CLINICAL PRACTICE GUIDELINE

EARLY MANAGEMENT OF HEAD INJURY IN ADULTS

MOH/P/PAK/304.15(GU)

Ministry of Health Malaysia

Neurosurgical Association of Malaysia

Academy of Medicine Malaysia

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http://www.moh.gov.my
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http://www.nam.org.my

Also available as an app for Android and iOS platform: MyMaHTAS

STATEMENT OF INTENT

These clinical practice guidelines (CPG) are meant to be guides for clinical practice, based on the best available evidence at the time of development. Adherence to these guidelines may not necessarily guarantee the best outcome in every case. Every healthcare provider is responsible for the management of his/her unique patient based on the clinical picture presented by the patient and the management options available locally. Priority of care in multisystem injuries is beyond the scope of this CPG.

REVIEW UPDATE

These guidelines were issued in 2015 and will be reviewed in a minimum period of four years (2019) or sooner if new evidence becomes available. When it is due for updating, the Chairman of the CPG or National Advisor of the related specialty will be informed about it. A discussion will be done on the need for a revision including the scope of the revised CPG. A multidisciplinary team will be formed and the latest systematic review methodology used by MaHTAS will be employed.

TABLE OF CONTENTS

LEVELS OF EVIDENCE

Level	Study design
I	Evidence from at least one properly randomised controlled trial
II-1	Evidence obtained from well-designed controlled trials without randomisation
II-2	Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one centre or group
II-3	Evidence from multiple time series with or without intervention. Dramatic results in uncontrolled experiments (such as the results of the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence
III	Opinions of respected authorities based on clinical experience; descriptive studies and case reports; or reports of expert committees

SOURCE: US / CANADIAN PREVENTIVE SERVICES TASK FORCE 2001

In line with the current development in CPG methodology, the CPG Unit of MaHTAS is in the process of adapting **Grading Recommendations, Assessment, Development and Evaluation (GRADE)** in its work process. The quality of each retrieved evidence and its effect size are carefully assessed/reviewed by the CPG Development Group. In formulating the recommendations, overall balances of the following aspects are considered in determining the strength of the recommendations:-

- overall quality of evidence
- balance of benefits versus harms
- values and preferences
- resource implications
- equity, feasibility and acceptability

GUIDELINES DEVELOPMENT AND OBJECTIVES

GUIDELINES DEVELOPMENT

The members of the Development Group (DG) for this Clinical Practice Guidelines (CPG) were from the Ministry of Health (MoH) and Ministry of Education. There was active involvement of a multidisciplinary Review Committee (RC) during the process of the CPG development.

A literature search was carried out using the following electronic databases: Guidelines International Network (G-I-N), Medline via Ovid, Pubmed and Cochrane Database of Systemic Reviews (CDSR) (refer to **Appendix 1** for **Example of Search Strategy**). The search was limited to literature published in the last ten years, on humans and in English. In addition, the reference lists of all retrieved literature and guidelines were searched to further identify relevant studies. Experts in the field were also contacted to identify further studies. All searches were conducted from 5 January 2014 to 9 July 2014. Literature searches were repeated for all clinical questions at the end of the CPG development process allowing any relevant papers published before 31 August 2015 to be included. Future CPG updates will consider evidence published after this cut-off date. The details of the search strategy can be obtained upon request from the CPG Secretariat.

References were also made to other CPGs on Head Injury such as Scottish Intercollegiate Guidelines Network (SIGN) - Early management of patients with a head injury (2009) and National Institute for Health and Clinical Excellence (NICE) - Head injury: Triage, assessment, investigation and early management of head injury in infants, children and adults (2014). The CPG was evaluated using the Appraisal of Guidelines for Research and Evaluation (AGREE) II prior to them being used as references.

A total of 27 clinical questions were developed under different sections. Members of the DG were assigned individual questions within these sections (refer to **Appendix 2** for **Clinical Questions**). The DG members met 25 times throughout the development of these guidelines. All literature retrieved were appraised by at least two DG members using Critical Appraisal Skill Programme checklist, presented in evidence tables and further discussed in each DG meetings. All statements and recommendations formulated after that were agreed upon by both the DG and RC. Where evidence was insufficient, the recommendations were made by consensus of the DG and RC. This CPG is based largely on the findings of systematic reviews, meta-analyses and clinical trials, with local practices taken into consideration.

The literature used in these guidelines were graded using the US/ Canadian Preventive Services Task Force Level of Evidence (2001), while the grading of recommendation was done using the principles of GRADE (refer to the preceding page).

On completion, the draft of the CPG was reviewed by external reviewers. It was also posted on the MoH Malaysia official website for feedback from any interested parties. The draft was finally presented to the Technical Advisory Committee for CPG, and the HTA and CPG Council MoH Malaysia for review and approval. Details on the CPG development by MaHTAS can be obtained from Manual on Development and Implementation of Evidence-based Clinical Practice Guidelines published in 2015 (available at http://www.moh.gov.my/index.php/pages/view/117).

OBJECTIVES

To provide evidence-based guidelines to those involved in the early management of head injury in primary and secondary/tertiary care

CLINICAL QUESTIONS

Refer to **Appendix 2**

TARGET POPULATION

Inclusion Criteria

Adult patients presenting with head injury (18 years old and above)

Exclusion Criteria

The guidelines do not cover definitive management of head injury:
- all surgeries pertaining to neurosurgery and post-operative care
- rehabilitation
- management of multisystem injuries

TARGET GROUP/USERS

This document is intended to guide healthcare professionals and relevant stakeholders including:
i. Doctors
ii. Pharmacists
iii. Allied health professionals
iv. Medical students and healthcare trainees
v. Professional societies
vi. Patients and carers/non-governmental organisations

HEALTHCARE SETTINGS

Primary or secondary/tertiary care settings

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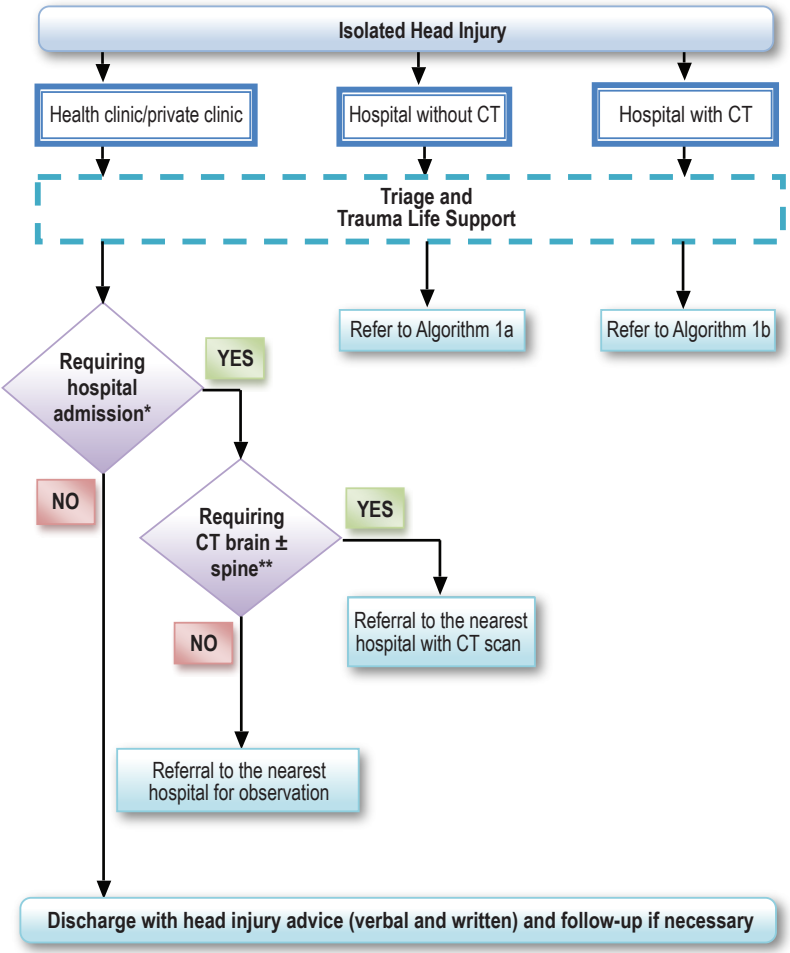
ALGORITHM 1. GENERAL MANAGEMENT OF ADULTS WITH ISOLATED HEAD INJURY



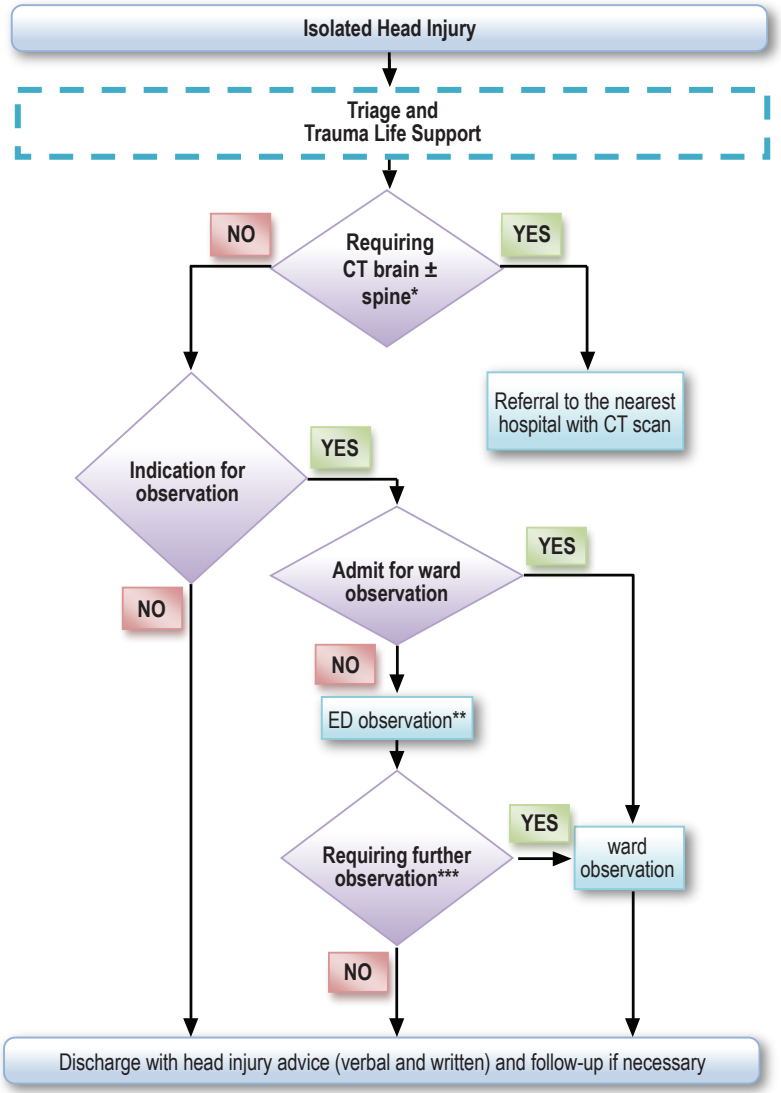
If a healthcare provider is unsure which hospital to refer to, the patient should then be referred to the nearest hospital.

* Refer to **Recommendation 3** on **Criteria of Referral to Hospital on Patients with MHI**.

** Refer to **Algorithm 3** on **Selection of Adults with Head Injury for Head CT**.

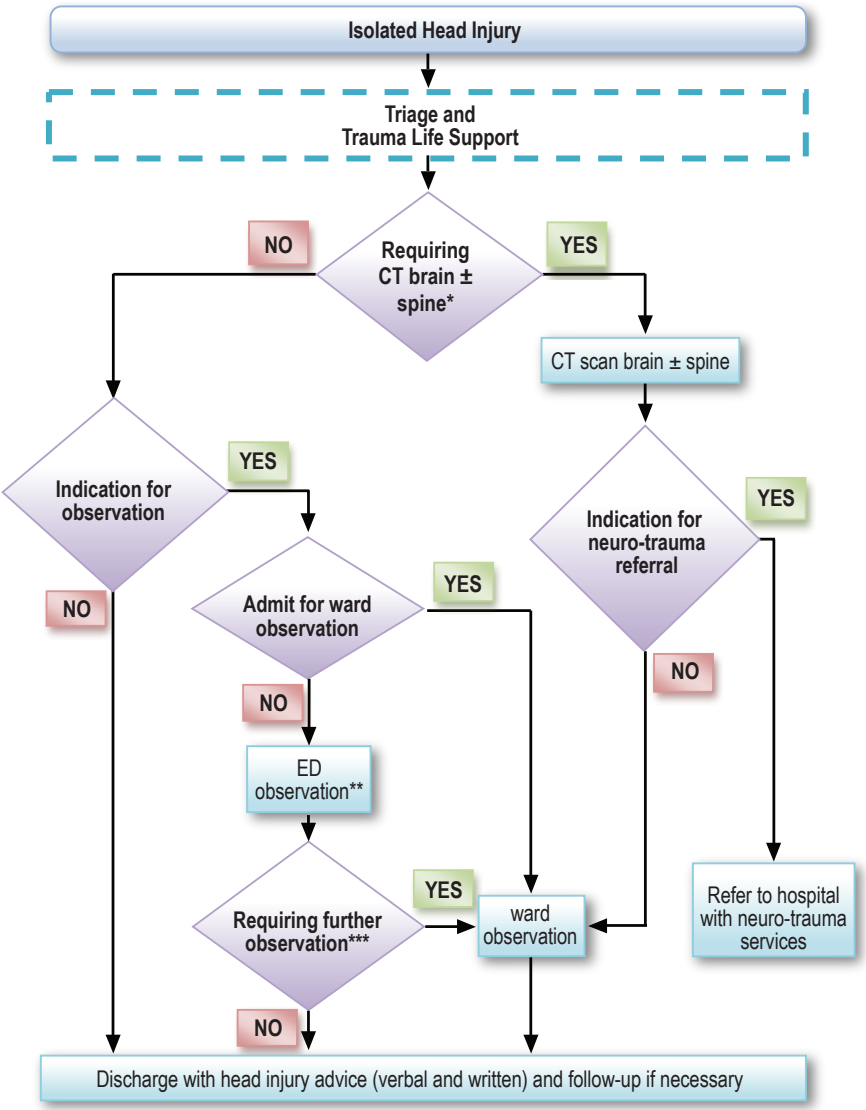
CT = computed tomography

ALGORITHM 1a. GENERAL MANAGEMENT OF ADULTS WITH ISOLATED HEAD INJURY IN HOSPITAL WITHOUT CT SCAN



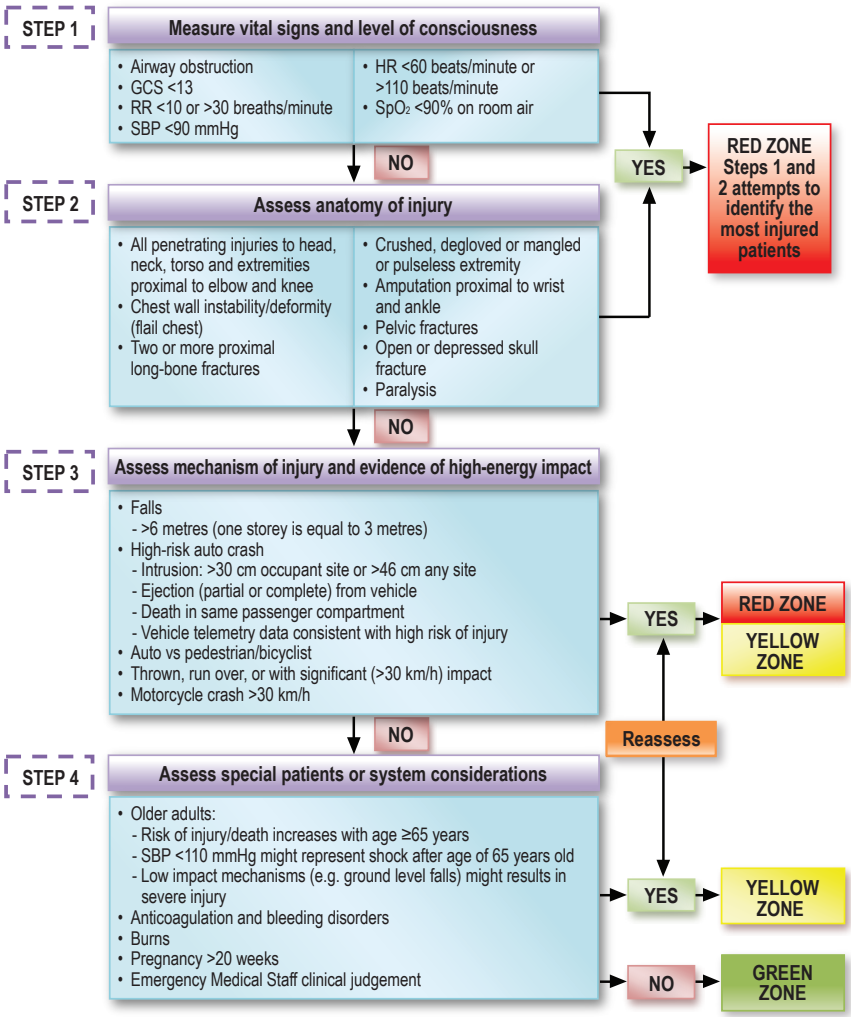
* Refer to **Algorithm 3** on **Selection of Adults with Head Injury for Head CT**.
** Refer to **Recommendation 7** on **Criteria of Patients with Mild Head Injury (MHI) who can be Safely Observed in ED**.
*** Refer to **Recommendation 10** on **Criteria for Admission of MHI Patients Post-observation in ED**.
CT = computed tomography; ED = Emergency Department

