

Ontario Psychological Association

OPA Guidelines
for Best Practices in the
Assessment of Concussion
and Related Symptoms

July 2016

ACKNOWLEDGEMENT

The Ontario Psychological Association would like to acknowledge and thank the members of the OPA Working Group on Concussion, the authors of this Guideline document, for their hard work and dedication:

Dr. Cherisse McKay and Dr. Diana Velikonja, Chairs; Dr. John Davis, Dr. Joanna Hamilton, Dr. Tobi Lubinsky, Dr. Valda Lopo, Dr. Eric Roy, Dr. Lorie Saxby, and Dr. Jane Storrie.

TABLE OF CONTENTS

PART 1: INTRODUCTION AND OVERVIEW	4
Section I: Purpose of Guidelines, Relevant Definitions and Terms of Reference	4
a) Purpose of Guidelines	4
b) Relevant Definitions/Terms of Reference	5
c) Typical Acute Effects and Recovery	5
Section II: Concussion-Related Conditions	6
a) Post-Concussive Syndrome (PCS)	6
b) Second Impact Syndrome (SIS)	11
c) Chronic Traumatic Encephalopathy (CTE)	12
PART 2: EVIDENCE-BASED ASSESSMENT OF CONCUSSION	15
Section I: Aspects of Concussion Assessment	15
a) Baseline Testing	15
b) Sideline Testing	16
c) Monitoring for Worsening Signs and Symptoms	17
d) Multidisciplinary Components of Acute Concussion Assessment	18
Section II: The Role of the Psychologist and Neuropsychologist	20
a) Self-Report Measures/Symptom Inventories	20
b) The Clinical Interview for Concussion Patients	21
c) Post-Injury Computerized Neurocognitive Testing/Screening	23
d) Neuropsychological Assessment	23
e) Symptom Validity for Concussion Patients	25
Section III: Differential Diagnosis and Comorbidities	26
PART 3: SPECIAL POPULATIONS AND CONSIDERATIONS	36
a) Concussion in Geriatric Patients (defined as individuals over age 65)	36
b) Concussion in Pediatric Patients	37
c) Concussions in Sports: The Effects of Multiple Concussions	42
PART 4: CONCLUSIONS AND EVIDENCE-BASED RECOMMENDATIONS	44
a) Typical Recovery and Post-Concussive Symptoms	44
b) SIS/CTE	45
c) Baseline Testing	45
d) Assessment/Diagnosis of Concussion	46
e) Differential Diagnosis of Concussion	46
f) Concussion in Older Adults	47
g) Concussion in Pediatric Populations	47
h) Multiple Concussions	48
APPENDIX A	49
APPENDIX B	50
REFERENCES	51

Ontario Psychological Association Evidence-Based Guidelines for the Assessment of Concussion and Related Symptoms

PART 1: INTRODUCTION AND OVERVIEW

Section I: Purpose of Guidelines, Relevant Definitions and Terms of Reference

a) Purpose of Guidelines

Concussion/mild traumatic brain injury (mTBI) has gained recent attention due to its prevalence in sports, combat, motor vehicle accidents, and its relationship to other conditions (chronic traumatic encephalopathy, depression, etc.). Despite this, there remains quite a bit of controversy, misunderstanding and debate over its diagnosis, assessment, treatment, and recovery. Given its complex and multifaceted nature, psychologists across competencies/scopes (neuropsychologists, rehabilitation psychologists, clinical psychologists, school psychologists) have become increasingly important to the assessment and treatment of concussion. While several guidelines regarding concussion (e.g., Ontario Neurotrauma Foundation) have been developed, there remains a large range in opinions, approaches, and treatment amongst health care providers, including psychologists. This, in part, is due to the almost-constantly changing empirical landscape and inconsistencies in the literature. As such, psychologists as a profession are also participating in varied practices when dealing with concussion.

Comprehensive concussion guidelines for multidisciplinary health professionals exist elsewhere and are a source of considerable information. For example, the Ontario Neurotrauma Foundation's *Guidelines for Concussion/mTBI and Persistent Symptoms (2nd Edition)* are a must-read for any health care provider working in this field. The purpose of these current Guidelines is not to duplicate these efforts. Instead, these Guidelines were created in the hopes of achieving the following goals:

1. To provide an objective 'psychologist-specific' overview of concussion, including its related conditions, comorbidities, and evidence-based assessment procedures for the purposes of informing psychologists who do not specialize in neuropsychology but may come in contact with individuals who have sustained concussions (e.g., clinical psychologists, rehabilitation psychologists, and school psychologists).
2. To provide contemporaneous and evidence-based assessment recommendations for those practicing clinical neuropsychology, including information pertaining to assessment components and clinical decision-making.

b) Relevant Definitions/Terms of Reference

One of the most recognized definitions of concussion is taken from the 2012 Zurich Conference (4th International Conference on Concussion in Sport). At that point, a panel discussion regarding the definition of concussion and its separation from mild traumatic brain injury (mTBI) was held. There was acknowledgement by the Concussion in Sport Group (CISG) that although the terms “mTBI” and “concussion” are often used interchangeably in the sporting context and particularly in the U.S. literature, others use the term to refer to different injury constructs. Based on the consensus results of the Zurich Conference, the following definition was put forth:

“Concussion is a brain injury and is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces. Several common features that incorporate clinical, pathologic and biomechanical injury constructs that may be utilised in defining the nature of a concussive head injury include:

- 1. Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an ‘impulsive’ force transmitted to the head.*
- 2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, symptoms and signs may evolve over a number of minutes to hours.*
- 3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies.*
- 4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. However, it is important to note that in some cases symptoms may be prolonged.”*

Given this definition, concussion should be considered a subset of traumatic brain injury. For the purpose of these guidelines, the terms concussion and mTBI will be used interchangeably.

c) Typical Acute Effects and Recovery

Following a concussion, many people experience a constellation of symptoms that can include headache, dizziness, fatigue, insomnia, irritability, depression, anxiety, impaired memory and concentration, and lowered tolerance for noise and light. These symptoms are thought to reflect the neurophysiology of concussion, which is conceptualized as a neurometabolic cascade and involves decreased protein synthesis and reduced oxidative capacity (Fischer & Vaca, 2004). Multiple independent meta-analytic studies have shown that the overwhelming majority of individuals who experience a concussion make excellent neurocognitive and neurobehavioral recovery within minutes to weeks following the injury (Binder, Rohling, & Larrabee, 1997; Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Rohling et al., 2011). However, it has always been recognized that a portion of individuals continues to be symptomatic beyond this period of time.

Rate of recovery from a concussion is one of the most important factors in the assessment and management of this injury. Most of the literature examining typical concussion recovery has come from sports concussion populations. The measure most often used in charting recovery is symptom expression, which reflects the athlete's self-report of symptoms. Many studies suggest that symptom expression returns to baseline level within 7-14 days (Henry, Elbin, Collins, Marchetti, & Kontos, 2016) indicating that recovery occurs over this time frame. Other studies, however, have identified symptom expression measures of recovery to be longer, between 21 and 28 days. The discrepancy appears due to the different measures of recovery. The '7-14 day' studies used return of symptoms to baseline as the outcome measure whereas the '21-28 day' studies used return to no symptoms at all as the measure of recovery. One of these latter studies found that symptoms declined rapidly over the first two weeks and then plateaued over the third and fourth weeks (Henry, et al., 2016).

A number of factors have been found to affect the time to recover, including age. As aforementioned, some studies have found that children take longer to recover than adults (Williams, Paetz, Giza, & Brolio, 2015). Other studies comparing high school versus university athletes have found differences in the pattern of recovery, depending on the measure examined. For example, high school athletes reported symptoms for a longer period post-concussion than did university athletes, although the two groups did not demonstrate differences in the pattern of recovery on neurocognitive measures. In other words, objective and subjective measures may show disparate results at times.

Another factor in recovery is gender as some studies have found that females take longer to recover. Females do not differ from males in initial self-reported symptoms but by two weeks post-concussion, males begin to show a more rapid decline in symptoms. While there are no differences in the pattern of recovery in neurocognitive function, males showed a larger decline in dizziness than did females (Henry, et al., 2016).

These sex and age differences in recovery demonstrate the importance of another factor - the measure used to reflect recovery. A recent study revealed that symptom expression, neurocognitive functions and vestibular function all appear to have different patterns of recovery.

Overall, a concussion is not a simple injury that has a singular recovery pattern but rather reflects an amalgamation of symptoms and functions that may recover in different trajectories. In order to fully understand recovery, it would seem imperative to use a multidimensional approach. Given the heterogeneity of these trajectories it is important to adopt an individualized approach to examining recovery.

Section II: Concussion-Related Conditions

a) Post-Concussive Syndrome (PCS)

In the scientific literature, the term postconcussion syndrome (PCS) has been used to refer to the persistence of symptoms following a concussion. PCS remains a much debated topic and for several good reasons. Firstly, there is currently no universally

accepted definition of this syndrome. The International Classification of Diseases Tenth Revision (ICD-10) offers diagnostic criteria for PCS, as does the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), in appendix form. The ICD-10 criteria includes:

“A history of head trauma with loss of consciousness preceding symptom onset by a maximum of 4 weeks and the presence of 3 or more of the following symptoms: Headache, dizziness, malaise, fatigue, noise intolerance, irritability, depression, anxiety, emotional lability, subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment, insomnia, reduced alcohol tolerance, and preoccupation with above symptoms and fear of brain damage with hypochondriacal concern and adoption of sick role (World Health Organization. International statistical classification of disease and related health problems (10th edition).”

The DSM-IV criteria, in contrast, includes:

1. *History of TBI causing "significant cerebral concussion";*
2. *Cognitive deficit in attention and/or memory;*
3. *Presence of at least 3 of 8 symptoms (e.g., fatigue, sleep disturbance, headache, dizziness, irritability, affective disturbance, personality change, apathy) that appear after the injury and persist for 3 months;*
4. *Symptoms that begin or worsen after injury;*
5. *Interference with social role functioning; and,*
6. *Exclusion of dementia due to head trauma and other disorders that better account for the symptoms.*

Criteria C and D require symptom onset or worsening to be contiguous to the head injury, distinguishable from preexisting symptoms, and have a minimum duration of three months.

It has been demonstrated that the rate in which PCS is diagnosed varies greatly based on whether ICD-10 or DSM-IV criteria is used (McCauley, Boake, Pedroza, Brown, Levin, Goodman, et al., 2008). Boake, McCauley, Levin, Pedroza, Contant, Song et al. (2005) performed a prospective study and demonstrated large differences between the prevalence of PCS using ICD-10 and DSM-IV criteria with the former being more inclusive. Furthermore, both criteria sets have been shown to have limited specificity to brain injury with the ICD-10 criteria not even requiring objective evidence of cognitive problems or an impairment to exist in a person's functional abilities. Additionally, while both the ICD-10 and DSM-IV criteria set require that an individual have sustained a TBI, neither offers an operational definition of TBI. The DSM-IV criteria does provide injury severity markers including a loss of consciousness greater than five minutes, a post-traumatic amnesia greater than 12 hours, or the new onset of seizures suggesting perhaps that it was the intent of the authors to imply that PCS would be unlikely in individuals who have not had these more significant injury severity markers.

Perhaps the most serious and obvious problem with the ICD-10 and DSM-IV criteria is that they causally link subjective, self-reported symptoms to the original concussion. The ICD-10 criteria do not consider other possible etiologies for the reported symptoms resulting in a corresponding lack of emphasis on differential diagnoses to explain the presented symptoms. Interestingly, PCS has not been included in DSM-5 in any diagnostic category, appendix, or area for further study. The DSM-5 provides criteria for *Major or Mild Neurocognitive Disorder due to Traumatic Brain Injury* to refer to the cognitive impairments that can develop following a traumatic brain injury (TBI). The DSM-5 recognizes that persons with concussion can have symptoms that co-occur including depression, irritability, fatigue, headache, photosensitivity, and sleep disturbance but they note that, like neurocognitive symptoms, these co-occurring symptoms tend to resolve in the weeks following a concussion and that subsequent deterioration in these areas, or deviation from the typical course of concussive injury, should trigger consideration of additional diagnoses.

In addition to an absence of an accepted definition, there is also no consensus on the prevalence of PCS as depending on the definition and the population examined and it has been estimated that anywhere from 5 to 90% of patients experience PCS symptoms shortly after a traumatic brain insult (Butler, 2013; Sullivan, Edmed, & Cunningham, 2013). A 15% prevalence of PCS has frequently been cited over the past 25 years, which was spawned from the results of a study in the mid-1970s by Rutherford and colleagues who followed people with concussions who were admitted to the hospital. They found that 14.5% of them had at least one symptom at the one-year post-injury mark. However, they also found that fewer than 5% had four or more symptoms at one year but this finding is rarely cited. A review of the literature suggests that many of the historical prospective studies that reported poor outcomes following brain injury were based on cases that had been hospitalized and/or seen in the emergency department. Patient groups were often broadly defined in terms of injury severity and many of these studies very likely included patients with more severe forms of TBI (such as complex mild or moderate TBIs).

There are also conflicting findings in the literature regarding the duration of PCS symptoms with a substantial minority of people reporting symptoms at one month (Bazarian & Atabaki, 2004; Kashluba, Casey, & Paniak, 2006), three months (Sigurdardottir, Andelic, Roe, Jerstad, & Schanke, 2009), six months (Hou, Moss-Morris, Peveler, Mogg, Bradley, & Belli, 2012), and 12 months (Ahman, Saveman, Stycke, Bjornstig, & Stalnacke, 2013; Stalnacke, Bjornstig, Karlsson, & Sojka, 2005) following injury. There is even evidence that some people who do not report significant PCS symptoms shortly following a concussion nevertheless go on to report symptoms many months or years post-injury (Dikmen, Machamer, Fann, & Temkin, 2010). This pattern in particular calls into question the exact etiology of reported symptoms, given the acute neurological effects of concussion.

In 2004, the World Health Organization (WHO) Collaborating Centre Task Force on mild TBI published the first systematic review of the literature on the course and prognosis after mild TBI/concussion in adults (Carroll, Cassidy, & Peloso, 2004). They concluded

that many of the post-traumatic symptoms reported following a concussion are also commonly reported in the acute stages of other injuries and are not specific to concussion. Specifically, it has been demonstrated that PCS symptoms are reported fairly frequently by healthy adults (Chan, 2001; Garden, Sullivan, & Lange, 2010), people with chronic pain (Smith-Seemiller, Fow, Kant, & Franzen, 2003; Stalnacke, 2012), as well individuals with post-traumatic stress disorder (Bryant, Creamer, O'Donnell, Silove, Clark, & McFarlane, 2009; Bryant, 2011) and depression (Lange, Iverson, & Rose, 2011). The Task Force concluded that while most individuals will recover from a concussion within a period of three months to one year, there are a number of factors that are associated with protracted recovery. This includes pre-existing physical limitations, prior brain injury, prior neurological condition, prior psychiatric history, premorbid personality traits, negative injury perceptions, and being involved in compensation-related litigation (Borg et al., 2004) with compensation and litigation factors being the single most stable predictor of prolonged PCS in concussion samples (Carrol et al., 2004). The implication is that because the course and outcome of a concussion varies with these factors, the mild severity and good prognosis of the initial brain injury sustained does not necessarily bear a strict relationship to the course of recovery.

In 2014, the International Collaboration on mild TBI Prognosis (Cassidy et al., 2014) updated the original Task Force's findings and reached similar conclusions, namely that postconcussion symptoms are equally as prevalent in individuals who have sustained non-head injuries (Davis, 2002; Landre, Poppe, Davis, Schmaus, & Hobbs, 2006; Rush, Malee, Moessnes & Brown, 2004) and that psychosocial factors are more strongly associated with outcomes than biomedical factors thought to determine recovery (Cassidy, Cancelliere, Carroll, Cote, Hincapie, Holm, et al., 2014). Consequently, they suggested that symptoms be regarded as "common reactions" to health stressors and that the term postconcussion *symptoms* be used instead of postconcussion *syndrome* given their widespread report.

Contextual factors have been shown to influence an individual's expectations for recovery following a concussion. This includes explicit or implicit messages from the media, healthcare providers/systems of care, and the forensic arena (Vanderploeg, Belanger, & Kaufmann, 2014). These factors have been referred to as nocebo effects, or inherently "inert" factors that may create negative expectancies for recovery and therefore impede a given person's progress and recovery. Moreover, it has been suggested that people's retrospective perception of their past functioning can also influence their perception of their current functioning. A person might falsely perceive a bigger difference than is actually the case between their past and present functioning due to over-estimating past abilities or to under-estimating past problems. This has been referred to as the "good-old-days bias" (Iverson, Zaler, & Lang, 2007).

From a neuropsychological perspective specifically, and in light of the multiple and often comorbid factors that can impact the onset and perpetuation of cognitive symptoms following a concussion, the task of successfully predicting the rate at which a person's cognitive functioning will improve following a concussion is rather challenging. In the

acute stage post-injury, cognitive impairment likely reflects the neurophysiology of the concussion. However, when faced with symptomatic patients who have sustained a single, uncomplicated concussion and who present in a chronic timeframe (i.e., more than one year following the injury), the research literature suggests that psychologists should not attribute the reported cognitive symptoms to the remote concussion, even if patients attribute their symptoms to it. In such a case, careful and thorough differential diagnoses should be considered to explain the perpetuation of symptoms. The neuropsychologist should assess and document the somatic, cognitive, and emotional/behavioural symptoms. The assessment should include a review of currently prescribed medications, over-the-counter medications, substance use, and symptoms of mood, anxiety, and adjustment reactions as each of these factors, both independently and collectively, can deleteriously affect a person's cognitive abilities. Assessments that do not take in to account consideration of premorbid factors (e.g., pre-existing physical conditions, prior brain injury, premorbid personality traits) as well as involvement in compensation-related litigation and placebo effects are likely overlooking important and relevant information that may be contributing to the onset and perpetuation of symptoms. Psychologists must keep in mind that if a message is sent that a person has permanent cognitive impairment as a result of the pathophysiological effects of a concussion, this can lead to treatment that is potentially deleterious both physically and/or psychologically, and can also lead to the perpetuation of actual impairment creating a more recalcitrant condition with a poorer prognosis. This is referred to as iatrogenesis, or adverse health effects caused by medical treatment (Iverson, Zaler, & Lang, 2007).

