



Expert Opinion

Medico-Legal Aspects of Concussion

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Concussion and the sequelae have been controversial medico-legal issues for over 150 years. The following topics which are also important in clinical practice are discussed: definitions of concussion, neuroimaging, onset and prognosis of headaches, cognitive impairment, cognitive rehabilitation, post-traumatic stress disorder, and risk of later development of dementia.

Key words: concussion, mild traumatic brain injury, post-concussion syndrome, post-traumatic headache, cognitive impairment, cognitive rehabilitation

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INTRODUCTION

In 1280, Lanfrancus distinguished *commotio* (shaking) *cerebri* with no damage from *contusio cerebri* with structural damage.¹ The word “concussion” (from Latin to shake) was first used in the 14th century.² In 1674, Boirel suggested that invisible brain damage might occur in concussion.³ Concussion has been a controversial medico-legal topic for over 150 years.^{4,5}

The following are composite cases that highlight the recurring issues which occur in medico-legal cases as well as in clinical practice.

CASES

Case 1.—A 24-year-old female was the restrained driver of a car that was broadsided. She hit her head and was dazed. CT of the brain in the emergen-

cy department was negative. She developed new onset headaches with a migraine phenotype 3 days a week starting 1 month after the accident. No prior history of headaches.

Case 2.—A 40-year-old male was the restrained front seat passenger of a pick-up truck hit from behind by an 18-wheeler. He denied loss of consciousness at the scene and in the emergency department where he reported only neck and shoulder pain. The head was atraumatic, the Glasgow Coma Scale score was 15, and the neurological exam was normal. CT of the brain was negative. He saw a chiropractor 1 week later and was followed for 1 month with complaints of only neck and shoulder pain.

Six months later, his attorney referred him to a primary care physician who obtained a history of brief loss of consciousness, frequent headaches, memory and concentration problems, anxiety, and depression. Neuropsychological testing was reported as supporting a diagnosis of major neurocognitive disorder due to a traumatic brain injury. He was referred to a neurologist who obtained a MRI of the

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brain with quantitative diffusion tensor imaging reported as showing significantly reduced functional anisotropy in multiple white matter fiber regions consistent with acceleration/deceleration-induced closed head injury resulting in diffuse axonal injury to both cerebral hemispheres.

Case 3.—A 22-year-old female was shopping in a warehouse store when a heavy box fell from a shelf hitting her on the head. She was dazed. The Glasgow Coma Scale score in the emergency department was 15 and the CT of the brain was negative. She had daily headaches, persistent difficulty with concentration and memory, vertigo, and tinnitus that persisted for over 1 year. She had to take a leave of absence from college. Neuropsychological testing 1 year after the injury was reported as showing major neurocognitive impairment and depression.

Questions.—Why are these 3 cases of interest? What is the definition of concussion? Is concussion a mild traumatic brain injury? Can concussion be under and overdiagnosed? Can concussion be present with a normal CT or MRI scan of the brain? Should quantitative diffuse tensor imaging be used to diagnose post-traumatic diffuse axonal injury in individual patients? Is the term “post-concussion syndrome” problematic? How long after the injury can headaches have an onset to be considered post-traumatic? How often do headaches occur after concussion? What is the prognosis of post-traumatic headaches? How often does cognitive impairment occur after concussion and in what domains? What is the prognosis? What percentage have permanent cognitive impairment? Can neuropsychologists misdiagnose or overdiagnose cognitive impairment? Why? What is the efficacy of cognitive rehabilitation for cognitive impairment after concussion? How often does post-traumatic stress disorder (PTSD) occur after concussion? Can PTSD be overdiagnosed? How do you determine return to work after concussion? Does concussion increase the risk for later development of dementia?

EXPERT OPINION

Why are these 3 Cases of Interest?—These cases raise questions which commonly arise in medico-legal cases and in clinical practice. For case 1, is a migraine phenotype headache with onset 1 month after

concussion a primary migraine, which arises by coincidence or a post-traumatic headache? Case 2 is a common scenario where the patient’s history changes weeks or months later from the initial report of no loss or alteration of consciousness. Can you/should you diagnose concussion based upon subjective symptoms in medico-legal cases? Are neuropsychological testing and diffusion tensor imaging MRI reliable methods for diagnosing traumatic brain injury? Finally, how often does a concussion without loss of consciousness result in headaches and clinically significant neurocognitive impairment for more than 1 year after the injury?

