# Neuropsychological Assessment of mTBI in Adults

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## Introduction

Mild traumatic brain injury (mTBI) refers to a wide range of brain injuries that are considered to be at the milder end of the TBI severity spectrum. mTBI has been classified as those injuries that result in a Glasgow Coma Scale (GCS) score of 13–15, a duration of loss of consciousness (LOC) of less than 30 minutes, and duration of post-traumatic amnesia (PTA) of less than 24 hours (Tables 1 and 2). PTA has been reported as a more effective measure of severity of mTBI than GCS in the context of predicting behavioral outcomes at 6 months post-injury [1]; however, the challenges of reliably assessing PTA in relation to mTBI is highlighted by Ruff and colleagues [2]. Even where there is an absence of PTA and/or LOC, cognitive abnormalities may be detected in the immediate aftermath of a suspected concussion [3].

**Table 1** Traumatic brain injury classification (civilian)

| TBI severity | Classification criteria[a] | | |
|---|---|---|---|
| | LOC | PTA | GCS |
| Mild | ≤30 minutes | <24 hours | 13–15 |
| Moderate | 30 minutes to 1 week | 24 hours to 1 week | 9–12 |
| Severe | >7 days | >7 days | ≤8 |

Note: *GCS* Glasgow Coma Scale Score, *LOC* duration of loss of consciousness, *PTA* duration of post-traumatic amnesia
[a]Data from Ref. [15]

mTBI is a common occurrence and is considered to be a major public health issue globally. The annual worldwide incidence has been estimated at 45 million [4], with over one million in the United States alone [5]. This estimate is known to be conservative as there is an absence of data on individuals suffering a mTBI who do not present to hospital, and those who do present, but are discharged at the emergency department (ED) [6]. Between 70% and 80% of all TBIs are classified as mild [7], but despite this, these injuries can be a continuing cause of disability, leading to cognitive, mood, and behavioral disorders [8]. For many people, post-injury symptoms usually resolve within days or weeks. Yet for a substantial

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**Table 2** Veteran Health Administration and Department of Defense TBI Classification Scheme

| TBI severity | Classification criteria | | | | |
|---|---|---|---|---|---|
| | LOC | Alteration of mental state | PTA | GCS | Structural imaging |
| Mild | 0–30 minutes | A moment up to 24 hours | 0–1 day | 13–15 | Normal |
| Moderate | 30 minutes to 1 week | >24 hours, severity based on criteria | 1–7 days | 9–12 | Normal or abnormal |
| Severe | >24 hours | >24 hours, severity based on criteria | >7 days | ≤8 | Normal or abnormal |

Note: Alteration of consciousness/mental state must be immediately related to the head
*GCS* Glasgow Coma Scale Score, *LOC* duration of loss of consciousness, *PTA* duration of post-traumatic amnesia

minority of individuals with mTBI, intracranial abnormality (referred to as "complicated" mTBIs) may be detected on computed tomography (CT) [9], with prevalence rates varying from 5% [10] to approximately 40% [11] between various studies [12]. Slow recovery (where symptoms persist beyond the initial weeks or months post-injury) occurs in 5–20% of mTBI individuals. These cases are referred to as suffering from persistent post-concussion syndrome (PCS) [13]. The provenance of such ongoing problems is controversial [14].

## Etiology

A TBI may occur from any number of causes and may vary according to gender, age, race, and geographical location. Falls have been reported as the leading cause of TBI, accounting for two in every five TBIs. Of individuals over 64 years, 81% of TBIs were a result of a fall, while in children under the age of 15 years, falls accounted for 55% of TBIs [4].

## Incidence Rates

The incidence of mTBI (approximately 131 cases per 100,000 people) far exceeds that of moderate TBI (15 cases per 100,000 people) and severe TBI (14 cases per 100,000 people) [15, 16].

## Definitions of Mild Traumatic Brain Injury and Persistent Concussion Symptoms

There are various terms used, often interchangeably, for the type of injury and subsequent symptoms associated with mTBI and PCS. In this chapter, the term mTBI will refer to the initial injury, and PCS will refer to persistent post-concussion symptoms following such injury (over weeks, months, and years). Immediate physical symptoms of mTBI may include headache, dizziness, nausea, unsteady gait, slurred speech, and cognitive signs, such as confusion or disorientation, reduced processing speed, memory disturbance, concentration difficulties, and executive dysfunction [17]. A LOC (e.g., GCS score of 13 or above) is considered a mild injury. However, amnesia, especially PTA, has been proposed as either an additional or an alternative diagnostic criterion to LOC, in conjunction with confusion [18]. Gradations of mTBI severity have been recommended in the past by the American Academy of Neurology [19, 20].

## Post-concussion Symptoms

There are numerous post-TBI self-report symptom inventories available to record subjective symptoms and the degree of impact or level of severity each endorsed symptom is having on an individual (e.g., Rivermead Post Concussion Symptoms Checklist [21], Concussion Signs and Symptoms Checklist). Residual signs and symptoms of sport-related concussion and mTBI may include those outlined in Table 3. In sport-related concussion, the large majority of athletes self-report resolution of symptoms within 7–10 days, and certainly within 1 month post injury [22]. This pattern of acute disturbance and recovery is remarkably consistent with the pattern of physiological disturbance and recovery described in neuroscience research [23, 24].

PCS is not a single pathophysiological entity. It is a term used to describe a constellation of nonspecific symptoms (e.g., memory disturbance,

**Table 3** Common signs and symptoms of mild TBI and sport-related concussion

| Cognitive | Physical | Emotional/mood | Sleep disturbance |
|---|---|---|---|
| Difficulty thinking clearly | Headache | Irritability | Sleeping more than usual |
| Difficulty remembering | Nausea/vomiting | Feeling more emotional | Sleeping less than usual |
| Difficulty concentrating | Neck pain | Sadness | Trouble falling asleep |
| Feeling slowed down | "Pressure in the head" | Anxiety | |
| Feeling like "in a fog" | Balance problems | Nervousness | |
| "Don't feel right" | Dizziness | | |
| Confusion | Sensitivity to noise | | |
| Drowsiness | Sensitivity to light | | |
| | Blurred vision | | |
| | Fatigue, lacking energy | | |

difficulty with concentration, irritability, anxiety, depression, apathy, headache, fatigue, sleep disturbance, balance problems, visual disturbance, sensitivity to light and/or noise) that are linked to several possible causes that do not necessarily reflect ongoing physiological brain injury [25]. The differential diagnosis of PCS includes depression, somatization, chronic fatigue, chronic pain, vestibular dysfunction, ocular dysfunction, or some combination of these conditions [26].

For the clinician, the challenge is to determine whether prolonged symptoms after mTBI reflect a prolonged version of the concussion pathophysiology as opposed to a manifestation of a secondary process, such as premorbid clinical depression or migraine headaches [27, 28]. Obtaining a prior medical history, performing a careful physical examination, and considering the response to physical or mental exertion (i.e., whether exertion reliably exacerbates symptoms) [29] when developing the differential diagnosis of persistent post-concussion symptoms are essential. This process may enable the clinician to link symptoms of post-concussion "syndrome" to one or more definable post-concussion "disorders" [30]. For example, establishing a pre-morbid history of migraine headaches, depression, anxiety, attention deficit hyperactivity disorder, or learning disability is crucial because mTBI can exacerbate these conditions, and they, in turn, can be responsible for ongoing symptoms [28]. It has been noted that a strong vulnerability factor in the development of PCS is older age compared to those typically presenting with milder head injury and that female gender is significant [31].

