## Subject Review

## Recovery from mild head injury

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The subjective and objective sequelae accompanying mild head injury (MHI) are discussed in an attempt to clarify MHI's immediate and long-term consequences. Areas covered include epidemiology, classification, the post-concussive syndrome (PCS), malingering, extent of recovery, rehabilitation and guidelines for clinical practice. Special emphasis is placed on the poor relationship between subjective complaint and objective measures of impairment. Also discussed are some of the methodological problems in the MHI literature, including attempts to match MHI subjects and controls with respect to cognitive and emotional complaint and the possible confounding effects of practice. The evidence for long-lasting (i.e. more than 1 year), subtle neurobehavioural impairment after MHI indicates that additional research is required on MHI 1 year or more after injury.

### Introduction

Mild or minor head injury (MHI) can occur quite easily. A motor vehicle accident, a fall in the bathtub, or a routine sports injury can all result in a MHI. Minor injuries may have unpleasant consequences that can linger for months if not years. This paper will address the effects of MHI on cognitive and emotional functioning. The paper is divided into two main sections. The first broadly covers classification, epidemiology and the post-concussive syndrome, while the second presents evidence for and against neurobehavioural impairment long after MHI. The paper concludes with a brief discussion of rehabilitation and guidelines for clinical practice, and suggestions for future research.

### Head injury classification

It is important to distinguish MHI from more severe forms of head trauma. It is also useful to separate MHIs, depending on the presence or absence of several factors. For instance, it has been argued that a distinction should be drawn between MHI, which describes any traumatic injury to the head and face, and mild traumatic brain injury, a subset of MHI in which brain damage is evident [1]. The rationale for this distinction is that MHI does not imply brain damage. Because it is difficult to detect underlying brain damage in many cases of MHI, the term MHI will be used throughout this review.

Several classification systems use the term concussion when characterizing a MHI. Concussion refers to an immediate and transient disturbance in neurological

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function caused by mechanical trauma [2] that may or may not involve loss of consciousness [3]. In a series of recent articles, Kelly and colleagues [3–5] present a concussion grading scale, followed by a brief sideline evaluation of head injured athletes, and guidelines for detecting and managing features and symptoms of concussion. Although developed for sports injuries, these guidelines should improve the management of other cases of concussion as well. All types of concussion are included in the defining criteria listed below.

Typical criteria for MHI diagnosis have included any one or more of the following: Glasgow Coma Scale (GCS) [6] rating of 13–15 upon hospital admission; absence of skull fracture or focal brain lesion; normal neurological examination; unconsciousness of less than 30 minutes (most studies now restrict inclusion to those individuals with less than 20 minutes unconsciousness); post-traumatic amnesia of less than 24 hours (here too the tendency of late has been to exclude cases where post-traumatic amnesia exceeds 1 hour) [7]. All of these criteria have their limitations.

For instance, Dacey *et al.* [8] conducted a prospective study of 610 MHI patients and found that an initial Glasgow Coma Scale score of 13–15 may belie more severe damage. In fact, 3% of the original patients required neurosurgery despite initial normal levels of alertness. Similar proportions have been reported elsewhere [9, 10]. On a related note, Dikmen and Levin [11] argue that alcohol intoxication at the time of injury may spuriously lower GCS rating upon admission to the hospital. Because alcohol intoxication is often associated with MHI, these authors argue cogently for its inclusion as an independent variable in MHI studies. Finally, recent evidence, based on a sample of 3370 consecutive cases of MHI, suggests that a GCS score of 15 should be separated from scores of 13 or 14 [12]. Studies such as these illustrate some of the problems associated with GCS ratings in MHI research.

With regard to intracranial pathology and MHI classification, Williams *et al.* [13] compared three groups of head injured patients ($n = 215$) to determine whether MHI complicated by intracranial lesion is more appropriately considered moderate head injury. Their findings indicate that depressed skull fracture can still be categorized as MHI if it is unaccompanied by an intracranial lesion.

Gronwall and Wrightson [14] studied patients' own estimates of post-traumatic amnesia over the first 3 months post-injury and found that 25% of their MHI subjects changed their estimates during this time. King *et al.* [15] report similar proportions, and also found that retrospective estimates of post-traumatic amnesia are far less reliable after MHI than after more severe head injury. For this reason, retrospective estimates of post-traumatic amnesia after MHI should be used with caution. Similar arguments have been levelled against self-reported estimates of length of unconsciousness [2]. These findings help illustrate the potential problems associated with MHI defining criteria. Such difficulties in MHI classification have spurred many authors to argue for stricter and more consistent defining criteria in MHI research (e.g. [11, 16]).

## Epidemiology

MHI occurs frequently. Depending on several factors such as gender, age, demographic location and socioeconomic status [17], the incidence of MHI is between 130 and 200 hospitalizations per 100 000 people annually [18–20]. However, this figure may be as high as 640 people per 100 000 when non-hospitalized cases are included [21]. There is good reason to believe that many cases of MHI go unre-

ported, depending on the definition of MHI. For example, in two retrospective self-report epidemiological studies of high school and university students, Segalowitz and colleagues [22, 23] defined MHI as any blow to the head causing one to stop whatever one was doing because of dizziness, pain, disorientation, or unconsciousness (an acceptable definition of MHI, according to Kay *et al.*'s [7] standards). The authors found that 25–35% of their samples reported MHI, although only 20–25% of MHI respondents received medical attention [23]. Thus, MHI may be far more prevalent than hospital surveys indicate.

Males between the ages of 15–24 years comprise the highest risk group for MHI. The most common cause of MHI is motor vehicle accidents, accounting for nearly 50% of all MHI. Falls, assaults, sports injuries and bicycle crashes comprise the remaining injuries (although, see [24] for a different ordering of these causes).

