REVIEW OPEN ACCESS

Sport-Related Concussion

A Cognitive Perspective

Harry Hallock, PhD, Maron Mantwill, MSc, Peter Vajkoczy, MD, Bernd Wolfarth, MD, Claus Reinsberger, MD, Amit Lampit, PhD, and Carsten Finke, MD

Correspondence
Dr. Finke
carsten.finke@charite.de

Neurology: Clinical Practice 2023;13:e200123. doi:10.1212/CPJ.0000000000200123

Abstract

Purpose of Review

The incidence of sport-related concussion (SRC) has been increasing in different sports and its impact on long-term cognitive function is increasingly recognized. In this study, we review the epidemiology, neuropathophysiology, clinical symptoms, and long-term consequences of SRC with a specific focus on cognition.

Recent Findings

Repeated concussions are associated with an increased risk of several neurologic diseases and long-term cognitive deficits. To improve cognitive outcomes in athletes with SRC, standardized guidelines for the assessment and management of SRC are vital. However, current concussion management guidelines lack procedures for rehabilitating acute and long-term cognitive symptoms.

Summary

Increased awareness for the management and rehabilitation of cognitive symptoms in SRC is needed in all clinical neurologists treating professional and amateur athletes. We propose cognitive training as a prehabilitation tool to alleviate the severity of cognitive symptoms and as a rehabilitative tool to improve cognitive recovery postinjury.

Cognitive impairment is a common sequela of acquired brain injury and a growing area of interest for the management of sport-related concussion (SRC). Challenges in managing cognitive impairment after SRC arise not only from the heterogeneity of neural injuries and resulting effects on cognition but also from the implications these may have within individual circumstances, goals, and prior cognitive abilities. These natural complexities are further compounded by challenges in measurement and monitoring, given the temporal dynamics of impact and measurement remain largely unclear.

In this article, we provide a state-of-the-art overview of current evidence and research directions in SRC, with a particular emphasis on the link between injury, cognition, and functional sequalae. We further introduce cognitive training as a potential mitigation and prevention measure and its potential role to improve field performance by targeting cognitive skills that underpin performance of different sports. We review potential mechanisms, practical considerations, and areas for further mechanistic and clinical research.

Definition and Epidemiology

Concussion, defined as a temporary disturbance of brain function, is a form of traumatic brain injury (TBI). TBI is listed as an intracranial injury in the *International Classification of Diseases,*

Berlin School of Mind and Brain (HH, MM, AL, CF), Humboldt-Universität zu Berlin; Departments of Neurology (HH, MM, AL, CF) and Neurosurgery (PV) and of Sports Medicine (BW), Charité Universitätsmedizin, Berlin; Institute of Sports Medicine (CR), University of Paderborn, Germany; and Department of Psychiatry (AL), University of Melbourne, Parkville, Australia.

Funding information and disclosures are provided at the end of the article. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

10th Revision (ICD-10) and can be mild, moderate, or severe. In addition to concussion, cerebral edema, diffuse and focal brain injury, and various forms of traumatic hemorrhage fall under TBI. Concussion is the mildest and most common variant of TBI, but given the heterogeneity in the literature regarding definitions, the term concussion is often interchangeably used with the term mild TBI; because the differences in injury are vague, there is debate on whether concussion and mild TBI are unique constructs.[1] This article will therefore use the term concussion to also represent mild TBI. The term TBI will be used when referring to all levels of TBI. Globally, 50 million people experience TBI annually[2]; however, given that many cases may receive outpatient treatment, are undiagnosed, untreated, or not the primary injury, the true prevalence is likely considerably greater.[3] Forward estimates suggest that half of the global population will have at least 1 TBI over their lifespan.[2] SRCs occur specifically due to biomechanical forces induced on the body or head, which in turn lead to acceleration, deceleration, and rotational forces on the brain. Repetitive head injuries (RHIs), which may follow similar mechanisms as concussion but do not meet the clinical criteria, are also very common in sport and can be less symptomatic or even asymptomatic. Contrary to common belief, more than 90% of concussions do not involve a loss of consciousness, which, combined with the absence of objective biomarkers, can make them challenging to diagnose.

SRCs have become such a serious concern that an international workgroup of leading researchers and clinicians was formed to guide policy, clinical practice, and future research.[1] Consensus statements[1] by this workgroup and the resulting awareness have led many sports to introduce new rules and mandatory requirements around player safety, assessment, management, and monitoring of concussion injuries and protective equipment.

Sports-Related Concussion Epidemiology

Beyond increasing public awareness, the statistics of SRCs tell an alarming story. Estimates suggest that 3.8 million concussions occur in the United States annually because of sport and recreation with up to 50% of concussions going unreported.[4] The incidence of concussion in sport has been steadily increasing over previous decades, with 2 comprehensive studies of collegiate athletes in North America showing an increase in concussion rates from 1.7 per 10,000 athlete exposures (1 athlete exposure is defined as 1 athlete participating in 1 practice or game) in 1988–1989 to 3.4 in 2003–2004[5] and to 4.47 in the period between 2009 and 2014.[6] Similar incidence trends are also found in high school athletes[7] and school children.[8] For comparison, the incidence of concussion injuries was almost double that of the seemingly more common anterior cruciate ligament injuries.[5] Exacerbating this is the fact that 35%–62% of concussion injuries in high school and collegiate athletes are not reported.[9] In collegiate athletes, the greatest number of concussion injuries were observed in men's American football, while the highest rates of concussion were noted in men and women's ice hockey and women's soccer.[5,6] In 2017, approximately 2.5 million American high school students self-reported having at least 1 physical activity–related or SRC in the preceding 12 months, while 1 million reported 2 or more.[10] At the elite level, concussions are more common, with an analysis of head injuries during the 2012–2013 and 2013–2014 National Football League (NFL) seasons illustrating a concussion rate of 66.1 (per 10,000 athlete exposures).[11]