For determining PCS, there are the International Classification of Diseases (ICD) section F07.2 (post-concussional syndrome) diagnostic criteria. The controversy regarding the validity of post-concussional disorder is reflected in the latest version of the Diagnostic and Statistical Manual of the American Psychiatric Association (i.e., DSM-V) [32]. There is no longer a category for post-concussional disorder, but a new disorder category known as the "neurocognitive disorders." Within the spectrum of neurocognitive disorders is a new category (i.e., "Major or Mild Neurocognitive Disorder due to Traumatic brain Injury"). There is reference to different categories of TBI, including mild, moderate, and severe. Neurocognitive symptoms associated with mTBI are noted to resolve within days to weeks after the injury, with complete resolution by 3 months (DSM-V). It is not known yet whether the next version of the ICD will revise the diagnostic category of PCS. The specific DSM-V criteria for neurocognitive disorder due to TBI are as follows:

1. The criteria are met for major or mild neurocognitive disorder.
2. There is evidence of a TBI—that is, an impact to the head or other mechanisms of rapid movement or displacement of the brain within the skull, with one or more of the following:
   - LOC
   - PTA
   - Disorientation and confusion
   - Neurological signs (e.g., neuroimaging demonstrating injury, a new onset of seizures, a

marked worsening of a preexisting seizure disorder, visual field cuts, anosmia, hemiparesis)
3. The neurocognitive disorder presents immediately after the occurrence of the TBI or immediately after recovery of consciousness and persists past the acute post-injury period.

## Consequences of mTBI/PCS

mTBI is "classically defined as an essentially reversible syndrome without detectable pathology" [33]. It is often noted that recovery following mTBI is rapid—with most acute symptoms resolving within hours, and then, typically, a person being symptom-free by around 10 days [22].

Typically, the more severe injuries occur from greater rotational acceleration–deceleration forces involved in the impact [34]. Following impact, a neurometabolic cascade ensues [24]. The short-term effects can include a lack of electro-chemical activity, hemorrhaging, and axonal shearing, especially in the frontal temporal lobe area, although in mTBI these early deficits may largely resolve themselves [35]. mTBI, therefore, tends to be characterized by dysfunction or neurobehavioral profile rather than underlying neuropathological changes [36]. Caution, though, is still warranted regarding signs of greater impact.

## Considerations Regarding Neuropsychological Testing

The common cognitive domains typically affected by mTBI include executive functions (a set of cognitive abilities that control and regulate volitional activities, such as planning, organizing, self-awareness, impulse control, mental flexibility, problem solving, and other self-regulatory functions), information processing speed (the speed, or how quickly, cognitive tasks are performed), sustained attention (the ability to maintain consistent behavioral responses over time to specific stimuli during an ongoing repetitive task), divided attention (the ability to respond to two or more different tasks at the same time), and memory (the ability to encode, store, and retrieve information within various time frames from the original encoding experience).

There are two main reasons for neuropsychological assessment for concussion: (i) to determine the presence of cognitive symptoms for early diagnosis of mTBI (in terms of severity and potential duration of injury) and (ii) to monitor recovery over days, weeks, months, or even years later [37, 38]. In the latter, there may be identification of lasting neuropsychological sequelae.

Neuropsychological testing needs to be specific, sensitive, reliable, and valid for identifying mTBI/PCS [39]. Validity is the accuracy of the measurement or the extent to which the test is measuring what it is purported to be measuring. Sensitivity and specificity refer to the likelihood of identifying either genuine positives or negatives, respectively. Sensitivity is the probability that someone in the category of interest (in this case, mTBI) is identified by the test. Conversely, if a test has a high level of specificity, it will reliably predict those who do have the condition versus those who do not have the condition. Reliability refers to the consistency of the measurement or the extent to which the test provides approximately the same result on each occasion it is used under the same set of conditions with the same participants.

Test–retest reliability is also an important consideration in view of the potential for serial assessment post-injury to track recovery trajectories. A large body of work has considered test–retest reliability at various intervals [40–46]. Recently Maerlender and colleagues [47] examined four sequential time points for the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) computerized battery and reported that the two memory composite scores increased significantly with successive administrations.

For further review on advanced topics in neuropsychological assessment following sport-related concussion, see Iverson and Schatz [48]. Some of these topics are discussed below.

## Baseline and Post-injury Assessment

At-risk populations, such as athletes of full contact and collision sports and military service members, are unique populations that offer the opportunity to employ a baseline (i.e., pre-injury exposure) post-injury model.

When considering cognitive tasks to use in a baseline (pre- and post-injury) model, test–retest reliability is especially important. This can be estimated by comparing the results of a test on the same population carried out at different times—e.g., using a correlation coefficient. However, such repeat testing can lead to practice effects, whereby the participant performs better in subsequent tests due to having "learned" from the previous experience [49].

Where it is known that if a test is susceptible to practice effect, then Reliable Change Indices (RCI) can be used to calculate what improvement would be expected from a person from baseline to post-concussion testing and what adjustment is needed to take account of such expected improvement [50]. The RCI is calculated by use of a control group to establish the average change between tests, and an additional correction is made for test variability and reliability using an error term which produces a standard score (*Z*). Furthermore, use of alternate versions of tasks can limit practice effects [51].

A number of studies have reported on reliable change, sensitivity, and predictive value for the commonly used ImPACT battery. Van Kampen and colleagues [52] reported 83% of concussed athletes had at least one ImPACT score that exceeded the reliable change index for that score, compared to 30% of the control group. Sensitivity was reported to increase by 19% with the addition of ImPACT result to a post-concussive symptom questionnaire. The predictive value of ImPACT, where at least one abnormal composite score was evident, was 83%, and the predictive value of a negative test result was 70%. Overall, 93% of concussed athletes were correctly identified as concussed when the post-concussive symptoms score, and at least one ImPACT score were determined to be abnormal [52]. The combined sensitivity of ImPACT and the concussive symptoms score in high school athletes has been reported at 89% and specificity at 82% [53]. Results suggest the ImPACT battery may be sensitive to the effects of sport-related concussion once subjective symptoms have resolved [52, 54, 55]. Being cognizant of false-positive rates of RCIs for concussion batteries is also an important reference point that can assist with interpretation of RCI output for multi-test batteries. For example, the majority of normal individuals would be expected to demonstrate significant declines on at least one RCI for batteries producing seven or more uncorrelated RCIs (80% confidence intervals), although expected rates are lower for tests with fewer indices, higher inter-RCI correlations, and more stringent impairment criteria [56].

The baseline post-injury model in at-risk populations (e.g., athletes and military service members) is vulnerable to "sandbagging" (incomplete effort) at baseline, and, as such, inbuilt measures of effort are incorporated in the neuropsychological test design. The motivation for intentional poor performance on baseline is to appear to be "recovered" post-injury and, therefore, able to return to activities sooner. The frequency of poor effort on baseline testing has been reported as 9% [57] to 11% in high school athletes [58], 6% in a collegiate sample [59], and 6% in a sample of US National Football League (NFL) players [60]. Intentional (or motivated) poor performance has been reported to be difficult on the ImPACT test, with one study reporting that only 11% of test takers were able to successfully underperform without detection from the inbuilt integrity measures [61].

When no initial baseline is available, it is still possible to consider reliable change post-mTBI, but such approaches are in early development. Through the use of intra-individual measures of quotients that are unlikely to be adversely affected by mTBI, analysis of test scores generated from neuropsychological assessment at the individual level can be used. Correlation coefficients that exist between tests may be utilized, together with the *z*-score distribution to undertake statistical analyses. Using standard error estimates, it is possible to calculate the probable range of scores that a person would have in testing, with scores falling increasingly further from the predicted range being increasingly improbable. Such discrepancy analyses are already available in analyses of discrepancies between WAIS-IV indices, for example, based upon these tests being co-normed. The approach advocated here facilitates such analyses between non-co-normed tests, which the authors are developing.

## Clinical Management of At-Risk Populations

There is a strong literature that has developed over a number of decades pertaining to athletes involved in full-contact and collision sports and concussion. The literature on mTBI and neuropsychological testing is dominated by studies conducted in populations engaged in sports activities. Studies with military populations have garnered considerable research attention, due in large part to the level of involvement of military forces from many nations in the Afghanistan and Iraq wars.