### Post-concussive syndrome

A constellation of symptoms is often reported after MHI. These can be roughly grouped into three categories: physical symptoms, including dizziness, fatigue, sleep difficulty, nausea, headache, blurred vision and insensitivity to intense light and sound; cognitive complaints, of difficulty concentrating, problems with memory, and impaired problem solving; behavioural and affective symptoms, including irritability, anger outbursts, depression, anxiety and poor social functioning [1, 25]. Together, these symptoms form what has been called the post-concussive syndrome (PCS), although some researchers challenge the validity of these symptom clusters [26, 27]. Questionnaires are typically employed in the assessment of PCS. The presence of several of the symptoms listed above after MHI is usually presumed to be evidence of PCS. Unfortunately, there is no precise definition of PCS presently agreed upon by MHI researchers. Many factors may be associated with PCS including age, sex, length of post-traumatic amnesia and unconsciousness, multiple MHIs, litigation and education. While Gronwall and Wrightson [28] have shown that multiple MHIs produce cumulative effects, the precise influence of the remaining variables listed above is a matter of debate. The extent to which PCS persists is also equivocal [21, 29, 30].

It has been demonstrated that PCS complaints are very common in the first weeks following MHI [31, 32]. These complaints have been documented 1 year post-injury [33, 34] up to 5 years post-injury [35–37], and as long as 23 years post-injury [38]. In the majority of cases, however, PCS symptoms mitigate over time and result in good recovery [21, 29]. This 'good recovery' may actually represent a behavioural adaptation rather than a return to pre-injury levels of functioning. Often, individuals with a MHI complain of persisting attentional problems, although it is difficult to demonstrate this with objective measures. Indeed, Seymonds [39] described some of the symptoms accompanying head injury (dizziness, headache, etc.) and questioned the reversibility of any concussion, regardless of severity.

The relationship between PCS, measured through self-report questionnaire, and objective cognitive impairment is obscure. King [40] has shown that questionnaires measuring stress and emotional factors given 7–10 days post-injury are far better predictors of PCS complaint at 3 months than are divided attention tasks given 7–10 days post-injury. One likely reason for these findings is that PCS checklists and questionnaire measures of stress and emotional factors share a similar methodology:

they are both questionnaires. Correlations between similar methods usually surpass those of dissimilar methods [41].

Indirect evidence for a link between PCS and objective cognitive deficits comes from the work of Klonoff *et al.* [38] who longitudinally assessed a cohort of children for 23 years. Ninety per cent of these children had a MHI. The authors found a significant relationship between subjective sequelae and objective, psychosocial measures of adaptation including educational lag, unemployment, current psycho-logical and psychiatric problems and relationships with family members. Unfortunately, subjects in this study were not administered cognitive tasks. Information was gathered via interviews. Arcia and Gualtieri [42] provide more direct evidence for the relationship between PCS and cognitive impairment. They report that memory complaints on a PCS symptoms checklist are the problems most commonly endorsed and most closely associated with neurobehavioural deficits in a group of mild to moderate head injured patients more than 1 year post-injury.

Many authors have questioned whether cognitive deficits remain in MHI patients who have PCS years after injury [43–47]. In fact, Alexander [48] has recently bemoaned the lack of diagnostic specificity between subjective PCS and objective neuropsychological impairment, maintaining that the two 'are equally compatible with diagnoses of depression, anxiety and chronic pain' (p. 1257). Binder [27] adds alcoholism, hypertension, migraine, and diabetes to this list.

In addition to the many voices asserting a lack of association between PCS complaint and cognitive impairment, there is evidence suggesting that neuro-behavioural deficits can persist many years after injury in the absence of PCS [49, 50]. While most studies have focused on groups of MHI patients with PCS, these latter reports are unique in that they used healthy, well-functioning indi-viduals with MHI and with no cognitive complaints. Thus, just because PCS and cognitive impairment may be poorly correlated does not imply that cognitive impairment does not exist after MHI.

### Malingering

A popular argument for the meagre relationship between PCS and performance found in so many studies is that patients presenting with PCS years after injury are motivated by financial incentives [51]. One detail offered in support of this thesis is that persistent PCS often disappears after financial settlement is reached. There is ample evidence arguing against this assertion [52].

MHI research on malingering has received much attention over the past 30 years. Some investigations have focussed entirely on tests and methods designed to detect malingering [e.g. 43, 53, 54]. Most suggest that the desire for compensa-tion may have contributed to poor performance. A recent study by Martin *et al.* [55] contributes to the growing body of evidence that PCS is linked to malingering. The authors found that, although a group of coached and uncoached college students, asked to feign deficits, can accurately simulate self-reported PCS symptoms, they cannot simulate objective clinical malingering test performance. These findings indicate that self-report PCS measures and clinical tests are differentially vulnerable to malingering.

One problem with this conclusion is that, if persistent PCS is related to mal-ingering, as the above studies indicate, then one might expect the layperson's knowledge of MHI to reflect an adequate understanding of the cognitive and

emotional sequelae surrounding MHI. However, Aubrey *et al.* [56] have demon-strated that college students with no formal knowledge of head injury have little understanding of the range of symptoms commonly reported after MHI. Moreover, this understanding is particularly poor with regards to cognitive deficits frequently reported after MHI.

Another problem with many of the malingering studies where controls are asked to fake deficits after a head injury is that these 'malingerers' often exaggerate deficits. The differences found between malingerers and controls tend to be much larger than the differences observed in many of the studies between matched controls and persons with MHI who report PCS. Given that judges are not always good at discriminating between the performance of volunteer malingerers and patients with MHI [16], if neurobehavioural deficits persist long after MHI (see evidence below), then such deficits will likely be subtle in nature. The malingering literature on controls asked to feign impairment may help identify the obvious opportunist, but it will unlikely tell us anything truly useful about the nature and extent of subtle cognitive impairment after MHI.

Despite these inconsistencies, a recent meta-analysis of 17 studies, including 2353 subjects, uncovered a modest overall effect size (0.47) for the impact of financial incentives on disability, symptoms and objective findings after head injury [57]. The authors conclude, reasonably, that their findings argue for more careful assessment in every case of head injury involving financial compensation.