Neuropathophysiology, Clinical Symptoms, and Long-term Consequences

Neuropathophysiology

SRCs are heterogeneous because severity and location of impact, age, and sex play key roles in the subsequent neuropathophysiology and clinical symptoms. Concussion generally results in diffuse injuries that can be difficult to detect and generally refer to axonal injury, but also include vascular injury and brain edema.[12,13] In a primary concussion phase, diffuse axonal injury is the most common form of injury and results from acceleration, deceleration, and rotational forces that cause the differently weighted brain tissues to move at different rates resulting in the shearing of axons.[12] In a secondary phase, diffuse axonal injury leads to a series of metabolic disturbances known as the hypermetabolic or neurometabolic cascade of concussion.[14] This is characterized by an influx of calcium and sodium ions and efflux of potassium ions, leading to an increased activation of the sodium-potassium pump and thus hyperglycolysis.[15] In addition, shearing of endothelial cells in small vessels can cause reduced cerebral blood flow,[12] leading to a mismatch between glucose supply and demand[15] and eventually an acute failure in neuronal function.[12] The aforementioned ionic dysregulation also results in the release of excitatory amino acids such as glutamate that binds to NMDA receptors and causes further depolarization. Concurrently, there is a calcium influx into mitochondria, which leads to oxidative stress and exacerbates the disruption in cellular energy supply.[16] Furthermore, excess intracellular calcium can also phosphorylate neurofilament microstructures leading to further axonal damage and disrupted axonal transport, continuing the cycle.[15]

Using a combination of rodent and human studies, links have been proposed between pathophysiologic changes and postconcussive symptoms. Cognitive impairment and slower speed of processing could be a consequence of axonal injury and altered neurotransmission; increased sensitivity to repeated injuries could be a result of the glycolysis imbalance.[15] In the long term, dysfunctional neurotransmission in NMDA receptors in the hippocampus could result in memory deficits and cognitive deterioration.[17] Recent hypotheses on cognitive dysfunction after concussion suggest that diffuse axonal injury in the hippocampus leads to persistent deficits in intrahippocampal synchronization and memory consolidation, to reduced input to hippocampal structures and diffuse damage to

long-range connections between the hippocampus and other brain regions such as the prefrontal cortex and amygdala,[18] explaining some of the most common postconcussive symptoms, such as impaired memory and attention deficits. However, the outlined neuropathophysiological cascade and its proposed links to postconcussive symptoms should be interpreted with caution. First, most pathophysiologic hypotheses are based on animal models that usually reflect moderate-to-severe TBI. Second, given the heterogeneity of concussion, the level of contribution of specific damage cascades to pathophysiology in individual cases is difficult to determine.

SRC Symptoms

Similar to its neuropathophysiology, symptoms after SRC can also be rather heterogeneous and difficult to separate from depressive mood or general fatigue induced by sport. The prevalence of certain symptoms after concussion in comparison with moderate-to-severe TBI is currently debated. Nonetheless, there is consensus[1] that symptoms of SRC may include 1 or more of the following:

1. Headache
2. Confusion
3. Disorientation
4. Emotional lability or irritability
5. Somnolence or drowsiness
6. Cognitive impairment
7. Gait unsteadiness
8. Amnesia
9. Loss of consciousness
10. Sleep disturbances
11. Vestibular-ocular impairments

When present, cognitive symptoms most commonly manifest as impairments in processing speed, attention, memory, and executive functions.[19,20] The duration of symptom resolution after SRC varies, but many studies illustrate cognitive symptoms resolve largely within a few days[21,22] to 1 month.[23] However, some observations indicate persistence of cognitive impairments beyond this interval, with a recent review suggesting approximately 50% of individuals who experience a single concussion (not specifically SRC) still exhibit cognitive impairment 3 or more months postinjury.[20] There is also evidence that RHI may affect cognition; increased heading in soccer players has been associated with lower memory scores,[24] and high school American football players, despite not being diagnosed with concussion, have been shown to exhibit cognitive impairment.[25] However, 2 well-sized systematic reviews of investigating RHI in soccer have concluded that either more research is needed[26] or that heading does not lead to adverse outcomes.[27]

Long-term Cognitive Effects

Symptom resolution may not mean complete cognitive recovery because neuropsychological testing may show persistent cognitive deficits in 80%–90% of individuals with typical recovery.[4] Longer recovery duration could be categorized as postconcussion syndrome (PCS), but whether PCS is a different to long-term effects of concussion remains unclear.[28] Studies on retired American football players with a history of head injuries have illustrated that recurrent concussion lead to an increased risk of late-life cognitive decline[29,30] and depression.[31] Furthermore, former athletes who sustained their last SRC more than 3 decades earlier were shown to have lower performance on tests of episodic memory and response inhibition.[32]

Concussion is a risk factor for several different diseases that affect cognition. There is clear evidence that previous concussion is the greatest risk factor for future concussion injuries,[33] with some studies illustrating an almost 6-fold increase in one's relative risk.[34] This could be caused by each subsequent concussion potentially reducing the level of impact needed to induce neuropathophysiological changes and clinical symptoms. There is increasing evidence suggesting that repetitive concussion can lead to chronic traumatic encephalopathy (CTE), a progressive neurodegenerative disease.[35] However, recent studies suggests that CTE may be linked more closely to head injury, irrespective of concussive symptoms (i.e., subconcussive damage).[36] CTE can result in perivascular tau pathology (e.g., neurofibrillary tangles and hyperphosphorylation), diffuse axonal injury, and cerebral atrophy. Clinical symptoms can include memory and executive function impairments, parkinsonism, impulsive behavior, emotional instability, apathy, depression, and suicidality.[35,37] A recent study found that from 202 brains of deceased American football players of all ages and levels examined, 87% had CTE neuropathology, including 110 from 111 former NFL players[37]. Despite the selection bias given the convenience sampling of donated brains, the results show a clear link between American football and CTE, likely resulting from repetitive head trauma. Furthermore, repeated concussions have been linked to an increased risk of multiple sclerosis,[38] Alzheimer disease,[39] stroke,[40] Parkinson disease,[41] and epilepsy.[42] Given the potential long-term consequences, the assessment and management of SRC is paramount, especially given that athletes are generally at a greater risk of repeated injury than nonathletes.

