



# Sports Concussion

Briana N. Perry, Kassondra Collins, Ellen O'Connor,  
Sharon R. Weeks, and Jack W. Tsao

## Introduction

Concussion, or mild traumatic brain injury (mTBI), is a persistent problem in athletic participation and competition. Full-contact sports, such as football, boxing, soccer, hockey, rugby, and basketball, report the highest incidence of sports concussions, but these injuries also occur in other sports and even recreational activities. An estimated 1.6 to 3.8 million sports-related concussions occur in the United States every year, and estimates reveal that as many as 50% go unreported [1, 2]. Therefore, defining, assessing, and treating these injuries have become a critical

focus for physicians, coaches, and players alike. Recent evidence that repeated concussions can have long-term or even fatal effects has raised policy questions on diagnosis and return-to-play (RTP) guidelines. Increased participation in athletics at both the high school and collegiate levels has exposed more and more youths to concussion risks [3]. The increase in participation and competition also means that elite athletes are sometimes subject to the effects of multiple concussions over many years of athletic competition prior to their college or professional careers. Some neurocognitive testing has been developed to assess the damage caused by concussions and to categorize injury severity and necessary treatment. Recent discoveries of chronic traumatic encephalopathy (CTE) or abnormal (pathological) deposition of tau protein in brain tissue during brain autopsies of National Football League (NFL) players highlight that there is still much to learn.

In this chapter, we review the literature on the etiology and sequelae of sports concussions, highlight areas of interest for future research, and present a summary of the compilation of guidelines published in the literature on triage and treatment of concussive injuries in both youths and adults.

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B. N. Perry

Department of Orthopedics and Rehabilitation,  
Walter Reed National Military Medical Center,  
Bethesda, MD, USA

K. Collins

Department of Health Sciences, University of  
Saskatchewan, Saskatoon, SK, Canada

E. O'Connor

Department of Neurology, University of Tennessee  
Health Sciences Center, Memphis, TN, USA

S. R. Weeks

Department of Surgery, Walter Reed National  
Military Medical Center, Bethesda, MD, USA

J. W. Tsao (✉)

Department of Neurology, University of Tennessee  
Health Science Center, Memphis, TN, USA

Children's Foundation Research Institute, Le  
Bonheur Children's Hospital, Memphis, TN, USA  
e-mail: [jtsao@uthsc.edu](mailto:jtsao@uthsc.edu)

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## Etiology and Symptoms of Sports Concussions

The etiology of sports concussions varies from sport to sport, but common mechanisms of injury

include rotational acceleration (shearing), linear acceleration (compressive and tensile stresses on axons), carotid artery injuries, and deceleration on impact [4, 5]. An increase in technological advances in protective gear and more aggressive play has increased the risk of catastrophic head injury associated with greater force of collisions and higher speed of play in full-contact team sports [6].

In football, the nature of the sport and the speed and frequency of player-to-player contact place the athlete at high risk for injury. One insurance company reported that the injury rates in organized high school football are double that of the general population [7]. The popularity of football across ages and regions of the United States further contributes to the public health concern, with estimates that as many as 1.5 million young men participate in American football at the high school and collegiate levels alone. An estimated 1.2 million football-related injuries are sustained annually, with concussions accounting for up to 5% of these injuries [7–10]. In the 2015 NFL season, there were 199 recorded concussions, with long snappers being the only position not to receive one and the highest instance of concussions recorded at the position of cornerback [11]. The majority of these injuries are likely sustained during direct competition; a 2-year study of over 6000 football players found that the rate of injury in games is 8.6 times higher than in practice, which is consistent with previous reports on other sports [9]. More recently, Meehan and colleagues [12] reported that approximately 78.5% of concussions occurred during game settings rather than practices in high school athletes. Dompier and colleagues found that while football practices were a major source of concussion for all three competition levels (youth, high school, and collegiate football), the rate for concussion was higher in games than in practice [13].

The risk of concussion in boxing is especially high since injury is a goal of the sport; a concussion is an objective rather than a competitive risk [14]. Boxers are subject to numerous and sometimes rapid, consecutive blows to the head, whether concussive or sub-concussive. As a result, these athletes often demonstrate a range of neurological defects [15]. A longitudinal study of

484 amateur boxers revealed statistically significant correlations between number of bouts completed before the baseline examination and changes in memory, visuospatial ability, and perceptual/motor ability 2 years later [16]. Another study of 41 boxers and 27 control subjects revealed that boxers performed worse on psychometric tests than controls and that the boxers with more bouts performed worse than the boxers with a smaller number [17]. Furthermore, boxers had more aberrations in cerebral perfusion than controls, as detected by positron emission tomography (PET) imaging. Incidence of CTE in boxers varies throughout the literature, due to differences in definitions and methods used to detect the condition. A 1969 study by Roberts [18] still holds as the best estimate of CTE in professional boxers. He sampled 244 random boxers from a pool of 16,781 retired professional boxers and found that 5% had severe CTE and 17% had lesions of the nervous system. The severity of the conditions was directly linked to the length of each boxer's career and the number of matches fought. Unfortunately, due to the nature of diagnosing CTE post-mortem, studies on the topic are retrospective and therefore unlikely to provide true incidence numbers [19].

Unlike football and boxing, soccer is not traditionally considered to be a high-risk sport. Recent studies, however, have revealed a high rate of concussive injuries among soccer players, which is particularly significant when you consider that soccer is the most popular sport in the world. Within the United States alone, there are an estimated 12.5 million [20] to 18.2 million [21] soccer players. This number dramatically increases to 265 million soccer players worldwide [22]. Covassin, Swanik, and Sachs [23] identified 22% of all soccer injuries as concussions. Comstock and colleagues [24] reported that player-to-player contact is the leading cause of soccer-related concussions, with head-to-ball contact (i.e., “heading” the ball) as the second leading cause. An overwhelming majority of these injuries are incurred during matches, rather than during training [25]. While both male and female soccer players are at a risk for suffering a concussion, multiple studies have shown that the symptoms due to

injury last longer in women than in men [26–28]; 8 days post-injury, female concussed athletes reported more total post-concussive symptoms than men, as well as scored worse on verbal and visual memory tests, after controlling for body mass index (BMI) [29]. BMI was controlled for due to the association between higher BMI and reduced cognitive function [30]. Moreover, younger female soccer players report higher concussion rates than women of older age groups, with most of the young concussed athletes continuing to remain in the game despite the presence of concussion symptoms [31]. Therefore, while all soccer players are at risk for sustaining a concussion, great attention should be paid to preventing, recognizing, and treating concussions among female athletes, specifically those of younger age. It is especially important to highlight the need to remove an athlete who is suspected of having a concussion in order to prevent further injury. The risk of brain changes secondary to playing soccer extends beyond those due to concussions alone and will be discussed further in the neuroimaging portion of this chapter [32–34].

