Mild traumatic brain injury and Postconcussion Syndrome: a neuropsychological perspective

W Huw Williams,[1] Seb Potter,[2] Helen Ryland[1]

[1]School of Psychology, Washington Singer Laboratories, Exeter University, Exeter, UK
[2]Lishman Brain Injury Unit, Maudsley Hospital, Denmark Hill, London, UK

Correspondence to
Professor W Huw Williams, School of Psychology, Washington Singer Laboratories, Exeter University, Exeter EX4 4QG, UK; w.h.williams@exeter.ac.uk

Received 17 August 2009
Revised 16 December 2009
Accepted 17 December 2009
Published Online First 27 August 2010

ABSTRACT

Symptoms of mild traumatic brain injury typically resolve within days or weeks. However, a significant group of patients may report symptoms of Post-concussional Syndrome (PCS) weeks, months and years postinjury. This review presents an overview of the pathogenesis, diagnosis and treatment options for PCS. The authors review the evidence for factors that may predict such symptoms. At early phases, there are associations between neurological signs and symptoms, neurocognitive functions and self reports. Over time, such associations become less coherent, and psychological issues become particularly relevant. An accurate understanding of neurological and psychosocial factors at play in PCS is crucial for appropriate management of symptoms at various points postinjury.

INTRODUCTION

Mild traumatic brain injury (MTBI) is a major public health issue. Around 80% of all traumatic brain injuries (TBIs) are mild, and of these, up to 15% may be associated with persisting symptoms.[1 2] It is unclear whether neurological or psychological factors account for such problems. We shall, first, provide an overview to issues in classification and diagnosis of MTBI and Post-Concussion Syndrome (PCS). We will then review the evidence for associations between neurological status and neuropsychological functions, and for psychological mediators of outcomes. We then provide guidance for assessment and intervention post-MTBI.

For the purpose of this review, we will use the terms MTBI for the initial injury and acute effects, and PCS for persistent symptoms (over weeks, months and years). Also, when referring to studies, we shall mostly use the terms used by authors of studies for their population of interest.[3]

MTBI and PCS: issues in classification and diagnosis

Epidemiology

TBI is a leading cause of death and disability, and accounts for a significant proportion of life-years of disability.[4] The yearly incidence of TBI is 180–250 per 100 000 people in the USA[5] and 229 per 100 000 in England.[6] Risk factors are age (very young (under 5), adolescence and young adulthood, and older age), male gender, urban dwelling and lower socio-economic level.[7] Common causes include road accidents, falls, sporting injuries and assaults. In non-sporting injuries, alcohol and/or drug influence is a key contributory factor.[8] In non-western areas, rates are likely to be very high and set to rise substantially.[9]

Definitions and classification of MTBI

MTBI is 'classically defined as an essentially reversible syndrome without any detectable pathology'[10]. Immediate symptoms of MTBI include headache, dizziness and nausea, as well as physical signs which may include unsteady gait, slurred speech, poor concentration and slowness when answering questions.[11] Recovery following MTBI within sports is rapid, with most acute symptoms resolving within hours, and then, typically, a person being symptom-free by around 10 days.[12] Recovery of functions across domains in patients may be differential—with physical and cognitive symptoms being less present that emotional symptoms (irritability, anxiety) at 6 weeks postinjury.[13] However, headaches appear to be relatively common at such a later follow-up. MTBI has a variety of clinical indicators, such as GCS of 13 or above,[14] post-traumatic amnesia (PTA) of no more than 24 h[15 16] and neurological signs such as double vision, headache, etc.[17–19] A recent study indicated that PTA is more effective than GCS for predicting behavioural outcomes at 6 months postinjury.[20] Criteria for diagnosis of MTBI are available from the American Congress of Rehabilitation Medicine[21] and the World Health Organization.[22] We also note that there is ongoing debate over whether MTBI is synonymous with 'concussion' or not.[2 23] It seems that MTBI and concussion have been used interchangeably,[24] although the latter is more commonly used in sports medicine, and MTBI in general medical contexts.

Relationship between MTBI and PCS

PCS is a constellation of symptoms in physical (eg, fatigue, headaches), cognitive (eg, difficulties with concentration and memory) and emotional (eg, irritability, anxiety) domains that persist for weeks, months and even years after an MTBI.[25] There are estimates of around 15% of individuals having such persistent symptoms.[26] In one study, it was reported that 47% of young adults with mild head injuries experienced moderate to severe disability at 1 year postinjury.[27] Methodological issues, such as recruitment bias,[28] may lead to such figures being overestimates.[29] For example, it was recently shown that attention-deficit/hyperactivity disorder may be a risk factor for early head injury.[30] This suggests a degree of reverse-causality for presence of symptoms. Furthermore, three interrelated issues cloud an understanding of the true scale and scope of the problem. First, there is disagreement between diagnostic systems on key criteria; second, lack of specificity of symptoms; third, as indicated above, a lack of clarity over pathogenesis.

There are two main diagnostic systems for PCS-ICD (F07.2) and (as postconcussional disorder) DSM-IV (research). While there is general agreement across the two sets of criteria in terms of general symptoms, within DSM-IV there are additional requirements for objective cognitive impairment and disturbance in social or occupational functioning and specification of threshold of 3 months for symptoms to persist.[31–34] Not surprisingly, a comparison of prevalence rates post-TBI of PCS according to each criteria revealed a striking difference between them, with DSM-IV criteria being met by 11% and 64% by the ICD criteria.[34]

Symptoms of PCS are not clearly specific to PCS, with a high rate of similar symptoms in non-brain-injured such as orthopaedic patients.[35] Overlap of symptoms with other clinical populations is considerable, including individuals with depression,[36] pain[37] and whiplash symptoms.[38] Although there is a lack of specificity to PCS, there does appear to be sufficient evidence of it being a clinical phenomenon that is sensitive to measurement. There is, for example, considerable consistency in symptoms across a range of PCS checklists and questionnaires,[39] and the structure of symptoms in cognitive, emotional and physical domains is relatively consistent across a variety of studies using different questionnaires and in different populations.[40] Assessment of the severity and impact of symptoms, using questionnaires such as the Rivermead Post Concusion Symptoms Questionnaire (RPCSQ), has been advocated, particularly as the presence and severity of symptoms are associated with quality of life[41] and return to work.[42]

There is much debate over whether persistent symptoms are 'driven' by neurological and/or psychological factors, and how premorbid issues may influence both sets of factors.[10 22 43 44] Female gender, previous psychiatric history and previous head injury[22] have been linked to poorer outcome, although much of the literature has been critiqued both conceptually and methodologically.[29] Diathesis-stressor models have been proposed to combine both 'organic' and 'psychogenic' factors for the development of PCS.[26 45 46] They typically have at their centre the idea proposed by Lishman[47] that early physiogenic mechanisms may be responsible for early PCS symptoms, but 'vicious cycles' that emphasise non-organic, psychological factors may be responsible for their persistence over time. For example, King[46] outlines a number of potential 'windows of vulnerability,' from early worries about symptom longevity and dissonance between injury severity and early symptoms.

