Neuromechanical Considerations for Postconcussion Musculoskeletal Injury Risk Management

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Abstract

Recent epidemiological studies have documented increased susceptibility to musculoskeletal injury after sport-related concussion, which raises questions about the adequacy of current clinical practices to ensure safe return to sport. A growing body of evidence derived from advanced neuroimaging and neurological assessment methods strongly suggests that mild traumatic brain injury has long-lasting adverse effects that persist beyond resolution of clinical symptoms. Plausible interrelationships among postconcussion changes in brain structure and function support the rationale for specific methods of clinical assessment and training to target the interaction of cognitive and motor function for reduction of musculoskeletal injury risk after concussion. The findings of preliminary clinical studies are presented to support suggested strategies for reduction of postconcussion musculoskeletal injury risk, and to identify novel approaches that we consider worthy areas for further research.

Introduction

An estimated 1.6 to 3.8 million sport-related head injuries occur each year (30). These injuries result in known deficits in neurocognitive performance, coordination, and balance that typically recover within 7 to 10 d after injury (40), but discoveries made over the past decade suggest that subtle motor control impairments persist well beyond relief of symptoms and recovery of basic neurocognitive functions. Specifically, laboratory-based studies have demonstrated prolonged delays in the transmission of neural signals within the brain and between the central and peripheral nervous systems (11,13,14,32,33,41,56,63,64). Changes in neural mechanisms that mediate visual-motor function, cognition, reflexive responses, postural alignment and hemispheric communication may be driving subtle perceptual and motor control deficits that could increase the risk for musculoskeletal injury upon return to participation in sport-related activity (8,12,15,19). We provide a review of current evidence relating to the influence of concussion on musculoskeletal injury risk, proposed physiological mechanisms by which concussion and musculoskeletal function are linked, and recommended methods for the assessment and reduction of injury risk during the concussion management process.

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Review of Epidemiological Evidence Linking Concussion to Musculoskeletal Injury

A total of 10 studies that have examined musculoskeletal injury rates upon return to sport after concussion have been published (5,6,9,20,26,34,37,50,51,53). The methodology and results of these studies are summarized in Table 1. Populations have included professional American, Australian, and European football, elite rugby, and both male and female college athletes. Variations in injury surveillance duration and injury definitions have generated somewhat inconsistent results, but the overall findings demonstrate increased musculoskeletal injury risk after return to sport.

The first published study pertaining to the possible effect of concussion on subsequent injury involved a cohort of elite Australian football players. Makdissi et al. (37) compared the incidence rate for injury of any kind during three consecutive games after return to play between 138 concussed players and an equal number of matched nonconcussed players. The incidence rate per 100 games was 7.25 for concussed players and 3.25 for the position-, age- and size-matched control players. Although the reported incidence rate ratio (IRR) of 2.23 (95% confidence interval [CI], 0.95, 5.04) clearly demonstrated a substantially larger injury rate for the concussed players, the authors interpreted the analysis result as a finding of no significant difference between the two groups.

Table 1.
Summary of research evidence relating to concussion association with musculoskeletal injury.