Hockey is a popular sport in both Canada and the United States. It is estimated that for the 2008–2009 season, roughly 550,000 youths (age 9–16 years) participated in ice hockey in Canada and 340,000 in the United States [35]. Concussions are the most common specific injury occurring in ice hockey players and account for more than 15% of all injuries in youth players [36, 37]. Body checking, or deliberately getting in the way of an opponent using one's own body, is associated with 45–86% of all injuries, including concussions [36, 38, 39]. A 2010 study conducted by Emery found that in leagues that body checking is acceptable, there is three times the risk of concussion and serious concussive injuries [35]. Concussion reporting in sports is not always seen as something of high importance. A study by Kroshus [40] found that targeting the perceived concussion reporting norms may be an avenue for repairing the underreporting of concussions among hockey players. The study found that players who believed most athletes reported symptoms of a concussion were more likely to report their own symptoms [40]. Thus, concus-

sion education and awareness is crucial to recognizing concussions and removing a player from the activity before further neurological damage occurs.

Recognition of head injury is easy when there is a loss of consciousness (LOC). The majority of sports concussions, however, occur without a LOC [41–43]. When it is difficult to make accurate sideline diagnoses, players are more likely to remain in the game or RTP too soon after injury. Internal and external pressures from players and their communities also increase the likelihood that they will not seek adequate medical attention immediately. As LOC may or may not occur with mild concussions, it is important to be aware and look out for other immediate effects of concussions including vacant stare, delayed verbal and motor responses, confusion, inability to focus attention, disorientation, slurred or incoherent speech, gross observable incoordination, disproportionate emotions, and memory deficits/post-traumatic amnesia [44]. As the brain is possibly the most variable of human organs in its response to external stimuli or insult [45], it should come as no surprise that the clinical presentation of concussed athletes varies significantly from individual to individual. In addition to individual differences, contributing factors to varied presentations include biomechanical forces involved and the athlete's prior history of injury, among others [46, 47].

Concussed individuals do commonly describe a similar set of symptoms after injury, including headaches, dizziness, confusion, disorientation, and blurred vision [48]. Balance problems are present after 30% of concussive events [48], and nausea and emesis are also common [49]. In children, symptoms typically include restlessness, lethargy, confusion, or irritability. The adult symptoms were classically thought to suggest intracranial lesions, but the data supporting these conclusions are sparse [49]. In fact, fewer than 1% of patients with minor head trauma have surgically significant lesions [50]. The consequences of a concussion can last for several days. McCrea and colleagues [51] found that concussed football players continue to show acute symptoms, such as balance problems, for at least 5 days,

**Table 1** Risk factors for concussive injury

High risk	Medium risk	Low Risk
Focal neurologic findings	Initial GCS score of 15	Currently asymptomatic
Asymmetric pupils	Brief LOC	No other injuries
Skull fracture on clinical examination	Post-traumatic amnesia	No focal deficits on examination
Multiple trauma	Vomiting	Normal pupils
Serious, painful, distracting injuries	Headache	No change in consciousness
External signs of trauma above the clavicles	Intoxication	Intact orientation/memory
Initial GCS score of 14 or 15		Initial GCS score of 15
Loss of consciousness		Accurate history
Post-traumatic confusion/anemia		Trivial mechanism
Progressively worsening headache		Injury more than 24 hours ago
Vomiting		No or mild headache
Post-traumatic seizure		No vomiting
History of bleeding disorder/anticoagulation		No preexisting high-risk factors
Recent ingestion of intoxicants		
Unreliable/unknown history of injury		
Previous neurologic diagnosis		
Previous epilepsy		
Suspected child abuse		
Age older than 60 years or younger than 2 years		

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with some cognitive impairments lasting up to 7 days post-injury. Every individual suspected of having a concussive injury needs a thorough neurological examination, but the need for additional diagnostic testing, including detailed mental status examinations, depends upon the individual's risk factors (Table 1).

## Lasting Effects of Sports Concussion

There is a plethora of research suggesting that sustaining a concussion increases the risk of incurring additional concussions [52, 53]. A study by Zemper [54] examining 15,304 football player-seasons at the high school and collegiate levels found that the relative risk for repeat concussions in individuals with a history of concussion is 5.8 times greater than in individuals with no prior history of concussion. In a study of 4251 player-seasons, Guskiewicz [55] also found a positive association between the reported number of previous concussions and the likelihood of incident concussion. Specifically, as compared to players with no concussive history, players who reported a history of 1, 2, or 3 or more previous concussions were 1.5, 2.8, and 3 times, respectively, more likely to have sustained a subsequent concussion.

The risk of sustaining multiple concussions is especially concerning since the long-term effects of concussions are still unknown but likely to be cumulative. Over 40 years ago, Gronwall [56] reported that the rate at which young adults process information is reduced more in those who have suffered two concussions compared to those who have suffered one concussion. More recently, research suggests that concussive effects become cumulative after 3 injuries. Collins [57] reported that athletes with three or more concussions are more likely to experience on-field LOC (6.7 times greater likelihood), anterograde amnesia (3.8 times), and confusion (4.1 times) after a subsequent concussion. Also, studies of high school and collegiate athletes using ImPACT testing (an automated neurocognitive test battery) showed that there are no detectable cumulative effects of only one or two previous concussions [58] but marked effects in athletes with three or more concussions [59]. This multitude of findings highlights the need for adequate prevention and treatment of concussive injuries.

The lasting effects of sports concussions were first highlighted in the public eye in the early 1990s when NFL players Al Toon and Merrill Hoge retired from the league because of prolonged post-concussion syndrome (PCS). Some reviews report an incidence of PCS of approximately

10–20% of concussed athletes [47]. Symptoms of the syndrome include headache, dizziness, anxiety, and impaired cognition and memory [60]. These symptoms affect more than 58% of patients 1 month after injury [61] and 15% of patients 1 year after injury [62]. PCS is characterized by lingering deficits due to the occurrence of a concussion. To be considered PCS, there must be a minimum of three symptoms present, for at least 3 months since the injury, as well as neuropsychological dysfunction [63]. The presence of headache, nausea, and dizziness during the acute head injury assessment increases the risk for subsequent development of PCS; a report of all three symptoms is associated with a 50% likelihood of PCS at 6 months post-injury, while an absence of all three symptoms reduces likelihood to 28%. The findings of lasting effects of concussive injuries and the underlying cause of PCS are still unknown. The pathophysiology of acute symptoms has been supported by animal studies demonstrating a neurochemical and metabolic cascade that detrimentally affects cognitive functions for up to 2 weeks after a concussive injury [64, 65]. Differences in development of PCS, however, point toward an additional psychopathological cause to this long-term disorder which we have yet to elucidate [63].