Assessment, Management, and Rehabilitation

Assessment—Neuropsychological and Behavioral

In addition to neurologic examination, patient medical history, and symptom questionaries, ancillary methods such as motor testing, neuropsychological testing, neuroimaging, and vestibular-ocular testing are used to aid TBI diagnosis. Vestibular-ocular symptoms such as dizziness and blurred vision are associated with a greater risk of protracted recovery.[43] Vestibular-ocular tests such as the Vestibular/Ocular Motor Screening (VOMS)[44] assess vestibular-ocular dysfunction by testing domains such as visual motion sensitivity, rapid eye movements, and smooth visual pursuits. While VOMS does not provoke symptoms in healthy individuals,[45] athletes with concussions will likely experience symptoms such as dizziness or nausea.

A number of sports have improved their safety guidelines and furthermore introduced compulsory baseline testing such as the Sport Concussion Assessment Tool 5 (SCAT-5). The SCAT-5 is currently the most commonly used clinical test for concussion, having been implemented by many professional bodies such as International Association Football Federation (FIFA) NFL, and World Rugby. Although not necessary for effectiveness, many teams administer a preseason baseline of the SCAT-5 for later postinjury comparison. The SCAT-5 combines athlete self-evaluation of symptoms with objective cognitive and neurologic screening administered by a healthcare professional. The self-evaluation can be disadvantageous because it could result in athletes underreporting their symptoms and thus misleading healthcare professionals. Furthermore, there have been reports that the orientation questions used (Maddock questions) [46] are sometimes memorized by athletes. Perhaps the largest limitation of SCAT-5 is that—similar to many standardized assessments for concussion—it currently cannot be effectively used as an on-field or sideline test for removal from activity. This is because many sports have strict rules about stoppage and substitution, which does not allow for the SCAT-5 to be conducted and scored in its entirety. For instance, in soccer, stoppage time is limited to 3 minutes and players cannot be substituted off for assessment.[47] Rule changes allowing for temporary concussion substitutions during assessment, and the introduction of technical aids replaying videos of a potential concussive event are being discussed to facilitate the SCAT-5–based diagnostic process.[48,49] Indeed, technical video and communication aids have already been implemented at the Union of European Football Associations (UEFA) European Championship 2020 and in the German professional football league.[48,50] Until changes to stoppage time or substitution guidelines have been fully implemented, SCAT-5 and similar standardized tests are better served to assist in the diagnosis of concussion and return-to-play decisions, but not necessarily in removal from activity decisions. There are a number of quicker commercial tests and products that claim to assist in the latter, but many of them can only focus on 1 aspect of concussion, e.g., vestibulo-ocular function, and thus may have limited sensitivity and specificity.[51]

Assessment—Imaging

There is an increasing use of brain imaging in the assessment of concussion. In severe cases, CT can rapidly be administered to rule out intracranial hemorrhage. However, given the lower sensitivity of CT for other concussion-related brain changes, MRI remains the preferred imaging modality, especially in the subacute and chronic phases, where recovery is worse than expected or neurologic deficits remain.[2,52] In research settings, advanced MRI techniques such as susceptibility-weighted imaging (SWI) and diffusion tensor imaging (DTI) are increasingly used, given their greater sensitivity to structural change. SWI has been shown to be specifically sensitive to microhemorrhage, a marker of white matter injury after TBI.[19] DTI shows promise as a potential diagnostic tool across varying TBI severities, with mean diffusivity (MD) being more sensitive to mild injuries such as SRC and fractional anisotropy to more severe injuries.[53] MD was significantly increased in acute and postacute phases of mild TBI, even when athletes showed no more symptoms and was associated with worse clinical outcome.[54] Using different imaging modalities such as functional imaging, magnetic resonance spectroscopy, and PET, recent studies have shown that SRC can lead to altered functional connectivity between brain regions,[55] neurometabolic changes,[56] and neuroinflammation.[57] While these advanced neuroimaging techniques show higher sensitivity than standard MRI and CT in detecting a multitude of acute, subacute, and chronic structural and functional brain changes and could have prognostic value in the future, their translation into clinical practice remains challenging, given the availability of appropriate equipment, temporal constraints of clinical environments, and a lack of standardization.[54] Given the extensive literature of imaging studies in concussion, an in-depth discussion of the topic is not within the scope of this article. Please see Koerte et al.[58] for a comprehensive review.

Assessment—Specimen Biomarkers

Blood biomarkers have received considerable interest recently, specifically to guide injury management and return-to-play decisions. Higher plasma tau levels after concussion (less than 6 hours postconcussion) have been linked to longer return-to-play time and correlate with the duration of symptoms.[59,60] Neurofilament light has been shown to be elevated in American football players after a season, both compared with their own baseline and noncontact athletes.[e1] Furthermore, neurofilament light has been used to identify individuals at risk of persistent PCS and similar to plasma tau, has the potential to assist in return-to-play decisions.[e2] A number of other measures, such as neurogranin, oligodendrocyte myelin glycoprotein, synuclein beta,[e3] high-sensitivity C-reactive protein,[e4] and S100 calcium-binding protein B,[e5] have likewise been proposed as potential blood-based biomarkers for concussion. Subacute biomarkers such as neurofilament light, as well as autoantibody biomarkers, and chronic biomarkers such as plasma tau are investigated for their potential in tracking disease progression, long-term consequences of TBI, and guide return-to-play decisions. Protein biomarkers such as glial fibrillary acidic protein and ubiquitin C-terminal hydrolase-L1 are closest to broader clinical implementation, with S100 calcium-binding protein B already being implemented in Scandinavian guidelines for patient selection for CT imaging after TBI.[2] Nevertheless, using serum biomarkers to detect concussion may be difficult because their low specificity could result in false-positives.[47] With advances in scientific methodology and technology, new pitch-side diagnostics are being investigated. Recently, specific MicroRNAs (miR-425-5p and miR-502) were discovered to be downregulated postconcussion.[e6] As a result, researchers are collecting urine and saliva samples from rugby players with suspected concussion, both during suspected concussion and throughout their recovery, with the aim of improving on-field diagnostics and return-to-play decisions.[e7]

