American Medical Society for Sports Medicine Position Statement: Concussion in Sport

Kimberly G. Harmon, MD,[1] Jonathan Drezner, MD,[1] Matthew Gammons, MD,[2] Kevin Guskiewicz, ATC, PhD,[3] Mark Halstead, MD,[4] Stan Herring, MD,[1] Jeff Kutcher, MD,[5] Andrea Pana, MD,[6] Margot Putukian, MD,[7] and William Roberts, MD[8]

Key Words: concussion, athlete, head injury, brain injury

(*Clin J Sport Med* 2013;23:1–18)

Executive Summary

Purpose of the Statement

- To provide an evidence-based, best practices summary to assist physicians with the evaluation and management of sports concussion.
- To establish the level of evidence, knowledge gaps, and areas requiring additional research.

Importance of an AMSSM Statement

- Sports medicine physicians are frequently involved in the care of patients with sports concussion.
- Sports medicine physicians are specifically trained to provide care along the continuum of sports concussion from the acute injury to return-to-play decisions.
- The care of athletes with sports concussion is ideally performed by healthcare professionals with specific training and experience in the assessment and management of concussion. Competence should be determined by training and experience, not dictated by specialty.
- While this statement is directed toward sports medicine physicians, it may also assist other physicians and healthcare professionals in the care of patients with sports concussion.

Definition

- Concussion is defined as a traumatically induced transient disturbance of brain function and involves a complex pathophysiologic process. Concussion is a subset of mild traumatic brain injury that is generally self-limited and at the less severe end of the brain injury spectrum.

Submitted for publication November 1, 2012; accepted November 21, 2012.

From the [1]University of Washington, Seattle, Washington; [2]Vermont Orthopaedic Clinic, Rutland, Vermont; [3]University of North Carolina at Chapel Hill, North Carolina; [4]Washington University, St Louis, Missouri; [5]University of Michigan, Ann Arbor, Michigan; [6]University of Texas, Austin, Texas; [7]Princeton University, Princeton, New Jersey; and [8]University of Minnesota, Minneapolis, Minnesota.

Address for correspondence: Kimberly G. Harmon, MD, Family Medicine and Orthopaedics and Sports Medicine, University of Washington, Box 35440, Seattle, WA 98195 (kharmon@u.washington.edu).

Pathophysiology

- Animal and human studies support the concept of postconcussive vulnerability, showing that a second blow before the brain has recovered results in worsening metabolic changes within the cell.
- Experimental evidence suggests the concussed brain is less responsive to usual neural activation, and when premature cognitive or physical activity occurs before full recovery the brain may be vulnerable to prolonged dysfunction.

Incidence

- It is estimated as many as 3.8 million concussions occur in the US per year during competitive sports and recreational activities; however, as many as 50% of concussions may go unreported.
- Concussions occur in all sports with the highest incidence in football, hockey, rugby, soccer, and basketball.

Risk Factors for Sports-related Concussion

- A history of concussion is associated with a higher risk of sustaining another concussion.
- A greater number, severity, and duration of symptoms after concussion are predictors of a prolonged recovery.
- In sports with similar playing rules, the reported incidence of concussion is higher in females than males.
- Certain sports, positions, and individual playing styles have a greater risk of concussion.
- Youth athletes may have a more prolonged recovery and are more susceptible to a concussion accompanied by a catastrophic injury.
- Preinjury mood disorders, learning disorders, attention deficit disorders (ADD/ADHD), and migraine headaches complicate diagnosis and management of concussion.

Diagnosis of Concussion

- Concussion remains a clinical diagnosis ideally made by a healthcare provider familiar with the athlete and knowledgeable in the recognition and evaluation of concussion.
- Graded symptom checklists provide an objective tool for assessing a variety of symptoms related to concussions, while also tracking the severity of those symptoms over serial evaluations.
- Standardized assessment tools provide a helpful structure for the evaluation of concussion, although limited validation of these assessment tools is available.

'Sideline' Evaluation and Management

- Any athlete suspected of having a concussion should be removed from play and assessed by a licensed healthcare provider trained in the evaluation and management of concussion.
- Recognition and initial assessment of concussion should be guided by a symptom checklist, cognitive evaluation (including orientation, past and immediate memory, new learning, and concentration), balance tests, and further neurologic physical examination.
- While standardized sideline tests are a useful framework for examination, the sensitivity, specificity, validity, and reliability of these tests among different age groups, cultural groups, and settings is largely undefined. Their practical usefulness with or without an individual baseline test is also largely unknown.
- Balance disturbance is a specific indicator of concussion but is not very sensitive. Balance testing on the sideline may be substantially different than baseline tests because of differences in shoe/cleat type or surface, use of ankle tape or braces, or the presence of other lower extremity injury.
- Imaging is reserved for athletes where intracerebral bleeding is suspected.
- There is no same-day return to play for an athlete diagnosed with a concussion.
- Athletes suspected or diagnosed with concussion should be monitored for deteriorating physical or mental status.

Neuropsychological Testing

- Neuropsychological tests are an objective measure of brain-behavior relationships and are more sensitive for subtle cognitive impairment than clinical exam.
- Most concussions can be managed appropriately without the use of neuropsychological testing.
- Computerized neuropsychological testing should be interpreted by healthcare professionals trained and familiar with the type of test and the individual test limitations, including a knowledgeable assessment of the reliable change index, baseline variability, and false positive and false negative rates.
- Paper and pencil neuropsychological tests can be more comprehensive, test different domains, and assess for other conditions that may masquerade as or complicate assessment of concussion.
- Neuropsychological testing should be used only as part of a comprehensive concussion management strategy and should not be used in isolation.
- The ideal timing, frequency, and type of neuropsychological testing have not been determined.
- In some cases, properly administered and interpreted neuropsychological testing provides added value to assess cognitive function and recovery in the management of sports concussions.
- It is unknown if use of neuropsychological testing in the management of sports concussion helps prevent recurrent concussion, catastrophic injury, or long-term complications.
- Comprehensive neuropsychological evaluation is helpful in the postconcussion management of athletes with persistent symptoms or complicated courses.

Return to Class

- Students will require cognitive rest and may require academic accommodations such as reduced workload and extended time for tests while recovering from concussion.

Return to Play

- Concussion symptoms should be resolved before returning to exercise.
- A return-to-play progression involves a gradual, stepwise increase in physical demands, sports-specific activities, and the risk for contact.
- If symptoms occur with activity, the progression should be halted and restarted at the preceding symptom-free step.
- Return to practice/play after concussion should occur only with medical clearance from a licensed healthcare provider trained in the evaluation and management of concussion.

Short-term Risks of Premature Return to Play

- The primary concern with early return to play is decreased reaction time leading to increased risk of repeat concussion or other injury and prolongation of symptoms.

Long-term Effects

- There is increasing concern that head impact exposure and recurrent concussions contribute to long-term neurological sequelae.
- Some studies have suggested an association between prior concussions and chronic cognitive dysfunction. Large-scale, epidemiological studies are needed to more clearly define risk factors and causation of any long-term neurological impairment.

Disqualification from Sport

- There are no evidence-based guidelines for disqualifying/retiring an athlete from sport after concussion. Each case should be carefully deliberated and an individualized approach to determining disqualification taken.

Education

- Greater efforts are needed to educate involved parties including athletes, parents, coaches, officials, school administrators, and healthcare providers to improve concussion recognition, management, and prevention.
- Physicians should be prepared to provide counseling regarding potential long-term consequences of concussion and recurrent concussion.

Prevention

- Primary prevention of some injuries may be possible with modification and enforcement of the rules and fair play.
- Helmets, both hard (football, lacrosse, and hockey), and soft (soccer, rugby), are best suited to prevent impact injuries (fracture, bleeding, laceration, etc) but have not

been shown to reduce the incidence and severity of concussions.
- There is no current evidence that mouth guards can reduce the severity of or prevent concussions.
- Secondary prevention may be possible by appropriate return-to-play management

Legislation

- Legislative efforts provide a uniform standard for scholastic and nonscholastic sports organizations regarding concussion safety and management.

Future Directions

- Additional research is needed to validate current assessment tools, delineate the role of neuropsychological testing, and improve identification of those at risk of prolonged postconcussive symptoms or other long-term complications.
- Evolving technologies for the diagnosis of concussion, such as newer neuroimaging techniques or biologic markers, may provide new insights into the evaluation and management of sports concussion.

BACKGROUND AND PURPOSE

The recognition and management of concussion in sport is an evolving and controversial topic with a myriad of groups and organizations producing statements and recommendations.[1–6] The purpose of this statement is to provide an evidence-based, best practices summary to assist physicians with the evaluation and management of sports-related concussion and to establish the level of evidence, knowledge gaps, and areas requiring additional research. The American Medical Society for Sport Medicine (AMSSM) represents over 2100 nonsurgical sports medicine physicians who have completed additional training in sports medicine after a residency program in family medicine, internal medicine, pediatrics, emergency medicine, or physical medicine and rehabilitation, many of whom have extended expertise in concussion evaluation and management.

Sports medicine physicians are frequently involved in the care of patients with sports concussion and are specifically trained to provide care along the continuum of sports concussion from the acute injury to return-to-play decisions. The care of athletes with sports-related concussions is ideally performed by healthcare professionals with specific training and experience in the assessment and management of concussion. Competence should be determined by training and experience, not dictated by specialty. While this statement is directed towards sports medicine-trained physicians, it may also be used by other physicians and healthcare professionals to improve the care of patients with sports-related concussion.

Level of Evidence

This statement uses the Strength of Recommendation Taxonomy (SORT) to grade recommendation based on athlete outcomes (Table 1).[7]

TABLE 1. Strength-of-Recommendation Taxonomy (SORT)

Strength of Recommendation	Basis for Recommendation
A	Consistent, good-quality patient-oriented evidence
B	Inconsistent or limited-quality patient-oriented evidence
C	Consensus, disease-oriented evidence, usual practice, expert opinion, or case series for studies of diagnosis, treatment, prevention, or screening

Definition of Concussion

Concussion is defined as a traumatically induced transient disturbance of brain function and is caused by a complex pathophysiologic process. Concussions have also been referred to as mild traumatic brain injuries (MTBI). While all concussions are MTBIs, not all MTBIs are concussions. Concussions are a subset of mild traumatic brain injury on the less severe end of the brain injury spectrum and are generally self-limited in duration and resolution.

Pathophysiology

Concussions occur when linear and/or rotational forces are transmitted to the brain. Currently, there is no known biomechanical threshold for a clinical concussion. A demonstrated cellular process, the "neurometabolic cascade" underlying the clinical presentation of concussive injury, describes a complex cascade of ionic, metabolic, and pathophysiological events that is accompanied by microscopic axonal injury.[8–10] This disruption of ionic balance and normal metabolism requires energy to reestablish homeostasis. However, the need for increased energy occurs in the presence of decreased cerebral blood flow and ongoing mitochondrial dysfunction, resulting in a mismatch of energy supply and demand.[8,10,11] Until normal brain cellular function is restored, animal and human studies support the concept of increased postconcussive vulnerability, showing that a second injury before the brain has recovered results in worsening cellular metabolic changes and more significant cognitive deficits.[8,9,11–16] Experimental evidence further suggests the concussed brain is less responsive to physiological neural activation.[9,10] Thus, excessive cognitive or physical activity before full recovery may result in prolonged dysfunction. Some of these pathophysiological perturbations are more pronounced in youth, raising concerns that the immature brain may be even more susceptible to repeat concussion before full recovery.[9]

Reported Incidence of Sports-related Concussion

Concussions occur commonly in helmeted and nonhelmeted sports, and recent data suggest a trend of increased annual concussion rates over the past decade.[17,18] Reasons for the apparent increased incidence are unknown, but it is widely speculated to be a result of the emphasis on concussion education and awareness leading to increased identification and reporting.[17,18] Despite the increased reported incidence of concussion in recent years, there has not been a corresponding increase in the incidence of sports-related catastrophic brain injuries such as subdural and epidural hematomas or malignant cerebral edema (ie, Second Impact Syndrome).

The Centers for Disease Control and Prevention (CDC) estimates that between 1.6 and 3.8 million sports-related concussive injuries occur annually in the United States[19] and account for 5% to 9% of all sport injuries.[20,21] Thirty percent of all concussions in individuals between 5 to 19 years of age are sport related and result in a significant number of emergency room visits.[6,22] The majority of concussions occurring in organized sports in the United States are sustained in football, wrestling, girls' soccer, boys' soccer, and girls' basketball. [20,21,23,24] (Table 2) Competition concussion rates are consistently higher than practice rates, and in high school and college sports with the same rules (basketball and soccer) there is an increased incidence of concussion reported in female athletes. [20,23,25] Several studies contend the true incidence is likely higher than documented because many athletes fail to report concussions.[26–28] With greater focus on concussion awareness and state legislation, the reported incidence is likely to continue to increase.

Signs and Symptoms

There are many signs and symptoms that can be observed with a concussion (Table 3). Headache is the most common reported symptom, with dizziness the second most common.[6,25,29,30] Loss of consciousness only occurs in about 10% of concussions.[5,6,30–34] Several symptoms of concussion are nonspecific, eg, nausea, vomiting, and headache are a common presentation of acute gastroenteritis, and dizziness is a common symptom of acute cardiac compromise. Some symptoms overlap with other disorders such as sleep disturbances, depression, and attention deficit disorder and it is helpful to determine whether these symptoms were present prior to the injury. (C) In college athletes, 59% reported concussion-like symptoms in the prior year with no history of head injury, and 50% to 84% of high school athletes reported similar symptoms of concussion at baseline testing.[34–36] There have been no consistently demonstrated differences in the symptoms reported between males and females.[34–39]

Most studies report that 80% to 90% of athletes will have symptom resolution by 7 days following their injury,[6,25,29,32,39] although symptom resolution may not always indicate a full cognitive recovery as persistent deficits may be present on neuropsychological testing.[32,40] However, the clinical importance of persistent neuropsychological testing changes in the absence of continued symptoms is unknown.

Risk Factors/Modifiers for Sports-related Concussion

A history of prior concussion, a greater number, severity or duration of symptoms after concussion, female sex, genetic predisposition, a history of a learning disorder, attention deficit disorder, migraines, or mood disorder, and playing certain positions have all been suggested to affect the risk of sustaining a concussion or having a more protracted course.

Previous Concussion

A history of concussion is associated with a 2 to 5.8 times higher risk of sustaining another concussion.[24,38,41–46] Athletes with a prior history of concussion may also report more symptoms at baseline than those without a history of concussion.[36,47–49] However, there is conflicting evidence on whether a prior concussion is associated with a prolonged recovery course.[50,51] Lau found no difference in history of concussion and time to recovery, while Slobounov demonstrated significantly slower recovery rates of neurological functions after a second concussion.[50,51] As with other sports injuries, the greatest risk factor for concussion is a previous concussion, and progressively prolonged symptoms with subsequent concussions is a concerning prognostic sign.

Number, Severity, or Duration

A greater number, severity, and duration of symptoms after concussion are predictors of a prolonged recovery.[47,51–53] Specific signs or symptoms may also predict recovery time. Dizziness at the time of injury was found to be the greatest predictor in high school football players for a recovery taking longer than 21 days,[50,54] and athletes who had more symptoms in the cognitive or migraine symptom clusters often required more recovery time.[50] In rugby players, headaches lasting longer

TABLE 2. Concussion Rates Per 1000 Athlete Exposures

Sport	Powell (1999) High School 1995-1997	Schultz (2004) High School 1996-1999	Hootman (2007) College 1988-2004	Gessel (2007) High School 2005-2006	Lincoln (2011) High School 1997-2008	Marar (2012) High School 2008-2010
Baseball	0.05	0.11	0.07	0.05	0.06	0.05
Softball	0.10	0.10	0.14	0.07	0.11	0.16
Boys' basketball	0.11	0.10	0.16	0.07	0.10	0.16
Girls' basketball	0.16	0.17	0.22	0.21	0.16	0.21
Boys' soccer	0.18	0.23	0.28	0.22	0.17	0.19
Girls' soccer	0.23	0.13	0.41	0.36	0.35	0.34
Football	0.59	0.33	0.37	0.47	0.60	0.64
Field hockey	0.09	NR	0.18	NR	0.10	0.22
Volleyball	0.02	NR	0.09	0.05	NR	0.06
Wrestling	0.25	0.09	0.25	0.18	0.17	0.22
Ice hockey			0.41			0.54
Overall		0.17	0.28	0.43	0.24	.024

Nr, Not reported.

TABLE 3. Signs and Symptoms of a Concussion

Physical
Headache
Nausea
Vomiting
Balance problems
Dizziness
Visual problems
Fatigue
Sensitivity to light
Sensitivity to noise
Numbness/tingling
Dazed
Stunned
Cognitive
Feeling mentally "foggy"
Feeling slowed down
Difficulty concentrating
Difficulty remembering
Forgetful of recent information and conversations
Confused about recent events
Answers questions slowly
Repeats questions
Emotional
Irritable
Sadness
More emotional
Nervousness
Sleep
Drowsiness
Sleeping more than usual
Sleeping less than usual
Difficulty falling asleep

than 60 hours, 3 or more symptoms at initial presentation, and the presence of fatigue/tiredness/fogginess were associated with a longer recovery.[29]

Sex

Recent data suggests that in sports with similar rules females sustain more concussions than their male counterparts.[20,23,25,55,56] In addition, females experience or report a higher number and severity of symptoms as well as a longer duration of recovery than males in several studies.[38,55–59] Decreased head-neck segment mass of females compared to male athletes may contribute to greater angular acceleration of the head after concussive impact as a mechanism for more severe injury.[60] Estrogen and differential cerebral blood flow may also play a role in influencing concussion severity and outcome.[15,61] Further study is needed to understand if sex is a risk factor for concussion and what mechanisms may account for it, or if sex is merely a predictor of symptom reporting.[62]

Age

Youth athletes may have a more prolonged recovery and are more susceptible to concussion accompanied by catastrophic injury. The developing brain differs physiologically from the adult brain when comparing the brain water content, degree of myelination, blood volume, blood-brain barrier, cerebral metabolic rate of glucose, blood flow, number of synapses, and geometry and elasticity of the skull's sutures.[63] Developmentally younger brains have less established engrams and may have less cognitive reserve than more mature brains.[9,64] This may account for the demonstrated increase in time to recovery from concussion seen in younger athletes.[65–68] It is difficult to compare studies at different levels of play (high school, college, and professional) as longer recovery times could reflect differences in study methodology, in risk tolerance and return-to-play protocols, or all of the above. Recovery patterns have not been adequately studied in athletes less than 15 years old. Catastrophic injury is more likely in younger athletes and is hypothesized to be related to the physiologic differences between younger and older brains.[69–71]

Sport, Position, and Style of Play

Certain sports, positions, and individual playing styles have a greater risk of concussion. The rate of concussion also varies by level of play. Position and style of play also appear to affect the risk of concussion. Mechanisms of concussive injury may vary based on the sport as well as the level of play. The most common mechanism of concussion is player-to-player contact.[25] It is not surprising, therefore, that sports and positions involved in frequent collision impacts sustain more concussions. Studies on professional football players have shown that "backs" (quarterbacks, wide receivers, running backs, and defensive backs) have a 3 times greater risk of concussion than "linemen,"[72] and kickoffs had a 4 times higher risk of concussion than rushing or passing plays.[72] In high school football players, linebackers were the most commonly concussed on the defense and running backs on the offense. In soccer players, concussions most commonly occur from player contact both at the high school level and at the college level.[73–75] At the high school level, 1 study demonstrated that 25.3% of concussions were associated with illegal activity. In a prospective study of college soccer players, the mechanism of concussion was again primarily player contact, and importantly none were related to purposeful heading.[76] In hockey the most common mechanism of concussive injury is checking.

Genetics

Studies on the association between concussion and genetic polymorphisms such as APOE e4, APOE G-219T promoter, or tau exon 6 are limited by small sample sizes, limited sports populations, retrospective study design, use of self-reported concussion history, and a lack of control groups.[77,78] Some studies have suggested potential associations, but methodological weaknesses do not support definitive conclusions. A study of college athletes showed prior self-reported concussion was associated with increased odds of having either 1 APOE e4 allele or at least 1 APOE G-219T 'T' allele. In other reports, a cross-sectional study showed college athletes with a self-reported history of concussion

were 2.7 times as likely to have APOE promoter G-219T 'TT' genotype after controlling for various cofounders,[77] and a small prospective cohort study showed no significant association between genotype and concussion risk.[46]

The largest prospective cohort study available (n = 234 athletes with 45 prospective concussions) showed no significant association between APOE, APOE G-219T, tau exon 6 Hist47Tyr, and Tau exon 6 Ser53Pro and concussion risk, although Tau exon 6 Ser53Pro was trending towards significance ($P = 0.09$).[79] A large prospective cohort study of a representative athletic population that controls for athletic exposure, prior concussion history, and other predisposing factors is necessary to determine if polymorphisms confer an increased risk for concussion, more severe concussions, or delayed neurocognitive recovery.

Mood Disorders

Mood disorders, either preexisting or as a result of a concussive episode, complicate both diagnosis and management of concussion. Symptoms of anxiety, depression, or irritability occur in 17% to 46% of high school and college athletes and affect the brain's mood centers including the hippocampus, amygdala, and prefrontal brain regions, which are also affected in concussion.[80,81] There is no evidence that the preexistence of a mood disorder predisposes athletes to concussion. However, when evaluating an athlete it is often difficult to determine which symptoms preceded the concussion, which have been caused by the concussion, and which symptoms are worsened after the concussion. An increased incidence of depression has been associated with a history of concussion among retired boxers and professional football players; however, these retrospective studies relied on a self-reported history and did not control for other factors that may cause depression.[42,82] Anxiety, depression, and other psychological impairments may also affect neuropsychological testing, either at baseline or at repeat testing, complicating test interpretation.[83,84] Knowing preinjury mood status may be beneficial to the evaluation of athletes with subsequent injury. (C)

Learning Disabilities and Attention Disorders

As with other conditions which share common symptomatology with concussion, it is important to take learning disorders into account in both diagnosis and management of concussion. Preinjury learning disabilities and attention deficit disorders (ADD/ADHD) may be associated with increased cognitive dysfunction and prolonged recovery after concussion. Collins found that athletes with learning disabilities and a history of concussion did proportionally worse on selected paper and pencil neuropsychological testing than those without learning disabilities.[85] In 108 athletes with concussion, Lau found there was no association between learning disability or attention deficit disorder and protracted recovery.[54,86,87] Learning and attention disorders share common features of concussion, such as difficulty with memory, attention, and concentration, making the diagnosis and management in these individuals more challenging. Baseline neuropsychological testing scores are lower in those with learning and attention disabilities[85,88] independent of concussion history making baseline testing more important in those with learning or attention disorders if neuropsychological testing is going to be used postinjury to assist in return-to-play decisions.

Migraines

A history of preexisting migraine headaches may be a risk factor for concussion and may be associated with prolonged recovery. 2.9% of NCAA college basketball athletes (0.9% of men and 4.4% of women) and 22% of Australian Rules football players report migraines meeting International Headache Society criteria for diagnosis compared to 10% of the general population.[89–91] An association between concussion and preexisting migraine was shown in 1 retrospective population study,[92] but no association between preexisting migraine and prolonged course of concussion has been demonstrated.[54,86,87]

Concussion can trigger a posttraumatic migraine, and athletes with postconcussion migraine usually have more symptoms and poorer performance on neuropsychological tests than athletes with other types of headache or no headache at all.[93] In addition, Lau found that athletes (without preexisting diagnosis of migraines) who developed symptoms in the "migraine symptoms complex", which included headaches, visual problems, dizziness, noise/light sensitivity, nausea/vomiting, balance problems, and numbness/tingling, had a more protracted recovery.[54] Similar to mood, learning, and attention disorders, it is important to understand preinjury cognitive or psychological disorders in order to optimize management.

Management of Concussion

Preseason

Preparation for the care of concussed athletes begins prior to any practice or competition with a preparticipation exam (PPE) and the development of an emergency action plan. (C) The preparticipation exam should include concussion-related questions including a past history of concussion (number, frequency, severity, and recovery) and the presence of mood, learning, attention, or migraine disorders.[94] (C) This information can be used to assess risk and for historical reference in the case of injury.

The exact role and impact on concussion management of baseline testing remains unclear, as no study has shown that use of these tests provides better short- or long-term outcomes for athletes with concussion. The preseason evaluation may also include baseline symptom scores, baseline balance testing, a baseline sideline evaluation tool (Sport Concussion Assessment Tool 2 [SCAT2], NFL Sideline Concussion Assessment Tool), and/or baseline computerized neuropsychological (NP) testing. This baseline testing may be more important in high-risk athletes with a prior history of concussion, with confounding conditions (learning disability, mood and attention disorders, migraine headaches) and sports with a higher incidence of concussion.[95,96] (C)

The reliability of preseason testing as a dependable baseline assessment to compare with postinjury testing performed weeks or months later is also controversial and for many tests unknown. While baseline testing is increasingly used in practice and may have a role in the preseason evaluation of high risk athletes, the role of baseline tests in

other settings is unknown.[97] (C) More research is needed to define which baseline tests should be performed and in which athletes.

Preseason testing requires honest effort on the part of an athlete. Balance testing is time intensive, but can be done by nonphysician personnel. Computerized NP testing requires adequate resources and a quiet environment for best results, but can be done in large groups. Neuropsychological testing does require health professionals who are competent in test interpretation.

On-Field Management

The first step in assessing a collapsed athlete is the check for airway, breathing, and heart function, followed by a physical evaluation to exclude cervical spine injury and/or more serious brain injury. (C) If cervical spine injury cannot be eliminated, neck immobilization and immediate transfer emergency department capable of advanced neurological imaging and management of cervical trauma should follow. (C) Emergency transfer should also occur if there are signs of a more serious brain injury such as deteriorating mental status, focal neurological findings (abnormal or unequal pupil reaction, abnormalities with extra-ocular movements, abnormalities on a screening motor/sensory exam), or worsening of symptoms. (C) If cervical spine and more serious brain injury can be excluded with history and physical exam, then a more detailed history of injury and an examination that includes symptoms, cognitive and balance assessment, and neurological examination to evaluate for concussion may be initiated. (C)

Sideline Assessment of Concussion

Any athlete suspected of having a concussion should be removed from play and assessed by a licensed healthcare provider trained in the evaluation and management of concussion. (C) It is useful to utilize a standardized approach in evaluating the athlete postinjury. Physical exam is primarily to rule out more serious brain injury and assess balance. History, cognitive testing, and balance testing comprise the majority of the evaluation. Previous medical history is important to understand confounding factors that may be present, such as mood or attention disorders, or orthopedic injury, which may affect the physical examination.

There have been several different sideline concussion assessment tools developed and in some settings the use of these tools is required. Standardized measures are designed to reduce the degree of subjectivity encountered by medical providers responsible for making a rapid and precise injury assessment and concussion diagnosis decision. When possible, sideline tests can be compared to a reliable preinjury baseline. (C) Baseline values of commonly used sideline tests can vary widely from athlete to athlete, and the results are dependent on age, sport, sex, and confounding medical conditions, making the use of sideline tests without baseline results difficult.[35,36,59,98] In addition, baseline tests may change as part of the normal maturation and developmental process and can be dependent on current mood, fatigue, and other factors. Familiarity with an athlete is an important component in the sideline evaluation of concussion, given the variability in presentation. (C)

The most common sideline measures include the use of symptoms scores, the Maddocks Questions, the Standardized Assessment of Concussion (SAC), and the Balance Error Scoring System (BESS) or modified BESS. The SCAT2 and the NFL Sideline Concussion Assessment Tool combine various assessment measures to give one score. The sensitivity and specificity of the diagnosis of concussion may increase when combining multiple assessment tools.[86]

The sensitivity (the likelihood that an athlete with concussion will be correctly identified), specificity (the likelihood that an athlete without a concussion will be correctly classified), and the false positive and false negative rates vary for the different sideline tests and are important to consider in the evaluation of an athlete (Table 4). The sensitivity and specificity of these tests may also change over the course of a concussion. A tool that is appropriate for sideline use may not be appropriate for office use. For example, balance testing typically returns to normal after 3 days, making it a potentially useful sideline test but not as useful for later follow-up. With our current sideline tests, as sensitivity goes up, the specificity goes down, so some athletes without a concussion may be held from return to play. Physicians evaluating concussion on the sideline are encouraged to err on the side of safety – "when in doubt sit them out." More research is needed to validate and improve sideline testing.

Balance Testing

Over the past decade, balance testing has become increasingly utilized in the diagnosis and management of sports-related concussion particularly on the sideline. Studies have identified temporary or permanent deficits in static and/or dynamic balance in individuals with mild-moderate traumatic brain injury (TBI)[99–102] and similar balance deficits have been reported in sports-related concussion.[32,82,103–105]

Balance Error Scoring System (BESS)

The test is easy to administer in 5 to 7 minutes and is inexpensive. Three stances (narrow double leg stance; single leg stance; and tandem stance) and 2 footing surfaces (firm surface/floor or medium density foam) are used for the complete test. Each stance is held, with hands on hips and eyes closed, for 20 seconds. "Error" point deductions are given for specific behaviors, including opening eyes, lifting hands off hips, stepping, stumbling, or falling. There is a maximum score of 60 points if both floor surfaces are used, or 30 if only 1 surface is used.

McCrea and colleagues[103] reported that BESS scores in concussed college football players varied from baseline an average of -5.7 points when measured immediately following the game or practice in which the injury occurred. Changes in BESS performance and rapid recovery of static balance have been reported in other studies of concussed athletes.[105–107] For most athletes, BESS performance returned to preseason baseline levels (average 12 errors) by 3 to 7 days postinjury. Sensitivity values for the BESS are reported as highest at the time of injury (sensitivity = 0.34). Specificity values for this instrument ranged from 0.91 to 0.96 across postinjury days 1 to 7.[32] One study questioned the clinical utility of the BESS suggesting it has low interrater and intrarater reliability, and that the minimum detectible change for the total

TABLE 4. Sideline Concussion Evaluation Tests

Test	Time to Administer, mins	Sensitivity, %	Specificity, %	False Positives, %	False Negatives, %
Symptom Scores	2–3				
Broglio (2007) 9-item		68			32
McCrea (2005) 17-item		89	100	0	11
Maddocks	<1				
CJSM (1995)		32-75	86-100	29-68	0-11
SAC	5				
Barr (2001)		94	76	24	6
McCrea (2005)		80	91	9	20
BESS	5				
McCrea (2005)		34	91	66	9
Modified BESS	2–3	unknown	unknown	unknown	unknown
SAC + BESS	10	unknown	unknown	unknown	unknown
NFL Sideline Concussion Assessment Tool (SAC+modified BESS+Symptoms score)	8-10	unknown	unknown	unknown	unknown
SCAT2 (SAC+ modified BESS+Glascow coma scale+Physical signs score +Maddock's score+coordination exam)	8-10	unknown	unknown	unknown	unknown

BESS score is between 7 and 9 points making its clinical use questionable.[108] Another study showed 42% of players without a clinically diagnosed concussion exceeded the reliable change on the BESS when tested after a game.[109]

Both the SCAT2 and the NFL Sideline Concussion Assessment Tool utilize a modified BESS. The modified BESS only includes testing on a firm surface and does not include testing on medium density foam. There are no reported intra- or interrater reliability studies using the modified BESS, no established minimal clinically meaningful change, and no sensitivity and specificity values available. In addition, there are practical issues with using the BESS in a sideline setting. Baseline testing is typically done in a training room on a firm surface in sneakers without tape or ankle braces. Sideline testing is typically done in cleats with ankles taped or braced on grass or a turf field. Despite these limitations, balance is often affected by concussion and should be evaluated when concussion is suspected. Ideally, baseline BESS testing should be done in "game" conditions. (C) More research to refine sideline balance testing is warranted.

The Sensory Organization Test (SOT) (NeuroCom International, Inc, Clackamas, Oregon) is a technical force plate system used to assess balance following concussion but is the gold standard in balance testing in research studies and clinical availability is limited. The SOT has proven to be a useful tool for detecting sensory interaction and balance deficits following concussion.[43,104,105,110,111] Research utilizing the SOT has identified deficits lasting an average of 3 days postinjury, similar to the studies involving clinical balance tests.[104,106] These studies indicate that the regions of the brain responsible for coordinating the sensory modalities (thalamus and its interconnective pathways to the cerebral cortex) may be disrupted postinjury and that the vestibular system is often affected following concussion.[97,99]

Sideline Management and Disposition

When a player is being evaluated for, or has been diagnosed with, a concussion, it is a good safety strategy to sequester an essential piece of playing equipment to avoid an "inadvertent" return to the game. A concussed player should not be left alone if the decision is made to keep the player on site, and regular monitoring for deteriorating physical or mental status is essential. (C) If after a full sideline assessment a concussion is not thought to have occurred and the player is allowed to continue play, serial evaluation should be performed after return to the game to ensure the decision was correct. (C)

Athletes diagnosed with a concussion should not return to play on the same day of practice or competition. (C) Athletes with concussion will need appropriate disposition to home, to remain on site until the end of the contest, or if needed, be transferred to an emergency facility. This is not always an easy or straightforward decision and regular reevaluation is desirable until a final disposition is determined. The medical provider should arrange or discuss the follow-up evaluation with a parent/guardian or *in loco parentis* representative for minor participants. The medical provider should also arrange for the athlete to be accompanied or monitored once allowed to leave the competition area. "Take home" information, ideally in written form, should be discussed with the athlete and any accompanying party, including signs or symptoms that should prompt an emergency room evaluation, avoiding any physical or cognitive exertion that can worsen or mimic signs of concussion, avoiding alcohol, and when to be seen in follow-up. (C)

Common advice previously given to those with concussion such as frequent awakening of the concussed athlete to "make sure they are okay" is no longer recommended. If level of consciousness is a concern, the athlete should be imaged and observed in a hospital setting; otherwise, sleep should not be interrupted, as it is likely restorative. Caretakers

should be informed that it is desirable to let the athlete sleep. Likewise, no data support that postinjury use of anticoagulation increases morbidity or mortality in head trauma;[112–114] however, because of the theoretical risk of bleeding, aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) are generally avoided postconcussion.

Postsideline and Office Management of Concussion

Follow-up Evaluation and Treatment

Athletes with concussion should have medical follow-up. (C) A detailed history of the event mechanism, course of symptoms, and previous history of concussion should be elicited. Serial monitoring of standardized symptoms scores can be helpful to more objectively assess resolution of symptoms or return to their preinjury baseline. In the vast majority of concussions balance disturbances are back to baseline by 3 days.[105–107] Worsening symptoms, pronounced amnesia, progressive balance dysfunction, or focal neurological deficits on examination could be signs of intracranial pathology and should prompt neurological imaging. (C)

The treatment of concussion consists of relative physical and cognitive rest. In the early stages of concussion recovery, an athlete should not engage in physical or cognitive activities that result in an increase in symptoms. (B)

Neuropsychological Testing

Neuropsychological testing in athletes began in the 1980s, and its use has expanded in the last decade with the availability of computerized testing.[2,115–117] Data suggest that cognitive impairment after concussion may last longer than subjective symptoms. Neuropsychological testing is a tool that can identify occult cognitive impairment and may also aid in documenting an athlete's recovery from a concussive injury.[2,5,29,116–120] Although variable by different test type, several domains of cognitive function are evaluated, focusing on those areas most affected by concussion such as memory, cognitive processing speed, and reaction time.[117,121] It is currently unknown if the use of NP testing alters the short-term risks (recurrence or catastrophic injury) or potential long-term complications.[122,123]

There are 2 main types of testing employed: paper and pencil and computerized. Paper and pencil tests are typically administered and interpreted by a neuropsychologist.[2,115] These tests are more comprehensive and have the advantage of testing additional domains, which may identify other conditions masquerading as a concussion or postconcussive syndrome.[121] However, paper and pencil testing is more costly and requires significantly more time to administer.[117,124] Computerized neuropsychological (CNP) testing has advantages in the athletic setting as it is less expensive, takes less time to administer, may be administered concurrently to groups of athletes, provides instant information to the provider, has more precise measures of reaction time, has multiple forms, and may be used for serial assessment.[2,116,117] Both paper and pencil and computerized testing have significant individual variability in regards to domains measured and performance measures such as validity, sensitivity, specificity, reliable change index, and baseline variability.[2,121,124–127]

Testing, particularly CNP, has been shown to have moderate sensitivity in the detection of postconcussive cognitive deficits.[117,118,126,128] Neuropsychological testing has not been validated as a diagnostic tool but has the ability to show cognitive deficits longer than athletes are symptomatic.[2,5,29,116–120,123] Because of this, testing has been promoted as a monitoring tool in the recovery from a concussive event.[4,5] Intuitively, baseline testing appears to have advantages over comparative normal values, but no studies have looked at this issue with regard to outcomes, and there is limited data to suggest age-related norms may be adequate to assist with management decisions.[97,129] No ideal interval for repeating baseline testing has been established for NP or CNP testing.[117,130,131] Test-retest reliability studies of currently available NP tests show baseline testing may need to be repeated, but an ideal interval has not been established.[98,119,125,127,132,133] Additionally, many intrinsic and extrinsic factors can affect performance on NP testing, particularly CNP testing.[116,130,131] In 1 study of college football players without a diagnosis of concussion, CNP testing 48 hours postgame showed that 75% exceeded reliable change in 1 category and 25% exceeded reliable change in 2 categories.[109] If testing is used, care should be taken to make the baseline and postinjury physiologic variables (ie, fatigue) and environmental variables (ie, distractions) as similar as possible.[115,116] Other conditions such as age, effort,[134] sex, cultural background, primary language, mood disorders, migraines, and history of concussion also can affect testing results.[2,67,115–117,124,125,135–139] No optimum postconcussion monitoring interval has been established and reported intervals vary from every few days to only testing asymptomatic athletes prior to return to sports.[2,5,126] No studies have evaluated these intervals in regard to long-term sequelae or recurrent concussive risk.

There are no universally agreed upon recommendations for use of NP testing, and existing recommendations are based on expert opinion. There are several potential reasons to consider testing. In high-risk athletes with prior concussion, testing adds additional information that may help with return-to-play decisions.[4,5] In athletes who may deny symptoms in order to return to play sooner, testing may play a role to identify athletes with persistent deficits.[119] It may also aid in the medico-legal or policy considerations in the management of these athletes.

Neuropsychological testing is only an adjunct to clinical assessment and judgment. (C) In the majority of cases, concussions can be managed appropriately without the use of NP testing. (C) However, testing may have added value in some settings, especially high-risk athletes. Neuropsychological testing should be interpreted by a healthcare professional trained and familiar with the type of test and the individual limitations of each test. (C) Neuropsychological testing should be used only as part of a comprehensive concussion management strategy and should not be used in isolation. (C)

Symptom Management

The appropriate management of concussion symptoms requires careful consideration of the timing and natural history of the injury. In the acute setting (0-10 hours postonset

of injury), drugs that could alter mental status, such as benzodiazepines, should be avoided. (C) After this acute phase, medications may be considered for symptomatic relief, although those that affect the central nervous system, such as stimulants, certain anti-nausea medications, and anti-depressants, should be used with caution as they may cloud the neurological and cognitive examination and impair the physician's ability to assess the injury. (C) When the patient is being considered for return to participation, medications that may mask symptoms of concussion must be avoided. During the entire course of the injury, activities and environmental conditions that exacerbate symptoms should be moderated appropriately.

There is no convincing evidence that any particular medication is effective in treating the acute symptoms of sports concussion specifically. Treatment should be based on common approaches to each specific symptom. (C) Symptoms such as headache, sleep difficulty, and depression may either be direct results of concussion or may represent exacerbations of preexisting conditions.

In the acute setting, treatment options for headache are limited. Acetaminophen offers possible benefit without significant increased bleeding risk. In general, aspirin (ASA) and NSAIDs are not recommended as discussed previously. Physical modalities (ie, massage, ice, contrast therapy, manual therapy) may be considered, especially if there is concurrent neck pain. A dim, quiet environment may moderate head pain, as well as symptoms of photophobia and phonophobia. If headache continues after 3 to 4 days, abortive treatment should be tailored to headache type (ie, migraine, tension, occipital neuralgia, etc). (C) Similarly, prophylactic treatment of a new or exacerbated chronic or recurrent headache syndrome should be tailored appropriately. Headaches that continue as part of a postconcussion syndrome (symptoms lasting longer than 6 weeks) often require a multidisciplinary approach.[140]

Sleep disturbance is a common and important symptom experienced throughout the course of concussion.[141] Immediately after concussion, patients may experience either increased sleep latency and frequent wakening, or decreased latency and longer sleep times. In either case, sleep issues in the first few days following injury onset should be addressed conservatively, without medications, and with particular attention to good sleep hygiene. Excessive daytime somnolence is also common, regardless of absolute sleep times. There is no evidence to support a role for stimulant- or sleep-promoting medications in the acute setting. Sleep difficulties may continue as part of a postconcussion syndrome. In these cases, both medical and cognitive therapies may be considered.

Alteration in mood is also a common manifestation of concussion, particularly in the acute setting.[142] While depression is perhaps the most common, any mood disturbance is possible. There is no established role for medications in the treatment of a concussion-induced mood disturbance. If mood issues persist beyond 6 to 12 weeks, either as part of a postconcussion syndrome, or as manifestations of an exacerbated mood disorder, treatment with medications and/or cognitive therapy should be considered. (C)

Cognitive symptoms, especially decreased attention, may also produce significant morbidity. There is no established role for stimulant medications in the treatment of acute attention difficulties following concussion. (C) Decreasing academic responsibilities and other cognitive demands should be considered for any significant decrease in cognitive performance.[143]

Symptoms of balance dysfunction and vertigo should be carefully evaluated prior to treatment. Medications such as meclizine or diazepam may be helpful for acute attacks of vertigo, but should be used cautiously early in concussion management as they may affect cognitive function, cause fatigue, and obscure the evaluation of concussion resolution.[144] Although only limited evidence exists, vestibular therapy may be considered for the treatment of dizziness or vertigo.[145] (C)

Return to School

There are no standardized guidelines for returning the athlete to school. If the athlete develops increased symptoms with cognitive stress, student-athletes may require academic accommodations such as a reduced workload, extended test taking time, days off, or a shortened school day.[146] Some athletes have persistent neurocognitive deficits following concussion, despite being symptom free. Consideration should be made to withhold an athlete from contact sports if they have not returned to their 'academic baseline' following their concussion.[126] (C) The CDC developed educational materials for educators and school administrators that are available at no cost and can be obtained via the CDC Web site. