



Recommendations for Diagnosing a Mild Traumatic Brain Injury: A National Academy of Neuropsychology Education Paper

Ronald M. Ruff^{a,b,*}, Grant L. Iverson^{c,d}, Jeffrey T. Barth^e, Shane S. Bush^f, Donna K. Broshek^e,
and the NAN Policy and Planning Committee[†]

^aSan Francisco Clinical Neurosciences, San Francisco, CA, USA

^bUniversity of California–San Francisco, San Francisco, CA, USA

^cUniversity of British Columbia, Vancouver, BC, Canada

^dBritish Columbia Mental Health and Addiction Services, Vancouver, BC, Canada

^eUniversity of Virginia School of Medicine, Charlottesville, VA, USA

^fLong Island Neuropsychology, P.C., Lake Ronkonkoma, NY, USA

Accepted November 2008

Abstract

A special interest group of the American Congress of Rehabilitation Medicine [ACRM; Mild Traumatic Brain Injury Committee. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 8 (3), 86–87.] was the first organized interdisciplinary group to advocate four specific criteria for the diagnosis of a mild traumatic brain injury (TBI). More recently, the World Health Organization (WHO) Collaborative Center Task Force on Mild Traumatic Brain Injury [Carroll, L. J., Cassidy, J. D., Holm, L., Kraus, J., & Coronado, V. G. (2004). Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, (Suppl. 43), 113–125.] conducted a comprehensive review of the definitions utilized in evidence-based studies with mild TBI patients. Based on this review, the WHO task force maintained the same four criteria but offered two modifications. The similarities and differences between these two definitions are discussed. The authors of the ACRM and the WHO definitions do not provide guidelines or specific recommendations for diagnosing the four criteria. Thus, we provide recommendations for assessing loss of consciousness, retrograde and post-traumatic amnesia, disorientation and confusion as well as clarification of the neurologic signs that can be indicative of a diagnosis of mild TBI. Finally, confounding factors mentioned in both definitions that should exclude a mild TBI diagnosis are summarized.

Keywords: Mild traumatic brain injury; Neuropsychology; Treatment; Assessment; Head injury

Introduction

Mild traumatic brain injury (TBI) occurs frequently. Across all severity levels of TBI, the Centers for Disease Control and Prevention (2006) estimated that approximately 2% of the U.S. population is living with some degree of disability as a result of a TBI. Of the annual incidence of 1.5 million TBIs, it is estimated that approximately 80% are of mild severity, with the remaining 20% split more or less evenly between moderate and severe TBIs (Kraus, McArthur, Silverman, & Jayaraman, 1996). Given that past incidence data for mild TBIs were primarily derived from patients evaluated in hospital emergency

[†] The Policy and Planning Committee of the National Academy of Neuropsychology (NAN) is charged with writing position and educational papers regarding important issues that affect the profession of Neuropsychology. Possible topics for the papers are suggested by the NAN Board of Directors, members of the NAN Policy and Planning Committee, or individual Academy members. Primary authors are identified and approved by the NAN Policy and Planning Committee. These authors can come from within or outside the Policy and Planning Committee. Selected outside reviewers provide extensive peer review for all papers. All topics and the final paper submissions are reviewed and approved by the NAN Board of Directors. This process of review and approval takes the place of the system of peer review typically conducted by the Editor and Editorial Board of this journal.

* Corresponding author at: 909 Hyde Street, Suite 620, San Francisco, CA 94109, USA. Tel.: +1-415-771-7833; fax: +1-415-922-5849.

E-mail address: ronruff@mindspring.com (R.M. Ruff).

Table 1. Diagnostic criteria for Mild TBI by the American Congress of Rehabilitation Medicine Special Interest Group on Mild Traumatic Brain Injury

A traumatically induced physiological disruption of brain function, as manifested by *at least* one of the following:

- any loss of consciousness
- any loss of memory for events immediately before or after the accident
- any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused) and focal neurologic deficit(s) that may or may not be transient

But where the severity of the injury does not exceed the following:

- loss of consciousness of approximately 30 min or less
- after 30 min, an initial Glasgow Coma Scale score of 13–15 and
- post-traumatic amnesia not greater than 24 hr

Note: Developed by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group (1993).

