

LETTER TO THE EDITOR-IN-CHIEF

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THE WHOLE PIE? MAYBE— BUT LET'S COMPARE APPLES WITH APPLES

Smith and Schneider⁵ highlight the importance of consideration of a full spectrum of potential mechanisms underlying whiplash pain. Arguably, few would disagree, but there are some inconsistencies in their argument that require mention.

Smith and Schneider⁵ cite clinical pre-post studies of radiofrequency neurotomy to make their case that, presumably, targeting nociception (in this case, from the zygapophyseal joints) delivers stronger effects on pain and disability than psychological or combined psychological and physical interventions. As a basis for their argument, they have drawn conclusions from papers with disparate study designs, from which conclusions should not be drawn. The radiofrequency neurotomy studies^{3,4} were clinical pre-post studies with no control group, no placebo, no randomization, no blinding of patients, practitioners or assessors taking outcome measures, and no prospective published protocol or trial registration. The effects they note from these studies are within-treatment effects (change over time). They then try to compare these changes (over time) to between-treatment effects found in randomized clinical trials, including the StressModex trial.⁶ In that high-quality trial (Physiotherapy Evidence Database scale score [PEDro] of 8/10), physical therapist-delivered stress inoculation training and exercise were compared to an active control treatment of exercise alone. Despite Smith and Schneider⁵ stating that

the effect size was small, this is not correct: as the blinded analysis showed (and was reported), a medium to large between-treatment effect on the primary outcome of pain-related disability was found (Cohen's $d = 0.7$ immediately after the 6-week intervention, 0.52 at 6 months, and 0.66 at 12 months). They are not the first authors to confuse within- and between-treatment effects, and this was the topic of a recent review by Kamper¹ in *JOSPT*. In other words, Smith and Schneider⁵ are comparing apples and oranges in order to frame their argument.

Additionally, the authors⁵ proposed that different mechanisms be identified and prioritized in individual patients, and that these then be targeted. They base this argument on the epidemiological causal pie models, which are pie charts with each component cause as a slice.² The assumption made by Smith and Schneider⁵ is that predictors identified after whiplash injury (eg, posttraumatic stress symptoms, poor recovery expectations, higher initial pain) are in fact causal contributors to persistent pain and disability, though causation has yet to be proven. There is also the difficulty of extrapolating an epidemiological model to individualized patient care. How do the authors⁵ propose to decide which discrete factors contribute more than others to an individual patient presentation? They themselves acknowledge that these factors interact, likely in a very complex way. A further question would be whether these factors are actually discrete. This proposal hints back to a time of mind-body dualism, when physical and psychological factors were considered as totally separate entities. If it existed, such a solution to the extremely complex problem of musculoskeletal pain would be utopic; however, in our current state of knowledge, it is unrealistic.

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The author certifies that she has no affiliations with or financial involvement in any organization or entity with a direct financial interest in the subject matter or materials discussed in the letter.

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RESPONSE

We thank Professor Sterling for her interest in our *JOSPT* Viewpoint. Professor Sterling noted 2 factors of concern in our commentary.⁹ One involves the effect sizes demonstrated within patients undergoing radiofrequency neurotomy when compared to effect sizes demonstrated between participants undergoing rigorously controlled trials. We do not disagree that the observational clinical studies⁶⁻⁸ we referred to have limitations. Importantly, large between-group effect sizes have previously been demonstrated for cervical radiofrequency neurotomy performed in patients with whiplash-

associated disorder (WAD) within a placebo-controlled randomized controlled trial.³ Cervical radiofrequency neurotomy is also supported by systematic reviews^{2,5} for those with persistent cervical pain, and was the most effective treatment for chronic WAD¹¹ in a review of therapeutic interventions for WAD. We do not disagree with the methodologic rigor of the recent clinical trial performed by Professor Sterling and colleagues.¹⁰ Although this study demonstrated a medium to large treatment effect, the stress inoculation training and exercise treatment group continued to have mild to moderate disability (considering the standard deviations around the mean Neck Disability Index scores) 12 months post whiplash injury. Our commentary was not a direct comparison of these disparate treatment approaches. We aimed to highlight that different treatments, even when performed as rigorously as those reported by Sterling et al,¹⁰ do not necessarily address the totality of impairments (ie, physical and psychological manifestations) in some individuals with WAD, providing further support for our viewpoint that complex mechanisms are involved.

This brings us to Professor Sterling's second concern. In our exploratory pie model, we used preinjury¹ factors identified in registry studies and systematic reviews of postinjury clinical features¹² to hypothesize that a range of factors likely would need to be addressed to optimize recovery at the individual level. We highlighted that the component causes for each individual are unknown and, as such, are not discrete factors. On the contrary, they are in opposition to a mind-body dualism. This approach requires health care providers to consider collection of outcome measures in data sets that would allow machine learning/artificial intelligence to assist with providing

a solution at the individual level for those with WAD. A recent study demonstrated the utility of this approach to identify predictive factors for persistent pain in rheumatoid arthritis.⁴ Twenty-one different demographic, patient-rated, and clinical factors were evaluated in a registry data set of 288 individuals in Sweden. Using these factors, patients could be correctly assigned to 3 distinct patient subgroups (low, moderate, high) with 70% accuracy. The authors' conclusion was that "... machine-learning is suited to extract knowledge from data queried from pain and disease related registries."⁴

Our contention is that the best pie involves a mixture of all ingredients, and although the recipe is not quite known at this time, the collaborative expertise both within our profession and between our peers may provide the most satisfying outcome for all.

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A Treatment-Based Profiling Model for Physical Therapy Management of Patients Following a Concussive Event

Concussion is an injury that affects individuals across the life span and across all aspects of activity participation. Over a 5-year period, children with a concussion accounted for an estimated 2.9 million emergency department visits and 1.9 million outpatient visits.⁴¹ The US Department of Defense estimates that 84% of traumatic brain injuries sustained by military service members are concussions.¹⁶ The Centers for Disease Control and Prevention reported a significant increase in the number of fall-related traumatic brain injuries in older adults.⁵⁸ There are approximately 3.8 million sports and recreation-related concussions occurring in the United States every year.³³

In a recent survey of clinical practice patterns, one third of physical therapists reported that concussions in the majority of their patients were caused by non-sport-related mechanisms (eg, fall, motor vehicle crash, assault).⁶⁵ The

same survey noted that physical therapists care for patients with concussion in a variety of settings, including acute care, inpatient rehabilitation, outpatient clinics, and schools. The timing between concussions and the initiation of physical therapy varied between patients and ranged from less than 48 hours to greater than 3 weeks.⁶⁵

The physical therapist is a key member of the interdisciplinary concussion management team, with a primary role

of providing assessment and management for patients who currently have, or who are likely to develop, disabling signs and symptoms of a multifaceted biopsychosocial nature after a concussive event.³⁸ The role of the physical therapist in concussion assessment and management may vary based on practice setting (eg, on the field, outpatient, school), injury mechanism (sport versus nonsport), injury acuity (acute versus chronic symptoms), availability of other disciplines on the concussion management team (eg, athletic trainers, physicians, neuropsychologists), and the defined roles of interdisciplinary team members.

Research supports physical therapy interventions for patients with persistent symptoms after concussion.^{2,26,31,51,54} Consistent with the paradigm shift toward endorsing active rehabilitation strategies for concussion, active and early physical therapy interventions or exercise are safe, feasible, and may be effective.^{35,50,53} However, evidence regarding the ideal timing to initiate therapy, dosing parameters, and prioritization approaches in patients with overlapping impairments is limited. Because patients with concussion exhibit heterogeneous impairments and variations in the acuity and severity of post-

• **SYNOPSIS:** Concussions are a public health concern that affects individuals across the life span. The multifaceted effects of concussion warrant an interdisciplinary management strategy that may include physical therapy. However, physical therapists may feel underprepared for clinical decision making following a concussive event. We propose a new treatment-based profiling model to help physical therapists manage patients following a concussive event. This profiling model, based on symptom type and intensity, disability

status, and response to movement, prioritizes treatment emphasis on (1) symptom management, (2) movement system optimization, or (3) performance optimization. We consider contextual factors that modify treatment decision making and present examples of each treatment-based profile. *J Orthop Sports Phys Ther* 2019;49(11):829-841. doi:10.2519/jospt.2019.8869

• **KEY WORDS:** concussion, mTBI, physical therapy, whiplash

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concussion symptoms,⁶⁵ a comprehensive model of assessment and treatment is warranted. In a comprehensive model of assessment and treatment, physical therapists recognize that some patients may require medical management and some patients may be amenable to self-care management (TABLE 1). Physical therapists also recognize that some patients may exhibit postconcussive symptoms despite the lack of a formal concussion diagnosis.

This clinical commentary suggests a model to support the design and delivery of physical therapy interventions for patients following a concussive event by prioritizing the treatment emphasis to one of the following: symptom management, movement system optimization, or performance optimization. The proposed treatment-based profiling model considers a variety of assessment domains and is presented using exemplary cases. We intend our model to provide general guidance to physical therapists caring for patients following a concussive event. Although the domains proposed in this model transcend practice patterns and can be applied in a variety of settings, the model should not be interpreted as encompassing all proper methods of care. We recognize that standards of care are

subject to change as scientific knowledge advances and patterns of care evolve. Therefore, we encourage physical therapists to integrate the proposed model with other acceptable methods of care aimed at achieving the same outcomes.

LIMITATIONS OF CURRENT CONCUSSION MANAGEMENT STRATEGIES

IN THIS SECTION, WE OUTLINE 6 LIMITATIONS of concussion management strategies that are relevant to physical therapists involved in concussion management. We then propose some strategies that may help physical therapists be more effective in managing patients with concussion.

Focusing on the Concussion Diagnosis at the Expense of Assessing the Concussive Event's Effects on Multiple Body Systems

Many concussion management approaches emphasize the need to determine a medical diagnosis of concussion. However, the diagnosis of concussion remains a clinical diagnosis, with minimum diagnostic criteria that may not cover the spectrum of impairments experienced by all patients after a concussive event. The

advantage of a patient carrying the diagnosis of concussion is that the diagnosis is a common trigger for a more thorough evaluation. However, exclusive of sideline and emergent management of concussion, when identifying serious and life-threatening impairments is paramount, the drawback of some management models is a specific focus on the concussion diagnosis as a brain injury and a potential underappreciation of the multisystem effects (eg, sensorimotor, vestibulo-ocular, cervicogenic) that can result from a concussive event.^{19,26,34}

Patients with concussion may present with impairments to the cervicogenic, sensorimotor, and vestibulo-ocular systems.^{10,15,20,42} For this reason, it is important not to forget about the event that resulted in the injury. We encourage physical therapists to view the concussive event as a biomechanical traumatic incident that may result in multisystem stress that is further influenced by individual personal and contextual factors specific to each patient. This reconceptualization recognizes that the clinical manifestations of a concussive event occur when interactions between multisystem stresses, individual vulnerabilities, and environmental demands exceed a threshold specific to that person. Expanding the focus from the brain injury alone encourages the clinician to consider the multisystem nature of impairments occurring from the biomechanical event, while considering dynamic individual vulnerabilities and environmental demands, even in the absence of formal concussion diagnosis. Focusing on the concussive event is analogous to considering whiplash from a motor vehicle crash as an event rather than a diagnosis.

Anatomy and Pathology Do Not Always Directly Relate to Concussion Symptoms

Current concussion management strategies often emphasize a diagnosis based on pathoanatomy,^{15,32} increasing the possibility of conflating pathology in a body structure with symptom severity. Current tests for pathology do not always accurately

TABLE 1

CONSIDERATIONS FOR PHYSICAL THERAPISTS PARTICIPATING IN CONCUSSION MANAGEMENT

- Recognize that some patients require medical management following a concussive event
 - Red flags (eg, worsening headaches, repeated vomiting, cervical spine ligamentous instability)^{34,42}
 - Comorbidities (eg, previous concussion history, migraine, depression, attention deficit hyperactivity disorder, sleep disorders)⁹
 - Patients required to seek medical management because of legislative oversight (eg, adolescents in the United States sustaining a sport-related concussion)⁹
- Recognize that some patients may be amenable to self-care management following a concussive event (eg, low levels of contextual distress, no or controlled comorbidities)
- Recognize that the categories of medical management, physical therapy management, or self-care management are not mutually exclusive; they are intended to help you conceptualize whether the patient is appropriate for physical therapy
- Recognize that physical therapy can be combined with medical management for some postconcussive effects (eg, affective/mood effects, cognitive effects, migraine symptoms, oculomotor deficits), or with self-care management of some postconcussive effects (eg, fatigue, sleep disturbance)
- Recognize the constantly evolving nature of the multifaceted effects of a concussive event and the recommended best practices for management of patients following a concussive event
- Be familiar with clinical practice guidelines^{25,37,40,46,47} and consensus statements^{3,42} that outline the effects of injury and recommended interventions. These guidelines provide an overall understanding of the interdisciplinary landscape of concussion care, and following them will likely improve patient-centered outcomes

identify the source of signs and symptoms. If physical therapists can classify patients according to the interventions most likely to improve symptoms and function, they may be more efficient at developing and delivering personalized, targeted, high-quality care.^{1,12,13,17,21-23,61}

Different People Have Different Symptoms Following a Concussive Event

People respond differently after sustaining a concussive event. One-size-fits-all recommendations for return to activity are inappropriate, especially when initiated after watchful waiting for self-resolution of symptoms.⁴³ Being proactive and using a profiling model to target ongoing symptoms and impairments may help clinicians in providing targeted therapy interventions.^{15,20} In addition, it is essential to recognize that many patients sustain injuries to multiple systems after a concussive event and present with a variety of overlapping impairments, each of which warrants identification (FIGURE 1). This will help the clinician to prioritize interventions that target primary, secondary, and tertiary sources of symptoms.^{26,29,38}

Different People Recover at Different Rates

Normal clinical recovery is often characterized using a time-based approach.⁴² The most recent consensus statement on concussion in sport describes normal recovery as recovery occurring within 14 days in adults and within 4 weeks in children.⁴² However, these time frames have not been systematically studied across the life span (eg, very young children, middle-aged or older adults) and mechanisms of injury (eg, non-sport-related injuries), and may not apply to patients who have signs of impairment in various systems. Because recovery is often based on patient-reported outcomes, the influence of contextual factors may also play an important role in how a patient perceives and reports symptoms. We encourage a criterion-based model that uses a staging process to help the physical therapist and

patient prioritize interventions. Symptom status, disability status, and response to movement are criteria that may guide progression. A criterion-based model applies to the diversity of physical therapy practice settings in which patients are seen by a physical therapist, at time points ranging from less than 48 hours to 3 weeks or more following concussion.⁶⁵

The Role of Contextual Factors in Recovery From Concussion Requires More Attention

Existing concussion literature varies in its recognition of the role that contextual factors play in recovery.^{15,20} Although contextual factors affecting the recovery from concussion include social factors,^{49,52,55} cognitive factors,^{5,55} psychological factors,^{18,55} and factors related to general health and lifestyle,^{49,55} existing models have mainly focused on a subset of psychological vulnerabilities (eg, anxiety, depression) and concussion recovery,^{15,55} without much emphasis on other contextual factors. Contextual factors can play a protective role (ie, resilience) that facilitates recovery or a provocative role (ie, vulnerability) that negatively affects recovery from concussion (TABLE 2). However, current considerations of contextual factors are focused on provocative factors.

Many proposed concussion management strategies do not specifically address how the presence of various pro-

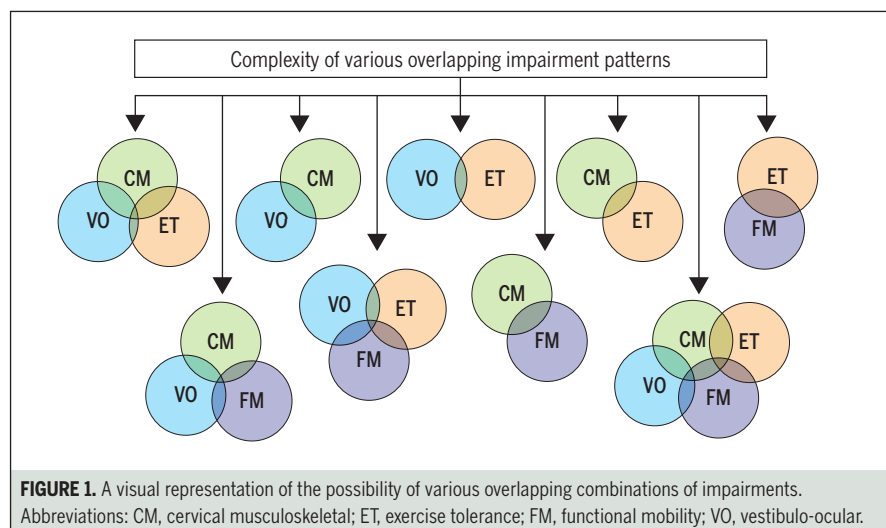
TECTIVE and provocative recovery factors can change the management of the individual patient. Our profiling model proposes a greater integration of the role of contextual factors in the clinical decision-making process.

Self-management Has an Underrecognized Role

We postulate that it is possible for an individual with low levels of contextual distress, who has no comorbidities or controlled comorbidities, to sustain a concussive event and not develop disabling symptoms that warrant medical and/or rehabilitation interventions. For these patients, self-management strategies that focus on education about favorable prognosis of nondisabling symptoms and progressive return to activities may be particularly beneficial. Some patients may successfully recover from concussive events without participating in physical therapy.

TREATMENT-BASED PROFILING MODEL OF PHYSICAL THERAPY MANAGEMENT

IN THIS SECTION, WE OUTLINE A PROFILING model of physical therapy management that is based on treatment provision (ie, treatment based). Our profiling model highlights the role of contextual



factors in recovery, and we provide exemplary patient cases. Our model prioritizes the treatment emphasis of physical therapy management into 3 profiles: (1) symptom management, (2) movement system optimization, or (3) performance optimization (**FIGURE 2**) by triangulating information obtained from 3 evaluation domains: symptom status, disability status, and response to movement (**FIGURE 3**). The interaction of profiles and evaluation domains dictates the needs of an individual patient and tailored care.

Evaluation Domains Used in the Treatment-Based Profiling Model

Symptom Status Complete a comprehensive evaluation of number, severity, onset, clustering, and triggers of self-reported symptoms through the following.

- Use a postconcussion symptom checklist, such as the Sport Concussion Assessment Tool symptom checklist,⁵⁶ Post-Concussion Symptom Scale,³⁶ or

Neurobehavioral Symptom Inventory,¹⁴ to quantify the following metrics.

- Total symptom score: the summation severity of all queried symptoms
- Positive Symptom Total: the number of experienced symptoms, regardless of intensity⁴⁴
- Global Severity Index: the severity of endorsed symptoms, controlling for the total number of queried symptoms⁴⁴ (total symptom score/number of queried symptoms)
- Positive Symptom Distress Index: the severity of endorsed symptoms, controlling for the total number of endorsed symptoms⁴⁴ (total symptom score/Positive Symptom Total)
- Divide symptom constellation into clusters such as cognitive, physical, emotional, or sleep constructs
- Gather information related to the common triggers for exacerbation of reported symptoms

- Evaluate symptom loading on possible sources of impairments (eg, vestibular, oculomotor, and cervical) and consider alternative self-report measures that probe these areas more in depth, rather than a more generic symptom scale, when signs and symptoms are in line with particular systems

- Evaluate whether currently reported symptoms preceded the concussive event. If symptoms were present before, then evaluate changes in frequency, severity, and triggers of symptoms since the concussive event

Disability Status Complete a comprehensive evaluation of perceived disability related to various aspects of movement-related postconcussive effects. Because concussive events can result in a heterogeneous presentation of signs, symptoms, and functional deficits, assessment of perceived disability cannot be achieved using a particular measure for all patients. Perceived disability can be evaluated by triangulating information from multiple outcome measures. Below, we list examples of outcome measures that could be used by physical therapists to characterize perceived disability. The list is intended to provide examples and is not all inclusive. We encourage physical therapists to consider the limitations of each of the measurements in their interpretation of its results.

- Child self-report Pediatric Quality of Life Inventory⁵⁹
- Perceived Quality of Life Scale⁴⁸
- Dizziness Handicap Inventory²⁸
- Neck Disability Index⁶⁰
- Convergence Insufficiency Symptom Survey⁸
- Barrow Neurological Institute Fatigue Scale⁶²
- Patient-Specific Functional Scale⁵⁷
- Headache Impact Test-6⁶
- Patient Health Questionnaire-9³⁰
- The Posttraumatic Stress Disorder Checklist⁷

Response to Movement Complete a symptom-guided physical examination to quantify the response to movement and to identify movement system deficits.

TABLE 2

PROTECTIVE AND PROVOCATIVE CONTEXTUAL FACTORS THAT MAY AFFECT RECOVERY AFTER CONCUSSION

Dimension	Protective/Resilience Factors	Provocative/Vulnerability Factors
Social ^{49,52,55}	• High socioeconomic status • Supportive family, work, or team relationship • Financial security • Educational attainment	• Low socioeconomic status • Poor family dynamic, negative work or team relationship • Ongoing litigations, entitlement, perceived injustice • Low educational level
Cognitive ^{5,55}	• High self-efficacy • Cognitive flexibility • Mindfulness • Positive beliefs • Strong academic performance	• Low self-efficacy • Catastrophizing behaviors • Stigmatization, maladaptive coping, hypervigilance • Negative beliefs • Poor academic performance
Psychological ^{18,55}	• Equanimity, perseverance, self-reliance • Meaningfulness • Existential aloneness, high self-reliance • Resilience¹⁸	• Depression • Anxiety, fear • High levels of stress, frustration, worry, grief • Lack of resilience
General health ⁴⁹	• Lack of comorbid health conditions • Lack of a past history of concussion, migraine, dizziness, falls	• Comorbid health problems such as chronic pain, chronic migraine, fatigue • Past history of concussion, migraine, dizziness, falls
Lifestyle ^{49,55}	• Physically active • Good sleep hygiene • Good conditioning, tolerant of progressive physical loading	• Sedentary behaviors • Poor sleep hygiene • Deconditioning, unpredictable response to physical loading

The scope and the extent of the physical examination are dictated by symptom exacerbation and perceived disability status and can include eye movement, head movement, neck movement, full-body movement, and/or visual-field movement.

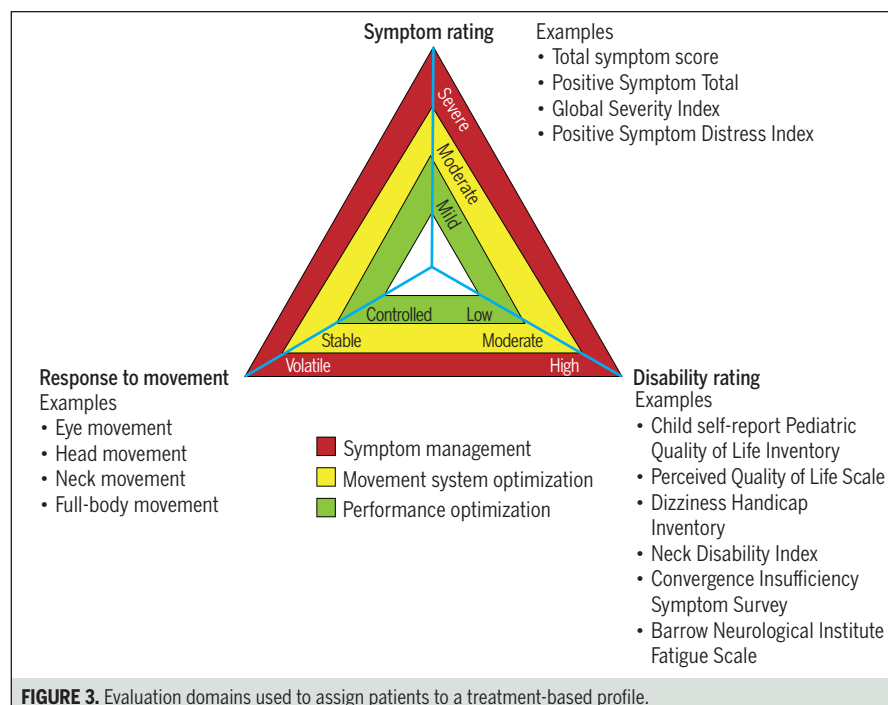
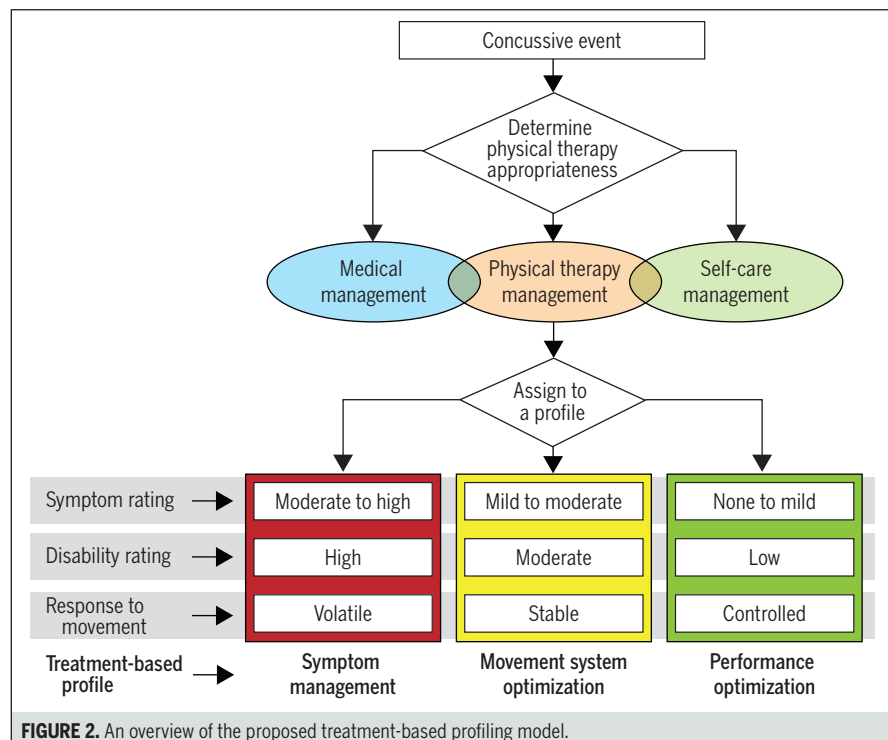
The goal of the physical exam is to quantify the patient's response to movement and to identify movement system deficits. Physical examination requires a thorough understanding of the human movement system, defined as "the collection of systems (cardiovascular, pulmonary, endocrine, integumentary, nervous, and musculoskeletal) that interact to move the body or its component parts."⁴ A movement system deficit can be attributed to insufficiencies in one or more systems. Therefore, a thorough physical examination is warranted, with particular emphasis on the systems contributing to the observed movement-specific deficit. Our profiling model avoids relying on identifying a pathoanatomical source of the deficits in favor of identifying signs associated with various systems contributing to the overall movement deficit.

Although the physical therapist will conduct the physical examination to quantify the patient's response to movement, the scope and extent of the physical examination can be modified based on the patient's symptom status and disability levels. The concept of irritability is useful here.³⁹ We use the term *irritability* in the context of the patient's response to movement, as opposed to an emotional state often associated with an injury suffered in a concussive event. Movement response irritability considers the vigor of the movement required to reproduce the patient's symptoms, the frequency of symptom provocation, the severity of the symptoms once provoked, how quickly the symptoms are provoked, which factors ease the symptoms, how much they reduce, and the time it takes for the symptoms to resolve. We propose the following terms to describe the response to movement after completion of the physi-

cal examination: *volatile*, *stable*, and *controlled* responses.

The response to physical examination in patients with a severe symptom rating and high levels of perceived disability is

volatile—characterized by exacerbation of symptoms with movement, regardless of the type, direction, and exertional demands of the movement. Patients with a severe symptom rating and high levels



of perceived disability can be assessed using a limited physical examination that mainly focuses on understanding symptom provocation and alleviation behaviors resulting from movement, rather than attempting to quantify specific movement deficits. The goal of the examination is to inform intervention choices that are mainly directed toward education, symptom management, and progressive resumption of activities.

The response to physical examination in patients with a moderate symptom rating and a moderate disability rating is more stable, characterized by exacerbation of symptoms after only a few movement triggers, with clear provoking and alleviating behaviors. Patients with moderate symptom and disability ratings can be examined using core standardized tasks pertinent to activities from their typical daily routine to quantify movement patterns and impairments. The goal of examination is to identify the source of movement system deficits (eg, cardiovascular, nervous, and musculoskeletal), which will help tailor the intervention strategy.

The response to physical examination in patients with low levels of symptoms and low levels of perceived disability is a controlled response, characterized by lack of symptom exacerbation or by transient exacerbation of symptoms only after sustained exertion. In patients with a mild symptom rating and low perceived disability, consider the demands of sustained performance upon return to work, school, sport, or military duty, and plan the physical examination accordingly. The goal of the examination is to identify patient-specific performance deficits and to guide progressive return to performance. New technologies such as wearables, telehealth, and virtual and augmented reality might help to bridge the gap between the clinical environment and the patient's environment.

Treatment-Based Profiles

Based on triangulating information related to symptom rating, disability rating, and response to movement, we prioritize

treatment emphasis into symptom management, movement system optimization, or performance optimization.

Symptom Management The symptom management profile can be used in patients who have experienced a concussive event and are currently exhibiting significant symptomatic features. Patients prioritized for the symptom management profile may present with a moderate to high symptom status, a high rating of perceived disability, and volatile or unpredictable nonspecific responses to movement where symptoms are aggravated, even in the absence of specific movement triggers. Patients may also have a high level of movement response irritability—a low tolerance for a movement-based clinical examination. Consequently, the scope and findings of an initial movement-based clinical exam may be limited due to the volatile and unpredictable response to movement. Patients profiled as symptom management may avoid environments with high sensory stimuli (eg, malls, schools, gyms, big crowds) and present with highly guarded movement patterns.

Patients prioritized for the symptom management profile may benefit most from interventions that emphasize effective symptom management and symptom-guided progressive resumption of activities. Individualized patient education for symptom management may emphasize reassurance that (1) the symptoms experienced are common and to be expected following a concussive event, (2) full recovery is expected in the majority of patients, and (3) occasional mild to moderate exacerbation of symptoms is expected and does not indicate harm to the brain or other systems. Education related to resumption of activities may emphasize that (1) return to activities is important but should be gradual and begin with resumption of daily activities; (2) mild but transient increases in symptoms are expected when returning to activities, although significant or prolonged exacerbation of symptoms is not conducive to recovery; and (3) environmental

accommodations for work or school can facilitate gradual resumption of activities.

Patients may receive multimodal physical therapy interventions to address specific symptoms (eg, neck pain, dizziness, and headache). These interventions may include subsymptom threshold exercises directed toward habituation. For example, a patient experiencing moderate to severe neck pain may have an early goal to reduce the frequency and intensity of the pain, as opposed to aggressively trying to improve range of motion. Similarly, for a patient with moderate to severe dizziness, the goal may be to reduce the frequency and intensity of dizziness through habituation exercises, as opposed to improving gaze stability via gaze stabilization exercises that may be appropriate at later stages (**FIGURE 4**). The patient's movement, function, and activity tolerance, with a gradual increase in intensity guided by the physical therapist and based on symptom response, should be assessed. As the patient at this stage improves, movement system optimization may be appropriate.

Movement System Optimization Patients who have mild-to-moderate symptoms and moderate disability that interferes with activities of daily living may be prioritized for the movement system optimization profile. These patients tend to have a more delineated and specific symptom profile: specific movements may trigger mild-to-moderate exacerbation of a limited number of symptoms, with a lower movement response irritability and return to baseline status shortly after the movement is completed. Specific movements or activities may modulate the symptoms of patients in this group. For example, a patient may have mild dizziness throughout the day that is exacerbated with simultaneous movement of the head and eyes but resolves to a mild level soon after head and eye movement stops.

Patients prioritized to this treatment profile require education focused on specific movements and activities that provoke their symptoms. Education may

emphasize that a mild-to-moderate exacerbation of symptoms with specific movement is to be expected, and reassure the patient that once exacerbated, the symptoms will resolve relatively quickly, and that symptom provocation is not harmful. An increase in symptoms is sometimes needed to promote recovery via habituation. For example, in a patient with mild-to-moderate levels of dizziness and reduced gaze stability, gaze stability exercises such as vestibulo-ocular reflex pattern 1 can be prescribed at an intensity appropriate to cause a mild and transient increase in dizziness.³

Multimodal treatment strategies may also be used to address deficits in one or more of the systems required for effective and efficient human movement. Examples of interventions include manual therapy for the cervical spine and exercises (aerobic, eye-head coordination, habituation, range of motion, strength, sensorimotor, or balance). Resumption of daily activities may identify more challenging situations that provoke symptoms. Careful assessment of those situations to identify specific provocative factors will facilitate a tailored and progressive plan of care (FIGURE 4).

Performance Optimization The performance optimization treatment emphasis profile may be most appropriate for patients who are relatively asymptomatic at rest and with activities of daily living but who need to be asymptomatic at higher levels of physical performance (eg, athletes, military personnel). These patients exhibit performance deficits with sustained exertion that can occur with or without symptom re-emergence. An athlete may no longer have dizziness at rest, during activities of daily living, or during routine exercises. However, the athlete's speed and power are impaired after sustained exertion upon return to sport, with or without re-emergence of symptoms.

Interventions would maximize the patient's physical performance at higher levels of sustained physical exertion (FIGURE 4). Patients who have recently sustained

a concussive event are at greater risk of subsequent injuries.^{11,24,27,45} Matching patients to a performance optimization profile may reduce the risk of subsequent injuries following a concussive event. Although addressing specific impairments, such as mobility and strength, may continue to play a role in treatment for patients in this profile, targeted interventions mainly consist of multidirectional sport- or work-specific activities. Training parameters may emphasize progressive loading and incremental increases in complexity of integrating simultaneous use of multiple systems. These parameters require mapping of the frequency, intensity, time, and type of given exercises to the specific demands required for the patient to return to peak performance status.

Considerations for Application of the Proposed Treatment-Based Profiling Model

Patients presenting to physical therapy following a concussive event may have coexisting impairments related to symp-

toms, movement system deficits, and performance deficits. However, the relative contribution of these deficits to the overall clinical presentation can vary, which is the rationale for prioritizing the treatment emphasis. As the patient's clinical status changes, treatment priorities should be updated to reflect changes in the relative contribution of symptoms, movement system deficits, and performance deficits to the overall clinical presentation. For example, a patient who is initially treated with interventions consistent with a movement system optimization profile due to moderate levels of symptoms and disability can move on to performance optimization interventions (FIGURE 4). A patient who initially receives treatments consistent with a movement system optimization profile might shift to receive more interventions for symptom management if his or her status deteriorates, while continuing treatment in the movement system category.

While there are many potential treatment options within each of the 3 treatment-based profiles, appropriate

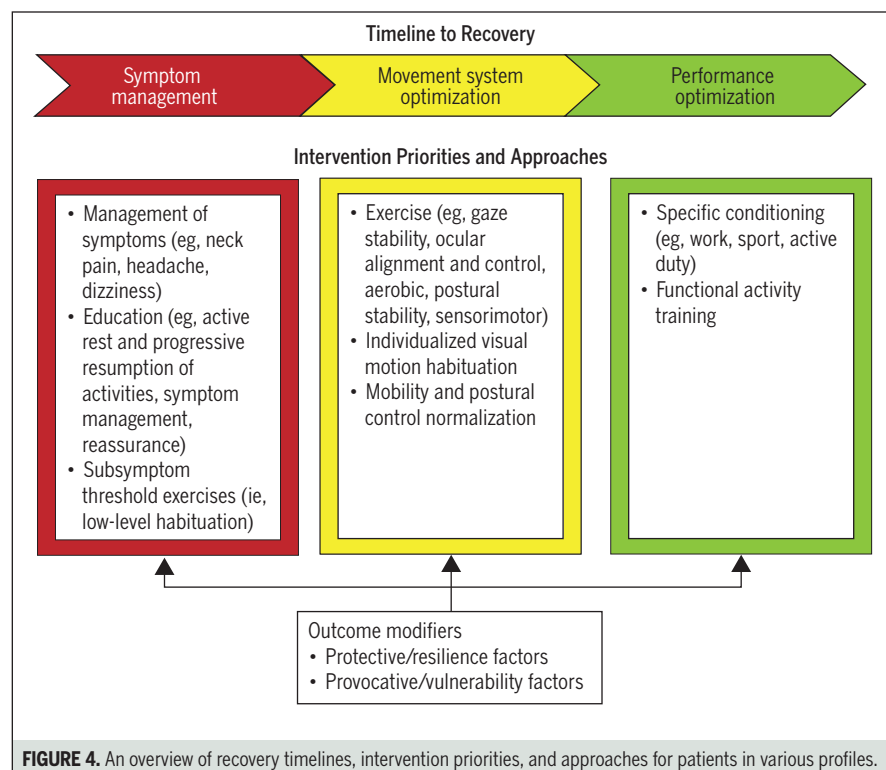


FIGURE 4. An overview of recovery timelines, intervention priorities, and approaches for patients in various profiles.

prioritization and sequencing of interventions must be considered. For instance, a patient treated with movement system optimization to address gaze stability and cervical mobility impairments may benefit from addressing cervical mobility and pain first,⁵⁴ given that full pain-free cervical range of motion is required for effective gaze stability training, and that gaze stability exercises may exacerbate cervical pain.

Application of Our Treatment-Based Profiling Model and Integration of Protective and Provocative Contextual Factors

We encourage physical therapists to adopt a practice model that goes beyond physical impairments and a treatment philosophy that incorporates protective and provocative contextual factors into patient management. Underpinning a context-informed practice model are factors identified at the individual level using patient-centered interviewing methods. These factors are then addressed through therapist reinforcement and delivery of context-informed

interventions focused on education and behavioral change.⁶⁴ In **TABLE 3**, we present exemplary cases to illustrate how to apply our model. We also illustrate how to incorporate protective and provocative contextual factors into patient management.

CONCLUSION

WE PRESENTED A TREATMENT-based profiling model for patients following a concussive event that addresses the limitations of existing strategies in directing the clinical decision-making process. Our model recognizes the concussive event as the presenting problem, resulting in physiological and psychological responses to what should be considered a tissue-based and stress-based interaction. The response to this traumatic event will be modulated by coexisting biological, psychological, and contextual factors, leading to a complex clinical presentation and a multidimensional recovery process. The proposed profiling model avoids the challenge of identifying the pathoanatomical cause

of symptoms, although observable signs of impairment are important to identify system involvement. Self-reported symptoms and disability and response to movement facilitate treatment-based profiles. Because risk factors are context and person dependent, we recommend that physical therapists embrace a context-informed practice approach, using risk identification practices to inform clinical decision making and treatment choices.

