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Thirty Complexities and Controversies in Mild Traumatic Brain Injury and Persistent Post-concussion Syndrome: a Roadmap for Research and Practice

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Abstract

Mild traumatic brain injury (MTBI) is a contentious topic in the field of psychological injury and law, especially in cases in which the symptoms persist in persistent post-concussion syndrome (PPCS). The article reviews 30 points related to MTBI/PPCS that workers in the field need to consider: understanding these syndromes, their symptoms, their prevalence, their causation, the influences and confounds in their diagnosis, best assessment practices, and legal aspects. Evaluators need to know the scientific literature, adopt an unbiased approach, undertake comprehensive assessments, consider all data and factors, and arrive at judicious decisions. The literature indicates few conclusive findings and conclusions related to MTBI/PPCS, except for finding much variability (even in terms of definition), uncertainty, inconclusiveness, and the need for extensive research. The literature supports the view that PPCS is biopsychosocial and that biological factors by themselves cannot account for MTBI/PPCS psychological presentations. The psychological factors can extent into symptom exaggeration, feigning, and malingering, and these confounds need to be assessed carefully before being ruled in or out. Ethically, evaluators should not have preconceived notions either way. When bias is evident in these regards, the weight of testimony or proffered reports in court and related venues will be reduced or they might be deemed inadmissible. As for recommendations, the article proposes that the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), creates a new category termed somatic symptom disorder with predominant post-concussion-like symptoms. PPCS should not be considered a syndrome or anything related to the original index concussion/MTBI and should be dropped from the lexicon in the field.

Keywords Mild traumatic brain injury · Persistent post-concussion syndrome · PPCS · Controversies in MTBI · Influences/confounds in MTBI · Neuropsychological assessment · Malingering · Psychological injury · Treatment · Law · Somatic symptom disorder

Mild traumatic brain injury (MTBI) and persistent post-concussion syndrome (PPCS) are common conditions in the general population, but are not well understood, promote confusion in practitioners, and are contentious conditions in court and legal venues. They are part of the conditions that are referred to as psychological injuries, which are psychological conditions that are the result of negligence, such as motor vehicle accidents (MVAs) and work injuries, and lead to seeking compensation in court and related venues (e.g., workers' compensation institutions). Their compensable nature can lead to symptom exaggeration, feigning, or outright malingering (Young, 2014).

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MTBI is well-researched, with two recent broadscale reviews (Mayer, Quinn, & Master, 2017; Polinder et al., 2018), but there is no consensus even of its definitions and classifications, prevalence, chronicity, consequences and outcomes (e.g., PPCS), best assessment and diagnostic practices, and standing in court. The paper reviews the recent literature on MTBI/PPCS and lists 30 points relevant to understanding MTBI/PPCS, practicing in the field, and dealing with court and related venues. The area is a dynamic one and each of the 30 points could be expanded with a vast literature review and commentary, but that is beyond the scope of the present paper, which serves as a statement for the current state of the art in the field and what it needs to deal with the 30 points raised.

As for the particular research findings in the field on long-term brain and behavioral effects of MTBI, whether in PPS or otherwise, this paper conducted a literature search using major data mining engines (Psychinfo, Scopus, Web of Science) and

was overwhelmed by the constant and ever-burgeoning publications in the field. There were very few broadscale review and meta-analyses and hundreds, if not thousands, of research studies that could be cited on the short-term and long-term effects of MTBI/PPCS, with many published recently, and so it is beyond the scope of the present paper to integrate all of them. Nevertheless, this paper selected many recent publications that illustrate the major themes of the present article, including that the long-term effects of MTBI/PPCS are far from established. Some findings support the validity of long-term effects due to MTBI in PPCS and others explain them away, even if found, by confounding and complicating variables, including in poor effort, malingering, and the like.

The data engine searches of the literature that were conducted by the author for purposes of the present article led to thousands of articles that were surveyed for their contents, but only the two reviews mentioned above and elaborated in the next section were broadband enough and covered the full range of topics of interest for the present article, even when considering the reviews that emerged in the literature up to the present in 2020. The other reviews that emerged in the search engine mining, and there were tens of them, were on more specialized topics, and they are integrated into the manuscript as it proceeds, if required. The author maintains that most if not all of the most relevant articles for the present work have been included, but any that have been inadvertently excluded would not change the general picture of the article. In the end, toward two hundred articles have been cited in the present paper.

Recent Integrative Reviews

The Reviews

In 2017–2018, two systematic literature reviews were conducted on MTBI and its consequences (Mayer et al., 2017; Polinder et al., 2018). The article by Polinder et al. (2018) is replete with caution on how the field lacks a solid definitional, conceptual, and empirical base. For example, mechanisms and influencing factors on MTBI are "poorly understood." Prevalence rates vary between 11 and 82% due to variations in the research in terms of diagnostic criteria, population, and timing of assessments. The research to date has "substantial limitations." There are "gaps" in our understanding. Aside from diagnostic uncertainty, "uncertainty" characterizes epidemiology, etiology, prognosis, and treatment. Post-concussive symptoms might not organize into a coherent, predictable syndrome, so they might not represent a particular syndrome. There are multiple confounding factors to consider, for example, related to demographic and pre-injury factors and to comorbidities. The research shows a high rate of "false positives" in the assessment. There could be "gross" misrepresentation in malingering, or there could be factors at play, such as misattributing daily symptoms to a brain injury, such as headaches. The authors concluded that PPCS symptoms are subjective; overlap significantly with other physical, neurological, and psychiatric conditions; are heterogeneous; and lack a clear predictor/biomarker profile, and the condition itself has been questioned as viable. They called for a comprehensive research program, including longitudinally.

Like Polinder et al. (2018), Mayer et al. (2017) also cautioned that the study of MTBI and PPCS is "fragmented," with "conflicting diagnostic criteria," "different nosologies," "incomplete understanding," "great barriers," a lack of clear biomarkers, and "nonspecific" symptomatology that could be "malingered" in nature. The authors called for comprehensive "phenotyping" in the assessment of MTBI/PPCS.

Definitions and Classifications

In terms of the specific research findings in their reviews, Polinder et al. (2018) and Mayer et al. (2017) noted the American Congress of Rehabilitation Medicine (Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine, 1993) definition of MTBI as involving an acute brain injury due to mechanical energy deriving from external physical forces to the head. The symptom type that could result include loss of consciousness (LOC) of equal to or less than 30 min, post-traumatic memory loss (amnesia, PTA) of up to 24 h, and a Glasgow Coma Scale (GCS) score of 13 or more out of 15 after 30 min post-injury or (upon post-injury examination) along with either signs of confusion (e.g., dazed, disoriented, confused) for an unspecified time or (transient) neurological abnormalities (e.g., focal signs, seizures) or both. Other diagnostic systems have been described for MTBI, including the American Academy of Neurology (1997). About 5 to 10% of MTBI patients will express complicated MTBI, that is, with associated scan abnormalities such as subarachnoid hemorrhage, intracranial contusion, or small extra-axial hematomas.

The major psychiatric diagnostic systems present nosologies on post-concussive symptoms, but in different ways. The Diagnostic and Statistical Manual of Mental Disorders (DSM) deal with post-concussion syndrome (PCS) in the fourth edition (Diagnostic and Statistical Manual of Mental Disorders, 4th ed.; DSM-IV, American Psychiatric Association, 1994; DSM-IV-TR; Diagnostic and Statistical Manual of Mental Disorders, 4th ed., Text Revision; American Psychiatric Association, 2000) but not in the fifth edition (Diagnostic and Statistical Manual of Mental Disorders, 5th ed., DSM-5, American Psychiatric Association, 2013). In the DSM-IV, PCS requires an immediate onset and chronicity of at least 3 months. The International Classification of Diseases (ICD-10; WHO, 1993) does not include such limitations to the definition.

At the time of the reviews under discussion in 2017–2018, the newest edition of the ICD (ICD-11) had not yet been published. In that edition, concussion is defined as loss or diminution of consciousness due to injury (ICD-11, WHO, 2020). The codes in the NA07 category allow for options with diffuse, focal, unspecified, and hemorrhagic intracranial injuries.

As for the DSM-5, it includes a category of neurocognitive disorder, which can be specified as mild and also due to a TBI. The DSM-5 refers to quantifiable evidence, symptoms as associated features, and differential diagnoses, such as when symptoms do not follow a dose-response relationship. It has been argued that the DSM-5 has a category of major neurocognitive disorder, which presents difficulties to neuropsychologists because a moderate degree of neurocognitive disorder typically is shunted into the minor category, thereby reducing the impact of the injury in diagnostic terms, with psychological and legal consequences for the injured party (Schultz, 2010, commenting on the DSM-5 draft).

As for epidemiology and prevalence, the two articles under review indicate a large lifetime prevalence rate worldwide, and with 10 to 25% exhibiting persistence in post-concussive symptoms to the extent that they become a syndrome (PPCS). In 3 to 6 months, according to the cited reviews, toward 80 to 95% of MTBI patients will be resolved according to neuropsychological test performance. However, for some, the symptoms persist nevertheless, implying biopsychosocial causation in the symptom maintenance.

Comment

Although comprehensive in nature, the reviews by Polinder et al. (2018) and Mayer et al. (2017) do not consider whether in the prevalence research cases of feigning and malingering have been screened and excluded in the estimates. Indeed, Mayer et al. (2017) wrote that malingering frequency is "rare", and that poor effort, symptom over-elaboration, or both do not necessarily indicate malingering. This paper agrees with this conclusion because the full gamut of the interview, testing, and document/record analysis need to be considered in attributing malingering. That said, the rate of malingering and other problematic presentations in these types of assessments has been estimated at up to 30% or more (Young, 2015a), keeping in mind that the base rate for malingering, *per se*, within this category is much smaller, and about 10%, if not lower in some cited literature, according to careful review of the literature in Young (2015a).

Figure 1 presents one way of modeling the factors that are involved in MTBI/PPCS. It includes a role for exaggeration, feigning, and malingering and the like in problematic presentations. Note that the latter term does not conflate with malingering alone. For example, the MND assessment procedure (Malingered Neurocognitive Dysfunction; Slick, Sherman, & Iverson, 1999) had been used to combine probable and definite malingering into the one category of malingering in the research. This sets the stage for the same use in practice, with potentially biased consequences for the evaluee.

The 30 Points on MTBI/PPCS

Introduction

The literature review that has been conducted suggests multiple points that need to be considered in practice and research related to MTBI and PPCS. Here, the article enumerates 30 points in this regard, adding other points to give a complete picture of the unresolved issues in the field or the important ones to consider for assessment, diagnosis, and court considerations. The author organized them into four major themes: basics, influences/confounds, assessment, and law (see Table 1). The basics in MTBI/PPCS concern their definitions, diagnostics, prevalence, and symptoms, as well as etiology and long-term outcomes. The influences/confounds cover the full range of biopsychosocial factors both before and after onset, including therapies. The assessment points concern best practices in testing and seeking inconsistencies, including those that help in attributing malingering. The law point is the thirtieth in the list but is crucial for effective assessment for court purposes.