ALGORITHM 1b. GENERAL MANAGEMENT OF ADULTS WITH ISOLATED HEAD INJURY IN HOSPITAL WITH CT SCAN



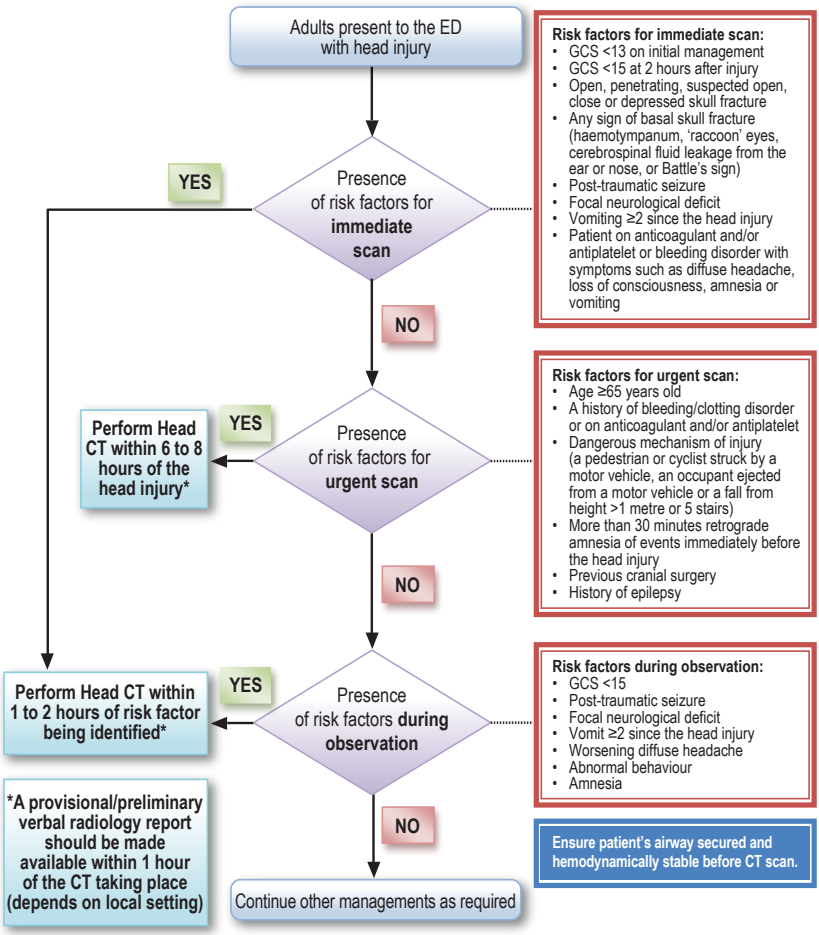
* Refer to **Algorithm 3** on **Selection of Adults with Head Injury for Head CT**.
** Refer to **Recommendation 7** on **Criteria of Patients with Mild Head Injury (MHI) who can be Safely Observed in ED**.
*** Refer to **Recommendation 10** on **Criteria for Admission of MHI Patients Post-observation in ED**.
CT = computed tomography; ED = Emergency Department

ALGORITHM 2. TRIAGING OF ADULTS WITH SUSPECTED HEAD INJURY IN PRE-HOSPITAL CARE OR EMERGENCY DEPARTMENT



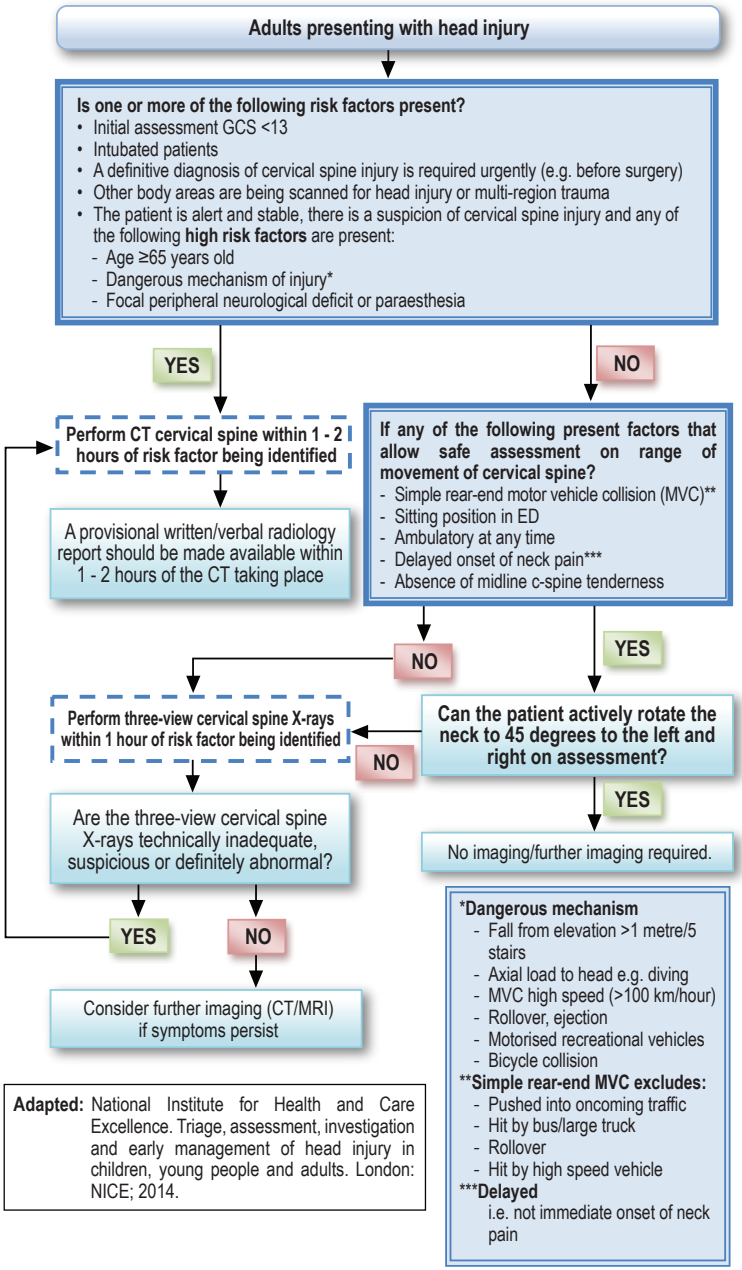
Adapted: Sasser SM, Hunt RC, Faul M, et al. Guidelines for field triage of injured patients: recommendations of the National Expert Panel on Field Triage, 2011. MMWR Recomm Rep. 2012 Jan 13;61(RR-1):1-20

ALGORITHM 3. SELECTION OF ADULTS WITH HEAD INJURY FOR HEAD CT



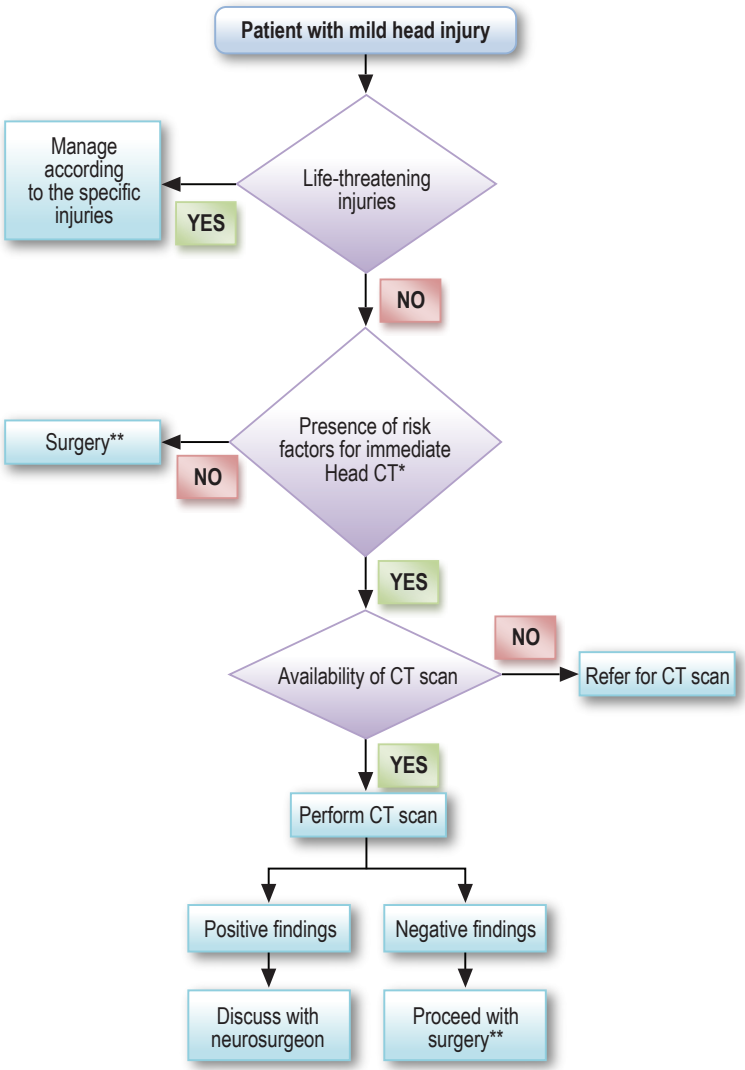
Adapted: National Institute for Health and Care Excellence. Triage, assessment, investigation and early management of head injury in children, young people and adults. London: NICE; 2014.

ALGORITHM 4. SELECTION OF ADULTS WITH HEAD INJURY FOR IMAGING OF THE CERVICAL SPINE



Adapted: National Institute for Health and Care Excellence. Triage, assessment, investigation and early management of head injury in children, young people and adults. London: NICE; 2014.

ALGORITHM 5. MANAGEMENT OF ADULTS WITH MILD HEAD INJURY REQUIRING URGENT SURGERY OTHER THAN CRANIAL SURGERY



* Refer to **Algorithm 3** on **Selection of Adults with Head Injury for Head CT**.
** Anaesthesia administration adheres to principles of neuroanesthesia.

1. INTRODUCTION

In the Malaysian National Trauma Database 2009 report, blunt trauma made up 96% of all injuries. Road traffic accident (RTA) accounted for 75% of cases with motorcyclists being most commonly injured. The younger age group of 15 - 34 years old (56.6%) is at highest risk of major trauma. A high proportion of those with major trauma (85%) had injuries to the head and neck with Abbreviated Injury Scale (AIS) ≥3.[1, level III]

Injury was the fifth (7.86%) commonest cause of hospitalisation in Malaysian public hospitals in 2014[2, level III] with RTA being the commonest cause of injury-related hospitalisation. Apart from that, head injury constituted the commonest diagnosis leading to intensive care unit (ICU) admission.[3, level III]

Based on the World Health Organization Global status report on road safety 2013, the road traffic fatality rate in Malaysia was higher than global rate (25 vs 18 per 100 000 population). Many lives can be saved with good pre-hospital care (PHC) and quick transportation to hospital.[4, level III]

Strengthening of emergency services and PHC were identified as crucial in improving secondary care as stated in the country health plan strategy 2011 - 2015 report. Subspecialties development will continue to be strengthened in regional hospitals including expansion and strengthening of tertiary care related to trauma.[5, level III]

A local study on the effectiveness of trauma services provided by secondary and tertiary hospitals showed an 83% mortality reduction in severe injuries among those admitted to hospital with specialist trauma services compared with those without such services. Nevertheless, they had a higher likelihood of disability and impairment upon discharge. It was concluded that improvement in access to trauma services for severely injured patients and standardisation in management of trauma care within hospitals in the country were required.[6, level III]

A specialty and subspecialty framework of MoH hospitals 10th Malaysia Plan (2010 - 2015) reported that Hospital Kuala Lumpur and 13 states hospitals will be provided with neurosurgery subspecialty to manage complicated head injury.[7, level III] However, currently there are only nine states and one district hospital with an additional three university hospitals providing neurosurgical services.

In view of the above issues, this first evidence-based local CPG has been developed with the aim of guiding healthcare providers in the early management of head injury. It intends to reduce clinical practice variation, provide optimum care for head injury patients and eventually

achieve significant reduction in mortality and morbidity related to the condition. It also helps in optimising limited neurosurgical subspecialty services in the country with a referral system that includes tele-health services.

2. DEFINITION

Head injury is defined as blunt and/or penetrating injury to the head (above the neck) and/or brain due to external force* with temporary or permanent impairment in brain function which may or may not result in underlying structural changes in the brain.

One or more of the following conditions from anatomical and physiological changes must be present in patients with head injury:[8]

1. Physiological changes
   - observed or self-reported loss of or decreased level of consciousness (any duration)
   - any loss of memory (amnesia) of events immediately before or after the injury
   - any alteration in mental state or neuropsychological abnormality at the time of the injury [such as loss of consciousness (LOC), confusion, disorientation and slow thinking]
   - objective neurological deficits (such as weakness, loss of balance, change in vision, praxis, paresis/paraplegia, sensory loss and aphasia) that may or may not be transient

2. Anatomical changes
   - scalp and/or facial wound or swelling
   - skull fracture (facial, basilar or vault) and/or clinical signs of skull fracture***
   - diagnosed intracranial lesion such as brain parenchyma injury, injury to intracranial blood vessels, injury to the dura mater, intracranial haemorrhage (ICH), subarachnoid haemorrhage or intraventricular haemorrhage

- To define head injury, criteria i and ii must be present with/without criteria iii:
  i. mechanism - presence of external force*
  ii. physiological - alteration in physiology of the brain
  iii. anatomical - scalp and/or face and/or skull and/or brain injury (internal and external)**

*The external forces may include any of the following events:
- the head being struck by an object
- the head striking an object
- the brain undergoing an acceleration/deceleration movement without direct external trauma to the head
- a foreign body penetrating the brain
- forces generated from events such as blast or explosion, or other forces yet to be defined

**The anatomical or mechanical changes in the brain such as axonal injury which may not be visualised in the CT scan does not rule out such injury.

***Clinical signs of skull base fractures such as periorbital ecchymosis ("Raccoon's eyes"), retro-auricular ecchymosis ("Battle's sign"), cerebrospinal fluid leakage either from the nose [cerebral spinal fluid (CSF) rhinorhoea] or ear (CSF otorhoea), or seventh and eighth cranial nerves deficits causing facial paralysis and hearing loss respectively.

3. CLASSIFICATION OF SEVERITY

The severity of head injury can be classified according the presenting Glasgow Coma Score (GCS):[9 level III]

- mild head injury (MHI): GCS 13 - 15
- moderate head injury: GCS 9 - 12
- severe head injury: GCS 3 - 8

The MHI group can be subdivided into two types as shown in **Table 1**.[10 - 11, level III]

Table 1. Classification of MHI

<table>
<tr><th colspan="3">Cerebral concussion</th><th colspan="3">Mild head injury</th></tr>
<tr><th colspan="3">GCS 15</th><th colspan="3">GCS 13 - 15</th></tr>
<tr><td colspan="3">Should only be used if there is no imaging evidence of brain injury</td><td colspan="3">With or without imaging evidence of brain injury</td></tr>
<tr><td colspan="3">With or without history of LOC</td><td colspan="3">Requires a LOC (≤30 minutes) or post-traumatic amnesia (≤24 hours)</td></tr>
<tr><td colspan="3">Can be further subdivided into:</td><td colspan="3">Can be further subdivided according to the risk of deterioration and expected outcome:</td></tr>
<tr><th>Grade 1</th><th>Grade 2</th><th>Grade 3</th><th>Low Risk</th><th>Medium Risk</th><th>High Risk</th></tr>
<tr><td>• History of confusion
• No history of LOC
• Concussion symptoms resolve in <15 minutes</td><td>• History of confusion
• No history of LOC
• Concussion symptoms last >15 minutes</td><td>• History of LOC either brief (seconds) or prolonged (minutes)</td><td>GCS 15
With one or more of the clinical findings</td><td colspan="2">GCS 14 - 15
With neurological deficits or skull fracture or risk factors with/without clinical findings</td><td>GCS 13</td></tr>
<tr><td colspan="3">Concussion symptoms are divided into four categories:
1. Physical
headache, fuzzy or blurry vision, sensitivity to noise and/or light, dizziness, feeling tired and lacking energy, and problems with balance
2. Cognition/memory
difficulty thinking clearly, feeling slowed down, trouble concentrating, and difficulty remembering new information
3. Emotional/mood
irritability, inexplicable sadness, nervousness and/or anxiety
4. Sleep disturbances
interruptions in normal sleep patterns</td><td colspan="3">Clinical findings:
LOC, amnesia, diffuse headache, vomiting

Risk factors:
Coagulopathy, alcohol consumption and/or drug misuse, age ≥65 years old, previous cranial surgery and history of pre-trauma epilepsy</td></tr>
</table>

Refer to **Appendix 3** on **Glasgow Coma Scale**.

4. DIFFERENTIAL DIAGNOSES

The differential diagnoses of adult head injury include the following:

- primary anoxic, inflammatory, infectious, toxic or metabolic encephalopathies, which are not complications of head trauma
- neoplasm
- brain infarction (ischaemic stroke) and intracranial haemorrhage (haemorrhagic stroke) without associated trauma
- alcohol intoxication, psychotropic drugs or substance abuse
- seizure

5. PRE-HOSPITAL CARE (PHC)

PHC is defined as an immediate assistance a patient receives from PHC providers before arriving at hospital. Mortality and morbidity due to head injury may be reduced by adequate pre-hospital trauma care.

The first responder who recognises the emergency situation is encouraged to rapidly activate emergency response system by dialling 999 and provide basic first aid measures. Most deaths in the first hour after injury are the result of airway and circulatory systems compromise. Appropriate assessment, stabilisation and care of trauma victim including referral and transportation to appropriate receiving facility will help avoid secondary brain injury.

5.1 Assessment and General Treatment

The Glasgow Coma Scale and Score are widely used in patients with head injury due to their good reliability and validity. Patient's best eye, motor and verbal response are assessed and categorised into mild (GCS 13 to 15), moderate (GCS 9 to12) and severe (GCS 3 to 8).[12] These are reliable tools to monitor and detect deterioration in patients with head injury. Alcohol intoxication, sedative medications, hypoxia and hypotension can influence the conscious level. Therefore any assessment of GCS should be repeated after resuscitation from cardiopulmonary insult or recovery from intoxication and sedation in patients with head injury. Other assessments that can be used other than GCS are Head (AIS) and Trauma Score - Injury Severity Score.

- GCS score can be influenced by alcohol intoxication, sedative medications, hypoxia and hypotension.
- Training on the use of GCS (technique and application) is essential for accurate assessment of patients with head injury.

Total GCS and GCS Motor Scores including those at pre-hospital are stronger predictors of 2-weeks mortality than Head AIS Score scales

after traumatic brain injury (TBI). In those aged ≥60 years, irrespective of motor scale component of GCS, total GCS score and head AIS score is an independent risk factor for the mortality.[13, level II-2]

Alcohol intoxication (blood alcohol concentration >0.08%) is an important confounder which may influence pre-hospital assessment of GCS in patients with TBI ($p<0.001$).[14, level II-2]

Recommendation 1
- Glasgow Coma Scale (GCS) and Glasgow Coma Scale Score (GCS score) should be used in the assessment of all patients with head injury by trained healthcare providers.

5.2 Initial Management

5.2.1 Airway and ventilation

The most important pre-hospital management in head injury is the maintenance of airway, breathing and circulation. Hypoxaemia [oxygen saturation (SpO_2) <90%] is the most important factor related to worse outcome (more mortality and severe disability with SPO_2 less than 90%) and therefore needs urgent attention.[15]

A systematic review of studies on pre-hospital intubation in severe head injury concludes:[16, level II-2]
- The adjusted ORs for in-hospital mortality ranged from 0.24 (95% CI 0.11 to 0.49) to 1.42 (95% CI 1.13 to 1.78).
- There were inconclusive results on functional outcome.
- Intubation failure or complication rates (e.g. pneumonia) ranged from 2.1% to 41.1%.