Perspectives on Future Research Directions

Research investigating rehabilitation interventions following a concussive event is in its infancy. There is an opportunity to structure research priorities and approaches to avoid common challenges that have hampered successful advancement of physical therapy practice across multiple diagnoses (eg, whiplash-associated disorders,⁶³ low back pain²²). The challenge for clinicians and researchers working with patients following a concussive event is not merely to find out which treatment will work; it is the concep-

TABLE 3

APPLICATION OF THE PROPOSED PROFILING MODEL USING EXEMPLARY CASES

	Case 1	Case 2	Case 3
History	- A 32-year-old woman with a chief complaint of headache (4/10) and neck pain (3/10). She is a data analyst who sustained a concussive event after slipping on ice 2 wk prior - She did not lose consciousness or memory. She was transferred to the emergency department and was diagnosed with possible concussion. Imaging findings were unremarkable - She was advised to rest and follow up with her primary care provider if her symptoms continued to persist after 1 wk - She followed up with her primary care physician, who referred her for physical therapy - Her job requires long hours working at a computer. She is unable to work more than a few hours at a time - She is a single mother of 2 children, aged 3 y and 1 y	- An 18-year-old man with a chief complaint of dizziness (3/10) and headache (4/10). He is a high school student-athlete on the varsity basketball team. A week prior, he sustained a concussive event after being elbowed during a basketball game - He was diagnosed on the sideline by the school athletic trainer and was referred to a concussion clinic - After being evaluated by a sport neurologist, he was referred for physical therapy - He reported having had 2 concussions prior to this one	- A 22-year-old female college student with chief complaints of fatigue (2/10), feeling nervous (2/10), and headache (2/10) - She is an undergraduate student who plays in a soccer club at college - She sustained a concussive event 2 wk ago after she hit her head against a bookshelf while studying at the library - She developed symptoms during the next few days that included headache, sleep problems, and fatigue - She was seen at the student health center on campus and was advised to follow up with a tertiary concussion clinic at an academic medical center affiliated with her college, but had not been seen yet due to lack of available times that do not conflict with her class/practice schedule - She was seen by a physical therapist during a wellness and performance clinic available for students participating in club sports

Table continues on page 837

TABLE 3

APPLICATION OF THE PROPOSED PROFILING MODEL USING EXEMPLARY CASES (CONTINUED)

	Case 1	Case 2	Case 3
Symptom rating	- Total symptom score: 60/132 - Symptom clusters* - PST: 18 - GSI: $60/22 = 2.7$ - PSDI: $60/18 = 3.3$ - No clear symptom-clustering pattern and no clear association with specific pathoanatomical sources - Aggravating factors: symptoms are exacerbated by any movement and by working on her computer - Severity - Headache, 4/10 - Neck pain, 3/10 - Irritability: headache and neck pain can worsen to 9/10 - Alleviating factors: headache is reduced to moderate levels after a few hours by wearing sunglasses, staying at home, and sleeping - Headache and neck pain can be reduced to 1/10 after a good night of sleep - Pre-existing conditions: she reports a pre-existing history of neck pain and migraine headache. However, this headache is different and is located "behind the eyes"	- Total symptom score: 60/132 - Symptom clusters* - PST: 9 - GSI: $60/22 = 2.7$ - PSDI: $98/9 = 10.9$ - Possible clustering of symptoms into physical and cognitive clusters: he reports headache, dizziness, blurred vision, double vision, sensitivity to light, feeling slowed down, feeling like being in a fog, difficulty concentrating, difficulty remembering, and "don't feel right" - Aggravating factors - Headaches: walking the school hallway, eating in the cafeteria, and worse toward the end of the school day - Dizziness: busy visual environment, lots of head movements - Severity - Headache, 4/10 - Dizziness, 3/10 - Irritability: headache can gradually worsen to 7/10 but only occasionally reaches this level. Dizziness can worsen to 7/10 but quickly reduces if he stops moving his head or visual environment is stabilized - Alleviating factors: headache is reduced with over-the-counter medications. Dizziness is reduced to mild levels on nonschool days and in the morning - Pre-existing conditions: no pre-existing symptoms	- Total symptom score: 6/132 - Symptom clusters* - PST: 3 - GSI: $6/22 = 0.27$ - PSDI: $6/3 = 2$ - She reports headache and feeling nervous - No clear clustering - Aggravating factors: no clear symptom exacerbation triggers or relieving factors - Severity - Headache, 2/10 - Feeling nervous, 2/10 - Fatigue, 2/10 - Irritability: headache can worsen to 5/10 after prolonged practices and in games. Feeling nervous can worsen to 4/10 during games. Fatigue can worsen to 7/10 after games - Alleviating factors: headache, feeling nervous, and fatigue are reduced to minimal by taking breaks during practices or by being replaced during games - Pre-existing conditions: she reports a pre-existing history of anxiety
Disability rating	NDI, 70% HIT-6, 66	DHI, 40 CISS, 30 HIT-6, 55	BNI-FS, 35/70 PSFS, 6/10 while participating in a 2-h soccer practice HIT-6, 40
Response to movement	Physical examination revealed: - Eye movement: no exacerbation of symptoms - Neck movement: - Exacerbation of neck pain (7/10) and headache (6/10) immediately with active movements. Neck pain eased after 1 min; headache persisted past several minutes - Passive segmental movement deferred due to pain - Head movement: exacerbation of dizziness (7/10) after 10 s, lasting 35 s - Visual-field movement: exacerbation of headache (5/10), dizziness (6/10), and balance lasting longer than 1 min - Full-body movement: not completed due to patient intolerance	Physical examination revealed: - Eye movement: exacerbation of headache (6/10) - Neck movement - No exacerbation of symptoms with active or passive ROM - Passive segmental movement is full and pain free - Weakness and poor endurance noted in cervical and capital flexors and extensors - Sensorimotor exam: poor ($>4.5^\circ$ error) cervical joint positioning - Head movement: exacerbation of dizziness (5/10). No change in headache - Visual-field movement: exacerbation of dizziness (6/10), headache (5/10), and blurred vision (5/10) - Full-body movement: exacerbation of dizziness (5/10) and light-headedness (4/10) - All symptoms return to baseline in 5 to 15 s	Physical examination revealed: - Eye movement: no exacerbation of symptoms - Neck movement - No exacerbation of symptoms with active or passive ROM - Passive segmental movement is full and pain free - Weakness and poor endurance noted in cervical and capital flexors and extensors - Sensorimotor exam: poor ($>4.5^\circ$ error) cervical joint positioning - Head movement: no exacerbation of symptoms - Visual-field movement: no exacerbation of symptoms - Full-body movement: no exacerbation of symptoms - Transient exacerbation of headache and fatigue is observed after practicing soccer drills, especially during feint and dribble, running with ball control, V runs, and during receiving and turning drills - Tolerates 30 to 45 min of drills - Symptoms ease after a few minutes' rest - A notable reduction in explosiveness is observed during roll and step to inside/outside cut, and during multidirectional hurdle jumps

Table continues on page 838.

[CLINICAL COMMENTARY]

TABLE 3

APPLICATION OF THE PROPOSED PROFILING MODEL USING EXEMPLARY CASES (CONTINUED)

	Case 1	Case 2	Case 3
Assigned profile	Based on the information above: • Symptom rating: moderate • Disability rating: severe • Response to movement: volatile This patient is profiled to symptom management (FIGURE 5A)	Based on the information above: • Symptom rating: moderate • Disability rating: moderate • Response to movement: stable This patient is profiled to movement system optimization (FIGURE 5B)	Based on the information above: • Symptom rating: mild • Disability rating: mild • Response to movement: controlled This patient is profiled to performance optimization (FIGURE 5C)
Contextual factors [†]			
Social	• A single mom with 2 children, aged 1 y and 3 y • Supportive team leader willing to have her work from home Assessment: 5	• Low socioeconomic status • Educational level: high school student (senior) • Lives at home; both parents work full-time Assessment: 4	• Low socioeconomic status • Educational level: some college classes • Strong family ties and support Assessment: 3
Cognitive	"I know that I can get through this, but I am trying to make sure that going through recovery does not impact my performance at work and my time with my children" Assessment: 4	"This is my third concussion, and I feel that I should not play basketball anymore if these concussions are going to damage my brain" Assessment: 8	"I know that I need to be in control of my destiny when it comes to overcoming fatigue and feeling nervous while playing" Assessment: 2
Psychological	"I am frustrated and angry that symptoms are continuing 3 wk after I fell. When I think about that, I get depressed. I'm afraid this will never resolve, because I have a friend who hit her head and is still suffering, and it's been 6 mo" Assessment: 8	"I am depressed because I will not be able to participate in college tryouts. If I do not get an athletic scholarship, I am not going to college. I can't stop thinking about missing that chance" Assessment: 8	"I am so aware now that I will get fatigued and nervous the longer I play" Assessment: 5
General health	Pre-existing history of migraine and neck pain Assessment: 6	Pre-existing issues: this is his third concussion Assessment: 4	Pre-existing history of anxiety Assessment: 4
Lifestyle	• Sedentary behaviors • Only 2-3 h of sleep at a time; takes 15-20 min to get back to sleep Assessment: 9	• Physically active, good physical conditioning • Sleeps 8-9 h; wakes up occasionally but able to return to sleep Assessment: 2	• Physically active • Sleeps through the night except for bathroom visit. Returns to sleep quickly Assessment: 2
Summary of contextual factors	FIGURE 6A	FIGURE 6B	FIGURE 6C

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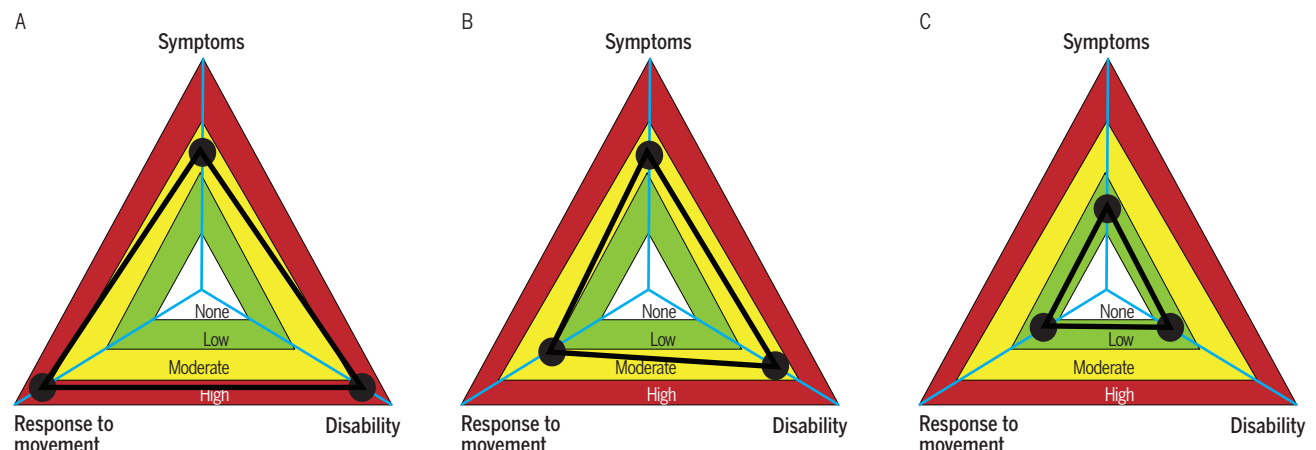


FIGURE 5. Assignment of patient cases (A) 1, (B) 2, and (C) 3 to a treatment-based profiling model based on symptom status, disability status, and response to movement.

TABLE 3

APPLICATION OF THE PROPOSED PROFILING MODEL USING EXEMPLARY CASES (CONTINUED)

	Case 1	Case 2	Case 3
Overall assessment	Patient is profiled to symptom management. She also exhibited high levels of contextual provocative factors, mainly related to psychological and lifestyle factors	Patient is profiled to movement system optimization. He also exhibited moderate levels of contextual provocative factors, mainly related to psychological and cognitive factors	Patient is profiled to performance optimization. She also exhibited low levels of provocative contextual factors related to psychological and general health factors
Treatment strategy	• Education that emphasizes - Positive recovery outcomes after concussion - The role of positive expectations in recovery - Importance of sleep hygiene to recovery - Pacing of work and family tasks - Capitalizing on supportive work environment by working from home, taking frequent breaks, etc • Gradual resumption of daily activities and light activities (ie, walking) • Symptom-guided exploration of limits of movement tolerance	• Education directed to the patient's statements and that emphasizes - The role of positive expectations in recovery - The longer duration of postconcussive symptoms does not always correlate to worse long-term brain health - Resumption of daily activities and subsymptom threshold activities - Transient mild exacerbation of symptoms is expected with therapeutic exercises • Exercises that include - Brock string/pencil-pushups for convergence insufficiency - Gaze stability exercises (VOR pattern 1) - Visual motion habituation exercises (VOR cancellation) - Cervical strength and endurance - Head repositioning accuracy	• Education that emphasizes - Fatigue is common after concussive events and can be attributed to prolonged rest and deconditioning, as opposed to being an effect of concussion - Prescribed exercises can be viewed as part of reconditioning • Exercises for - Cervical strength and endurance - Head repositioning accuracy • Soccer-specific drills, including feint and dribble, running with ball control, V runs, and receiving and turning drills • Soccer-specific exercises for explosiveness, such as roll and step to inside/outside cut and multidirectional hurdle jumps

Abbreviations: BNI-FS, Barrow Neurological Institute Fatigue Scale; CISS, Convergence Insufficiency Symptom Survey; DHI, Dizziness Handicap Inventory; GSI, Global Severity Index; HIT, Headache Impact Test; NDI, Neck Disability Index; PSDI, Positive Symptom Distress Index; PSFS, Patient-Specific Functional Scale; PST, Positive Symptom Total; ROM, range of motion; VOR, vestibulo-ocular reflex.

*PST: the number of experienced symptoms, regardless of intensity. GSI: the severity of endorsed symptoms, controlling for the total number of queried symptoms ($GSI = \text{total symptom score}/\text{number of queried symptoms}$). PSDI: the severity of endorsed symptoms, controlling for the total number of endorsed symptoms ($PSDI = \text{total symptom score}/PST$).

[†]To illustrate the contribution of contextual factors, we scored each category on a Likert scale (0-10). In general, higher scores indicate factors that are more provocative and lower scores indicate factors that are more protective.

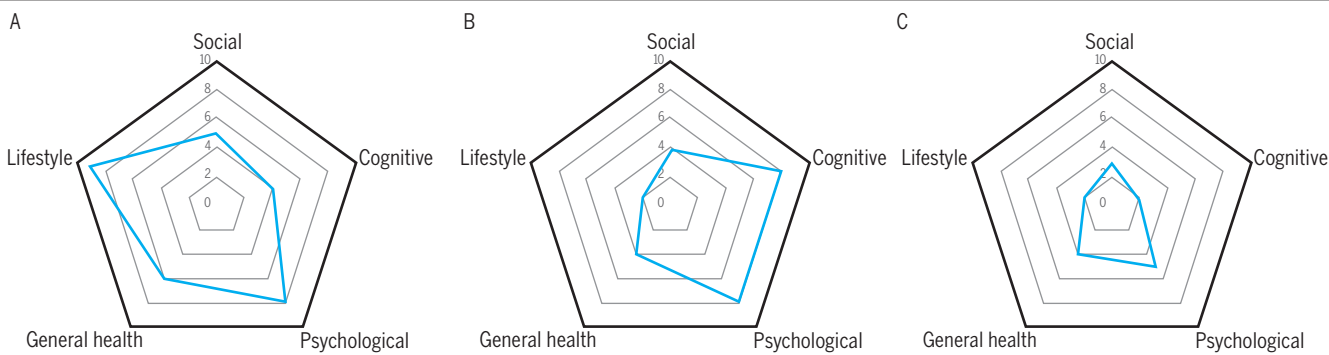


FIGURE 6. Application of contextual factors to patient cases (A) 1, (B) 2, and (C) 3.

tual shift in the management approach for patients with multisystem stresses. Accounting for contextual and psychological factors will enable clinicians to provide personalized physical therapy aimed at prescribing the right treatment, for the right person, at the right time.

Systematic quantification of clinical outcomes through comparative effectiveness research and learning health systems will offer timely, generalizable, and clinically applicable solutions for patients presenting to physical therapy following a concussive event. ●

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Brain Drain: Psychosocial Factors Influence Recovery Following Mild Traumatic Brain Injury—3 Recommendations for Clinicians Assessing Psychosocial Factors

Mild traumatic brain injuries (TBIs) account for the largest number of deaths and disabilities worldwide.¹⁹ Up to 90% of TBIs are mild, with over 1.5 million new cases annually in the United States alone.^{7,15} People with mild TBI may experience a variety of physical, behavioral/emotional, and cognitive symptoms, such as headaches, dizziness, nausea, fatigue, memory and concentration difficulties, irritability, changes in mood and emotions, anxiety, and depressive symptoms.^{9,19}

Although most people with mild TBI recover within 3 months, approximately 20% report persistent postconcussion symptoms up to 1 year or longer and typically receive a diagnosis of postconcussion syndrome.^{2,3,7,14} The symptoms can be

debilitating and interfere with, or delay return to, work, school, and sport.⁴ Post-concussive symptoms are not specific to people with mild TBI.¹² These symptoms are also reported after other injuries, such as orthopaedic injuries, and by uninjured members of the community.^{2,21,22} Perhaps postconcussive symptoms are



more related to a trauma in general rather than to a specific head trauma. The purpose of this clinical commentary was to provide 3 recommendations for clinicians to assess psychosocial factors in

patients after concussion, and to argue a case for clinicians to improve their skills in assessing psychosocial factors.

UNDERSTANDING THE BIOLOGICAL, PSYCHOLOGICAL, AND SOCIAL DETERMINANTS OF HEALTH

IN THE 16TH CENTURY, THE BODY AND mind were considered separate. This philosophy continued after the scientific revolution, ultimately giving rise to the currently and most widely used biomedical model.²⁰ In the 20th century, sequencing the human genome reinforced a linear, biomedical model of health: genes cause disease. Patients present with single disorders that respond predictably to specific evidence-based treatments, and pills are the best treatment.¹⁷ The mind and psychology of patients were afforded cursory consideration.

In the late 1970s, Engel proposed a biopsychosocial model of health to ac-

• **SYNOPSIS:** Mild traumatic brain injury is a major global public health concern. While most people recover within days to months, 1 in 5 people with mild traumatic brain injury report persistent, disabling symptoms that interfere with participation in work, school, and sport. People with injuries to regions other than the head may report similar symptoms. The biopsychosocial model of health explains this phenomenon in terms of factors associated with recovery that are not biomedical. Important psychosocial factors include poor recovery expectations and pretraumatic and posttraumatic psychological symptoms. Recent clinical practice guidelines recommend that clinicians examine all relevant biopsychosocial factors that may contrib-

ute to persistent postconcussive symptoms and consider them when helping their patients make health-management decisions. However, because clinical training continues to prioritize biomedical symptoms, clinicians may not feel confident in the psychosocial domain. Our objective is to provide 3 recommendations for clinicians to assess psychosocial factors in patients after concussion, and to argue a case for clinicians to improve their skills in assessing psychosocial factors. *J Orthop Sports Phys Ther* 2019;49(11):842-844. Epub 1 Jun 2019. doi:10.2519/jospt.2019.8849

• **KEY WORDS:** brain concussion, postconcussion syndrome, psychology, recovery of function

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count for multiple facets of a patient's biology, psychology, social behaviors, and exposures.²⁰ Psychosocial factors have one of the strongest impacts on health.⁵ By introducing the biopsychosocial model, Engel aimed to enhance the existing biomedical model by incorporating the social, behavioral, and psychological constructs of a patient.^{5,20}

There are extensive studies of the pathophysiology of mild TBI and postconcussive symptoms. The evidence suggests that persistent postconcussion symptoms can be better understood using a biopsychosocial model rather than a biomedical one. The following 3 recommendations and the **TABLE** provide guidance for clinicians when assessing and managing psychosocial symptoms in patients who have experienced a mild TBI.

Clinical Recommendation 1: Address Psychosocial Factors Early

Clinical Consideration When a patient presents with multiple symptoms, clinicians should focus on those that can be more easily managed or could delay recovery first, before targeting more complex or difficult-to-treat symptoms.¹⁶ Primary symptoms include psychosocial symptoms, such as depression and anxiety, in addition to other common symptoms such as sleep disturbance and headache. Primary symptoms can also exacerbate other symptoms. For example, feeling anxious and depressed can lead to fatigue, dizziness, and cognitive impairment.

Clinicians should screen for anxiety and depression using validated measures such as the Generalized Anxiety Disorder 7-item scale and the Patient Health

Questionnaire 9-item scale (**TABLE**). Screening for anxiety and depression should be done at any point when the clinician observes anxious or depressive symptomatology.

Clinical Recommendation 2: Ask All Patients About Their Recovery Expectations as Early as You Can

Clinical Consideration Poor recovery after mild TBI is strongly associated with psychosocial factors, including poor recovery expectations,⁶ more emotional stress or depressive symptoms, poorer preinjury mental status, and lower education.^{7,10} People who expect to recover more slowly after injury often do, compared to those who expect a faster recovery.^{1,8,11} Recent clinical practice guidelines recommend early education and reassurance concerning recovery times. This

TABLE

OVERVIEW OF APPROACH TO THE ASSESSMENT AND MANAGEMENT OF INDIVIDUALS WITH MILD TRAUMATIC BRAIN INJURY (FOCUS ON PSYCHOSOCIAL FACTORS)*

Assessment	Tool
• Screen for common mental disorders (preinjury or concurrent), including depressive disorders, anxiety disorders (including PTSD), behavioral changes (eg, apathy, aggression, irritability), emotional regulation issues, substance use disorders, and somatoform disorders. Note that these factors may delay recovery • Monitor • Offer management strategies • Collaborate with other health care providers as appropriate	ACE PHQ-9 (depression) GAD-7 PC-PTSD-5 PCL-5 CAGE and CAGE-Adapted to Include Drugs questionnaires
Management	Tool
1. Focus on education and reassurance: provide verbal and printed information to patients and support persons at the initial assessment and throughout care as required. Include information on a. Symptoms and expected outcomes: expect full recovery in the majority of patients (within days, weeks, or months) b. Normalizing symptoms: current symptoms are expected and common c. A gradual return to activities as tolerated (ie, does not result in significant or prolonged exacerbation of symptoms); some symptoms with return to activity are common and expected 2. Self-management techniques (eg, sleep hygiene, stress management) 3. Cognitive behavioral therapy (eg, to alter negative expectations of recovery) 4. Consider nonpharmacological interventions (with a focus on active versus passive care) as the first line of management a. Manage specific symptoms (eg, headache, sleep difficulties) according to evidence-based practice b. Target symptoms that can be managed more easily or could delay recovery first, before focusing on more complex or difficult-to-treat symptoms, as some symptoms may exacerbate others (eg, headache and sleep difficulties may contribute to anxiety and memory problems) 5. Goals for management are to return to preinjury status and prevent/reduce persistent symptoms that may limit function and potentially contribute to disability	Brain Injury Advice Card (long and short versions) Use tools according to specific symptoms (eg, MIDAS, PSQI, MoCA)

Abbreviations: ACE, Acute Concussion Evaluation; GAD-7, Generalized Anxiety Disorder 7-item scale; MIDAS, Migraine Disability Assessment questionnaire; MoCA, Montreal Cognitive Assessment; PCL-5, Posttraumatic Stress Disorder Checklist for DSM-5; PC-PTSD-5, Primary Care Posttraumatic Stress Disorder Screen for DSM-5; PHQ-9, Patient Health Questionnaire 9-item scale; PSQI, Pittsburgh Sleep Quality Index; PTSD, posttraumatic stress disorder.

**Refer to the Ontario Neurotrauma Foundation guideline¹⁶ for details and a comprehensive approach to the management and assessment of adults with mild traumatic brain injury. The tools listed are those recommended by the guideline¹⁶ and are available in the guideline appendices (along with others).*

may help normalize the experience of initial symptoms, reduce poor recovery expectations, and ultimately improve patient outcomes.¹⁶

Clinical Recommendation 3: Patients With a Positive Screen for Depression, Anxiety, Posttraumatic Stress Disorder, or Other Mental Health Disorders Should Be Referred to the Appropriate Health Care Provider or Be Comanaged

Clinical Consideration Patients with positive screening for a mental health disorder should be managed with evidence-based treatments such as cognitive behavioral or pharmacological therapy, as these conditions often complicate recovery.^{13,16,18}

WHERE TO FROM HERE?

WHILE RESEARCHERS CONTINUE TO investigate the determinants of poor recovery after mild TBI, as with the many health challenges of our time, it will be important to work within the biopsychosocial model of health. Some of the strongest predictors of poor recovery after mild TBI and other conditions are psychosocial in nature. Clinical training often emphasizes “physical” conditions and continues to focus on the biomedical domain. Therefore, clinicians may feel ill equipped to deal with the psychosocial aspect of injury and health. Clinicians and trainees need evidence-based guidance and point-of-care tools for assessing and managing psychosocial factors.

Future research must identify which factors (eg, health history; the influences of cultural, work, and family; beliefs about pain) may influence recovery expectations and can be modified by specific interventions for mild TBI. As technology, brain imaging, and assessment continue to advance, clinicians must remember that it is not only the brain they must concern themselves with, but also the person with the brain. ●

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Cutting to the Pathophysiology Chase: Translating Cutting-Edge Neuroscience to Rehabilitation Practice in Sports-Related Concussion Management

Mild traumatic brain injury, or concussion, is a common sports injury, with an annual estimate of 300 000 new cases in the United States.^{6,4} Objective, clinically relevant biomarkers can improve our understanding of concussion diagnosis, risk, and recovery, and are necessary to inform clinical decision making and guide sports safety regulations. A biomarker is “a characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacological response to a therapeutic intervention.”¹⁴

Concussion is a complex sequela of events affecting the brain after either a direct or indirect blow to the head. It typically results in a range of transient clinical symptoms, and functional disruption that may involve a brief loss of consciousness (less than 30 minutes) or alteration of consciousness (less than 24 hours), or a brief period of posttraumatic amnesia (less than 24 hours).^{5,6,3} Brain injury begins with direct damage to brain tissue and vasculature, including blood-brain barrier disruption and axonal shearing due to rotational forces.^{93,113} Secondary injury occurs more gradually⁹³ through ionic imbalances (eg, increased intracel-



lular calcium ions), excitotoxicity, inflammation, and apoptosis following glutamate release.¹¹³

The cell processes needed to re-establish ionic equilibrium deplete energy stores and increase metabolic stress, leading to necrosis. Ionic imbalances and axonal shearing result in disruption of axonal transport and accumulation of proteins, resulting in axonal swelling within deep gyri of the brain.¹¹³ These processes may also set the stage for repair and remodeling (ie, neuroplasticity) to promote recovery.

In this commentary, we highlight 5 areas in sport-related concussion that require further exploration and describe 6 cutting-edge approaches applicable to these areas.

AREAS OF RESEARCH NEED

IN THIS SECTION, WE IDENTIFY 5 AREAS in concussion research that need to be addressed in future studies.

Devices to Measure Impact Force

In the absence of clear signs and symptoms immediately following a head impact (eg, loss of consciousness, lack of balance while sitting or standing, memory

• **SYNOPSIS:** Mild traumatic brain injury, or concussion, is a common sports injury. Concussion involves physical injury to brain tissue and vascular and axonal damage that manifests as transient and often nonspecific clinical symptoms. Concussion diagnosis is challenging, and the relationship between brain injury and clinical symptoms is unclear. The purpose of this commentary was to translate cutting-edge neuroscience to rehabilitation practice. We (1) highlight potential biomarkers that may improve our understanding of concussion and its recovery, (2) explain why researchers must address the paucity of concussion research in

female athletes, and (3) present female-specific factors that should be accounted for in future studies. Integrating objective, quantitative measures of concussion pathophysiology with concussion history, genetics, and genomics will help caregivers identify concussed athletes, tailor recovery protocols, and protect athletes from potential long-term effects of cumulative head impact. *J Orthop Sports Phys Ther* 2019;49(11):811-818. Epub 1 Jun 2019. doi:10.2519/jospt.2019.8884

• **KEY WORDS:** eye movement tracking, female athletes, magnetic resonance imaging, mild traumatic brain injury, neuroscience

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disruptions, impaired judgment and/or speech), diagnosis of concussion relies heavily on subjective report of symptoms from the patient,⁶⁹ which are often underreported.⁶⁸ New devices that measure biomechanical forces to the head in real time are an emerging alternative, although few current commercial devices accurately diagnose concussion.^{15,79} Diagnosis is further complicated by nonspecific postconcussive symptoms (eg, mood changes, sleep disturbance, and cognitive complaints) that can also be observed in people who do not have concussion.⁴⁹

Cumulative Effect of Prior Concussions and Repetitive Subconcussive Hits

For a single concussion occurring in otherwise healthy adolescents, clinical recovery occurs between 1 and 3 months.⁴⁸ However, for some, symptoms may persist.¹⁷ One factor that may influence concussion risk and recovery is a history of previous concussion. Among both athlete and veteran samples, prior concussion increases the risk of developing subsequent concussions.^{41,78,91} With increased injuries, there is risk for greater symptom severity and functional impairment.^{45,87,110}

Exposure to subclinical repetitive head trauma incurred during sports participation (eg, soccer heading) may also influence concussion risk and recovery. Brain changes may reflect pathways through which repeated subthreshold head trauma increases vulnerability to concussion and protracted recovery.^{2,95,96} For example, magnetic resonance imaging of high school collision-sport athletes shows significantly different resting-state functional connectivity in the default-mode network compared to non-collision-sport athletes both before and after a single season, and this functional connectivity change persists up to 6 months after the end of the season.^{1,2,51}

The default-mode network is a brain network that is active during mind wandering, deactivated during goal-oriented tasks, and disrupted in numerous brain disorders, including Alzheimer's disease, schizophrenia, and autism.¹⁶ Changes in

functional connectivity suggest a compensatory rewiring of brain networks in response to physical impact. Similarly, structural connectivity (measured by diffusion imaging) and brain metabolite composition (measured by magnetic resonance spectroscopy) differentiate contact-sport and non-contact-sport athletes in the absence of diagnosed concussion. There is elevated fractional anisotropy and reduced mean diffusivity in white matter (indicating more hindered water diffusion) and a reduced N-acetyl-aspartate-creatine ratio in the brains of contact-sport athletes, and larger alterations in athletes with a history of concussion.¹⁸ Even in the absence of clinically diagnosable concussive symptoms, impact from participation in collision sports might be sufficient to alter functional, structural, and chemical characteristics of brain tissue.

Genetics

Specific genotypes may be associated with greater risk for concussion.⁸¹ Apolipoprotein E and brain-derived neurotrophic factor^{26,31,32,54,73,100} genes have been implicated in collegiate athletes experiencing multiple concussions.^{99,100} In addition to these genes, the genotype of KIAA0319, a gene implicated in dyslexia, significantly predicted number of previous concussions among college football players.¹⁰³

Because they are peripherally accessible, expressed in the brain, and have been implicated in other brain disorders⁶⁵ or trauma exposures,⁶¹ microRNAs (short, noncoding RNAs [20-23 nucleotides] that inhibit messenger RNA and protein expression)^{10,43} may be potential biomarkers for concussion. There is differential microRNA expression in animal models of concussion, where expression profiles fluctuate post injury (unpublished data). In humans, microRNAs have above-average diagnostic accuracy for mild to moderate traumatic brain injury,¹³ and may predict spatial memory abnormalities over the course of a football season without concussion in collegiate football.⁸² Ongoing research

suggests that these markers, potentially in conjunction with metabolomics metrics,³⁴ may be important for the diagnosis and staging of illness.⁹⁵

Return to Play

Return-to-play decisions are currently guided by clinical considerations and guidelines developed by the National Collegiate Athletic Association, the National Football League, the Concussion in Sport Group consensus statement,⁶⁹ and state legislation.^{92,94} Typically, injured athletes are cleared through a stepwise process requiring symptom resolution with increased activity. Yet, brain changes measured by neuroimaging persist beyond clinical symptom resolution.^{23,74} Therefore, establishing an evidence-informed metric of brain recovery following concussion and exposure to repetitive head trauma incurred during sports participation is essential for personalized care in injured athletes in the short and long term.

Understanding Female-Specific Factors in Concussion

Most research has focused on male athletes and male military populations. Studying female athletes is crucial because women may experience a greater number of injuries and a longer recovery from concussion than men.^{11,20,21,36} One physiological explanation is that the female brain may be more vulnerable to the effects of trauma at the cellular level, evident by increased axonal microtubule breakage, calcium-ion dysregulation, and axonal swelling after injury.³⁰ Lower neck strength,⁴⁶ hormonal fluctuations, menstrual cycle phase at time of injury, and contraceptive use⁸⁹ may also influence injury risk and concussion recovery among female athletes.

Hormones fluctuate over the course of the menstrual cycle. Hormonal contraceptives radically attenuate these natural fluctuations. Four in every 5 premenopausal, sexually experienced women in the United States have used oral contraceptives, and approximately 1 in 3 have used other hormonal methods of con-

traception (eg, contraceptive patch).²⁵ Therefore, excluding women using hormonal contraception³ from concussion research limits the generalizability of findings. Collegiate female athletes using hormonal contraceptives had significantly lower postconcussive symptom severity than their peers who were not using hormonal contraception.³⁶ Women injured during the luteal phase of their menstrual cycle had worse symptoms than those taking oral contraceptives or those who were injured during the follicular phase.¹¹² New methodology recommendations are needed to include women using hormonal contraceptives, as well as to account for the effects of hormonal fluctuations across the menstrual cycle.

Ignoring the impact of menstrual cycle phase may impair interpretation of clinical and research biomarkers. There are different patterns of functional activation in response to emotional processing,^{6,37,39} verbal memory tasks,^{22,55,58,109} and visuospatial tasks^{29,90} across the menstrual cycle. Similar findings have also been reported for structural²⁷ and resting-state functional connectivity,⁸⁴ indicating that menstrual cycle phase may induce changes at both structural and functional levels. Contraception use may also affect cognitive functions.^{85,88} Accounting for these variations will improve the sensitivity of clinical and research biomarkers to concussive injury.

CUTTING-EDGE NEUROSCIENCE TOOLS TO STUDY CONCUSSION

IN THIS SECTION, WE HIGHLIGHT 6 emerging neuroimaging research tools that are simple to administer and interpret.

Static Measures of Cerebral Blood Flow

Cerebral blood flow is tightly linked to neural activity. Magnetic resonance imaging can measure cerebral blood flow either with an exogenous contrast agent (dynamic susceptibility contrast) or by manipulating blood signal with radio-

frequency pulses, such that it becomes an endogenous contrast in arterial spin labeling. Although dynamic susceptibility contrast is more commonly used clinically, arterial spin labeling is noninvasive.^{28,59}

Concussion studies using arterial spin labeling primarily report decreases in regional cerebral blood flow immediately after injury, coinciding with cognitive impairment and acute symptom severity. Most studies reported a negative correlation between cerebral blood flow and symptom severity, cognitive performance, and stress and anxiety scores.^{71,106} With increased time after injury, some studies reported persistent reductions in cerebral blood flow up to several months following injury,^{66,105-107} while others have found cerebral blood flow to normalize within a month.⁷¹ Some studies also reported an increase in either global or regional cerebral blood flow, although this appears to be primarily associated with symptomatic participants.^{9,97} Most studies only included male athletes,^{71,107} and sex differences or hormonal influences on cerebral blood flow are unclear.

Dynamic Measures of Vascular Reactivity

Cerebral blood flow provides information about tissue health at rest but does not illuminate whether the blood vessels can dynamically adapt when there are increased demands of brain function. The link between changes in neural function and a corresponding increase in cerebral blood flow (neurovascular coupling) is often disrupted when the vascular system is compromised by disease or injury.³⁸

Cerebrovascular reactivity allows researchers to examine neurovascular coupling using a global vasoactive stimulus, such as acetazolamide, carbon dioxide-enriched air, or breath holding, which causes dilation of vessels throughout the brain. This changes the local concentration of deoxyhemoglobin and the measured magnetic resonance signal—measured via blood oxygenation level-dependent imaging.⁸⁰ Alternatively, vascular reactivity can also be measured using transcranial Doppler ultrasonogra-

phy, which can detect changes in blood velocity of major arteries supplying the brain. While transcranial Doppler ultrasonography is easier to implement, it does not provide vascular reactivity measures in specific brain regions.

Use of neuroimaging-based cerebrovascular reactivity in studies of concussion is a novel development, and findings are mixed due to variations in the vasoactive stimulus used, time after injury, and head impact load.^{18,76,77,98} Despite discrepancies due to injury heterogeneity, treatment approaches, baseline physiology, and time after injury, cerebrovascular reactivity is consistently altered by both concussive injury and subconcussive hits and dynamically evolves during the recovery period.

Brain Network Connectivity

Blood oxygenation level-dependent imaging is typically used to investigate specific functional tasks. Yet, in the resting brain, the “noise” of the blood oxygenation level-dependent time course also contains a wealth of information, including oscillations at 0.01 to 0.1 Hz, representing spontaneous brain activity. Brain areas with correlated oscillations are thought to be functionally connected and form a functional network. Multiple resting-state networks have been identified.^{4,24} The connectivity strength within these networks correlates with behavioral measures and differs between diseased and healthy control populations.³⁵ In addition to its sensitivity to brain activity and health, resting-state connectivity can be measured while participants are at rest inside the scanner, minimizing cognitive burden after injury, and is not confounded by differences in task performance.

Resting-state connectivity findings in sport-related concussion are related to time after injury. In general, increased connectivity in the default-mode network, a network active at rest, is observed during the acute phase (24 hours). At a week post injury, connectivity within the default-mode network is reduced,^{52,67,114}

but across the entire brain, both hyperconnectivities and hypoconnectivities have been reported, and the connectivity strengths correlate with verbal and visual memory deficits.^{70,74} At a month post injury, default-mode network connectivity approaches baseline levels but is still abnormal.¹¹⁴ Brain areas involved in executive function have hyperconnectivity at 6 months post injury.²³ Connectivity changes dynamically during recovery and can be used to determine whether an athlete deviates from a normal recovery trajectory.

White Matter Integrity

Diffusion imaging refers to the study of microscopic movement of water by encoding diffusion with magnetic gradient fields in differing orientations. While diffusion in nature is truly random, diffusion within tissue is restricted by tissue components such as cell membranes, fibers, and micromolecules.⁶⁰ Diffusion imaging is particularly well suited to study white matter microstructure due to the organization of myelinated axonal fiber bundles, which restrict molecular diffusion perpendicular to the fiber.

