CHAPTER SIX

More than a bump to the head: An overview of the long-term effects of concussion

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Abstract

Concussion is often depicted as a short-term injury whereby symptoms assessed in the clinical setting typically resolve within two to four weeks. Recent high-profile cases involving post-concussion syndrome, chronic traumatic encephalopathy, and disturbing behavioral alterations in veterans and athletes participating in high-impact sports have begun a new discussion within both the general public and research community centered around the potential long-term effects of concussion. Innovative research, employing experimental measures of neural and behavioral function, have begun to contradict this notion of transience and suggests that concussed individuals can have lasting effects for years following initial injury. This chapter will provide context for this new perspective. We start by providing a brief description of concussion, outlining its symptomology and the existing guidelines for assessing clinical "recovery". Additionally, to help clarify their distinction from the evidence herein, we find it crucial to define two disorders commonly linked to long-term concussion effects: Post-Concussion Syndrome and Chronic Traumatic Encephalopathy. Fundamental to this chapter, we will review the evidence of subtle yet persistent functional alterations in

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individuals with a history of concussion, expanding upon the insights provided by neurophysiological techniques and highlighting their implications for clinical recovery and future research directions.

1. Introduction

The brain's ability to maintain functionality throughout a human's lifespan, despite its delicate structure and the near-constant biomechanical forces exerted upon it, emphasizes its remarkable nature. Such resilience is aided by surrounding anatomical defenses (i.e., the skull, meninges, and cerebrospinal fluid) which help redistribute and absorb external forces transmitted toward the brain (Sakka, Coll, & Chazal, 2011; Walsh et al., 2021; Yoganandan et al., 1994). However, when biomechanical forces exceed the capacity of endogenous protective mechanisms to mitigate damage, traumatic brain injury (TBI) occurs, disrupting functionality (Werner & Engelhard, 2007). Estimating the true incidence of TBI is challenging, but it is believed that annually 64–74 million cases occur worldwide (Dewan et al., 2018). Distinct from moderate to severe TBI—which is associated with protracted loss of consciousness, altered mental status, and structural pathology (e.g., intracranial hemorrhage, lesions, or edema) or penetrating injury (e.g., skull fracture) (Maas, Stocchetti, & Bullock, 2008); the term "concussion" is used to refer to the substantial percentage of TBI (70–90%) that are classified as "mild" in severity (Cassidy et al., 2004). Roughly 1 in 5 adolescents report a lifetime prevalence of at least one concussion, with these estimates approaching 1 in 3 individuals in adulthood (Daugherty, DePadilla, Sarmiento, & Breiding, 2020; Veliz, Eckner, Zdroik, & Schulenberg, 2019). Despite significant functional impairment, concussion is described as a "silent" injury due to the general absence of gross structural pathologies or abnormalities and absence of sustained loss of consciousness/altered mental status, rendering it less likely that individuals will seek medical care (Patricios, Schneider, et al., 2023). As clinical symptomology is popularly asserted to resolve within two to four weeks after injury onset (Putukian et al., 2023), the general absence of detectable pathology and transient symptoms has historically shaped the perception that concussion is merely a "bump to the head" with brief and self-resolving symptoms. However, research over the past two decades has revealed that the effects of concussion are less temporary than commonly believed (Martini & Broglio, 2018).

Two of the most concerning long-term outcomes linked to concussion history are post-concussion syndrome, characterized by the persistence of concussion-like symptoms beyond the typical recovery timeframe, and the manifestation of neurodegenerative diseases later in life (Barlow, 2016; Gardner & Yaffe, 2015). While presenting potentially troublesome prognoses, ongoing research has started to identify non-concussion-related factors influencing their likelihood (see Sections 2.2–2.3). Tremendous efforts have been made to enhance concussion prevention strategies and reduce unnecessary exposure to head impacts by developing more advanced equipment technology, implementing safer rules for sport (May et al., 2023), improving education (Feiss, Lutz, Reiche, Moody, & Pangelinan, 2020), and refining diagnostic and management protocols (Broglio et al., 2024; Eliason et al., 2023). Nevertheless, fear of the potential deleterious effects has intensified (Baugh, Kroshus, Kiernan, Mendel, & Meehan, 2017; Walton et al., 2022). Out of an abundance of caution, nearly 1 in 5 of adults now report that they would never allow their children to participate in high-contact sports such as American tackle football (Waltzman, Sarmiento, & Daugherty, 2024). Considerable discourse has arisen regarding whether the potential long-term effects linked to concussion history outweigh the benefits of sport participation (Malcolm, Matthews, & Wiltshire, 2024; Piggin, Parry, & White, 2024). As such, ongoing scientific inquiry has sought to further elucidate the long-term effects of concussion.

Advancements in research methods have uncovered underlying behavioral and physiological differences between individuals with and without a history of concussion, implying residual alterations despite individuals being considered as clinically recovered (Martini & Broglio, 2018). There is growing concern that these alterations, while subtle, may negatively impact daily functioning and quality of life, and contribute to an increased risk of injury (Reneker, Babl, & Flowers, 2019). The present chapter aims to review modern evidence on the long-term effects of concussion, consider their potential impact on functionality throughout the lifespan, and present how neurophysiological approaches can be implemented to further our understanding of these effects. We start by defining concussion and outlining how this injury is typically managed. Additionally, we find it crucial to explain how differences in timelines, clinical burden, and contributing factors necessitate that post-concussion syndrome and neurodegenerative diseases are treated as distinct conditions despite sharing similar symptomology and history of head trauma. We conclude by examining how these emergent findings may guide future research directions. A paucity of data prompts us

to periodically reference studies covering a wider spectrum of TBI severity (including concussion) throughout this chapter. We recommend caution when extrapolating these findings to concussion specifically, but this approach may unveil avenues for further exploration.

2. Defining concussion

Historical inconsistencies in the terminology, definitions, and criteria for concussion have made it difficult even for experts to coherently synthesize research findings. Therefore, before proceeding, it is necessary to outline how diverse terminology has been consolidated into the general conceptual definition as it stands today. Colloquial terms such as "ding", "bump", "bell ringer", or "rattled" have generally been deemed as outdated/improper terminology that should be avoided in favor of the terms "mild TBI" and "concussion"—which are generally recognized as interchangeable. Recognizing that certain injury characteristics and return-to-activity/work considerations may be unique to the context in which head trauma occurred, it has been suggested that the use of the term "concussion" be restricted to injuries sustained in a sport-related context. More unitary perspectives of TBI however argue that context does not adequately differentiate concussion from other forms of mild TBI, rather that the term "concussion" may be more appropriately used to denote a subset of mild TBI with relatively minor clinical outcomes (King, 2019; McCrory et al., 2013). As a result, concussive injuries or injuries producing concussion-like symptoms after a single or repeated impact(s) have been historically characterized as minor TBI, minor head trauma, or minor head (brain) (Gronwall, 1991; Kibby & Long, 1996; Weight, 1998). Consistent with such a view, the term "concussion" has been suggested as the preferred term when speaking with patients to avoid perceptions of "permanent brain damage" (Management of Concussion/mTBI Working Group, 2009). However, these terms lack distinct operational definitions and there is no objective test or biomarker that clinicians utilize for diagnosis. Importantly, their injury characteristics and outcomes fall within the broadest criteria set for diagnosing concussion (McCrory et al., 2013). Fundamentally, the lack of a common definition represents a critical failure within this area of research and medicine. It is further important to acknowledge that while concussion reflects the lowest clinical severity from a unitary perspective of TBI, the historical tendency has been to present

concussions as "no big deal", something that can be brushed aside or played through, and/or lacking long-term implications—either due to a research emphasis oriented towards more severe head trauma or sporting contexts oriented towards enabling athletes to return to participation. Within the present chapter, the term "concussion" is preferentially used, although studies using related terms may be referenced due to their inherent overlap in diagnostic criteria.

Beyond the limitations in appropriately characterizing the literature in this area, the lack of consistent terminology also has implications for diagnosis and assessment. At least 17 published definitions for concussion and mild TBI requiring blunt head trauma have been proposed (Crowe et al., 2018). Substantial variability in these definitions makes it difficult for clinicians to diagnose the injury with assurance. The Glasgow Coma Scale is the conventional method used to determine a suspected TBI's severity within the first 6 h of injury, with scores ranging from 3 (completely unresponsive) to 15 (fully conscious) (Sternbach, 2000). A person with a concussion would obtain a score between 13 and 15, indicating mild to no impairment in consciousness, alertness, orientation, and responsiveness to eye-opening and motor commands. However, a high Glasgow Coma Scale score alone does not guarantee the absence of underlying structural pathology and clinical neuroimaging techniques including Computerized Tomography and Magnetic Resonance Imaging may be used at the clinician's discretion.

