CHAPTER 16

Mild traumatic brain injury

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Abstract

Background The incidence of traumatic brain injury (TBI) is high, varying between 229 and 1967 per 100 000, with the highest incidence occurring in men, aged 15–24 years. Approximately 90–95% of all TBIs are considered mild. Intracranial complications of mild traumatic brain injury (MTBI) are infrequent but potentially life-threatening, and may require neurosurgical intervention in a minority of cases (0.2–3.1%). Hence, a true health management problem exists because of the need to exclude the small chance of a life-threatening complication in large numbers of individual patients.

Objective To construct from the literature acceptable evidence-based guidelines for initial management with respect to ancillary investigations, hospital admission, observation, and follow-up after MTBI.

Methods Systematic review.

Results A systematic review of the literature revealed risk factors with sufficient sensitivity to predict the presence of intracranial complications (evidence Level I–IV) including Glasgow Coma Scale Score at the time of hospital admission, presence of persistent anterograde amnesia, retrograde amnesia longer than 30 min, trauma above the clavicles including facial or cranial soft tissue injury and clinical signs of skull fracture (skull base- or depressed skull fracture), severe headache, nausea, vomiting (≥2 times), focal neurological deficit, cranial nerve deficit, motor deficit, dysphasia, seizure, age, coagulation disorders, high-energy accident (dangerous mechanism of injury) and intoxication with alcohol/drugs.

Conclusion The guidelines in this paper present evidence for the importance of careful neurological

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examination, assessment of trauma history, recognition of risk factors and use of CT to detect all intracranial complications after MTBI.

Background

Trauma of the head can cause brain injury (Denny-Brown and Russell, 1941; Frowein and Firsching, 1990) and is a common cause of morbidity and mortality (Kraus *et al.*, 1996). After mild traumatic brain injury (MTBI), that is, patients with a hospital admission Glasgow Coma Score (GCS) of 13–15) (Teasdale and Jennett, 1974), mortality, almost exclusively caused by intracranial haemorrhage, is very low (between 0.04 and 0.29%) (Klauber *et al.*, 1989; af Geijerstam and Britton, 2003). The International Classification of Diseases (ICD-10) classifies acute traumatic brain injury (TBI) as S-02, S-04, S-06, S-07, S-09 in combination with dizziness or vomiting, retrograde or anterograde amnesia, impaired consciousness, skull fracture, and/or focal neurological impairment (Bellner *et al.*, 2003). As the incidence of TBI is high, varying between 229 and 1967 per 100 000, with the highest incidence occurring in men, aged 15–24 years (Jennet, 1996; Kraus *et al.*, 1996; von Wild and Wenzlaff, 2005) and 90–95% of all TBIs are considered mild, formal evidence-based clinical decision rules are warranted (Haydel *et al.*, 2000; Meerhoff *et al.*, 2000; Stiell *et al.*, 2001). There are practically no Class I and II data on TBI management in the literature. These guidelines try to provide a set of rules aimed at early recognition of symptoms and signs known to increase the risk of development of an intracranial haemorrhage after MTBI (Krau *et al.*, 1996; Jennet, 1996; Stiell *et al.*, 2001).

Search strategy

The Task Force systematically searched the English literature in the medline, EMBASE, Cochrane database (1966–2005) using the key words 'minor head injury', 'mild head injury', 'mild traumatic brain injury', 'traumatic brain injury', 'guidelines' and 'management'. Additional articles were identified from the bibliographies of the articles retrieved (including those in the German language), and from textbooks. Articles were included if they contained data on the classification system used (i.e. admission GCS 13–15) and outcome data (CT abnormalities, need for neurosurgical intervention, mortality) or management. Articles judged to be of historical value were also included. Initially, 540 articles were retrieved. Articles were reviewed by one author (PEV). For the purpose of this report, a total of 109 papers were finally included. Additional information can be found on the European Federation of Neurological Societies (EFNS) website. Where appropriate, a classification of evidence level (EL) was given for interventions, diagnostic tests, and grades of recommendation for management according to the neurological management guidelines of the EFNS (Brainin *et al.*, 2004). Where there was a lack of evidence but consensus was clear we have stated our opinion as Good Practice Points (GPP).

Mechanisms of traumatic brain injury

Focal impact or contact injuries may cause closed and open skull fractures, extradural haematoma, subdural haematoma, cortical contusion, rupture of the dura mater with CSF leakage and/or prolaps of brain tissue. Direct collisional forces acting on the skull might compress the underlying tissue structures (coup) or of tissue remote from the site of the impact (contre-coup) (Pudenz and Shelden, 1946; Sellier, 1963).

Impact to the head *per se* is not mandatory to evoke brain dysfunction or brain damage. Except for skull fracture and extradural haematoma, all types of brain injury can be produced by (angular) acceleration of the head without impact, provided that there is a period of loss of consciousness (Gennarelli *et al.*, 1982; Birbamer *et al.*, 1994). Shear forces generated in the brain upon sudden rotation may cause damage to axons and blood vessels (Houlbourn, 1943; Gennarelli, 1983; Grcevic, 1988; Gerstenbrand and Stepan, 2001). Controversy exists whether diffuse brain dysfunction can occur in MTBI without there being structural damage (Trotter, 1924; Spatz, 1936).