Because diffuse axonal damage is one of the primary mechanisms of injury in concussion, diffusion tensor imaging is a useful tool. The most commonly reported measures for diffusion imaging are (1) fractional anisotropy—a normalized scalar, with values ranging between 0 and 1 describing whether diffusion is isotropic (equal in all directions) or restricted along a single axis; and (2) mean diffusivity—the average apparent diffusion along the 3 major diffusion axes.

Most concussion studies, but not all,²⁴ report decreased fractional anisotropy and increased mean diffusivity in people after concussive injury.^{62,72,75,111} Axial diffusivity correlated with the Brief Symptom Inventory, and participants with higher fractional anisotropy had higher Standardized Assessment of Concussion cognitive scores.⁷⁵ Changes in fractional anisotropy and mean diffusivity are consistent with axonal injury and are easily

measured clinically. However, more advanced diffusion models are needed to improve understanding of the mechanism underlying these changes and extend our ability to probe gray matter changes.

Microhemorrhages and Tissue Integrity

Magnetic susceptibility—the differential response of substances when exposed to a magnetic field—is the basis of a new type of magnetic resonance contrast called “susceptibility-weighted imaging.”⁴² This technique is especially sensitive to microhemorrhages, correlates with neurological symptoms, and may predict long-term outcomes following brain injury.^{7,12,19,42}

Quantitative susceptibility mapping is a quantitative extension of susceptibility-weighted imaging that can measure tissue iron content and venous oxygen saturation.^{86,104} The application of quantitative susceptibility mapping to concussion is relatively new. In 2 studies,^{40,108} there was no change in susceptibility either post injury or after a season among high school football players.^{40,108} However, in the most recent publication, with a larger cohort of collegiate and high school football athletes followed longitudinally, there were significant increases in white matter susceptibility that persisted after clinical symptom resolution and correlated with return-to-play duration.⁵⁷

Eye Movement (Infrared Imaging)

Reliable tools to assess subtle cognitive impairments that occur after head trauma are needed to assess impairment and track change over time. Eye movement testing is a noninvasive and reliable measure of sensorimotor and cognitive functioning,⁵⁶ with promise for distinguishing individuals with and without a history of recent head trauma.^{8,44,101}

Rapid movements of the eyes (saccades) from one point of gaze to another are tested. The tests are either stimulus driven (eg, prosaccade task) or under executive control (eg, antisaccade and memory-guided saccade tasks). Saccades are executed by hierarchically organized brain regions, many of which are asso-

ciated with pathophysiological changes following symptomatic concussion, as well as with pathology signifying chronic traumatic encephalopathy. Eye movement test performance is associated with white matter changes in the splenium of the corpus callosum among individuals with recent concussion, suggesting an association between performance and neurostructural integrity among individuals with recent concussion.¹⁰¹

Some individuals with concussion perform worse than controls, particularly on classic eye movement tasks with increased executive-control demands (eg, antisaccade and memory-guided saccade tasks). People with concussion tend to have increased directional error, decreased saccade accuracy, and, less consistently across studies, prolonged latencies.^{33,44,47,53,101}

Studies of eye movement following concussion have primarily been conducted among men presenting to the emergency room. Therefore, further research is needed to determine whether eye movement testing is sensitive to the effects of concussion in women and in those with sport-related concussion.

SUMMARY AND PERSPECTIVES

INCREASED AWARENESS OF THE LONG-term impact of concussions is driving growth in concussion research. However, there is much work to be done. In this commentary, we stress the importance of establishing objective biomarkers across the research areas of behavior, brain, molecular biology, and genetics to aid concussion diagnosis. Panels of biomarkers will allow caregivers to assess concussion risk in athletes, evaluate their resilience over many seasons of play, and identify concussed athletes more accurately to initiate the recovery protocol as early as possible.

In the second part of this commentary, we outlined some methodological advances that can help (a) better assess concussion pathophysiology, (b) link

pathophysiology to clinical symptoms, and (c) improve understanding of the recovery process. Each technique is sensitive to a different component of brain injury. Therefore, the most powerful approach may be to integrate these findings with other common clinical tests for a comprehensive assessment and informed plan of care. Collecting information about an individual's concussion history, genetics, and genomics might enhance diagnosis, prognosis, and recovery planning—a handful of studies have already recognized the importance of multimodal data integration.^{50,83,102} While some techniques are not easily deployable on the sidelines for immediate assessment, their quantitative nature can help researchers identify surrogate peripheral biomarkers that may be more easily applied on the field.

Aside from overt sport-related concussion injury with clinically evident symptoms, mounting evidence suggests that repetitive subconcussive events may lead to underlying brain structure and function changes.^{51,95,96} Whether this results in increased vulnerability to subsequent concussions or increased risk of neurodegenerative disease later in life requires longer-term prospective study with multimodal data integration.^{50,83,102}

Female athletes are more vulnerable to injury, experience a higher number of injuries, and have longer recovery times than male athletes. However, women are dramatically underrepresented in concussion research. Women have lower neck strength and greater hormonal fluctuations that may explain sex differences. These findings need to be investigated on a larger scale to guide return-to-play and postinjury care, and to inform educational resources for injured athletes and their caregivers. ●

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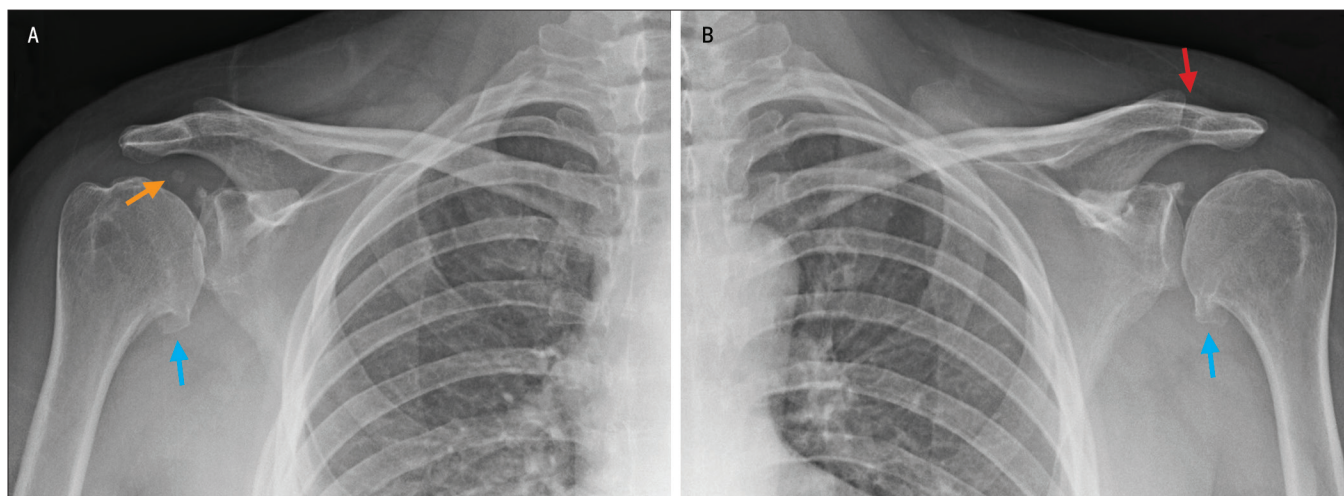


FIGURE 1. Anteroposterior radiographic views of the right shoulder (A) and the left shoulder (B) in external rotation revealing decreased bone density, severe glenohumeral joint arthrosis, and a large osteophyte (blue arrows) projecting off the inferior aspect of the humeral head. Small calcifications superior to the right humeral head (orange arrow) may reflect calcific tendinitis. There is moderate left acromioclavicular joint arthrosis (red arrow).

Acromegalic Arthropathy

JASON T. CIROLIA, PT, DPT, OCS, AdventHealth Sports Medicine and Rehabilitation, Orlando, FL.

A 30-YEAR-OLD WOMAN WAS REFERRED to physical therapy for bilateral shoulder and thoracic spine pain concurrent with loss of range of motion (ROM), resulting in limitations in activities such as dressing and swimming. Notable medical history included surgical removal of a pituitary adenoma 6 years prior. The adenoma resulted in excessive growth hormone secretion, developing into acromegaly with hallmark physical features.

A shoulder examination revealed bilateral capsular-pattern limitations, normal strength through the available ROM, and a numeric pain scale score of 7/10 with both active and passive motions. Functionally, reaching behind the back was limited to the lateral hip and overhead reaching was limited to the

anterior base of the neck, with a score of 35% disability on the shortened version of the Disabilities of the Arm, Shoulder and Hand questionnaire (QuickDASH). The thoracic spine was grossly hypomobile, with restrictions in extension and rotation bilaterally.

Treatment consisted of neurodynamics and mobility exercises for 16 visits over a 10-week time span. Additionally, radiographs were requested to evaluate the extent of arthropathies associated with acromegaly. Radiographs revealed advanced arthropathies of the bilateral acromioclavicular and glenohumeral joints, along with loss of disc height and anterolisthesis throughout the thoracic spine (FIGURE 1; FIGURES 2 and 3, available at www.jospt.org). Due to the severity of the arthrosis, atypical at this age, the

patient was referred to an orthopaedist, who recommended continued nonsurgical management. At discharge from physical therapy, the patient's ROM had plateaued, her numeric pain scale score was 3/10, and her QuickDASH disability score was 23%.

Acromegalic arthropathy, which occurs in about 50% of patients with acromegaly, is a thickening of soft tissue and cartilage affecting both the peripheral and axial skeletons, due to increased bone turnover from excessive growth hormone.^{1,2} While surgical removal of the pituitary tumor is the preferred treatment, persistent elevated levels of growth hormone and insulin-like growth factor 1 may require long-term medical management.¹ ● *J Orthop Sports Phys Ther* 2019;49(11):864. doi:10.2519/jospt.2019.8302

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Determining Outcome in Children and Adolescents After Concussion: Viewing Things More Holistically

Concussions, or mild traumatic brain injuries (TBIs), are common in individuals younger than 18 years of age. Every year, thousands of children and adolescents sustain a concussion requiring medical attention in the context of sports or other activities.³⁵ Although many children and adolescents do not seek immediate care after a concussion,³ incidence estimates based on hospital or physician office visits help illustrate the scope of the problem. In the United States, emergency department TBI-related visits, of which 80% to 90% are mild TBI, vary from 2193 per 100 000 in children 0 to 4 years of age to 981.9 per 100 000 in young people 15 to 24 years of age. This clearly indicates a substantial public health concern.²⁹ In Canada in 2010, the overall rate for emergency department or physician's office visits for 3-to-17-year-olds was 601 per 100 000 individuals in Ontario,³¹ with similar trends in Quebec for the same year.²⁵

Making a diagnosis of concussion in children and adolescents is challenging, whether the injury is considered on the spectrum of TBI or a separate entity.³⁹ The heterogeneity of brain injury, the complexity inherent to brain function, and the variability of clinical presentations have set the stage for different definitions of concussion over the last decade.³² While clinicians await imaging or fluid biomarkers, which could confirm or refute the presence of structural dam-

age after these injuries, they must rely on their expertise and clinical judgment. Critical to proper concussion management is efficient and timely assessment. This paper reflects on how to approach the evaluation of children and adolescents after a concussion, from diagnosis to return to play.

Diagnosis and the First 48 Hours: Initial Assessment of Children and Adolescents With Concussion

The diagnosis of mild TBI/concussion continues to rely on the treating physician's clinical judgment, constructed from the amalgamation of often limited and nonspecific clinical information (signs and symptoms).^{34,46} One such item, headache, is both the most common symptom of a concussion³⁷ and a very common single symptom for individuals presenting to an emergency department with a variety of conditions, making its presence alone a poor marker of concussion.²¹ Relying on asking children "how they feel" or whether they have a headache at the time of diagnosis is insufficient.⁴ The clinician must broaden the assessment to include domains other than self-reported symptoms.¹⁸

Complementary to a general physical exam, adding elements of a neurological exam, of cognitive assessment, and of balance evaluation is now advocated by international recommendations³³ and

● **SYNOPSIS:** Pediatric and adolescent concussion is an increasingly high-profile public health issue, but it is also a highly heterogeneous phenomenon. Many factors interact dynamically to influence the recovery trajectory of adolescents and children. Diagnostic assessment must include domains other than self-reported symptoms, yet many prognostic models of outcome focus solely on the presence or absence of postconcussion symptoms to determine recovery. Function after concussion (recovery or persistence of problems) is the result of an interaction between biological, psychological, and social factors. Despite biopsychosocial models of assessment being advocated in rehabilitation for the last 20 years, they are still not routinely implemented in the evaluation

of concussions, along the recovery trajectory, in children and adolescents. The International Classification of Functioning, Disability and Health is a framework anchored in a biopsychosocial perspective that can guide clinicians and researchers to include multiple perspectives in their assessments or research designs. By focusing on the patient as a person, researchers and clinicians can provide a more holistic approach that has the potential to contribute to a more successful and sustainable pediatric and adolescent concussion care model. *J Orthop Sports Phys Ther* 2019;49(11):855-863. Epub 9 Oct 2019. doi:10.2519/jospt.2019.8918

● **KEY WORDS:** mild traumatic brain injury, outcome measures, youths

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experts in the field,¹⁸ to exclude other critical diagnoses such as an intracranial bleed or other neurosurgical emergencies. Screening tools and standard assessments may help guide initial diagnosis and management in the acute phase post injury.^{11,13,19,23} Examples include the Centers for Disease Control and Prevention Acute Concussion Evaluation¹⁹ or the Sport Concussion Assessment Tool. Both tools promote the use of a multifaceted, albeit limited, approach and can distinguish children with and without concussions.⁵ The fifth iteration of the Sport Concussion Assessment Tool (SCAT5)¹³ and its pediatric equivalent (Child SCAT5)¹¹ are currently the proposed standards for assessment of acute concussions.^{33,41} Their utility, however, decreases with time and is questionable beyond 3 to 5 days after injury.¹² An alternative must therefore be considered to track recovery over time, beyond simply monitoring postconcussion symptoms, until complete and unrestricted return to activities has been achieved.

Tracking Recovery After Concussion

Clinicians often rely on the presence or absence of postconcussion symptoms when making judgments about prognosis, because they lack a standard, multidomain assessment tool.^{43,54} Symptom resolution may be a proxy for the child or adolescent having returned to preinjury levels of function. However, symptom resolution does not account for many of the deficits or physiological changes that have been identified through various research. Persistent problems, such as difficulties with complex balance skills,⁴⁷ disrupted cerebral blood flow,³⁶ or electrophysiological abnormalities,⁴² found at time points when symptoms are largely resolved^{7,24} point to a discordance between the recovery curve and self-reported symptoms. In addition, prolonged recovery in these diverse spheres of functioning is frequently studied in isolation. While excellent at identifying neuropsychological,^{6,22} physical,^{15,30,38} or psychosocial impairments,⁴⁴ the body of research

on prolonged recovery leaves clinicians wanting for a more holistic view of the child or adolescent that could be applied to their own practice, where the focus on overall well-being is replacing more traditional and narrower views of physically defined medical conditions.²⁸ Biopsychosocial models can help clinicians and patients understand concussion recovery and track change over time.

Fifteen years ago, biomedical models, where health was defined as the absence of disease and the resolution of any structural or physiological disruptions, dominated. Biopsychosocial models are contemporary alternatives that help rehabilitation specialists better understand the experiences of individuals with illness or injuries through the examination of biological, psychological, and social dimensions of their lives.⁵⁰ Rehabilitation is embracing a vision of health that is positive, where clinicians focus on patient capacity and strengths. Assessments and interventions are positioned in a perspective of not only decreasing impairments, but also increasing participation in a real-world (ecological) context.^{1,40}

The field of TBI rehabilitation has seen the International Classification of Functioning, Disability and Health (ICF)⁵³ emerge as a useful biopsychosocial model to better characterize functioning after more severe types of TBI. However, the field of concussion has been slower to embrace this view. Even if a concussion is thought of as a milder form of brain injury, people experience its effects in ways that sometimes appear unrelated to the physiological nature of the initial injury itself.^{10,14} Function after concussion can thus be better described when ascertained through a biopsychosocial lens, acknowledging that its impact may not solely be explained by changes in structure/physiology. Achieving this understanding, however, requires the selection and interpretation of assessment tools that are appropriate for the patient and the context, and that possess the measurement properties required to facilitate good clinical decisions.

When applied to any health condition, the ICF presents functioning and disability as a dynamic interaction between body function and structure changes, activities, and participation, as well as environmental and personal factors. A child with a concussion who is subjected to a metabolic and physiological cascade of events (body structures)²⁰ may have impairments of eye movements (body function), limitations in reading class material (activity), and restrictions in participating in meaningful class activities (participation). However, the interaction may be such that contextual factors, such as effective school accommodations or helpful teachers (environmental factors) and high resilience (personal factor), can help the child maintain higher levels of participation in social and community settings.

Studies approaching concussions from a biopsychosocial perspective are rare. However, the models usually focus on linearly linking imaging findings to a limited number of functional outcomes,^{51,52} failing to take advantage of the dynamic relationships allowed by the model. More efforts to examine how best to use the ICF to assess functioning post injury and allow the emergence of new rehabilitation targets for intervention, especially for children and adolescents with prolonged recovery, must be deployed.

There are barriers to the successful use of biopsychosocial models in clinical care and research projects. For example, to capture all areas of functioning, clinicians risk overburdening children and their families with long and repeated assessment sessions, as they may already be fatigued from their concussion. Overtesting in any context may perpetuate iatrogenic consequences, as problems could be induced by the increased attention given to a potentially mild condition.⁴⁸ More work on the operationalization of biopsychosocial models in the field of concussion is needed.

The CanPedCDE Initiative

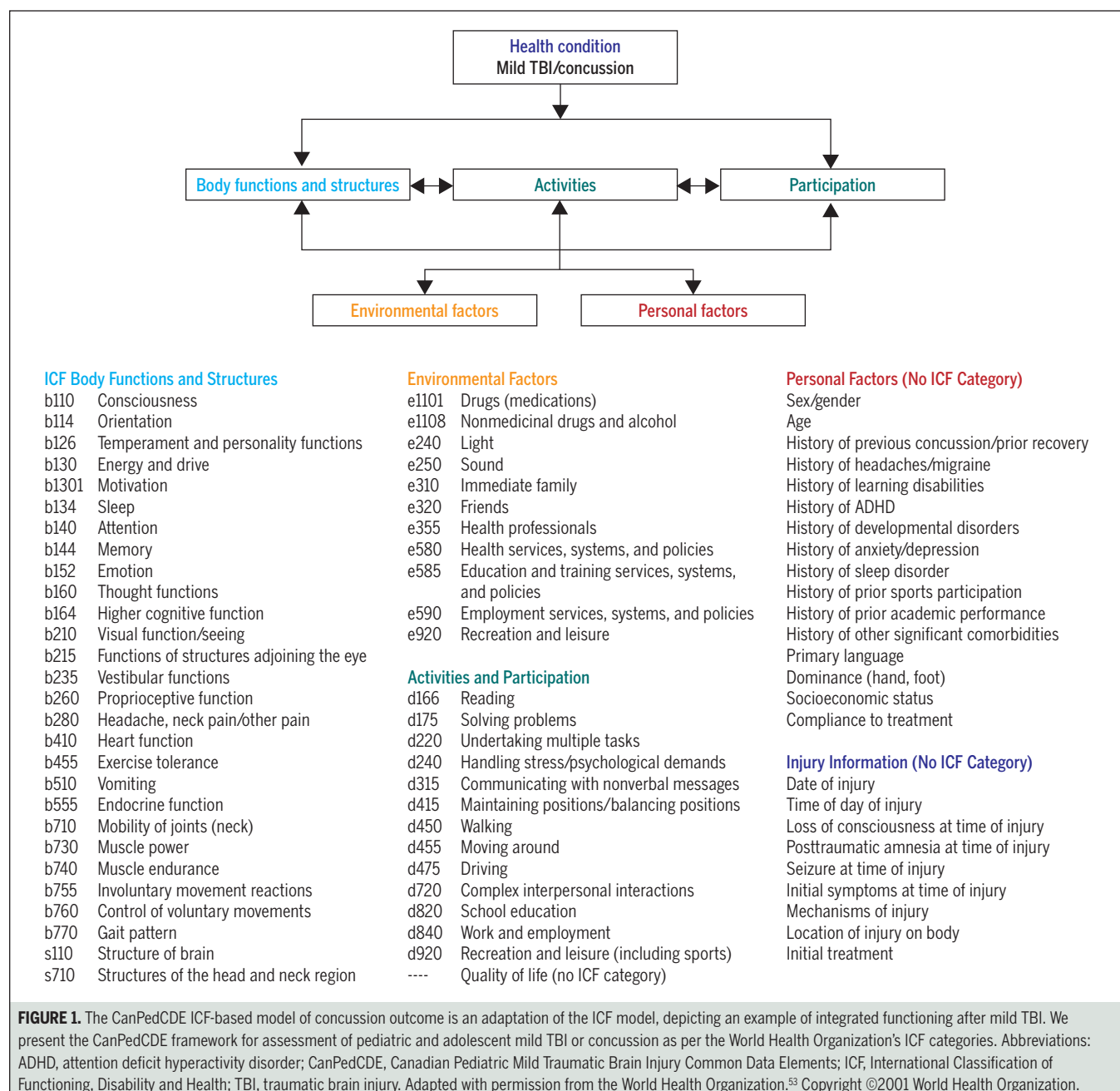
The Canadian Pediatric Mild Traumatic Brain Injury Common Data Elements

(CanPedCDE) is an initiative involving researchers, clinicians, patients, and other stakeholders to propose a comprehensive assessment of pediatric mild TBI and concussion by varied mechanisms. A decade of work by international initiatives has suggested a need to standardize the clinical presentation of concussion in a broad and comprehensive manner. The National Institute of Neurological Disorders

and Stroke, through its initiative of Common Data Elements for TBI² and for sport-related concussions,⁸ has proposed a list of domains and outcome measures thought to be appropriate at key time points. Although assessment tools for single constructs (eg, 3 tools capturing quality of life) have been useful in sharing data across TBI or sport-related concussion studies, their multiplication and lack

of specificity to the pediatric population have made their clinical utility difficult.

Using the National Institute of Neurological Disorders and Stroke Common Data Elements as a starting point, the CanPedCDE consensus exercise led to the proposition of an ICF-based model of functioning after concussion, with related assessment tools (see Gagnon et al¹⁶ and the summary in **FIGURES 1**



and 2 and TABLE 1). To address some of the limitations of previous initiatives, which advocated an impressive number of elements and assessment tools without actually testing how they should be implemented with children and adolescents, the CanPedCDE examined the feasibility of executing its framework in the context of a pilot, multicenter longitudinal study.¹⁶

Feasibility of Administering Multiple Assessment Tools to Children and Adolescents With Concussion

To explore the burden of administration of the selected tools, the CanPedCDE initiative included pilot data collection in 6 clinical mild TBI/concussion follow-up programs located in Montreal (Montreal Children's Hospital-McGill University Health Centre and Centre hospitalier uni-

versitaire Sainte-Justine), Quebec City (Centre hospitalier universitaire de Québec-Université Laval), Ottawa (Children's Hospital of Eastern Ontario), Hamilton (McMaster Children's Hospital), and Victoria (University of Victoria). Sites recruited children and adolescents seen in their programs, and followed them for 6 months (48 hours; 2 weeks; and 1, 3, and 6 months post injury). Ethics approval was

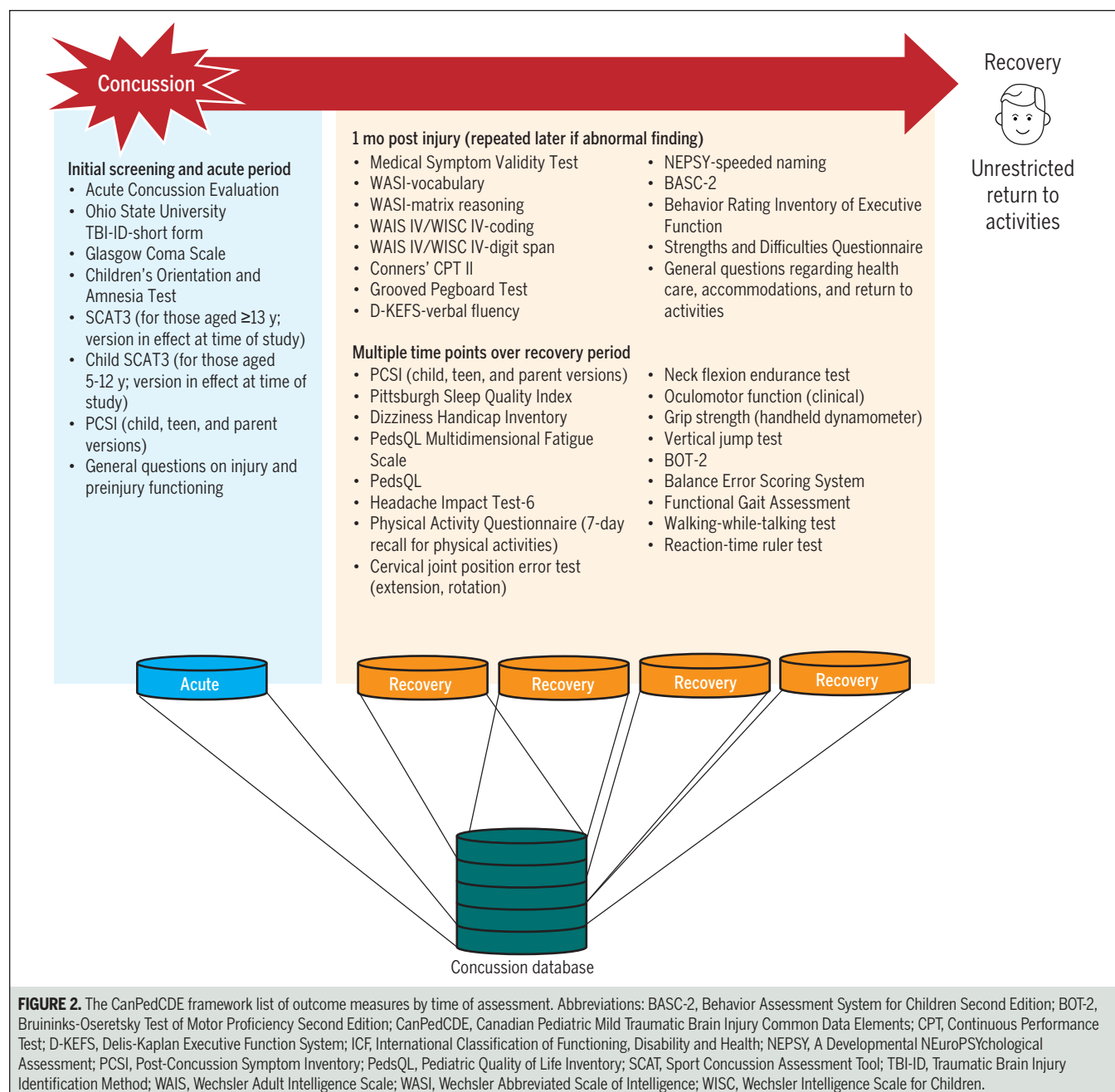


FIGURE 2. The CanPedCDE framework list of outcome measures by time of assessment. Abbreviations: BASC-2, Behavior Assessment System for Children Second Edition; BOT-2, Bruininks-Oseretsky Test of Motor Proficiency Second Edition; CanPedCDE, Canadian Pediatric Mild Traumatic Brain Injury Common Data Elements; CPT, Continuous Performance Test; D-KEFS, Delis-Kaplan Executive Function System; ICF, International Classification of Functioning, Disability and Health; NEPSY, A Developmental NEuroPSychological Assessment; PCSI, Post-Concussion Symptom Inventory; PedsQL, Pediatric Quality of Life Inventory; SCAT, Sport Concussion Assessment Tool; TBI-ID, Traumatic Brain Injury Identification Method; WAIS, Wechsler Adult Intelligence Scale; WASI, Wechsler Abbreviated Scale of Intelligence; WISC, Wechsler Intelligence Scale for Children.

obtained from each institution prior to initiation of the project. Consecutive children presenting to the emergency department or follow-up programs of the participating institutions were included in the project if they (1) were 6 to 17 years of age, (2) had a

mild TBI,⁹ (3) had suffered the initial injury in the previous 28 days, and (4) were proficient in English or French. Children were excluded if they had a severe developmental delay or a polytrauma requiring hospitalization for something other than

mild TBI. Participating children and families were introduced to the project as they presented to emergency departments or on initial referral to mild-TBI programs, and provided informed consent for their deidentified data to be used.

TABLE 1

FINAL LIST OF OUTCOME MEASURES INCLUDED IN THE CANPедCDE FRAMEWORK VERSION 1

Outcome Measure	ICF Category	Specific Domain/Construct
Acute Concussion Evaluation	Injury information and personal factors	Multiple
Ohio State University TBI-ID-short form	Personal factors	Previous concussion and recovery history
Glasgow Coma Scale	Body functions	Consciousness
Children's Orientation and Amnesia Test	Body functions	Orientation
SCAT3 (for those aged ≥13 y; version in effect at time of study)	Body functions	Multiple
Child SCAT3 (for those aged 5-12 y; version in effect at time of study)	Body functions	Multiple
PCSI (child, teen, and parent versions)	Body functions	Multiple
Pittsburgh Sleep Quality Index	Body functions	Sleep
Dizziness Handicap Inventory	Body functions	Vestibular
PedsQL Multidimensional Fatigue Scale	Body functions	Energy
PedsQL	None	Quality of life
Headache Impact Test-6	Body functions	Pain (headache)
Physical Activity Questionnaire (7-day recall for physical activities)	Activities and participation	Sport participation
Cervical joint position error test (extension, rotation)	Body functions	Proprioceptive function
Neck flexion endurance test	Body functions	Muscle power/endurance
Oculomotor function (clinical)	Body functions	Functions of structures adjoining the eye
Grip strength (handheld dynamometer)	Body functions	Muscle power/endurance
Vertical jump test	Body functions	Muscle power/endurance
BOT-2	Body functions	Multiple
Balance Error Scoring System	Body functions	Involuntary movement reactions (postural control)
Functional Gait Assessment	Activities and participation	Walking, moving around
Walking-while-talking test	Activities and participation	Undertaking multiple tasks, walking
Reaction-time ruler test	Body functions	Control of voluntary movements
Medical Symptom Validity Test	Body functions	Motivation, memory
WASI-vocabulary	None	General intelligence
WASI-matrix reasoning	None	General intelligence
WAIS IV/WISC IV-coding	Body functions	Thought processing
WAIS IV/WISC IV-digit span	Body functions	Memory
Conners' CPT II	Body functions	Attention
Grooved Pegboard Test	Body functions	Control of voluntary movements (fine motor)
D-KEFS-verbal fluency	Body functions	Higher cognitive function
NEPSY-speeded naming	Body functions	Higher cognitive function
BASC-2	Body functions	Emotional functions
Behavior Rating Inventory of Executive Function	Body functions	Higher cognitive function
Strengths and Difficulties Questionnaire	Body functions	Emotional functions
General questions regarding health care, accommodations, and return to activities	Environmental factors	Multiple

Abbreviations: BASC-2, Behavior Assessment System for Children Second Edition; BOT-2, Bruininks-Oseretsky Test of Motor Proficiency Second Edition; CanPedCDE, Canadian Pediatric Mild Traumatic Brain Injury Common Data Elements; CPT, Continuous Performance Test; D-KEFS, Delis-Kaplan Executive Function System; ICF, International Classification of Functioning, Disability and Health; NEPSY, A Developmental NEUROPSYchological Assessment; PCSI, Post-Concussion Symptom Inventory; PedsQL, Pediatric Quality of Life Inventory; SCAT, Sport Concussion Assessment Tool; TBI-ID, Traumatic Brain Injury Identification Method; WAIS, Wechsler Adult Intelligence Scale; WASI, Wechsler Abbreviated Scale of Intelligence; WISC, Wechsler Intelligence Scale for Children.

[CLINICAL COMMENTARY]

In the pilot implementation project, 484 participants were recruited. At least 10 participants (children and their parents) and 2 clinicians from each site were asked to respond to an online questionnaire addressing the feasibility and acceptability of completing or administering assessment tools over the 6-month visit cycle. Age-appropriate surveys for children 10 years of age and older were created and tested prior to administration. The survey asked questions about the impact of the entire testing battery, in terms of usefulness for concussion management and the participation process. In addition, clinicians were surveyed for burden on health care services. The questions were scored on a 5-point Likert scale from 1 (I don't agree at all) to 5 (I totally agree) that targeted the data-collection process ("I think the measures used are comprehensible, logical, in good order, easily performed, complete, too long . . ." and participant experience ("I think that participating was interesting,

personally relevant, useful . . ." Finally, clinicians were asked about changes to their practice, burden of administration, and burden on existing resources in the context of sustainability outside of the context of the study. All responses were compiled by participant group to allow for contrasts to emerge.

Respondents to the feasibility survey included 97 parents, 59 children, and 39 clinicians or research assistants. **TABLE 2** presents the percentage of participants who were in agreement with the presented statements. A large majority of children, parents, and clinicians agreed that participating in the follow-up project was useful for them, and that the tests used were helpful to track recovery or understand the concussion itself. Clinicians did not fully recognize how helpful the comprehensive approach was and how reassuring their participation was for the child and parents. Clinicians reported that the outcome measures contributed to providing them with a comprehensive

view of the child's functioning and that all measures could be administered as part of a clinical care visit. However, a significant minority of them were concerned with the sustainability of administering such a high volume of measures without the support of a research project.

It appears feasible to approach concussion follow-up from a more global perspective and to go beyond the assessment of patient-reported symptoms to determine children's function. However, to address clinicians' concerns about sustainability, it is essential to re-examine the proposed outcome measures and determine, through empirical analysis, whether all retained tools included in the CanPedCDE initiative are necessary to capture children's experiences of living with a concussion through a biopsychosocial lens.

What Can Be Done Right Now?

While understanding of the usefulness and validation of biopsychosocial models

TABLE 2

CANPEDCDE FEASIBILITY SURVEY RESULTS*

Survey Statement	Parents (n = 97)	Children (n = 59)	Clinicians (n = 39)
Common items for all groups			
Participating in the concussion study was useful for me	81	80	92
Participating in the concussion study helped me understand my/the child's concussion	79	86	64
Participating in the concussion study was reassuring	73	71	64
The tests performed during the visits were helpful to see how I/the child got better	79	76	92
The visits took a lot of my time	20	30	77
The visits lasted longer than I was told	11	0	21
The number of visits was just right	79	86	74
Common items for parents/children			
Everything I did will help future patients and their families	94	93	
Everything I had to do was well explained by the research staff	98	100	
I trusted the research staff during this study	99	98	
The visits were fun	90	61	
Thinking about the experience of being in the study, I would definitely be part of this study again	87	63	
Additional items for clinicians			
The number of tests and questionnaires that I had to administer as part of the concussion study could be done in my clinical setting			87
The extra resources required for the concussion study visits could be sustained in my clinical setting			67
The interdisciplinary nature of the testing provided a complete picture of the child's recovery			82

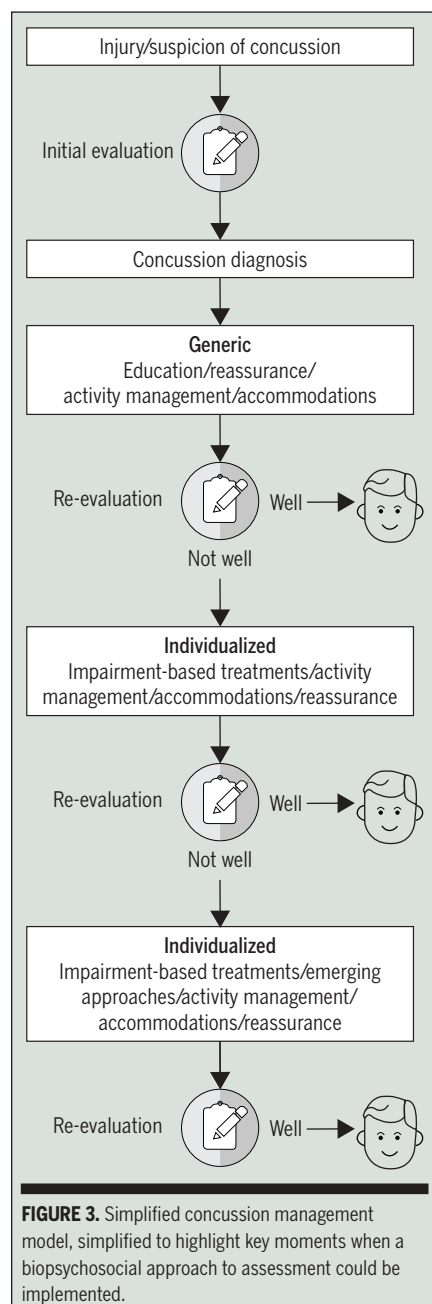
Abbreviation: CanPedCDE, Canadian Pediatric Mild Traumatic Brain Injury Common Data Elements.

*Values are percent of participants who agreed or strongly agreed.

of function after concussions continues to evolve, the question of how to integrate current understanding in follow-up clinics delivering postconcussion care is important. **FIGURE 3** identifies 4 key moments, proposed by our group,¹⁷ when intervention can be guided by adopting a multimodal approach to assessment. Clinical decisions in these key moments of transition rely heavily on how assess-

ments are performed to identify needs. While the initial evaluation may require addressing a larger number of domains with more screening measures, re-evaluations would be focused on thorough assessments of fewer constructs. For example, evaluating patient-reported postconcussion symptoms at the time of initial evaluation might consist of simple yes/no questions regarding key symptoms,¹⁹ while a later assessment would require rating a longer list of symptoms with a scale allowing for sensitivity to severity.⁴⁵ Evaluation of participation domains at the initial evaluation may be limited to documenting preinjury levels of school, leisure, and sport participation. The same participation domains would require a detailed assessment of level, performance, or satisfaction with participation at later stages of recovery. Documenting environmental factors, such as access to care or support from the school environment, would help clinicians target and tailor specific interventions for the child and family.

