanning none of these 99 pts required neurosurgical intervention or a change in management.  | MHI = GCS 13-15  |
| Smits <i>et al</i> <sup>54</sup><br>2005  | Prospective cohort<br>n=3181<br>LOE II    | good          | Are the New Orleans Criteria (NOC) and the Canadian CT Head Rule (CCHR) equally sensitive & specific for identifying the need for neurosurgery, the presence intracranial traumatic CT findings or clinically important CT findings in MHI pts? | Sensitivity for predicting neurosurgery was 100% for both the NOC and the CCHR. The NOC had greater sensitivity for intracranial traumatic CT findings and clinically important CT findings (97.7%, 99.4%) than the CCHR (83.4%, 87.2%). Specificity was lower for the NOC for intracranial trauma & clinically important CT findings (3.0%, 5.6%) than for the CCHR (37.2%, 39.7%)   | MHI = GCS 13-15<br>The estimated reduction in CT scanning for MHI pts using the NOC would be 3.0%, using the CCHR would be 37.3%<br>This study included additional risk factors as well as those in the NOC and the CCHR (anticoagulation status, posttraumatic seizure, neurological deficit)   |
| Smits <i>et al</i> <sup>47</sup><br>2007  | Prospective cohort<br>n=3181<br>LOE III-1 | good          | Which clinical risk factors predict the presence of a clinically important intracranial injury on CT scanning in patients with MHI with or without LOC?<br>(MHI GCS13-15)   | CT required if:<br>Any one of the following:<br>Pedestrian/cyclist vs vehicle,<br>Ejected from vehicle,<br>Vomiting<br>Posttraumatic amnesia ≥ 4 hours,<br>Clinical signs of skull fracture,<br>GCS < 15,<br>GCS drop ≥2 points after presentation,<br>Anticoagulation therapy,<br>Posttraumatic seizure,<br>Age ≥ 60 years.<br>At least two of the following:<br>Fall from any height,<br>Persistent anterograde amnesia,<br>Posttraumatic amnesia 2 – 4 hours,<br>Contusion of the skull,<br>Neurologic deficit,<br>LOC,<br>GCS drop of 1point (1h post presentation),<br>Age 40 – 60 years | Did not use LOC or amnesia in the definition of MHI.<br>Noted that an accurate history of LOC, amnesia and dangerous mechanism are difficult to obtain.<br>Identified prolonged posttraumatic amnesia as major risk factor<br>Limitations included CHIP Rule not yet externally validated and any rule should only be used as decision-support tool to aid clinical judgment |

| Author and year                           | Study type & LOE  | Study quality | Study question / objective  | Study outcomes/findings relevant to question  | Comments  |
|---|---|---------------|---|---|---|
| Smits <i>et al</i> <sup>159</sup><br>2007 | Prospective cohort<br>n=2462<br>LOE III-2                                   | good          | Should LOC and/or PTA be included in the definition of MHI?   | Intracranial injury was more common in MHI pts with LOC or PTA than those without (8.7% vs 4.9% p=0.001), however the rate of neurosurgical intervention was the same in both groups (0.5%)   | MHI group included those with an admission GCS of 13-15. Significant intracranial injury may occur in the absence of LOC or amnesia. LOC and PTA should not be included in the definition of MHI. |
| Stein <i>et al</i> <sup>180</sup><br>2006 | Systematic review of cohort studies & case series<br>n=40,000<br>LOE IV     | fair          | Cost-effectiveness study comparing the following strategies for identifying MHI pts that require CT scanning:<br>1. Observation (6hrs ED)<br>2. Admission of all MHI pts for 24h<br>3. Skull radiography<br>4. Selective CT based on CCHR<br>5. Universal CT scanning | Adverse outcomes for a 'missed' or delayed diagnosis of intracranial haemorrhage were calculated as more costly than universal screening of all MHI pts, based on analysis using a 'model' 20yr old male MHI pt                                       |   |
| Stein <i>et al</i> <sup>181</sup><br>2008 | Systematic review of cohort studies & case series<br>n=28 studies<br>LOE IV | fair          | Cost-effectiveness review of routine serial CT scanning vs CT scanning after signs of neurological deterioration for MHI pts presenting with an intracranial lesion on initial CT scan  | Calculations based on the model of a 20year old MHI pt.<br>Routine serial CT scanning is slightly more effective (not stat sig) than waiting for neurological deterioration. The benefit of routine CT serial scanning increases with increasing age. | MHI = GCS 14-15<br>Results also depend on frequency & comprehensive nature of neurological observations, availability & cost of CT scanning, and time taken to act on abnormal CT findings.       |

| Author and year                                     | Study type & LOE   | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments   |
|---|--|---------------|---|--|--|
| Stein <i>et al</i> <sup>48</sup><br>2009            | Retrospective analysis of cohort<br>n=7955<br>LOE III-2  | good          | Comparison of 6 clinical decision instruments for identifying MHI pts requiring a CT scan; Canadian CT Head Rule (CCHR), Neurotraumatology Committee of the World Federation of Neurosurgical Societies (NCWFNS), New Orleans (NOC), National Emergency X-Ray Utilisation Study (NEXUS-II), National Institute of Clinical Excellence (NICE), Scandinavian Neurotrauma Committee (Scandinavian) | The 6 decision instruments' sensitivities for predicting surgical haematomas could not be statistically distinguished (Range 98.1% - 100.0%)<br>Sensitivity for any intracranial lesion was highest for Scandinavian (95.7%) Specificity was also highest for Scandinavian (52.9%). NEXUS II or Scandinavian favoured in discussion. | MHI = GCS 14-15<br>Most clinical decision rules performed well but need to be used to assist clinical judgment.  |
| Stiell <i>et al</i> <sup>37</sup><br>1997           | Retrospective, multicentre cohort<br>n=1699<br>LOE III-3 | fair          | Description of the use of CT scanning for MHI pts with a GCS 13-15 with LOC/amnesia   | In this cohort, 6.2% had abnormal findings on CT (0.5% extradural). There was significant variability in the use of CT scanning for between hospitals.   | Routine CT scanning approach missed as many or more patients with significant injury as clinically guided approach. Authors concluded that CT scanning was not necessarily useful if use is not standardised; a clinical decision rule was needed. |
| Stiell <i>et al</i> <sup>35</sup><br>2001<br>(CCHR) | Prospective cohort<br>n=3121<br>LOE II                   | good          | Validation of Canadian CT Head Rule (CCHR) 'high risk' criteria in MHI (GCS 13 – 15 with LOC or amnesia) pts  | CCHR was 100% sensitive, 69% specific for predicting need for neurosurgery, using high risk criteria.<br>High risk and medium risk criteria together were 98% sensitive and 50% specific for clinically important brain injury   | High risk factors [OR]:<br>GCS <15 @ 2h post injury[7.3]<br>Base of skull fracture [5.2]<br>Other skull fracture [3.6]<br>Age >65yrs [4.1]<br>Emesis [3.8]<br>Medium Risk [OR];<br>Dangerous mechanism [2.8]<br>Retrograde amnesia [1.4]           |

| Author and year                              | Study type & LOE                           | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments   |
|--|--|---------------|--|--|--|
| Stiell <i>et al</i> <sup>56</sup><br>2005    | Prospective cohort<br>n=2707<br>LOE III-1  | good          | Comparison of NOC and CCHR for predicting adverse outcomes in MHI pts  | NOC and CCHR both had 100% sensitivity for neurosurgery; NOC had 12.1% specificity, CCHR had 76.3% specificity<br>NOC and CCHR both had 100% sensitivity for clinically important brain injury; NOC specificity was 12.7%, CCHR specificity was 50.6%              | MHI = GCS 13-15 with NO LOC<br><br>Use of the CCHR would result in lower CT rates (52.15 vs 88.0%)   |
| Teasdale <i>et al</i> <sup>74</sup><br>1990  | Prospective cohort<br>n=8406<br>LOE III-2  | fair          | Determination of the factors influencing the risk of an acute traumatic intracranial haematoma in children and adults with a recent head injury              | Initial GCS <15, skull fracture or GCS15 PLUS LOC/amnesia were associated with an increased risk of an acute intracranial haematoma  | History of LOC/amnesia with an initial GCS of 15 was only a minor absolute risk (1 in 6663 vs 1 in 31370 if no LOC/amnesia)  |
| Turedi <i>et al</i> <sup>81</sup><br>2006    | Prospective cohort<br>n=240<br>LOE III-2   | fair          | In a cohort of pts with a GCS of 13-15 at presentation, what clinical factors predicted an intracranial lesion on CT?  | MHI pts with a GCS of 13 or 14 were significantly more likely to have an intracranial lesion (86%, 59%) than those with a GCS of 15 (24%) p<0.0005   | Cohort included adults and children  |
| Velmahos <i>et al</i> <sup>136</sup><br>2006 | Retrospective cohort<br>n=179<br>LOE III-3 |               | Is routine repeat CT scanning indicated for MHI pts with an initial CT indicating traumatic pathology and no subsequent signs of neurological deterioration? | 7/179 MHI pts with an initial CT+ required neurosurgery, all 7 showed clinical signs of neurological deterioration. A further 30/179 MHI pts showed signs of evolution of intracranial injury on repeat CT; however none required a change in clinical management. | MHI = GCS 13-15<br>GCS 13 or 14, multiple intracranial lesions, or time to initial CT <90mins post hospital arrival all independently predicted a worse repeat CT result                 |
| Vilke <i>et al</i> <sup>75</sup><br>2000     | Prospective case series<br>n=58<br>LOE IV  | fair          | In this series of MHI pts, would an initial GCS of 15 and a normal comprehensive neurological assessment identify all pts with intracranial injury?          | 2 of the 3 pts with acute intracranial injuries had normal neurological examinations   | Significant brain injury and need for CT scanning cannot be excluded in patients with minor head injury despite a GCS = 15 and normal complete neurological examination on presentation. |



**Evidence Table 2: What are the clinically important complications of mild CHI?**

| Author and year                                      | Study type & LOE   | Study quality | Study question / objective  | Study outcomes/findings relevant to question  | Comments  |
|--|--|---------------|---|---|---|
| Af Geijerstam <i>et al</i> <sup>46</sup><br>2003     | Meta-analysis of comparative studies and case series (n=24249)<br>LOE IV | fair          | Estimate of the incidence of complications, mortality and pathological CT findings in MHI pts | Mortality = 0.1%<br>Complications (neurosurgery)=0.9%<br>Pathological findings on CT=8%   | MHI= GCS15 on admission<br>Meta-analysis included case series, hence lower LOE                                |
| Alves <i>et al</i> <sup>93</sup><br>1986             | Cohort (n=847)<br>LOE III-2  | fair          | Description of post-traumatic symptoms in patients following MHI                              | Persistent headache most commonly reported symptom.<br>Post-concussive symptoms generally resolve by three months post-injury.  |   |
| Carroll <i>et al</i> <sup>182</sup><br>2004<br>(WHO) | Meta-analysis of comparative studies<br>LOE III-3                        | good          | Prognosis following MHI   | Majority of adults have resolution of post-concussive symptoms within 3 months<br>Mortality rates post MHI 0.0 – 0.9%   | No definition of MHI<br>Unable to provide pooled estimates due to heterogeneity of studies                    |
| Chambers <i>et al</i> <sup>142</sup><br>1996         | Prospective cohort<br>n=129<br>LOE III-2                                 | fair          | Assessment of post-injury symptoms/complications in MHI pts.                                  | Post-concussive symptoms are commonly reported and gradually reduce with time.<br>Headaches or memory problems are most frequently reported.  |   |
| Cushman <i>et al</i> <sup>8</sup><br>2001            | EAST guidelines  | Recommended   | Systematic review based guidelines to facilitate the management of MTBI                       | Mild cognitive impairment is common and generally resolves within 1 month post-injury (LOEII)<br>Pts with post-concussive symptoms persisting > 6/52 should undergo formal neuropsychological testing (LOEII)   | Persistent post concussive symptoms may identify a subgroup at increased risk of prolonged cognitive deficits |
| Franko <i>et al</i> <sup>78</sup><br>2006            | Retrospective analysis of case series (n=1493)<br>LOE IV                 | fair          | Effect of age and anticoagulant therapy on mortality risk post TBI                            | Mortality of TBI pts >70 years is significantly higher than TBI pts <70 (p<0.001)<br>Anticoagulated TBI patients have a six-fold higher risk of mortality than non-anti-coagulated pts.<br>Linear relationship between increased INR and increased mortality in TBI pts , especially with INR>4 | Included all TBI patients   |

| Author and year                               | Study type & LOE                            | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments  |
|---|---|---------------|---|--|---|
| Hughenoltz <i>et al</i> <sup>94</sup><br>1988 | Prospective cohort<br>(n=22)<br>LOE III-2   | fair          | Determination of time to resolution of symptoms post MHI  | Headaches and mild cognitive impairment with attention and information processing deficits most common symptoms. These are reported to gradually resolve over the 3 months post-injury   |   |
| Kraus <i>et al</i> <sup>95</sup><br>2009      | Cohort<br>(n=2005)<br>LOE III-2             | good          | What are the sequelae of MHI pts at 3 months post-injury compared with ED patients without a head injury? | MHI patients report significantly more post-concussive symptoms and decreased sleep quality compared with control group. (Rivermead post-concussion symptoms questionnaire)  | MHI=GCS13-15 + LOC<br>All subjects between 18- 64 yrs age   |
| Lannsjö <i>et al</i> <sup>96</sup><br>2009    | Cohort<br>(n= 2523)<br>LOE III-2            | fair          | What is the prevalence of post-concussive symptoms three months post MHI?                                 | 56% reported no post-concussive symptoms, 24% reported 3 or more symptoms, 10% reported 7 or more symptoms   | MHI=GCS15 + LOC   |
| Lee <i>et al</i> <sup>69</sup><br>1995        | Prospective cohort<br>(n=1812)<br>LOE III-2 | fair          | Three month outpatient follow up of MHI patients (GCS 15)   | 57% of patients that deteriorated post-MHI did so within 24hrs post injury. Persistent lethargy, emesis and headache were early predictors of deterioration. At 3 months post-injury most pts had good outcome                     | Initial GCS 15  |
| Nell <i>et al</i> <sup>109</sup><br>2000      | Prospective cohort<br>n=561<br>LOE III-2    | fair          | Evaluation of the extended Glasgow Coma Scale for assessing amnesia in MHI patients                       | The extended GCS is a easily used by clinicians and may be a potentially useful tool to flag MHI pts at increased risk of cognitive impairment   |   |
| Rimel <i>et al</i> <sup>98</sup><br>1981      | Cohort<br>(n=538)<br>LOE III-2              | fair          | An evaluation of disability levels 3 months post-injury   | 79% reported persistent headaches, 59% reported persistent memory problems, 34% remained unemployed.   | Initial GCS 13-15   |
| Savola <i>et al</i> <sup>183</sup><br>2003    | Prospective cohort<br>n=172<br>LOE III-2    | fair          | Evaluation of potential early clinical predictors for post concussive symptoms in MHI pts (GCS=15)        | Best predictors for post concussive symptoms (at 1 month post injury) were skull fracture, dizziness on admission or headache on admission. Serum S100B was also found to be a good specific predictor of post concussive symptoms | Initial GCS and duration of PTA were not reported as good predictors of post concussive symptoms at 1 month post injury |

| Author and year                              | Study type & LOE                           | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments   |
|--|--|---------------|--|--|--|
| Thornhill <i>et al</i> <sup>99</sup><br>2000 | Prospective cohort<br>n=549<br>LOE III-2   | fair          | One year follow-up of head injury patients (mild, moderate & severe) from one hospital   | For CHI pts with no pre-existing problems ~ 1/3 had failed to achieve a good outcome at 12 months post injury. Primary dysfunctions were cognitive, behavioural & employment associated.   | Significant disability occurred in the undifferentiated GCS 13-15 group defined as MHI in this cohort. |
| Vos <i>et al</i> <sup>10</sup><br>2002       | EFNS guidelines                            | Recommended   | Systematic review based guidelines to facilitate the management of mild traumatic brain injury   | Pts with high risk mild head injury admitted to hospital should have outpatient follow up<br>Post concussive symptoms are common but usually resolve by 3-6 months   | Post concussive symptoms persisting after 6 months may benefit from neuropsychological testing.        |
| Williams <i>et al</i> <sup>76</sup><br>1990  | Prospective cohort<br>(n=215)<br>LOE III-2 | good          | Comparison of neurobehavioural outcomes for uncomplicated MHI (GCS 13-15), complicated MHI (GCS 13-15) with brain injury or skull fracture and moderate head injury (GCS 9-12). Follow up was over six months. | Outcome for uncomplicated MHI was better than for complicated MHI or moderate head injuries (which were similar). Increasing age was associated with poorer outcome.<br>An abnormal intracranial lesion was more predictive of poor outcome than an isolated skull fracture. |  |

