HEAD INJURY & TRAUMA

There are no effective therapies that can be applied after an injury, to reverse most pathologic aspects of traumatic brain injury (TBI)...

Epidemiology.

- Leading causes are *falls & MVAs*.
- TBI is the leading cause of traumatic death in people < 25 years.

Head Injury can be subdivided;		
Mild	= GCS 14-15	= 80% of cases
Moderate	= GCS 9-13	= 10% of cases.
Severe	= GCS ≤ 8	= 10% of cases.

Principles of Disease.

Anatomy & Physiology.

- *Scalp* consists of 5 layers. ➔

Skin
Close connective tissue & cutaneous vessels & nerves.
Aponeurosis (epicranial aponeurosis)
Loose connective tissue (scalping layer, "Danger Zone")
Pericranium (periosteum of skull bones)

- *Skull* comprises of:
  - Frontal, 2x parietal, 2x temporal, ethmoid, sphenoid & occipital bones.
  - Smooth outside / protrusion & ridges on inside.

- *Dura:*
  - Lines inner aspect of skull & the outermost aspect of brain.
  - Reflects upon itself to compartmentalise the brain
    - *falx cerebri & tentorium cerebelli*
  - Within the margins of dural reflections are the large *dural venous sinuses.*

- *Brain & CSF:*
  - Occupies ~80% of cranial vault. Utilises ~20% of body's oxygen volume.
  - Divided into cerebrum, cerebellum & brainstem.
  - Covered by three distinct membranes;
    - Meningeal *dura mata.*
    - *Arachnoid mata*
    - *Pia mata*
  - Brain is suspended in CSF (a buffer against trauma)
  - Normal CSF pressure is 6-20cm H_2O.

- *Cerebral Haemodynamics.*
  - Blood-brain-barrier (BBB) maintains brain microenvironment.
    - Neuroactive drugs must be lipophilic to cross.
  - Cerebral blood flow (CBF) is maintained by autoregulation.
    - CBF is maintained at constant levels with a MAP of 60-150mmHg.
      - Outside this range; CBF varies in a linear fashion.

HTN, alkalosis & hypocarbia promote vasoconstriction.
Hypotension, acidosis & hypercarbia promote vasodilatation.

- Linear relationship b/ween BCF & pCO2 (20-60mmHg)
    - 1mmHg drop = 2-3% diameter decrease.
    - PCO2 < 20mmHg = profound vasoconstriction
        - Leads to cerebral ischaemia.
    - Over 12-24hrs vessels adapt to the new CO2 and likely dilate.
- As PO2 declines, cerebral vessels dilate to ensure adequate oxygen supply.
- *Cerebral perfusion pressure;*
    - = MAP - ICP.
    - CBF remains constant with a CPP of 50-160mmHg.
    - CPP < 40mmHg = critical ischaemia.

- *Biomechanics of Head Trauma.*
    - Direct injury = direct impact with the head (by object) or arrest of motion (by another object)
        - skull provides considerable resistance & absorption of applied energy.
        - can have associated skull fracture.
        - shock waves can distort/disrupt intracranial contents & alter regional ICP.
    - Indirect injury = cranial contents are set in motion by forces other than direct contact with the skull.
        - common example = acceleration/deceleration
        - brain moves within the skull
        - bridging subdural vessels are strained & haematomas may result.
        - diffuse axonal injury results from differential shear forces

Brain Cellular Damage & Death.

- *Primary & Secondary Brain Injuries.*
    - the acute clinical picture of a TBI-patient is the sum of their primary & secondary injuries

Primary Brain injury = mechanical irreversible damage that occurs at the time of head trauma. Includes; lacerations, haemorrhages, contusions & tissue avulsion. No specific intervention can reverse or repair primary brain injury.	**Secondary Brain injury** = results from intra & extra-cellular derangements initiated at time of trauma with massive depolarisation of brain cells & subsequent ionic shifts. Potentially avoidable & reversible.

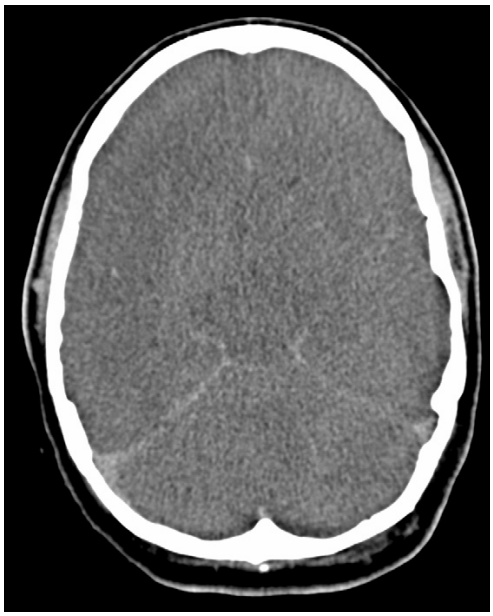
- *Secondary Systemic Insults.*
    - Influences the final neurological outcome following TBI.
    - Affected by premorbid & comorbid conditions.
    - **Hypotension.**
        - Systolic BP < 90mmHg --> potentially *doubles* mortality.
        - Reduces CPP & results in subsequent ischaemia/infarction.
    - **Hypoxia.**
        - pO2 < 60mmHg --> morality doubles/quadruples !!!
        - Multifactorial: apnoea, airway obstruction, chest/lung injury...

- **Hyperpyrexia.**
    - Temp > 38.5*C = worsened outcomes.
    - Mechanism is poorly understood.
- **Anaemia.**
    - usu. from blood loss (in traumatic setting).
    - Haematocrit < 0.30 = mortality increase.

Pathophysiology.

- *Increased ICP.*
    - recall Monro-Kellie doctrine.
    - defined as:
        - >15mmHg or >19.5cmH_2O
    - intracranial compensation can allow for up to 50-100mL of increased volume.
        - CSF displaced into spinal canal
        - Brain tissue can be somewhat compressed.
    - "*small increases in volume result in dramatic increases in pressure".*
    - Once CPP is compromised massive vasodilatation occurs
        - systemic pressure is transmitted to capillaries
        - outpouring of fluid into extravascular space --> *vasogenic oedema.*
    - When ICP > systemic arterial pressure = brain death ensues.

- *Brain Swelling & Cerebral Oedema.*
    - Congestive brain swelling = increased intracranial blood volume
    - Cerebral oedema = increase in brain volume from increased cerebral tissue water content.
        - On CT:
            - Bilateral ventricular compression
            - Loss of grey-white differentiation
            - Effacement of basal cisterns.
    - Vasogenic oedema:
        - Transvascular leakage & failure of BBB.
        - usu. associated with white matter.
        - Assoc. with focal contusions or haematomas.
    - *Cytotoxic oedema.*
        - Intracellular pathology --> pump failure.
        - Post-traumatic ischaemia or hypoxia.
        - Begins when CBF is < 40% of normal



- *Altered Levels of Consciousness.*
    - The hallmark of TBI.
    - Contributing factors; *hypoxia, hypotension, intoxication, seizures or hypoglycaemia*.

