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DIAGNOSTIC PROCEDURES IN MILD TRAUMATIC BRAIN INJURY: RESULTS OF THE WHO COLLABORATING CENTRE TASK FORCE ON MILD TRAUMATIC BRAIN INJURY

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We examined diagnostic procedures in mild traumatic brain injury by a systematic literature search. After screening 38,806 abstracts, we critically reviewed 228 diagnostic studies and accepted 73 (32%). The estimated prevalence of intracranial CT scan abnormalities is 5% in patients presenting to hospital with a Glasgow Coma Scale score of 15 and 30% or higher in patients presenting with a score of 13. About 1% of all treated patients with mild traumatic brain injury require neurosurgical intervention. There is strong evidence that clinical factors can predict computerized tomography scan abnormalities and the need for intervention in adults, but no such evidence for mild traumatic brain injury in children. We found evidence that skull fracture is a risk factor for intracranial lesions, but the diagnostic accuracy of radiologically diagnosed skull fracture as an indication of intracranial lesions is poor. There is only a little evidence for the diagnostic validity of cognitive testing and other diagnostic tools for mild traumatic brain injury.

Key words: mild traumatic brain injury, concussion, diagnosis, computerized tomography scan, skull X-ray, loss of consciousness, post-traumatic amnesia, cognitive function.

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INTRODUCTION

The diagnostic procedure is critically important in the acute management of patients with traumatic brain injury (TBI). Firstly, it allows the attending clinician to classify the severity of the injury as mild, moderate or severe, which in turn, can determine the prognosis for the patient. Secondly, it guides the application of diagnostic tests to identify intracranial lesions that require immediate management and/or problems of cognitive function that can determine long-term recovery. In the first instance, the clinical diagnosis and classification of mild traumatic brain injury (MTBI) is usually based on the length of loss of consciousness (LOC) and/or the length of post-traumatic amnesia (PTA) and the physical and neurological examination. These, in turn, determine whether or not the attending clinician will order more advanced diagnostic tests, such as skull radiographs, computed tomographic (CT) scans, magnetic resonance imaging (MRI) of the brain or other tests. With respect to the diagnostic classification of TBI, it is generally accepted that patients with MTBI have no more than 30 minutes of LOC and/or 24 hours of PTA (1). If the Glasgow Coma Scale (GCS) is used, MTBI falls within the range 13–15, with the majority scoring 15. There is some debate about the proper classification of patients with GCS 13–14 and those with neurological deficit(s) due to intracranial lesion(s). Although falling within the mild category according to the GCS score, their outcomes can be as poor as moderate cases. Carroll et al. (2) address some of these issues of clinical definition and classification of MTBI elsewhere in this supplement. However, this paper deals with the scientific evidence for the application of diagnostic tests to identify important lesions or deficits in patients already classified as MTBI, rather than dealing with the case definition of MTBI *per se*.

Diagnostic testing is often taken to mean laboratory tests, but it can and does also include information taken from the clinical history, the physical and neurological examination, neuropsychological testing and diagnostic imaging. Regardless of the source of information, to be useful, a diagnostic test must yield valid information, that is, it must be relatively free of random error (good reliability) and systematic error (free of bias). Although reliability (i.e. repeatability as tested by intra- and inter-rater reliability) is a necessary step in the validation of a diagnostic test, it is not sufficient to establish validity. A reliable test may still have too much systematic error to be useful. Recently Sackett & Haynes (3) (Table I) have established 4 levels or phases of validation studies for diagnostic tests that help to differentiate exploratory or hypothesis-generating studies of diagnostic criteria from confirmatory or hypothesis-testing studies that establish the clinical validity of diagnostic tests. We have found this classification of diagnostic evidence useful, and we use it here to guide our decisions about the recommendation of diagnostic tests in patients with MTBI.

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Table I. *Phases of diagnostic test designs (Based on Sackett & Hayes (3))*

Phase	Research question	Inclusion criteria	Independence[1] and Blinding[2]	
I	Do test results in patients with the target disorder differ from those in normal people?	Sample subjects from normal controls and from patients with the disorder	No	Exploratory
II	Are patients with positive test results more likely to have the disorder than patients with negative test results?	Sample subjects from normal controls and from patients with the disorder	No	Exploratory
III	Do test results distinguish patients with/without disorder?	Representative sample of patients suspected to have the disorder[3]	Yes	Confirmatory
IV	Does the application of the test improve health outcomes?	Representative sample of patients suspected to have the disorder[3]	Yes	Confirmatory and therapeutic

[1]The reference (gold) standard has been applied regardless of the result of the diagnostic test.
[2]The reference (gold) standard has been applied and interpreted in total ignorance of the diagnostic test result and vice versa.
[3]All study patients have undergone both the diagnostic test and the reference (gold) standard evaluation.

Specifically, phase I and II studies do not support the recommendation of a test in the clinical setting because of the selection of subjects into the study (selection bias) and the lack of independence and blinding in the assessment of the results (information bias). They can suggest that further studies be recommended (i.e. phase III and IV studies) to confirm the validity of the diagnostic test in a representative sample of subjects with independence and blinding of the test application and interpretation. In keeping with these principles, a diagnostic procedure or test must be supported by at least phase III evidence to be recommended in clinical practice.

Two other issues are important in deciding which diagnostic tests to order in patients with MTBI and how to interpret the results. For example, in the early assessment of patients with MTBI, the examining clinician might be concerned with detecting an intracranial lesion. If so, he or she would want to apply diagnostic criteria (from the history and/or physical examination) that are sensitive enough to detect patients who require further studies, such as advanced imagining. Then, in determining the type of imaging to order, the clinician would want a test sensitive enough to detect any intracranial lesion that might be present. A sensitive test is usually positive in the presence of the target disease or condition and should be chosen when there is reason to suspect a dangerous, but treatable lesion (e.g. intracranial bleeding). It is defined as the probability of a positive test in those with the target disorder, and a highly sensitive test will rarely miss the disorder (Table II). In this respect, a sensitive test is most helpful to the clinician when it is negative because it "rules out" the condition. Sensitivity is a diagnostic test property that guides the decision of whether or not to do the test. However, once the test is done, whether positive or negative, sensitivity is no longer relevant because the value is based on past data collected from those known to have the condition. Therefore, once the test is done, a second diagnostic test property, known as the negative predictive value (−PV) (3), becomes important for the interpretation of the results (Table II). The −PV is defined as the probability of not having the target condition when the test result is negative. This issue is important in the triage of patients with MTBI suspected of having an intracranial lesion. A negative test might guide the decision not to pursue further testing (e.g. a CT scan), or to discharge the patients from the emergency room, rather than admit them to hospital for a neurosurgical consultation or further observation. In this paper we have emphasized the importance of these values in interpreting the results of diagnostic studies.

The task force performed a systematic search of the literature on MTBI as outlined in detail elsewhere (4). In this paper, the aims are to report the evidence found on diagnostic procedures related to the acute management of patients with MTBI, to make evidence-based recommendations and to identify important areas for further research.