Table 2. Concussion grading system by the American Academy of Neurology

Grade 1	Grade 2	Grade 3
No LOC	No LOC	LOC (seconds or minutes)
Transient confusion	Transient confusion	
Symptoms (e.g., headaches and dizziness) and mental status changes (e.g., befuddlement, inability to focus attention, or post-traumatic amnesia) resolve in less than 15 min	Symptoms or mental status changes last more than 15 min	

LOC = loss of consciousness.

rooms, these incidence rates are underestimates because the majority of persons who sustain a mild TBI either consult their primary care physician days after the injury or seek no care at all (Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992; Langlois et al., 2003; Mellick, Gerhart, & Whiteneck, 2003; Sosin, Sniezek, & Thurman, 1996). Hence, the 1998 National Institute of Health (NIH) Consensus statement concluded that mild TBIs were underdiagnosed (NIH, 1998).

In comparison with moderate and severe brain injuries, mild TBIs are often more challenging to diagnose. This is due to rapid resolution of acute signs and symptoms (e.g., loss of consciousness [LOC], post-traumatic amnesia [PTA], disorientation, and confusion) and the typical absence of objective evidence of injury on neuroimaging. The lack of a universally agreed definition of mild TBI contributes to this diagnostic challenge. Moreover, the lack of guidelines for assessing specific diagnostic criteria can lead to diagnostic disagreements. To better understand and serve this patient population, the National Academy of Neuropsychology's Policy and Planning Committee wrote this paper to provide recommendations for diagnosing a mild TBI.

The American Congress of Rehabilitation Medicine and World Health Organization Definitions

The American Congress of Rehabilitation Medicine (ACRM; Mild Traumatic Brain Injury Committee, 1993) was the first organized interdisciplinary group to advocate four specific criteria for the diagnosis of a mild TBI (Table 1). This ACRM definition has been widely used, especially in the field of rehabilitation and neuropsychology. With a focus on managing sports-related concussions, the American Academy of Neurology (1997) also published a definition of mild TBI (Table 2). For a detailed review of sports-related concussion, see the National Academy of Neuropsychology's recent position paper (Moser et al., 2007). For the purpose of the present paper, the term "concussion" is used synonymously with "mild TBI."

Eleven years after the ACRM definition was published, the World Health Organization (WHO) Collaborative Center Task Force on Mild Traumatic Brain Injury (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004) published a comprehensive review of definitions utilized in research studies and identified significant definitional discrepancies. For example, 62% of the definitions reviewed incorporated Glasgow Coma Scale (GCS) scores, yet the range of scores varied, including scores of 13–15, 14–15, or only 15. Similarly, although LOC or amnesia was required in some definitions, the duration of LOC varied. Thus, the WHO task force concluded that "the literature would greatly benefit by common criteria" (p. 115).

The WHO task force then advanced a standardized definition. In doing so, it acknowledged that the definition was derived from the ACRM definition and was also similar to the definition proposed by the Centers for Disease Control and Prevention's Mild TBI Working Group (Center for Disease Control and Prevention, 2003). The WHO task force operational definition is reprinted below.

MTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for healthcare. These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury (p. 115).

Differences between the ACRM and WHO Definitions

When the ACRM definition (Table 1) is compared with the WHO definition, the following differences emerge. First, one of the ACRM criteria is stated as “any alteration of mental state at the time of accident (dazed, disoriented, or confused)” whereas the WHO definition simplified the wording to “confusion and disorientation.” Thus, the word “dazed” was omitted from the WHO definition. Secondly, the ACRM definition describes the neurologic criterion as follows: “Focal neurologic deficit(s) that may or may not be transient” whereas the WHO definition captures this by stating “Other transient neurological abnormalities, such as focal signs, seizure, and intracranial lesion, which are not requiring surgery.” Thus, a potential difference emerges as to whether the focal neurologic signs are *transient* or not.