There is, therefore, uncertainty over how, and why, MTBI leads to PCS. What is clear is that acute indicators of injury severity, and concomitant neurocognitive dysfunction, may be important considerations for understanding later presentation of PCS symptoms. We will now review evidence for neurocognitive sequelae to MTBI. We shall then explore whether there is any evidence to link such symptoms with neuroradiological data, principally imaging. We shall then consider how, and why, psychological factors may be related to persistence of symptoms.

Neurocognitive consequences of MTBI

There are two main types of neurocognitive studies, those of athletes at 'risk' of injury from contact sports, and of patient groups—typically attendees at emergency departments. Studies are focussed on determining the presence of neurocognitive symptoms for early diagnosis of MTBI and for monitoring recovery for guiding return to activities.[48–51] Systems include traditional neurocognitive measures and/or computerised tests[52].

There are important distinctions to be drawn between sports and patient group studies. First, athletes may 'down play' symptoms to enable return to play.[53] Second, athletes may be assessed as being concussed for relatively minor disturbances in consciousness compared with patients. Third, patients may have a greater heterogeneity of issues to consider, such as premorbid factors (educational, socio-economic, etc). Fourth, the nature of the injury may mean very different degrees of biomechanical forces at play—for example, acceleration and deceleration forces are typically far higher in road incidents than in sports.

Sports

There are many sports 'return to play' studies that indicate that single concussive episodes leave no lasting neurocognitive consequence.[54] A meta-analytical review of postacute neurocognitive effects of concussion in sports by Belanger and Vanderploeg[53] identified 21 of 69 studies between 1970 and 2004 that met key inclusion criteria (such as including a control or baseline comparisons). They reported that there were mild–moderate effects of concussion in the first 24 h on global measures of functioning, and larger deficits on memory. However, there was full resolution of functions by 7–10 days postinjury. They did note, however, that practice effects may have led to an underestimation of concussion effects. They also noted that studies that excluded prior 'head injury' had a smaller effect size than those that did not exclude such athletes.

A landmark study in the area by McCrea *et al*[12] illustrates key points regarding recovery trajectories. They followed up a concussed group (n=94) and an uninjured control group (n=56) of American college football players selected from a cohort of 1631. They were tested preseason, and then immediately after injury. They were subsequently tested at 3 h, then at 1, 2, 3, 5, 7 and 90 days postinjury. By 7 days, there was no difference between the concussed and non-concussed group on the Standardised Assessment of Concussion (SAC, which addresses orientation, balance and coordination, neurological signs and delayed memory). However, it is noteworthy that the concussed group performed 'less well' than controls on verbal fluency 7 days and 90 days post, and that 10% of players needed more than a week for symptoms to resolve. Importantly, there was no evidence of 'lingering symptoms,' or cognitive impairments, at 90 days. Assessment using computerised systems has shown a similar resolution of symptoms, albeit with some variation in recovery. Iverson GI, Brooks BL, Collins *et al*[55] followed up concussed athletes (n=30) from baseline at 1–2 days, 3–7 days and 1–3 weeks post using Immediate Post-Concussion Assessment and Cognitive Testing.[56] The athletes' scores on a range of measures (memory, speed, reaction time) were significantly reduced at day 1, but there were significant improvements by 5 days postinjury, although, at 10 days postinjury, 37% of athletes had two or more composite scores that were lower than preseason. Two or more existing head injuries, or the presence of headaches, were suggested to be associated with compromised recovery. Collins *et al*[57] also identified repeat injury as related to poorer outcomes. In a sample of 393 American Football players, assessed on annual baselines, they found that a history of multiple concussions was associated with lowered performance for divided attention and visuomotor speed. Similarly, Wall *et al*[54] showed that jockeys with repeated concussions, compared with those concussed once, were less efficient on tasks involving executive functions and attention. Younger age appeared to account for this discrepancy, suggesting that either younger age of injury, or greater repeat injury within a shorter time span, may be important considerations when gauging recovery.

In light of a possibility that multiple MTBIs may have a cumulative effect, it is important to note that such effects are not consistently found. Indeed, it has been argued that the cross-sectional research designs typically employed in such studies do not allow confident causal inferences to be made between multiple injury and current status.[58] Furthermore, some prospective studies have not indicated increased impairment from cumulative injury. For example, Moriarty *et al*,[59] in a prospective study of 82 amateur boxers participating in a 7-day tournament, found no evidence of short-term cognitive impairment. Importantly, though, they did find that there was cognitive dysfunction in those who had had their bout stopped by the referee.