Author(s)	Population	Comparison Time Frame(s)	Injury Definition	Results[a]
Makdissi et al., 2009 (37)	Australian football players Cx cases, n = 138 Controls, n = 138	3 wks before injury; 3 wk post-RTP	Injury database included any injuries occurring in games	IRR = 2.23 (0.95–5.04) Cx injury rate: 7.25 per 100 games Control injury rate: 3.25 per 100 games
Nordström et al., 2014 (50)	Premier European soccer players, Cx cases, n = 66 Controls, n = 1599	12 months before injury; 0–3 months, 3–6 months, and 6–12 months post-RTP	Injuries resulting in inability to fully participate in training or match play	Before injury: HR = 1.47 (1.05–2.05) 0–3 mo: HR = 1.56 (1.09–2.23) 3–6 mo: HR = 2.78 (1.58–4.89) 6–12 mo: HR = 4.07 (2.14–7.76)
Nyberg et al., 2015 (51)	Swedish elite ice hockey players, Cx Cases, n = 144 Controls, n = 81	7 d, 21 d, and 42 d post-RTP	Injuries that lead to absence or a medical intervention	7 d: Cx = 13/140, Con = 4/98 (P = 0.12) 21 d: Cx = 35/138, Con = 22/97 (P = 0.64) 42 d: Cx = 56/122, Con = 35/90 (P = 0.31)
Burman et al., 2015 (6)	Swedish hospital patients, Cx Cases, n = 281 Controls, n = 1259	24 months before and after the indexed injury	Retrospective review of all injuries presenting to emergency department	Before injury: OR = 1.98 (1.45–2.72) After injury: OR = 1.72 (1.26–2.37)
Pietrosimone et al., 2015 (53)	Professional American football players, N = 2429	Playing career (retrospective self-reported injury history)	Self-reported frequency of injuries sustained among 9 categories	1 cx: OR = 1.59 (1.30–1.94) 2 cx: OR = 2.29 (1.85–2.83) 3+ cx: OR = 2.86 (2.36–3.48)
Lynall et al., 2015 (34)	Division I collegiate athletes, Cx Cases, n = 44 Controls, n = 58	12 months before injury; 90, 180, and 365 d post-RTS	Any musculoskeletal injury documented by the athletic trainer (in electronic medical records)	12 months preinjury: RR = 0.69 (0.41–1.17) 90 d post-RTP: RR = 2.10 (0.91–4.81) 180 d post-RTP: RR = 2.02 (1.08–3.78) 365 d post-RTP: RR = 1.97 (1.19–3.28)
Cross et al., 2016 (9)	Professional rugby players, Cx Cases, n = 135 Controls, n = 660	2 seasons of play post-RTP; comparing typical vs. prolonged recovery	Injuries resulting in 24-h time-loss	OR = 1.6 (1.4–1.9) RR = 1.1 (0.8–1.5) Typical vs. prolonged RTP: RR = 1.3 (0.9–2.0)

Brooks et al., 2016 (5)	Division I collegiate athletes, Cx Cases, n = 87 Controls, n = 182	90 d post-RTP	Noncontact acute fractures, muscle strains/tears, or ligament sprains/ruptures of the lower extremity	Cx injury incidence: 15/87 = 17% Con injury incidence: 17/182 = 9% OR, 2.48 (1.04–5.91)
Gilbert et al., 2016 (20)	NCAA collegiate athletes (all divisions), N = 335	Collegiate playing career (injury history questionnaire completed at conclusion of college career)	Self-reported history of ankle sprain, knee injury, and muscle strain of the lower extremity	Ankle sprain: OR = 1.79 Knee injury: OR = 2.13 Muscle strain: OR = 1.61
Herman et al., 2017 (26)	Division I collegiate athletes, Cx Cases, n = 90 Controls, n = 148	90 d post-RTP	Time-loss lower-extremity musculoskeletal injury including sprains, strains, dislocations, or ruptures	Cx injury incidence: 45/90 = 50% Con injury incidence: 30/148 = 20% OR = 3.39 (1.90–6.05

Cx, concussion group; Con, control group; RTP, return to participation; IRR, injury rate ratio; RR, risk ratio; HR, hazard ratio; OR, odds ratio.
[a] 95% CI presented in parentheses.

In 2104, Nordström et al. (50) reported the results of an analysis of all time-loss injuries sustained during the 12 months preceding and after a concussion among 66 elite European soccer players, which were compared with those for 1599 players who sustained nonconcussive injuries. Concussion was found to increase risk of subsequent injury on a time-dependent basis. The hazard ratio (HR) progressively increased from 1.56 at 3 months after return to sport to 4.07 at 12 months. The mean number of injuries over the 12 months of follow-up were 2.2 times greater for the concussed players (11.5 vs 5.0). Interestingly, the concussed players appeared to be more prone to injury in the year preceding the concussion than the players who sustained nonconcussive injuries (mean number of injuries, 1.8 vs 0.9). However, after controlling for preconcussion injury rates, the increase in postconcussion injury risk after to play remained significant, with a 12-month HR of 1.47 (95% CI, 1.05–2.05). The results also demonstrated that concussion can lead to increased risk for all acute injuries, including fractures, contusions, ligament sprains, and muscle strains (HR, 1.70; 95% CI, 1.20–2.41), but no effect was evident for overuse injuries, such as tendinopathies, stress fractures, and musculoskeletal pain (HR, 1.00; 95% CI, 0.60–1.66).