Assessment—Medical Devices

There has also been promising research into the biomechanics and physics of collisions, with rugby union players wearing

accelerometers to measure the *g*-force of impact as a potential measure of severity of concussion.[e8] However, it has been argued that most forms of head impact measurement systems such as accelerometers, telemetry, and sensors are not sensitive or specific enough to detect concussion.[e9] The United States Food and Drug Administration (FDA) has approved a number of medical devices for the assessment of head injury using a range of different approaches, including analysis of blood samples for known protein biomarkers of head injury, computerized neuropsychological tests and devices based on portable imaging modalities (EEG and near infrared spectroscopy) and eye tracking. There are also a number of companies marketing unapproved "medical devices" for the diagnosis, treatment, and management of concussion, and the FDA has recently had to release a statement warning consumers against these and that no medical device can assess head injuries without the presence of a healthcare professional.[61]

While combined neurologic, neuropsychological, and vestibular-ocular testing are still the primary methods for assessing potential SRC, the potential of specimen biomarkers and the creation of new highly mobile medical devices illustrate a new era that could enable faster and more precise pitch-side assessments of SRC. Ultimately, these different methods could be combined to produce more sensitive and objective measures for removal from activity decisions, SRC diagnoses, and return-to-play decisions.

Management

Given that concussion may often present with symptoms that do not seem to warrant medical intervention, many individuals who have sustained a concussion do not see a medical professional. However, as outlined earlier, concussion can result in a series of underlying neuropathophysiological changes, which, in some cases, can lead to long-term cognitive, behavioral, and psychosocial impairment. For moderate and severe TBI, management and treatment after acute care generally revolves around occupational, speech, physical, and psychological rehabilitation and improving independent functioning. However, cognitive rehabilitation is sometimes overshadowed by rehabilitation of these other domains. For SRC, most sporting bodies now follow specific management guidelines published by the Concussion in Sport Group[1] (Table). After an initial 24–48 hours of rest, the concussed player can begin the graded return to sport. The guidelines suggest a minimum of 24 hours between each stage, which in theory could result in a concussed athlete returning to play within a week.

These guidelines reduce the immediate likelihood of sustaining an additional concussion, which is vital given that further concussion during this acute phase can exacerbate the neuropathophysiological cascade. As described earlier, the immediate neuropathophysiological consequences of a concussion are an uncoupling of cerebral blood flow and glucose metabolism, making cells particularly vulnerable during recovery. Any further dysregulation of energy demand due to additional injury in the immediate aftermath of a concussion might render the recovery of an injured cell impossible and instead lead to cell death. Other sources of potentially increased vulnerability such as intracellular Ca^{2+} accumulation or alterations in NMDA receptor composition can last up to 4 weeks postconcussion,[17] making graded return-to-sports guidelines imperative. The guidelines can also mitigate short-term physical symptoms, but one could argue they fail to holistically rehabilitate the player. For example, rehabilitation of a torn muscle fiber will involve stretching, weight-bearing, deep-tissue massage, or dry needling among other activities. Thus, the injured muscle is actively rehabilitated before return to play. Furthermore, these activities are not restricted to postinjury and are often suggested for injury prevention as prehabilitation. By contrast, this form of rehabilitation is rarely followed for cognition after SRC because a disproportionate emphasis is placed on motor and vestibular-ocular symptoms. This is also noted with moderate and severe TBI in a clinical setting because more focus is placed on rehabilitation of physical abilities over cognitive and psychosocial symptoms. However, this approach is changing with recent clinical guidelines, suggesting cognitive remediation should play a larger role,[2] especially restorative treatments and compensatory strategies.[e10]

Perhaps the cornerstone of current concussion management, the recommendation of cognitive and physical rest is now being questioned. While there is broad evidence supporting rest, some experts are in agreement that there is inadequate empirical

Table Graded Return-to-Sport Guidelines From the Fifth International Conference on Concussion[1]

Stage	Aim	Activity	Goal of each step
1	Symptom-limited activity	Daily activities that do not provoke symptoms	Gradual reintroduction of work/school activities
2	Light aerobic exercise	Walking or stationary cycling at slow to medium pace. No resistance training	Increase heart rate
3	Sport-specific exercise	Running or skating drills. No head impact activities	Add movement
4	Noncontact training drills	Harder training drills, e.g., passing drills. May start progressive resistance training	Exercise, coordination, and increased thinking
5	Full contact practice	Following medical clearance, participate in normal training activities	Restore confidence and assess functional skills by coaching staff
6	Return to sport	Normal game play	

evidence for the effectiveness of prescribed physical and cognitive rest and furthermore that active rehabilitation may improve symptom recovery.[e11] Firsthand investigations have shown that cognitive rest is not significantly associated with time to symptom recovery[e12] and in some cases too much rest could prolong recovery.[e13,e14] Individualized subthreshold aerobic exercises following SRC have been shown to speed recovery time compared with stretching,[e15] a result also supported by a systematic review on rest after SRC.[e16] This raises the question as to whether subthreshold cognitive exercise could have a similar effect. Of interest, there is evidence showing that moderate activity (both physical and cognitive) leads to optimal neurocognitive outcomes postconcussion when compared with high activity and no activity.[e17] More research, specifically RCTs, is needed to investigate the effects of immediate postconcussive cognitive activity on neurocognitive outcomes. Other factors beyond activity intensity, such as type of cognitive activity, whether it is combined with physical activity, the duration of the activity, and when it is instigated after concussion, could all hypothetically have an effect on neurocognitive outcomes. Ultimately, while rest may still play an important factor in recovery, perhaps it should not be a blanket recommendation but instead a guideline that could be superseded by cognitive activity to a level of intensity as tolerated by the patient.