Alongside PCS, the discovery of the rare “second impact syndrome” (SIS) has led to considerable concern in athletes of all levels. First noted in 1984 [66], there have since been several other reports on this syndrome in the literature [67, 68]. In 2011, the death of a 22-year-old starting full-back for the Frostburg State University Bobcats was attributed to uncontrollable brain swelling that may have been caused by repeat concussions leading to SIS [69]. With SIS, athletes suffer a concussion, usually mild, and sometimes, but not always, with LOC. The athlete typically returns to play within a few days of the first hit and sometimes even within the same match as the initial injury. Cantu [70] outlined ten cases of suspected SIS, with six individuals surviving the repeated blows but incurring devastating neurological cognitive, visual, motor, and sensory deficits. Four of the reported cases resulted in death. Following the second impact, none of the athletes lost consciousness immediately, but within min-

utes all fell into a coma, with symptoms of blown pupils, respiratory arrest, and signs of brain herniation – all of which are typical of SIS [70]. These cases of SIS show that repeated episodes of mild brain injury occurring within a short period of time can be catastrophic and even fatal. This highlights a drastic need for proper initial diagnosis and subsequent removal from play until complete recovery, as well as increased education and improved vigilance surrounding athletes with head injuries.

One of the more severe consequences of multiple concussions an athlete may suffer over the course of a career is CTE. First described by Harrison Martland [71] in 1928 as dementia pugilistic, CTE is characterized by early symptoms of slight mental confusion, a slowing of muscular movements, hesitancy in speech, and hand tremors. In time, these symptoms become more severe and progressive, including speech and gait disturbances, pyramidal tract dysfunction, memory impairment, extrapyramidal features, behavior or personality changes (including increased aggression), and psychiatric disease [72–75]. In 1973, Corsellis [15] identified the neuropathology of this syndrome in the brains of 15 deceased boxers, 8 of whom were national or world champions. Through autopsy, he found that the neuropathology of CTE was characterized by cavum septum pellucidum, degeneration of substantia nigra, septal fenestrations, cerebellar scarring, diffuse neuronal loss, and prominent neurofibrillary tangles, now known to be composed of tau protein. In the mid-2000s, the term, “CTE,” entered the public lexicon when the first documented cases of symptoms suggestive of CTE in retired NFL players were published [76, 77]. Since these initial reports, the presence of neuropathological changes associated with CTE has been confirmed at autopsy in numerous professional football players via an ongoing prospective research program entitled C.O.N.T.A.C.T (Consent to Offer Neural Tissue of Athletes with Concussive Trauma). This program was initially comprised of 150 former athletes, including 40 retired and 3 active NFL players, but has grown to include more than 196 brain tissue donations and 700 registered future donors [78]. All participants have

agreed to be interviewed annually by phone throughout their lives and, upon their death, to donate their brains for examination by the Center for the Study of Traumatic Encephalopathy (CSTE), an independent academic research center located at the Boston University School of Medicine [79]. In 2009, McKee and colleagues [80] reviewed all 47 of the neuropathologically confirmed CTE cases and reported on 3 additional CSTE cases, noting the overall athletic makeup of the subjects: 43 boxers, 5 American football players, 1 professional wrestler, and 1 soccer player. This report also explored the timeline of CTE symptomology, finding that symptoms of half of the athletes were observed within 4 years of their retirement, often while they were in their early 40s, and continued to progress in an irreversible fashion.

In 2017, Mez and colleagues published results from a convenience sample of 202 deceased football players in which a very high proportion of players (87%) had neuropathological evidence of CTE, leading the researchers to hypothesize that prior participation in football may be related to the development of the disease [81]. Of the 202 deceased players, 3 out of 14 high school, 48 out of 53 college, 9 out of 14 semiprofessional, 7 out of 8 Canadian Football League, and 110 out of 111 NFL players were neuropathologically diagnosed with CTE. Neither of the two pre-high school players had evidence of the disease. Athletes with severe pathology were more likely to be involved in the highest level of play than those with mild CTE severity.

The phosphorylated tau (p-tau) protein pathology of CTE is typically classified into four stages, where stages I and II are “mild” and III and IV are “severe” [81–83]. Stage I consists of 1–2 isolated perivascular epicenters of p-tau neurofibrillary tangles and neurites located deep in the cerebral sulci of the frontal, temporal, or parietal cortices. Stage II is characterized by superficial neurofibrillary tangles located along the sulcal wall and gyral crests and three or more CTE lesions found in multiple cortical regions. P-tau pathology is widespread in stage III, with the greatest severity of neurofibrillary degeneration in the frontal and temporal lobes, concentrated in

the depths of the sulci [82]. Neurofibrillary pathology is also seen in the amygdala, hippocampus, and entorhinal cortex. In stage IV, lesions and neurofibrillary tangles are spread through most regions of the cerebral cortex and brain stem, along with neuronal loss, gliosis, and astrocytic p-tau pathology [81].

Currently, CTE can only be definitively diagnosed by a post-mortem neuropathological examination [83]. Distinctive clinical features, however, do exist that often follow a progressive course. In a study by McKee and coauthors [82], the clinical symptoms of each stage of CTE were recorded using medical record review and family interviews. They found that in stage I, four out of six subjects reported headache and loss of attention/concentration, two had trouble with executive function and explosivity, and three had short-term memory problems, depression, and aggressive tendencies. In stage II, individuals reported loss of attention/concentration, short-term memory loss, depression and mood lability, headache, and explosivity. Executive dysfunction, language difficulties, impulsivity, and suicidality were also present, although less common. In stage III, individuals most commonly reported symptoms of explosivity, attention/concentration difficulties, and executive dysfunction. Depression and mood swings, aggression, and visuospatial problems were also frequently found at this stage, as well as the less common symptoms of apathy, headaches, suicidality, and impulsivity. Seventy five percent of the stage III subjects were cognitively impaired. Finally, stage IV clinical symptoms included executive dysfunction, profound loss of concentration/attention, paranoia, depression, gait and visuospatial difficulties, and explosivity and aggressive tendencies. All subjects in stage IV developed severe memory loss with dementia during their course [82].