It should be pointed out that there is nothing magical about the 30 points underscored in the present roadmap for MTBI/PPCS. For example, sleep disturbance is a central driver of other symptoms in their prolongation, and can stand alone as a roadmap point. Also, substance abuse could be considered part of other confounds and not given a separate point status in the roadmap. Further, different comorbidities can be separated out to create additional road map points. However, for present purposes, the 30 points are considered essential and remain separate. Moreover, they can be split or added to in future work expanding and filling out with research the roadmap.

Finally, the literature review in the paper is expansive, but the reader will want certain points amplified with detailed literature reviews beyond the one provided. The length of the manuscript precludes adding further to the literature review already undertaken. Indeed, certain of the 30 points raised in the article require full-length article or chapter treatment.

In the following, the article cites some supportive research for the 30 points, but mostly explain them for practitioners, researchers, and the court and related venues. The explanations highlight not only research findings and practice recommendations in prior research (e.g., Young, 2014) but also indicate how to use the points in assessments and in court/related venues, if at all appropriate in these regards.

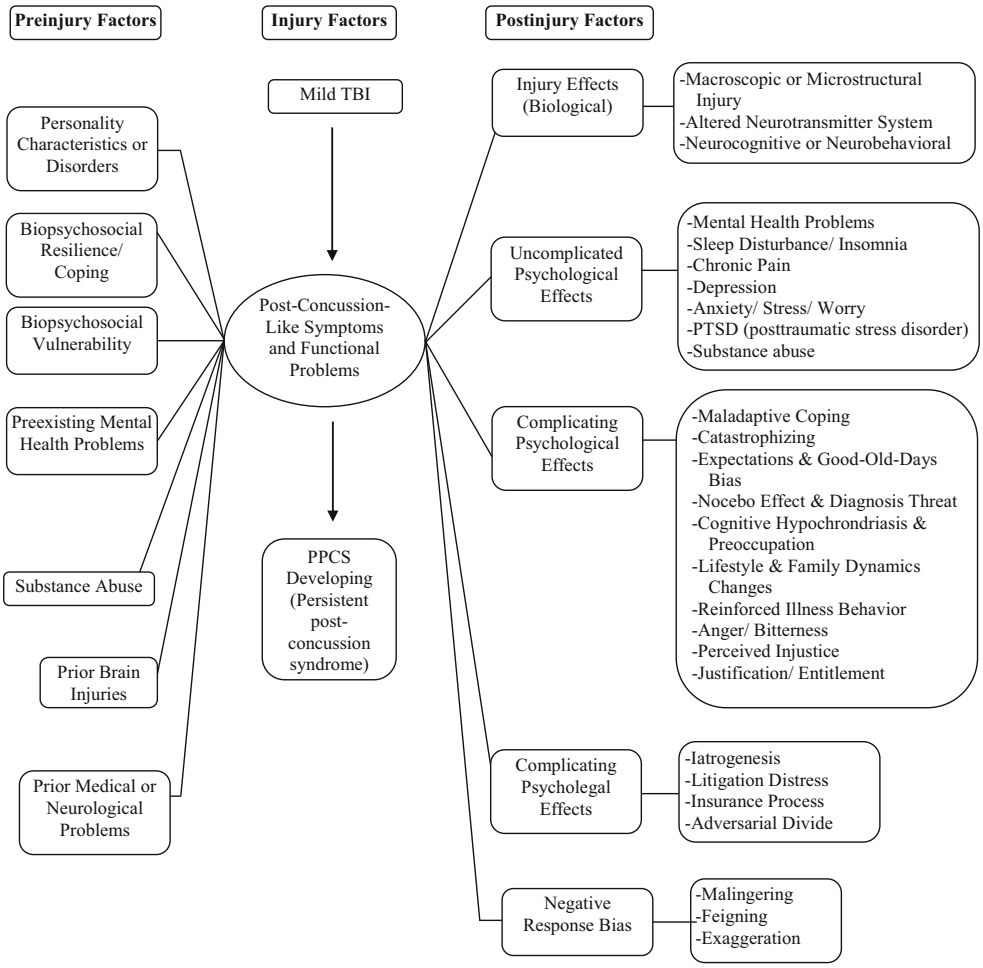

Fig. 1 A biopsychosocial conceptual model of poor outcome after mild traumatic brain injury. The original figure of their model of poor outcome from mild traumatic brain disorder (TBI) did not mention malingering, feigning, and exaggeration (negative response bias), nor did it include separately complicating legal effects (such as the role of litigation distress, iatrogenesis, the insurance process, and the adversarial (plaintiff-defense) divide). The original figure has been altered to put distal preinjury factors to the left, injury factors medially, and the post-injury factor to the right. Adapted from Young (2016)

Explanations

Basics in MTBI/PPCS

Terms (1) What are basic terms in the neuropsychological field (there is a lack of consensus)? What are the diagnostic symptoms? Are any markers that help in specificity? These types of questions constitute the fundamentals in the area of MTBI/PPCS. Gasquoine (2020) presented a useful review of the evolution of terminology in the field. Mayer et al. (2017) presented a spectrum of ideal terms that are separated and hopefully will be amenable to finding valid, differentiating biomarkers. This approach needs to be applied to the full range of terms in the field. Diagnostic manuals take different approaches to establishing nosologies. The DSMs (Diagnostic and Statistical Manuals) use an overlapping approach without listing unique symptoms for each disorder. The ICDs (International Classification of Diseases) seem more constricted in the range of symptoms in their lists, with the different approaches in the manuals to PTSD serving as a good example (21 symptoms vs. 6, respectively). The ICDs give a minimum definition of concussion, for example related to a possible LOC. Nevertheless, the latter approach of reduced symptomology does not provide unique marker for each disorder in the manual.

For MTBI/PPCS, there are many common symptoms that cut across many medical and psychological conditions, and they are vague, as well. Therefore, part of the difficulty in establishing terminology in the field relates to establishing the approach to creating terms, which is itself an outstanding issue, as this discussion indicates. The plethora of terms in the area constitutes another confound in these regards. Perhaps a central scheme is needed, such as being more proximal to the injury and more-symptom focused, and being more distal and conceptual, requiring neurological and neuropsychological investigations/tests and determinations.

Definitions (2) What are all relevant definitions not only for MTBI and PPCS, but also for other terms? What are the exact components of the latter two terms, e.g., post-traumatic amnesia (PTA), loss of consciousness (LOC)? The first major and still classic definition of MTBI was developed by the American Congress of Rehabilitation Medicine (ACRM) in 1993 (Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine, 1993).

Table 1 Thirty points on MTBI/PPCS in psychological injury and law

Number	Point	Description
Basics in MTBI/PPCS		
1	Terms	Differences among subconcussive blow, repetitive head injury, head injury, closed head injury, acquired brain injury, concussion, alteration of consciousness, loss of consciousness, orientation × 3; MTBI, complex MTBI, PCS, PPCS, chronic traumatic encephalopathy
2	Definitions	Differences in different definitions of MTBI
3	Classifications	Differences in classification in the DSMs and the ICDs
4	Prevalence	Variations and their validity in MTBI
5	Cognitive symptoms	Acute/immediate cognitive deficits and their typical duration in MTBI
6	Emotion/social symptoms	Acute/immediate emotional and social deficits and their typical duration in MTBI
7	Physical/bodily symptoms	Acute/immediate neurovegetative deficits and their typical duration in MTBI
8	Etiology	Mechanisms in MTBI: coup/contra coup, diffuse axonal injury/damage, etc.; predisposing, precipitating, propagating factors; stress-diathesis model; biopsychosocial model
9	Recovery	Mechanisms and variations in recovery
10	PPCS	Long-term chronic outcomes, the miserable minority—true MTBI or biopsychosocial influences?
11	Outcome	Long-term residua in MTBI related to processing speed and white matter loss?
12	Validity	How much of a significant empirical difference in MTBI research is clinically significant?
Influences and confounds in MTBI/PPCS		
13	Prior state	Influence of prior state; can they explain everything about the MTBI symptoms
14	Confounders	Influence of confounders, e.g., reaction to medicines or anything else that can give the same symptoms
15	Cognitive dynamics	Influence of cognitive dynamics, exacerbatory beliefs in MTBI
16	Social dynamics	Influence of social dynamics, family, support, community
17	Comorbidities	Influence of comorbidities that complicate, e.g., depression, PTSD
18	Exacerbators	Propagating and maintaining factors
19	Deactivator/protective factors	Mitigating and buttressing factors
20	Substance abuse	The role of alcohol and illicit drugs
21	Treatment	Best treatment regime for MTBI
Assessment		
22	Scans	Best scan techniques for MTBI; their validity in court
23	Data predictors	Best empirical/scale predictor measures of MTBI outcome
24	Real-life predictors	Best real-life predictor measures of MTBI outcome
25	Test predictors	Best neuropsychological measures of MTBI outcome
26	Malingering	Best indicators of exaggerated MTBI, feigned MTBI, malingered MTBI
27	Neuropsychological tests	Best neuropsychological tests and measures for MTBI
28	Scales	Best scales to use in MTBI assessment
29	Inconsistencies	Are there inconsistencies in the record, behavior, and symptoms?
Law		
30	Court	MTBI in court; recent case law

MTBI: mild traumatic brain injury, *PPCS*: persistent post-concussion syndrome

Because it still is used preferentially in court and related legal settings, here, the article notes its major components, with one or more having to be present. It refers to a traumatic induction of brain function. Note that this term is vague, e.g., not

specifying a physiological disruption, the application of an external mechanical force, and the like, the relatively minor nature of the blow to the head, the possibility that acceleration/deceleration forces might be involved rather than a blow to the head, and so on. The definition allows for any period of loss of consciousness, although qualified as 30 min maximum. Note that this term is vague because some approaches to MTBI definitions require its presence. The ACRM definition refers to loss of memory either before or after the event, with the loss happening immediately. This aspect of the definition is vague in its time frames, at least for pre-injury amnesia (in the definition, post-traumatic amnesia should not be greater than 24 h). The fourth criterion of MTBI in this definitional approach is that there might be focal (transient) neurological signs. This definition is vague because self-report by the injured party does not equate with a neurological examination. A fifth criterion relates to Glasgow Coma Scale (GCS) score, which must be between 13 and 15 out of a total possible score of 15 within the first 30 min after the injury. This criterion is vague when trained assessors cannot establish the GCS score within the first 30 min. Other aspects that are vague in the definition of MTBI concern the degree of late onset allowed for the diagnosis to be valid. Finally, symptoms are distinguished from the medical/physiological event that had taken place, and they fall into the categories of physical, cognitive, and behavioral/emotional changes. In the present approach, as per below, neurological/sensorial type symptoms are included with the physical ones and social type symptoms are included with the behavioral/emotional ones. Any definition of MTBI has the usual disclaimers that its signs and symptoms cannot be accounted for by other factors or conditions, that they have to be clearly linked to the initiating event, and that they can lead to functional impairments.