Classification of mild traumatic brain injury

Many terms exist to describe and define MTBI (Frowein and Firsching, 1990; Williams *et al.*, 1990; Evans, 1992; Pople *et al.*, 1993; Birbamer *et al.*, 1994; Stein and Spettell, 1995; Teasdale, 1995; Culotta *et al.*, 1996; Gomez *et al.*, 1996; Saab *et al.*, 1996; Arienta *et al.*, 1997; Ingebrigsten, *et al.*, 2000; Gerstenbrand and Stepan, 2001; von Wild and Terwey, 2001; Stepan *et al.*, 2001). Here, a classification for MTBI is proposed based on admission GCS, trauma history (i.e., the duration of loss of consciousness [LOC] and post-traumatic amnesia [PTA]), age, neurological signs and symptoms), and risk factors for intracranial complications. Several sub-classifications are recognized to facilitate initial management decisions (Recommendation Level B) (table 16.1, figure 16.1).

RECOMMENDATIONS

MTBI is defined as the consequence of blunt (non-penetrating) impact with sudden acceleration, deceleration, or rotation of the head (ICD-10 codes: S-02, S-04, S-06, S-07, S-09) with a GCS score of 13–15 at the time of hospital admission (table 16.1).

Table 16.1 Classification of traumatic brain injury.

Classification	Admission Glasgow Coma Scale Score (GCS) and clinical characteristics modified from the Dutch, Scandinavian and American classification systems (Maas *et al.*, 1997; Stein and Spettell, 1995; Ingebrigsten *et al.*, 2000; Twijnstra *et al.*, 2001)
Mild	GCS = 13–15
Category	
0	GCS = 15 No LOC, no PTA = head injury, no TBI No risk factors
1	GCS = 15 LOC < 30 min, PTA < 1 h No risk factors
2	GCS = 15 and risk factors present*
3	GCS = 13–14 With or without risk factors present*
Moderate	GCS = 9–12
Severe	GCS ≤ 8
Critical	GCS = 3–4, with loss of pupillary reactions and absent or decerebrate motor reactions

Abbreviations: TBI, traumatic brain injury; GCS, Glasgow Coma Scale; LOC, loss of consciousness; PTA, post-traumatic amnesia.

*Risk factors are shown in table 16.2.

Admission GCS

The risk of intracranial complications and the need for neurosurgical interventions are inversely related to the admission GCS (Gomez *et al.*, 1996; Culotta *et al.*, 1996). Reported rates depend on the definition of neurosurgical intervention and vary between 0.6 and 13% in patients with an admission GCS of 15 to 25–37.5% in patients with an admission GCS of 13 (Teasdale *et al.*, 1990; Stein and Ross, 1992; Stein *et al.*, 1995; Gomez *et al.*, 1996; Culotta *et al.*, 1996; Dunham *et al.*, 1996; Haydel *et al.*, 2000; Steill *et al.*, 2001; af Geijerstam and Britton, 2003). A meta-analysis in patients with a GCS=15 found a complication rate (CI), defined as neurosurgical procedure, medical treatment of brain oedema, start of intracranial pressure monitoring or transfer to intensive care, of 0.9 (0.6–1.2)% (5). The time between the accident and hospital admission can influence the GCS (Jennett, 1996; Haydel *et al.*, 2000; Stiell *et al.*, 2001). The GCS is also the most frequently used scoring tool in children; however, it is less appropriate for very young children whose motor and verbal skills are not yet fully developed, and for this reason alternative scales have been developed (Reilly *et al.*, 1988; Simpson *et al.*, 1991; Durham *et al.*, 2000).

Duration of loss of consciousness

Verification of whether LOC has occurred and assessment of the duration of LOC are essential because LOC increases the risk of intracranial complications (EL = Class III) (Teasdale *et al.*, 1990; Stein and Spettell, 1995; Gomez *et al.*, 1996).