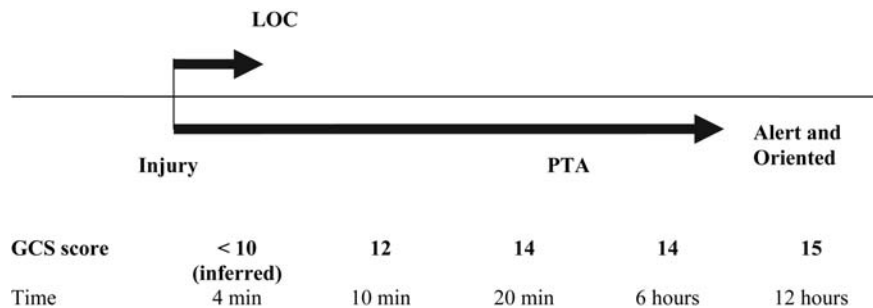
Although both the ACRM and WHO definitions identify the same four diagnostic criteria, the rationale by the WHO task force for making these two changes was not stated explicitly in their publication. The primary purpose of this paper is to identify various challenges and provide recommendations for assessing the presence or absence of LOC, PTA, confusion and disorientation, and neurologic signs.

Recommendations for Assessing Mild TBI

Assessing LOC

Diagnosing a mild TBI is challenging because the majority of these patients are evaluated by neuropsychologists weeks or even months after the injury. Even when patients are reviewed by emergency medical personnel at the scene of the injury, various acute symptoms, including a brief LOC, might have been present prior to their arrival at the scene. The mildest of injuries can be difficult to identify, even by first responders.

Because post-traumatic confusion or amnesia usually persists for a period beyond LOC (Fig. 1), patients typically are unable to accurately self-report if—and for how long—they were unconscious. Relying on the observations of others when assessing LOC reduces the likelihood of making two fundamental mistakes. First, some patients assume that they were unconscious during the period for which they have no recall. For example, if a patient’s first recall is of being in the ambulance, then the person might assume that he had been unconscious up to that time. When this occurs, the patient often says: “I woke up in the ambulance.” However, when the neuropsychologist reviews the paramedic records, it may turn out that the patient was talking at the scene of the accident. This can lead to an apparent contradiction, which may be incorrectly



LOC = loss of consciousness; PTA = post-traumatic amnesia; GCS = Glasgow Coma Scale. The GCS less than 10 was inferred because the patient was unconscious and unresponsive with a witness. However, the ambulance crew had not arrived so no GCS score was obtained at that time.

Fig. 1. Example of changing Glasgow Coma Scale scores across time.

interpreted as an intentional misrepresentation on the patient's part. The patient, however, simply reported a period of PTA as if it were a period of unconsciousness. Second, some patients deny experiencing LOC when in fact they did. For example, LOC may have occurred for 30 s after impact, yet the patient denies LOC, because the first memory is that of standing by the car or talking with a paramedic rather than of regaining consciousness. In the absence of recalling that they regained consciousness, the patient simply assumes that he was not knocked out. For these reasons, asking the patient "Did you lose consciousness?" or "Did you get knocked out?" should be replaced by "Has anyone told you that you were unconscious? Who saw you unconscious?" When available, obtaining collateral information regarding the presence or absence of LOC is strongly recommended.

LOC attributed to a TBI should result from the impact and should not be due to other medical factors (e.g., syncope). In those cases in which a brief LOC was witnessed (e.g., observed by reliable witness, paramedic, or physician), then a mild TBI can be diagnosed with a high degree of certainty. If the patient was initially conscious and LOC occurs minutes following the impact, then other causes for the LOC must be investigated. Of course, it is possible to sustain a mild TBI without being rendered unconscious. Agreement exists among the definitions put forth by the American Academy of Neurology, the ACRM, and the WHO Mild TBI Task Force that LOC is not essential for the diagnosis. In fact, the vast majority of injuries in sports (Guskiewicz, Weaver, Padua, & Garrett, 2000; Macciocchi, Barth, Alves, Rimel, & Jane, 1996; McCrea et al., 2003) and many injuries in medical trauma settings (e.g., Hanlon, Demery, Martinovich, & Kelly, 1999; Lovell, Iverson, Collins, McKeag, & Maroon, 1999; Paniak, MacDonald, Toller-Lobe, Durand, & Nagy, 1998) are not associated with LOC. Thus, in the absence of LOC or focal neurologic signs, either a period of altered consciousness (i.e., some period of confusion) or PTA (gap in memory following injury) is required. PTA is frequently the primary and most specific diagnostic indicator of injury.