Nyberg et al. (51) performed a retrospective review of 28 seasons of injury data for a Swedish elite ice hockey club to compare injuries after return to play for athletes who sustained a concussion to those who sustained a knee sprain without clinical instability. Subsequent injuries of any type that resulted in time loss or medical intervention for the two groups were compared for periods of 7, 21, and 42 d after return to play. Although concussed players sustained more than twice as many subsequent injuries as players with a previous knee injury within 7 d after return to play (risk ratio [RR], 2.27; 95% CI, 0.76–6.77), the authors interpreted the finding as a nonsignificant difference. A significant difference was reported for severe injuries that resulted in more than 28 d of time loss within 21 d after return to play (17.5% of concussed players vs 0% of players with previous knee injury). Also, players who had a history of more than one concussion sustained 51% more injuries within 42 d of return to play compared to players with previous knee injury (RR = 1.51; 95% CI: 1.09, 2.08).

Burman et al. (6) performed a retrospective analysis of data derived from injury records maintained by a Swedish hospital to compare injury occurrences for male and female ice hockey, soccer, floorball, and handball players who sustained a concussion with those who sustained an ankle sprain. All injury occurrences for the two groups were compared for both the 2-yr period before and the 2-yr period after the index injury. Concussed athletes were found to sustain more injuries than the ankle sprain comparison group of athletes both before and after the index injury (odds ratio [OR], 1.98 and OR, 1.72, respectively), which the authors suggested might have been due to a more aggressive style of play among the athletes who had sustained concussions. Only injuries documented by the hospital emergency department were included, and there was no means to quantify exposures before or after the concussion or ankle sprain.

Pietrosimone et al. (53) reported a positive association between risk of musculoskeletal injury and number of concussions reported by 2429 retired National Football League

(NFL) players. Self-report of any lower-extremity injury demonstrated an OR of 1.59 (95% CI, 1.30–1.94) for those with history of only one concussion compared with those with none. The OR increased to 2.29 among those with a history of two concussions, and it further increased to 2.86 among those with a history of three or more concussions. This study also examined incidence of knee- or ankle-specific injuries, with the knee injury OR ranging from 1.36 for one concussion to 1.92 for three or more concussions and the corresponding ankle injury OR ranging from 1.36 to 2.37 for the ankle. This study was limited by its retrospective design and reliance on self-reported injury history. Because the NFL does not archive medical records, the amount of time that elapsed between concussion and musculoskeletal injury could not be determined, nor could the accuracy of self-reported concussion and musculoskeletal injury history be validated. Thus, musculoskeletal injury may have preceded concussion occurrence in some cases.

Herman et al. (28) reported the preliminary results of a study of 6 yr of injury data for American college athletes in a 2013 professional conference abstract. Using a 90-d follow-up period after return to play for the occurrence of lower-extremity sprain or strain that resulted in time loss, an OR of 3.79 (95% CI, 2.01–7.14) was reported for a comparison of 61 concussion cases to 179 matched control athletes who had not sustained a concussion during the previous 12 months. A complete report was published by the same group of researchers in 2017 (26), which included two additional years of injury data for 90 concussion cases and 148 matched control athletes. The reported incidence of lower-extremity injury after return to play was 50% for athletes who had sustained a concussion, whereas the corresponding 90-d incidence for uninjured control athletes who were matched on age, exposure level, and position was 20%, which corresponds to an RR of 2.5 (OR, 3.39; 95% CI, 1.90–6.05). Using similar methods, Brooks et al. (5) matched 87 concussion cases to 182 control athletes for sport/sex and game/match exposure level and documented the occurrence of noncontact lower-extremity acute fracture, strain, or sprain over a 90-d surveillance period. The odds for lower-extremity musculoskeletal injury were found to be 2.48 times greater for concussed compared with matched control athletes (95% CI, 1.04–5.91). Neither Herman et al. (31) or Brooks et al. (5) assessed differences in injury incidence before concussion.