METHODS

Search strategies and evaluation procedures are described in detail elsewhere in this supplement (4). Briefly, we performed a comprehensive, systematic search of the world literature on MTBI. Medline and PsycINFO where searched from 1980 to 2000, Cinhal from 1982 to 2000 and Embase from 1988 to 2000. All languages were included. Indexed thesaurus terms (e.g. Medical Subject Headings for Medline) and text words, such as concussion, mild brain/head injury and others were used, to ensure that all relevant articles were captured. The retrieved abstracts were screened for relevancy to the mandate of the task force (5) by applying our inclusion/exclusion criteria. Only articles that contained data on more than 10 subjects with MTBI were included, with the exception of papers reporting rare complications such as delayed, severe intracranial complications and second impact syndrome. We did not consider studies using animals, biomechanical simulations or cadavers.

The published papers of relevant abstracts were screened to ensure the study met our inclusion criteria. Relevant papers were then reviewed in detail for methodological quality using *a priori* criteria for scientific acceptance. We carefully considered the merits and biases of each paper separately, and our final decision on the scientific admissibility was made by informed group consensus.

Table II. *Calculating sensitivity (Se) and negative predictive value (−PV) of a diagnostic test*

		Disease/Condition		
		Present	Absent	
TEST	Positive	a	b	
	Negative	c	d	−PV = d/c+d
		Se = a/a+c		

We also screened the reference lists of all reviewed papers to identify additional studies that might be relevant. These would include studies published prior to 1980 and literature not indexed in the electronic databases that we searched. We also solicited papers from experts in the field, brain injury associations, and other sources such as Internet sites and professional associations. In the spring of 2002, we screened Medline one last time, but reviewed only studies that were relevant and of high impact. These included randomized clinical trials of interventions, large well-designed cohort and case-control studies and other studies that addressed gaps in the knowledge on MTBI. We also included high impact studies published in 2002 that came to our attention, but we did not undertake a systematic search (4).

In this report, we focus on the papers that address any aspect of the diagnosis of MTBI. To better delineate the strength of the evidence on diagnosis for MTBI, we used the method given by Sackett & Haynes (3). Data from accepted papers were extracted into evidence Tables that summarize our findings and form the basis of our recommendations. Our evidence tables contain diagnostic data, as reported in the results of the original study, or as calculated by us from the raw data presented in each study using Statsdirect (6).

RESULTS

We accepted 73 studies, or 32% of the 228 articles identified in this area. Of the studies that comprise our best-evidence synthesis, 44 address radiological examinations, 7 address tests of cognitive function and 22 address various other diagnostic tools.

Studies related to CT scan

Of the 44 accepted radiographic studies, 29 addressed the use of CT scans in patients with MTBI (7–35). The evidence shows that CT can detect unsuspected intracranial lesions in patients with MTBI. However, the use of CT scan examination is variable, as illustrated by 2 recent studies showing large variations in use and yield of CT scan in community and teaching hospitals (32) and paediatric hospitals (21). To be useful, clinical decision rules in this area are expected to have a high sensitivity and negative predictive value, so as not to overlook any important lesion (36).

Of these 29 studies using CT scans in MTBI, 17 are cohort studies, 1 is a cross-sectional study and 6 are case series. Only 2 of these had a phase III design. Five were systematic reviews.

What is the prevalence of intracranial abnormalities by CT scan, need for surgical intervention and death?

In order to develop and validate diagnostic tools for the detection of clinically important MTBI complications, knowledge about the prevalence of these complications is required. Extracted data on frequencies of MTBI complications from 26 studies are summarized in Table III. The accepted studies show that the frequency of CT scan abnormalities, need for surgery, and deaths in MTBI vary with study design, inclusion/exclusion criteria and setting. Some studies are retrospective and CT scan examination was not always done according to a fixed protocol, but according to various clinical indications. Thus, the reported frequencies of CT scan abnormalities are likely falsely high due to the selection of patients with more severe injuries (i.e. confounding by indication). Frequencies might also differ due to inclusion or exclusion of skull fractures, which is not explicitly stated in all studies. Furthermore, most studies do not differentiate among intracranial lesions (e.g. epidural, subdural haematoma, subarachnoidal bleeding and contusion) and only a few identify these requiring clinical interventions. Most early studies are from US Level I Trauma Centres and include transferred patients, many of whom were transferred because of the severity of their injuries. As such, there is a great potential for over-representation of patients exposed to high-energy trauma and more severe injury. Other studies include less selected samples of patients with MTBI with ordinary "concussions". All studies are restricted to hospital samples, and not all concussed patients present to hospital. All these aspects have to be considered when interpreting the frequency data in Table II. However, there are several good cohort studies yielding evidence on the prevalence of CT scan abnormalities, need of surgical intervention and death in patients with MTBI attending hospital.

Five cohort studies, with representative patient samples and independent assessment of CT scans (15, 22–24, 33), show that in patients with MTBI with a GCS score of 15, the prevalence of intracranial CT scan abnormalities is about 5%. A systematic review of studies addressing CT scans in patients with MTBI with a GCS score 15 reports a higher estimated mean prevalence of CT scan abnormalities, at about 8% (8). However, as pointed out in that report, CT scan was not performed in all patients in the included studies, and not all studies differentiated intracranial lesions and skull fractures.

Several studies show that the prevalence of CT scan abnormalities is higher in the more severely injured patients who present with a lower GCS score of 14 or 13. Four large cohort studies, which report data differentiated by GCS score 13–15, show that the prevalence of CT scan abnormalities is around 20% in patients with a GCS score of 14, and around 30% or higher in patients with a GCS score of 13 (9, 13, 33, 35). A comprehensive study of 3121 patients with MTBI at 10 Canadian community and teaching hospitals (33), reports a prevalence of clinically important intracranial lesions of 4.8% in patients presenting with a GCS score of 15, 17.2% in patients presenting with a GCS score of 14, and 40.9% in patients presenting with a GCS score of 13.

We found only 6 studies in this area that specifically address use of CT scans for children with MTBI (7, 11, 12, 21, 26, 29). These studies suggest that the prevalence of CT scan abnormalities in children with MTBI is similar to that reported in adult patients with MTBI. However, study design and inclusion criteria, as well as indications for CT scan examination, varied in these studies, and therefore, they must be interpreted with caution. Only 1 study reported data specific for elderly patients with MTBI (13). This study yields evidence that the frequency of intracranial lesions is higher in patients aged over 60 years when compared with patients aged 14–60 years.