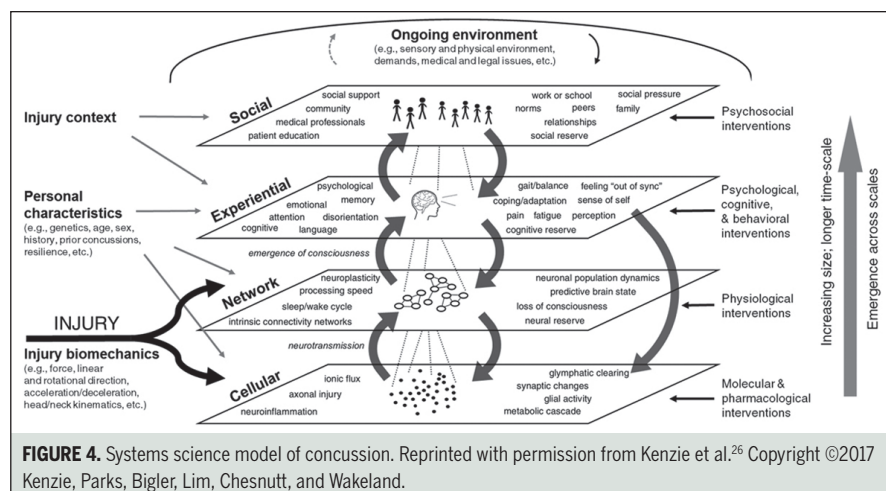
result of complex interactions between an individual's physical, psychological, social, and environmental factors. While there are undeniable underlying structural/physiological changes following concussion, other factors will contribute to determine how the individual copes. Children and adolescents will vary widely in how they respond to their symptoms, and also differ in how they cope. By adopting a more global view, clinicians and researchers could provide a holistic approach and contribute to a more successful and sustainable pediatric concussion care model. Promising avenues, such as that of Kenzie's²⁷ systems science-inspired model, propose an even more integrated and dynamic linking of key aspects of concussion injury and recovery than do biopsychosocial models (**FIGURE 4**).^{26,27,49} As our understanding of these complex processes increases, clinicians providing concussion care will need to adapt to this more encompassing way of approaching the condition and see symptoms as only one of the objects of our attention. ●



SUMMARY

MOST CURRENT PREDICTIVE MODELS of concussion recovery limit their definition of recovery to an absence of postconcussion symptoms, and thus appear grounded in more biomedical models of health. Biopsychosocial models recognize that disability is the

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The Epidemiology of Sport-Related Concussion: What the Rehabilitation Clinician Needs to Know

Concussion is a common injury in sport^{3,4} and occurs in athletes of all ages, with varying incidence rates across contact and limited-contact sports.⁷⁷ Historically, the incidence of concussion in athletes has been difficult to estimate due to lack of standardization of injury definitions and methods used to collect and report data from injury surveillance systems, underreporting, and underdiagnosis.⁵⁹ Concussion is also common in daily life and military service, and its epidemiology in the civilian and military populations is reported in a number of reviews.^{2,28,36,57} The purpose of this commentary is to summarize key concepts related to the epidemiology of sport-related concussion (SRC) in 2 sections: (1) the

methodological issues of concussion definition, surveillance systems, and advances in concussion knowledge; and (2) a summary of the data on SRC epidemiology.

Glossary of Terms

Epidemiology: the study of the distribution and determinants of disease, and

its application to the control of diseases and the measurement of disease outcomes in relation to a population at risk

Prevalence: the number of cases of a disease/injury/problem that are present in a particular population at a given time

Incidence: the rate at which the number of new events of a disease/injury/problem occur over a specific period

Point prevalence: the proportion of individuals in a population who have a disease, injury, or problem at a particular time, sampled at a single time point

Period prevalence: the proportion of the population that has or develops the disease, injury, problem within a specified period

PART 1: ISSUES IN SRC EPIDEMIOLOGY

A KEY CHALLENGE IN INJURY EPIDEMIOLOGY is clearly defining the injury, so that cases can be reliably counted. Definitions of injury should include the injury cause and the outcome.⁵⁴

• **SYNOPSIS:** Sport-related concussion is common in full-contact and collision sports. Epidemiology studies use different types of surveillance systems and concussion definitions. Concussion incidence rates vary across age, sex, sport, and level of competition. Incidence rates are increasing, likely due to higher rates of reporting following improved knowledge and increased regulations. In this review, we summarize 7 key concepts related to concussion epidemiology: concussion definition, changes in concussion knowledge, reliability and

accuracy of injury surveillance systems, conservative management and return to play, reliability of self-report, incidence of concussion across levels of play, and understanding the behaviors of players, coaches, and medical personnel from a multidisciplinary management perspective.

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• **KEY WORDS:** athletes, concussion, epidemiology, injury surveillance, management

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Definition of Injury and Definition of Concussion

Though a standard definition of concussion has been available for many years,^{61,63} consistently applying it has not been easy. The severity of concussion has not been defined in most sports, except in rugby, which defines it as “time loss” (ie, the number of days away from sport).

The range of injury definitions in sports injury surveillance systems includes injuries that result in presentation to an emergency department or sports medicine clinic treatment,^{1,21,23,71,82,93} injuries that result in absence from match play^{7,81} or a training session,^{11,86} and injuries that result in symptoms reported by the athlete.⁴² These different injury definitions will produce different injury rates.¹⁷

Epidemiological studies relating to concussion use different methods of data capture (surveillance systems) and different definitions of concussion. Some rely on data (often a diagnosis) provided by athletic trainers,^{12,18,48,73,80,88} and some use physician diagnosis.⁸

Examples of definitions of concussion used in epidemiological studies include the following:

1. “A trauma-induced alteration in mental status that may or may not involve loss of consciousness.”¹⁰
2. “A blow to the head followed by a variety of symptoms that may include any of the following: headache, dizziness, loss of balance, blurred vision, ‘seeing stars,’ feeling in a fog or slowed down, memory problems, poor concentration, nausea, or vomiting. Getting ‘knocked out’ or being unconscious does not always occur with a concussion.”^{47,59}
3. “When a blow or jolt to the head causes problems such as headaches, dizziness, being dazed or confused, difficulty remembering or concentrating, vomiting, blurred vision, or being knocked out.”²²

The Concussion in Sport Group⁶¹ defines concussion as “a traumatic brain injury induced by biomechanical forces,”

including “several common features that may be utilised in clinically defining the nature of a concussive head injury”:

- (1) “Caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head”
- (2) “Results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously; however, in some cases, signs and symptoms evolve over a number of minutes to hours”
- (3) “May result in neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies”
- (4) “Results in a range of clinical signs and symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive features typically follows a sequential course. However, in some cases symptoms may be prolonged”⁶¹

Loss of consciousness is not required for a diagnosis of concussion. There are no specific criteria provided to define or quantify “short-lived impairment of neurological function.” For example, retrograde amnesia, posttraumatic amnesia, and confusion are not discussed in the definition.⁶¹

There is no consensus on the lowest threshold for defining and diagnosing concussion.⁶⁴ In its mildest form, a transient neurological event⁵³ might resolve spontaneously in seconds or minutes, with no clinical manifestations being detectable after this resolution. Whether this represents the lowest threshold of injury or a so-called “subconcussive” blow to the head^{4,20,33,83} remains an open question. The criteria and/or threshold used for the definition of concussion have a direct effect on the reported concussion incidence.

Operational definitions of concussion have also been proposed^{74,75} to expand on theoretical or conceptual definitions and

to increase recognition and sensitivity of diagnosis.⁷⁵ World Rugby has recognized the variable natural history of concussion, appreciating the possibility that a concussion may be transient, fluctuate, have a delayed onset, or have signs and symptoms that evolve over time. As such, a 3-stage diagnostic process was introduced that included (1) immediate postinjury assessment, (2) repeated assessment within 3 hours, and (3) a follow-up assessment at 36 to 48 hours post injury.⁷⁵ Concussion can be diagnosed at any of these time points. Given the natural history of the condition, it is not possible to rule out or exclude a concussion until the athlete has been re-evaluated 36 to 48 hours following the event.⁷⁵ The criteria adopted to define concussion will have a direct effect on the reported concussion incidence.

Diagnosis of Concussion

Variations in the definition of an injury and the definition of concussion make it challenging to compare studies and produce reliable incidence data. It is important to consider the reliability of reported incidence rates of concussion diagnosis. Many professional leagues (eg, the National Rugby League, Australian Football League, World Rugby, National Hockey League, and National Football League) have adopted policies that allow a team physician to remove an athlete from play following a suspected concussion. The team physician can examine the athlete to determine his or her fitness to return to play during the same game (referred to in rugby union and rugby league as the “head injury assessment” [HIA]). Some athletes who are removed from play under the HIA and who do not return to play in the same game may be kept out of play for reasons other than a concussion. In such circumstances, it is important to clarify whether the injury has been recorded in surveillance systems as a concussion, because incidence rates can be overestimated when all HIA events resulting in a player being permanently removed from play are recorded as a concussion.

Impact of Surveillance Systems on Reported Concussion Rates

The people who collect the data and their incentives can impact concussion rates. In a study of South African schoolboy rugby injuries, there was substantial underreporting of concussion by schools that obtained data by sending forms (instruction forms, weekly report forms, and injury questionnaire forms) to all 20 of the participating schools' teacher-coaches and requiring them to complete the injury report forms every week. The data were more accurate when collected by researchers.⁷⁸

There is limited information about the proportion of concussions that are not captured by injury surveillance systems, because any or all of the following apply:

- A concussion was sustained by a player, but no signs of concussion were apparent to observers (an "unobserved" concussion).
- A concussion was sustained by a player and the event may or may not have been observed, but the player did not report his or her injury (an "unreported" concussion).
- A concussion was sustained by a player and the player reported the injury to a medical practitioner or data collector, but the medical practitioner or data collector did not make a diagnosis of concussion, or the person classified the injury as something other than a concussion (an "undiagnosed" concussion).

Changes in Knowledge and Awareness of Concussion

Greater knowledge and awareness of injury among athletes, coaches, parents, athletic trainers, and physicians increase the likelihood of self-identification, injury surveillance reporting, and physician diagnosis,⁸⁰ ultimately influencing concussion incidence and lifetime prevalence rates.^{18,48,55,77,94} The number of concussion diagnoses is increasing.³ A 10-year study of 25 high schools found that the rate of reported concussions increased more than 4-fold over the study period.⁵⁵

High school athletes have longer recovery times, fewer athletes have signs typically considered to be more severe (eg, loss of consciousness and amnesia), and more athletes are reporting symptoms (eg, drowsiness, irritability, light sensitivity), which suggests that clinicians may have lowered the threshold in diagnosing concussion.¹⁸ In the United States, nationwide state-level concussion legislation and associated education efforts have facilitated recognition of possible lingering symptoms and contributed to a longer documented symptom resolution time.¹⁸ Emergency department visits for concussion have increased,^{25,44} likely because of increased knowledge and awareness of (and concern for) concussion rather than increased incidence.⁷²

Reliability and Accuracy of Surveillance Systems

National injury databases rely on estimates that are based on administrative claims,⁵ which are limited to fit an International Classification of Diseases code. The International Classification of Diseases code has limited sensitivity and specificity for SRC, and is prone to underestimation.⁵ Surveillance systems need better capacity for recording comprehensive injury information. High School Reporting Information Online,¹⁵ the National Collegiate Athletic Association Injury Surveillance Program, and the National Electronic Injury Surveillance System All Injury Program are examples of next-generation injury databases. Each of these systems is web based and enables a broad range of incident information to be uploaded by athletic trainers.

There are no effective mechanisms to consistently and inexpensively link various surveillance data sets, or to follow individual athletes across sports, tracking systems, or the age continuum—key limitations to current surveillance systems.⁵ In 2012, the Ivy League and Big Ten conferences in the United States combined to establish a surveillance system. Athletic trainers complete a data-collection in-

strument, entered online, for each concussive event.¹²

The Impact of Management and Return-to-Play Decision Making on Injury Incidence Rates

In the 1980s and 1990s, it was relatively common for athletes to return to play following concussion during the same game if their signs and symptoms resolved. It was also relatively common for athletes to return to play while still symptomatic. In the 21st century, there has been increased vigilance toward identifying concussion and avoiding same-day return to play.⁶² It is more common for athletes to be managed more conservatively, from a medical perspective, and to follow a gradual return-to-play procedure.⁶⁰⁻⁶³ Epidemiological studies documenting "time loss" or recovery times published in the 1990s, 2000s,¹⁸ and in recent years^{30,72} may yield different findings, based, in part, on the evolution of how the injury is medically managed. Documenting the recovery times may or may not overcome some of the limitations pertaining to the accuracy of the data entered into national surveillance systems.

Reliability and Accuracy of Self-reported Concussion

The source of information regarding concussion history is most often athlete self-report. Adolescent and young adult athletes may underreport or inconsistently report their concussion history.^{59,66,91} Among adolescent ice hockey players asked to report their concussion history on 2 self-report instruments administered during the same session, 1 in every 5 players provided inconsistent information; 1 in 10 reported fewer concussions and 1 in 10 reported more concussions.⁶⁶

Among professional National Football League players, 7% reported fewer concussions during a second administration of a screening tool completed 9 years after the first administration.⁴⁶ Among approximately 5000 high school student-athletes with concussion, only a small proportion (n = 181, 4%) provided

inconsistent information about their concussion history when reassessed between 90 days and 3 years later. In a subgroup of injured athletes ($n = 587$), 17% reported no change in their concussion history on postinjury testing.⁹² Athletes reporting a greater number of lifetime concussions at initial assessment had greater disagreement in concussion reporting at a subsequent assessment,^{66,92} suggesting that consistent recall of concussion history may be more difficult for adolescent athletes with a greater number of concussions at baseline.⁹²

Athletes may not report concussion for many reasons, including (1) lack of awareness of the injury, (2) belief that concussion is not a serious injury, (3) not wanting to be withheld from participation, (4) not wanting to let the team down, and (5) feeling pressure to maintain their position on the team.^{14,16,19,59,65,69,87} This is a problem because the athlete may be more vulnerable to repeated injury, and possibly an exacerbation of symptoms and a longer recovery time. Among Australian high school rugby union players with suspected concussion, 4 of every 5 returned to play prior to seeking medical clearance.⁸⁵ The rate of compliance with medical advice in community rugby union players revealed that of the 22% of players who had been provided with return-to-play advice, none complied with the return-to-play regulation.³⁹

Injury Incidence Rates: The Challenge of Assessing Exposure Time

An injury incidence rate is calculated as the number of events—in this case, concussions (the numerator)—divided by some unit of athlete time for a population at risk (the denominator).^{34,84} Numerators may vary based on underreporting or overreporting of concussion or inaccurate diagnosis, and denominators can be difficult to accurately track.³⁴ For team sports, exposure is best assessed as the number of minutes each athlete plays, because different playing positions have different exposure rates. Using team exposure can lead to the underestimation

or overestimation of individual athletes. However, it can be very challenging, time consuming, and resource intensive to collect these data.⁸⁴

When individual-level data are unavailable, sports injury epidemiologists typically calculate either “athletes at risk” or “athlete participation.”⁸⁴ The athletes-at-risk method uses the number of players on the field multiplied by either the team’s games played or game-hours. This may be represented as injuries per athlete-games or, more commonly, as injuries per 1000 athlete-games. The athlete-participation method uses athlete-exposure (AE), meaning 1 athlete participating in 1 practice or game. In some instances, “participation” may be defined as athletes who were involved in the game, while in other instances, all athletes recorded on the team sheet (even those who did not actually enter the game) are included. That is, all participating athletes are counted equally, even though exposure time ranges from full to partial to (potentially) no involvement.⁸⁴

Estimates of risk may change dramatically depending on whether hours of participation are tracked or a seasonal or annual risk of concussion is determined.³⁴ The American Medical Society for Sports Medicine concluded that seasonal or annual risk may be a more readily understood concept.³⁴ The above limitations require consideration when assessing the reported rates of concussion and comparing these rates within a sport over time, or comparing rates across various sports.⁵¹

PART 2: EPIDEMIOLOGY OF SRC

THIS SECTION PROVIDES EPIDEMIOLOGICAL information relating to injury rates and a review of lifetime history and sport-specific injury rates. It also discusses injury rates in relation to level of competition and type of protective equipment worn.

Lifetime History of Concussion and Sport-Specific Rates

Children and Adolescents Concussion is a common injury among young athletes.^{3,58,68}

TABLE 1

INCIDENCE RATES OF SPORT-RELATED CONCUSSION IN HIGH SCHOOL ATHLETES DURING THE 2008-2009 AND 2009-2010 SEASONS ACROSS SPORTS*

Sport	Concussions per 100000 Athletic-Exposures
Boys football	76.8
Boys ice hockey	61.9
Boys lacrosse	46.6
Girls soccer	33.0
Girls lacrosse	31.0
Girls field hockey	24.9
Boys wrestling	23.9
Boys basketball	21.2
Boys soccer	19.2
Girls basketball	18.6
Girls softball	16.3
Cheerleading	11.5
Girls volleyball	8.6
Girls gymnastics	8.2
Boys baseball	4.6
Boys track and field	3.5
Girls track and field	1.4

*Adapted with permission from Meehan et al.⁶⁸

It has been reported to represent between 9%⁴³ and 13%⁵⁸ of all high school athletic injuries. There may be higher rates of concussion in high school athletes compared to adult athletes.^{56,67} The incidence

of concussion in children and adolescent athletes (younger than 18 years of age) across 12 sports was reported as 0.23/1000 AEs. Rugby, ice hockey, and American football were the sports with

the highest incidence rates of concussion among this age range (4.18, 1.20, and 0.53/1000 AEs, respectively).⁷² A meta-analysis of incidence rates of concussion in schoolboy rugby union (n = 11 studies) reported an incidence rate of 0.62 concussions per 1000 player-match hours (range, 0.19-10.6).²⁷ A summary of the incidence of SRC in high school athletes across 2 academic years is presented in **TABLE 1**.⁶⁸

Adolescent Student-Athletes Iverson and colleagues⁴¹ reported lifetime history of concussion rates in a large cohort of adolescent student-athletes. Between 2009 and 2013, 32 487 students between 13 and 18 years of age (mean $\pm$ SD, 15.5 $\pm$ 1.3 years), from the state of Maine in the United States, completed baseline preseason testing with Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT Applications, Inc, San Diego, CA), a computerized program that includes measures of symptom ratings and cognitive functioning intended for concussion management. As part of the program, students completed a health survey relating to attention deficit hyperactivity disorder (ADHD), learning problems, health problems, and concussion history. In the total sample, 17% reported a history of 1 or more prior concussions, 5% reported 2 or more prior concussions, and 2% reported 3 or more prior concussions. Stratifying the total sample by sex, 20% of boys and 14% of girls reported 1 or more prior concussions, 6% of boys and 4% of girls reported 2 or more prior concussions, and 2% of boys and 1% of girls reported 3 or more prior concussions. Youth with ADHD reported a greater lifetime history of concussion. Specifically, 26% of boys and 21% of girls with ADHD reported a history of 1 or more prior concussions, compared to 19% of boys and 13% of girls who reported that they did not have ADHD.

College Athletes Concussion represents 6% of all collegiate athletic injuries.⁹ In collegiate football, the reported lifetime prevalence of concussion has ranged from 21%⁷⁹ to 25%.⁴⁰ College football

TABLE 2

CONCUSSION RATES IN HIGH SCHOOL AND COLLEGIATE ATHLETES IN THE UNITED STATES FOR THE 2005-2006 SCHOOL YEAR*

Sport/Division	Concussions per 1000 Athletic-Exposures		
	Practice	Competition or Match Play	Overall
Football			
High school	0.21	1.55	0.47
Collegiate	0.39	3.02	0.61
Boys soccer			
High school	0.04	0.59	0.22
Collegiate	0.24	1.38	0.49
Girls soccer			
High school	0.09	0.97	0.36
Collegiate	0.25	1.80	0.63
Volleyball			
High school	0.05	0.05	0.05
Collegiate	0.21	0.13	0.18
Boys basketball			
High school	0.06	0.11	0.07
Collegiate	0.22	0.45	0.27
Girls basketball			
High school	0.06	0.60	0.21
Collegiate	0.31	0.85	0.43
Wrestling			
High school	0.13	0.32	0.18
Collegiate	0.35	1.00	0.42
Baseball			
High school	0.03	0.08	0.05
Collegiate	0.03	0.23	0.09
Softball			
High school	0.09	0.04	0.07
Collegiate	0.07	0.37	0.19
Boys overall total			
High school	0.13	0.61	0.25
Collegiate	0.30	1.26	0.45
Girls overall total			
High school	0.07	0.42	0.18
Collegiate	0.23	0.74	0.38
Overall total			
High school	0.11	0.53	0.23
Collegiate	0.28	1.02	0.43

*Concussion rates reported from the High School Sports-Related Injury Surveillance Study and the National Collegiate Athletic Association Injury Surveillance System, as reported in Gessel et al.²⁹ Adapted with permission from Gessel et al.²⁹

TABLE 3

INCIDENCE OF CONCUSSION IN THE NATIONAL FOOTBALL LEAGUE IN THE PRESEASON, REGULAR SEASON, AND POSTSEASON FROM 2012 THROUGH 2017*

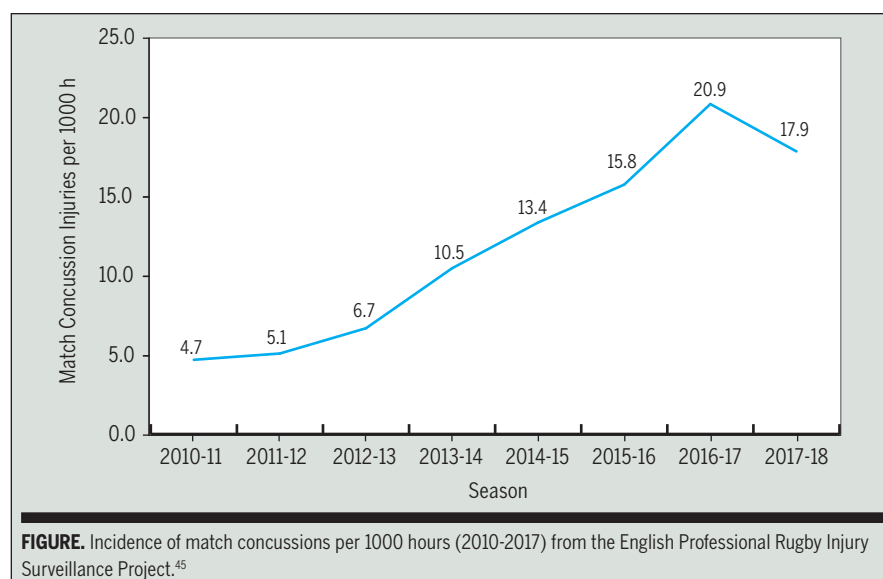
Year	Preseason			Regular Season and Postseason			Full Season		
	Practice	Game	Total	Practice	Game	Total	Practice	Game	Total
2012	42	43	85	3	177	180	45	220	265
2013	39	38	77	4	163	167	43	201	244
2014	42	41	83	8	121	129	50	162	212
2015	29	54	83	9	187	196	38	241	279
2016	26	45	71	7	172	179	33	217	250
2017	45	46	91	11	189	200	56	235	291
Total	223	267	490	42	1009	1051	265	1276	1541

*Adapted with permission from <https://www.playsmartplaysafe.com/newsroom/reports/2017-injury-data/>.

players with a history of previous concussions were more likely to have future concussions than were collegiate football players with no history of concussion.³¹ One in 15 concussed football players sustained a subsequent concussion in the same season.³¹ Players who sustained a concussion in a season were 3 times more likely to sustain a second concussion in the same season compared with uninjured players.³²

A large majority of same-season repeat concussions occurred within the first 2 weeks of the first concussion,¹³ which has been far less likely in recent years due to more conservative management and a gradual, stepwise return-to-play approach. In collegiate-level ice hockey, 1 study reported concussion rates based on the sex and level of play (ie, Division I versus Division III).⁷⁶ The highest concussion rate was found in Division I men (0.83 per 1000 AEs), followed by Division III women (0.78 per 1000 AEs), Division I women (0.65 per 1000 AEs), and Division III men (0.64 per 1000 AEs).⁷⁶ A summary of concussion rates reported in the High School Sports-Related Injury Surveillance Study and the National Collegiate Athletic Association Injury Surveillance System is presented in TABLE 2.

Professional Athletes Annual injury reports in professional leagues have been produced for a number of years. However, the definition of injury, and the definition



of concussion, has varied across sports, making comparisons challenging. The criteria for diagnosing concussion within the same professional competition have also changed over time. The National Football League's reported incidences of concussion from 2012 through 2017 are presented in TABLE 3.

A systematic review of the concussion incidence rates from the National Hockey League published in 2016⁵² reported a 10-fold increase in the rate of concussion from the 1986-1987 season to the 2011-2012 season. The lowest incidence rate was during the 1986-1987 season (0.42 concussions per 100 games),⁹⁰ and the second highest and highest in-

cidence rates were in 2010-2011 (4.35 concussions per 100 games) and in 2011-2012 (4.88 concussions per 100 games), respectively.²⁴

Prior to the National Hockey League lockout (a labor dispute between the National Hockey League and the National Hockey League Players' Association resulted in the cancellation of the 2004-2005 season), the concussion incidence rate was 2.89 concussions per 100 games. The rate decreased 28% to 2.07 concussions per 100 games in the season following the lockout, but has risen each season since. "Rule 48" was created in an effort to eliminate a type of game play known to be a risk factor for concussion (ie, checks to the head) and was

implemented at the beginning of the 2010-2011 season.

A summary of the incidence rates of concussion during the regular season in the National Hockey League is provided in **TABLE 4**.⁵² Donaldson et al²⁴ reported the rates of “suspected concussion,” which likely accounts for the increased rates reported for seasons 2009-2010, 2010-2011, and 2011-2012. All other rates reported are medically diagnosed concussions.

In Australian professional rugby league, the concussion incidence rates vary dramatically across studies, in large part due to different criteria for defining injury. Using the more liberal injury definition, between 8.0 and 17.1 injuries per 1000 playing-hours have been reported.²⁶ In rugby league, concussion accounted for 29% of all injuries associated with illegal play and 9% of injuries sustained in legal play,²⁶ suggesting that rule modification and stricter officiating might re-

duce the rate of concussion in that sport.

A pooled analysis of concussion injury incidence revealed 7.7 concussions per 1000 match-hours (95% confidence interval [CI]: 6.8, 8.7) in rugby league.⁵⁰ A meta-analysis of incidence rates of concussion in professional rugby union players (n = 16 studies) reported an incidence rate of 1.19 concussions per 1000 player-match hours (range, 0.33-7.8).²⁷ King and colleagues⁴⁹ reported an overall incidence of 51 concussions per 1000 player-hours. There was a 5-fold difference between witnessed (n = 8) and unwitnessed (n = 44) concussions. The incidence rate of unwitnessed concussions was 51 concussions per 1000 player-hours.⁴⁹

In the Rugby Football Union injury surveillance report from the 2016-2017 season,⁴⁵ the most commonly reported match injury was concussion, accounting for 20% of all match injuries. The incidence of concussions during match play between 2010 and 2018 is illustrated in the **FIGURE**. The mean severity of medically diagnosed match concussions, defined as time lost from sport, in 2017-2018 was 19 days. Concussion accounted for 18% of all injuries to the ball carrier and 37% of all injuries to the tackler.⁴⁵

Protective Equipment

The protective benefit of headgear is a topic that requires further investigation. The prevailing opinion has been that headgear provides limited or no effect on reducing the risk of concussion, although this may be partly due to a lack of evidence being interpreted as a lack of effect. However, in recent years, biomechanical research relating to helmets and other types of protective headgear suggests that there are important differences between helmet and headgear types in regard to reducing both linear and rotational acceleration to the head and brain.

There are a number of factors besides the potential direct benefits of wearing headgear that require consideration. For example, full-contact and collision-sport athletes who choose to wear headgear may be the type of players who

TABLE 4

A SUMMARY OF THE INCIDENCE RATE OF CONCUSSION IN THE REGULAR SEASON OF THE NHL*

Study/Season	Concussion Rate†
Wennberg and Tator ⁹⁰	
1986-1987	0.42
1987-1988	0.83
1988-1989	0.71
1989-1990	0.71
1990-1991	0.54
1991-1992	0.46
1992-1993	0.69
1993-1994	0.69
1994-1995	0.64
1995-1996	0.80
1996-1997	1.27
Benson et al ⁶ ; Wennberg and Tator ^{89,90}	
1997-1998	2.52 ± 0.46
1998-1999	3.54 ± 0.48
1999-2000	2.95 ± 0.23
2000-2001	3.62 ± 0.73
2001-2002	3.05 ± 0.75
Benson et al ⁶ ; Wennberg and Tator ⁸⁹	
2002-2003	3.07 ± 0.20
2003-2004	2.89 ± 0.06
2004-2005	NHL lockout
Wennberg and Tator ⁸⁹	
2005-2006	2.07
2006-2007	2.36
2007-2008	2.52
2008-2009	No reported data
Donaldson et al ²⁴	
2009-2010	2.76
2010-2011	4.35
2011-2012	4.88

Abbreviation: NHL, National Hockey League.

*Adapted with permission from Kuhn and Solomon.⁵²

†Incidence rate reported in concussions per 100 games.

‡Reported rates include “suspected concussion” as determined by the authors.

also take fewer risks during games, and may therefore adopt a style of play that places them at less risk of injury.³⁸ In contrast, wearing so-called “protective equipment” like headgear may encourage some players to play more aggressively and therefore place themselves at greater risk of injury.

In a cohort of 3207 nonprofessional male rugby union players aged 15 years and older from metropolitan Sydney, Australia, players who “always” wore headgear during games had a significantly reduced risk of concussion.^{37,38} Players who reported that they rarely wore headgear during games (incidence rate = 12.6; 95% CI: 8.4, 18.3) had almost double the incidence of concussion compared to players who reported that they always wore headgear (incidence rate = 7.4; 95% CI: 5.6, 9.7).³⁸ However, the concussion incidence in players who reported never wearing headgear was almost identical to the incidence in players who always wore headgear (7.5 [95% CI: 6.0, 9.2] versus 7.4 [95% CI: 5.6, 9.7]).³⁸ In a 3-season study of 757 professional rugby players in the United Kingdom, players who did not wear headgear had a higher rate of concussion (4.6 per 1000 player-hours; 95% CI: 3.7, 5.7) than those who did wear headgear (2.0; 95% CI: 1.0, 4.2).⁴⁵

A rigorous evaluation of the benefits (or lack thereof) of appropriate headgear and the relationship with concussion in full-contact and collision-sport athletes is required. A summary of the level of evidence by Navarro⁷⁰ shows that the use of customized mandibular orthosis in American football has a high level of evidence (4), but no effectiveness in preventing concussion. In rugby, a mouthguard and headgear have a low level of evidence (2), but have no effectiveness and inconclusive effectiveness, respectively. In soccer, headgear has no effectiveness and a level of evidence of 3 for preventing concussion. And in field hockey and ice hockey, a face shield has inconclusive and no effectiveness, respectively, and levels of evidence of 4 and 2 for preventing concussion, respectively.

Directions for Future Research

Future epidemiological studies should consider the longitudinal examination of SRC incidence while considering differences stratified by age, sex, ethnicity, level of competition, and playing position. However, when considering incidence rates both within sports over time (intrasport comparisons) and across sports (intersport comparisons), until there is universal agreement on the operational definition of concussion for the numerator and a standardized exposure measure for the denominator, such comparisons are impossible. It is critical for epidemiologists to lead the field to consensus on these issues in order to produce meaningful comparisons.

SUMMARY

THERE ARE A NUMBER OF FACTORS TO take into account when considering the epidemiology of SRC.^{64,74,75} One of the greatest challenges is determining reliable methods for calculating and comparing concussion incidence rates, both within a sport over time and between sports. Factors such as the definition of concussion, changes in knowledge, more conservative management, increased readiness of athletes to self-report, and the calculation of the denominator are all important. In providing incidence rates, it has been suggested that seasonal or annual risk may be a more readily understood concept.³⁵ Estimates of risk for concussion may change dramatically when hours of participation are tracked or a seasonal or annual risk of concussion is determined.³⁵ Researchers, the medical community, and the general public are interested in injury data that generally are not presented in articles, such as the injury rates for total participants in a single season for a specific sport at a specific level of play. Injury rates per 1000 playing-hours are important to present for standardization and cross-comparison purposes, but they are difficult to interpret. ●

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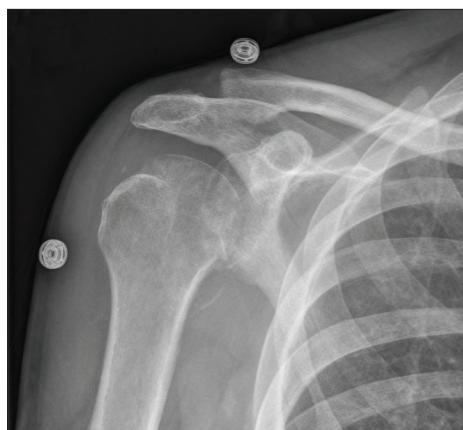


FIGURE 1. An anteroposterior radiograph of the right shoulder showing decreased bone density, osteolysis, and fragmentation of the glenohumeral head and articular surfaces. Bony fragments are seen scattered throughout the capsule.

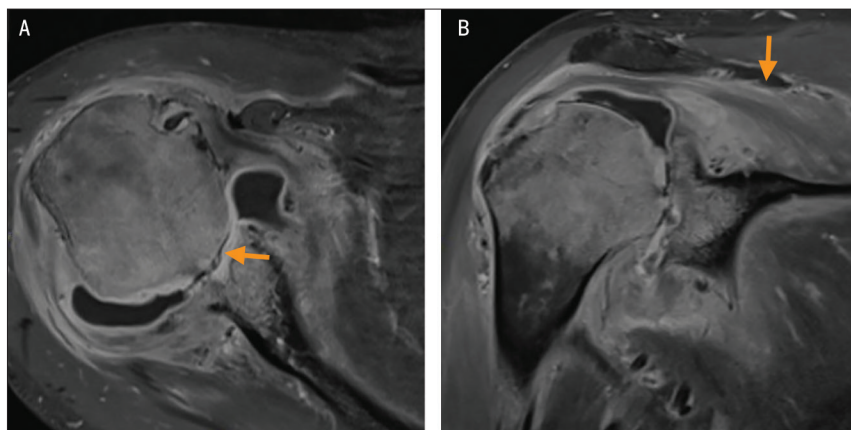


FIGURE 2. T1-weighted, fat-saturated, contrast magnetic resonance image of the right shoulder, with the (A) axial view showing osseous erosion and destruction of the medial aspect of the humeral head and glenoid fossa (arrow), consistent with osteomyelitis. (B) The coronal oblique view shows edema of the supraspinatus (arrow), consistent with myositis. Enhancing T1 marrow replacement is visualized in the glenoid and humeral head extending to the surgical neck.

Osteomyelitis of the Glenohumeral Joint

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A 60-YEAR-OLD MAN PRESENTED TO the emergency department with constant right shoulder pain of insidious onset that had lasted for 1 month. Available history included cervical spine osteomyelitis and C6-T1 laminectomy 5 months prior, with loss to follow-up after completing two 6-week courses of intravenous antibiotics. He described severe pain with movement and alleviation with the elbow flexed and the shoulder adducted and internally rotated against his abdomen. He reported homelessness, “long-time” daily alcohol consumption, and 1-month relapse to intranasal cocaine use for pain management, after 5 years of sobriety.

While awaiting initial radiograph results, the patient was referred to the emergency department’s physical therapist

for frozen shoulder. Examination revealed no active right shoulder motion due to pain. Passive range was pain limited to 0° of external rotation and 20° of flexion, with an empty end feel. Palpation of the quadrangular space produced radiating symptoms to the anterior shoulder. There was no fever, joint erythema, warmth, or edema. The left shoulder demonstrated pain-free limited motion in a capsular pattern. The cervical spine screen revealed no significant findings.

Based on history of osteomyelitis, drug relapse, pain severity, and side-to-side differences in shoulder examination, the physical therapist recommended magnetic resonance imaging of the right shoulder for suspected infection.¹ Upon receiving physical therapist recommendations and reviewing initial radiographs

(FIGURE 1), the physician ordered magnetic resonance imaging, which revealed shoulder osteomyelitis and myositis (FIGURE 2; FIGURE 3, available at www.jospt.org). The patient was admitted for intravenous antibiotics, irrigation, debridement, and antibiotic spacer placement, with eventual reverse total shoulder arthroplasty (FIGURE 4, available at www.jospt.org).

This case highlights a person-centered approach to clinical decision making in light of atypical and vague musculoskeletal findings. In the absence of classic joint infection signs and symptoms, the physical therapist had to consider patient history and social situation in the clinical decision for further diagnostic imaging. ● *J Orthop Sports Phys Ther* 2019;49(11):865. doi:10.2519/jospt.2019.9101

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How Do We Meet the Challenges of Assessing and Managing Concussion?

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The past decade has witnessed many advances in the assessment, diagnosis, prognosis, and management of the patient with mild traumatic brain injury (mTBI) or concussion and its varied sequelae. However, the condition continues to frustrate clinicians and researchers due to the lack of (1) clear definition of the injury, (2) identification of the cause (sports related, trauma based, a fall or other external forces impacting the head/neck) of the injury, (3) consistent evidence-based interventions, and (4) valid biomarkers to explain the outcome on a patient-by-patient basis across the life span.

Because many mTBI cases go unrecognized, underreported, and potentially untreated, concussion continues to be a

significant public health concern. While many patients seek and receive care in an emergency setting, some also receive primary and follow-on care from a number of health care professionals, including physicians, athletic trainers, chiropractors, exercise physiologists, neuropsychologists, and physical therapists. All of these professionals undoubtedly intend to effectively assess and manage the pa-

tient with mTBI (concussion). However, given the variability of the clinical presentation, dissimilar screening and evaluation processes further contribute to practice variability. The specialty nature of physical therapy practice can raise challenges in this regard. Therapists who are expert in managing one type of impairment (eg, musculoskeletal, vestibular) may be more thorough in identifying and managing deficits in their area of expertise.

Though a few screening tools have been developed specifically for concussion (eg, the Rivermead Post Concussion Symptoms Questionnaire,¹⁰ the Post-Concussion Symptom Scale,¹¹ and the

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Child Sport Concussion Assessment Tool Fifth Edition⁵), the vast majority of outcomes measures have been adapted from other conditions, such as headache (with or without migraine), neck pain (eg, the Neck Disability Index¹⁵), whiplash-associated disorders, vestibular deficits (eg, Dizziness Handicap Inventory⁸), and other neuromusculoskeletal conditions. The overlapping and interactive nature of multiple body systems in postconcussion presentation challenges the acumen of even the most astute clinician. Impairments following brain injury can be diverse, nonspecific, and have many possible causes (eg, brain injury, autonomic or sleep dysfunction, visual impairments, cervicogenic causation). Accurately identifying the underlying cause of concussion impairments helps the clinician prioritize assessment and management, including appropriate referral to other clinicians with complementary specialties.