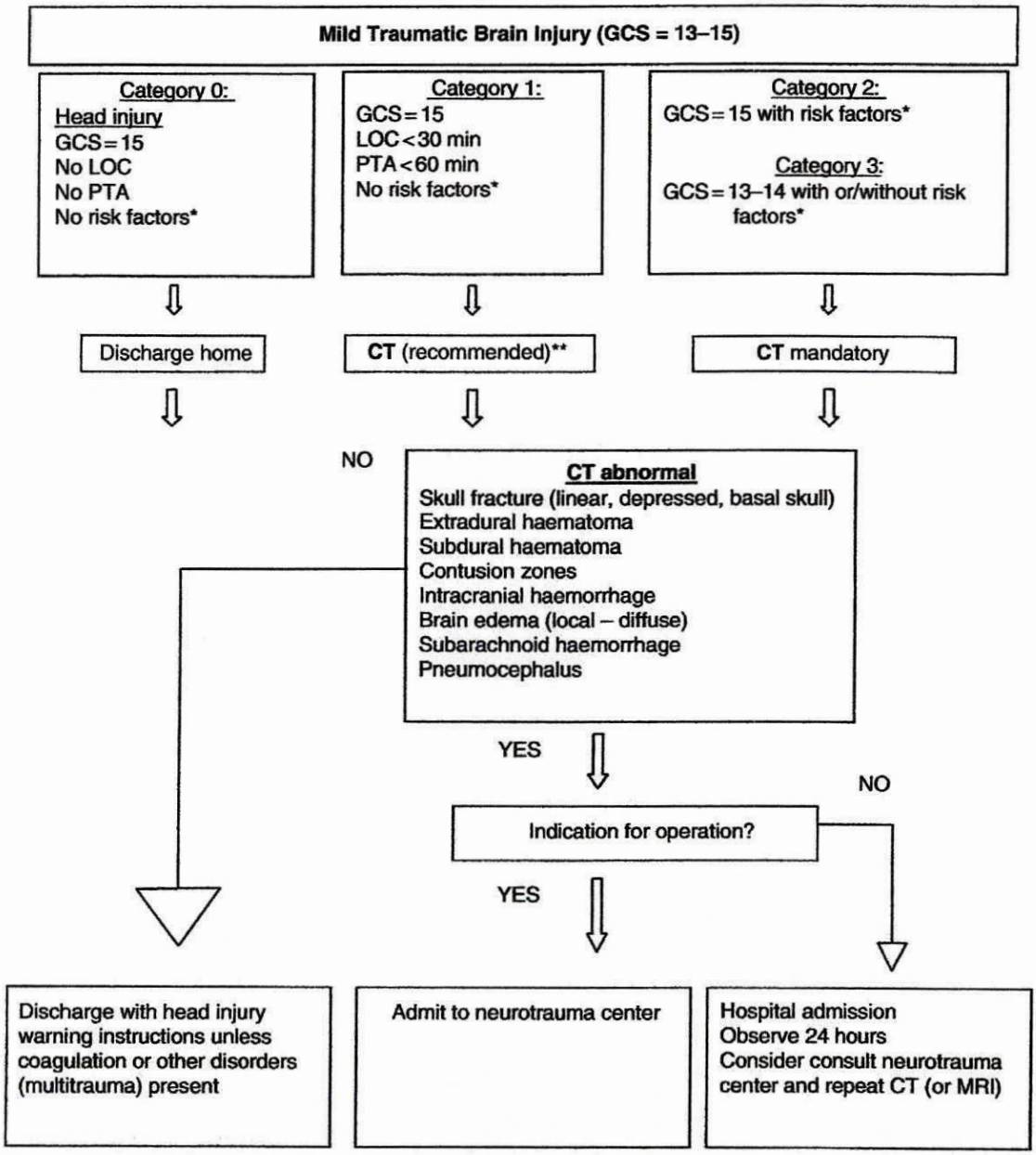

Figure 16.1. Decision scheme for initial management in mild traumatic brain injury (modified from the Dutch and Scandinavian guidelines) (Ingebrigtsen *et al.*, 2000; Twijnstra *et al.*, 2001)
GCS, Glasgow Coma Scale; LOC, loss of consciousness; PTA, post-traumatic amnesia; TBI, traumatic brain injury; CT, computed tomography; MRI, magnetic resonance imaging. *Risk factors are shown in table 16.2. **If CT availability is limited, conventional skull radiography can be performed but the sensitivity and specificity for intracranial abnormalities is unacceptably low.

Compatible with a diagnosis of MTBI are LOC duration times of 5–30 min (Rimel *et al.*, 1981; Williams *et al.*, 1990; Evans, 1992; Hahn and McLone, 1993; Mild Traumatic Brain Injury Committee, 1993; Gomez *et al.*, 1996; Jennett, 1996; Haydel *et al.*, 2000; Ingebrigsten *et al.*, 2000). Although LOC increases the risk of intracranial complications, long-term outcome is not necessarily adversely affected by a short period of LOC. In children, a 100% good outcome was

found if LOC was less than 15 min (Hahn and McLeone, 1993) (EL = II). Also, the number of post-traumatic subjective complaints, neurocognitive performance, and pre-existing emotional risk factors does not correlate with the duration of LOC (EL = II) (Ruff and Jurica, 1999). A duration of altered consciousness of less than 15–30 min can be considered as mild (EL = IV) (Jennett, 1996).

RECOMMENDATIONS

A duration of LOC of 30 minutes maximum is considered compatible with MTBI (Level B).

PTA

Post-traumatic (or anterograde) amnesia is the period of inability to lay down continuous memories (amnesic for ongoing events) and is often characterised by confusion (Levin *et al.*, 1979; Tate *et al.*, 2000). A distinction is usually made between disorientation and amnesia because the two do not always disappear at the same time (Tate *et al.*, 2000). Retrograde amnesia is the loss of memory for the period before the accident.

Failure to reach GCS 15 within 2 h post injury and deficits in short-term memory increase the risk of intracranial complications (table 16.2) (Haydel *et al.*, 2000; Stiell *et al.*, 2001). Retrograde amnesia >30 min increases the risk for neurological intervention (Stiell *et al.*, 2001).

RECOMMENDATIONS

If the duration of LOC is maximally 30 min and PTA is less than 1 h, outcome is considered good (mortality 0.1%) especially in the absence of risk factors (Level B).

Of note is that the presence of PTA is synonymous with a GCS of 14 in patients with an otherwise normal GCS score.