The Management of Concussion/mTBI Working Group (2009) definition of MTBI is quite similar to the ACRM definition, but it adds that, at least for any degree of severity of a TBI, neurological signs include physical weakness, loss of balance, changes in the visual modality, praxis deficits, paresis/plegia, sensory loss, aphasia, etc. Also, it specifies that alteration of consciousness (AOC) can stretch out to 24 h. It includes being dazed, being uncertain, being confused, having difficulty in thinking clearly, or responding appropriately to questions on mental status.

Ruff, Iverson, Barth, Bush, Broshek, and the NAN Policy and Planning Committee (2009) compared the ACRM definition of MTBI with that of the World Health Organization (WHO; Carroll, Cassidy, Holm, Kraus, & Coronado, 2004). They noted that the latter omitted the word "dazed" in reference to MTBI and the former indicated that neurological abnormalities may not be transient, unlike the case for the ACRM. Ruff, Iverson, Barth, Bush, and Broshek (2009) provide guidelines/recommendations for the proper diagnosis of the vague terminology in both definitions. This includes how to evaluate the GCS score absent any professional being present in the 30 min time frame required. In this regard, the GCS score can be assigned in a time frame after 30 min according to Carroll et al. (2004).

Silverberg, Iverson, et al. (2020) described that the ACRM definition of MTBI is being revised. They noted that a lack of a universally accepted definition of MTBI in the field hinders both research and practice. They conducted an international cross-sectional web-based survey of experts in the field toward the revision. They found that objective signs were rated as more valuable in diagnosing MTBI than subjective ones; that said, acute subjective symptoms are diagnostically important. Of the signs, fatigue was rated as the least helpful. Cognitive and balance impairments were deemed the most helpful, and the full list of most helpful symptoms in the diagnosis included LOC, disorientation, confusion, feeling dazed, balance problems, memory difficulties, dizziness, and inappropriate behavior. Note that feeling dazed was considered among the most useful diagnostic signs, in contrast to the previously mentioned exclusion of this sign by the WHO in its definition. Further, diagnoses should consider the symptoms in the first 3 days after the injury. Finally, the diagnostic process should be probabilistic, with disproportionate weightings of diagnostic features.

Mayer et al. (2017) included a table with the multiple attempts to define MTBI, and there is no clear consensus. There are variations in symptoms, which are cardinal, and which are associated features. In future work in this area toward structuring reliably and validly those critical symptoms related to MTBI/PPCS, they specified symptoms should be organized into a hierarchical structure. As well, which core ones could be potential biomarkers should be indicated. The field also should specify how the specific symptoms had been derived, for example, by (a) self-report, (b) collateral nonprofessional sources, (c) collateral professional sources, (d) file/record/document review, (e) observation, (f) qualitative data, (g) quantitative data, (h) neuropsychological assessment/testing, and/or (i) neurological assessment/scanning/investigation.

Establishing symptom networks is critical to understanding symptoms in MTBI/PPCS. The classic factorial approach to grouping statistics into ensembles that reflect putative underlying latent variables is being challenged and replaced by approaches that deny causal validity to such underlying latent variables. Network approaches qualify, instead, that the drivers of symptom expression reside in the dynamics of symptom interplay and their (reciprocal) causal effects (Borsboom, 2008; Young, 2015b). For example, poor sleep could reside centrally in a cluster of symptoms (nodes) having links (edges) to each other that constitutes a "small world" or tightly linked cluster. It would be important to know whether symptom clusters resemble the typical split into cognitive, socio-affective, and sensorial-central clusters, or are there overlaps across these categories. Moreover, the network-

based clusters might relate differentially to pre-event and precipitating causal factors and to propagating ones and to equivalent networks in the brain.

With different definitions of MTBI and related terms, both research and practice are hampered in arriving at reliable and valid findings. At the research level, scientists need to find and use the best definitions in their investigations and justify their use in their publications, so that a better consensus is built on the definitions. This will have throughput effects into practice assessments and court deliberations on acceptable science on the matter.

Toward resolving the definitional conundrums related to defining critical terms in the area, here, the paper provides a comprehensive, new definition of MTBI, one partly based on the cited sources. This definition adds the degree of the physiological impact involved, unlike other definitions, and acknowledges that the degree of physical impact does not dictate the diagnosis. It considers also the usual qualifiers. To the traditional approaches to the definition, here, the paper adds the subjective aspect and its indeterminate/nonspecific nature (lack of (bio)markers). This definition is not meant to supplant others but to consider in the upcoming changes to the ACRM definition, as discussed above (Silverberg, Iverson, et al., 2020).

Given these considerations, *mild traumatic brain injury* (*MTBI*) can be defined as a relatively minor physiological disruption of brain function due to an external mechanical force to the head (which more than likely will be relatively minor). The MTBI is manifested by one or more of the following: (1) any period of LOC (loss of consciousness); (2) any memory loss for events immediately preceding or post the index injury: (3) any alteration in mental state just after the injury (e.g., feeling dazed/disoriented/confused); and (4) focal neurological deficit(s), whether transient or not. These subjective symptoms relate to physical, emotional, cognitive, and (apparent) neurological symptoms, but none as yet have been shown to be (bio)markers (partly after Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine, 1993; Denning & Shura, 2019; McInnes, Friesen, MacKenzie, Westwood, & Boe, 2017).

Here, the paper took the same approach to defining *persistent post-concussion syndrome* (*PPCS*): a chronic condition in which PCS symptoms persist beyond 3 months. The symptoms are subjective, with no (bio)markers; concern physical, emotional, cognitive, and (apparent) neurological symptoms; and are, to date, considered sequelae of psychosocial factors propagating and maintaining the symptoms after the initial initiating event (partly after McInnes et al., 2017; Rickards, Cranston, & McWhorter, 2020).

Classifications (3) What are the different definitions in the DSM-IV, DSM-5, ICD-10, and ICD-11? Professional organizations, especially in neurology, have established nosological systems related to acquired brain injury and their effects (Mayer et al., 2017). Without a unified approach, both research and practice will find it difficult to develop proper research tools and determine reliable and valid prevalence estimates for MTBI/PPCS. The different approaches encouraged diversity in thought and continued reflection, but certain components should be considered essential and common to different nosologies, such as inclusion and exclusion criteria, what constitutes the acute stage and the chronic stage, whether neuropsychological and neurological investigations are required, and so on. Beyond that, the DSMs use a polythetic approach to classification, the ICDs more a prototypical one; the DSMs use longer lists of symptoms, the ICD shorter lists; the DSMs have been considered to lower the bar in diagnosis, the ICDs less so, etc. (Young, 2016). A common diagnostic approach for MTBI/PPCS should consider these different approaches to nosology and emerge with a system with both clinical utility and research applicability.

Prevalence (4) Prevalence estimates vary between the teens and the 80s (Polinder et al., 2018); why and what is valid? For work in epidemiology/prevalence rates, the nature of the research undertaken is critical in establishing the rates found. First, epidemiological research is population-based, and the samples should be representative of the population. When there are gaps in groups used in the research, the prevalence estimates will be biased (Young, 2020). Second, a common sample recruiting strategy in research on MTBI/PPCS uses convenience samples. By definition, samples such as these are biased by the referral sources for the practitioners involved. This strategy has attendant risks of a different nature, for example, relating to the adversarial, or plaintiff-defense, divide. Third, prevalence estimates need to be specified for lifetime and point (e.g., year) rates, which are quite different. Fourth, whatever the sample used in prevalence estimates, the different methodologies used to determine MTBI/PPCS symptoms can disproportionately tilt the estimates to the lower end or higher end. The most reliable and valid, as well as commonly accepted, methods and tools need to be used in this type of research in order to obtain realistic prevalence estimates. There are psychometric consequences in obtaining prevalence estimates; for example, calculation of test specificities and sensitivities depend, in part, on base rates as determined by epidemiological/prevalence research in these regards (e.g., Young, 2014).

Cognition Symptoms (5) This refers to confusion, taking longer to think/speed of processing, dazed/stunned/disoriented/feeling foggy, memory problems, concentration problems, attention problems, executive function problems, perceptual problems, speech/language problems/slurring, judgment affected, etc. MTBI can induce pre-trauma retrograde amnesia as well as post-traumatic anterograde amnesia (PTA). The

primary locus of MTBI/PPCS is related to cognitive problems, which begin immediately or right after awakening from a LOC. The person feels confused, dazed, and disoriented, often referred to as being in shock, and cannot think clearly through whatever problem is at hand, for example, related to safety. Memory issues relate to the inability to recall the events immediately preceding the trauma, the trauma itself, and post-traumatic events. Memory is considered re-established in the present, at least for immediate and post-event recall, when there is no more waxing and waning of memory, going in and out of LOC, and so on, such that the person can recall more or less at an acceptable level the events of the day with consecutive memory after the event at issue/emerging from a confused state, or from LOC, although the person will report, too, that everything was a blur and the like.

As the MTBI continues into the next days, weeks, and months, the ability to focus, use working memory, and so on remains compromised in terms of concentration; short-term memory; attention, e.g., sustained attention, distractibility/mind wandering, persistence/pace/staying on track; etc. Problem-solving and deploying executive function toward problem-solving in daily life (i.e., inhibition skills, shifting set, working memory) could take a while to re-establish and lead to deficits in planning/organization, decision-making, and engaging effectively in daily functions, such as home care, parenting, studying, work, and partner and social relations. The person might claim long-term memory issues, but there is little in the research to support any noticeable direct effects on memory from MTBI along these lines (McInnes et al., 2017).

Emotions/Social Symptoms (6) This refers to (a) worry/anxiety, preoccupation with symptoms/anxiety sensitivity, depression/sadness, irritability/quickness to anger/aggression/frustration, bitterness, fear, etc.; and to (b) poor emotional regulation/responsivity/lability/disinhibition/impulsivity/behavior problems/restlessness/hyperactivity/agitation, personality disturbance, inappropriate behavior, affected self-confidence/self-worth; and to (c) interference in social skills/social cognition, etc. MTBI can induce changes in all the major negative emotions, from anxiety to depression to irritability to fear. The clinician might decide to diagnose a comorbid condition such as MDD (major depressive disorder), GAD (generalized anxiety disorder), PTSD (post-traumatic stress disorder), and so on, depending on the extent of the emotional symptoms and social disruptions. The emotion/social symptoms can get quite complex, for example leading to poor self-control and self-regulatory skills emotionally, socially, verbally, motorically, and cognitively. The personality will seem disturbed, and latent personality disorder characteristics might be exacerbated, or they might arise seemingly de novo or out of character. Ultimately, functionality is affected, and the person cannot engage with equilibrium and effectiveness in social tasks and interactions, which might lead to self-awareness of the deficits and loss of personal esteem and self-confidence, aggravating anxiety, depression, and other negative emotions.