Neuropsychological assessment and management models in at-risk populations are designed to promote the screening of large numbers of people in order to establish an individual standard for each person. The model is distinctly different from more traditional models of neuropsychological evaluation that utilize extensive, thorough (but time-consuming) test batteries. The baseline evaluation is not meant to represent a comprehensive assessment, but is targeted to assess cognitive domains that are most often affected by mTBI/concussion, such as memory, attention and concentration, executive function, speed of mental processing, and reaction time [62]. It has been proposed that the most effective use of neuropsychological test data to help determine post-injury return to activity occurs by obtaining a "baseline" level of function prior to sustaining an injury [63]. Baseline testing is typically conducted at pre-season training camps for athletes and incorporated into the routine pre-deployment preparations for the military. Individuals who are suspected of sustaining a concussion are then retested (the timing of the post-injury testing will be contingent upon the clinical question). It is considered standard practice that an individual's cognitive performance must return to baseline or better, prior to re-commencing regular (at-risk) activities, in order to avoid the possibility of re-injury prior to making a full recovery [62, 64]. Determining cutoff scores in neuropsychological performance and post-concussion symptom clusters for classifying protracted recovery in concussed athletes may assist in setting numerical thresholds for clinicians to predict recovery [65].

## Military-Related mTBI Studies

Military service members are another at-risk group for sustaining mTBI/concussion. Cognitive complaints in military service members following combat exposure are common, particularly in individuals who have sustained mTBI, with some 15–20% of military service members reporting a history of mTBI [66]. TBI as a result of combat action may occur from blast injury, penetrating head injury, or via other non-blast exposure. The ongoing development of military armor for use in combat and the more common use of improvised explosive devices on the battlefields of modern conflicts has led to an increase in exposure to blast-related injury. The effects of blast-related mTBI on behavior and cognition continue to be a controversial topic.

In a study examining clinical outcomes in US military personnel with blast-related versus non-blast-related TBI, neuropsychological outcomes (together with global outcomes, headache severity, depression, and PTSD) were not found to be significantly different between the two groups, although both groups had higher rates of moderate to severe overall disability than the respective control groups [67]. Another recently published study reported that US Marines who sustained a concussion during a combat deployment had more post-deployment symptoms than Marines who were exposed to explosive blasts who, in turn, reported greater numbers of clinical symptoms than Marines who were not exposed to blasts and did not sustain a mTBI/concussion during the deployment [68].

A recent meta-analysis of the cognitive outcomes of blast-related mTBI found that executive function (specifically, set-shifting), delayed memory, and information processing speed were the most sensitive cognitive domains affected by blast-related mTBI [69]. Interestingly, post-traumatic stress disorder (PTSD) was not found to be a significant moderator in predicting cognitive effects sizes [69]. Lange and colleagues [70] found that there were no significant differences when comparing the neuropsychological outcome in US military service members suffering from uncomplicated mTBI, complicated mTBI, and moderate TBI, within the previous 6 months. In another sample of US military service members who had sustained a mTBI, the self-reported cognitive complaints were not found to be associated with neuropsychological test performance, but were associated with psychological distress [71]. In an examination of neuropsychological profiles of US military populations, military personnel reporting "brain injury with current symptoms" were two times more likely to function at below average levels compared to those reporting "no previous TBI" [72].

## Sport-Related Concussion Studies

An increasing awareness of the effects of sport-related concussion on cognition has led to sports physicians seeking fast and accurate assessment of cognitive function to facilitate management decisions about time of recovery and resumption of participation in sports. In a number of high profile sports, these are now done on the sidelines and may dictate return-to-play (RTP). Neuropsychological testing has been recognized as a unique and invaluable method for not only assisting with assessment of post-mTBI sequela, but also in tracking recovery over time [63].

Neuropsychological testing, the domain of the neuropsychologist, typically involves the administration of a variety of tests assessing cognitive abilities. The interpretation of neuropsychological test data assists athletes by identifying and tracking post-concussion symptoms and cognitive sequelae, lending valuable information for managing RTP decisions and focusing on the best interests of the athlete. Results of these tests, coupled with other clinical information (such as medical history, neuroimaging, and interviews with family members), give credence to the neuropsychologist for making important clinical and diagnostic decisions pertaining to disorders of the central nervous system [73]. The aim of neuropsychological assessment with respect to concussive injury is to detect and quantify residual cognitive and behavioral deficits [62].

The utility of neuropsychological testing in assessing concussion was proposed as early as the 1880s [74] and has been documented empirically since the early 1980s [75]. The development of sport-related neuropsychological testing occurred concurrently in both North America and Australia at this time. Barth and colleagues at the University of Virginia in the late 1980s [76] demonstrated the potential usefulness of neuropsychological testing to monitor and document cognitive recovery in the first week following a sport-related concussion. Although this pioneering work was the foundation for the field of neuropsychology to contribute to sport-related concussion, this project initially did not result in the widespread adoption of neuropsychological testing. In the early 1990s, a series of events transpired that promoted the use of neuropsychological testing of athletes in the clinical arena. Initially, concussive injuries to a number of high profile professional athletes resulted in implementation of baseline neuropsychological testing by several NFL teams. Almost immediately following this, the US National Hockey League (NHL) mandated baseline neuropsychological testing for every athlete subsequent to career ending injuries of a number of elite athletes. Coincident with this trend was the publications of several large-scale studies of collegiate athletes [77]. These studies provided further support for the implementation of neuropsychological testing of athletes suspected of sustaining a concussion.

Specifically, neuropsychological testing allowed individual baseline and post-injury analysis of the subtle aspects of cognition likely to be affected by sport-related concussion. Neuropsychological testing is now widely regarded as a valid clinical strategy for assessing the cognitive sequelae of sport-related concussion [62, 78–80].

## Recovery from Sport-Related Concussion

The general consensus within the field of sports medicine is that isolated concussions in sports are often self-limiting injuries that are not associated with long-term cognitive or neurobehavioral problems [55, 81–83]. Most neuropsychological deficits appear to resolve within 10 days following a concussion [84, 85]. Studies from the sports concussion literature have shown that age [86], gender [87–89], learning disability/attention deficit disorder [78, 84], headache status, concussion history [80, 90–93], sleep and vigilance [94], and demographic and biopsychosocial factors [60] may have effects on baseline and post-concussion neuropsychological performances. For this reason, among others, the interpretation of neuropsychological test data should be conducted by a clinical neuropsychologist who is uniquely qualified to translate the test data into recommendations for clinical management [62].

## Neuropsychological Impact of mTBI

Belanger and colleagues [95] conducted a meta-analyses reviewing the neuropsychological impact of mTBI across nine cognitive domains (an analysis that included 39 studies comprising 1463 mTBI cases and 1191 controls). The overall effect of mTBI on neuropsychological functioning was moderate ($d = 0.54$), with findings moderated by cognitive domain, time since injury, patient characteristics, and sampling methods. Mild neuropsychological impairments across domains were observed within the first 90 days, with specific and relatively large deficits in fluency ($d = 0.89$) and delayed memory recall ($d = 1.03$). However, by 90 days post-injury, no individual cognitive domain was found to be significantly different from zero ($d = 0.04$). In contrast, clinic-based samples and samples including participants in litigation were associated with greater cognitive sequelae of mTBI ($d = 0.74$ and 0.78, respectively) at 3 months or longer after the injury. Participants in litigation had an overall acute effect size ($d = 0.52$ at <90 days since injury) compared to unselected samples ($d = 0.63$). However, overall the results of this meta-analysis suggest that for the mTBI sample (unselected sample at large), there is full neuropsychological recovery by 3 months post-injury.

## Conventional "Pencil and Paper" Neuropsychological Tests

Initially, neuropsychological testing was conducted on athletes and military personnel using pencil and paper measures [76, 84]. Many individuals suffering from mTBI have neuropsychological decrements detectable using conventional paper–pencil neuropsychological tests in the initial hours, days, and potentially weeks post-injury [63, 96–107]. The primary focus of the initial research examining cognitive function following sport-related concussion tended to relate to retrograde amnesia and memory retention [108]. The results indicated that athletes developed progressive retrograde amnesia and memory difficulties approximately 3–20 minutes after a concussion. Despite this relative success in detecting cognitive deficits, it became apparent that only assessing memory-related performance was not an effective way to evaluate the multi-dimensional cognitive sequelae typically observed following a sport-related concussion. As a result, the early focus on memory was expanded in subsequent studies to include multiple cognitive domains, including processing speed, reaction time, attention, and concentration as well as complex problem solving [76]. Concussed athletes were found to consistently perform poorly on these multidimensional neuropsychological tests [37, 76].