What is the Definition of Concussion?—There are different definitions of concussion.

According to the American Association of Neurological Surgeons, “A concussion is an injury to the brain that results in temporary loss of normal brain function. Medically, it is defined as a clinical syndrome characterized by immediate and transient alteration in brain function, including alteration of mental status or level of consciousness that results from mechanical force or trauma.”⁶

Definitions have been provided for concussion in sport.

According to the American Academy of Neurology concussion in sport guideline, “Concussion is recognized as a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness (LOC).”⁷

The Concussion in Sport Group defines concussion “as a complex pathophysiologic process induced by biomechanical forces ... results in a range of clinical signs and symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive features typically follows a sequential course. However, in some cases symptoms may be prolonged.”⁸

Although the definitions of concussion in sport have been arbitrarily provided to meet the demands of sporting organizations for guidelines on recovery and safe return to play, the same definitions should apply to non-sport concussions.⁹

Is Concussion a Mild Traumatic Brain Injury (mTBI)?—Yes. Concussion is more accurately defined as a minimal mTBI.¹⁰ Different definitions of mTBI have been proposed. According to the Ameri-

can Congress of Rehabilitation Medicine, mTBI can occur after the head being struck, the head striking an object, or the brain undergoing an acceleration/deceleration movement without direct external trauma to the head and is “a traumatically induced physiological disruption of brain function, as manifested by at least one of the following: (1) any period of loss of consciousness; (2) any loss of memory for events immediately before or after the accident; (3) any alteration in mental state at the time of the accident (eg, feeling dazed, disoriented, or confused); and (4) focal neurological deficit(s) that may or may not be transient; but where the severity of the injury does not exceed the following:

- loss of consciousness of approximately 30 minutes or less;
- after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and
- posttraumatic amnesia (PTA) not greater than 24 hours.”¹¹

The distinction between concussion and mTBI has not been clearly defined.¹² The initial Glasgow Coma Scale score is between 13 and 15 for mTBI but would reasonably be 15 for concussion although this is not part of the definitions. The definitions do not provide an upper limit for the duration of LOC to distinguish concussion from mTBI while the upper limit for LOC for mTBI is 30 minutes.

In the past, one distinguishing factor between concussion and mTBI was opined to be that concussion was fully reversible which is not true in a minority of cases. Conversely, most people with mTBI make a full recovery.

Can concussion Be Under and Overdiagnosed?—Yes. Concussion is a clinical diagnosis. The following statement for sport-related concussions (SRC) applies for all concussions: “Currently, there is no gold or reference standard for SRC diagnosis, and the diagnostic properties of the various definitions have not been studied.”⁹

Underdiagnosis of Concussion.—Patients may not be aware that they had LOC and a witness may not have been present. The duration of LOC provided may not be accurate because the patient may incorrect-

ly believe that the duration of the post-traumatic amnesia was the duration of the LOC. Patients may not report or be asked about the acute symptoms of alteration of mental state for example, feeling dazed, disoriented, or confused.

The duration of LOC can be assessed in some cases by review of medical records (reports of witnesses, first responders, emergency department (ED) records, and subsequent treaters). However, an adequate history may not be taken in the ED to establish the diagnosis. In a prospective American ED study of 197 adults in Seattle, 56% of mTBI patients were not identified.¹³ Although the patients were typically asked about LOC, other symptoms typical of concussion such as confusion were not assessed. In a Canadian prospective ED study of 250 adults, 16% were not diagnosed with concussion despite meeting criteria.¹⁴ In a retrospective Australian ED study of 351 adults, only 23.1% with confirmed mTBI had this diagnosis clearly recorded in the medical notes.¹⁵ Subsequent non-neurologist treaters may also not take an adequate history to establish a diagnosis of concussion.

Overdiagnosis of Concussion.—It can be difficult to determine whether a concussion actually occurred and concussion can be easily overdiagnosed based upon subjective symptoms. After a non-brain injury, people may experience confusion and disorientation due to a strong emotional reaction¹⁶ which can be misdiagnosed as a concussion.