To summarise then, in sports it appears that the effects of a single MTBI typically resolve quickly, although there can be delayed recovery in some, but there appears to be a very low risk of long-term effects. There is, though, preliminary evidence of risk of cumulative damage from repeat injury.[23 60]

Patient groups

One of the earliest, well-controlled, patient studies—comparing 22 participants with MTBI versus 19 matched controls—revealed that a single minor head injury in persons with no prior compromising condition was associated with mild but 'probably clinically non-significant difficulties at 1 month after injury.[61] Neurocognitive problems were largely related to concentration and new learning but were not apparent at 1 year postinjury. It was noted that disruptions of everyday activities were extensive when other 'system injuries' were also present. In a meta-analytical review of neurocognitive studies (from 1970 to 2004) of patients with MTBI, Belanger and colleagues[62] reported that, of eight cognitive domains, with unselected samples (recruited prospectively and not based on symptoms), the largest effect sizes were for verbal fluency and delayed memory. Neurocognitive outcomes of those who were 'unselected' were equal to control participants at 90 days postinjury. However, in those where litigation was involved, the average effect size increased after 90 days postinjury. Symptom validity tests did not explain these effects. In another meta-analysis, Schretlen and Shapiro[63] reported that the cognitive performance of MTBI patients could not be distinguished from matched controls at 1 month post-injury. Caution has been expressed regarding acceptance that meta-analyses confirm that MTBI leave no lasting consequence. Pertab *et al*[64] noted that there was significant statistical heterogeneity in the effects sizes of neuropsychological measures used, criteria adopted for defining MTBI and populations, and mechanisms of injury of the MTBI samples. Furthermore, lasting neurocognitive deficits have been shown within subsets of neuropsychological measures suggesting that a 'likelihood of mTBI individuals that have lingering symptoms exists within the larger group of individuals without symptoms'[64] (p 504).

Relationships between imaging and neurocognitive processing

There is emerging evidence linking neurocognitive dysfunction to neuroimaging findings post-MTBI. We shall now review the strength of such relationships. A neurocognitive study of outcomes at 2 weeks in a group of patients with 'day of injury' CT scan showing 'abnormalities' (hence 'complicated,' compared with uncomplicated), showed that complicated MTBI was associated with worse performance.[65] Executive and attention functions were particularly affected. However, effect size was smaller than predicted, and logistical regression indicated that performance was more similar than different between the groups. In a further study, 20 'complicated' MTBI (based on CT scan results or GCS falling between 13 and 15) and 'uncomplicated,' well-matched MTBI patients were compared on neurocognitve tasks within days of injury.[66] The complicated MTBI performed worse on memory and verbal learning. In a recent study of 'complicated' patients (abnormal CT scan within 24 h of injury) and non-patient controls, it was found that the complicated group were poorer on speed, attention and executive functions at 1 month post, but by 3 months, speed and divided attention were much improved. However, sustained attention and aspects of executive functions were still not fully resolved.[67] In an MRI study of neuropsychological functions in 30 MTBI patients, compared with matched controls, it was found that patients with traumatic lesions performed more poorly on neurocognitive tasks within 4 days of injury.[68] The 'complicated' group differed from controls on immediate and delayed recall, and on complex reaction time. In another MRI study, with imaging 1–3 days postinjury, with 80 patients from an Emergency Department, abnormalities were found in 26—although only in five were there signs attributable to the injury.[69] There was a weak correlation between MRI abnormalities and neuropsychological dysfunction (memory, attention and executive skills) in acute period. However, there was no difference in terms of whether those with normal, or abnormal scans, returned to work. In an MRI with single-photon emission CT study, it was found that 57% and 61% of 21 and 18 (GCS on average 14.48) had abnormalities on MRI and SPECT imaging respectively within 5 days after injury.[70] There was also associated brain atrophy at 6 months. Those with complicated MTBI were slower on reaction-time tasks.

In contrast, a prospective study over 1 year in Norway of 115 patients with mild (separated into 'presence' or 'absence' of abnormality—including use of MRI), moderate and severe TBI found that the mild group reported greater PCS symptoms at 3 months but not at 1 year post.[71] Also, at 3 months, there was no difference in the mild group between those meeting PCS criteria between those with and without intercranial pathology as detected by MRI. Most recently, diffuse tensor imaging (DTI) MRI has been developed to measure the integrity of white-mater tracts and critical structures. Within the acute and subacute period postinjury, there are preliminary data suggesting involvement of the internal capsule and corpus callosum.[50 72] In that DTI provides a measure of axonal injury, not death, it is suggested that it may become more relevant for prognostic purposes in future.[73] These studies therefore provide some evidence linking early neurological scan data, neurocognitive dysfunction and delayed recovery. However, the evidence is not compelling regarding later PCS and social role outcomes—such as return to work.

Another means to indicate whether MTBI has any effects on neurological systems linked to cognition is to establish whether there are any changes in activation patterns postinjury. Functional imaging studies have indicated that there may, indeed, be differential patterns of activity following concussion. In an fMRI study of 18 MTBI patients at 1 month post, there were significant changes in activation patterns.[74] The patient group, compared with controls, had differential complexity in activation patterns on working memory tasks—particularly in right such as in bilateral frontal and parietal areas. In an fMRI study using a working memory task with concussed athletes, it was found that several (of 15 'symptomatic' participants), who had sustained their last injury from 1 to 14 months previously, had differential activity patterns compared with a control group.[75] It was noted that only one had shown abnormality on standard structural MRI. The region of interest (ROI) identified in

controls involving self-monitoring on a working memory task was mid-dorsolateral prefrontal cortex and anterior insula. On fMRI, the 'symptomatic' participants showed weaker activity in the ROI identified in controls and increased activity outside the ROI. Chen and colleagues[76] conducted a further fMRI imaging on two groups of athletes self-rated for severity of symptoms, 'low' (n=9), 'moderate' (n=9) and a further control group, with no concussion in the past year (n=10)—with a working memory task. Participants were seen at least 1 month (and on average 5 months) postinjury. All participants had normal MRI scans. The moderate group showed less activation in the ROI identified in controls for the tasks—the prefrontal cortex—and both concussed groups had increased activation in temporal area. Associations between neurocognitive performance and neurological activation have recently been investigated over a long term with TMS.[77] In this study, 21 healthy, uninjured, athletes were compared with 19 former athletes who had had concussions 30 years prior to testing. The authors reported that the concussed group were poorer on tasks of memory and response inhibition, and had a longer duration of Cortical Silent Period (CSP) on TMS. There are important limitations that relate to a number of these studies. First, there is insufficient information as to whether those who displayed a differential activation pattern may have had premorbid factors relevant to such functions. Second, particularly at long-term postinjury, there is a possibility that participants may have been inaccurate in their reports on the severity and number of MTBIs. Third, numbers of participants tend to be low, and retention rates for follow-up studies are particularly low. Consequently, samples may not be representative of the MTBI population.