Rehabilitation—Cognitive Training

Cognitive training is a general term that covers an array of structured interventions aiming to enhance or maintain cognitive performance using repeated practice of cognitively challenging tasks.[e18] It is one of the 3 overarching types of cognition-focused interventions (interchangeably referred to in the literature as neuropsychological, neurocognitive, or simply cognitive interventions), alongside cognitive rehabilitation and cognitive stimulation (for a comprehensive review, see Wilson[e19]). Cognitive rehabilitation focuses on the cognitive underpinning of individual functional deficits and is more individualized in nature. Cognitive stimulation is a more generic approach that aims to create opportunities for cognitively engaging activities, which is much more common in populations with severe impairments (e.g., people with dementia) than those with potentially reversable cognitive impairment. The overarching idea of cognitive training is to induce experience-dependent neuroplasticity through structured and controlled learning events, in contrast to the more sporadic exposure to cognitive challenges experienced with cognitive rehabilitation or stimulation.[e20] Training will often combine explicit teaching of compensatory strategies alongside restorative drill and practice exercises and typically delivered to people with brain injury as part of a comprehensive rehabilitation program.[e19]

If properly targeted, such neural changes could build over time and lead to reduced cognitive dysfunction (rehabilitation), lead to a reduced risk of severe cognitive dysfunction after trauma (prerehabilitation), or improve cognitive functioning, which could translate to superior performance in the field (enhancement). Similar to how professional athletes undertake preseason strength and conditioning training to increase sporting performance and lower the risk of injury throughout the season (i.e., prerehabilitation),[e21,e22] cognitive training has the potential to work in a similar capacity, by improving sporting performance,[e23] reducing the risk of injury, and supporting rehabilitation.

Despite robust evidence for its efficacy on cognitive performance in healthy individuals[e24,e25] and those with acquired brain injury[e26,e27] or other cognitive disorders,[e28,e29] the mechanisms underlying cognitive training effects—and therefore their clinical implications—remain largely unclear. Despite some claims that cognitive training effects do not extend beyond trained tasks,[e30] the bulk of the evidence points to robust efficacy on untrained cognitive tasks within the same domain (i.e., near transfer) and limited "far" transfer to domains not targeted by the intervention.[e18,e25,e31] Rehabilitation interventions based on explicit teaching of cognitive strategies would tend to be task specific and therefore not directly relevant or efficacious for other cognitive domains.[e19,e32] There is even less clarity about the mechanisms underlying process-based training (predominantly computerized cognitive training) because these may simply improve problem-solving efficiency without changing cognitive capacity, a notion partially supported by neuroimaging studies, although the link between training content and its associated neural effects are not well understood.[e18,e33]

Although solid mechanistic theory is still lacking, several important practical implications can already be drawn. Perhaps most critical is that cognitive training must be targeted at the cognitive skills that are most important for the individual and their specialization. For example, recent studies in soccer players[e34] and lacrosse players[e35] have shown that cognitive training can improve sport-specific performance, with soccer players improving on-field passing and lacrosse players improving shooting accuracy. These studies tailored the intervention to specific cognitive skills believed to underpin performance in the respective sports, such as field of view, processing speed, or visuospatial skills, whereas other unrelated cognitive abilities (e.g., verbal memory) are less likely to be relevant to on-field performance. Similarly, the content and approach in postinjury rehabilitation must take into account individual areas of impairment, individual goals, and critical abilities required to return to activity.[e19]

Planning cognitive prehabilitation may be more complex because the impact of injury is less predictable. In populations such as in surgical patients, prehabilitation interventions tend to be comprehensive and multidomain, i.e., training a wide array of processes targeting global cognitive performance and requiring larger doses and exposure than more targeted approaches.[e36] Theoretically, cognitive prehabilitation can leverage on cognitive reserve, commonly defined as the adaptivity of cognitive abilities and therefore their potential susceptibility to injury.[e37] Cognitive reserve has been used to explain why individuals with similar brain pathology may present significant differences in cognitive performance and is often linked to lifestyle exposures such as educational attainment, occupation, physical exercise, and general cognitive abilities before brain injury.[e37] In light

of these interindividual differences, studies of rehabilitative methods will need to carefully balance their study populations to provide actionable results.

Because high cognitive reserve in the form of higher educational attainment has been associated with less cognitive impairment after TBI,[e38,e39] undertaking cognitive training as prehabilitation, or as part of general training and conditioning, may have the potential to reduce the severity of cognitive side effects.[e23] Indeed, cognitive training has already been shown to be effective in improving cognition in postacute TBI,[e26] with several feasibility studies also highlighting the potential of cognitive training as a therapeutic tool in patients with moderate-to-severe TBI with varying time since injury.[e40,e41] Whether the effects are similar during the acute phase (i.e., immediately after injury) is still unknown. Given that there is now agreement that active rehabilitation could improve symptom recovery, future research should focus on investigating the potential of CCT to alleviate cognitive symptoms immediately after SRC.

Conclusion

The incidence of SRC has become a major concern for many sporting bodies and athletes. Given its heterogeneity, research in the field is mainly targeted toward making diagnoses methods more sensitive and return-to-play decisions more objective. SRC can often lead to cognitive symptoms, which, in some cases, develop into long-term impairments. Currently, concussion management guidelines lack procedures for rehabilitating these acute or long-term cognitive symptoms. Cognitive training could be an added option because it has the potential to improve sport-specific performance, reduce the risk of cognitive symptoms postconcussion, and rehabilitate cognitive impairment. However, more research, specifically randomized controlled trials, are needed to investigate the effectiveness and feasibility of cognitive training for SRC.

Study Funding

This review was funded by the Bundesinstitut für Sportwissenschaft (Aktenzeichen: ZMVI4-070106/19–20).

Disclosure

H. Hallock was using ImPACT Applications, Inc then as a paying customer for separate ongoing research; C. Reinsberger receives scientific funding by the Federal Institute of Sports Sciences (Germany) and the Heinz Nixdorf Westfalian Foundation. He examines and treats patients with sport-related concussion on a regular basis in an outpatient concussion clinic. He is a member of the medical committee of the German Football association (DFB) and provides counselling to the Union of European Football Associations (UEFA); C. Finke received scientific funding by the Federal Institute of Sports Sciences (Germany). C. Finke was using ImPACT Applications, Inc then as a paying customer for separate ongoing research. The other authors report no relevant disclosures. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

Publication History

Received by *Neurology: Clinical Practice* May 6, 2022. Accepted in final form November 3, 2022. Submitted and externally peer reviewed. The handling editor was Associate Editor Jack W. Tsao, MD, DPhil, FAAN.

Appendix Authors

Name	Location	Contribution
Harry Hallock, PhD	Berlin School of Mind and Brain, Humboldt-Universität zu Berlin, Berlin, Germany; Department of Neurology, Charité Universitätsmedizin, Berlin, Germany	Drafting/revision of the article for content, including medical writing for content; major role in the acquisition of data; study concept or design; analysis or interpretation of data; and was using ImPACT applications, Inc then as a paying customer for separate ongoing research
Maron Mantwill, MSc	Berlin School of Mind and Brain, Humboldt-Universität zu Berlin, Berlin, Germany; Department of Neurology, Charité Universitätsmedizin, Berlin, Germany	Drafting/revision of the article for content, including medical writing for content; major role in the acquisition of data; study concept or design; and analysis or interpretation of data
Peter Vajkoczy, MD	Department of Neurosurgery, Charité Universitätsmedizin, Berlin, Germany	Study concept or design
Bernd Wolfarth, MD	Department of Sports Medicine, Charité Universitätsmedizin, Berlin, Germany	Study concept or design
Claus Reinsberger, MD	Institute of Sports Medicine, University of Paderborn, Paderborn, Germany	Study concept or design
Amit Lampit, PhD	Berlin School of Mind and Brain, Humboldt-Universität zu Berlin, Berlin, Germany; Department of Neurology, Charité Universitätsmedizin, Berlin, Germany; Department of Psychiatry, University of Melbourne, Parkville, Australia	Drafting/revision of the article for content, including medical writing for content; major role in the acquisition of data; study concept or design; and analysis or interpretation of data
Carsten Finke, MD	Berlin School of Mind and Brain, Humboldt-Universität zu Berlin, Berlin, Germany; Department of Neurology, Charité Universitätsmedizin, Berlin, Germany	Drafting/revision of the article for content, including medical writing for content; major role in the acquisition of data; study concept or design; and analysis or interpretation of data

References

1. McCrory P, Meeuwisse W, Dvořák J, et al. Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin. *Br J Sports Med.* 2017;51(11):838-847. doi. 10.1136/bjsports-2017-097699.
2. Maas AIR, Menon DK, Adelson PD, et al. Traumatic brain injury: integrated approaches to improve prevention, clinical care, and research. *Lancet Neurol.* 2017; 16(12):987-1048. doi. 10.1016/S1474-4422(17)30371-X.
3. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil.* 2006;21(5):375-378. doi. 10.1097/00001199-200609000-00001.
4. Harmon KG, Drezner JA, Gammons M, et al. American Medical Society for Sports Medicine position statement: concussion in sport. *Br J Sports Med.* 2013;47(1):15-26. doi. 10.1136/bjsports-2012-091941.

5. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train.* 2007; 42(2):311-319.
6. Zuckerman SL, Kerr ZY, Yengo-Kahn A, Wasserman E, Covassin T, Solomon GS. Epidemiology of sports-related concussion in NCAA athletes from 2009-2010 to 2013-2014: incidence, recurrence, and mechanisms. *Am J Sports Med.* 2015;43(11): 2654-2662. doi. 10.1177/0363546515599634.
7. Schallmo MS, Weiner JA, Hsu WK. Sport and sex-specific reporting trends in the epidemiology of concussions sustained by high school athletes. *J Bone Joint Surg Am.* 2017;99(15):1314-1320. doi. 10.2106/JBJS.16.01573.
8. Pfister T, Pfister K, Hagel B, Ghali WA, Ronksley PE. The incidence of concussion in youth sports: a systematic review and meta-analysis. *Br J Sports Med.* 2016;50(5): 292-297. doi. 10.1136/bjsports-2015-094978.
9. Kerr ZY, Register-Mihalik JK, Marshall SW, Evenson KR, Mihalik JP, Guskiewicz KM. Disclosure and non-disclosure of concussion and concussion symptoms in athletes: review and application of the socio-ecological framework. *Brain Inj.* 2014;28(8): 1009-1021. doi. 10.3109/02699052.2014.904049.
10. DePadilla L, Miller GF, Jones SE, Peterson AB, Breiding MJ. Self-reported concussions from playing a sport or being physically active among high school students—United States, 2017. *MMWR Morb Mortal Wkly Rep.* 2018;67(24):682-685. doi. 10.15585/mmwr.mm6724a3.
11. Nathanson JT, Connolly JG, Yuk F, et al. Concussion incidence in professional football: position-specific analysis with use of a novel metric. *Orthopaedic J Sports Med.* 2016;4(1):232596711562262. doi. 10.1177/2325967115622621.
12. Blennow K, Hardy J, Zetterberg H. The neuropathology and neurobiology of traumatic brain injury. *Neuron.* 2012;76(5):886-899. doi. 10.1016/j.neuron.2012.11.021.
13. Mckee AC, Daneshvar DH. The neuropathology of traumatic brain injury. *Handbook Clin Neurol.* 2015;127:45-66. doi. 10.1016/B978-0-444-52892-6.00004-0.
14. McKee AC, Daneshvar DH, Alvarez VE, Stein TD. The neuropathology of sport. *Acta Neuropathol.* 2014;127(1):29-51. doi. 10.1007/s00401-013-1230-6.
15. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. *Neurosurgery.* 2014;75(suppl 4):S24-S33. doi. 10.1227/NEU.0000000000000505.
16. Signoretti S, Lazzarino G, Tavazzi B, Vagnozzi R. The pathophysiology of concussion. *PM R.* 2011;3:S359-S368. doi. 10.1016/j.pmrj.2011.07.018.
17. Giza CC, Hovda DA. The neurometabolic cascade of concussion. *J Athl Train.* 2001; 36(3):228-235.
18. Wolf JA, Koch PF. Disruption of network synchrony and cognitive dysfunction after traumatic brain injury. *Front Syst Neurosci.* 2016;10:43. doi. 10.3389/fnsys.2016.00043.
19. Sharp DJ, Jenkins PO. Concussion is confusing us all. *Pract Neurol.* 2015;15(3): 172-186. doi. 10.1136/practneurol-2015-001087.
20. McInnes K, Friesen CL, MacKenzie DE, Westwood DA, Boe SG. Mild Traumatic Brain Injury (mTBI) and chronic cognitive impairment: a scoping review. *PLoS One.* 2017;12(4):e0174847. doi. 10.1371/journal.pone.0174847.
21. McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA concussion study. *JAMA.* 2003;290(19):2556. doi. 10.1001/jama.290.19.2556.
22. Bleiberg J, Cernich AN, Cameron K, et al. Duration of cognitive impairment after sports concussion. *Neurosurgery.* 2004;54(5):1073-1080. doi. 10.1227/01.NEU.0000118820.33396.6A.
23. Henry LC, Elbin RJ, Collins MW, Marchetti G, Kontos AP. Examining recovery trajectories after sport-related concussion with a multimodal clinical assessment approach. *Neurosurgery.* 2016;78(2):232-241. doi. 10.1227/NEU.0000000000001041.
24. Lipton ML, Kim N, Zimmerman ME, et al. Soccer heading is associated with white matter microstructural and cognitive abnormalities. *Radiology.* 2013;268(3):850-857. doi. 10.1148/radiol.13130545.
25. Talavage TM, Nauman EA, Breedlove EL, et al. Functionally-detected cognitive impairment in high school football players without clinically-diagnosed concussion. *J Neurotrauma.* 2014;31(4):327-338. doi. 10.1089/neu.2010.1512.
26. Tarnutzer AA, Straumann D, Brugger P, Feddermann-Demont N. Persistent effects of playing football and associated (subconcussive) head trauma on brain structure and function: a systematic review of the literature. *Br J Sports Med.* 2017;51(22): 1592-1604. doi. 10.1136/bjsports-2016-096593.
27. Kontos AP, Braithwaite R, Chrisman SPD, et al. Systematic review and meta-analysis of the effects of football heading. *Br J Sports Med.* 2017;51(15):1118-1124. doi. 10.1136/bjsports-2016-096276.
28. Martini DN, Broglio SP. Long-term effects of sport concussion on cognitive and motor performance: a review. *Int J Psychophysiology.* 2018;132:25-30. doi. 10.1016/j.ijpsycho.2017.09.019.
29. Guskiewicz KM, Marshall SW, Bailes J, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurgery.* 2005;57(4):719-726. doi. 10.1227/01.NEU.0000175725.75780.DD.
30. Manley G, Gardner AJ, Schneider KJ, et al. A systematic review of potential long-term effects of sport-related concussion. *Br J Sports Med.* 2017;51(12):969-977. doi. 10.1136/bjsports-2017-097791.
31. Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. *Med Sci Sports Exerc.* 2007;39(6): 903-909. doi. 10.1249/mss.0b013e3180383da5.
32. De Beaumont L, Theoret H, Mongeon D, et al. Brain function decline in healthy retired athletes who sustained their last sports concussion in early adulthood. *Brain.* 2009;132(3):695-708. doi. 10.1093/brain/awn347.
33. Abrahams S, Fie SM, Patricios J, Posthumus M, September AV. Risk factors for sports concussion: an evidence-based systematic review. *Br J Sports Med.* 2014;48(2):91-97. doi. 10.1136/bjsports-2013-092734.
34. Zemper ED. Two-year prospective study of relative risk of a second cerebral concussion. *Am J Phys Med Rehabil.* 2003;82(9):653-659. doi. 10.1097/01.PHM.0000083666.74494.BA.
35. Gavett BE, Stern RA, McKee AC. Chronic traumatic encephalopathy: a potential late effect of sport-related concussive and subconcussive head trauma. *Clin Sports Med.* 2011;30(1):179-188. doi. 10.1016/j.csm.2010.09.007.