Lynall et al. (34) examined lower-extremity musculoskeletal injury incidence per 1000 athlete exposures during the year before concussion and the year after return to play in 44 college athletes, which was compared with those for 55 nonconcussed athletes who were matched on sport/sex, game/match exposure, age, height, and weight. The analysis of lower-extremity injuries included any acute sprain, strain, fracture, or contusion, regardless of time loss from participation. Consistent with the findings of Nordström et al. (50), they demonstrated that injury rates for the year after a concussion were time-dependent. Compared to the injury rate for the previous year, the concussion cases did not demonstrate a significantly higher injury rate at 90 d after return to sport, but the IRR was 2.02 at 180 d (95% CI, 1.08–3.78) and 1.97 at 365 d (95% CI, 1.19–3.28). Before concussion, no group differences in injury incidence were found at any time point (IRR at 90 d, 0.66; 95% CI, 0.28–1.55; IRR at 180 d, 0.66; 95% CI, 0.35–1.26; IRR at 365 d, 0.69; 95% CI, 0.41–1.17). The lack of an elevated preconcussion injury incidence rate is contrary to the finding of Nordström et al. (50), which may have been due to a wider variety of sports, a more stringent standard for concussion diagnosis, and better matching of controls. For the year after concussion, comparison of the cases to matched controls demonstrated an IRR of 1.64 (95% CI, 1.07–2.51).

The association of rugby-related concussion with subsequent time-loss injury of any type was examined in 810 English Premiership players over 2 seasons of play by Cross et al. (9). A total of 181 concussions occurred during the study period, with an overall incidence rate of 8.9 concussions per 1000 h of participation. The IRR for comparison of 135 players who returned to play after a concussion to the rest of the cohort was 1.6 (95% CI, 1.4–1.9), which matches the IRR value reported by Lynall et al. (34) for postconcussion case-control comparison. No significant difference in injury rate was found between groups for the period before concussion occurrence (IRR, 1.1; 90% CI, 0.8–1.5). The mean time to subsequent injury among concussed athletes was 52 d, whereas a mean of 114 d was documented for a group of randomly selected players who had sustained a nonconcussive injury.

Relationships between reported, unrecognized, and unreported concussion and region-specific lower-extremity injuries were examined by Gilbert et al (20). When including any concussion, whether self-reported, potentially unrecognized, or knowingly unreported, significant associations were found with the occurrence of ankle sprains (OR, 1.79), knee injuries (OR, 2.13), and muscle strains (OR, 1.61). This study did not account for temporal relationships between musculoskeletal injury and concussion occurrence, but did examine rates of unreported and potentially unrecognized concussion, which apparently occurred among 36.4% of the surveyed athletes (122/335). No significant differences were found between injury rates, but potentially unrecognized concussions had stronger associations with ankle sprain (OR, 2.29) and muscle strain (OR, 1.90) than self-reported and knowingly unreported concussions. Although an aggressive style of play may be associated with both underreporting of concussion symptoms and risk for musculoskeletal injury, the influence of intentionally unreported and unrecognized concussions may be responsible for underestimation of the association between concussion and musculoskeletal injury risk.

The multifactorial nature of musculoskeletal injury susceptibility makes precise estimation of the isolated effect of concussion exceedingly difficult, if not impossible. Despite this challenge to interpretation of epidemiological data, the collective findings of the 10 studies that have examined the problem strongly suggest elevation of musculoskeletal injury risk after postconcussion return to play. The 2017 Consensus Statement of the Concussion in Sport Group (40) acknowledges that neurobiological recovery might extend beyond clinical recovery in some athletes, and that group statistical analyses can obscure subgroup results and individual differences. Furthermore, there was an acknowledgement that the science of sport-related concussion is evolving, which makes clinical judgment essential for the management of individual cases and return-to-play decisions. Because the available evidence suggests that physiological dysfunction may outlast current clinical measures of recovery, clinicians should

consider theoretical explanations that are relevant to clinical management of concussion.