Deficits in speed of information processing or psychomotor speed are also apparent [109], and a number of pencil and paper tests have been developed specifically examining this neuropsychological construct [35]. Thus, mTBI testing batteries routinely incorporate at least one measure of processing speed [109]. The tasks frequently employed in "pencil and paper" testing include tests, such as Digit Span [110] which tests working memory with mental rotation, Speed of Comprehension and Language Processing [110] which tests general cognitive level and speed of processing, Trail-Making Tests A and B [110] which test sustained and divided attention, Stroop Color and Word [110] which tests executive skills (especially inhibition), and Symbol Digit Modalities Test (SDMT), a measure of visual-spatial and motor speed and accuracy [111].

The perceived value of neuropsychological testing for assessing and managing sport-related concussion was highlighted by the implementation in late 1990s of pencil and paper neuropsychological testing protocols in all NHL and the majority of NFL franchises. Such data were used extensively to determine more objective and individualized RTP parameters in athletes sustaining a concussion [64]. The Concussion in Sport group has endorsed neuropsychological testing as "one of the cornerstones of concussion evaluation and contributed significantly to both understanding of the injury and management of the individual" [96].

Conventional pencil and paper methods, however, were originally designed to examine gross impairment at a single point in recovery. That is, they were not designed to be serially administered to detect the very minor deficits in cognition often observed in sport-related concussion. Furthermore, conventional pencil and paper tests are time consuming and require trained, on-call clinical personnel to be properly administered [53, 112, 113]. This method of assessment may be feasible at the professional level. However, very few collegiate and high school programs have implemented this approach given the limitations of time, personnel, and finances [64].

## Computerized Neuropsychological Tests

As a result of the inherent limitations of conventional pencil and paper neuropsychological tests and in parallel to the widespread proliferation of advanced technology, several researchers have developed computerized neuropsychological testing batteries and symptom evaluations as an alternative. These enable quick and efficient baseline evaluations of large groups of individuals [107, 114]. The use of comprehensive computerized neuropsychological batteries has largely supplanted the use of traditional neuropsychological measures in most concussion management programs [109]. Neuropsychological testing in the computerized format is considered to have several advantages and few limitations compared with conventional testing procedures. The documented advantages of this format of testing include:

- *Time efficiency* – The approach allows large numbers of athletes to be tested with minimal time and effort, promoting the testing of an entire team within a reasonable time period.
- *Easy storage of information* – Data collected from testing can be stored electronically (i.e., on the hard drive of the computer) and can be easily accessed at a later date.
- *More accurate measurement* – The use of a computerized format promotes more accurate measurement of cognitive processes, such as reaction time and information processing speed (the computerized format allows for accuracy to 0.01 of a second). This has inevitably resulted in an increase in the validity of detecting subtle changes in cognitive processes, particularly those related to speed of response.
- *Randomization* – The use of a computerized format allows for test stimuli to be randomized, which, in turn, should improve reliability across multiple administration periods, minimizing the practice effects inherent within multiple exposure to testing. Limiting the influence of practice effects on testing allows a direct interpretation of post-injury data with baseline performance of the athlete to determine whether or not full cognitive recovery has occurred.
- *Automatic scoring* – The computerized format allows for automatic scoring, eliminating the possibility for human error and enabling immediate feedback of the athlete's performance [63].

**Table 4** Properties of conventional "pencil and paper" and computerized neuropsychological tests

| | Conventional "pencil and paper" tests | Computerized tests |
|---|---|---|
| *Psychometric considerations* | | |
| Alternative forms | None or very few | Infinite |
| Stimulus randomization | Within test only | Within test, between test and between subjects |
| Test–retest reliability | Wide range | Generally high for RT measures |
| Normative data | Mainly cross sectional, little LT | Very little for most tests |
| Practice effects | Large due to lack of alternative forms | Small: alternative forms and randomization |
| Output | Level of performance | Level of performance and variability |
| *Practical considerations* | | |
| Administration time | 1 minute–4 hours | 1 minute–2 hours |
| Support required | NP or trained technician for admin | Self-admin and auto scored |
| Accessibility | Poor—requires a NP | High—may be internet delivered |
| Data storage and analysis | Time consuming and costly | Automated |

*NP* neuropsychologist, *RT* response time, *LT* longitudinal, auto, automatic; admin, administration

In essence, a computerized approach appears to be more sensitive, reliable, practical, and certainly more cost-effective than conventional pencil and paper approaches. Because computerized neuropsychological testing is self-paced and self-directed, trained athletic trainers and other properly trained sports medicine staff members can administer baseline and follow-up tests [115]. However, this perceived advantage also has a distinct limitation, in that there is no real opportunity for the neuropsychologist to observe the athlete completing the test directly (i.e., qualitative information regarding the athlete cannot be collected and used for assisting with clinical decisions). See Table 4 for a summary of the properties of conventional "pencil and paper" neuropsychological testing and computerized neuropsychological testing.

There are a number of computer-based concussion management tools available or under development [116]. There are two with the largest share of the commercial market: ImPACT Applications® (San Diego, CA, USA) [117] and AxonSports (Scottsdale, AZ, USA; formally, CogState Ltd.'s CogSport©) [118]. An alternative test is commonly used by the US military (Automated Neuropsychological Assessment Metrics, or ANAM) [119]. A number of unique characteristics exist between these tests, and each is at a different stage of validation [116]. Each computerized battery has been developed to collect an individual baseline performance for comparison to post-concussive performance(s) should an athlete sustain a concussion during the season. As with conventional pencil and paper neuropsychological tests, issues pertaining to sensitivity, reliability, and validity of the respective options should be given careful scrutiny prior to implementation within the clinical setting [63].

## Limitations of Neuropsychological Testing

Despite the accumulating evidence supporting the clinical utility of neuropsychological tests in this area, a number of limitations have also been documented [51, 115, 120]. A number of shortcomings of both conventional and computerized neuropsychological assessment tools have been highlighted, and the need for neuropsychological testing in managing mTBI has been challenged. A strong case has been put forth that neuropsychological testing contributes nothing when considering decisions related to return to activity and, therefore, the clinical benefit of such assessment has been questioned. In a sporting context, however, if an athlete is symptomatic, current guidelines do not permit RTP or resumption of training. In this context neuropsychological assessment provides the only current objective criteria to inform decisions around fitness to re-engage in a given activity.

There is a lack of support for the utility of neuropsychological tests in detecting residual neuropsychological impairments following more obvious resolution of concussive symptoms, which is also problematic. This has further fueled the view that neuropsychological testing could not add clinical value to management and RTP decision-making. This is a moot point in the context of a recent review by Randolph, McCrea, and Barr [115], which highlighted that there are real risks involved in premature RTP that have never been clearly defined and, further, that no assessment technique or management intervention has ever been demonstrated that clearly attests to risk modification. As such, Randolph and colleagues did not endorse athletic teams allocating significant resources to implementation of an unproven method (neuropsychological testing) in an attempt to modify an unknown risk. In terms of the evidence for risks of sport-related concussion and the potential for risk modification from a neuropsychological perspective, prolonged recovery, same season repeat concussion, and late-life consequences, there is no current evidence to suggest that any specific guidelines or the use of baseline testing is of utility in modifying outcome from sport-related concussion [121].

While these criticisms may have some merit, we advocate that it is a narrow view to consider that neuropsychological testing has little value once symptoms have resolved. It is universally acknowledged that athletes are notorious at under reporting their symptoms following a concussion [122–126]; therefore, relying solely on the athlete's self-report, as is implied by this line of argument, is an unreliable management strategy that increases risk. Athletes may still be suffering from discrete residual cognitive deficits when reporting resolution of their post-concussion symptoms.