There is ongoing epidemiologic debate as to what concussion is, the time required for recovery, who should best assess and manage concussion, and whether best practice is defined by targeting (1) the cardinal signs/symptoms resulting from injury to head/neck structures, (2) the adverse neuropsychological sequelae, or (3) both. Although the onetime standard practice, “rest until asymptomatic,” is no longer advised, there is limited evidence to clearly define the timing of rehabilitation versus allowing natural recovery to occur with a gradual return to typical activities.

As the field progresses, there is a point of interdisciplinary convergence around assessment, prognosis, management, and referral. This special issue supports and represents an interdisciplinary approach to assessment, management, and treatment of mTBI/concussion. The papers cover the epidemiology of sport-related concussion⁷; innovative imaging and molecular biomarkers⁴; injury classification schemas^{1,2}; patient-centered models of individualized assessment, diagnosis, and management^{12,14}; new evidence highlighting psychosocial factors that can drive the

clinical course³; links to cervical spine disorders⁹ (such as whiplash injury¹³); and current concepts in evaluating outcomes in children and youth after concussion.⁶ The articles further highlight the evolving role of the physical therapist throughout the clinical course post concussion.

Substantial work in sports and military concussion has provided many important lessons to guide current practice. But there is much to be learned about concussion recovery in other populations commonly seen in physical therapy (eg, children, older adults, and individuals who wish to return to work post concussion). Physical therapists interact with patients across the life span and are ideally placed to (1) screen for and identify concussion in those who may not have been diagnosed, and (2) provide intervention for impairments that stem from the musculoskeletal, vestibulo-ocular, and exertional systems.

It is incumbent on every clinician to recognize when to refer to other professionals, within or outside their practice, who may have specialty knowledge and experience in treating these patients. Interdisciplinary collaboration to study approaches and models for assessing, diagnosing, and treating concussion will help clinicians provide optimal care. It is with gratitude to the authors who contributed to this special issue of the *Journal of Orthopaedic & Sports Physical Therapy* that we present the evidence and practices currently available to clinicians in the assessment and treatment of mTBI/concussion. ●

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Can the Neck Contribute to Persistent Symptoms Post Concussion?

A Prospective Descriptive Case Series

Concussion is a condition that can affect anyone, at any stage of life. The incidence of mild traumatic brain injury is estimated to be 749 cases (95% confidence interval: 709, 790) per 100 000 person-years.¹¹ Approximately half (47.9%) of people report substantial persistent symptoms 1 year following injury.⁴⁵ The economic and social burdens of concussion injury are increasingly recognized, with New Zealand data reflecting international trends.^{5,26,34} For the purposes of this paper, concussion is considered synonymous with mild traumatic brain injury.³³

Clinicians must consider the neck as an alternative source of symptoms when evaluating people following a potential concussion. While concussion is defined as a brain injury,³⁴ it is well recognized

that persistent symptoms post concussion can arise from other structures.^{9,27,32} Many hallmark symptoms post concussion are not specific to concussion,^{1,27,29,44} and several of the most common symptoms, including headache, dizziness, and neck pain,^{7,19} are characteristic of neck conditions.^{2,25,42,46} Persistent symptoms post concussion “do not necessarily reflect ongoing physiological injury to the brain.”³⁴ This is reflected in several studies that draw parallels between whiplash-associated disorder and concussion: similar cognitive deficits,^{1,44} biomechanical injury forces,⁸ and injury occurrence.¹⁵ It is also consistent with descriptions of upper cervical dysfunction in people with headache post concussion.⁴⁷ Multimodal assessment, including a focused physical examination, must identify specific issues that may contribute to persistent symptoms.³⁴ Concussion can affect multiple systems concurrently, and assessment and management of persistent symptoms are best performed by a multidisciplinary team on an individual basis.^{31,34,42}

When neck-related problems that may contribute to persistent symptoms post concussion are identified, clinicians can offer specific tailored treatment.^{32,34,42,43} This has great potential, as high-quality randomized controlled trials report that neck-related headaches,¹⁷

● **BACKGROUND:** Persistent symptoms post concussion can arise from a range of sources, including the neck. There is little description of neck assessment findings in people with persistent symptoms post concussion.

● **OBJECTIVES:** To assess people with persistent symptoms following a concussion and determine whether the neck has also been injured, and to evaluate the potential of the neck to contribute to their symptoms.

● **METHODS:** A consecutive series of participants (n = 20) referred for neck assessment were prospectively recruited by 2 providers of a multidisciplinary concussion service for people with persistent symptoms. Data were collected at initial assessment and on completion of neck treatment, which included standard questionnaires (Rivermead Post Concussion Symptoms Questionnaire, Neck Disability Index, Dizziness Handicap Inventory); patient-reported measures of headache, dizziness, and neck pain; physical examination findings; and details of comorbidities.

● **RESULTS:** Participants were evaluated at a mean of 7.5 weeks post concussion (median, 5 weeks). On neck assessment, 90% were considered by the clinician to have a neck problem contributing to their current symptoms. Multiple findings were consistent with this view, including moderate-to-severe Neck Disability Index scores (mean ± SD, 33.4 ± 9.5 points), frequent neck pain (85%), frequent moderate-to-severe pain on occiput-C4 segmental assessment (85%), a positive flexion-rotation test (45%), and muscle tenderness (50%-55%).

● **CONCLUSION:** Multiple findings were indicative of concurrent neck injury, particularly involving the upper cervical spine. These neck-related findings are important to recognize, as they have the potential to contribute to persistent symptoms post concussion and may respond to neck treatment. This study was prospectively registered with the Australian New Zealand Clinical Trials Registry (ACTRN12616001183471). *J Orthop Sports Phys Ther* 2019;49(11):845-854. Epub 1 Jun 2019. doi:10.2519/jospt.2019.8547

● **KEY WORDS:** brain concussion, cervical spine

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dizziness,³⁹ and neck pain²⁴ can be effectively treated by physical therapists. In sport-related concussion, a combination of neck and vestibular physical therapy can decrease time to medical clearance to return to sport in people with persistent headaches, dizziness, and neck pain.⁴³ It is reasonable to consider that offering evidence-based treatment for neck-related symptoms may reduce the impact of persistent symptoms post concussion.

While our understanding of concussion continues to improve, the specific role of the neck in people with persistent symptoms post concussion is not well understood. How neck problems should be identified post concussion, the extent to which the neck contributes to symptoms, and whether treatment of the neck is effective remain unclear. Symptoms alone have limited value in distinguishing between neck and brain injury in people with concussion.^{21,27} Preliminary research suggests that the physical examination may be more likely to identify neck (particularly upper neck) problems when they are present after concussion,^{6,21} and highlights the need for further prospective study. The purpose of this study was to assess people with persistent symptoms following a concussion who were referred for neck assessment within a multidisciplinary concussion service, to (1) determine whether the neck had also been injured, and (2) evaluate the potential for the neck to contribute to their symptoms.

METHODS

THIS PROSPECTIVE CASE SERIES WAS conducted within a multidisciplinary concussion service. Ethical approval for this research was received from the University of Otago Human Ethics Committee (H16/089) and the Accident Compensation Corporation Ethics Committee (number 314). This study was prospectively registered with the Australian New Zealand Clinical Trials Registry (ACTRN12616001183471).

Clinical Setting

Participants were prospectively recruited by 2 providers of a multidisciplinary concussion service. This service is nationally funded and provided by local contract holders nationwide. It is designed to provide further assessment and care for people with persistent symptoms post concussion who are at risk of a prolonged recovery. The service accepts referrals from medical practitioners (most commonly general practitioners). The concussion service provides a multidisciplinary assessment to confirm the diagnosis, evaluate the source(s) of symptoms, consider any barriers to recovery, and develop an individual management plan. A “key worker” (either a physical therapist or occupational therapist) performs an initial assessment for new referrals, which is then reviewed, along with any other clinical notes available, by a multidisciplinary team including a medical doctor, neuropsychologist, occupational therapist, and physical therapist. The team then decides which assessments are necessary for each patient, and these are performed. Other specialists are consulted as appropriate. Information from these assessments is collated and overall recommendations for management are developed. As the concussion service providers were in a main city to which people from surrounding areas might travel to access care, the individual management plan could be completed where the person lived by local clinicians.

All patients using the concussion service received basic support from the key worker (an occupational therapist or physical therapist), including education about concussion, advice on how to manage a graduated return to daily activities, and case management.

Participants

This study included patients who received a neck assessment from 1 of 3 experienced physical therapists with postgraduate training in orthopaedic manual therapy. Where neck treatment was completed with a study physical therapist,

the treatment was tailored to the patient’s needs, and the initial measures were repeated at the final appointment, upon completion of neck treatment. Data were collected from the physical therapy assessment and from the concussion service providers.

Selection Criteria

Key workers in the concussion service screened consecutive patients for eligibility. The selection criteria were based on current clinical practice at both sites. Patients were eligible to participate if they had persistent (more than 10 days) headaches, dizziness and/or neck pain, a history suggesting that the neck might contribute to their symptoms, and attended a referral for neck assessment with a study physical therapist. Patients were excluded if they had contraindications to manual neck assessment (eg, fracture, inflammatory joint conditions, or infection), other significant neurological conditions, or were under 16 years of age. All participants provided written informed consent, and their rights were protected.

Recruitment was performed over a 2-month period at 2 clinic sites, with an initial target of 30 participants. As a descriptive study, the recruitment target reflected referral expectations.

Data Collection

Data were recorded at the initial physical therapy neck assessment, and again at the final appointment for neck treatment provided by the study physical therapists. Data collected included demographic data, the Rivermead Post Concussion Symptoms Questionnaire (RPQ), the Neck Disability Index, the Dizziness Handicap Inventory, patient-reported findings, and physical assessment using a standard assessment form (APPENDIX, available at www.jospt.org). The cause of concussion was recorded according to the International Statistical Classification of Diseases and Related Health Problems-10th Revision external-cause classification, grouped into falls (unin-

tentional), transport accidents, exposure to mechanical force (eg, struck by/against an object), and assault (interpersonal violence).

The RPQ measures the severity of a range of postconcussion symptoms,²² and is recommended as a core measure of traumatic brain injury-related symptoms.⁴⁹ Scores of 2 or greater indicate that the symptom has increased since the concussion injury. In addition, the RPQ-3 and RPQ-13 subscales are reported.¹⁰ The Neck Disability Index and Dizziness Handicap Inventory measure the impact of neck disability⁴⁸ and dizziness,¹⁶ respectively, on daily activities.

Subjective and physical examination findings were recorded on a standard assessment sheet developed in collaboration with the clinicians and reflecting their routine clinical practice (APPENDIX). Headache frequency (headache days in the last week), duration (hours), and severity (0-10 numeric scale) were recorded.¹⁷ Dizziness frequency was recorded on a 6-point scale^{20,40} (0, never; 1, less than once per month; 2, 1 to 4 episodes of dizziness per month; 3, 1 to 4 episodes of dizziness per week; 4, dizziness once daily; 5, dizziness more than once a day or constant), and dizziness duration on a similar scale (0, nil; 1, dizziness lasting up to 10 seconds; 2, dizziness lasting up to a minute; 3, dizziness lasting up to 5 minutes; 4, dizziness lasting up to 10 minutes; 5, dizziness lasting longer than 10 minutes or constant). Dizziness severity/intensity was measured using a 0-to-10 numeric scale.⁴⁰ Neck pain severity was recorded using the numeric pain-rating scale (calculated as the average of the current, best, and worst neck pain in the last 24 hours, on a 0-to-10 scale for each).

Physical measures included neck range of motion, measured using the CROM device (Performance Attainment Associates, Lindstrom, MN); the flexion-rotation test¹³; a segmental assessment of the neck, recording pain and stiffness from the occiput to C4⁵⁰; and tenderness of the paraspinal and

suboccipital muscles. Descriptive measures included whether headaches were provoked by movement, the analysis of the assessing physical therapist, and the amount of neck treatment recommended (recorded as hours and time span). The analysis of the assessing physical therapist included judging whether the neck was considered to be contributing to the patient's current symptoms from their most recent concussion injury. If the clinician determined the neck to be contributing to the patient's current symptoms, then neck treatment was recommended as appropriate. Treatment for pre-existing conditions is not permitted in this service. Neck treatment is only recommended when symptoms are considered to arise from the recent concussion injury.

Further details were retrieved from the multidisciplinary concussion service reports, including details of comorbidities and other treatment received. Where the multidisciplinary team recommended and included specific follow-up treatment (as distinct from the initial assessment) in the care plan, this was recorded as "other treatment received." Furthermore, the extent to which neck and vestibulo-ocular treatments were received concurrently was recorded as "overlap in neck and vestibulo-ocular treatment." This was due to neck and vestibulo-ocular assessment/treatment being completed by separate, specialized physical therapists. Last, adverse effects from treatment were monitored.

Data Analysis

In line with recommendations for case-series design,^{4,23} most results are presented individually as descriptive statistics, such as frequency (percentage) for categorical data or mean $\pm$ SD for continuous data. Median values have been presented where appropriate to ensure the data were represented accurately, and differences were noted. For the segmental findings, mean pain scores of 3 or less were excluded to better represent moderate-to-severe cases and exclude "normal" tenderness.

RESULTS

PARTICIPANT FLOW THROUGH THE concussion service and study is detailed in **FIGURE 1**. After screening, 20 individuals completed the initial neck assessment, and 11 completed neck treatment with the study physical therapists, including posttreatment reassessment. A majority of people attending the concussion service were referred for a neck assessment (29/39, 74%; including 2 who saw nonstudy physical therapists), and most of those who were eligible participated (20/26, 77%; excluding 1 considered later not to have a concussion).

Demographic characteristics of participants ($n = 20$) are presented in **TABLE 1**, and the symptom characteristics are presented in **TABLE 2**. Participants were evaluated at a mean of 7.5 weeks post concussion (median, 5 weeks). All participants reported headaches. The headaches were frequent (mean, 5.6 headache days in the past week), of variable duration (mean $\pm$ SD, 8.9 ± 10.4 hours), and of moderate severity (mean, 4.6/10). Fifteen participants reported dizziness. The dizziness was typically frequent (median, 5/5), of short duration (median, 2/5), and of moderate severity (mean, 4.7/10).

Physical examination and analysis findings are presented in **TABLE 3**, with further detail on segmental findings shown in **FIGURE 2**. Other physical examination findings commonly documented were lower cervical or cervicothoracic junction pain (4 patients) and tightness/tenderness in the trapezius or sternocleidomastoid (6 patients). Overall, 18 (90%) participants were considered to have a neck problem that was contributing to their current symptoms post concussion. For these participants, a mean of 3.4 ± 1.6 hours of neck treatment was recommended over 4.3 ± 1.8 weeks.

Treatment information, including comorbidities and overlap in treatment for participants completing neck treatment ($n = 11$), is reported in **TABLE 4**. The median time span of treatment was 3 weeks. While all patients were consid-

ered to have vestibular/functional balance comorbidities, treatment for this did not always overlap with neck treatment. There were 3 cases in whom no overlap occurred (cases 1, 2, and 6). These participants received vestibular treatment only after neck treatment was completed. Pre- and post-neck treatment comparisons for headache and dizziness variables (frequency, duration, and severity) and neck pain are presented in **FIGURES 3** through **5**.

This highlights that changes in one variable (eg, headache frequency) were not always reflected in others. No adverse effects from treatment were reported.

DISCUSSION

THIS PROSPECTIVE CASE SERIES DESCRIBES multiple findings indicative of concurrent neck injury in 18 of 20 participants with persistent symp-

toms post concussion. Participants were recruited from a multidisciplinary concussion service and represent those with persistent symptoms (mean, 7.5 weeks; median, 5 weeks post concussion at initial assessment), consistent with previous descriptions.^{7,14,45} The neck-related findings have the potential to contribute to participant symptoms and may respond to neck treatment.

Concussion and neck injuries can be concurrent. While previous studies have proposed^{9,28,32,42} or given limited support to the role of the neck,^{21,37,43,47} to date, there has been a lack of prospective data describing the nature of neck issues in those with persistent symptoms post concussion. In this study, physical therapists considered the neck to contribute to current symptoms in 90% of cases referred for assessment. Participants reported neck disability, neck pain, headaches provoked by neck movement/position, muscle tenderness, and cervical segmental pain and stiffness. The presence of multiple neck-related findings lends further support to the idea that the neck has the potential to contribute to symptoms following a concussion injury. This does not diminish the role of brain injury or other comorbidities (eg, vestibular or oculomotor dysfunction) in postconcussion symptoms, but highlights the potential for concurrent neck injury in those with persistent symptoms. In these cases, there is value in having a member of the multidisciplinary team with the skills to identify and manage neck-related symptoms.

Assessment findings help clarify the nature of neck problems that may present following a concussion. The clinician who works with people who have postconcussion symptoms and suspects neck involvement may focus on the upper cervical spine. Relevant findings in this series of participants include moderate-to-severe pain and stiffness on segmental assessment of the upper cervical spine, a positive flexion-rotation test, asymmetry of neck movement in rotation, and tenderness of the suboccipital muscles.

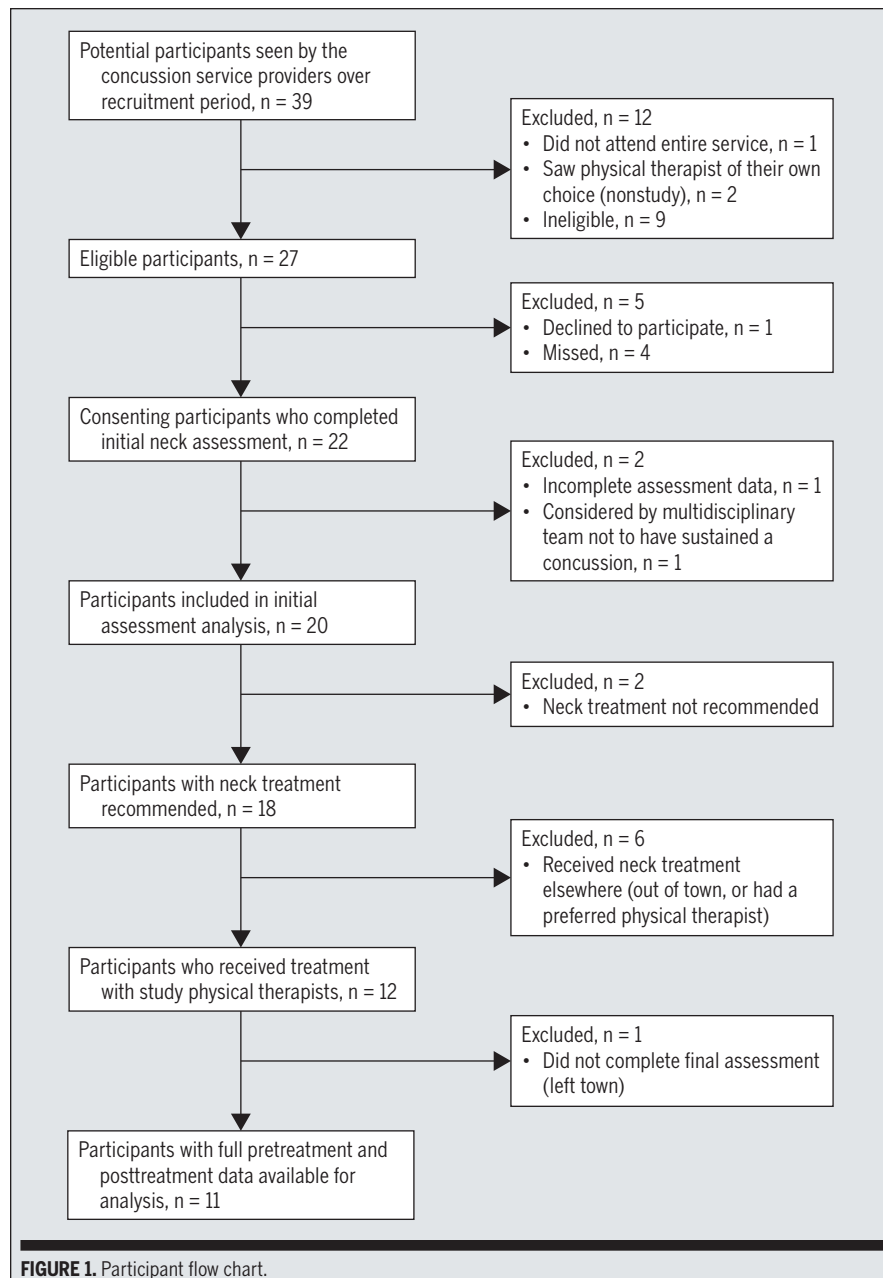


FIGURE 1. Participant flow chart.

These findings indicate frequent dysfunction at the C1-2 level, a particularly mobile segment involved in axial rotation.³ Given that headache, neck pain, and dizziness are the most common physical symptoms reported post concussion,¹⁹ findings consistent with upper cervical spine dysfunction are relevant. People with cervicogenic headache or dizziness and no history of concussion also report pain, stiffness, a positive flexion-rotation test, movement asymmetry, and suboccipital muscle tenderness.^{17,18,36,38} The

convergence of findings might suggest that a focus on neck range of movement, the flexion-rotation test, palpation, and segmental assessment could be appropriate to identify neck contributions to symptoms in people with persistent postconcussion symptoms.

The nature of the neck findings in this study is consistent with neck problems for which treatment is available. This is good news, as identifying a neck disorder opens a pathway for treatment in addition to other postconcussion care. The

clinicians identified 3 main categories of neck problems: (1) possible cervicogenic headache, (2) possible cervicogenic dizziness, and (3) neck injury. The broad term *neck injury* reflects issues identified with the neck not related to headaches or dizziness. While a working diagnosis of possible cervicogenic headache might be challenged,² the clinicians gathered appropriate, relevant subjective and physical information⁴¹ to make an informed decision. Specific findings, such as frequent pain at the C1-2 level and a positive

TABLE 1

DEMOGRAPHIC CHARACTERISTICS (N = 20)

Case	Age, y	Sex	Ethnicity	Employment Status	Time Since Injury, wk	Cause of Concussion	Injury Setting	Past History
1	19	Female	NZ European	School or tertiary study	4	Exposure to mechanical force	Sport	Unremarkable
2	26	Male	NZ European	Employed full-time	4	Exposure to mechanical force	Nonsport	Unremarkable
3	21	Male	Other European	Employed full-time	4	Exposure to mechanical force	Nonsport	Unremarkable
4	40	Female	NZ European	Employed full-time	4	Fall	Nonsport	Unremarkable
5	31	Female	Other European	Employed part-time	2	Exposure to mechanical force	Nonsport	Migraines; previous concussion 2 mo prior
6	46	Female	NZ European	Employed full-time	5	Fall	Nonsport	Unremarkable
7	66	Female	NZ European	Not employed or retired	42	Transport accident	Nonsport	Multiple previous concussions; previous neck injury 1 y prior
8	23	Male	NZ European	School or tertiary study	10	Assault	Nonsport	Unremarkable
9	44	Female	NZ European	Not employed or retired	2	Exposure to mechanical force	Nonsport	Unremarkable
10	62	Male	Other (Irish)	Employed part-time	3	Transport accident	Nonsport	Unremarkable
11	19	Female	NZ European	Employed part-time	5	Exposure to mechanical force	Nonsport	Migraines
12	17	Female	NZ European	School or tertiary study	13	Fall	Nonsport	Unremarkable
13	47	Female	NZ European	Employed part-time	6	Transport accident	Nonsport	Migraines
14	56	Male	NZ European	Employed full-time	5	Transport accident	Nonsport	Migraines as teenager; no neck injury history
15	21	Female	NZ European	School or tertiary study	17	Fall	Sport	Previous concussion 12 y prior; previous neck injury 5 y prior
16	20	Female	Maori	School or tertiary study	7	Fall	Sport	Unremarkable
17	64	Male	NZ European	Employed full-time	5	Exposure to mechanical force	Nonsport	Chronic neck pain; dizziness related to low blood pressure
18	38	Male	NZ European	Employed part-time	3	Transport accident	Sport	Chronic neck stiffness and pain
19	37	Female	NZ European	Employed full-time	5	Transport accident	Sport	Multiple previous concussions
20	45	Female	NZ European	Employed full-time	3	Transport accident	Nonsport	Unremarkable
Total	37 ± 16	65% female*	80% NZ European†	40% employed full-time‡	7.5 ± 8.9§	35% exposure to mechanical force or transport accident¶	75% nonsport†	

Abbreviation: NZ, New Zealand.

*35% male.

†15% other, 5% Maori.

‡25% employed part-time, 25% school or tertiary study, 10% not employed or retired.

§Median, 5 weeks.

¶25% fall, 5% assault.

†25% sport.

RESEARCH REPORT

flexion-rotation test, support the clinical reasoning.^{12,36}

A challenge in treating persistent postconcussion symptoms is the high potential for headaches to arise from multiple sources³⁰ (eg, brain, neck, oculomotor, etc). For a physical therapist trying to decide whether conservative neck treatment is worth exploring, a low-threshold clinical diagnosis of possible cervicogenic headache might be appropriate, particularly considering that manual therapy and exercise have good evidence for treating cervicogenic

headache.^{2,17} All participants had signs of concurrent vestibulo-ocular issues. Therefore, the presence or absence of cervicogenic dizziness was unclear. A neck injury was considered to affect approximately half of those assessed based on the history, questionnaires, and examination, and might be less controversial to identify.

Participants' symptoms changed from the initial to the posttreatment neck assessments. Given the observational study design, these improvements are certainly not all attributable to neck

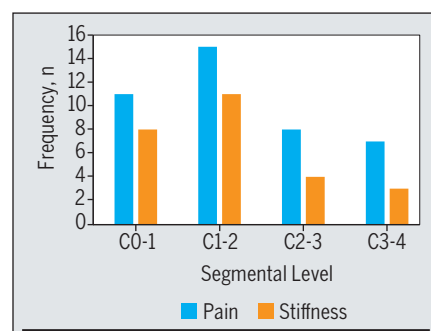


FIGURE 2. Frequency of segmental findings from the occiput to C4 (C0-C4). These data represent findings on the left or the right side (n = 20), and only include moderate-to-severe pain scores (4/10 or greater).

TABLE 2

SYMPTOM CHARACTERISTICS (N = 20)

	RPQ*			DHI				Headache				Dizziness				Neck Pain		
	Full	RPQ-3	NDI	Full	F	E	P	Yes/No	Fr, d [†]	D, h	S	Provoked [‡]	Yes/No	Fr	D	S	Yes/No	NPRS
Case	(0-64)	(0-12)	(0-100)	(0-100)	(0-36)	(0-36)	(0-28)	(side)			(0-10)			(0-5)	(0-5)	(0-10)		(0-10) [§]
1	25	5	...	...	...	...	...	Yes (B)	7	0.3	5	No	No				No	
2	35	10	26	60	26	18	16	Yes (U) [¶]	7	24	9	Unsure	Yes	5	2	6	Yes	0.7
3	36	5	30	62	24	22	16	Yes (B)	7	0.2	6	Yes	Yes	5	1	3	Yes	4.7
4	42	9	31	36	10	8	18	Yes (B)	7	2	3	Unsure	Yes	5	1	5	Yes	2.3
5	42	8	34	42	18	6	18	Yes (U) [¶]	7	0.5	4	Unsure	Yes	5	1	5	Yes	4.3
6	31	6	36	58	28	8	22	Yes (B)	4	2	6	Yes	Yes	5	1	4	Yes	3.7
7	42	10	42	60	22	22	16	Yes (B)	3	24	2	No	Yes	4	4	7	Yes	3.0
8	29	6	42	40	12	16	12	Yes (B)	7	2	4	Yes	Yes	4	1	4	Yes	2.7
9	26	3	40	26	8	6	12	Yes (U) [¶]	7	4	3	Unsure	Yes	3	2	4	Yes	6.0
10	34	4	28	56	28	16	12	Yes (B)	1	1	1	No	Yes	4	2	1	Yes	2.7
11	47	10	20	78	34	24	20	Yes (U) [¶]	7	24	4	Yes	Yes	5	3	8	Yes	4.7
12	38	8	...	30	10	8	12	Yes (U) [¶]	7	2.5	8	No	Yes	5	2	6	No	
13	27	6	...	44	18	10	16	Yes (U) [¶]	4	12	7	No	Yes	5	3	7	No	
14	28	5	36	...	...	...	...	Yes (B)	7	1	6	Yes	No				Yes	4.3
15	54	9	44	34	10	8	16	Yes (B)	7	24	3	Unsure	Yes	3	1	2	Yes	4.0
16	24	4	44	40	18	12	10	Yes (B)	3	2	5	Yes	Yes	5	1	2	Yes	3.3
17	25	5	20	...	...	...	...	Yes (B)	7	24	2	No	No				Yes	2.0
18	32	6	26	...	...	...	...	Yes (B)	7	2	3	Unsure	No				Yes	1.7
19	43	9	50	78	30	28	20	Yes (B)	4	2	8	Unsure	Yes	5	5	6	Yes	6.3
20	26	3	18	...	...	...	...	Yes (U) [¶]	2	24	3	Yes	No				Yes	2.7
Total	34.3 ± 8.5	6.6 ± 2.4	33.4 ± 9.5	49.6 ± 16.3	19.7 ± 8.4	14.1 ± 7.3	15.7 ± 3.5	100% yes	5.6 ± 2.1 [#]	8.9 ± 10.4	4.6 ± 2.2	35% yes	75% yes	5**	2**	4.7 ± 2.1	85% yes	3.5 ± 1.5

Abbreviations: B, bilateral; D, duration; DHI, Dizziness Handicap Inventory; E, emotion; F, function; Fr, frequency; NDI, Neck Disability Index; NPRS, numeric pain-rating scale; P, physical; RPQ, Rivermead Post Concussion Symptoms Questionnaire; S, severity; U, unilateral.

*As recommended by Eyres et al,¹⁰ a separate score for the RPQ-3 (headaches, dizziness, nausea) is provided (and the RPQ-13 can be calculated).

†Headache days in the past week.

‡By neck movement or positions.

§Calculated as the average of the current, best, and worst neck pain in the last 24 hours on a 0-to-10 scale for each.

¶Side shift.

¶No side shift.

*Median, 7 days.

**Values are median.

TABLE 3

PHYSICAL EXAMINATION AND ANALYSIS (N = 20)

Case	AROM*	F-R Test	Segmental Levels With MSP†	Suboccipital Tenderness	Paraspinal Tenderness	Headache Provoked by PE	Other Relevant Findings	Analysis‡	NCCCS‡	NTR‡
1	Right rotation	Positive	C2-C3, right	No	Right	No	Pain at CTJ	C2-C3 dysfunction; possible CH	Yes	Yes
2	No	Negative	C0-C2, bilateral	Right	Right	No	Unremarkable	Possible CH	Yes	Yes
3	No	Negative	C0-C4, left; C1-C4, right	Left	Left	No	C6-C7, left pain: 8/10	Lower cervical neck pain	Yes	Yes
4	No	Negative	C0-C2, right	No	No	Yes	Upper cervical flexion reproduced headache	Possible CH and dizziness	Yes	Yes
5	No	Positive	No (mild only)	Bilateral	Right	No	Tight upper trapezius	Neck injury	Yes	Yes
6	No	Negative	C0-C3, right	Right	Right	Yes	SCM tender, bilateral	Possible CH and dizziness	Yes	Yes
7	No	Positive	C1-C2, right	No	No	No	Unremarkable	Possible CH and dizziness. Chronic neck issue related to several concussions/whiplash	Yes	Yes
8	Right rotation	Negative	C1-C3, right	No	No	Yes	SCM tenderness, right more than left	Possible CH and dizziness. Neck injury	Yes	Yes
9	No	Positive	No (mild only)	Left	Left	No	Paraspinal/suboccipital muscle tightness	Neck injury: left neck pain and severe spasm	Yes	Yes
10	Left rotation, right side flexion	Negative	C3-C4, bilateral; C1-C3, right	No	No	No	C5-C6, left pain at CTJ	Lower cervical injury: left side	Yes	Yes
11	Right side flexion	Negative	C0-C4, right	Right	Right	No	Unremarkable	Neck injury: right upper cervical. Headache not reproduced	Yes	Yes
12	No	Negative	C0-C2 and C3-C4, bilateral; C2-C3, right	No	Right	No	Stiffness and pain at T2-T3: reproduced her day-to-day pain	Main problem: upper thoracic spine pain/stiffness	Yes§	Yes§
13	No	Positive	C0-C4, left	No	No	Yes	Right upper cervical side-bend tighter than left	Possible CH	Yes	Yes
14	Right rotation, right side flexion	Positive	C0-C2, right	Right	Right	Yes	Unremarkable	Possible CH	Yes	Yes
15	Left rotation	Positive	C0-C2, bilateral	Bilateral	Bilateral	No	Unremarkable	Neck injury	Yes	Yes
16	Right rotation	Negative	C1-C2, right	Right	No	No	Tender trapezius, bilateral	Possible CH and neck injury	Yes	Yes
17	No	Negative	No (mild only)	No	No	No	Upper cervical flexion and right sidebend tight	No acute neck injury or CH. Has chronic thoracic spine pain; will continue current treatment for this	No	No
18	No	Negative	C0-C1, bilateral	No	No	Yes	Reduced left rotation (C1-C2) and right upper cervical sidebend	Underlying chronic neck stiffness; no worse as a result of this injury	No	No
19	No	Positive	C1-C2, right	Right	No	No	Tender right trapezius. Fracture of right clavicle concurrent with concussion	Possible CH and dizziness. Upper cervical dysfunction at C1-C3	Yes	Yes
20	Left rotation	Positive	C0-C4, bilateral	Bilateral	No	No	SCM tender, bilateral	Possible CH and dizziness; C0-C2 dysfunction	Yes	Yes
Total	40% ≥10° loss	45% positive	85% MSP	55% yes	50% yes	30% yes			90% yes	90% yes

Abbreviations: AROM, active range of motion; CH, cervicogenic headache; CTJ, cervicothoracic junction; F-R, flexion-rotation; MSP, moderate-to-severe pain; NCCCS, neck considered contributing to current symptoms; NTR, neck treatment recommended; PE, physical examination; SCM, sternocleidomastoid.

*Loss of 10° or greater in rotation or side flexion.

†Pain rated as 4/10 or greater.

‡Represents the clinical opinion of the assessing physical therapist; NCCS refers to symptoms arising from the patient's most recent concussion injury.

§Although the upper thoracic spine was involved, this case was managed similarly to a neck injury, by the same provider and within the concussion service. To reflect this situation, the case has been recorded as NCCCS and NTR.

TABLE 4

TREATMENT INFORMATION, INCLUDING COMORBIDITIES AND OVERLAP IN TREATMENT (N = 11)*

	Value
Treatment type (more than one could apply), n	
Manual therapy	10
Soft tissue techniques	3
Specific exercises (eg, posture, control)	8
Number of sessions [†]	4.45 ± 1.1 (3-6)
Time span of treatment, wk [‡]	3.2 ± 2.3 (1-9)
Neck treatment considered beneficial (yes), n (%) [‡]	11 (100)
Comorbidities identified, n	
Vestibular/functional balance	11
Oculomotor	6
Mental health	1
Other musculoskeletal (eg, fracture)	1
Other treatment received, n	
Vestibulo-ocular rehabilitation	11
Psychology	0
Other musculoskeletal treatment	1
Occupational therapy	5
Overlap in neck and vestibulo-ocular treatment, n	
No overlap	3 (cases 1, 2, 6)
Single session	2 (cases 8, 10)
2 sessions	3 (cases 3, 4, 9)
3 or more sessions	3 (cases 5, 7, 11)

*Data were retrieved from the multidisciplinary concussion service summary.

[†]Values are mean ± SD (range).

[‡]As reported by the treating physical therapist.

treatment. Early support from the key worker might influence participant recovery and outcomes. Participants were initially assessed at a mean of 7.5 weeks (median, 5 weeks) post concussion, a stage where fast symptom resolution is no longer expected³⁵ and active rehabilitation is recommended.^{28,42} The time between the initial and posttreatment assessments was relatively short (mean, 3.2 weeks; 8 participants completed treatment within 3 weeks). That substantial symptom improvements are observable for multiple and chronic symptoms within a short period suggests that the overall model of care is appropriate. Improvements in neck pain in particular, along with headache and dizziness variables, lend some additional support to clinician impressions of a cervicogenic contribution to symptoms. However, the extent of neck involvement relative to other variables is unclear. Participants who did not achieve resolution of headache or dizziness reported changes in frequency, duration, or severity, which may be clinically relevant. These preliminary results suggest that neck treatment is worth exploring in further

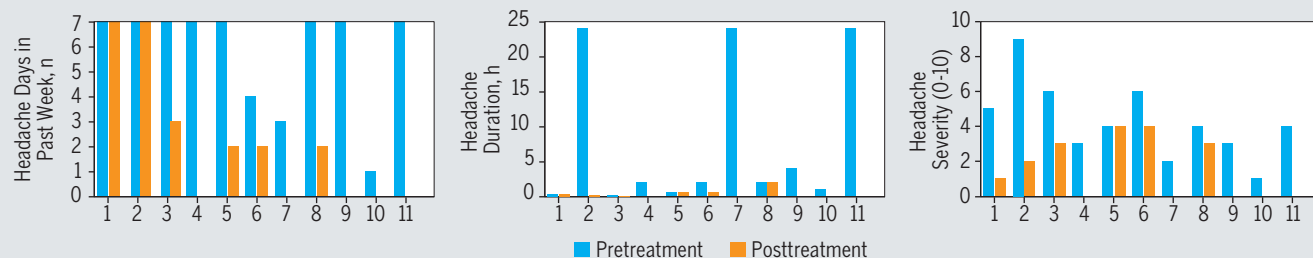


FIGURE 3. Individual headache data, pretreatment versus posttreatment (n = 11). Participants 1, 2, and 6 had no overlap with other treatment.