Proposed Physiological Link Between Concussive Injury and Musculoskeletal Injury Risk

The results of studies involving electroencephalography (EEG), transcranial magnetic stimulation (TMS), functional magnetic resonance imaging (fMRI), and other neurophysiological assessment methods have demonstrated the importance of neural processes to the dynamic stabilization of body segments (46). The term "neuromechanical coupling" has been used in the context of dynamic joint stability to refer to modulation of muscle tone that can optimize joint stiffness in response to perceptual inputs to the central nervous system (47). Although the intrinsic neural processes of neuromechanical decoupling after concussion (Fig. 1) are difficult to quantify clinically, the capability to generate optimal motor responses to varied visual, cognitive, and sensory demands can be measured to assess "neuromechanical responsiveness" (Fig. 2). Both impact avoidance and restraint of abnormal joint displacements require rapid initiation of appropriate muscle activation patterns (neuromechanical responsiveness) that depend on brain processing of sensory inputs (neuromechanical coupling).

Accumulating evidence indicates that concussion has long-lasting adverse effects on transmission of neural signals within the brain (11,13,14,32,33,41,56,63,64). However, investigations into the precise neural activity or structural changes after concussion have produced mixed results, which may be attributed to the heterogeneous nature of mild traumatic brain injury, variability in participant characteristics (*i.e.*, age, sport, and gender), differing definition of concussive injury, time from injury, and method for quantification of neurological status across studies (Table 2). However, a common finding across many investigations of brain structure and function after concussion is the existence of changes that could affect motor control (Fig. 1). Disruptions in the structural integrity of neurons and supporting glial and astrocyte cells, brain metabolism, neurotransmitter levels, intracortical inhibition, and connectivity of the motor cortex to other brain regions are likely responsible for the inhibited and delayed motor cortex communication with peripheral muscles, thereby reducing neuromechanical responsiveness (Fig. 2). Further supporting the link between concussion and musculoskeletal injury risk are the findings of several investigations that have documented diminished motor performance in parallel with changes in brain activity or structural connectivity. These studies have demonstrated postconcussion degradation of white matter integrity in the corpus callosum (intrahemispheric connectivity) and the anterior corona radiate (motor projections), associated with adverse effects on reaction time (48), attention (43), gaze control (39), and executive function (29).

This connection between disrupted neural signals and neuromechanical deficits is evidenced by the direct relationship between cortical excitability and muscle tone (*i.e.*, continuous low-level muscle contraction at rest), which is believed to contribute to a state of readiness for rapid generation of a high level of muscle tension (47). The ability to effectively modulate tension in multiple muscles simultaneously is required to maintain dynamic stability of the lower-extremity joints (59). The decreased cortical excitability and impaired intracortical inhibition after concussion may reduce muscle tone, impair reflexive muscle responses, coordination, and the capability to protect joints through rapid generation of muscle stiffness in response to perturbation (31,54,58). The concurrent changes in cortical excitability after concussion along with impairments in postural control and movement coordination, likely contribute to elevated musculoskeletal injury risk (36).

The association of concussion with depression, anxiety, and stress (17), along with evidence that cognitive, affective,

