

Mild traumatic brain injury and Postconcussion Syndrome: A neuropsychological Perspective

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ABSTRACT

Symptoms of mild traumatic brain injury typically resolve within days or weeks. However, a significant group of patients may report symptoms of Post-concussion 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 USA⁵ 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 than 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 Medicine²¹ 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 inter-related 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 pain³⁷ 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 Concussion Symptoms Questionnaire (RPCSQ), has been advocated, particularly as the presence and severity of symptoms are associated with quality of life⁴¹ 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 injury²² 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 Lishman⁴⁷ 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, King⁴⁶ 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 tests⁵².

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 Vanderploeg⁵³ 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 colleagues⁶² 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 Shapiro⁶³ reported that the cognitive performance of MTBI patients could not be distinguished from matched controls at 1 month postinjury. 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 neurocognitive 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-matter 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 colleagues⁷⁶ 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 King⁸⁶ 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' phenomenon⁹²), 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 colleagues⁹⁴ 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 symptoms⁴⁷ 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 Gunstad⁹⁹ 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 1997¹⁰¹ 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 anger¹⁰⁷ 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 complementary 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.

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