The duration of post-traumatic amnesia may not be accurate. Causes for inaccurate durations include the following: psychogenic amnesia (due to an acute stress disorder or PTSD); the effect of alcohol or drugs (either taken by the patient or administered by paramedics or physicians); and other physical injuries and pain.⁵

Later reports from the patient of their recollection may not be accurate because they may incorrectly remember what happened or include third party information which they believe is their own. Secondary gain and malingering can also result in inaccurate reporting.

The symptoms of post-concussion syndrome (PCS) are not specific to concussion and their presence alone may not determine that a concussion occurred.¹⁷ PCS mimics include the following: base rate misattribution,^{18,19} depression,²⁰ attention-deficit/hyperactivity disorder,²¹

post-traumatic stress disorder,²² chronic pain,²³ obstructive sleep apnea,²⁴ litigation,²⁵ and malingering.²⁶

Can Concussion Be Present With a Normal CT or MRI Scan of the Brain?.—Yes. In a meta-analysis, the estimated prevalence of intracranial CT abnormalities of patients presenting to the hospital with a GCS score of 15 is 5%.²⁷ In a prospective study of 20 patients with concussion who had MRI scans within 24 hours and after 3 months compared to controls, there were no cases of definite intracranial abnormalities due to trauma.²⁸

MRI is more sensitive than CT in detecting the following: small amounts of parenchymal, subdural, and epidural hemorrhage; contusion; and posterior fossa, brainstem, and diffuse axonal injuries. In a prospective study of 135 mTBI patients evaluated acutely in the ED, 27% of those with normal CT scans had an abnormal MRI scan performed 12 ± 3.9 days after injury.²⁹

Should Quantitative Diffusion Tensor Imaging (DTI) Be Used to Diagnose Diffuse Axonal Injury in Individual Patients With Concussion?.—No. Douglas et al conclude the following: “DTI is sensitive at detecting TBI at the group level (TBI-group vs control group), but there is insufficient evidence to suggest that DTI plays a clinical role for diagnosing mild TBI at the individual patient level.”³⁰

Is the Term “PCS” Problematic?.—Yes. PCS is a symptom complex with many symptoms that may include 1 or more of the following: headache, dizziness, neuropsychiatric, and cognitive.³¹ PCS is a heterogeneous disorder with controversy over the definition, incidence, etiology, and pathophysiology.³² Many clinicians use the term PCS as a convenient label for the sequelae of mTBI, not just concussion. People who have not had a concussion can have symptoms which meet the criteria for PCS and be overdiagnosed. It is more accurate to use the term post-mTBI syndrome rather than PCS (although it is unlikely that the use of this term will cease just as sciatica is still used). Most guidelines and authors prefer the use of the term “postconcussion symptoms” rather than “syndrome” because of the nonspecificity of the complaints.³³

To Be a Post-Traumatic Headache, What Can Be the Greatest Latency Between the Concussion and Onset?.—According to the International Classification

of Headache Disorders, 3rd edition (ICHD-3), “Headache attributed to trauma or injury to the head and/or neck require that headache must be reported to have developed within seven days following trauma or injury”³⁴

The latency is arbitrary and might underdiagnose some cases especially for post-traumatic headaches with a migraine phenotype where the pathophysiology is not understood. A latency of up to 3 months has been proposed but may overdiagnose nontraumatic headaches as traumatic.³⁵

Consider the 24-year-old female in case 1 with new onset headache with a migraine phenotype starting 1 month after the concussion. Could she have developed unrelated new onset primary migraine? Migraine incidence peaks in the 20- to 24-year-old age group in women at 18.2 per 1000 person-years³⁶ so the monthly incidence is 0.15%

Non-neurologists may not obtain a headache history so it may not be possible to reliably determine the onset and clinical course of post-traumatic headaches from the medical records. Conversely, the patient’s recall may be inaccurate and they could have had preexisting headaches incorrectly diagnosed as post-traumatic.