To confirm head trauma, a plausible mechanism of injury must be first identified whereby acceleration, deceleration, angular, and/or rotational forces were transferred directly or indirectly to the brain—although an important note is that a collision between the brain and the skull (e.g., a coup-countercoup injury) is not necessary. It is generally understood that such forces cause a shearing or stretching of neural tissue, initiating a complex pathophysiological cascade that aligns with concussion's symptomology (Giza & Hovda, 2014). Nonetheless, there is significant ambiguity in asserting the magnitude of forces required to produce these consequences. Therefore, injury characteristics immediately following the event are further utilized to help diagnosis TBI, including loss of consciousness, post-traumatic amnesia, disorientation/confusion, tonic posturing, seizure, and other focal neurologic deficits reported by the patient or witnesses (Silverberg et al., 2023). Generally, loss of consciousness duration must be ≤ 30 min and post-traumatic amnesia duration must be ≤ 24 h to exclude moderate/severe TBI. However, loss of consciousness

and post-traumatic amnesia are not required for diagnosing concussion, and more conservative definitions which apply specific duration requirements raise concerns regarding the extent to which the individual can accurately recall the period following injury (Crowe et al., 2018).

The "cornerstone" of concussion diagnosis and management relies on assessing clinical symptoms indicative of neurological dysfunction (see Section 2.1). It is crucial to assess clinical symptoms during the acute period (i.e., within 72 h post-injury) when symptoms are most severe, as they may evolve over hours or days (Patricios, Schneider, et al., 2023). Relying solely on symptom profiles for decision-making can often result in misdiagnosis, as concussion symptomology is non-specific (i.e., not unique to concussion/TBI). Diagnostic criteria require that clinicians ensure that concussion-like symptoms are not solely attributable to drug, alcohol, or medication usage, cervical injuries, psychological disorders, or other comorbid conditions. The Concussion Recognition Tool-6 combines signs of TBI, injury characteristics, clinical symptoms, and modified Maddocks questions to strengthen diagnostic assurance (Echemendia, Ahmed, et al., 2023).

While consensus groups have moved toward establishing more standardized criteria, we reiterate that there is no unanimous definition for concussion, and its diagnosis relies on the clinician's judgement. For a more accurate diagnosis, the methods detailed above may be utilized but are not explicitly required. Four overlapping criteria consistently appear across the expert consensus derived definitions for concussion/mild TBI: (i) A plausible mechanism of injury resulting in the transfer of biomechanical forces to the brain must have occurred, (ii) Acute symptoms and/or evidence of neurological impairment attributable to the injury must be present upon clinical evaluation, (iii) In the absence of acute symptomology, injury characteristics or neuroimaging must be attributable to the injury, and (iv) Criteria (ii) and (iii) cannot be accounted for by confounding factors or more severe TBI (Davis, Patricios, Schneider, Iverson, & Silverberg, 2023; Harmon et al., 2019; Silverberg et al., 2023).

2.1 Clinical assessment of concussion recovery

Current guidelines on concussion management call for assessment of an individual's recovery progress using a comprehensive multimodal evaluation, preferably conducted by a multidisciplinary team (Broglio et al., 2024; Patricios, Schneider, et al., 2023), in order to suppress any one individual bias or perspective. There is no universal set of assessments for clinicians to use in their evaluation. Instead, clinicians have discretion in selecting which

symptom domains to assess and the tools used to evaluate them. Again, such a "wild-west" approach represents a critical failure within this area of research and medicine given that concussion symptomology is non-specific and heterogeneous (i.e., symptoms as well as their onset, duration, and severity can vary widely among individuals). The argument for this approach, however, is that attempting to assess the full breadth of potential concussion symptomology would be costly and potentially burdensome to both the patient and clinician (Broglio et al., 2024).

To reduce assessment redundancy and the need to build individualized assessment plans, clinicians regularly utilize a symptom rating scale within a battery of tools to identify common symptoms profiles and their relative severity (Kontos, Sufrinko, Sandel, Emami, & Collins, 2019; Kontos et al., 2020; Lempke et al., 2023). Popular standardized assessment batteries, such as the Sport Concussion Assessment Tool-6 (SCAT6) and the Sport Concussion Office Assessment Tool-6 (SCOAT6)—which offer child (8–12 years) and adolescent/adult (≥13 years) versions—are valuable in discriminating between concussed and non-concussed individuals during the acute and subacute (i.e., 72 h to weeks postinjury) periods, respectively (Echemendia, Brett, et al., 2023; Patricios, Davis, et al., 2023; Patricios, Schneider, et al., 2023). Clinicians may seek to further enhance decision-making by employing neurocognitive test batteries (Alsalaheen, Stockdale, Pechumer, & Broglio, 2016), coordination/balance assessments (Buckley, Munkasy, & Clouse, 2018), measurements of heart rate, blood pressure, and symptoms in response to postural change or physical exertion (Haider et al., 2021), cervical spine and neurological evaluation (Mohai et al., 2022), Vestibular-Ocular Motor Screening (Mucha et al., 2014), and/or validated mental health or sleep screening instruments (Patricios, Schneider, et al., 2023).

Once the clinical assessment of a concussion is made, the current standard of practice is to address the symptoms which manifest following the injury. Although historical strict rest protocols such as 'dark room" or "cocoon" therapy are no longer recommended, consensus statements emphasize a relative rest period within the first 24–48 h postinjury (Broglio et al., 2024; Patricios, Schneider, et al., 2023). During this period, relative rest protocols often encourage individuals to maintain activities of daily living to the best of their ability, rest physically—to address dysfunctions associated with postural/motor control and perceptual sensitivities and avoid tasks which require cognitive engagement (such as screen time, school, and work)—to address dysfunctions associated with high level cognitive operations and memory (Patricios, Schneider, et al., 2023).

Beyond this, the standard of practice encourages a graduated return-to-activity/work protocol, where individuals progressively return to doing those activities that do not exacerbate their symptoms while avoiding high-risk activities (such as contact, collision, or falls) to mitigate the risk of reinjury (Leddy et al., 2023; Putukian et al., 2023). To aid this protocol, clinicians may choose to employ graded aerobic exercise testing—such as the Buffalo Concussion Treadmill or Bike Tests—which gradually increase exercise intensity up until the point at which concussion symptoms are exacerbated or re-manifest, as exercise intolerance due to symptom exacerbation may indicate persistent physiological dysfunction (Haider et al., 2019; Leddy & Willer, 2013). Even "cutting edge" therapeutic practices still utilize a concussion symptom abatement approach, focusing only on addressing symptomologies, having patients undergo balance, vision, and cognitive training to minimize symptom presentation within that domain. Pharmacological treatments for concussion have similarly followed suit with 1 in 10 clinicians prescribing Amantadine—a neurostimulant originally used as an antiviral agent—in an attempt to offset post-injury cognitive impairments (Reddy, Collins, Lovell, & Kontos, 2013). When such a symptom abatement approach is considered in the context of physical injuries such as a broken bone, it is unsurprising that long-term issues persist following concussion. Treating only those symptoms that manifest following a concussion is the equivalent of only using Tylenol to treat a broken bone—the treatment would center around pain reduction but would fail to address the actual injury. While some injuries would heal and go away on their own; a sizable portion of individuals would exhibit persistent limitations or dysfunctions, eventually resulting in an inability to continue their normal activities or even impaired health. Fundamentally, this summarizes the current situation surrounding concussion and the emergence of persistent post-concussion syndrome, along with recognition that concussion is a risk factor for subsequent mental health issues (Guskiewicz et al., 2007; Rice et al., 2016, 2018; Solomon, Kuhn, & Zuckerman, 2016).

Although the determination of clinical recovery varies widely across clinical settings, it is generally based upon: (1) a reduction in symptom ratings to a marginal or pre-injury level, (2) a general absence of neurological dysfunction due to concussion (e.g., normative neurocognitive performance, adequate postural stability, lack of symptom provocation during cognitive/physical exertion or VOMS), and (3) a complete return to learning environments and activity (work/play) (Broglio et al., 2024; Patricios, Schneider, et al., 2023). Thus, a majority of concussed individuals

will return to their regular activities and exhibit marginal symptomatology within two to four weeks postinjury (Putukian et al., 2023). While such return to regular activity is popularly asserted as a reflection of clinical recovery—even if the individual still experiences some residual symptomatology; true physiological recovery extends far beyond this timeframe as evidence from more sensitive techniques has consistently observed long-term effects months to years following injury, even in asymptomatic presentations (Kamins et al., 2017). As many of the assessment approaches within this literature are essentially minor evolutions of 1940s era paper-and-pencil screening tools to detect gross-abnormalities in function, the question remains as to the clinical significance of more modern and sensitive assessment techniques. Specifically, if an individual presents with relatively normal functioning but still exhibits impairments on more sensitive assessments, it is unclear how clinical decision-making should change—as well as how willing individuals in sporting contexts may be to delay returning to full participation. The current literature further lacks explicit consideration of temporality, often relying on observed differences between individuals with (i.e., presumably deemed "clinically recovered") and without a history of concussion to extrapolate potential long-term effects, without assessing the persistence of these effects from injury onset. As modern approaches appear to be sensitive to alterations in the brain and cognition in response to a wide array of behaviors (e.g., caffeine, sleep, physical activity, etc.), some caution is warranted in directly attributing differences between groups to a single underlying cause.