The rate of need for surgery is low and fatal outcome is rare in patients with MTBI. Four cohort studies (9, 23, 24) demonstrate that, in patients with MTBI presenting with a GCS score of 15, the prevalence of surgical intervention is 0.5% or less. As demonstrated for CT scan abnormalities, the reported frequency

Table III. *Frequencies of computerized tomography scan, abnormalities, need for surgery and mortality by Glasgow Coma Scale (GCS) score*

Authors	Study design	Setting, Sample size (*n*) and age of subjects	Patients with LOC and/or PTA: Proportion of sample (%)	GCS	CT abnormality[1]: proportion of sample (%) by GCS	Surgery: proportion of sample (%) by GCS	Mortality: proportion of sample (%) by GCS
Britton et al., 2000 (8)	Systematic review	80 studies; $n = 14{,}175$ in 9 high-quality studies; Age: all	100	15	7.5–8.9	1.14	0.10
Schutzman et al., 2001 (26)	Systematic review	32 studies; Age: <2 years	NR	Majority 13–15	3.4–2[2]	NR	NR
Servadei et al., 2001 (27)	Systematic review	42 studies; $n = 11{,}859$ for the presence of intracranial lesion and craniotomy; Age: excluded studies restricted to children	NR	13 14 15	27–58 16–35 4–18	4.5–20.0 1.6–6.0 0.3–3.3	 NR
Borczuk, 1995 (9)	Cohort	Level 1 Trauma Center, US; $n = 1448$; Age: >16 years	100	13 14 15	27.5 18.2 5.9	7.5 3.6 0.08	NR
Davis et al., 1994 (11)	Cohort	Level 1 Trauma Center and General Children's Hospital, US; $n = 185$; Age: 2–17 years	100	15	7.6[2]	1.0	NR
Davis et al., 1995 (12)	Cohort	Level 1 Trauma Center, US; $n = 400$; Age: 0–17 years	59	13–15	18	0.25	0
Dunham et al., 1996 (13)	Cohort	Trauma Center, US; $n = 2252$; Age: 14–60 years and >60 years	100	13–15 13 14 15	NR 25.0–30.8[2,3] 12.4–28.1[2,3] 3.0–10.0[2,3]	0.3–0.5[2,3]	NR
Haydel et al., 2000 (15)	Cohort	Level 1 Trauma Center, US; $n = 1429$; Age: >2 years	100	15	6.5 (95% CI 5.2–7.7)	0.4 (95% CI 0.1–0.7)	NR
Holmes et al., 1997 (16)	Cohort	Trauma Center, US; $n = 264$; Age: NR	100	14	13.2	1.5	NR
Hsiang et al.,1997 (17)	Cohort	University Hospital, Hong Kong; $n = 1360$; Age: >10 years	NR	13 14 15	57.8 35.5 18.5	20 5.1 2.2	2.22 0.72 0.17
Klassen et al., 2000 (21)	Cohort	9 Pediatric Hospitals, Canada; $n = 1164$; Age: <17 years	100	13–15 13 14 15	NR 60 28 12	0.2	0
Livingston et al., 2000 (22)	Cohort	Level 1 Trauma Center, US; $n = 2152$; Age: >15 years	100	14–15	14.3 5.6[2]	1.8	0.14
Miller et al., 1996 (23)	Cohort	Level 1 Trauma Center, US; $n = 1382$; Age: NR	100	15	6.1 4.8[2]	0.2	NR
Miller et al., 1997 (24)	Cohort	Level 1Trauma Center, US; $n = 2143$[4]; Age: NR	100	15	6.4 5.1[2]	0.2	NR
Shackford et al., 1992 (28)	Cohort	8 Trauma Centers (Level 1 & 2), US; $n = 2766$; Age: mean 26.9 (SD 17.4) years	100	13–15	30.5 21.6[2]	4.2	0.18

Authors	Study design	Setting, Sample size (*n*) and age of subjects	Patients with LOC and/or PTA: Proportion of sample (%)	GCS	CT abnormality[1]: proportion of sample (%) by GCS	Surgery: proportion of sample (%) by GCS	Mortality: proportion of sample (%) by GCS
Stiell et al., 1997 (32)	Cohort	7 Teaching Hospitals, Canada; *n* = 1699; Age: >15 years	100	13–15	6.2 5.8[2]	3.1	0.1
Stiell et al., 2001 (33)	Cohort	10 Community and Teaching hospitals, Canada, including referrals; *n* = 3121; Age: >15 years	100	13–15 13 14 15	11 40.9[2] 17.2[2] 4.8[2]	1.4	0.13
Wang et al., 2000 (34)	Cohort	Transfers to Trauma Centers, Los Angeles County, US; *n* = 157; Age: <15 years	33	13–14[5]	27.4 19.1[2]	3.2	0
Viola et al., 2000 (35)	Cohort	Neurosurgery Department, Italy; *n* = 859; Age: >11 years	78	14–15 14 15	10.2 62.0 8.7	0.7	0.1
Simon et al., 2001 (29)	Cross sectional	Level 1 Trauma Center, US; *n* = 429; Age: <16 years	46	14 15	21.2[2] 13.5[2]	0 0.07	NR
Culotta et al., 1996 (10)	Case series	Level 1 Trauma Center, US; *n* = 3370; Age: >13 years	100	13 14 15	28[2] 16[2] 4[2]	4.5 1.6 0.4	1.1 0.01 0.01
Iverson et al., 2000 (18)	Case series	Trauma Center, US; *n* = 912; Age: mean 35.3 (SD 16.5) years	70	13–15	15.8	NR	NR
Jeret et al., 1993 (20)	Case series	Trauma Center, US; *n* = 712; Age: >17 years	100	15	9.4	0.28	0.14
Nagy et al., 1999 (25)	Case series	Level 1 Trauma Center, US; *n* = 1,170; Age: mean 33.8 (SD 12.9) years	100	15	3.3 2.1[2]	0.3	NR
Stein et al., 1990 (30)	Case series	Regional Trauma Center, US; *n* = 658; Age: NR	100	13 14 15	40.3 22.5 13.0	12.9 5.6 3.7	NR
Stein et al., 1992 (31)	Case series	Regional Trauma Center, US; *n* = 106; Age: NR	NR	13	44.3	NR	NR

[1]Abnormality could include intracranial lesion and/or skull fracture.
[2]Intracranial lesion specific.
[3]Lower value in age 14–60 years, higher value in age >60 years.
[4]Includes the sample from Miller et al., 1996.
[5]Field GCS score.
LOC = loss of consciousness; PTA = post-traumatic amnesia; GCS = Glasgow Coma Scale; CT = computerized tomography; NR = not reported; CI = confidence interval; SD = standard deviation.

of patients with a need of surgical intervention is higher when patients within the whole MTBI severity spectrum are considered. In patients presenting with a GCS score of 13–15, the prevalence of surgical intervention is about 1% (12, 13, 33). In 1 cohort study of 1448 patients with MTBI, which reports data differentiated by GCS score, the need for surgery was 0.08% in patients with a GCS score of 15, 3.6% in patients with a GCS score of 14, and 7.5% in patients with a GCS score of 13 (9). When reported, mortality rates for all MTBI is around 0.2% or less (22, 28, 33). In 1 study that reports mortality rates by GCS score, the mortality is 0.01% in patients with GCS scores of 14 or 15, and 1.1% in patients with a GCS score of 13 (10).

Can clinical variables be used to predict a CT scan abnormality and need for surgical intervention?

Nine studies examined the diagnostic value of clinical variables as predictors of CT scan abnormality or the need for surgery. Only 2 of these studies (15, 33) have a phase III design, according to the criteria of Sackett & Haynes (3), and neither phase III study addresses this issue in children (Table IV).