Subsequent to this critique, studies have found 38% of concussed athletes demonstrated impaired performance on at least one ImPACT variable following resolution of their symptoms [127]; a decline from baseline performance on divided attention scores on the CogSport battery has been reported in athletes no longer reporting symptoms; symptomatic and asymptomatic athletes examined on the CogSport battery following sport-related concussion demonstrated a significant decline from individual baseline performance in motor function and attention in symptomatic athletes. Further, there was a significant decline in divided attention in asymptomatic athletes [128].

## Cognitive Function in mTBI and Neuroimaging

In over 90% of mTBI cases, CT and structural MRI investigations are unremarkable [129, 130]. However, with more sophisticated brain function-related techniques, abnormalities may be detected. While many mTBIs tend to result in a recovery period of days or weeks, this is not the case for all mTBIs. In attempting to draw together the neuroimaging literature in mTBI, methodological heterogeneity within these studies, particularly pertaining to imaging data acquisition, is a source of challenge to coherence in interpreting the neuroimaging data across studies [131, 132].

Mu, Catenaccio, and Lipton [131] conducted a comprehensive review of various neuroimaging techniques (structural MRI, functional MRI [fMRI], diffusion tensor imaging [DTI], fluorodeoxyglucose positron emission tomography, electroencephalography, and magnetoencephalography) investigating blast injury. The authors found that four of the five structural MRI studies reported decreased cortical thickness and decreased thalamus and amygdala volume. The corpus callosum and superior longitudinal fasciculus were the neuroanatomical regions that revealed abnormality in 8 of 18 DTI studies. Resting-state fMRI studies reported a variety of functional network differences. Other functional imaging studies showed diffuse changes in activity, especially in the frontal, parietal, temporal, and cingulate regions. fMRI studies tended to examine executive function in the task-based studies and typically revealed widespread task-related activation in blast-related mTBI participants compared to control subjects [131]. In a general sense these studies do attest to both structural and functional changes after mTBI; however, a dominant and conclusive method which precisely extrapolates neural correlates has yet to emerge.

A systematic review of DTI studies in sport-related concussion [132] found 7 of 8 eligible studies had at least some type of DTI abnormality. While neuroanatomical location was inconsistent, the variance in location is unsurprising given the heterogeneity of concussion and the variability between time of injury and DTI scanning. Changes in some regions, such as the corpus callosum, internal capsule, and longitudinal fasciculus, are reported more often than others, which may further indicate that a useful approach lies in consideration of *neural connectivity* models and the vulnerability of associated structures to axonal injury in concussion. Diffuse decrease in fractional anisotropy using tract-based spatial statistics (TBSS) were demonstrated in retired aging collision sport athletes compared to non-concussed matched controls [133].

A systematic review of magnetic resonance spectroscopy (MRS) studies in sport-related concussion [134] found that 9 of 11 studies reported differences in MR spectra between concussed athletes and controls. The MRS findings suggest that metabolic disruption continues beyond the resolution of symptoms and other objective measures in some athletes.

## (Neuro-) Psychological Treatment

Active psychological and neuropsychological rehabilitation addressing persistent PCS has previously had limited empirical support [135]. Controlled trials of psychosocial approaches to interventions have predominantly focused on early intervention and prophylaxis. Education and reassurance (e.g., discussing typical symptoms, expected recovery time, and making graded increases in activity), offered either directly by clinicians [136] or via information leaflets [137], can reduce symptoms at 3–6 months post-injury [26, 138]. However, not all studies show a benefit for these approaches. Targeting at-risk groups, such as those with pre-injury psychiatric difficulties [139], may be warranted though.

A developing body of research indicates that various appraisals and coping responses may influence whether symptoms endure, such as symptom interpretation, recovery expectations, the "good old days" bias, and all-or-nothing coping [140–142]. Addressing these and associated vicious cycles that maintain or exacerbate symptoms using cognitive behavioral therapy (CBT) has been proposed should difficulties persist [135]; treatment may go beyond addressing comorbid anxiety and depression to focus on other processes that may contribute towards problems, such as fatigue and cognitive difficulties. Two randomized controlled trials of CBT, one with additional cognitive rehabilitation components [143] and one without [144], both indicated positive findings compared with waiting list controls. Reducing symptoms and improving quality of life may, therefore, be possible for individuals with persistent difficulties.

## Summary and Conclusions

Neuropsychological functions appear to recover rapidly early post-mTBI. Neuroimaging studies largely demonstrate functional, rather than structural, changes post-mTBI; however, in some cases, especially in "complicated" mTBI, structural changes may also be present. Studies examining the association between neuropsychological status and radiographic neuroanatomic data suggest the functional changes in brain activation may resolve readily, but in those "complicated" cases, especially where structural changes are present, delayed recovery (at 3 months to a year) may be anticipated. There appears to be concordance between neurological findings and cognitive functions early after injury, but, with time, such associations dissipate. The relationship between subjective complaints and cognitive function also appears to weaken with time. Empirical support for the use of cognitive rehabilitation is sparse, but the role of psychoeducation and the treatment and modification of other psychosocial factors that may exacerbate post-mTBI symptoms has gained increasing support. It is crucial, therefore, that neuropsychological assessments of mTBI cases are undertaken not only to identify neuropsychological processing but also to identify and manage related issues, with a careful eye toward monitoring return to activities. With a better understanding of the multiple causal variables that interplay in mTBI and PCS, patients and relatives may be given better advice to ensure that recovery is maximized.

## References


1. Tellier A, Marshall SC, Wilson KG, Smith A, Perugini M, Stiell IG. The heterogeneity of mild traumatic brain injury: where do we stand? Brain Inj. 2009;23(11):879–87.
2. Ruff RM, Iverson GL, Barth JT, Bush SS, Broshek DK. Recommendations for diagnosing a mild traumatic brain injury: a National Academy of neuropsychology education paper. Arch Clin Neuropsychol. 2009;24(1):3–10.
3. McCrea M, Kelly JP, Randolph C, Cisler R, Berger L. Immediate neurocognitive effects of concussion. Neurosurgery. 2002;50(5):1032–40.
4. Faul M, Xu L, Wald MM, Coronado VG. Traumatic brain injury in the United States: emergency department visits, hospitalizations, and deaths. Atlanta: Centers for Disease Control and Prevention; 2010.
5. Arciniegas DB, Anderson CA, Topkoff J, McAllister TW. Mild traumatic brain injury: a neuropsychiatric approach to diagnosis, evaluation, and treatment. Neuropsychiatr Dis Treat. 2005;1(4):311–27.
6. Ratcliff JJ, Adeoye O, Lindsell CJ, Hart KW, Pancioli A, McMullan JT, et al. TRACK-TBI investigators. Emergency department disposition of the GCS 13-15 traumatic brain injury patient: analysis of the TRACK-TBI Study. Am J Emerg Med. 2014;32(8):844–850.
7. Kraus JF, Nourjah P. The epidemiology of mild, uncomplicated brain injury. J Trauma. 1988;28(12):1637–43.
8. Fleminger S, Pondsford J. Long term outcome after traumatic brain injury. Br Med J. 2005;331:1419–20.
9. Panenka WJ, Lange RT, Bouix S, Shewchuk JR, Heran MKS, Brubacher JR, et al. Neuropsychological outcome and diffusion tensor imaging in complicated versus uncomplicated mild traumatic brain injury. PLoS One. 2015;10(4):e0122746.
10. Ono K, Wada K, Takahara T, Shirotani T. Indications for computed tomography in patients with mild head injury. Neurol Med Chir (Tokyo). 2007;47(7):291–8.
11. Thiruppathy SP, Muthukumar N. Mild head injury: revisited. Acta Neurochir. 2004;146(10):1073–5.
12. Iverson GL, Lange RT, Waljas M, Liimatainen S, Dastidar P, Hartikainen KM, et al. Outcome from complicated versus uncomplicated mild traumatic brain injury. Rehabil Res Pract. 2012;2012:415740.
13. Ruff RM, Jamora CW. Myths and mild traumatic brain injury. Psychol Inj Law. 2009;2:34–42.
14. Williams WH, Potter S, Ryland H. Mild traumatic brain injury and Postconcussion syndrome: a neuropsychological perspective. J Neurol Neurosurg Psychiatry. 2010;81(10):1116–22.
15. Rao V, Lyketsos C. Neuropsychiatric sequelae of traumatic brain injury. Psychosomatics. 2000; 41(2):95–103.
16. Syed AT, Lone NA, Afzai Wani M, Bhat AS. Clinical management of patients with minor head injuries. Int J Health Sci (Qassim). 2007;1(1):131–40.
17. McCrory P. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Br J Sports Med. 2005;39(Supplement 1):i78–86.
18. Alexander MP. Mild traumatic brain injury: pathophysiology, natural history, and clinical management. Neurology. 1995;45(7):1253–60.
19. Giza CC, Kutcher JS, Ashwal S, Barth J, Getchius TSD, Gioia GA, et al. Summary of evidence-based guideline update: evaluation and management of concussion in sports. Neurology. 2013;80(24): 2250–7.
20. McCrory P. Traumatic brain injury: revisiting the AAN guidelines on sport-related concussion. Nat Rev Neurol. 2013;9(7):361–2.
21. King NS, Crawford S, Wenden FJ, Moss NE, Wade DT. The Rivermead post concussion symptoms questionnaire: a measure of symptoms commonly experienced after head injury and its reliability. J Neurol. 1995;242(9):587–92.
22. McCrea M, Guskiewicz KM, Marshall SW, Barr W, Randolph C, Cantu RC, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2556–63.
23. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery. 2014;75(Suppl 4):S24–33.
24. Giza CC, Hovda DA. The neurometabolic cascade of concussion. J Athl Train. 2001;36(3):228–35.
25. Leddy JJ, Sandhu H, Sodhi V, Baker JG, Willer B. Rehabilitation of concussion and post-concussion syndrome. Sports health: a multidisciplinary approach. Sports Health. 2012;4(2):147–54.
26. Al Sayegh A, Sandford D, Carson AJ. Psychological approaches to treatment of postconcussion syndrome: a systematic review. J Neurol Neurosurg Psychiatry. 2010;81(10):1128–34.
27. Dimberg EL, Burns TM. Management of common neurologic conditions in sports. Clin Sports Med. 2005;24(3):637–62.
28. Kutcher J, Eckner JT. At-risk populations in sports-related concussion. Curr Sport Med Rep. 2010;9(1):16–20.
29. Leddy JJ, Willer B. Use of graded exercise testing in concussion and return-to-activity management. Curr Sports Med Rep. 2013;12(6):370–6.
30. Leddy JJ, Baker JG, Merchant A, Picano J, Gaile D, Matuszak J, et al. Brain or strain? Symptoms alone do not distinguish physiologic concussion from cervical/vestibular injury. Clin J Sport Med. 2015;25(3):237–42.