Risk factors

Several symptoms, signs and risk factors associated with an increased risk of intracranial injury have been identified (EL = I-III) (See table 16.2 for overview) (Masters *et al.*, 1987; Chan *et al.*, 1990; Arienta *et al.*, 1997; Haydel *et al.*, 2000; Stiell *et al.*, 2001).

RECOMMENDATIONS

Recognition of risk factors is important and such factors should be included in a classification system to further assess the risk of immediate complications (intracranial haemorrhage) (Level B).

Complications

Intracranial abnormalities

Post-traumatic intracerebral complications can be divided into: (1) Intracranial (mass) lesions, that is, abnormalities that (often) need neurosurgical intervention (extracerebral haematoma, depressed skull fracture, growing skull fracture, secondary haemorrhagic contusion, subdural effusions, malignant brain oedema with diffuse brain swelling). (2) Intracranial lesions that are treated conservatively (contusion zones, brain oedema, diffuse axonal injury, small haemorrhages, traumatic subarachnoid haemorrhage, pneumocephalus) (Teasdale *et al.*, 1990; Lloyd *et al.*, 1997; Ingebrigsten *et al.*, 2000).

Computerised Tomography (CT) is very sensitive in the detection of extracerebral haematoma and other intracranial abnormalities and is the gold standard in imaging acute TBI, although no formal CT classification for MTBI exists. The incidence of intracranial abnormalities varies with the definitions used, the clinical inclusion criteria, and the radiography method used (Stein *et al.*, 1995; Culotta *et al.*, 1996). Normal CT findings may predict the absence of late disease progression. The negative predictive value of a normal CT for neurosurgical intervention was 100% in a study involving 2032 patients and 99.7% in a study of 2124 patients (EL = Class II) (Dunham *et al.*, 1996; Livingston *et al.*, 2000).

Table 16.2 Risk factors for intracranial complications after MTBI.

Reference	Risk factor	Level of evidence	N	Follow up	% CT	Endpoint
	Glasgow Coma Scale Score					
Stiell *et al.*, 2001	GCS < 15 at 2 h after injury	II	3121	14 day telephone interview	67	[1]Neurosurgery, CT
	Continued post-traumatic amnesia*					
Haydel *et al.*, 2000	Persistent anterograde amnesia	I	1429	Discharge	100	CT
	Retrograde amnesia longer than 30 min					
Stiell *et al.*, 2001	Amnesia before impact	II	3121	14 day telephone interview	67	[1]Neurosurgery, CT
	Trauma above the clavicles including clinical signs of skull fracture					
	(skull base- or depressed skull fracture)					
Haydel *et al.*, 2000	Any external evidence of injury including contusions, abrasions, lacerations, deformities, and signs of facial or skull fracture	I	1429	Discharge	100	CT
Borczuk 1995	Cranial or facial injury	III	1448	—	100	CT
Madden *et al.*, 1995	Facial injury- signs of basilar skull fracture, depressed skull fracture	II	813	—	100	CT
Miller *et al.*, 1997	Signs of depressed skull fracture	I	2143	Discharge	100	[2]Neurosurgery, CT
Jeret *et al.*, 1993	Signs of basilar skull fracture	II	712	Discharge	100	[3]Neurosurgery, CT
Stiell *et al.*, 2001	Signs of basal skull fracture or depressed skull fracture	II	3121	14 day telephone interview	67	[1]Neurosurgery, CT
Dunham *et al.*, 1996	Cranial soft tissue injury	II	2252	?	91.3	CT
	Headache					
Haydel *et al.*, 2000	Any	I	1429	Discharge	100	CT
Miller *et al.*, 1997	Severe headache	I	2143	Discharge	100	[2]Neurosurgery, CT
	Nausea					
Miller *et al.*, 1997	n.d.	I	2143	Discharge	100	[2]Neurosurgery, CT
	Vomiting					
Miller *et al.*, 1997	n.d	I	2143	Discharge	100	[2]Neurosurgery, CT
Haydel *et al.*, 2000	Any	I	1429	Discharge	100	CT
Stiell *et al.*, 2001	≥2 times	II	3121	14 day telephone interview	67	[1]Neurosurgery, CT
	Focal neurological deficit					
Gomez *et al.*, 1996	Cranial nerve deficit, motor deficit, dysphasia	III	2484	—	7.5	[4]Neurosurgery, CT
	Seizure					
Haydel *et al.*, 2000	Suspected or witnessed seizure after the event	I	1429	Discharge	100	CT

Table 16.2 Contd.