2.2 Post-concussion syndrome

Nevertheless, within those individuals who incur a concussion, up to 30% may experience signs of protracted clinical recovery (Voormolen et al., 2018; Yeates et al., 2023). This condition, referred to as persistent post-concussion symptoms or Post-Concussion Syndrome (PCS)—the latter used for consistency herein, is broadly characterized by a symptom course lasting more than three months after initial concussion diagnosis (Mittenberg & Strauman, 2000). While referrals to specialized physicians and targeted interventions may be implemented to better manage PCS, the central assessment remains consistent with assessment recommendations for the subacute period (Yeates et al., 2023). Diagnosing PCS is as challenging as diagnosing concussion itself. As concussion symptoms are non-specific, when these symptoms appear well after the typical recovery timeframe it poses a substantial challenge in determining their biological underpinnings.

Indeed, up to 20% of healthy individuals report concussion-like symptoms consistent with PCS criteria (Asken, Snyder, Smith, Zaremski, & Bauer, 2017). Therefore, PCS diagnosis depends upon clinical discretion when evaluating an individual's recovery progress and confirming that their symptoms are not better explained by an extraneous underlying condition (s) (Harmon et al., 2019).

Extensive research has focused on identifying predictors of PCS and at-risk populations to facilitate earlier intervention, hopefully reducing its prevalence. An elevated risk of PCS is observed in females and older adults (Mavroudis, Balmus, Ciobica, & Hogas, 2024; Varriano et al., 2018), as well as in individuals with pre-existing mental health disorders (Broshek, De Marco, & Freeman, 2015; Morgan et al., 2015; Ponsford et al., 2019), somatization (Root et al., 2016), and headache/migraine disorders (Scott, Uomoto, & Barry, 2020). However, it should be noted that individuals with these risk factors may not necessarily develop PCS, and conversely, PCS may be developed absent these risk factors. In populations experiencing symptomatology which overlaps with that of concussion, clinical decision-making may be made more difficult. It has been suggested that one reason for the elevated risk of PCS in such populations may be a reflection upon individuals being prematurely cleared to return to school, work, or play as residual impairments/symptoms are misattributed (Bullard et al., 2022; Coffman et al., 2023). Importantly however, findings in Attention-Deficit Hyperactivity Disorder (ADHD) populations indicate that although ADHD-related and concussion-related symptomatology are similar, they exhibit distinct factor structures (Bullard et al., 2022). Such findings indicate that the elevated risk of PCS in individuals with ADHD is not a function of overlapping constructs being assessed and confused, but rather may reflect symptom exacerbation as a result of some other means. It has been proposed that concussion may trigger mechanisms in individuals with a natural predisposition to develop symptomatology in these domains, further exacerbating their symptoms (Broshek et al., 2015). Additionally, emotional distress (e.g., anxiety, fear, post-traumatic stress, or depression) associated with changes to habitual activities or injury outcomes may cause patients to attribute their difficulties erroneously to concussion (Wijenberg, Stapert, Verbunt, Ponsford, & Van Heugten, 2017; Wood, McCabe, & Dawkins, 2011).

The pathophysiological consequences associated with clinical symptom presentation generally resolve within the subacute period. Pre-existing characteristics or psychological factors that overlap with or exacerbate

symptomatology may contribute to seemingly prolonged concussion effects, representing a distinct syndrome, PCS. In contrast, the long-term effects reviewed herein are not detected by traditional clinical techniques during the subacute period and are presumed to be present even in cases of typical recovery. Nonetheless, our current understanding of what constitutes "typical" recovery is limited outside the context of clinical symptomology. Without extensive longitudinal data, it is difficult to determine whether PCS and the long-term effects of concussion are mutually exclusive from a pathophysiological perspective. Indeed, it may be that all post-concussion presentations exist along the same continuum of pathophysiological and symptomological recovery, influenced by a myriad of factors which determine the chronicity and severity of symptoms. While we believe that such a unified approach warrants further consideration, at present, PCS is viewed as distinct from other long-term effects of concussion and will be described as such our review of the literature.

2.3 Chronic traumatic encephalopathy and other neurodegenerative diseases

If the general public were asked, "What long-term effects do you associate with concussion?", "CTE" would likely be the most common response, with many mistakenly identifying its first letter as "concussion". However, the general public is not at fault for these misconceptions. Over the past decade, perceptions of this concept have been largely influenced by widespread alarming media reports and a blockbuster film titled "Concussion" which dramatized the discovery of Chronic Traumatic Encephalopathy (CTE) in former American Football players. During the same period, researchers failed to establish reliable evidence linking CTE to concussion history (Godbolt et al., 2014; Stein, Alvarez, & McKee, 2015). Considerable refinements have been made to CTE criteria and its proposed etiology in light of this new research (McKee, Stein, et al., 2023), yet nearly 70% of the general public still express concern that a single concussion event could increase one's risk of CTE (Daugherty & Sarmiento, 2018; Merz, Van Patten, & Lace, 2017; Salisbury, Kolessar, Callender, & Bennett, 2017; Zurlinden et al., 2024). As such, we believe it is imperative to describe the current view on CTE and its relationship to concussion to avoid past mistakes in knowledge translation.

Chronic Traumatic Encephalopathy is a rare neurodegenerative disease characterized by the abnormal deposition of tau, an essential structural protein found in neurons, within the frontal and temporal cortices, as well

as in crevices of the brain called sulci (McKee, Stein, et al., 2023). While at present CTE can only be diagnosed through post-mortem pathohistological examination, it is associated with a progressive array of nonspecific clinical features typically during mid-to-late adulthood (e.g., behavioral disturbance, suicidal behaviors, cognitive decline, and overt clinical dementia) termed Traumatic Encephalopathy Syndrome (TES) (Katz et al., 2021). Alarmingly, CTE has also been identified in contact-sport athletes who were younger than 30 years old at the time of their death (McKee, Mez, et al., 2023). The causal etiology of CTE is proposed to result from long-term exposure to repetitive head impacts, which may or may not include a history of concussion (McKee, Stein, et al., 2023). Proponents of this hypothesis argue that there is preponderance of evidence supporting it, citing that 97% of CTE cases occur in retired professional athletes from collision/contact sports (McKee, Stein, et al., 2023). However, critics have called for the abandonment of this hypothesis, arguing that it is overly reductionist (Barr, 2020; Carson, 2017; Malcolm et al., 2024), and highlighting the lack of empirical characterization in the diagnostic criteria for CTE and TES (Hazrati et al., 2013; Iverson & Gardner, 2020; Randolph, 2018; Terry et al., 2024). Studies heavily rely on brain donors, often lack control groups, and rarely account for additional lifestyle factors which may occur within these populations (e.g., alcohol, drug, or pain medication overuse, as well as psychiatric conditions) and simultaneously contribute to tau accumulation. Clearly, more diagnostic refinements and investigations into causal criteria are needed before a consensus is reached on whether repeated head impacts or concussion contributes to CTE/TES development.

Epidemiological evidence suggests that a history of concussion may confer an increased risk for other neurodegenerative diseases including amyotrophic lateral sclerosis (ALS) (Chen, Richard, Sandler, Umbach, & Kamel, 2007), Alzheimer's (Fleminger, Oliver, Lovestone, Rabe-Hesketh, & Giora, 2003; Mortimer et al., 1991; Schofield et al., 1997), Parkinson's (Jafari, Etminan, Aminzadeh, & Samii, 2013), and other dementias (Kalkonde et al., 2012; Lee et al., 2013; Rosso et al., 2003). In neurodegenerative tauopathies (e.g., CTE, Alzheimer's, and frontotemporal dementia), the accumulation of tau—and amyloid-β peptides in some diseases—deposits both intracellularly and extracellularly, forming aggregates called neurofibrillary tangles (Lee, Goedert, & Trojanowski, 2001). These aggregates are believed to be neurotoxic, triggering chronic inflammation and subsequent neuronal dysfunction, damage, or death—although it is important to acknowledge growing perspectives that the accumulation of tau and amyloid-β may be physiological

markers of neurotoxic environments rather than causal factors (Langworth-Green et al., 2023). Healthy individuals without these neurodegenerative tauopathies maintain functional waste-clearing mechanisms, inhibiting their accumulation (Nedergaard, 2013; Tanaka, Mizushima, & Saeki, 2012). For example, the glymphatic system, preferentially active during sleep, facilitates the clearance of brain waste by a convective flow of interstitial and cerebral spinal fluids through perivascular spaces (Nedergaard, 2013). Researchers have hypothesized that sleep disturbances following concussion can hinder glymphatic clearance, allowing for extracellular tau accumulation to propagate and thereby increasing the risk of developing neurodegenerative tauopathies (Giza & Hovda, 2014; Iliff et al., 2014; Medina & Avila, 2014). However, this theory does not help explain the increased risk of ALS or Parkinson's, and in order for it to be considered viable, further empirical evidence is required. Additionally, the current epidemiological findings are inconsistent in their conclusions (Helmes, Østbye, & Steenhuis, 2011; Mehta et al., 1999) and their studies are considered methodologically limited, as they rarely address other factors known to be important for brain health (Patricios, Schneider, et al., 2023).