The phase III study by Stiell et al. (33), reports on the validity of a CT decision rule consisting of a set of clinical high-risk factors to indicate the need for neurological intervention and a set of clinical medium-risk factors to indicate the presence of a clinically important intracranial lesion on CT scanning. The study is restricted to patients aged over 15 years and presenting with a GCS score of 13–15. Primary outcome was defined as either death within 7 days, or need for any of the following within 7 days: craniotomy, elevation of depressed skull fracture, intracranial pressure monitoring, or intubation for head injury. Clinically important brain injury (secondary outcome) was defined as any acute brain finding on CT, which would normally require admission to hospital and neurological follow-up. In neurologically intact patients, the following CT findings were not considered important: solitary contusion less than 5 mm in diameter, localized subarachnoid blood less than 1 mm thick, haematoma less than 4 mm thick, isolated pneumocephaly and closed depressed skull fracture not through the inner table. Good inter-observer agreement for clinical factors and CT scan results was demonstrated. Logistic regression analysis yielded a model with good accuracy for discriminating cases with clinically important brain injury. Recursive partitioning analysis was done to decrease the number of baseline features in the decision rule. This yielded the following 5 conditions to identify high-risk patients requiring neurological intervention: a GCS score less than 15 at 2 hours after injury, suspected open or depressed skull fracture, any sign of basal skull fracture, vomiting 2 or more times, and age of 65 years or older. Two more questions stratified individuals as medium risk for clinically important intracranial lesion on CT scan: anterograde amnesia of more than 30 minutes, and dangerous injury mechanism.

The 5 high-risk factors were 100% sensitive (95% CI 92–100%) for predicting the need for neurological intervention. Using all 7 risk factors in the decision rule (that is, the so-called Canadian CT Head Rule) (33), yielded a sensitivity of 98.4% (95% CI 96–99%) and a −PV of 99.7 (95% CI 99.3–99.9%) for clinically important brain injury. Only 4 of 254 clinically important injuries on CT scan (small contusions) would have been missed and none of these needed any intervention. Cross-validation analysis indicated that using the 5 high-risk factors for decisions would have yielded a sensitivity of 92% (95% CI 88–94%) for identifying patients with any injury on CT, including unimportant injuries. Use of these clinical factors for decisions about administering CT scans would yield an estimated 25–50% reduction in head CT scanning. It should be pointed out that success of these prediction rules is dependent on the physician's ability to carry out the necessary clinical examinations.

Recently, these findings have been validated in a study of 2588 patients with MTBI at 9 tertiary care emergency departments (37). In these patients, with a mean age of 38.4 years (range 18–99 years) and with a GCS score of 13–15, clinically important brain injury on CT scan was present in 8.2% and unimportant injury in 3.6%. Neurological intervention was required by 1.6% and the mortality rate was 0.2%. The 5 high-risk factors classified patients for neurological intervention with a sensitivity of 100% (95% CI 91–100%) and would have required CT scan for 35.7%. The 7 high- and medium-risk factors classified 212 important brain injuries with a sensitivity of 100% (95% CI 98–100%) and would have required CT scan for 62.4%.

In another phase III study, Haydel et al. (15) developed (in 520 patients) and validated (in 909 patients) a set of clinical factors for predicting a positive CT scan in patients over 2 years of age and presenting with a GCS score of 15 (Table IV). A positive CT scan was defined as the presence of any of the following: subdural, epidural, or parenchymal haematoma; subarachnoid haemorrhage; cerebral contusion; and depressed skull fracture. All patients with a positive CT scan had one or more of 7 clinical findings: headache, vomiting, age over 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicles and seizure. The sensitivity of the 7 findings combined was 100% (95% CI 95–100%) and the −PV was 100% (95% CI 98–100%). It is not clear how many children were included in this study, and thus the findings cannot be generalized to children.

Both these studies (15, 33) yield good evidence that clinical factors can be used to predict acute complications in adult patients with MTBI, and the evidence from 1 study (15) has been used in recently published guidelines for the management of patients with MTBI with a GCS score of 15 (19). One major strength of the Canadian study is the use of outcome measures that have an obvious clinical relevance (33). The outcome measure used in the American study (i.e. any intracranial lesion or depressed skull fracture) would also capture clinically insignificant findings, such as minimal brain contusions, that would not alter the acute management of MTBI (15). Thus, in application, this approach would unnecessarily triage patients to CT scan or hospital observation. Nevertheless, we have taken a conservative approach by using both these studies to make our recommendations regarding acute triage for adult patients with MTBI (Fig. 1, discussed in detail on p. 74).

Table IV. *Clinical indicators for need of computerized tomography (CT) scan for diagnosis of mild traumatic brain injury, phase III studies (3)*

Study	Diagnostic tools assessed	Outcome measures	Sensitivity % (95% CI)	Specificity % (95% CI)	–PV(%), +PV(%), +LR (95% CI)
Haydel et al., 2000 (15)	In patients with GCS 15: One or more of • Headache • Vomiting • Age >60 years • Drug/alcohol intoxication • Short-term memory deficits • Physical evidence of trauma above the clavicles • Seizure	CT abnormality defined as any intracranial lesion or depressed skull fracture.	100 (95–100)[1]	25 (22–28)[1]	–PV = 100 (98–100)[1] +PV = 8.2 (6.3–10.5)[1] +LR = 1.3 (1.2–1.4)[1]
Stiell et al., 2001 (33)	In patients with GCS 13–15: 1. High-risk factors: one or more of: • GCS <15 at 2 hours post-injury • Suspected open or depressed skull fracture • Signs of basal skull fracture • Vomiting ⩾2 episodes • Age ⩾65 years	Need of neurological[1] intervention	100 (92–100)	68.7 (67–70)	–PV = 100 (99.8–100)[1] +PV = 4 (3–6)[1] +LR = 3.2 (2.9–3.3)[1]
	2. Medium risk factors: 1 or more of: • Amnesia before impact >30 minutes • Dangerous mechanism of injury, including pedestrian struck by vehicle, ejected from vehicle, and fall from >3 feet or 5 stairs.	Clinically important brain injury[3]	98.4 (96–99)	49.6 (48–51)	–PV = 99.7 (99.3–99.9)[1] +PV = 15 (13–17)[1] +LR = 2.0 (1.9–2.0)[1]

[1]Task force analysis.
[2]Need of neurological intervention is defined as death within 7 days, or need of any of the following: craniotomy, elevation of depressed skull fracture, intracranial pressure monitoring, or intubation for head injury.
[3]Clinically important brain injury is defined as any acute brain finding on CT, which would normally require admission to hospital and neurological follow-up.
CI: confidence interval; –PV = negative predictive value; +PV = positive predictive value; +LR = likelihood ratio positive test; GCS = Glasgow Coma Scale.