Assessing Retrograde Amnesia and PTA

False-negative diagnoses can occur with patients who have no memory for the accident, but are fully oriented by the time the emergency personnel arrive. For example, the documentation in the medical records of the patient being fully oriented at the scene does not preclude that the patient experienced a gap in memory prior to the evaluation of the paramedics. Therefore, when the possibility of a mild TBI exists, it is necessary to assess the patient's status *retrospectively*. Although different types of questions can be posed, for these answers to accurately assess retrograde and PTA, *it is essential to determine what the patient remembers versus what he or she has been told or has surmised*. This can be challenging. There is often an intermingling between what a person remembers and what they have been told or have later deduced. Thus, it is necessary to ask very specific questions, such as: "What is the first event you remember *after* your injury?" A follow-up question could be: "Can you describe in as much detail as possible what you can remember immediately *after* your injury?" To ascertain whether the patient has a brief period of retrograde amnesia, a query about pre-impact events is necessary, such as: "Tell me the last event you remember *before* the accident." Some clinicians have the patient, in a free-flowing and open-ended manner, discuss the events before, during, and after the accident. Examples of such questions are: "How did it happen?" "Tell me about the accident/injury." "Tell me as many details as possible." However, this approach carries the risk of not being able to distinguish between what the patient actually remembers versus what he or she has garnered from other sources (e.g., police report, observations of third parties, medical records, or discussions with attorneys). Thus, the clinician using that approach needs to clarify what the patient actually remembers versus what has been pieced together from other sources. In sum, if retrograde amnesia occurred, then a definite gap should exist between the patient's recall of the last events *before* the accident and the impact itself. If PTA occurred, then a definite gap should exist between the impact and the patient's first recall *after* the injury. According to the ACRM and WHO definitions, no minimal duration for PTA is specified, thus even a brief PTA of a few seconds qualifies. However, if the duration of PTA exceeds 24 hr, both definitions state that the TBI should no longer be considered as mild.

There are further challenges to the reliable diagnosis of PTA. The issue of psychogenic amnesia should be considered, especially in patients, who prior to the accident, experienced a significant emotional trauma (e.g., rape, robbed at gunpoint, in a falling airplane, in a multiple roll-over car accident). For example, a person with an acute stress disorder (or post-traumatic stress disorder) can have partial amnesia. Poor memory can also be caused by intoxication with alcohol or drugs. Severe physical injuries and pain may also influence accurate recall. Medication, administered by paramedics or physicians, can also result in memory gaps. It is even possible to overestimate PTA by not carefully considering the timeline of events and failing to notice in the hospital records that the patient was asleep for several hours during the reported period of amnesia. Thus, the presence of PTA should be carefully considered if such significant factors played a role. PTA is not present if the person's recall is preserved before, during, and immediately after the accident. For example, if the only memory gap occurred 10 min after the

injury to the head, then alternative explanations are highly likely, such as impaired memory due to severe pain or feelings of panic.

Assessing Confusion and Disorientation

The ACRM definition describes this diagnostic criterion as “Any alteration of mental state at the time of accident (dazed, disoriented, or confused)” (Mild Traumatic Brain Injury Committee, 1993, p. 86) (Table 1) and the WHO definition provides the descriptors of “confusion and disorientation” (Carroll et al., 2004, p. 115). Thus, the word “dazed” is dropped in the WHO definition.

The diagnostic criterion of confusion and disorientation is frequently the most challenging to establish. A survivor of an unexpected event that placed him or her in serious danger can often experience a sense of being shocked and overwhelmed (Harvey & Bryant, 1999; Hunter, Sierra, & David, 2004). The challenge for the diagnostician is to differentiate between such a strong emotional reaction and evidence of external forces to the head that likely resulted in a biomechanically induced alteration of the person’s mental awareness. In some circumstances, this can be extraordinarily difficult. In most cases, however, it can be determined with a reasonable degree of accuracy whether the person was frightened, stressed, and overwhelmed at the time of the accident. The clinician could ask questions like: “Were you scared after the accident? Did you feel stressed, worked up, or overwhelmed? Was your heart beating rapidly? Did you have an anxiety attack?” In many cases, the circumstances of the injury were not directly experienced as distressing or traumatic—thus, it is easier to differentiate biomechanically induced confusion from psychologically induced confusion.