It is important to note that, in addition to the evidence for an association between persistent PCS and the desire for financial compensation, there is a grow-ing body of work suggesting that cognitive, emotional and behavioural complaint and impairment can exist in the absence of financial gain [52]. In such cases, persistent PCS is unlikely due to malingering. Nevertheless, there are sufficient data in the literature to argue for the inclusion of litigation as an independent variable in MHI studies.

### Psychiatric sequelae

Psychiatric sequelae have also been reported following MHI [58–60]. Merskey and Woodforde [58] found that nearly half of their MHI patients (10 of 23) assessed for psychiatric sequelae at a median of 4 years post-injury showed no improvement. More recently, Fenton and McClelland [60] conducted a prospective study of 45 MHI patients and matched controls. Thirty-nine per cent of the MHI patients were diagnosed as psychiatric cases at 6 weeks post-injury. Approximately half of these cases complained of symptoms at 6 months. Overall, the MHI patients displayed four times the mean level of chronic social difficulties compared to controls. Lishman [61, 62] contends that several factors interact to produce psychiatric se-quelae after head injury. The same author also proposes a model of PCS where initial complaints are most likely biogenic in origin, whereas long-term sequelae are probably psychogenic. This view has some support in the literature [33, 47].

A recent model of long-term PCS attempts to invalidate Lishman's [61] biogenic/psychogenic dichotomy. Jacobson [52] contends that persistent PCS is maintained by a combination of organic, psychosocial and behavioural factors. Instead of focuss-ing on symptoms or pathophysiology, Jacobson's model centres on the patient's beliefs and coping responses. Whether long-term sequelae result from biogenic and/or psychogenic causes, the impairment may be real.

*Dissociation between cognitive impairment and complaint*

An important methodological issue arising from MHI research concerns the rela-
tionship between objective cognitive impairment and subjective cognitive com-
plaint. Many models of functional disability after MHI rely on the assumption
that individuals are not only aware of their deficits, but also that they are good
judges of the types of deficits they have. According to one such model [1], people
may exaggerate symptoms or choose to ignore and over-compensate for their
problems. Whichever way people react during recovery from a MHI, according
to this model, they are *aware* of their deficits. Unfortunately, it is also quite possible
to be unaware of deficits. In other words, there are reasons to believe in a possible
dissociation between subjective complaint and objective measures of performance.

This dissociation refers to either a lack of subjective complaint in the presence of
cognitive impairment, or to the lack of cognitive impairment in the presence of
cognitive complaint. A person may think that s/he is cognitively impaired, but s/he
performs adequately on most tests. Conversely, a person who thinks s/he is cogni-
tively sound may, in truth, be impaired. Another possibility is that people are just
poor at identifying the nature of their deficit. That is, a subjectively perceived
inability to concentrate may, in fact, be a problem with integrating novel informa-
tion. Whatever the reason, more attention should be paid to the possibility that
people are inaccurate judges of their own cognitive abilities.

*Methodological considerations*

Most MHI investigations utilize patients who evidence PCS. Many of these patients
are referred to a neuropsychologist for standard assessment. In such instances,
patients are administered a battery of neurobehavioural tasks and may be given a
PCS checklist or questionnaire. MHI patients are then matched with controls for
age, sex, education and intelligence. Controls generally complete only the neuro-
behavioural battery.

There are two problems associated with this methodology. First, MHI patients
with and without PCS and complicating factors (e.g. pre-existing emotional prob-
lems, previous head injury in the last 10 years, alcohol consumption at time of
injury) differ on neurobehavioural tasks and on PCS questionnaires [32, 63, 64].
Second, controls often complain of many of the symptoms associated with PCS [46,
65]. In order to counter these problems, researchers should separate individuals with
MHI into subgroups based on cognitive and emotional complaint. Moreover,
researchers should administer PCS measures to both controls and MHI patients.
Taylor *et al.* [66] present a novel solution. They matched patients with MHI to
patients with Chronic Pain Syndrome.

Finally, it should be noted that some authors have cautioned against the use of
unstandardized PCS questionnaires and checklists in MHI research (e.g. [40]). To
better distinguish groups of MHI patients with and without PCS, standardized PCS
measures should be used. It is unacceptable to assume that a list of possible symp-
toms on a checklist adequately reflects PCS just because those are the symptoms
most commonly endorsed by persons with MHI. A related problem here is that
PCS questionnaires should contain distractor items that have nothing to do with
PCS. There are several standardized PCS and PCS-related questionnaires [65, 67–
69]. More consistency in PCS questionnaire usage, coupled with the use of

psychometrically sound PCS inventories, would produce a more coherent body of MHI research.

### Conclusions about PCS

Despite PCS's contentious nature, the constellation of symptoms indicative of PCS is remarkably similar across most experimental series worldwide [52]. The current trend in MHI research is underscored by a reliance on biogenic explanations of PCS. However, the definition of probable cause of PCS must be expanded to encompass multiple psychological and psychosocial factors. These include emotional responses to injury (e.g. post-traumatic stress disorder); pre-existing conditions such as learning disabilities [70], psychiatric problems, neurological disorders, head injury, alcohol and other drug abuse; and circumstances following the accident (e.g. litigation).

## Evidence for and against long-term neurobehavioural impairment

The following sections outline some of the evidence for and against an array of persistent cognitive and behavioural deficits after MHI, without regard to a particular model or to specific types of impairment. The discussion is limited to studies performed on adult samples (see [71] for a review of MHI in children and adolescents).

Early work on the neurobehavioural sequelae of head injury by Strauss and Savitsky [72] outlined the subtle effects of brain injury on behaviour. Ruesch [73] later found evidence that individuals with both mild and severe head injuries demonstrated problems with sustained effort, mental speed and visual judgement. Other neurobehavioural deficits generally associated with MHI include impaired information processing capacity [74], poor memory and visuospatial skills [75], slower reaction times and inconsistent performance on complex tasks requiring focussed and divided attention [31, 76].