There is strong evidence that suggests that providing individuals with education about the mechanisms of a concussion, an explanation of symptoms, expectations for recovery, and graduated reintegration into physical activity, work, and/or school decreases the severity and duration of symptoms. It is recommended that health care providers and/or clinicians provide education to their clients post-injury as quickly as possible. Mittenberg, Tremont, Zielinski, Fichera, and Rayls (1996) demonstrated that giving a patient an opportunity to meet with a clinician, supervised by a neuropsychologist, for one hour before hospital discharge during which they were provided with psychoeducation resulted in significantly shorter symptom duration and significantly fewer symptoms at 6-month follow-up compared to a sample of matched controls who received routine hospital care. Symptomatic treatment following a concussion is also critical. This involves ensuring that any underlying and/or comorbid conditions such as depression, anxiety, and insomnia are targeted. It is also often helpful to assist patients to reduce stress, facilitate adaptive coping mechanisms, problem solve life stressors, increase exercise, and resume a more active and normal lifestyle. In sum:

- There is no universally agreed upon etiology, pathophysiology, definition, prevalence, or criteria for PCS
- While post-concussion symptoms in the acute stage of injury likely reflects the neurophysiology of the concussion, clinicians should keep in mind that PCS is a non-specific cluster of symptoms that can be mimicked by a number of pre-existing or co-morbid conditions

- Careful and thorough differential diagnoses should be considered when recovery and/or outcome severity is atypical and does not follow the natural course expected from such injuries
- Individuals benefit from the provision of psychoeducation as this has been shown to result in significantly shorter symptom duration and significantly fewer symptoms at 6-month follow-up
- Treating the underlying and/or comorbid condition(s) is imperative for improvement in a person's functional abilities and outcomes

b) Second Impact Syndrome (SIS)

Second Impact Syndrome (SIS) is a term attributed to Saunders and Harbaugh (1984) to describe circumstances in which an athlete suffering from post-concussive symptoms returns to play and sustains a second head injury that can result in diffuse cerebral swelling, brain herniation and death.

Pathophysiology of Suspected SIS: Following an initial concussion, a patient may develop cerebral edema, accounting for loss of consciousness, memory impairment, disorientation and headache. Typically, the brain's regulatory mechanisms compensate for this physiologic stress and protect against massive swelling. This is thought to be accomplished by acutely limiting cerebral blood flow, which leads to accumulation of lactate and intracellular acidosis (DeSalles, Kontos, Ward, Marmarou, & Becker, 1987).

After the initial phase, a state of altered cerebral metabolism occurs and may last up to ten days, involving decreased protein synthesis and reduced oxidative capacity. Research suggests that the loss of consciousness after head injuries, the development of secondary brain damage, and the enhanced vulnerability of the brain after an initial insult can be explained largely by characteristic ionic fluxes, acute metabolic changes, and cerebral blood flow alterations that occur immediately after cerebral concussions. Extracellular potassium concentration can increase massively in the brain after concussion, followed by hypermetabolism that makes the brain more vulnerable and susceptible to herniation and death after a second sub-lethal insult of even less intensity (Fischer & Vaca, 2004).

Evidence for SIS: Support for SIS gained momentum in the literature with Robert Cantu's identification of 35 cases in American high school football players from a review of data collected by National Center for Catastrophic Sport Injury Research between 1980 and 1993 (Cantu, 1998). Paul McCrory, however, challenged the existence of SIS by noting that it appeared to be a phenomenon unique to the United States (McCrory, 2001). His review of the literature failed to find any incidence of SIS in Asia, Europe, or Australia, where football has a concussive injury rate some 15 times higher than in American football.

McCrory (2001) also reviewed 17 of Cantu's 35 cases to determine whether they met four basic criteria ascribed to a definitive diagnosis of SIS (i.e., medical documentation after first impact, documented ongoing symptoms secondary to first impact, witnessed second impact with rapid cerebral deterioration, and neuropathological or neuroimaging evidence of cerebral swelling). Not a single case met the criteria, leading McCrory to

conclude that the existence of SIS was in doubt. Further, he notes that in the overwhelming majority of these cases, there was neuroradiologic evidence of intracranial bleeding (typically acute subdural hematoma). The traditional view of SIS is that it occurs in the absence of structural injury.

A current review of the literature demonstrates that there is still little epidemiological data supporting SIS, with most articles referencing anecdotal case reports or series (McCrory, Davis, & Makdissi, 2012; Mori, Katayama, & Kawamata, 2006; Ropper & Gorson, 2007). Muller and Cantu (2011) note that the National Center for Catastrophic Sport Injury Research has identified 145 cases of catastrophic cerebral injury through 2010, only one of which was considered to be “possible” SIS. In this case, however, there was neuroimaging confirmation of an acute subdural hematoma.

Summary: Thirty years after introduction of the term SIS, there remains little consensus or systemic evidence for its existence. McCrory, Davis and Makdissi (2012) make a compelling argument that the term is misleading, and should be replaced with “diffuse cerebral swelling.” Herniation and death following brain injury, while exceedingly rare, can result following a single insult. It may be that an underlying genetic susceptibility is responsible rather than a response to impact alone.

It is recommended that evidence-based practice in concussion avoid use of the term SIS, particularly as the threat of catastrophic cerebral deterioration and death can cause unnecessary alarm. The focus should remain on prevention, safety and adherence to return-to-activity guidelines.

c) Chronic Traumatic Encephalopathy (CTE)

CTE is described as a progressive degenerative brain disease considered secondary to a history of repetitive brain trauma. Symptoms include impairments in memory and executive functioning, behavioral and personality disturbances (e.g., apathy, depression, paranoia, irritability, impulsiveness, aggression, suicidality), and parkinsonism. CTE is thought to result from the build-up of an abnormal protein called Tau, and changes in the brain can begin months or decades after the last traumatic event. CTE can only be definitively diagnosed on autopsy.

CTE was first described in boxers in 1928 by pathologist Harrison Martland, who referred to the condition as “punch drunk.” In 1937, Millspaugh wrote about the same constellation of symptoms under the term “dementia pugilistica.” Over time, it became clear that boxers were not the only ones suffering from this neurodegeneration and the term “chronic traumatic encephalopathy” was introduced by Miller (1966). CTE has since been identified clinically and neuropathologically in players of football, wrestling, soccer, hockey, and following non-sporting activities associated with repeated mild head trauma, such as physical abuse and seizures.

Onset: CTE onset appears most typically in mid-life. In some, the first symptoms are behavioural with family and friends noticing irritability or apathy. In others, cognitive symptoms such as memory difficulties and changes in executive functioning are the first signs. As CTE progresses, impairments in speech, vision and movement may be

observed. The development of dementia in CTE appears to be relatively infrequent, although this finding may be obscured by early death in those with neuropathologically diagnosed CTE (through suicide, accident, or overdose).

Neuropathology: Neuropathological studies of athletes with a history of repeated mild head injuries have demonstrated anterior cavum septum pellucidum fenestrations and ventricular enlargement. The former may be the result of head impact forces moving through the ventricular system. Other features of CTE identified on gross examination include atrophy of the frontal and temporal cortices and the medial temporal lobe, thinning of the hypothalamic floor, shrinkage of the mammillary bodies, substantia nigra pallor, and hippocampal sclerosis. Atrophy of the cerebrum, diencephalon, basal ganglia, brainstem, and cerebellum may result in an overall reduction in brain mass (McKee, Cantu, Nowinski, et al., 2009).

Microscopically, CTE is characterized by an abundance of neurofibrillary inclusions, in the form of neurofibrillary and glial tangles, and neuropil threads. The main protein that composes neurofibrillary tangles is Tau. While there are similarities to Alzheimer's Disease, in CTE the distribution of Tau pathology is in the superficial cortical laminae in layers II and III, rather than in the large projection neurons of layers III and IV. And while the distribution of cortical neurofibrillary tangles in Alzheimer's is uniform, Tau pathology is irregular and largely confined to uneven foci in the frontal and temporal cortices in CTE. The density of tangles in the medial temporal lobe is greater in CTE than in Alzheimer's, and prominent in the diencephalon, basal ganglia, and brainstem (McKee, Cantu, Nowinski, et al., 2009).

The mechanism underlying neurofibrillary tangle formation following repeated brain trauma remains unclear. It has been suggested that initial impact results in diffuse axonal injury that is aggravated by subsequent injuries. Traumatic axonal injury produces changes in axonal membrane permeability, ionic shifts, and the release of caspases and calpains that might trigger Tau phosphorylation, misfolding, truncation, and aggregation, as well as breakdown of the cytoskeleton with dissolution of microtubules and neurofilaments (Giza & Hovda, 2001).

Beta-amyloid (A β) deposits are found in 40–45% of individuals with CTE, but in almost all cases of Alzheimer's Disease. Neuritic plaques, which are necessary for a diagnosis of Alzheimer's are rarely found in CTE (McKee, Cantu, Nowinski et al., 2009).

Diagnosis: Clinical diagnosis of CTE is difficult because of the similarity of presenting complaints to Alzheimer's Disease and frontotemporal dementia. In addition, there are currently no consensus diagnostic criteria and no large-scale longitudinal studies comparing clinical and pathological diagnoses.

Remote history of head injury is insufficient to suggest a diagnosis of CTE since brain trauma has been implicated as a risk factor in Alzheimer's, Parkinson, ALS and other neurodegenerative diseases (Chen, Richard, Sandler, Umbach, & Kamel, 2007; Goldman, Tanner, Oakes, Budhikanok, Gupta, & Langston, 2006).

At this point in time, neuropathological confirmation of a CTE diagnosis is required.

Incidence and Prevalence: While the incidence and prevalence of CTE is currently unclear, it is considered likely that it varies by sport or activity, duration of exposure, age, genetic predisposition, and other factors.

From 1928 through 2009, there were only 49 cases consistent with CTE described in the medical literature, 39 of whom were boxers. CTE was not well known in sports outside of boxing until a Pittsburgh medical examiner named Bennet Omalu identified CTE in two former Pittsburgh Steelers who died in his jurisdiction in 2002 and 2005. This work drew the attention of Sports Legacy Institute (SLI) co-founder Chris Nowinski, and he began reaching out to families of recently deceased former athletes to review symptoms and clinical course and, where possible, obtain permission for post-mortem brain studies.

In 2008 Nowinski and Dr. Robert Cantu, another co-founder of SLI, partnered with the Boston University School of Medicine to create the Center for the Study of Traumatic Encephalopathy (BU CSTE), the world's first research center dedicated to studying CTE.

In 2009, BU Professor Ann McKee, a neuropathologist and one of the world's foremost neurodegenerative disease experts, published what is considered the seminal paper on all known cases of CTE ever identified in the medical literature, which by this time numbered 52.

Concussion and CTE: The relationship between concussion and CTE has not been well-established. It has been suggested that repetitive axonal derangement may trigger metabolic, ionic, membranous, and cytoskeletal disturbances that initiate the pathological cascade that results in CTE in those susceptible (Giza & Hovda, 2001; Yuen, Browne, Iwata, & Smith, 2009).

At this time, there is only limited evidence of a link between sports concussions and an increased risk of late-life cognitive and neuropsychiatric impairments (McCrory, Meeuwisse, Aubry, et al, 2013). According to Karantzoulis and Randolph (2013), there are a number of issues with both ascribing symptoms to CTE and drawing a correlation between CTE and concussion:

- CTE has been diagnosed following autopsies of brains donated from families reporting premorbid cognitive and behavioural symptoms. Non-random samples can, however, bias findings because they may not be representative of the entire population of retired players.
- The largest epidemiological study of retired NFL athletes, which included 3,439 players, found that suicide rates were actually substantially lower among the athletes than the general population. This appears at odds with the description of suicidality as a key feature of CTE.

- Some studies of retired NFL players suffering from mild cognitive impairment, have demonstrated virtually the same symptom constellation in non-athletes. These findings cast doubt on the notion that CTE is unique to athletes who have sustained concussions.

Most significantly, the presence of abnormal Tau proteins in the brain may not be a reliable indicator of CTE. Various case studies have found that between 20 and 50 percent of subjects with abnormal Tau deposits were asymptomatic. It is well known that non-athlete seniors found to on autopsy to have Alzheimer's Disease pathology, including Tau deposition, did not demonstrate associated cognitive or behavioural symptoms pre-morbidly (Karantzoulis & Randolph, 2013).

Summary: While CTE may represent a distinct taupathy, the incidence in athletic populations remains unknown. Given that there is not yet consensus as to a direct relationship between concussion and CTE, it would be wise to proceed cautiously when discussing the likelihood of future neurodegeneration with patients. The focus should remain on prevention, safety and the implementation of evidence-based return-to-activity guidelines.

PART 2: EVIDENCE-BASED ASSESSMENT OF CONCUSSION

At its best, concussion assessment and management should be multidisciplinary and comprehensive. Although not all of the recommended components are conducted by psychologists, it is important for one working with the population to understand all of the key components. As such, the following section contains a description of the current literature pertaining to 'gold-standard' multidisciplinary concussion assessment.

Section I: Aspects of Concussion Assessment

a) Baseline Testing

Baseline testing is typically conducted pre-season to establish an athlete's normal cognitive functioning. Post-injury test results can then be compared to the baseline to identify concussion signs and symptoms, inform treatment and direct return to activity (school, work, play, etc). Popular baseline tests include:

- Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT)
- King-Devick Test for Concussions
- Computerized Cognitive Assessment Tool (CCAT)
- Concussion Resolution Index (CRI)
- Automated Neuropsychological Assessment Metrics (ANAM)

ImPACT: Developed by neuropsychologists, ImPACT is the most-widely used computerized concussion evaluation system in North America. It has also been the most rigorously studied and scientifically validated tool. ImPACT is to be administered by specific professionals (e.g., psychologists, neuropsychologists, physicians) who have

completed a training program prior to using the test, which includes a commitment to a best practice model. Clinicians can achieve expert status by seeking the Credentialed ImPACT Consultant (CIC) designation, which requires report review and an oral examination. ImPACT is endorsed by leading sports leagues (eg., CFL, NFL, NHL, MLB, NASCAR) and authorities, governing bodies, and colleges and universities.

Baseline Testing Cautions: There are a number of factors that can undermine the validity of baseline testing, including:

- Distracting environment
- Reading difficulties
- Attentional or learning difficulties
- Effort
- Fatigue
- Illness
- Right-left confusion
- Colour blindness.

Sandbagging: Sandbagging is an attempt by athletes to perform poorly on baseline testing so that, in the event of a concussion, post-injury testing results appear to be within normal limits. In most sports, there is great incentive to remain in the game, and sandbagging is thought to be quite common. It is vital that concussion tests include validity indicators that will help trained clinicians identify discrepancies indicative of sandbagging.

Uncontrolled and unsupervised access to online concussion baseline testing undermines the validity of baseline testing and should never be allowed. Testing should always take place in person, and under the supervision of a trained healthcare provider. ImPACT automatically flags suspicious test protocols.

b) Sideline Testing

While it is unlikely that psychologists would be involved in sideline testing, we should be aware of the various tests developed for this use. According to the Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport Held in Zurich (McCrory, et al., 2013):

“Sideline evaluation of cognitive function is an essential component in the assessment of this injury. Brief neuropsychological test batteries that assess attention and memory function have been shown to be practical and effective. Such tests include the SCAT3, which incorporates the Maddocks’ questions and the Standardized Assessment of Concussion (SAC). It is worth noting that standard orientation questions (e.g., time, place and person) have been shown to be unreliable in the sporting situation when compared with memory assessment.

It is recognized, however, that abbreviated testing paradigms are designed for rapid concussion screening on the sidelines and are not meant to replace comprehensive neuropsychological testing which should ideally be performed by trained neuropsychologists who are sensitive to subtle deficits that may exist beyond the acute

episode; nor should they be used as a stand-alone tool for the ongoing management of sports concussions.

It should also be recognized that the appearance of symptoms or cognitive deficit might be delayed several hours following a concussive episode and that concussion should be seen as an evolving injury in the acute stage.”

Evaluation on the sideline is typically used to determine whether an athlete can continue playing or must sit out. It is important to note that, in concussion, neurological findings are within normal limits with the exception of perhaps mental status and balance deficits. Subtle signs and symptoms of concussion, though, are difficult to identify through standard orientation questions or traditional balance testing. In an attempt to determine whether an athlete has sustained a concussion, sideline assessment should include a physical examination in addition to a concussion assessment tool. It is also essential to monitor for subsequent deterioration. If there is any suspicion that an athlete has sustained a concussion, they should not return to play. Common sideline tools include:

- Standardized Assessment of Concussion (SAC)
- Sport Concussion Assessment Tool-3 (SCAT3)
- Concussion Assessment and Response: Sport Version (CARE)
- Mayo Concussion Test (MCT) and Post-Concussion Symptoms Scale (PCSS)
- Pocket Concussion Recognition Tool
- VHSL On-Mat Concussion Evaluation Protocol
- Hospital-Based Concussion Evaluation.

c) Monitoring for Worsening Signs and Symptoms

Individuals suspected of having sustained a concussion should be closely monitored for worsening signs and symptoms in the 24 to 48 hours following the injury. They should be checked frequently while sleeping to ensure normal respiration. There are conflicting opinions on whether waking them to determine how easily they are roused is indicated, or detrimental to recovery. To err on the side of caution, in the event of a complication such as hemorrhage, it would seem prudent to awaken the individual at least once to ensure they readily do so and to check for complaints.

If any of the following signs are evident, the individual should be taken to the emergency room for evaluation:

- Severe or increased headache
- Double vision
- Unequal pupils
- Convulsions
- Unusual/increased drowsiness
- Bleeding/clear fluid from the ear/nose
- Projectile or repeated vomiting
- Unusual stiffness in the neck area
- Severe personality changes
- Weakness in either arm(s) or leg(s)

- Numbness in the face/extremities
- Loss or altered consciousness
- Confusion or disorientation
- Imbalance or motor problems

d) Multidisciplinary Components of Acute Concussion Assessment

Overall, acute concussion is best assessed and treated in a contemporaneous and multidisciplinary way. A suggested algorithm for the assessment of concussion (Scorza, Raleigh & O'Connor, 2012) can be found in Appendix B.