RECOVERY OF NEUROCOGNITIVE FUNCTIONS: SUMMARY

It may be helpful to consider MTBI as a spectrum disorder, with the 'dosage' of injury—depending on biomechanical factors—being important in setting a context for recovery and/or resolution of symptoms. It appears that concentration, attention, executive function, memory and complex attention are all, to a degree, affected but that there is differential recovery of these functions. Sustained attention and executive functions are subject to greater delay. Such problems recover rapidly in the context of sports, but there is a tendency for symptoms to linger in a subgroup of patients. Of particular note, studies linking brain imaging and neurocognitive functions suggest two levels of neurological involvement. At one level, there may be functional changes in brain activation where, for the same cognitive task or demand, there is a differential 'load' in those who are concussed.[73] Related symptoms may resolve readily in such cases. At another, level, there may be structural changes, particularly when there are signs of 'complicated' injury which may be associated with delayed recovery. Signs of potential for complicated injury appear to be: abnormal imaging findings, prior MTBI, greater LOC/PTA, longer duration of initial symptoms and younger age. Subjective complaints may be more closely associated with neurocognitive performance early on, but it appears that there is a loosening of associations between neurological profile, neurocognitive functions and subjective self-reports over time.

Psychological mechanisms and persistent postconcussional symptoms

The evidence that MTBI may be associated with PCS is, therefore, equivocal. In some cases, there may be a biological vector that is linked to outcomes. In others, psychological variables may have a key role to play in genesis and/or maintenance of symptoms. These may be in two overlapping areas: symptoms may reflect psychological reactions better conceptualised within a psychiatric nosology, and the role of more idiosyncratic appraisals and attributions of symptoms after MTBI.

Psychological reaction

It is well established that there are elevated rates of psychiatric comorbidity in PCS groups.[78] This may represent a response to persisting effects of brain injury on cognition and associated limitations in functioning. However, the role of Post-Traumatic Stress Disorder (PTSD)—in the context of other mood issues, particularly depression—has emerged as a critical issue in explaining PCS.

It had been thought that TBI and PTSD were incompatible: without a memory of the event, the survivor of trauma might not have source material for intrusive thoughts to drive avoidance behaviour.[79] However, a number of potential mechanisms have been identified for PTSD post-TBI—such as islands of memory, confabulated memory, external causal attributions and fear conditioning.[80 81] Although rates of PTSD after TBI vary greatly between studies (from 0 to 48% prevalence in one review),[82] there is accumulating evidence for its presence at various levels of severity.[83] For example, a large-scale study of 920 trauma patients in Australia by Bryant and colleagues showed that MTBI patients were more likely to develop PTSD compared with non-TBI controls (11.8% vs 7.5%).[84] They also found that a longer PTA was a protective factor.

Importantly, it appears that PTSD not only occurs post-MTBI but can contribute to PCS symptomology. For example, following general trauma or mild–moderate TBI, rates for persisting PCS at 3 months were over three times higher for individuals with PTSD,[85] while symptoms of depression, anxiety and PTSD at 7–10 days post-MTBI predicted PCS symptoms at 3–6 months later (see also King[86] and King et al[87]). It seems that PTSD not only may coexist but also may be a mediator of outcomes post-MTBI. Two recent studies with military populations provide insights into this process. In a retrospective review of 2525 US soldiers 3–4 months after their return from deployment to Iraq, post-concussional symptoms were elevated in individuals exposed to MTBI compared with other injuries.[88] However, PTSD, along with depression, emerged as major factors mediating the relationship between the two. In a study by Belanger *et al*[89] with 225 participants, predominantly active duty or veteran military personnel, it was found that those with MTBI endorsed more PCS symptoms than those with moderate to severe injuries. However, when controlling for variance due to the effect of PTSD, the MTBI group were no different from the other groups—across all three domains of affective, somatic and cognitive symptoms. These findings suggest that there is a role for PTSD in explaining PCS post-MTBI. It may be that PTSD decreases attenuation of stress response by contributing to a neurogenic process for its persistence and/or lessening coping skills to deal with problems.[83] However, it may be that PTSD is simply misinterpreted as PCS, particularly as the relationship between PTSD and PCS is complicated by overlapping symptoms.[90] It is also worth noting that PTSD questionnaires may, in turn, lack validity in that they may be sensitive to the effects of non-traumatic stressors and to personality traits such as negative affectivity.[91] As noted by Stein and McAllister, 'the literature (on PCS and PTSD) is far from consistent and serves mainly to raise new, challenging questions about mutual pathophysiology'[83] (p. 768).

Attributions and expectations

There may be a role of 'expectation as aetiology' in maintaining symptoms post-MTBI. Individuals with persistent PCS may tend to under-report normal 'postconcussional' symptoms they experienced prior to their head injury (described as the 'good old days' phenomenon[92]), while uninjured controls can report expecting postconcussional symptoms after reading head-injury vignettes.[93] The role of other aspects of symptom appraisals in the development of persistent symptoms was also indicated by Whitaker and colleagues[94] in a longitudinal study. Individuals who initially viewed their injury as having serious and persisting negative consequences soon after injury were shown to have a greater presence of symptoms at 3 months.

Involvement in a medicolegal or compensation claim may well lead to a context for expectations to be modulated. There remains a consistent finding of involvement in medicolegal action and poorer outcomes.[29 95] However, as noted above, this association should not be presumed as being synonymous with malingering: aspects of being involved in a medicolegal process, from the repeated rehearsal of symptoms[47] to an emphasis on blame and culpability,[96] may play a role. In this context, it is interesting to note the finding that individuals involved in tort insurance claims had slower recovery trajectories compared with no fault claimants.[97] There is, however, evidence that at least a proportion of individuals with persisting difficulties after MTBI can show evidence of at least suboptimal effort on formal neurocognitive assessment.[98] Nonetheless, other 'psychological' variables may impact on neurocognitive test performance in more subtle but significant ways. Suhr and Gunstad[99] for example, administered a battery of measures of memory, attention and executive functioning to two groups of undergraduates who had reported a history of MTBI. One group had their attention drawn to their head injury and typical cognitive effects prior to testing. This 'diagnosis threat' group showed significantly worse performance on a number of commonly used tests, with this effect apparently independent of mood or effort.