Although there is much evidence in the literature to suggest that most MHI cases involving cognitive and/or behavioural deficits resolve within 6 months [17, 44, 46, 64, 66, 77–80], there are mounting data that indicate long-term impairment [49, 50, 75, 81–90] (see tables 1 and 2, respectively). There are also several investigators who have argued that the effects of MHI may be permanent [14, 48, 59, 91].

### Evidence for rapid recovery after MHI

A number of initial reports suggesting rapid recovery from MHI served to instill an overly optimistic view of recovery from MHI (see table 1). McLean *et al.* [46] tested a group of head injured patients over the first month of recovery and found that cognitive decrements at 3 days improved by 1 month. The main problem with this study is that the authors did not differentiate between mild and moderate head injury. MacFlynn *et al.* [79] followed a group of 45 MHI patients for 6 months after injury. This group showed markedly improved reaction times relative to controls. Unfortunately, in this study, controls were only tested once, whereas MHI patients were tested three separate times on the same task and then compared to the controls

Table 1.   *Evidence for rapid recovery after MHI*

| Study | Subjects | Time since injury | Primary findings |
|---|---|---|---|
| McLean et al. [46][a] | 20 HI<br>52 Controls | 3 days<br>1 month | HI group impaired on neuropsychological battery at 3 days, but not at 1 month. |
| Levin et al. [17] | 57 MHI<br>57 Controls | 1 week<br>1 month<br>3 months | MHI worse on 5/5 measures at 1 week; MHI normal performance on all five tests by 3 months. |
| MacFlynn et al. [79] | 45 MHI<br>45 Controls | 1 day<br>6 weeks<br>6 months | MHI slower on 4-choice RT at 1 day; marked improvement by 6 months. |
| Dikmen et al. [44] | 20 MHI<br>19 Controls | 1 month<br>1 year | MHI worse on 2/21 tests at 1 month (sustained attention and delayed recall); no difference at 1 year. |
| Dikmen et al. [77] | 161 MHI<br>121 Controls | 1 month<br>1 year | No difference on any of the measures at 1 year. Results of 1 month testing not given. |
| Bohnen et al. [64][b] | 11 MHI<br>11 MHI<br>11 Controls | 12–34 months | MHI w/ complaints worse than other two groups on 1/4 measures (sustained attention). |
| Alterman et al. [78][a,c] | 25 MHI<br>25 Controls | No given | No difference on any of the measures. |
| Taylor et al. [66][d] | 15 MHI<br>10 HI<br>24 Controls | 4 years[e] | No difference on measures of sustained or divided attention (used PASAT and Consonant Trigram Test). |
| Bornstein et al. [80][a,f] | 24 MHI<br>24 Controls | 14.8 years[e] | No difference on any of the measures. |

[a]HI = mild to moderate head injury.
[b]In Bohnen et al. [64], 11 MHI Ss with PCS complaints; 11 MHI Ss without PCS complaints.
[c]In Alterman et al. [78], Ss were chronic alcoholics with and without mild to moderate HI.
[d]In Taylor et al. [66], HI group was moderate to severe; Controls were patients with Chronic Pain Syndrome.
[e]Average time since injury.
[f]In Bornstein et al. [80], Ss were seropositive for the human immunodeficiency virus (HIV-1+).

each time. There  are two problems with this approach. First, repeated testing on the same task is subject to practice effects (see discussion below). Second, little can be assumed about the controls' performance relative to the MHI group unless both groups undergo similar testing protocols.

More reliable evidence for a relatively swift return to normal functioning after MHI comes from two seminal studies [17, 44]. Dikmen et al. [44], found that a MHI group performed little differently from matched controls at 1 month and no differently at 1 year. Perhaps the most widely cited study in support of the notion that MHI is generally associated with good recovery within the first few months is that of Levin et al. [17]. These authors prospectively studied 57 patients with uncomplicated MHI (i.e. no pre-existing psychiatric problems, head injuries, neuro-logical abnormalities or substance abuse) over the first 3 months post-injury and compared their performance on a neurobehavioural battery to that of 56 matched controls. Testing was done at 1 week, 1 month and 3 months at three different hospitals. Neurobehavioural impairment, self-reported cognitive problems, somatic complaints and emotional malaise observed in the MHI group at 1 week post-injury typically resolved by 3 months. These findings led the authors to conclude that a single uncomplicated MHI produces no permanent disabling neurobehavioural impairment in the great majority of patients.

Table 2. *Evidence for long-term neurobehavioural impairment after MHI*

| Study | Subjects | Time since injury | Primary findings |
|---|---|---|---|
| Rimel *et al.* [81] | 69 MHI | 3 months | Ss impaired on tests of new problem solving skills, attention and concentration. |
| Barth *et al.* [75] | 71 MHI | 3 months | Ss impaired on tests of memory and visuospatial skills. |
| Gentilini *et al.* [82][a] | 48 MHI<br>48 Controls | 1 month<br>3 months<br>5 months | MHI slower than controls on tests of sustained and divided attention at all intervals. |
| Cremora-Meteyard and Geffen [83][b] | 6 MHI<br>8 MHI<br>9 Controls | 1 year<br>$\geq$ 1 year | MHI impaired visuospatial attention. |
| Haaland *et al.* [84][c] | 40 HI<br>88 Controls | 1 month<br>1 year | HI impaired finger tapping and grip strength at 1 month; HI impaired finger tapping at 1 year. |
| Arcia and Gualtieri [85][c] | 26 HI<br>23 ADD[d]<br>25 Controls | 10 months[e] | HI worst overall; HI worse than controls on simple motor response, response speed and attention, memory and learning; HI best recall on self-paced symbol-digit pairs test; HI and ADD impaired sustained attention. |
| Stablum *et al.* [86] | 26 MHI<br>26 Controls | 2–184 days<br>2 years | MHI worse at both testing on dual task involving visual discrimination; effects due to age and loss of consciousness; no difference between groups on Stroop-like letter discrimination. |
| Leininger *et al.* [88] | 53 MHI<br>23 Controls | 1–22 months | MHI worse than Controls on 5/8 neuropsychological tests, including auditory and visual memory, information processing, executive functioning and visuomotor integration. |
| Cicerone [87] | 15 MHI<br>9 Controls | 1.5 years[e] | MHI slower on dual task, but equal accuracy. |
| Sangal and Sangal [89] | 8 MHI<br>32 Controls | 3.1 years[e] | MHI slower on visual discrimination; MHI longer visual P300 latency, but equal P300 amplitude. |
| Carlsson *et al.* [90][f] | 1112 Men | 'a long time' (p. 197) | Closed HI with unconsciousness correlated with performance on finger tapping and reaction time tasks. |
| Bernstein *et al.* [50] | 10 MHI<br>12 Controls | 6.4 years[e] | MHI worse on sustained and divided attention tasks; MHI reduced P300 amplitudes on 4/4 auditory discrimination tasks, but equal P300 latencies. |
| Klein *et al.* [49][c,g] | 25 HI<br>20 HI<br>45 Controls | 30 years[e] | 2 HI groups worse on all aspects of primary and secondary memory and on most reaction time tasks. |