Mez and coauthors [81] found similar results with regard to a progressive clinical course of symptoms with CTE. Behavioral and mood symptoms were common in athletes with both mild (96%) and severe (89%) CTE pathology. Their study examined 111 cases of standardized informant reports of clinical symptoms finding that 48 (43.2%) individuals first presented with behavioral or mood symptoms, 47 (42.3%) first

presented with cognitive symptoms, and 16 (14.4%) initially presented with both behavioral or mood and cognitive symptoms.

It is crucial to note that the clinical presentation of CTE is separate from the accumulation of symptoms attributed to post-concussive syndrome, or other long-term sequelae of concussion, as CTE symptoms are due to progressive neuronal death and/or progressive decline in functioning neurons [84]. For example, when neuronal death occurs in a certain region of the brain, symptoms may present that coincide with that region's function. It is common for symptoms to appear in midlife, often years after the end of the traumatic exposure(s). Symptoms typically present slowly and gradually, often over decades, and broaden in scope and severity over time [84].

While repetitive brain trauma is the most notorious risk factor for the development of CTE, other risk factors may include genetics, family history, chronic inflammation, type of brain trauma exposure, age and duration of brain trauma exposure, frequency of brain trauma exposure, gender, race, and cognitive reserve [84]. It is currently unclear whether symptomatic hits that lead to concussions are riskier than sub-concussive hits that remain asymptomatic but accumulate over time [81]. It is also unclear whether specific biomarkers exist for CTE diagnosis. Recent research has suggested that CCL11 (a chemokine that has been associated with age-related cognitive decline) may be a potential diagnostic biomarker in the brains and CSF of people with CTE [85]. Cherry and coauthors examined the level of expression of CCL11 in the dorsolateral frontal cortex of subjects with neuropathologically verified Alzheimer's disease, CTE, and normal controls and found that the total levels of CCL11 were significantly elevated in those with CTE as compared to the subjects with Alzheimer's disease or the controls. This increase was also correlated with years of exposure to American football – CCL11 levels were significantly increased in subjects with CTE and exposure to 16 or more years of American football, as compared to controls with no exposure to sports and subjects with CTE and less than 16 years of exposure. Furthermore,

using post-mortem CSF samples, a trend was found in which increased CCL11 levels were present in those with CTE but not Alzheimer's disease, compared with controls [85]. Hopefully, with ongoing research and public awareness, preventing the onset of CTE will be possible.

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## Differences Due to Age and Developmental Level

Age differences in concussion diagnosis and management were not given much attention until the early 2000s when studies began to reveal marked differences in the way that youths and adults respond to and recover from concussions. Multiple studies have now shown that high school athletes require more time to recover cognitive performance than collegiate athletes [86–88], even though collegiate athletes had a greater prior incidence of concussion, which typically slows recovery [89]. Lovell and colleagues (2003, 2004) revealed a heightened vulnerability to concussions in younger athletes (ages 13–17 years), leading them to propose that currently accepted RTP guidelines for adults may be too liberal for adolescents. It has been suggested that the immature brain's sensitivity to glutamate [90, 91], a neurotransmitter involved in the metabolic cascade following concussion, may partly explain these differences in recovery time [92]. In addition, the young brain is still developing and differs from the adult brain in many areas including the brain water content, amount of myelination, total blood volume, structure of the blood-brain barrier, metabolic rate of processing glucose, level of blood flow, amount of synapses, and elasticity of the skull itself [93]. These findings collectively suggest that clinicians need to exercise increased caution in returning young athletes to play following a concussion or display of concussive symptoms.

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## Triage and Treatment

It should be noted that most people recover successfully from a concussion with no noticeable long-term effects. McCrea [51] found that 91%

of concussed football players returned to their pre-injury baselines within a week following injury. Nevertheless, the severe conditions that can result from sports head injuries in a small but noteworthy number of cases highlight the necessity of taking concussions seriously and being conservative in RTP guidelines.

The frequency of concussive sports injuries has encouraged the development of easy-to-administer neurocognitive tests that can be given on the sidelines of a playing field, immediately after a suspected concussion, to improve diagnostic accuracy. Of the neurocognitive tests reported in the literature, the Standardized Assessment of Concussion, or the SAC, is possibly the most popular and well-studied. The SAC takes approximately 5 minutes to administer, requires no prior experience in neuropsychological testing, and consists of four components: orientation, immediate memory, concentration, and delayed recall [94]. An assessment of strength, sensation, and coordination is included, as is the documentation of LOC, retrograde amnesia, and post-traumatic amnesia. The total composite score on the exam has been shown to be sufficient in differentiating between non-concussed controls and players who have suffered even mild concussions. A study of this test in 141 high school football players demonstrated that its demanding cognitive measures could be sensitive enough to detect mild concussions [94]. These findings were later supported by a larger study of 568 high school and college football players [95]. Normative data from more than 2500 male and female junior high, high school, college, and professional athletes have shown that the SAC is reliable over repeated administrations and is free of significant gender effects. It is also acceptable for use at all competitive and educational levels [96].

In addition to the SAC, the Second International Symposium on Concussion Prague 2004 developed a sideline assessment entitled the Sport Concussion Assessment Tool, or the SCAT [97]. The SCAT was created by combining several common tools into one standardized test and includes a neurologic screen, cognitive and memory assessments, and a query of symptoms, such as LOC, convulsive activity, and balance prob-

lems. The SCAT was updated to include the calculation of the SAC score and the Maddocks questions for sideline concussion assessment [98, 99]. The SCAT-3 was later developed to improve upon the reliability and sensitivity of the SCAT by adding a Glasgow Coma Scale, as well as assessments of symptom severity, neurocognitive function, and balance function [100].

In 2016, the Berlin meeting of the Fifth International Consensus Conference on Concussion in Sport led to the development of the SCAT-5. The SCAT-5 improves upon the SCAT-3 by clarifying administration and RTP guidelines. It states that the test needs at least 10 minutes to be appropriately administered, that the athlete should be in a resting state while completing the symptom checklist, and that a written clearance by a healthcare professional is needed before the athlete may RTP. The SCAT-5 also includes additional assessments, such as the Rapid Neurological Screen, which evaluates an athlete's speech, balance, visual tracking, cervical exam, reading abilities, and finger-to-nose coordination [101].