31. King N. Permanent post concussion symptoms after mild head injury: a systematic review of age and gender factors. NeuroRehabilitation. 2014;34(4):741–8.
32. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-5. Washington, DC: American Psychiatric Association; 2013.
33. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. Brain. 1974;97(4):633–54.
34. Guskiewicz KM, Weaver NL, Padua DA, Garrett WE. Epidemiology of concussion in collegiate and high school football players. Am J Sports Med. 2000;28(5):643–50.
35. Lezak MD, Howieson DB, Bigler ED, Tranel D. Neuropsychological assessment. 4th ed. Oxford: Oxford University Press; 2004.
36. Iverson GL. Outcome from mild traumatic brain injury. Curr Opin Psychiatry. 2005;18(3):301–17.
37. Macciocchi SN, Barth JT, Alves W, Rimel RW, Jane JA. Neuropsychological functioning and recovery after mild head injury in collegiate athletes. Neurosurgery. 1996;39(3):510–4.
38. Davis GA, Iverson GL, Guskiewicz KM, Ptito A, Johnston KM. Contributions of neuroimaging, balance testing, electrophysiology and blood markers to the assessment of sport-related concussion. Br J Sports Med. 2009;43(Suppl 1):i36–45.
39. Iverson GL, Lovell MR, Collins MW. Validity of ImPACT for measuring processing speed following sports- related concussion validity of ImPACT for measuring processing speed following sports-related concussion. J Clin Exp Neuropsychol. 2005;27(6):683–9.
40. Cole WR, Arrieux JP, Schwab K, Ivins BJ, Qashu FM, Lewis SC. Test-retest reliability of four computerized neurocognitive assessment tools in an active duty military population. Arch Clin Neuropsychol. 2013;28(7):732–42.
41. De Monte VE, Geffen GM, Kwapil K. Test-retest reliability and practice effects of a rapid screen of mild traumatic brain injury. J Clin Exp Neuropsychol. 2005;27(5):624–32.
42. Van Dyke SA, Axelrod BN, Schutte C. Test-retest reliability of the traumatic brain injury screening instrument. Mil Med. 2010;175(12):947–50.
43. Womble MN, Reynolds E, Schatz P, Shah KM, Kontos AP. Test-retest reliability of computerized neurocognitive testing in youth ice hockey players. Arch Clin Neuropsychol. 2016;31:305–12.
44. Schatz P. Long-term test-retest reliability of baseline cognitive assessments using ImPACT. Am J Sports Med. 2010;38(1):47–53.
45. Schatz P, Ferris CS. One-month test-retest reliability of the ImPACT test battery. Arch Clin Neuropsychol. 2013;28(5):499–504.
46. Nakayama Y, Covassin T, Schatz P, Nogle S, Kovan J. Examination of the test-retest reliability of a computerized neurocognitive test battery. Am J Sports Med. 2014;42(8):2000–5.
47. Maerlender AC, Masterson CJ, James TD, Beckwith J, Brolinson PG, Crisco J, et al. Test–retest, retest, and retest: growth curve models of repeat testing with immediate post-concussion assessment and cognitive testing (ImPACT). J Clin Exp Neuropsychol. 2016;38(8):869–74.
48. Iverson GL, Schatz P. Advanced topics in neuropsychological assessment following sport-related concussion. Brain Inj. 2015;29(2):263–75.
49. Collie A, Maruff P, McStephen M, Darby DG. Psychometric issues associated with computerised neuropsychological assessment of concussed athletes. Br J Sports Med. 2003;37(6):556–9.
50. Hinton-Bayre AD, Geffen G. Severity of sports-related concussion and neuropsychological test performance. Neurology. 2002;59(7):1068–70.
51. Collie A, Darby DG, Maruff P. Computerised cognitive assessment of athletes with sports related head injury. Br J Sports Med. 2001;35(5):297–302.
52. Van Kampen D, Lovell MR, Pardini JE, Collins MW, Fu FH. The "value added" of neurocognitive testing after sports-related concussion. Am J Sports Med. 2006;34(10):1630–5.
53. Schatz P, Pardini JE, Lovell MR, Collins MW, Podell K. Sensitivity and specificity of the ImPACT test battery for concussion in athletes. Arch Clin Neuropsychol. 2006;21(1):91–9.
54. Broglio SP, Macciocchi SN, Ferrara MS. Sensitivity of the concussion assessment battery. Neurosurgery. 2007;60(6):1050–7.
55. Iverson GL, Brooks BL, Collins MW, Lovell MR. Tracking neuropsychological recovery following concussion in sport. Brain Inj. 2006;20(3):245–52.
56. Nelson LD. False-positive rates of reliable change indices for concussion test batteries: a Monte Carlo simulation. J Athl Train. 2015;50(12):1319–22.
57. Schatz P, Neidzwski K, Moser RS, Karpf R. Relationship between subjective test feedback provided by high-school athletes during computer-based assessment of baseline cognitive functioning and self-reported symptoms. Arch Clin Neuropsychol. 2010;25(4):285–92.
58. Hunt T, Ferrara M, Miller L, Macciocchi S. The effect of effort on baseline neuropsychological test scores in high school football athletes. Arch Clin Neuropsychol. 2007;22(5):615–21.
59. Schatz P. Long-term test-retest reliability of baseline cognitive assessments using the ImPACT. Am J Sports Med. 2010;38(1):47–52.
60. Solomon G, Haase R. Biopsychosocial characteristics and neurocognitive test performance in National Football League players: an initial assessment. Arch Clin Neuropsychol. 2008;23(5):563–77.
61. Erdal K. Neuropsychological testing for sports-related concussion: how athletes can sandbag their baseline testing without detection. Arch Clin Neuropsychol. 2012 Aug;27(5):473–9.