**Evidence Table 3: What is the optimal management strategy for high-risk MHI patients when CT scanning is not available?**

| Author and year                                   | Study type & LOE   | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments |
|---|--|---------------|---|--|----------|
| Borg <i>et al</i> <sup>101</sup><br>2004<br>(WHO) | Systematic review of 73 comparative studies, no meta-analysis<br>LOE III-3 | good          | What is the treatment for high-risk MHI patients when CT scanning is not available? | Closed head injury pts with an admission GCS of 15 plus any of the following risk factors should be admitted for observation:<br>Age >60yrs, dangerous mechanism of injury, suspected skull fracture, signs of supra-clavicular trauma, anterograde amnesia, emesis, headache, seizure, drug or alcohol intoxication |          |

**Evidence Table 4: What are the proven treatments for moderate to severe head injury?**

| Author and year                               | Study type & LOE                      | Study quality        | Study question / objective   | Study outcomes/findings relevant to question   | Comments |
|---|---------------------------------------|----------------------|--|--|----------|
| Alderson <i>et al</i> <sup>16</sup><br>2005   | Systematic review<br>n=11792<br>LOE I | good                 | Do corticosteroids improve any outcomes for acute brain injury patients compared with no corticosteroids?    | The increase in mortality with steroids in the one trial (CRASH 2005) suggests that steroids should no longer be routinely used in people with traumatic head injury.<br>Not recommended.  |          |
| Arango <i>et al</i> <sup>17</sup><br>2008     | Systematic review<br>n=574<br>LOE I   | good                 | Does magnesium improve mortality or GOS outcomes for acute brain injury patients compared with no magnesium? | Insufficient evidence showing improved patient outcomes to make a recommendation   |          |
| Bennett <i>et al</i> <sup>18</sup><br>2004    | Systematic review<br>LOE I            | good                 | To assess the benefits and harms of adjunctive HBOT for treating traumatic brain injury.                     | Insufficient evidence showing improved patient outcomes to make a recommendation   |          |
| Brain Trauma Foundation <sup>15</sup><br>2007 | Brain Trauma Foundation Guidelines    | Strongly Recommended | Systematic review of the management & prognosis of severe traumatic brain injury                             | <p>Recommendations:</p> <p>OVERALL MANAGEMENT STRATEGIES:</p> <ul style="list-style-type: none"> <li>- Organised trauma systems</li> <li>- Initial ABCDE resuscitation fundamental to successful neurological outcome</li> <li>- Prevention of 2o brain injury from hypoxaemia or hypotension crucial to outcome</li> <li>- Specific therapy aimed at raised intracranial pressure should not interfere with systemic resuscitation</li> </ul> <p>HYPOXAEMIA / HYPOTENSION:</p> <ul style="list-style-type: none"> <li>- Systemic hypoxaemia (SaO<sub>2</sub>&lt;90) &amp; hypotension (SBP&lt;90) following head injury are both associated with poor outcome</li> <li>- Adequate oxygenation &amp; fluid resuscitation should be the priority in multiply injured patients</li> </ul> <p>ICP MONITORING:</p> <ul style="list-style-type: none"> <li>- ICP monitoring should be used as a guide to optimise cerebral perfusion</li> </ul> |          |

| Author and year | Study type & LOE | Study quality | Study question / objective | Study outcomes/findings relevant to question  | Comments |
|-----------------|------------------|---------------|----------------------------|---|----------|
|                 |                  |               |                            | <ul style="list-style-type: none"> <li>- Specific indications for ICP monitoring include:               <ul style="list-style-type: none"> <li>(i) GCS 3-8 with abnormal CT scan</li> <li>(ii) GCS 3- 8 with normal scan if two of the following factors are also present – age &gt;40yrs, motor posturing or SBP&lt;90mmHg</li> </ul> </li> <li>SUPPORTIVE CARE ABCDEs:               <ul style="list-style-type: none"> <li>- Supportive care with attention to stabilising ABCDE, adequate nutrition, appropriate posture (30o head up), basic nursing care and prevention of complications has been shown to be more effective than most other interventions. Full nutritional replacement should be commenced by 7 days post injury. Mechanical DVT prophylaxis should be commenced.</li> </ul> </li> <li>ANTICONVULSANTS:               <ul style="list-style-type: none"> <li>- Anticonvulsants such as phenytoin are effective at preventing early posttraumatic seizures but do not prevent late posttraumatic seizures</li> </ul> </li> <li>HYPERVENTILATION:               <ul style="list-style-type: none"> <li>- Routine hyperventilation (PaCO<sub>2</sub>&lt;35mmHg) is associated with poor outcome &amp; should be avoided</li> <li>- Acute hyperventilation (PaCO<sub>2</sub> 25 - 35mmHg) has been shown to be effective for short term reduction of raised ICP associated with acute neurological deterioration.</li> </ul> </li> <li>MANNITOL:               <ul style="list-style-type: none"> <li>- Mannitol (0.5 – 1.0g/kg) is effective at reducing raised ICP</li> <li>- Care should be taken to avoid hypovolaemia or arterial hypotension (SBP&lt;90mmHg)</li> <li>- Mannitol should be largely reserved for pts with acute neurological deterioration.</li> </ul> </li> </ul> |          |

| Author and year                              | Study type & LOE  | Study quality | Study question / objective  | Study outcomes/findings relevant to question  | Comments |
|--|---|---------------|---|---|----------|
|  |   |               |   | <p>THERAPIES NOT SHOWN TO BE EFFECTIVE IN ACUTE MANAGEMENT:</p> <ul style="list-style-type: none"> <li>- barbiturates</li> <li>- steroids</li> </ul> <p>THERAPIES WITH INSUFFICIENT EVIDENCE TO MAKE A CLEAR RECOMMENDATION:</p> <ul style="list-style-type: none"> <li>- hypertonic saline</li> <li>- therapeutic hypothermia</li> <li>- prophylactic antibiotics</li> <li>- pharmacological DVT prophylaxis</li> </ul> <p>FACTORS ASSOCIATED WITH A POOR PROGNOSIS:</p> <ul style="list-style-type: none"> <li>- GCS (Lower GCS = worse outcome with motor component most predictive)</li> <li>- Age &gt; 60yrs (trend)</li> <li>- Absent pupillary reflexes (when systemic ABCDE causes eliminated)</li> <li>- Hypotension (SBP &lt; 90mmHg)</li> <li>- Hypoxaemia (SaO<sub>2</sub> &lt; 90%)</li> </ul> |          |
| Fleminger <i>et al</i> <sup>19</sup><br>2006 | Systematic review<br>LOE I                              | good          | To evaluate the effects of drugs for agitation and/or aggression following acquired brain injury  | $\beta$ -blockers may be effective in reducing aggression and agitation in the long term  |          |
| Forsyth <i>et al</i> <sup>20</sup><br>2010   | Systematic review<br>LOE I                              | good          | Is routine ICP monitoring in acute coma beneficial compared with no ICP monitoring?   | <p>No randomised controlled studies of ICP monitoring by invasive or semi-invasive means in acute coma (traumatic or non-traumatic aetiology) versus no ICP monitoring (that is, clinical assessment of ICP) were located.</p> <p>There is insufficient evidence to make a recommendation</p>   |          |
| Kalina <i>et al</i> <sup>184</sup><br>2008   | Prospective cohort with historical control<br>LOE III-3 | fair          | Does the administration of a prothrombin complex concentrate compared to administration of vitamin K and FFP, improve outcomes for haemorrhagic brain injury pts on warfarin therapy with a raised INR? | <p>The prothrombin group had decreased time to INR normalisation, increased rate of reversal of coagulopathy, decreased time to operative intervention.</p> <p>There were no differences in ICU stay or mortality.</p>  |          |

| Author and year                               | Study type & LOE                          | Study quality | Study question / objective   | Study outcomes/findings relevant to question  | Comments |
|---|---|---------------|--|---|----------|
| Ker <i>et al</i> <sup>21</sup><br>2008        | Systematic review<br>LOE I                | good          | To assess the safety and effectiveness of beta-2 receptor antagonists for TBI.   | Insufficient evidence showing improved patient outcomes to make a recommendation  |          |
| Langham <i>et al</i> <sup>22</sup><br>2003    | Systematic review<br>LOE I                | good          | To estimate the effects of calcium channel blockers in patients with acute traumatic brain injury  | There is insufficient evidence showing improved patient outcomes to make a recommendation   |          |
| Morgalla <i>et al</i> <sup>185</sup><br>2008  | Prospective case series<br>n=33<br>LOE IV | fair          | What are the outcomes at 3yrs post-surgery for deteriorating severe head injury pts with uncontrollable ICP >30mmHg, systolic flow only on T/C Doppler and <60yrs age who undergo decompressive craniectomy? | 19/33 pts recovered completely, or with mild deficits. 7/33 died, 7/33 permanent vegetative state.<br>Younger age was associated with better outcomes.  |          |
| Roberts <i>et al</i> <sup>25</sup><br>1999    | Systematic review<br>LOE I                | good          | To assess the effects of barbiturates in reducing raised ICP, mortality and morbidity in acute traumatic brain injury  | There is no evidence that barbiturates improve any patient outcomes, and may cause hypotension.   |          |
| Roberts <i>et al</i> <sup>24</sup><br>2009    | Systematic review<br>LOE I                | good          | To quantify the effect of hyperventilation on death and neurological disability following head injury.   | The data available are inadequate to assess any potential benefit or harm that might result from hyperventilation in severe head injury.<br>There is insufficient evidence to make a recommendation |          |
| Sahuquillo <i>et al</i> <sup>26</sup><br>2006 | Systematic review<br>LOE I                | good          | To assess the effects of secondary decompressive craniectomy on outcome and quality of life in patients with severe TBI in whom conventional medical therapeutic measures have failed to control raised ICP  | Possible benefit in paediatric population but insufficient evidence showing improved patient outcomes to make recommendations   |          |



| Author and year                               | Study type & LOE  | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments |
|---|---|---------------|---|--|----------|
| Scheirhout <i>et al</i> <sup>27</sup><br>2001 | Systematic review<br>LOE I                              | good          | To determine the effects of prophylactic anti-epileptic agents for acute traumatic head injury  | Prophylactic anti-epileptics are effective in reducing early seizures, but there is no evidence that prophylactic anti-epileptics reduce the occurrence of late seizures, or have any effect on death and neurological disability. Insufficient evidence is available to establish the net benefit of prophylactic treatment at any time after injury. |          |
| Shafi <i>et al</i> <sup>186</sup><br>2008     | Multicentre retrospective cohort<br>n=1646<br>LOE III-3 | fair          | Is the use of ICP monitoring in severe head injury patients (GCS 3-6) associated with better outcomes compared to no ICP monitoring?  | After adjusting for GCS, age, BP, head AIS, ISS, ICP monitoring was associated with a 45% reduction in survival.   |          |
| Sydenham <i>et al</i> <sup>28</sup><br>2009   | Systematic review<br>LOE I                              | good          | To estimate the effect of mild hypothermia for traumatic head injury on mortality and long-term functional outcome complications  | Possible benefit but insufficient evidence showing improved patient outcomes to make recommendations   |          |
| Wakai <i>et al</i> <sup>29</sup><br>2007      | Systematic review<br>LOE I                              | good          | To assess the effects of different mannitol therapy regimens, of mannitol compared to other ICP lowering agents, and to quantify the effectiveness of mannitol administration given at other stages following acute traumatic brain injury. | There are insufficient data on the effectiveness of pre-hospital administration of mannitol.<br>There is insufficient evidence to make a recommendation  |          |
| Willis <i>et al</i> <sup>30</sup><br>2003     | Systematic review<br>LOE I                              | good          | To assess systematically the efficacy of excitatory amino acid inhibitors on improving patient outcome following traumatic brain injury   | Insufficient evidence showing improved patient outcomes to make a recommendation   |          |

**Evidence Table 5: When should patients with a closed head injury be transferred to a hospital with neurosurgical facilities?**

| Author and year                               | Study type & LOE                           | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments   |
|---|--|---------------|---|--|--|
| Devrill <i>et al</i> <sup>187</sup><br>2007   | Retrospective cohort<br>n=261<br>LOE III-3 | fair          | What are the clinical outcomes for patients with an extra-dural haemorrhage presenting to neurosurgical centres compared with patients presenting to hospitals with no neurosurgical facilities?  | All 5 deaths and 4/7 serious disabilities occurred in the group that were transferred from a hospital with no neurosurgical facilities to a neurosurgical centre before undergoing craniotomy. This group had a median time to surgery of 8hrs 5min. 8/9 pts who received emergency burr-hole treatment in non-neurosurgical hospitals had good outcomes | This study was characterised by the especially lengthy inter-hospital transfer times (rural Queensland).   |
| Fabbri <i>et al</i> <sup>41</sup><br>2008     | Prospective cohort<br>n=700<br>LOE III-2   | good          | What are the effects on 6month outcomes for mild and moderate head injury pts with adverse CT findings not requiring immediate surgery when observed in a non-neurosurgical facility compared with those transferred to a neurosurgical centre? | The outcome was unfavourable for 18% of pts transferred to a neurosurgical centre for observation, compared with 10% for pts kept in non-neurosurgical centres for observation (NS: p=0.143)   | Transfer times 30 – 60 minutes.<br>The hospital with no neurosurgical facilities had access to neurosurgical expertise via a teleradiology system<br>MHI GCS 14-15<br>Moderate HI GCS 9 – 13 |
| McConnell <i>et al</i> <sup>188</sup><br>2005 | Retrospective cohort<br>n=542<br>LOE III-2 | fair          | Do CHI pts transferred to US level I trauma centres have a reduced mortality rate relative to matched pts transferred to US level II trauma centres?  | The inter-hospital transfer of CHI pts to level I trauma centres results in a significant mortality benefit (10% reduction) compared to those transferred to a level II trauma centre.   |  |