Implications for psychosocial treatment

While MTBI may set the conditions for PCS to occur, there does appear to be a role for psychological mechanisms in persistence of symptoms—which provides potential avenues for treatment. The majority of the current literature on treatment of persistent PCS primarily focuses on the benefits of early interventions (typically in the first week to month postinjury) that focus on prophylactic prevention of persistent symptoms.[100] Such interventions typically provide individuals with information about PCS as a common but transient phenomenon after MTBI. A meta-analysis of five studies up to 1997[101] found a modest, positive effect size average of 0.32 in terms of reduction in persistent PCS, and similar results have been replicated subsequently.[102 103]

In contrast, systematic studies for psychosocial interventions with persistent symptoms remain limited, although single case studies or trials with limited controls do provide some evidence of improving symptoms with use of cognitive behavioural approaches such as for dizziness,[104] headache,[105] depression,[106] anger[107] and PTSD.[108] One randomised control trial with mild–moderate TBI individuals (n=20) used intensive individual cognitive rehabilitation tasks (eg, remediation and compensation for attentional difficulties) with cognitive behavioural elements (eg, modifying coping strategies). Improvements were found for affective symptoms and attention.[109] This trial highlights the tension for clinicians between seeing PCS as related to a brain injury that needs to be compensated for, or as being largely maintained by psychological mechanisms that may be managed.[110] However, these two positions might also be viewed as complimentary and capable of being integrated.[111 112] Practising tasks involving sustained attention might be framed as a way of 'boosting' attentional resources, or desensitisation to improve tolerance to fatigue, and/or as a method of testing and challenging concerns about competence and abilities to learn new skills. The likely heterogeneity of persistent symptoms may make defining a particular treatment protocol difficult. Moreover, clinicians must be vigilant for identifying relevant mood-related issues that might respond better to specific treatment, such as Trauma Focussed Cognitive Behaviour Therapy (TF-CBT) for PTSD.[79]

CONCLUSION

The aphorism commonly attributed to Hippocrates that 'No head injury is too severe to despair of, nor too trivial to ignore' reminds us that caution is needed in the care of any head injury. Caution is needed to ensure that patients are not provided with scenarios in which they imply that their lives are necessarily and forever shattered following MTBI: with any TBI, there is a need for a careful formulation of the neurological and psychosocial issues that may be at play. We have argued that there are associations between acute indicators of injury severity, particularly when there are signs of 'complicated' injury, and early neurocognitive dysfunction, which may indicate delayed recovery. However, psychological factors are important in persistence of symptoms of PCS. In particular, mood disorders such as PTSD, as well as appraisals and attributions of symptoms, are likely to play a significant role. Crucially, patients and relatives need guidance to ensure that recovery is maximised, and any risks are managed.

Funding WHW Economic and Social Research Council (ESRC) Grant Res-062-23-0135 HJR; CASE-ESRC studentship with British Horseracing Authority (BHA).

Competing interests None.

Provenance and peer review Commissioned; externally peer reviewed.

REFERENCES

1. **Kraus F,** Chu LD. Epidemiology. In: Silver JM, McAllister TW, Yudofsky SC, eds. *Textbook of traumatic brain injury*. Washington DC: American Psychiatric Publishing, 2005:3–26.
2. **Ruff RM,** Weyer Jamora C. Myths and mild traumatic brain injury. *Psychological Injury and Law* 2005;**2**:34–42.
3. **Bigler ED.** Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. *J Int Neuropsychol Soc* 2008;**14**:1–22.
4. **Fleminger S,** Ponsford J. Long term outcome after traumatic brain injury. *BMJ* 2005;**331**:1419–20.
5. **Bruns J,** Hauser WA. The epidemiology of traumatic brain injury: a review. *Epilepsia* 2003;**44**:2–10.
6. **Tennant A.** Admission to hospital following head injury in England: incidence and socioeconomic associations. *BMC Public Health* 2005;**5**:21.
7. **Yates PJ,** Williams WH, Harris A, *et al*. An epidemiological study of head injuries in a UK population attending an emergency department. *J Neurol Neurosurg Psychiatry* 2006;**77**:699–701.
8. **Kolakowsky-Hayner SA,** Kreutzer JS. Pre-injury crime, substance abuse, and neurobehavioural functioning after traumatic brain injury. *Brain Inj* 2001;**15**:53–63.
9. **Hyder AA,** Wunderlich CA, Puvanachandra P, *et al*. The impact of traumatic brain injuries: a global perspective. *Neurorehabilitation* 2007;**22**:341–53.
10. **Ommaya AK,** Gennarelli TA. Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. *Brain* 1974;**97**:633–54.
11. **McCrory P,** Johnston K, Meeuwisse W, *et al*. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *Br J Sports Med* 2005;**39**:196–204.
12. **McCrea M,** Guskiewicz KM, Marshall SW, *et al*. Acute effects and recovery time following concussion in collegiate football players. *JAMA* 2003;**290**:2556–63.
13. **Rutherford WH,** Merrett JD, McDonald JR. Sequelae of concussion caused by minor had injuries. *Lancet* 1977;**1**:1–4.
14. **Teasdale G,** Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet* 1974;**2**:81–4.