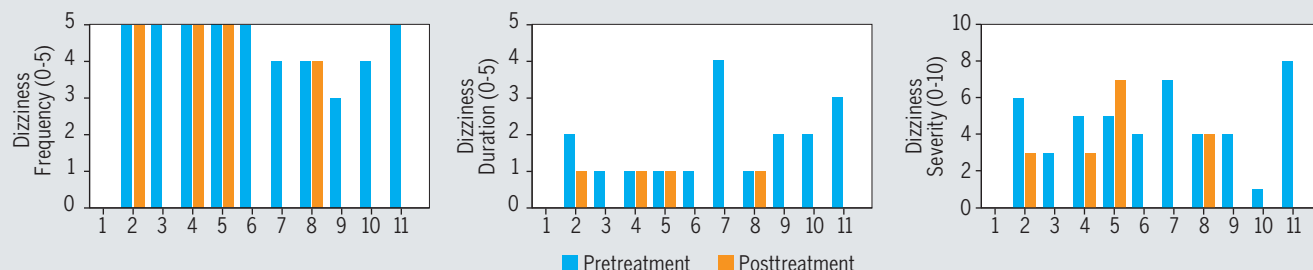


FIGURE 4. Individual dizziness data, pretreatment versus posttreatment (n = 11). Participants 1, 2, and 6 had no overlap with other treatment.

controlled studies. Persistent symptoms post concussion are complex, and assessment and treatment of the neck are relatively accessible and affordable.

Limitations

This study is an observational case series designed for descriptive reporting. The outcome data are exploratory and intended to stimulate ideas for future research and practice. Participants' health care was not controlled or modified and represents clinical rather than research conditions. Each participant had a history, presentation, and set of issues addressed by the concussion service's health professionals. This study reports variables routinely collected by the clinicians involved in the concussion service. Participants represent those suspected to have neck problems and referred for neck assessment, rather than all patients accessing the concussion service. Therefore, the generalizability of these results to all patients with postconcussion symptoms is uncertain. We are unable to draw conclusions regarding the extent to which neck problems contributed to postconcussion symptoms. Persistent symptoms post concussion are multifactorial, and this study focuses specifically on findings considered relevant to the neck. When considering the pre/post-neck treatment data, the contribution of the wider concussion service team—in particular, key worker support and specific vestibulo-ocular treatment—should be accounted for.

CONCLUSION

PAIN AND RESTRICTION IN THE UPPER cervical spine were similar to those described in cervicogenic headache and cervicogenic dizziness studies. Neck-related findings are important to recognize, as they have the potential to contribute to persistent symptoms post concussion and may respond to neck treatment. Along with a detailed history, a physical examination including cervical range of motion, the flexion-rotation test, palpation, and segmental examination may help clinicians identify neck-related problems. ●

KEY POINTS

FINDINGS: Pain and stiffness in the upper cervical spine were similar to those described in cervicogenic headache and cervicogenic dizziness research.

IMPLICATIONS: Neck-related findings are important to recognize, as they have the potential to contribute to persistent symptoms post concussion and may respond to neck treatment. Neck assessment may help clinicians evaluate potential sources of symptoms post concussion.

CAUTION: The case-series design is suitable for descriptive reporting. Further controlled studies are needed to clarify the effects of neck treatment on persistent symptoms post concussion.

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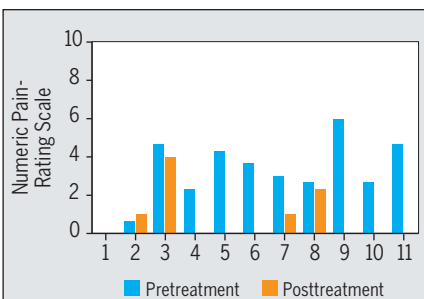


FIGURE 5. Individual neck pain data, pretreatment versus posttreatment (n = 11). Participants 1, 2, and 6 had no overlap with other treatment.

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APPENDIX

Standard concussion neck assessment - INITIAL

Name: _____

Date: _____

Physiotherapist: _____

Subjective

Brief current history: _____

Brief past history: _____

Main current problems: _____

Headaches: Yes / No Description: _____

Provoked by neck movements or positions? Yes / No / Don't know

Location: Unilateral without side shift Unilateral with side shift Bilateral

Notes: _____

Frequency: _____ headache days in the past week

Duration: _____ minutes / hours (*typically*)

Severity: _____ / 10 (*typically*)

Dizziness: Yes / No Description: _____

Provoked by neck movements or positions? Yes / No / Don't know

Frequency: _____ per day / week / month (*circle*) Constant

Duration: _____ seconds / minutes (*typically*)

Severity: _____ / 10 (*typically*)

Neck pain: Yes / No Description: _____

Constancy: Constant / intermittent

Severity: _____ / 10 current _____ / 10 best, last 24 h _____ / 10 worst, last 24 h

Other relevant issues (*describe*):

Physical examination

Range of movement	CROM measure (degrees)		Notes
Flexion			
Extension			
Retraction			
Protraction			
Side flexion	Left:	Right:	
Rotation	Left:	Right:	
Flexion-rotation test	Left:	Right:	Positive / negative (<i>circle</i>)

Note: The flexion-rotation test is positive when the visually estimated range is reduced by more than 10° from the anticipated normal range (44° ± 8°).¹²

APPENDIX

Standard concussion neck assessment - INITIAL

Name: _____ Date: _____

Physiotherapist: _____

VBI screen	Clinical judgment	Notes
Subjective	Positive / negative	
Physical examination	Positive / negative	

Ligament testing	Clinical judgment	Notes
Alar ligaments	Positive / negative	
Transverse ligament	Positive / negative	

Segmental exam	Left		Right	
	Pain (NPRS)	Restriction (7-point scale)	Pain (NPRS)	Restriction (7-point scale)
C0-C1				
C1-C2				
C2-C3				
C3-C4				

Restriction on 7-point scale: hypermobile (1, 2, 3), normal (4), hypomobile (5, 6, 7).

Neurological exam	Clinical judgment	Notes
Indicated?	Yes / no	
Upper motor signs	Positive / negative	
Sensation	Positive / negative	
Motor	Positive / negative	
Reflex	Positive / negative	

Muscle tenderness: Suboccipital Left / Right Paraspinal Left / Right

Other: _____

Other relevant findings (eg, lower cervical spine, thoracic spine)

Headache provoked during physical examination? Yes / No If so, due to _____

Dizziness provoked during physical examination? Yes / No If so, due to _____

Brief summary / analysis:

Overall, do you consider the neck as contributing to symptoms? Yes / No

If yes, evidence of (circle): Cervicogenic headache Cervicogenic dizziness Neck injury

Other: _____

Recommend further neck treatment? Yes / No

If yes, estimated treatment required is _____ hours over a period of _____ weeks

Comments: _____

Signature: _____

Abbreviations: NPRS, numeric pain-rating scale; VBI, vertebrobasilar insufficiency.

Considerations for Diagnosis and Management of Concussion

About 3 million patients each year seek medical attention for traumatic brain injury (TBI) in the United States, with the vast majority of cases classified as mild TBI or “concussions.”^{29,87} As half of injuries might go unrecognized or unreported, concussion continues to be a significant public health concern.^{102,119,144} Just as concussions are seen with increasing frequency in emergency settings,²⁰ surveys reflect that concussion is increasingly represented in the clinical practices of many health care professions.^{53,148,151,154} While most patients recover in a few weeks with minimal complications, some patients experience persistent posttraumatic complaints affecting daily activities.^{22,92,150} Nearly 14% of school-aged children and approximately one third of adult patients report symptoms and reduced function 3 months after a concussion.^{6,14,101} Many factors, such as age, sex, psychiatric history, and migraine history, as well as posttraumatic manifestations of migraine, psychiatric, vestibular, and oculomotor dysfunction, may influ-

ence recovery following a concussion and contribute to postconcussion syndrome.^{74,82,108,117,142,152} As a result, the evaluation process in concussion management is complex and multifaceted.

The challenges for differential diagnosis are the absence of biomarkers and paucity of clinical tests specific to concussion. As a result, tools developed to assess other conditions have been borrowed and adapted to evaluate many of the effects of concussion. For example, there are no concussion-specific headache or psychiatric assessments, despite the high prevalence of posttraumatic findings in these

areas. In addition, many common presentations, such as migraine, mood and emotional symptoms, exercise intolerance, and vestibular and oculomotor impairments, may be injury related but may also present independent of concussion.

This clinical commentary focuses on the evaluation and differential diagnosis of concussion to help guide patient assessment and management in the acute and postacute phases of recovery, including return to high-risk sport and life activities. As clinician-researchers who manage concussions of sport and nonsport etiology in a multidisciplinary setting, we intend this commentary to be applicable across populations and ages. We use the terms “mild TBI” and “concussion” interchangeably, as the distinction between the two is ill defined.

ACUTE ASSESSMENT (3 OR FEWER DAYS POST INJURY)

IN THE ACUTE PHASE, THE MOST IMPORTANT principles are (1) to protect the patient from further injury (primarily by removal from play or high-risk activity), and (2) to quickly evaluate the patient and rule out more serious injury, including cervical injury, skull fractures, and intracranial hemorrhage.¹⁰⁰ Several groups have validated clinical criteria to identify patients with mild TBI who are at low risk for serious injury and do not need to undergo head and cervical computed tomography.^{54,60,80,129,133,134} In addition, a blood test was recently approved by the

• **SYNOPSIS:** Concussion is an ongoing concern for health care providers. The incidence rates continue to be high and the rate of recovery is variable due to potential risk factors. With no valid biomarkers, diagnosis and assessment of concussion remain a clinical challenge. The heterogeneity in presentation following injury provides an additional level of complexity, requiring the screening and evaluation of diverse body systems, including oculomotor, vestibular, autonomic, psychiatric, cervical, and cognitive symptoms. While a few tools, such as the Vestibular/Ocular Motor Screening and Balance Error Scoring System, have been developed specifically for concussion, the vast majority of tests are adapted from other conditions. Further

complicating the process is the overlapping and interactive nature of the multiple domains of postconcussion presentation. This commentary illustrates how clinicians can conceptualize the multiple profiles that present following concussion and describes tools that are available to assist with screening and evaluation of each area. The multifaceted nature of concussion warrants broad clinical screening skills and an interdisciplinary approach to management. *J Orthop Sports Phys Ther* 2019;49(11):787-798. doi:10.2519/jospt.2019.8855

• **KEY WORDS:** assessment, concussion, diagnosis, heterogeneity

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US Food and Drug Administration to detect the presence of intracranial lesions.⁴⁹ Despite utility in identifying more serious injury, none of these measures is specific to concussion management.

Instruments for acute concussion assessment in sport and military settings continue to be revised and studied, including the Standardized Assessment of Concussion,⁹⁸ the multidimensional Sport Concussion Assessment Tool,^{99,100} and the Military Acute Concussion Evaluation.⁵⁰ While each of these measures has sensitivity and specificity for concussion diagnosis in the acute environment,^{7,13,96,98} their utility in detection of impairment is limited to the first few days after injury.^{25,26,31,38,95}

POSTACUTE ASSESSMENT (MORE THAN 3 DAYS POST INJURY)

THE PRIMARY GOALS OF POSTACUTE assessment are (1) to provide appropriate management of the concussion, and (2) to minimize the extent and duration of morbidity. This may include further medical assessments; recommendations for school, work, or sport participation; generalized activity prescription; and interventions including therapies and medication. Heterogeneity in clinical presentation following a concussion^{27,34,44} necessitates a multifaceted clinical assessment approach rather than a single measure.

We frame postacute assessment in the context of the following domains, where the clinician might expect to see signs and symptoms: ocular, vestibular, autonomic, mood/psychiatric, posttraumatic migraine, cervical, and cognition. Although these domains may overlap, the postacute assessment of concussion should consider each of these areas. Due to the variable nature of concussion, a multidimensional evaluation can form the basis for identifying primary, secondary, and tertiary clinical profiles presenting in each injury and provide a useful framework for guiding individualized management. For exam-

ple, patients with ocular profiles following a concussion may be prescribed vision exercises to facilitate recovery, whereas patients with posttraumatic migraine profiles may require pharmacological intervention. After reading this section, clinicians will become familiar with various tools and assessments that assist in the evaluation and management of patients with concussion in the postacute phase as they relate to the domains of ocular, vestibular, autonomic, psychiatric, posttraumatic migraine, cervical, and cognitive function.

Symptoms

Evaluation of concussion in all phases typically starts with a thorough inventory of symptoms. In the postacute phase, this subjective information can form the basis for identifying a patient's unique clinical profile when combined with objective measures and medical history. Symptoms can be highly variable and relate to a host of cognitive, physical, mood, and sleep issues. Symptoms such as dizziness and imbalance suggest a vestibular clinical profile, while reporting nervousness or sadness may point to a psychiatric presentation. Several self-report measures—including those developed specifically for athletes, children, and the general population—are available and may be

used by clinicians to assess postconcussion symptoms (**FIGURE 1**). Up to 28% of adolescents report concussion-like symptoms despite no history of a concussion.⁶⁸ Therefore, symptom assessments should not be stand-alone measures and should be considered in combination with other subjective and objective tests for greater accuracy.

Ocular Assessment

Impairment in ocular function, a frequent marker of concussion, is found in 42% to 55% of athletes.^{39,117} These deficits are strongly associated with cognitive and gait impairments^{67,117} and protracted recovery.^{39,42} Symptoms of ocular dysfunction may include blurred vision, double vision, eye fatigue, poor eye tracking, and frontal headaches associated with visual activity. Convergence deficits, accommodation dysfunction, saccadic and pursuit impairment, and disorders of ocular misalignment are common after a concussion.^{21,94} While full assessment of the oculomotor system involves a detailed examination by a vision specialist, screening tools can be helpful in identifying potential impairment in the ocular system (**FIGURE 2**). Most ocular assessments were not developed specifically for concussion and are best used in combination with other postconcussion

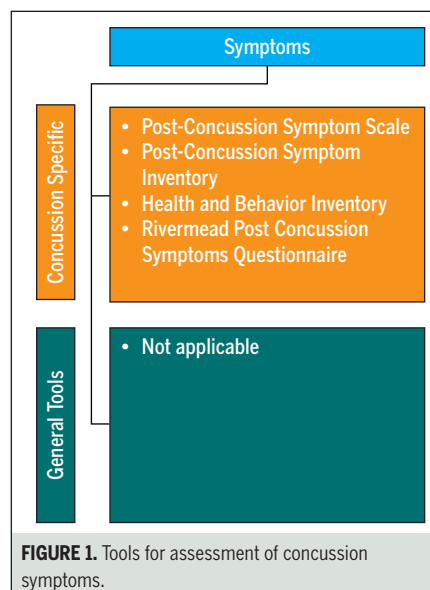


FIGURE 1. Tools for assessment of concussion symptoms.

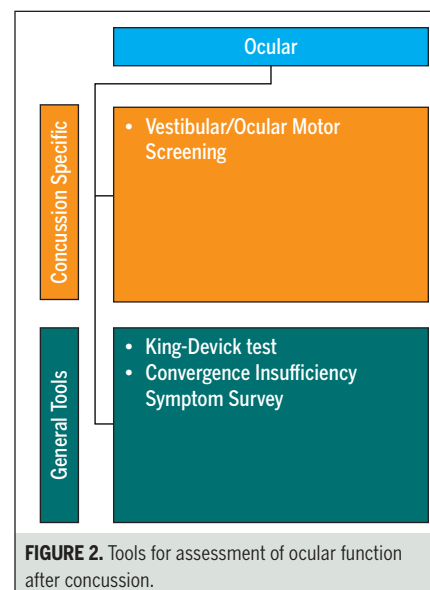


FIGURE 2. Tools for assessment of ocular function after concussion.

measures. When ocular findings present after a concussion, management often includes oculomotor exercises.¹⁶

The King-Devick test,¹¹⁵ developed to assess eye movement in children with reading difficulty, quantifies saccadic movements and has been proposed for both oculomotor assessment and acute diagnosis in patients with concussion. Due to variability in performance^{1,2} on the King-Devick test, patients require a baseline measurement for valid postinjury comparison. Additional challenges include high rates of abnormal baseline performance,^{105,106} high rates of false-positive findings after exercise (32%-36%),^{40,146} practice effects,¹¹⁴ and inadequate sensitivity (60%-62%) and specificity (39%-84%) in concussion identification.^{51,104,113} The benefits of the King-Devick test are its ease and short time to administer (less than 2 minutes).

The Vestibular/Ocular Motor Screening (VOMS)¹⁰⁹ was developed as a simple tool to identify potential oculomotor and vestibular dysfunction not assessed by other available instruments following sport-related concussion. Recognizing that oculomotor and vestibular impairments are common and often interdependent, the VOMS assesses near point of convergence and symptom provocation with the ocular functions of pursuit, saccades, and convergence, and the vestibular subtests of vestibular-ocular reflex (VOR) and visual motion sensitivity. Administration of the VOMS is standardized, with symptom scores totaling 2 or greater on individual VOMS items or a near point of convergence of greater than 5 cm considered significant for increased likelihood of concussion.¹⁰⁹ Studies have found the VOMS to have low false-positive rates (between 2% and 11%).^{76,146,149} The VOMS measures aspects of oculomotor and vestibular function other than those tested by the King-Devick test and the Balance Error Scoring System (BESS), with good reliability.^{107,109,146,149} Abnormalities on the VOMS may be associated with delayed recovery after sport-related concussion in youth and adolescents.³

Key Points

- Oculomotor signs and symptoms are distinct from and require different management than other postconcussive findings.
- Screening tools such as the King-Devick test and VOMS may help to identify common oculomotor deficits.
- A full oculomotor exam is indicated when screening tools identify deficits.

Vestibular Assessment

Approximately 60% of athletes have vestibular impairment and symptoms, such as dizziness, nausea, and imbalance, following sport-related concussion.¹⁰⁹ Vestibular symptoms may be an important early marker of a concussion, as prolonged recovery has been linked to the presence of dizziness as an acute symptom.^{30,82,126} Postconcussion vestibular impairment presents functionally as balance dysfunction, VOR impairment, visual motion sensitivity, and/or posttraumatic benign paroxysmal positional vertigo (BPPV).¹⁶ Because of these diverse presentations, screening for vestibular dysfunction should be multidimensional (FIGURE 3). Implementation of vestibular rehabilitation is recommended when deficits are identified in any of these areas.¹⁶

Balance deficits in sensory organization are frequently observed acutely and subacutely following concussion.⁵⁵ Therefore, sensory organization testing instruments have a role in the diagnosis of concussion, along with other on-field measures. The BESS¹²³ has been studied extensively in sport-related concussion as a measure of balance function.¹¹¹ Acutely, the BESS has a sensitivity of 34% and specificity of 91% to 96% in the differential diagnosis of concussion.²⁴ A modified BESS, which excludes the surface challenge (the condition of standing on a cushion in the original version), is included in the sideline Sport Concussion Assessment Tool, but research on the instrument is limited.¹⁸ The BESS appears to be best suited as an acute measure, as it is insensitive to

deficits after the third day of injury.¹¹¹ As deficits in maintaining balance under conditions of divided attention appear to persist into the postacute phase, balance testing utilizing dual cognitive task paradigms may be more sensitive for subacute assessment.^{23,47,116} However, at this time, no standardized methods exist for dual-task balance and gait assessment.

In addition to oculomotor items, the VOMS includes tests to identify vestibular constructs of VOR function and visual motion sensitivity in a standardized format. While nonconcussed individuals rarely have symptoms and impairments on the VOMS, the vestibular items on the VOMS are likely to provoke the most symptoms following a concussion (unpublished data).^{109,146} When abnormalities are identified with the basic screening and tests described above, more in-depth clinical evaluation of the vestibular system may be indicated. In particular, the Dix-Hallpike and supine roll tests should be performed when posttraumatic BPPV is suspected.¹⁰ Clinical or laboratory testing of VOR function, including but not limited to the dynamic visual acuity test, head impulse test, and head-shake nystagmus test, may be helpful for clinicians with expertise in applying these measures.^{58,59,90}

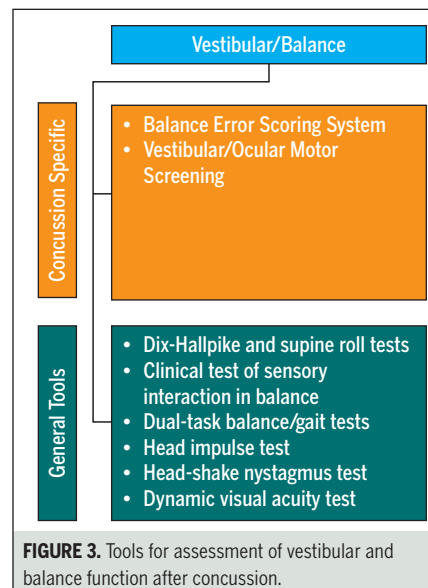


FIGURE 3. Tools for assessment of vestibular and balance function after concussion.

Key Points

- Vestibular symptoms and impairments are common and potentially contribute to prolonged recovery.
- Postconcussion vestibular impairments require different assessments and include BPPV, imbalance, VOR dysfunction, and visual motion sensitivity.
- The VOMS, BESS, and positional testing (Dix-Hallpike and supine roll tests) are helpful tools for assessing vestibular system impairment in patients with concussion.
- Vestibular rehabilitation targeted to deficits should be implemented when findings are present.

Autonomic Assessment

Persistent autonomic nervous system dysfunction is thought to be the underlying mechanism for the physiological postconcussion disorder subtype described by Ellis and colleagues.⁴³ Patients with this clinical profile following a concussion tend to have exercise intolerance and become symptomatic with cognitive activity. Regulating cerebral blood flow is one of the main roles of the autonomic nervous system thought to be implicated following concussion,¹¹⁸ and symptom provocation may be due to decreased efficiency of the brain in increasing blood supply to brain networks that are best suited to task demands.¹¹⁸ This type of autonomic nervous system dysfunction, cardiovascular autonomic dysfunction, has been suggested as a biomarker for concussion⁸¹ and investigated through measures of heart rate variability, blood pressure, and orthostatic intolerance.⁶² Despite small sample sizes and variability in methodology, recent research suggests that there is at least transient cardiovascular autonomic dysfunction that may resolve by 72 hours post injury,³⁷ and a recent meta-analysis concluded that concussion “likely” causes anomalies in autonomic nervous system functioning.¹¹⁸

Concussion symptoms associated with autonomic nervous system dysfunction may extend beyond exercise intoler-

ance and provocation of symptoms with cognitive activity. Several symptoms or features of other clinical profiles may be driven by or linked to autonomic nervous system dysfunction, including emotional distress, migraine, and vestibular disorders.^{11,19,110,118,128,145} Notably, autonomic nervous system dysfunction, especially orthostatic intolerance, occurs as a result of deconditioning,^{28,32} which is a common consequence of overresting and inactivity following concussion.

The most common tools or measures used to assess autonomic nervous system dysfunction following concussion are specific to exercise tolerance (FIGURE 4). The Buffalo Concussion Treadmill Test⁸⁴ is adapted from the Balke cardiac treadmill stress test⁴ and relies on measuring heart rate and blood pressure while gradually increasing workload until the first sign of symptom exacerbation. Details of the protocol are available elsewhere.⁸³ This approach has been presented as both an assessment and treatment modality following concussion, particularly for the physiological postconcussion subtype.^{43,83} Similar to treadmill paradigms, the McMaster All-Out Progressive Continuous Cycling Test is another exercise-tolerance test that has been used to assess symptoms and cardiorespiratory response to increased workload following concussion.³⁵

Other methods to assess autonomic nervous system function, including heart rate variability, the Valsalva maneuver, and head upright tilt table testing, are used infrequently in routine clinical care and are typically reserved for severe presentations.⁴⁶ While these assessment methods for autonomic nervous system function have been proposed, interpretation and management of findings are controversial. As autonomic dysfunction appears to manifest more subtly following concussion compared to primary dysautonomia, exercise-tolerance testing paradigms likely provide the best information for identifying impairment and guiding management. However, additional research is needed to determine

whether the existing protocols are optimal for assessing individuals with autonomic signs and symptoms following concussion.

Key Points

- Autonomic dysregulation can occur after a concussion and contribute to exercise intolerance.
- Exercise-tolerance testing is an important component of assessment following a concussion.
- Constructs of the optimal exercise-tolerance test are not known at this time.

Mood/Psychiatric Assessment

Following a concussion, emotional symptoms are common, with a quarter of patients displaying an anxiety/mood profile.⁷⁷ Mood changes may be challenging to assess due to complex etiology and overlap in symptoms with other clinical profiles. For example, dizziness and nausea are both prominent symptoms of both anxiety and vestibular clinical profiles following concussion. The relationship between profiles is further complicated by the overlap in physiology of vestibular function and emotional regulation.¹²⁰

Neurometabolic disruptions of the emotional circuitry have been identified in neuroimaging studies following concussion.⁴⁵ Neurometabolic alterations are similar in patients with concussion

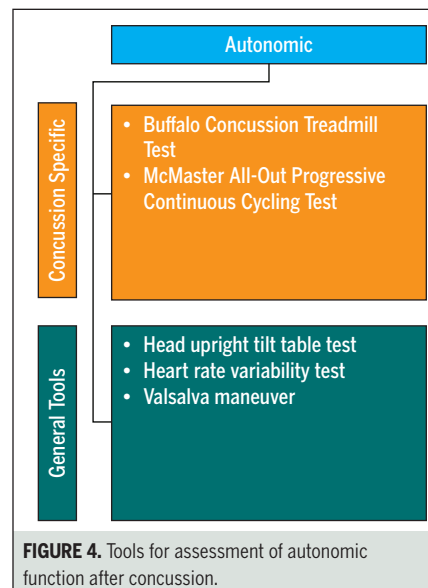


FIGURE 4. Tools for assessment of autonomic function after concussion.

and those with clinical depression on advanced neuroimaging, suggesting overlap in physiological substrate. It is not entirely clear when the pathophysiological changes that are linked to emotional symptomology fully resolve, or whether some neurochemical changes persist for weeks to months in some patients.

Concussion symptom inventories can assist the clinician in screening for mood-related impairment, as these tools ask patients to report the presence and intensity of affective symptoms such as sadness, emotionality, and nervousness. However, evaluating for mood changes and anxiety after a concussion requires more in-depth interview questions pertaining to current mood and stressors. There are many standardized clinical tools that can be borrowed from the psychology field to assess postconcussion emotional symptomology (FIGURE 5). These measures are typically validated and reliable self-report questionnaires to measure depression, anxiety, adjustment, and coping strategies, with comprehensive lists of these tools used in concussion patients summarized elsewhere.¹²⁴ However, no emotional or mood measures are specific to concussion, nor can they be used to diagnose concussion or differentiate etiology of symptoms. Rather, questionnaires for depression and anxiety can measure the

severity and type of distress, along with functional impact.

Key Points

- The presence of emotional symptoms can complicate recovery from a concussion.
- Concussion symptom inventories can assist in screening for psychiatric influences.
- Instruments borrowed from the psychiatric field may be helpful to further assess mood-related impairment.

Posttraumatic Migraine

Headache is the most common symptom following a concussion and occurs in approximately 70% of cases.^{63,64,75} Similar to concussion itself, posttraumatic headache is quite heterogeneous in presentation and may arise from many sources, including the cervical spine, medication overuse, and autonomic, psychiatric, tension, and migraine influences.⁷⁰ When examining features of posttraumatic headache, migraine has been identified as the most common posttraumatic headache type in both sport and nonsport populations, representing up to half of the cases.^{74,91,136}

Primary migraine is defined as episodic headache attacks of moderate to severe pain intensity, typically unilateral and of pulsating quality and lasting 4

to 72 hours if untreated. Migraines are aggravated by normal physical activity (eg, walking or climbing stairs) and frequently occur with nausea and/or vomiting or photophobia and phonophobia. Some migraines may involve aura, with transient sensorimotor symptoms, particularly visual changes.⁶¹ The pathophysiology of migraine involves abnormal activation of trigeminovascular pain pathways that results in headache and associated symptoms. While the mechanism behind posttraumatic migraine is not clearly understood, it does not appear to merely be a manifestation of a pre-existing migraine diagnosis, as rates of posttraumatic migraine far exceed rates of migraine in the general population.⁸⁶ Posttraumatic migraine is likely the result of interactive effects of concussion-induced cortical hyperexcitability and genetic predisposition.^{5,15}

Differentiating migraine from other headache types following concussion is important for at least 2 reasons: (1) posttraumatic migraine headache (as opposed to nonmigraine headache) is specifically associated with a worse clinical course, including more severe symptoms, increased neurocognitive deficits, and protracted recovery^{74,103}; and (2) migraine headache requires distinct management that may include preventative or abortive medication and identification and regulation of triggers. Additionally, a migraine variant—vestibular migraine—is a frequent cause of episodic dizziness that appears to have greater prevalence in younger patients.⁸⁵ Posttraumatic vestibular migraine is likely a common cause of dizziness following concussion.

At this time, posttraumatic migraine diagnosis is based on the International Headache Society clinical criteria for primary migraine.⁶¹ The Migraine Assessment Tool, based on international criteria, has been developed and validated to clinically diagnose migraine.⁹³ Other inventories, such as the Migraine Disability Assessment test¹³² and the Headache Impact Test,⁷⁹ may help to quantify pain and disability due to migraine (FIGURE 6).

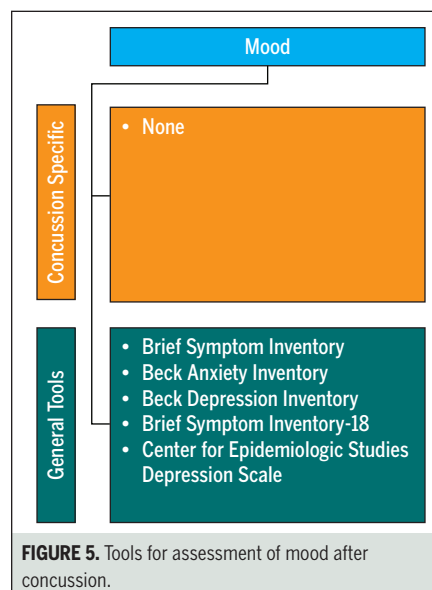


FIGURE 5. Tools for assessment of mood after concussion.

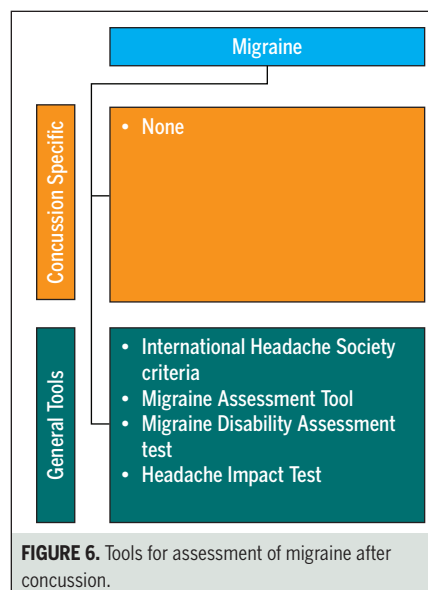


FIGURE 6. Tools for assessment of migraine after concussion.

Key Points

- Posttraumatic migraine is a distinct postconcussion clinical presentation that influences recovery time.
- Migraine must be managed differently from other headache types.
- The diagnosis of posttraumatic migraine is currently based on international classification criteria for primary migraine.

Cervical Considerations

Many of the symptoms of concussion and whiplash-associated disorder are similar. Neck pain, headaches, dizziness, impaired balance, impaired oculomotor control, sleep disruption, fatigue, hypersensitivity, cognitive dysfunction, and affective symptoms have all been documented independently in both concussion and whiplash-associated disorder. Therefore, in cases of trauma where these symptoms are present, it is important to determine whether concussion, whiplash-associated disorder, or both are contributing to clinical presentation in order to provide evidence-based treatment. Although several tools are in use, none of them have sufficient sensitivity and specificity to differentiate the cervicogenic source of many symptoms (FIGURE 7). An important clinical differentiation may simply be whether symptomatic relief (eg, reduced headache

or dizziness) can be achieved with treatment of the cervical spine in a reasonable amount of time.

Whiplash may result in a constellation of symptoms, likely due to the participation of cervical afferents in higher-order processes such as postural control, eye movements, and gaze stabilization.⁵⁶ However, as with many issues related to concussions, these symptoms may have a noncervicogenic etiology as well. In the differential exam, consider cervical instability, postural deficits, active motion, segmental motion, soft tissue, motor control, or sensory/reflex changes that are consistent with cervical dysfunction and/or pathology.¹²¹

Cervicogenic Headache As discussed earlier, headache is common following trauma, with various etiologies including cervicogenic, medication overuse, migraine, and other causes. Cervicogenic headache, by definition, begins within 7 days of whiplash injury and may or may not be associated with neck pain.⁶¹ Importantly, cervical pain is neither necessary for the diagnosis of nor specific to cervicogenic headache. Cervical pain is a known symptom of migraine, due to activation of the upper cervical dorsal root afferents relaying to the trigeminocervical complex within the trigeminovascular pain system.⁹ However, cervicogenic headache is often associated with other findings, such as postural deficits, range-of-motion limitation, and upper cervical dysfunction.^{57,69,153,155} Sensorimotor findings, such as tissue hypersensitivity^{131,135,141,153} and balance disturbance,¹⁴⁰ may also occur with cervicogenic headache; however, these issues may characterize other conditions, such as migraine, vestibular disorders, and anxiety.

The prevalence of cervicogenic headache is unclear. In a large prospective study, posttraumatic cervicogenic headache was diagnosed in only 4% of those with posttraumatic headache, while migraine, probable migraine, and tension-type headaches were identified at much higher rates.⁹¹ It's important that clinicians recognize the overlap in symptoms

when managing posttraumatic headaches and apply cervical intervention in appropriate cases, while referring other headache types for medical management.

Cervicogenic Dizziness Dizziness is another common postconcussive symptom with potential cervicogenic etiology. Much like headache, posttraumatic dizziness can be attributed to multiple causes, such as peripheral and central vestibular dysfunction, autonomic dysfunction, vestibular migraine, or anxiety.⁵² However, cervicogenic dizziness should be considered when dizziness is episodic, has a close temporal relationship to neck pain, and is brought on by specific neck movements or positions rather than whole-body movements.¹³⁹ Cervicogenic dizziness has been associated with other findings, including altered postural control, abnormal oculomotor function, and impaired kinesthetic awareness of neck position.^{89,139}

The best tests to differentiate cervicogenic influences on sensorimotor control likely involve movement of the body with a stable head in order to activate the cervical spine while eliminating vestibular influences. In the cervical torsion test (CTT),⁸⁹ nystagmus is recorded with the head and body in neutral and with the head still and body rotated to the right and left. A study⁸⁹ examining patients with BPPV and cervicogenic dizziness reported that nystagmus of greater than 2°/s in any position is considered a positive test for cervicogenic dizziness. Conversely, symptoms or nystagmus with en bloc motions point to a noncervicogenic cause of symptoms. The CTT requires nystagmography, which may not be available to many clinicians.

Similar to the CTT, variations of cervical relocation testing have been proposed to provide additional differentiation capability.⁶⁵ These paradigms hold promise but require additional empirical evidence before they are suitable for clinical use. Head relocation tests designed to identify cervical proprioceptive deficit have found increased impairment in patients with cervical pathology.^{36,89,122} However,

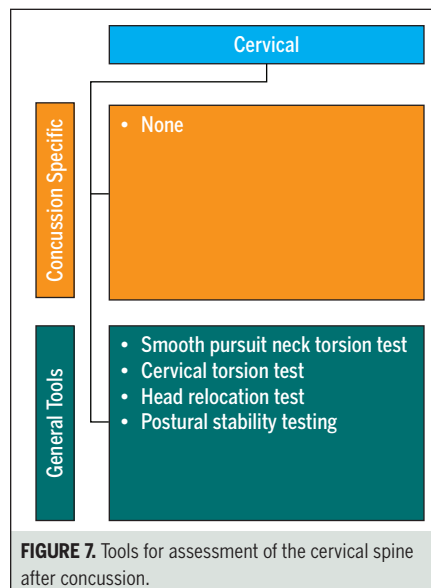


FIGURE 7. Tools for assessment of the cervical spine after concussion.

increased deficits in joint position testing have also been found in patients with vestibular disorders, making this a nonspecific measure.⁶⁵ The smooth pursuit neck torsion test¹³⁸ was developed to assess cervical output on oculomotor function. Early studies were encouraging, finding differences in the gain of eye movement response with the head rotated¹³⁸; however, more recent research has not reproduced these findings.⁷¹ Postural stability is reduced in patients with whiplash.^{12,112} Balance sway measures may only be useful in ruling out cervicogenic influences when measures are normal.⁵⁶

Key Points

- Cervical impairments frequently overlap with concussion.
- Headaches and dizziness are symptoms that commonly result from cervical dysfunction as well as concussion.
- Treatment of the cervical spine is recommended when cervicogenic etiology is supported.

Cognitive Assessment

Cognitive deficits following concussion have been documented for many decades, and neuropsychological tests were some of the first tools utilized for concussion assessment and management.⁸ It is well established that many adolescent patients with sport-related concussion experience

transient cognitive decline within the first days to weeks post injury,^{33,73,97,125} with some studies suggesting that deficits can persist for a few months following concussion in a small subset of patients.^{88,147} Although deficits are often mild⁷² and some patients do not notice overt changes in mental capacity in day-to-day functioning, they exhibit impairments in attention, processing speed, and memory processes when evaluated on objective neurocognitive testing^{48,127,143} (FIGURE 8).

Neurocognitive testing is a helpful diagnostic tool for concussion,⁷⁸ and can be particularly useful in situations where patients are not forthcoming about their symptoms (eg, athletes trying to hide injury to avoid removal from competition) and have no other objective deficits to accurately diagnose injury.¹⁷ Cognitive deficits following concussion potentially have multiple pathophysiological underpinnings, and severity is influenced by a variety of personal and injury factors. As with each postconcussion domain, a bidirectional relationship exists between the cognitive and nearly every other domain, including sleep, ocular, vestibular, mood, and migraine.

Key Points

- Cognitive deficits are a consistently demonstrated marker of concussion.
- Clinical assessment of concussion is aided by neurocognitive testing.
- Preinjury (baseline) cognitive testing is helpful, but not necessary, in the evaluation of concussion.

ESTABLISHING RECOVERY FROM CONCUSSION

MUCH LIKE ESTABLISHING A CONCUSSION diagnosis, determining concussion recovery poses a unique challenge for health care providers. There are no recognized biomarkers or objective measures of recovery from concussion. Often, “clinical” recovery does not align with physiological recovery—abnormalities in the electrical responses, metabolic balance, and oxygen consumption of neurons persist several months af-

ter clinical testing is normal and patients are symptom free.^{41,130} At present, a comprehensive, multidomain evaluation is the gold standard for determining recovery status, where establishing that the patient has returned to normal preinjury function is the goal. Provocative examinations of neurocognitive function, exercise tolerance, and vestibular-oculomotor function are important to perform, as symptoms and performance are typically normal in the resting state. When possible, comparing findings to baseline test results may help the clinician identify postinjury neurocognitive deficits.⁶⁶ Baseline testing for other domains of functioning, such as vestibular-oculomotor screening, has been initiated as a standard of care for collegiate athletes.⁷⁶

Return to Sport

Return-to-sport criteria include the requirement that the person be symptom free at rest and with physical activity.¹⁰⁰ Normal cognitive function is also a criterion mandated by a number of organizations and school districts,¹³⁷ and neurocognitive testing is often considered a “cornerstone” for appropriate management with return to sport.¹⁰⁰ These criteria loosely represent recovery from the diverse concussion impairments or profiles elaborated throughout this article (eg, neurocognitive testing determines normal cognitive function and exertion testing rules out persistent dysautonomia). No return-to-sport protocols include criteria for normal vestibular or oculomotor function. However, providers frequently require these domains of functioning to normalize before allowing their patients to return to sport, due to the elevated risk of reinjury during sport participation with persistent deficits (eg, poor balance or gaze stability) and the consequences of chronic impairments from repetitive trauma to these brain systems.