3. Evidence of long-term functional alterations

Despite its apparent flaws, the current clinical assessment approach is particularly useful for categorizing symptom profiles following concussion and evaluating their interrelatedness. The clinical profiles model, popularized by Collins and colleagues, categorizes symptoms into five, often co-occurring, clinical profiles: (1) cognitive/fatigue (e.g., difficulty remembering/concentrating, memory problems, feeling 'slow'), (2) vestibular (e.g., dizziness, nausea, balance problems, motion sensitivity), (3) ocular (e.g., blurry vision, difficulty focusing, difficulty reading), (4) migraine (e.g., headache with nausea and/or phono/photosensitivity, visual aura), and (5) anxiety/mood (e.g., depression, irritability, anxiousness), with two modifying factors: (a) sleep (e.g., trouble falling asleep, sleeping more/less than usual) and (b) cervicogenic injury (e.g., neck pain or stiffness, headache originating toward the back of the head, numbness or tingling) (Collins, Kontos, Reynolds, Murawski, & Fu, 2014; Kontos et al., 2019). Such an approach enables clinicians to identify secondary symptom manifestations, allowing resources and therapeutic efforts to be allocated toward addressing the particular symptomological profile. For example, if a patient's symptoms primarily align

with the anxiety/mood profile but they also present with exaggerated headaches and sleep disturbance, the clinician may determine that there is a shared psychological explanation for the reported symptoms and focus the patient's treatment accordingly. Nevertheless, the clinical profiles approach is only as sensitive as the assessment tools used to inform it. If the assessment tools chosen to have suboptimal sensitivity, underlying or subclinical symptoms/profiles can go unrecognized, despite their potential to develop into long-term functional impairments if left unaddressed. As such, our review will be framed in the context of this significant gap, focusing on the flaws of more traditional assessment tools and detailing evidence from studies using experimental measures that appear sensitive enough to detect long-term alterations in four often-neglected symptom domains following concussion.

3.1 Mental health

Historically, concussion practices and guidelines have focused on ameliorating physical pain and other somatic symptoms, inadvertently overlooking the assessment of mental health status. An increased emphasis has been placed on mental health assessment more recently as nearly 1 in 4 concussed patients report psycho-affective symptoms (Fish et al., 2023; Iverson, Greenberg, & Cook, 2022; Kontos et al., 2019; Yang, Peek-Asa, Covassin, & Torner, 2015). Further, psychiatric difficulties are among the strongest predictors of protracted recovery and symptom severity following concussion (D'Alonzo et al., 2022; Daley et al., 2024; Iverson et al., 2017, 2022; Ponsford et al., 2012; Weber et al., 2018; Zemek et al., 2016). As such, mental health screening instruments, primarily focusing on the manifestation of anxiety, depression, irritability, and emotional instability, have become some of the most valuable tools within a clinician's arsenal.

Mental health status following a concussion does not solely reflect its pathophysiological consequences but is also influenced by pre-injury psychiatric difficulties (Yang et al., 2015) and non-specific injury effects, such as detraining, boredom, isolation, or other sociocultural factors (Putukian, 2016). Indeed, it is important to consider that secondary effects of injury could even exacerbate underlying mental health concerns as a result of activity restrictions, separation from peers or group members, and concerns about regaining the opportunity to resume typical daily activities. Ideally, psycho-affective symptoms would be treated uniquely based on their causal factors. However, mental health assessment following concussion is often constrained to just a few items on a symptom rating scale or a brief

questionnaire (Crawford, Wenden, & Wade, 1996; Echemendia et al., 2023; Kontos et al., 2020; Patricios et al., 2023). While the inclusion of such items reflects a considerable improvement over historic standards of care and can serve as an initial trigger for referring a patient for more focused follow-up assessment and care; such a limited characterization makes it challenging for clinicians to disentangle the heterogeneous factors contributing to psycho-affective symptomology. This approach ultimately contributes to inadequate treatment for a subset of patients, risking symptom persistence, exacerbation, or even temporary masking, with symptoms reemerging after patients are deemed "clinically recovered".

If one were to focus within the typical clinical recovery period (i.e., injury onset to full medical clearance), psycho-affective symptoms would appear to be transient, following a similar but slightly extended time course to other concussion symptoms (Covassin et al., 2023; Mainwaring et al., 2004; Sicard, Harrison, & Moore, 2021). However, higher rates of mental health disturbances are observed in individuals with a history of concussion, even years after the initial injury (Brett, Nelson, & Meier, 2022; Burns et al., 2024; Chrisman & Richardson, 2014; Izzy et al., 2021; Keightley et al., 2014; Ledoux et al., 2022; Russell, Walld, Bolton, Chateau, & Ellis, 2023). A recent meta-analysis found that individuals with a history of concussion had nearly 3.3 times greater odds of being diagnosed with depression compared to their non-concussed counterparts (Hellewell, Beaton, Welton, & Grieve, 2020). These increased odds were stable from 6 months to more than 10 years post-injury and consistent across biological sex, age at injury/assessment, and injury etiology. Thus, the trajectory of psycho-affective symptoms following concussion may not be transient; rather, it may follow a biphasic pattern, with symptom regression during the acute and subacute periods and reemergence around 6 months post-injury.

While this evidence certainly points toward an association between concussion history and mental health disturbance, much of the available research is limited by its cross-sectional design, and the presence of pre-injury mental health diagnoses cannot be ruled out, making it challenging to infer causality. Nonetheless, there is limited evidence supporting the notion that concussion is indeed a significant risk factor for depression and other affective disorders. Delmonico and colleagues, using a longitudinal matched cohort design, demonstrated that even concussed individuals with no prior history of affective disorders had greater odds of developing depressive disorders (OR = 1.9) and anxiety disorders (OR = 1.7) up to 48 months post-injury compared to uninjured controls (Delmonico, Theodore, Sandel, Armstrong, & Camicia, 2022).

Furthermore, select studies have observed a frequency-response relationship in contact sport athletes, where a greater lifetime history of concussion is associated with increased depressive symptom severity (Brett, Kerr, et al., 2022; Didehbani, Munro Cullum, Mansinghani, Conover, & Hart, 2013; Gouttebarge, Aoki, Lambert, Stewart, & Kerkhoffs, 2017; Kerr, Thomas, Simon, McCrea, & Guskiewicz, 2018; Pryor, Larson, & DeBeliso, 2016; Walton et al., 2021). The precise number of concussions required to produce a clinically significant increase in depressive symptom severity remains unknown. Generally, studies note that while one or two concussions marginally increase the likelihood of a concurrent depression diagnosis, the likelihood becomes significantly greater with three or more concussions (Guskiewicz et al., 2007; Kennedy et al., 2024; Kerr, Marshall, Harding, & Guskiewicz, 2012; Kerr et al., 2014). Such evidence could be interpreted by instead suggesting that a history of repetitive non-concussive head trauma is the true underlying cause; however, former contact athletes across all levels of competition do not exhibit an increased risk of psychiatric disorder (Bohr, Boardman, & McQueen, 2019; Deshpande et al., 2017; Deshpande, Hasegawa, Weiss, & Small, 2020; Iverson & Terry, 2022; Iverson, Merz, & Terry, 2021; Russell et al., 2020). The collective findings indicate that experiencing multiple concussions may have a cumulative and detrimental effect on mental health; yet it is important to note that not all studies observe such an effect (Hellewell et al., 2020; Kay et al., 2022) and athletes exhibit poorer recall of concussion history when they experience greater mental health difficulties (Brett, Kerr, et al., 2022). This phenomenon likely represents a more complex interaction between concussion history and pre-existing risk factors (Moore et al., 2023).