How Often Do Headaches Occur After Concussion?.—Headaches have been reported by up to 90% of those who are symptomatic after mTBI.³⁷ In a large prospective ED study of subjects with mTBI with a median Glasgow Coma Scale score of 15, 45% reported headaches in the ED and 51% at 2 weeks (defined as new or worsening preexisting headache beginning within 7 days after trauma).³⁸ In collegiate athletes, headaches were reported by 92.2% after concussions.³⁹

Preexisting migraines may increase following concussion.⁴⁰ According to ICHD-3, “When a preexisting headache with the characteristics of a primary headache disorder becomes chronic or is made significantly worse (usually meaning a twofold or greater increase in frequency and/or severity) in close temporal relation to such trauma or injury, both the initial headache diagnosis and a diagnosis of Headache attributed to trauma or injury to the head and/or neck (or 1 of its types or subtypes) should be given, provided that there is good evidence that the disorder can cause headache.”³⁴ Medication overuse can occur for both preexisting migraines and post-traumatic migraine-type headaches

when acute headache medications are overused or with the frequent use of pain medications for other injuries.

What is the Prognosis of Post-Traumatic Headaches?.—The long-term prognosis of headaches after concussion is not certain as studies have investigated the prognosis of headaches after mTBI, not just concussion. Headaches may still be present at 3 months in up to 75% and even at 4 years in 20%.⁴¹

How Often Does Cognitive Impairment Occur After Concussion and in What Domains?.—Cognitive impairment can occur following a TBI of any severity⁴² and even very mild TBIs have been shown to temporarily impact cognitive functioning.⁴³ However, the incidence of cognitive impairment post-concussion is not certain from the literature. Sample characteristics and methodology (eg, operational definitions of injury) can make it difficult to interpret findings across studies and to generalize results given the possible influence of sociodemographic variables, reported symptomatology, preexisting medical and psychiatric conditions, and administered cognitive measures (eg, sensitivity and specificity).

Research has documented cognitive declines in the domains of attention, memory, processing speed, and in cognitive (eg, planning, organizing, sequencing, set-shifting, and problem solving) and emotional/behavioral aspects (inhibition, emotional regulation, behavior monitoring) of executive functions.⁴⁴⁻⁴⁹ Memory declines typically associated with mTBI include difficulties with acquisition and/or with the strategic aspects involved in the encoding process, rather than with the consolidation or retention of information.⁵⁰ Declines in serial position learning, semantic organization, and interference effects can be observed via standardized neuropsychological testing.⁵¹

What is the Prognosis? What Percentage Have Permanent Cognitive Impairment?.—Prognosis following concussion is generally good. Cognitive impairment, as measured by neuropsychological outcome measures has been shown to improve and resolve in the initial, days, weeks, or months.^{52,53} Several systematic reviews involving meta-analysis have shown that the impact of mTBI on neurocognitive performance becomes undetectable at the group-level by 3 months post injury.⁵⁴⁻⁵⁹ Symptoms beyond 1-year post injury are referred to as persistent (PCS) and while some literature indicates

this condition is estimated to impact approximately 15% of individuals with a first-time concussion,⁶⁰ findings should be interpreted with caution as the symptom constellation of PCS is non-specific and overlaps with other conditions, such as chronic fatigue, fibromyalgia, and mood disorders.⁶¹

Can Neuropsychologists Under or Overdiagnose Cognitive Impairment? Why?.—Under and overdiagnosing of cognitive impairment is possible and neuropsychological data should be interpreted within the examinee's context and with consideration of the individual's academic, medical, and psychiatric history. Underdiagnosis can occur in individuals with high levels of premorbid functioning (eg, superior, very superior abilities prior to the injury) where neuropsychological data evidences subtle declines (eg, low average scores), but the clinical classification of scores is below the threshold (less than 1.5 or 2 standard deviations below the mean) to meet criteria for a cognitive diagnosis. In these cases, the clinician's acumen is imperative in understanding the obtained cognitive profile, the type of typical cognitive changes found post-concussion, and the psychometric properties of the administered measures.