SUMMARY

CONCUSSION IS CHALLENGING TO evaluate and manage due to the nonspecific nature of symptoms

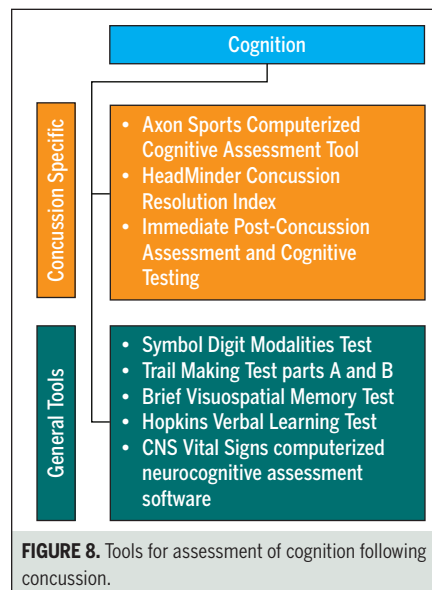


FIGURE 8. Tools for assessment of cognition following concussion.

and impairments characteristic of injury, coupled with lack of valid biomarkers to diagnose concussion and determine recovery. The acute diagnosis, postacute evaluation and management, and determination of recovery from concussion rely predominantly on clinical assessment. No single instrument is sufficient to assess concussion in isolation due to the significant heterogeneity in presentation, and research supports comprehensive, multidomain assessment approaches to manage concussion. Significant overlap in symptoms and impairments (eg, oculomotor problems following both concussion and whiplash-associated disorder) and interaction between profiles (eg, vestibular symptoms due to migraine) can occur post concussion. Although most clinical tools have been successfully borrowed and adapted to patients with concussion, a majority of measures are not exclusive to concussion and require additional research and refinement for this subpopulation. It is important for clinicians to understand the strengths and limitations of the tools and instruments used to diagnose and manage concussion, and for researchers to contribute empirically to this body of knowledge. ●

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Concussion in Combination With Whiplash-Associated Disorder May Be Missed in Primary Care: Key Recommendations for Assessment and Management

Whiplash and its associated disorders (WAD)⁷³ are the most common injury arising from a motor vehicle collision (MVC), with an annual Western-world incidence of between 200 and 300 per 100 000 population.^{36,58,68} Rehabilitation costs for persistent WAD exceed those associated with other common, and not radiologically occult, catastrophic injuries such as spinal cord injury.¹⁶ One in 2 people with WAD will develop long-term neck-related disability, and up to 1 in 4 will report widespread

bodily pain.^{38,70} The cardinal features of WAD remain neck pain and lack of mobility. Other signs/symptoms of the clinical course include motor weakness,⁵⁴ sensory hypersensitivity,⁷¹ hypoesthesia,¹⁴ arousal,^{21,87,88} and cognitive deficits.⁶⁵ The signs and symptoms of WAD are strikingly similar to those reported in known concussion cases. However, assessment and management of WAD and concussion are often considered separately.

The global incidence of concussion, often described as a “mild” traumatic brain injury (mTBI), is expected to rise, considering the increase in motor vehicle use, particularly in developing countries.^{44,45} In the United States, of the 1.4 million cases of mTBI reported annually, 45% are suggested to have resulted from an MVC.²⁵ A more recent study suggested that concussions occur in about 1 of 61 occupants in noncatastrophic crashes, with an increased risk occurring in rollover crashes.⁸⁶

Similar Symptoms Arise After WAD and Concussion

The most common and shared symptoms, some of which may persist for up

• **SYNOPSIS:** Whiplash and concussion may have similar presenting symptoms, biomechanical mechanisms, and neurophysiological sequelae, but neither enjoys a gold standard diagnostic test. Guidelines for whiplash and concussion are developed and implemented separately. This disparate process may contribute to misdiagnosis, delay appropriate primary care management, and impair patient outcomes. In our clinical commentary, we present 3 cases where signs and symptoms consistent with whiplash were identified in primary care. Symptoms in all cases included neck pain, headache, dizziness, and concentration deficits, raising suspicion of coexisting postconcussion syndrome. All cases were referred for specialist physical therapy. Characteristics consistent with poor recovery in both whiplash and postconcussion syndrome were confirmed, and multidisciplinary management, drawing from both whiplash and concussion guidelines, was implemented. All patients reported improvement in activities of daily

living after tailored management addressing both neck and head injury-related factors, suggesting that these conditions were not mutually exclusive. Self-reported outcomes included reductions in neck disability and postconcussion symptoms of between 20% and 40%. It may be appropriate for whiplash and concussion guidelines to be amalgamated, enhanced, and mutually recognized on a patient-by-patient basis. Primary health care professionals might consider minimum screening to identify postconcussion syndrome in patients following motor vehicle collision by administering questionnaires and assessing cranial nerve function, balance, and cognition. Management should then incorporate principles from both whiplash and concussion guidelines and harmonize with available imaging guidelines for suspected spine and head trauma. *J Orthop Sports Phys Ther* 2019;49(11):819-828. doi:10.2519/jospt.2019.8946

• **KEY WORDS:** mild traumatic brain injury, neck trauma, screening

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to 5 years,⁷⁹ across WAD and mTBI are neck pain, headache, dizziness, fatigue, and cognitive impairment. After neck pain, the most common symptoms experienced by people with WAD (70%-80% of people) are headache,^{5,35} dizziness,^{67,83} blurred vision,⁸² fatigue,³⁵ and cognitive impairment.³⁹ The most common symptoms after mTBI are pain, headache,⁹ and cognitive deficits, including impaired information processing and reduced attention.^{34,43,50} While cognitive impairment following whiplash and mTBI appears to be similar, some aspects, including long-term memory, reasoning, and accuracy in problem solving, were worse in patients with whiplash compared with mTBI.¹⁰ Therefore, in the presence of these symptoms, a diagnosis of either WAD or mTBI could be present and contribute to the clinical course.

Imaging Cannot Differentiate WAD and Concussion

There are no gold standard diagnostic tests capable of differentiating WAD and mTBI.^{13,44} For both conditions, controversy persists regarding the relationship between imaging findings of structural pathology and diagnosis, prognosis, and outcomes. For example, magnetic resonance imaging (MRI) scans of the brain in healthy people with no history of concussion have revealed “incidental findings” that are benign (eg, sinus problems, enlarged glands, and cysts of no medical significance).⁸⁵ Furthermore, specific patient populations (eg, athletes or military personnel) may have pre-existing differences in brain structure, function, and connectivity as a result of previous repetitive, subconcussive impacts and history of concussion.⁵¹

Routine use of early diagnostic imaging tests following whiplash or other common spinal conditions is challenged for multiple reasons, including abnormal or variant morphology of the cervical⁵² and lumbar spines of asymptomatic participants (false positives)¹² and lack of imaging findings in some patients following whiplash.^{1,3,26,29,48,53,84} Some clini-

cians may be skeptical and question the merit of pathoanatomical findings and outcomes.

One guideline that may assist primary health care clinicians to make appropriate imaging decisions is the American College of Radiology Appropriateness Criteria (ACR-AC).^{2,72} The ACR-AC are a set of consensus-developed, evidence-derived guidelines to assist in decision making for imaging based on the potential condition requiring investigation. Most applicable within the ACR-AC for patients with WAD are the clinical condition categories of “suspected spine trauma”¹⁹ and “head trauma.”⁷¹ Further imaging may be considered appropriate when, for example, concerns of significant relevant pathology may not be consistent with a favorable outcome.⁶⁴

One characteristic that has traditionally assisted the clinician to distinguish whiplash from postconcussion syndrome (PCS) is either loss of consciousness or posttraumatic amnesia. Traumatic brain injury is usually classified as mild, moderate, or severe, based on the initial Glasgow Coma Scale score (part of the criteria for appropriate imaging referral in head trauma) recorded in the emergency room, the duration of loss of consciousness, and the duration of posttraumatic amnesia.⁴³ However, not all patients can be assessed using these criteria. For example, some patients may have briefly lost consciousness after the MVC but regained it before being assessed. Some patients do not present to the emergency room.¹³ Drugs or alcohol may confound results.⁴⁵ Some patients may have other injuries that require priority assessment and management.^{9,20}

Guidelines for WAD and Concussion Are Poorly Implemented in Primary Care

Guidelines for the assessment and management of patients with mTBI presenting to the emergency room exist.²⁰ However, different hospitals and emergency care providers adopt different guidelines.^{28,81} Irrespective of diagnosis and management in the emergency

room, health care professionals working in primary care often do not have access to information, including initial loss of consciousness or posttraumatic amnesia. So, the diagnosis is likely to be made according to how patients present in primary care.

The suggestion that whiplash and concussion are similar is not innovative or new. There are similarities in the acute and chronic clinical profiles and even results of somatosensory-evoked-potential testing following whiplash and mTBI.^{1,11,30,32,37,91} Nineteenth-century clinical reports of whiplash, or “railway spine,” implicated brain injury as a possible explanation for the long-lasting symptoms reported by those afflicted.²⁴ Risk factors for persistent symptoms following whiplash and mTBI are similar (older age, female sex, and acute symptom report).⁵⁹ Contemporary work provides new insights into biomechanical links between WAD and concussion and supports the assessment of the brain and the musculoskeletal system following MVC to inform management and, hopefully, expedite recovery.²³

Despite similarities in symptoms, neurophysiology, and biomechanical forces, guidelines for managing whiplash and guidelines for managing concussion have been separately developed and implemented. Separate guidelines have been developed for managing concussion in sport (eg, the Sport Concussion Assessment Tool Fifth Edition)²² and concussion due to other mechanisms.³¹ Different diagnostic criteria are employed in these guidelines, both within and between conditions.

Recommendations for Management in Primary Care

Guidelines for WAD include specific suggestions for management. Guidelines for patients with mTBI suggest, after significant trauma has been ruled out, providing verbal and written information on discharge and that a brief, routine follow-up appointment may be required.⁸¹ Recently, in one Australian state (New

South Wales), legislation was passed⁵⁷ that restricts compensation—and therefore rehabilitation—for people with minor injury (including whiplash) to a period of 6 months. For people with WAD who may also have comorbid concussion, the implications are that they may not have access to care after 6 months.

Here, we present 3 cases of WAD and apparent comorbid PCS. We (1) discuss the implications for assessment and management, and (2) make recommendations for how clinicians should approach routine concussion screening for people presenting with whiplash in primary care.

CASE DESCRIPTIONS

Case 1

A 45-YEAR-OLD WOMAN (Ms M) WAS involved in a low-speed, rear-end MVC 4 months prior. She could not recall whether she lost consciousness and reported no posttraumatic amnesia. Presenting symptoms included neck pain, headache, dizziness, nausea, blurred vision, and memory and concentration difficulties (FIGURE 1). When Ms M presented to the emergency department after the collision, X-rays excluded

cervical fracture and further work-up excluded serious pathology. Management in primary care included medication (tricyclic antidepressant), with no change to symptoms, and physical therapy (manual therapy), which aggravated most symptoms.

Ms M had been referred to a neurosurgeon, who advised that surgery was not indicated, and to an optometrist, who reported that her visual acuity was 6/6. Despite MRI not being warranted according to the ACR-AC, Ms M's general practitioner referred her for brain MRI. The report concluded, "multiple small left frontal and smaller right parietal subcortical white matter intensity—nonspecific finding." Her cervical spine MRI was essentially normal, and she reported being reassured by these findings. However, given the lack of response to treatment, the insurer referred her to a specialist physical therapist, who performed further examination. In Australia, specialist physical therapists must complete an additional 2-year fellowship in the Australian College of Physiotherapists and are recognized for their expertise in the management of complex presentations. The findings of

the specialist physical therapist examination were as follows:

- Physical examination: cervical active range of extension reproduced dizziness (TABLE 1)
 - Oculomotor examination: abnormal, with vertical saccadic eye movement during the smooth pursuit examination and poor near-point convergence observed
 - Upper-limb neurological examination: normal
 - Vestibular system examination: normal
 - Sensorimotor system and palpation of the cervical spine: symptomatic
- Self-reported questionnaires (TABLE 2) indicated high disability due to neck pain and dizziness, high levels of postconcussion symptoms, and low function.

Case 2

A 24-year-old woman (Ms B) was involved in a simple rear-end MVC 6 weeks prior, with no imaging performed in primary care. Ms B recalled hitting her head on the steering wheel and had a bruised cheekbone, but did not recall losing consciousness. She attended her general practitioner, who referred her to a sports and exercise physician the day after the collision. Ms B was diagnosed with WAD and PCS and referred for physical therapy. Presenting symptoms included severe headache, nausea, dizziness, loss of balance, photophobia, phonophobia, bilateral upper-limb paresthesia, and neck and arm pain (FIGURE 2). Given these symptoms, and following the ACR-AC, appropriate referral was made for MRI of the cervical spine and brain, and findings were reported as normal. Relevant past medical history included Ehlers-Danlos syndrome (a group of connective tissue disorders, although the patient's specific subtype was unknown) and Raynaud's disease. The physical therapist findings were as follows:

- Physical examination: marked global restriction in cervical active range of motion, with noticeable muscle guarding/spasm

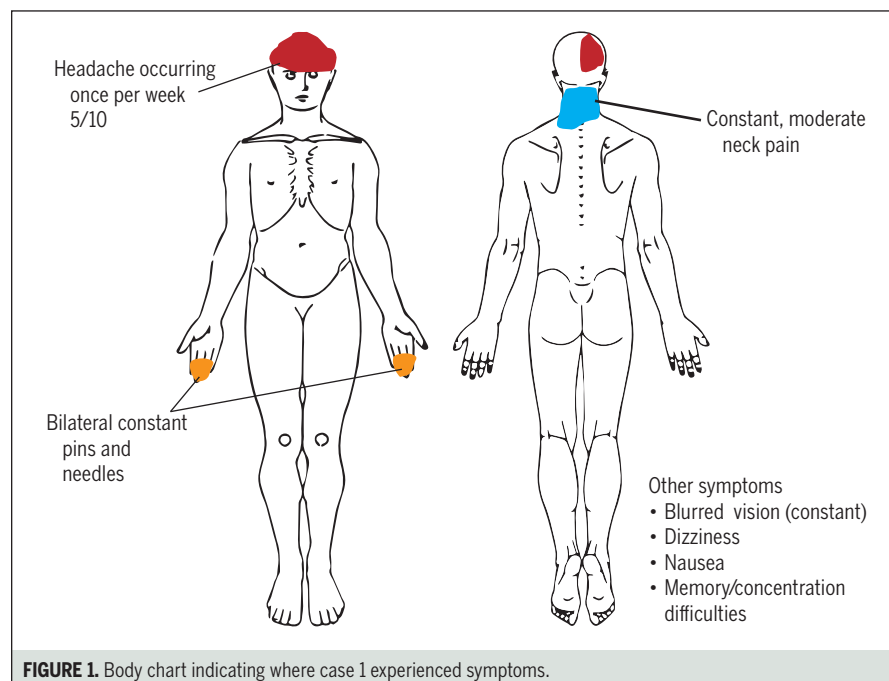


FIGURE 1. Body chart indicating where case 1 experienced symptoms.

CLINICAL COMMENTARY

- Cranial nerve (CN) examination: vertical saccadic eye movements during the smooth pursuit component of the oculomotor nerve (CN III) examination and reduced facial muscle coordination during facial nerve (CN VII) examination; normal upper motor

neuron/upper-limb neurological examination, including normal responses to both the Babinski and Hoffmann signs

- Sensory examination: evidence of temporal summation to repeated pinprick,⁶⁶ allodynia, cold hyperalgesia,

and pressure hyperalgesia; therefore, as expected and consistent with the mechanism of injury, tenderness to palpation of the cervical spine; a positive finding (increased mobility and symptom reproduction) on an alar ligament stress test

The positive finding on the alar ligament stress test, together with the history of Ehlers-Danlos syndrome, may be significant to physical therapists, as together they suggest joint hypermobility with or without craniovertebral instability.⁵⁶ Self-reported questionnaires indicated high self-reported disability due to neck pain, a high score on the Impact of Event Scale-revised,⁹⁰ and high severity of PCS (TABLE 2).

Case 3

A 44-year-old man (Mr S) was involved in a high-speed, dangerous-mechanism, head-on MVC on the motorway 12 weeks prior. The estimated speed of impact was greater than 100 km/h. Mr S believed he lost consciousness for a short time and recalled waking up in the wreckage of the vehicle. He was trapped in the vehicle until paramedics could cut him free. He was taken by helicopter to the hospital.

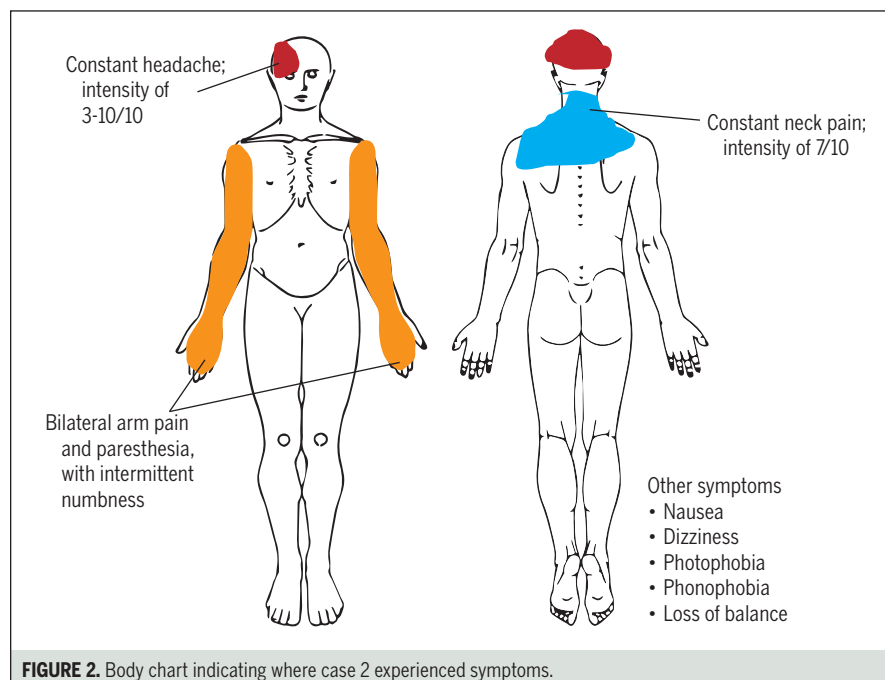


FIGURE 2. Body chart indicating where case 2 experienced symptoms.

TABLE 1

PHYSICAL EXAMINATION FINDINGS OF THE 3 CASES

System/Impairment Examined	Case 1	Case 2	Case 3
Cervical ROM	Reduced cervical extension (20°; increasing headache and dizziness)	Reduced cervical AROM in all directions (<10°)	Reduced cervical AROM in all directions (<10°)
Neurological			
Cranial nerves	Abnormal oculomotor examination*	Abnormal oculomotor examination* and facial nerve (VII) examination (poor coordination)	Abnormal oculomotor examination* and hypoglossal (XII) nerve examination (poor coordination)
Upper-limb/upper motor neuron lesion	Normal, including normal responses to Babinski and Hoffmann tests†	Normal, including normal responses to Babinski and Hoffmann tests†	Clonus-elicited bilateral hyperreflexia
Pain sensitivity	Normal PPTs, pinprick hyperalgesia (left and right upper limbs), normal CPTs	Reduced PPTs globally, temporal summation, reduced CPTs globally	Temporal summation, widespread allodynia
Vestibular/balance	Negative HIT, 5 s in tandem stance, failed by stumble (maximum score of 10)	2 s in tandem balance; HIT not performed due to reduced cervical AROM	Unable to perform tandem balance; HIT not performed due to reduced cervical AROM
Sensorimotor	Positive SPNTT, JPE >6° (extension and rotation)	Not tested initially	Not tested initially
Palpation	PAIVM at C1-C2 reproduced dizziness and headache	Generalized tenderness, positive alar ligament stress test	Not tested initially

Abbreviations: AROM, active range of motion; CPT, cold pain threshold; HIT, head impulse test; JPE, joint position error; PAIVM, passive accessory intervertebral movement; PPT, pressure pain threshold; ROM, range of motion; SPNTT, smooth pursuit neck torsion test.

*Observable vertical saccade during SPNTT examination and reduced near-point convergence.

†Babinski and Hoffmann tests performed to screen/exclude upper motor neuron lesion.

A cervical spine computed tomography scan excluded fracture, further work-up excluded serious pathology, and Mr S was discharged home after 2 nights in the hospital. Initial follow-up was with his general practitioner, who then referred him for physical therapy. Treatment to date had included exercise and manual therapy, which aggravated symptoms and produced “muscle spasms.” Mr S was then referred for specialist physical therapy. Presenting symptoms included constant back and neck pain (FIGURE 3), dizziness, poor balance, and fatigue. Investigations included cervical spine MRI, which was reported as normal. While the acute computed tomography scan was warranted, the subsequent MRI scan, based on presenting symptoms, was considered inappropriate according to the ACR-AC guidelines (chronic neck pain variant 1).² The specialist physical therapist findings were as follows:

- Physical examination: global restriction of cervical active range of motion, with all directions measuring less than 10°;

- noticeable cervical muscle guarding
- Neurological examination: abnormal response to the CN III examination,

with reduced eye coordination; difficulty controlling the coordination of his tongue during assessment of the

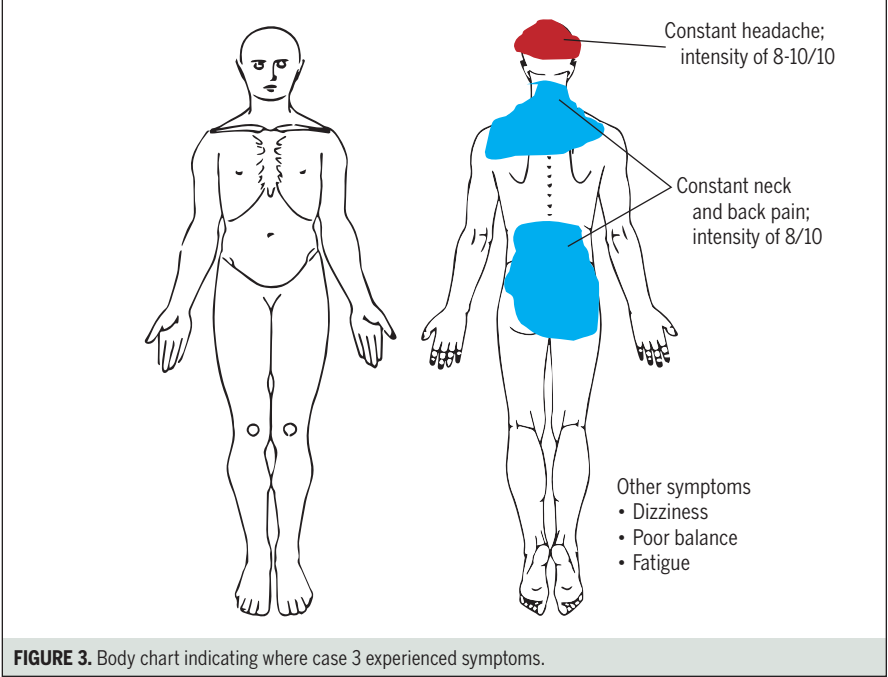


TABLE 2		SELF-REPORTED QUESTIONNAIRE RESULTS FOR ALL 3 CASES					
	Case 1		Case 2		Case 3		
Construct/Measure	Baseline	12-mo Follow-up	Baseline	12-mo Follow-up	Baseline	12-mo Follow-up	
Pain/disability							
NDI	33/50	18/50	34/50	10/50	48/50	28/50	
Dizziness (DHI)	75/100						
Central sensitization (CSI)					53/100	28/100	
Whiplash clinical prediction rule	High risk		High risk		High risk		
Psychological							
IES-R	22/75	13/75	49/75	19/75			
PTSDS					21/51	12/51	
Concussion symptoms (RPCSQ)							
3 items	8/12	4/12	11/12	3/12	5/12	3/12	
13 items	45/52	28/52	39/52	19/52	27/52	17/52	
Function (PSFS)							
Work	0/10	4/10	0/10	2/10	0/10*	8/10	
Reading	5/10 [†]	8/10	1/10*	6/10			
Driving	2/10 [†]	6/10	1/10*	4/10			
Abbreviations: CSI, Central Sensitization Inventory; DHI, Dizziness Handicap Inventory; IES-R, Impact of Event Scale-revised; NDI, Neck Disability Index; PSFS, Patient-Specific Functional Scale; PTSDS, Posttraumatic Stress Diagnostic Scale; RPCSQ, Rivermead Post Concussion Symptoms Questionnaire.							
*Activity limited by dizziness and nausea and cognitive impairment.							
[†] Activity limited by dizziness and blurred vision and cognitive impairment.							

hypoglossal nerve (CN XII); following clinical assessment of the spinal cord, responses consistent with hyperarousal (eg, brisk bilateral upper- and lower-limb reflexes, but a decreasing Babinski sign); a repeated beat on clonus testing

- Sensory examination: widespread allodynia over the neck and back; temporal summation with repeated pinprick over the forearm, inducing bilateral spasms of the upper and lower limbs

Self-reported questionnaires also revealed high scores for self-reported disability, central sensitization, and post-traumatic stress symptoms (TABLE 2).

OUTCOMES

Management and Interpretation of Cases

ALL 3 CASES PRESENTED WITH SYMPTOMS that could be classified as either WAD (grade II)⁷³ or PCS. In the initial primary care phase, the patients were diagnosed with WAD and managed accordingly. The apparent PCS was initially missed. Delayed diagnosis of sport-related concussion is associated with delayed recovery.⁴ A similar scenario could occur in the context of concussion after MVC.

In all 3 cases, once referred for a second opinion, the apparent PCS was identified. Patient management was then multidisciplinary, including medical, physical, and psychological therapy. Patients were referred for appropriate pain management and concurrently referred to a psychologist. In one case, a neuropsychologist directed the cognitive rehabilitation. The approach to the physical management incorporated principles from both WAD and PCS guidelines.

For patients with WAD, in the presence of altered pain processing, standard management (such as manual therapy) is often contraindicated. Rather, patient management is advised by experts to be directed at identified impairments (which are often sensorimotor or neuromuscu-

lar) and to avoid pain provocation with the targeted exercises prescribed.^{60,62,75}

Concussion guidelines recommend that the approach to recovery of function follow specific principles, based on reaching target heart rates and increasing by specific increments weekly. Outcomes included significant reduction in symptoms and return to driving and work in all 3 cases (TABLE 2).

DISCUSSION

THESE CASES AND THE SUPPORTING literature suggest that clinical guidelines for symptoms arising after MVC are needed for primary health care practitioners to screen patients with WAD for PCS. If screening could occur during the history and physical examination, it would allow for appropriate management and further assessment (eg, a specific type of imaging).

Implications for Patient History Taking

Recommendations from mTBI/concussion guidelines could be implemented in whiplash guidelines to screen patients with WAD for PCS when taking a history in primary care. While whiplash guidelines recommend assessment of pain, disability, and psychological distress,^{69,74} those for cognitive impairment and PCS do not. Given that cognitive deficits occur at a similar frequency and to a similar extent in both whiplash and PCS,^{10,39} and are associated with high pain and reduced quality of life,¹⁸ assessment of cognitive impairment could be recommended. While there are tools that assess cognitive impairment, constructs such as attention and immediate and delayed recall are easily assessed by questionnaire.^{10,39} For example, recently, the Perceived Deficits Questionnaire was validated and recommended for this purpose in whiplash.⁸⁰

Assessment of PCS is also not routinely recommended in people with whiplash. By the time patients present to primary care, acute assessment tools such as the Sport Concussion Assessment Tool Fifth Edition²² or the Abbreviated Westmead

Post Traumatic Amnesia Scale, as recommended in acute or emergency department assessment,⁵⁵ may not be relevant. However, postconcussion symptom questionnaires appropriate for the subacute stage, such as the Rivermead Post Concussion Symptoms Questionnaire,⁴⁰ are recommended.⁵⁵ The cases presented here and elsewhere³⁵ have used this tool.

Implications for the Physical Examination

Minimal concussion screening for people with WAD during the physical examination may be warranted. Recommended domains in clinical guidelines for WAD include assessing for physical impairments, such as active range of motion, muscle function, and sensorimotor impairment, if indicated.⁷⁴ An upper-limb neurological examination is recommended to screen for the potential presence of radiculopathy; however, clinical examination of brain function is not recommended.

When people with WAD have experienced loss of consciousness, posttraumatic amnesia, or have symptoms of PCS (eg, a high score on the Rivermead Post Concussion Symptoms Questionnaire⁴⁰), we recommend that primary health care practitioners conduct minimal screening of brain function. Signs and symptoms should guide the tests chosen. At a minimum, conduct a CN examination. A stronger focus on testing balance and coordination may be required.^{27,47} In all 3 cases presented in this paper, CN III and balance testing was abnormal and enabled detection of potential PCS. These tests take only a few seconds to administer, and would not be burdensome to implement in primary care.

Although uncommon, there have been previous case reports of abnormal facial (CN VII)⁴⁹ and hypoglossal (CN XII)¹⁵ function after head and neck trauma. We hypothesize that this may be due to head trauma (concussion) rather than neck trauma (whiplash). Given that the hypoglossal nerve traverses the anterolateral cervical spine, it is plausible that neck trauma may result in hypoglossal

nerve injury. Given that abnormal facial and hypoglossal nerve function was not a common finding in these case studies or in the literature, less emphasis should be placed on these tests for routine screening.

Because the vestibular system is implicated in concussion,⁴¹ routine vestibular and oculomotor screening is commonly recommended. However, in whiplash, painful restriction of cervical range of motion may limit valid testing of the vestibular system (eg, the head impulse test). Instead, the clinician might choose the oculomotor assessment as the differential test. The vertical saccade observed during smooth pursuit examination is more suggestive of concussion than vestibular involvement. Therefore, modifications to the recommended vestibular and oculomotor screening may need to be made in the presence of whiplash.

Implications for Imaging

In the course of routine care of the patient with suspected head/neck injury, primary care practitioners recommend diagnostic tests on a patient-by-patient basis. The ACR-AC guidelines, and other available guidelines informed by the same body of evidence and by the same clinical criteria,^{77,78} are available worldwide.

While the ACR-AC provide guidance as to who should be referred for imaging, and what kind, they do not inform clinicians about how they should or could correlate nonemergent imaging findings with the subsequent clinical course. Pre-injury neck pain, older age, high baseline pain intensity, higher self-reports of disability, and female sex are associated with poor recovery from whiplash^{64,89} and mTBI.⁵⁹ However, it is unclear whether indeterminate imaging findings, beyond frank pathology, may be associated with the subsequent clinical course of whiplash and mTBI/concussion.

In all 3 cases, imaging was ordered, but not always in line with current guideline recommendations. Among the proposed benefits of adhering to the ACR-AC are cost savings, reductions in

exposure to ionizing radiation, avoiding the identification of pathology that may simply represent normal variants, and more informed clinical decision making. While there was no radiological evidence of more serious pathology (eg, spinal cord injury or moderate-to-severe brain injury) in any of the cases, conventional MRI findings should not be necessarily dismissed as “normal,” given the low likelihood of conventional imaging techniques to detect brain lesions following mTBI.⁴²

In the future, findings from advanced imaging, combined with other known risk factors for poor recovery, may help identify key factors influencing the clinical course. Until that time, clinicians are encouraged to follow the imaging guidelines, refer patients for appropriate imaging, embrace emerging technologies, and recognize the opportunity to examine our own clinical instincts when managing patients with more complex, and seemingly inexplicable, signs and symptoms of whiplash or PCS.

Implications for Management

Diagnosis or presence of concussion symptoms in people who had initially been managed for whiplash directed care and promoted recovery. All cases were managed initially as if they were “whiplash,” with minimal awareness that concussion might be present.

Due to the lack of a gold standard diagnostic test for both WAD and concussion, current guidelines for both^{46,74} recommend management according to the presenting symptoms and signs. In the case of WAD, guidelines recommend advice to remain active, analgesia, and exercise aimed at the impairment.⁷⁴ Similarly, concussion guidelines recommend managing the presenting symptoms (such as headache and dizziness) as well as providing advice on graded physical activity.^{33,46} One of the key differences, however, is that concussion guidelines recommend cognitive restructuring as part of rehabilitation,^{33,46} whereas WAD guidelines do not. In the 3 cases presented, the reason for limitations in activity participation and failure

to return to work was primarily due to dizziness and cognitive impairments. Addressing cognitive impairments was essential for recovery.

Despite whiplash and concussion sharing common pathophysiological and pathomechanical processes, outcomes are often evaluated separately. We might be ready to improve characterization of whiplash and concussion through superior understanding of the pathogenesis of these 2 non-mutually exclusive conditions. Therefore, our call to clinicians is to recognize the highly comorbid conditions: try not to evaluate outcomes separately. Future guidelines should aim to reconcile that a proportion of people with whiplash may have concussion, and vice versa.

One barrier for primary health care practitioners to implementing guideline recommendations is the copious information they receive. Compared to common conditions such as diabetes and hypertension, WAD and concussion present infrequently in primary care.¹⁷ Recommendations from clinical guidelines for conditions such as WAD and concussion are implemented poorly.^{6,7} An alternative health service delivery model gaining favor for busy primary health care professionals is to perform routine minimal-risk screening for nonrecovery or poor prognosis (regardless of diagnosis) and refer patients early to specialized care.^{29,61}

Comprehensive assessment (eg, neurological, psychological distress, pain sensitivity, physical impairment, and vestibular screening) is required to direct care. Because not every person with WAD will present with these impairments, it can be inefficient for primary health care professionals to routinely assess all domains. Those at risk of poor prognosis early after injury more commonly have complex impairments.⁷⁶ In the 3 cases presented, a validated risk-stratification tool for whiplash⁶³ determined that all 3 cases were at “high risk” of nonrecovery. In this situation, clinical guidelines recommend referral to a specialist clinician, who then undertakes a more comprehensive assessment to determine further

care, in consultation with the primary health care professional.⁷⁴ The service delivery model assists primary health care professionals by determining who can be managed well in primary care and who may need referral. This model of care is feasible⁶ and can improve care in patients with WAD⁸ and low back pain.²⁹

Implications for Compensation and Cost

Whiplash-associated disorder and concussion are frequently managed in compensable settings. However, eligibility for compensation differs. In New South Wales, Australia, compensation law changed in 2017 to restrict compensation entitlements for people with WAD to a 6-month time frame.⁵⁷ The intent of this compensation change was to benefit “wellness,” not “sickness.” However, the casualties may be cases of WAD where undiagnosed concussion coexists. Our 3 cases did not recover within the 6-month time frame, and were ineligible for compensation after this period—a substantial financial burden. All 3 patients eventually returned to work, but required longer to do so. Without the financial support of their partners and families, it is likely that this outcome would not have been possible.

SUMMARY

WHIPLASH AND PCS SHARE SYMPTOMS and pathophysiology. However, they are often considered separately in clinical practice. Whiplash and PCS may often coexist, and primary health care professionals should consider minimal screening so that PCS is not missed. Likewise, for researchers and guideline developers, the call is to consider where whiplash and PCS guidelines may interact. Novel risk-based health service delivery pathways may provide a mechanism to translate recommendations into clinical practice. ●

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Adapting the Dynamic, Recursive Model of Sport Injury to Concussion: An Individualized Approach to Concussion Prevention, Detection, Assessment, and Treatment

Sport-related concussion is among the most frequently reported injuries in sport and recreation.⁸⁰ A sport-related concussion is “a traumatic brain injury induced by biomechanical forces.”⁸⁴ Symptoms and signs that occur following a concussion are believed to represent a functional rather than structural injury, as structural neuroimaging studies do not detect abnormalities.⁸⁴ Recovery can occur in the initial days to weeks for most adults, but up to one third of children and youth may take longer than 4 weeks to recover.^{108,127}

Each individual can present with a unique set of symptoms and aggravating or relieving factors following concussion.⁸⁴ Individualized assessments are

imperative to best understand the etiology of symptoms.^{37,78,102,106} A multifaceted, interdisciplinary approach to assessment and management is vital.

The purpose of this clinical commentary was to summarize prevention, detection, assessment, and treatment



factors that affect individuals across the continuum of concussion care, using the dynamic, recursive model of sport injury⁸⁸ framework. In part 1 of this commentary, we address etiology, risk factors, and detection of concussion. In part 2, we address concussion assessment and management.

An Introduction to the Dynamic, Recursive Model of Sport-Related Concussion Etiology

Individual athletes have their own risk factors that predispose them to concussion (FIGURE 1). These factors may change over time. During sport, athletes are exposed to different events in which no concussion or injury occurs. In these cases, the athlete continues to play, and ongoing adaptation and changes in the set of risk factors to which the athlete is exposed may occur, resulting in higher or lower concussion risk. In youth athletes, growth and development may result in changes in performance and adaptations.