15. **Cantu RC.** Posttraumatic Retrograde and Anterograde Amnesia: Pathophysiology and Implications in Grading and Safe Return to Play. *Journal of Athletic Training* 2001;**36**:244—248.
16. **Meares S,** Shores EA, Taylor AJ, *et al*. Mild traumatic brain injury does not predict acute postconcussion syndrome. *J Neurol Neurosurg Psychiatry* 2008;**79**:300—6.
17. **Cantu RC.** Return to play guidelines after a head injury. *Clin Sports Med* 1998;**17**:45—60.
18. **Colorado Medical Society.** *Report of the sports medicine committee: guidelines for the management of concussion in sports*. Denver, CO: Medical Society, 2005.
19. **American Academy of Neurology.** Practice parameter: the management of concussion in sports (summary statement). *Neurology* 1997;**48**:581—5.
20. **Tellier A,** Marshall SC, Wilson KG, *et al*. The heterogeneity of mild traumatic brain injury: where do we stand? *Brain Inj* 2009;**23**:879—87.
21. **American Congress of Rehabilitation Medicine.** Report of Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group. Definition of mild traumatic brain injury. *J Head Trauma Rehabil* 1993;**8**:86—7.
22. **Carroll LJ,** Cassidy JD, Holm L, *et al*. Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *J Rehabil Med* 2004;**S43**:113—25.
23. **McCrory P,** Meeuwisse W, Johnston K, *et al*. Consensus statement on concussion in sport: the 3rd International Conference on Concussion in sport held in Zurich, November 2008. *Clin J Sports Med* 2009;**19**:185—200.
24. **Gerberding JL,** Binder S. *Report to congress on mild traumatic brain injury in the United States: steps to prevent a serious public health problem*. Atlanta, GA; Centres for Disease Control and Prevention, 2003.
25. **Ruff RM,** Camenzuli L, Mueller J. Miserable minority: emotional risk factors that influence the outcome of a mild traumatic brain injury. *Brain Inj* 1996;**10**:551—66.
26. **Wood RLL.** Understanding the 'miserable minority': a diathesis-stress paradigm for post-concussional syndrome. *Brain Inj* 2004;**18**:1135—53.
27. **Thornhill S,** Teasdale GM, Murray GD, *et al*. Disability in young people and adults one year after head injury: prospective cohort study. *BMJ* 2000;**320**:1631—5.
28. **McCullagh S,** Feinstein A. Outcome after mild traumatic brain injury: an examination of recruitment bias. *J Neurol Neurosurg Psychiatry* 2003;**74**:39—43.
29. **Carroll LJ,** Cassidy JD, Peloso PM, *et al*. Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *J Rehabil Med* 2004;**36**:84—105.
30. **Keenan HT,** Hall GC, Marshall SW. Early head injury and attention deficit hyperactivity disorder: retrospective cohort study. *BMJ* 2008;**337**:a1984.
31. **Boake C,** McCauley SR, Levin HW, *et al*. Limited agreement between criteria-based diagnoses of postconcussional syndrome. *J Neuropsychiatry Clin Neurosci* 2004;**16**:493—9.
32. **McCauley SR,** Boake C, Pedroza C, *et al*. Correlates of persistent postconcussional disorder: DSM-IV criteria versus ICD-10. *J Clin Exp Neuropsychol* 2007;**30**:360—79.
33. **Kashluba S,** Casey JE, Paniak C. Evaluating the utility of ICD-10 diagnostic criteria for postconcussion syndrome following mild traumatic brain injury. *J Int Neuropsychol Soc* 2006;**12**:111—18.
34. **Boake C,** McCauley SR, Levin HS, *et al*. Diagnostic criteria for postconcussional syndrome after mild to moderate traumatic brain injury. *J Neuropsychiatry Clin Neurosci* 2005;**17**:350—6.
35. **Bazarian JJ,** Wong T, Harris M, *et al*. Epidemiology and predictors of post-concussive syndrome after minor head injury in an emergency population. *Brain Inj* 1999;**13**:173—89.
36. **Iverson GL,** Lange RT. Examination of 'post-concussion-like' symptoms in a healthy sample. *Appl Neuropsychol* 2003;**10**:137—44.
37. **Smith-Seemiller L,** Fow NR, Kant R, *et al*. Presence of post-concussion syndrome symptoms in patients with chronic pain vs. mild traumatic brain injury. *Brain Inj* 2003;**17**:199—206.
38. **Haldorsen T,** Waterloo K, Dahl A, *et al*. Symptoms and cognitive dysfunction in patients with the late whiplash syndrome. *Appl Neuropsychol* 2003;**10**:170—5.
39. **Alla S,** Sullivan SJ, Hale L, *et al*. Self-report scales/checklists for the measurement of concussion symptoms: a systematic review. *Br J Sports Med* 2009;**43**:i3—12.
40. **Potter S,** Leigh E, Wade D, *et al*. The Rivermead post concussion symptoms questionnaire. *J Neurol* 2006;**253**:1603—14.
41. **Stålnacke B-M.** Community integration, social support and life satisfaction in relation to symptoms 3 years after mild traumatic brain injury. *Brain Inj* 2007;**21**:933—42.
42. **Nolin P,** Heroux L. Relations among sociodemographic, neurologic, clinical and neuropsychologic variables, and vocational status following mild traumatic brain injury: a follow-up study. *J Head Trauma Rehabil* 2006;**21**:514—26.
43. **Lishman WA.** *Organic psychiatry: the psychological consequences of cerebral disorder*. 3rd edn. Oxford: Blackwell Scientific, 1988.
44. **Iverson GL.** Outcome from mild traumatic brain injury. *Curr Opin Psychiatry* 2005;**18**:301—17.
45. **Alexander MP.** Mild traumatic brain injury: pathophysiology, natural history and clinical management. *Neurology* 1995;**45**:1253—60.
46. **King NS.** Post-concussion syndrome: clarity amid the controversy? *Br J Psychiatry* 2003;**183**:276—8.
47. **Lishman WA.** Physiogenesis and psychogenesis in the 'post-concussional syndrome.' *B J Psychiatry* 1988;**153**:460—9.
48. **Barth JT,** Alves WM, Ryan TV, *et al*. Mild head injury in sports: neuropsychological sequelae and recovery of function. In: Levin HL, Eisenberg HM, Benton AL, eds. *Mild head injury*. New York: Oxford University Press, 1989.