- *Cushing's Reflex.*
    - Progressive hypertension with associated bradycardia & diminished respiratory effort.
    - Associated w/ potentially lethal increases in ICP.

- *Cerebral Herniation.*
    - Can result from traumatic brain swelling, oedema formation or trauma mass lesion (haematoma or contusion).
    - When signs of herniation are present, mortality approaches 100% without rapid implementation of temporising measures & neurosurgical intervention !

Uncal Herniation.

- Most common herniation syndrome.
- Associated with extra-axial haematomas (in lateral middle fossa or temporal lobe).
- Ipsilateral temporal lobe *uncus* puts pressure on tentorium cerebelli & is forced through the hiatus.
    - Results in 3rd nerve compression (unequal pupils, abnormal EOM)
    - Fixed, dilated pupil is the end-point of ongoing compression.
    - Contralateral *Babinski's* occurs early.
    - Contralateral hemiparesis --> decerebrate posturing --> decorticate posturing.
    - Brainstem compression leads to *coma & cardiorespiratory alterations.*

Central Transtentorial Herniation.

- Rostrocaudal neurological deterioration.
- Results from expanding lesion at the vertex, frontal or occipital pole.
- Results in:
    - Subtle mental state changes --> depressed LOC
    - Bilateral motor weakness
    - Pinpoint pupils (< 2mm)
    - Bilateral hypertonicity & Babinski's
    - Progressive deterioration of respiratory function (yawn, sigh, tachypnoea)
        - Peri-arrest shallow, slow, irregular breaths.

Cerebellotonsillar Herniation.

- Results from cerebellar tonsils herniating downward through the foramen magnum.
- Usually from cerebellar mass or large central vertex mass.
- Results in:
    - Rapid respiratory & cardiovascular collapse w/ medulla impingement.
    - Pin-point pupils.
    - Flaccid quadriplegia (bilateral corticospinal tract compression).
- Mortality approaches 70%.

Upward Transtentorial Herniation.

- Occurs with expanding posterior fossa lesions.
- LOC rapidly declines
- Pin-point pupils from pontine compression.

Clinical Features & Diagnostic Strategies.

History.

- Mechanism of injury.
- Pre-morbid conditions & comorbidities.
- Medications (particular anticoagulants). Alcohol or drug use.
- Post-injury seizures, apnoea & duration of time until return to current LOC.

Acute Neurologic Examination.

- General:
    - Primary Survey
    - Mental state / GCS / Pupillary size & reactivity / Motor strength & symmetry.
- GCS:
    - Score out of 15.
    - Designed for assessment of patients with isolated head injury.
- Pupillary Exam:
    - Size & reactivity.
    - Traumatic mydriasis can confound interpretation.
- Motor Exam (Posturing):
    - Strength & symmetry
    - Ideally assess this prior to intubation/paralysis
    - *Hemiparesis contralateral to a dilated pupil = herniation syndrome.*
    - *Decorticate* posturing = abnormal flexion
        - Lesion above midbrain.
    - *Decerebrate* posturing = abnormal extension.
        - Worse prognosis (a more caudal injury).
- *Brainstem Function.*
    - Assessed by respiratory function, pupillary size and eye movements.
    - *Oculocephalic response = 'Doll's Eyes Test'.*
        - Test only after C-spine clearance.
    - *Oculovestibular response = 'Cold water calorics'.*
        - Cold water into ear should normally elicit nystagmus.
    - Other cranial nerves are affected with more profound head injury.
- *Deep tendon reflexes & pathological reflexes.*
    - including Babinski's
    - Rectal tone & anal reflexes should be assessed.

Other Examination Findings.

- Head & neck assessed for external evidence of trauma.
- Evidence of basilar skull fracture (*see box).*
- Don't forget C-spine protection.

BOX 38-1 Clinical Characteristics of Basilar Skull Fractures

Blood in ear canal
Hemotympanum
Rhinorrhea
Otorrhea
Battle's sign (retroauricular hematoma)
Raccoon sign (periorbital ecchymosis)
Cranial nerve deficits
  Facial paralysis
  Decreased auditory acuity
  Dizziness
  Tinnitus
  Nystagmus

Management.

Severe Traumatic Brain Injury.

- Post-resuscitative GCS ≤ 8 within 48 hours.
- Overall mortality approaches 60%.
- Clinical prognostic indicators:
    - initial motor activity
    - pupillary responsiveness
    - patients age & premorbid condition
    - occurrence of secondary systemic insult

Out of Hospital Care.

- Airway interventions / management.
    - Prehospital RSI issues include:
        - Apnoea/hypoxia/bradycardia
        - unintentional hyperventilation
        - Prolonged scene times.
    - Literature is unclear on benefit behind semi-elective pre-hospital RSI.
        - Depends on the setting of study & who performed the intubation.
- IV access & treatment of trauma-related hypotension.
    - Cardiac monitoring is compulsory.
    - Scalp lacerations can lead to significant haemorrhage
    - (apply 'less bulky' bandage).
- Intubation/sedation may also be indicated for agitated & noncompliant patients.
- Prompt transfer to hospital.

Emergency Department.

- **Airway.**
    - RSI for obtunded or agitated patients.
        - Brief neurologic examination must be performed prior to induction.
    - ICP spikes can occur with airway manipulation.
        - Theoretical benefit from *lignocaine* (1.5-2mg/kg IV).
- **Hypotension.**
    - Rarely caused by isolated head trauma (except as a terminal event).
    - If this occurs; rapid & thorough assessment takes place to identify the culprit.
        - Haemorrhage, haemorrhage, haemorrhage !!
        - Neurogenic shock (from spinal injury)
        - Others; obstructive (pneumothorax, tamponade) etc.
    - Normal saline vs Hartmann's vs Hypertonic saline.
- **Hyperventilation.**
    - Goal = *pCO_2 of 30-35mmHg* (can reduce ICP by up to 25%).
    - Onset in 30 seconds.
- **Osmotic Agents.**
    - Mannitol:
        - 0.25 - 1gram/kg.
        - Creates osmotic gradient prevent water movement out of vascular space; thereby reducing brain volume.
        - Effect onset within minutes and lasts 6-8 hours.
    - Hypertonic Saline:
        - use remains controversial.