62. Moser RS, Iverson GL, Echemendia RJ, Lovell MR, Schatz P, Webbe FM, et al. Neuropsychological evaluation in the diagnosis and management of sports-related concussion. Arch Clin Neuropsychol. 2007;22(8):909–16.
63. Lovell M. The management of sports-related concussion: current status and future trends. Clin Sports Med. 2009;28(1):95–111.
64. Kontos AP, Collins M, Russo SA. An introduction to sports concussion for the sport psychology consultant. J Appl Sport Psychol. 2004;16(3):220–35.
65. Lau BC, Collins MW, Lovell MR. Cutoff scores in neurocognitive testing and symptom clusters that predict protracted recovery from concussions in high school athletes. Neurosurgery. 2012;70(2):371–9.
66. Hoge CW, McGurk D, Thomas JL, Cox AL, Engel CC, Castro CA. Mild traumatic brain injury in U.S. soldiers returning from Iraq. N Engl J Med. 2008;358(5):453–63.
67. Mac Donald CL, Johnson AM, Wierzechowski L, Kassner E, Stewart T, Nelson EC, et al. Prospectively assessed clinical outcomes in concussive blast vs nonblast traumatic brain injury among evacuated US military personnel. JAMA Neurol. 2014;71(8):994–1002.
68. Tsao JW, Stentz LA, Rouhanian M, Howard RS, Perry BN, Haran FJ, et al. Effect of concussion and blast exposure on symptoms after military deployment. Neurology. 2017;89:2010–6.
69. Karr JE, Areshenkoff CN, Duggan EC, Garcia-Barrera MA. Blast-related mild traumatic brain injury: a Bayesian random-effects meta-analysis on the cognitive outcomes of concussion among military personnel. Neuropsychol Rev. 2014;24(4):428–44.
70. Lange RT, Brickell TA, French LM, Merritt VC, Bhagwat A, Pancholi S, et al. Neuropsychological outcome from uncomplicated mild, complicated mild, and moderate traumatic brain injury in US military personnel. Arch Clin Neuropsychol. 2012;27(5):480–94.
71. French LM, Lange RT, Brickell TA. Subjective cognitive complaints and neuropsychological test performance following military-related traumatic brain injury. JRRD. 2014;51(6):933–50.
72. Proctor SP, Nieto K, Heaton KJ, Dillon CC, Schlegel RE, Russell ML, et al. Neurocognitive performance and prior injury among U.S. Department of Defense Military Personnel. Mil Med. 2015;180(6):660–9.
73. Echemendia RJ. Sports neuropsychology: assessment and management of traumatic brain injury. New York: Guilford Press; 2006.
74. Walton GL. Possible cerebral origin of symptoms usually classed under “railway spine”. Bost Med Surg J. 1883;109:337–40.
75. Rimel RW, Giordani B, Barth JT, Boll TJ, Jane JA. Disability caused by minor head injury. Neurosurgery. 1981;9(3):221–8.
76. Barth J, Alves W, Ryan T, Macciocchi S, Rimel R, Jane J, et al. Mild head injury in sports: neuropsychological sequelae and recovery of function. In: Levin EH, Benton A, editors. Mild head injury. New York: Oxford University Press; 1989. p. 257–75.
77. Guskiewicz KM, Ross SE, Marshall SW. Postural stability and neuropsychological deficits after concussion in collegiate athletes. J Athl Train. 2001;36(3):263–73.
78. Collins MW. Relationship between concussion and neuropsychological performance in college football players. JAMA. 1999;282(10):964.
79. Echemendia RJ, Putukian M, Mackin RS, Julian L, Shoss N. Neuropsychological test performance prior to and following sports-related mild traumatic brain injury. Clin J Sport Med. 2001;11(1):23–31.
80. Moser RS, Schatz P, Jordan BD. Prolonged effects of concussion in high school athletes. Neurosurgery. 2005;57(2):300–6.
81. Belanger HG, Vanderploeg RD. The neuropsychological impact of sports-related concussion: a meta-analysis. J Int Neuropsychol Soc. 2005;11(4):345–57.
82. Bleiberg J, Cernich AN, Cameron K, Sun W, Peck K, Ecklund J, et al. Duration of cognitive impairment after sports concussion. Neurosurgery. 2004;54(5):1073–80.
83. Carroll L, Cassidy JD, Peloso P, Borg J, von Holst H, Holm L, 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(43 Suppl):84–105.
84. Collins MW. Current issues in managing sports-related concussion. JAMA. 1999;282(24):2283.
85. Collins MW, Hawn KL. The clinical management of sports concussion. Curr Sports Med Rep. 2002;1(1):12–22.
86. Pellman EJ, Lovell MR, Viano DC, Casson IR, Tucker AM. Concussion in professional football: neuropsychological testing part 6. Neurosurgery. 2004;55(6):1290–305.
87. Covassin T, Schatz P, Swanik CB. Sex differences in neuropsychological function and post-concussion symptoms of concussed collegiate athletes. Neurosurgery. 2007;61(2):345–50.
88. Covassin T, Swanik CB, Sachs M, Kendrick Z, Schatz P, Zillmer E, et al. Sex differences in baseline neuropsychological function and concussion symptoms of collegiate athletes. Br J Sports Med. 2006;40(11):923–7.
89. Covassin T, Swanik CB, Sachs ML. Epidemiological considerations of concussions among intercollegiate athletes. Appl Neuropsychol. 2003;10(1):12–22.
90. Broglio SP, Ferrara MS, Piland SG, Anderson RB, Collie A. Concussion history is not a predictor of computerised neurocognitive performance. Br J Sports Med. 2006;40(9):802–5.
91. Collie A, McCrory P, Makdissi M. Does history of concussion affect current cognitive status? Br J Sports Med. 2006;40(6):550–1.
92. Iverson GL, Brooks BL, Lovell MR, Collins MW. No cumulative effects for one or two previous concussions. Br J Sport Med. 2006;40:72–5.