Moreover, depression does not manifest in isolation but is accompanied by other psycho-affective and neurocognitive changes following concussion. For example, impulsivity, either as a risk factor or a long-term effect, is correlated with concussion history (Beidler et al., 2021). Researchers have posited that impulsivity moderates the relationship between depression and suicidal behaviors, suggesting that while severely depressed individuals may report suicidal ideation, those who are both depressed and impulsive are at the greatest risk of attempting suicide (Dumais et al., 2005; Mann et al., 2009; Wang, Jiang, Cheung, Sun, & Chan, 2015). A recent meta-analysis found that individuals with a history of concussion were twice as likely to commit suicide compared to those without a history of concussion (Fralick, Thiruchelvam, Tien, & Redelmeier, 2016). Nevertheless, suicide remains relatively rare, as absolute risk ranged from 0.28%

to 0.59% among concussed individuals (Fralick et al., 2016). In the United States, middle school and high school students who sustained at least one concussion had 1.69- and 1.60-times greater odds of attempting suicide within the same year, respectively (Mantey, Omega-Njemnobi, Barroso, & Kelder, 2020; Mantey, Omega-Njemnobi, & Kelder, 2021). More recent analysis of the high school data indicates that these risks become more meaningful when accounting for concussion frequency and demographic risk factors (e.g., race, ethnicity, and biological sex) (Eagle et al., 2022; Kay et al., 2022). For example, Kay and colleagues found that male students with two or more concussions in the past year had double the odds of attempting suicide compared to those with a single concussion, however, a frequency-response relationship was not observed for female students (Kay et al., 2022). While an increased risk of suicide represents a particularly concerning long-term consequence of concussion, we reiterate that much of the current literature is still limited to cross-sectional studies.

Some researchers have hypothesized that post-concussion factors act as stressors for individuals with a diathesis toward psycho-affective disturbance, enabling the development of clinically significant mental health disorders after injury (Moore, Kay, & Ellemberg, 2018). While this potentially explains the association between concussion and the development of depression, anxiety, and other affective disorders, it does not account for the biphasic trajectory of psycho-affective symptoms. In addition to suboptimal assessment sensitivity and inadequate treatment, this unique time course may reflect that concussed patients are temporarily concealing their psycho-affective symptoms to expedite medical clearance. Of course, we cannot discount the additional possibility that participants recruited in these cross-sectional studies may have undiagnosed PCS, making them unrepresentative of most individuals with a history of concussion. Without more extensive longitudinal data, extant literature offers limited guidance for clinical practice in mental health assessment and treatment following concussion. At the moment, it is imperative to prioritize concussion prevention and ensure that individuals struggling with mental health issues following a concussion are provided with a comprehensive support system.

3.2 Sleep

Within the broader discussion of concussive injuries, a particularly dominant perspective is the view that "too much sleep" contributes toward worse outcomes following a concussion. Such views reflect pervasive misconceptions that sleep must be avoided or periodically interrupted to

prevent deterioration shortly after a concussion (Rieger et al., 2018). Yet, the extant evidence in this area has largely dispelled such ideas. In fact, acute pleiosomnia (i.e., requiring more sleep than before injury) has been proposed as a necessary behavior following concussion to maximize recovery processes and glymphatic clearance (Imbach et al., 2016; Sommerauer, Valko, Werth, & Baumann, 2013). Problematically, sleep-wake disturbances also occur with concussion, partially due to disruption of orexin/wake-promoting pathways following injury (Baumann et al., 2009; Maerlender et al., 2020). These interconnected factors and inconsistencies in communicating the importance of maintaining healthy sleep habits likely not only impair recovery but could also contribute to a cycle of long-term issues (Kureshi, Stowe, Francis, & Djalilian, 2023).

A common research approach to assess post-concussion sleep health involves using various symptom rating scales and questionnaires to evaluate dimensions such as **S**atisfaction with sleep, **A**lertness during waking hours, **T**iming of sleep, sleep **E**fficiency, and sleep **D**uration, together known as **SATED** (Buysse, 2014). As such, studies have consistently found that individuals with a history of concussion report poorer sleep quality, excessive daytime sleepiness, insomnia, and altered sleep duration; with prevalence ranging from 28% to 41% (Gosselin et al., 2009; Pillar et al., 2003; Theadom et al., 2015). Similar reports of sleep disturbances are observed at high rates (~29–72%) during the symptomatic period as well (Chan & Feinstein, 2015; Fisher, Wiseman-Hakes, Obeid, & DeMatteo, 2022; Hoffman, O'Connor, Schmidt, Lynall, & Schmidt, 2019; Howell, Oldham, Brilliant, & Meehan, 2019). Furthermore, prevalence appears to increase with prior concussion history (Blake, McVicar, Retino, Hall, & Ketcham, 2019; Bryan, 2013; Oyegbile, Dougherty, Tanveer, Zecavati, & Delasobera, 2020; Schatz, Moser, Covassin, & Karpf, 2011). These findings indicate that underlying sleep disturbances are inadequately addressed in clinical assessments. There may be several explanations for this oversight. First, inconsistent incorporation of all five SATED dimensions could contribute to ineffective management, as it would overlook the heterogeneity of sleep symptoms, allowing certain sleep-wake disturbances to remain untreated. Additionally, behavioral techniques, medications, and/or supplements (e.g., melatonin) prescribed by the clinician to abate symptoms associated with difficulty sleeping may temporarily alleviate sleep disturbances during the clinical recovery phase, only for these disturbances to resurface upon discontinuation. Whether these interventions continue to alleviate sleep-wake disturbances long-term is currently unknown.

Identifying which long-term sleep complaints are linked to objective changes in sleep-wake behavior is essential for determining necessary additions to clinical assessments and tailoring interventions to effectively address sleep-related issues. However, research utilizing actigraphy, a method for quantifying sleep-wake behavior through movement changes, indicates that longer total sleep time, prolonged nighttime sleep onset latency, and greater wake after sleep onset observed during the symptomatic period do not seem to persist past the typical recovery timeframe (Fisher et al., 2022; Hoffman et al., 2019; Khoury et al., 2013; Maerlender et al., 2020; Raikes & Schaefer, 2016; Stevens, Appleton, Bickley, Holtzhausen, & Adams, 2023). Indeed, individuals with a history of concussion exhibit only slightly lower sleep efficiency (~4%) compared to controls, along with small, inconsistent alterations in total sleep time, sleep onset latency, and wake after sleep onset (Barlow et al., 2020; Kaufman et al., 2001; Williams, Lazic, & Ogilvie, 2008). Multiple sleep latency tests, an objective measure of daytime sleepiness, do reveal quicker daytime sleep onset latency in those with a history of concussion regardless of subjective sleepiness (Imbach et al., 2015, 2016; Schreiber et al., 2008). The absence of objective differences in sleep-wake behavior, coupled with the presence of sleep complaints and increased daytime sleepiness, aligns with the features of nonrestorative sleep (Stone, Taylor, McCrae, Kalsekar, & Lichstein, 2008).

Undoubtedly, a lack of longitudinal research on the subject prevents inference as to whether these sleep-wake disturbances originate due to concussion itself or are exacerbated by other long-term sequelae, such as psycho-affective disturbances (Alvaro, Roberts, & Harris, 2013; Reynolds & Banks, 2010). Unfortunately, the poor alignment between subjective complaints and objective sleep-wake behavior may indicate poor sensitivity in detecting subtle, long-term effects of concussion with the current techniques (Allan et al., 2017; Barlow et al., 2020; Berger, Obeid, Timmons, & DeMatteo, 2017; Lan Chun Yang, Colantonio, & Mollayeva, 2021). As such, delving into the intricacies of the neurophysiological organization and structure of sleep (i.e., sleep architecture) throughout the night may provide a deeper understanding of concussion's long-term impact on sleep-wake processes and neurological function.

Polysomnography (PSG; a comprehensive sleep analysis utilizing EEG, actigraphy, muscle activity, and other physiological parameters) or EEG alone can capture the gradual transition of predominant patterns of sustained oscillatory brain activity and specific EEG events to classify stages

and features of sleep architecture (Berry et al., 2015). A number of investigations have begun to utilize such approaches to characterize patterns of sleep architecture related to TBI, with meta-analytic reviews indicating that reduced duration of NREM sleep stage 2 (N2) and increased duration of NREM sleep stage 3 (N3 or slow wave sleep) are observable more than 6 months after moderate to severe TBI (Mantua, Henry, Garskovas, & Spencer, 2017). Although such investigations have generally failed to observe consistent alterations in sleep architecture in individuals with a history of concussion, it is important to note the general paucity of evidence specific to concussion and that much of the literature is plagued by problematically small sample sizes which contribute to inconsistent findings.