Although there was insufficient evidence to suggest any recommendation, benefit and harm of pre-hospital intubation in severe TBI depend on organisation of PHC services, skills of staff, risk of procedure failure and expected transport times.[16, level II-2]

An airway should be established by the most appropriate means available in severe TBI (GCS <9) to maintain an adequate airway or when hypoxaemia is not corrected by supplemental oxygen.[15]

Patients should be maintained with normal breathing rates [End tidal carbon dioxide (ET CO_2) 35 - 40 mmHg]. Hyperventilation (ET CO_2 <35 mmHg) should be avoided unless the patients shows sign of cerebral herniation.[15]

Manual in line stabilisation must be performed in suspected cervical spine injury and rapid sequence intubation is the preferred choice of

intubation technique as it is proven to be not associated with increased risk of adverse neurological outcomes.[17, level I]

If tracheal intubation is required, manual in-line stabilisation is provided by an assistant during intubation by using the fingers and palms of both hands to stabilise the patient's occiput and mastoid processes to gently counteract the forces of airway intervention. Prior to this maneuver, the anterior half of a semi rigid collar is removed. The assistant stands at the head or side of the patients. Manual in-line stabilisation anchors the occiput and the torso anchors the lower cervical spine.[18, level III] The procedure should be performed with caution as laryngoscopic forces can be translated to mid-cervical spine.

5.2.2 Cervical spine

Unstable spinal injury may occur concomitantly with TBI and may lead to permanent neurological deficit if not addressed appropriately.[19, level III] Hence, cervical injury must be considered in all head injury.

Cervical spine immobilisation should be carried out in TBI patients with any of the following risk factors:[20]

- GCS <15 on initial assessment by the healthcare professional
- paraesthesia in the extremities
- neck pain or tenderness
- focal neurological deficit
- any other clinical suspicion of cervical spine injury

Maintain cervical spine immobilisation until full risk assessment including clinical assessment (and imaging if deemed necessary) indicates it is safe to remove the immobilisation device.[20]

5.2.3 Circulation

Hypotension is defined as a SBP <90 mmHg in adults.[15] Untreated hypotension will produce secondary brain insult and studies have demonstrated that a single episode of hypotension significantly worsens the outcome.[21, level III] Treatment includes fluid and vasopressors to maintain a mean arterial pressure (MAP) of 80 mmHg or more.[20]

There is no benefit on survival or functional outcome, and also no difference in hospital length of stay by using hyperosmolar crystalloid or colloid solutions over isotonic crystalloids in pre-hospital fluid resuscitation of patients with TBI. Hypotension and hypoxia however must be avoided, and fluid resuscitation should be sufficient to maintain cerebral perfusion.[22, level I]

A multicentre Saline Albumin Fluid Resuscitation Trial showed that the use of albumin was associated with higher mortality in patients with

severe head injury. Among patients with severe brain injury (GCS 3 - 8), 41.8% of them died in the albumin group as compared with 22.2% patients in the saline group. However in those with GCS scores of 9 - 12, death occurred in eight of 50 patients in the albumin group (16.0%) and eight of 37 patients in the saline group (21.6%).[23, level I]

Recommendation 2

- Hypoxaemia [oxygen saturation (SpO_2) <90%] should be avoided in head injury and corrected immediately upon identification. Oxygen supplementation should be given to all patients with head injury to prevent hypoxaemia.
- An airway should be established in head injury patients, by the most appropriate means* available in the following conditions:
  - severe head injury (Glasgow Coma Scale ≤8)
  - inability to maintain an adequate airway
  - hypoxaemia not corrected by supplemental oxygen

  Manual in-line immobilisation should be performed during intubation.
- Cervical collar should be applied in head injury patients with suspected cervical injury**.
- Isotonic crystalloid is the preferred choice of intravenous fluid resuscitation in head injury.

* Use either definitive (e.g. endotracheal intubation) or non-definitive airway (e.g. laryngeal mask airway) depending on expertise and resources.

**Refer to the related preceding text.

5.3 Referral or Discharge at Primary Care Setting

The need to make a decision to refer or discharge a patient with head injury who is seen in a primary health setting can be very challenging. This is especially so in presumably mild cases as there is a possibility that the cases may have pre-existing risk factors to intracranial bleeding or symptoms masked by intoxication.

Patients presenting with MHI with the following findings may eventually require neurosurgical intervention:[24, level III]

- restlessness
- decrease in GCS score within six hours
- severe headache
- focal temporal blow
- vomiting
- confusion

Male gender (OR=3.82, 95% CI 1.60 to 9.13) and alcohol intoxication (OR=12.44, 95% CI 2.14 to 72.38) are important risk factors of delayed

referral.[25, level III] A healthcare provider should be aware that a quick and accurate decision of referral for patient with presumably mild head injury can be hindered if the patient is male and intoxicated by alcohol.

There is a possibility of ICH after minor head trauma in patients taking anticoagulant and/or antiplatelet medication. The frequency of a positive computed tomography (CT) with regards to anticoagulation is 27%, antiplatelet 41% and combined therapy 14%.[26, level III]

Low platelet count in TBI patients on antiplatelet therapy contributed to negative outcome. Patients on clopidogrel therapy and high-dose aspirin therapy (325 mg) (p=0.001) are more likely to have progression of ICH and require neurosurgical intervention (p=0.01) compared with patients on low-dose aspirin therapy (81mg). Platelet count of ≤135,000/μL is the strongest predictor for progression of initial ICH (p<0.001) on repeat head computed tomography (RHCT). A lower platelet count i.e. ≤95,000/μL is the strongest predictor (p<0.001) for neurosurgical intervention.[27, level III]

In older patients (≥55 years old) with TBI, those on anticoagulation therapy are 4.6 times (95% CI 1.08 to 19.6) more likely to have a secondary intracranial haemorrhage event (SIHE) compared with antiplatelet users and non-users on follow-up. The anticoagulant users are also more likely to have progression of initial haemorrhage.[28, level II-2]

Predictors of mortality for patients who present with isolated moderate to severe TBI were assessed using a multivariable logistic model. Age, male gender, ISS ≥16, GCS score ≤8, systolic blood pressure, and diabetes mellitus (DM) were significant predictors of mortality after moderate to severe TBI. DM was associated with an increased for mortality OR of 1.5 (95% CI 1.29 to 1.74).[29, level III]

In a cohort study of isolated TBI patients, pre-hospital hypertension of >160 mmHg was an predictor for in-hospital mortality:[30, level III]

- OR for pre-hospital SBP of 160 - 180 mmHg was 1.33 (95% CI 1.22 to 1.44)
- OR for pre-hospital SBP of 190 - 230 mmHg was 1.97 (95% CI 1.76 to 2.21)

A deviation from admission heart rate (HR) of 70 beats per minute (bpm) to 89 bpm was associated with increased mortality (p<0.001).

Another cohort study factors showed that factors associated with mortality in TBI patients were:[31, level II-2]

- GCS <9 (HR=2.08, 95% CI 1.25 to 3.46)
- abnormal pupil (HR=2.08, 95% CI 1.25 to 3.46)
- ISS score ≥25 (HR=2.62, 95% CI 1.48 to 4.63)
- SBP <90 mmHg (HR=2.13, 95% CI 1.12 to 4.04)
- hypothermia <35°C (HR=1.42, 95% CI 1.00 to 2.01)

In local context, social issues such as transport, communication problem or no supervision by a responsible adult should be considered when referring patients with mild head injury.

Recommendation 3

- Referral of patients with mild head injury to the nearest hospital should be considered especially if they have any of the following factors:
  - Glasgow Coma Scale (GCS) of 15 but symptomatic such as amnesia, headache, vomiting or restlessness
  - age ≥65 years old
  - treated with antiplatelets or anticoagulants
  - GCS <15 and/or declining GCS score
  - alcohol intoxication and substance misuse
  - focal temporal blow
  - social issues
  - indicated for head CT (refer to **Algorithm 3** on **Selection of Adults with Head Injury for Head CT)**

5.4 Transportation

Surgical neuro-trauma management in Malaysia is provided by general and/or neurosurgeons. Many patients with head injuries are transferred to these hospitals by road or occasionally by air or river/sea. During transportation, patients with impaired consciousness and physiological instability may develop secondary insults and are predisposed to worse outcome. Adhering to best practice directed by evidence-based protocols or management prior and during transport can help minimise these adverse events.[12]

Patients with head injury should be transferred in a safe and timely manner. The mode and rapidity of transferring patients with head injury in the pre-hospital setting are based on the risk of intracranial complications and severity of head injury.

Refer to **Appendix 8** on Equipments for Transfer.

5.4.1 Transferring head injury patients from the scene of injury to hospital

Patients with suspected TBI preferably should be transported from the scene directly to a centre where TBI can be managed in its entirety (centre with resources necessary to resuscitate, investigate and manage any patients with major trauma or suspected polytrauma). Alternatively patients can be directly transported to a centre capable of initial management. Calls should be made earlier to inform the ED to standby especially in cases of GCS ≤8. Cervical spine immobilisation should

be maintained during transfer until a full assessment and appropriate investigations are performed. The mode of transport selected should minimise total pre-hospital time. On scene management of TBI patient and transfer must follow current standard management. Unnecessary delay in transfer must be avoided.[32]

Transferring polytrauma patients with suspected TBI to an appropriate facility with established trauma system is associated with a 15% reduction in overall mortality.[32]

5.4.2 Transfer of Head Injury Patients from Clinic to Hospital

- Criteria for rapid transfer:[32]
  - deterioration in patient's condition
  - GCS <15
  - focal neurological deficit
  - seizure
  - suspected skull fracture or penetrating head injury
  - high impact head injury
  - suspected neck injury

Patients referred from primary care should be accompanied by a competent adult. The referring health care providers should determine whether an ambulance or other mode of transport is required. Public transport and car may be an appropriate alternative provided the patient is clinically stable and accompanied. The referring health care providers should contact the referral hospital by phone of the impending transfer.[20] The decision to transfer patients from a rural location should be discussed between the referring and receiving healthcare providers.[12] The rapidity and mode of transport from the pre-hospital setting to ED depend on the risk of clinically important head injury and the acute complications of head injury.

5.4.3 Transfer of Head Injury Patient from ED/Hospital without Neuro-trauma Service to Neuro-trauma Centre

Patients with persistent hypotension despite resuscitation should have the cause of hypotension identified and managed in a hospital with surgical services. Advanced airway management including intubation may be required prior to transfer and must be done by trained healthcare providers.[33, level I] The risk of secondary brain injury during transfer to tertiary centre is high if poorly executed. Transfer purely for the purpose of imaging in unstable patients should be avoided.[12]

- Initial management including resuscitation and stabilisation are essentials, and should be completed before transferring TBI patients:[15; 19, level III; 20]
  - Intubate* and ventilate patients with TBI if:
    - GCS ≤8 or significant drop of total GCS score ≥2 or motor component ≥1
    - unstable fractures of the facial skeleton
    - copious bleeding into mouth (e.g. from skull base fracture)
    - hypoxaemia
    - seizure
    - respiratory irregularities
    - hyperventilation causing hypocarbia (keep ET CO_2 35 - 40 mmHg)
  - Cause of persistent hypotension should be identified and managed in hospital with surgical services.
  - If patient has seizures, treat with anticonvulsants (refer to **Chapter 8.4** on **Anticonvulsant**).

  *if intubation is not possible, LMA insertion is acceptable

During transfer, hypotension, hypercarbia and hypoxia are the main causes of secondary brain damage in patients with TBI. Hence, it is important to maintain adequate oxygenation and ventilation with appropriate short-acting sedation and analgesia and to avoid SpO_2 <90%.[19, level III]

The general principles of safe transfer are:
- ensure haemodynamic optimisation (Refer to **Section 5.2** on **Pre-hospital Management** and **Section 8.2** on **Intravenous Fluid**)
- secure bleeding prior to transfer e.g. apply haemostatic suturing for bleeding scalp wounds and splints for long bone fractures.[19, level III]
- intubation if required (Refer to **Section 5.2** on **Pre-hospital Management**)
- cervical immobilisation
- the transfer team should be trained in neuro-trauma management
- reliable communication equipment is essential to allow effective communication between the transfer team with their hospital and the neurosurgical team
- completed transfer checklist for neurosurgical patients (Refer to **Appendix 9** on **Transfer Checklist for Neurosurgical Patients**)

Stretcher is preferable to traditional spinal board. Hence, patient should be taken off the spinal board while awaiting transfer. There is insufficient evidence of safe duration for patient immobilisation on spinal board.[17, level I]

- The fundamental requirements prior to transfer include adequate oxygenation delivery and optimised vital parameters:[32]
  - target MAP >80 mmHg, PaO_2 >97.5 mmHg (13 kPa), $PaCO_2$ within 35 - 40 mmHg, SpO_2 >90%
- Parameters to monitor during transfer include:
  - cardiac monitoring, BP, pulse oximetry, capnography (if available), pupil size and reaction to light ± urine output
- Investigations before transfer when necessary are*:
  - full blood count, coagulation screen, blood sugar, blood group and cross-match, arterial blood gases, radiological investigations [CT scan (where available), chest x-ray, pelvic X-rays and other investigations as appropriate]

*The investigations should not delay transfer.

A copy of the summary and transfer record should be kept in the referring hospital for audit purposes and potential medico-legal consideration.[19, level III]

Recommendation 4

- Hypotension, hypoxia, hypocarbia and hypercarbia, and inadvertent cervical injury should be avoided before and during transfer of patients with head injury.
- Continuous monitoring of vital signs should be conducted during transfer of head injured patients*.
- Effective communication should be established between the transfer team and receiving hospital of head injury cases.

*Refer to **Appendix 9** on **Transfer Checklist for Neurosurgical Patients**.

6. MANAGEMENT IN EMERGENCY DEPARTMENT (ED)

6.1 Triage

The purpose of triage is to ensure the right patient receives appropriate timely treatment in the right place before she or he deteriorates. A systematic method ensures quick work flow process. Triage of patients with head injury in many countries has been based on American College of Surgeon-Committee on Trauma/Centre for Disease Control (ACOS-COT/CDC) 2011 Physiologic and Anatomical Guidelines for field triaging of injured patients.[34, level III]

Based on this guideline, there is increased likelihood of death in TBI with the following physiological factors:[34, level III]

- GCS ≤13 (OR=17.4, 95% CI 10.7 to 28.3)
- Respiratory rate (RR) <10 breaths per minute and >29 breaths per minute in adults (OR=20.3, 95% CI 13.4 to 30.8)
- SBP <90 mmHg (OR=18.6, 95% CI 14.0 to 24.7)

Combination of all three physiologic criteria markedly increases the OR to 67.8 (95% CI 48.3 to 95.3).

In trauma patient, with the following anatomical criteria outlined in the same guidelines, there is increased likelihood of death:[35, level III]

- flail chest (LR=42.4, 95% CI 6.3 to 284.0)
- open or depressed skull fracture (LR=18.9, 95% CI 11.6 to 30.8)
- pelvic fracture (LR=12.3, 95% CI 9.1 to 16.5)
- penetrating injury to the head, neck, torso and extremities (LR=49.7, 95% CI 42.3 to 58.3)
- two or more proximal long-bone fractures (LR=7.1, 95% CI 5.0 to 10.1)

An earlier study showed that the predictors of mortality in patients with blunt TBI were:[36, level II-2]

- age (OR=1.04. 95% CI 1.01 to 1.07)
- GCS <9 (OR=19.29, 95% CI 5.04 to 73.82)
- skull bone fracture (OR=10.44, 95% CI 3.59 to 30.38)

A four-step algorithm based on physiologic abnormalities, anatomic injuries, mechanism of injuries, and co-morbidities and age (modified from ACOS-COT/CDC triaging guidelines) shows that the following factors are associated with a higher risk of undertriage:[37, level III]

- isolated head injury with GCS 13-15 (talk and deteriorate) (OR=14.0, 95% CI 4.55 to 43.3)
- age of 45-54 years old (OR=10.8, 95% CI 1.88 to 61.7)
- isolated pelvic injury (OR=14.2, 95% CI 2.58 to 78.0)
- night time arrival (OR=2.45, 95% CI 1.04 to 5.79)

Triage of patients suspected of head injury should follow an Algorithm 2 on Triaging of Adults with Suspected Head Injury in PHC or ED which has been adapted from ACOS-COT/CDC triaging guidelines.[34, level III]

Recommendation 5
- Triage of patients suspected of head injury in pre-hospital care or on arrival in emergency department should follow a four-step algorithm* based on physiologic abnormalities, anatomic injuries, mechanism of injuries, co-morbidities and age.

*Refer to **Algorithm 2** on **Triaging of Adults with Suspected Head Injury PHC or ED**.

6.2 General Treatment

Initial management of patients with head injury should be performed according to primary survey of trauma patients as recommended by Advanced Trauma Life Support guidelines:[38, level III]
- airway patency and cervical spine protection
- breathing
- circulation and haemorrhage control
- disability
- exposure

- Stabilisation of airway, breathing and circulation is the priority for all ED patients before attending to other injuries.
- In patients with depressed conscious level, head injury need to be ruled out before diagnosis of intoxication is made.
- All healthcare providers in ED should be trained in the assessment of patients with head injury and identification of risk factors for CT head and cervical spine imaging.

In patients with head injury with GCS of ≤8, intubation is recommended for airway protection. To prevent secondary injuries, parameters such as PO_2, PCO_2, BP, PR, SpO_2 and glucose level should be assessed and be normalised.[38, level III]

National Trauma Data Bank study, which included mild and moderate head injury, base deficit and mortality analysis, showed that the first presenting SBP <110 mmHg in hospital was a more clinically relevant definition of hypotension and hypoperfusion than SBP <90 mmHg. This was independent of both age and gender of the patients. There was an increase of approximately 6% in mortality for every 10 mmHg decrease below SBP 115 mmHg, with a maximum of 40% mortality

at SBP 60 mmHg. However, further studies are warranted to support these findings.[39, level III]

In traumatic ICH, those who do not require critical care interventions are patients with full GCS, isolated head injury and age <65 years (AUC=0.74, 95% CI 0.70 to 0.77).[40, level II-2]

There is a significant association between death and age (OR=1.04), GCS (OR=0.59), ISS (OR=1.03), mean arterial BP (OR=0.71) and RR (OR=0.82) in patients with TBI.[41, level III]

Recommendation 6

- Initial assessment and management of patients with head injury in emergency department (ED) includes:
  - airway patency and cervical spine protection
  - breathing (to detect any intrathoracic injury)
  - circulation and haemorrhage control
  - disability including Glasgow Coma Scale (GCS), pupil size and reaction to light
  - exposure including log roll
- Secondary survey (head to toe examinations) should be done in patients with head injury which includes signs of base of skull fracture*.
- Head chart which includes serial GCS, blood pressure, pulse rate and pupil size should be done at least hourly on patients with head injury in ED including while awaiting computed tomography or transfer to another ward/hospital.
  - Monitor for signs of intracranial hypertension such as decreased pupillary response to light, hypertension with bradycardia, posturing or respiratory abnormalities.

*Refer to **Chapter 7** on **Imaging - Skull X-ray**.