- **Barbiturates:**
  - Reduces cerebral metabolic demands.
- **Steroids:**
  - No benefit.
- **Hypothermia:**
  - More numbers needed --> trend towards survival benefit.
- **Cranial Decompression.**
  - Involve burr-holes & craniectomy.
  - Obviously involves Neurosurgical consultation.
- **Seizure Prophylaxis.**
  - Early post-traumatic seizures occur in up to 12% of blunt-trauma & 50% of penetrating head injury patients.
  - Post-traumatic seizures have no predictive value for future seizures or epilepsy.
    - *They do however, worsen secondary brain injury.*

BOX 38-2 INDICATIONS FOR ACUTE SEIZURE PROPHYLAXIS IN SEVERE HEAD TRAUMA

Depressed skull fracture
Paralyzed and intubated patient
Seizure at the time of injury
Seizure at emergency department presentation
Penetrating brain injury
Severe head injury (Glasgow Coma Scale score ≤8)
Acute subdural hematoma
Acute epidural hematoma
Acute intracranial hemorrhage
Prior history of seizures

Treatment involves:
- Benzodiazepines
- Phenytoin / Fosphenytoin

Reduces risk of early seizures by 66%

- **Antibiotic Prophylaxis.**
  - For penetrating injury, open skull #s & complex scalp lacerations.

Ancillary Evaluation.
- **Laboratory Tests.**
  - Directed to physical examination.
- **Neuroimaging.**

Table 38-2 Comparison of Head Imaging Modalities

	COMPUTED TOMOGRAPHY SCANS	MAGNETIC RESONANCE IMAGING	ANGIOGRAPHY	SKULL RADIOGRAPHY
Advantages	Fast Patient accessible for monitoring Defines acute hemorrhages, mass effects, bone injuries, hydrocephalus, intraventricular blood, edema	Defines contusions and pericontusion edema, post-traumatic ischemic infarction, brainstem injuries	Helps localize acute traumatic lesions Defines vascular injuries, injuries to venous sinuses Detects mass effects	Readily available May help screen some patients for further imaging studies
Disadvantages	Artifacts arise from patient's movement, foreign bodies Streak artifacts may obscure brainstem or posterior fossa	Slow Patients not easily accessible for monitoring Does not define most acute hemorrhagic lesions Not useful for bone injuries	Does not define nature of acute lesion Does not detect infratentorial masses	Does not indicate presence or absence of intracranial injury
Indications	Acute severe head trauma Acute moderate head trauma Suspected depressed skull fracture High-risk minor head trauma Suspected child abuse in minor head trauma Deteriorating neurologic status	Persistent symptoms with postconcussive syndrome Suspected post-traumatic ischemic infarction Suspected contusions not seen on CT scan	Suspected vascular injury CT scan not available	CT scan may not be done Penetrating head trauma

Disposition.

- All patients with severe head trauma require *neuroimaging*.
- Neurosurgical consultation obtained as soon as possible.
- The haemodynamically unstable multi-trauma patient with concomitant head injury is a difficult case.
    - Sequence of repair/intervention is based on addressing the most life-threatening pathology first.
    - ie. ideally a trauma laparotomy for an unstable patient with free intraperitoneal fluid would occur prior to CT imaging of the head.

Moderate Head Trauma.

- Post-resuscitative GCS 9-13.
- Most survive to hospital presentation.
- ~40% will have an abnormal CT.
- Benefit largely from avoidance of secondary insults.

Clinical Features.

- A wide variety of clinical presentations occur w/ moderate head injury.
- Often have:
    - a change in conscious state at time of injury
        - often confused & somnolent @ time of presentation
    - progressive headache & vomiting
    - post-traumatic seizures
    - post-traumatic amnesia.
- Many have concomitant serious facial injuries.
- Rarely, “Talk & Die” phenomenon of rapid deterioration
    - usu. *epidural haematomas*
    - can also be subdurals or contusions w/ subsequent oedema.

Acute Management.

- Close observation is mandatory (?mental status deterioration vs focal findings).
- Early CT-scanning & avoidance of secondary systemic insults.
- Medical management of acute herniation syndromes.
- Neurosurgical intervention.

Disposition.

- All patients with moderate head injury should be admitted for observation, even with a normal CT scan.
- Low threshold for repeat CT if focal findings develop (or if no improvement at 48hrs).

Complications.

- Overall mortality is ~20%. Morbidity is substantial.
- Most patients remain symptomatic for extended periods following their injury.
- At 3 months;
    - 90% have memory difficulties
    - 70% unable to return to work.
    - 50% have long-term disability interfering with daily functioning.
- Delayed MRI can be helpful for prognostication & directing future rehabilitation.

Mild Head Trauma.

- GCS 14-15.
- Temporary/brief interruption of neurologic function.
- A clinical diagnosis.
- ~5% will have an abnormal CT
    - < 1% will need neurosurgical intervention
- GCS itself is not sensitive enough to prognosticate minor TBI...

Clinical & Historical Features.

- The vast majority of minor TBI patients arrive to ED with resolving or completely resolved symptoms.
- Most common complaints:
    - Headache.
    - Nausea & vomiting.
    - Disorientation / confusion / amnesia.
- Subtle findings include:
    - Balance deficits
    - Impaired verbal memory
    - Delayed language comprehension
    - Slowed speech.

Post-traumatic amnesia is a better predictor of injury severity & eventual outcome than duration of retrograde amnesia or GCS.

The goal of the ED assessment is to identify the 'high-risk' patients with minor TBI.

Imaging Studies.

- Major controversial decision is, 'who to scan?'
- Early scan vs prolonged observation.
- CT scan should be performed on *all patients who stratify into being 'high-risk'.*
    - see table below (from ITIM, NSW Health).
- Skull xray is unhelpful.
    - Suspicion of skull # = CT scan.
- Other modalities include:
    - MRI (very sensitive for DAI & ischaemia)
    - PET scan.

BOX 38-3 Risk Stratification in Patients with Minor Head Trauma

High Risk
Focal neurologic findings
Asymmetrical pupils
Skull fracture on clinical examination
Multiple trauma
Serious, painful, distracting injuries
External signs of trauma above the clavicles
Initial Glasgow Coma Scale score of 14 or 15
Loss of consciousness
Post-traumatic confusion/amnesia
Progressively worsening headache
Vomiting
Post-traumatic seizure
History of bleeding disorder/anticoagulation
Recent ingestion of intoxicants
Unreliable/unknown history of injury
Previous neurologic diagnosis
Previous epilepsy
Suspected child abuse
Age >60 yr, <2 yr

Medium Risk
Initial Glasgow Coma Scale score of 15
Brief loss of consciousness
Post-traumatic amnesia
Vomiting
Headache
Intoxication

Low Risk
Currently asymptomatic
No other injuries
No focality on examination
Normal pupils
No change in consciousness
Intact orientation/memory
Initial Glasgow Coma Scale score of 15
Accurate history
Trivial mechanism
Injury >24 hr ago
No or mild headache
No vomiting
No preexisting high-risk factors

Risk factors indicating potentially significant mild head injury

- GCS <15 at 2 hours post injury
- Deterioration in GCS
- Focal neurological deficit
- Clinical suspicion of skull fracture
- Vomiting (especially if recurrent)
- Known coagulopathy / bleeding disorder
- Age >65 years
- Post traumatic seizure
- Prolonged loss of consciousness (>5 min).
- Persistent post traumatic amnesia (AWPTAS <18/18)*
- Persistent abnormal alertness / behaviour / cognition*
- Persistent severe headache*
- Large scalp haematoma or laceration.**
- Multi-system trauma**
- Dangerous mechanism**
- Known neurosurgery / neurological deficit.**
- Delayed presentation or representation**

* particularly if persists at 4 hours post time of injury
**clinical judgement required

Ancillary Studies.