36. Tagge CA, Fisher AM, Minaeva OV, et al. Concussion, microvascular injury, and early tauopathy in young athletes after impact head injury and an impact concussion mouse model. *Brain.* 2018;141(2):422-458. doi. 10.1093/brain/awx350.
37. Mez J, Daneshvar DH, Kiernan PT, et al. Clinicopathological evaluation of chronic traumatic encephalopathy in players of American football. *JAMA.* 2017;318(4):360. doi. 10.1001/jama.2017.8334.
38. Montgomery S, Hiyoshi A, Burkill S, Alfredsson L, Bahmanyar S, Olsson T. Concussion in adolescence and risk of multiple sclerosis: concussion and MS. *Ann Neurol.* 2017;82(4):554-561. doi. 10.1002/ana.25036.
39. Hayes JP, Logue MW, Sadeh N, et al. Mild traumatic brain injury is associated with reduced cortical thickness in those at risk for Alzheimer's disease. *Brain.* 2017;140(3): 813-825. doi. 10.1093/brain/aww344.
40. Burke JF, Stulc JL, Skolarus LE, Sears ED, Zahuranec DB, Morgenstern LB. Traumatic brain injury may be an independent risk factor for stroke. *Neurology.* 2013;81(1): 33-39. doi. 10.1212/WNL.0b013e318297eecf.
41. Jafari S, Etminan M, Aminzadeh F, Samii A. Head injury and risk of Parkinson disease: a systematic review and meta-analysis: head injury and risk of Parkinson disease. *Mov Disord.* 2013;28(9):1222-1229. doi. 10.1002/mds.25458.
42. Walsh S, Donnan J, Fortin Y, et al. A systematic review of the risks factors associated with the onset and natural progression of epilepsy. *NeuroToxicology.* 2017;61:64-77. doi. 10.1016/j.neuro.2016.03.011.
43. Lau BC, Kontos AP, Collins MW, Mucha A, Lovell MR. Which on-field signs/symptoms predict protracted recovery from sport-related concussion among high school football players? *Am J Sports Med.* 2011;39(11):2311-2318. doi. 10.1177/0363546511410655.
44. Mucha A, Collins MW, Elbin RJ, et al. A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions: preliminary findings. *Am J Sports Med.* 2014;42(10):2479-2486. doi. 10.1177/0363546514543775.
45. Kontos AP, Sufrinko A, Elbin RJ, Puskar A, Collins MW. Reliability and associated risk factors for performance on the vestibular/ocular motor screening (VOMS) tool in healthy collegiate athletes. *Am J Sports Med.* 2016;44(6):1400-1406. doi. 10.1177/0363546516632754.
46. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. *Clin J Sport Med.* 1995;5(1):32-35. doi. 10.1097/00042752-199501000-00006.
47. Hubertus V, Marklund N, Vajkoczy P. Management of concussion in soccer. *Acta Neurochir.* 2019;161(3):425-433. doi. 10.1007/s00701-019-03807-6.
48. Union of European Football Associations. *UEFA Introduces UEFA EURO 2020 Concussion Charter*; 2021. Accessed September 2, 2022. uefa.com/returntoplay/news/026a-128036869e67-553222de49ed-1000–uefa-introduces-uefa-euro-2020-concussion-charter/.
49. Professional Footballers' Association. *Current Concussion Protocols Failing to Prioritise Player Safety*; 2022. Accessed September 2, 2022. thepfa.com/news/2022/2/20/current-concussion-protocols-failing-to-prioritise-player-safety.
50. Deutsche Fussball Liga. *Baseline Screening Obligatory in the Bundesliga and Bundesliga 2 as of the 2019-20 Season*; 2019. Accessed September 2, 2022. dfl.de/en/news/baseline-screening-obligatory-in-the-bundesliga-and-bundesliga-2-as-of-the-2019-20-season/.
51. Yue JK, Phelps RRL, Chandra A, Winkler EA, Manley GT, Berger MS. Sideline concussion assessment: the current state of the art. *Neurosurgery.* 2020;87(3): 466-475. doi. 10.1093/neuros/nyaa022.
52. Provenzale JM. Imaging of traumatic brain injury: a review of the recent medical literature. *Am J Roentgenology.* 2010;194(1):16-19. doi. 10.2214/AJR.09.3687.
53. Cubon VA, Putukian M, Boyer C, Dettwiler A. A diffusion tensor imaging study on the white matter skeleton in individuals with sports-related concussion. *J Neurotrauma.* 2011;28(2):189-201. doi. 10.1089/neu.2010.1430.
54. Wu YC, Harezlak J, Elsaid NMH, et al. Longitudinal white-matter abnormalities in sports-related concussion: a diffusion MRI study. *Neurology.* 2020;95(7):e781-e792. doi. 10.1212/WNL.0000000000009930.
55. Churchill NW, Hutchison MG, Graham SJ, Schweizer TA. Connectomic markers of symptom severity in sport-related concussion: whole-brain analysis of resting-state fMRI. *NeuroImage Clin.* 2018;18:518-526. doi. 10.1016/j.nicl.2018.02.011.
56. Vagnozzi R, Signoretti S, Cristofori L, et al. Assessment of metabolic brain damage and recovery following mild traumatic brain injury: a multicentre, proton magnetic resonance spectroscopic study in concussed patients. *Brain.* 2010;133(11): 3232-3242. doi. 10.1093/brain/awq200.
57. Coughlin JM, Wang Y, Minn I, et al. Imaging of glial cell activation and white matter integrity in brains of active and recently retired national football league players. *JAMA Neurol.* 2017;74(1):67. doi. 10.1001/jamaneurol.2016.3764.
58. Koerte IK, Lin AP, Willems A, et al. A review of neuroimaging findings in repetitive brain trauma: neuroimaging findings in repetitive brain trauma. *Brain Pathol.* 2015; 25(3):318-349. doi. 10.1111/bpa.12249.
59. Gill J, Merchant-Borna K, Jeromin A, Livingston W, Bazarian J. Acute plasma tau relates to prolonged return to play after concussion. *Neurology.* 2017;88(6):595-602. doi. 10.1212/WNL.0000000000003587.
60. Shahim P, Tegner Y, Wilson DH, et al. Blood biomarkers for brain injury in concussed professional ice hockey players. *JAMA Neurol.* 2014;71(6):684. doi. 10.1001/jamaneurol.2014.367.
61. U.S. Food and Drug Administration. FDA warns public not to use unapproved or uncleared medical devices to help assess or diagnose a concussion. Published April 10, 2019. Accessed February 14, 2023. https://www.fda.gov/news-events/press-announcements/fda-warns-public-not-use-unapproved-or-uncleared-medical-devices-help-assess-or-diagnose-concussion.

eReferences available from links.lww.com/CPJ/A400.

How to cite this article: Hallock H, Mantwill M, Vajkoczy P, et al. Sport-related concussion: a cognitive perspective. *Neurol Clin Pract.* 2023;13(2):e200123. doi: 10.1212/CPJ.0000000000200123.

Sport-Related Concussion: A Cognitive Perspective

Harry Hallock, Maron Mantwill, Peter Vajkoczy, et al.

Neurol Clin Pract 2023;13;
DOI 10.1212/CPJ.0000000000200123