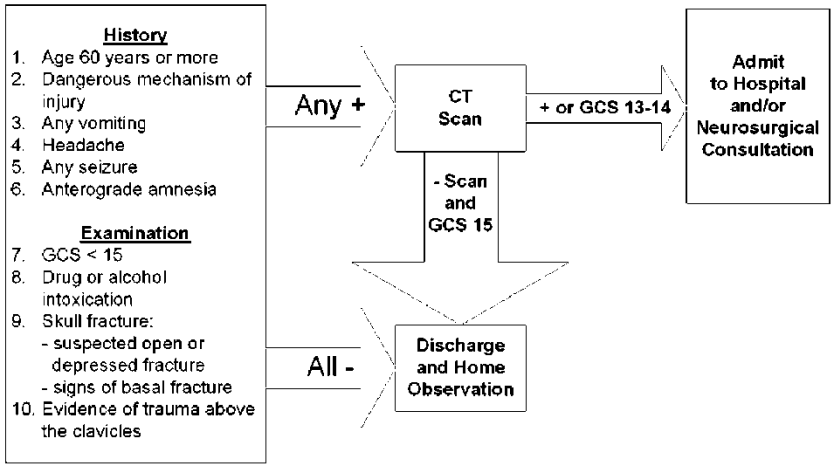

Fig. 1. Evidence-based approach to the acute diagnostic management of mild traumatic brain injury (MTBI) in adults. GCS = Glasgow Coma Scale, CT = computerized tomography.

Six studies yield phase II evidence that is consistent with the phase III studies we have just reviewed. Two studies suggest that, in patients presenting with a GCS score of 15, depressed skull fracture, vomiting, nausea and headache are good clinical predictors for intracranial lesions (23, 24). However, when these predictors were applied in another study of patients presenting with a GCS score of 14, the sensitivity was only 51% (16). Another study yields phase II evidence that age of 60 or more years and craniofacial soft-tissue injury are risk factors for intracranial haemorrhage on CT scan (13).

None of the previously discussed studies contain evidence on the predictive value of an early normal CT scan for intracranial complications in patients with clinical risk factor(s). However, approximately 73% of the patients with a normal CT scan were discharged in the study by Stiell et al. (33) (Stiell IG, personal communication). This question is addressed in 2 phase II studies (22, 28). In 1 of these, a large cohort of 2152 patients presenting with a GCS score of 14 or 15 were followed for 20 hours, or until discharge from hospital (22). The −PV of the CT scan for the need of any intervention was 99.7% (95% lower confidence limit 99.2%) and for need of craniotomy it was 99.94% (95% lower confidence limit 99.6%). However, the possible impact of other clinical findings was not considered. Another large cohort study of 2766 patients with MTBI, of whom 2166 (78%) underwent a CT scan examination, suggests an increased risk for intracranial complications and need of surgical intervention in patients with an initially abnormal CT scan or an abnormal neurological examination (28). However, CT scan examination was not performed in all patients and CT scan use was even lower in patients with a GCS score of 15. Thus, we identified no strong evidence that an early CT scan adds diagnostic information to the clinical findings from these studies.

Six studies (7, 11, 12, 26, 29, 34), including 2 phase II studies (29, 34), specifically addressed clinical prediction in children with MTBI.

One of these studies yields weak phase II evidence (11) on risk factors for intracranial bleeding on CT scan in children with MTBI aged 2–17 years. None of 49 neurologically normal children without signs of depressed or basilar skull fracture had a complicated brain injury. However, the study sample is small and no data on the reliability of the CT scan interpretation or the clinical examination are reported.

Other studies with explorative designs yield preliminary data on various other clinical risk factors for intracranial lesions in children with MTBI. One of these studies (34) reports GCS scores from the field prior to emergency room presentation. The results show that a field GCS score of 13 or 14, or a decreasing GCS score, or skull fracture are poor predictors of intracranial lesions. In another study of 400 children followed 1 month post-injury a normal initial CT scan examination predicted an uneventful clinical course (12). In 1 study, which includes a large proportion of children with no LOC or PTA, cranial soft-tissue injuries and skull fractures were associated with CT scan abnormalities (29). Also, the task force accepted 2 guidelines (7) addressing the acute management of MTBI in children (38). One of these, based on a review of the literature, reports that skull fracture, altered mental status, focal neurological findings, scalp swelling, younger age and inflicted injury are risk factors for important clinical lesions (26). The authors further report that no predictor, or combination of predictors, is 100% sensitive for identifying intracranial lesions. The search methodology and quality assessment of the reviewed studies was not detailed. Another guideline concludes, "Much remains to be learned about minor closed head injury in children (7)."

Studies related to skull fracture and use of plain skull X-ray

Of the 15 studies in this area (39–53), 7 are cohort studies, 2 are cross-sectional, 5 are clinical descriptive studies and there is 1 systematic review. No phase III studies were identified. Data from 7 of the accepted studies are summarized in Table V.

The 15 accepted studies vary with regard to study settings and also in many other respects. Not all studies use the same or specified inclusion criteria. The proportion of patients with a history of LOC or PTA varies, and some studies include patients with a "trivial" or a "low risk" head injury (patients with no LOC or amnesia). Not all studies report GCS scores, but use other clinical descriptions of injury severity such as "talk and walk". The proportion of patients who underwent radiological exam-

ination varies and the information on possible other non-cranial injuries is not routinely reported.

Frequency and clinical prediction of skull fracture

Available data from 1 cohort study (46) and 1 case series (39), indicate that the frequency of skull fractures is below 5% in treated patients with MTBI. In patients with a trivial head injury, i.e. in patients without altered consciousness or amnesia, the reported frequency of skull fracture is around 1% (46, 49, 51, 53).

Two studies yield phase II evidence on clinical risk factors for skull fracture. The frequency of skull fracture is much higher (almost 10 times) for patients with LOC (46). In 1 study, which included patients with head injuries of all severity, vomiting was an independent risk factor for skull fracture in both adults and children, but repeated vomiting did not confer a higher risk than a single episode of vomiting (50).

What is the diagnostic value of radiological skull fracture for intracranial lesions?

Several studies in the current review suggest that skull fracture is a risk factor for intracranial haematomas in both adults and children (39, 41, 42, 44–47, 52, 53), but the diagnostic accuracy of skull X-rays is questionable (51).

In a systematic review by Hofman et al. (43), the prevalence of intracranial haemorrhage (ICH) in patients with MTBI and the diagnostic value of skull fracture for ICH in these patients were examined. Only studies with more than 50 subjects and patients with a GCS score of 13–15 were included. For the prevalence study, 50% of the included patients had to have undergone a CT scan examination. When CT scan was not performed, an uneventful recovery was considered as having no ICH. For the diagnostic study, it was required that diagnostic test data were reported. No other quality assessment of the original studies was mentioned. The prevalence studies include 12,750 patients, and the diagnostic studies include 48,619 patients. There were large inter-study differences with respect to how many of the study patients had a history of LOC or PTA. The reported mean prevalence of ICH after mild head injury was 10% (range 3–18%) and the estimated sensitivity of skull fracture for ICH was 38% (range 13–75%).

In summary, there is consistent evidence from many explorative studies and 1 systematic review that the presence of radiologically diagnosed skull fracture increases the risk of intracranial lesions in patients with MTBI, as also demonstrated in phase III and phase II studies related to CT scan examination (as discussed above). However, there is also evidence from 1 systematic review that the diagnostic accuracy of radiologically demonstrated skull fracture for intracranial lesions is poor.