- Urine toxicology screen & blood ethanol levels *may be helpful.*

Disposition.

- Most patients with *low-risk minor TBI* can be discharged after a normal examination & reasonable period of observation (ie. 4-6 hours).
- Detailed follow-up & 'return to ED' instructions should be discussed.
    - Any doubt to safety for discharge = brief inpatient admission.
- Low threshold for imaging patients who 'return to ED'.
- **Concussion.**
    - A type of minor TBI.
    - Results in distortion of axons, vasculature & brain neuroanatomy.
    - Imaging (CT & MRI) is generally negative.
    - Functional studies suggest abnormal glucose uptake and blood flow.
    - Symptoms include ongoing headache, dizziness, confusion & amnesia.
        - Children w/ concussion can be vomiting, tachycardic and pale !!
        - Generally resolve w/in 6 hours, but can last 5-7 days.
    - Avoidance of contact sports following injury is important.
        - High rate of re-injury within 10 days !!
    - ***Second-impact Syndrome:***
        - A second concussion (before being completely asymptomatic from the first injury) can lead to a rapid (sometimes fatal) neurologic decline.
        - Associated with marked brain swelling & subsequent herniation.
        - Mandates at least 1 week break off sport, prior to 'return to play'.
- Most patients have a rapid & complete resolution of symptoms.
- Persistence of symptoms = ***post-concussive syndrome.***
    - Headache, sensory insensitivity, memory & concentration difficulties.
    - Irritability, sleep disturbance & depression.

Paediatric Head Injuries.

Pathophysiology.

- Children's skulls are more distendable than adults (until cranial sutures close)
    - Therefore, young children often sustain less TBI than adults.
- Unfortunately, very young children appear to have worse outcomes from mild TBI.
    - ?more difficult to assess.
    - ?reluctance to initiate imaging (radiation risk, sedation required etc).
- Children have less traumatic mass lesions.
    - More commonly diffuse brain swelling & DAI.

Clinical Features.

- **History:**
    - Mechanism of injury
    - Appearance of child immediately before & after injury
    - Subsequent events.
    - Children have a higher incidence of post-traumatic seizures.
- **Examination:**
    - GCS / Pupillary exam / Motor function & lateralisation.
    - Modified GCS for children < 5 years.
    - Evidence of head trauma (especially *non-frontal scalp haematoma*) or concomitant injury.

Impact Seizures:
- Can occur w/ minor injury.
- Do not predict subsequent post-traumatic seizures.

Post-concussive Blindness:
- Serious complication of concussive injury.
- Assoc. with occipital trauma/impact.
- Temporary loss of vision (mins-hours).

- Post-traumatic intracranial lesions can be very subtle.
    - Beware the irritable toddler.
- Consider and investigate the possibility of *non-accidental injury*.
    - Does the alleged mechanism match (1) the injury sustained & (2) the age-appropriate physical capability of the child ??
- *Shaken-Baby Syndrome:*
    - Retinal haemorrhages, SDHs, subarachnoid blood.
    - No evidence of external head trauma.

Diagnosis & Management.

- Initial assessment involves primary survey, C-spine immobilisation & assessment for other major trauma.
- Specifically for head injury the primary goals are;
    - Prevention of secondary insults, prevention of increasing ICP and detection of traumatic lesions.
- Initial management of airway, circulation etc mirrors that for adult care (*see above)*.
- Avoid hypoxia & hypotension.

Hypovolaemic hypotension *CAN* occur with isolated head trauma.

- up to 80% of children with severe TBI will have elevated ICPs.
- Management of raised ICP is similar to adult-care.
    - Intubation & controlled hyperventilation (CO_2 30-35mmHg)
    - Mannitol (0.25-0.5 g/kg) *or* Hypertonic (3%) Saline (0.1-1.0 mL/kg).
- CT scan is imaging modality of choice (in moderate-severe TBI).
    - Strongly consider in *high risk, minor TBI*.
    - Risk vs Benefit = diagnosis vs irradiation & need for sedation.
- Skull radiographs:
    - Controversial, but occasionally appropriate.
- The presence of a skull fracture significantly increases the likelihood of intracranial pathology.
    - The converse is not true.
- Leptomeningeal cysts are complications from linear skull fractures
    - Represent CSF leakage (from dural injury).

Over all, children with severe TBI have better mortality and a better neurologic outcome, than comparably injured adults.

Parents should be educated about *warning signs & symptoms* of delayed complications of minor head injury prior to discharge home.

Multiple studies & clinical prediction rules exist on *high risk mild-TBI in children.*

- Kuppermann, N. et al. (2009). Identification of children at very low risk of clinically-important brain injuries after head trauma: a prospective cohort study. *The Lancet*, *374*(9696), 1160–1170. (**PECARN**)
- Dunning, J. et al. (2006). Derivation of the children's head injury algorithm for the prediction of important clinical events decision rule for head injury in children. *Archives of disease in childhood*, *91*(11), 885. **(CHALICE)**

The children's head injury algorithm for the prediction of important clinical events rule

A computed tomography scan is required if any of the following criteria are present.

- *History*
  - Witnessed loss of consciousness of >5 min duration
  - History of amnesia (either antegrade or retrograde) of >5 min duration
  - Abnormal drowsiness (defined as drowsiness in excess of that expected by the examining doctor)
  - ⩾3 vomits after head injury (a vomit is defined as a single discrete episode of vomiting)
  - Suspicion of non-accidental injury (NAI, defined as any suspicion of NAI by the examining doctor)
  - Seizure after head injury in a patient who has no history of epilepsy
- *Examination*
  - Glasgow Coma Score (GCS)<14, or GCS<15 if <1 year old
  - Suspicion of penetrating or depressed skull injury or tense fontanelle
  - Signs of a basal skull fracture (defined as evidence of blood or cerebrospinal fluid from ear or nose, panda eyes, Battles sign, haemotympanum, facial crepitus or serious facial injury)
  - Positive focal neurology (defined as any focal neurology, including motor, sensory, coordination or reflex abnormality)
  - Presence of bruise, swelling or laceration >5 cm if <1 year old
- *Mechanism*
  - High-speed road traffic accident either as pedestrian, cyclist or occupant (defined as accident with speed >40 m/h)
  - Fall of >3 m in height
  - High-speed injury from a projectile or an object

If none of the above variables are present, the patient is at low risk of intracranial pathology.