Physical/Bodily Symptoms (7) This refers to (a) headaches, pain, poor sleep, fatigue/apathy/lethargy, arousal/stress responses, shock, panics, dizziness/light-headedness/vertigo, loss of balance/unsteady gait/motor incoordination, and nausea/vomiting; (b) visual disturbance (blurriness, double vision, light sensitivity; difficulties in smooth pursuits, saccades, convergence), auditory disturbance/noise intolerance/sensitivity, and tinnitus; and (c) (transient) neurological abnormalities/seizures, sensitivity to alcohol, etc. The physical/bodily symptoms associated with MTBI are divided into (a) neurovegetative symptoms, (b) soft sensorium ones, and (c) hard neurological ones. The first category represents common and vague symptoms that are not specific or unique to MTBI/PPCS. That state of affairs confounds their diagnosis and differentiation from other disorders and syndromes. The second category is particular to brain-based symptomatology, but not necessarily in a biomarker manner, or unique to TBI. For example, they might accompany migraine headaches or anoxia from multiple conditions, including panic. The third category of symptoms is more directly associated with TBI and it requires neurological assessment for determining their status. For example, there might be pseudo-seizures involved rather than investigation-confirmed seizures.

Etiology (8) This refers to coup contrecoup, DAI, white matter effects; the frontal lobe as a locus, etc.; petechial hemorrhages, neuroinflammation, altered cerebral blood flow, altered neural connectivities, macroscopic or microstructural injury, and altered neurotransmitter system; and physiological/metabolic cascade; what is the etiological pathway or neurobiological cascade from a physical impact to the head to PCS; and are there biomarkers or psychological markers (or their test score representations) that have the required sensitivity and specificity. Etiology and mechanisms in MTBI are important to establish in the research, but nomothetic research evidence cannot be applied directly to an idiographic, individual case. The assessor can determine how the head was struck in the event at issue, the likely effects in a coup contrecoup rebound effect, the loci of the brain regions possibly involved, and indicate that usually there are transfers through kinetic energy and rotational effects to the frontal lobe because of the bony protuberances in the brow region, and so on. However, nothing definitive can be made based on the research profile of the typical MTBI victim for the case at hand. That said, the research on etiology and mechanisms in TBI is important and should continue in earnest because the court might opine out of ignorance or bias that

the symptoms of MTBI were all psychological, to begin with; they were all in the head or mind or psychology of the person; they were all non-organic, psychogenic, functional, vague, and non-compensable; and so on. Granted, the article has raised validity issues about PPCS, but this should not negate the train of events neurophysiologically that takes place during and just after the event at issue, which together help understand the initial symptoms and why they might persist, propagate, and stabilize even permanently in some victims. Ultimately, the quest for understanding etiological mechanisms in MTBI/PPCS should trace the cascade of physiological/metabolic activities from neuron, to the central nervous system, to intra-regional and intra-regional neuroconnectivities, and to behavior and its organization after the event at issue. Also, it should aim to find the unique biomarkers in the cascades at each level, if any.

The central nervous system (CNS) typically is underscored as the locus of MTBI impacts and consequent effects. However, the notions of contributory neuroinflammation and stress responses and the like indicate that the autonomic nervous system (ANS) is intimately tied to the CNS in mediating the MTBI response to the external mechanical force applied to the head (and brain). This opens the understanding of MTBI/PPCS to its understanding as a psycho-neuro-immunological process. To what extent are there the twin processes in the traumatic response of adrenaline and cortisol release that exacerbate the CNS effects, and how do individual differences in the peritraumatic response affect the successive metabolic-physiological cascade that underwrites the behavioral symptoms that manifest?

There are complications to be aware of in a research program such as this one. For example, there will be no one cascade that characterizes all MTBI/PPCS. Similarly, there will be individual differences in trajectory profiles, ranging from rapid recovery to persistent chronicity, and the latter might have differential emphases on cognitive, socio-affective, and physical/bodily symptomatology. Third, markers in psychiatric disorders might not be biological but psychological or even social. For example, the sensorial symptoms in MTBI might be the predominant ones, or the cognitive ones might predominate in this regard, and even then be different from one person to the next, or test results that represent behavioral and central impacts might be what stands out in determining endophenotypes in MTBI/PPCS. The latter might relate to pre-event factors, such as extreme pre-existing traumas, abuse, and psychiatric vulnerabilities and conditions.

Finally, in the etiological/mechanistic processes that govern or associate with MTBI/PPCS, both researchers and practitioners need to be aware of individual differences related to gender, minority status, age, SES (socioeconomic status) strata, and other demographic variables because, ultimately, the field needs to understand the factors that promote vicious circles in MTBI/PPCS and how to mitigate/control and resolve them. There is no one formula that fits all.

In the end, more integrative models of the etiological process in all psychological injuries (sequelae of MTBI, PTSD, chronic pain) need to be adopted toward better understanding them and elucidating the pathway in each individual from onset to outcome (Young, 2014). Classic models in this regard relate to stress-diathesis, biopsychosocial, and predisposing, precipitating, and propagating factors. In court, the question always arises to what extent the predisposing or psychosocial factors can fully explain the presenting condition, especially if it includes functional impairments and disability, leading to compensable claims. Moreover, ruling out of malingering is critical to even considering etiological options such as these.

MTBI/PPCS are described in multifactorial terms (Prince & Bruhns, 2017) and, after the precipitating event, a host of psychosocial factors can exacerbate, propagate, and maintain it in negative "dysfunctional" feedback loops. Furthermore, there might be a range of biological (including genetic factors and prior MTBIs), psychological, and social factors that precede it and, together, constitute viable and valid predisposing factors that can either explain ongoing symptom presentation either in full or in part. The event is the "stressor," and the pre-event psychological status or pre-existing vulnerability/psychopathology is the "diathesis." The "neurogenic" precipitation is displaced by the "psychogenic" continuing etiology in the causation of any continuing symptomatology due to the MTBI. In these scenarios, the typical post-event course of full resolution does not take place, which happens in most cases of MTBI (80–90%), thus precluding development of PPCS.

Biopsychosocial models of MTBI PPCS should cover the biological (e.g., O'Reilly and Tom (2020) on neuroinflammation; Hoffman and Taylor (2019) on stress reactivity), and the psychosocial (e.g., Faulkner et al. (2020) on psychological inflexibility; Stenberg et al. (2020), Teodoro, Edwards, and Isaacs (2018) on factors that increase bodily focus; Anderson and Fitzgerald (2018) on problematic coping and illness perceptions). Together, factors such as these at play in MTBI/PPCS work together physiologically and psychologically to underwrite the expressed symptoms (van der Horn et al., 2019), with the biological factors being precipitants and the psychological processes being perpetuating and maintaining factors. As these authors argued, an interaction takes place among original cellular injury, inflammation, and stress, and the mediation by pre-injury style of coping, personality variations, and so on.

MTBI symptoms persist in some cases, and some of them for many years. Generally, the studies involved do not screen for the host of confounding factors that might be involved other than the original injury. Their participants vary in characteristics, as well, and the measures used vary, making cross-study generalizations difficult. That said, Carroll et al. (2020) reported a prolonged growth curve in recovery for some

patients (28%) into 2 years. Some of the factors involved included executive function measures, harmful drinking, depression, poor sleep, and headaches. Roy et al. (2019) found some predictive value in persistence especially with initial LOC, but not after 3 months post-injury. Rona, Jones, Jones, Fear, and Wessely (2020) found a 7-year persistence in MTBI onset of dizziness and loss of concentration. Theadom, Starkey, Barker-Collo, Jones, Ameratunga, and Feigin (2018) found a 4-year effect of MTBI on residual self-reported cognitive symptoms, which appeared to impact community participation, relative to controls. Regression analyses that controlled relevant co-variates implicated prior TBIs, medical factors and baseline variables beyond the cognitive, including for depression and anxiety, in explaining the variance in self-reported cognitive symptoms 4 years post-injury. Stubbs et al. (2020) evaluated patients with "prolonged recovery from their MTBIs, and found that somatization symptoms not associated with MTBI distinguished the patients (gastrointestinal upset, musculoskeletal complaint, cardiovascular complaint). This reminds of Young's (2008) multifactorial, biopsychosocial model of somatization, which has 100 possible factors involved.

The most recent work presenting a biopsychosocial model of MTBI/PPCS is by Iverson (2019). He embeds his biopsychosocial model in a network and systems perspective, much like Young (2015b, 2021) in his general approach to psychological causality. The nested complex system levels refer to the intra-cellular, neuronal/brain network, psychological/experiential, and social (Kenzie et al. (2017)). In keeping with an integrated approach to MTBI modeling, Stenberg et al. (2020) found that somatic and emotional factors are associated with self-reported cognitive symptoms in MTBI but not cognitive performance changes. Theadom et al. (2018) found for MTBI, among others, a history of pre-existing TBI a factor in symptomatology 4 years post-injury. van der Naalt et al. (2017) referred pre-injury health problems, education level, and age as early predictors, among others, for possible 6-month recovery trajectories after MTBI. Silverberg et al. (2015) referred to a multivariate model in predicting outcome in MTBI, and included factors such as pre-injury mental health, in this regard. Pre-injury factors in MTBI were studied as a major factor by Taylor and Seebeck (2019) and by Voormolen et al. (2020). Perceptions of symptoms, resilience, self-efficacy, etc. have effects on symptoms in MTBI (e.g., respectively, Mah, Hickling, and Reed (2018), Vos, Poritz, Ngan, Leon-Novelo, and Sherer (2019), and the three articles by Belanger et al. (2020), Scheenen, van der Horn, de Koning, van der Naalt, and Spikman (2017), and Yehene, Lichtenstern, Harel, Druckman, and Sacher (2020)). The social aspects of MTBI are considered in Bannon, Greenberg, Goldson, O'Leary, and Vranceanu (2020). These cited articles give only a flavor of the factors impinging on MTBI symptomology that can be considered biopsychosocial in nature.

Returning to the network model of Iverson (2019), he concluded that a network approach to MTBI/PPCS might help identify multiple syndromes or clusters of prominent symptoms, or subgroups of persons having the MTBI/PPCS. Iverson does not elaborate on this suggestion, but growth curve research over time might help specify the types of subgroups in MTBI/PPCS, even among those with PPCS after many years. If pre-injury factors are taken into account along with other biopsychosocial variables, the subgroups might vary in their multiple system level and their biopsychosocial specifics. (Also see Broshek, De Marco, and Freeman (2014) for modeling on PPCS that includes psychosocial variables.)

Other approaches to subcategories of populations in MTBI have not been sufficiently effective in arriving at defined subgroups for diagnostic purposes (Theadom, Barker-Collo, Greemwood, Parmar, Jones, Starkey, McPherson, Feigin, on behalf of the BIONIC Study Group (2017)). Lumba-Brown et al. (2019) argued that those with oculo-motor, vestibular, and cervical-strain symptomatology might constitute separate subtypes. Lumba-Brown, Teramoto, Bloom, Brody, Chesnutt, Clugston, Collins, Gioia, Kontos, Lal, Sills, and Ghajar (2020) referred to headache/migraine, cognitive, vestibular, anxiety/mood, oculo-motor, and vestibular subtypes being possible at 3 days post injury. Agtarap et al. (2020) referred to stable factors of "general" and emotional/cognitive/visual on the Rivermead Questionnaire 1-year post-injury. Broadway et al. (2019) studied delayed memory deficits in MTBI, finding executive function and semantic clustering deficits as predictors.