Over-diagnosing of cognitive impairment post-concussion can occur when preexisting factors are not considered. Previous medical diagnoses of headaches,^{62,63} migraines,⁶⁴ Attention-Deficit Hyperactivity Disorder (ADHD),⁶⁵⁻⁶⁷ learning disability,⁶⁸ and psychiatric conditions⁶⁹⁻⁷² may influence symptom presentation and these conditions are known to contribute to a high baseline symptom score pre-concussion and worsened symptom score and neuropsychological test performance post-concussion.^{73,74} Moreover, sleep difficulties, pain, and side effects of medication (eg, topiramate for migraine prevention) can influence performance on neurocognitive outcome measures and confound findings.⁷⁵ A pre-and post-concussion comparison of medical and psychological symptoms should be conducted to assist with data interpretation, as it can be difficult to disentangle the examinee's current presentation from preexisting conditions.

The literature has warned against the negative impact of over-diagnosing individuals with cognitive impairment because such labels can engender misattribution bias by the examinee.^{61,76} Furthermore,

inaccurate cognitive diagnoses may result in un-needed examinations, interventions, and management services that are not warranted given the examinee's medical evidence.

Neuropsychologists must employ performance validity testing throughout the assessment to examine level of engagement and confirm validity of obtained data.^{77,78} In addition, the influence of sociodemographic variables, including cultural and linguistic factors, should be considered when examining cognitive symptoms in ethnic minorities and non-English speakers as lack of attention to these possible influences can result in misdiagnosis.⁷⁹

What is the Efficacy of Cognitive Rehabilitation for Cognitive Impairment After Concussion?.—mTBI is characterized as a heterogeneous disorder with a varied clinical presentation. Cognitive rehabilitation programs differ in regard to addressed cognitive domains, level of complexity in regard to assigned work, duration of intervention, and generalizability of noted improvements to real world settings (ecological validity). Research has shown that a tailored, active, targeted approach to mTBI symptoms and impairments will yield improvements in cognition and social functioning.⁸⁰

A literature review conducted by Vanderploeg and colleagues⁸¹ concerning the effectiveness of cognitive rehabilitation and CBT methods on mTBI symptoms revealed a dearth of standardization in established and studied cognitive rehabilitation procedures. Interventions focused solely on cognitive rehabilitation spanned in general over 12-15 weeks and hours were protocol dependent with varying distribution across days. Cognitive rehabilitation in combination with cognitive behavioral therapy (CBT) programs revealed generally longer intervention periods and this combination intervention proved most efficacious in alleviating PCS when compared to cognitive rehabilitation only. Authors encourage providers to focus on the comorbid and underlying the mental health symptoms rather than cognitive complaints, especially for those who reported PCS.⁸¹

Cognitive rehabilitation in the early stages of recovery may serve as a buffer against exacerbation of future cognitive complaints and impairments. Sample demographics of participants in published studies, intervention type (cognitive rehabilitation and combined

cognitive rehabilitation with a psychological or psychoeducational component) and duration of treatment should be examined to better assess the possible benefits of cognitive rehabilitation post-concussion. A tailored treatment approach is recommended once the individual's cognitive areas of strength and weakness have been identified via standardized neuropsychological testing.

How Often Does PTSD Occur After Concussion?.—Population based estimates of PTSD in the general community range from 7 to 8% and higher rates (up to approximately 20%) have been reported in American service members returning from deployment⁸²⁻⁸⁴ Research utilizing a screening measure to examine symptoms of PTSD in patients admitted to the hospital and diagnosed with mTBI described PTSD as a rare comorbidity following their injury (2.8%).⁸⁵ Nelson et al observed a link between PTSD symptoms and concussion in a sample of veterans; yet findings lack generalizability to the general population.⁸⁶

Prevalence of PTSD in cases of mTBI is difficult to gauge due to the varied and at times incorrect manner in which the diagnosis of PTSD is made. Mood disorders are highly prevalent with depression, anxiety, and somatic preoccupation being most common in mTBI^{87,88} and some of these symptoms can be incorrectly interpreted as symptoms of PTSD. This condition is unique among psychological disorders in that diagnostic criteria cannot be met without exposure to a traumatic event. The DSM-V defines a traumatic event as one in which a person has "exposure to actual or threatened death, serious injury, or sexual violence" and if this criterion is not met, reported symptoms do not meet criteria for a diagnosis of PTSD.⁸⁹ Given the possible symptom overlap in mental health disorders, a diagnosis of PTSD should come from a medical/mental health provider who is knowledgeable in the criteria required to meet this diagnosis.