Reference	Risk factor	Level of evidence	N	Follow up	% CT	Endpoint
	Age					
Masters *et al.*, 1987	<2 years	IV	7035	National Health Statistics	?	Intracranial injury
Haydel *et al.*, 2000	>60 years	I	1429	Discharge	100	CT
Stiell *et al.*, 2001	>65 years	II	3121	14 day telephone interview	67	[1]Neurosurgery, CT
Gomez *et al.*, 1996	Linear	IV	2484	—	7.5	[4]Neurosurgery, CT
	Coagulation disorders					
Fabbri *et al.*, 2004	Warfarin	III	501	instructions	100	CT
Li *et al.*, 2001	Warfarin	IV	144	—	100	CT
	High-energy accident**					
Stiell *et al.*, 2001	Dangerous mechanism of injury	II	3121	14 day telephone interview	67	[1]Neurosurgery, CT
Jeret *et al.*, 1993	Pedestrian hit by car or victim of assault	II	712	Discharge	100	[3]Neurosurgery, CT
	Intoxication with alcohol/drugs					
Haydel *et al.*, 2000	Alcohol or drugs	I	1429	Discharge	100	CT

*Continued post-traumatic amnesia may be interpreted as a GCS verbal reaction of 4 and hence be defined as GCS <15.
**According to Advanced Trauma Life Support principles, a high-energy (vehicle) accident is defined as initial speed >64 km/h, major auto-deformity, intrusion into passenger compartment >30 cm, extrication time from vehicle >20 min, falls >6 m, roll over, auto–pedestrian accidents, or motor cycle crash >32 km/h or with separation of rider and bike (American College of Surgeons, 1997; Bartlett *et al.*, 1998). Neurosurgery defined as: [1]death within 7 days, craniotomy, elevation of skull fracture, intracranial pressure monitoring or intubation for head injury; [2]craniotomy, or placing of monitoring bolt; [3]death or craniotomy; [4]craniotomy, elevation of depressed skull fracture, ICP monitoring.

RECOMMENDATIONS

CT is the gold standard for the detection of intracranial abnormalities (Level B). The term post-traumatic intracranial complication includes all extracerebral, and intracerebral abnormalities in relation to head trauma that can be visualized on CT and that are likely to be the result of the head trauma (Level C).

Neurosurgical intervention

Absolute indications for emergency decompressive neurosurgical intervention are signs and symptoms of an existing or rapidly developing intracranial mass lesion including deterioration of consciousness, functional motor impairment and brain stem compression signs. Intracranial haemorrhage (extradural or subdural) often needing quick neurosurgical intervention occurs in 0.2–3.1 % (Stein and Ross, 1992; Shackford *et al.*, 1992; Borczuk, 1995; Culotta *et al.*, 1996; Dunham *et al.*, 1996; Hsiang *et al.*, 1997; Haydel *et al.*, 2000; Steill *et al.*, 2001; af Geijerstam and Britton, 2003).

The mortality of MTBI, after systemic (multiple) injuries are excluded, is very low and is almost exclusively caused by the late or missed diagnosis of deterioration in patients with an intracranial haemorrhage (specifically an

extradural haematoma) (EL = II-III) (Mendelow *et al.*, 1979; Klauber *et al.*, 1989; Shackford *et al.*, 1992; Culotta *et al.*, 1996; Dunham *et al.*, 1996; Gomez *et al.*, 1996; Jennett, 1996; Mendelow and Bartlett, 1998; Servadei *et al.*, 2001; Stiell *et al.*, 2001). The prognosis of extradural haematoma is good, especially when it is detected early in fully conscious patients and surgery is performed as soon as possible (Paterniti *et al.*, 1994; Servadei *et al.*, 1995; Servadei, 1997). However, when rapid neurological deterioration occurs or when patients are already in coma, mortality rises sharply with the delay between deterioration and surgery (EL = III) (Mendelow *et al.*, 1979; Seelig *et al.*, 1984; Servadei, 1997).

Growing skull fractures are rare (frequency 0.05–0.6%). It is most likely to occur in children younger than 6 years old, when a dural tear beneath a skull fracture is present. Neurosurgical treatment is mandatory when systolic–diastolic pulsations result in widening of the fracture margins and interposition of leptomeninges or brain tissue into the fracture. It is mentioned here as a long-term complication that occurs if early diagnosis and intervention are deferred.

RECOMMENDATIONS

The primary goal of initial management in MTBI is to identify the patients at risk of intracranial abnormalities and especially those that may need neurosurgical intervention. Use of a clinical decision scheme based on risk factors may facilitate this process (Level B) (see figure 16.1).

The extracerebral haemorrhage (extradural haematoma) is potentially the most threatening complication after MTBI (Level B). An extradural haematoma can be easily identified with CT, which should be carried out urgently (Level B).

Seizures

Patients with MTBI have only a slightly increased risk of developing post-traumatic seizures including early post-traumatic seizures (= a seizure occurring in the first week) (Schierhout and Roberts, 1997; Annegers *et al.*, 1998). Prophylactic antiepileptic treatment is not warranted. A systematic review of randomised controlled trials including 2036 patients showed that prophylactic antiepileptic treatment did not reduce mortality, neurological disability, or late seizures (EL = I) (Schierhout and Roberts, 1998). If recurrent seizures occur, treatment is probably necessary and alternative explanations (i.e. delayed haematoma, Wernicke-Korsakoff syndrome, alcohol withdrawal or electrolyte disturbances) should be taken into account.

RECOMMENDATIONS

Prophylactic antiepileptic treatment is not indicated (Level A).