This latter approach illustrates the multiple level approach, finding mediating factors in MTBI symptomatology. These various attempts to differentiate variability in course after MTBI vary with measure, population, duration post-injury, and so on, but they suggest avenues for the biopsychosocial/network/systems approach. The latter approach implicates a multilevel data set, from genes/epigenetic factors (e.g., Johnson, Lundgren, Henrich, and Phillips (2020), Bertogliat, Morris-Blanco, and Vemuganti (2020), respectively) to possible biomarkers to cellular/neuronal/network and brain function, and to psychosocial variables, that is not present in the various studies cited. Moreover, the degree to which pre-existing factors are included in the data sets used is not inclusive, as it should be. Perhaps all trajectories post-injury onset will have associations with pre-existing factors one way or another, as well as with other confounds.

The field of research in MTBI/PPCS is heavily biologically and brain oriented. The question becomes to what extent can the findings be replicated for long-term cases and, if so, actually help explain residual deficits in these cases. That said, a brief survey of this biological/brain component in MTBI/PPCS cannot be dismissed by indicating simply that biological factors inevitably drop out in explaining persistent symptoms. For research on autonomic nervous system dysfunction

along these lines, see Purkayastha, Stokes, and Bell (2019). They related this dysfunction to difficulties in sleep, with headaches, and to cognitive and emotional symptoms. As for the CNS, white matter microstructure and tracts have been implicated (Patel, Wilson, Oakes, Santhanam, & Weaver, 2020; Sorg et al., 2020). A role for the prefrontal cortex emerges in the research by Dall'Acqua et al. (2017). Biagianti, Stocchetti, Brambilla, and Van Vleet (2020) implicate the decreased perfusion along white matter tracts, and reduced connectivities within several resting-state networks. Other research on functional connectivities in functional networks include the research by Dailey, Smith, Vanuk, Raikes, and Killgore (2018) and by Li, Lu, Chen, Wang, Zhang, Chen, and Yin (2020), Papadaki et al. (2020) referred to cerebral perfusion deficits in the left anterior cingulate gyrus. Also, see Coyle, Ponsford, and Hoy (2018). For forensic issues related to the brain in court, see Bertozzi et al. (2020), and Shenton, Price, Levin, and Edersheim (2018). Despite the promise, caution is recommended in these regards.

Workers have attempted to find common mechanisms underlying all MTBI/PPCS symptomatology, and the biopsychosocial model pretty much covers many of them. Workers might emphasize one or the other among the compendium of factors associated with each of the biological, psychological, and social factors involved. More likely, they will refer to the multifactorial and systemic nature of the factors holistically. However, Young (2021) has proposed a general causal factor that cuts across neuron, brain, and behavior, and that he integrated into his biopsychosocial/network/systems model of behavioral causality. The concept originated in his work on manual lateralization in infancy, in which he found in 1-month-olds that the right hand is better coordinated than the left hand in activating the reach and in inhibiting interference during the reach. He characterized the coordination involved as activation-inhibition coordination and posited that the left hemisphere is specialized beginning from prenatal development and across the lifespan for this function, in contrast to outright inhibition specialized in the right hemisphere. This generalized skills can help explain the left hemisphere's verbal and motor functions, given the complexity involved in these behaviors.

The research on inhibition deficits in persistent cases of MTBI supports this model, although not directly. For example, Xu et al. (2017) referred to deficits in inhibitory control in the post-acute phase of MTBI. The inhibition related to harder inhibitory tasks involving occasional response conflicts (go/no go tasks). The results showed altered neural activity by event-related fMRI, involving the cerebellar-thalamo-cortical and the frontal-basal-ganglia networks, which are involved in regulating inhibitory control. Control measures in the study related to attention, and they did not differentiate groups. Sample size was small, and the results should be replicated with larger samples. Also, deficits in response inhibition have been found in other research in MTBI, but the findings are not necessarily replicated (Holiday et al., 2020).

Activation-inhibition coordination is referred to indirectly at the physiological levels in MTBI research. Yasen, Lim, Weymann, and Christie (2020) referred to altered excitability and inhibition in the neurotransmitter system of the motor cortex in symptomatic MTBI cases. Specifically, they took measures of cortico-spinal excitability and found lower activity in symptomatic cases. However, their inhibitory measures yielded no relevant group differences. Nagappan, Chen, and Wang (2020) referred to inhibitory brakes and facilitators at the cellular level in the parasympathetic nervous system, and proposed equivalent factors in the central nervous system.

The concept of activation-inhibition coordination might help explain different groupings of MTBI patients in the long term. Possibly, different groups have different incoming problems in activation-inhibition coordination, different compromised integrity of the function with the injury, and different post-event trajectories characterized by different underpinnings in activation-inhibition coordination. From a multilevel systems perspective, the proposed activation-inhibition coordination function might help explain in a common way across the levels of the person's system any and all difficulties experienced in long-term MTBI/PPCS. Is coping compromised in terms of that capacity, for example? Are social skills hindered because of lack of coordination in these regards? Are behavioral/emotional difficulties, including in impulsivity and disinhibition, reflective of deficits along these lines? Does recovery, in contrast, take place because of return to better functioning in deploying activation-inhibition coordination skills and capacities over multiple levels of the biological/psychological/social system?

Recovery (9) What is the recovery pathway and its variations that lead to the resolution of MTBI/PPCS and the problems in regulating the neurobiological cascade in that recovery process? As much as we need to know the etiology and mechanisms behind MTBI/PPCS and the multiple trajectories and individual differences therein, the field needs to establish the mechanisms that underwrite the improvement and recovery process. Granted, there are factors that will be emphasized, such as taking time, but exactly what is the underlying reverse process to the etiological mechanisms in such profiles? Do the biophysiological cascades simply dissipate, or reverse engineer, and can interventions speed up the process (or delay it if inappropriate/non-scientific)?

As a side note, although etiological mechanisms in MTBI and PPCS involve physio-neurobiological mechanisms to a degree, and the same applies to the improvement/recovery process, it is not appropriate to indicate that these biological processes, and the impacts on the brain, if any, are "responsible" for the symptoms and syndromes. They play a factor, and, for some case, critical ones, but they need to be seen as part of the larger biopsychosocial causation that typically

characterizes any type of behavioral disturbance or disorder, especially where the brain and central nervous system are implicated (Young, 2014). It is important not to isolate biological factors from psychosocial ones, context, environment, development, etc., in causality analysis, and, in parallel, in assessments, investigations, interventions, and treatments.

PPCS (10) Is PPCSv valid psychologically, neuropsychologically, and neurologically? Or is it vague, unreliable, invalid, a MUS, conversion disorder, factitious, disorder, somatic symptom disorder, the product of an original valid biological injury for which psychosocial factors propagate it after the biological injury heals, etc.? The roadmap described to this point in the article has intimated that PPCS is problematic, and in this tenth of the 30 points under discussion in this regard, the paper proposes that PPCS is highly problematic and even should be removed from the lexicon in the field, as per the recommendations that end the paper. The literature reviews by Polinder et al. (2018), and Mayer et al. (2017) that were summarized at the outset of this paper did not go that far, but they did recommend further research on the questions of symptoms, diagnosis, classifications, and so on, given the lacks in the field, and queried whether alternate explanations for alleged PPCS can play a role in individual cases, such as conversion disorder. The biopsychosocial perspective (Young, 2014, 2019) is a general one that can apply to any symptom, psychological disturbance, or psychiatric disorder. It is transdiagnostic and refers equally to causation, symptom expression, and treatment. It implies in the rehabilitation context that ignoring one component of the triad will increase the probability that interventions/treatments will not be as effective or will not work. It does not suggest that all three components have equal roles in any symptom, syndrome, or disorder, but all three should be accounted for, monitored, etc. In this regard, a simple bad habit that had developed in a biologically intact person might not have relevant biological factors to consider, aside from understanding how relearning, extinction, cognitive behavioral therapy, or any other type of treatment has corresponding biological indices. But for complex cases involving alleged PPCS, medically unexplained symptoms (MUS), conversion and factitious disorder, or any somatization condition or disorder, the biological component in the biopsychosocial formula that applies to the causation, symptom expression, and treatment appears more relevant.

However, there could be exacerbations in these regards from psychosocial factors; the biological factors can dissipate in ongoing maintenance and propagation, or even disappear in the condition at issue, and so on. The paper argues that the latter trajectory is the one most likely to be valid for PPCS. It may start off as uniquely biological due to the mechanical forces applied to the head by an external impact. Then, other factors in the biopsychosocial complex take over and are responsible for the propagation and maintenance of the syndrome in chronicity and disability. In these regards, PPCS should be removed from the lexicon in the field. Many labels in psychiatry and neurology are no longer included in the diagnostic lexicon, and confusing ones, in particular, are discarded. The same should apply to PPCS.

Granted, there will be cases in which classic PPCS symptoms are produced, and there does not appear to be confounds from heightened symptom sensitivity and anxiety, negative emotions, social factors, and the like. In this case, for the proposed replacement in Table 2 at the end of the paper, the diagnostician can refer to features of or a subsyndromal disorder rather than to PPCS.

Outcome (11) Are there long-term repercussions in the brain and consequently in behavior for MTBI/PPCS? Has the research been replicated? Posing this question might seem illogical because the paper has just advocated for removing PPCS from the lexicon in the field. Practically, the paper continues to use the terminology of PPCS when discussing long-term persistence of MTBI symptoms instead of replacing it with the new proposed terminology. There are isolated research studies on findings that implicate long-term outcomes due to MTBI, but as far as is known, meta-analyses have not been conducted confirming the reliability of the findings.

Specifically, the paper sought at least one study in which there were evident long-term brain-related effects of MTBI that could then influence behavior and symptoms long term. If research such as this has been published, then it could be argued that MTBI does not necessarily dissipate and resolve in the short term in every case. The argument could be made in court and related venues that, in a particular case, it cannot be unequivocally argued that long-term brain-based effects of MTBI do not exist, as per the literature. That said, any such finding would have to be replicated, would have to be linked to plausible pathways and linkages to the behaviors and symptoms in question, and so on. Therefore, any such findings would need a strong evidence base with it to support its utility clinically and in court.