49. **Macciocchi SN,** Barth JT, Alves W, *et al*. Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery* 1996;**39**:510—14.
50. **Davis GA,** Iverson GL, Guskiewicz KM, *et al*. Contributions of neuroimaging, balance testing, electrophysiology and blood markers to the assessment of sport-related concussion. *Br J Sports Med* 2009;**43**:i36—45.
51. **Echemendia RJ,** Putakian M, Mackin SR, *et al*. Neuropsychological test performance prior to and following sports-related mild traumatic brain injury. *Clin J Sport Med* 2001;**11**:23—31.
52. **Collie A,** Darby DG, Maruff P. Computerised cognitive assessment of athletes with sports related head injury. *Br J Sports Med* 2001;**35**:297—302.
53. **Belanger HG,** Vanderploeg RD. The neuropsychological impact of sports-related concussion: a meta-analysis. *J Int Neuropsychol Soc* 2005;**11**:34—57.
54. **Wall SE,** Williams WH, Cartwright-Hatton S, *et al*. Neuropsychological dysfunction following repeat concussion in jockeys. *J Neurol Neurosurg Psychiatry* 2006;**77**:518—20.
55. **Iverson GI,** Brooks BL, Collins MW, *et al*. Tracking neuropsychological recovery following concussion in sport. *Brain Injury* 2006;**20**:245—252.
56. **Maroon JC,** Lovell MR, Norwig J, *et al*. Cerebral concussion in athletes: evaluation and neuropsychological testing. *Neurosurgery* 2000;**47**:659—72.
57. **Collins MW,** Grindel SH, Lovell MR, *et al*. Relationship between concussion and neuropsychological performance in college football players. *JAMA* 1999;**282**:964—70.
58. **Iverson GL.** Sport-related concussion. In: Schoenberg MR, Scott JG, eds. *The black book of neuropsychology: a syndrome based approach*. 1st Edition., 2011, 430 pages. Hardcover ISBN: 978-0-387-70703-7. Due: 28 November 2010.
59. **Moriarty J,** Collie A, Olson D, *et al*. A prospective controlled study of cognitive function during an amateur boxing tournament. *Neurology* 2004;**62**:1497—502.
60. **Collie A,** McCrory P, Makdissi M, *et al*. Does history of concussion affect current cognitive status? *Br J Sports Med* 2006;**40**:550—1.
61. **Dikmen S,** McLean A, Temkin N. Neuropsychological and psychosocial consequences of minor head injury. *J Neurol Neurosurg Psychiatry* 1986;**49**:1227—32.
62. **Belanger H,** Curtiss G, Demery A, *et al*. Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *J Int Neuropsychol Soc* 2005;**11**:215—27.
63. **Schretlen DJ,** Shapiro AM. A quantitative review of the effect of traumatic brain injury on cognitive functioning. *Int Rev Psychiatry* 2003;**15**:341—9.
64. **Pertab JL,** James KM, Bigler ED. Limitations of mild traumatic brain injury meta-analyses. *Brain Inj* 2009;**23**:498—508.
65. **Iverson GL.** Complicated vs. uncomplicated mild traumatic brain injury: acute neuropsychological outcome. *Brain Inj* 2006;**20**:1335—44.
66. **Lange R,** Iverson GL, Franzen M. Neuropsychological functioning following complicated vs. uncomplicated mild traumatic brain injury. *Brain Inj* 2009;**23**:83—91.
67. **Kwok FY,** Lee TMC, Leung CHS, *et al*. Changes of cognitive functioning following mild traumatic brain injury over a 3-month period. *Brain Inj* 2008;**22**:740—51.
68. **Kurca E,** Sivak S, Kucera P. Impaired cognitive functions in mild traumatic brain injury patients with normal and pathologic magnetic resonance imaging. *Neuroradiology* 2006;**48**:661—9.
69. **Hughes DG,** Jackson A, Mason DL, *et al*. Abnormalities on magnetic resonance imaging seen acutely following mild traumatic brain injury: correlation with neuropsychological tests and delayed recover. *Neuroradiology* 2004;**46**:550—8.
70. **Hofman PA,** Stapert SZ, van Kroonenburgh MJ, *et al*. MR imaging, single-photon emission CT, and neurocognitive performance after mild traumatic brain injury. *Am J Neuroradiol* 2001;**22**:441—9.
71. **Sigurdardottir S,** Andelic N, Roe C, *et al*. Post-concussion symptoms after traumatic brain injury at 3 and 12 months post-injury: a prospective study. *Brain Inj* 2009;**23**:489—97.
72. **Kumar R,** Gupta RK, Husain M, *et al*. Comparative evaluation of corpus callosum DTI merics in acute mild and moderate traumatic brain injury: its correlation with neuropsychometric tests. *Brain Inj* 2009;**23**:675—85.
73. **Belanger HG,** Vanderploeg RD, Curtiss G, *et al*. Recent neuroimaging techniques in mild traumatic brain injury. *J Neuropsychiatry Clin Neurosci* 2007;**19**:5—20.
74. **McAllister TW,** Sparling MB, Flashman LA, *et al*. Differential working memory load effects after traumatic brain inury. *NeuroImage* 2001;**14**:1004—1012.
75. **Chen J-K,** Johnston KM, Frey S, *et al*. Functional abnormalities in symptomatic concussed athletes: an fMRI study. *Neuroimage* 2004;**22**:68—82.
76. **Chen J-K,** Johnston KM, Collie A, *et al*. A validation of the post-concussion symptom scale in the assessment of complex concussion using cognitive testing and functional MRI. *J Neurol Neurosurg Psychiatry* 2007;**78**:1231—8.
77. **De Beaumont L,** Théoret H, Mongeon D, *et al*. Brain function decline in healthy retired athletes who sustained their last sports concussion in early adulthood. *Brain* 2009;**132**:695—708.
78. **Ruff RM,** Jurica P. In search of a unified definition for mild traumatic brain injury. *Brain Inj* 1999;**13**:943—52.
79. **McMillan TM.** Errors in diagnosing post-traumatic stress disorder after traumatic brain injury. *Brain Inj* 2001;**15**:39—46.
80. **McMillan TM,** Williams WH, Bryant R. Post-traumatic stress disorder and traumatic brain injury: a review of causal mechanisms, assessment, and treatment. *Neuropsychol Rehabil* 2003;**13**:149—64.