Skull base fracture

A skull base or temporal bone fracture or open fracture increases the risk of cerebrospinal fluid (CSF) leakage and CSF fistula formation (Dagi *et al.*, 1983; Brodie, 1997; Brodie and Thompson, 1997). The reported incidence of CSF leakage after basal skull fracture varies from approximately 10 to 20%, and the incidence of bacterial meningitis from 2 to 50% (Leech and Paterson, 1973; Dagi *et al.*, 1983; Helling *et al.*, 1988; Marion, 1991; Brodie, 1997; Brodie and Thompson, 1997). The role of antibiotic prophylaxis in open or basilar skull fractures remains controversial; the conclusions of two recent meta-analysis on the prophylactic use of antibiotics were contradictory (Demetriades *et al.*, 1992; Working Party of the British Society for Antimicrobial Chemotherapy, 1994; Brodie, 1997; Villalobos, 1998).

RECOMMENDATIONS

There is insufficient proof for prophylactic antibiotic treatment against meningitis in patients with clinical signs of a skull base fracture (Level C).

Patients on anticoagulation

No randomised clinical trials exist on the discontinuation of anticoagulation therapy after head injury. The scarce available literature shows conflicting results. Post hoc analysis of a large Italian cohort with mild head injury showed increased odds of coagulopathy for intracranial abnormalities as shown with CT (Fabbri *et al.*, 2004). Retrospective studies concluded that elderly patients on warfarin may have an increased risk for intracranial haemorrhage as well as equal morbidity and mortality after head injury (Kennedy *et al.*, 2000; Karni *et al.*, 2001).

The question what to do in a patient under anticoagulation for a cardiovascular cause and with an intracranial haematoma is hence not easy to answer. The indications for antiocoagulation and the underlying cardiovascular disease should be reviewed. A retrospective study in 39 patients with intracranial haemorrhage and mechanical heart valves without previous evidence of systemic embolization revealed that discontinuation of warfarin therapy for 1 to 2 weeks had a low probability of embolic events (Wijdicks *et al.*, 1998). It is uncertain however if these findings are applicable to head injury patients on anticoagulation therapy.

GOOD PRACTICE POINTS

All patients with head injury should be questioned about the use of anticoagulation therapy (Level C). All patients with head injury on anticoagulation therapy should have their international normalized ratio (INR) checked and the indication for anticoagulation reviewed (Level C). These patients should be admitted for neurological observation (Level C) (Saab *et al.*, 1996). If CT demonstrates an intracranial haematoma, the INR should be corrected immediately. (Over-) anticoagulation can be best corrected with fresh frozen plasma and vitamin K. If spontaneous coagulation disorders or additional injuries with bleeding exist consultation of a coagulation specialist should be sought (Good Practice Point).

Ancillary investigations

Skull radiography versus CT

The diagnostic value of plain skull radiography(Masters *et al.*, 1987; Teasdale *et al.*, 1990; Mendelow *et al.* 1983; Borczuk 1995; Mendelow and Bartlett 1998; Nee *et al.* 1999) is now considered insufficient to demonstrate intracranial complications (Masters *et al.*, 1987; Teasdale *et al.*, 1990). Mendelow *et al.* (1983), Borczuk (1995), Mendelow and Bartlett (1998) and Nee *et al.* (1999). showed that sSkull radiography is of little value in the initial assessment of MTBI (EL = I) (Hofman *et al.*, 2000). A meta-analysis of 13 studies, in which at least 50a CT of the brain, showed that the estimated sensitivity of radiographic evidence of skull fracture for a diagnosis of intracranial haemorrhage was only 0.38 with a corresponding specificity of 0.95 (EL = 1) (Hofman *et al.*, 2000).

RECOMMENDATIONS

Skull radiography is of insufficient value in the detection of intracranial abnormalities in patients with MTBI (Level A).

Clinical decision rules for CT

Two large prospective studies investigated a clinical decision rule for use of CT to demonstrate the need for neurosurgical intervention or clinically important brain injury after MTBI (Haydel *et al.*, 2000; Stiell *et al.*, 2001). In a North-American prospective study involving 1429 patients with minor head injury (defined as LOC and an admission GCS of 15), seven predictors (headache, vomiting, seizure, PTA, trauma above the clavicles, drug or alcohol intoxication, or age over 60 years) were retrieved after Chi-square analysis and determination of likelihood ratios for each criterion. This model showed 100% (95% CI: 95–100%) sensitivity for intracranial complications.

In a Canadian prospective multicentre study involving 3121 patients with minor head injury (defined as blunt trauma with LOC and/or amnesia or disorientation and initial ED GCS = 13–15),

250 patients (8%) had clinically important brain injury and 31(1%) required neurosurgical intervention. Five high-risk factors (failure to reach GCS of 15 within 2 h, suspected open skull fracture, any signs of basal skull fracture, vomiting >2 episodes, or age >65 years) were derived which had 100% sensitivity (95% CI: 92–100%) for predicting the need for neurosurgical intervention (Stiell *et al.*, 2001). Interestingly, this would lead to a CT ordering proportion of 32%. In addition, two medium-risk factors (amnesia before impact >30 min and dangerous mechanism of injury) were 98.4% sensitive (95% CI 96–99%) and 49.6% specific for predicting clinically important brain damage. This would lead to a CT ordering proportion of 54%. Both studies concluded that in patients with MTBI the use of CT can be safely limited to those who have certain clinical findings (Haydel *et al.*, 2000; Stiell *et al.*, 2001). However the generalisability of existing guidelines has been questioned. In an independent sample of 1101 patients, the reliability (detection of intracranial abnormalities) of 11 existing guidelines was lower than described in the original studies (Ibanez *et al.*, 2004).