Delayed intracranial complications

Five studies (12, 54–56 and 1 submitted study*) address the prevalence, timing and risk factors for delayed, intracranial complications in patients with MTBI. Two studies show that the prevalence of delayed clinical deterioration with a need of surgery is low, but not negligible (54)*. In 1 cohort study, the clinical course of 1812 patients with GCS 15 and LOC or PTA of less than 30 minutes was followed (54). Twenty-eight patients (1.5%) deteriorated, 23 patients (1.3%) needed surgery and 5 had non-surgical problems. Of those 28 patients that deteriorated, 16 (57%) deteriorated within the first 24 hours. In the other 14 patients, all except 1 patient with a subdural haematoma deteriorated within 21 days after the injury. Thus, in this cohort, the frequency of severe complications occurring later than the first 24 hours after the injury is 0.8%. In a population-based, case-control study of 100,784 patients discharged from Swedish hospitals with a single diagnosis of concussion, the frequency of readmissions due to severe, intracranial complications until 3 weeks after the injury was 0.13%*. One reason for the lower rate in this study is that some patients were hospitalized for more than 24 hours after the injury, which would capture more complications.

Other studies related to delayed intracranial complications yield only preliminary data. One retrospective study of 606 patients who were discharged "walking and talking" after a head injury of varying severity showed that 34% had an intracranial abnormality on CT scan when re-attending 6 days after the injury (56). Furthermore, 16.5% required neurosurgical intervention. However, this study included patients with penetrating trauma.

In a 1-month, follow-up study of 400 children with an initially normal CT scan examination and who were followed for 1 month post-injury, 4 children were readmitted and only 1 of these had an intracranial lesion (a subdural haematoma) requiring surgical intervention (12). In 1 case series of 194 patients examined at least 12 hours post-injury, 101 underwent a CT scan and 6 (3.3%) revealed an abnormality (57). Another case series describes the late deterioration due to diffuse brain swelling that might occur in children at varying time intervals after MTBI or trivial head injury (55).

Is anti-coagulative medication a risk factor for intracranial lesions?

No high-quality study providing evidence on the risk of anti-coagulative medication or disorder for intracranial lesions was identified. One phase III study excluded these patients (33), and they were under-represented in the other (15). The findings in 2 case series of patients with trivial head injuries were inconsistent (58).

Tests of cognitive function

There were 8 studies addressing the reliability and validity of cognitive assessment, or reporting observations of cognitive dysfunction, in MTBI (59–66). Three are cohort studies, 2 are

*Nygren C, Bellocco R, af Geijerstam J-L, Borg J, Adami J. Delayed, intracranial complications after concussion: a population-based nested case-control study in Sweden. Unpublished submitted 2003.

Table V. *Diagnostic value of skull fracture in detecting intracranial lesion*

Authors	Study design	Setting and country	Sample size (*n*) and brain injury severity	Age spectrum	Outcomes	Main findings
Hofman et al., 2000 (43)	Systematic review	13 studies of the diagnostic value of skull X-ray. 13 studies for the prevalence of intracranial haemorrhage	*n* = 27,389 for X-ray, *n* = 12,750 for prevalence of intracranial haemorrhage. GCS 13–15	Studies with paediatric or geriatric patients only were excluded	Frequency of intracranial haemorrhage. Sensitivity and specificity of skull fracture for the diagnosis of intracranial haemorrhage	Mean frequency of intracranial haemorrhage was 8.3%. In studies where at least 50% had CT, sensitivity of skull fracture was 38% and specificity 95%
Gomez et al., 1996 (41)	Cohort	University hospital, Spain	*n* = 2484 GCS 13–15	>15 years	Neurological deterioration and need for surgery	Incidence of skull fracture detected by X-ray and abnormal CT findings was higher in those with GCS 13 than those with GCS 15 and GCS 14
Hung et al., 1996 (44)	Cohort	Hospital based setting from Taipei City and Hualien County, Taiwan, China	*n* = 7000 GCS 13–15	Not reported	SSIH and skull fracture as risk factor for SSIH	476 had a SSIH (6.8%). Skull fracture is a risk factor for SSIH with a crude odds ratio of 5.5‡. Sensitivity of skull fracture (by skull X-ray or CT) in predicting SSIH was 37.8%, specificity 90.2%, −PV 95.2%, +PV 95.2%, +LR 3.9 (CI 3.34–4.39), −LR 0.69 (CI 0.64–0.74). Accuracy 86.6% with a prevalence of skull fracture of 6.8%
Masters et al., 1987 (46)	Cohort	31 Emergency departments Maryland, USA	*n* = 7035 Moderate risk group is head injury with LOC or PT and the group that best corresponds to MTBI[1]	Not reported	Incidence of skull fracture, intracranial injury and mortality	Skull fractures were found in 0.4% of low risk group, 4.2% of moderate risk group and 21.5% of the high-risk group. None of low risk patients had an intracranial injury but 4% of the moderate and 29% of high-risk patients had intracranial injury. No sensitivity and specificity data presented
Teasdale et al., 1990 (53)	Cohort	General hospitals and 1 neurosurgery unit, UK	*n* = 9326 All severities of brain injury and 8051 were fully conscious at arrival to accident and emergency department	Not reported	Absolute and relative risk of surgically operated intracranial haematoma and incidence of intracranial haematoma. Children compared with adults	Children are at less risk than adults for intracranial haematoma requiring surgery: one in 2100 children vs 1 in 34 adults. Those with no skull fracture and no history of altered consciousness are at low risk. The crude risk of intracranial haematoma requiring surgery in fully conscious adults with no history of altered consciousness and without a skull fracture was 1 per 31,370, and with a skull fracture it was 1 per 81
Nee et al., 1999 (50)	Cross-sectional	One emergency department, UK	*n* = 5416 All severities of brain injury	Not reported	Vomiting as a risk factor for skull fracture	Relative risk of skull fracture for one vomit was 4.3 (95% CI 2.7–7.0) and multiple vomits 3.7 (95% CI 1.6–9.0). Sensitivity and specificity for vomiting as a predictor of skull fracture were 28% and 93% for adults, vs 33% and 88% for children. In patients who were fully alert at presentation, post-traumatic vomiting was associated with a two-fold increased risk for a skull fracture
Servadei et al., 1988 (52)	Cross-sectional	Two hospitals without neurosurgery service, Italy	*n* = 182 GCS 14–15	>13 years	Skull fracture as a risk factor for intracranial abnormalities	38% of patients with skull fractures had intracranial abnormalities opposed to 6% of those without skull fractures. 1 per 105 with skull fracture required surgery, and those without fracture did not require surgery

[1]Task force analysis. GCS = Glasgow Coma Scale; CT = computerized tomography; SSIH = surgically significant intracranial haematoma;–PV: negative predictive value, +PV = positive predictive value; LR = likelihood ratio (+ is positive and − is negative); CI = 95% confidence interval; LOC = loss of consciousness; PTA = post-traumatic amnesia; MTBI = mild traumatic brain injury.

cross-sectional and 3 are clinical case series. Two of the cohort studies have a phase II design (Table VI) (59, 60).