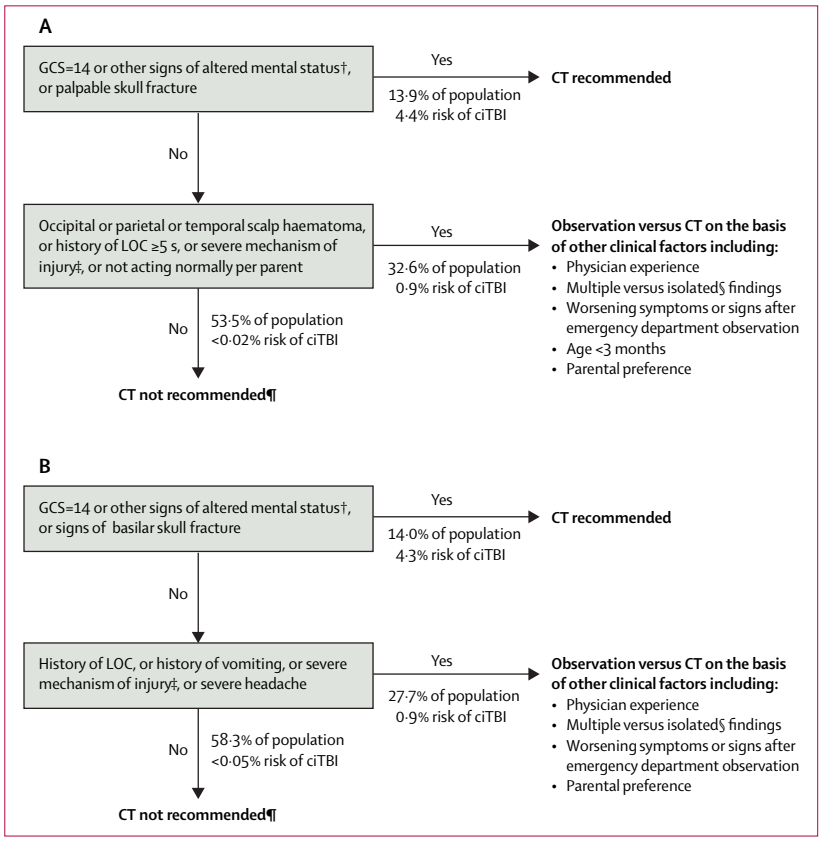


Figure 3: **Suggested CT algorithm for children younger than 2 years (A) and for those aged 2 years and older (B) with GCS scores of 14–15 after head trauma***

PECARN Study

CHALICE Study

Penetrating Head Injuries.

- The most common mechanism of injury is GSW.
- Dramatic injuries & dramatic presentations.
- Mortality approaches 90%.
- Mortality predicted by GCS & pupillary responsive upon arrival.
  - GCS < 5 = 100% mortality.
  - GCS > 8 w/ reactive pupils = 75% survival.

Pathophysiology.

- There are several different patterns of damage.
  - *Tangential injuries*: high velocity, low energy missiles may not penetrate skull.
    - may have focal cortical contusions at point of impact.
  - *Perforating wounds:* high-velocity projectiles causing 'through & through' injuries of the brain.
    - entrance wound is usu. smaller than exit wound.

        - *Penetrating missile wounds:* mod-high velocity projectiles from close range.
            - may travel through the entire skull, but more prone to ricochet off inner table of skull.
- Wounding capacity of projectile is proportional to its kinetic energy.
    - As bullet passes through tissue, a cavity is created.
    - Cavity can be up to 10x the diameter of the actual missile.
- Morbidity & mortality is dependent on:
    - intracranial path
    - speed of entry
    - size & type of penetrating object.

> Projectiles that cross the midline, pass through ventricles or come to rest in posterior fossa carry extremely high mortality.

Clinical Features.

- Primary survey, with focus on presenting *GCS and pupillary reaction.*
- Devastating physiologic changes can occur immediately after injury.
- ICP rapidly increases, with loss of CBF autoregulation.

Management.

- Care is focussed at reducing the occurrence of secondary systemic insults.
- Management should be aggressive until prognosis is established.
- Intubation is nearly always indicated.
- IV antibiotics is mandatory.
- Seizure prophylaxis (30-50% will develop seizures).
    - Should *not* continue beyond a week.
- CT scan; ASAP !
    - Angiography can be added to assess for vascular injury.
- Impalement --> leave the object in place.

Complications After Head Injury.

Neurological Complications.

- **Seizures.**
    - Very common in post-traumatic phase.
    - *Box 38-2 (above) - lists indications for prophylaxis.*
- **CNS infections.**
    - *Meningitis after Basilar Fractures:*
        - Ceftriaxone + vancomycin is a reasonable combination.
        - ABx prophylaxis is currently not indicated for CSF leaks.
    - *Brain Abscess.*
        - Infrequently associated w/ penetrating brain injury.
        - Dx made with contrast-enhanced CT.
        - Requires operative drainage.
    - *Cranial Osteomyelitis.*
        - Pain, tenderness, swelling.
        - Can be diagnosed on plain radiograph (skull) or *technetium bone scan.*

Medical Complications.

- **DIC.**
    - Brain is major source of thromboplastin & results in massive extrinsic clotting system activation.
    - Increased morbidity & mortality after severe TBI.
    - Correct coagulopathy and thrombocytopenia.
    - Repeat CT in stable patients who deteriorate (?extension of haemorrhage).
- **Neurogenic Pulmonary Oedema.**
    - Occurs minutes to days following TBI.
    - Can be reversed by ICP management.
- **Cardiac Dysfunction.**
    - A variety of dysrhythmias & conduction abnormalities can occur after TBI.
    - Ensure adequate cardiac output is maintained.
    - Occurs in up to 70% of SAH patients.
    - SVTs are most common.
    - Diffuse large (upright or inverted) T-waves, ST-segment changes & QT changes can all occur.

Specific Injuries.

Scalp Wounds.

- Extremely common.
- A source of significant bleeding.
- *Management* involves:
    - Direct digital compression.
    - Infiltration of lignocaine w/ adrenaline.
    - Ligation of identified culprit blood vessels.

Closure of the wound is the most effective way to achieve haemostasis.

- Irrigation of debris is crucial, as is removal of blood clots
- Inspect & palpate for underlying skull fractures.
- Avoid embedding hair in the laceration.

Skull Fractures.

- All proven skull fractures require assessment for other traumatic injuries.
- Fractures can result in pneumocranium, damage to underlying vessels (eg. middle meningeal artery, or dural venous sinuses)
- Best diagnosed on CT.
- **Linear fractures.**
    - Extend through full skull thickness.
    - Can disrupt vascular structures & result in epidural haematomas.
    - Can be difficult to differentiate from sutures.
    - *Sutural diastasis & comminuted #s* lie on this spectrum.
- **Depressed fractures.**
    - Predispose to significant underlying brain injury, infections and seizures.
    - Bone can penetrate tissue & lacerate dura.
    - *Surgical elevation* is required if the free piece of bone is depressed further than the adjacent inner table of skull.
- **Basilar fractures.**
    - Fractures of the base of skull & can often communicate with subarachnoid space, sinuses or middle ear.

        - Can result in entrapment or compression of cranial nerves.
        - Vascular injury (from involvement of cavernous sinus) can occur.
        - Mechanical deafness can result (ossicle dislocation or fracture).
    - **Open fractures.**
        - A fracture with overlying scalp laceration, or with disruption of sinuses or middle ear.

Diffuse Axonal Injury.

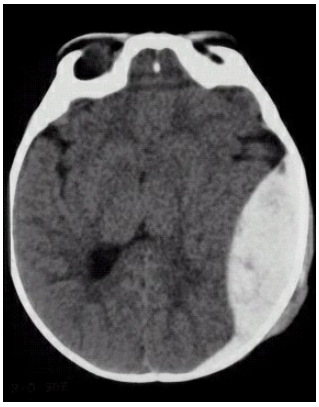
- Axons are stretched & twisted by shear & tensile biomechanical forces
    - Same forces that produce concussion.
    - Damaged axons become oedematous & separate from each other.
    - Causes widespread disruption of cortical physiology & microanatomy.
- Cause of persistent traumatic coma that begins immediately at the time of trauma.
    - Occurs in up to 50% of all severe TBI.
- No focal traumatic lesions are seen on CT.
    - MRI more sensitive in making diagnosis.
- Severity is determined by the clinical course
    - **Mild.**
        - Coma for 6-24 hours, then follow commands.
        - Mortality ~ 15%
    - **Moderate.**
        - Most common, with coma > 24 hours.
        - Transient decorticate / decerebrate posturing; recovering purposeful movements later.
        - ~25% die of complications of prolonged coma.
    - **Severe.**
        - Prolonged coma.
        - Demonstrate persistent brainstem functioning (posturing) & autonomic dysfunction.
        - Swelling can lead to raised ICP, and herniation syndromes can occur.
        - Most are severely disabled in a persistent vegetative state.

Contusions.

- "Bruises" to the surface of the brain.
- usu. occur at the *poles of the brain*, & the *inferior surfaces of temporal/frontal* lobes.
- *Coup* vs *Contrecoup* injuries.
- Initially petechial haemorrhages; develop subsequent oedema (& haemorrhage) leading to mass effect.
- Most make uneventful recoveries.

Epidural (extradural) Haematoma.

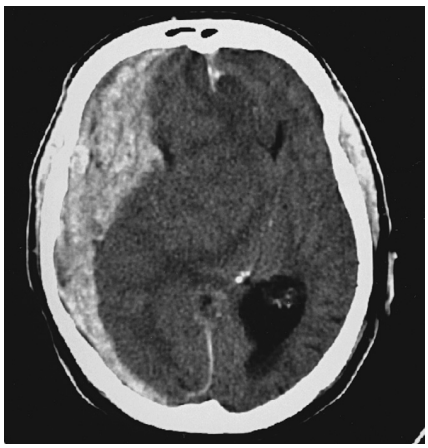
- Clots between the *inner table of the skull & dura.*
- Mostly caused by direct impact injury causing forceful skull deformity.
    - Often #'s occur across the *middle meningeal artery, vein or dural sinus.*
    - usu. arterial bleeding that dissects the dura away.
- Temporoparietal region is most common.
- Typically *unilateral* w/ rapid & dramatic deterioration.
    - can have other intracranial lesions (SAH, SDH, contusions).
- Classically have a 'lucid' period prior to decompensation.
    - Assoc. w/ headache, drowsiness, N&V & dizziness.

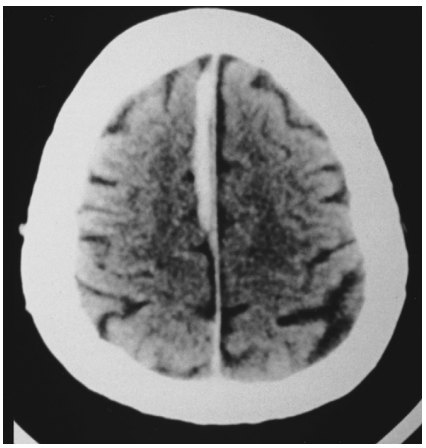


- on CT:
    - Hyperdense, biconvex, ovoid & lenticular.
    - Does not extend beyond suture lines.
    - Margins are sharply defined.
    - Can have mixed density.
    - Don't forget to look at the posterior fossa.
- Management involves surgical drainage.
    - EDH > 30cm^3 *or*
    - presentation of coma w/ asymmetric pupils.

Subdural Haematoma.

- Clots between the *dura & the brain.*
- Caused by movement of brain relative to skull (acceleration / deceleration)
- More common in atrophic brains (alcoholic / elderly) due to *superficial bridging vessels* traversing greater distances.
- Slower bleeding (than EDHs); delays the signs & symptoms.
- Often have concomitant DAI.
- ***Acute SDH*** = < 24 hours after trauma.
    - Mortality of those requiring neurosurgical intervention = 40-60%.
    - Early consultation w/ Neurosurgery is required for all SDHs.
        - There are consensus guidelines on conservative vs operative Mx of these.
        - Recommendation for operative drainage if:
            - Acute SDH > 10mm thickness
            - Midline shift > 5mm.
    - Often extend beyond suture lines & follows the tentorium/falx (*below right*).

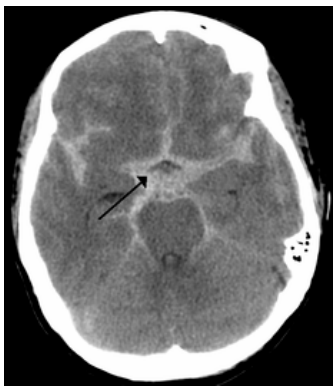




- ***Subacute SDH*** = symptomatic b/ween 24 hours & 2 weeks after injury.
    - May appear hypodense or isodense on CT.
    - Contrast increases detection.
    - Most require surgical drainage of their lesion.
- ***Chronic SDH*** = symptomatic after 2 weeks from injury.
    - Often present w/ very subtle or non-specific findings. Often are *bilateral*.
    - Most cannot recall their head injury or LOC.
    - on CT; iso/hypodense, typically with mass effect. Blood of various ages.
    - Treatment is controversial, but require drainage if symptomatic.
    - Mortality effected by concomitant injury and comorbidities/age.
        - Posterior fossa SDHs = *high* mortality.
- *Suspect NAI for children diagnosed with SDH.*

Subdural Hygroma.