Hospital or Physician Concussion Evaluation: Evaluation in the hospital or by the individual's primary care physician should include the following:

- Comprehensive history, including observations of witnesses
- Neurological evaluation of mental status, cognitive functioning, gait and balance
- Medical assessment, including a comprehensive history
- Determination of the need for emergent neuroimaging

Neurological Evaluation: The following table presents neurologic examination findings suggestive of more severe injury in patients with suspected concussion:

Type of assessment	Findings
Balance	Romberg sign, postural instability, unsteadiness
Cranial nerves	Vision problems; unequal or fixed, dilated pupils; abnormal extraocular movements; or other abnormal cranial nerve findings may be suggestive of brainstem lesion
Deep tendon reflexes	Hyperreflexia or Babinski reflex suggests upper motor neuron lesion
Finger-to-nose test	Abnormal findings suggest coordination deficit
Gait	Ataxic gait may suggest cerebellar dysfunction
Mental status	Prolonged loss of consciousness (more than 60 seconds); somnolence or confusion; disorientation; deficit in language, speech, or long-term memory
Muscular strength	Weakness or unequal strength, decreased tone; involuntary movements may indicate basal ganglia or cerebellar injury
Sensory assessment of dermatomes	Numbness or abnormal sensation can be traced to spinal nerve root

Neuroimaging: In concussion, conventional neuroimaging (CT, MRI) is typically found to be within normal limits but should be ordered when exclusion of an intracerebral or

structural lesion, skull fracture, or cervical spinal injury is warranted given issues with consciousness or worsening symptoms.

While research has demonstrated fMRI activation patterns that correlate with concussion symptom severity and recovery, this modality is not often available in Ontario hospitals. Other imaging technologies (e.g., positron emission tomography, diffusion tensor imaging, magnetic resonance spectroscopy) are proving interesting in studies but are still only in the early stages of development.

Generally, imaging in the ER is overused and contributes little to the management of concussion. Neuroimaging conducted immediately after the injury does not rule out the potential for emerging issues such as intracranial hemorrhage. Even when imaging proves to be within normal limits, patients must be carefully monitored for worsening signs and symptoms, and returned to the ER for further evaluation as required. Indications for neuroimaging can be found in Appendix A.

Balance Testing and Postural Stability: Balance testing, either clinical or using force plate technology, can reveal acute deficits in postural stability in the 72 hours following a concussion. These tests have proven to be a reliable indicator in athletes who have sustained a concussion and should be part of the evaluation process where available. The assessment of balance and postural stability has been found to be the most sensitive within 24 hours post-injury.

Dynamic Stability Assessment (gait): Postconcussive impairments in dynamic stability can include slowed ambulation speed, shorter steps, and increased mediolateral motion of the body. These deficits may be present up to 28 days post-injury, and are typically assessed using a dual-task paradigm where the individual must perform a cognitive task while walking.

Postural Stability Assessment (balance): Balance problems have been shown to persist up to 30 days post-injury, but most typically is seen for only 3 to 5 days. In addition to the Romberg Test, the most common measures are the Balance Error Scoring System (BESS) and the Sensory Organization Test (SOT), which uses a force plate. While these are sensitive to concussion diagnosis, there is little evidence that repeated testing is useful for monitoring recovery.

Vestibular/Ocular Assessment: Common measures used to detect deficits in vestibular or visual function indicative of concussion include:

- King-Devick (K-D) Oculomotor Test
- Vestibular/Oculomotor Screening (VOMS)
- Dizziness Handicap Inventory (DHI)
- Activities-specific Balance Confidence Scale (ABC)
- Visual Vertigo Scale (VVS)

Electrophysiological Studies: There has been considerable research using electrophysiological recording techniques (e.g., evoked response potential, cortical

magnetic stimulation and electroencephalography) that has revealed abnormalities in post-concussed individuals. Unfortunately, the reliability of these studies to differentiate concussed athletes from control subjects remains poor. As a result, the clinical significance of electrophysiological changes secondary to concussion has not been established.

Section II: The Role of the Psychologist and Neuropsychologist

As an expert in brain-behaviour relationships, neuropsychologists hold a unique position in the assessment of concussion and its consequences. Given the complex and multifaceted nature of concussion, especially in cases where symptoms persist into chronic stages, neuropsychologists offer critical and irreplaceable insight into the interplay of neurological, psychological, and behavioural factors. Although this knowledge and skill set leads neuropsychologists to be an integral member of the rehabilitation treatment team, the focus of these Guidelines is the role of neuropsychologists in the assessment domain. As a regulated expert in brain-behaviour relationships, neuropsychologists, along with medical practitioners, are able to diagnose concussion. As it is not within the scope of practice for a non-neuropsychologist (i.e., Clinical, Health, or Rehabilitation Psychologists) to diagnose concussion/brain injury, this section will focus more on the role of neuropsychologists. However, it serves non-neuropsychologists well to understand concussion and how to screen for its effects and how a history of concussion may affect a client's clinical presentation.

a) Self-Report Measures/Symptom Inventories

Symptom inventories have long been popular tools in the evaluation of concussive symptoms. There is a wide range of measures available to clinicians, including:

- Acute Concussion Evaluation (ACE)
- Graded Symptom Checklist/Scale (GSS/GCS)
- Concussion Symptom Inventory (CSI)
- SCAT Post Concussion Symptom Scale (SCAT-PCSS)
- Post-Concussion Symptom Inventory (PCSI)
- Rivermead Post-Concussion Symptoms Questionnaire
- Head Injury Scale (HIS)
- Neuromotor/Neurosensory Evaluation

Given the difficulty in differentiating post-concussive symptoms from psychological comorbidities, self-report measures assessing psychoemotional functioning can also be helpful in evaluating the individual's symptom presentation and etiology. By administering both a concussion and psychoemotional inventory, the psychologist can evaluate and identify overlapping/redundant symptoms to determine how many 'concussive-specific' symptoms remain after controlling for psychological distress and vice versa. Popular psychoemotional assessments include:

- Beck Depression Inventory (BDI)
- Beck Anxiety Inventory (BAI)
- Sport Anxiety Scale (SAS)

- Athletic Coping Skills Inventory (ACSI).

Although symptom checklists can help the clinician determine the presence of symptoms and monitor symptom progression, significant issues arise when they are the only tool used for actual diagnosis and assessment of concussion. As aforementioned in the section describing PCS, when evaluating symptom presentation it is important for the clinician to appreciate the respective specificity and sensitivity of 'post-concussion' symptoms. Many post-concussive symptoms (e.g., headache, sleep disturbance, fatigue, memory problems) are non-specific in that many other factors can lead to similar symptomatology. In contrast, clinicians should ensure they ascertain whether the patient is demonstrating symptoms that are more specific to concussion (e.g., nausea, noise sensitivity). Strict reliance on the number of endorsed concussive symptoms is ill-advised and can lead to a high false positive rate. For example, the base rate of post-concussion symptoms among normal people is quite high (Chan, 2001). Healthy individuals with no history of head injury have been found to endorse many 'post-concussive' symptoms at very high rates, including 'longer time to think' (66%), poor concentration (59%), forgetfulness (59%), fatigue (54%), and sleep disturbance (51%). In contrast, symptoms such as nausea and/or vomiting (13%), double vision (13%), and noise sensitivity (2%) were not endorsed by healthy individuals.

b) The Clinical Interview for Concussion Patients

As the model for concussion assessment continues to adapt and change, the clinician is often asked to complete basic concussion screenings to comprehensive neuropsychological assessments. Regardless of assessment, a clinical interview is an integral part of concussion assessment and there are certain factors that need to be addressed during a clinical interview. The content of a clinical interview should be guided by empirical evidence in that it should include an evaluation of recognized neurotrauma indicators, medical history (e.g., has the person sustained previous concussions?), and symptom presentation to allow for evaluation of extraneous factors (e.g., anxiety, whiplash, etc.).

Specifically, clinicians completing clinical interviews with individuals who have sustained a documented or suspected concussion should evaluate the following:

1. Medical/Psychiatric and Psychological History:

- a. Is there a previous history of chronic medical conditions (diabetes, fibromyalgia, etc.)
- b. Previous history of neurological disorders (multiple sclerosis, stroke, etc.)?
- c. Previous history of seizures? If so, what type and when was the last one he/she had? Is the patient on any medications?
- d. Previous history of migraines or headaches? If yes, how frequently and what treatment were they receiving?
- e. Previous history of syncopal episodes or loss of consciousness?
- f. Previous history of suspected/documentated concussions? If yes, then ascertain the neurotrauma indicators and information pertaining to each (e.g., how and when it occurred, symptom course afterwards)

- g. Previous history of any mental health concerns (e.g., depression, anxiety, substance dependence, eating disorders).

2. *Current occupational/academic/recreational status and history:*

- a. Is/was there a history of diagnosed learning disability, ADHD, intellectual disability, or other challenges in school? If there was a diagnosis, who made the diagnosis?
- b. Educational level and general academic history (e.g., strong student, failed several classes, etc.)
- c. Occupational status and daily work demands (e.g., is it predominantly computer work, standing for long periods, typical work hours)
- d. Recreational status (e.g., how many sports or activities is the patient involved in? Intensity/frequency)
- e. Social status (e.g., relationship status, social support system, family involvement)
- f. Recent stressors prior to concussion and currently (e.g., dissolution of marriage, financial strain, children, bereavement).

3. *Accident details/Neurotrauma Indicators:*

- a. Type of injury and mechanisms involved (e.g., motor vehicle accident and if so, was the patient in a vehicle or a pedestrian? Damage to vehicle? Blunt force trauma, assault, slip and fall, sports injury, etc.)
- b. Was there a loss of consciousness or a decline in conscious awareness? Is this based on the patient's report or witnesses?
- c. What is the extent of retrograde and anterograde amnesia? Ensure that you ascertain what the patient recalls on their own, not what they have been told by others.
- d. What forces were involved (e.g., acceleration-deceleration forces, rotational forces, direct striking of the head, etc.)
- e. Acute hospitalization and concomitant diagnoses (e.g., whiplash, facial fractures).

4. *Symptomatology and Course:*

- a. Immediate symptomatology (within the first 24-48 hours) – evaluate physical/sensory, cognitive, and affective symptoms
- b. Symptom progression: what symptoms have improved, stayed the same, worsened? Were there symptoms that had a delayed onset? If so, when did they start?
- c. Current symptoms (physical/sensory, cognitive, affective) including severity, variability, and recognizable patterns (e.g., symptom free at home but not at work, etc.).
- d. Symptom exacerbation: do the symptoms worsen with exertion (cognitive and/or physical)? Are there any consistent triggers (e.g., computer work, physical movement)?

5. Current medications and other treatment/assessments:

- a. What, if any, medications were the patient taking at the time of the injury event?
- b. What medications is he/she currently taking?
- c. What other treatment providers are involved? What information has the patient been told by other providers regarding his/her symptoms and/or diagnosis and if applicable, concussion management?

c) Post-Injury Computerized Neurocognitive Testing/Screening

According to the Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport Held in Zurich (McCrory, et al., 2013):

“Brief computerized cognitive evaluation tools are the mainstay of these (concussion) assessments worldwide, given the logistical limitation in accessing trained neuropsychologists; however, it should be noted that these are not substitutes for formal neuropsychological assessment. At present, there is insufficient evidence to recommend the widespread routine use of baseline neuropsychological testing.”

Popular computerized neurocognitive tests include: ANAM (www.vistalifesciences.com), Axon Sports (www.axonsports.com), and ImPACT (www.impacttest.com). The use of computerized tests has increased due to the following advantages over traditional pencil and paper neuropsychological tests, including: rapid scoring, ease of administration (less time and labour-intensive), increased test-retest reliability, and greater accessibility. There are, however, limitations to computerized testing because these measures do not test all the cognitive domains that are implicated in concussion. Other limitations include:

- Questions regarding test reliability
- Validity, sensitivity, and specificity
- Required user training and qualifications
- Hardware and software issues inherent to computerized testing
- User costs
- Inflexibility
- Smaller normative data bases
- Increased likelihood of misuse (e.g., poor control of environment, interpretation by under-qualified individuals).

d) Neuropsychological Assessment

Neuropsychological assessment has been shown to be sensitive to deficits less than 24 hours post-injury and may detect persisting deficits even after the individual appears symptom-free. According to the Consensus Statement on Concussion in Sport: The 4th International Conference on Concussion in Sport Held in Zurich (McCrory, P., et al., 2013):

“The application of neuropsychological (NP) testing in concussion has been shown to be of clinical value and contributes significant information in concussion evaluation. Although cognitive recovery largely overlaps with the time course of symptom recovery in most cases, it has been demonstrated that cognitive recovery may occasionally precede or more commonly follow clinical symptom resolution, suggesting that the assessment of cognitive function should be an important component in the overall assessment of concussion and, in particular, any return-to-play (RTP) protocol. It must be emphasised, however, that NP assessment should not be the sole basis of management decisions. Rather, it should be seen as an aid to the clinical decision-making process in conjunction with a range of assessments of different clinical domains and investigational results.

It is recommended that all athletes should have a clinical neurological assessment (including assessment of their cognitive function) as part of their overall management. This will normally be performed by the treating physician often in conjunction with computerised neuropsychological screening tools.

Formal NP testing is not required for all athletes; however, when this is considered necessary, it should ideally be performed by a trained neuropsychologist. Although neuropsychologists are in the best position to interpret NP tests by virtue of their background and training, the ultimate RTP decision should remain a medical one in which a multidisciplinary approach, when possible, has been taken. In the absence of NP and other (eg, formal balance assessment) testing, a more conservative RTP approach may be appropriate.

NP testing may be used to assist RTP decisions and is typically performed when an athlete is clinically asymptomatic; however, NP assessment may add important information in the early stages following injury. There may be particular situations where testing is performed early to assist in determining aspects of management, for example, return to school in a paediatric athlete. This will normally be best determined in consultation with a trained neuropsychologist.

Baseline NP testing was considered by the panel and was not felt to be required as a mandatory aspect of every assessment; however, it may be helpful to add useful information to the overall interpretation of these tests. It also provides an additional educative opportunity for the physician to discuss the significance of this injury with the athlete. At present, there is insufficient evidence to recommend the widespread routine use of baseline neuropsychological testing.”

Studies have found no significant differences in sensitivity 14 days post-concussion between pencil and paper tests, computer-based neurocognitive testing, and the SAC sideline assessment tool. Neuropsychological assessment, however, is still considered the gold standard of concussion evaluation and remains vital to determining strengths and weaknesses that may require treatment or rehabilitation.

e) Symptom Validity for Concussion Patients

Measurement of symptom validity is a standard of practice for neuropsychological assessment (Heilbronner, et al., 2009). A corollary is that measurement of performance validity should be a standard of practice in the assessment of mild traumatic brain injury. A recent text (Carone & Bush, 2013) reviews a comprehensive range of issues related to assessment of symptom validity in mild traumatic brain injury.

Ziegler and Boone (2013) make a series of recommendations for the evidence-based assessment of symptom validity and noncredible test performance:

- Evaluation context (e.g., personal injury, forensic, capacity) should be considered when selecting measures for the test battery because context influences how the subject may feign or exaggerate symptoms.
- Symptom validity tests (SVTs) should be selected that are most appropriate for the subject's claimed condition.
- Subjects should be encouraged to give maximum effort and not warned about the use of symptom validity tests.
- Several independent neurocognitive symptom validity tests (SVTs) should be interspersed throughout the test battery.
- Both forced-choice and non-forced-choice SVTs should be used.
- Both free-standing and embedded SVTs should be used.
- SVTs that are not highly intercorrelated should be used.
- Failed SVTs are more informative than passed SVTs.
- "Passed SVTs do not 'cancel out' failed SVTs" (p. 27). Regardless of the number of passed SVTs, failure of three SVTs is almost 100% predictive of noncredible performance.
- "Interpretation of SVT data should consider whether the test taker is in a group at high risk for failure despite performance to true capability (e.g., low intelligence, dementia, etc.)" (p.27).
- "Individuals who demonstrate invalid test performance on cognitive-based assessments still may obtain scores on some standard neurocognitive tasks in the low-average and higher ranges, and when this occurs, these performances may be interpreted as representing minimum ability level" (p. 27).
- "In evaluating validity of self-reported complaints (e.g., cognitive, psychological, somatic), it is best to use psychometric instruments with built-in validity indicators that measure a range of response styles (e.g., MMPI-2-RF)" (p. 27).
- "Validity of cognitive and psychiatric data should be assessed and interpreted separately" (p. 27).
- "Substantial inconsistencies between test data and 'real-world' functionality, and between self-report and historical records, should be considered in documentation of symptom invalidity" (p. 27).
- "When performance on a claimed condition (e.g., mild TBI) is largely discrepant from what is demonstrated in the research, credibility of symptom report and test data should be carefully considered" (p. 27).
- Examiners should stay up-to-date with current research on SVTs.

- Examiners should stay up-to-date with the literature related to various medical and psychiatric conditions.

Diagnostic criteria for malingered neurocognitive dysfunction (MND) have been proposed (e.g., Slick, Sherman, & Iverson, 1999). Four criterion domains are specified. The first is the presence of a substantial external incentive. The second domain includes poor performance on well-validated tests designed to measure exaggeration, discrepancy between test data and known patterns of brain functioning, discrepancy between test data and observed behavior, discrepancy between test data and collateral report, and discrepancy between test data and documented background history. These have been organized into a decision tree by Larrabee, Greiffenstein, Greve, and Bianchini (2007). Slick et al. specify the diagnostic criteria for three categories of MND: definite, probable, and possible. These differ based on the magnitude of test findings, and the number of discrepancies from test data and self-report data. These authors summarize research related to the validity of the Slick et al. criteria, and propose modifications based on their review of the research. The first modification is to allow failure of multiple SVTs alone to define probable MND. Use of only one, rather than two, test findings that are discrepant with documented history is the second modification. Third, the use of a conservative decision strategy with subjective criteria is recommended. That is, additional discrepancies involving subjective criteria should be required before reaching a diagnosis of probably MND. Last, Larrabee et al. recommend giving self-report discrepancies that same weight as test-based discrepancies in the diagnosis of MND.

Section III: Differential Diagnosis and Comorbidities

a) Concussion and Co-Morbid Medical/Physical Conditions

When evaluating concussive effects, it is of critical importance to consider the individual's comorbid and/or pre-existing medical and neurological conditions as many of these conditions present with similar symptomatology. Although it is not within the scope of these Guidelines to provide an exhaustive overview of all potential confounding medical/neurological conditions, there are several conditions that a clinician should pay particular attention to due to the high comorbidity rates with concussion.

Whiplash – Due to the biomechanics involved in concussive injuries, particularly those suffered during motor vehicle accidents (e.g., acceleration-deceleration and rotational forces), whiplash and concussion often co-occur. As such, appreciation of whiplash sequelae is essential in concussion evaluation.