93. Gardner A, Shores EA, Batchelor J. Reduced processing speed in rugby union players reporting three or more previous concussions. Arch Clin Neuropsychol. 2010;25(3):174–81.
94. Gosselin N, Lassonde M, Petit D, Leclerc S, Mongrain V, Collie A, et al. Sleep following sport-related concussions. Sleep Med. 2009;10(1):35–46.
95. Belanger HG, Curtiss G, Demery JA, Lebowitz BK, Vanderploeg RD. Factors moderating neuropsychological outcomes following mild traumatic brain injury: a meta-analysis. J Int Neuropsychol Soc. 2005;11(3):215–27.
96. Aubry M, Cantu R, Dvorak J, Graf-Baumann T, Johnston K, Kelly J, et al. Summary and agreement statement of the first international conference on concussion in sport, Vienna 2001. Phys Sportmed. 2002;30(2):57–63.
97. Barr WB, McCrea M. Sensitivity and specificity of standardized neurocognitive testing immediately following sports concussion. J Int Neuropsychol Soc. 2001;7(6):693–702.
98. Barr WB, McCrea M, Randolph C. No neuropsychology of sports-related injuries. In: Morgan J, Ricker JH, editors. Textbook of clinical neuropsychology. New York: Psychology Press; 2008. p. 660–78.
99. Boutin D, Lassonde M, Robert M, Vanassing P, Ellemberg D. Neurophysiological assessment prior to and following sports-related concussion during childhood: a case study. Neurocase. 2008;14(3):239–48.
100. Casson IR, Pellman EJ, Viano DC. Concussion in the national football league: an overview for neurologists. Phys Med Rehabil Clin N Am. 2009;20(1):195–214.
101. Covassin T, Stearne D, Elbin R III. Concussion history and Postconcussion neurocognitive performance and symptoms in collegiate athletes. J Athl Train. 2008;43(2):119–24.
102. Erlanger DM, Kutner KC, Barth JT, Barnes R. Neuropsychology of sports-related head injury: dementia pugilistica to post concussion syndrome. Clin Neuropsychol. 1999;13(2):193–209.
103. Goldberg LD, Dimeff RJ. Sideline management of sport-related concussions. Sport Med Arthrosc Rev. 2006;14(4):199–205.
104. Henry GK, Gross HS, Herndon CA, Furst CJ. Nonimpact brain injury: neuropsychological and behavioral correlates with consideration of physiological findings. Appl Neuropsychol. 2000;7(2):65–75.
105. Mrazik M. Neuropsychological assessment of college athletes. Arch Clin Neuropsychol. 2000;15(8):813–4.
106. Roebuck-Spencer T, Sun W, Cernich AN, Farmer K, Bleiberg J. Assessing change with the automated neuropsychological assessment metrics (ANAM): issues and challenges. Arch Clin Neuropsychol. 2007;22(Suppl 1):S79–87.
107. Lovell M. The neurophysiology and assessment of sports-related head injuries. Phys Med Rehabil Clin N Am. 2009;20(1):39–53.
108. Yarnell PR, Lynch S. The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines. Lancet. 1970;295(7652):863–4.
109. Bruce JM, Echemendia RJ. History of multiple self-reported concussions is not associated with reduced cognitive abilities. Neurosurgery. 2009;64(1):100–6.
110. Lezak MD. Neuropsychological assessment. Oxford: Oxford University Press; 1995.
111. Smith A. Symbol digit modalities test manual. Los Angeles: Western Psychological Services; 1982.
112. Lovell MR. Is neuropsychological testing useful in the management of sport-related concussion? J Athl Train. 2006;41(2):137–8.
113. Lovell MR, Collins MW. Neuropsychological assessment of the college football player. J Head Trauma Rehabil. 1998;13(2):9–26.
114. Collins M, Stump J, Lovell MR. New developments in the management of sports concussion. Curr Opin Orthop. 2004;15(2):100–7.
115. Randolph C, McCrea M, Barr WB. Is neuropsychological testing useful in the management of sport-related concussion? J Athl Train. 2005;40(3):139–54.
116. Lovell M. The neurophysiology and assessment of sports-related head injuries. Neurol Clin. 2008;26(1):45–62.
117. Maroon JC, Lovell MR, Norwig J, Podell K, Powell JW, Hartl R. Cerebral concussion in athletes: evaluation and neuropsychological testing. Neurosurgery. 2000;47(3):659–72.
118. Collie A, Maruff P, Makdissi M, McCrory P, McStephen M, Darby D. CogSport: reliability and correlation with conventional cognitive tests used in postconcussion medical evaluations. Clin J Sport Med. 2003;13(1):28–32.
119. Cernich A, Reeves D, Sun W, Bleiberg J. Automated neuropsychological assessment metrics sports medicine battery. Arch Clin Neuropsychol. 2007;22(SUPPL. 1):101–14.
120. Randolph C, Kirkwood MW. What are the real risks of sport-related concussion, and are they modifiable? J Int Neuropsychol Soc. 2009;15(4):512–20.
121. Kirkwood MW, Randolph C, Yeates KO. Returning pediatric athletes to play after concussion: the evidence (or lack thereof) behind baseline neuropsychological testing. Acta Paediatr. 2009;98(9):1409–11.
122. Collins MW, Iverson GL, Lovell MR, McKeag DB, Norwig J, Maroon J. On-field predictors of neuropsychological and symptom deficit following sports-related concussion. Clin J Sport Med. 2003;13(4):222–9.
123. Delaney JS, Abuzeyad F, Correa JA, Foxford R. Recognition and characteristics of concussion in the emergency department population. J Emerg Med. 2005;29(2):189–97.

124. Delaney JS, Al-Kashmiri A, Drummond R, Correa JA. The effect of protective headgear on head injuries and concussions in adolescent football (soccer) players. Br J Sports Med. 2008;42(2):110–5.
125. McCrory P, Johnston KM, Mohtadi NG, Meeuwisse W. Evidence-based review of sport-related concussion: basic science. Clin J Sport Med. 2001;11(3):160–5.
126. Thunnan DJ, Branche CM, Sneizek JE. The epidemiology of sports related concussion in the United States: recent developments. J Head Trauma Rehabil. 1998;12(2):1–8.
127. Broglio SP, Macciocchi SN, Ferrara MS. Neurocognitive performance of concussed athletes when symptom free. J Athl Train. 2007;42(4):504–8.
128. Collie A, Makdissi M, Maruff P, Bennell K, McCrory P. Cognition in the days following concussion: comparison of symptomatic versus asymptomatic athletes. J Neurol Neurosurg Psychiatry. 2006;77(2):241–5.
129. Jeret JS, Mandell M, Anziska B, Lipitz M, Vilceus AP, Ware JA, et al. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. Neurosurgery. 1993;32(1):6–9.
130. Stein SC, Ross SE. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. Neurosurgery. 1993;33(2):339–40.
131. Mu W, Catenaccio E, Lipton ML. Neuroimaging in blast-related mild traumatic brain injury. J Head Trauma Rehabil. 2017;32(1):55–69.
132. Gardner A, Kay-Lambkin F, Stanwell P, Donnelly J, Williams WH, Hiles A, et al. A systematic review of diffusion tensor imaging findings in sports-related concussion. J Neurotrauma. 2012;29(16):2521–38.
133. Henry LC, Tremblay S, De Beaumont L. Long-term effects of sports concussions: bridging the neurocognitive repercussions of the injury with the newest neuroimaging data. Neurosci. 2017;23(5):567–78.
134. Gardner A, Iverson GL, Stanwell P. A systematic review of proton magnetic resonance spectroscopy findings in sport-related concussion. J Neurotrauma. 2014;31(1):1–18.
135. Conder R, Conder AA. Neuropsychological and psychological rehabilitation interventions in refractory sport-related post-concussive syndrome. Brain Inj. 2015;29(2):249–62.
136. Mittenberg W, Canyock EM, Condit D, Patton C. Treatment of post-concussion syndrome following mild head injury. J Clin Exp Neuropsychol. 2001;23(6):829–36.
137. Ponsford J, Willmott C, Rothwell A, Cameron P, Kelly A-M, Nelms R, et al. Impact of early intervention on outcome following mild head injury in adults. J Neurol Neurosurg Psychiatry. 2002;73(3):330–2.
138. Mittenberg W, Tremont G, Zielinski RE, Fichera S, Rayls KR. Cognitive-behavioral prevention of postconcussion syndrome. Arch Clin Neuropsychol. 1996;11(2):139–45.
139. Ghaffar O, McCullagh S, Ouchterlony D, Feinstein A. Randomized treatment trial in mild traumatic brain injury. J Psychosom Res. 2006;61(2):153–60.
140. Iverson GL, Lange RT, Brooks BL, Rennison VLA. "Good old days" bias following mild traumatic brain injury. Clin Neuropsychol. 2010;24(1):17–37.
141. Hou R, Moss-Morris R, Peveler R, Mogg K, Bradley BP, Belli A. When a minor head injury results in enduring symptoms: a prospective investigation of risk factors for postconcussional syndrome after mild traumatic brain injury. J Neurol Neurosurg Psychiatry. 2012;83(2):217–23.
142. Whittaker R, Kemp S, House A. Illness perceptions and outcome in mild head injury: a longitudinal study. J Neurol Neurosurg Psychiatry. 2007;78(6):644–66.
143. Tiersky LA, Anselmi V, Johnston MV, Kurtyka J, Roosen E, Schwartz T, et al. A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. Arch Phys Med Rehabil. 2005;86(8):1565–74.
144. Potter SD, Brown RG, Fleminger S. Randomised, waiting list controlled trial of cognitive-behavioural therapy for persistent postconcussional symptoms after predominantly mild-moderate traumatic brain injury. J Neurol Neurosurg Psychiatry. 2016;87(10):1075–83.