[a] In Gentilini *et al.* [82], $n = 44$ at 3 months and $n = 12$ at 5 months.
[b] Cremora-Meteyard and Geffen [83] ran two experiments. In experiment 1, six MHI Ss. compared to controls at 1 year. In experiment 2, eight MHI compared to controls at 1 year or more.
[c] HI = mild to moderate head injury.
[d] ADD = Attention Deficit Disorder (adults).
[e] Average time since injury.
[f] In Carlsson *et al.* [90], Ss were 30, 50 and 60 year old men; roughly 50% reported mild to moderate HI.
[g] In Klein *et al.* [49], 25 middle aged and 20 elderly Ss.

   Despite its lasting appeal and importance, the study by Levin *et al.* [17] may be criticized on two fronts. First, it is unclear whether control subjects were tested more than once. If they were tested only once, then, like in MacFlynn *et al.*'s [79] study, it is difficult to conclude much about the improvement of the MHI groups relative to controls. Second, Levin *et al.*'s brief dismissal of practice effects as a valid account for their findings is inadequate. Contrary to their arguments, practice effects could explain the improvement in neurobehavioural performance observed in the MHI groups. Repeated testing on all the neurobehavioural measures used in this study could lead to markedly improved scores over a 3 month period. Here it is especially important that controls be tested in tandem with the MHI groups on each of the three occasions to chart the improvement in both groups over time. Improvement in control performance will provide an accurate index of the effect of practice, which can then be directly compared to improved scores in the MHI groups. Unfortunately, even if controls were tested more than once and their performance improved less over time relative to MHI performance, this does not mean that the effects observed are independent of practice; nor does this imply that the MHI group has recovered. Initially lower scores in the MHI groups will likely exhibit more improvement relative to the controls' high baseline scores, which begin high and, therefore, have less chance of improving (i.e. the ceiling effect).

   The negative findings of Levin *et al.* [17] and others [44, 79] helped establish the view that MHI typically results in rapid and complete recovery. Consequently, there are relatively few published studies reporting no difference between indi-viduals with MHI and controls more than 1 year post-injury. A study by Alterman *et al.* [78] provides some tentative and indirect support; unfortunately, this study fails to provide time since injury. Other notable and recent studies do provide such evidence. Dikmen *et al.* [77] prospectively studied 161 patients with MHI and 121 controls for 1 year. Testing occurred at 1 month and 1 year post-injury. The authors found no difference between these groups, despite large sample sizes and the use of a comprehensive neuropsychological battery. Although both groups were tested on both occasions, the results still may be due partly to practice (see discussion above).

   Bohnen *et al.* [64] compared two groups of MHI patients—those with and without post-concussive complaints—to matched controls 12–34 months post-injury. The two MHI groups showed no overall impairment on attention and information processing tasks relative to controls. Taylor *et al.* [66] compared 15 whiplash patients (MHI group), 10 moderate to severe head injured patients and 24 patients with chronic pain 4 years post-injury on tests of divided attention. No differences were found among the three groups. This study is laudatory for its inclusion of controls matched in terms of cognitive and emotional complaints; however, because most research on MHI sequelae includes controls with no cog-nitive complaint, it is difficult to compare this study to the majority of MHI studies. Finally, Bornstein *et al.* [80] assessed the long-term effects of MHI associated with HIV-1 infection, and found no differences relative to matched controls. Interestingly, although not statistically tested, the MHI group was slower on 14/ 14 reaction time measures in comparison to controls. Overall, these studies provide evidence suggesting that MHI is not associated with long-term neurobehavioural impairment. The next section summarizes studies that indicate the existence of long-term effects (see table 2).

### Evidence for long-term impairment after MHI

Two of the first studies to demonstrate the potentially lasting consequences of MHI were marred by their lack of a control group [75, 81]. In more controlled studies, individuals who have sustained a MHI may demonstrate slower information pro-cessing, problems on tasks involving divided attention, impaired focussed attention and overall inconsistency in performance up to 3 months post-injury, '...well beyond the period of posttraumatic symptoms' ([31] p. 857). Gentilini *et al.* [82] have found similar attention deficits 5 months post-injury.

More recent work has extended the picture of neurobehavioural impairment up to 3 years after MHI [83–89]. Two of these studies [84, 85] included patients with more severe head injuries in their MHI groups. In both cases, however, the majority of the HI group had MHI. Many of these studies employed highly demanding tasks to uncover differences between controls and individuals with MHI. For instance, Cicerone [87] compared a MHI group to matched controls on a dual task involving digit cancellation, mental arithmetic, and attending to or ignoring a radio talk show. Despite equal accuracy, dual task demands significantly slowed processing speed in the MHI group relative to controls. No difference emerged between the groups on the digit cancellation task alone, suggesting a more subtle form of neurobehavioural impairment in the MHI group.