Similar to concussion, whiplash has been shown to lead to acute neuropsychological changes, including tests of attention and concentration (Ettlin, et al., 1992). Neurophysiological studies utilizing somatosensory evoked potentials (SEPs) have shown that both whiplash and concussion alter processing of the middle-latency SEP component in the acute post-traumatic period (both at 48 hours and at 3 months post) when compared to normal controls. The SEPs did not differ from whiplash and

concussion patients, suggesting that the overlapping clinical symptomatology post-whiplash and concussion may reflect a similar underlying mechanism (Zumsteg, Wennberg, Gutling, & Hess, 2006).

Whiplash also leads to chronic subjective symptom complaints that are similar to concussion. In a large Scandinavian study (Styrke, Sojka, Bjornstig, & Stalnacke, 2014), the most common symptoms five years after whiplash injury were fatigue (41%), poor memory (39%), headache (37%), inability to sustain previous workload (44%), and fatigue at work (43%).

Neuropsychological studies have shown individuals with chronic whiplash injuries to demonstrate reduced information processing speed, (Radanov, Hitlinger, Di Stefano, & Valach, 1992), attention (Gimse, Bjorgen, Tjell, Tyssedal, & Bo, 1997), divided attention (Di Stefano & Radanov, 1995; Radanov, Dvorak, & Valach, 1992), and learning and memory (Gimse, et al., 1997). A meta-analysis conducted by Kessels and colleagues (Kessels, Aleman, Verhagen, & Van Luitelaar, 2000) compiled 22 neuropsychological studies and suggested a consistent overall pattern of cognitive dysfunction after chronic whiplash injury, both compared to healthy and to asymptomatic controls. Specifically, they found deficits in working memory, attention, immediate and delayed recall, visuomotor tracking, and cognitive flexibility. Significant improvements were noted in many of these measures by 6 months post-injury.

Overall, the evaluation of concomitant whiplash and/or neck injuries is of paramount importance when evaluating concussion due to not only its high comorbidity but the fact that it can be a significant cause of post-traumatic headaches (PTHA), and subsequently, neurocognitive complaints and symptoms. Whiplash and concussion share many symptoms as well, which complicates diagnostic reliability given that the severity and number of symptoms have been suggested to have such large diagnostic value in both conditions.

Chronic Pain (CP) – Post-concussive symptoms are often difficult to evaluate due to the fact that many of the symptoms are present in other conditions, including chronic pain (CP). Very high comorbidities have been found for MTBI and CP, with one study finding that 95% of MTBI clients who presented for treatment had a comorbid CP condition (Uomoto & Esselman, 1993). For example, ‘post-concussive-like’ symptoms such as disturbed sleep, fatigue, and irritability are also reported by the majority of chronic pain patients (Iverson & McCracken, 1997). On self-report questionnaires evaluating ‘post-concussive’ symptoms (e.g., Rivermead Post-Concussion Questionnaire), no group differences have been found between MTBI and CP in terms of overall scores (Smith-Seemiller, Fow, Kant, & Franzen, 2003). Similarly, CP samples frequently complain of forgetfulness, difficulties finishing tasks, and attention difficulties; 54% of CP patients were found to report at least one cognitive symptom (McCracken & Iverson, 2001). Overall, the culmination of research suggests that most people with CP would be identified as suffering from post-concussion syndrome based on self-report symptoms alone. Researches have warned that self-reports of cognitive or emotional

dysfunction “cannot be reliably used to differentiate patients with CP from people with MTBI (p. 205).”

There is also a large amount of research that has shown chronic pain patients to demonstrate neuropsychological deficits similar to those seen in individuals with concussion. For example, individuals with CP have been found to perform more poorly on measures of sustained attention, psychomotor speed, information processing, and working memory when compared to healthy controls (Sjogren, Christrup, Petersen, & Hojsted, 2005). A large review article completed by Hart, Martelli, & Zasler (2000) noted that numerous studies have demonstrated impairment in patients with CP, particularly on measures assessing attentional capacity, processing speed, and psychomotor speed.

Overall, there is a substantial amount of literature that has shown that individuals with CP experience similar symptomatology to those with concussion or MTBI. These similarities are noted with both subjective (self-report) and objective (neurocognitive testing) data, suggesting that in chronic pain must be evaluated and considered within the context of concussion evaluation.

Headaches/Migraines - Headaches are a significant and common consequence of concussion. In fact, headache is among the most frequently reported symptoms following injury to the head, brain, or neck with incidence rates reported as high as 80% to 100% (McAllister & Arciniegas, 2002). There are many different types of headaches, including tension-type, migraines, cluster headaches, and posttraumatic. Due to their prevalence following concussion, as well as the potential impact on cognitive functioning, clinicians need to sufficiently evaluate both pre-concussion and post-concussive headache history.

Post-traumatic headaches (PTHA) can result from a variety of neurological events (e.g., stroke, anoxia, traumatic brain injury), including concussion. The very complexity and comorbidity of neurological conditions that can lead to the development of PTHA make the identification of a single neuropsychological profile essentially impossible. As with studies on the neuropsychology of migraine, evaluation of PTHA neurocognitive deficits are far from consistent. Some studies have shown evidence of impaired memory and concentration (Fioravanti et al., 1983), whereas others have found no relationship between PTHA and neuropsychological deficits in patients with mild head injury (Tsushima & Newbill, 1996; Tsushima & Tsushima, 1993). Interestingly, studies that have found neuropsychological differences amongst individuals with PTHA have often included non-head injury populations (e.g., whiplash).

Research pertaining to the neuropsychological effects of *migraine* has been by far the most studied of headache types but is not conclusive. In a comprehensive review of the literature, O'Bryant and colleagues (O'Bryant, Marcus, Rains, & Penzien, 2006), they noted that most of the studies documenting significant neuropsychological deficits have been drawn from populations seeking medical treatment, such as specialty clinics, where ones failing to find significant results commonly evaluated migraine patients who

were not seeking treatment from specialty clinics. One of the most compelling studies from researchers in New Zealand followed a cohort of headache sufferers from age 3 until 26. Mild impairments were noted in verbal intelligence and verbal reasoning in migraine sufferers relative to those with tension-type headache (Waldie, Hausmann, Milne, & Poulton, 2000). Despite the ongoing inconsistencies in neuropsychological findings, there have been several important findings. Migraine patients with aura appear to experience more cognitive deficits than those without auras, including problems with visuospatial skills, visual memory, motor tests, information processing speed, sustained attention, and executive functioning (Ardila & Sanchez, 1988; Le Pira, et al., 2000; Mongini, Keller, Deregibus, Barbalonga, & Mongini, 2003; Mulder, Linssen, Passchier, Orlebeke, & de Geus, 1999). It is important to note that these studies have focused on persistent interictal cognitive deficits in migraine patients and were not only observed in the context of an acute migraine pain. In contrast, a recent meta-analysis of the literature (Suhr & Seng, 2012) showed only 'weak' evidence for deficits in processing speed, attention, verbal memory, verbal skills, working memory, sustained attention, and inhibition in migraine sufferers relative to healthy controls. Based on the review of the research, they concluded that cognitive dysfunction is seen only in a subset of migraine sufferers.

Migraine history and presentation is also important within the context of concussion due to their reported relationship. Migraine and concussion may be associated through a number of mechanisms. Concussion may trigger migraine, migraine may be misdiagnosed by concussion, or they may co-occur either through similar mechanisms or by chance (Solomon, 1998). Mild head trauma can trigger both typical migraine and acute confusional migraine; distinguishing the two may be difficult although some research has indicated that those with concussion are less likely to have agitation (Neinstein & Milgrom, 2000; Solomon, 1998). Misdiagnosing migraine as concussion is also possible as the International Headache Society criteria for migraine (2004) are so broad that many post-traumatic headaches would qualify as migraine (Margulies, 2000). An exploratory analysis using a large cross-sectional survey was conducted through the Canadian Community Health Survey of individuals ages 12 to 24 (Gordon, Dooley, & Wood, 2006). The results indicated that risk factors for concussion were younger age, male gender, and reported migraine headaches. This study was correlational in nature, however, so causality could not be determined between migraine and concussion (concussion causing migraine or vice versa). However, it revealed a strong co-occurrence between migraine and concussion on a large population-based study.

While the literature devoted to understanding the neurocognitive deficits of migraine sufferers is growing, much less research has been conducted in terms of the neuropsychology of *tension-type headache*. In fact, one of the only studies conducted was by Waldie and associates who evaluated adults who had been diagnosed with tension-type headache and *retroactively* evaluated their academic records. Those with a history of childhood headache who were later diagnosed with tension-type headache and those whose headache did not persist consistently performed more poorly on verbal IQ measures (Waldie, et al., 2000). Overall, the paucity of research precludes the drawing of conclusions. It remains possible that tension-type headache patients do no

exhibit any consistent neuropsychological deficits or they exhibit a similar pattern of deficits found in chronic pain patients.

Finally, the research examining neuropsychological effects of individuals with *cluster headache* has been equivocal. Some studies (Dresler et al., 2012) have shown individuals with chronic and active episodic cluster headaches experience poorer performance on measures of prefrontal functioning (e.g., Trail Making Test, Stroop tasks, inhibition). Other studies have shown little to no functional differences between cluster headache sufferers and healthy controls across measures of intelligence or executive functioning (Jorge, Leston, Arndt, & Robinson, 1999; Sinforiani, Farina, Mancuso, Manzoni, Bono, & Mazzucchi, 1987; Torkamani et al., 2015). There has been some limited support for subtle and relative differences in working memory in individuals with episodic and chronic cluster headaches but the functional implications of these difference were deemed minimal as the individuals' scores remained in the average range (Torkamani et al., 2015). Overall, a review of the literature shows inconclusive findings stemming from various studies that were limited by small sample sizes and selection biases in terms of how samples were obtained.

Based on the available research findings, it is clear that a clinician evaluating concussion should evaluate whether a patient has a history of headaches, migraines (with or without aura), and if they have been formally diagnosed with migraine and/or posttraumatic headache since the impact since they can impact one's neurocognitive functioning and may explain some cognitive inefficiencies attributed to 'post-concussive' etiology.

Sleep Deprivation/Chronic Sleep Disruption - Sleep disturbance is a common complaint following concussion. Chronic partial sleep deprivation (defined as sleep restriction to less than 7 hours per 24-hour period) demonstrates profound neurocognitive deficits that accumulate over time, even in the face of subjective adaptation to the sensation of sleepiness (Durmer & Dinges, 2005). Sleep deprivation associated with disease-related sleep fragmentation (i.e., sleep apnea, restless legs syndrome) also results in neurocognitive performance decrements similar to those seen in sleep restriction studies. It can also lead to increased negative mood states, fatigue, and confusion (Durmer & Dinges, 2005). Meta-analytic studies have also shown that overall sleep deprivation negatively affects mood more than cognitive or motor performance, with chronic partial sleep deprivation (consistent with that often seen in concussion, chronic pain, and other conditions) having a more profound effect on functioning than either long-term or short-term sleep deprivation (Pilcher & Huffcutt, 1996). In terms of neuropsychological functioning, however, sleep loss reliably produces reductions in speed of processing and attention. Higher order cognitive functions are affected to a lesser extent, and there is sparing on tasks of crystallized abilities (Waters & Bucks, 2011). This is of particular interest to neuropsychologists evaluating post-concussive effects since the two populations appear to present with very similar neurocognitive profiles.

Neuropsychologists evaluating concussion should ascertain information pertaining to a patient's sleep quality, both prior to and since the concussive injury. Conditions such as sleep apnea, restless leg, anxiety, and chronic sleep deprivation (less than 7 hours of sleep per night) can hinder an individual's functioning and may mimic the effects of concussion. Moreover, post-concussive sleep disruption is a likely contributor to cognitive and affective complaints and needs to be considered a significant factor in the introduction and exacerbation of post-concussive symptoms.

b) Concussion and Co-Morbid Psychological Conditions

When evaluating concussive effects, it is of critical importance to consider the individual's comorbid and/or pre-existing psychological conditions as many of these conditions present with similar symptomatology. Although it is not within the scope of these Guidelines to provide an exhaustive overview of all potential confounding psychological conditions, there are several conditions that a clinician should pay particular attention to due to the high comorbidity rates with concussion.

As aforementioned, symptoms commonly used in post-concussive symptom checklists share many similar diagnostic symptoms used in self-report measures for psychological conditions (e.g., Beck Depression Inventory (BDI) – difficulties concentrating, fatigue, low energy, irritability, agitation; Beck Anxiety Inventory (BAI) – numbness/tingling, etc.). Qualitatively, concussion shares many subjective complaints with psychological disorders, including difficulties sleeping, aversion to chaotic/social environments, headache, general malaise, psychomotor slowing, difficulties performing work tasks, etc. Therefore, timing, severity and history of symptom presentation is required over and above isolated symptom endorsement for differential diagnosis. When evaluating recovery from concussion, early studies showed that only level of psychological distress (not even severity of injury) was strongly associated with symptomatology (Karzmark, Hall & Englander, 1995). Of various factors, Ponsford and colleagues (2000) showed individuals with persistent concussive symptoms were more likely to have a history of previous head injury, psychiatric problems, and to have been injured in a MVA (which can be considered a traumatic event). These findings all suggest that psychogenic factors play a large role in the development, perpetuation, and recovery of post-concussive symptoms. Given their ability to mimic 'post-concussive' symptomatology, a comprehensive concussion assessment must include an evaluation of the individual's psychological history as well as current psychological status.

Depression - In cases where individuals do not recover within the expected timeline, it is very common for them to be unable to return to premorbid activities, including work, recreational activities, and activities of daily living. With these limitations, they are at a greater risk of developing increasing psychological distress and depressive symptoms. In individuals who are exhibiting chronic post-concussive symptoms, an evaluation of depression is of paramount importance due to the overlap in symptomatology and the role depression plays in the exacerbation and maintenance of disability. Depression and concussion share many symptoms, which requires a careful examination of the respective roles of each in a patient's presentation. Depression can often mimic post-concussive effects, which leads to a risk of misdiagnosis and ill-informed treatment. For

example, using the Postconcussion Symptom Inventory, researchers have found that the rate of symptom endorsement ranged from 31.2% to 85.6% in a depressed sample; 9 out of 10 patients with depression met liberal criteria for post-concussive syndrome; 5 out of 10 met conservative criteria (Iverson, 2006). In addition, depression after mild TBI is associated with self-reported increases in the number and perceived severity of symptoms, including headache, dizziness, and blurred vision (Silver, McAllister, & Arciniegas, 2009).

The neuropsychological profile of depression also shares similar features to concussion. Studies have shown individuals with major depression show deficits consistent with global-diffuse impairment of brain functions, with particular involvement of frontal lobes (including Stroop tasks, cognitive flexibility)(Castaneda, et al., 2008; Veiel, 1997). Depression has also been found to have an adverse effect on immediate verbal recall but intact retrieval and retention (Kizilbash, Vanderploeg, & Curtiss, 2002).

Overall, when evaluating concussion, especially in the post-acute stages, it is of paramount importance to evaluate an individual's mood. Depression and concussion share many symptoms and also exhibit potential similarities on cognitive testing. As such, it is very much possible that in some patients, depressive symptoms are being misdiagnosed and misattributed to concussion.

Anxiety - Similar to depression, anxiety and concussion share many symptoms (e.g., dizziness, agitation, sleep disruption, difficulties concentrating, etc.). It is also reasonable to assume that anxiety and concussion co-exist even during the acute stage due to the traumatic cause of many concussive injuries (e.g., car accidents, assaults, etc.). This is of particular importance since research has found that premorbid and concurrent anxiety increases the risk for prolonged concussion recovery (Broshek, De Marco, & Freeman, 2015). Conversely, it is possible that concussion can result in increased anxiety as well. For example, animal models of concussion suggest that it can result in anxiety and fear reactions. Given this interplay between concussion and anxiety, effective assessment and treatment of post-concussive effects must include a careful consideration of anxiety effects. Qualitatively, individuals who are anxious in nature are more likely to become hypervigilant to perceived symptoms. As they become more focused on symptoms, they are likely to experience them, consistent with self-fulfilling prophecy literature. Similarly, anxious individuals are more prone to misattribution of symptoms (e.g., attributing a headache to post-concussive effects rather than stress-related tension), which suggests why anxiety increases the risk for prolonged concussion recovery. As such, most recent reviews of the literature (Broshek et al., 2015) recommend that effective treatment to shorten the length of post-concussive symptoms include introduction of anxiety reduction techniques and cognitive-behavioural therapy for cognitive biases and misattribution. In contrast, medically-prescribed excessive rest and symptom monitoring is likely to be counter-productive and deleterious to recovery due to the frequent comorbidity of anxiety and fear (Broshek, et al., 2015)

From a neuropsychological perspective, it is also important to delineate anxiety and concussive effects. There are variable findings regarding cognitive effects of anxiety, mainly due to the heterogeneity of the population. For example, mixed anxiety disorder groups have been found to exhibit significant impairments in episodic memory and executive functioning, with different subgroups showing different neuropsychological functioning (Airaksinen, Larsson, & Forsell, 2005). Individuals with specific phobias and Generalized Anxiety Disorder have not shown any consistent neuropsychological deficits, whereas individuals with Panic Disorder and Obsessive Compulsive Disorder have shown changes. Comorbid anxiety has also been identified as a partial contributor to memory performance in depressed individuals (Kizilbash, et al., 2002).

Overall, of all of the potential comorbid psychological conditions that exist when dealing with concussion, anxiety is the most important to consider given the often traumatic/anxiety-provoking nature of concussive injuries (e.g., accidents, assaults, sports injuries), and its propensity to exaggerate concussion symptoms and prolong/delay recovery.

Post-Traumatic Stress Disorder (PTSD) - As aforementioned, concussion often occurs in the context of a traumatic event, including motor vehicle accidents, assaults, or combat. As such, it is important to evaluate the etiology of the concussive event and whether PTSD is present. One of the intriguing findings has been that concussion appears to increase the risk for PTSD. For example, Fann and colleagues (2004) reported that in a large-scale sample, patients with a history of concussion were 2.8 times likely to develop a psychiatric disorder than patients with no brain injury history. Similarly, military studies showed that 16% of troops who sustained a bodily injury indicated PTSD, whereas 44% of those with mild brain injury screened positive for PTSD (Hoge, et al., 2008).