Nevertheless, the pattern of sleep architecture observed following injury is consistent with restorative theories of sleep which suggest that the brain preferentially adopts greater time in slow wave sleep to support cellular repair and renewal processes. Synchronous firing of neurons during slow wave sleep is also associated with an influx of glymphatic fluids enlarging the perivascular spaces in the brain, facilitating waste clearance (Hablitz et al., 2020). Recent work has observed the presence of enlarged perivascular spaces in veterans with a history of TBI—including concussion, with their severity (i.e., number and volume) notably correlated with sleep complaints, diminished total sleep time, and reduced NREM sleep (Opel et al., 2019; Piantino et al., 2021). As enlarged perivascular spaces are thought to be a marker of impaired glymphatic clearance contributing to a potentially neurotoxic environment through the accumulation of interstitial solutes and waste; then the alterations in sleep architecture (i.e., greater time spent within slow wave sleep) may be an adaptive response or at least mechanistically linked to such impairments (Opel et al., 2019). Of course, replication is necessary, but exploring these associations may offer deeper insight into the pathogenesis of a variety of long-term consequences following concussion, particularly in relation to neurodegenerative processes.

Refined quantification techniques of sleep EEG enable researchers to examine the duration, frequency, and spatial distribution of oscillatory activities and EEG features within specific sleep stages, known as sleep microarchitecture. Sleep microarchitecture and its distinct features, such as k-complex and sleep spindle density, are thought to play a vital role in cognitive processing and sleep-dependent memory consolidation but have been minimally investigated following concussion (Astori, Wimmer, &

Lüthi, 2013; Cote et al., 2015; Djonlagic et al., 2021). Of the research conducted, individuals with a history of concussion exhibit subtle alterations in beta and delta power during sleep (Arbour et al., 2015; Khoury et al., 2013; Rao, Bergey, Hill, Efron, & McCann, 2011; Mantua, Mahan, Henry, & Spencer, 2015). The conflicting nature of these findings across different brain regions and sleep stages makes it challenging to ascertain whether changes in sleep microarchitecture are inherent to concussion or even linked to its cognitive deficits. The study of long-term effects of concussion on sleep is certainly in its infancy, necessitating extensive replication and further characterization; however, its potential insights into cognition and neurodegenerative disease underscore its importance as a pivotal area of research moving forward.

3.3 Cognition

Prominent dysfunctions with cognitive abilities, difficulty sustaining attention, and exacerbated symptoms associated with cognitive engagement have made the assessment of cognition following concussion a particularly popular area of study (Walton et al., 2022). Individuals with a history of concussion report heightened cognitive symptomology, showing an apparent dose-response relationship (i.e., more concussions sustained result in greater reported dysfunction) (Brooks et al., 2013; Cunningham, Broglio, O'Grady, & Wilson, 2020; Kaye et al., 2019; Register-Mihalik, Mihalik, & Guskiewicz, 2009; Walton et al., 2022). Yet, such cognitive complaints are non-specific and remain poorly characterized (Vynorius, Paquin, & Seichepine, 2016; Weber et al., 2018), making the use of symptom rating scales particularly ineffective for detecting objective cognitive impairment in the presence of more dominant comorbid factors such as elevated psychological distress or fatigue (Brett et al., 2023; Bryant et al., 2023).

To introduce objectivity, commercialized cognitive batteries or select neuropsychological measures are frequently integrated as central components of the clinical evaluation during the acute and subacute periods. The Immediate Post-Concussion Assessment and Cognitive Test (ImPACT) is among the most widely used computerized cognitive battery for concussion evaluation; however, concerns regarding its reliability and sensitivity beyond the acute period are warranted (Bruce, Echemendia, Meeuwisse, Comper, & Sisco, 2014; Kontos, Sufrinko, Womble, & Kegel, 2016). Indeed, extensive research demonstrates that individuals with and without a history of concussion generally perform similarly on the ImPACT and

other computerized batteries, regardless of number of prior concussions (Alsalaheen et al., 2017; Broglio, Pontifex, O'Connor, & Hillman, 2009; Brooks et al., 2016; Bruce & Echemendia, 2009; Collie, McCrory, & Makdissi, 2006; Dretsch, Silverberg, & Iverson, 2015; Martini, Eckner, Meehan, & Broglio, 2017; Olson, Brush, Ehmann, Buckman, & Alderman, 2018; Pontifex, O'Connor, Broglio, & Hillman, 2009; Rosenblum et al., 2020; Singh et al., 2014; Tsushima, Geling, Arnold, & Oshiro, 2016). A critical issue in this area is that although such measures provide standardized approaches, they often rely upon a very small number of trials to assess a given cognitive domain. Also, they often assess performance across a wide range of cognitive domains while also suiting individuals with varying cognitive abilities (e.g., 12 years old to 80 years old). As a result, such assessments compromise their ability to detect more subtle changes in performance in favor of identifying gross-level differences. Indeed, evidence indicates that the ImPACT fails to detect well-established diurnal variation in cognitive performance (Anderson, Elbin, Schatz, Henry, & Covassin, 2021). Despite their widespread popularity and adoption, it is important to acknowledge that it is unclear if such computerized cognitive batteries provide utility for clinical decision-making beyond detecting egregious performance degradation—which likely could be detected by clinical staff without such assessments, raising questions about their comparative value against cheaper and less burdensome symptom rating scales.

Such null findings within individuals with a history of concussion seem to extend to many traditional neuropsychological assessments as well (Bruce & Echemendia, 2009; Clough et al., 2018; Cunningham et al., 2020; De Beaumont, Brisson, Lassonde, & Jolicoeur, 2007; De Souza et al., 2021). While Moore and colleagues have identified that some standardized neuropsychological tests (e.g., Raven's Coloured Progressive Matrices, Wide Range Achievement Test-3, and Comprehensive Trail Making Test—trials requiring interference control) may be sensitive enough to detect cognitive alterations in children with a history of concussion (Moore et al., 2019), even these measures produce mixed findings (Little et al., 2016; Moser, Schatz, & Jordan, 2005). Importantly, domain-specific alterations across these studies demonstrate that higher-order cognitive processes such as executive control appear most likely to manifest with residual impairments associated with a history of concussion (Cunningham et al., 2020).

In particular, a number of investigations have detected persistent impairments associated with concussion across classic domains of executive

control. Specifically, impairments in inhibitory control (De Beaumont et al., 2009; Ellemberg, Leclerc, Couture, & Daigle, 2007; McGowan et al., 2019; Moore, Hillman, & Broglio, 2014; Moore et al., 2015; Ornstein, Haden, & Hedrick, 2004; Parks et al., 2015; Pontifex et al., 2009; Pontifex et al., 2012), working memory (Elbin et al., 2012; Hudac, Cortesa, Ledwidge, & Molfese, 2018; Ozen, Itier, Preston, & Fernandes, 2013; Sicard & Moore, 2022; Sicard, Moore, & Ellemberg, 2018; Sicard, Moore, & Ellemberg, 2019), and cognitive flexibility (McGowan et al., 2018; Redlinger, Sicard, Caron, & Ellemberg, 2022) have been observed months to years following injury. However, it is important to note that despite these findings' relative consistency, if the task is not carefully considered, even assessments of these domains of executive control may fail to detect these persistent cognitive deficits. Interestingly, two perspectives have been put forward in this regard to explain why utilization of a particular task might be sensitive while other similar tasks might fail to detect differences. Simply put, tasks which are not sufficiently difficult may enable the use of alternative strategies, processes, and even neural degeneracy to compensate for underlying cognitive difficulty (Parks et al., 2015). As a result, performance in an impaired individual is indistinguishable from that of a healthy individual. Tasks which require lower-order cognitive operations and tasks which are not adequately calibrated to avoid ceiling effects (performance at or near perfect/maximal levels) are unlikely to provide utility in understanding the long-term effects of concussion. While tasks that are inherently more effortful—such as executive control tasks—serve to tax neural resources fundamentally limiting the extent to which compensatory processing could suitably mask residual impairments in cognition; such tasks can also fail to observe effects if they are not sufficiently challenging for the population. This concept makes it difficult to implement measures of executive control within clinical batteries as tasks must be tailored to the population of interest. Critically, within the present literature and diagnostic battery approach, assessments of executive control often neglect such tailoring in favor of adopting a one-size fits all approach.