The accuracy of identifying disorientation and confusion can be enhanced if the clinician carefully establishes a timeline for this experience. The disorientation and confusion should be directly caused by the impact to and/or very rapid acceleration–deceleration of the brain. For example, if an individual fully recalls the accident and then describes a feeling of being dazed, confused, or even disoriented after he or she realizes the extent of the bodily injuries or detrimental consequences to others, then this is likely not due to the mild TBI *per se*. Thus, the confusion and disorientation should not follow the patient’s conscious awareness of what took place, but rather must be directly linked to the presumed cause (e.g., or acceleration–deceleration forces) of the trauma to the brain.

As previously noted, the word “dazed” was dropped in the WHO definition. If the descriptor of “dazed” captures an emotional reaction, then that would justify the omission. Thus, if a patient describes feeling “dazed” after the accident, then the clinician should use follow-up questions to differentiate between a concussion-mediated state of confusion or disorientation and a sense of being overwhelmed or in emotional shock.

Neurologic Signs and Symptoms Associated with Mild TBI

The ACRM definition (Table 1) states that a mild TBI can be diagnosed on the basis of “focal neurologic deficit(s) that may or may not be transient” (p. 86). Similarly, the WHO definition included neurologic deficits “such as focal signs, seizure, and intracranial lesion not requiring surgery” (p. 115).

Neither definition has specified the focal neurologic signs that should be used to diagnose a mild TBI. Focal neurologic signs may be associated with injury to one or more of the systems affecting vision, hearing, language, sensory–perceptual, or motor functions. The most common focal signs of brain injury, including those in the WHO definition, are: post-traumatic seizures; intracranial lesions (e.g., contusion, hematoma, hemorrhage, or edema); anosmia/hyposmia; other cranial nerve deficits; visual field cuts, diplopia, or other visual symptoms caused by CNS injury; acute non-fluent (expressive) aphasia; gait/balance problems caused by CNS injury.

In order to rely on one or more of these focal neurologic signs as the *only* basis for diagnosing a mild TBI, the sign(s) should be caused by forces applied to the head and/or neck and, if possible, supported with data by the appropriate health care provider. It is important to note that mild TBIs usually do not cause focal signs.

The DSM-IV-TR (American Psychiatric Association, 2000), in its proposed criteria for diagnosing Post-Concussional Disorder, provides the following *physical symptoms* as being caused by a concussion: physical fatigue, disordered sleep, headaches, or vertigo/dizziness.

Based on the International Classification of Diseases (10th Edition, ICD-10; World Health Organization, 1992), the following physical symptoms are expanded to include the symptoms: tinnitus and hyperacusis (increased sensitivity to sounds), photosensitivity, and reduced tolerance to alcohol or medications.

According to DSM-IV and ICD-10 some or most of these physical *symptoms* should be present in the initial days following the injury. The clear presence of some of these symptoms, combined with a plausible mechanism of injury and one or more injury severity markers (e.g., LOC or PTA), should increase the clinician’s confidence that a mild TBI has occurred. These

symptoms should be documented carefully, but they should not be the *sole basis* on which a diagnosis of mild TBI is made (especially long after the injury). Many other factors, such as diverse medical problems, chronic pain, depression, and anxiety problems can cause these symptoms (Foa, Cashman, Jaycox, & Perry, 1997; Fox, Lees-Haley, Ernest, & Dolezal-Wood, 1995; Gasquoin, 2000; Iverson, 2006; Iverson & McCracken, 1997; Lees-Haley & Brown, 1993; Mickeviciene et al., 2004; Radanov, Dvorak, & Valach, 1992; Smith-Seemiller, Fow, Kant, & Franzen, 2003).