The military used SCAT metrics to develop the Military Acute Concussion Evaluation (MACE), which is used by combat medics and corpsmen on the battlefield to evaluate service members in whom a concussion is suspected [102, 103]. The MACE uses many of the same examination tasks as the SAC as well as includes a collection of demographic and injury incident details. The SAC, MACE, and Maddocks questions are summarized in Table 2.

Considering that balance is often affected by concussive injuries, neurological assessments would be improved by including clinical balance tests [48]. Balance, or the maintaining of the body's center of gravity, is controlled through a complex connection of neural networks within the brain involving the cerebral cortex, cerebellum, brain stem, and spinal cord. An incorrect interaction at any point within this system can cause failure to maintain proper balance. The Balance Error Scoring System (BESS) is a cost-effective, easily administered, quantifiable test designed to determine balance deficits obtained after a potentially concussive event [48]. To administer it, one needs



**Table 2** Immediate assessments for concussion

Assessment	SAC	MACE	Maddocks questions
<i>Orientation</i>			Which field are we at? Which team are we playing? Who is your opponent at present? Which half/period is it? How far into the half is it? Which side scored the last touchdown/goal/point? Which team did we play last week? Did we win last week?
	Month, date, day of the week, year, time	Month, date, day of the week, year, time	
<i>Immediate memory</i>			
	Recall a list of five words immediately, three trials	Recall a list of five words immediately, three trials	
<i>Concentration</i>	Reverse strings of digits (3–6 digits in length)	Reverse strings of digits (3–6 digits in length)	
	Reverse the months of the year	Reverse the months of the year	
<i>Delayed recall</i>	Recall list of five words 5 minutes later	Recall list of five words 5 minutes later	
<i>Neurologic screening</i>	Recollection of injury, strength, sensation, coordination	Pupil size and reactivity, speech fluency and word finding, pronator drift, gait and coordination	
<i>Exertional measures</i>	40-yard sprint, 5 sit-ups, 5 push-ups, 5 knee bends	None	

Data from Coldren et al. [103], Maddocks et al. [99], McCrory et al. [149]

The SAC and Maddocks questions are typically used for sideline assessments for sports injuries; the MACE was developed for battlefield screening for military personnel suspected of concussion

only a stopwatch and a piece of foam. Athletes are asked to stand with their hands on their hips and their eyes closed for 20 seconds in each of three stances – double, single, and tandem – first on solid ground and then on a piece of foam. An error is recorded if the athletes step, stumble, fall, lift their foot, lift their hands off their hips, open their eyes, or flex or abduct their hips more than 30°, and do not correct their footing within 5 seconds [48]. A study by McCrea [104] found that after a concussive event, a change from baseline scores in the BESS averages 5.7 points initially and then 2.7 points at 1 day. There are limitations to the BESS: an effect of fatigue, ankle instability, and learning or practice has been observed after repetitive administration [105–107]. Despite these limitations, the use of a comprehensive sideline assessment, like the BESS, that considers neurological and balance function should be conducted whenever an athlete is suspected of sustaining a concussion.

In addition to neurocognitive testing, neuropsychological testing is becoming common among sports health professionals. The wide range of tests currently available is sensitive to concussive impairments. The conventional neuropsychological assessments include the Trail

Making Tests A and B [108], Digit Symbol Substitution Test [109], Controlled Oral Word Association (COWA) Test [110], Hopkins Verbal Learning Test [111], and Stroop Word Color Test [112]. There are also computerized assessments available, which include the Automated Neuropsychological Assessment Metrics (ANAM) [113], Axon Sports (Scottsdale, AZ, USA; formally, CogState Ltd.'s CogSport©) [114], Headminder Cognitive Stability Index (CSI) [115], BrainCheckers test (Behavioral Neuroscience Systems, Springfield, Missouri, USA) [116], CNS Vital Signs test [117], Immediate Post-Assessment of Concussion Test (ImPACT Applications®, San Diego, CA, USA) [118], and Defense Automated Neurobehavioral Assessment (DANA) [119] (Table 3). With advancing technology and increased access to hand-held devices, companies have begun to develop easily downloadable applications to assist in sideline assessments of concussions. Cleveland Clinic, for example, developed the C3 application (Cleveland Clinic Concussion) to use as a tool for assessing concussive symptoms and guiding therapy and recovery for individual athletes [120]. Other applications include the CRR (Concussion Recognition and Response™, PAR

**Table 3** Conventional tests for assessment of mild head injury

Assessment	Description
Trail Making Tests A and B	Part A involves drawing lines between 25 numbered circles, in sequential order, which are randomly arranged. Part B requires subjects to connect circles containing the letters A through L and numbered 1 through 13 by drawing lines alternating between numbers and letters in sequential order. Subjects are instructed immediately on their mistakes and continue from the last correct circle. The test takes approximately 5–10 minutes to complete. The test evaluates information processing speed, visual scanning ability, integration of visual and motor functions, letter and number recognition and sequencing, and the ability to maintain two different trains of thought.
Digit Symbol Substitution Test	Paper-pen test consisting of digit symbol pairs followed by a list of digits. The subject writes as many of the corresponding symbols on the list of digits as possible within the allowed time. Is sensitive to brain damage.
Hopkins Verbal Learning Test-Revised	Verbal learning and memory test requiring the use of both working and episodic memory. Subjects are asked to recall a repeated list of words several times. The words fall into discrete categories. Learning ability and total immediate recall and delayed recall are recorded.
Controlled Oral Word Association (COWA)	Spoken word test. The examinee has 1 minute to name as many words as possible that begin with particular letters. Examinee is then given 1 minute to name as many animals as possible. This test is a measure of verbal fluency, specifically for letters, requiring initiation and maintenance, both considered to be aspects of frontal lobe function.
Stroop Word Color Test	Provides diagnosis of brain dysfunction and the evaluation of stress, personality cognition, and psychopathology. Assesses cognitive flexibility, resistance to interference from outside stimuli, creativity, and psychopathology by requiring subject to read through words, name ink colors of symbols, and name ink colors of color words that do not match. Five minutes to administer.
Automated Neuropsychological Assessment Metrics (ANAM)	Computer-administered neuropsychological battery. Specifically designed for military use. Consists of 9 subtests and a questionnaire of symptoms. Assesses energy-fatigue level, predominant mood state, visuomotor response timing, visual search, sustained attention, working memory, processing efficiency, computational skills, spatial processing, and visuospatial working memory.
Braincheckers	Computer-administered neuropsychological battery. Consists of 6 subtests and a questionnaire of symptoms. Assesses energy-fatigue level, predominant mood state, visuomotor response timing, visual search, sustained attention, working memory, processing efficiency, computational skills, spatial processing, fronto-executive functioning, and visuospatial working memory.
CogState Sport	Battery of four card-based games. Assesses psychomotor function, processing speed, visual attention/vigilance, visual learning, and memory.
Headminder Cognitive Stability Index (CSI)	Web-based neurocognitive test protocol. Subtests relevant to general cognitive screening techniques. Adaptable for repeatable, longitudinal assessments. Ten subtests; 30 minutes in length.
CNS Vital Signs	Computerized neurocognitive test battery. Comprised of seven tests: verbal and visual memory, finger tapping, symbol digit coding, the Stroop Test, a test of shifting attention, and the continuous performance test. Sensitive to malingers and patients with conversion disorder. Suitable as a screening instrument.
Immediate Post-Assessment of Concussion Test (ImPACT)	Computerized neuropsychological test battery. Six individual cognitive test modules assess cognitive functioning, including attention, memory, reaction time, and processing speed. Modules include word memory, design memory, Xs and Os, symbol match, color match, and three letters. Composite scores are derived in the areas of memory, reaction time, and processing speed.
Defense Automated Neurobehavioral Assessment (DANA)	Portable neurocognitive assessment tool administered on an android device. There are three different versions with varying lengths: DANA Rapid (5 minutes), DANA Brief (15 minutes), and DANA Standard (45 minutes). Depending on the battery chosen, assessments include simple reaction time, procedural reaction time, go/no go, spatial discrimination, code substitution simultaneous, code substitution delayed, Sternberg memory search, matching to sample, insomnia screening index, primary care PTSD screen, patient health questionnaire, Pittsburgh sleep quality index, combat exposure scale, PTSD checklist military version, and the deployment stress inventory.

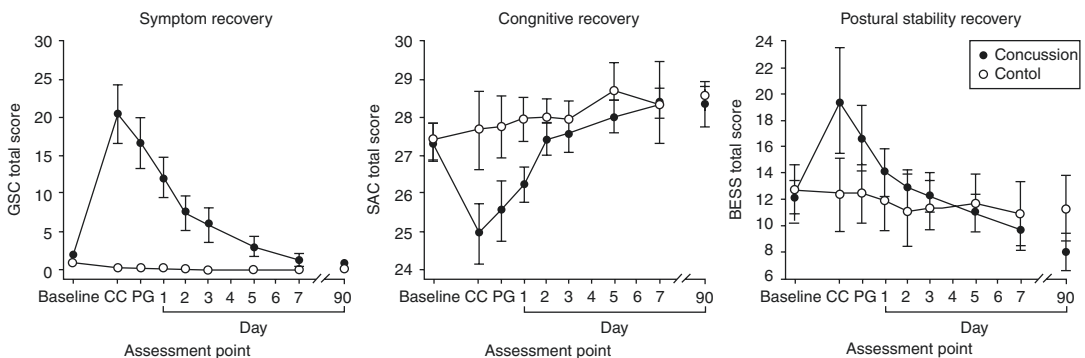
Inc., Lutz, FL, USA), the Concussion App from Sports Safety Labs LLC, and Play It Safe from Concussion Health LLC [121].

The popularity of sideline assessment tests has increased, thanks to research showing a need to assess higher cognitive functioning directly, rather than by relying on reports of LOC and amnesia. One study found that the presence of amnesia, not LOC, was most predictive of difficulties 3 days post-injury [122]. Similarly, another study found that impairment of immediate recall was much more frequent than disorientation post-injury and suggested that evaluating cognitive function and disability by asking the concussed athlete to state the day, time, month, and year may not be the most clinically useful evaluation task [123]. A third study found that athletes who reported memory problems following injury had significantly more symptoms, longer durations of symptoms, and significantly decreased performances on neurocognitive testing [115]. These results indicate that the conventional focus on LOC and disorientation as predictors for severity of a concussion may be misplaced. Moreover, with LOC occurring in less than 10% of sports-related concussions, it is essential to instead evaluate memory and immediate recall following suspected concussion [122].

In their NCAA Concussion Study, McCrea and colleagues [51] examined the timeline of concussive injury symptoms in 1631 football

players from 15 US colleges. They found that the most severe symptoms occurred immediately after concussion and were followed by a recovery period lasting 5–7 days. Normal cognitive functioning often returned by day five after injury, whereas a full 7 days were needed for clinical symptoms to return to baseline and control levels (Fig. 1). This large cohort study supported the clinical experience of many professionals and contributed scientific evidence to RTP guidelines by suggesting a gradual reintroduction to sport over the course of several days to weeks, depending on the severity of injury.

Despite growing research and interest in addressing sports concussions, there is still little consensus in the field on the best approach to post-injury care, especially regarding when and how to return athletes with head injuries to play. Hunt and Asplund [124] suggest that whatever assessment tools are used should include a cognitive assessment, some measure of balance testing, and a self-reported symptom assessment. Many guidelines promote allowing athletes to recover from all symptoms before testing, so as to prevent learning effects. In the US military, exercise to a target heart rate is recommended prior to neurocognitive testing to assess whether clinical symptoms, such as headache, have fully resolved [125]. Many institutions have started mandating baseline neurocognitive testing for



**Fig. 1** Symptom, cognitive, and postural stability recovery in concussion and control participants. Higher scores on the Graded Symptom Checklist (GSC) indicate more severe symptoms; lower scores on the Standardized Assessment of Concussion (SAC) indicate poorer cognitive performance; and higher scores on the Balance Error

Scoring System (BESS) indicate poorer postural stability. Error bars indicate 95% confidence intervals. CC indicates time of concussion; PG postgame/postpractice. On the BESS, multiple imputation was used to estimate means and 95% confidence intervals for control participants for the CC and PG assessments

athletes at risk of head injury to obtain an individualized standard in the event of a concussion, although there is little evidence that this approach changes clinical outcomes. These preseason baselines account for any comorbidities that may affect testing, such as learning disabilities, previous concussion history, medication usage, and mental conditions. While controlling for baseline performance is ideal, this current system of assessment can be circumvented; athletes have reported intentionally underperforming on these tests to decrease the scores needed for them to RTP post-injury [126].

The decision about when to return an athlete to play without limitations is an issue of considerable importance in the sports medicine field, especially given the potential for external or internal pressures on an athlete to return prematurely. There are many published guidelines in the literature, but they are based largely on clinical experience and expertise in the field rather than on rigorous study and objective evidence. Most guidelines currently recommend a gradual, stepwise return to full activity that is overseen by a qualified health professional and begun only after the athlete has become asymptomatic. If an injured athlete exhibits symptoms with increased activity, then the athlete is returned to a previous step in the recovery process, such as rest. In November 2008, the guidelines proposed by the 3rd International Conference on Concussion in Sport in Zurich, Switzerland [98], improved upon the guidelines presented by prior conferences in Vienna, Austria [127], and Prague, Czech Republic [97]. The recommendations of the Zurich conference are presented in Table 4.

Research on the effect of multiple concussions has prompted clinicians to differentiate RTP guidelines based on the severity of the concussion and the athlete's concussion history. Guidelines by Cantu [128], which are now outdated, are presented in Table 5. For the purposes of historical background, at that time the concussion grades in these guidelines included Grade 1, no LOC and post-traumatic amnesia (PTA) or post-concussive symptoms lasting less than 30 minutes; Grade 2, LOC less than 1 minute and PTA or post-concussive symptoms 30 minutes to 24 hours in duration; and Grade 3, LOC lasting more than 1 minute

**Table 4** Zurich Guidelines, Graduated Return-to-Play Protocol

Rehabilitation stage	Functional exercise at each stage of rehabilitation	Objective of each stage
No activity	Complete physical and cognitive rest	Recovery
Light aerobic exercise	Walking, swimming, or stationary cycling, keeping intensity to 70% of maximum predicted heart rate; no resistance training	Increase heart rate
Sport-specific exercise	Skating drills in hockey, running drills in soccer, no head impact activities	Add movement
Noncontact training drills	Progression to more complex training drills, e.g., passing drills in football and ice hockey; may start progressive resistance training	Exercise, coordination, and cognitive load
Full-contact practice	Following medical clearance, participate in normal training activities	Restore athlete's confidence; functional skills
Return to play	Normal game play	

Used with permission from McCrory et al. [149]

Athlete should continue to the next level if asymptomatic at the current level. Generally, each step should take 24 hours, so that an athlete would take approximately 1 week to proceed through the full rehabilitation protocol once asymptomatic at rest and with provocative exercise. If any post-concussion symptoms occur while in the stepwise program, then the patient should drop back to the previous asymptomatic level and try to progress again after a further 24-hour period of rest has passed

or PTA lasting longer than 24 hours with post-concussion signs or symptoms lasting longer than 7 days [128]. This system was revised from his previous grading system [41] based on evidence from prospective studies on PTA and persistence of post-concussive symptoms. Several other grading systems for concussion also exist in the literature. Those commonly cited are the Colorado Medical Society [129], the American Academy of Neurology [130], Jordan B.J. [131], Ommaya [132], Nelson [133], Roberts W.O. [134], and Torg Grading Systems for Concussion [135].

During early 2013, when concussion became a topic of popular discussion, multiple new and

**Table 5** Cantu Guidelines for Return to Play (RTP) After Concussion

	First concussion	Second concussion	Third concussion
Grade 1 (mild)	May RTP if asymptomatic for 1 week	RTP in 2 weeks if asymptomatic at that time for 1 week	Terminate season; may RTP next season if asymptomatic
Grade 2 (moderate)	RTP after asymptomatic for 1 weeks	Minimum of 1 month; may RTP then if asymptomatic for 1 week; consider terminating the season	Terminate season; may RTP next season if asymptomatic
Grade 3 (severe)	Minimum of 1 month; may RTP if asymptomatic for 1 week	Terminate season; may RTP next season if asymptomatic	

Used with permission of Elsevier from Cantu [150]

Note. Asymptomatic means no headache, dizziness, or impaired orientation, concentration, or memory during rest or exertion

updated guidelines and position statements were published. These updated guidelines were generated by groups like the American Medical Society for Sports Medicine, the American Academy of Neurology (Box 1), and the Zurich Consensus. All three groups agreed that no single assessment test can be used to determine the occurrence of a concussion but that any athlete who is suspected of having a concussion should be removed from play immediately [136]. In addition, the consensus was that there is no golden rule for returning an athlete to play. All athletes should be treated on an individual basis following a gradual stepwise RTP routine that allows the athlete to advance to more strenuous activities only once the athlete is asymptomatic at the current level [136].

#### Box 1 American Academy of Neurology Guidelines for Return to Play (RTP) After Concussion

1. Athletes must be assessed by an experienced LHCP with training both in the diagnosis and management of concussion and in the recognition of more severe TBI before returning to play.
2. Persons supervising athletes should prohibit any athlete with concussion from RTP/practice (contact-risk activity) until the athlete is asymptomatic.
3. Persons supervising athletes of high school age or younger with diagnosed concussion should treat them more conservatively than older athletes regarding RTP.

Data from Giza et al. [151]

## Neuroimaging and Concussions

Computed tomography (CT) and magnetic resonance imaging (MRI) remain the imaging techniques of choice for initial assessment of acute head injury for skull fractures and intracranial hemorrhage. MRI is the also standard of care for the evaluation of subacute or chronic traumatic brain injury [137]. Despite the use of these modalities, the neuroimaging of concussions has not been thoroughly explored; most mTBIs/concussions do not result in abnormalities that can be detected by either CT or standard MRI studies [138]. Research has suggested that less than 10% of patients with minor head injuries have positive CT findings and that less than 1% require neurosurgical intervention [139]. The resulting reliance on neurocognitive testing and symptom checklists for concussion diagnosis has motivated clinicians and researchers to use advanced imaging techniques to better quantify and define structural injuries in the brain following concussion. Possible techniques with increased sensitivity over traditional neuroimaging modalities include MR diffusion tensor imaging (DTI), functional MRI (fMRI), magnetic resonance spectroscopy (MRS), and positron emission tomography (PET). DTI provides a measurement modality for white matter integrity and connectivity. Functional MRI offers the opportunity to receive real-time feedback on cerebral metabolism and brain activation patterns during specific cognitive or motor tasks. MRS and PET provide images that indicate

functional cerebral metabolism. Each of these modalities, however, requires relatively long collection times and, with the exception of PET, requires post-imaging data processing. These advanced imaging technologies are not currently used in clinical assessments of sports concussions but may play a future role with increased investigation.

Early studies have used advanced imaging modalities in a research setting to observe brain changes in soccer players without a history of symptomatic concussion and compared results to images of brains from athletes participating in noncontact sports. In a small study of soccer players and swimmers, advanced DTI demonstrated increased radial diffusivity in the brains of the soccer players only – a finding similar to what may be observed in persons with mTBI [32]. Further, a study using MRS found neuroinflammatory changes among former professional soccer players and not in matched noncontact sports athletes (table tennis players, runners, and ballroom dancers) [33]. Additionally, a study by Lipton and coauthors [34] observed microstructural abnormalities in the temporo-occipital white matter of amateur soccer players that was associated with poorer memory performance on neurocognitive tests. These findings suggest that repetitive sub-concussive head impacts may lead to brain changes detectable by neuroimaging techniques, but further study into the specificity and sensitivity of these techniques is certainly needed.

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## Prevention

Prevention of sports-related concussions needs to be encouraged through further education of players, coaches, and referees, as well as through the use of research-based guidelines by sports health professionals. The teaching of safe athletic techniques, promotion of protective equipment, and encouraging of symptom reporting could further decrease the incidence and consequences of concussions.

Take the sport of football as an example. Mueller and Schindler [140] noted that coaches and referees must do a better job of emphasizing

and enforcing the rules against targeting the head as an initial contact point and tackling head on. The latter rule protects the impacted player by decreasing the contributing torso mass of the tackling player, through a “head-up” stance, resulting in lower effective mass and lower force on the impacted player, presumably lowering the risk of concussion in the player being struck [141]. It is particularly important to decrease the force on the impacted player since a study of NFL athletes found concussions occurred in the impacted and not the tackling players [123]. The difference in force with proper tackling technique translates directly to a difference in peak head acceleration, which was found to be statistically correlated to whether a collision resulted in a concussed or an uninjured player [123].

Other rules, such as the kickoff distance, have also been changed in hopes of reducing the number of concussions reported each season. In 2011 the NFL moved the kickoff spot up 5 yards to cause more touchbacks and less returning of the ball. Kickoff returns are chaotic and one of the most violent plays in football. Due to this rule change, concussions that occurred on kickoffs decreased from 35 in the 2010 season to 20 in the 2011 season [142]. Currently college teams, such as those in the Ivy League, are implementing non-contact practices to reduce the amount of injuries players incur by hitting their own teammates. Since eliminating tackling at practices, players are experiencing fewer concussions, as well as fewer shoulder and neck injuries. Moreover, players are becoming better at tackling: by focusing on avoiding head collisions, the number of missed tackles in games has fallen by more than half [143].

In addition to adjusting tackling techniques, improved helmet design has been shown to reduce the incidence of concussion in football. Rowson and colleagues [144] explored the impact of helmet design on concussive injury by using helmet-mounted accelerometer arrays to collect head impact data on 1833 collegiate football players. They found that players wearing Revolution helmets sustained significantly fewer concussions per head impact than players wearing VSR4 helmets (3.86 vs. 8.37 concussions per 100,000 impacts, respectively). Since the

Revolution helmets allow less head acceleration post-impact than the VSR4 helmets, designs to minimize head acceleration may help to protect against concussive injury [144]. It is important to note that, in this study, several authors had a financial interest in the instrumentation used to collect the biomechanical data.

Like helmets, the use of headgear has been suggested to decrease the incidence of concussions. In the sport of soccer, for example, athletes who have previously sustained concussions, goalies, and children have been advised to wear headgear during practices and games [145]. A cross-sectional study of soccer players ages 12–17 years found that a total of 47.8% of players might have sustained a concussion (based upon self-report of symptoms which was extrapolated to a diagnosis of concussion based upon the symptoms) during a single season. Out of these athletes, 52.8% were not wearing headgear and 26.9% were wearing headgear [146]. Further research is needed to deduce the ability of headgear to decrease the incidence of concussions in non-helmet wearing athletes.

Another approach to preventing sports-related concussions may lie in strengthening the neck muscles of athletes. In a single study of 6704 high school athletes, Collins C.L. and colleagues [147] found that overall neck strength served as a significant predictor of concussion, even after adjusting for gender and sport. In fact, the odds of concussion decreased by 5% for every 1 pound increase in neck strength. By measuring neck strength, one might be able to identify athletes at a higher risk of concussion and use this information to both educate players and coaches and inform strength-training regimens.

To prevent worsened symptoms or second injuries post-concussion, athletes need to be immediately removed from play and evaluated by a qualified health professional. To do this, players need to be educated on how to recognize symptoms suggestive of a concussion as well as encouraged to report suspected injuries to coaches and other players. Kroshus and coauthors [40] explored concussion reporting in 328 male and female collegiate athletes and found that almost half of those surveyed reported continuing to play

in a game or practice despite experiencing post-impact symptoms consistent with a possible concussion. Moreover, one-quarter of those surveyed reported being pressured by their coaches, teammates, fans, or parents during the previous year to continue playing after a head impact [40]. Clearly, progress needs to be made in encouraging athletes to report their symptoms immediately and in creating an environment of health advocacy within athletics, not only at the collegiate and professional levels but also in youth, elementary, middle, and high school levels of play.

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## Areas for Future Research

Public interest in sports concussions has increased research in the area; however, many details about the mechanisms, etiologies, and best treatments of concussive injuries remain understudied. Both large-scale studies and anecdotal evidence from practitioners indicate that the great individual variability of the human brain significantly contributes to differences in concussion incidence and resolution. Further research will do best to explore the effect of specific comorbidities, as well as hereditary and environmental factors, on an individual's risk for and recovery from concussion. Screening has improved to allow practitioners to better assess injury on the sidelines, to request additional neurocognitive testing, and to supervise RTP regimens. For any tests to be useful, however, they must continue to be validated in different populations and to incorporate new technologies. Otherwise, some tests may remain inapplicable for a wide population of athletes or difficult to either administer or evaluate. As research continues to improve advanced neuroimaging of concussive injury, these modalities will also begin to play a role in clinical care, helping to improve the treatment and prognosis of concussive injury.

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## Conclusions

The complex and wide-ranging presentation of concussions make the study and care of concussed athletes an important issue for the medical

community. With recent studies elucidating the potential long-term effects and increased future risks caused by concussive injuries, it is our hope that increased awareness among the public and medical professionals will lead to the evolution and application of evidence-based practices for the diagnosing and treating of concussions. Since concussion is perhaps the single most common form of acquired brain injury in the young and middle-aged, it is imperative that health providers, sports professionals, and athletes themselves develop a better understanding of the risks, prevention, diagnosis, and treatment of sports concussions.

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