**Evidence Table 6: When can patients with a MHI be safely discharged home?**

| Author and year                                   | Study type & LOE   | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments  |
|---|--|---------------|--|--|---|
| Borg <i>et al</i> <sup>101</sup><br>2004<br>(WHO) | Systematic review of 73 comparative studies, no meta-analysis<br>LOE III-3 | good          | When can MHI pts be safely discharged home?  | MHI pts with an admission GCS of 15 and NONE of the following risk factors can be safely discharged home without a CT scan:<br>Age >60yrs, dangerous mechanism of injury, suspected skull fracture, signs of supra-clavicular trauma, anterograde amnesia, emesis, headache, seizure, drug or alcohol intoxication<br>Pts with an admission GCS of 15 plus any risk factors may be safely discharged home if a subsequent CT scan is negative. |   |
| Dunham <i>et al</i> <sup>126</sup><br>1996        | Prospective cohort<br>n= 2587<br>LOE III-2                                 | fair          | What clinical risk factors predicted abnormal findings on CT?  | All pts requiring a craniotomy deteriorated within 4hrs of arrival.<br>No pt with a negative initial CT required neurosurgery  | Safe discharge was implied if:<br>(1) Initial GCS 15 and no evidence of a skull fracture, no Neurodeficit, no headache and no emesis<br>(2) Initial GCS 13-14 and negative CT findings, no persistent Neurodeficit, no persistent headache and no persistent emesis |
| Fabbri <i>et al</i> <sup>125</sup><br>2004        | Prospective cohort<br>n=1480<br>LOE III-2                                  | fair          | What were the outcomes for MHI pts discharged to home observation compared with those monitored in hospital? | There was no significant difference in six month adverse outcomes between the two groups   |   |

| Author and year                            | Study type & LOE                                       | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments   |
|--|--|---------------|---|--|--|
| Fabbri <i>et al</i> <sup>31</sup><br>2004  | Prospective, multicentre cohort<br>n=5578<br>LOE III-1 | fair          | Validation of a set of criteria to classify MHI pts into different risk categories for management checked against incidence of post-traumatic lesions, incidence of neurosurgical intervention and clinical outcome at 6/12 | <p><b>Low Risk MHI (n=1676):</b><br/>-1 missed intracranial injury<br/>-52 post-concussive symptoms</p> <p><b>Medium Risk MHI (n=1200):</b><br/>-22 intracranial injuries (0 missed)<br/>-49 post-concussive symptoms</p> <p><b>High Risk MHI (n=2702):</b><br/>-301 intracranial injuries (15 missed)<br/>-76 post-concussive symptoms</p> <p>No association was found between risk category and the frequency of post-concussive symptoms.</p> <p>In pts with negative initial CT scans, 8 cases of delayed intracranial haemorrhage occurred (8/1774) – CT scan may miss delayed subdural haemorrhage, will generally identify extradural haemorrhages.</p> | <p>Safe to discharge MHI pts if:</p> <p>(1) Initial GCS of 15 (without a CT scan) AND all of:<br/>-no LOC<br/>-no headache / emesis<br/>-no neurodeficit<br/>-no skull fracture<br/>-brief ED observation<br/>-no risk factors</p> <p>(2) Initial GCS 15 with LOC/ amnesia or emesis or headache AND all of:<br/>-no neurodeficit<br/>-no skull fracture<br/>-no risk factors<br/>-negative CT scan + brief ED obs OR normal skull x-ray + 24hr hospital obs OR 24hr hospital obs.</p> |
| Fung <i>et al</i> <sup>155</sup><br>2006   | Retrospective analysis                                 | n/a           | Do currently available post MHI discharge forms contain information identifying the 6 evidence-based predictors of intracranial haemorrhage? (ie GCS<15, vomiting, amnesia, headache, seizure, neurodeficit)                | <p>Only one of the 15 forms outlined all 6 of the risk factors.</p> <p>The forms were generally confusing, and none made clear that the primary reason for close observation was to detect a possible haemorrhage.</p>   | Authors give an example of a concise, precise and readable discharge form  |
| Hsiang <i>et al</i> <sup>152</sup><br>1997 | Prospective cohort<br>n=1360<br>LOE III-2              | fair          | What clinical factors define a 'high risk' mild head injury?  | MHI pts with negative CT findings and no fractures on skull x-ray have good outcome (at 6/12) and can be safely discharged from ED   | MHI = GCS 13-15  |

| Author and year                                 | Study type & LOE                                    | Study quality | Study question / objective   | Study outcomes/findings relevant to question   | Comments   |
|---|---|---------------|--|--|--|
| Jagoda <i>et al</i> <sup>9</sup><br>2008        | Clinical policy /<br>systematic review<br>LOE III-2 | fair          | Can a pt with MHI be safely discharged from ED if a non-contrast CT scan shows no acute injuries?  | Excluded were MHI pts with any of the following:<br>GCS < 15<br>Coagulopathy<br>Focal neurological deficit<br><Multi-system trauma | MHI pts can be safely discharged from ED if they have:<br>GCS 15, normal neurological examination, negative CT findings, 6hrs observation, and are discharged to the care of a responsible observer. Detailed written discharge advice should be given.  |
| Lee <i>et al</i> <sup>69</sup><br>1995          | Prospective cohort<br>n=1812<br>LOE III-2           | fair          | What clinical risk factors predicted neurological deterioration in this cohort of MHI pts?   | Risk factors were: Age >60yrs, abnormal mental state, focal neurological deficit, headache or emesis                               | Deterioration most frequently occurred in the first 24hrs after presentation, as a result of an extradural haematoma. Delayed deterioration was generally due to subdural lesions and occurred up to 1/52 later. Initial CT scans may not rule out the risk of deterioration due to SIADH, subdural haematoma or seizure |
| Livingstone <i>et al</i> <sup>131</sup><br>2000 | Prospective cohort<br>LOE III-2                     | fair          | What is the negative predictive value of CT scanning in MHI?<br>Is admission for observation mandatory after a negative diagnostic evaluation for MHI? | 1/1788 MHI pts with negative CT findings deteriorated, subsequently requiring neurosurgical intervention.                          | MHI = GCS 14-15 and LOC/<br>amnesia<br>Safe to discharge pts home if CT findings are negative and the pt shows clinical improvement  |
| Nagy <i>et al</i> <sup>89</sup><br>1999         | Prospective cohort<br>n=1170<br>LOE III-2           | fair          | Can CT scanning identify MHI pts who will deteriorate or who have an intracranial lesion?  | No MHI pts who had a negative initial CT scan deteriorated in the following 24hrs  | MHI (GCS15) pts can be safely discharged home if their initial CT findings are negative.   |

| Author and year                              | Study type & LOE  | Study quality | Study question / objective  | Study outcomes/findings relevant to question   | Comments   |
|--|---|---------------|---|--|--|
| Shackford <i>et al</i> <sup>53</sup><br>1992 | Multicentre retrospective cohort<br>n=2166<br>LOE III-2       | fair          | What is the risk of a MHI pt with a normal neurological examination & negative CT scan having an intracranial lesion requiring surgical intervention? | None of the 1170 pts with normal findings on CT required neurosurgical intervention. 59/2112 pts with a normal neurological examination subsequently underwent a craniotomy<br>Significant risk factors were; GCS 13 or focal neurological deficit | Reliable pts with a MHI and a normal neurological examination and negative CT scan can be safely discharged.<br>The sensitivity of the CT scan to detect neurosurgical lesions was 100%, with positive predictive value of 10%, negative predictive value of 100%, and a specificity of 51%. |
| Stein <i>et al</i> <sup>189</sup><br>1990    | Retrospective cohort<br>n=658<br>LOE III-3                    | fair          | Do MHI patients with no abnormal findings on CT clinically deteriorate  | None of the 542/658 pts who had a MHI and no abnormalities on CT deteriorated  | It is safe to discharge MHI pts if their CT findings and neurological examination are normal.<br>MHI = GCS13-15 & LOC/amnesia  |
| Stein <i>et al</i> <sup>90</sup><br>1992     | Retrospective cohort<br>n=1538<br>LOE III-2                   | fair          | Do MHI patients with no abnormal findings on CT clinically deteriorate?   | None of the 1339 / 1538 pts who had a MHI and no abnormalities on CT deteriorated  | It is safe to discharge MHI pts if their CT findings and neurological examination are normal.<br>MHI = GCS13-15 & LOC/amnesia  |
| Taheri <i>et al</i> <sup>190</sup><br>1993   | Retrospective cohort<br>n=310<br>LOE III-3                    | fair          | Which MHI pts could have been safely discharged home from the emergency department?   | MHI (GCS 15) pts who required neurosurgical intervention had either a skull fracture or a neurological deficit.  | Safe discharge required GSC of 15, no skull fracture (clinically or radiologically) and no neurological deficit  |
| Teasdale <i>et al</i> <sup>74</sup><br>1990  | Prospective cohort<br>n=8406 (children & adults)<br>LOE III-2 | fair          | What were the clinical predictors of intracranial haematoma?  | Best predictors of intracranial haematoma were: abnormal level of consciousness, focal neurological deficit or a skull fracture  | MHI pts can be safely discharged if they have an initial GCS of 15, no focal deficit and no skull fracture   |

## Appendix 1: Definitions of mild head injury

| Study                                      | Source   | Terminology                 | Initial GCS | Inclusion criteria                      | Exclusion criteria  | Clinical findings associated with sub-classification of increased risk of intracranial injury within mild head injury category   |
|--|--|-----------------------------|-------------|---|---|--|
| Haydel <i>et al</i> <sup>32</sup><br>2000  | Prospective study<br>New Orleans Criteria (NOC)    | Minor head injury           | 15          | LOC/amnesia                             | Minimal head injury (no LOC/amnesia)<br>Penetrating head injury<br>Neurodeficit. Coagulopathy (insufficient enrolled to assess)                 | Seizure, emesis, drug or alcohol intoxication, evidence of supra-clavicular trauma, headache, age >60 years, deficits in short-term memory   |
| Cushman <i>et al</i> <sup>8</sup><br>2001  | Guideline (EAST)                                   | Mild traumatic brain injury | 13-15       | Amnesia<br>LOC <20min<br>Normal CT scan | Minimal head injury<br>Penetrating head injury<br>Neurodeficit<br>Seizure   | Used CT scanning to define mild head injury  |
| Stiell <i>et al</i> <sup>35</sup><br>2001  | Prospective study<br>Canadian CT Head Rules (CCHR) | Minor head injury           | 13-15       | LOC/amnesia                             | Minimal head injury (no LOC/amnesia)<br>Penetrating head injury<br>Neurodeficit<br>Seizure<br>Coagulopathy<br>Representation<br>Unstable vitals | Suspected open skull fracture, signs of basal skull fracture, failure to reach GCS 15 within 2hrs, emesis ≥ two episodes, age ≥ 65 years, anterograde amnesia >30minutes or dangerous mechanism of injury. |
| Servadei <i>et al</i> <sup>6</sup><br>2001 | Guideline (W.F.N.S.)                               | Mild head injury            | 14-15       | All                                     | Penetrating head injury   | Sub-classification into mild, medium and high risk based on:<br>(i) initial GCS<br>(ii) LOC/amnesia<br>(iii) risk factors  |
| Vos <i>et al</i> <sup>10</sup><br>2002     | Guideline (EFNS)                                   | Mild traumatic brain injury | 13-15       | LOC <30min<br>PTA <60min                | Penetrating head injury   | Sub-classified categories 0-3 based on:<br>(i) initial GCS<br>(ii) LOC/amnesia<br>(iii) risk factors   |

| Study                                      | Source   | Terminology                 | Initial GCS | Inclusion criteria                      | Exclusion criteria  | Clinical findings associated with sub-classification of increased risk of intracranial injury within mild head injury category   |
|--|--|-----------------------------|-------------|---|---|--|
| Haydel <i>et al</i> <sup>32</sup><br>2000  | Prospective study<br>New Orleans Criteria (NOC)    | Minor head injury           | 15          | LOC/amnesia                             | Minimal head injury (no LOC/amnesia)<br>Penetrating head injury<br>Neurodeficit. Coagulopathy (insufficient enrolled to assess)                 | Seizure, emesis, drug or alcohol intoxication, evidence of supra-clavicular trauma, headache, age >60 years, deficits in short-term memory   |
| Cushman <i>et al</i> <sup>8</sup><br>2001  | Guideline (EAST)                                   | Mild traumatic brain injury | 13-15       | Amnesia<br>LOC <20min<br>Normal CT scan | Minimal head injury<br>Penetrating head injury<br>Neurodeficit<br>Seizure   | Used CT scanning to define mild head injury  |
| Stiell <i>et al</i> <sup>35</sup><br>2001  | Prospective study<br>Canadian CT Head Rules (CCHR) | Minor head injury           | 13-15       | LOC/amnesia                             | Minimal head injury (no LOC/amnesia)<br>Penetrating head injury<br>Neurodeficit<br>Seizure<br>Coagulopathy<br>Representation<br>Unstable vitals | Suspected open skull fracture, signs of basal skull fracture, failure to reach GCS 15 within 2hrs, emesis ≥ two episodes, age ≥ 65 years, anterograde amnesia >30minutes or dangerous mechanism of injury. |
| Servadei <i>et al</i> <sup>6</sup><br>2001 | Guideline (W.F.N.S.)                               | Mild head injury            | 14-15       | All                                     | Penetrating head injury   | Sub-classification into mild, medium and high risk based on:<br>(i) initial GCS<br>(ii) LOC/amnesia<br>(iii) risk factors  |
| Vos <i>et al</i> <sup>10</sup><br>2002     | Guideline (EFNS)                                   | Mild traumatic brain injury | 13-15       | LOC <30min<br>PTA <60min                | Penetrating head injury   | Sub-classified categories 0-3 based on:<br>(i) initial GCS<br>(ii) LOC/amnesia<br>(iii) risk factors   |



| Study                                     | Source   | Terminology                 | Initial GCS | Inclusion criteria             | Exclusion criteria   | Clinical findings associated with sub-classification of increased risk of intracranial injury within mild head injury category   |
|---|--|-----------------------------|-------------|--------------------------------|--|--|
| Jagoda <i>et al</i> <sup>9</sup><br>2002  | Guideline (ACEP)                               | Mild traumatic brain injury | 15          | LOC/amnesia                    | Minimal head injury<br>Penetrating<br>Neurodeficit<br>Coagulopathy<br>Multisystem trauma | Headache, emesis, age ≥ 60 years, deficits in short-term memory, evidence of supra-clavicular trauma, seizure, drug or alcohol intoxication  |
| Fabbri <i>et al</i> <sup>31</sup><br>2004 | Prospective study                              | Mild head injury            | 14-15       | All                            | Penetrating head injury  | As per Servadei <i>et al</i> (2001)  |
| Ibanez <i>et al</i> <sup>33</sup><br>2004 | Prospective , observational                    | Mild head injury            | 14-15       | With or without LOC            |  | MHI patients further sub-classified into:<br>a. Minimal: GCS 15 & no LOC<br>b. Mild: GCS14-15 & LOC<5min or PTA<br>c. Moderate: GCS14-15 & LOC ≥ 5min  |
| Mower <i>et al</i> <sup>58</sup><br>2005  | Prospective study<br>NEXUS II                  | Minor head injury           | 15          | All                            | Penetrating head injury  | Risk factors for intracranial injury identified as any one of the following:<br>Neurological deficit (included GCS<15)<br>Abnormal alertness<br>Abnormal behaviour<br>Persistent vomiting<br>Skull fracture<br>Scalp haematoma<br>Age > 65<br>Coagulopathy   |
| Smits <i>et al</i> <sup>47</sup><br>2007  | Prospective, observational study.<br>CHIP Rule | Minor head injury           | 13 - 15     | With or without LOC or amnesia | Penetrating head injury  | Any one of the following: Pedestrian/cyclist vs vehicle, ejected from vehicle, emesis, posttraumatic amnesia ≥ 4 hours, clinical signs of skull fracture, GCS < 15, GCS drop ≥2 points after presentation, current anticoagulation therapy, posttraumatic seizure, age ≥ 60 years.<br>At least two of the following:<br>Fall from any height, persistent anterograde amnesia, posttraumatic amnesia 2 – 4 hours, contusion of the skull, neurologic deficit, LOC, GCS drop of 1point (1h post presentation), age 40 – 60 years |

| Study                                    | Source   | Terminology                    | Initial GCS | Inclusion criteria   | Exclusion criteria  | Clinical findings associated with sub-classification of increased risk of intracranial injury within mild head injury category   |
|--|--|--------------------------------|-------------|--|---|--|
| Jagoda <i>et al</i> <sup>9</sup><br>2008 | Guideline<br>ACEP / CDC  | Mild traumatic<br>brain injury | 14 - 15     | Presentation<br>within 24h post<br>injury with or<br>without LOC or<br>amnesia | Penetrating head injury   | LOC with any one of the following:<br>Headache, vomiting, Age > 60y, drug or alcohol<br>intoxication, deficit in short-term memory, physical<br>evidence of supra-clavicular trauma, posttraumatic<br>seizure, GCS<15, focal neurologic deficit, coagulopathy.<br>No LOC with any one of the following:<br>Focal neurologic deficit, severe headache, Age ≥65y,<br>physical signs of basilar skull fracture, GCS<15,<br>coagulopathy, dangerous mechanism of injury. |
| Stein <i>et al</i> <sup>48</sup><br>2009 | Prospective<br>study comparing<br>six clinical<br>decision<br>guidelines | Mild traumatic<br>brain injury | 14 – 15     | Presentation<br>within 24h post<br>injury                                      | Penetrating head injury<br>Unstable vital sign<br>Re-presentation | Application of the Nexus II or Scandinavian Guidelines<br>resulted in the highest sensitivity and specificity in this<br>sample.   |

## Appendix 2: Initial GCS versus abnormal CT/Neurosurgery

Summary of studies examining the relationship between initial GCS and frequency of abnormal findings on CT scans and/or neurosurgical intervention.

| Author and Year                       | Level of Evidence | Quality | GCS 13                       |                      |                       | GCS 14                      |                      |                       | GCS 15         |                      |                       |
|---------------------------------------|-------------------|---------|------------------------------|----------------------|-----------------------|-----------------------------|----------------------|-----------------------|----------------|----------------------|-----------------------|
|                                       |                   |         | Patients (No.)               | CT Scan Abnormal (%) | Neuro SX Required (%) | Patients (No.)              | CT Scan Abnormal (%) | Neuro SX Required (%) | Patients (No.) | CT Scan Abnormal (%) | Neuro SX Required (%) |
| Dacey et al <sup>51</sup><br>1986     | III-2             | fair    | 18                           | N/A                  | 33                    | 59                          | N/A                  | 6.5                   | 533            | N/A                  | 1.5                   |
| Teasdale et al <sup>74</sup><br>1990  | III-2             | fair    |                              |                      |                       |                             |                      |                       | 7838           |                      | 0.03                  |
| Shackford et al <sup>53</sup><br>1992 | III-2             | fair    | 221                          | 33                   | 10.8                  | 646                         | 17.5                 | 3.8                   | 1899           | 14.8                 | 3.2                   |
| Stein et al <sup>90</sup><br>1992     | IV                | fair    | 120                          | 37.5                 | -                     | 301                         | 24.2                 | -                     | 1117           | 13.2                 | -                     |
| Jeret et al <sup>68</sup><br>1993     | III-2             | fair    |                              |                      |                       |                             |                      |                       | 712            | 9.4                  | 0.3                   |
| Borcuk <sup>49</sup><br>1995          | III-2             | poor    | 40                           | 27.5                 | 7.5                   | 197                         | 18.2                 | 3.6                   | 1211           | 5.9                  | 0.1                   |
| Dunham et al <sup>36</sup><br>1996    | III-2             | fair    | 1160 Age 14-60<br>13 Age >60 | 25<br>30             | 3.5<br>-              | 435 Age 14-60<br>57 Age >60 | 12.4<br>28           | 0.5<br>-              | 1481 Age 14-60 | 3.0<br>10            | 0.13<br>-             |
| Culotta et al <sup>50</sup><br>1996   | III-2             | fair    | 173                          | 28                   | 4.5                   | 755                         | 16                   | 1.6                   | 150 Age >60    | 4                    | 0.4                   |
| Hsiang et al <sup>52</sup><br>1997    | III-2             | fair    | 45                           | 57.8                 | 20                    | 138                         | 35.5                 | 5.1                   | 2179           | 18.5                 | 2.2                   |

| Author and Year                     | Level of Evidence | Quality | GCS 13         |                      |                       | GCS 14         |                      |                       | GCS 15         |                      |                       |
|-------------------------------------|-------------------|---------|----------------|----------------------|-----------------------|----------------|----------------------|-----------------------|----------------|----------------------|-----------------------|
|                                     |                   |         | Patients (No.) | CT Scan Abnormal (%) | Neuro SX Required (%) | Patients (No.) | CT Scan Abnormal (%) | Neuro SX Required (%) | Patients (No.) | CT Scan Abnormal (%) | Neuro SX Required (%) |
| Miller et al <sup>88</sup><br>1997  | III-2             | fair    | -              | -                    | -                     | -              | -                    | -                     | 2143           | 6.4                  | 0.2                   |
| Nagy et al <sup>89</sup><br>1999    | III-2             | fair    | -              | -                    | -                     | -              | -                    | -                     | 1170           | 3.3                  | 0.34                  |
| Haydel et al <sup>32</sup><br>2000  | II                | good    | -              | -                    | -                     | -              | -                    | -                     | 1429           | 6.5                  | 0.4                   |
| Stiell et al <sup>35</sup><br>2001  | II                | good    | 110            | 41                   | -                     | -              | 17                   | -                     | 2489           | 4.8                  | -                     |
| Ibanez et al <sup>33</sup><br>2004  | II                | good    |                |                      |                       |                | 33                   | -                     | 1050           | 6.3                  | -                     |
| Clement et al <sup>77</sup><br>2006 | III-2             | good    |                |                      |                       |                |                      |                       | 4551           | 7.7                  | 0.6                   |
| Smits et al <sup>47</sup><br>2007   | III-1             | good    | 151            | 20.5                 | 1.3                   | 1.3            | 13.6                 | 0.9                   | 2462           | 5.5                  | 0.4                   |

## Appendix 3: Westmead PTA Scale

The Westmead Post Traumatic Amnesia (PTA) Scale, developed by Shores et al<sup>191</sup> consists of 7 orientation questions and 5 memory items designed to objectively measure the period of PTA. The Westmead PTA Scale is a standardised and prospective measure of PTA. A person is said to be out of PTA if they can achieve a perfect score on the Westmead PTA Scale for 3 consecutive days.

The Westmead PTA Scale form (as seen by the example on the following page) and 9 picture cards are required to perform the test. As the test was designed to measure PTA in a standard fashion to enable comparison of patients from different hospitals, the supplied picture cards must be used. They are available for purchase with instruction on their use from the Department of Rehabilitation Medicine, Westmead Hospital, Westmead NSW 2145 for a minimal fee.

More information is available on the Westmead PTA Scale website at [www.psy.mq.edu.au/pta/index.html](http://www.psy.mq.edu.au/pta/index.html) or email [sue.meares@mq.edu.au](mailto:sue.meares@mq.edu.au)

## Patient Label

Date of Onset: \_\_\_\_\_

1001

1001

Adapted by S.Swan, Queensland Health Occupational Therapy Gold Coast Hospital and Royal Brisbane & Women's Hospital, 2009; J.E., Sandanam, J. & Batchelor, J. (1986). Preliminary validation of a clinical scale for measuring the duration of post-traumatic amnesia. *Medical Journal of Australia*, 144, 569-572.

## Appendix 4: Abbreviated Westmead PTA Scale

### ABBREVIATED WESTMEAD PTA SCALE (A-WPTAS) GCS & PTA testing of patients with MTBI following mild head injury

Abbreviated Westmead PTA Scale (A-WPTAS)  
incorporating Glasgow Coma Scale (GCS)

MRN sticker here

|                |                                  |                             |                          |                          |                          |                          |
|----------------|----------------------------------|-----------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| Date:          |                                  | T1                          | T2                       | T3                       | T4                       | T5                       |
| Time           |                                  |                             |                          |                          |                          |                          |
| Motor          | Obeys commands                   | 6                           | 6                        | 6                        | 6                        | 6                        |
|                | Localises                        | 5                           | 5                        | 5                        | 5                        | 5                        |
|                | Abnormal flexion                 | 4                           | 4                        | 4                        | 4                        | 4                        |
|                | Withdraws                        | 3                           | 3                        | 3                        | 3                        | 3                        |
|                | Extension                        | 2                           | 2                        | 2                        | 2                        | 2                        |
|                | None                             | 1                           | 1                        | 1                        | 1                        | 1                        |
| Eye Opening    | Spontaneously                    | 4                           | 4                        | 4                        | 4                        | 4                        |
|                | To speech                        | 3                           | 3                        | 3                        | 3                        | 3                        |
|                | To pain                          | 2                           | 2                        | 2                        | 2                        | 2                        |
|                | None                             | 1                           | 1                        | 1                        | 1                        | 1                        |
| Verbal         | Oriented **<br>(tick if correct) | 5                           | 5                        | 5                        | 5                        | 5                        |
|                | Name                             | <input type="checkbox"/>    | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
|                | Place                            | <input type="checkbox"/>    | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
|                | Why are you here                 | <input type="checkbox"/>    | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
|                | Month                            | <input type="checkbox"/>    | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
|                | Year                             | <input type="checkbox"/>    | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
|                | Confused                         | 4                           | 4                        | 4                        | 4                        | 4                        |
|                | Inappropriate words              | 3                           | 3                        | 3                        | 3                        | 3                        |
|                | Incomprehensible sounds          | 2                           | 2                        | 2                        | 2                        | 2                        |
|                | None                             | 1                           | 1                        | 1                        | 1                        | 1                        |
|                | <b>GCS</b>                       | <b>Score out of 15</b>      | <b>/15</b>               | <b>/15</b>               | <b>/15</b>               | <b>/15</b>               |
|                | Picture 1                        | Show pictures<br>(see over) |                          |                          |                          |                          |
|                | Picture 2                        |                             |                          |                          |                          |                          |
|                | Picture 3                        |                             |                          |                          |                          |                          |
| <b>A-WPTAS</b> | <b>Score out of 18</b>           |                             | <b>/18</b>               | <b>/18</b>               | <b>/18</b>               | <b>/18</b>               |

#### Use of A-WPTAS and GCS for patients with MTBI

The A-WPTAS combined with a standardised GCS assessment is an objective measure of post traumatic amnesia (PTA).

Only for patients with **current GCS of 13-15 (<24hrs post injury)** with impact to the head resulting in confusion, disorientation, anterograde or retrograde amnesia, or brief LOC. **Administer both tests at hourly intervals** to gauge patient's capacity for full orientation and ability to retain new information. Also, **note the following:** poor motivation, depression, pre-morbid intellectual handicap or possible medication, drug or alcohol effects. **NB:** This is a screening device, so exercise clinical judgement. In cases where doubt exists, more thorough assessment may be necessary.

#### Admission and Discharge Criteria:

A patient is considered to be out of PTA when they score 18/18.

Both the GCS and A-WPTAS should be used in conjunction with clinical judgement.

Patients scoring 18/18 can be considered for discharge.

For patients who do not obtain 18/18 re-assess after a further hour.

Patients with persistent score <18/18 at 4 hours post time of injury should be considered for admission.

Clinical judgement and consideration of pre-existing conditions should be used where the memory component of A-WPTAS is abnormal but the GCS is normal (15/15).

Referral to GP on discharge if abnormal PTA was present, provide patient advice sheet.

#### Target set of picture cards



\*\* must have all 5 orientation questions correct to score 5 on verbal score for GCS, otherwise the score is 4 (or less).

| PUPIL ASSESSMENT | T1 |   | T2 |   | T3 |   | T4 |   | T5 |   | +  | = | REACTS BRISKLY |
|------------------|----|---|----|---|----|---|----|---|----|---|----|---|----------------|
|                  | R  | L | R  | L | R  | L | R  | L | R  | L |    |   |                |
| Size             |    |   |    |   |    |   |    |   |    |   | SL | = | SLUGGISH       |
| Reaction         |    |   |    |   |    |   |    |   |    |   | C  | = | CLOSED         |
|                  |    |   |    |   |    |   |    |   |    |   | -  | = | NIL            |

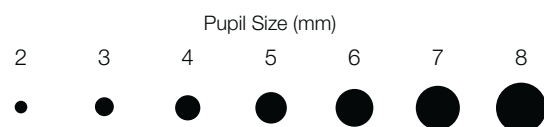
#### Comments

.....

.....

.....

.....



Shores & Lamme (2007) - further copies of this score sheet can be downloaded from <http://www.psy.mq.edu.au/GCS>

## GLASGOW COMA SCALE (GCS) AND ABBREVIATED WESTMEAD PTA SCALE (A-WPTAS)

### Administration and Scoring

#### 1. Orientation Questions

**Question 1: WHAT IS YOUR NAME?**

The patient must provide their full name.

**Question 2: WHAT IS THE NAME OF THIS PLACE?**

The patient has to be able to give the name of the hospital. For example: Westmead Hospital. (NB: The patient does not get any points for just saying 'hospital'.) If the patient can not name the hospital, give them a choice of 3 options. To do this, pick 2 other similar sized hospitals in your local area or neighbouring region. In Westmead Hospital's case the 3 choices are 'Nepean Hospital, Westmead Hospital or Liverpool Hospital'.

**Question 3: WHY ARE YOU HERE?**

The patient must know why they were brought into hospital. e.g. they were injured in a car accident, fell, assaulted or injured playing sport. If the patient does not know, give them three options, including the correct reason.

**Question 4: WHAT MONTH ARE WE IN?**

For emphasis the examiner can ask what month are we in now? The patient must name the month. For example, if the patient answers 'the 6th month', the examiner must ask the further question 'What is the 6th month called?'.

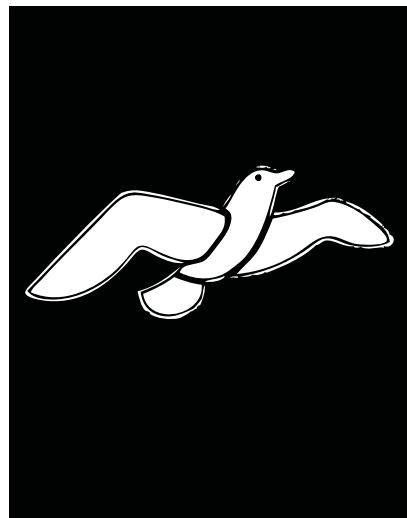
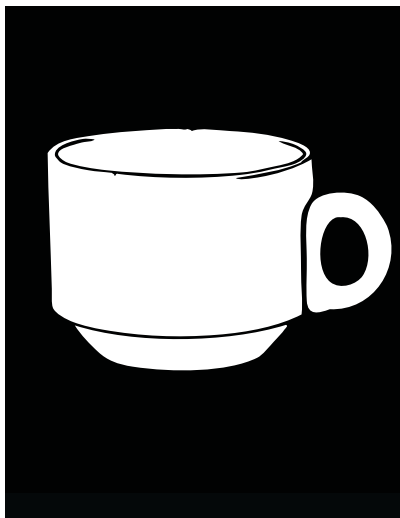
**Question 5: WHAT YEAR ARE WE IN?**

It is considered correct for patients to answer in the short form '08', instead of '2008'. Also, an acceptable alternative prompt (for the rest of the 2000's) is 'The year is 2000 and what?'

#### 2. Picture recognition

Straight after administering the GCS (standardised questions), administer the A-WPTAS by presenting the 3 Westmead PTA cards. Picture Cards the first time - T1 : Show patients the target set of picture cards for about 5 seconds and ensure that they can repeat the names of each card. Tell the patient to remember the pictures for the next testing in about one hour. Picture Cards at each subsequent time T2-T5: Ask patient, "What were the three pictures that I showed you earlier?" Scoring:

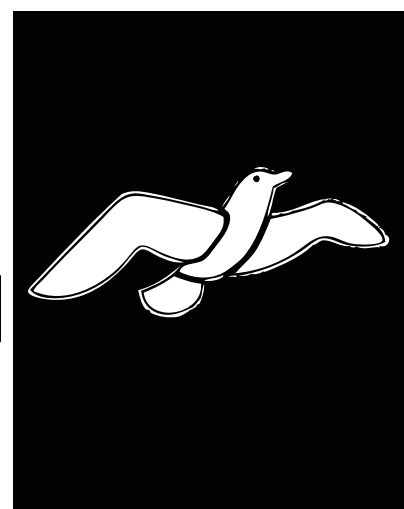
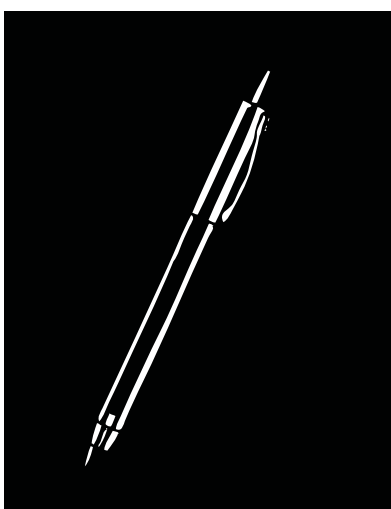
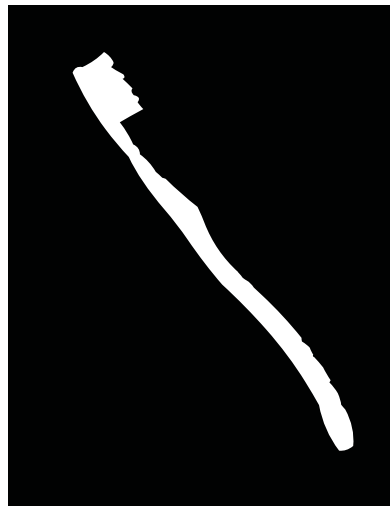
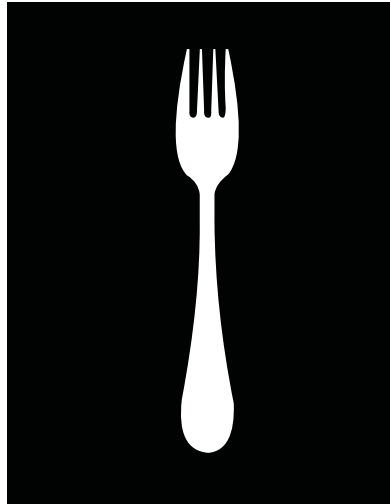
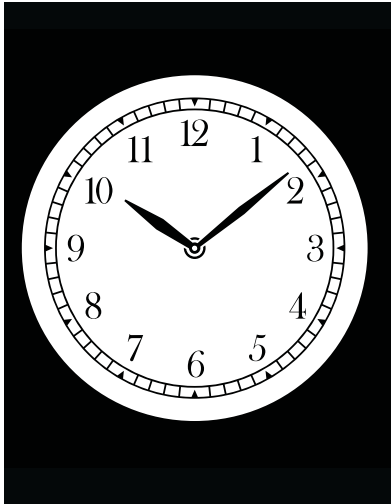
- For patients who free recall all 3 pictures correctly, assign a score of 1 per picture and add up the patient's GCS (out of 15) and A-WPTAS memory component to give the A-WPTAS score (total = 18). Present the 3 target pictures again and re-test in 1 hour.
- For patients who can not free recall, or only partially free recall, the 3 correct pictures, present the 9-object recognition chart. If patient can recognise any correctly, score 1 per correct item and record their GCS and A-WPTAS score (total = 18). Present the target set of pictures again and re-test in 1 hour.
- For patients who neither remember any pictures by free call nor recognition, show the patient the target set of 3 picture cards again for re-test in 1 hour.



Shores & Lammel (2007) - further copies of this score sheet can be downloaded from <http://www.psy.mq.edu.au/GCS>

Research and development of the A-WPTAS supported by the Motor Accidents Authority NSW





Shores & Lammell (2007) - further copies of this score sheet can be downloaded from <http://www.psy.mq.edu.au/GCS>

## Appendix 5: The Glasgow Coma Scale - a practical implementation guide

Associate Professor Paul M Middleton

### Introduction

Impairment of consciousness is one of the most consistent features of head injury, and the Glasgow Coma Scale (GCS) was described by Teasdale and Jennett in 1974<sup>192</sup>, based on a theoretical model of level of consciousness. It was introduced as a simple tool, not to allow absolute distinctions between levels of consciousness, but to be an effective method of accurately describing the various states of impairment within the continuum of consciousness.<sup>192</sup> As the authors stated in the original paper “In the acute stage, changes in conscious level provide the best indication of the development of complications such as intracranial haematoma, whilst the depth of coma and its duration indicate the degree of ultimate recovery which can be expected”. Prior to this most descriptions of altered levels of consciousness revolved around very subjective portrayals such as “comatose”, “drowsy”, “obtunded”, and “stuporose”.<sup>193</sup>

The GCS was originally described by the authors as a repeated bedside assessment of the “...depth and duration of impaired consciousness and coma”<sup>192</sup>, and was used to objectively determine the severity of coma and underlying brain dysfunction at six hours following head trauma. This time frame was chosen to avoid overestimation of brain damage produced by temporary factors such as alcohol, hypoxia or hypotension,<sup>193</sup> and similarly the GCS should be recorded prior to any sedation.

The GCS has also been incorporated as the neurological component of assessment into various aggregate scores such as APACHE and TRISS,<sup>193</sup> and it has been found that taking out this neurological component worsened predictive ability, which led to the presumption that neurological status is the best predictor of overall functional outcome.<sup>194</sup> The GCS makes up 17% of the theoretical maximum Acute Physiology Score (APS) in APACHE II, 19% of the APS in APACHE III<sup>195</sup> and is the basis of the World Federation of neurosurgeons (WFNS) subarachnoid haemorrhage (SAH) grading scale.<sup>196</sup>

The GCS, in actual fact, has evolved through both design and common usage to fulfil multiple functions, which are

summarised in the list below. The GCS can be said to...

- Aid in clinical decision making in interventions such as airway management or intensive care admission
- Describe, quantitate and add structure to the assessment of coma
- Facilitate and standardise communication between clinicians
- Enable monitoring of change in both component and overall scores, i.e. trends in the early stages after injury, allowing rapid detection of complications and discriminating between those at higher or lower risk of complications
- Be an indicator of the severity of illness
- Facilitate comparison between groups of patients
- Allow triage of patients after injury
- Provide a tool for prognostication
- Allow standardisation of patients and patient groups for research

The important primary uses for the GCS can be distilled from this list to...

- Act as an indicator of the level injury and illness, allowing triage and immediate intervention when required, as well as to enable monitoring by the provision of valid measurements and trends of level of consciousness
- Facilitate understanding, clear description and communication between clinicians. This should enable one clinician to describe the level of consciousness to another, whether face-to-face or remotely, in the sure knowledge that this description precisely represents the injury, physiological and functional state of the patient and that the receiving clinician may accurately comprehend this from the description.

The original authors believed that measurement of consciousness should not depend on a single measure, so the GCS was designed to utilise the three domains of eye opening, verbal response and motor response.<sup>193</sup> These domains were chosen as they represent differing aspects of central nervous system function, measured independently of each other, with scores in rank order that indicate the degree of dysfunction. The domains are represented by three different behavioural responses, each assessable in the absence of the others, and the GCS was therefore considered to be more appropriate and effective than the imposition of subjective “levels” of function. The total GCS is time-efficient, and considered to be easy to sum.<sup>197</sup>

The eye opening component refers to the processing of information by the cerebral cortex and the level of arousal or wakefulness.<sup>193</sup> The verbal response domain measures

integration within the nervous system and the presence of speech represents a high degree of this integration.<sup>193</sup> The motor response is considered a good indicator of the ability of the nervous system to function properly due to the variety of possible motion patterns<sup>192, 193</sup> and is also considered to represent that part of the central nervous system least affected by trauma.<sup>193</sup> Total GCS up to 8 largely reflects changes in motor response, referring to patients with no eye opening or verbal response; response scores from 9-15 depend more on eye opening and verbal response.<sup>198</sup> Changes in the eye and verbal responses, and thus higher overall scores, are useful in discriminating between patients with less severe impairment of consciousness. One research group found that increasing scores in the 9-15 range (reflecting improving eye and verbal performances) are associated with a doubling of the rate of good recovery in survivors of head injury.<sup>199</sup>

#### The Glasgow Coma Scale

| Domain               | Level of response                   | Score |
|----------------------|-------------------------------------|-------|
| Eye opening          | Spontaneous                         | 4     |
|                      | To speech                           | 3     |
|                      | To pain                             | 2     |
|                      | None                                | 1     |
| Best Verbal Response | Oriented                            | 5     |
|                      | Confused                            | 4     |
|                      | Inappropriate words                 | 3     |
|                      | Incomprehensible sounds             | 2     |
|                      | None                                | 1     |
| Best Motor Response  | Obeying commands                    | 6     |
|                      | Localising                          | 5     |
|                      | Normal flexor response / withdrawal | 4     |
|                      | Abnormal flexor response            | 3     |
|                      | Extensor posturing                  | 2     |
|                      | None                                | 1     |

Importantly, to facilitate these uses, an exact understanding of the terminology encompassed in the GCS, of the pathophysiological reactions underpinning response, and of the methodology by which various clinicians examine and describe the level of consciousness using the GCS, are essential. Unfortunately, not only are many clinicians unaware what the descriptions of patient reaction to a stimulus mean, but there also appears to be very variable teaching and practice in the detail of how to perform the examination. Studies have shown varying degrees of agreement between groups of clinicians performing the examination and assessing the level of consciousness with the GCS. Despite the high degree of consistency reported by the authors of the GCS,<sup>200</sup> one 2004 study<sup>201</sup> showed only moderate agreement between two emergency physicians who assessed the GCS of a broad range of patients with differing pathologies, and a further

emergency physician-based study by the same authors comparing different types of score found similarly low values.<sup>201</sup> An Australian emergency department study comparing an emergency physician with a registered nurse found excellent agreement in the verbal and total GCS scores, but only intermediate agreement in the motor and eye scores.<sup>202</sup> Given other work which suggests that the motor score is the most discriminating part of the GCS,<sup>203</sup> this is a cause for some concern.

Given that there is little formal training in the application of the GCS and that definitions of the appropriate stimuli to apply and the details of the responses to observe, are similarly scanty, it was considered useful to review the literature and produce a didactic guide which can be used by all practitioners, and attempt to increase the agreement in the clinical setting.

## Detailed breakdown of GCS components

### Eye opening component

| Level of response | Score | Details of response   |
|-------------------|-------|---|
| Spontaneous       | 4     | Indicative of activity of brainstem arousal mechanisms, but not necessarily of attentiveness (primitive ocular-following reflexes at subcortical level) |
| To speech         | 3     | Tested by any verbal approach (spoken or shouted); not necessarily the command to open the eyes   |
| To pain           | 2     | Tested by a stimulus in the limbs (supraorbital pressure may cause grimacing and eye closure)   |
| None              | 1     | No response to speech or pain   |

### Verbal component

| Level of response       | Score | Details of response   |
|-------------------------|-------|---|
| Oriented                | 5     | Awareness of the self and the environment (who / where / when / why)  |
| Confused                | 4     | Responses to questions with presence of disorientation and confusion. |
| Inappropriate words     | 3     | Speech in a random way, no conversational exchange                    |
| Incomprehensible sounds | 2     | Moaning, groaning   |
| None                    | 1     | No response   |

### Motor component

| Level of response                   | Score | Details of response   |
|-------------------------------------|-------|---|
| Obedying commands                   | 6     | The rater must rule out grasp reflex or postural adjustment   |
| Localising                          | 5     | Movement of limb as to attempt to remove the stimulus, the arm crosses midline, and moves to more than one site of noxious stimulus   |
| Normal flexor response / withdrawal | 4     | Rapid withdrawal and abduction of shoulder  |
| Abnormal flexor response            | 3     | Adduction of upper extremities, flexion of arms, wrists and fingers, extension and internal rotation of lower extremities, plantar flexion of feet, and assumption of a hemiplegic or decorticate posture |
| Extensor posturing                  | 2     | Adduction and hyperpronation of upper extremities, extension of legs, plantar flexion of feet, progress to opisthotonus (decerebration)   |
| None                                | 1     | The observer must rule out an inadequate stimulus or spinal transection   |

## Assessment

### Eyes

An eye component score of 3 or 4 implies that information processing is occurring and that the related arousal mechanisms at the brain stem are functioning,<sup>193</sup> whereas a eye component score of 2 indicates that lower levels of the brain are functioning.<sup>193</sup> It is not true however that eye opening indicates awareness; for instance patients in a persistent vegetative state may have spontaneous eye opening, and in this instance this is a reflexive action and does not indicate awareness of self or surroundings.<sup>197</sup>

### Verbal

As stated above, presence of speech implies a high level of integration in the nervous system, although it is important to remember that a lack of speech may be attributed to other factors such as tracheostomy or dysphasia. A lack of speech due to local factors such as this need to be carefully considered, as including a low score cause by local factors into a GCS, especially when only the sum is being used, falsely decreases the score. It is important to remember that the GCS is a measure of level of consciousness, and use a tracheostomy in an otherwise fully conscious patient to give a decreased GCS is clearly counter-intuitive and incorrect.

In terms of the gradations of verbal component score, oriented indicates that the patient is aware of his / her self and the surrounding environment,<sup>197</sup> and is usually described in terms of questions about patient's name, the role of the person asking the questions, the month and year, and the name of the hospital or health care facility. Confused patients can carry on a conversation but the content betrays disorientation and misunderstanding of the components described above. Inappropriate words describes clear and comprehensible speech,<sup>197</sup> but using random words or swearing and cursing. Repeating words or perseveration also falls into this category. Incomprehensible sounds refers to moaning and groaning without recognisable words,<sup>197</sup> even when an attempt to articulate words is being made. It is important to differentiate between a patient with a decreased level of consciousness and reduced cognition, who is unable to form words in response to stimulus, and an awake stroke patient for instance; whose dysphasia may make the task impossible. Clearly, the second case does not represent the situation which the GCS is designed to measure. None means that the patient is unable to verbalise at all,<sup>197</sup> and is subject to the factors described above.

## Motor

Motor component scores of 6, 5 and 4 imply the presence of a degree of cerebral function and thus the ability to react appropriately to a noxious stimulus. Obeys commands indicates an ability to process and obey verbal commands;<sup>197</sup> localisation means that the patient is able to identify the location of a painful stimulus and attempt to remove it, an action often accompanied by the upper extremity of a patient purposefully crossing the midline to remove the stimulus<sup>193, 197</sup>. Withdrawal means that the patient is attempting to move away from the noxious stimulus, sometimes by adopting a fetal position.<sup>197</sup> This last position is particularly important when there is an inexperienced observer, as differentiating a localising response from an abnormal flexion response may prove difficult.<sup>195</sup>

A motor component score of 3, or an abnormal flexor response, implies that the lesion is located in the cerebral hemispheres or internal capsule,<sup>193</sup> whereas a score of 2 describes a midbrain to upper pontine damage.<sup>193</sup> Abnormal flexor response is complex, but involves adduction of upper limbs, with flexion of arms, wrists and fingers. Accompanying this are extension and internal rotation of lower limbs, and plantar flexion of feet. This must be differentiated from the normal flexor response or withdrawal, but also from extensor posturing which indicates a lesion lower in the central nervous system, and therefore reflecting CNS function at a lower level.<sup>193</sup> Extensor posturing includes the same lower limb appearances as in abnormal flexion, but with the upper limbs adopting a different position; this is described as extension of the upper limbs along the sides of the body, accompanied by pronation of the forearms. Abnormal flexion and extensor posturing are often known by the terms decerebrate and decorticate response, implying the level of loss of CNS function, and studies have shown that patients showing extensor posturing are more likely to have a poor outcome than those with abnormal flexion.<sup>197</sup> If a patient demonstrates flexion on one side of the body, and extension on the other, the best of the two responses needs to be recorded.<sup>193</sup>

None means that the patient is flaccid, and does not make any movement in response to a painful stimulus. In these circumstances, it is essential to check that the patient is not pharmacologically or pathologically paralysed. Bear in mind again, that as the GCS is endeavouring to measure cognition and that abnormal motor responses, due to the presence of anaesthetic paralysis or spinal cord injury

invalidates the motor score at this time, in this patient, as a means to measure consciousness. Another important caveat to the measurement of the motor score component of the GCS is that the simple “squeeze my fingers” is NOT sufficient or appropriate to demonstrate this function.<sup>197</sup> A grasp reflex can be elicited in many patients with decreased cognition, similar to that found in babies, and attempts at least have to include the command to release the fingers after squeezing them, and this must be seen to be obeyed. More specific commands such as “show me two fingers” are more appropriate.

In the sections above, there are various conditions discussed which invalidate the measurement of specific domains of the GCS, however it must always be borne in mind that this tool is designed to assess consciousness and cognition, and that local lesions of many descriptions invalidate this measurement, and should therefore not be counted into an overall score at all, and should not be counted into a domain score without documented explanation. If a domain of the GCS, such as eye opening or verbal response is confounded by local lesions, then both snapshots and trends should be limited to the use of the other domains.<sup>192, 193</sup> Since the motor score has been shown to contain most of the predictive power of the GCS, especially in the more severely head injured patients, it would be reasonable to use this alone in these circumstances. When this occurs, it has been recommended that a 1 is scored,<sup>197</sup> however if this is done it has to be accompanied by a written explanation and the caveat that this cannot be used in an overall score.

Conditions such as alcohol, drugs, inability to understand commands due to language barriers, and hearing impairment are all conditions that may confound the performance of a GCS, and once again consideration needs to be given to the reason for measuring cognition. For example, if the reason the GCS is being measured is to assess the level of consciousness associated with a head injury or pathological cause of decreased conscious level, conditions such as alcohol or sedative drugs are a confounder which invalidate the GCS; however, if the measurement is being used to assess the effect of drugs on the level of consciousness, this is then the relevant effect being measured. In these circumstances, however, many of the correlates of a decreased level of consciousness in head injury measured by GCS may not be accurate; such as an inability to protect the airway associated with a GCS  $\leq 8$ , which is often not true in patients obtunded with certain drugs of abuse. An extended list of potential confounders is shown below.

The GCS is NOT a scale to measure an altered sensorium, so cannot be used to test sensation. It is also not substitute for either a full neurological examination or an assessment of orientation.<sup>193, 197</sup> It also does not account for true lateralisation as it measures the best response rather than the worst.<sup>193</sup>

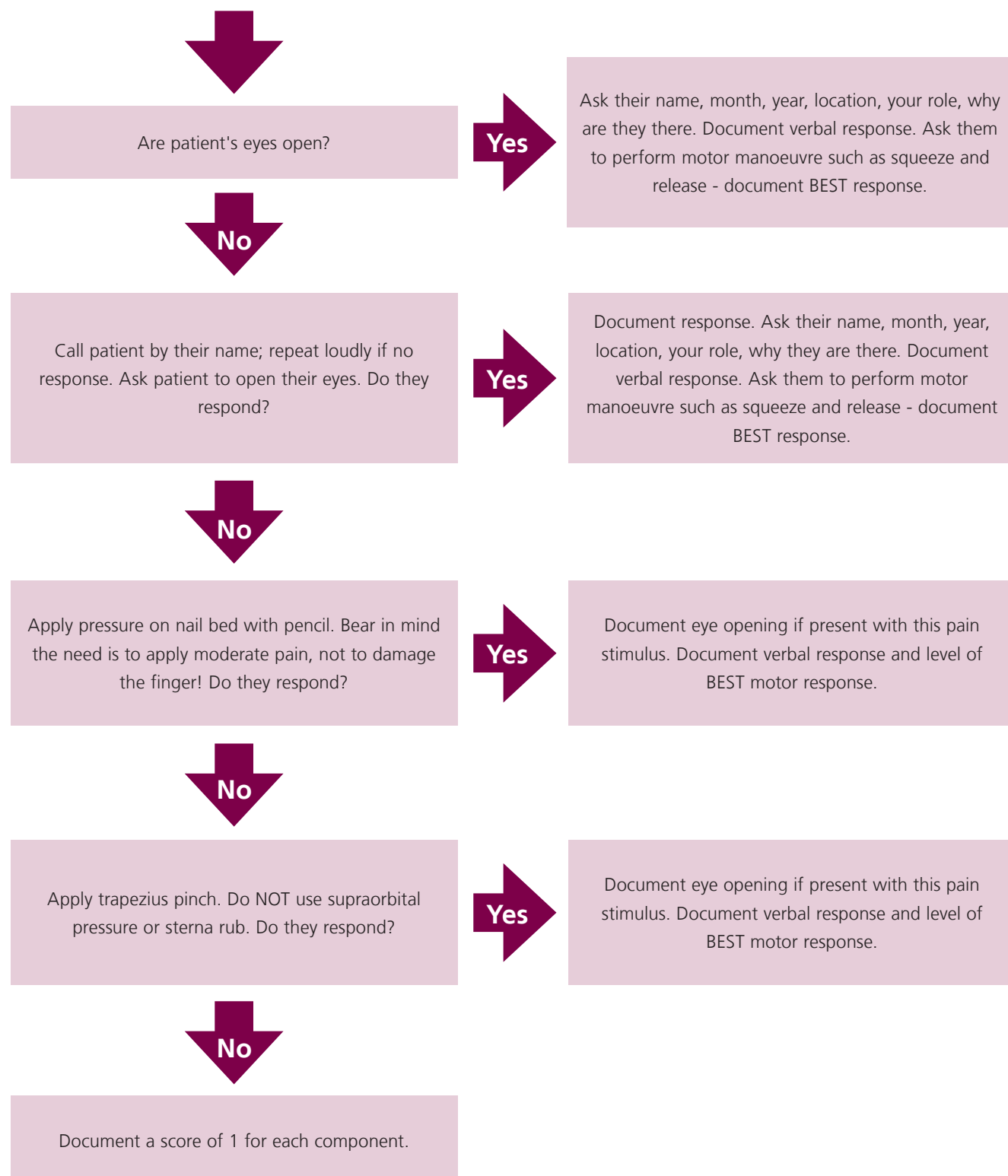
The sternal rub has been documented to cause injury, particularly pressure area damage<sup>204</sup> and cannot be recommended. Supraorbital nerve pressure has caused damage and is less reliable and consistent than other methods or applying a central noxious stimulus.<sup>205</sup> The trapezius pinch causes no damage as it simply comprises pressure on a large muscular area, but does provide a suitable painful stimulus.<sup>204</sup> Ensure that you note whether the upper limb localised to the painful stimulus by crossing the midline or not; if it does not, carefully assess to discriminate between this and abnormal flexion.

Conditions that affect the calculation of the three components of the GCS

| Conditions   | Eyes | Verbal | Motor |
|--|------|--------|-------|
| Ocular trauma  | +    |        |       |
| Cranial nerve injuries                                 | +    |        |       |
| Pain   | +    |        | +     |
| Intoxication (alcohol, drugs)                          |      | +      | +     |
| Medications (anaesthetics, sedatives)                  |      | +      | +     |
| Dementia   |      | +      | +     |
| Psychiatric diseases                                   |      | +      | +     |
| Developmental impairments                              |      | +      | +     |
| No comprehension of spoken language                    |      | +      | +     |
| Intubation, tracheostomy, laryngectomy                 |      | +      |       |
| Oedema of tongue                                       |      | +      |       |
| Facial trauma  |      | +      |       |
| Mutism   |      | +      |       |
| Hearing impairments                                    |      | +      |       |
| Injuries (spinal cord, peripheral nerves, extremities) |      |        | +     |

## Practical measurement of the Glasgow Coma Scale

Assess global condition of patient, particularly noting any evidence of local injuries that might affect the measurement of the Glasgow Coma Scale. These include presence of endotracheal tube, tracheostomy or other airway adjunct, traumatic injury to eyes, mouth or limbs. The medication chart should be checked to determine if there have been any sedating or paralysing drugs administered, and patient notes should be checked for a history of recent alcohol or substance use. Document the presence of any of the above on the observations chart, or ensure that they have already been noted. Check the patient's correct name, and that they speak English.





## Appendix 6: Mild Head Injury Discharge Advice

### Mild Head Injury Advice

2008

#### Important points about Mild Head Injury

You had a mild head injury. Most people recover rapidly following a mild head injury. A few people may suffer from symptoms over a longer period.

There is a small risk of you developing serious complications so **you should be watched closely by another adult for 24 hours after the accident. Please read the following. It outlines what signs to look out for after a head injury and what you need to do if you have problems.**



#### Warning Signs

If you show any of these symptoms or signs after your head injury, or you get worse, go to the nearest hospital, doctor or telephone an ambulance immediately.

- ❖ Fainting or drowsiness - or you can't wake up
- ❖ Acting strange, saying things that do not make sense (change in behaviour)
- ❖ A constant severe headache or a headache that gets worse
- ❖ Vomiting or throwing up more than twice
- ❖ Cannot remember new events, recognise people or places (increased confusion)
- ❖ Pass out or have a blackout or a seizure (any jerking of the body or limbs)
- ❖ Cannot move parts of your body or clumsiness
- ❖ Blurred vision or slurred speech
- ❖ Continual fluid or bleeding from the ear or nose

#### The first 24-48 hours after injury



##### Warning Signs

**You should be observed and return to hospital if you develop any of the above warning signs.**



##### Rest / Sleeping

Rest and avoid strenuous activity for at least 24 hours. It is alright for you to sleep tonight but you should be checked every four hours by someone to make sure you are alright.



##### Driving

Do not drive for at least 24 hours. You should not drive until you feel much better and can concentrate properly. Talk to your doctor.



##### Drinking / Drugs

Do not drink alcohol or take sleeping pills or recreational drugs in the next 48 hours. All of these can make you feel worse. They also make it hard for other people to tell whether the injury is affecting you or not.



##### Pain Relief

Use **paracetamol** or **paracetamol/codeine** for headaches. **Do not use aspirin or anti inflammatory pain reliever** such as ibuprofen or naproxen (NSAIDs), which may increase the risk of complications.



##### Sports

Do not play sports for at least 24 hours.



MOTOR ACCIDENTS  
AUTHORITY

**See your local doctor if you are not starting to feel better within a few days of your injury.**

Adapted from "Mild Head Injury Discharge Advice" author Dr Duncan Reed (2007) Director of Trauma Gosford Hospital. NSW Institute of Trauma and Injury Management

This discharge advice information is taken from the Motor Accidents Authority of NSW, Guideline for mild traumatic brain injury following closed head injury – first edition 2008, ISBN 978-1-921422-08-9

## The first 4 weeks after injury

You may have some common effects from the head injury which usually resolve in several weeks to three months. These are called **post concussive symptoms** (see below). Tiredness can exaggerate the symptoms. Return to your normal activities gradually (not all at once) during the first weeks or months. **You can help yourself get better by:**



### Rest / Sleeping

Your brain needs time to recover. It is important to get adequate amounts of sleep as you may feel more tired than normal.



### Driving

Do not drive or operate machinery until you feel much better and can concentrate properly. Talk to your doctor.



### Drinking / Drugs

Do not drink alcohol or use recreational drugs until you are fully recovered. They will make you feel much worse. Do not take medication unless advised by your doctor.



### Work / Study

You may need to take time off work or study until you can concentrate better. Most people need a day or two off work but are back full time in less than 2 weeks. How much time you need off work or study will depend on the type of job you do. See your doctor and let your employer or teachers know if you are having problems at work or with study. You may need to return to study or work gradually.



### Sport / Lifestyle

It is dangerous for the brain to be injured again if it has not recovered from the first injury. Talk to your doctor about the steps you need to take to gradually increase sports activity and return to play. **If in doubt "sit it out"**.



### Relationships

Sometimes your symptoms will affect your relationship with family and friends. You may suffer irritability and mood swings. See your doctor if you or your family are worried.

## Recovery

You should start to feel better within a few days and be 'back to normal' within about 4 weeks. See your local doctor if you are not starting to feel better.

Your doctor will monitor these symptoms and may refer you to a specialist if you do not improve over 4 weeks up to 3 months.

## Post Concussion Symptoms

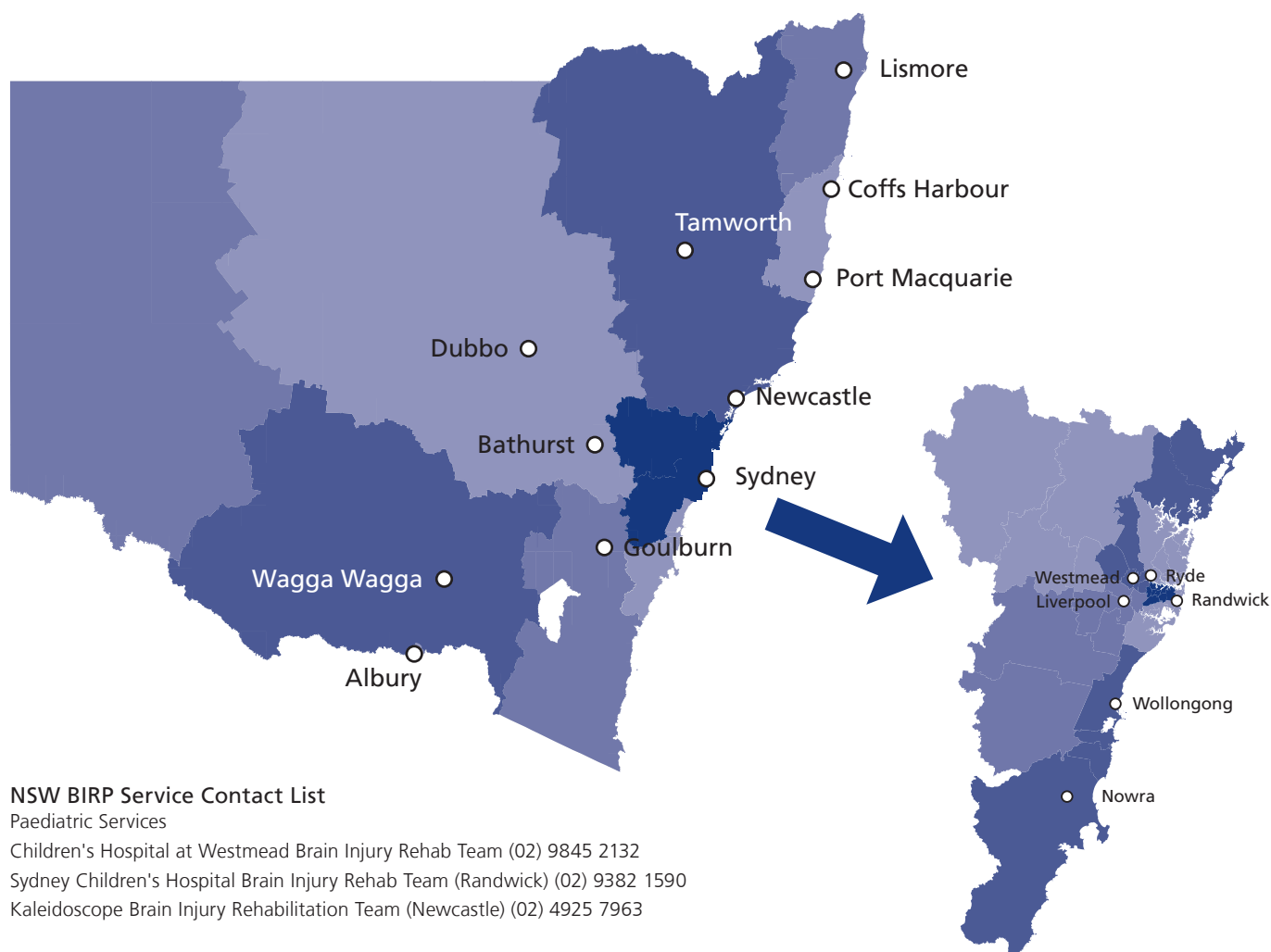
There are common symptoms after a mild head injury. **They usually go away within a few days or weeks.**

Sometimes you may not be aware of them until sometime after your injury like when you return to work.

- ❖ Mild headaches (that won't go away)
- ❖ Having more trouble than usual with attention & concentration
- ❖ Having more trouble than usual with remembering things (memory difficulties/forgetfulness)
- ❖ Feeling dizzy or sick without vomiting (nausea)
- ❖ Balance problems
- ❖ More difficulty than usual with making decisions and solving problems, getting things done or being organised
- ❖ Feeling vague, slowed or "foggy" thinking
- ❖ Feeling more tired than usual and lacking energy (fatigue)
- ❖ Irritability. Losing your temper and getting annoyed easily
- ❖ Mood swings
- ❖ Anxiety or depression
- ❖ Mild behavioural change
- ❖ More sensitive to sounds or lights
- ❖ Change in sleep patterns. Trouble sleeping or sleeping too much
- ❖ Reduced tolerance to alcohol

### Local service information

## Appendix 7: NSW Brain Injury Rehabilitation Program



### Greater Metropolitan Sydney Services

Liverpool Hospital Brain Injury Rehabilitation Unit (02) 9828 5495  
Royal Rehabilitation Centre Sydney Brain Injury Rehab Team (02) 9807 1144  
Westmead Brain Injury Rehabilitation Service (02) 9845 7941  
Hunter Brain Injury Service (Newcastle) (02) 4929 3100  
Illawarra Brain Injury Service (02) 4223 8470

### Rural Services

Dubbo Brain Injury Rehabilitation Program (02) 6841 8505  
Mid Western Brain Injury Rehabilitation Program (Bathurst) (02) 6330 5114  
New England Brain Injury Rehabilitation Service (Tamworth) (02) 6767 8350  
North Coast Brain Injury Rehabilitation Service:  
■ Lismore (02) 6620 2111  
■ Port Macquarie (02) 6584 3300  
■ Coffs Harbour (02) 6652 2856  
Southern Area Brain Injury Service (Goulburn) (02) 4823 7911  
South West Brain Injury Rehabilitation Service:  
■ Albury (02) 6041 9902  
■ Wagga Wagga (02) 69710151

For more information please contact the **NSW Brain Injury Rehabilitation Directorate** on (02) 9828 6133.

## Appendix 8: Methodology

### A8.1 General search strategy

Comprehensive search strategies for both Medline and Embase were guided by each of the clinical questions. A general text-word based strategy was used to search the Cochrane Database of Systematic Reviews (CDSR), the Cochrane Central Register of Controlled Trials (CENTRAL) and SCOPUS. The searches were executed for the period 1980 to 30th October 2004 (1st Ed.) and updated for the 2nd Ed. (2004-2010). The results from each of these searches were filtered in accordance with the exclusion and inclusion criteria (see below) and then assessed for relevance to the clinical questions. The search strategies are listed in Appendix 9.

In addition, reference lists of previous guidelines and key papers were used to identify other key references, including pre-2004 literature. SCOPUS and Google Scholar were used to execute author-based searches, citation mapping and grey literature searching. The following websites were also searched (using relevant free text terms):

Scottish Intercollegiate Guidelines Network  
[www.sign.ac.uk](http://www.sign.ac.uk)

Bandolier  
[www.medicine.ox.ac.uk/bandolier](http://www.medicine.ox.ac.uk/bandolier)

TRIP database  
[www.tripdatabase.com](http://www.tripdatabase.com)

ClinicalTrials.gov  
[www.ClinicalTrials.gov](http://www.ClinicalTrials.gov)

National Guideline Clearing House  
[www.guideline.gov](http://www.guideline.gov)

Brain Trauma Foundation  
[www.braintrauma.org](http://www.braintrauma.org)

National Institute for Health & Clinical Evidence  
[www.nice.org.uk](http://www.nice.org.uk)

Agency for Healthcare Research & Quality  
[www.ahrq.gov](http://www.ahrq.gov)

### A8.2 Inclusion and exclusion criteria

| Conditions   | Exclusion Criteria  |
|--|---|
| <ul style="list-style-type: none"><li>■ Closed head injury studies</li><li>■ Aged &gt; 16 years</li><li>■ Meta-analyses, systematic reviews, clinical guidelines incorporating systematic reviews, controlled trials, comparative studies.</li></ul> | <ul style="list-style-type: none"><li>■ Penetrating head injury or brain damage from stroke/ cerebrovascular incidents</li><li>■ Aged &lt; 16 years</li><li>■ Narrative reviews, letters, editorials, case studies/ series</li><li>■ Studies using non-human subjects</li></ul> |

### A8.3 Strength of recommendations

This guideline uses the National Health and Medical Research Council (NHMRC) overall grades of recommendation to indicate the strength of the body of evidence underpinning each recommendation. The body of evidence reflects the evidence components of all the studies relevant to each recommendation. The evidence components are assessed according to the NHMRC body of evidence matrix (see table below). The overall grade of the recommendation is determined based on a summation of the rating for each individual component of the body of evidence. Please note that a recommendation cannot be graded A or B unless the evidence base and consistency of the evidence are both rated A or B.<sup>44</sup>

Overall grade A or B recommendations are generally based on a body of evidence that can be trusted to guide clinical practice, whereas Grades C or D recommendations must be applied carefully to individual clinical and organisational circumstances and should be interpreted with care<sup>44</sup> This guideline also utilises an additional grade of "Consensus" where appropriate.

The recommendation boxes of each clinical question addressed in this guideline contain clear recommendations with an associated strength of recommendation grade as per above. Where appropriate, the author has also added relevant clinical points to the boxes which support the given recommendation.

Body of evidence matrix<sup>44</sup>

| Components       | A   | B   | C  | D  |
|------------------|---|---|--|--|
|                  | Excellent   | Good  | Satisfactory   | Poor   |
| Evidence base    | one or more level I studies with a low risk of bias or several level II studies with a low risk of bias | one or two level II studies with a low risk of bias or a SR/several level III studies with a low risk of bias | one or two level III studies with a low risk of bias, or level I or II studies with a moderate risk of bias  | level IV studies, or level I to III studies/ SRs with a high risk of bias  |
| Consistency      | all studies consistent  | most studies consistent and inconsistency may be explained  | some inconsistency reflecting genuine uncertainty around clinical question   | evidence is inconsistent   |
| Clinical impact  | very large  | substantial   | moderate   | slight or restricted   |
| Generalisability | population/s studied in body of evidence are the same as the target population for the guideline        | population/s studied in the body of evidence are similar to the target population for the guideline           | population/s studied in body of evidence differ to target population for guideline but it is clinically sensible to apply this evidence to target population | population/s studied in body of evidence differ to target population and hard to judge whether it is sensible to generalise to target population |
| Applicability    | directly applicable to Australian healthcare context  | applicable to Australian healthcare context with few caveats  | probably applicable to Australian healthcare context with some caveats   | not applicable to Australian healthcare context  |

| Grade of recommendation | Description   |
|-------------------------|---|
| A                       | Body of evidence can be trusted to guide practice   |
| B                       | Body of evidence can be trusted to guide practice in most situations  |
| C                       | Body of evidence provides some support for recommendation(s) but care should be taken in its application  |
| D                       | Body of evidence is weak and recommendation must be applied with caution  |
| Consensus               | When limited literature was available, the author and editorial group utilised the best available clinical expertise, practices and accepted teachings to reach a consensus on the recommendation |

#### A8.4 Level of evidence

'Level of Evidence' refers to the study design used to minimise bias. The articles were classified according to their general purpose and study type in accordance with

the NHMRC publication: A guide to the development, evaluation and implementation of clinical practice guidelines.<sup>45</sup> From this, each article was allocated a level of evidence as follows:

|             |   |
|-------------|---|
| Level I     | Evidence obtained from a systematic review of all relevant randomised control trials  |
| Level II    | Evidence obtained from at least one properly-designed randomised control trial  |
| Level III-1 | Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method)  |
| Level III-2 | Evidence obtained from comparative studies (including systematic reviews of such studies) with concurrent controls and allocation not randomised, cohort studies, case-control studies, or interrupted time series with a control group |
| Level III-3 | Evidence obtained from comparative studies with historical control, two or more single arm studies or interrupted time series without a parallel control group  |
| Level IV    | Evidence obtained from a case-series, either post-test or pre-test/post-test  |

#### A8.5 Quality assessment for individual studies used in guidelines

##### A8.5.1 Introduction

The quality of each study is an assessment of the methodological quality (or internal validity), and is the extent to which the study's design, conduct and analysis has minimised selection, measurement and confounding biases. The process used to assess the studies included in

this guideline was adapted from the NHMRC publication: *How to review the evidence: systematic identification and review of the scientific literature*<sup>206</sup> and the MERGE assessment tool.<sup>207</sup>

Studies are allocated the following ratings based on the extent to which they address the quality items in each study type specific checklist:

|              |  |   |
|--------------|--|---|
| Good studies | Low risk of bias   | Have <i>most or all</i> of the relevant quality items   |
| Fair studies | Susceptible to some bias, but not sufficient to invalidate results | Have <i>some</i> of the relevant items  |
| Poor studies | High risk of bias arising from significant methodological flaws    | Have <i>few or none</i> of the relevant quality items (these studies are generally not included in the evidence tables) |

##### A8.5.2 Checklists of study-specific quality items:

###### 1. Checklist for the quality assessment of systematic reviews:

- Was a comprehensive and explicit search strategy used?
- Were the included studies assessed for quality?

c. Were the characteristics and results of the studies summarised appropriately?

d. Were sources of heterogeneity explained?

###### 2. Checklist for the quality assessment of evidence-based guidelines:

- Was a comprehensive and explicit search strategy used?

b. Have all relevant interventions and outcomes been considered, both benefits and harms?

c. Is the level and quality of evidence for each recommendation given?

d. Do the recommendations address benefit versus harm according to the level of risk in different patient sub-groups?

c. Was follow-up for final outcomes adequately long and complete?

d. Has measurement or misclassification bias been minimised? (Were outcomes measured blind?)

### 3. Checklist for the quality assessment of *intervention* studies:

a. Has selection bias (including allocation bias) been minimised?

b. Have adequate adjustments been made for residual confounding?

c. Was the follow-up for final outcomes adequate? (Follow-up rate reported and adequately high?)

d. Has measurement or misclassification bias been minimised? (Blinding of outcome measurements?)

### 4. Checklist for the quality assessment of *diagnostic* studies:

a. Has selection bias been minimised? (Were participants consecutively enrolled?)

b. Have adequate adjustments been made for residual confounding? (Were interventions blind to the test result?)

c. Was follow-up for final outcomes adequate? (Were all enrolled verified by the reference standard?)

d. Has measurement or misclassification bias been minimised? (Was the reference standard validated and measured blindly?)

### 5. Checklist for the quality assessment of *prognostic* studies (cohort studies):

a. Has selection bias been minimised? (A random/ consecutive sample of participants at the same point in their disease?)

b. Were all potentially important prognostic factors assessed?

A8.6

## Quality assessment for guidelines referred to in this document

The Appraisal of Guidelines for Research and Evaluation (AGREE) instrument,<sup>208</sup> a validated tool, was used for the quality appraisal of all guidelines referenced in this document. Each appraised guideline is scored on six criteria resulting in an overall assessment of strongly recommended, recommended, would not recommend or unsure.

For further information please see the AGREE Collaboration website [www.agreecollaboration.org](http://www.agreecollaboration.org).

## Appendix 9: Search Strategies

### 1st Edition:

The following search phrases were used in Medline:

1. exp Head Injuries, Closed/
2. exp \*tomography, x-ray/
3. Patient Discharge/
4. Patient Transfer/
5. intubation/ or exp intubation, intratracheal/
6. \*Intracranial Pressure/
7. Drainage/
8. (7 and (ventricular or intra?ventricular or extra?ventricular).mp.) or ((ventricular or intra?ventricular or extra?ventricular) adj drain\$).mp.
9. (icp monitor\$ or intracranial pressure monitor\$).mp.
10. exp Aggression/
11. exp Mannitol/
12. exp Hyperventilation/
13. Adrenal Cortex Hormones/
14. Craniotomy/
15. Trephining/
16. exp emergency treatment/
17. exp \*head injuries, closed/su, th or (exp head injuries, closed/ and management.mp.)
18. (or/2-6) or (or/8-16)
19. (1 and 18) or 17

The following search phrases were used in Embase:

1. (head injury/ and closed\$.mp.) or (closed head injury or closed head trauma\$).mp.
2. exp computer assisted tomography/
3. patient transport/ or discharg\$.mp.
4. exp RESPIRATORY TRACT INTUBATION/ or INTUBATION/
5. Intracranial Hypertension/
6. cerebrospinal fluid drainage/
7. ((ventricular or intra?ventricular or extra?ventricular) adj drain\$).mp.
8. (icp monitor\$ or intracranial pressure monitor\$).mp.
9. exp aggression/
10. Mannitol/
11. exp Hyperventilation/
12. exp Corticosteroid/
13. craniotomy/
14. (trephin\$ or burr hole\$).mp.
15. exp emergency treatment/
16. (su or th).fs. or management.mp.
17. 1 and (or/2-16)
18. limit 17 to human



## 2nd Edition:

For the 2nd Edition, searches were constructed for each clinical question as per below:

### 1. What is the definition of Mild Head Injury?

#### *MEDLINE*

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab.
3. (defin\$ or classif\$ or (risk adj stratif\$)).ti,ab.
4. (GCS adj4 (admission or arrival or initial or present\$)).ti,ab.
5. (guideline\$ OR (emergency ADJ (management OR treatment)).ti,ab.
6. exp emergency treatment/
7. 1 OR 2
8. OR/3-6
9. 7 AND 8
10. LIMIT 9 to (English language and (comparative study or controlled clinical trial or guideline or meta analysis or randomized controlled trial) and last 5 years)

n = 516 citations retrieved

#### *EMBASE*

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. OR/1-3
5. (minor or minimal or mild).tw
6. ((Glasgow coma scale NEXT/3 (14 or 15)).tw
7. ((Glasgow coma score NEXT/3 (14 or 15)).tw
8. (gcs NEAR/3 (14 or 15)).tw
9. OR/ 5-8
10. 4 and 9
11. (defin\* or classif\* or "risk NEXT stratif\*").tw
12. (GCS NEAR/5 (admission or arrival or initial or present\*)).tw
13. emergency treatment/exp
14. OR/10-13
15. 10 and 14
16. LIMIT 15 to (English language AND (yr=2005-2010))

N=162 citations retrieved

## 2. What are the clinically important complications of Mild Head Injury?

### *MEDLINE*

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab.
3. (mild OR minim\$ OR minor).ti,ab
4. 1 OR 2
5. 3 AND 4
6. (complication\$ or risk\$ or sequelae or morbid\$ or mortalit\$).ti,ab.
7. (post?concuss\$ or concuss\$ ).ti,ab.
8. ((headache\$ or dizziness or fatigue) or (cognitive adj deficit\$) or ((behav\$ or social) adj3 (dysfunction\$ or function\$))).ti,ab
9. ((intracranial or intra?cranial or sub?dural or intra?dural or epidural or sub?arachnoid or structural) adj (haematoma\$ or hematoma\$ or haemorrhage\$ or hemorrhage\$ or contusion\$ or lesion\$)).ti,ab.
10. ((skull or cranial) adj fracture\$).ti,ab.
11. OR /6-10
12. 5 AND 11
13. limit 12 to (english language and humans and yr="2005 -Current" and (clinical trial, all or controlled clinical trial or government publications or guideline or meta analysis or randomized controlled trial))

### *EMBASE*

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. (minor or minimal or mild).tw
5. OR/1-3
6. 4 AND 5
7. (complication\* or risk\* or sequelae or morbid\* or mortalit\*).tw
8. (post?concuss\* or concuss\* ).tw
9. ((headache\* or dizziness or fatigue) or (cognitive NEXT deficit\*)).tw
10. ((behav\* or social) NEXT3 (dysfunction\* or function\*)).tw
11. ((intracranial or intra?cranial or sub?dural or intra?dural or epidural or sub?arachnoid or structural) NEXT (haematoma\* or hematoma\* or haemorrhage\* or hemorrhage\* or contusion\* or lesion\*)).tw
12. ((skull or cranial) NEXT fracture\*).tw
13. OR /7-12
14. 6 AND 13
15. limit 14 to (English language AND (yr=2005-2010))

### 3. How should patients with Mild Head Injury be assessed?

#### **MEDLINE**

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab.
3. (mild OR minim\$ OR minor).ti,ab
4. 1 OR 2
5. 3 AND 4
6. (assessment or observation\$ or classification\$ or stratification or risk or examination or (clinical adj (history OR assessment)) OR (neurologic\$ adj (history OR assessment OR observation\$))).ti,ab.
7. ((GCS or (glasgow adj coma adj (score or scale))) adj3 (admission or arrival or initial or present\$ or deteriorat\$ or serial or abnormal)).ti,ab.
8. ((PTA or (post?traumatic adj amnesia)) adj (testing or scor\$ or persistan\$)).ti,ab.
9. ((clinical adj (decision or diagnostic) adj (rule\$ or tool\$)) or (guideline\$ or protocol\$ or algorithm\$) or management).ti,ab.
10. OR/ 6-9
11. 5 AND 10
12. limit 11 to (english language and humans and (clinical trial, all or controlled clinical trial or government publications or guideline or meta analysis or practice guideline or randomized controlled trial) and last 5 years)

#### **EMBASE**

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. (minor or minimal or mild).tw
5. OR/1-3
6. 4 AND 5
7. (assessment or observation\* or classification\* or stratification or risk or examination or (clinical NEXT (history OR assessment)) OR (neurologic\* NEXT (history OR assessment OR observation\*))).tw
8. ((GCS or (glasgow adj coma NEXT (score or scale))) NEXT3 (admission or arrival or initial or present\* or deteriorat\* or serial or abnormal)).tw
9. ((PTA or (post?traumatic adj amnesia)) NEXT (testing or scor\* or persistan\*)).tw
10. ((clinical NEXT (decision or diagnostic) NEXT (rule\* or tool\*)) or (guideline\* or protocol\* or algorithm\*) or management).
11. OR/7-10
12. 6 AND 11
13. limit 12 to (English language AND (yr=2005-2010))

#### 4. Which patients with Mild Head Injury require a CT scan?

The literature searches for question 4 and 5 were combined, as most evidence addressing indications for CT scanning also address alternate management strategies.

#### 5. What is the optimal management strategy for high-risk Mild Head Injury patients when CT scan is unavailable?

##### *MEDLINE*

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab
3. (mild OR minim\$ OR minor).ti,ab
4. 1 OR 2
5. 3 AND 4
6. exp skull fractures/
7. ((skull or cranial) ADJ fracture\$).ti,ab
8. ((intracranial or intra?cranial or sub?dural or intra?dural or epidural or sub?arachnoid or structural) adj (haematoma\$ or hematoma\$ or haemorrhage\$ or hemorrhage\$ or contusion\$ or lesion\$)).ti,ab.
9. ((cerebral or brain) ADJ (?edema OR lesion\$)).ti,ab
10. exp anticoagulants/
11. OR/ 6-10
12. exp Tomography, X-ray computed/
13. (CT OR computed tomograph\$).ti,ab
14. ((Canadian adj3 ct) or nexus?II or NICE or (SIGN) or (Scottish adj intercollegiate adj guidelines adj network) or (brain adj trauma adj foundation)).ti,ab
15. ((clinical adj (decision or diagnostic) adj (rule\$ or tool\$)) or (guideline\$ or protocol\$ or algorithm\$) or management).ti,ab.
16. exp X-Rays/
17. exp Referral and Consultation/
18. or/12-17
19. 11 AND 18
20. 5 AND 18
21. 19 OR 20
22. limit 21 to (english language and humans and (clinical trial, all or controlled clinical trial or government publications or guideline or meta analysis or practice guideline or randomized controlled trial) and last 5 years)

##### *EMBASE*

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. (minor or minimal or mild).tw
5. OR/1-3
6. 4 AND 5
7. skull fracture/exp
8. ((skull or cranial) NEXT fracture\*).tw
9. ((intracranial or intra?cranial or sub?dural or intra?dural or epidural or sub?arachnoid or structural) NEXT (haematoma\* or hematoma\* or haemorrhage\* or hemorrhage\* or contusion\* or lesion\*)).tw
10. ((cerebral or brain) NEXT (?edema OR lesion\*)).tw
11. OR/ 7-10
12. computer assisted tomography/exp
13. (ct OR computed tomograph\*).tw
14. ((Canadian NEXT ct) or nexus?II or NICE or (national institute for health and clinical excellence) or (SIGN) or (Scottish intercollegiate guidelines network) or (brain trauma foundation)).tw
15. or/12-14
16. 11 AND 15
17. 6 AND 14
18. 16 or 17
19. limit 18 to (English language AND (yr=2005-2010))

N = 1305 citations retrieved

## 6. When can patients with mild head injury be safely discharged?

### MEDLINE

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab
3. (mild OR minim\$ OR minor).ti,ab
4. 1 OR 2
5. 3 AND 4
6. exp Patient Discharge
7. exp risk assessment
8. discharge\$.ti,ab
9. OR/6-8
10. 5 AND 9

N = 519 citations retrieved

### EMBASE

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. (minor or minimal or mild).tw
5. OR/1-3
6. 4 AND 5
7. hospital discharge/exp
8. 6 AND 8

N = 51 citations retrieved

## 7. What are the proven treatments for patients with 'moderate' to 'severe' head injury?

### MEDLINE

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab
3. 1 OR 2
4. exp emergency treatment
5. exp emergency service, hospital
6. 4 or 5
7. hypnotics / tu
8. exp intracranial pressure
9. subarachnoid hemorrhage / dt
10. exp drainage
11. exp hypothermia, induced
12. exp hyperventilation
13. exp neuroprotective agents
14. anti-inflammatory agents / tu
15. seizures / pc
16. saline solution, hypertonic / tu
17. OR/7-16
18. 3 AND 16 AND 17

N = 505 citations retrieved

### EMBASE

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. OR/1-3
5. emergency treatment/exp
6. intensive care/exp
7. OR/5-6
8. therapeutic hyperventilation/exp
9. cerebrospinal fluid drainage/exp
10. induced hypothermia/exp
11. sodium chloride/exp
12. sedative agent/exp
13. hypnotic sedative agent/exp
14. anticonvulsive agent/exp
15. decompressive craniectomy/exp
16. analgesic agent/exp
17. neurosurgery/exp
18. OR/8-16
19. 4 AND 7 AND 18
20. limit 19 to (English language AND (yr=2005-2010))

N = 1288 citations retrieved

**8. When should patients with closed head injury be transferred to hospitals with neurosurgical facilities?**

**MEDLINE**

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab
3. 1 OR 2
4. exp patient transfer
5. 3 AND 4
6. limit 5 to (english language and humans and yr="2004-Current")

N = 59 citations retrieved

**EMBASE**

1. head injury/ exp
2. (craniocerebral trauma or cranio-cerebral trauma or cerebral trauma).tw
3. (head injur\* or brain injur\* or brain trauma or head trauma).tw
4. OR/1-3
5. interhospital NEAR/3 transfer
6. patient transport/exp
7. 5 OR 6
8. 4 AND 7
9. limit 8 to (English language AND (yr=2005-2010))

N = 169 citations retrieved

**9. Which patients with closed head injury should receive anticonvulsants?**

**MEDLINE**

1. exp craniocerebral trauma/
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) ADJ (injur\$ OR traum\$).ti,ab
3. Or / 1-2
4. exp phenytoin/
5. exp pentobarbital/
6. exp carbamazepine/
7. exp valproic acid/
8. (pentobarbit\* or carbamazepine\* or valpro\* or fosphenytoin\* or phenytoin\*).ti,ab.
9. (anticonvul\* or anti-convul\* or antiseiz\* or anti-seiz\* or antiepilep\*).ti,ab.
10. Or/4-9
11. exp epilepsy, post-traumatic/
12. 3 or 11
13. 10 AND 12

**EMBASE**

1. head injury/exp
2. (head OR brain OR cerebral OR cranial OR cranio?cerebral) NEXT (injur\$ OR traum\$).ti,ab
3. (post-traumatic or posttraumatic).mp. AND (seizure\* or epilep\*).tw.
4. exp traumatic epilepsy/
5. OR/1-4
6. phenytoin/exp
7. pentobarbital/exp
8. carbamazepine/exp
9. valproic acid/exp
10. fosphenytoin/exp
11. (pentobarbit\* OR carbamazepine\* OR valpro\* OR fosphenytoin\* OR phenytoin\*).ti,ab.
12. (anticonvul\* OR anti-convul\* OR antiseiz\* OR anti-seiz\* OR antiepilep\*).ti,ab.
13. OR/6-12
14. 5 AND 13

## 10. What analgesia should patients with closed head injury receive?

### *MEDLINE*

1. exp craniocerebral trauma/
2. ((head or brain or cerebral or cranial) adj (traum\$ or injur\$)).tw.
3. OR/1-2
4. exp Analgesia/
5. analges\$.tw.
6. exp Analgesics, Opioid/
7. exp Morphinans/
8. exp Anti-Inflammatory Agents, Non-Steroidal/
9. exp Narcotics/
10. exp Narcotic Antagonists/
11. (paracetamol OR acetaminophen).tw.
12. (pain ADJ rel\$).tw.
13. (morphine or NSAID\$ OR fentanyl OR remifentanyl OR diclofenac OR meperidine OR alfentanyl OR sulfentanyl OR tramadol OR codeine OR oxycodone OR dihydromorphine).tw.
14. OR/4-13
15. 3 AND 14
16. limit 15 to (english language AND humans)

### *EMBASE*

1. craniocerebral Trauma/exp
2. ((head OR brain OR cerebral OR cranial) NEXT (traum\$ OR injur\$)).tw.
3. diffuse axonal injur\$.tw.
4. OR/1-3
5. Analgesia/exp
6. analges\$.tw.
7. Analgesics, Opioid/exp
8. Morphinans/exp
9. Anti-Inflammatory Agents, Non-Steroidal/exp
10. Narcotics/exp
11. Narcotic Antagonists/exp
12. (paracetamol OR acetaminophen).tw.
13. (pain NEXT rel\$).tw.
14. (morphine OR NSAID\$ OR fentanyl OR remifentanyl OR diclofenac OR meperidine OR alfentanyl OR sulfentanyl OR tramadol OR codeine OR oxycodone OR dihydromorphine).tw.
15. OR/5-14
16. 4 AND 15
17. limit 16 to (human AND english language)

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