Few studies have compared individuals with MHI to matched controls many years after injury. Evidence for impairment more than 5 years after MHI has come mainly from retrospective self-reported incidence of MHI [49, 50, 90]. For ex-ample, Bernstein *et al.* [50] assessed neurobehavioural performance in a group of high-functioning university students with self-reported MHI an average of 6.4 years after injury. Seven of the 10 individuals in the MHI group reported having experi-enced more than one MHI. In comparison to matched controls, the MHI group had significantly lower discrimination $(d')$ values on two complex auditory tasks, accompanied by reduced P300 evoked potential amplitudes on all four auditory discrimination tasks used. The authors argue for subtle neurobehavioural impair-ment long after MHI, supporting Cicerone's [87] conclusion described above.

In two additional studies, evidence for permanent impairment after MHI has been reported. Carlsson *et al.* [90] studied 1112 Swedish men aged 30, 50 or 60 years, chosen to represent the general population. The authors found a significant relationship between closed head injury accompanied by unconsciousness (70–75% of cases reporting less than 30 minutes of unconsciousness) and post-concussional symptoms, self-assessed health variables and performance on finger-tapping and reaction time tasks. In another study, Klein *et al.* [49] compared two groups of individuals with self-reported head injury to age and sex matched controls 30 years after injury. Despite what would be termed 'good recovery'—no cognitive or emotional complaint and normal intellectual functioning—the head injured groups performed worse on all aspects of primary and secondary memory and on most reaction time tasks used. Unfortunately, in both of these studies, the authors failed to adequately discriminate between mild and moderate head injury; therefore, generalization from these samples to the majority of individuals with MHI may be problematic. Despite this difficulty, these two reports demonstrate that cognitive impairment may persist many years after mild to moderate head injury.

The main problem with the three studies just discussed is that self-report data are far less reliable than hospital records. Sadly, the large number of MHI cases that go

unreported cannot be studied without resorting to retrospective self-report. For this reason, retrospective self-report methods are vital to MHI research.

Overall, the three retrospective studies [49, 50, 90], coupled with the evidence of impairment up to 3 years post-injury [81–89], offer some support for long-term neurobehavioural impairment after MHI. These data suggest that the rosy picture of cognitive recovery after MHI painted by so many investigators (e.g. [16, 17]) may have to be re-drawn to include permanent impairment. Indeed, in a recent meta-analysis of eight studies (seven of which are included in the present paper) involving neuropsychological outcome among patients without cognitive complaint at least 3 months after MHI, Binder *et al.* [92] found a small but significant overall effect size (*d*). The most powerful effects reported by Binder *et al.* involved measures of attention, a finding that concurs with the evidence cited in table 2. Because many of the effects reported by Binder *et al.* are relatively small, it might be more appropriate to conclude that MHI may be associated with long-term, *subtle* cognitive impairment.

### *Why the inconsistent findings? Methodological issues*

Several pre-injury and post-injury factors are believed to be linked to cognitive impairment after MHI. The list of factors related to PCS is also applicable here. Alcohol and other drug intoxication at time of injury and hospital admission, age, education, occupation, personality characteristics, emotional adjustment prior to and after injury, length of unconsciousness and post-traumatic amnesia, number of head injuries and time since injury have all been shown to be associated with neurobehavioural outcome [11, 16, 86, 93]. The relationship among these variables remains unclear.

For instance, Leininger *et al.* [88] found that MHI patients with and without loss of consciousness (but all with post-concussive complaints) performed worse than matched controls, but no differently from each other, on a battery of neuropsycho-logical tests 1–22 months post-injury. Moreover, time since injury was found to be unrelated to performance. Thus, neurobehavioural deficits can occur in the absence of loss of consciousness, and time since injury may have little overall effect on neurobehavioural performance more than 1 month post-injury. Gronwall and Wrightson [94] attempted to determine the relationship among information pro-cessing capacity, post-traumatic amnesia and memory impairment after differing degrees of head injury severity. They found highly variable relations among these measures, suggesting that it may be too simplistic to expect a single index of head injury severity to account for neurobehavioural outcome.

Unfortunately, relatively few investigators have followed MHI patients for more than 6 months. Fewer still have followed patients in excess of 1 year. Those studies that have followed patients longitudinally are mostly interested in the complaints indicative of PCS, using tests designed to tap more general rather than specific memory and attentional deficits.

This raises a problem with the MHI literature. Most studies have utilized a neuropsychological battery that taps various aspects of attention and memory. This makes sense given that these measures are reliable and valid. However, there is a need for more demanding measures that can detect *subtle* deficits like impaired divided attention and information processing speed and capacity deficits. Gronwall's [95] Paced Auditory Serial-Addition Task (PASAT) in which subjects mentally

add consecutively presented numbers, and the original and modified versions of the Stroop Color Word Test [32, 96] have been successfully used to this end. As Stuss and colleagues [75, 97] illustrate, divided attention is critical in a variety of settings (e.g. driving a car, working in a busy office). Thus, concerns with issues of divided attention are not new to MHI research, but they may help delineate more subtle forms of impairment after injury. Gronwall [24] maintains that a return to normal performance on neuropsychological tests does not imply a return to normal functioning. On a related note, Mateer and Mapou [98] question the ecological validity of many well-structured testing situations where normal per-formance does not necessarily denote normal functioning in a noisy social or work environment.

Another methodological problem present in the MHI literature pertains to the re-testing of participants on the same cognitive or behavioural tasks (e.g. [17, 77, 99]). Research involving significant improvements in performance, over a specified length of time after MHI, may be confounded by practice effects. Often, controls start at ceiling on many of these tasks, thus preventing a marked improvement in their scores over time. Participants with MHI, though, most often display a gradual improvement in their performance over the first few weeks and months. One solution to this problem is to use a staggered group design in which a single group of individuals is subdivided and given different tasks on different occasions. Another solution is to develop a psychometrically sound neurobehavioural test battery and to administer part of it right after injury and then to administer the other part months after injury. Either approach would eliminate practice effects, because the same participant would not complete the same task more than once.