The ACRM definition specifies “Focal neurologic deficit(s) that may or may not be transient” whereas the WHO criterion addresses this by stating “transient neurologic abnormalities such as focal signs.” Thus, agreement exists between the two definitions that the neurologic deficits or signs should be *focal*, and that *transient neurologic signs* can be part of the diagnosis of a mild TBI. A potential disagreement emerges as to the distinction between “may or may not be transient” as stated in the ACRM definition whereas the WHO definition refers only to “transient neurologic signs.” Because a definition of a mild TBI has to include criteria that allow for a diagnosis immediately following the injury, a separation between transient and or persistent neurologic symptoms is not feasible. For example, anosmia may not resolve shortly after injury or even in the months following the injury. Despite the expectation that most neurologic signs are transient, the ACRM definition indicating that signs “may or may not be transient” is arguably more accurate.

Diagnostic Exclusion Criteria

As noted by the WHO task force, the signs and symptoms of a mild TBI should not be caused by drugs, alcohol, or medications. Similarly, the effects of non-neurologic physical injuries or their treatment should not be the cause (e.g., systemic injuries, facial injuries, or intubation). Moreover, clinicians should rule out the influence of other causes, such as psychologic trauma, language barrier, or coexisting medical conditions as playing a primary role in the presentation of neurologic signs and symptoms.

As stated in the ACRM definition, a mild TBI should not be diagnosed if the GCS score is less than 13 at 30 min post-injury, the LOC exceeds 30 min, or if the PTA lasts longer than 24 hr because these criteria would indicate a more severe TBI. However, the WHO task force noted a practical problem with applying these criteria to all cases.

We agree with the ACRM definition, which specifies that the GCS score of 13–15 be assessed after 30 min post-injury. However, we recognize the practical concern that individuals with mild TBI will rarely be assessed at an emergency department within this time frame. Therefore, although an assessment of GCS score just after 30 min post-injury remains the ideal, our proposed definition permits diagnostic use of a GCS score assessed by a qualified healthcare provider at the first opportunity. (p. 115)

Given that the GCS might be administered several times, it is essential that *all GCS scores are interpreted according to a timeline*. See Fig. 1 for an example of different GCS scores over time. This patient had a witnessed LOC of approximately 3–4 min during which time he was unresponsive. Due to his unresponsiveness, his GCS would have been 10 or less had he been evaluated. An ambulance crew arrived at approximately 10 min post-injury and assessed him with a GCS of 10. They reassessed him at 20 min post-injury with a GCS of 14. The patient’s GCS was 14 at the hospital at 6 hr post-injury and a GCS of 15 was documented 12 hr post-injury. It would be a mistake in this case to assume that the patient had a moderate TBI based on the initial low GCS score of 10, obtained approximately 10 min post-injury, because the GCS was within the mild range (i.e., 13–15) within the first 30 min post-injury. Therefore, a mild TBI should be diagnosed.

Relying on medical records without knowing how LOC or PTA was assessed can lead to inaccurate conclusions. For example, a GCS of 15 at the scene of the accident does not provide sufficient evidence to rule out a mild TBI. Thus, neuropsychologists should independently examine patients even if this examination takes place days, weeks, or months post-accident. One should not assume that all health-care providers carefully assess retrospective gaps in memory (e.g., PTA). Therefore, a detailed examination of the patient’s recall, even if conducted days, weeks, or months post-injury, is essential. It is important to note, however, that the assessment of PTA long after an injury can be influenced by numerous factors, such as normal forgetting, deliberate misrepresentation (e.g., to avoid liability issues or to make oneself seem more injured), or memory that is confounded by what a person has learned (and come to believe) over time. These factors, obviously, can reduce the accuracy of PTA estimates.

Summary, Conclusions, and Future Directions

To date, there is no single definition of mild TBI that is uniformly applied across the disciplines of neurology, neurosurgery, psychiatry, physical medicine and rehabilitation, and neuropsychology. Clinical decision-making and research, including meta-analyses based on studies with different definitions of mild TBI, are weakened by heterogeneous diagnostic inclusion criteria. However, when the ACRM definition (Mild Traumatic Brain Injury Committee, 1993) was, essentially, endorsed by the WHO’s Collaborative Center Task Force on Mild Traumatic Brain Injury (Carroll et al., 2004), a definition emerged

that is likely acceptable to most neuropsychologists. Although we discussed the differences between these two definitions, it might be helpful to have a consensus conference in the future with the aim of bridging these differences and possibly considering further modifications. In the interim, this paper provides recommendations for those clinicians and researchers who are less experienced in diagnosing mild TBIs.