6.3 Observation in ED

Head injury is one of the common reasons for ED visits. Most cases are mild and self-limiting. However they usually require further observation after initial systematic assessment and treatment. The primary aim is to detect promptly patients who deteriorate neurologically and to ensure safe home discharge.

Early imaging, rather than admission and observation for neurological deterioration, will reduce the time to detection for life-threatening complications and is associated with better outcomes.[11, level II-2] Patients with MHI (who do not have criteria for CT scan) can be managed in ED.

The management at ED observation wards is efficient at dealing with short stay observation patients.[20]

6.3.1 Patients who can be safely observed in ED

Patients with MHI (who do not meet the criteria for head CT) can be managed in the ED observation ward.

In the management of head injury, patients with GCS score of 15 and normal neurological findings who either have CT scan performed immediately or undergo routine observation have similar extended Glasgow outcome score (GOS) at three months follow-up.[42, level I]

In a RCT, all adult patients with mild TBI (GCS 13 - 15), without a history of inherited coagulopathy or anticoagulant therapy, platelet aggregation inhibitor therapy, intoxication or multiple associated injuries, who present with a single ICH with maximum diameter <5 mm regained full GCS within two hours. These patients may not require 24-hour observation.[43, level I]

Patients with MHI and ICH of ≤4 mm, normal neurological examination, no skull fracture, not on any warfarin (anticoagulant), aspirin or clopidogrel (antiplatelet) and not intoxicated, can be safety observed for six hours without neurosurgical consultation.[44, level III]

Patients with minor head injury (GCS 15), ≥65 years old, on low dose aspirin with systolic blood pressure <150 mmHg and presenting with initial negative primary head CT, should have a routine repeat head CT within 12 - 24 hours to accurately identify head injury or such patients should be subjected to a prolonged hospital observation for at least 48 hours.[45, level III]

Recommendation 7

- Patients with mild head injury in whom computed tomography scan is not indicated and with all the following criteria can be safely observed in emergency department* for a minimum of six hours:
  - Glasgow Coma Scale score 15 on arrival or two hours later
  - no neurological abnormality
  - age <65 years old
  - not on any anticoagulant or antiplatelet therapy
  - no history of coagulopathy
  - no multiple injuries
  - not intoxicated and not under influence of psychotropic drugs

*For hospital without observation ward, these patients may be admitted.

- Patients with MHI who are ≥65 years old and/or on anticoagulant/ antiplatelet should be admitted directly to ward irrespective of CT findings.

6.3.2 Parameters for observation in ED

It is well established that the risk of intracranial complications and subsequent need for surgery increases as GCS score declines. Extensive studies have supported their repeatability and validity.[12]

A prospective observational study comparing GCS score of patients done in the field, with the GCS score obtained by emergency physicians upon arrival in ED showed a moderate relationship (r=0.45, p=0.003). Assuming most GCS scores improve from field to ED, there is concordance between ECP and physicians in assessment of TBI patients using the GCS.[46, level III]

Variables that are highly accurate in predicting clinically significant outcomes of mild TBI patients include GCS (OR=11.74, 95% CI 8.42 to 16.37), neurological deficit (OR=1.90, 95% CI 1.33 to 2.71), clinical findings [such as amnesia, diffuse headache, vomiting and LOC (OR=4.31, 95% CI 2.81 to 6.61)], skull fracture (OR=31.01, 95% CI 20.36 to 47.21) and risk factors [such as coagulopathy, age >60 years old, previous neurosurgery, post-traumatic seizure (PTS) and alcohol/ drug abuse (OR=2.12, 95% CI 1.62 to 3.01)].[11, level III]

In patients with TBI, the significant risk factors for mortality are increasing age, lower GCS, higher ISS, lower mean arterial BP and abnormal RR (>25 and <10).[41, level III]

In patients with head injury admitted for observation, the minimum acceptable documented neurological observations are GCS, pupil size and reactivity, limb movements, RR, HR, BP, temperature and blood oxygen saturation.[20] Even though the evidence is based on patients admitted for observation in hospital, the same principles apply to patients observed in ED.

- Emergency care providers (ECP) should use a standard head injury proforma in their documentation when assessing and observing patients with head injury (refer to **Appendix 4** on **Head Chart**).
- All healthcare providers caring for patients with head injury should be capable of performing observations of the standard parameters effectively.
- The acquisition and maintenance of observation and recording skills require dedicated training and this should be available to all relevant staff.
- When using GCS, document and communicate each response (for example a patient scoring 13/15 based on scale of 4 on eye opening, 4 on verbal response and 5 on motor response should be communicated verbally as E4 opening eyes spontaneously, V4 confused, M5 localising pain) and written as E4V4M5.

Recommendation 8

- The following parameters should be used for observation of patients with head injury:
  a) Glasgow Coma Scale and Score
  b) Vital signs
     - Respiratory rate and oxygen saturation
     - Pulse rate
     - Blood pressure
     - Pain score
     - Temperature
  c) Neurological deficits
     - Pupil size and reactivity
     - Limb movement
     - Unusual behaviour, temperament or speech impairment
  d) Other symptoms such as persistent vomiting, seizure, amnesia and diffuse headache

Refer to **Appendix 4** on **Head Chart**.

6.3.3 Method, frequency and duration of observation/assessment in ED

There is insufficient evidence on method, frequency and duration of observation/assessment of head injury in ED. In management of head injury, pre-emptive investigation to detect lesions before they lead to further neurological deterioration has superseded the earlier deterioration dependent approach.[12]

i. Method

Safe assessment of TBI in ED requires consistent observation and re-evaluation as the injury is dynamic.[12]

- During handover of patient care, ECP should communicate with healthcare providers in ED on details of the mechanism and type of injury, and records of neurological progress since arrival in ED.
- Head chart should be used to monitor patient's progress. Any discrepancy in assessment should be discussed immediately with the senior medical officer or EP.

ii. Frequency

Frequency of observation should relate to risk of clinically important findings in order to detect early deterioration of head injury.

The risk of rapid deterioration due to intracranial complications is high during the first six hours and diminishes after that. Therefore, observations should be more frequent during this period.[20]

a. In-patients with GCS 15/15

Patients with head injury, who warrant admission, should have neurological observations carried out at least in the following frequency starting after initial assessment in the ED:[12]

- half hourly for two hours
- hourly for four hours
- two hourly for six hours
- four hourly thereafter until agreed to be no longer necessary

b. In-patients with GCS <15/15

Perform and record observation on a half-hourly basis until GCS 15 has been achieved. Thereafter, the minimum frequency of observation are as follows:[20]

- half-hourly for two hours
- hourly for next four hours
- 2-hourly thereafter
- should there be any deterioration at any time after the initial 2-hour period, observations should revert to half-hourly and follow the original frequency schedule

iii. Duration

Patients with MHI can be safely observed for six hours without neurosurgical consultation if they have the following features:[44, level III]

- ICH of ≤4 mm
- normal neurological examination
- no skull fracture
- not on any warfarin, aspirin or clopidogrel
- not intoxicated

Patient who have been provided with emergency care and having clinical and/or physiological conditions that are expected to deteriorate or resolve and require close observation may be admitted to ED observation ward. These patients should have a pre-defined care plan within stipulated time frame of **eight hours** or prerogative of EP in charge as clinically indicated.[47, level III]

Recommendation 9

- Patients with head injury should be monitored with findings recorded immediately using head chart.
  - Any deterioration should prompt immediate re-evaluation of the patients by the attending doctor.
- Observations may be performed and documented hourly in head injury patients. Should there be any deterioration at any time after the initial 2-hour period, the minimum frequency of observation respectively are as follows until GCS 15 is achieved:
  - half-hourly for first two hours
  - hourly for next four hours
  - 2-hourly for six hours
  - 4-hourly thereafter until no longer necessary and/or fit for discharge
- Patients with mild head injury in whom computed tomography scan is not indicated can be safely observed in emergency department for a minimum of six hours.

6.3.4 Admission after observation in ED

The primary reasons for in-hospital admission after observation in ED is for further management of patients who develop worsening post-concussion symptoms or presence of features indicating risk of further complications. Patients with persistent impaired consciousness or neurological impairment will need continued observation and in-patient care.[20]

Some clinical risk factors can be used as a guide to identify those who need neurosurgical intervention. In pre-injury warfarin and clopidogrel use, the risk factors for ICH are vomiting (OR=3.68, 95% CI 1.55 to 8.76) and abnormal mental status (OR=3.08, 95% CI 1.60 to 5.94).[48, level III] GCS (OR=11.74, 95% CI 8.42 to 16.37), neurological deficit (OR=1.90, 95% CI 1.33 to 2.71), clinical findings [such as amnesia, diffuse headache, vomiting and LOC (OR=4.31, 95% CI 2.81 to 6.61)], skull fracture (OR=31.01, 95% CI 20.36 to 47.21) and risk factors [such as coagulopathy, age >60 years old, previous neurosurgery, PTS and alcohol/drug abuse (OR=2.12, 95% CI 1.62 to 3.01)] was found to be highly accurate in predicting clinically significant outcomes of mild TBI patients.[11, level III]

CT can be used as a safe modality to triage patients for admission as it leads to similar clinical outcomes compared with observation in hospital.[42, level I]

The criteria below are used for admitting patients to hospital following a head injury:[12; 20]

- patients with new, clinically significant abnormalities on imaging
- patients whose GCS has not returned to 15 after imaging, regardless of the imaging results
- when a patient has indications for CT scan but this cannot be done within appropriate period, either because CT is not available or because the patient is not sufficiently cooperative to allow scanning
- continuing worrying signs (e.g. persistent vomiting, unresolved amnesia and headache) of concern to the clinician
- other sources of concern to the clinician (e.g. anticoagulant use, drug or alcohol intoxication, other injuries, shock, suspected non-accidental injury, meningism, cerebrospinal fluid leak)
- social problems or cannot be supervised by a responsible adult

Recommendation in this section is formulated based on extrapolation from other guidelines and tailored to local context.

Recommendation 10

- Patients with mild head injury who have been observed for six hours in emergency department should be admitted to ward if they have:
  - clinically significant abnormalities on head computed tomography (CT) imaging
  - Glasgow Coma Scale Score <15*
  - worrying signs (e.g. vomit ≥2 times, seizure, diffuse headache, amnesia, abnormal behaviour or neurological deficit)*
  - other body system injuries requiring admission
  - social problem** including no supervision by a responsible adult

* Patients should have a head CT before admission.

**Transport issue, no communication, stay in remote area, suspected abuse case or other factors affecting the monitoring and safety of patients.

6.3.5 Discharge home from ED without observation

It is neither feasible nor desirable to admit all MHI patients attending ED for observation especially those who have recovered and at low risk of intracranial complications. Furthermore, patients with MHI who present with a GCS of 15 to the ED rarely require urgent neurosurgical intervention or experience precipitous deterioration.[24, level II-2]

Patients with low risk MHI can be discharged home after assessment without observation. Low risk patients are those with:[11, level III; 49, level II-2]

- GCS of 15
- no clinical finding (amnesia, vomiting, diffuse headache and LOC)
- no neurological deficit
- no skull fracture
- no risk factors (coagulopathy, age <60 years, previous neurosurgery, pre-trauma epilepsy and/or alcohol and/or drug misuse)

- Criteria to be met by patients of head injury prior to discharge:
  - Presence of a willing responsible adult for at least 24-hour observation
  - Verbal and written discharge advice given to responsible care givers and discussed prior to discharge
  - Easy access to an emergency response system e.g. 999
  - Living within reasonable access to medical care.
  - Availability of home transport.

Adapted: Scottish Intercollegiate Guidelines Network. Early management of patients with a head injury. Edinburgh; SIGN; 2009

Recommendation 11

- Patient with low risk mild head injury* and who is living with a reliable care giver can be discharged home safely without observation from emergency department.

*Refer to preceding paragraph.

7. IMAGING

Management of moderate and severe head injury depends largely on imaging to detect intracranial lesions. Timely imaging expedites appropriate surgical and medical interventions. Exclusion or demonstration of intracranial injury can guide decision about extent and duration of observation in such cases.

7.1 Head CT

There are a few head CT rules available to guide the need for CT scan. Canadian CT Head Rule (CCTHR) is used to select patients with MHI (GCS 13 - 15) for CT head. CCTHR identifies five high risk factors (refer to yellow box below) which predict neurological intervention [sensitivity of 100% (95% CI 92 to 100%) and specificity of 68.7% (95% CI 62 to 70)].[50, level II-2]

CCTHR also identifies two medium-risk factors (refer to yellow box below) which predict clinically important brain injury [sensitivity 98.4% (95%CI 96 to 99%) and specificity 49.6% (95% CI 48 to 51%)].[50, level II-2]

However, CCTHR is not applicable in the presence of seizure, penetrating skull injury, focal neurological deficit and use of anticoagulant.[50, level II-2]

The New Orleans Criteria (NOC) derived a set of clinical findings to identify a group of minor head injury patients who need CT scan. Presence of any the following findings will predict positive head CT [sensitivity of 100% (95% CI 95 to 100%) and specificity of 25% (95% CI 22 to 28%)]:[51, level II-2]

- headache
- vomiting
- age >60 years
- drug or alcohol intoxication
- deficits in short-term memory
- physical evidence of trauma above the clavicles
- seizure

Both CCHTR and NOC had been externally validated in detecting all patients who require neurosurgical intervention in two studies. For CCTHR, the sensitivities remained the same but with variable specificities (37.2% and 58%). For NOC, the sensitivities were also high (96% and 100%) but specificities were low (5.3% and 26%).[52 - 53, level II -2]

Scandinavian Neurotrauma Committee has concluded that the presence of the following criteria warrants a head CT:[54]

- GCS ≤14
- LOC

- repeated (≥2) vomiting
- anticoagulant or coagulation disorders
- clinical signs of depressed or basal skull fracture
- PTS
- focal neurological deficits

There is no retrievable evidence on the timing of head CT. However, the Scottish International Guidelines Network (SIGN) guidelines list down factors for immediate (within one hour) and urgent (within eight hours) CT scan in head injury. The timing for CT scan in MHI for local context is based on expert opinion of CPG DG.

Canadian CT Head Rule

CT Head Rule is only applicable for patients with minor head injuries and any of the following:

A. High risk (for neurological intervention)
   - GCS score <15 at two hours after injury
   - Suspected open or depressed skull fracture
   - Any sign of basal skull fracture (haemotympanum, 'racoon' eyes, cerebrospinal fluid otorrhoea/rhinorrhoea, Battle's sign)
   - Vomiting ≥2 episodes
   - Age ≥65 years

B. Medium risk (for brain injury on CT)
   - Amnesia before impact >30 minutes
   - Dangerous mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height >3 feet or 5 stairs)

   Minor head injury is defined as witnessed loss of consciousness, definite amnesia or witnessed disorientation in a patient with a GCS score of 13 - 15.

Patients on anticoagulation or antiplatelet therapy have a positive head CTs of 29% despite having a GCS score of 15. LOC is a predictor of a positive CT result (p=0.008), suggesting that head CT should be strongly considered in these patients.[26, level III]

In polytrauma patients with TBI, priority for initial management including timing of head CT is complex. Primary cause of hypotension must be urgently sought and treated, e.g. patients with exsanguinating intra-abdominal injuries should be operated on and stabilised before obtaining head CT. Neurologic examination of patients with hypotension is unreliable. Closed head injury will not cause haemorrhagic shock. Hypotension may occur with concomitant spinal cord injury or when medullary failure supervenes in the terminal stages of ICH.[38, level III]

Recommendation 12
- Canadian Computed Tomography Head Rule (CCTHR) may be used to decide on the need of Computed Tomography (CT) of the head in mild head injury (MHI).
- Patients with MHI on anticoagulation and/or antiplatelet should be considered for head CT.
- Urgency* for head CT in head injury patients should be based on severity of the injury and risk factors.
- Patient's cardiopulmonary status should be stabilised before performing head CT.

*Refer to the preceding text.

7.2 Skull X-ray

The definitive imaging for head injury is head CT. The scan is indicated when there is anatomical and/or physiological evidence of head injury. In MHI group with GCS of 15, asymptomatic and no neurological deficit, external evidences of head injury especially skull fracture is an indication for head CT prior to a definitive management plan.

Skull fractures may occur in the cranial vault or skull base. They may be linear or comminuted, and open or closed. Open fractures are diagnosed when any of the following is present:[38, level III]
- obvious evidence of penetrating head injury (involving skull bone and structure beneath it)
- skull fracture visualised beneath scalp laceration wound
- clinical signs suggestive of skull base fracture

Closed fracture is diagnosed when skull depression is detected by scalp palpation with or without overlying scalp swelling or hematoma.

Skull fracture is an important finding to predict intracranial lesion following MHI which require a head CT.
- If skull fracture is present, the probability of an ICH is 4.9 times higher than those without skull fracture.[55, level III]
- Skull fracture, with or without clinical signs, in MHI is an independent risk marker of neurosurgically relevant intracranial lesion (RR=3.9, 95% CI 2.2 to 7.1).[56, level II-2]

Head CT is the investigation of choice to identify ICH following head injury. The plain skull x-ray is ineffective screening tool in predicting ICH in MHI with a mean sensitivity of 50.0% (ranges from 13.0% to 75.0%) and a mean specificity of 97.0% (ranges from 91.0% to 99.5%).[55, level III]

Skull fracture can only be ruled out by radiographic imaging such as skull x-ray and head CT. In the absence of clinical signs of skull fracture,

one cannot rule out the presence of skull fracture. This is because the sensitivities and specificities of clinical signs are low compared to radiographic imaging, as shown in the **Table 2** below.[56, level II-2]

Table 2. Sensitivity and specificity of clinical assessment on skull fracture

Clinical findings	Sensitivity	Specificity
Clinical assessment (both clinical signs of skull fracture and scalp wound)	51.6%	71.4%
Clinical signs of skull fracture alone with absence of scalp wound	36.1%	87.5%
Scalp wound alone	15.4%	50.0%

If CT scan is not immediately available and patient does not fulfil clinical criteria for CT scan of head, a skull x-ray can be performed. The patients should undergo CT scan of head if skull x-ray is positive.[56, level II-2]

- A normal skull x-ray does not exclude ICH and may miss a fracture.

Recommendation 13
- Skull x-ray may be considered in mild head injury patients who do not fulfil the criteria for head CT*.

*Refer to the **Chapter 7.1** on **Indications for CT**.

7.3 Repeat Head CT

Given the high cost and radiation risk associated with head CT, the practice of routine repeat CT scan of patients with head injury needs to be addressed.