Can PTSD Be Overdiagnosed?.—Yes. The Clinician Administered PTSD Scale for *Diagnostic Statistical Manual 5th edition* (CAPS-5) is the gold standard for PTSD assessment and diagnosis.⁹⁰ This measure along with a detailed clinical interview is imperative in assessing whether the criteria for a diagnosis of PTSD are met. Mental health providers must utilize their clinical acumen, as deviations from the proscribed language are

sometimes needed to ensure all required information is obtained from the examinee.

What is the Rate of Return to Work After mTBI?.—A number of studies have investigated rates of return to work and risk factors for not returning to work for mTBI but not specifically for concussion. In litigation cases, secondary gain and rare cases of malingering might further decrease the rate of returning to work.

A 1-year prospective study of a cohort with mTBI found a significant negative association between return to work at 12 months and psychological distress, global functioning post injury, and the sick leave trajectory of the subjects before and after the injury.⁹¹ Another 1-year prospective study of those with mTBI found that the rates of return to work increased from 34% at 2 weeks to 77% at 12 months with partial return to work varying from 8 to 16% throughout the year. Occupational factors influenced short-term return to work while post-traumatic complaints and early signs of psychological distress were short- and long-term predictors.⁹² In 1 study, the odds of successful return to work were more than cut in half for each unit increase in the numerical pain rating scale.⁹³

Does Concussion Increase the Risk of Later Onset Dementia?.—Concussion has not been conclusively linked to chronic cognitive dysfunction. Barr reinstated previous research findings indicating concussion may result in a temporary, “limited functional disturbance” over a prolonged disruption to cognitive performance that could develop into dementia/Major Neurocognitive Disorder.⁹⁴ Larrabee indicated that the norm following a single, uncomplicated mTBI in regard to cognitive functioning is full recovery with no long-term residual deficits.⁹⁵ While a scoping review suggested “a single mTBI chronically impairs cognitive functioning”⁹⁶ and subsequent articles^{97,98} have cited this study as illustrating a high rate of cognitive impairment secondary to concussion, Iverson and colleagues highlight the methodological errors that resulted in their erroneous conclusion.⁴² Overall, the literature has pronounced the lack of research support for long-term cognitive or psychological deficits secondary to concussion.⁵²⁻⁵⁴ In light of this highly supported conclusion, healthcare professionals are encouraged to seek alternative explanations for reported post-concussion

symptoms, other than the concussion and a PCS diagnosis. Some alternatives may include chronic pain,⁹⁹ mental health symptoms,¹⁰⁰ including personality disorders,¹⁰¹ substance use,¹⁰² medication side effects,^{103,104} and litigation.¹⁰⁵

Research has linked moderate and severe traumatic brain injury to a greater risk of developing Major Neurocognitive Disorder/Dementia. A prospective study by Plassman and colleagues¹⁰⁶ found that veterans who had sustained a severe TBI (defined as LOC or post-traumatic amnesia lasting longer than 24 hours) were 4.5 times as likely to have dementia compared with controls. Those who had experienced a moderate TBI (defined as LOC or post-traumatic amnesia lasting longer than 30 minutes but less than 24 hours) were at 2.3 times greater risk. The study did not find an increased risk for the veterans who had sustained mTBI (LOC or post-traumatic amnesia fewer than 30 minutes).

Emerging evidence indicates that repeated mTBIs may be linked to a greater risk of chronic traumatic encephalopathy (CTE), a neurodegenerative condition which in addition to cognitive impairments in complex attention, processing speed, memory, and executive functioning usually presents with neuropsychiatric disturbances, such as apathy, depression, irritability, impulsiveness, and suicidality.¹⁰⁷ The clinical presentation of CTE is distinct from the symptoms associated with PCS (for those who support the syndrome), as it is not the accumulation of symptoms from the earlier injuries.¹⁰⁸ The number of years of exposure to head trauma has been significantly associated with increased tau pathology in CTE; chronic and repetitive nature of head trauma, irrespective of concussion symptoms, has been identified as the central factor of disease^{50,109}

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