● **SYNOPSIS:** The risk factors of concussion may be categorized as intrinsic (internal factors specific to the individual) or extrinsic (external factors related to the environment or sport). Identifying these factors is part of an individualized, patient-centered approach to prevention, assessment, and management of concussion. In most cases, the symptoms of concussion resolve in the initial few days following the injury, and a strategy involving a gradual return to sport and school is recommended. When symptoms persist for longer than 7 to 10 days, a multifaceted interdisciplinary assessment

to guide treatment is recommended. This article applies the dynamic, recursive model of sport injury to sport-related concussion and summarizes the process of individualized assessment and management following concussion in athletes of all ages, with a focus on physical rehabilitation. *J Orthop Sports Phys Ther* 2019;49(11):799-810. doi:10.2519/jospt.2019.8926

● **KEY WORDS:** assessment, clinical care, concussion, prevention, rehabilitation

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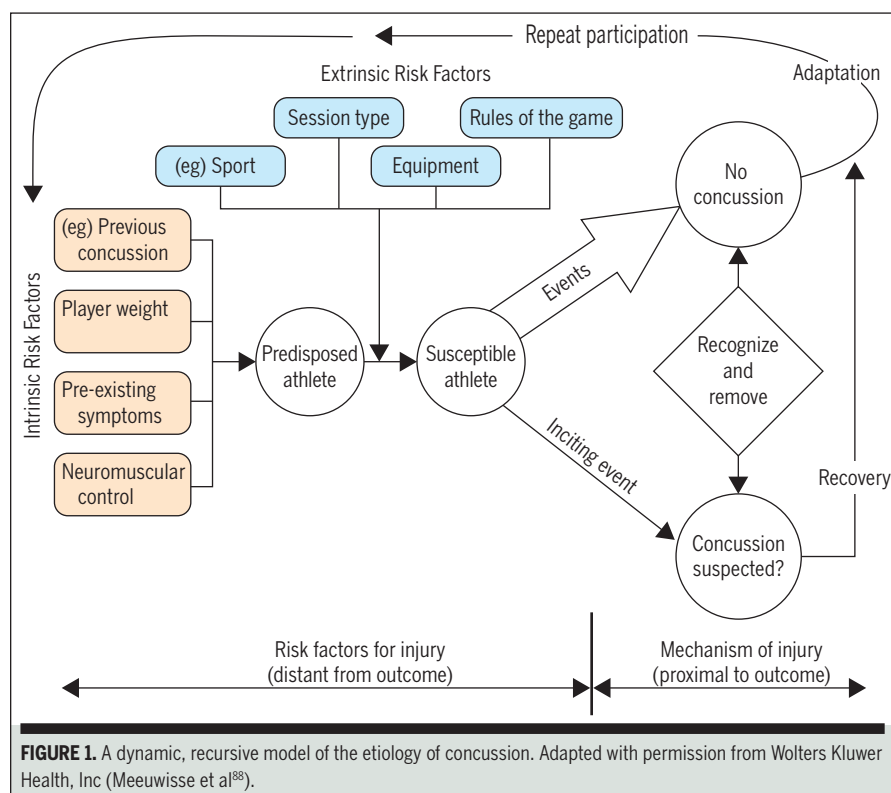


FIGURE 1. A dynamic, recursive model of the etiology of concussion. Adapted with permission from Wolters Kluwer Health, Inc (Meeuwisse et al⁸⁸).

Following concussion, it is necessary to recognize and remove the player from additional risk and refer the player to appropriate medical management as early as possible.^{28,84} Trauma followed by observable signs or symptoms of concussion should trigger an assessment to screen for concussion.^{28,84} A multifaceted assessment can inform appropriate management.^{37,78,84,102} Once the player has recovered and received clearance to return to play, the player may re-enter the dynamic process of adapting through recurrent participation.

PART 1: ETIOLOGY, RISK FACTORS, AND DETECTION OF CONCUSSION

A WIDELY REFERENCED MODEL in the area of sport injury research has proposed that multiple factors influence the etiology of sport injury.⁸⁸ Various etiological factors can vary over time and change the risk that is associated with injury.⁸⁸ The literature in the

area of concussion is evolving and, as such, enables adaptation of this model to better understand the etiology of concussion.

Increasing knowledge regarding concussion burden and identifying factors contributing to multifaceted and recursive risk for concussion will inform the development and evaluation of effective concussion prevention strategies. The best way to decrease the burden of concussion is to prevent the injury before it occurs (ie, through primary prevention).

One frequently referenced models of injury prevention is the van Mechelen model.¹²² In this model, understanding the overall burden of injury in the population and identifying risk factors inform interventions aimed at injury prevention. Knowledge of modifiable risk factors helps to efficiently direct injury prevention efforts, and knowledge of nonmodifiable risk factors helps the clinician achieve an understanding of the overall risk to the athlete and informs return-to-play decision making.

Risk Factors for Concussion

Each individual who participates in an activity brings a specific set of intrinsic and extrinsic factors (FIGURE 1). Understanding the impact that these factors may have on assessment, management, and return to activity/sport helps to ensure a well-balanced and evidence-informed approach to care. In this section, we summarize the key intrinsic and extrinsic risk factors for concussion.

Intrinsic Risk Factors for Concussion Intrinsic risk factors may be modifiable (such as neuromuscular or sensorimotor control) or nonmodifiable (such as previous history of concussion, sex, age, and genetics).

Previous history of concussion is a risk factor for future concussion.¹ The exact mechanism by which this occurs is not yet well understood and may be related to genetics, epigenetics, sensorimotor or neuromuscular control, and other factors.

The literature is inconsistent regarding sex as a risk factor for concussion. In sports with similar rules, women may be at greater risk of concussion than men.^{1,16,43,71,79} Risk may differ due to physical characteristics or because women may be more likely to report symptoms.^{23,117}

As age increases through adolescence, the risk of concussion increases, before declining in the early twenties.^{1,34,43,50}

History of attention deficit hyperactivity disorder or learning disability may increase the risk of concussion and/or detection of concussion in youth and collegiate athletes.^{10,42}

Pre-existing symptoms of dizziness, neck pain, and headache may increase the risk of concussion in male youth ice hockey players.¹⁰⁵ Possible explanations for the increased risk include altered neuromuscular control, sensorimotor control, balance, or cervical spine strength.¹⁰¹

High school athletes with lower neck strength may have a greater risk of concussion.¹⁹ In youth ice hockey players, an increased risk of concussion has been reported in players who did not meet the Canadian recommendations for daily physical activity (1 hour of daily physical

activity) in the 6 weeks prior to study entry.⁹ Player skills and strategy of sport-specific techniques may also influence concussion risk. In elite rugby, the majority of concussions occur to the tackler, and the risk of concussion increases when the tackler accelerates, travels at a high speed, and has head contact.²⁰

Extrinsic Risk Factors for Concussion The environment in which an athlete plays includes factors that can influence the risk of concussion, many of which may be modifiable.

Contact and collision sports, such as rugby, American football, and ice hockey, have the highest reported incidence of concussion.⁹³ In youth ice hockey leagues where bodychecking is permitted, there is nearly a 4-fold increase in risk of concussion in the 11- to 12-year age group (Pee Wee).³³ Game play has greater concussion risk than practice.^{1,22,57}

Interventions Can Mitigate Risk The protective effects of helmets in reducing the risk of more severe traumatic brain injury are well documented,⁷⁶ as is the protective effect of mouthguards in reducing orofacial injury. However, while protective equipment, such as helmets, headgear, and mouthguards, may mitigate the risk of concussion, the literature is inconclusive. Studies in basketball, hockey, and rugby have suggested a protective effect of mouthguards on concussion risk; however, a meta-analysis found no significant effect.³² In American youth football, appropriate helmet fit was associated with lower symptom severity and shorter duration of symptoms.⁴⁴ In ice hockey, appropriate helmet fit may protect against concussion, although further research is needed.⁴¹ Studies examining the use of headgear in rugby and soccer are inconclusive.^{32,95} Further research is needed to better understand the role of protective equipment by sport.

Interventions aimed at primary prevention have shown promise in decreasing the risk of concussion. In youth rugby players, there was a reduction in overall risk of game-related concussion when a neuromuscular training program was

performed at least 3 times weekly.⁴⁹ A vision training program may reduce the risk of concussion in collegiate football players.¹⁸ There was a 67% reduction in the risk of concussion in youth ice hockey following rule changes to disallow bodychecking.³² Tackle training and rules related to tackling in rugby as a way of decreasing risk of concussion are areas of ongoing evaluation.⁴⁷ Finally, restricting the number of collision practices in youth football may reduce the frequency of head impacts in games and practice.¹⁴

Detecting Concussion: Recognize and Remove From Sport Participation

A concussion should be suspected after a direct blow to the head or following trauma to the body by which force has been transmitted to the head.⁸⁴ Some of the observable signs of concussion are lying motionless, clutching the head, unsteadiness, or appearing dazed or confused immediately following a concussion (see the Concussion Recognition Tool Fifth Edition²⁸). Symptoms may be reported by the player, such as headaches, dizziness, nausea, sensitivity to light or noise, fatigue, and feeling as though in a fog. Symptom onset can be delayed, with the duration of the delay predicting a longer time to recovery following injury. Once a concussion is suspected, the player should be removed from play and further assessed by a qualified health care professional (**FIGURE 2**).

Concussion Screening Tools

The Concussion Recognition Tool Fifth Edition (CRT5) is a sideline tool that can be used by parents, coaches, officials, and players to recognize when a concussion may have occurred.²⁸ In some sports, a “spotter” watches for potential signs of concussion and identifies individuals who may require screening for concussion. Immediate removal from activity may improve outcomes.⁴

At the time of injury, screening for more severe injury (eg, intracranial bleeding, cervical spine fracture) is imperative. Clinicians should use a multifaceted assessment that includes symptoms,

a neurological screen, and assessment of multiple clinical domains.^{27,37} The Sport Concussion Assessment Tool Fifth Edition (SCAT5)²⁹ includes an immediate/on-field assessment that incorporates red flags, observable signs, memory assessment (ie, the Maddocks questions), the Glasgow Coma Scale, and a cervical spine assessment. The office (off-field) assessment portion of the SCAT5 includes history, symptoms, cognitive screening (from the Standardized Assessment of Concussion, which includes orientation, immediate and delayed memory questions, and digits and months of the year in reverse order), a neurological screen (including reading, cervical spine range of motion, ocular motor function, coordination, and balance), and a modified version of the Balance Error Scoring System.²⁹ The Child Sport Concussion Assessment Tool Fifth Edition should be used with children aged 5 to 12 years.²¹

The clinical utility of the SCAT5 diminishes after the initial 3 to 5 days following injury.⁸⁴ However, the symptom scale on the SCAT5 can be used to evaluate change in symptoms over time. Other screening tools, such as the Vestibular/Ocular Motor Screening and a combination of optokinetic stimulation, gaze stabilization testing, and near point of convergence, may have clinical utility as screening tools for concussion in the subacute period (2-10 days) following concussion.^{85,89} The SCAT5 and Vestibular/Ocular Motor Screening tools can be used as part of the clinical assessment but should not replace other aspects of the clinical exam that may be warranted, based on the individual circumstances of the injury.^{21,29,84}

PART 2: ASSESSMENT AND MANAGEMENT OF CONCUSSION

THE INITIAL MANAGEMENT OF CONCUSSION involves both cognitive and physical rest for the first 24 to 48 hours following injury.^{84,103} After this time, gradually and progressively increase activities of daily living, as long as symptoms do

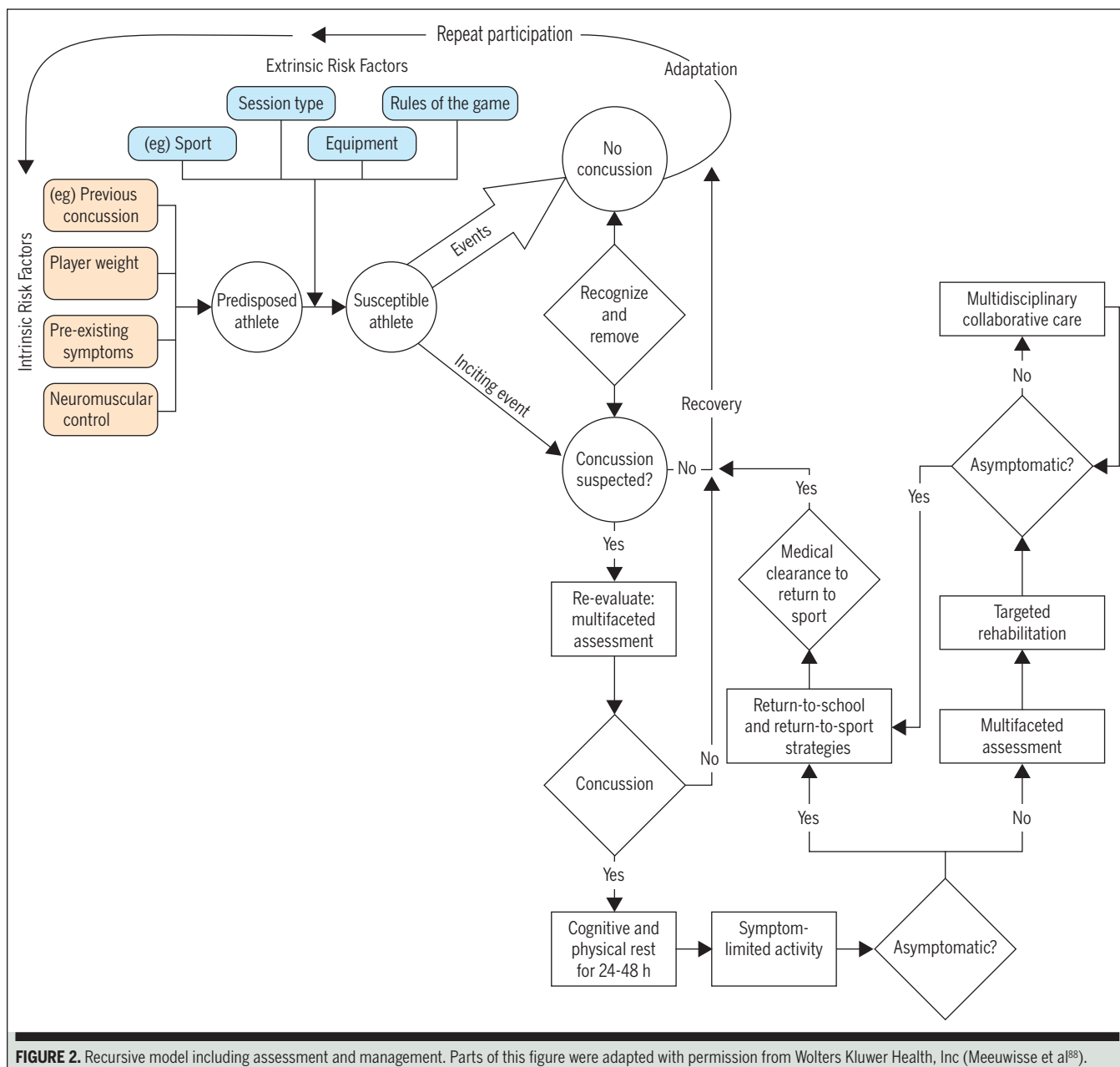


FIGURE 2. Recursive model including assessment and management. Parts of this figure were adapted with permission from Wolters Kluwer Health, Inc (Meeuwisse et al¹⁸).

not increase.^{84,103} Once concussion-related symptoms have resolved with typical activities, gradually resume physical and cognitive activities (FIGURE 2).

Return to school and return-to-sport strategies can occur simultaneously.⁸⁴ Each step of the return-to-school and return-to-sport protocols should take a minimum of 24 hours. If symptoms recur, then the athlete should move back to the previous step.

Return to School

The return-to-school protocol includes 4 steps: (1) daily activities that do not provoke symptoms, (2) school activities outside of school, (3) part-time return to school, and (4) full-time return to school (FIGURE 3).^{29,84} To facilitate return to school, a medical letter including recommendations for individual accommodations is recommended.⁹⁴ Accommodations at school may include

reduced hours at school, more time to complete assignments and examinations, frequent breaks, reduced screen time, and working in a quiet area.²¹ Return to school should occur before return to contact activity or full competition.

Return to Sport

The return-to-sport strategy includes 6 steps: (1) symptom-limited activity, (2) light aerobic exercise, (3) sport-specific

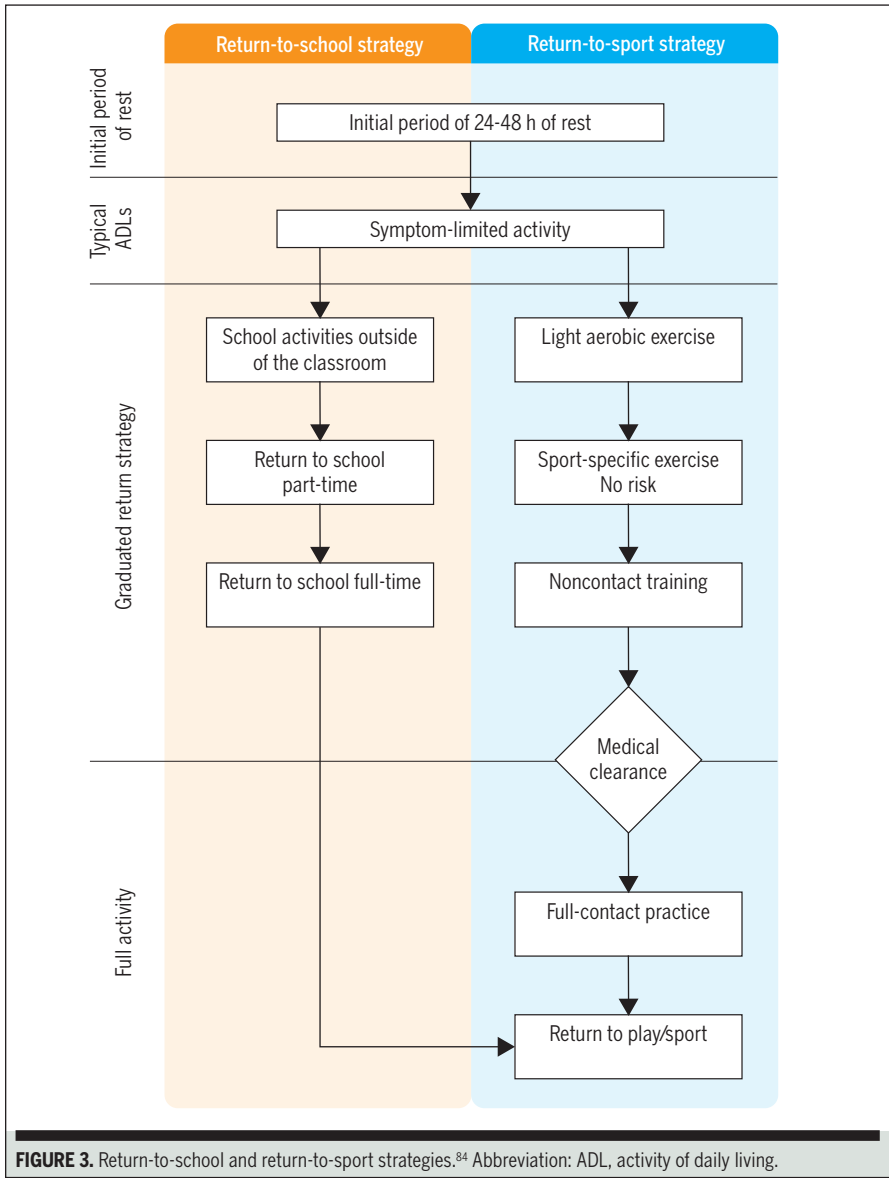


FIGURE 3. Return-to-school and return-to-sport strategies.⁸⁴ Abbreviation: ADL, activity of daily living.

exercise, (4) noncontact training drills, (5) full-contact practice, and (6) return to sport (FIGURE 3).^{29,84} Medical clearance to return to sport occurs once the individual is able to complete the return-to-sport protocol with no symptom exacerbation and when no other clinical assessment findings suggest ongoing problems that would preclude returning to sport.⁸⁴

Return to Work

Return-to-work recommendations are based on similar principles as those of return to school and return to sport.⁹¹

Gradually and progressively increase activities, provided there is no increase in symptoms. If symptoms recur or are exacerbated, reduce the demands of the task to a level that does not provoke symptoms. Consider occupation-specific activities (eg, cognitive and physical demands, safety requirements) in any return-to-work recommendations.^{15,91} Positive health outcomes have been reported with return to work or staying at work.⁹⁹ However, reintroduction of risk and timing in the early recovery period must be carefully monitored.⁹¹

If symptoms persist following the initial days to weeks following injury, a multifaceted assessment to identify areas that may require rehabilitation is warranted.^{37,65,78,103} Refer the patient for additional assessment and rehabilitation if the individual has not recovered in the 10 to 14 days following injury. In some cases, targeted rehabilitation (eg, vestibular rehabilitation, cervical spine rehabilitation, subsymptom threshold aerobic exercise) is warranted.¹⁰⁶ In other cases, further medical investigations, referral to additional interdisciplinary health care professionals, or referral for interdisciplinary care may be required (FIGURE 4).

In this section, we outline 9 common persistent symptoms following concussion,^{7,58,106} describe differential diagnoses, and offer an overview of evidence-based rehabilitation approaches.

Headaches

Headaches are the most frequent symptom following concussion. A post-traumatic headache (1) is a secondary headache that can be attributed to the injury when a new headache occurs following trauma, and (2) must occur within the initial 7 days after the trauma.⁴⁶ If a preinjury headache worsens or becomes persistent, the primary headache diagnosis, in addition to the posttraumatic diagnosis as described above, is to be used.⁴⁶ Headache diagnoses following concussion might also include medication overuse headache, migraine headache (with or without aura), tension-type headache, cervicogenic headache, occipital neuralgia, and mixed headache type.^{73,74,126}

Diagnosis directs appropriate management. In many cases, medical management is required; for some headache types, such as cervicogenic headaches, physical therapy may be of benefit.^{54,125} Botulinum toxin injection can be considered for posttraumatic chronic migraine headaches.²⁴ Repetitive transcranial magnetic stimulation may be effective in the treatment of posttraumatic headache.^{60,69,70} Often, a multimodal approach to headache management

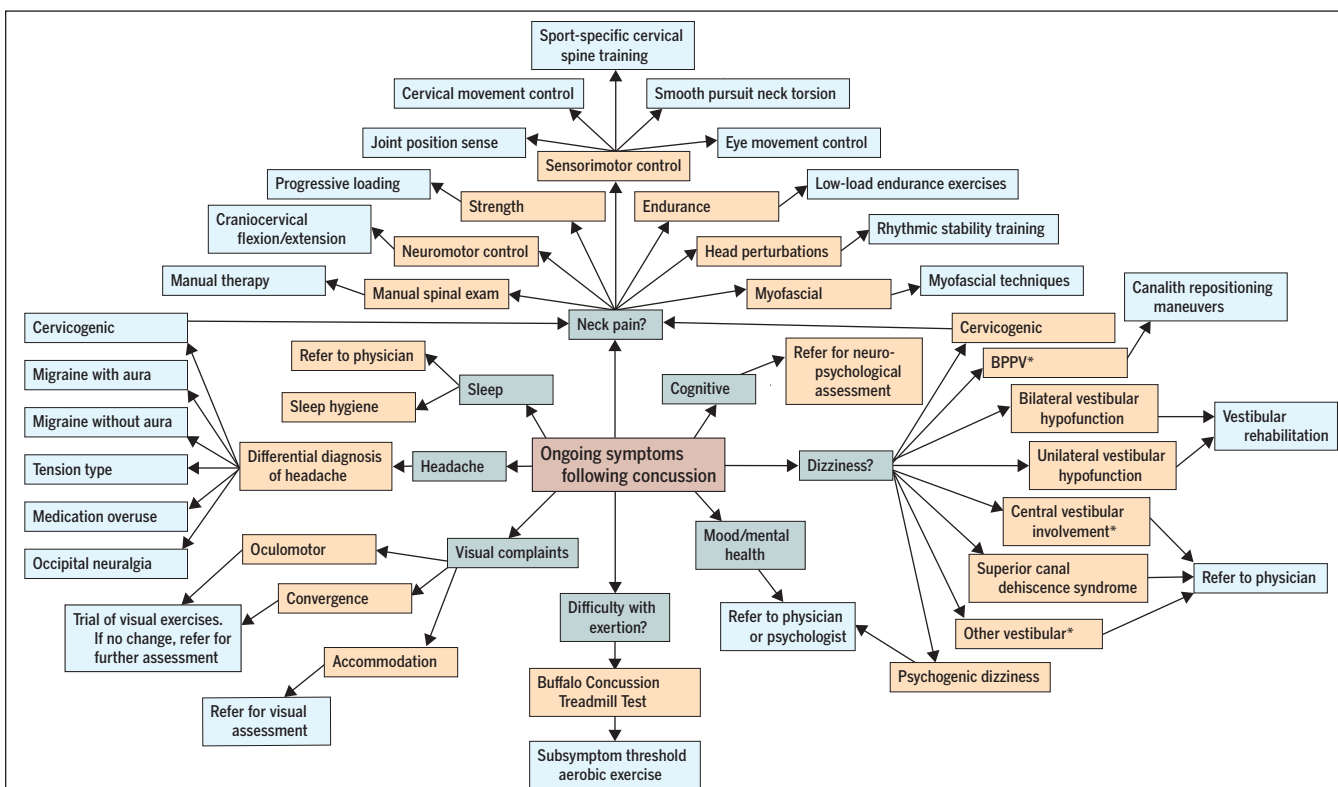


FIGURE 4. Symptoms, targeted clinical assessment, and management following concussion. *May also benefit from vestibular rehabilitation. Abbreviation: BPPV, benign paroxysmal positional vertigo.

includes both acute and prophylactic medication.

Cervical Spine Pain

Cervical spine pain may be accompanied by cervicogenic headache or cervicogenic dizziness.^{7,106} Cervical spine findings are common following concussion (eg, impairments following anterolateral strength, the head perturbation test, joint position to the left, or the cervical flexor endurance test).¹⁰⁷ Many of the symptoms reported following whiplash are similar to those reported following concussion,⁵¹ suggesting that cervical spine injury might have occurred at the same time as the concussion. When the 2 injuries occur concurrently, they must be treated appropriately.

Assessment of the cervical spine should include range of motion, manual spinal exam, general strength, and cervical sensorimotor and neuromotor control.^{61,118,120} The clinical tests that have established

utility in the cervical spine literature, including joint position sense, cervical movement control, the craniocervical flexion test, cervical flexor and extensor endurance, the cervical flexion-rotation test, and manual spinal exam, may be useful in identifying potential areas of dysfunction in concussion.^{53,55,61,97,106,107,119}

Combining specific exercises with manual therapy is effective for treating cervical spine pain.⁵⁴ After concussion, include neuromotor control, sensorimotor control, manual therapy, and soft tissue techniques, in combination with vestibular rehabilitation.¹⁰⁶ A sequential approach to addressing headaches and cervical spine findings (including neuromotor control) as an initial step of rehabilitation is appropriate, given the connections between the upper cervical spine and the vestibular and visual systems.

Treatment may be more effective when initiated early in the recovery process.¹⁰⁴ However, further research is war-

ranted to identify the ideal timing and type of intervention. In the presence of ongoing cervical spine findings, further interventional procedures, such as comparative controlled medial branch blocks (to confirm facet joint-mediated pain), trigger point injections (in the presence of ongoing myofascial pain), and greater occipital nerve blockades (in the presence of greater occipital neuralgia), may be considered.^{26,98}

Dizziness

Dizziness is typically the second most common symptom following concussion.⁷ As with headache, identifying the source of dizziness is important to direct treatment—some disorders respond well to physical therapy (eg, benign paroxysmal positional vertigo [BPPV], unilateral peripheral vestibular hypofunction), while others require medical evaluation and management (eg, superior semicircular canal dehiscence).^{2,45,48}

Dizziness symptoms can be vertigo (ie, sensation of spinning of the environment or the person), light-headedness, presyncope, or a sense of disorientation. In addition to a thorough history, targeted assessment tests help clinicians to understand the source of dizziness. Neurological examination, assessment of vestibulo-ocular reflex function, static balance, dynamic balance, assessment of nystagmus with and without fixation, positional testing, and vestibular function testing can inform diagnosis and management.

The duration of a dizziness episode can also provide a clue as to the source of dizziness following concussion. For example, BPPV may occur in approximately 5% of cases of ongoing dizziness following concussion.^{2,106} Suspect BPPV when the patient describes seconds of vertigo with positional changes (eg, lying down/getting up, rolling in bed, looking up, bending over).⁸ For BPPV to be diagnosed, a positive Dix-Hallpike test with seconds of vertigo and a characteristic pattern of nystagmus should be present.⁸ Canalith repositioning maneuvers (eg, the Epley maneuver) are effective for treating BPPV (up to 98% of cases resolve within 3 treatments).^{8,75}

In up to 10% to 26% of cases of ongoing dizziness following concussion, assessment findings suggest peripheral vestibular hypofunction (ie, decreased vestibular labyrinth function).^{11,12} Suspect a peripheral vestibular problem in patients who report intense dizziness and unsteadiness following the concussion, followed by a gradual improvement of symptoms over the initial few weeks. Symptoms are often provoked with rapid head motions, and blurred vision may be reported in association with head movement (suggesting altered vestibulo-ocular reflex dysfunction). Unsteadiness or imbalance may also be reported (suggesting difficulty with maintaining an upright position in space, possibly related to dysfunction of vestibulospinal function, proprioception, vision, or other systems that contribute to upright bal-

ance) and should be assessed. In other cases, findings suggest that central vestibular involvement may be present. Less common diagnoses may include temporal bone fracture (with resultant damage to the eighth cranial nerve), labyrinthine concussion, peri-lymphatic fistula, and semicircular canal dehiscence.^{12,35}

Vestibular rehabilitation may be of benefit for individuals with peripheral vestibular disorders (including BPPV) and stable central vestibular disorders.^{8,45,75} Positive effects on recovery following vestibular rehabilitation after concussion have been reported in the literature.^{2,106} Typically, vestibular rehabilitation includes canalith repositioning maneuvers (for BPPV) and individually targeted exercises aimed at facilitating sensorimotor compensation (including adaptation, habituation, substitution, and standing and dynamic balance exercises).^{3,8,106}

Vision

Blurred vision, double vision, and difficulty reading may be reported following a concussion. Findings of convergence insufficiency have been identified in children following concussion; however, further research is needed to identify whether these deficits are pre-existing or have their onset following trauma. Of the children with convergence insufficiency, 46% had their symptoms resolve in the initial 4.5 weeks following injury, and another 41% reported recovery following vestibular rehabilitation that included convergence training.¹¹⁰ Deficits in smooth pursuit, saccades, near point of convergence, and accommodation also have been reported following concussion, often in the presence of vestibulo-ocular reflex deficits and altered balance.⁸¹ Thus, visual assessment should include smooth pursuit, saccades, near point of convergence, and accommodation, in combination with a vestibular and balance examination.

Many individuals who have suffered a concussion may report difficulty with reading at school, work, or during screen

time (eg, computers, smartphones, tablets). Individuals with visual symptoms following concussion may benefit from accommodations to enable earlier return to school or work in a less visually provocative environment (eg, printed materials rather than electronic, change in contrast on a screen).⁹⁴ Frequent breaks, pacing of activities, and working in a quieter environment may facilitate return to function.

Exertion

Autonomic function may be disrupted following concussion.²⁵ An increase in symptoms can occur for some individuals when they increase the intensity of physical activity. This type of difficulty has been termed *physiological postconcussion syndrome* and has been hypothesized to occur secondary to autonomic nervous system involvement.^{67,68} For individuals who report difficulty with exertion, the Buffalo Concussion Treadmill Test is a widely used clinical test (based on the Balke test) that progressively increases the level of physical activity by increasing the incline and then speed of a treadmill.⁶⁷ The test is stopped when symptoms increase (termed the *symptom threshold* and defined as at least a 2-point increase in symptom severity on a 0-to-10 rating scale) or when the individual reaches voluntary exhaustion.

Exercise may facilitate recovery following concussion.^{40,66,67} Two different paradigms of exercise have demonstrated benefit for symptoms and function: (1) subsymptom aerobic exercise training at 80% of the maximal heart rate that was achieved on the Buffalo Concussion Treadmill Test, 5 days per week⁶⁷; and (2) exercising at 60% of maximal heart rate (calculated as $220 - \text{age} \times 60\%$) for up to 15 minutes, combined with guided imagery and sport-specific coordination exercises.^{39,40} However, some studies have reported an increase in symptoms with exercise in children and youth, and others have reported no change.^{77,103} Given the known general positive benefits of exercise, consideration of aerobic exer-

cise training following the initial return to activity after concussion is warranted in the absence of contraindications to exercise.¹⁰³

Cognitive Difficulties

Cognitive symptoms can include difficulties remembering and concentrating, slowed processing, decreased attention, and difficulty with learning.^{58,86} Cognitive symptoms often resolve over the initial days to weeks following injury. Sometimes, cognitive symptoms persist and may be associated with ongoing difficulties with school and with occupational, sport, and social activities.⁸⁶ Cognitive symptoms often occur in the presence of other symptoms, such as pain, headaches, difficulties with vision, and sleep problems.⁸⁷ Referral to a neuropsychologist may be warranted for a thorough assessment to clarify the etiology of the cognitive complaints and to assist in developing an individualized management plan. Evidence for cognitive remediation following concussion is very limited.

There may be alterations in gait, reduced gait velocity, and increased sway when dividing attention following concussion.^{38,59} Further research to better understand changes in the ability to divide attention while accounting for growth and development is warranted.

Mood and Mental Health

Irritability, sadness, anxiety, and feeling more emotional than normal are often reported following concussion, although they may not be acute.⁵⁸ The psychological response to concussion may be similar to that to musculoskeletal injury, and improves over time.¹²¹ Some adults may have generalized anxiety disorder, panic attacks, and posttraumatic stress disorder following injury, which may reflect a new diagnosis or an exacerbation of a previous condition.¹²⁸ Anxiety and depression are more common in women than in men, and may predict a longer recovery.^{52,109} Ongoing psychological or psychiatric problems are rare in children and youth without preinjury problems.³¹ Management of

mental health problems will depend on the specific diagnosis (eg, pharmacological or psychological treatment). Use standardized tools when screening for mood and mental health problems.¹³

Sleep Problems and Fatigue

Up to 1 in 2 individuals with concussion report sleep problems (insomnia, difficulty falling asleep, difficulty staying asleep).^{17,82} In the early postinjury phase, individuals may be more fatigued than normal and require more sleep.⁹⁶ Individuals with pain may also require more sleep.^{64,112} Addressing sleep difficulties is important to improve recovery.⁹²

Treatment of sleep disorders often includes pharmacological and nonpharmacological management.^{123,124} Education regarding sleep hygiene may improve sleep quality. Cognitive behavioral therapy may be beneficial for insomnia.³⁶ Melatonin may have benefits for sleep following concussion, but is not currently recommended for sleep onset or maintenance problems.¹⁰⁰ In the presence of ongoing sleep difficulties, refer to a sleep specialist to further investigate potential underlying causes.

Key sleep recommendations include⁹¹:

- Avoid caffeine and alcohol for several hours before bedtime.
- Set a consistent wake time.
- Monitor sleep time with a diary.
- Relax for an hour prior to going to bed.
- Only go to bed when tired.
- Use the bed only for sleeping.
- Limit naps to less than 1 hour, and prior to mid afternoon.
- Limit screen time prior to sleeping.

Neuroendocrine Dysfunction

Neuroendocrine dysfunction, caused by injury to the hypothalamic-pituitary axis following mild to severe traumatic brain injury^{5,6,90,114} and sport-related concussion,^{72,113,114,116} has been reported. Growth hormone is the most commonly affected hormone following concussion.^{56,63,114,115} Individuals with symptoms consistent with alteration in sex hormones, hypothyroidism, adrenal dysfunction, diabetes

insipidus, syndrome of inappropriate antidiuretic hormone secretion, or growth hormone deficiency (fatigue, disrupted sleep patterns, and cognitive difficulties) should be investigated for hypothalamic-pituitary axis dysfunction.¹¹⁴

Predicting Recovery

People with more, and more severe, acute and subacute symptoms take longer to recover following concussion.⁵² Adolescent age, female sex, the presence of a migraine history, and pre-existing mental health problems are predictors of slower recovery.⁵² Many other factors (eg, previous history of concussion, preschool age, race, genetics) have been evaluated as potential predictors of longer recovery, with mixed results.⁵² Attention deficit hyperactivity disorder and learning disabilities are unlikely to be risk factors for prolonged recovery.⁵² Among youths 5 to 18 years of age who presented to an emergency department, female sex, older than 13 years of age, migraine history, previous concussion with symptoms for greater than 1 week, sensitivity to noise, fatigue, headache, parent reporting that the child answers questions slowly, and more than 3 errors on the Balance Error Scoring System-tandem stance were predictors of longer recovery.¹²⁷ Children with visual, vestibular, and cervical spine findings also recover more slowly.^{30,81}

An Overview of Rehabilitation Following Concussion

After an initial 24 to 48 hours of cognitive and physical rest,^{84,103} initiate a strategy of gradual return to school and sport.⁸⁴ If symptoms persist beyond 7 to 10 days following injury, targeted treatment may be warranted.^{84,103} Rehabilitation following concussion should be informed by a multifaceted, interdisciplinary assessment aimed at identifying underlying sources of ongoing symptoms.^{78,103}

In the presence of headache, differential diagnosis of headache type is imperative to inform management. For individuals with ongoing dizziness, neck pain, and headaches, cervicovestibular

physical therapy can be beneficial.^{103,104,106} Sport-specific training, related to the context in which the individual would be participating, should form an integral part of the rehabilitation program.¹⁰⁶ For children and adolescents with visual and vestibular findings, vestibular rehabilitation may be of benefit.¹¹¹ In addition, low-level aerobic exercise may promote recovery following concussion.^{40,62,67}

Collaborative care, including cognitive-behavioral therapy, care management, and psychopharmacological evaluation, has positive effects on symptom reduction after 6 months.⁸³ An active approach to rehabilitation, including aerobic exercise, visualization, and coordination, has positive effects on symptoms and function.^{39,40} Future research to best understand timing, order, frequency, and other parameters of combination treatments is warranted.¹⁰³

Return-to-sport and return-to-school strategies include a gradual return to activities, which may vary depending on the environment to which the athlete returns. Sport-specific and performance-related skills may be necessary to return to full participation. For instance, the skills required of a volleyball player will differ from those expected of an ice hockey player. Thus, consideration of sport-specific skills should be an integral part of a rehabilitation program. In addition, decisions regarding return to sport may be affected by the intrinsic and extrinsic factors that characterize the individual athlete. Discussion among the health care team and with the individual and his or her family can facilitate appropriate return-to-sport and return-to-school decisions.

SUMMARY

AS CONCUSSIONS DIFFER SO WIDELY, an awareness of risk factors and individual clinical characteristics can facilitate an individualized approach. A multifaceted assessment for each patient should include postconcussive symptom reports; a neurological screen; assessment

of cervical spine, vestibular, visual, and exertion-related symptoms; plus sleep, mood, cognitive, and related domains. Once the individual has completed 1 to 2 days of rest, a gradual return to sport and school/work is recommended. Return to participation in sport should occur along a continuum, with respect to the individual's risk of concussion and the characteristics of the environment to which the person is returning. Such a rehabilitation strategy, tailored to the individual, can facilitate high-quality, evidence-informed care and injury prevention. ●

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