Assessing MTBI (concussion) in athletes

One study yields phase II evidence that recent memory questions are more sensitive than orientation questions in the assessment of cognitive function in concussed athletes (59). Another phase II study provides some evidence on the validity of a brief measure of cognitive functioning, the Standardized Assessment of Concussion, to detect the immediate effects of MTBI on cognition (60). Other studies yield limited evidence on the use of various tools for diagnosing disturbed cognitive function in patients with MTBI. One study compared concussed individuals with normative data on the Digit Symbol subtest from the Wechsler Adult Intelligence Scale – Revised, and it suggests that age is an important determinant of the results (64). Those 20–24 years of age had better test scores than both younger and older age groups, and no difference was observed between football players who reported a concussion when compared with those players who had not reported a concussion during the prior 6 months.

Assessing post-traumatic amnesia

PTA has been proposed as a more informative clinical measure than the GCS score for classifying severity of MTBI. However, there are no high-quality studies to substantiate this view. In 1 accepted study, classification by GCS and PTA gave different impressions of injury severity (62). Another study compared a simple, questionnaire-based quantitative test of PTA with PTA estimates by neurosurgeons, and these matched closely (66). One study explored the correlation between assessment of 2 observers by using the Rivermead PTA protocol across different time intervals and different TBI severity grades (63). Injury severity grade and time delay from injury to assessment seemed to affect the reliability of PTA assessment, but no data on acute PTA assessment are presented. One study that compared GCS scores and PTA in concussed and healthy children showed that duration of coma and PTA are related (65). In summary, the accepted studies in this area yield no consistent evidence.

Other studies related to diagnosis

There were 15 studies related to various other aspects of diagnostic procedures in MTBI (67–81). Two are cohort studies, 2 are cross-sectional studies, 10 are clinical case series or other descriptive studies, and 1 is a systematic review. There were no phase III diagnostic studies in this group.

Two studies (67, 68) examine the diagnostic value of vomiting in children with MTBI. They suggest that vomiting is associated with a past history of recurrent vomiting, motion sickness or migraine headaches rather than head injury. However, these findings are in contrast to a study on clinical prediction of skull fractures (50) that reports that vomiting is an independent risk factor for skull fracture in both adults and children.

Table VI. *Phase II studies on cognitive function in mild traumatic brain injuries (3)*

Authors	Study design	Setting	Inclusion/exclusion criteria	Outcome measures	Main findings
Maddocks et al., 1995 (59)	Cohort	Australian rules football players	Inclusion: senior players. Cases: players with a concussion (n = 28) during play (defined as LOC or altered consciousness and development of post-impact symptoms). Controls: players suffering other injuries and no concussion within the previous 12 months (n = 28)	The sensitivity of 8 orientation questions compared with 6 questions relating to recent memory items	Recent memory questions were more sensitive than orientation questions. Sensitivity of the 2 best memory items was 71–75% and the specificity was 86–100%[1]. Sensitivity of the 2 best orientation items was 18–57% and the specificity was 68–100%[1]
Barr & McCrea, 2001 (60)	Cohort	High-school and university football players, USA	Inclusion: players available for baseline testing with a brief measure of cognitive functioning between 1997 and 1999. Cases (n = 50) were re-tested immediately following a play-related concussion. Controls (n = 68) had no injury	The reliability and validity of the Standardized Assessment of Concussion (SAC)	One point decline on SAC differentiated injured and non-injured participants with a sensitivity of 94% and a specificity of 76%

[1]Task force analysis.
LOC = loss of consciousness.

Blood chemistry

We accepted 1 study that suggests that hypokalemia occurs in children with acute MTBI, but resolves within 24 hours (69). Another study explored the presence of inappropriate secretion of antidiuretic hormone in a large case series of 1808 patients with head injuries of all severity grades, including 842 with MTBI (70). The reported prevalence of this condition was low (0.6%) in the patients with MTBI. However, its clinical significance in MTBI is not clear. The task force accepted only 1 study related to the diagnostic value of the serum protein S100 (71). This study suggests that a small proportion of patients with MTBI has elevated S100 levels, but there are no data on the diagnostic validity of the test.

Documentation of MTBI in medical records and studies

Retrospective clinical studies require high-quality documentation of relevant, clinical data. This issue was addressed in 2 exploratory studies. One UK study reports how diagnosis was documented in patients admitted for head injury (72). Twenty-four out of 47 patients did not have the injury documented, and this occurred more commonly in the presence of other injuries, and when the head injury was trivial. A Canadian study reported on the quality of MTBI diagnostic and clinical data in 119 patients at 2 hospitals, 1 of which was a university hospital (73). These authors performed patient interviews within 3 weeks after the injury and found that lack of reporting MTBI in emergency records was common. Similar findings were reported in another study that reviewed this issue. According to the authors, various criteria are used to define MTBI in children and most studies poorly document many aspects of TBI in children (74).

Other aspects

Various other aspects of MTBI diagnosis were addressed in several clinical descriptive studies, including acute motor and convulsive symptoms in sport-related concussion (75), the workload of a regional head injury service before and after 2 policy changes (76), and pre-hospital triage to a regional trauma centre (77). We found only 1 study concerning the use of magnetic resonance imaging (MRI) in patients with MTBI (78). CT scan was performed promptly after the presentation to the emergency room, usually within 4 hours. MRI was performed within 24–96 hours post-injury. In this case series of 20 patients with GCS 13–15 and a normal CT scan (usually performed within 4 hours post-injury), MRI (performed within 24–96 hours post-injury) demonstrated abnormalities in 6 of the patients, including signs of diffuse axonal injury. The diagnostic value of MRI is an important area for further research.

DISCUSSION

It is striking that about two-thirds of the 230 articles addressing diagnostic procedures in MTBI were judged as scientifically inadmissible. Many were single case studies or small case-series, and others used a design unsuited to the research question, or had too vague a case definition, or highly selected samples. This reflects the methodological difficulties in clinical diagnostic research on MTBI and the lack of uniform case definitions. It might also reflect that clinical research in this area has not been given high priority in the hospital setting, where patients with MTBI are often seen by junior doctors and managed according to established routines that are not necessarily backed by scientific evidence. However, there has been an increasing interest in diagnostic validity during the last decade, probably related to the increasing availability of CT scan. Thus, most scientific diagnostic evidence identified in this area concerns the use of CT scan as a gold standard outcome measure for clinical evaluation, or as a diagnostic tool to predict clinical course.

Most studies concerned the validity of clinical variables (symptoms and signs) to predict intracranial complications, as defined by CT scan or clinical course. CT scan abnormalities, including skull fractures, are present in varying proportions of patients depending on the setting of the study and the distribution of GCS scores. About 1% of the total MTBI population seen in hospital need intervention, and the mortality rate is close to zero. Thus, severe complications and the need for surgical intervention are uncommon, and diagnostic studies related to these complications require large numbers of patients to be conclusive.

The strongest diagnostic evidence comes from recent well-designed studies showing that clinical factors can be used to predict CT scan abnormalities and the need for surgical intervention in adult patients with MTBI. However, only 1 study (33) had a sufficiently long enough follow-up to demonstrate that clinical factors can be used to predict the need for intervention. In that study, patients were followed for 1 week after the injury, and a subgroup was followed for 2 weeks. Recently, the findings in that study have been validated (37). Other studies showed that intracranial complications rarely occur later than 1 week post-injury in the majority of patients with MTBI with GCS 15.