RECOMMENDATIONS

CT is a gold standard for the detection of life-threatening (and other intracranial) abnormalities after MTBI and is recommended in those with documented LOC and/or PTA and is considered mandatory in all patients with certain clinical findings (GCS = 13–14, or GCS = 15 in the presence of risk factors) (Level B).

Clinical decisions for MRI

Cerebral MRI is not routinely used in TBI. The relationship between intracranial abnormalities on MRI and outcome is not entirely clear, and more research is needed (Voller *et al.*, 2001). When early MRI (within 21 days from the injury) and late MRI (between 5 and 18 months) findings were compared in patients with mild, moderate or severe TBI, measures of neuropsychological outcome correlated with late MRI findings only (Wilson *et al.*, 1988).

RECOMMENDATIONS

MRI may be of value for the detection of structural brain damage in patients without CT abnormalities, and especially in those with long-term complaints (Level B).

PET and SPECT-examination

Positron emission tomography (PET) and technetium ^{99m}-hexa-methylpropyleneamineoxime SPECT may show abnormalities in the acute and chronic stages when CT or MRI and neurological examination do not show damage (Ichise *et al.*, 1994; Jacobs *et al.*, 1994; Ruff *et al.*, 1994). Normal SPECT findings within 1–4 weeks after mild and moderate TBI predicted good outcome after 1 year, with a negative predictive value of 97% (Jacobs *et al.*, 1994). The specificity of abnormal findings, however, has been questioned (Alexander, 1998). Similar patterns of hypometabolism in the frontopolar and lateral temporal cortices and the basal ganglia have been reported among patients with depression but no injury (Mayberg, 1994; Dolan *et al.*, 1994).

RECOMMENDATIONS

No recommendations for the use of PET or SPECT in the initial phase after MTBI can be given at present.

Biochemical markers of traumatic brain injury

Brain-specific proteins, in particular S100β and neuron-specific enolase, may be released into the circulation after TBI. Serum levels of S100β are higher in patients with intracranial pathology and correlate with clinical outcome and the severity of primary and secondary brain damage (EL = II)

(Raabe *et al.*, 1999; Romner *et al.*, 2000). Undetectable or normal serum levels of S100β are predictive of normal intracranial findings on CT, and thus S100β could be used to select patients for CT after MTBI (Class II) (Romner *et al.*, 2000; Biberthaler *et al.*, 2004). These results have to be confirmed in large prospective studies. Although this finding has already been questioned: normal serum S100β levels may be present after epidural haematoma (Unden *et al.*, 2005). In the future, this may be of relevance in the medical–legal context to prove that the acute symptoms and signs and/or the long-term disability or neuropsychological impairments after MTBI are indeed a consequence of structural brain damage or of psychological stress in reaction to the event, alcohol intoxication, pre-existent disorders, systemic injury, or other causes (see also Romner *et al.*, 2000).

RECOMMENDATIONS

The study of biochemical markers of MTBI is of considerable interest (especially the negative predictive value of normal serum concentrations for the absence of intracranial abnormalities), but at present no recommendations can be given and more research is needed (Level B).

Initial patient management

According to the Advanced Trauma Life Support (ATLS) guidelines, any patient with trauma should be evaluated for surgical trauma (EL = III) (American College of Surgeons, 1997). Proper triage includes assessing the airways, breathing, and circulation, and the cervical spine. A neurological examination is obligatory and should include level of consciousness, presence of anterograde or retrograde amnesia and disorientation, higher cognitive functions, presence of focal neurological deficit (asymmetrical motor reactions or reflexes, unilateral paresis or cranial nerve deficit), pupillary responses, blood pressure, and pulse rate (Valadka and Narayan, 1996; Ingebrigsten *et al.*, 2000; Tate *et al.*, 2000). In addition, the presence of frontal lobe signs, cerebellar symptoms, or sensory deficits should be actively investigated.

GOOD PRACTICE POINTS

Following acute TBI all patients should undergo urgent neurological examination, in addition to a surgical examination. Furthermore, accurate history taking (including medication), preferably with information being obtained from a witness of the accident or personnel involved in first-aid procedures outside the hospital, is important to ascertain the circumstances (mechanism of injury) under which the accident took place and to assess the duration of LOC and amnesia (Good Practice Point).

An algorithm for the initial management of MTBI is given in figure 16.1.

RECOMMENDATIONS

Hospitals should have a protocol for resuscitation and triage of patients with MTBI (Level C). Category 2 and 3 patients should be admitted to a neurotrauma centre. All children with MTBI should be seen by a paediatrician or a child neurologist (Level C). CT is recommended for category 1 patients and is mandatory for all category 2 and 3 patients (see figure 16.1) (Level B)*. If CT findings are normal, adult category 1 patients can be discharged and head injury warning instructions should be given to the patient and family members. Compliance is greater if both verbal and written instructions are given (EL = III) (de Louw *et al.*, 1994; Valadka and Narayan, 1996; Ingebrigsten *et al.*, 2000). A repeat CT should be considered if the admission CT findings were abnormal or if risk factors are present (table 16.2) (Level C).