One of the biggest problems inherent in MHI research is that it is difficult, if not impossible, to prove that observed deficits are the result of injury [27]. For instance, it may be that if deficits exist prior to injury, they might somehow predispose an individual to MHI. Because many of the deficits observed after MHI involve attention [92], perhaps a general lack of attention will leave the individual vulner-able to accidents resulting in MHI. Animal studies [100–103], post-mortem exam-inations of the brains of individuals with MHI [104–106] and neuroimaging work [107–109] have helped establish the view that MHI produces diffuse axonal injury. These structural changes are believed to underlie the neurobehavioural deficits observed after MHI; however, the precise relationship between injury severity and neurobehavioural outcome remains obscure. More work is needed in which individuals (e.g. athletes) are tested before and long after sustaining MHI, e.g. [110]. Such work can control for pre-injury cognitive status, as well as help clarify the relationship between injury and outcome.

On a different note altogether, a potential confound in MHI research that has been all but ignored is the amount and quality of sleep participants receive prior to testing. Because MHI is often associated with sleep disturbances (e.g. [23]), and because sleep disturbances have been shown to produce performance decrements in healthy controls (e.g. [111]), sleep problems may significantly alter the performance of both controls and individuals with MHI. Moreover, it is possible that sleep problems may differentially affect the performance of controls and individuals with MHI. The likely pattern of effects here is that individuals with MHI will perform more poorly than controls after equal amounts of sleep deprivation. This idea needs testing. If such evidence is found, individuals with MHI may prove to be at greater risk of traffic and industrial accidents after sleep loss. Sleep may prove to be

another useful variable to distinguish the performance of controls from that of individuals with MHI.

Two additional factors that could account for some of the inconsistencies in the findings listed above are the number of MHIs sustained and the amount and timing of clinical intervention and treatment received by patients with MHI. On the latter note, O'Hara [112] has argued forcefully that patients admitted to hospital with MHI, who undergo immediate neuropsychological evaluation and treatment, will likely exhibit better and faster recovery than will people who do not receive similar medical attention. This might explain the different results obtained by those who maintain that MHI leads to relatively swift and full recovery [17, 38, 77, 79] and others who argue that MHI produces long-term impairment [49, 50, 87]. In the former group of studies, patients with MHI received immediate medical care, while in the latter studies, individuals with MHI often received no immediate medical care.

Regarding the number of MHIs sustained, it is possible that the evidence in favour of persistent neurobehavioural impairment is the result, at least in part, of multiple MHIs. Gronwall and Wrightson [28] have shown that the effects of MHIs tend to be cumulative. As such, multiple MHIs might be associated with more persistent cognitive impairment. Unlike the Dikmen *et al.* [44] and the Levin *et al.* [17] studies, which strictly used patients with a single MHI, most of the studies discussed above showing long-term neurobehavioural deficits failed to mention the prior incidence of MHI. It would be useful to know whether a single uncompli-cated MHI can produce long-term deficits.

Finally, there is a great need for longitudinal assessment (e.g. 5 years or more) of MHI using demanding neurobehavioural tasks. Such a study might help clarify the present picture of PCS, including the detection of possible malingering and the establishment of symptom development and abatement. By following a large cohort of individuals with MHI (e.g. $n = 200$) and a group of matched controls, investi-gators may be able to identify symptoms that are most likely to present problems for the MHI group during recovery. Preferably, such a study would include both symptom checklists/questionnaires administered to both groups, as well as cognitive and neurobehavioural performance measures. These measures could be given every few months the first year and every 6 months thereafter, bearing in mind the possible confounds due to practice. A longitudinal assessment of this nature may help delineate the path of recovery after MHI. Indeed, Lezak [113] has argued for more attention to be paid to the longitudinal aspects of recovery from traumatic brain injury

### *Conclusions about long-term impairment after MHI*

There is suggestive evidence for long-term (up to 30 years) neurobehavioural impairment associated with MHI, but the evidence is not strong. While it is now virtually uncontested that the majority of mild head injuries result in good recovery, it is still uncertain what deficits persist in which people and why. Also, it is unclear what exactly is meant by the term 'good recovery'. Because 'good recovery' does not imply the absence of cognitive deficits, it is difficult to determine whether the recovery is physiological, psychological or psychosocial (e.g. compensation and coping), or a combination of these factors. Additional work will help clarify the present picture regarding the length and extent of recovery following MHI.

*Rehabilitation and clinical practice*

Posthuma and Wild [114] maintain that MHI patients exhibit acute distress over their injury because: (1) they are more aware of their injury than are severely head injured patients, and/or (2) they have higher expectations of themselves because they do not view themselves as significantly impaired. Regardless of the reason, persons with MHI may encounter grave difficulty adjusting after injury. In a recent review, King [26] forcefully argues that anxiety reduction is of paramount importance in the treatment of PCS. Minderhoud *et al.* [47] mention the benefits of information and encouragement in the amelioration of PCS symptoms caused by MHI; however, Wade *et al.* [15] recently conducted a large scale randomized control trial and found that early psychological intervention (e.g. information and encouragement) did little to reduce social morbidity and severity of PCS after MHI. Mateer *et al.* [69] stress the importance of prospective memory assessment (e.g. remembering to keep appointments, return phone calls, take medications, etc.) in head injury, while Mateer and Mapou [98] mention the potential effectiveness of repeated practice on adaptive cognitive functioning.

Finally, Mittenberg and Burton [116] randomly selected 470 US neuropsychologists and asked them a variety of questions regarding the treatment of PCS. One hundred and sixty-five respondents endorsed the following five treatments as most effective: (1) education (82%); (2) support/reassurance (74%); (3) graded exposure to activity (56%); (4) antidepressant medication (45%); and (5) cognitive restructuring (44%). Overall, respondents chose psychological interventions as more effective than pharmacological treatments, although the authors conclude that these two treatments were viewed as interdependent.