Another study (15) yields phase III evidence in the same area, and we have used the evidence in both phase III studies to make recommendations for the acute management of adult patients with MTBI. We did not find corresponding evidence for the usefulness of clinical variables to predict intracranial complications in children. It seems critical that these studies be performed in children soon.

It is not known if, or to what extent, non-surgical intracranial lesions on CT scan are important for the long-term outcome. Thus, further diagnostic research in terms of phase IV studies (3) is an important area that has not received any consideration (82).

Plain skull X-ray was the standard diagnostic tool before the CT scan era. Our literature review yields consistent evidence that skull fracture is associated with intracranial lesions. However, we also found evidence that the diagnostic accuracy of skull fracture as a marker for intracranial lesions is poor.

Studies on the diagnostic value of cognitive function assessments in acute MTBI were surprisingly few. One reason might

be that these studies are not easy to perform in an emergency setting in patients who might be distracted by factors like other injuries, alcohol or drugs, and the general stress of an injury. Most of the cognitive data comes from studies in sports activities. There was some evidence that the Standardized Assessment of Concussion is a useful diagnostic tool, and that questions on recent memory are more sensitive than questions on orientation in assessing athletes who have sustained a concussion. However, further studies are needed to prove the validity of these findings.

In summary, confirmatory studies of diagnostic procedures in MTBI are scarce. Obviously, diagnostic research in MTBI faces a number of important challenges, including the development of cost-effective acute management and follow-up routines. As pointed out, there is need for confirmatory studies of the findings that clinical factors can be used to predict CT scan abnormalities and the clinical course after injury. There is also an urgent need for phase III evidence for the identification of intracranial lesions in the paediatric MTBI population. New imaging techniques, such as MRI and positron emission tomography, as well as biochemical injury markers specific for different cell population in the brain, and cognitive tests, will probably be useful to improve the basic understanding of acute MTBI pathophysiology and might help improve our diagnostic procedures in the future. Furthermore, these technologies could be useful in phase IV studies of the possible impact subtle brain lesions might have for persisting symptoms and disability. Moreover, progress in diagnostic research will probably help define MTBI and further stratify patients into meaningful severity groups (19, 27, 74).

CONCLUSION AND RECOMMENDATIONS

There is consistent evidence that clinical factors can be used for predicting intracranial lesions in adult patients with MTBI with the sensitivity and negative predictive values that are required. The strongest evidence is from the 2 phase III studies (15, 33). The findings from these studies are supported by phase II evidence from other accepted studies, and no contradictory evidence was identified.

When the findings of these studies are translated to management recommendations, the aforementioned strengths and limitations of both studies have to be considered. Therefore, the task force favours a cautious interpretation that takes into account all clinical factors that are shown to be risk factors for CT scan abnormalities in both studies. Even in doing so, the number of CT scan examinations and admissions for hospitalized observation will probably be reduced in many settings if our recommendations are implemented.

One of the studies only included patients with an arrival GCS score of 15 (15), while the other included patients within the whole severity spectrum, including those with an arrival GCS score of 13–15 (33). In the latter study, a persisting GCS score below 15 at 2 hours after the injury is a high-risk factor for intracranial lesions. Since the time interval between the injury and the first examination in the emergency room might be unknown, or the estimate of this time period unreliable, the task force recommends that the GCS score at the first examination be used for risk classification.

Some of the other risk factors identified in the 2 studies overlap. Both studies show that older age is a risk factor for intracranial lesions and the reported age limit differs only by 5 years. Injury mechanism is considered only in 1 of the studies, which shows that a dangerous injury mechanism is a high-risk factor (33). A dangerous injury mechanism means any of the following: pedestrian struck by motor vehicle, occupant ejected from a motor vehicle, and fall from a height more than 3 feet or 5 stairs. Suspected or open skull fracture and any signs of basal skull fracture, as identified in 1 study (33), are covered by "physical evidence of trauma above the clavicles", as identified in the other study (15). Both studies yield evidence that anterograde amnesia is a risk factor, again with the difference that the criterion is more restrictive (duration more than 30 minutes) in the study by Stiell et al. (33). The task force recommends that any anterograde amnesia that persists at the first examination be considered a risk factor. Vomiting, either any vomit (15), or vomiting 2 or more times (33), is a risk factor in both studies. A cautious interpretation would be to consider any vomit as a risk factor.

Three other risk factors were identified in only 1 of the studies (15). One of these is headache that is defined as any head pain whether diffuse or local. This is indeed a very broad criterion, but given the evidence, it has to be considered. Seizure, defined as a suspected or witnessed seizure after the trauma, is an identified risk factor in one study (15), while seizure was an exclusion criterion in the other study (33). There is both clinical and biological rationale to consider seizure as an important risk factor for intracranial lesion. Drug or alcohol intoxication, "determined on the basis of the history obtained from the patients or a witness and suggestive findings on physical examination", is an identified risk factor in 1 study (15). Therefore, the task force recommends that the following factors be used to identify patients at risk for intracranial lesions: age limit over 60 years, dangerous injury mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, and fall from height more than 3 feet or 5 stairs), an arrival GCS score of 13 or 14, suspected or open skull fracture, any signs of basal skull fracture or other physical evidence of trauma above the clavicles, anterograde amnesia, vomit, headache (defined as any head pain whether diffuse or local), seizure (defined as a suspected or witnessed seizure after the trauma) and drug or alcohol intoxication.

In order to properly diagnose acute MTBI, the task force recommends that a minimum data set be collected on all acute patients presenting with suspected MTBI. This must include age, mechanism of injury, vomiting since the injury, presence of headache since the injury, presence of seizures since the injury, presence of anterograde amnesia since the injury, GCS score, evidence of suspected or open skull fracture, signs of basal skull fracture and evidence of trauma above the clavicles.

Patients with acute MTBI presenting with a GCS score of 15 and without any of the risk factors can be discharged home

without a CT scan examination, if there is no other reason for hospital admission such as other injuries. Patients with MTBI presenting with a GCS score of 15 and one or more of the risk factors should undergo a CT scan. If the CT scan is normal, the patient can be discharged for home observation, if there is no other reason for hospital admission such as other injuries. If the CT scan shows traumatic abnormality, the patient should be admitted, unless a qualified interpreter judges the CT scan finding as clinically not important and the patient can be reliably observed at home. If there is limited access to CT scan examination, careful hospitalized observation is an alternative. Patients with MTBI with a presenting GCS score of 13 or 14 should undergo a CT scan examination and be admitted for hospitalized observation. These recommendations are summarized in Fig. 1.

There is no evidence on how long a hospitalized observation period should be. Therefore detailed recommendations are not possible. Generally, the current clinical risk factor(s), the clinical course, and CT scan findings must all be considered. The task force finds it reasonable, with regard to available evidence and clinical experience, to recommend 24 hours as the minimal time for hospitalized observation when the current recommendations for CT scan and admission are used.

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