In contrast, research has also indicated that comorbid PTSD can predict the presence of lingering concussive symptoms. In a study comparing a concussion group from a non-concussion trauma group during acute hospitalization, those with concussion performed worse on cognitive testing measures but did not differ from the trauma group in terms of subjective post-concussive symptoms reports. Concussion symptoms were also significantly related to emotional distress at the time of hospitalization (Landre, Poppe, Davis, Schmaus, & Hobbs, 2006). In a large sample study involving long-term post-deployment outcomes in soldiers with PTSD and concussion (Polusny, et al., 2011), combat-related PTSD was strongly associated with post-concussive symptoms and psychosocial outcomes at 1 year post incident. In contrast, there was little evidence of a long-term negative impact of concussion on these same outcomes after accounting for PTSD. Those with PTSD symptoms more strongly predicted post-deployment symptoms and outcome than did concussion. This indicated the likely misdiagnosis and mistreatment of post-concussive symptoms when dealing with individuals who also suffer from PTSD. PTSD has been found to hinder cognitive performance in a way that could be misconstrued as concussion. For example, PTSD and other deployment-based trauma in war veterans has been shown to compromise performance on sustained attention, verbal learning, and visual-spatial memory (Vasterling, Proctor, Amoroso,

Kane, Heeran & White, 2006). Similarly, Vasterling and colleagues also found that although soldiers with mTBI/concussion showed cognitive recovery, those with PTSD and depression showed lingering cognitive deficits on testing (Vasterling, Brailey, Proctor, Kane, Heeran, & Franz, 2012).

Overall, a thoughtful review of the overlap and interplay between PTSD and concussion has been written by Dr Richard Bryant (2011). In that review, he concludes that the non-specific symptoms associated with 'post-concussive' syndrome share many features with PTSD and other traumatic-based psychological disorders (e.g., headaches, irritability, emotional lability, noise sensitivity, concentration problems, etc.). As such, he concludes that the 'likelihood that the presumed sequelae of MTBI are actually attributed to psychological responses to the traumatic experience is becoming more apparent (pp. 259)."

c) Concussion and Pre-existing Cognitive and Learning Conditions

Pre-injury cognitive factors must be considered when looking at the recovery of functioning following concussion. While it is known that the majority of individuals who sustain this type of injury present with acute cognitive challenges, there are those who continue to report difficulties well beyond the normal expected recovery time. When examining individuals post-concussion, it is important to consider pre-injury factors, including a history of cognitive challenges such as learning disability or Attention Deficit Hyperactivity Disorder (ADHD). However, although these issues are considered to be modifying factors in the management of individuals following concussion, few studies are available that report on their effects. Studies that examine cognitive risk factors often focus on more seriously injured individuals or do not differentiate between types of injury severity. In addition, the majority of studies examining individuals with learning disability or ADHD examine children. No studies looking at adults with pre-existing learning issues were found.

Ponsford and colleagues (2000) examined individuals with persisting problems following concussion. They identified a subgroup of 20 individuals from a group of 84 concussion cases who were reporting ongoing difficulties at three months post-injury. While a number of factors (including pain and a further head injury) were found to impact performance, it was noted that those who were students generally had difficulty with their studies. Although it was not clear as to whether these difficulties pre-dated the initial injury, the study included individuals with a prior history of learning difficulties.

Attention Deficit Hyperactivity Disorder: Bonfield, Lam, Lin, & Greene (2013) retrospectively identified patients with concussion and a history of ADHD and compared their performance to that of those with concussion and no history of ADHD. They found that children with a history of concussion and ADHD had worse outcome on the KOSCHI (King's Outcome Scale for Childhood Head Injury). There was more disability, as measured by the KOSCHI, for that group of children when seen at follow-up, even when adjusting for other factors including gender, age, length of stay, mechanism of injury, and presence of other injuries. Other factors (such as older age and mechanism of injury) were also predictive of a worse outcome in both ADHD and control groups. It

was noted that children with ADHD might be more disabled than their peers initially and that it was possible that the TBI might unmask characteristics of a premorbid ADHD. In addition, it was noted that ADHD might, in fact, be serving as a “proxy” for lower socioeconomic status as it is known that there is an association between lower SES and the incidence of ADHD. Bonfield and colleagues. (2013) concluded that individuals with ADHD who sustain concussions were more likely to be moderately disabled than those sustaining brain injuries in the absence of a premorbid attentional issue. Of those patients without premorbid ADHD, 84% had recovered completely by the last follow-up (mean 7.2 weeks) whereas 25% of those with ADHD were moderately disabled (as rated by the KOSCHI) and 56% were completely recovered.

Mautner, Sussman, Axtman, Al-Farsi and Al-Adawi (2015) identified that young athletes with ADHD took on average three days longer than those without ADHD to return to baseline on neurocognitive assessment. They studied young athletes and measured their performance on ImPACT. While it was identified that individuals with ADHD scored lower on baseline neurocognitive testing, the delayed recovery in this study was posited to be concussion-related. However, the diagnosis of ADHD was self-reported by the young athlete, which reflects a limitation to this study. Furthermore, no stand-alone performance validity measures were administered as part of this study.

ADHD is both a risk factor for concussions and for poorer outcome following injury. Alosco, Fedor & Gunstad (2014) identified that ADHD was prevalent in NCAA Division 1 athletes and associated with a history of prior concussion. While this study reflects a self-report history, approximately 50% of those athletes who reported ADHD, also reported a history of at least one prior concussion.

Learning Disability: There are few studies that examine the effect of brain injury on individuals with learning disability. Studies have shown that children who have pre-injury learning problems demonstrate greater difficulties with memory functioning, attention, executive functioning, adaptive behaviour and behavioural functioning when compared to children without these difficulties (Farmer et al., 2002; Ponsford et al., 1999; Sesma et al. 2008; Woodward et al. 1990). However, these studies include children with brain injuries ranging from mild to severe. Donders and Strom (1997) identified that *moderate to severe* brain injuries in children with learning disabilities resulted in significant additional cognitive impairment. While there are studies demonstrating greater impairments in functioning in those with pre-existing challenges, it is not entirely clear as to how applicable these findings are to individuals who have sustained mild traumatic brain injury or concussions, given the nature of the participants in those studies.

Developmental Disability: Research is also limited in the area of concussion in individuals with developmental disability. What limited research is available indicates that there are greater declines in such cognitive areas as memory, verbal abilities, attention and executive functioning (Johnstone et al. 1995) in children following mild TBI and, in adults, poorer emotional adjustment (Wood and Rutterford, 2006).

Overall, when examining an individual with mild traumatic brain injury/concussion, the use of a biopsychosocial model is paramount and will result in a more integrated and thorough understanding of the effects of brain injury. It is important to evaluate premorbid and post-injury variables that can impact on outcome. At present, the research in the area of concussion in cognitively vulnerable individuals is limited but provides preliminary evidence suggesting that vulnerable individuals demonstrate greater challenges following concussion than those individuals who do not demonstrate cognitive difficulties prior to their injury. In general, it appears, however, that learning and cognitive difficulties that pre-date the injury are associated with poorer outcomes. Further research is, however, needed in this area.

PART 3: SPECIAL POPULATIONS AND CONSIDERATIONS

a) Concussion in Geriatric Patients (defined as individuals over age 65)

Epidemiology: Thompson, McCormick, and Kagan (2006) report that the age-adjusted rate of hospitalization for adults over 65 with TBI (155.9 per 100,000) (Coronado, Thomas, Sattin et al., 2005) is more than twice that found in the general population (60.6 per 100,000) (Centers for Disease Control, 2010). Cheng, Lin, Lee et al. (2014) controlled for a number of relevant variables and found that even concussion or mild head injury is an independent and clinically significant risk factor for death in the elderly. These authors concluded that increased emphasis on prevention is worthwhile. The most frequent causes for TBI in seniors are falls (51%) followed by motor-vehicle-related injury (9%) (Langlois, Rutland-Brown, & Thomas, 2004). Further, the highest rate of suicide is among persons over 65 (CDC, 2002), and attempted suicide is the third leading cause of injury in this age group (Binder, 2002). However, the mechanism of traumatic brain injury is unknown in a sizable percentage (21%) (Langlois, et al., 2004).

Assessment: Consistent with the Ontario Neurotrauma Foundation (ONF) Guideline (2013) recommendations, standardized and comprehensive assessment should be conducted as soon as possible following injury. This should include cognitive screening measures, as well as a thorough history, assessment of post-concussion symptoms and mental health. Self-reported history should be supplemented by collateral informants and a review of health records (Lezak, Howieson, Bigler, & Tranel, 2012). Comorbidities and differential diagnoses should be considered in the patient's presentation. Some of the most relevant conditions entering into the differential diagnosis are depression and anxiety, dementia, physical illness and injury, pain, insomnia, and polypharmacy (Raskin & Mateer, 2000).

Regarding test administration, additional time and care to educate the patient about the nature and purpose of testing should be taken. Standardized psychometric measures that are validated for persons over 65 should be used (Lezak et al., 2012). Adjustments should be made for fatigue, sensory and motor limitations, and attitudes about psychometric testing. Consideration should also be given to the use of untimed tests.

Because access to psychologists is limited in many contexts, psychologists may need to organize collaborative networks with other health professionals with the aim of ensuring that early comprehensive assessment integrates appropriate measures and interpretation of findings. It can be helpful to train and supervise those who have the earliest contact with the patient to administer screening measures, and refer for more comprehensive neuropsychological assessment when warranted. Collaboration with primary care providers and allied health professionals is recommended to facilitate early referral and to integrate findings into ongoing care. Because recovery can be prolonged and incomplete, follow-up assessment is recommended when initial neuropsychological assessment reveals neurocognitive deficits.

Persistent Symptoms: Because of the interaction of concussion and co-occurring medical and neurological factors, follow-up assessment should be conducted to determine whether a more detailed assessment is warranted due to persistent symptoms (Raskin & Mateer, 2000).

Finally, research in the area of the older adult is also limited. While it is known that older age is associated with poorer outcome, there is limited research into the effects of mild brain injury in individuals with dementia. Thompson, McCormick & Kagan (2006) note that while older age is associated with poorer outcome, selection bias is an issue in studies that examine this area.

b) Concussion in Pediatric Patients

General Findings: Concussions account for 80 to 90% of all treated cases of pediatric brain injury (Kirkwood et al., 2008). The development of evidence-based approaches to assist with the assessment and management of these injuries is imperative to ensure that children and adolescents are appropriately treated. Similar to what is described in the adult literature, children and adolescents experience a constellation of somatic, cognitive and behavioural/emotional difficulties that include headache, dizziness, fatigue, sensory sensitivity, poor attention and memory, and increased levels of anxiety. Recovery is reported to be quite variable, with some studies demonstrating that, between two to three months post-injury, cognitive and achievement difficulties are not recognized (Carroll et al, 2004; Satz, 2001; Satz et al., 1997), and other studies indicating that a sizeable minority report more persistent problems (Yeates & Taylor, 2005). Yeates and Taylor (2005) identified that children with concussion were more likely than children with orthopaedic injuries to demonstrate recoveries that had high levels of acute symptoms that either resolved or persisted over time. Both injury and non-injury factors are likely involved in predicting outcome following injury and include severity, age at injury, premorbid “brain reserve,” genetic vulnerability, premorbid learning and behavioural functioning, history of prior concussions, family expectations and functioning, comorbid post-injury stress or pain, motivational factors, litigation, and post-injury management (Kirkwood et al., 2008).

Neuropsychologists are well positioned to assist in the clinical management of children with these injuries. Care must be individualized to the child or adolescent, his/her

developmental level, and the circumstances of the child. However, children must be understood in a different manner than adults and this requires the implementation of assessment measures that are appropriate from both an age and developmental perspective (Gioia, Schneider, Vaughan & Isquith, 2009). Another critical difference between assessment of children and adults is the role that parents and other adults play in children's recovery and in the assessment process. Parents and teachers can provide a wealth of information about behaviour and functional status in home and school environments that can supplement standardized assessment data.

Karlin (2011) noted that concussions in the pediatric population have the potential to impact on a child's neurodevelopment. While research in this area is growing, it remains sparse particularly when compared to the research using adults. Using practice guidelines and principles that have been developed on adult populations is not recommended. Karlin (2011) identified that normalization to baseline following a sports-related concussion is slightly longer (10 to 14 days) in high school athletes when compared to college-level and adult athletes and that neurocognitive deficits may persist long after self-reported symptoms have normalized. Again, it is noted that neuropsychological assessment should be developmentally sensitive and take into account the expected improvements in performance over time.

Cognitive complaints are frequently reported following pediatric concussion. Assessment of these complaints by neuropsychologists assists in identifying the nature of those deficits and with the development of appropriate treatment plans. Assessing children and adolescents, however, requires specialist approaches and an understanding of neural development.

Cognitive Assessment: Similar to adults, cognitive assessments occur both on the sideline, as well as after the typically expected timeline for recovery has passed and ongoing cognitive complaints persist. Psychologists are not typically involved in sideline assessments in children and adolescents who have sustained a concussion as the result of a sporting injury. Adult measures designed to assess symptoms of concussion are not appropriate for the pediatric population and assessment measures specifically designed for this population must be utilized. These include the SCAT3, the pediatric version of ImPACT, and the Standardized Assessment of Concussion, along with other measures. These measures have been utilized within this population and can provide information about functioning and recovery. They can assist in documenting the course of recovery in the acute phase following injury. Comprehensive neuropsychological evaluation is not typically warranted in the acute stage; however, incorporating cognitive assessment at this stage can assist in providing a good description of the injury and assist with diagnosis. In addition, when exploring symptoms at this stage, it is important to gather information from both the child/adolescent and parents. It is also important to review information (e.g., school records) about pre-injury status and functioning.

Issues pertaining to the assessment of children and adolescents following concussion include the development and use of measures appropriate to that population and the timing of abbreviated and more comprehensive neuropsychological assessments.

Furthermore, as baseline and post-concussion computerized testing becomes more widespread, a need for standards for proper administration and interpretation of those findings is important. Moser, Schatz, & Lichtenstein (2015) summarized research in this area and identified that inadequately trained or untrained individuals often administer or direct sports concussion management programs. They may not fully understand standardized assessment procedures or factors that might affect testing or test results. While programs designed for professional athletes are typically managed by neuropsychologists, the same is not true for those programs developed for schools.

Psychometric Issues: One issue that must be addressed relates to the psychometric properties of the assessment measures utilized. There is limited research available that explores the psychometric properties of baseline measures of cognitive functioning in the child and adolescent populations. In addition, while test-retest statistics are often presented for short retest intervals (e.g., 1 or 2 weeks), in reality this is shorter than what occurs between pre-season and post-injury assessment (Kirkwood, Randolph & Yeates, 2009). Gioia et al. (2009) examined the psychometric properties of a number of clinical measures of post-concussion symptoms (Acute Concussion Evaluation, Post Concussion Symptom Checklist, Post Concussion Symptom Inventory, Graded Symptom Scale; Health and Behaviour Inventory, and Rivermead Post Concussion Symptom Questionnaire) and found that there was sparse data for those children in the youngest age group (aged 5 to 12) and no data for children under the age of 8. There was greater information for adolescents and young adults (aged 13 to 22). The majority of the studies reviewed indicated evidence for concurrent validity of the measures but few reported evidence for reliability and other forms of validity. In addition, few studies explored the age-specificity of the scales or reliable change statistics.

Valovich McLeod, Barr, McCrea, and Guskiewicz (2006) examined the test-retest reliability of the Standardized Assessment of Concussion (SAC) in a group of young athletes between the ages of 9 and 14. While the test-retest reliability was moderate, construct validity was weak. It was noted that there was a weak relationship between the SAC and other neuropsychological measures, suggesting that the various tools were not measuring the same thing. It was recommended that the SAC be included along with a more comprehensive battery when assessing adolescents with concussion. Other research has identified that the psychometric properties of the Acute Concussion Evaluation (ACE), a structured clinical interview, are strong for an initial assessment tool (Gioia, Collins & Isquith, 2008).

McKay, Brooks, Mrazik, Jubinville & Emery (2014) evaluated the use of ImPACT in elite athletes, aged 13 to 17. Their research demonstrated that older players had better visual-motor processing speed and impulse control than younger players and that girls had more symptoms than boys. Reference values for this population were provided. Newman, Reesman, Vaughan, & Gioia (2013) also evaluated the validity of the pediatric ImPACT. It was noted that performance on the Response Speed Composite of this measure was more strongly associated with other measures of cognitive processing speed than measures of working memory and learning and memory. Evidence for convergent and discriminant validity was identified.

Echemendia and colleagues (2013) noted that evaluation and management of concussion in children posed unique challenges. They identified the lack of published studies examining postconcussive impairments in children below high school age and noted that this was largely due to the fact that there were fewer age appropriate measures available for this population. They examined three computerized measures: CogSport, Pediatric ImPACT, and CNS Vital Signs. It was noted that CogSport demonstrated clear evidence for developmental changes and that the Pediatric ImPACT was developmentally appropriate for those aged 5 to 12. In addition, CNS Vital Signs had norms available from the age of 8. They concluded, however, that computerized batteries lacked the necessary evidence regarding clinical utility. They further identified that, although the literature in this area is growing, there is limited research identifying reliable metric changes for individuals and normative data is not available for a number of measures.

Yeates et al. (2012) looked at reliable change in postconcussive symptoms in 8 to 15-year-old children. They identified that children with mild TBI were more likely than those with orthopaedic injuries to show reliable increases in cognitive and somatic symptoms. From a cognitive perspective, group differences were noted to persist up to 12 months. These reliable changes were more common for those with complicated brain injury or with a loss of consciousness.

It is clear that further research needs to be conducted in the reliability of measures that are employed and how useful they are to assist with decision-making processes in the pediatric population. In addition, Yeates (2010) has identified that long-term studies and assessments are needed to identify whether concussion results in persisting symptoms in children. Kirkwood et al. (2009) note, however, that regardless of the concern with respect to the utility of neuropsychological assessment in the baseline and acute period, individuals should be referred for neuropsychological consultation if they fail to recover within the usual time frame, if they have a history of multiple concussions, or if their symptoms are severe, persistent, or functionally disruptive.