On a different note, O'Hara [112] cautions against the tightening of research methodology on MHI where those individuals who need to be studied most (e.g. patients with psychiatric problems, previous head injuries, delayed diagnosis) will be overlooked. O'Hara contends that the exclusion of such cases:

> '...leaves rehabilitation professionals...in a near vacuum...explaining "the exceptions to the rule" to dubious colleagues, family members, employers and litigators' (p. 32).

This raises a potential dilemma for MHI research. While few would argue against the benefits of stricter methodologies, including more consistency in MHI classification, such tightening of procedures may limit the generalizability of the research. If O'Hara is correct in her plea for the inclusion of the same patient groups that serve to muddy the subject pools in MHI research, then it might be useful to separate MHI into subgroups based on the presence of previous head injury, psychiatric disturbance, etc.

In addition to these recommendations, several authors have emphasized the need for doctors, clinicians and family members to be more aware and accepting of the subtle dysfunctions that may possibly result from MHI. A noteworthy report comes from a neurosurgeon who suffered a MHI in a ski accident [117], and who subsequently experienced cognitive sequelae that he believed might be difficult to identify and substantiate. Gronwall's [24] admonition that 'The cause of the impairment is less important than how it affects the patient's total ability to function' (p. 257) may help raise the sensitivity level of those interacting with and treating individuals with MHI.

Several of the aforementioned tactics may help reduce the stress caused by PCS. Van Zomeren and Van den Burg [118] have argued that PCS is mainly due to a stress reaction from a chronic inability to cope after head trauma. The inability to cope arises from one's continuous attempts to compensate for cognitive deficits. Whether or not Van Zomeren and Van den Burg are correct, sensitivity and informative counsel on the part of clinicians and doctors will undoubtedly aid patients in their recovery from MHI. Andrasik and Wincze [119] remind clinicians that there is no one treatment for MHI, and that treatment must be tailored toward the person's specific PCS complaints.

O'Hara [112] emphasizes a comprehensive 'whole person' treatment approach to PCS which includes a cognitive/teaching component, an emotional/processing and resolving component and a behavioural/skills-building component. Gronwall [24] reminds clinicians and case managers that the first month is the most critical in the management of MHI. During this period, patients and their families should receive education, information and support.

Finally, a recent supplement in the *Archives of Physical Medicine and Rehabilitation* [120] outlines methods for developing a national information system for traumatic brain injury. The system would include, among other things, a database to provide information regarding prognostic indicators in medical rehabilitation and a population-based follow-up system calling for the random selection and follow-up of 20% of patients with mild to moderate head injury. If successful, this system could provide an invaluable source of information to investigators of MHI, doctors and clinicians, and to individuals with MHI.

### Future directions

From this overview of the MHI literature, it is apparent that additional work is needed. Why do we not have a clearer picture of MHI and its sequelae? Methodological differences with respect to sampling populations, lack of controls, symptoms checklists and neurobehavioural measures, repeated testing on the same tasks, and widely fluctuating definitions of MHI have served to obfuscate understanding of MHI. How good are we at predicting the sequelae associated with MHI? Although most people will exhibit little to no overt signs of dysfunction, especially long after MHI, subtle impairment may lie undetected.

Several methodological issues must be addressed and many possible avenues are open to investigation. The following points may help clarify understanding of MHI and its sequelae:

(1) It is imperative that MHI researchers adopt a more precise, universally accepted definition of MHI. Within this definition, it is critical that multiple independent variables be assessed, such as the number of previous MHIs, age, litigation, presence of cognitive complaint (PCS), psychiatric problems, and the amount and quality of sleep prior to testing.

(2) More research should be performed on unhospitalized cases of MHI. These are by far the majority of MHI cases; thus, they deserve more attention.

(3) There is a need for more meta-analytic studies of MHI and neurobehavioural outcomes.

(4) In an attempt to clarify the relationship between subjective cognitive complaint and objective neurobehavioural deficit, MHI subjects should be separated into subgroups based on the presence or absence of cognitive complaint. Controls should then be matched to MHI subjects in terms of age, sex, education and cognitive complaint.

(5) A large, standardized neurobehavioural assessment battery, including standardized PCS inventories, is needed. Such a battery, or a staggered group design in which a single experimental group is divided into subgroups, could help researchers avoid re-testing MHI samples on the same tests, thereby eliminating practice effects.

(6) More demanding measures to detect subtle cognitive deficits in divided attention and in information processing speed and capacity should be developed, standardized and used.

(7) There is a need for more studies examining sequelae 1 year or more after injury.

(8) More longitudinal work is needed.

(9) There is a need for more prospective studies which include pre-morbid cognitive status. Such work can help reduce the number of factors associated with long-lasting PCS complaints and neurobehavioural impairment.

(10) Regarding rehabilitation and clinical practice, anxiety reduction through information, support and encouragement may help individuals deal more effectively with MHI.

Finally, it is important that health professionals and laypersons alike begin to accept the notion that MHI may not be such a mild form of injury after all. Indeed, Jane *et al.* [100] say of the pervasive nature of MHI that:

'…from a sociological point of view, it is obviously necessary to reevaluate our society's somewhat permissive attitude regarding these injuries' (p. 99).

In this vein, there is a need for more acceptance of the possibility that even uncomplicated MHI can lead to long-lasting, if not permanent, neurobehavioural deficits. The notion that 'minor [head] injuries are compatible with full intellectual recovery' [61 (p. 308)] needs to be redressed. The opinion that an uncomplicated MHI can lead to permanent sequelae is not popularly held. Indeed, 20 years after Lishman's comment, Van Zomeren and Brouwer [121] have come to largely the same conclusion. The evidence from this review indicates that MHI may be associated with long-term, subtle neurobehavioural impairment. Nonetheless, further work is required to determine whether this is, in fact, the case.

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