The diagnosis of mild TBI is based on injury characteristics. Neuroimaging is adjunctive, but in the absence of positive findings not conclusive. Neuropsychologic testing examines the consequences of a mild TBI, but cannot be used as the basis for the initial diagnosis, which must be determined on the basis of LOC, PTA, confusion and disorientation, or neurologic signs. It is well established that neuropsychologic test results can also be influenced by numerous demographic, situational, preexisting, co-occurring, and injury-related factors. Therefore, the diagnosis of a mild TBI is primarily based on a clinical interview, collateral interviews, and record review. Records of the day of injury and the first few medical contacts following the date of injury can be most helpful for an accurate diagnosis. However, records that contain an initial GCS of 15 are insufficient to rule out a mild TBI. Additional information is necessary. A thoughtful and deliberate approach should be used that *retrospectively* assesses the presence of loss or altered consciousness, gaps in memory or amnesia (retrograde and post-traumatic), and focal neurologic signs. One cannot assume that such a deliberate approach was taken by health care providers at the scene or in the emergency department.

This paper focuses on recommendations for assessing the diagnostic criteria. This paper does not address the long-term outcome subsequent to mild TBI. However, it is well established in the literature that most patients appear to recover fully within days, weeks, or months of injury. Regrettably, some patients have poor long-term outcome. When patients have a poor outcome, it can be challenging to determine why. The questions become: why did they not follow the typical recovery pattern, and what are the risk factors or individual vulnerability factors? Have these individuals actually suffered more serious injuries? The spectrum of mild TBI is very broad, falls on a continuum, and some injuries are much closer to a moderate TBI than a minor concussion sustained, for example, in sports. Thus, more refined gradations may assist us in the future. However, further issues must be explored. Are secondary gain and poor motivation influencing symptom reporting? Are there pre-injury risk factors such as depression, stress, anxiety disorders, substance abuse, compromised cognitive function, advanced age, previous neurologic injury, or poor medical health? Are there additional barriers to recovery such as chronic pain, sleep disorder, limited social support systems, or a lack of adequate information provision and follow-up medical care? Defining and accurately diagnosing mild TBI is merely the starting point, yet it is an important foundation for pursuing answers in the future to these critical outcome questions.

Conflict of Interest

None declared.

References

- American Academy of Neurology. (1997). Practice parameter: the management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology*, 48 (3), 581–585.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (text revision). Washington, DC: American Psychiatric Association.
- Carroll, L. J., Cassidy, J. D., Holm, L., Kraus, J., & Coronado, V. G. (2004). Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine*, (Suppl. 43), 113–125.
- Center for Disease Control and Prevention. (2003). *Report to Congress on mild traumatic brain injury in the United States: Steps to prevent a serious public health problem*. Atlanta: Centers for Disease Control and Prevention.
- Centers for Disease Control and Prevention. (2006). *National center for injury prevention and control*. Retrieved February 23, 2006, from <http://www.cdc.gov>.
- Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, 9 (4), 445–451.
- Fox, D. D., Lees-Haley, P. R., Ernest, K., & Dolezal-Wood, S. (1995). Post-concussive symptoms: Base rates and etiology in psychiatric patients. *The Clinical Neuropsychologist*, 9, 89–92.
- Gasquoin, P. G. (2000). Postconcussional symptoms in chronic back pain. *Applied Neuropsychology*, 7 (2), 83–89.
- Guskiewicz, K. M., Weaver, N. L., Padua, D. A., & Garrett, W. E., Jr. (2000). Epidemiology of concussion in collegiate and high school football players. *American Journal of Sports Medicine*, 28 (5), 643–650.
- Hanlon, R. E., Demery, J. A., Martinovich, Z., & Kelly, J. P. (1999). Effects of acute injury characteristics on neurophysical status and vocational outcome following mild traumatic brain injury. *Brain Injury*, 13 (11), 873–887.
- Harvey, A. G., & Bryant, R. A. (1999). Acute stress disorder across trauma populations. *Journal of Nervous and Mental Disease*, 187 (7), 443–446.
- Hunter, E. C., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalisation and derealisation. A systematic review. *Social Psychiatry and Psychiatric Epidemiology*, 39 (1), 9–18.