The literature search that was conducted emerged with several lines of research in this regard. The most notable publication on long-term brain-based changes due to MTBI relied on a systematic review of different neuroimaging methodologies and outcome measures within 1-year post-injury (Biagianti et al. (2020)). The technologies included research with decreased resting state (fMRI) (functional magnetic resonance imaging), diffusion-weighted imaging, and H-MR (nuclear magnetic resonance) spectroscopy. The review of the results in the studies showed alterations in MTBI in diffusion along white matter fiber tracts, alteration of perfusion, disrupted metabolism, and reduced connectivity within several resting-state networks. Some specific findings included finding decreased fractional anisotropy values in the association, commissural, and projection white matter fiber tracts (Messé et al., 2012), less DMN (default mode network) connectivity strength (Sours, Zhuo, Roys, Shanmuganathan, & Gullapalli, 2015),

Table 2 Proposal for DSM 5.1: somatic symptom disorder with predominant post-concussion-like symptoms (diagnostic criteria)

Criterion	Explanation
I. Symptoms: apparent authentic biopsychosocial presentation/causation	
A	Post-concussion-like symptoms [as in the note below to the table on the symptoms] are distressing and are the predominant focus of the clinical presentation
B	The symptoms cause clinically significant impairment in social, occupational, or other important areas of functioning (post-symptom onset complications)
C	Psychological factors are judged to have an important role in the onset, severity, exacerbation, or maintenance of the symptoms (excessive, persistent, maladaptive thoughts, feelings, or behaviors), as manifested by at least two of the following: (a) Thoughts about the symptoms' seriousness (b) Anxiety about the experienced symptoms and their perceived consequences (c) Time and energy expended about them
D	Social/ecological/environmental factors are judged to possibly have an important role in the onset, severity, exacerbation, or maintenance of the symptoms (excessive, persistent, maladaptive thoughts, feelings, or behaviors), as manifested by at least one of the following: (a) Family dynamics involved, e.g., encouraging the sick role (b) Social support lacking, socially isolated, or withdrawing (c) Institutional support perceived as lacking, e.g., by the workplace, third party payors, the court or related venues, health professionals involved; a sense of injustice or even undue entitlement in consequence
E	The symptoms or deficits are not intentionally produced or feigned (as in factitious disorder or malingering).
F	The symptoms are not better accounted for by another disorder.
II. Specifiers	
A	Duration: acute: < 3 months; chronic: ≥ 3 months; persistent chronic: ≥ 6 months
B	A. Severity: specify in the boxes below 1. Mild: consider not diagnosing SSD as clinical, given its manageability at this level 2. Moderate: consider diagnosing SSD as a feature or sub-syndromally, although even this level is hard to manage 3. Severe: this level is definitely SSD Reported: symptoms □ □ □ distress □ □ □ impairment □ □ □
III. Feigning	
A	If confusing or complicated presentation/causation, specify degree of feigning, if any • Minor exaggeration • Gross exaggeration • Outright malingering
B	Specify source of confusion, if any: • Can be fully explained by pre-existing factors (e.g., psychopathology) • Pre-existing factors exacerbate the symptoms • Post-onset factors exacerbate the symptoms (e.g., family, work, litigation, distress) • Incidental factors exacerbate the symptoms (e.g., death in family, societal unrest)
IV. Certainty of these ratings	
A	Specify: □ unsure □ some data □ clear data
V. Note	
A	All terms and qualifiers that could be ambiguous or contentious must be attributed only if clearly evidenced and documented and go beyond the minimal/mild and, if applicable, the moderate range, as the case may be, for example: (a) Excessive and persistent psychological factors in the symptom experience (b) Severe symptoms/distress/impairment (c) Gross exaggeration/malingering, and (d) Pre-existing and post-onset factors, as well as any extraneous factors (e.g., an unrelated death of a loved one)

The symptoms: specify the specific symptoms and if they have been derived by the following: (a) self-report, (b) collateral nonprofessional sources, (c) collateral professional sources, (d) file/record/document review, (e) observation, (f) qualitative data, (g) quantitative data, (h) neuropsychological assessment/testing, and/or (i) neurological assessment/scanning/investigation. For example, if using this form for the evaluee record, put the alphabet representation of the data source above the symptoms for the symptoms below that are being expressed by the evaluee in order to indicate their presence and source

Cognition symptoms: confusion, taking longer to think/speed of processing, dazed/stunned/disoriented/feeling foggy, memory problems, concentration problems, attention problems, executive function problems, perceptual problems, speech/language problems/slurring, judgment affected, etc. MTBI can induce pre-trauma retrograde amnesia as well as post-trauma anterograde amnesia (PTA)

Emotions/social symptoms: (a) worry/anxiety, preoccupation with symptoms/anxiety sensitivity, depression/sadness, irritability/quickness to anger/frustration/aggression, bitterness, fear, etc.; (b) poor emotional regulation/responsivity, lability/disinhibition/impulsivity, behavior problems/restlessness/hyperactivity/agitation, personality disturbance, inappropriate behavior, affected self-confidence/self-worth; and (c) interference in social skills/social cognition, etc.

Physical/bodily symptoms: (a) headaches, pain, poor sleep, fatigue/apathy/lethargy, arousal/stress responses, shock, panics, dizziness/light-headedness, vertigo/loss of balance/unsteady gait/motor incoordination, nausea/vomiting; (b) visual disturbance (blurriness, double vision, light sensitivity; difficulties in smooth pursuits, saccades, convergence), auditory disturbance/noise intolerance/sensitivity, tinnitus; and (c) (transient) neurological abnormalities/seizures, sensitivity to alcohol, etc.

and less connectivity within some posterior regions (Palacios-Berraquero et al., 2017).

These findings are significant for the field and should be replicated. Moreover, before being useful for court purposes, continued research is required to determine the efficacy in differentiating long-term MTBI outcomes on an individual basis for these types of neuroimaging methods and how the findings help explicate individual functional outcomes, including in disability. Finally, the necessary control groups would have to be used in the research in order to establish that chronic MTBI effects are not due to pre-injury, predisposing factors either greatly or fully. Necessarily, research participants should be screened for malingering as an exclusion factor. Or groups related to probable and definite malingerers according to test performance and other indicators should be given the same method protocols as other groups in order to determine the differential brain-based activity patterns in the neuroimaging that relates to genuine MTBI cases that do not resolve in the long term.

In other research related to brain-based changes in MTBI/PPCS, in an fMRI study, relative to controls, Dretsch et al. (2019) found that chronic (up to 5 years) MTBI is still active in military service members which was associated with activation in the pre- and post-central gyrus, inferior parietal lobe, insula, and limbic regions in an emotion enhance condition to negative and neutral combat-related visual stimuli. The authors referred to hypervigilance/lower emotional regulation in the chronic MTBI sample relative to controls. They called for further research pertaining to whether injury pathology or other factors account for the findings, and the PPCS involved but discounted PTSD comorbidity as a factor in that it was controlled in the analyses. As a comment, this finding does not generalize to non-combat civilian MTBI, but it does illustrate that long-term effects of MTBI are possible in fMRI tasks related to attention. In a civilian population that was studied 1-year post-MTBI (Booker, Sinha, Choudhari, Dawson, & Singh, 2019a), predictors of outcome according to the functional-based GOSE included the nature of the injury (assault being the most predictive), alcohol consumption at the time of the injury, a GCS score < 15, and psychiatric history, with CT (computed tomography) scan findings playing no role. These results supported a multifactorial model of MTBI/PPCS.

Validity (12) If long-term deficits have been found, are they clinically significant? For example, does a 2% drop or difference in any measure, as in neuropsychological testing results, really mean anything in daily life and can be pursued legally for deleterious effects on quality of life and on functional impairments? It would depend on the evaluee's job to some extent; a banker is quite different in these regards than a laborer. What are long-term deficits in longitudinal studies being published on this type of question, and what are the further studies that need to be undertaken?

This type of question relates to two main points that might bedevil MTBI cases in court. If there is an effect, is it relevant given that, in the grand scheme of things, it is likely to be minor? Moreover, how much can we generalize from neuropsychological data to real-world settings and assume ecological validity? These issues apply to psychological injuries, generally, which are misunderstood in court and diminished in importance. They are considered invisible injuries, and mental ones are excluded often unless they are shown to be physical-mental in characteristic. Thus, mental health workers, such as neuropsychologists, and attorneys might supply to the court in their testimony/report quantitative data to indicate the severity of the consequences for the evaluee, either in the nomothetic research generally, or ideographically for the case at hand. But data have importance only when considered in context, and cannot be treated in isolation. Moreover, research data are manipulable, might not be replicated, and might be a product of litigation science or otherwise of conflict of interest. And the misgivings can apply to idiographic data brought to court. Neuropsychological batteries are used, but there is debate about whether they should be fixed or variable, the decision for which creates a whole range of data variability for one person and one practitioner to the next. As for individual neuropsychological tests, the confidence intervals or cut scores might vary for neuropsychological test from one practitioner to the next. Further, the range of tests used for a particular domain of neuropsychological function can vary.

To summarize, in cases of MTBI/PPCS, there are no gold standard, batteries, tests, and sequences in neuropsychological assessments, so that data will always vary from one person to the next and one practitioner to the next, even in comparable circumstances of injury and consequences. Also, the data brought forward in a case can be selective, manipulated, biased, overinflated or underinflated, and even at odds with best practices and standards in the field. Data that are significant clinically could be trivialized or explained away. Data that are tangential, even if statistically significant, could be magnified in importance and used to explain away other less supportive data for the theory of the case for a case at hand. The 2% difference could mean a 0% ecological significance, or, to the contrary, a 100% difference in one case compared to another. Ethically, the neuropsychologist needs a balanced, scientifically informed, and comprehensive assessment in which all possible explanations are considered for all data that are gathered reliably in a particular case, and any non-supportive data to the preferred explanation, opinion, or conclusion (and recommendation) are addressed as to why the preferred explanation/opinion/conclusion (and recommendation) are still viable.

Influences and Confounds in MTBI/PPCS

The paper has shown to this point the complexity in the etiology and symptom expression of MTBI/PPCS. It has focused on the peritraumatic event and the traumatic consequences, in particular. However, the pre-existing state and influences on the specific manifestation of MTBI/PPCS effects will vary with other factors, and there is no dose-response relationship with the severity of impact and outcome. Indeed, it can be argued that the compendium of pre-event factors in MTBI/PPCS is so large that they can be used to explain away any effects of the event itself on the resulting neurological and psychological condition. However, a reasonable approach to this type of question would call for an extensive literature review and would examine the mediators and moderators that influence and causally affect the outcomes in MTBI/PPCS. Once more, it is beyond the scope of the present paper to conduct such an in-depth literature review. Polinder et al. (2018), in particular, have pointed out relevant literature, which we do not try to supplement here. Rickards et al. (2020) have reviewed the lengthy list of predisposing factors that can influence MTBI outcome, and it is quite consistent with the Polinder et al. list and what this paper has added to the list. The goal in the next section is to illustrate the compendium of pre-event factors that need to be considered on the causal question on MTBI/PPCS. This listing will help orient future research and also individual assessment for court and related purposes.

That said, the danger is that a valid case of MTBI and long-term effects related to biopsychosocial factors is dismissed in court simply by arguing that there are so many pre-existing factors possible according to the literature that any apparent long-term effects are impossible, exaggerated, feigned, malingered, and so on. This paper maintains that in such cases, in which the latter explanations have been ruled out, a person could express valid long-term symptoms that even affect functionality for work and the like through peritraumatic reactions that exacerbate, propagate, and maintain the symptoms, such as extreme initial shock, anxiety, depression, catastrophizing, and the like. The proper diagnosis in such cases, however, should not be PPCS but more emotionally based disorders, such as anxiety, depression, adjustment disorder, or one like the recommendations made at the end of the paper, as the case may be. The initiating event may be biological, but the follow-through will be psychosocial in such cases.

Prior State (13) What are the confounds from (a) previous/sequential TBIs, neurological history, and medical and health history; (b) demographic variables such as sex, prior psychological/psychiatric vulnerabilities, conditions, or disorders, including disturbed personality characteristics and disorders, and alexithymia; (c) poor coping skills and resilience; (d) prior poor education/LD (learning disorder)/ADHD (attention-deficit hyperactivity disorder), and lower intelligence; (e) prior alcohol and drug use/abuse/addictions; (f) prior negative family and relationship dynamics and social integration, and family psychiatric history; (g) prior work performance and satisfaction, and prior financial problems; (h) life satisfaction; and (i) extraneous variables, such as criminal record, if any, etc.?

For demographics related to MTBI/PPCS, on the matter of sex/gender differences, consult Cogan, McCaughey, and Scholten (2020), Gray et al. (2020), Gupte, Brooks, Vukas, Pierce, and Harris (2019), Merritt, Padgett, and Jak (2019), Niemeier (2019), and Solomito, Reuman, and Wang (2019). For age differences, consult Abdulle et al. (2018), Asselstine, Kristman, Armstrong, and Dewan (2020), McCulloch, Osborne, and Ramsey (2020), and Richey et al. (2020).

This category of influences and confounds is the critical one in psychological injury cases because the plaintiff will attempt to show that they are not contributory to the claim at issue, or minimally so, whereas the defense will attempt to show that they are so strongly present that they can explain in full or almost fully the presenting state of the person. The issue relates to the heart of the causation argument in court; the event at issue causally must be beyond the *de minimus* range, or considered a substantial or material contributor to the outcome despite the presence of predisposing factors, if any.

Confounders (14) About other confounds and mimics, medicines can create a state comparable to MTBI, syncope, too, etc. This category is another way of explaining away the presenting condition of the plaintiff that could be used in court, or one that needs to be carefully scrutinized by the plaintiff in order to rule it out.

Social Dynamics (15) What are the complications of inappropriate family dynamics such as reinforcing illness behavior? What are the repercussions of negative social effects? This category relates to psychosocial variables that can either exacerbate the presenting condition negatively or moderate it through more constructive elements. Does the family encourage the sick role, impeding normative progress? Is the person lacking in normative social support, or withdraw socially after the injury at issue?

Cognitive Dynamics (16) Involves misattribution, expectancy effects, diagnostic threat, good-old-days thinking, catastrophic thinking, symptom hypervigilance, perceived injustice/justification/sense of entitlement, and doctor shopping. This category is crucial because, in a CBT (cognitive behavioral therapy) framework, maladaptive cognition fuels symptomatology and negative emotionality. Cognition is the mediator that needs careful scrutiny in MTBI/PPCS cases because it might lead the person toward (a) giving up in the recovery or the like, (b) having stress responses that aggravate the original injury

effects, or (c) waiting for a monetary settlement in the legal process beyond what the original injury merits.

Comorbidities (17) What are the complications from comorbid depression, anxiety, PTSD, dissociation, pain disorders, and polytrauma? For example, Terry, Brassil, Iverson, Panenka, and Silverberg (2019) referred to depression in this regard, and Roberge et al. (2020) referred to PTSD. Other research to consult for these comorbidities include the following: for depression, Barker-Collo et al. (2018), Hellewell, Beaton, Welton, and Grieve (2020), Larsen, Larson, Hunt, Lorber, and deRoon-Cassini (2020), and Wallace et al. (2020). For PTSD, consult Hebert, Forster, Stearns-Yoder, Penzenik, and Brenner (2018), Iljazi et al. (2020), Lange et al. (2020), Loignon, Ouellet, and Belleville (2020), Moore et al. (2020), and Ramanathan-Elion, Baydoun, and Johnstone (2020).

For sleep disturbance, consult the following. In this regard, see Kalmbach et al. (2018), Lowe, Neligan, and Greenwood (2020), Lu, Reid, Cooper, and Kennedy (2019), Martindale, Morissette, Rowland, and Dolan (2017), Mollayeva et al. (2017), Mollayeva, Stock, and Colantonio (2019), Mumbower et al. (2019), Saksvik et al. (2020), and Wickwire et al. (2018).

For pain and headaches, the influence on MTBI/PPCS is considerable. The reader should consult in this regard Anderson (2020), Chaput, Lajoie, Naismith, and Lavigne (2016), Greenberg et al. (2020), Hoffman, Herbert, Crocker, DeFord, Keller, Jurick, Sanderson-Cimino, and Jak (2019), King, McCrea, and Nelson (2020), Leung (2020), Mares, Dagher, and Harissi-Dagher (2019), Ofoghi, Dewy, and Barlow (2019); Silverberg, Martin, and Panenka (2019), Snell et al. (2018), and Lucas et al. (2016).

Exacerbators (18) This refers to the stress response to stakeholder dynamics, being put off work or other disruptions in daily responsibilities, litigation distress, etc. This refers to extraneous factors for the person beyond her or his control that add to the stress impacting her or him in the post-injury period. What if the disability regime in the case (e.g., the insurer) plays hardball inappropriately, subjecting the person to uncalled-for stress? What if the legal representative adds to the litigation distress in her/his own way? What if the employer is unfair in accommodating the injured worker and prematurely terminates the job, the associated benefits, and so on?

Deactivators/Protective Factors (19) This refers to (a) social support, systemic support, e.g., benevolent employer, supportive WSIB/RTW specialist, caring family doctor/personal care physician, and (b) good skills in accessing resources. The psychosocial factors that could influence MTBI/PPCS outcomes include possible supportive factors as opposed to ones that encourage vicious circles and deterioration. These include social, institutional, professional, and family support that enhances recovery and RTW (return to work).

Substance Abuse (20) This refers to substance use disorder and self-medication, excluding if it is pre-existing (this is covered in the category of prior state), or the opposite, an aversion to alcohol as a symptom. This category of factors is crucial as another possible exacerbatory one. Indeed, many MTBIs are associated with alcohol use at the time of the original injury. Some of the recent research in the area that the reader should consult includes Booker, Sinha, Choudhari, Dawson, and Singh (2019b), Dismuke-Greer et al. (2018), Gallant and Good (2019), Hanson et al. (2016), Herrold, Pape, Li, and Jordan (2017), Scheenen et al. (2016), and Uccella, Bongetta, Fumagalli, Raffa, and Zoia (2020).

Treatment (21) What cognitive rehabilitation program is best, and how much can it help? Or are standard CBT and other therapies more appropriate? When considered together, several publications (Arbabi et al., 2020; Chen, Lin, Huda, & Tsai, 2020; Sullivan et al., 2020; Teo, Fong, Chen, & Chung, 2020; Vanderploeg, Belanger, Curtiss, Bowles, & Cooper, 2019) found that more standard CBT approaches constitute the best practice approaches in rehabilitation of MTBI/PPCS. Register-Mihalik, DeFreese, Callahan, and Carneiro (2020) advocated for a biopsychosocial approach. As for cognitive rehabilitation, the reader should consult Cicerone et al. (2019). For psycho-pharmacological interventions, see Beedham, Belli, Ingaralingam, Haque, and Upthegrove (2020), and Johansson, Andréll, Rönnbäck, and Mannheimer (2020).

Assessment

The present roadmap on MTBI/PPCS does not delve deeply into assessment (points 22–29) because it requires an in-depth presentation. A follow-up paper in the journal will explore assessment in relation to MTBI/PPCS. However, this article does describe some recent findings in relation to point 26 on malingering.

Scans (22) This refers to CT, MRI, MRS (magnetic resonance spectroscopy), SPECT (single photon emission computed tomography), fMRI, DTI (diffusion tensor imaging), etc. Which are valid scientifically, valid in court, controversial, etc. In MTBI, that is not complicated; none of these neuroimaging techniques should be useful for cases that have long-term repercussions leading to PPCS. That said, subtle neuroimaging findings are elucidating regional and connectivity abnormalities in long-term outcome cases, as shown above. Aside from replicability issues, this research needs a solid program to inform the court how the abnormalities relate to an individual's presenting condition and consolidate the evidence that indeed the PPCS is valid neurologically and neuropsychologically. This paper is skeptical that such an outcome to the proposed research programme will be reliable for court purposes for the legal issues at hand.

Data Predictors (23) This refers to GCS (Glasgow Coma Scale), GOSE (Glasgow Outcome Scale), the four spheres of the AMA Guides to the Evaluation of Permanent Impairment's (activities of daily living (ADLs); socializing; concentration/persistence/pace; and adaptation/decompensation/coping in work or work-like (i.e., role) settings) (Rondinelli, 2007), etc. Which of these indicators are valid scientifically, valid in court and related legal settings, controversial, etc. For the most part, this category refers to tests and measures that are not based on standardized and empirically derived measures. However, they are useful to court because they qualify impairment ratings, functional losses, and disability.

Real-Life Predictors (24) This refers to quality of life (QOL), RTW, and return to other roles. Which are valid scientifically, valid in court, controversial, etc. This category refers to outcome measures and adapting life roles outside of the ones in the prior category that are also useful to court. The plaintiff could argue that, despite an RTW or other daily responsible role, such as studying or caregiving, the person's quality of life has been greatly impaired and is deserving of compensation for pain and suffering.

Test Predictors (25) This refers to processing speed, attention measures, and anything else the MTBI research is finding. Which are valid scientifically, valid in court, controversial, etc. This category relates to specific empirical findings in the long-term effects of MTBI/PPCS that are merging in the literature, and are being replicated, if any. As with other research for court purposes in MTBI/PPCS, the findings will be more probative than prejudicial to a case at hand when malingering has been screened as an exclusionary factor or controlled in other ways, and the findings help explain the pathways to the deficits and impairments/disabilities being claimed.

Malingering (26) What are the best performance validity tests (PVTs) and symptom validity tests (SVTs) to use in assessment? Tests include the TOMM (Test of Memory Malingering), and the MSVT (Nonverbal Medical Symptom Validity Test), for example, as per Polinder et al. (2018). Which are valid scientifically, valid in court, controversial, etc.? A new test in this arena is the Inventory of Problems-29 (IOP-29) (Viglione, Giromini, & Landis, 2017). Is the MND valid, or are there other ways of determining the validity of a profile? For example, see Denning and Shura (2019) on the cost of malingering to the VA (Veterans Benefits Administration). The authors used the MSVT (Nonverbal Medical Symptom Validity Test, Green, 2008) and the TOMM (Test of Memory Malingering, Tombaugh, 1996) in their study. However, failure on just one of the two tests was deemed sufficient to be attributed to malingering. The field needs to be wary in determining the malingering motivation based on just one test result.

Moreover, systems that typically require more than one test failure, such as in the MND, also have their difficulties (Young, 2014). That said, in a study with a relatively small civilian sample (Elias, MacLaren, Brien, & Metcalfe, 2018), the MND helped distinguish probable malingerers from non-malingerers and then functional impairments were found to differ in the groups. However, probable and definite malingerers were collapsed into one group in this study because of the small sample size, so that the findings are not clear on the value of the MND.

The problems inherent to the MND has led to its revision (Sherman, Slick, & Iverson, 2020). This indicates that its past use in research led to some questionable findings. Also, its future use, especially if applied to evaluees, should be tested for its reliability and validity. Premature application to evaluees might lead to misclassification of genuine malingerers and those who are not.

Specifically, Sherman et al. (2020) indicated, in particular for present purposes, that the revised MND has expanded to consider not only cognitively mediated dysfunction that could indicate malingering but also somatic- and psychiatric-mediated function. Establishing compelling inconsistencies and marked discrepancies is relegated to non-test aspects of a file. For testing, the evaluee is evaluated for PVT and SVT performance. The criteria indicate that two or more PVTs should be administered in assessments and that the ratio of failed and passed PVTs is critical to determining "invalid" presentation. With respect to testing results, invalid presentations as defined are used to determine the presence of malingering. The PVTs used in the revised MND should have a low false positive rate (related to its accuracy, e.g., 10), either alone or in combination, or the evaluee fails a two-option, forced-choice SVT at the statistically below-chance level. For SVTs in the revised MND, one or more SVTs need to be in the invalid range in this regard, and once more the tests should have a low false positive rate of 10 or lower.

It is not clear that the revised MND has avoided new minefields that undermine its reliability, validity, and accuracy in attributing malingering. For example, it is not clear why the evaluee should be given at least two PVTs, but in the case of SVTs, only one is sufficient. Moreover, SVTs, such as in the MMPI-2-RF and the PAI, come in multiples, and how to use them is not specified. It is not clear what exactly is the ratio of failed PVTs to passed PVTs that would meet MND requirements. The text to the revised MND maintains that the ratio should not be reducible to one fail out of the number of PVT administered (e.g., two fails out of 14 is like one fail out of 7, so that ratio is not indicative of profile invalidity). Ideally, the PVTs used in assessments should tap different domains and not be much correlated in the research, but, according to text for the revised MND, that kind of proviso does not have hard guidelines in the literature that can be applied to the revised MND. For SVTs, the revised MND paper indicated that if

more than one is used, they should provide "nonredundant" information. Specifically, the text to the revised criteria indicates that the construct validity of existing SVTs is in doubt because, generally, they have overlapping cognitive, somatic, and psychiatric items in one way or another. Of note, the revised MND removed the distinction between definite and probable malingering attribution categories, partly because, against expectations, "a minority" examinees actually met the MND criteria for it. This is telling perhaps could it be that too much stringency in the original MND has led to more relaxed standards in attributing malingering for the revised version? Non-contentiously, the authors conclude that evaluees who fall in the gray zone should not automatically be given an attribution of valid presentation. Also, those who present and perform invalidly might have valid reasons for the result and might not be necessarily malingering.

What about some specific recent research on the MMPI-2-RF and the PAI? In their study of a military sample with a history (3 months +) of MTBI, Jurick et al. (2018) found the validity scales of the MMPI-2-RF effective in effectively distinguishing symptom exaggerators and non-exaggerators, with PVTs less effective in these regards. In a military sample, Ramanathan-Eliona, Baydoun, and Johnstone (2020) found the negative impression management (NIM) scale on the PAI (Personality Assessment Inventory; Morey, 2007), which measures symptom exaggeration (or negative impression management), and helped predict indicators of outcome on the FIS, but so did concurrent PTSD. The authors concluded that elevated NIM scores in the sample were not related to malingering.

Neuropsychological Tests (27) What is the best battery to use in neuropsychological testing for MTBI? Tests include the Rey Auditory Verbal Learning Test; Delis-Kaplan Executive Function System—Verbal Fluency; Trail Making Test; Cognitive Battery-NIH toolbox; WASI-2; and WAIS, as per Polinder et al. (2018). It would be helpful to know the scope of neuropsychological testing undertaken for MTBI/PPCS; the domains involved, e.g., memory, concentration, attention, verbal skills, and intelligence; which ones are emphasized more than in the standard TBI case; and which ones are not even used because of the milder nature of the injuries.

In this regard, Prince and Bruhns (2017) indicated the functional domains that should be assessed by neuropsychologists in their assessments. They include premorbid estimate, performance validity, motor, attention/working memory, processing speed, language, visuospatial, memory, executive function, mood, and symptom self-report. Here, we note that performance validity testing in MTBI neuropsychological assessment should include not only multiple embedded tests (e.g., the ones listed by Prince and Bruhns (2017), the RDS, and the CVLT-II forced-choice procedure), but also stand-alone ones (e.g., the TOMM, IOP-29, and SIMS; respectively, the Test of Memory Malingering, Tombaugh (1996); the Inventory of Problems-29; Viglione et al. (2017); and Structured Inventory of Malingered Symptomatology (SIMS; Smith & Burger, 1997) with the qualification that their cutoff scores should be determined according to the changing research on the instruments). The cognitive areas with results to consider include social cognition, emotion recognition, theory of mind, self-awareness, complex attention (sustained, divided, selective), processing speed, learning and memory (including for free and cued recall, recognition memory, semantic and autobiographical (episodic) memory, and implicit learning), executive function (including panning, decision-making, feedback response, working memory, response inhibition, and cognitive flexibility), language (including word finding and naming, verbal fluency, grammar and syntax, and receptive language), and perceptual-motor function (including visual perception, visuo-constructional reasoning, and perceptual-motor coordination) (Calvillo & Irimia, 2020).

Scales (28) As per Polinder et al. (2018), use the following scales: (a) for self-report—Neurobehavioral Symptom Inventory, and the Rivermead; (b) for psychological/psychiatric status—BDI II, PHQ-9, the MMPIs, the PCL, the SMEQ, and the AUDIT. The research supports the use of the PAI and its NIM scale, as well as shown above. The MMPIs include the MMPI-2 (Butcher et al., 2001) and the MMPI-2-RF (Ben-Porath & Tellegen, 2008/2011). The latter personality test includes five evaluee validity scales that are useful for court purposes (F-r, Fp-r, Fs, FBS, RBS). More research is needed to determine what combination of "failed" results on these scales indicate better than otherwise possible malingering. Results from psychometric testing constitute only one source of evidence in malingering determination, with interview data and documentation/record review constituting other sources.

Inconsistencies (29) Are there inconsistencies? Are the symptoms vague/product of a poor historian? Are the symptoms out of proportion to the severity of the original head trauma, not being in a dose-response relationship? Inconsistencies must be compelling before malingering is attributed. The clearest one concerns video surveillance of an injured working claiming disability who is found to be working surreptitiously. Other inconsistencies refer to gross differences in presentation and evidence derived from collaterals, documents, and testing. Also, verbal and nonverbal information in the interview could be highly discrepant, or the person could present in a way that is grossly out of proportion to the severity of the original trauma. Inconsistencies by themselves are insufficient to attribute malingering. Moreover, they should not be culled from the evidence for a long list of minor inconsistencies to be used to deny valid claims. Refer to Young (2014) and to Sherman et al. (2020) for detailed analysis of types of inconsistencies in this area of assessment.

Law

Court (30) How does MTBI fare in court? What are some case law precedents? Review of this category is beyond the scope of the present paper, but it is crucial to the forensic task of presenting testimony to court. The field needs to know both nationally and locally what court decisions lay the groundwork for assessment procedures of psychological injury cases, including of MTBI/PPCS. In the USA, the decisions of the Daubert trilogy (Daubert, 1993; Frye, 1923; Kumho, 1999) outline what evidence is admissible to court. The equivalent decision in Canada is R. v. Mohan (1994). In the end, the court expects scientifically informed testimony, including in reports proffered to court, that reaches the bar of reliability or being useful to the court in its deliberations (in psychological terms, this means the reports are reliable and valid). Other decisions include Frye (1923), which involves general acceptance of the evidence behind the testimony/report rather than having scientific validity. A good proportion of states in the USA adheres to this legal doctrine rather than *Daubert* or its equivalents. Finally, Federal Rules of Evidence (Michigan Legal Publishing Ltd, 2016) specify *Daubert*-like rules that define testimony to court.

When legal standards are not met in proffered testimony to court, the testimony could be subject to admissibility challenges and subsequently excluded from court. Alternatively, the weight given to the testimony could be reduced and subject to withering cross-examinations. The assessor in MTBI/PPCS cases should follow best legal, practice, and ethical guidelines and standards and know the laws and rules related to admission of evidence to court in her/his jurisdiction in order to contribute constructively to court proceedings, maintain reputation (and referral sources), and offer evidence that is helpful to the referral source. Indeed, even if the referral source expects a supportive testimony to the case at hand, and the outcome is not in favor of the source, the assessor should submit the testimony/report as consistent with legal, professional, and ethical obligations, and withstand undue pressure to alter the testimony/report.

Conclusions

Summary

The article has reviewed the complicated and evolving field of MTBI and organized a list of 30 points that workers need to consider toward functioning in the assessment of these types of brain injuries and in determining their long-term consequences, if any. The article took a biopsychological, forensic approach, in which the arrays of multiple factors both before and after the index injury need to be evaluated comprehensively and scientifically, and in an impartial way, including for possible malingering.

The field is replete with too many conundrums, controversies, confounds, and gaps in the literature such that definitive practice and legal position statements would be premature. Nevertheless, the 30 points enumerated that the field in this article needs to consider pointing the way in establishing its gaps, the research needed to accommodate them, and the general directions the field needs to take, as well as recommendations toward disentangling its conundrums, controversies, and gaps.

Well-constructed longitudinal research with appropriate definitions and classifications of different groups in the samples that are studied constitutes the best research practice in the field. Well-argued and impartial conceptualizations on MTBI/PPCS constitute the best assessment and legal practice in the field. This paper has offered conceptual clarifications, suggestions for empirical investigation, and cautions for assessors in court and related venues that work toward these goals.

Recommendations

This article recommends that PPCS be removed from the definitional and diagnostic lexicon related to MTBI because of its invalidity and confusing status. Persistent symptoms related to the original mechanical force applied to the head that leads to concussion-like symptoms might be valid in a particular case, but the maintenance and propagation of the symptoms from the acute to the chronic stage, or after a few weeks to a few months, can hardly be due to continuing physiological impacts that continue for purely biological reasons. Rather, in these regards, the symptoms should resolve, as happens in most cases of MTBI. When they do not resolve and become chronic, factors such as psychosocial, ecological, environmental, secondary, iatrogenic, forensic (e.g., litigation distress), and pre-event history might all play a role. This argument assumes that, in terms of gross exaggeration, poor effort and response bias and feigning and malingering have been ruled out. Also, it assumes the case is not a complicated MTBI with associated CNS signs. In the standard case of PPCS, for this article, there are no post-concussion symptoms but symptoms that propagate because of somatization, a host of psychosocial factors, and where applicable, legal-forensic ones. Therefore, according to this logic, the DSM-5 should add to its diagnostic categories the one of somatic symptom disorder with predominant post-concussion-like symptoms (see Table 2). The present proposal for SSD with predominant PPCS-like symptoms for inclusion in the DSM-5.1 is based on a similar proposal on how SSD with predominant pain could be re-organized in the DSM 5.1 (Young, 2016).

Compliance with Ethical Standards

Conflict of Interest The author declares that they have no conflict of interest.

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