Timing of Assessments: In the post-acute state when the child is still actively recovering, comprehensive neuropsychological evaluation will not typically be indicated. An abbreviated assessment, however, could be conducted. Kirkwood et al. (2008) reported that assessments can be conducted two weeks post-injury, as many adolescents demonstrate complete cognitive recovery following a concussion by this time. They noted that research with younger children was less clear but two weeks would appear to be an appropriate waiting time. An abbreviated assessment could be completed initially although it must allow for sufficient breadth of evaluation. Standardized cognitive assessment should be included along with collection of injury-related information and a comprehensive developmental and educational history. At this stage of recovery the use of post-concussional symptom checklists should be included. Kirkwood et al. (2008) identified that measures of speeded responding, memory, attention, and executive functioning are the most sensitive to injury and must be included. In addition, measures that are insensitive to the effects of brain injury (e.g.,

single word reading) in concussion must be included. Post-traumatic anxiety, pain and sleep should also be evaluated.

When there are ongoing concerns about cognitive functioning, a decision to make a referral for more in-depth neuropsychological assessment should be considered. Similar to research in the adult population, cognitive challenges that persist beyond three to six months should be more carefully evaluated (Kirkwood et al., 2008; Echemendia et al., 2013). Neuropsychological assessment should include measures of academic functioning, as well as an evaluation of mood. Child and caregiver accounts of the injury should be supplemented by objective information and records (e.g., hospital and physician records). Consideration of non-injury factors must also be made carefully. A comprehensive psychosocial history and a review of academic history must be obtained to determine if there are pre-existing learning and/or attentional issues. Information pertaining to family history and stressors must also be included. Assessment should continue to evaluate post-concussive symptoms, general somatic issues (e.g., pain) and cognitive domains that are sensitive to brain injury. A broad assessment of cognitive, psychosocial and achievement functioning is required at this stage.

Issues related to effort: The evaluation of effort in an assessment is necessary so that appropriate conclusions can be drawn and recommendations made. Assessment following concussion in children and adolescents must also include measures of effort. This includes assessments ranging from baseline evaluations to comprehensive batteries. Lichtenstein, Moser, & Schatz (2014) identified that both age and test setting impacted the validity of baseline scores on ImPACT. Younger children were more likely to produce invalid baseline results, the prevalence of which increased when testing was completed in a large group or nonclinical setting.

Kirkwood & Kirk (2010) examined suboptimal effort on the Medical Symptom Validity Test (MSVT) in children and adolescents aged 8 to 17 who had sustained a concussion. The base rate of suboptimal effort was found to be 17%. Only one failure was thought to be related to litigation. Kirkwood & Kirk (2010) identified that a sizable minority of children put forth suboptimal effort. Kirkwood, Connery, Kirk & Baker (2014) explored performance on embedded indicators of effort. In a sample of 439 children with concussion, between the ages of 8 and 17, it was found that 13% failed the MSVT and also performed below cutoffs on the TOMM or the Wechsler Digit Span subtest. In addition, they performed worse than the group passing the MSVT on measures of automatic sequencing (e.g., reciting the alphabet, counting to 20, saying the days of the week, reciting the months of the year). The sequencing tasks were found to be of promise in detecting invalid performance in children and adolescents with concussion.

Araujo et al. (2014) examined the relationship between suboptimal effort and post-concussion symptoms in both children and adolescents with concussion. They examined 382 children and adolescents between the ages of 8 and 16. Those who demonstrated non-credible performance (20% of their sample) presented with a greater number of symptoms than those with credible performance. In addition, those with suboptimal performance had poorer performance on measures of focused attention and

processing speed. Non-credible performance was identified using reliable digit span and age-corrected scaled scores from the Numbers subtest of the Children's Memory Scale. Kirkwood, Peterson, Connery, Baker & Grubenhoff (2014) also found that, in a group of 191 patients aged 8 to 17, 12% failed the MSVT and this group endorsed significantly more postconcussive symptoms than those children and adolescents who passed the MSVT.

The rate of suboptimal effort in children and adolescents with concussion appears to be in the range of 20% (Araujo et al., 2014; Kirkwood & Kirk, 2010; Kirkwood, Hargrave, & Kirk, 2011). In these studies, individuals with suboptimal effort had no known involvement in litigation. This percentage of individuals is reported to be higher than that identified in adults with concussion in non-forensic contexts (estimated to be between 4 and 7%; Araujo et al., 2014). In the study by Araujo et al. (2014) it was identified that a greater proportion of non-credible performers had received some form of special educational services compared with credible performers.

Age effects: Review of a number of studies indicates that as children age there is a higher number of post-concussion symptoms. This might be because older individuals are more aware of their symptoms as they have greater insight or recognize how their symptoms interfere with the day-to-day functioning (Araujo et al., 2014). This observation argues strongly for a developmentally sensitive approach to assessment in children and adolescents with concussion.

Conclusions: The research is limited in the area of psychometric properties for baseline assessment measures developed for children. The development of standardized administration and interpretation of baseline measures in children is imperative and should involve neuropsychologists. Further research is needed in this area. In addition, there is a need to assess the long-term outcome of children who have sustained mild traumatic brain injury. It is important to ensure that assessment measures are age and developmentally appropriate. Furthermore, assessment must be broad and include input from parents and teachers. The implementation of a comprehensive assessment is appropriate when symptoms persist beyond the expected recovery phase, although abbreviated assessments can be implemented earlier in the child's recovery. Finally, it is important that assessment batteries from baseline to comprehensive neuropsychological assessments include measures of effort.

c) Concussions in Sports: The Effects of Multiple Concussions

While the effect of a single concussion has been relatively well studied, the impact of multiple concussion injuries is much less understood. For the most part, this issue has been studied almost exclusively within the sports literature with mixed findings. Iverson (2007) found no relationship between prior concussion history and subsequent recovery time. In contrast, Collins, Lovell, Iverson, Cantu, Maroon, and Field (2002) reported that high school athletes with a self-reported history of three prior concussions were more than nine times more likely than those with no prior concussions to display abnormal markers of injury when injured again, including the presence of loss of consciousness, anterograde or retrograde amnesia, and confusion. These authors argued that the

findings reflected a lowered concussion threshold but they also noted the possibility that results might have reflected a sample that was selectively vulnerable to concussion injuries. Additional limitations of this study concerned the fact that the concussions were self-reported and that the severity of impact and the temporal proximity of prior concussions were not considered. In 2003, Guskiewicz, McCrea, Marshall, Cantu, Randolph, Barr, et al. demonstrated that college football players with a history of concussion were more likely to have future concussion injuries. These authors observed an increase in the likelihood of recurrent injury with each successive previous injury. Following three concussions, athletes were found to have a three-fold greater risk of future concussion injuries compared to those athletes who had never sustained such injury. They also demonstrated that re-injury often took place within a short window of time, usually 7 to 10 days following the first concussion. Animal studies have described an acute neurometabolic cascade involving accelerated glycolysis and increased lactate production immediately following concussion. Increased lactate is thought to leave neurons more vulnerable to secondary ischemic injury and it has been argued that it may predispose one to further injury (Giza 2000 and 2001). Animal models also suggest decreased cerebral blood flow lasting approximately 10 days following a concussion, which is consistent with Guskiewicz's findings of a 7 to 10 day period of increased susceptibility to additional injury. Further research in humans is still required.

These studies examined the risk of additional concussions following one such injury and the relationship between prior concussion and recovery; however, they did not examine the effect of multiple concussions on neuropsychological performance. Macciocchi, Barth, Littlefield, and Cantu (2001) reported no difference between pre-season and post-concussion neuropsychological test scores in athletes who reported multiple prior concussions and athletes who sustained a single prior concussion. However, the very small sample size ($n = 12$) examined in the study may have accounted for the lack of significant findings. Belanger and Vanderploeg (2005) conducted a meta-analysis and compared athletes involved in risky sports such as boxing and soccer to control participants in less risky sports (e.g., track and field) and found a significant and moderate effect ($d = .31$) on cognitive measures with the largest effects found in the domains of delayed memory, executive function, and language. These studies were, however, quite variable in terms of the selection of participants and their length of participation in sports and number of previous head injuries. Furthermore, there was no discussion about measures of symptom validity used to ascertain the reliability of the cognitive data in the individual patient assessments.

In 2010, Belanger, Spiegel, and Vanderploeg conducted a meta-analysis and found no significant main effect for repetitive injury on cognitive symptoms. Secondary analyses, however, found small effect sizes for multiple concussions and delayed memory and executive functioning, but the clinical significance of these findings was uncertain. Furthermore, the included studies did not examine the impact of prior concussions on recovery from a recent concussion. Thus, additional research is required.

A few recent studies have shown an association between multiple concussions and persistent emotional symptoms. Bryan and Clemens (2013) found an association

between lifetime number of concussions and suicidal ideation in a sample of concussed military personnel independent of the effects of depression and PTSD. However, whether these symptoms persisted weeks after the injury was not assessed. In 2014, Spira, Lathan, Bleiberg, and Tsao examined 646 United States Marines in good standing (i.e., not on limited duty medical status or pending medical or disciplinary charges) and showed that having had three or more lifetime concussions was associated with worse emotional distress and PCS. However, at this time it is unknown whether these findings translate to non-military personnel. It should also be noted that it is unclear whether the marines in this study suffered from concomitant orthopedic or soft tissue injuries, which could have produced pain conditions and associated increased emotional distress and PCS.

Overall, the effect of multiple concussions remains largely unknown as the findings in the literature are mixed. However, in general, the TBI literature argues against the prospect of catastrophic neuropsychiatric, functional, and neuropathological outcomes following a modest number of concussion injuries separated by an adequate inter-injury recovery period.

PART 4: CONCLUSIONS AND EVIDENCE-BASED RECOMMENDATIONS

a) Typical Recovery and Post-Concussive Symptoms:

- Concussion symptoms (physical, cognitive, affective) should typically resolve within 7 to 28 days, although symptoms may recover at different rates (e.g., neurocognitive versus vestibular versus subjective)
- Many post-traumatic symptoms reported following concussion are also commonly reported in acute stages of other injuries and are not specific to concussion
- While post-concussion symptoms in the acute stage of injury likely reflect the neurophysiology of the concussion, clinicians should keep in mind that PCS is a non-specific cluster of symptoms that can be mimicked by a number of pre-existing or comorbid conditions
- Careful and thorough differential diagnoses should be considered when recovery and/or outcome severity is atypical and does not follow the natural course expected from such injuries
- Many factors associated with protracted recovery and PCS, including pre-existing conditions, psychiatric conditions/history, negative injury perceptions, compensation, and litigation are the most stable predictors of prolonged PCS
- Psychosocial factors more strongly associated with outcomes than biomedical factors
- Increasing evidence indicates that many factors previously identified as symptoms of PCS are actually indicative of psychological responses and generalized trauma reaction. It has been shown that recovery is undermined in

patients who mistakenly perceive these symptoms as indicators of brain injury that may be permanent. Accurate identification of the true nature and cause of concussion-related symptoms is imperative as mistakenly attributing stress-related disturbances to neurological factors may deprive patients effective treatments that can, in most cases, alleviate the symptoms

- Terminology should be changed to *post-concussion symptoms* instead of *post-concussion syndrome* given that many 'post-concussive' symptoms are now regarded as 'common reactions' to health stressors
- Psychologists should not necessarily attribute persisting cognitive symptoms to remote concussion (i.e., more than 1 year post-injury), even if patients believe this is the case; psychologists should avoid suggesting that a person has permanent cognitive impairment as a result of the pathophysiology of concussion, as it will likely be deleterious to recovery and lead to perpetuation of actual impairment and poorer prognosis
- Strong evidence to suggest that providing patients with education about concussion, symptoms, and expectations for recovery, combined with graduated reintegration into physical activity, work, and/or school decreases the severity and duration of symptoms
- Treating the underlying and/or comorbid condition(s) is imperative for improvement in a person's functional abilities and outcomes

b) SIS/CTE:

- Incidence of SIS is controversial as it has only been reported in North America and not in countries where sports-related concussion rates are much higher (e.g., Australian football)
- Existence of SIS is questionable due to methodological limitations in previous studies (i.e., positive neuroimaging observed during the initial brain injury, which is inconsistent with concussion definition)
- There is little epidemiological data, consensus or systematic evidence of the existence of SIS
- Evidence-based practice in concussion requires avoidance of the term SIS, as it may cause unnecessary alarm; focus should instead remain on prevention, safety, and adherence to return-to-activity guidelines
- Consensus regarding the direct relationship between concussion and CTE remains unclear; therefore psychologists should proceed with caution when discussing the likelihood of future neurodegeneration with patients; focus should again remain on prevention, safety, and adherence to return-to-activity guidelines

c) Baseline Testing:

- Uncontrolled and unsupervised access to online concussion baseline testing undermines the validity and should never be allowed; testing should always take place in person, and under the supervision of a trained healthcare provider
- Baseline tests should include validity indicators that will help psychologists identify discrepancies indicative of suboptimal performance or 'sandbagging'

d) Assessment/Diagnosis of Concussion:

- Base rates of post-concussive symptoms among normal people are quite high, with some symptoms less 'concussion specific' than others; strict reliance on the number of endorsed concussive symptoms on symptom checklists is ill-advised and will lead to poor specificity and high false positive rates; instead, assessments should include differentiation of low specificity symptoms (e.g., fatigue, poor concentration) and high specificity symptoms (e.g., noise sensitivity); diagnosis based solely on symptom checklists/inventories is contraindicated
- Inadequately trained or untrained individuals often administer or direct sports concussion management programs; they may not fully understand standardized assessment procedures or extraneous factors that might affect testing results or recovery and should therefore not be considered reliable
- Concussion management programs developed for schools have often been developed and overseen by inadequately trained individuals, which is cause for concern; whenever possible, school-based concussion management programs should involve consultation by pediatric neuropsychologists
- Clinical interview is a critical component of concussion assessments to evaluate relevant medical/psychiatric history, symptom presentation/progression, and comorbidities
- Neuropsychological assessment is still considered the gold standard of chronic concussion evaluation but is not clinically indicated during the acute stage where typical recovery is expected
- Symptom validity tests (SVTs) should be included in all neuropsychological assessments of concussion, given the common comorbidities of extraneous factors affecting recovery, including litigation and psychological factors

e) Differential Diagnosis of Concussion:

- The evaluation of concomitant whiplash and/or neck injuries is of paramount importance when evaluating concussion due not only to its high comorbidity but the fact that it can be a significant cause of post-traumatic headaches, and subsequently, neurocognitive complaints and symptoms
- Individuals with chronic pain experience similar symptomatology to those with concussion in terms of subjective (self-report) and objective (neurocognitive testing) data, suggesting that chronic pain must be evaluated and considered within the context of concussion evaluation
- Psychologists should evaluate whether a patient has a history of headaches or migraines (with or without aura), and whether they have been formally diagnosed with migraine and/or posttraumatic headache since sustaining a concussion as these can impact a patient's neurocognitive functioning and may account for cognitive inefficiencies attributed to 'post-concussive' etiology
- Neuropsychologists evaluating concussion should ascertain information pertaining to a patient's sleep quality, both prior to and since the concussive

injury; conditions such as sleep apnea, restless leg syndrome, anxiety, and chronic sleep deprivation can hinder a patient's functioning and may mimic symptoms of concussion

- Psychogenic factors (including depression, anxiety, and PTSD) play a large role in the development, perpetuation, and recovery of post-concussive symptoms. Given their ability to mimic 'post-concussive' symptomatology, a comprehensive concussion assessment must include an evaluation of the individual's psychological history as well as current psychoemotional status
- Non-specific symptoms associated with 'post-concussive' syndrome share many features with anxiety, PTSD, and other traumatic-based psychological disorders; it is becoming more apparent, therefore, that presumed sequelae of concussion should be conceptualized rather as a generalized psychological response to trauma
- ADHD is both a risk factor for concussion and for poorer outcome following injury; as such, it needs to be addressed in concussion assessments
- Current literature is unclear regarding the relationship between learning and developmental disabilities and concussion, although there are some studies showing greater impairment in those with pre-existing learning and intellectual/developmental challenges

f) Concussion in Older Adults:

- Concussion is an independent and clinically significant risk factor for death in the elderly
- Older age appears to be associated with poorer outcome following concussion, although selection bias may hinder reliability and validity of empirical findings

g) Concussion in Pediatric Populations:

- Similar to adults, recovery from concussion in pediatric populations has been found to be variable, with similar injury and non-injury factors affecting outcome
- Concussions in pediatric populations may have the potential to impact a child's development; however, long-term studies and assessments are still required to identify whether concussion results in long-term effects in children
- A critical difference between concussion assessment of children and adults is the role that parents and other adults play in recovery and management; therefore, proper psychoeducation of parents and teachers is of paramount importance in the prevention of misunderstanding and symptom perpetuation
- Using practice guidelines and principles that have been developed for adult populations is not recommended in children; similarly, adult measures designed to assess symptoms are not appropriate for pediatric populations and only assessment measures specifically designed for pediatric populations must be utilized (e.g., SAC, ACE)
- In pediatric cases, full neuropsychological assessment is not warranted during the acute stages of injury; however, children should be referred for neuropsychological consultation if they fail to recover within the usual time frame,

if they have a history of multiple concussions, or if their symptoms are severe, persistent, or functionally disruptive

- Symptom validity tests should be incorporated into neuropsychological assessment of concussion in pediatric populations; sequencing tasks may hold the most promise in detecting invalid performance in children and adolescents with concussion although further research is needed

h) Multiple Concussions:

- The effect of multiple concussions remains largely unknown as the findings in the literature are mixed
- In general, the TBI literature argues against the prospect of catastrophic neuropsychiatric, functional, or neuropathological outcomes following a modest number of concussion injuries separated by an adequate inter-injury recovery period

APPENDIX A

Indications for Neuroimaging Guidelines from the American College of Emergency Physicians Jagoda, A.S., Bazarian, J.J., Burns, J.J., et al. (2008)

Imaging is indicated in patients with a loss of consciousness or amnesia if at least one of the following is present: headache (diffuse), vomiting, age older than 60 years, intoxication, deficits in short-term memory, evidence of trauma above the clavicle, seizures, GCS score of less than 15, focal neurologic deficits, coagulopathy.

Imaging is indicated in patients with no loss of consciousness or amnesia if at least one of the following is present: focal neurologic deficit, vomiting, severe headache, age older than 65 years, signs of basilar skull fracture, GCS score of less than 15, coagulopathy, significant mechanism of injury (e.g., ejection from vehicle, pedestrian struck by vehicle, fall from a height greater than 3 feet or five stairs).