In a recent meta-analysis of 16 cohort studies, intervention rates were higher in patients with MHI who had CT scan done based on neurological deterioration compared with those who had routine repeat CT regardless of neurological changes (2.7% vs 0.6%, $p<0.001$). The findings suggest that it is unnecessary to repeat a CT scan after MHI when neurological status remain unchanged or improves.[57, level II-2] Neurological deterioration is an independent predictor on the need for neurosurgical intervention in patients with TBI who are not on antiplatelet or anticoagulation therapy (OR=3.98, 95% CI 1.7 to 9.1).[58, level II-2]

Patients with MHI who are ≥65 years old and on low dose aspirin, with initial negative primary head CT, have secondary ICH accurately identified in routine repeat head CT within 12 hours to 24 hours ($p<0.00001$). For those who do not have a repeat scan, they should be subjected to prolonged in-hospital observation for at least 48 hours.[45, level III]

In another meta-analysis of low quality cohort studies, repeat CT in patients with TBI resulted in change of management for a minority of patients. Better designed studies are needed to address the value of repeat CT in the management of TBI.[59, level II-2]

- There is insufficient evidence to recommend routine repeat head CT in MHI.

7.4 Cervical Imaging

- All patients with head injury are presumed to have cervical spine injury until proven otherwise. Protection of the cervical spine in these patients is a priority.

Assessment for the need of cervical spine imaging in patients with head injury depends on their conscious level and ability to cooperate in clinical examination. Two independent decision rules [Canadian Cervical Spine Rule (CCR) and National Emergency X-radiography Utilization Study (NEXUS)] are available to assist such assessment. Refer to **Appendix 6** and **Appendix 7** on CCR and NEXUS criteria.

In a study comparing these two decision rules of cervical radiography, CCR was more sensitive (99.4% vs 90.7%, $p<0.001$) and more specific (45.1% vs 36.8%, $p<0.001$) than Nexus criteria for detection of cervical spine injury in alert and stable patients with trauma to the head and neck. The use of CCR when compared with Nexus criteria resulted in lower radiography rates (55.9% vs 66.6%, $p<0.001$).[60, level I]

Validation of the CCR, using CT scan as a gold standard, to identify cervical spine fracture demonstrated that CCR was very sensitive (100%) but had very low specificity (0.60%). The study evaluated 19 of the 20 clinical findings described in the CCR excluding 45° rotation of the neck and found eight predictors of cervical spine fracture which were:[61, level III]

- midline tenderness
- GCS <15
- age ≥65 year
- paraesthesia
- high speed motor vehicle collision (MVC) 100 km/hour

- rollover MVC
- MVC with ejection/ejection
- patients who were never in sitting position in ED

In another study, several factors were significantly associated with cervical spine injury (CSI) in patients with TBI. Patients in the older age group (>65 years old) have a higher odds ratio of CSI when compared to younger age patients in MVA but not in fall related injuries (OR=1.26, 95% CI 1.15 to 1.39). Skull/face fracture, other spine fracture/dislocation, upper limb injury, thorax injury, and hypotension were significantly associated with CSI (OR range from 32 to 3.34).[62, level III]

The incidence of CSI in patients with blunt polytrauma and reduced levels of consciousness (GCS <15) ranges between 5.2% and 13.9%.[63, level III] In a meta-analysis of seven studies, cervical CT was more sensitive (98%, 95% CI 96 to 99) than cervical spine plain radiography (52%, 95% CI 47 to 56) in detecting CSI.[64, level I] Thus, it is a good practice to include cervical spine when performing head CT.

Recommendation 14

- Canadian Cervical Spine Rule (CCR) or National Emergency X-radiography Utilization Study (NEXUS) criteria may be used as selection criteria for cervical radiograph in alert and stable head injury patients.
- Head Computed Tomography (CT) for patient with a Glasgow Coma Scale <15 should have cervical spine CT at the same setting.

Refer to **Algorithm 4** on **Selection of Adults with Head Injury for Imaging of the Cervical Spine**.

8. MEDICATION FOR INITIAL MANAGEMENT

Agitation, pain and seizure may potentially contribute to elevation in intracranial pressure (ICP), BP and body temperature. Analgesia, sedative and anticonvulsant are administered to treat these conditions.[65]

Diuretic may be used to control raised ICP. Intravenous (IV) fluid is administered to restore and maintain the systemic and cerebral perfusion.

8.1 Analgesia and Sedative

Ideal properties of analgesia and sedative in neuro-trauma patients:

- quick onset and offset
- predictable clearance independent of end-organ function
- easily titrated to effect
- reduce ICP by cerebral blood volume reduction or cerebral vasoconstriction
- maintain cerebral autoregulation
- permit normal cerebral vascular reactivity to changes in $PaCO_2$
- minimal cardiovascular depressant effects
- inexpensive

- Analgesia and sedative are commonly administered in adults with head injury for one or more of the following indications:
  - to induce anxiolysis
  - to control agitation/restlessness
  - to control pain
  - to facilitate mechanical ventilation
  - to improve ICP and cerebral perfusion pressure (CPP)

A systematic review showed that etomidate and propofol improved ICP and CPP in adults with severe TBI. Boluses or short infusions of opioids such as fentanyl and morphine resulted in clinically and statistically significant increases in ICP and decreases in MAP and CPP. However, there was no strong evidence to support that one agent was more efficacious than another for improvement of favourable neurological outcome (defined as a GOS score of 4 to 5) and mortality rate.[66, level I] In a meta-analysis on severe TBI, there was no difference between the use of propofol and midazolam in term of neurological outcome (OR=1.139, 95% CI 0.397 to 3.273) and mortality (OR=0.758, 95% CI 0.237 to 2.424).[67, level I]

A Cochrane systematic review revealed no significance in mortality and severe disability between the use of barbiturate and control group. However, barbiturate therapy resulted in increased occurrence of

hypotension (OR=1.8, 95% CI 1.19 to 2.7) which offset any ICP lowering effect on CPP and lowered the mean body temperature.[68, level I] This is supported by a large, multicentre cohort study which showed high dose barbiturate decreased ICP but also caused hemodynamic instability leading to increased use of high doses of vasopressors in severe TBI.[69, level III]

In a meta-analysis of 19 RCTs, naloxone was safe to be used in severe TBI in terms of mortality, ICP and prognosis.[70, level I]

There is no recent evidence retrieved on the use of analgesia/sedation or their reversal agents in mild to moderate head injury. However, CPG DG is of the opinion that this issue should be addressed based on expert opinion.

For MHI, oral analgesic such as paracetamol, non-steroidal anti-inflammatory drugs and tramadol can be used provided there is no definite contraindication.

8.2 Intravenous Fluids

In patients with head injury, resuscitation fluids are fundamental components of the restoration and maintenance of the systemic and cerebral perfusion.

A systematic review found no evidence to support the use of hyperosmolar crystalloid or colloid solutions over isotonic crystalloids for pre-hospital fluid resuscitation of patients with TBI.[22, level I] In a RCT, fluid resuscitation with albumin was associated with higher mortality rates and fewer favourable neurologic outcomes when compared with saline in severe TBI patients in ICU.[23, level I]

A systematic review found only one RCT showing no significant differences in mortality and morbidity between pre-hospital and delayed fluid resuscitation (mean transfer time to hospital of 19 minutes).[22, level I]

8.3 Diuretic

Mannitol is widely used in the control of raised ICP following brain injury. However, there is uncertainty over the optimal treatment regimen, the effectiveness of mannitol as compared with other ICP lowering agents and the usefulness of mannitol given at other stages following head injury, for example in the pre-hospital setting prior to volume resuscitation.

A Cochrane systematic review showed that mannitol therapy for raised ICP had no significant beneficial or detrimental effect on mortality when compared with pentobarbital or hypertonic saline. ICP-directed

treatment had small beneficial effect compared to treatment directed by neurological signs and physiological indicators.[71, level I]

In view of insufficient evidence on the use of IV fluid regime for resuscitation in PHC and the important of the issue to be addressed, the CPG DG formulates the recommendations based on their expert opinion.

8.4 Anticonvulsant

PTS is defined as a recurrent seizure disorder due to TBI. PTS can be divided into three groups: immediate seizure (occurring within 24 hours of injury), early seizure (occurring within seven days of injury) and late seizure (occurring more than seven days after injury). The peak incidence of early PTS occurs within the initial 48 hours of the causative head injury.[72, level III]

The incidence of early and late PTS without anticonvulsant prophylaxis has been estimated from 4% to 25% and 9% to 42% respectively.[65] Effective prophylaxis of early PTS reduces brain metabolic demands, therefore reducing intracranial pressure and neurotransmitter release. This can prevent secondary brain injury.[73, level III]

The risk factors for PTS include:[74, level I]

- GCS score <10/15
- cortical contusions
- depressed skull fractures
- early intracranial haematoma
- wounds with dural penetration
- prolonged length of coma (>24 hours)
- prolonged length of post-traumatic amnesia (>24 hours)
- damage in the region adjacent to the temporal sulcus

In a systematic review, IV prophylactic phenytoin administered within eight hours of injury (ideally in the first one hour) was efficacious in reducing the incidence of early PTS in moderate to severe head injury.[74, level I]

There is no clinically significant advantage between levetiracetam and phenytoin with regards to seizure control, adverse drug reaction, complication and mortality.[75, level II-2]

Recommendation 15

- Analgesia and sedative should be used in severe head injury patients who are intubated and ventilated.
- In mild to moderate head injury:
  - analgesia should be used to control pain
  - short-acting sedative agent may be offered in titrated dose to control agitation/restlessness
- Naloxone may be used as opioid reversal in head injury.
- Isotonic crystalloid is the preferred choice of intravenous fluid in head injury.
- Mannitol, hypertonic saline or frusemide may be used to reduce intracranial pressure in head injury after consultation with a specialist.
  - Diuretics should not be used in hypotensive patients.
- Phenytoin should be given as prophylaxis against early post-traumatic seizure in head injury with risk factors*.

*Refer to preceding paragraph.

Refer to **Appendix 11** on **Drug Dosing Regimen for Initial Management of Head Injury**.

9. SPECIAL CONSIDERATION

9.1 Reversal of Antiplatelet and Anticoagulant in Patients with Head Injury

Patients with pre-injury use of antiplatelet agents (e.g. clopidogrel and aspirin) are at an increased risk for mortality with blunt traumatic ICH.[76, level III] It is prudent to achieve rapid control of haemorrhage as the haemorrhagic expansion in traumatic ICH occurs within the first 24 hours after injury[77, level III] and is an independent predictor of mortality.[78, level III]

The transfusion of platelet in patients with mild TBI does not significantly increase cardiac and pulmonary events, or mortality compared with patients without transfusion. It is also not significantly associated with progression of injury based on imaging. Combined transfusion of platelet, fresh frozen plasma and/or factor VII is associated with clinical deterioration, specifically cardiac and respiratory events (OR=5.8, 95% CI 1.2 to 28.2).[79, level II-3]

A systematic review of five articles on the utility of platelet transfusion in adults with pre-injury antiplatelet and traumatic ICH showed inadequate evidence to support the routine use of platelet transfusion in the ED.[80, level II-2]

- There is insufficient evidence to recommend routine transfusion of platelet in patients with MHI and on antiplatelet therapy.

Anticoagulant-associated ICH has a high risk of bleeding expansion, disability or death. Pre-injury use of warfarin is a significant predictor of mortality in patients with traumatic ICH. The important aspect of management of anticoagulant-associated ICH is urgent reversal of coagulopathy with rapid decrease of the International Normalized Ratio (INR) to a value of <1.4, preferably to <1.2. Interventions aimed at preventing the haematoma expansion are paramount as larger haematomas are associated with poorer functional outcomes.[78, level III]

International consensus-based guidelines emphasise immediate discontinuation of anticoagulant and reversal of anticoagulation in ICH by administration IV vitamin K, fresh frozen plasma (FFP), prothrombin complex concentrate (PCC) or recombinant factor VIIa as indicated. However there is no specific target INR for adequate reversal in ICH patients on warfarin.[81, level III]

In a review of treatments for reversing warfarin anticoagulation in patients with acute ICH, PCC was statistically faster than FFP in correcting INR. Recombinant factor VIIa rapidly reversed the effect of

warfarin on INR with a reported 5% thromboembolic risk. Vitamin K has slow onset of action. It carries a risk of allergic reaction. Hence it should be given slowly to all patients with warfarin-associated ICH. Despite the multiple treatment options available, the review did not demonstrate an improved patient survival with any particular treatment option.[82, level III]

A study was conducted on 13 coagulopathic patients with traumatic ICH and treated with recombinant factor VIIa. It demonstrated rapid correction of INR to a threshold of 1.3 prevented expansion of bleeding and facilitated urgent surgical intervention.[83, level II-3]

Recommendations 16

- Anticoagulants should be immediately stopped and reversed in patients with intracranial bleeding by using vitamin K (only for warfarin), fresh frozen plasma, prothrombin complex concentrate or recombinant factor VIIa as indicated and there should be consultation on this matter among relevant specialties.

9.2 Restarting Antiplatelet/Anticoagulant in Patients with Head Injury

Reinitiating warfarin therapy in a patient with recent warfarin associated intracranial haemorrhage (WAICH) is a challenging decision for the physician. The risks include the reoccurrence of ICH once warfarin is restarted versus the risk of thromboembolism without warfarin therapy. Careful control of anticoagulation level decreases the risk of ICH.[84, level III]

A long-term clinical study indicated that recurrent WAICH is uncommon when anticoagulant therapy is resumed. The end point of thromboembolic events and WAICH is not statistically significant between those restarted and not restarted on warfarin (p=0.62). Clinical decision to resume anticoagulant therapy after WAICH should include patient's general medical condition, risk of falls and risk factors for systemic haemorrhage. Patients without these risks may benefit from restarting warfarin therapy. Failure to resume therapy may subject the patient to thromboembolic complications.[85, level III]

A systematic review concluded that it may be advisable to reinitiate anticoagulation earlier with the timing and intensity modified based on predictors of thromboembolic and haemorrhagic complications. The predictors for haemorrhagic complications are:[86, level II-2]

- younger patients (p<0.0001)
- traumatic causes (p=0.002)
- subdural haemorrhage (p=0.049),
- patients anticoagulated for cardiac indications (p=0.0047)

- failure to reverse anticoagulation (OR=3.633, 95% CI 1.431 to 9.226)

Predictors for thromboembolic complications are:[86, level II-2]

- younger patients (p=0.048)
- spinal haemorrhage, multiple haemorrhage and non-traumatic causes (p=0.046)
- anticoagulation started at lower intensity (p=0.0001)

- There is insufficient evidence on when to restart antiplatelet and anticoagulant in patients with head injury.
- If there is an urgent need for anticoagulation but risk of bleeding is still considered reasonably high, decision on when and how to start anticoagulant should be decided by a multidisciplinary team.

9.3 Patient with Mild Head Injury Requiring Non-Life Threatening other than Cranial Surgery

Head injury may co-exist with other injuries such as compound femur fracture that may need urgent surgery. Any delay may lead to morbidity and mortality.[87, level III]

Reported benefits of early long-bone stabilisation in polytrauma patients include increased patient mobilisation by eliminating the need for traction and reducing pulmonary morbidity, hospital care costs, mortality, hospital length of stay, intensive care unit and ventilator days.[88, level II-2]

The concerns for patients with head injury include:

- possibility of secondary brain injury
- timing for performing non-urgent surgery
- interruption in GCS monitoring for patients while undergone anaesthesia
- MHI with no indication for a CT scan but require surgery

As the types of non-urgent surgery is limitless and choices aplenty, the CPG DG has decided to use compound fracture of the femur as a surrogate indicator. It is probably the most major among non-life-threatening or limb-threatening surgeries. Urgent intervention for a compound injury is to prevent osteomyelitis.[87, level III]

There is no retrievable evidence on indications for CT scan in patients with MHI who do not fulfil the CCHTR criteria but require urgent surgery. The decision on the need of CT scan should be made by a multidisciplinary team. The period of greatest risk for delayed ICH

(causing significant morbidity and mortality) following trauma is the first six hours.[12]

- Delayed ICH should be considered in patients who do not regain full consciousness or develop new neurological deficit post-anaesthesia.

Early fixation of long bone fracture under general anaesthesia (GA) in patients with head injury is an acceptable practice. In multisystem trauma patients with TBI and chest injury, early timing of orthopaedic femur fracture fixation under GA is not associated with worse outcome than those without femur fracture and no surgery.[89, level II-2] Orthopaedic and facial fracture fixation under GA (<24 hours) is not associated with worse neuropsychological or functional outcome than late surgery. As there is no evidence that early stabilisation has any detrimental effect, it is preferable to perform early long-bone stabilisation in polytrauma patients.[89, level II-2]

Recommendation 17
- Early extracranial surgery in head injury patients can be performed safely under general anaesthesia.

Refer to **Algorithm 5** on **Adults with Mild Head Injury Requiring Urgent Surgery Other Than Cranial Surgery**.

9.4 Safe Extubation of Patients with Head Injury

Patients may have to be pre-emptively intubated for certain situations (e.g. intoxicated, restlessness and for radiological or diagnostic procedures). However, there may be a need to extubate these patients after that. Extubation of these patients demands consideration of airway patency, respiratory parameters, neurological status and associated injury. Points to be considered include timing and place of extubation, and post-extubation care.

There is limited evidence on the above issues. An observational study showed that it was safe to extubate patients in ED provided that certain rules and criteria were followed as the following:[90, level III]
- pre-extubation
  - resolution of the clinical issue requiring intubation
  - fraction of inspired oxygen (FiO_2) ≤40%
  - positive end-expiratory pressure (PEEP) ≤5 cm H_2O
  - SpO_2 >95%, SBP >100 mmHg, HR <130 bpm
  - patient not known to be a case of difficult intubation
- post-extubation
  - patient should receive close monitoring for at least 60 minutes

A person trained in airway and ventilator management preferably an anaesthesiologist or EP should carry out the extubation.

- For patients who have been intubated for certain situations (eg. intoxicated, restlessness and for radiological and diagnostic procedures), the following points are to be considered prior to extubation:
  - Equipments for intubation with a person trained in airway management should be readily available.
  - At least 60 minutes post-extubation close observation (GCS, SpO_2, BP, PR, RR and sign of hypoxia or airway compromise) is required.
  - When in doubt, extubation should not be attempted.
  - Patients are preferably placed in High Dependency Unit (HDU) or Critical Care Area.

*Refer to **Appendix 10** on **Safe Extubation Criteria in Head Injury**.