Alternatively, the failure of some executive control tasks to detect persistent impairments associated with concussion may be the result of the extent to which the task relies upon visuospatial attention (McGowan et al., 2018). Tasks such as the flanker task, attentional network task, spatial working memory tasks, and perceptually based cognitive flexibility tasks all rely upon intact visuospatial attention and are those tasks most commonly utilized within investigations observing long-term impairments; whereas

tasks such as the Stroop task, serial working memory tasks, and rule-based cognitive flexibility tasks—tasks which tap the same underlying aspects of cognition without being dependent upon visuospatial attention—have been observed to fail to detect residual impairments beyond the acute phase of concussion (McGowan et al., 2018). Accordingly, it is critically important to consider not just the overall domain of cognition being assessed, but also to consider the way in which the assessment is tapping that domain. Such an idea is not mutually exclusive with consideration of cognitive difficulty and sensitivity; but rather these perspectives align to reflect important aspects of the way assessments are designed that may impact upon their utility in this area of research.

Beyond the assessment of overt behavioral performance, event-related brain potentials (ERPs) offer insight into covert cognitive operations which appear sensitive to the long-term effects of concussion. The vast majority of research in this area has focused upon the P3 (also known as the P300 or P3b), which provides a neural index of the allocation of attentional resources during stimulus engagement (i.e., P3 amplitude) and stimulus classification and processing speed (i.e., P3 latency). Even when behavioral performance is equivalent between individuals with and without a history of concussion, studies consistently find that persistent reductions in the allocation of attentional resources, as indexed by decreased P3 amplitude (Baillargeon, Lassonde, Leclerc, & Ellemberg, 2012; Broglio et al., 2009; De Beaumont et al., 2007, 2009; Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000; Gosselin, Theriault, Leclerc, Montplaisier, & Lassonde, 2006; Gosselin et al., 2012; Lavoie, Dupuis, Johnston, Leclerc, & Lassonde, 2004; Moore et al., 2014, 2015, 2016; Ozen et al., 2013; Parks et al., 2015; Theriault, de Beaumont, Gosselin, Filipinni, & Lassonde, 2009), and delays in stimulus classification and processing speed, as indexed by P3 latency (De Beaumont et al., 2009; Gaetz, Goodman, & Weinberg, 2000; Gosselin et al., 2006; Ledwidge & Molfese, 2016), are apparent even years post-injury. Within a four-year longitudinal design, Clayton and colleagues observed that alterations in P3 amplitude were most dramatic during the acute phase of concussion and gradually diminished during the graded return-to-activity phase. Interestingly however, not only were alterations in the P3 still apparent when behavioral patterns had returned to pre-injury levels; but those individuals who failed to show normalization of the amplitude of the P3 following injury were also more likely to exhibit persistent symptomatology and were more prone to repeated concussions (Clayton et al., 2020).

Another prominent ERP component of interest is the error related negativity (ERN) which provides a neural index of action-monitoring processes signaling the need for further control (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Miltner et al., 2003). However, there is a paucity of investigations assessing this component within concussed individuals, and the studies that have been conducted present conflicting findings. While some evidence appears to indicate that persistent reductions in action monitoring as indexed by smaller ERN amplitude are apparent even years following a concussion (De Beaumont, Beauchemin, Beaulieu, & Jolicoeur, 2013; Moore et al., 2015; Pontifex et al., 2009), other findings have failed to observe any relationship (Larson, Clayson, & Farrer, 2012), or have observed greater action monitoring as indexed by larger ERN amplitude for individuals with a history of concussion (Olson et al., 2018). Although it is unclear what methodological differences in studies may have contributed to such discrepant findings, it is important to acknowledge that the ERN component has been observed to be particularly alterable in response to strategic initiatives. Thus, even minor alterations in instruction (e.g., emphasizing speed vs accuracy) and adaptative strategic control (e.g., proactive vs reactive control) can drastically alter how this component may manifest. Nevertheless, future investigation may benefit by assessing the extent to which this ERP component may exhibit diminished modulation in response to such goal representations in individuals with a history of concussion.

Although ERPs such as the P3 component appear particularly sensitive to the residual effects of a concussion, a critical question that has yet to be resolved is to what extent this information is clinically relevant. As articulated by Clayton and colleagues (2020), absent any other behavioral or symptom manifestation, should altered P3 amplitude be sufficient cause to delay returning an individual to work/play contexts? Arguably, at present there is little compelling evidence for such a perspective given the relative dearth of longitudinal evidence. However, as the ERP approach enables assessment of neural processes underlying our behavioral interactions with the environment, there may well be key relationships such as risk of subsequent reinjury or neurodegenerative implications that warrant careful consideration in such discussions.

3.4 Motor performance

Gross deficits in motor control (e.g., balance and gait difficulties, motor incoordination, and ataxia) are among the most immediate signs and quickest

resolving symptoms of a concussion (Guskiewicz, Ross, & Marshall, 2001; Patricios, Schneider, et al., 2023). While recent efforts have been made to enhance the clinical assessment of motor control following concussion, these assessments remain focused on identifying prominent balance and gait impairments and evaluating closely related sensory and vestibular dysfunction (Echemendia, Brett, et al., 2023; Mucha et al., 2014; Patricios, Schneider, et al., 2023). Given that many guidelines are oriented toward returning athletes or military personnel to their pre-injury performance levels with minimal risk of re-injury, healthcare practitioners are tasked with ensuring that patients can maintain sufficient motor performance during both physical and cognitive exertion throughout the return-to-activity phase (Patricios, Schneider, et al., 2023). Yet, evidence of an elevated risk of injury among individuals with a history of concussion would indicate that current practices are inadequate in identifying more subtle, persistent motor deficits (Gardner et al., 2024; McPherson, Nagai, Webster, & Hewett, 2019; Reneker et al., 2019; Wilkerson et al., 2021, 2024); and unfortunately, there is a dearth of literature in this area.

Of the research available, dynamic stability and gait parameters are the most well-studied and have been shown to be altered well beyond clinical recovery (Baker & Cinelli, 2014; Fino, Nussbaum, & Brolinson, 2016; Martini et al., 2011; Sosnoff, Broglio, Shin, & Ferrara, 2011). Although these findings provide valuable insight into long-term motor control after concussion, the clinical relevance of the complex measures needed to produce consistent findings (e.g., center of mass parameters and non-linear dynamics)—while promising (Chou et al., 2023; Johnston et al., 2019)—requires further investigation. Recent studies, utilizing tasks that more closely resemble the movement demands of sport (i.e., jump cut maneuvers and drop-landing maneuvers), observe distinct biomechanical patterns associated with a greater risk of lower extremity injury in individuals with a history of concussion (DuBose et al., 2017; Lapointe et al., 2018; Avedesian, Covassin, & Dufek, 2020). Although such tasks require advanced techniques, they could be particularly useful in clinical settings and additional research aimed at developing tasks that mimic the specific movement demands of various patient populations would further enhance their utility.

One hypothesis as to why traditional clinical tasks may fail to detect persistent motor control deficits is that concussed individuals can compensate for compromised neural efficiency when tasks impose little demand on the motor system. Indeed, dual gait tasks requiring divided attention and

those necessitating cognitive-motor integration are more likely to elicit observable motor control impairments in individuals with a history of concussion (Brown, Dalecki, Hughes, Macpherson, & Sergio, 2015; Howell, Beasley, Vopat, & Meehan, 2017; Howell, Osternig, & Chou, 2018; Howell et al., 2020; Hurtubise, Gorbet, Hamandi, Macpherson, & Sergio, 2016; Ketcham et al., 2019; Lapointe et al., 2018). In an effort to detect these alterations earlier in recovery, the SCAT-6 and SCOAT-6 have recently added a timed tandem gait task, as well as optional complex tandem gait and dual tasks, into their assessment batteries (Echemendia, Brett, et al., 2023; Patricios, Davis, et al., 2023). At present, it remains too early to determine whether the incorporation of these tools in their current form will lead to extended recovery times or even impose sufficient demand to capture more subtle motor control deficits. Additional longitudinal research will provide an opportunity to integrate higher-order measures of motor control into clinical settings and return-to-activity protocols. While this research will be crucial for understanding the chronicity and breadth of behavioral alterations in motor control following concussion, it lacks the ability to probe neurophysiological etiology.

Transcranial Magnetic Stimulation (TMS) is a unique neurophysiological approach to motor assessment that has greatly enhanced our understanding of the functional alterations within and between the primary motor cortices following concussion. While TMS is primarily known for its versatile therapeutic applications as non-invasive brain modulation technique, it is also capable of providing reliable measures of corticomotor function by assessing muscle activity in response to stimulation of the primary motor cortex (Lefaucheur, 2019). As such, TMS research has greatly enhanced our understanding of the functional alterations within and between the primary motor cortices following concussion. Such evidence has generally failed to observe any differences in the excitability of corticomotor pathways after injury (Davidson & Tremblay, 2016; King et al., 2019; Pauhl, Yasen, & Christie, 2022; Pearce et al., 2015; Pearce, Rist, Fraser, Cohen, & Maller, 2018; Stokes et al., 2020). That is, when a single TMS pulse is applied, the magnitude of responses to proportionally identical intensities and the minimum intensity needed to elicit a muscular response are comparable between previously concussed individuals and their healthy counterparts. However, a relatively consistent feature in individuals with a history of concussion is prolonged duration of the corticospinal silent period (cSP; i.e., the succeeding, brief interruption in muscle activity opposite the stimulated hemisphere), which indicates

increased intracortical inhibition (De Beaumont et al., 2009, 2011; King et al., 2019; Pearce et al., 2018; Tremblay, de Beaumont, Lassonde, & Théoret, 2011). Interestingly, when concussed individuals are symptomatic such alterations are not consistently observed (Pauhl et al., 2022; Pearce et al., 2015; Schmidt et al., 2021; Seeger et al., 2017).

Paired-pulse paradigms, which use two TMS pulses delivered at set intervals to the same location, help confirm these findings while offering a more nuanced view. Specifically, short- (2–3 ms) and long-interval (~100 ms) paradigms index different intracortical inhibitory mechanisms mediated by γ-aminobutyric acid (GABA) type A and B receptor activity, respectively (Sanger, Garg, & Chen, 2001). With the exception of a few studies conducted with PCS patients (Pearce, Tommerdahl, & King, 2019; Pearce et al., 2021), null findings in short-interval paradigms are quite common following concussion (Lewis, Hume, Stavric, Brown, & Taylor, 2017; Meehan, Mirdamadi, Martini, & Broglio, 2017; Pearce et al., 2015; Schmidt et al., 2021; Seeger et al., 2017). In contrast, long-interval paradigms demonstrate a similar pattern to cSP findings—both mediated by GABA type B receptor activity—whereby enhanced intracortical inhibition is exhibited in individuals with a history of concussion but not in earlier, more symptomatic periods (De Beaumont, Tremblay, Poirier, Lassonde, & Théoret, 2012; Lewis, Hume, Stavric, Brown, & Taylor, 2017; Pearce et al., 2015; Pearce, Rist, Fraser, Cohen, & Maller, 2018; Powers, Cinelli, & Kalmar, 2014; Tremblay, de Beaumont, Lassonde, & Théoret, 2011). While it is possible these results reflect a delayed manifestation of corticomotor effects, pathophysiological factors associated with clinical symptomology may instead have a masking effect, inhibiting consistent observation in recently concussed individuals and those with PCS (King et al., 2019; Pearce et al., 2020). Unfortunately, research early after concussion is fairly limited, as TMS can be particularly challenging for reliable clinical use due to its sensitivity to inter-individual variability and nuances in technique. More consistent deviations are evident in those with a history of multiple concussions and older individuals—or are at least more challenging to capture in youth—indicating that these effects may also be cumulative and impacted by age-related changes in neural reserve (De Beaumont et al., 2011; Stokes et al., 2020).

Inhibitory modulation does not appear to be confined to intracortical pathways either. Limited yet consistent findings reveal that interhemispheric processes are functionally altered in individuals with a history of concussion, as they display prolonged transcallosal conduction time

(i.e., duration for neural signals to propagate from the stimulated hemisphere across the corpus callosum to the opposite hemisphere) and reduced duration of the ipsilateral silent period (iSP; i.e., the succeeding, brief interruption in muscle activity on the same side of the stimulated hemisphere) (Davidson & Tremblay, 2016; King et al., 2019; Locke et al., 2020; Schmidt et al., 2021). Such findings may be unsurprising given the existing evidence of long-term microstructural alterations in the corpus callosum (Churchill et al., 2017a; Churchill, Caverzasi, Graham, Hutchison, & Schweizer, 2017b; Dean, Sato, Vieira, McNamara, & Sterr, 2015; Henry et al., 2011), but together suggest that transcallosal integrity is compromised both structurally and functionally in the long-term following concussion.

The extant TMS literature provides convincing evidence that enhanced GABAergic activity, particularly linked to type B receptor activity within the primary motor cortex, as well as inefficiencies in interhemispheric communication, are long-term effects of concussion. Yet, further investigations are required to determine whether these effects truly impair interrelated behaviors or simply represent compensatory mechanisms. Indeed, some studies note correlations among corticomotor metrics, task kinematics, and reaction time following concussion (De Beaumont et al., 2009; Pearce et al., 2015, 2020; Schmidt et al., 2021). However, their potential associations with motor control, motor learning, and bimanual or gait coordination (Fling & Seidler, 2012; Paci, Di Cosmo, Perrucci, Ferri, & Costantini, 2021; Sarwary, Wischnewski, Schutter, Selen, & Medendorp, 2018; Swanson & Fling, 2018), particularly in the context of increased injury risk, have not been examined. While this may seem to reiterate our calls for inquiry into the relationship between underlying physiological alterations and measurable behavioral outcomes, it is important to note that TMS techniques are uniquely capable of answering such research questions while also offering therapeutic applications as a neuromodulation tool, broadening its potential for use in our field.

4. Conclusion

This chapter highlights a clear misalignment between the clinical perception of concussion transience and the chronicity of its effects as documented in the current literature. The field is familiar with translational lapses between research, clinical, and public perception. However, in this case, we posit that such misalignment likely stems from the poor sensitivity

of traditional clinical measures in detecting subtle, long-term alterations following concussion. While we have reviewed studies on four important symptom domains, it's essential to acknowledge that long-term functional alterations extend beyond these domains and the techniques discussed herein. Indeed, a number of neuroimaging techniques (e.g., diffusion tensor imaging, magnetic resonance imaging, positron emission tomography, etc.), which were not covered in the current review, have also demonstrated sensitivity to persistent changes in individuals with a history of concussion (Henry, Elbin, Collins, Marchetti, & Kontos, 2016; Manley et al., 2017). While neurophysiological techniques are undoubtedly valuable from a research perspective for identifying the underlying pathophysiological consequences of concussion, they lack the standardization required for clinical decision-making. Furthermore, positive neurophysiological findings following concussion are often presented without any associated behavioral or symptomatic manifestations, rendering them clinically irrelevant within the current treatment approach.

One possible explanation for these seemingly inconsistent findings could be that the brain compensates for neurological inefficiencies following concussion, similar to how we compensate for declining cognitive efficiency as we reach older age (Reuter-Lorenz & Cappell, 2008). That is, the brain has the ability, through physical and functional redundancies which constitute an individual's reserve capacity (Stern, 2002), to reorganize or recruit alternate neural resources in response to brain damage, utilizing additional resources that might not typically be engaged to meet task demands. In many cases, neural compensation does not result in any behavioral alterations, and performance is maintained as it would be in a non-concussed individual. However, in situations where task demands require the recruitment of a sufficiently damaged network (e.g., the fronto-executive network) or need to be completed under resource-demanding conditions (e.g., high-intensity exercise, sleep deprivation, increased states of anxiety, or divided attention tasks), behavioral alterations and performance deficits can manifest (Ledwidge & Molfese, 2016; Sicard, Caron, Moore, & Ellemberg, 2021). This "Reorganization/Compensation" hypothesis explains why older adults often exhibit more frequent long-term effects of concussion compared to younger individuals. As age-related neurodegeneration advances, older adults with a history of concussion must compensate not only for the neural inefficiencies caused by the concussion but also those associated with aging. The inclusion of adolescent or young adult samples in many reviewed studies may mask long-term behavioral alterations due to concussion, given their enhanced

reserve capacity. Similarly, it explains why individuals with a history of multiple concussions may show a higher likelihood of long-term behavioral alterations. With each additional concussion, their reserve capacity diminishes, leaving fewer resources available for compensation. Examining the long-term effects of concussion under this model of reserve capacity can help us understand the conditions, tasks, and populations in which a history of concussion is most likely to result in clinically significant functional alterations. Nonetheless, the absence of clear causative indicators linking concussion directly to these long-term effects suggests that neurophysiological techniques may not be immediately necessary outside of research settings.

While the chapter does not cover the entire breadth of existing literature on concussion's long-term effects, it effectively underscores the importance of further investigation before considering significant changes to clinical concussion management. Yet, given the significant public fear surrounding the long-term effects of concussion and the imperative to address patients' desire for symptom relief regardless of the burden imposed, there may be a case for incorporating more sensitive techniques into clinical assessment as a supplemental tool or within targeted interventions as the outcome of interest. Such an approach would enable a more nuanced understanding of the chronicity of concussion effects, prompting a shift from symptom abatement to the implementation of more targeted treatments. The evidence of long-term effects following concussion is convincing, however, moving forward it is crucial to acknowledge the limitations inherent in defining causality, potential selection biases, and the challenge of disentangling effects directly caused by concussion from contaminate effects of other long-term sequelae. We encourage our readers to continue pursuing research avenues related to the long-term effects of concussion within their respective specialties, striving to illuminate this topic through rigorous investigation.

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