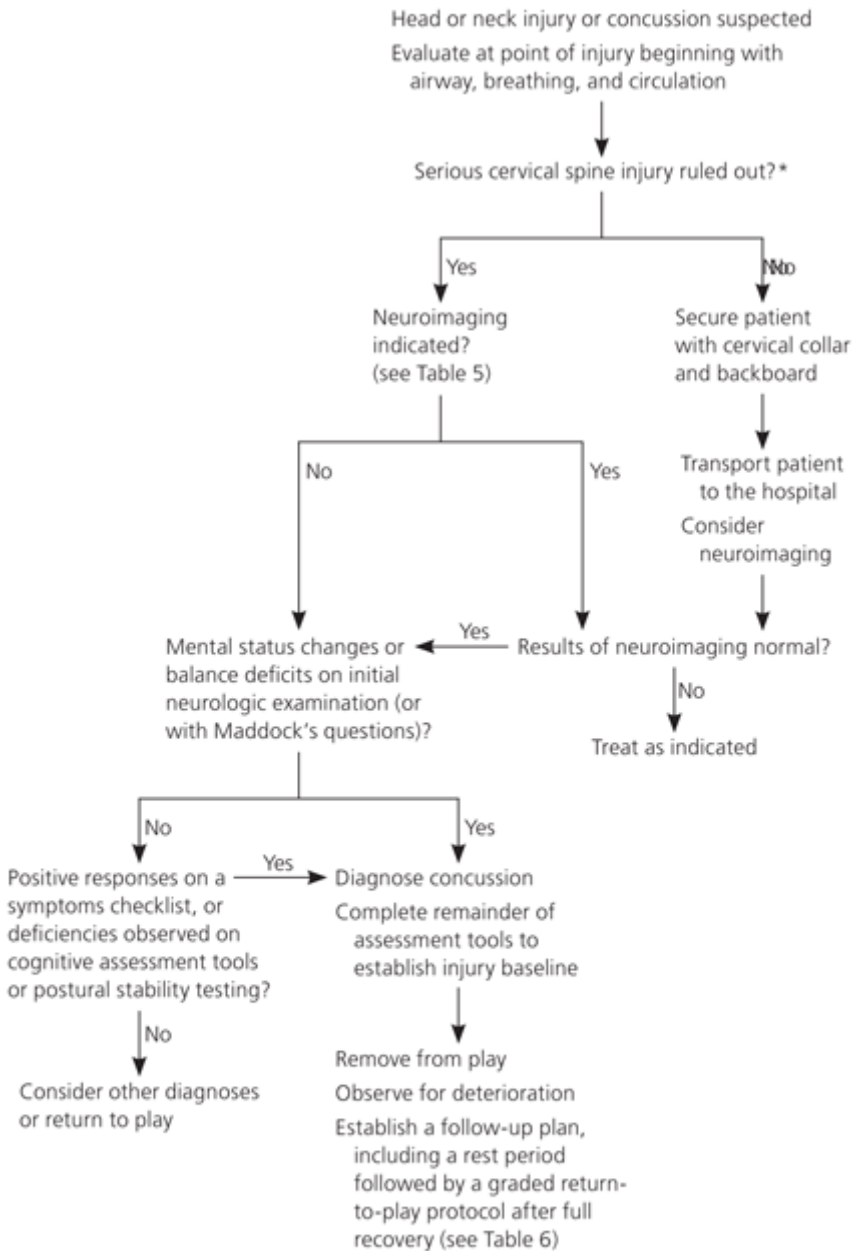
Indications for Neuroimaging: Guidelines from the American Academy of Pediatrics and the American Academy of Family Physicians Scorza, K.A., Raleigh, M.F., & O'Connor, F.G. (2012)

Perform imaging in patients with loss of consciousness of greater than 60 seconds, evidence of skull fracture, or focal neurologic findings.

Consider imaging or observation if patient has brief loss of consciousness.

Note that nonspecific signs (e.g., immediate seizures, headache, vomiting, lethargy) increase the likelihood of intracranial injury, but have very limited predictive value..

APPENDIX B



*—Cervical spine injury can be ruled out and the patient may be moved if all of the following are normal (perform evaluations without moving the head or neck): peripheral strength and sensation, absence of asymmetric spasm or spinal tenderness, isometric neck strength, active range of motion of the neck, Spurling test. Exceptions: altered level of consciousness, intoxication, distracting injuries, midline tenderness, focal neurologic deficits.

REFERENCES

- Ahman, S., Saveman, B. I., Styrke, J., Bjornstig, U., & Stalnacke, B. M. (2013). Long-term follow-up of patients with mild traumatic brain injury: a mixed-method study. *Journal of Rehabilitation Medicine, 45*, 758-764.
- Airaksinen, E., Larsson, M., & Forsell, Y. (2005). Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction. *Journal of Psychiatric Research, 39*(2), 207-214.
- Alosco, M. L., Fedor, A.R., & Gunstad, J. (2014). Attention deficit hyperactivity disorder as a risk factor for concussions in NCAA division-1 athletes. *Brain Injury, 472-474*.
- Araujo, G. C., Antonini, T. N., Monahan, K., Gelfius, C., Klamar, K., Potts, M., Yeates, K. O., & Bodin, D. (2014). The relationship between suboptimal effort and post-concussion symptoms in children and adolescents with mild traumatic brain injury. *The Clinical Neuropsychologist, 28*, 786-801.
- Ardila, A., & Sanchez, E. (1988). Neuropsychologic symptoms in the migraine syndrome. *Cephalalgia, 8*, 67-70.
- Bazarian, J. J., & Atabaki, S. (2001). Predicting postconcussion syndrome after minor traumatic brain injury. *Academic Emergency Medicine, 8*, 788-795.
- Belanger, H. G., Curtiss, G., Demery, J. A., Lebowitz, B. K., & Vanderploeg, R. D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society, 11*, 215-227.
- Belanger, H. G., Spiegel, E., & Vanderploeg, R. D. (2005). The neuropsychological impact of sports-related concussions: A meta-analysis. *Journal of International Neuropsychological Society, 11*, 215-227.
- Belanger, H. G., Spiegel, E., & Vanderploeg, R. D. (2010). Neuropsychological performance following a history of multiple self-reported concussions: A meta-analysis. *Journal of International Neuropsychological Society, 16*, 262-267.
- Binder S. (2002). Injuries among older adults: The challenge of optimizing safety and minimizing unintended consequences. *Injury Prevention, 8*(Suppl. 4), IV2-IV4.
- Binder, L.M., Rohling, M. L., & Larrabee, G. J. (1997). A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical Experimental Neuropsychology, 19*, 421-431.

- Boake, C., McCauley, S. R., Levin, H. S., Pedroza, C., Contant, C.F. Song, J. X., et al. (2005). Diagnostic criteria for postconcussional syndrome after mild to moderate traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neuroscience*, 17, 350-356.
- Bonfield, C.M., Lam, S., Lin, Y., & Greene S. (2013). The impact of attention deficit hyperactivity disorder on recovery from mild traumatic brain injury. *Journal of Neurosurgery: Pediatrics*, 12, 97-102.
- Broshek, D., De Marco, A., & Freeman, J. (2015). A review of post-concussion syndrome and psychological factors associated with concussion. *Brain Injury*, 29(2), 228-237.
- Bryan, C. J., & Clements, T. A. (2013). Repetitive traumatic brain injury, psychological symptoms, and suicidal risk in a clinical sample of deployed military personnel. *Journal of American Medical Association Psychiatry*, 70, 686-691.
- Bryant, R. (2011). Post-traumatic stress disorder versus traumatic brain injury. *Dialogues Clinical Neuroscience*, 13, 251-262.
- Bryant, R. A., Creamer, M., O'Donnell, M., Silove, D., Clark, C. R., & McFarlane, A. C. (2009). Post-traumatic amnesia and the nature of post-traumatic stress disorder after mild traumatic brain injury. *Journal of International Neuropsychological Society*, 15, 862-867.
- Butler, I. J. (2013). Postconcussion syndrome after mild traumatic brain injury in children and adolescents requires further detailed study. *Journal of American Medical Association*, 1-2.
- Carone, D. A., & Bush, S. S. (Eds.). (2013). *Mild traumatic brain injury: Symptom validity assessment and malingering*. New York: Springer Publishing.
- 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*, 36 (Suppl. 43), 113-125.
- Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., et al. (2004). Prognosis for mild traumatic brain injury: Results of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine*, 36, 84 – 105.
- Cassidy, J. D., Cancelliere, D.C., Carroll, L. J., Cote, P., Hincapie, C. A., Holm, L. W. et al. (2014). Systematic review of self-reported prognosis in adults after mild traumatic brain injury: Results of the international collaboration of mild traumatic

- brain injury prognosis. *Archives of Physical Medicine and Rehabilitation*, 95, S132-151.
- Castenada, A., Tuulio-Henriksson, A., Marttunen, M., Suvisaari, J., & Lonnqvist, J. (2008). A review on cognitive impairments in depressive and anxiety disorders with a focus on young adults. *Journal of Affective Disorders*, 106(1-2), 1-27.
- Centers for Disease Control (2010). Injury and violence prevention [October 18, 2004] www.healthypeople.gov/Document/HTML/Volume2/15Injury.htm
- Chan, R. (2001). Base rate of post-concussion symptoms among normal people and its neuropsychological correlates. *Clinical Rehabilitation*, 15, 266-273.
- Chen, H., Richard, M., Sandler, D.P., Umbach, D.M., & Kamel, F. (2007). Head injury and amyotrophic lateral sclerosis. *American Journal of Epidemiology*, 166, 810–816.
- Cheng, P.-O., Lin, H.-Y., Lee, Y.-K., Hsu, C.-Y., Lee, C.-C., & Su, Y.-C. (2014). Higher mortality rates among the elderly with mild traumatic brain injury: A nationwide cohort study. *Scandinavian Journal of Trauma, Resuscitation, and Emergency Medicine*, 22.
- Collins, M. W., Lovell, M. R., Iverson, G. L., Cantu, R. C., Maroon, J. C., & Field, M. (2002). Cumulative effects of concussion in high school athletes. *Neurosurgery*, 51, 1175-1181.
- Coronado, V. G., Thomas, K. E., Sattin, R. W., et al. (2005). The CDC traumatic brain injury surveillance system: Characteristics of persons aged 65 years and older hospitalized with a TBI. *Journal of Head Trauma Rehabilitation*, 20, 215-228.
- Davis, C. H. (2002). Self-perception in mild traumatic brain injury, *American Journal of Physical Medicine and Rehabilitation*, 81, 609 -621.
- Dikmen, S., Machamer, J., Fann, J. R., and Temkin, N. R. (2010). Rates of symptom reporting following traumatic brain injury. *Journal of International Neuropsychological Society*, 16, 401-411.
- Di Stefano, G. & Radanov, B. (1995). Course of attention and memory after common whiplash: A two-years prospective study with age, education and gender pair-matched patients. *Acta Neurologica Scandinavica*, 91, 346-352.
- Donders, J., & Strom, D. (1997). The effect of traumatic brain injury on children with learning disability. *Pediatric Rehabilitation*, 1, 179-184.

- Dresler, T., Lurding, R., Paelecke-Habermann, Y., Gaul, C., Henkel, K., Lindwurn-Spath, A., Leinisch, E., & Jurgens, T. (2012). Cluster headache and neuropsychological functioning. *Cephalalgia*, 33(11), 813-821.
- Durmer, J., & Dinges, D. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology*, 25(1), 117-129.
- Echemendia, R. J., Iverson, G. L., McCrea, M., Macciocchi, S. N., Gioia, G. A., Putakian, M. & Comper, P. (2013). Advances in neuropsychological assessment of sport-related concussion. *British Journal of Sports Medicine*, 47, 294-298.
- Ettlin, T., Kischka, U., Reichmann, S., Radii, E., Heim, S., Wengen, D., & Benson, D. (1992). Cerebral symptoms after whiplash injury of the neck: A prospective clinical and neuropsychological study of whiplash injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 55, 943-948.
- Fann, J., Burington, B., Leonetti, A., Jaffe, K., Katon, W., & Thompson, R. (2004). Psychiatric illness following traumatic brain injury in an adult health maintenance organization population. *Archives of General Psychiatry*, 61, 53-61.
- Farmer, J., Kanne, S., Haut, J., Williams, J., Johnstone, B., & Kirk, K. (2002). Memory functioning following traumatic brain injury in children with premorbid learning problems. *Developmental Neuropsychology*, 22, 455-469.
- Fioravanti, M., Ramelli, L., Napoleoni, A., et al. (1983). Post-traumatic headache: Neuropsychological and clinical aspects. *Cephalalgia*, 3, 221-224.
- Fischer, J., & Vaca, F. (2004). Sport-related concussions in the emergency department. *Topics in Emergency Medicine*, 26, 260-266.
- Gimse, R., Bjorgen, I., Tjell, C., Tyssedal, J., & Bo, K. (1997). Reduced cognitive functions in a group of whiplash patients with demonstrated disturbances in the posture control system. *Journal of Clinical and Experimental Neuropsychology*, 19, 838-849.
- Gioia, G. A., Collins, M. & Isquith, P. K. (2008). Improving identification and diagnosis of mild traumatic brain injury with evidence: Psychometric support for the acute concussion evaluation. *Journal of Head Trauma Rehabilitation*, 23, 230-242.
- Gioia, G.A., Schneider, J.C., Vaughan C.G., & Isquith, P. K. (2009). Which symptom assessments and approaches are uniquely appropriate for pediatric concussion? *British Journal of Sports Medicine*, 43 (Suppl 1), i13-i22.
- Giza, C. C., & Hovda, D. A. (2000). Ionic and metabolic consequences of concussion. *Neurologic Athletic and Spine Injuries*. 36, 80-100.

- Giza, C. C., & Hovda, D. A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training*, 36, 228-235.
- Goldman, S.M., Tanner, C.M., Oakes, D., Bhudhikanok, G.S., Gupta, A., & Langston, J.W. Head injury and Parkinson's disease risk in twins. *Annals of Neurology*, 60, 65–72.
- Goldstein, F. D., & Levin, H. S. (2001). Cognitive outcome after mild and moderate traumatic brain injury in older adults. *Journal of Clinical and Experimental Neuropsychology*, 23, 739-753.
- Gordon, K., Dooley, J., & Wood, E. (2006). Is migraine a risk factor for the development of concussion? *British Journal of Sports Medicine*, 40(2), 184-185.
- Guskiewicz, K. M., McCrea, M., Marshall, S. W., Cantu, R. C., Randolph, C., Barr, W., et al. (2003). Cumulative effects associated with recurrent concussion in collegiate football players. *Journal of American Medical Association*, 290, 2549-2555.
- Hart, R.P., Martelli, M.F., & Zasler, N.D. (2000). Chronic pain and neuropsychological findings. *Neuropsychology Review*, 10(3), 131-149.
- Headache Classification Subcommittee of the International Headache Society (2004). The international classification of headache disorders. *Cephalalgia*, 24 (supplement 1), 9-160.
- Heilbronner, R. L., Sweet, J. J., Morgan, J. E., Larrabee, G. J., Millis, S. R., et al. (2009). American Academy of Clinical Neuropsychology consensus conference statement on the neuropsychological assessment of effort, response bias, and malingering. *The Clinical Neuropsychologist*, 23, 1093-1129.
- Henry, L., Elbin, R., Collins, M., Marchetti, G., & Kontos, A. (2016). Examining recovery trajectories after sport-related concussion with a multimodal clinical assessment approach. *Neurosurgery*, 0, 1-9
- Hoge, C., McGurk, D., Thomas, J., Cox, A., Engel, C., & Castro, C. (2008). Mild traumatic brain injury in US soldiers returning from Iraq. *New England Journal of Medicine*, 358, 453- 463.
- Hou, R., Moss-Morris, R., Peveler, R., Mogg, K., Bradley, B. P., & Belli, A. (2012). When a minor head injury results in enduring symptoms: a prospective investigation of risk factors for postconcussional syndrome after mild traumatic brain injury. *Journal of Neurology, Neurosurgery, and Psychiatry* 83, 217-223.
- Iverson, G. (2006). Misdiagnosis of the persistent postconcussive syndrome in patients with depression. *Archives of Clinical Neuropsychology*, 21(4), 303-310.