**Note: If CT availability is limited, conventional skull radiography can be performed but because of low sensitivity and specificity for intracranial abnormalities it is insufficient for patient management.*

Clinical observation

Another issue is the necessity for and duration of neurological observation after MTBI. Patients

in category 1 can be discharged to home with head injury warning instructions if CT findings are normal (Appendix available on the web site of the EFNS: http://www.efns.org/) (Warren and Kissoon, 1989; Ward *et al.*, 1992; Valadka and Narayan, 1996). Patients in category 2 or 3 should preferably be admitted to the hospital for observation, although the necessity of this can be questioned in some patients in category 2 (e.g. patients older then 60 years of age who are not on anticoagulation therapy). Most guidelines recommend an observation period of minimally 12–24 h (Masters *et al.*, 1987; Bartlett *et al.*, 1998; American Academy of Pediatrics, 1999; Ingebrigsten *et al.*, 2000; Twijnstra *et al.*, 2001). The main goal of clinical observation is to detect, at an early stage, the development of extradural or subdural haematoma or diffuse cerebral oedema. A secondary goal is to determine the duration of PTA.

An extradural haematoma usually develops within 6 h, and thus the initial CT may be false negative when performed very early (within 1 h) (Frowein *et al.*, 1989; Smith and Miller 1991; Servadei *et al.*, 1995). Repeated neurological observation (see above) is therefore obligatory for the timely detection of clinical deterioration and other neurological deficits (such as sensory deficits, frontal lobe signs, cerebellar symptoms, etc.).

RECOMMENDATIONS

A complete neurological examination is mandatory after admission and should include assessment of the GCS. Repeat neurological examination should be carried out, its frequency being dependent on the clinical condition of the patient. The patient should be examined every 30 min and if no complications or deterioration occurs, every 1–2 h. The use of a neurological checklist may be helpful to document the neurological condition and its course. If deterioration occurs, possible intracranial causes should be evaluated with (repeated) CT (Level C).

Rules for bed rest

No randomised trials exist on the value and duration of bed rest and on the duration of sick leave after MTBI. A survey among various European hospitals showed major differences in management with regard to the ordering (and duration) of bed rest, home observation, sick leave, and follow-up examination (de Kruijk *et al.*, 2001). When patients were randomised for complete bed rest (for a period of 6 days) versus no bedrest no treatment effect was found on the number of post traumatic complaints and quality of life 6 months after the trauma (de Kruijk *et al.*, 2002). Graded resumption of activities after discharge and follow-up may beneficially influence the recovery process (EL = IV) (Alexander, 1995; Kibby and Long, 1997; Ingebrigsten *et al.*, 1998).

RECOMMENDATIONS

No recommendations can be given for the need for or duration of bed rest. Early graded resumption of activities (including return to work) is probably the best strategy (Level B).

Follow-up

It has been shown that regular specialised outpatient follow-up visits are effective in reducing social morbidity and the severity of symptoms after MTBI (Wade *et al.*, 1998). In a large randomised controlled trial, patients with a PTA shorter than 7 days who received specialist intervention had significantly less social disability and fewer post-concussion symptoms 6 months after injury than those who did not receive the service (EL = II) (Wade *et al.*, 1998).

RECOMMENDATIONS

It is recommended that all patients in MTBI category 3 who have been admitted to hospital should be seen at least once in the outpatient clinic in

the first 2 weeks after discharge (Level C) (Wade *et al.*, 1998). Patients who are discharged immediately with head injury instructions should contact their general practitioners, who can decide to refer the patient to the neurologist if complaints persist (Level C).

Conclusions

The guidelines presented in this paper stress the importance of careful neurological examination, assessment of trauma history and extensive use of CT. Moreover, the use of a clinical decision rule for CT and hospital admission after MTBI may increase the use of CT compared with other existing protocols.

CT is the preferred imaging method for MTBI even though MRI is more sensitive. As MRI becomes more widely available, it may have a greater role in the evaluation of more subtle intracranial abnormalities in patients with MTBI (Haydel *et al.*, 2000; Voller *et al.*, 2001).

Conflicts of interest

The authors declare that they have no conflict of interest regarding this Chapter.

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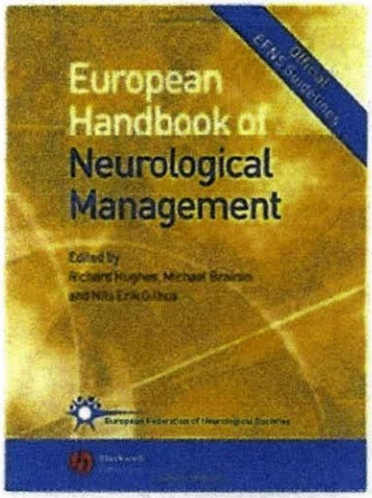

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Summary

This chapter contains section titled:

- Abstract
- Background
- Search strategy
- Mechanisms of traumatic brain injury
- Classification of mild traumatic brain injury
- Ancillary investigations
- Biochemical markers of traumatic brain injury
- Clinical observation
- Rules for bed rest
- Follow-up
- Conclusions