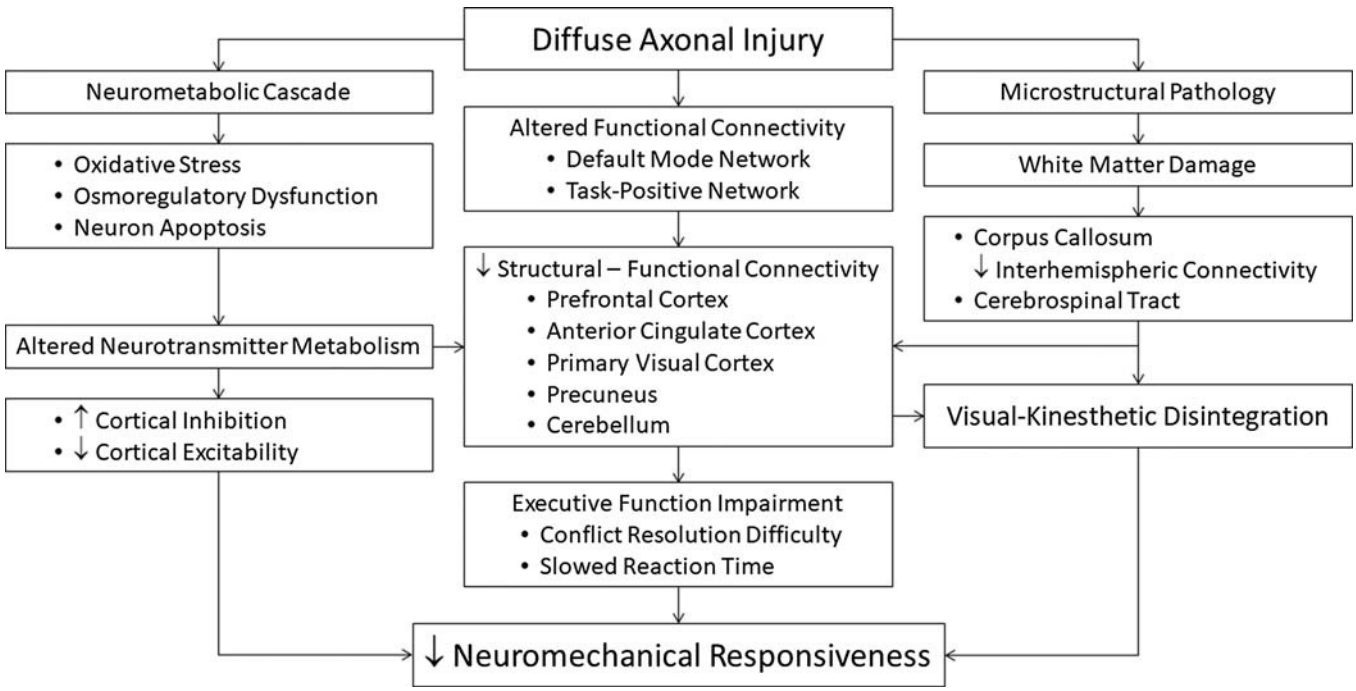

Figure 1: Pathophysiologic factors that may contribute to postconcussion susceptibility to musculoskeletal injury.

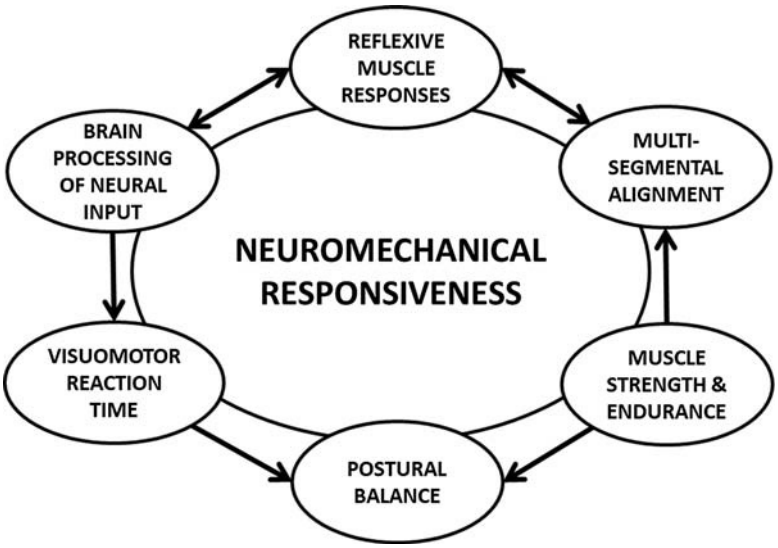

Figure 2: Interrelated quantifiable attributes and capabilities that may contribute to an optimal level of neuromechanical responsiveness.

and motor processes interact with one another, makes the influence of psychosocial factors on injury risk an important consideration (38,49,70). Stress imposed by negative life events has been associated with a greater rate of musculoskeletal injury occurrence (1), which may be due to slowed visual-motor reaction time and a narrowed peripheral visual field (68). Neuromechanical responsiveness also may be adversely affected by fatigue (3) and sleep deprivation (16), either of which may be exacerbated by concussion. Any factor that overloads cognitive processing capacity may increase injury risk by disrupting anticipatory motor responses (22,42,60).

Postconcussion Musculoskeletal Injury Risk Assessment

Preparticipation injury risk screening and postconcussion status assessment for return to sport should involve a multifaceted battery of objective tests of neurocognitive function, postural stability, visual-motor reaction time, and perhaps most importantly, tests that integrate cognitive, visual, somatosensory, and motor function. Although computerized neurocognitive test results may identify athletes who possess elevated risk for musculoskeletal injury (61,66), a more accurate assessment may be derived from clinical tests that quantify complex relationships among interrelated factors, such as visual perception of environmental changes, brain processing of sensory inputs, and rapid generation of appropriate mechanical forces (15,24,25). A computerized neurocognitive test may provide evidence of a deficiency in the speed of information processing, but mouse-click responses to visual stimuli presented on a monitor have no similarity to the nature of the physical responses required in a competitive sport environment (24). Similarly, a physical movement test battery may identify malalignment of body segments or asymmetrical movement patterns, but these are typically assessed in isolation to any of the cognitive or visual challenges of sport. The predictive power of functional tests may increase when they challenge an athlete's capability to rapidly plan and execute a complex motor activation pattern under cognitive or visual stress (25).

High-order cognitive processes responsible for visual stimulus identification, motor response selection, and memory may be particularly vulnerable to the effects of concussions (23,45,55,63), as well as the effects of subconcussive head blows (4,62,65). Subtle changes in brain processing of visual input have been shown to persist in young adults for 6 to 7 yr after having experienced a concussion (45), which could play an important role in elevation of risk for musculoskeletal injury. Reduced speed of response to peripheral visual stimuli after concussion has been documented (8), which may adversely affect anticipatory muscle activation that is heavily reliant on peripheral visual awareness (35,44). Visual-motor reaction time in isolation has been shown to be prospectively associated with injury occurrence (67), but similar to cognitive dual-tasking, the simultaneous use of central vision to perform a cognitive task while executing rapid motor responses to

Table 2.
Methods used for assessment of postconcussion changes in brain structure and function.

Method	Measurement
fMRI	BOLD signal
DTI	FA
	AD
	RD
MRS	Neurometabolite concentrations
TMS + EMG	Intracortical inhibition:
	CSP duration
	Corticospinal excitability:
	MEP amplitude
EEG	ERP amplitude/latency

AD, axial diffusivity; BOLD, Blood oxygen level-dependent; CSP, cortical silent period; DTI, diffusion tensor imaging; EMG, electromyography; ERP, event-related potential; FA, fractional anisotropy; MEP, motor-evoked potential; MRS, magnetic resonance spectroscopy; RD, radial diffusivity.

peripheral visual stimuli appears to be a better indicator of an individual's susceptibility to a sport-related injury (21,69). Simultaneous imposition of cognitive and motor demands may identify subtle deficiencies that would otherwise remain unrecognized (27). If an injury has persisting effects, the imposition of a "cognitive load" typically causes deterioration in the performance of one or both tasks (52,57). Importantly, the speed of reflexive responses to peripheral visual stimuli has been shown to be modifiable through such cognitive-motor dual-task training, possibility reducing concussion risk through improved integration of central and peripheral visual input (69).

Proposed Approaches for Clinical Integration of Concepts and Future Research

Clinical tests of neuromechanical responsiveness are needed to ensure adequate recovery from concussion and reduced musculoskeletal injury risk. One such test is the Erikson flanker test for assessment of high-order cognitive processes relating to rapid stimulus recognition and visual conflict resolution (12,23,45,55). A verbal or motor response is provided to identify the direction indicated by a centrally-located arrow within a 5-arrow visual display that is congruous (*i.e.*, >>>>> or <<<<<) or incongruous (*i.e.*, >><>> or <><><). Performance is assessed by reaction time, overall response accuracy, and comparison of congruent versus incongruent performance values. To illustrate the point that combined cognitive-visual-motor tests may provide more information than isolated component testing, screening data for 66 college football players demonstrated that isolated measures of single-leg postural balance and visual-motor reaction time provided good discrimination between players who reported a history of diagnosed concussion and those who denied such a history with 50% sensitivity, 81% specificity, and an OR of 4.3 (7). However, the results of dual-task tests that included the Erikson flanker test provided a 10-fold increase in discriminatory power for concussion history, with 50% sensitivity, 98% specificity and an OR of 47. Furthermore, suboptimal dual-task visual-motor performance (adjusted for high exposure to game conditions) was found to have a strong prospective association with the occurrence of a core or lower-extremity sprain or strain in the same cohort of college football players (HR, 5.3), which suggests that such testing has strong value (21).

Testing that incorporates reactive whole-body movement responses may provide further information to better quantify injury risk (2). A recent cohort study that involved 71 college football players identified a strong association between asymmetrical whole-body reactive movement responses and history of a diagnosed concussion (18). The TRAZER Sports Stimulator (Traq Global Ltd, Westlake, OH) was used to quantify side-shuffling responses to visual targets that appear on a large monitor in a random sequence of right and left positions. Side to side differences in reaction time (≥16%), movement velocity (≥6%), and deceleration (≥7%) effectively discriminated players who reported a previously diagnosed concussion (n = 22) from those who denied such a history (n = 49). Having any two of the three indicators of asymmetrical performance provided strong discrimination between the players (86% sensitivity; 61% specificity; OR, 10.00). This association between concussion history and asymmetrical side-to-side movement capabilities, may be secondary to disruption of white matter integrity in the corpus callosum associated with concussion that degrades inter-hemispheric connectivity (39,43,48). Collectively, the results of these preliminary studies suggest that long-lasting effects of concussion that elevate risk for musculoskeletal injury may be identified through specific dual-task screening tests of cognitive function, balance, and visual-motor reaction time.

The speed of motor responses to visual stimuli has been shown to be modifiable through dual-task training on the Dynavision D2™ System (Dynavision International, West Chester, OH) that involved simultaneous manual contact with illuminated targets and oral recitation of text (67). Follow-up studies have demonstrated that this type of dual-task training enhances responsiveness to targets in the peripheral visual field (10), which appeared to reduce risk for subsequent injury occurrence (69). Other approaches that may enhance neuromechanical responsiveness include balance training with cognitive or visual challenges, use of stroboscopic glasses to partially occlude visual feedback during task performance, and imposition of fatigue or other stressful demands during the performance of sport-specific training activities.

Conclusions

Concussions, and possibly subconcussive head blows, may have cumulative subclinical effects that persist for a much longer period than previously believed (4,63). Current postconcussion clinical assessments may not be adequate to identify a diminished capacity for neuromechanical responsiveness to rapidly changing demands imposed by a competitive sport environment, which may be a key factor that elevates musculoskeletal injury risk. Clinical tests for quantification of neuromechanical responsiveness offer the potential for identification of athletes who are not ready to return to sport participation, but large-scale studies are needed to confirm the reported findings of the preliminary studies. Similarly, there appears to be strong potential for injury risk reduction through neuromechanical responsiveness training, but more research is needed to document a lowered injury incidence rate, and to quantify the training dose-response relationship. Most importantly, we advise clinicians to be cognizant of the fact that subtle neuromechanical deficiencies are likely to persist beyond resolution of overt concussion symptoms. We anticipate continued advancements in concussion management that will be based on strong research evidence to support specific clinical tests and interventions, as well as quantitative performance standards for safe return to sport participation.

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