- Iverson, G. (2007). Predicting slow recovery from sport-related concussion: The new simple-complex distinction. *Clinical Journal of Sport Medicine*, 17, 31-37.
- Iverson, G.L. & McCracken, L.M. (1997). 'Postconcussive' symptoms in persons with chronic pain. *Brain Injury*, 11(11), 783-790.
- Iverson, G. L., Zasler, N. D., & Lange, R. T. (2007). Post-concussive disorder. *Brain injury medicine: Principles and practice*, 373-405.
- Jagoda, A.S., Bazarian, J.J., Burns, J.J., et al. (2008). Neuroimaging and decision making in adult mild traumatic brain injury in the acute setting. *Annals of Emergency Medicine*, 52, 714-748.
- Johnstone, B., Hexum, B., & Ashkazi, G. (1995). Extent of cognitive decline in traumatic brain injury based on estimates of premorbid intelligence. *Brain Injury*, 9, 377-384.
- Jorge, R., Leston, J., Ardnt, S., & Robinson, R. (1999). Cluster headaches: Association with anxiety disorders and memory deficits. *Neurology*, 53, 543-547.
- Karantzoulis, S., & Randolph, C. (2013). Modern chronic traumatic encephalopathy in retired athletes: What is the evidence? *Neuropsychology Review*, 23(4), 350-360.
- Karlin, A. M. (2011). Concussion in the pediatric and adolescent population: "Different population, different concerns". *Physical Medicine and Rehabilitation*, 3, 369-379.
- Karzmark, P., Hall, K., & Englander, J. (1995). Late-onset post-concussion symptoms after mild brain injury: the role of premorbid, injury-related, environmental, and personality factors. *Brain Injury*, 9(1), 21-26.
- Kashluba, S., Casey, J. E., & Paniak, C. (2006). Evaluating the utility of ICD-10 diagnostic criteria for postconcussion syndrome following mild traumatic brain injury. *Journal of International Neuropsychological Society*, 12, 111-118.
- Kessels, R., Aleman, A., Verhagen, W., & Van Luitelaar, E. (2000). Cognitive functioning after whiplash injury: A meta-analysis. *Journal of the International Neuropsychological Society*, 6, 271-278.
- Kirkwood, M. W., Connery, A. K., Kirk, J.W., & Baker D. A. (2014). Detecting performance invalidity in children: Not quite as easy as A, B, C, 1, 2, 3 but automated sequences appears promising. *Child Neuropsychology*, 20, 242-252.
- Kirkwood, M. W., Hargrave, D. D., & Kirk, J. W. (2011). The value of the WISC-IV

- Digit Span subtest in detecting noncredible performance during pediatric neuropsychological examinations. *Archives of Clinical Neuropsychology*, 26, 377-384.
- Kirkwood, M. W., & Kirk, J. W. (2010). The base rate of suboptimal effort in a pediatric mild TBI sample: Performance on the Medical Symptom Validity Test. *The Clinical Neuropsychologist*, 24, 860-872.
- Kirkwood, M. W., Peterson, R. L., Connery, A. K., Baker, D. A., & Grubenhoff, J. A. (2014). Postconcussive symptom exaggeration after pediatric mild traumatic brain injury. *Pediatrics*, 133, 643-650.
- Kirkwood, M. W., Randolph, C., & Yeates, K. O. (2009). Returning pediatric athletes to play after concussion: The evidence (or lack thereof) behind baseline neuropsychological testing. *Acta Paediatrica*, 98, 1409-1411.
- Kirkwood, M. W., Yeates, K. O., Taylor, H. G., Randolph, C., McCrea, M., & Anderson, V. A. (2008). Management of pediatric mild traumatic brain injury: A neuropsychological review from injury through recovery. *The Clinical Neuropsychologist*, 22, 769-200.
- Kizilbash, A., Vanderploeg, R., & Curtiss, G. (2002). The effects of depression and anxiety on memory performance. *Archives of Clinical Neuropsychology*, 17(1), 57-67.
- Landre, N., Poppe, C., Davis, N., Schmaus, B., & Hobbs, S. (2006). Cognitive functioning and postconcussive symptoms in trauma patients with and without mild TBI. *Archives of Clinical Neuropsychology*, 21, 255-273.
- Lange, R. T., Iverson, G. L., & Rose, A. (2011). Depression strongly influences postconcussion symptom reporting following mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 26, 127-137.
- Langlois, J.A., Rutland-Brown, W., & Thomas, K.E. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths. National Center for Injury Prevention and Control; Atlanta, GA: 2004.
- Larrabee, G. L., Greiffenstein, M. F., Greve, K. W., & Bianchini, K. J. (2007). Refining diagnostic criteria for malingering. In G. L. Larrabee (Ed.), *Assessment of malingered neuropsychological deficits* (pp. 334-317).
- Le Pira, F., Lanaia, F., Zappala, G., et al. (2004). Relationship between clinical variables and cognitive performances in migraineurs with and without aura. *Functional Neurology*, 19, 101-105.
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological*

- assessment* (5th ed.). New York: Oxford University Press.
- Lichtenstein, J. D., Moser, R. S., & Schatz, P. (2014). Age and test setting affect the prevalence of invalid baseline scores on neurocognitive tests. *American Journal of Sports Medicine*, 42, 479-484.
- Macciocchi, S. N., Bath, J. T., Littlefield, L., & Cantu, R. C. (2001). Multiple concussions and neuropsychological functioning in collegiate football players. *Journal of Athletic Training*, 36, 303-306.
- Margulies, S. (2000). The postconcussion syndrome after mild head trauma. Part II: Is migraine underdiagnosed? *Journal of Clinical Neuroscience*, 7, 495-499.
- Martland, H. (1928). Punch drunk. *Journal of the American Medical Association*, 91, 1103-1107.
- Mautner, K., Sussman, W.I., Axtman, M., Al-Farsi, Y., & Al-Adawi, S. (2015). Relationship between Attention-Deficit Hyperactivity Disorder and post-concussion recovery in youth athletes. *Clinical Journal of Sport Medicine*, 25, 355-360.
- McAllister, T., & Arciniegas, D. (2002). Evaluation and treatment of postconcussive symptoms. *Neurorehabilitation*, 17, 265-283.
- McCauley, S. R., Boake, C., Pedroza, C., Brown, S. A., Levin, H. S., Goodman, H. S., and Merritt, S. G. (2008). Correlates of persistent postconcussional disorder: DSM-IV criteria versus ICD-10. *Journal of Clinical Experimental Neuropsychology*, 30, 360-379.
- McCracken, L., & Iverson, G. (2001). Predicting complaints of impaired cognitive functioning in patients with chronic pain. *Journal of Pain and Symptom Management*, 21(5), 392-396.
- McCrory, P., Meeuwisse, W.H., Aubry, M., et al. (2013). Consensus statement on concussion in sport: The 4th International Conference on Concussion in Sport held in Zurich, November 2012. *British Journal of Sports Medicine*, 47, 250-258.
- McKay, C. D., Brooks, B. L., Mrazik, M., Jubinville, A. L., & Emery, C. A. (2014). Psychometric properties and reference values for the ImPACT neurocognitive test battery in a sample of elite youth ice hockey players. *Archives of Clinical Neuropsychology*, 29, 141-151.
- McKee, A., Cantu, R., Nowinski, C., Hedley-White, E.T., Gavett, B.E., Budson, A.E.,

- Santini, V.E., Less, H.S., Kubilus, C.A., & Stern, R.A. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury. *Journal of Neuropathology and Experimental Neurology*, 68, 709–735.
- Miller, H. (1966). Mental after-effects of head injury. *Proceedings of the Royal Society of Medicine*, 59, 257–261.
- Millspaugh, J.A. (1937). Dementia Pugilistica. *US Navy Medical Bulletin*, 35, 297–303.
- Mittenberg, W., Tremont, G., Zielinski, R. E., Fichera, S., & Rayls, K. R. (1996). Cognitive-behavioral prevention of postconcussion syndrome. *Archives of Clinical Neuropsychology*, 11, 139–145.
- Mongini, F., Keller, R., Deregibus, A., Barbalonga, E., & Mongini, T. (2005). Frontal lobe dysfunction in patients with chronic migraine: A clinical-neuropsychological study. *Psychiatry Research*, 133(1), 101-106.
- Moser, R. S., Schatz, P., & Lichtenstein, J. D. (2015). The importance of proper administration and interpretation of neuropsychological baseline and postconcussion computerized testing. *Applied Neuropsychology: Child*, 4, 41-48.
- Mulder, E., Linssen, W., Passchier, J., Orlebeke, J., & de Geus, E. (1999). Interictal and postictal cognitive changes in migraine. *Cephalalgia*, 19, 557-565.
- National Center for Injury Prevention and Control. Injury Fact Book. Centers for Disease Control and Prevention; Atlanta, GA: 2002.
- Neinstein, L., & Milgrom, E. (2000). Trauma-triggered migraine and acute confusional migraine. *Journal of Adolescent Health*, 27, 119-124.
- Newman, J. B., Reesman, J. H., Vaughan, C. G., & Gioia, G. A. (2013). Assessment of processing speed in children with mild TBI: A “first look” at the validity of pediatric ImPACT. *The Clinical Neuropsychologist*, 27, 779-793.
- O’Bryant, S., Marcus, D., Rains, J., & Penzien, D. (2006). The neuropsychology of recurrent headache. *Headache, October*, 1364-1376.
- Pilcher, J., & Huffcutt, A. (1996). Effects of sleep deprivation on performance: A meta-analysis. *Sleep*, 19(4), 318-326.
- Ponsford, J., Willmott, C., Rothwell, A., Cameron, P., Ayton, G., Nelms R., et al. (1999). Cognitive and behavioural outcomes following mild traumatic head injury in children. *The Journal of Head Trauma Rehabilitation*, 14, 360-372.
- Ponsford, J., Willmott, C., Rothwell, A., Cameron, P., Kelly, A-M., Nelms, R., Curran, C., & Ng., K. (2000). Factors influencing outcome following mild traumatic

- brain injury in adults. *Journal of the International Neuropsychological Society*, 6, 568-579.
- Polusny, M., Kehle, S., Nelson, N., Erbes, C., Arbisi, P., & Thuras, P. (2011). Longitudinal effects of mild traumatic brain injury and posttraumatic stress disorder comorbidity on postdeployment outcomes in National Guard soldiers deployed to Iraq. *Archives of General Psychiatry*, 68(1), 79-89.
- Radanov, B., Dvorak, J., & Valach, L. (1992). Cognitive deficits in patients after soft tissue injury of the cervical spine. *Spine*, 17, 127-131.
- Radanov, B., Hitlinger, I., Di Stefano, G., & Valach, L. (1992). Attentional processing in cervical spine syndromes. *Acta Neurologica Scandinavica*, 85, 358-362.
- Rapoport, M. J., Harrmann, N., Shammi, P., Kiss, A., Phillips, A., & Feinstein, A. (2006). Outcome after traumatic brain injury sustained in older adulthood: A one-year longitudinal study. *American Journal of Geriatric Psychiatry*, 14, 456-465.
- Rapoport, M. J., Kiss, A., & Feinstein, A. (2006). Impact of major depression on outcome following mild-to-moderate traumatic brain injury in older adults. *Journal of Affective Disorders*, 92, 273-276.
- Raskin, S. A., & Mateer, C. A. (Eds.) (2000). *Neuropsychological management of mild traumatic brain injury*. New York: Oxford University Press.
- Rohling, M. L., Binder, L. M., Demakis, G. J., Larrabee, G. J., Ploetz, D. M., & Langhinrichsen-Rohling, J. (2011). A meta-analysis of neuropsychological outcome after mild traumatic brain injury: Re-analyses and reconsiderations of Binder et al. (1997), Frencham et al. (2005), and Pertab et al. (2009). *Clinical Neuropsychologist*, 25, 608 –623.
- Rush, B. K., Malee, J. F., Moessnes, A. M., Brown, A. W. (2004). Preinjury personality traits and the prediction of early neurobehavioural symptoms following mild traumatic brain injury, *Rehabilitation Psychology*, 49, 275 – 281.
- Satz, P. (2001). Mild head injury in children and adolescents. *Current Directions in Psychological Science*, 10, 106-109.
- Satz, P., Zaucha, K., McCleery, C., Light, R., Asarnow, R., & Becker, D. (1997). Mild head injury in children and adolescents: A review of studies (1970-1995). *Psychological Bulletin*, 122, 107-131.
- Scorza, K.A., Raleigh, M.F., & O'Connor, F.G. (2012). Current concepts in concussion: Evaluation and management. *American Family Physician*, 85(2), 123-132.
- Sesma, H., Slomine, B., Ding, R., McCarthy, M., & The Children's Health After Trauma

- Study Group. (2008). Executive functioning in the first year after pediatric traumatic brain injury. *Pediatrics*, 121, 1246-1247.
- Sigurdardottir, S., Andelic, N., Roe, C., Jerstad, T., & Schanke, A. K. (2009). Post-concussion symptoms after traumatic brain injury at 3 and 12 months post-injury: A prospective study. *Brain Injury*, 23, 489-497.
- Silver, J.M., McAllister, T.W., & Arciniegas, D.B. (2009). Depression and cognitive complaints following mild traumatic brain injury. *American Journal of Psychiatry*, 166, 653-661.
- Sinforiani, E., Farina, S., Mancuso, A., Manzoni, G., Bono, G., & Mazzucchi, A. (1987). Analysis of higher nervous functions in migraine and cluster headache. *Functional Neurology*, 2, 69-77.
- Sjorgen, P., Christup, L., Petersen, M., & Hojsted, J. (2005). Neuropsychological assessment of chronic non-malignant pain patients treated in a multidisciplinary pain centre. *European Journal of Pain*, 9, 453-462.
- Slick, D. J., Sherman, E. M. S., & Iverson, G. L. (1999). Diagnostic criteria for malingered neurocognitive dysfunction: Proposed standards for clinical practice and research. *The Clinical Neuropsychologist*, 13, 545-561.
- Smith-Seemiller, L., Fow, N., Kant, R., & Franzen, M. (2003). Presence of post-concussion syndrome symptoms in patients with chronic pain vs. mild traumatic brain injury. *Brain Injury*, 17(3), 199-206.
- Solomon, S. (1998). Posttraumatic migraine. *Headache*, 38, 772-778.
- Spira, J. L., Lathan, C. E., Bleiberg, J. & Tsao, J. W. (2014). The impact of multiple concussions on emotional distress, post-concussive symptoms, and neurocognitive functioning in active duty United States Marines independent of combat exposure or emotional distress. *Journal of Neurotrauma*, 31, 1823-1834.
- Stalnacke, B. M. (2012). Postconcussion symptoms in patients with injury-related chronic pain. *Rehabilitation Resource Practice*, 2012, 1-5.
- Stalnacke, B. M., Bjornstig, U., Karlsson, K., & Sojka, P. (2005). One-year follow-up of mild traumatic brain injury: post-concussion symptoms, disabilities and life satisfaction in relation to serum levels of S-100B and neurone-specific enolase in acute phase. *Journal of Rehabilitation Medicine*, 37, 300-305.
- Styrke, J., Sojka, P., Bjornstig, U., & Stalnacke, B. (2014). Symptoms, disabilities, and life satisfaction five years after whiplash. *Scandinavian Journal of Pain*, 5, 229-236.

- Suhr, J., & Seng, E. (2012). Neuropsychological functioning in migraine: Clinical and research implications. *Cephalalgia*, 32(1), 39-54.
- Sullivan, K. A., Edmed, S. L., Cunningham, L. C. A comparison of new and existing mild traumatic brain injury vignettes: Recommendations for research into post-concussion syndrome (2013). *Brain Injury*, 27, 19-30.
- Thompson, H. J., McCormick, W. C., & Kagan, S. H. (2006). Traumatic brain injury in older adults: Epidemiology, outcomes, and future implications. *Journal of the American Geriatric Society*, 54, 1590-1595.
- Tiersky, L. A., Anselmi, V., Johnston, M. V., Kurtyka, J., Roosen, E., Schwartz, T., et al (2005). A trial of neuropsychologic rehabilitation in mild-spectrum traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 86, 1565-1574.
- Torkamani, M., Ernst, L., Sze Cheung, L., Lambru, G., Matharu, M., & Jahanshahi, M. (2015). The neuropsychology of cluster headache: Cognition, mood, disability, and quality of life of patients with chronic and episodic cluster headache. *Headache, February*, 287-300.
- Tsushima, W., & Newbill, W. (1996). Effects of headaches during neuropsychological testing of mild head injury patients. *Headache*, 36, 613-615.
- Tsushima, W., & Tsushima, V. (1993). Relation between headaches and neuropsychological functioning among head injury patients. *Headache*, 33, 139-142.
- Uomoto, J. M. (2008). Older adults and neuropsychological rehabilitation following acquired brain injury. *NeuroRehabilitation*, 23, 415-424.
- Uomoto, J., & Esselman, P. (1993). Traumatic brain injury and chronic pain: Differential types and rates by head injury severity. *Archives of Physical Medicine and Rehabilitation*, 74, 61-64.
- Valovich McLeod, T. C., Barr, W. B., McCrea, M., & Guskiewicz, K. M. (2006). Psychometric and measurement properties of concussion assessment tools in youth sports. *Journal of Athletic Training*, 41, 399-408.
- Vanderploeg, R. D., Belanger, H. G., & Kaufmann, P. M. (2014). Nocebo effects and mild traumatic brain injury: Legal implications. *Psychological Injury and Law*, 7, 245-254.
- Vasterling, J., Brailey, K., Proctor, S., Kane, R., Heeran, T., & Franz, M. (2012). Neuropsychological outcomes of mild traumatic brain injury, post-traumatic stress disorder, and depression in Iraq-deployed army soldiers. *British Journal of Psychiatry*, 208(4), 1-7.

- Vasterling, J., Proctor, S., Amoroso, P., Kane, R., Heeran, T., & White, R. (2006). Neuropsychological outcomes of army personnel following deployment to the Iraq war. *Journal of the American Medical Association*, 296(5), 519-529.
- Veiel, H.O. (1997). A preliminary profile of neuropsychological deficits associated with major depression. *Journal of Clinical and Experimental Neuropsychology*, 19(4), 587-603.
- Waldie, K., Hausmann, M., Milne, B., & Poulton, R. (2000). Migraine and cognitive function: A life-course study. *Neurology*, 59, 904-908.
- Waters, F. & Bucks, R.S. (2011). Neuropsychological effects of sleep loss: Implication for neuropsychologist. *Journal of the International Neuropsychological Society*, 17, 1-16.
- Williams, R., Paetz, T., Giza, C., & Brolio, S. (2015). Concussion recovery time among high school and collegiate athletes: A systematic review and meta-analysis. *Sports Medicine*, 43, 893-903.
- Wood, R., & Rutterford, N. (2006). The impact of mild developmental learning difficulties on neuropsychological recovery from head trauma. *Brain Injury*, 20, 477-484.
- Woodward, H., Winterhalther, K., Donders, J., Hackbarth, R., Kuldane, A., & Sanfilippo, D. (1999). Prediction of neurobehavioural outcome 1-5 years post pediatric traumatic head injury. *The Journal of Head Trauma Rehabilitation*, 14, 351-359.
- World Health Organization. International statistical classification of disease and related health problems (10th edition). Geneva: Author; (1992).
- Yeates, K. O. (2010). Mild traumatic brain injury and postconcussive symptoms in children and adolescents. *Journal of the International Neuropsychological Society*, 16, 953-960.
- Yeates, K. O., Kaizar, E., Rusin, R., Bangert, B., Dietrich, A., Nuss, K., Wright, M., & Taylor, H.G. (2012). Reliable change in postconcussive symptoms and its functional consequences among children with mild traumatic brain injury. *Archives of Pediatric Adolescent Medicine*, 166, 615-622.
- Yeates, K. O. & Taylor, H. G. (2005). Neurobehavioural outcomes in children and adolescents. *Pediatric Rehabilitation*, 8, 5-16.
- Yuen, T.J., Browne, K.D., Iwata, A., & Smith, D.H. (2009). Sodium channelopathy induced by mild axonal trauma worsens outcome after a repeat injury. *Journal of Neuroscience Research*, 87, 3620-3625.

- Ziegler, E. A., & Boone, K. B. (2013). Symptom invalidity on neuropsychological testing. In P. A. Arnett (Ed.), *Secondary influences on neuropsychological test performance: Research findings and practical applications* (pp. 7-38). New York: Oxford University Press.
- Zumsteg, D., Wennberg, R., Gutling, E., & Hess, K. (2006). Whiplash and concussion: Similar acute changes in middle-latency SEPs. *Canadian Journal of Neurological Sciences*, 33, 379-386.