- Iverson, G. L. (2006). Misdiagnosis of persistent postconcussion syndrome in patients with depression. *Archives of Clinical Neuropsychology*, 21 (4), 303–310.
- Iverson, G. L., & McCracken, L. M. (1997). 'Postconcussive' symptoms in persons with chronic pain. *Brain Injury*, 11 (11), 783–790.
- Kay, T., Newman, B., Cavallo, M., Ezrachi, O., & Resnick, M. (1992). Toward and neuropsychological model of functional disability after mild traumatic brain injury. *Neuropsychology*, 6 (4), 371–384.
- Kraus, J. F., McArthur, D. L., Silverman, T. A., & Jayaraman, M. (1996). Epidemiology of brain injury. In R. K. Naravan, J. E. Wilberger, & J. T. Povlishock, (Eds.), *Neurotrauma*. New York: McGraw-Hill.
- Langlois, J. A., Kegler, S. R., Butler, J. A., Gotsch, K. E., Johnson, R. L., & Reichard, A. A. et al. (2003). Traumatic brain injury-related hospital discharges. Results from a 14-state surveillance system, 1997. *Morbidity and mortality weekly report. Surveillance summaries*, 52 (4), 1–20.
- Lees-Haley, P. R., & Brown, R. S. (1993). Neuropsychological complaint base rates of 170 personal injury claimants. *Archives of Clinical Neuropsychology*, 8 (3), 203–209.
- Lovell, M. R., Iverson, G. L., Collins, M. W., McKeag, D., & Maroon, J. C. (1999). Does loss of consciousness predict neuropsychological decrements after concussion? *Clinical Journal of Sport Medicine*, 9 (4), 193–198.
- Macciocchi, S. N., Barth, J. T., Alves, W., Rimel, R. W., & Jane, J. A. (1996). Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery*, 39 (3), 510–514.
- McCrea, M., Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., & Cantu, R. C. et al. (2003). Acute effects and recovery time following concussion in collegiate football players: The NCAA Concussion Study. *Journal of the American Medical Association*, 290 (19), 2556–2563.
- Mellick, D., Gerhart, K. A., & Whiteneck, G. G. (2003). Understanding outcomes based on the post-acute hospitalization pathways followed by persons with traumatic brain injury. *Brain Injury*, 17 (1), 55–71.
- Mickeviciene, D., Schrader, H., Obelieniene, D., Surkiene, D., Kunickas, R., & Stovner, L. J. et al. (2004). A controlled prospective inception cohort study on the post-concussion syndrome outside the medicolegal context. *European Journal of Neurology*, 11 (6), 411–419.
- Mild Traumatic Brain Injury Committee, A. C. o. R. M., Head Injury Interdisciplinary Special Interest Group. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 8 (3), 86–87.
- Moser, R. S., Iverson, G. L., Echemendia, R. J., Lovell, M. R., Schatz, P., & Webbe, F. M. et al. (2007). Neuropsychological evaluation in the diagnosis and management of sports-related concussion. *Archives of Clinical Neuropsychology*, 22 (8), 909–916.
- National Institutes of Health. (1998). Rehabilitation of persons with traumatic brain injury. *NIH Consensus Statement*, 16 (1), 1–41.
- Paniak, C., MacDonald, J., Toller-Lobe, G., Durand, A., & Nagy, J. (1998). A preliminary normative profile of mild traumatic brain injury diagnostic criteria. *Journal of Clinical and Experimental Neuropsychology*, 20 (6), 852–855.
- Radanov, B. P., Dvorak, J., & Valach, L. (1992). Cognitive deficits in patients after soft tissue injury of the cervical spine. *Spine*, 17 (2), 127–131.
- Smith-Seemiller, L., Fow, N. R., Kant, R., & Franzen, M. D. (2003). Presence of post-concussion syndrome symptoms in patients with chronic pain v. mild traumatic brain injury. *Brain Injury*, 17 (3), 199–206.
- Sosin, D. M., Snizek, J. E., & Thurman, D. J. (1996). Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury*, 10 (1), 47–54.
- World Health Organization. (1992). *International statistical classification of diseases and related health problems* (10th ed.). Geneva, Switzerland: World Health Organization.