81. **Williams WH,** Evans JJ, Wilson DA, *et al*. Brief report: prevalence of post-traumatic stress disorder symptoms after severe traumatic brain injury in a representative community sample. *Brain Inj* 2002;**16**:673—9.
82. **Harvey AG,** Bryant RA, Tarrier N. Cognitive behaviour therapy for post traumatic stress disorder. *Clin Psychol Rev* 2003;**23**:501—22.
83. **Stein MB,** McAllister TW. Exploring the convergence of posttraumatic stress disorder and mild traumatic brain injury. *Am J Psychiatry* 2009;**166**:768—76.
84. **Bryant RA,** Creamer M, O'Donnell M, *et al*. Post-traumatic amnesia and the nature of post-traumatic stress disorder after mild traumatic brain injury. *J Int Neuropsychol Soc* 2009;**15**:862—7.
85. **McCauley SR,** Boake C, Levin HS, *et al*. Postconcussional disorder following mild to moderate traumatic brain injury: anxiety depression and social support as risk factors and comorbidities. *J Clin Exp Neuropsychol* 2001;**23**:792—808.
86. **King NS.** Emotional neuropsychological and organic factors: their use in the prediction of persisting postconcussion symptoms after moderate to mild head injury. *J Neurol Neurosurg Psychiatry* 1996;**61**:75—81.
87. **King NS,** Crawford S, Wenden FJ, *et al*. Early prediction of persisting post-concussion symptoms following mild and moderate head injuries. *Br J Clin Psychol* 1999;**38**:15—25.
88. **Hoge CW,** McGurk D, Thomas JL, *et al*. Mild traumatic brain injury in US soldiers returning from Iraq. *N Engl J Med* 2008;**358**:453—63.
89. **Belanger H,** Kretzmer T, Vanderploeg RD, *et al*. Symptom complaints following combat-related traumatic brain injury: relationship to traumatic brain injury severity and posttraumatic stress disorder. *J Int Neuropsychol Soc* 2009; doi:10.1017/S1355617709990841.
90. **Chalton LD,** McMillan TM. Can 'partial' PTSD explain differences in diagnosis of PTSD by questionnaire self-report and interview after head injury? *Brain Inj* 2009;**23**:77—82.
91. **Shapinsky AC,** Rapport LJ, Henderson MJ, *et al*. Civilian PTSD scales: relationships with trait characteristics and everyday distress. *Assessment* 2005;**12**:220—30.
92. **Iverson GL,** Lange RT, Brooks BL, *et al*. 'Good old days' bias following mild traumatic brain injury. *Clin Neuropsychol* 2010;**24**:17—37.
93. **Mittenberg W,** DiGuilio DV, Perrin S, *et al*. Symptoms following mild head injury: expectation as aetiology. *J Neurol Neurosurg Psychiatry* 1992;**55**:200—4.
94. **Whittaker R,** Kemp S, House A. Illness perceptions and outcome in mild head injury: a longitudinal study. *J Neurol Neurosurg Psychiatry* 2007;**78**:644—6.
95. **Binder LM,** Rohling ML. Money matters: a meta-analytic review of the effects of financial incentives on recovery after closed-head injury. *Am J Psychiatry* 1996;**153**:7—10.
96. **Jacobson RR.** The post-concussional syndrome: physiogenesis psychogenesis and malingering, an integrative model. *J Psychosom Res* 1995;**39**:675—93.
97. **Cassidy JD,** Carroll LJ, Cote P. Mild Traumatic Brain Injury after traffic collisions: A population-based cohort study. *Journal of Rehabilitation Medicine* 2004;(43 Suppl):15—21.
98. **Larrabee GJ.** Detection of malingering using atypical performance patterns on standard neuropsychological tests. *Clin Neuropsychol* 2003;**17**:410—25.
99. **Suhr JA,** Gunstad J. Further exploration of the effect of 'diagnosis threat' on cognitive performance in individuals with mild head injury. *J Int Neuropsychol Soc* 2005;**11**:23—9.
100. **Snell DL,** Surgenor LJ, Hay-Smith JC, *et al*. A systematic review of psychological treatments for mild traumatic brain injury: an update on the evidence. *J Clin Exp Neuropsychol* 2009;**31**:20—38.
101. **Mittenberg W,** Canyock EM, Condit D, *et al*. Treatment of post-concussion syndrome following mild head injury. *J Clin Exp Neuropsychol* 2001;**23**:829—36.
102. **Ponsford J,** Willmott C, Rothwell A, *et al*. Impact of early intervention on outcome following mild head injury in adults. *J Neurol Neurosurg Psychiatry* 2002;**73**:330—2.
103. **Bell KR,** Hoffman JM, Temkin N, *et al*. The effect of telephone counselling on reducing post-traumatic symptoms after mild traumatic brain injury: a randomised trial. *J Neurol Neurosurg Psychiatry* 2008;**79**:1275—81.
104. **Gurr B,** Moffat N. Psychological consequences of vertigo and the effectiveness of vestibular rehabilitation for brain injury patients. *Brain Inj* 2001;**15**:387—400.
105. **Martelli MF,** Grayson RL, Zasler ND. Posttraumatic headache: neuropsychological and psychological effects and treatments implications. *J Head Trauma Rehabil* 1999;**14**:49—69.
106. **Rees RJ,** Bellon ML. Post concussion syndrome ebb and flow: longitudinal effects and management. *Neurorehabilitation* 2007;**22**:229—42.
107. **Medd J,** Tate RL. Evaluation of an anger management therapy programme following acquired brain injury: a preliminary study. *Neuropsychol Rehabil* 2001;**10**:185—201.
108. **McGrath J.** Cognitive impairment associated with post-traumatic stress disorder and mild head injury: a case report. *Neuropsychol Rehabil* 1997;**7**:231—9.
109. **Tiersky L,** Anselmi V, Johnston M, *et al*. A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Arch Phys Med Rehabil* 2005;**86**:1565—74.
110. **Paniak C,** Toller-Lobe G, Durand A, *et al*. A randomized trial of two treatments for mild traumatic brain injury. *Brain Inj* 1998;**12**:1011—23.
111. **Mateer CA,** Sira CS, O'Connell ME. Putting Humpty Dumpty together again: the importance of integrating cognitive and emotional interventions. *J Head Trauma Rehabil* 2005;**20**:62—75.
112. **Barlow DH,** Allen LB, Choate ML. Toward a unified treatment for emotional disorders. *Behav Ther* 2004;**35**:205—30.

Mild traumatic brain injury and Postconcussion Syndrome: a neuropsychological perspective

W Huw Williams, Seb Potter and Helen Ryland

J Neurol Neurosurg Psychiatry 2010 81: 1116-1122 originally published online August 27, 2010
doi: 10.1136/jnnp.2008.171298