- A clear, xanthochromic blood-tinged fluid in the dural space.
- Pathogenesis uncertain. May result from CSF leakage from arachnoid tear.
- Features of raised ICP may be present on examination.
- Common to be bilateral.

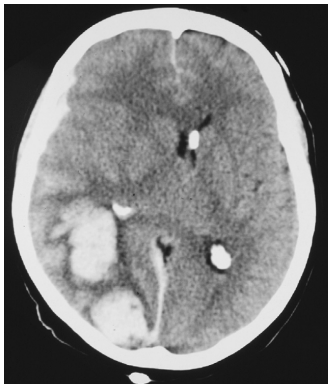


Traumatic Subarachnoid Haemorrhage.

- Blood within the CSF and meningeal intima.
- Present in ~44% of all severe TBI patients.
- Carries increased incidence of skull #s & contusions.
- The *amount of blood directly correlates with outcome !*
- Headache & photophobia are common symptoms.
- CT demonstrates:
    - Blood @ basal cisterns, inter-hemispheric fissures and sulci.
- Complications include *vasospasm* inducing ischaemia.
    - Nimodipine is used in ICU setting to reduce the incidence of this.

Intracerebral Haematoma.

- Formed deep within the brain tissue, caused by sheering & tensile forces.
    - Tearing of deep small-caliber arterioles.
- Results in small petechial haemorrhages which coalesce.
- 85% are found in frontal / temporal lobes.
    - Often in conjunction w/ EDHs.
- Signs & symptoms depend on size, location & ongoing bleeding.
    - 50% report an LOC at time of injury.
- On CT;
    - Well-defined, hyperdense homogenous area of haemorrhage.
- Many patients require emergent intervention/surgery for management of ICP.
    - Extension into ventricles or cerebellum carry higher mortality.



Traumatic Intracellebellar Haematoma.

- Rare.
- Result from direct occipital blow.
- Often have associated skull# or posterior fossa SDH.
- Present with posterior fossa symptoms
- Mortality is very high !!

Initial Management of Adult Mild Closed Head Injury

Initial GCS 14-15 on arrival following blunt head trauma

Stabilise ABCDEs and assess clinical risk factors.

Commence minimum of hourly clinical observations of vital signs, GCS, pupils, PTA and clinical symptoms

Low risk mild head injury

No indication for CT scan if all of...

- GCS 15 at 2 hours post injury.
- No focal neurological deficit.
- No clinical suspicion of skull fracture.
- No vomiting
- No known coagulopathy or bleeding disorder.
- Age <65 years.
- No seizure
- Brief loss of consciousness (<5 mins).
- Brief post traumatic amnesia (<30 mins)
- No severe headache.
- No large scalp haematoma or laceration
- Isolated head injury
- No dangerous mechanism.
- No known neurosurgery / neurological impairment.
- No delayed presentation or representation

NOTE:
Mild acute clinical symptoms such as lethargy, nausea, dizziness, mild headache, mild behavioural change, amnesia for event and mild disorientation are common and are not associated with increased risk of intracranial injury. These clinical symptoms usually start to improve within 2 to 4 hours of time of injury.

Continue minimum of hourly clinical observations until at least four hours post time of injury.

Clinically deteriorates or clinical symptoms not improving during observation period

Clinical symptoms IMPROVING or remain normal during period of observation.

Clinical symptoms IMPROVING at 4-6 hours post time of injury.

Clinical symptoms NOT IMPROVING at 4-6 hours post time of injury.

Clinically safe for discharge for home observation if:

- GCS 15/15
- No persistent post traumatic amnesia (nb A-WPTAS 18/18)
- Alertness / behaviour / cognition returning to normal
- Clinically improving after observation.
- Normal CT scan or no indication for CT scan.
- Clinical judgment required regarding discharge and follow up of elderly patients or patients with known coagulopathy or bleeding disorder due to increased risk of delayed subdural haematoma.

Clinically safe for discharge for home observation if:

- Responsible person available to take home and observe.
- Able to return if deteriorates.
- Discharge advice is understood.

Discharge for home observation if above criteria met:

- Provide written patient advice sheet
- Provide discharge summary for GP
- All patients should be advised to see their GP for follow up if they are not feeling back to normal within 2 days
- Any patients who have minor CT abnormalities, who suffered significant clinical symptoms or who had prolonged post traumatic amnesia should be routinely referred to their GP for follow up due to an increased risk of post concussion symptoms.

High risk mild head injury

Strong indication for CT scan if...

- GCS <15 at 2 hours post injury. #1
- Deterioration in GCS.
- Focal neurological deficit.
- Clinical suspicion of skull fracture #2
- Vomiting (especially if recurrent) #3
- Known coagulopathy or bleeding disorder #4
- Age >65 years. #5
- Seizure #6
- Prolonged loss of consciousness (>5 mins).
- Persistent post traumatic amnesia (A-WPTAS <18/18 at 4hrs post injury) #7
- Persistent abnormal alertness / behaviour / cognition #8
- Persistent severe headache.

Relative indication for CT scan if...

- Large scalp haematoma or laceration #9
- Multi-system trauma. #10
- Dangerous mechanism. #11
- Known neurosurgery / neurological impairment. #12
- Delayed presentation or representation. #13

Note
The presence of multiple risk factors is more concerning than a single isolated risk factor. In most uncomplicated mild head injury patients clinical symptoms start to improve by 2 hours post injury and are returning to normal by 4 hours post injury. Clinical symptoms that are deteriorating or not improving by 4 hours post injury on serial observation such as abnormal alertness / behaviour / cognition, PTA, vomiting or severe headache are very concerning.

Indication for CT scan. Continue clinical observations.

Normal CT scan

Abnormal CT scan

CT scan unavailable

Consider transfer for CT scanning particularly if:

- Persistent GCS <15.
- Deterioration in GCS.
- Focal neurological deficit.
- Clinical suspicion of skull fracture
- Known coagulopathy (esp if INR>4)
- Persistent abnormal alertness, behaviour, cognition, PTA, vomiting or severe headache at 4 hours post injury

Consult senior clinician and network neurosurgical service regarding further management and disposition. Continue clinical observations in hospital.

Explanatory notes for risk factors

1. Using GCS<15 at 2 hours post injury allows clinical judgement for patients who present soon after injury or who have drug or alcohol intoxication. Drug or alcohol intoxication has not been shown to be an independant risk factor for intracranial injury but persistent GCS<15 is a major risk factor and mandates CT.
2. Clinical suspicion of skull fracture includes history of focal blunt assault or injury; palpable skull fracture; large scalp haematoma or laceration; signs of base of skull fracture – haemotympanum / CSF leak / raccoon eyes / Battles sign.
3. Recurrent vomiting more concerning than isolated vomiting but both are indications.
4. Known coagulopathy is both a strong indication for early CT scan and to check the INR. Early reversal of anticoagulation if abnormal CT scan and consider reversal if initially normal CT scan with high INR (>4) depending on clinical situation.
5. Elderly patients have increasing risk of intracranial injury with increasing age; routine CT scanning indicated unless totally asymptomatic patient with no other risk factors.
6. Brief generalised seizures immediately following head injury are not significant risk factors. Prolonged, focal or delayed seizures are risk factors for intracranial injury.
7. Post traumatic amnesia may manifest as repetitive questioning or short term memory deficits and can be objectively tested using the A-WPTAS. PTA > 30 mins is a minor risk factor and PTA > 4 hours a major risk factor for intracranial injury.
8. Abnormal alertness/behaviour/cognition detects subtle brain injury better than GCS and should be part of the bedside assessment. Family may help establish what is normal.
9. Multi-system trauma – beware patient with unstable vital signs or distracting injuries or who receive analgesia or anaesthesia, as significant head injury is easily missed.
10. Clinical judgement required as to what is a large scalp haematoma or laceration.
11. Dangerous - MVA ejection / rollover; pedestrians / cyclists hit by vehicle; falls >own height or five stairs; falls from horses / cycles etc; focal blunt trauma, eg bat / ball / club.
12. Known neurosurgery/neurological impairment – conditions such as hydrocephalus with shunt or AVM or tumour or cognitive impairment such as dementia make clinical assessment less reliable and may increase risk of intracranial injury.
13. Delayed presentation should be considered as failure to clinically improve during observation. For representation consider both intracranial injury and post concussion symptoms and have a low threshold for CT scanning if not done initially.

Initial Management of Adult Closed Head Injury

Initial Assessment and Stabilisation of ABCDEs

Trauma Team activation if initial GCS 3-13 or otherwise indicated

Commence minimum of hourly clinical observations of vital signs, GCS, pupils, PTA (if applicable) and clinical symptoms

GCS 3-8

Severe Head Injury (10%)

- Early intubation
- Supportive care of ABCDEs
- Prevent secondary brain injury by avoiding hypoxaemia and hypotension
- Early CT scan
- Early neurosurgical consult
- Early retrieval consult if transfer required
- Consider use of anticonvulsants
- Consider ICP monitoring
- ICU admission
- Brain injury rehabilitation consult

NB. Minimum supportive care aims to prevent secondary brain injury:

- PaO_2 >60
- SaO_2 >90
- $PaCO_2$ 35-40
- Systolic BP >90
- Head up 30°

GCS 9-13

Moderate Head Injury (10%)

- Supportive care of ABCDEs
- Prevent secondary brain injury by avoiding hypoxaemia and hypotension
- Early CT scan
- Period of clinical observation
- Consider intubation in the event of clinical deterioration or to facilitate management
- Early neurosurgical consult if not clinically improving and/or abnormal CT scan
- Early retrieval consult if transfer required
- Admit to hospital for prolonged observation unless rapid clinical improvement to GCS 15, normal CT scan and absence of other risk factors (as per mild head injury)
- Routine post traumatic amnesia testing and consider referral to brain injury rehabilitation service due to significant risk of cognitive behavioural social sequelae

GCS 14-15

Mild Head Injury (80%)

- Initial assessment followed by period of clinical observation to detect risk factors for significant intracranial injury.
- CT scan not routinely indicated unless one or more risk factors listed below are present.
- Discharge for home observation with head injury advice sheet at 4 hours post injury if clinically improving with either no risk factors indicating the need for CT scan or normal CT scan if performed.
- Consider hospital admission and consult network neurosurgical service if abnormal CT scan.
- Consider hospital admission for observation if clinically not improving at 4 hours post injury irrespective of CT scan result.
- Consider hospital admission for observation if elderly, known coagulopathy or socially isolated.
- Advise patients to see their local doctor if they do not return to normal within 48 hours so they can be reassessed and monitored for post concussion symptoms.

NB. Also see separate Mild Head Injury Algorithm.

Risk factors indicating potentially significant mild head injury

- GCS <15 at 2 hours post injury
- Deterioration in GCS
- Focal neurological deficit
- Clinical suspicion of skull fracture
- Vomiting (especially if recurrent)
- Known coagulopathy / bleeding disorder
- Age >65 years
- Post traumatic seizure
- Prolonged loss of consciousness (>5 min).
- Persistent post traumatic amnesia (AWPTAS <18/18)*
- Persistent abnormal alertness / behaviour / cognition*
- Persistent severe headache*
- Large scalp haematoma or laceration.**
- Multi-system trauma**
- Dangerous mechanism**
- Known neurosurgery / neurological deficit.**
- Delayed presentation or representation**

* particularly if persists at 4 hours post time of injury

**clinical judgement required

What should be done when patients with closed head injury acutely deteriorate?

Early signs of deterioration

- Confusion
- Agitation
- Drowsiness
- Vomiting
- Severe headache

Late signs of deterioration

- Decrease in GCS by two or more points
- Dilated pupil(s)
- Focal neurological deficit
- Seizure
- Cushing's response – bradycardia and hypertension

Clinical approach

- Resuscitate ABCDEs and exclude non head injury cause
- Supportive care of ABCDEs
- Early intubation if indicated
- Immediate CT scan
- If clinical or CT evidence of raised ICP/mass effect consult with network neurosurgical and retrieval services re;
  - short term hyperventilation to $PaCO_2$ 30-35
  - bolus of mannitol (1g/kg)
  - local burr holes/craniectomy when more than 2 hours from neurosurgical care
  - prophylactic anti-convulsants

AMRS (adult) **1800 650 004**

'formerly the MRU'

NETS (children) **1300 362 500**

Network neurosurgical service

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

Potential indications

Patient with severe head injury

Patient with moderate head injury if:

- clinical deterioration
- abnormal CT scan
- normal CT scan but not clinically improving
- CT scan unavailable.

Patient with mild head injury if:

- clinical deterioration
- abnormal CT scan
- normal CT scan but not clinically improving within 4-6 hours post injury
- mild head injury with CT scan unavailable, particularly if:
  - Persistent GCS<15
  - Deterioration in GCS
  - Focal neurological deficit
  - Clinical suspicion of skull fracture
  - Persistent abnormal mental status
  - Persistent vomiting
  - Persistent severe headache
  - Known coagulopathy (particularly if age >65 or INR >4)

Clinical approach

- When in doubt consult you network neurosurgical service.
- Patients with closed head injuries should be observed in facilities that can manage any complications that are likely to arise. Clinical judgment regarding risk of deterioration is required and neurosurgical consultation may be appropriate.
- Patients with closed head injuries should be transferred to the nearest appropriate hospital with neurosurgical facilities if there is significant risk of intracranial injury. The transfer of patients to hospitals with CT scan facilities but without neurosurgical services should be avoided.