A RCT showed that GCS score ≥8 and partial pressure arterial oxygen/fraction of inspired oxygen ratio (PaO_2/FiO_2) >200 were associated with increased odds of successful extubation.[91, level I]

10. TELECONSULTATION

Observation in a general hospital with telemedicine facilities does not put patients with mild to moderate head injury and positive CT scan at a significant risk provided there are:[92, level II-2]

- careful initial assessment
- continuous observation
- easy access to neurosurgical services in the event of progression

Teleconsultation avoids unnecessary transfer of one third of patients with head injury (34%). It changes the treatment at the referring hospital on the advice of the neurosurgeon (42%).[93, level II-3]

A local study showed that teleradiology significantly reduced the number of inter-hospital transfer by 37%. However 20.1% of patients who were not transferred based on clinical data alone would have to be transferred when the clinical data and images were reviewed. On multiple logistic regression analysis, MHI was twice as likely to be transferred if both clinical data and images were reviewed compared with clinical data alone.[94, level III]

iPad2 is equally efficacious as LCD monitor in interpretation of radiologic images during teleconsultation (AUC of 0.900 and 0.935 respectively, p=0.183).[95, level II-3]

Video consultation (VC) may be a safe mode of consultation in emergency neurosurgery compared with teleradiology (TR) and telephone consultation (TC). Although the time taken for VC is significantly longer, VC and TR have unequivocally better diagnostic accuracy than TC alone. VC is more likely to result in logistical problems, as reflected by the unacceptably high failure rate of 30.1%.[96, level I]

Recommendation 18

- Teleconsultation should be used in the management of head injury if available.

- Patient's confidentiality must always be upheld at all times when utilising teleconsultation.

11. DISCHARGE ADVICE

It is important to give careful verbal and written discharge advice to patients with head injury and their care givers. This will help them to identify alarming features that need immediate medical attention and those who need post-concussion rehabilitation.[97, level III; 98, level I] The discharge form should include phone contact number of the hospital in the event of a emergency or related queries.[99, level III]

A standardised head injury discharge advice is beneficial provided that the staff are trained and well versed with it.[97, level III] Factors that may influence the comprehension of the advice are age group, years of schooling, literacy level and simplicity of the discharge form.[100, level I] Therefore a discharge form should be standardised and comprehensible at all levels. Patient with minor TBI may be discharged from ED provided there is a responsible care giver. Carers should understand the given discharge advice.[101, level III]

- All patients or their care givers should be given verbal and written discharge advice. This must be documented.
  - They should be asked to repeat the advice to ensure that they understand the content upon discharge.
- Where possible, head injury discharge form should be available in multiple local languages.

Discharge instructions to patients and care giver should highlight the following alarming features:[99, level III]

- GCS <15 (unable to open eyes spontaneously, disorientated or unable to obey command)
- vomiting
- headache
- amnesia
- seizure
- neurological deficit

Recommendation 19

- Head injury discharge advice form should be comprehensible. It should include instructions to recognise alarming features and phone contact number of local healthcare facilities*.

*Refer to **Appendix 5** on **Discharge Advice for Head Injury**.

12. FOLLOW-UP

Patients with minor head injury can be followed up by telephone conversation within 48 hours on their ability to recall alarming features and well-being.[101, level III] Early rehabilitation significantly aids patients with MHI who continue to experience difficulties with everyday activities.[102, level I]

Patients with moderate or severe head injury should be discharged with routine follow-up. Rehabilitation programme improves patients' condition by reducing social disabilities and post-concussion symptoms.[98, level I]

Recommendation 20
- Moderate to severe head injury should have scheduled clinic follow-up.
- Mild head injury may have follow-up via clinic visit or telephone call within 48 hours.

13. IMPLEMENTING THE GUIDELINES

The management of head injury should be guided by evidence-based approach in order to provide quality care to the patients. Several factors may affect the implementation of recommendations in the CPG.

13.1 Facilitating and Limiting Factors

Existing facilitators for implementations of the recommendations in the CPG include:

1. wide dissemination of the CPG (soft- and hard-copies) to healthcare providers
2. regular update on HI management at conferences and scientific meeting locally

Existing barriers for implementations of the recommendations of the CPG are:

1. evolving understanding of the illness and its treatment
2. insufficient resources for integrated care at different level of service delivery
3. variation in treatment practice and preferences
4. no national registry for HI for further planning of services

13.2 Potential Resource Implications

To implement the CPG, there must be strong commitment to:

1. ensure widespread distribution of the CPG to healthcare providers via printed and electronic copies
2. reinforce regular training of healthcare providers, with adequate funding
3. ensure trained multidisciplinary team is available at different levels of healthcare
4. ensure widespread distribution of updated patient education materials

13.3 Clinical Audit Indicator

The following is proposed as clinical audit indicator for quality management:

$$\text{Percentage of patients with isolated mild head injury initially treated as inpatient and died} = \frac{\text{Number of patients with isolated mild head injury initially treated as inpatient and died during admission within one year period}}{\text{Number of patients with isolated mild head injury initially treated as inpatient in the same period}} \times 100\%$$

Inclusion criteria: All MHI who are treated at all healthcare facilities

Implementation strategies will be developed following the approval of the CPG by MoH which include Quick Reference and Training Module.

REFERENCES

1. Jamaluddin SF, Abd Wahab M , Abdul Wahab MY , et al. National Trauma Database January 2009 To December 2009– Fourth Report; Malaysia: 2011.
2. Health Informatics Centre. Health Facts 2015; Ministry of Health, Malaysia, 2015.
3. Technical Committee of Malaysian Registry of Intensive Care. 6th Report of Malaysian Registry of Intensive Care 2008; Ministry of Health, Malaysia; May 2009.
4. World Health Organization. Global status report on road safety 2013: supporting a decade of action. WHO: Geneva; 2013.
5. Medical Development Division. Country Health Plan 10th Malaysia Plan 2011-2015; Ministry of Health, Malaysia.
6. Sethi D, Aljunid S, SB S, et al. Comparison of the effectiveness of trauma services provided by secondary and tertiary hospitals in Malaysia. Ann Emerg Med. 2007 49(1):52-61.
7. Medical Development Division. Specialty and subspecialty Framework of Ministry of Health hospitals 10 MP (2010-2015); Ministry of Health, Malaysia; Dec 2011.
8. Department of Veterans Affairs (VA) and The Department of Defense (DoD). VA/DoD Clinical Practice Guideline for Management of Concussion/Mild Traumatic Brain Injury (mTBI); Washington: 2009.
9. Jennett B, Teasdale G. Aspects of coma after severe head injury. Lancet. 1977;23;1(8017):878-959.
10. Carney N, Ghajar J, Jagoda A, et al. Concussion guidelines step 1: systematic review of prevalent indicators. Neurosurgery. 2014;75 Suppl 1:S3-15.
11. Fabbri A, Servadei F, Marchesini G, et al. Prospective validation of a proposal for diagnosis and management of patients attending the emergency department for mild head injury. J Neurol Neurosurg Psychiatry. 2004;75(3):410-416.
12. Early management of patients with a head injury. Scottish Intercollegiate Guidelines Network. SIGN. Edinburgh:2009.
13. Timmons SD, Bee T, Webb S, et al. Using the abbreviated injury severity and Glasgow Coma Scale scores to predict 2-week mortality after traumatic brain injury. J Trauma. 2011;71(5):1172-1178.
14. Shahin H, Gopinath SP, Robertson CS. Influence of Alcohol on Early Glasgow Coma Scale in Head-Injured Patients. The Journal of Trauma: Injury, Infection, and Critical Care. 2010;69(5):1176-1181.
15. Badjatia N, Carney N, Crocco TJ, et al. Brain Trauma Foundation; BTF Center for Guidelines Management. Guidelines for prehospital management of traumatic brain injury 2nd edition. Prehosp Emerg Care. 2008;12 Suppl 1:S1-52.
16. von Elm E, Schoettker P, Henzi I, et al. Pre-hospital tracheal intubation in patients with traumatic brain injury: systematic review of current evidence. Br J Anaesth. 2009;103(3):371-386.
17. Ahn H, Singh J, Nathens A, et al. Pre-hospital care management of a potential spinal cord injured patient: a systematic review of the literature and evidence-based guidelines. J Neurotrauma. 2011;28(8):1341-1361.
18. Austin N, Krishnamoorthy V, Dagal A, et al. Airway management in cervical spine injury. . International Journal of Critical Illness and Injury Science. 2014;4(1):50-56.
19. Association of Anesthetists of Great Britain and Ireland. Recommendations for the Safe Transfer of Patients with Brain Injury. London: AAGBI; 2006.
20. The National Institute for Health and Care Excellence. Triage, assessment, investigation and early management of head injury in children, young people and adults. UK: NICE; 2014.
21. Chowdhury T, Kowalski S, Arabi Y, et al. Pre-hospital and initial management of head injury patients: An update. Saudi J Anaesth. 2014;8(1):114-120.
22. Tan PG, Cincotta M, Clavisi O, et al. Review article: Prehospital fluid management in traumatic brain injury. Emerg Med Australas. 2011;23(6):665-676.
23. SAFE Study Investigators; Australian and New Zealand Intensive Care Society Clinical Trials Group; Australian Red Cross Blood Service; George Institute for International Health, Myburgh J, Cooper DJ, Finfer S, et al. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. . N Engl J Med. 2007;357(9):874-884.

24. Clement CM, Stiell IG, Schull MJ, et al. Clinical features of head injury patients presenting with a Glasgow Coma Scale score of 15 and who require neurosurgical intervention. Ann Emerg Med. 2006;48(3):245-251.
25. Raj R, Siironen J, Kivisaari R, et al. Factors correlating with delayed trauma center admission following traumatic brain injury. Scand J Trauma Resusc Emerg Med. 2013;21:67.
26. Brewer ES, Reznikov B, Liberman RF, et al. Incidence and predictors of intracranial hemorrhage after minor head trauma in patients taking anticoagulant and antiplatelet medication. J Trauma. 2011;70(1):E1-5.
27. Joseph B, Pandit V, Meyer D, et al. The significance of platelet count in traumatic brain injury patients on antiplatelet therapy. J Trauma Acute Care Surg. 2014;77(3):417-421.
28. Peck KA, Calvo RY, Schechter MS, et al. The impact of preinjury anticoagulants and prescription antiplatelet agents on outcomes in older patients with traumatic brain injury. J Trauma Acute Care Surg. 2014;76(2):431-436.
29. Ley EJ, Srour MK, Clond MA, et al. Diabetic patients with traumatic brain injury: insulin deficiency is associated with increased mortality. J Trauma. 2011;70(5):1141-1144.
30. Barmparas G, Liou DZ, Lamb AW, et al. Prehospital hypertension is predictive of traumatic brain injury and is associated with higher mortality. J Trauma Acute Care Surg. 2014;77(4):592-598.
31. Tohme S, Delhumeau C, Zuercher M, et al. Prehospital risk factors of mortality and impaired consciousness after severe traumatic brain injury: an epidemiological study. Scand J Trauma Resusc Emerg Med. 2014;7:22:21.
32. Ayson M. Best-evidence Review of Transfer Protocols for Moderate to Severe Traumatic Brain Injury. ACC: 2011.
33. Ryynänen O-P, Iirola T, Reitala J, et al. Is advanced life support better than basic life support in prehospital care? A systematic review. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine. 2010;18(1):62.
34. Pearson WS, Ovalle F, Faul M, et al. A Review of Traumatic Brain Injury Trauma Center Visits Meeting Physiologic Criteria from the American College of Surgeons Committee on Trauma/ Centers for Disease Control and Prevention Field Triage Guidelines. Prehospital Emergency Care. 2012;16(3):323-328.
35. Lerner EB, Roberts J, Guse CE, et al. Does EMS Perceived Anatomic Injury Predict Trauma Center Need? Prehospital Emergency Care. 2013;17(3):312-316.
36. Hsiao KY, Hsiao CT, Weng HH, et al. Factors predicting mortality in victims of blunt trauma brain injury in emergency department settings. Emergency Medicine Journal. 2008;25(10):670-673.
37. Nakahara S, Matsuoka T, Ueno M, et al. Predictive Factors for Undertriage Among Severe Blunt Trauma Patients: What Enables Them to Slip Through an Established Trauma Triage Protocol? The Journal of Trauma: Injury, Infection, and Critical Care. 2010;68(5):1044-1051.
38. American College of Surgeon Committee on Trauma. Advanced Trauma Life Support Student Course Manual (9th Edition). Chicago: 2012.
39. Eastridge BJ, Salinas J, McManus JG, et al. Hypotension Begins at 110 mm Hg: Redefining Hypotension With Data. The Journal of Trauma: Injury, Infection, and Critical Care. 2007;63(2):291-299.
40. Nishijima DK, Shahlaie K, Echeverri A, et al. A clinical decision rule to predict adult patients with traumatic intracranial haemorrhage who do not require intensive care unit admission. Injury. 2012;43(11):1827-1832.
41. Saadat S, Akbari H, Khorramirouz R, et al. Determinants of mortality in patients with traumatic brain injury. Turkish Journal of Trauma and Emergency Surgery. 2012;18(3):219-224.
42. af Geijerstam JL, Oredsson S, M B. OCTOPUS Study Investigators. Medical outcome after immediate computed tomography or admission for observation in patients with mild head injury: randomised controlled trial. BMJ. 2006;333(7566):465.
43. Schaller B, Evangelopoulos DS, Muller C, et al. Do we really need 24-h observation for patients with minimal brain injury and small intracranial bleeding? The Bernese Trauma Unit Protocol. Emergency Medicine Journal. 2010;27(7):537-539.
44. Joseph B, Friese RS, Sadoun M, et al. The BIG (brain injury guidelines) project. Journal of Trauma and Acute Care Surgery. 2014;76(4):965-969.

45. Tauber M, Koller H, Moroder P, et al. Secondary Intracranial Hemorrhage After Mild Head Injury in Patients With Low-Dose Acetylsalicylate Acid Prophylaxis. The Journal of Trauma: Injury, Infection, and Critical Care. 2009;67(3):521-525.
46. Bazarian JJ, Eirich MA, SD S. The relationship between pre-hospital and emergency department Glasgow coma scale scores. Brain Inj. 2003 17(7):553-560.
47. Medical Development Division. Emergency Medicine and Trauma Services Policy. Putrajaya: MoH, Malaysia; 2012.
48. Nishijima DK, Offerman SR, Ballard DW, et al. Risk of Traumatic Intracranial Hemorrhage In Patients With Head Injury and Preinjury Warfarin or Clopidogrel Use. Academic Emergency Medicine. 2013;20(2):140-145.
49. Fabbri A, Servadei F, Marchesini G, et al. Which type of observation for patients with high-risk mild head injury and negative computed tomography? Eur J Emerg Med. 2004;11(2):65-69.
50. Stiell IG, Wells GA, Vandemheen K, et al. The Canadian CT Head Rule for patients with minor head injury. Lancet. 2001 357(9266):1391-1396.
51. Haydel MJ, Preston CA, Mills TJ, et al. Indications for computed tomography in patients with minor head injury. N Engl J Med. 2000 343(2):100-105.
52. Bouida W, Marghli S, Souissi S, et al. Prediction value of the Canadian CT head rule and the New Orleans criteria for positive head CT scan and acute neurosurgical procedures in minor head trauma: a multicenter external validation study. Ann Emerg Med. 2013;61(5):521-527.
53. Smits M, Dippel DW, de Haan GG, et al. External validation of the Canadian CT Head Rule and the New Orleans Criteria for CT scanning in patients with minor head injury. JAMA. 2005;294(12):1519-1525.
54. Undén J, Ingebrigtsen T, Romner B, et al. Scandinavian Neurotrauma Committee (SNC). Scandinavian guidelines for initial management of minimal, mild and moderate head injuries in adults: an evidence and consensus-based update. BMC Med. 2013;11:50.
55. Hofman PA, Nelemans P, Kemerink GJ, et al. Value of radiological diagnosis of skull fracture in the management of mild head injury: meta-analysis. J Neurol Neurosurg Psychiatry. 2000 68(4):416-422.
56. Muñoz-Sánchez MA, Murillo-Cabezas F, Cayuela-Domínguez A, et al. Skull fracture, with or without clinical signs, in mTBI is an independent risk marker for neurosurgically relevant intracranial lesion: a cohort study. Brain Inj. 2009;23(1):39-44.
57. Almenawer SA, Bogza I, Yarascavitch B, et al. The value of scheduled repeat cranial computed tomography after mild head injury: single-center series and meta-analysis. Neurosurgery. 72. 2013;1(56-62):discussion 63-64.
58. Joseph B, Aziz H, Pandit V, et al. A three-year prospective study of repeat head computed tomography in patients with traumatic brain injury. J Am Coll Surg. 2014;219(1):45-51.
59. Reljic T, Mahony H, Djulbegovic B, et al. Value of repeat head computed tomography after traumatic brain injury: systematic review and meta-analysis. J Neurotrauma. 2014 31(1):78-98.
60. Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med. 2003 349(26):2510-2518.
61. Duane TM, Wilson SP, Mayglothling J, et al. Canadian Cervical Spine rule compared with computed tomography: a prospective analysis. J Trauma. 2011;71(2):352-355.
62. Fujii T, Faul M, Sasser S, et al. Risk factors for cervical spine injury among patients with traumatic brain injury. J Emerg Trauma Shock. 2013;6(4):252-258.
63. Thomas M, Teece S. Towards evidence based emergency medicine: best BETs from Manchester Royal Infirmary. Computed tomography and the exclusion of upper cervical spine injury in trauma patients with altered mental state. Emerg Med J. 2002;19(6):551-552.
64. Holmes JF, Akkinepalli R. Computed tomography versus plain radiography to screen for cervical spine injury: a meta-analysis. . J Trauma. 2005;58:902-905.
65. Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons, et al. Guidelines for the management of severe traumatic brain injury. II. Hyperosmolar therapy. J Neurotrauma. 2007;24 Suppl 1:S14-20. Erratum in: J Neurotrauma. 2008 Mar;25(3):276-8.
66. Roberts DJ, Hall RI, Kramer AH, et al. Sedation for critically ill adults with severe traumatic brain injury: a systematic review of randomized controlled trials. Crit Care Med. 2011;39(12):2743-2751.

67. Gu JW, Yang T, Kuang YQ, et al. Comparison of the safety and efficacy of propofol with midazolam for sedation of patients with severe traumatic brain injury: a meta-analysis. J Crit Care. 2014;29(2):287-290.
68. Roberts I, Sydenham E. Barbiturates for acute traumatic brain injury. Cochrane Database Syst Rev. 2012;12:12 CD000033.
69. Majdan M, Mauritz W, Wilbacher I, et al. Barbiturates use and its effects in patients with severe traumatic brain injury in five European countries. J Neurotrauma. 2013;30(1):23-29.
70. Zafar SN, Khan AA, Ghauri AA, et al. Phenytoin versus Leviteracetam for seizure prophylaxis after brain injury - a meta analysis. BMC Neurol. 2012;12:30.
71. Wakai A, Roberts I, Schierhout G, et al. Mannitol for acute traumatic brain injury. Cochrane Database Syst Rev. 2007;24(1):CD001049.
72. Grisar T, Bottin P, de Borchgrave d'Alténa V, et al. Prophylaxis of the epilepsies: should anti-epileptic drugs be used for preventing seizures after acute brain injury? Acta Neurol Belg. 2005;105(1):5-13.
73. Practice parameter: antiepileptic drug treatment of posttraumatic seizures. Brain Injury Special Interest Group of the American Academy of Physical Medicine and Rehabilitation. Arch Phys Med Rehabil. 1998 May;79(5):594-7.
74. Khan AA, Banerjee A. The role of prophylactic anticonvulsants in moderate to severe head injury. Int J Emerg Med. 2010;22;3(3):187-191.
75. Inaba K, Menaker J, Branco BC, et al. A prospective multicenter comparison of levetiracetam versus phenytoin for early posttraumatic seizure prophylaxis. J Trauma Acute Care Surg. 2013;74(3):766-771.
76. Wong DK, Lurie F, Wong LL. The effects of clopidogrel on elderly traumatic brain injured patients. J Trauma. 2008;65(6):1303-1308.
77. Narayan RK, Maas AI, Servadei F, et al. Traumatic Intracerebral Hemorrhage Study Group. Progression of traumatic intracerebral hemorrhage: a prospective observational study. J Neurotrauma. 2008;25(6):629-639.
78. Davis SM, Broderick J, Hennerici M, et al. Recombinant Activated Factor VII Intracerebral Hemorrhage Trial Investigators. Hematoma growth is a determinant of mortality and poor outcome after intracerebral hemorrhage. Neurology. 2006;66(8):1175-1181
79. Washington CW, Schuerer DJ, Grubb RL Jr. Platelet transfusion: an unnecessary risk for mild traumatic brain injury patients on antiplatelet therapy. J Trauma. 2011;71(2):358-363.
80. Nishijima DK, Zehtabchi S, Berrong J, et al. Utility of platelet transfusion in adult patients with traumatic intracranial hemorrhage and preinjury antiplatelet use: a systematic review. J Trauma Acute Care Surg. 2012;72(6):1658-1663.
81. Ansell J, Hirsh J, Hylek E, et al. American College of Chest Physicians. Pharmacology and management of the vitamin K antagonists: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest. 2008;133(6 Suppl):160S-198S.
82. Bechtel BF, Nunez TC, Lyon JA, et al. Treatments for reversing warfarin anticoagulation in patients with acute intracranial hemorrhage: a structured literature review. Int J Emerg Med. 2011;4(1):40.
83. Bartal C, Freedman J, Bowman K, et al. Coagulopathic patients with traumatic intracranial bleeding: defining the role of recombinant factor VIIa. J Trauma. 2007;63(4):725-732.
84. Broderick J, Connolly S, Feldmann E, et al. American Heart Association; American Stroke Association Stroke Council; High Blood Pressure Research Council; Quality of Care and Outcomes in Research Interdisciplinary Working Group. Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: a guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. 38. 2007;6(2001-23).
85. Claassen DO, Kazemi N, Zubkov AY, et al. Restarting anticoagulation therapy after warfarin-associated intracerebral hemorrhage. Arch Neurol. 2008;65(10):1313-1318.
86. Hawryluk GW, Austin JW, Furlan JC, et al. Management of anticoagulation following central nervous system hemorrhage in patients with high thromboembolic risk. J Thromb Haemost. 2010;8(7):1500-1508.

87. Nau T, Aldrian S, Koenig F, et al. Fixation of femoral fractures in multiple-injury patients with combined chest and head injuries. ANZ J Surg. 2003;73(12):1018-1021.
88. Dunham CM, Bosse MJ, Clancy TV, et al. EAST Practice Management Guidelines Work Group. Practice management guidelines for the optimal timing of long-bone fracture stabilization in polytrauma patients: the EAST Practice Management Guidelines Work Group. J Trauma. 2001;50(5):958-967.
89. Wang MC, Temkin NR, Deyo RA, et al. Timing of surgery after multisystem injury with traumatic brain injury: effect on neuropsychological and functional outcome. J Trauma. 2007;62(5):1250-1258.
90. Weingart SD, Menaker J, Truong H, et al. Trauma patients can be safely extubated in the emergency department. J Emerg Med. 2011;40(2):235-239.
91. Namen AM, Ely EW, Tatter SB, et al. Predictors of successful extubation in neurosurgical patients. Am J Respir Crit Care Med. 2001;163(3Pt 1):658-664.
92. Fabbri A, Servadei F, Marchesini G, et al. Observational approach to subjects with mild-to-moderate head injury and initial non-neurosurgical lesions. J Neurol Neurosurg Psychiatry. 2008;79(10):1180-1185.
93. Stormo A, Sollid S, Størmer J, et al. Neurosurgical teleconsultations in northern Norway. J Telemed Telecare. 2004;10(3):135-139.
94. Hassan R, Siregar JA, NA. ARM. The implementation of teleneurosurgery in the management of referrals to a neurosurgical department in Hospital Sultanah Amninah Johor Bahru. Malays J Med Sci. 2014;21(2):54-62.
95. Park JB, Choi HJ, Lee JH, et al. An assessment of the iPad 2 as a CT teleradiology tool using brain CT with subtle intracranial hemorrhage under conventional illumination. J Digit Imaging. 2013 26(4):683-690.
96. Wong HT, Poon WS, Jacobs P, et al. The comparative impact of video consultation on emergency neurosurgical referrals. Neurosurgery. 2006 59(3):607-613.
97. Kerr J, Swann IJ, Pentland B. A survey of information given to head-injured patients on direct discharge from emergency departments in Scotland. Emerg Med J. 2007;24(5):330-332.
98. Wade DT, King NS, Wenden FJ, et al. Routine follow up after head injury: a second randomised controlled trial. J Neurol Neurosurg Psychiatry. 1998;65(2):177-183.
99. Fung M, Willer B, Moreland D, et al. A proposal for an evidenced-based emergency department discharge form for mild traumatic brain injury. Brain Inj. 2006;20(9):889-894.
100. Yates K, Pena A. Comprehension of discharge information for minor head injury: a randomised controlled trial in New Zealand. N Z Med J. 2006;119(1239):U2101.
101. Heng KW, Tham KY, How KY, et al. Recall of discharge advice given to patients with minor head injury presenting to a Singapore emergency department. Singapore Med J. 2007 48(12):1107-1110.
102. Wade DT, Crawford S, Wenden FJ, et al. Does routine follow up after head injury help? A randomised controlled trial. J Neurol Neurosurg Psychiatry. 1997;62(5):478-484.

APPENDIX 1

EXAMPLE OF SEARCH STRATEGY

The following MeSH terms or free text terms were used either singly or in combination, search was limit to English, human and last 10 years:-

1. Craniocerebral Trauma/
2. (traum* adj1 (craniocerebral or cranial or frontal region or frontal or parietal region or parietal or temporal region or temporal or occipital region or occipital or forehead or head or skull or posterior fossa)).tw.
3. (traum* adj1 (occipital or parietal or frontal)).tw.
4. (head injur* adj1 (multiple or superficial or minor or open or closed)).tw.
5. (injur* adj1 (cranial or head or craniocerebral or multiple head or superficial head or minor head or open head or skull)).tw.
6. Skull Fractures/
7. (skull adj1 fracture*).tw.
8. (skull fracture* adj1 (linear or non depressed or non-depressed)).tw.
9. (fracture* adj1 (non-depressed skull or non depressed skull linear skull or compound skull)).tw.
10. Brain Injuries/
11. (injur* adj1 (traum* brain or mild traum* brain or brain traum* mild or diffuse brain or focal brain or acute brain or brain)).tw.
12. (brain injur* adj1 (traum* or acute or focal or diffuse)).tw.
13. (encephalopath* adj1 (post concussive or post-concussive or traum* or post-traum* or post traum*)).tw.
14. (brain adj1 (traum* or laceration* or contusion*)).tw.
15. (cortical adj1 contusion*).tw.
16. traum* brain injury.tw.
17. tbi*.tw.
18. Diffuse Axonal Injury/
19. (injur* adj1 diffuse axonal).tw.
20. diffuse axonal injury.tw.
21. axonal injur* diffuse.tw.
22. Post-Concussion Syndrome/
23. ((post concuss* or post-concuss*) adj1 (syndrome* or symptom*)).tw.
24. Brain Concussion/
25. (concussion* adj1 (cerebral or brain or severe or mild or moderate or intermediate)).tw.
26. Skull Fracture, Depressed/
27. (compound depressed adj1 skull fracture*).tw.
28. (skull fracture* adj1 depressed).tw.
29. Multiple Trauma/
30. ((injur* or wound* or traum*) adj1 multiple).tw.
31. polytrauma*.tw.
32. Contrecoup Injury/
33. (injur* adj1 (contre-coup or contre coup or contrecoup)).tw.
34. Brain Hemorrhage, Traumatic/
35. (h?emorrhage* adj1 (traumatic cerebellar or traum* brain)).tw.
36. (traum* adj1 (cerebellar h?emorrhage* or brain h?emorrhage*)).tw.
37. Brain Stem Hemorrhage, Traumatic/
38. (traum* adj1 (brainstem h?em* or brain stem h?em* or bulbar h?em* or medullary h?em* or pontine h?em* or midbrain h?em* or h?em* brain stem or h?em* brainstem)).tw.
39. (h?em* adj1 (traumatic medullary or traumatic bulbar or post-traumatic brainstem)).tw.
40. Cerebral Hemorrhage, Traumatic/
41. (traum* adj1 (cerebral h?em* or intracerebral h?em* or cerebral parenchymal h?em* or brain h?em* cerebral or cerebral intraparenchymal h?em*)).tw.
42. (h?em* traum* adj1 (intracerebral or cerebral)).tw.
43. Epilepsy, Post-Traumatic/
44. (seizure* adj1 (disorder* post traum* or disorder* post-traum* or early post-traum* or early post traum* or impact)).tw.
45. ((post-traum* seizure* or post traum* seizure*) adj1 (disorder* or early)).tw.
46. (epileps* adj1 (post-traum* or post traum* or traum*)).tw.
47. (concussive adj1 convulsion*).tw.
48. Pneumocephalus/
49. (pneumocephalus adj1 (traum* or tension or pressure)).tw.
50. Cerebrospinal Fluid Otorrhea/
51. ((otorrh?ea or rhinorrh?ea*) adj1 (post-traum* cerebrospinal fluid or post traum* cerebrospinal fluid or cerebrospinal fluid traum* or traum* cerebrospinal fluid or cerebrospinal fluid post- traum*)).tw.
52. (cerebrospinal fluid adj1 (post-traum* otorrh?ea or post traum* otorrh?ea or otorrh?ea traum* or traum* otorrh?ea or otorrh?ea post-traum* or otorrh?ea post traum* or post-traum* rhinorrh?ea* or post traum* rhinorrh?ea* or rhinorrh?ea* traum* or traum* rhinorrh?ea* or rhinorrh?ea* post-traum* or rhinorrh?ea* post traum*)).tw.
53. Cerebrospinal Fluid Rhinorrhea/
54. Coma, Post-Head Injury/
55. (coma* adj1 (post-traum* or post traum* or post-head injury or post head injury or post-concussive or post concussive)).tw.
56. Head Injuries, Closed/
57. ((head injur* or head traum*) adj1 (closed or nonpenetrating or blunt or penetrating)).tw.
58. ((traum* or injur*) adj closed head).tw.
59. OCULOMOTOR NERVE INJURIES/
60. ((traum* or injur*) adj1 (third-nerve or third nerve or third-nerve palsy or third nerve palsy or oculomotor nerve or oculomotor neuropath* or third cranial nerve or cranial nerve iii or second-nerve or second nerve or second-nerve palsy or second nerve palsy or optic nerve or optic neuropath* or second cranial nerve or cranial nerve ii)).tw.
61. ((oculomotor or optic) adj (nerve traum* or nerve injur*)).tw.
62. Optic Nerve Injuries/
63. HEAD INJURIES, PENETRATING/
64. (penetrating adj1 (craniocerebral traum* or brain traum* or brain injur* or cranial traum* or head traum* or head injur*)).tw.
65. ((traum* or injur*) adj1 (penetrating head or penetrating cranial or penetrating craniocerebral)).tw.
66. Intracranial Hemorrhage, Traumatic/
67. (intracranial h?em* adj traum*).tw.
68. (traum* intracranial adj1 h?em*).tw.
69. Brain Hemorrhage, Traumatic/
70. ((traum* cerebellar or traum* brain) adj1 h?em*).tw.
71. (traum* adj1 (brain h?em* or cerebellar h?em*)).tw.
72. HEMATOMA, EPIDURAL, CRANIAL/
73. ((cranial or intracranial) adj1 (extradural h?em* or epidural h?em*)).tw.
74. HEMATOMA, SUBDURAL/
75. (subdural adj1 h?em*).tw.
76. (traum* adj1 subdural h?em*).tw.
77. HEMATOMA, SUBDURAL, ACUTE/
78. (subdural adj1 h?em* acute).tw.
79. (h?em* adj1 (acute subdural or subdural acute)).tw.
80. HEMATOMA, SUBDURAL, INTRACRANIAL/
81. (subdural h?em* adj2 (intracranial or traum*)).tw.
82. (subdural adj1 h?em* intracranial).tw.
83. (h?em* adj1 (subdural intracranial or subdural cranial or intracranial subdural)).tw.
84. Skull Fracture, Basilar/
85. ((frontobasilar or basilar) adj1 skull fracture*).tw.
86. (fracture* adj1 basilar skull).tw.
87. (battle* adj1 sign).tw.
88. Subarachnoid Hemorrhage, Traumatic/
89. ((traum* or post-traum* or post traum*) adj1 subarachnoid h?em*).tw.
90. (h?em* adj (traum* or post-traum* or post traum*)).tw.

91. or/1-90
92. (prehospital adj1 emergency care).tw.
93. ((pre-hospital or primary care) adj (assess* or evaluat*)).tw.
94. Abbreviated Injury Scale/
95. (abbreviated adj1 injur* scale*).tw.
96. Injury Severity Score/
97. iss score*.tw.
98. injur* severity score*.tw.
99. score* injury severity.tw.
100. GLASGOW COMA (coma adj1 scale Glasgow).
101. coma scale Glasgow.tw.
102. Glasgow Coma Scale.tw.
103. 92 or 93 or 94 or 95 or 96 or 97 or 98 or 99 or 100 or 101 or 102
104. 91 and 103

91. or/1-90
92. tomography, x-ray computed/ or tomography, spiral computed/ or multidetector computed tomography/
93. (helical adj1 ct*).tw.
94. ((compute*-assisted or compute* assisted or compute*) adj1 (tomography spiral or tomography helical)).tw.
95. (spiral adj1 (c?t scan* or compute* tomography or ct*)).tw.
96. (scan* adj1 spiral c?t*).tw.
97. (tomography adj1 (spiral compute* or spiral compute*-assisted or helical computed)).tw.
98. ((ct scan* or cat scan* or ct) adj1 (x-ray* or x ray* or cine)).tw.
99. ((scan* x-ray* or scan x ray*) adj (ct or cat)).tw.

91. or/1-90
92. (anticoagulant adj1 agent*).tw.
93. anticoagulant*.tw.
94. (thrombin inhibitor adj1 indirect).tw.
95. Inhibitors indirect thrombin.tw.
96. PLATELET AGGREGATION INHIBITORS/
97. (antiplatelet adj1 drug*).tw.
98. (platelet adj1 antogonist*).tw.
99. Blood platelet adj1 antagonist*).tw.
100. antiplatelet agent*.tw.
101. Anticoagulants/
102. (platelet adj1 inhibitor*).tw.
103. (blood platelet adj1 antiaggregrant*).tw
104. (platelet adj1 antiaggregant*).tw.
105. blood platelet aggregation inhibitor*.tw.
106. platelet aggregation inhibitors.tw.

100. (tomodensitometry.tw.
101. (tomograph* adj1 (x-ray* compute* or x ray* compute* or xray* compute* or electron beam or compute* x-ray* or compute* x ray* or compute* xray* or transmission compute*)).tw.
102. ((compute* tomograp* or tomograp* compute*) adj1 (x-ray* or x ray* or xray* or transmission)).tw.
103. ((x-ray* compute* or xray* compute* or x ray* compute* or electron beam compute*) adj1 (assisted tomograph* or tomograph* or axial tomograph*)).tw.
104. tomograph* x ray* or tomograph* xray* or tomograph* x-ray*) adj1 (compute* assisted or compute* axial)).tw.
105. tomography multisection computed.tw.
106. tomography multidetector-row computed.tw.
107. ((multidetector row or multidetector-row or multidetector or multislice or multisection) adj1 computed tomography).tw.
108. tomography multislice computed.tw.
109. tomography multidetector computed.tw.
110. 92 or 93 or 94 or 95 or 96 or 97 or 98 or 99 or 100 or 101 or 102 or 103 or 104 or 105 or 106 or 107 or 108 or 109
111. 91 and 110
107. Aspirin/
108. (acid adj1 acetylsalicylic).tw.
109. aspirin.tw.
110. Ticlopidine/
111. ticlopidine.tw.
112. (ticlopidine adj1 hydrochloride).tw
113. ticlid.tw.
114. 92 or 93 or 94 or 95 or 96 or 97 or 98 or 99 or 100 or 101 or 102 or 103 or 104 or 105 or 106 or 107 or 108 or 109 or 110 or 111 or 112 or 113
115. 91 and 114

APPENDIX 2

CLINICAL QUESTIONS

1. What is the definition of head injury?
2. What are the differential diagnoses in suspected head injury?
3. What are the classifications of severity in head injury?
4. What is the safe and effective pre-hospital/primary care assessment in a patient with head injury by healthcare providers (e.g. GCS)?
5. What is the safe and effective pre-hospital care/primary care in a patient with head injury?
6. What are the criteria for discharge or referral of a patient with head injury at primary care?
7. What are the safe and effective measures to prevent secondary brain injury during transfer of a patient with head injury to hospital?
8. What are the triage criteria for a patient with head injury in ED?
9. What is the safe and effective initial management of a patient with mild/moderate/severe head injury in ED?
10. What are the criteria for observation of a patient with head injury in ED?
11. What are the safe and effective method, frequency and duration of observation/assessment in a patient with mild head injury in ED?
12. What are the criteria for admission of a patient with head injury (post-observation at ED)?
13. What are the criteria for discharge of a patient with head injury from ED without observation?
14. What are the indications for urgent/immediate head CT in head injury?
15. What are the criteria for skull x-ray in head injury if CT is not available?
16. What are the criteria for a repeat head CT in a patient with head injury who does not require surgical intervention?
17. What are the indications for cervical imaging in head injury?
18. Is anaesthesia for non-urgent and non-life threatening extracranial surgery safe for a patient with mild head injury?
19. What is the safe and effective method of reversing or restarting antiplatelet or anticoagulant in a patient with head injury on the medications?
20. What are the criteria for safe extubation in a patient with head injury?
21. What is the safe and effective analgesia in a patient with head injury?
22. What is the safe and effective sedation and reversal of sedation in a patient with head injury?
23. What are the criteria to start anticonvulsant in a patient with head injury?
24. What are the safe and effective IV fluids (e.g. hypertonic saline)/ diuretic in a patient with head injury?
25. Is tele-consultation safe and effective in management of head injury?
26. What discharge advice should be provided to a patient with head injury or his/her care givers?
27. When and how should a patient with head injury be followed-up?

APPENDIX 3

GLASGOW COMA SCALE AND SCORE

The GCS is based on a 15-point scale used for estimating and categorising the severity of brain injury following a TBI. The scale measures the best motor, verbal and eye opening response.

Component	Response	Score
Eye Opening Response	Spontaneous	4
	To voice/sound	3
	To pressure/pain	2
	None	1
Verbal Response	Orientated	5
	Confused	4
	Words	3
	Sounds	2
	None	1
Motor Response	Obeys commands	6
	Localising	5
	Flexion withdrawal	4
	Abnormal flexion	3
	Extension	2
	None	1

Glasgow Coma Score

The score is the cumulative sum of the scale in each component. The levels of head injury severity are classified as:

Mild	GCS 13 - 15
Moderate	GCS 9 - 12
Severe	GCS 3 - 8

APPENDIX 4

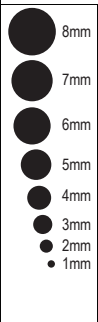
HEAD CHART

Name:_____ Age:_____ Sex: Male / Female

IC:_____ RN:_____ Ward: _____

Date																	
Time																	
STATUS		Temp(°c)	Pulse														
	TEMP(+)	41	180														
		40	160														
	Pulse (Blue)	39	140														
		38	120														
		37	100														
	BP (Red)	36	80														
		35	60														
		34	40														
	Pain Score																
	Respiratory Rate																
	Oxygen (L/ min or %)																
	Oxygen saturation %																
	Pupils	Right	Size														
			Reaction														
		Left	Size														
			Reaction														
BEST COMA SCALE	(E) >1 YR	SPONTANEOUS															
		TO VOICE/SOUND															
		TO PRESSURE/PAIN															
		NONE															
	(V) >1 YR	ORIENTATED															
		CONFUSED															
		WORDS															
		SOUNDS															
		NONE															
	(M) >2 YR	OBEYS COMMANDS															
		LOCALISING															
		FLEXION WITHDRAWAL															
		ABNORMAL FLEXION															
		EXTENSION															
		NONE															
TOTAL GCS																	
LIMBS MOVEMENT	ARMS	NORMAL POWER															
		MILD WEAKNESS															
		SEVERE WEAKNESS															
		SPASTIC FLEXION															
		NO RESPONSE															
	LEGS	NORMAL POWER															
		MILD WEAKNESS															
		SEVERE WEAKNESS															
		SPASTIC FLEXION															
		NO RESPONSE															

PUPILS SCALE



(E) EYE CLOSED DUE TO SWELLING (C)

(V) ENDOTRACHEAL TUBE OR TRACHEOSTOMY (T)

(M) USUALLY RECEIVED BEST ARM RESPONSE

(E) <1 YR	SPONTANEOUS	
	TO VOICE/SOUND	
	TO PRESSURE/PAIN	
	NONE	
(V) <1 YR	SMILE, COOS APPROPRIATELY	
	CONSOLABLE CRIES	
	INCONSOLABLE CRIES	
	GRUNTS, AGITATED, RESTLESS	
	NONE	
(M) <2 YR	NORMAL SPONTANEOUS MOVEMENTS	
	LOCALISING	
	NORMAL FLEXION	
	ABNORMAL FLEXION	
	EXTENSION	
	NONE	

RECORD RIGHT (R) AND LEFT (L) SEPARATELY IF THERE IS A DIFFERENCE BETWEEN THE TWO SIDES

CLINICAL EVENTS:

vomit ≥2x	
seizure	
diffuse headache	
amnesia	
abnormal behaviour	

APPENDIX 5

DISCHARGE ADVICE FOR HEAD INJURY

Patient's name:	Contact no.:
Care giver's name:	Date:
IC/RN:	

If any of the following alarming symptoms are present, you or your care giver should immediately contact the hospital for advice.

If within the next 24 hours you experience/as observed by care giver:

- Fainting or sleepiness 
- Increasing confusion, inability to recognise time, place or people 
- Change in behaviour 
- Constant headache which is worsening 
- Vomiting 
- Inability to remember new events 
- Jerking or seizures, abnormal speech 
- Blood or fluid coming out of the ear 
- Inability to move any part of your body 

What you should and should not do

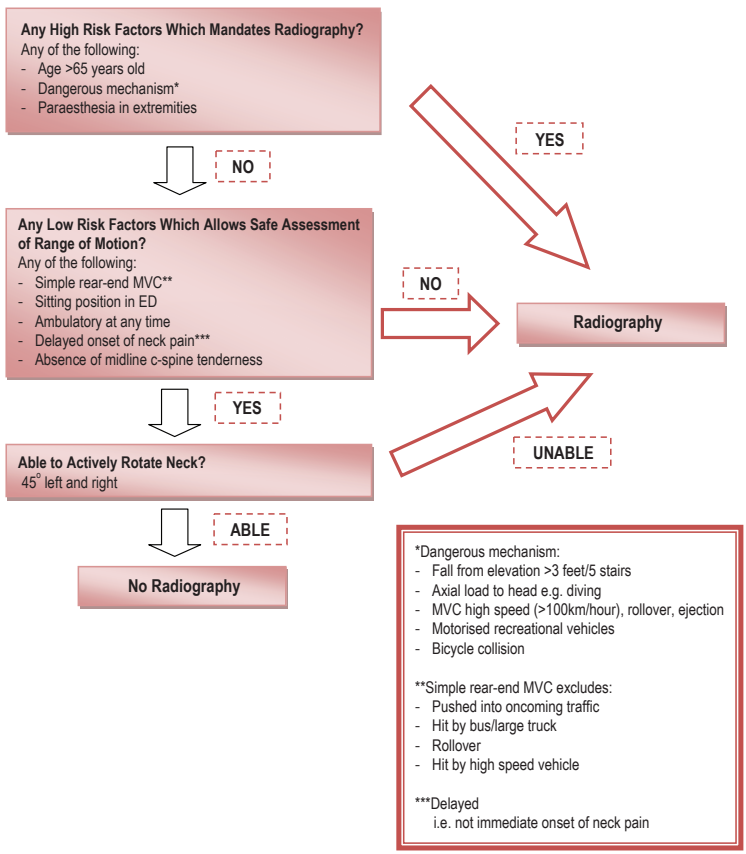
DO	DON'T (until advised by your doctor)
✓ Take paracetamol for headache	× Take sleeping pills and anticoagulants/ antiplatelets
✓ Take your usual medications	× Take alcohol
✓ Mild exercise when you feel better	× Play contact sport
✓ Take rest or a few days off from work	× Drive

Name of Doctor:
Emergency Contact:
Witness:

APPENDIX 6

CANADIAN CERVICAL RULE (CCR)

- For alert (GCS=15) and stable trauma patients where cervical spine injury is concern



Source: Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med. 2003 349(26):2510-2518

APPENDIX 7

THE NEXUS LOW-RISK CRITERIA FOR LOW PROBABILITY OF CERVICAL INJURY

Cervical-spine radiography is indicated for patients with trauma unless they meet all of the following criteria:

- No posterior midline cervical-spine tenderness[a]
- No evidence of intoxication[b]
- A normal level of alertness[c]
- No focal neurologic deficit[d]
- No painful distracting injuries[e]

Criteria are from Hoffman and colleagues.

a. Midline posterior bony cervical-spine tenderness is present if the patient reports pain on palpation of the posterior midline neck from the nuchal ridge to the prominence of the first thoracic vertebra, or if the patient evinces pain with direct palpation of any cervical spinous process.

b. Patients should be considered intoxicated if they have either of the following: a recent history provided by the patient or an observer of intoxication or intoxicating ingestion, or evidence of intoxication on physical examination such as an odour of alcohol, slurred speech, ataxia, dysmetria, or other cerebellar findings, or any behaviour consistent with intoxication. Patients may also be considered to be intoxicated if tests of bodily secretions are positive for alcohol or drugs that affect the level of alertness.

c. An altered level of alertness can include any of the following: a GCS score of 14 or less; disorientation to person, place, time, or events; an inability to remember three objects at five minutes; a delayed or inappropriate response to external stimuli; or other findings.

d. A focal neurologic deficit is any focal neurologic finding on motor or sensory examination.

e. No precise definition of a painful distracting injury is possible. This category includes any condition thought by the clinician to be producing pain sufficient to distract the patient from a second (neck) injury. Such injuries may include, but are not limited to, any long-bone fracture; a visceral injury requiring surgical consultation; a large laceration, degloving injury, or crush injury; large burns; or any other injury causing acute functional impairment. Physicians may also classify any injury as distracting if it is thought to have the potential to impair the patient's ability to appreciate other injuries.

Source: Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med. 2003 349(26):2510-2518.

APPENDIX 8

EQUIPMENTS FOR TRANSFER

A. Essential equipments:
- portable mechanical ventilator
- adequate supply of O_2
- portable battery powered multifunction monitor to include:
  - ECG
  - non-invasive BP set
  - pulse oximetry
  - temperature (optional)
  - invasive pressure monitoring (optional)
  - capnography (optional)

B. Other equipments:
- suction
- battery powered syringe pumps
- battery powered IV volumetric pumps (infusion by gravity is unreliable during transfer)
- intubation equipment
- self-inflating bag, valve and mask
- venous access equipment
- defibrillator/AED
- spare batteries
- blanket

C. An adequate supply of essential drugs to go with the patient:
- sedative agents (e.g. midazolam)
- muscle relaxants (e.g. suxamethonium) may be required for re-intubation
- analgesics (e.g. fentanyl)
- anticonvulsants (e.g. diazepam)
- vasoactive drugs (e.g. ephedrine, dopamine, noradrenaline)
- atropine
- sodium bicarbonate
- intravenous fluids (e.g. 0.9%NaCl)
- ±mannitol 20%, frusemide

D. Communication equipments

Adapted: Best-evidence Review of Transfer Protocols for Moderate to Severe Traumatic Brain Injury. (Available at http://www.acc.co.nz/PRD_EXT_CSMP/groups/external_providers/documents/reports_results/wpc111560.pdf)

APPENDIX 9

TRANSFER CHECKLIST FOR NEUROSURGICAL PATIENTS

	Checklist	YES	NO
Respiration	SpO_2 >90%? Airway clear? Airway protected adequately? (where applicable) Intubation and ventilation required? (where applicable)		
Circulation	BP MAP >80 mmHg? (adults) Pulse <100/min? (adults) Continuous cardiac monitoring? Peripheral perfusion adequate? Two reliable large bore IV cannulae in situ? Estimated blood loss already replaced? Arterial line? (optional) Central venous access? (optional)		
Head injury	Admission GCS recorded? GCS before transfer recorded? Pupillary size and reactivity recorded? Focal signs recorded? Seizures controlled? Raised ICP appropriately managed?		
Others	Cervical spine injury (cervical spine protection), chest injury, fractured ribs, pneumothorax addressed? Intra-thoracic, intra-abdominal bleed addressed? Pelvic fracture addressed? Long bone injuries splinted? Continuous bladder drainage? Nasogastric tube? (where applicable) Blood available? (where applicable) Chest tube function? (where applicable)		
Escort	Appropriate personnel escorting adequately experienced and trained? Instructed about this case? Adequate equipment and drugs? Sufficient oxygen supplies? (should be additional one hour or twice the estimated journey time) Referral letter attached? CT scans/X-rays films/CD attached? Where to go in the neurosurgical unit? Telephone numbers programmed into mobile phone? Electronic and communication equipment battery fully function? Name and contact number of receiving doctor?		

The development of standardised transfer documentation is encouraged according to the local setting.

Adapted: Best-evidence Review of Transfer Protocols for Moderate to Severe Traumatic Brain Injury. (Available at http://www.acc.co.nz/PRD_EXT_CSMP/groups/external_providers/documents/reports_results/wpc111560.pdf)

APPENDIX 10

SAFE EXTUBATION CRITERIA IN HEAD INJURY

If the patients are to be safely extubated in ED, the following criteria should be met:

No.	Criteria	YES	NO
1.	Resolution of the process necessitating intubation		
	The original airway or breathing problem has resolved		
2.	Able to oxygenate and ventilate on low ventilator settings		
	A. Options for assessing ability to oxygenate and ventilate would include oxygen saturation >92% or a normal arterial blood gas B. A common assessment of respiratory strength would be negative inspiratory force (NIF) <-30cm H_2O (normal NIF values would be -90 to -120 cm H_2O)		
	C. Typical low ventilator settings would include the following: • Pressure support ≤10 mmHg • PEEP ≤5 cm H_2O • Tidal volumes ≥5 mL/kg • FiO_2 ≤0.40%		
3.	Able to maintain a patent airway		
	A. Anatomically easy airway for laryngoscopy and intubation		
	B. Patient follows commands (opens eyes, grasps hand, lifts head off bed)		
	C. A cuff leak is present: with the cuff of the endotracheal tube deflated, a leak >25% should be present (exhaled volume less than inhaled volume due to leak)		
4.	Anticipated clinical course does not require mechanical ventilation		
	A. Good cough with acceptable secretions		
	B. Haemodynamically stable, with no vasopressors		
	• SpO_2 >92% on FiO_2 <0.4		
	• HR <100 beats/min		
	• RR <30 breaths/min		
	• SBP >90 mmHg		
	• No active cardiac ischemia		
	C. No other mitigating factor		
	• Cervical spine injury or instability		
	• Acute lung injury		
	• Acute pharyngeal injury		
	• Plan for imminent operation or transport		
	• Attending physician feels uncomfortable with extubation		

Preferably patients should be placed in HDU or Critical Care Area.

Source: Gray SH, Ross JA, Green RS. How to safely extubate a patient in the emergency department: a user's guide to critical care. CJEM. 2013 Sep;15(5):303-6.

APPENDIX 11

DRUG DOSING REGIMEN FOR INITIAL MANAGEMENT OF HEAD INJURY

A. Analgesia/Sedation

Drug	Dose	Adverse Reaction (>10%)
Benzodiazepines Midazolam	**Sedation:** IV infusion 0.05 - 0.1 mg/kg over at least 2 - 5 minutes	**Respiratory**: Decreased respiratory rate (23%) Apnoea (13%)
Opioids		
Fentanyl	**Analgesia**: 0.5 - 2 µg/kg/h continuous infusion	**Cardiovascular**: bradycardia, oedema **Central Nervous (CN)**: CN depression, confusion, dizziness, drowsiness, fatigue, headache, sedation **Endocrine and metabolic**: dehydration **Gastrointestinal (GI)**: Constipation, nausea, vomiting, xerostomia **Local**: application site reaction **Neuromuscular and skeletal**: chest wall rigidity (high dose IV), muscle rigidity, weakness **Ocular**: miosis **Respiratory**: dyspnoea, respiratory depression **Miscellaneous**: diaphoresis
Morphine sulphate	**Analgesia :** 0.05 - 0.1 mg/kg/hr continuous infusion	**Cardiovascular**: bradycardia, hypotension **CN**: drowsiness (9 - 48%), dizziness (6 -20%) **GI**: xerostomia (78%), constipation (9 -40%), nausea (7 - 28%)

B. Anticonvulsant

Drug	Dose	Adverse reaction
Phenytoin	**Loading dose:** 15 - 20 mg/kg IV infusion over 30 - 60 minutes **Rate:** should not be more than 50 mg/minute (to avoid hypotension) **Maintenance dose:** IV 100 mg given three times daily	**IV effects**: hypotension, bradycardia, cardiac arrhythmia, cardiovascular collapse (especially with rapid IV use),venous irritation and pain **Concentration related effects**: Nystagmus, blurred vision, diplopia, ataxia, slurred speech, dizziness, drowsiness, lethargy, coma, rash, fever, nausea, vomiting, confusion, mood changes

	Oral 300 mg ON for a total of seven days	>20 µg/mL: far lateral nystagmus >30 µg/mL: 45° lateral gaze nystagmus and ataxia >40 µg/mL: decreased mentation >100 µg/mL: death
Levetiracetam	**Loading dose**: 20 mg/kg IV (rounded to the nearest 250 mg and administered over 60 minutes) **Maintenance dose**: 1000 mg IV every 12 hours (given over 15 minutes) The dose may be adjusted as needed for therapeutic effect up to 1500 mg every 12 hours (3000 mg/day).	**CN**: behavioural symptoms (agitation, aggression, anger, anxiety, apathy, depersonalization, depression, emotional lability, hostility, hyperkinesias, irritability, nervousness, neurosis, and personality disorder (5 - 13%), somnolence (8 - 23%), headache (14%), hostility (2 - 12%) **GI**: vomiting (15%)

Source:

- Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons et al. J Neurotrauma. 2008 Mar;25(3):276-8.
- Flower O, Hellings S. Sedation in traumatic brain injury. Emerg Med Int. 2012;2012:637171
- American Pharmacists Association. Drug Information Handbook, 22th Edition Hudson: Wolters Kluwer Clinical Drug Information, Inc.; 2013

LIST OF ABBREVIATIONS

µg	microgramme
µL	microlitre
AIS	Abbreviated Injury Scale
BP	blood pressure
bpm	beats per minute
ACOS-COT/ CDC	American College of Surgeon – Committee on Trauma /Centre for Disease Control
CCR	Canadian Cervical Spine Rule
CCTHR	Canadian CT Head Rule
CI	confidence interval
CN	central nervous
CPG	clinical practice guidelines
CPP	cerebral perfusion pressure
CSI	cervical spine injury
CSF	cerebral spinal fluid
CT	computed tomography
DG	Development Group
DM	diabetes mellitus
ECP	emergency care providers
ED	emergency department
EP	emergency physician
ET CO_2	end tidal carbon dioxide
FFP	fresh frozen plasma
FiO_2	fraction of inspired oxygen
GA	general anaesthesia
GCS	Glasgow Coma Score
GI	gastrointestinal
GOS	Glasgow Outcome Scale
HDU	High Dependency Unit
HR	heart rate
ICH	intracranial haemorrhage
ICP	intracranial cerebral pressure
ICU	Intensive care unit
INR	international normalized ratio
IPH	intraparenchymal haemorrhage
ISS	Injury Severity Score
IV	intravenous
kg	kilogramme
kPa	kilopascal
LOC	loss of consciousness
LR	likelihood ratio
NEXUS	National Emergency X-radiography Utilization Study
NOC	New Orleans Criteria
MAP	mean arterial pressure
mg	milligramme
MoH	Ministry of Health
MHI	mild head injury
mL	millilitre

mm	millimetre
mmHg	millimetre mercury
MVC	motor vehicle collision
OR	odd ratio
PCC	prothrombin complex concentrate
PEEP	positive end-expiratory pressure
PHC	pre-hospital care
PTS	post-traumatic seizure
RC	Review Committee
RCT	randomised control trial
RR	respiratory rate
RTA	road traffic accident
SAH	subarachnoid haemorrhage
SBP	systolic blood pressure
SpO_2	oxygen saturation
TBI	traumatic brain injury
vs	versus
WAICH	Warfarin-associated intracranial haemorrhage

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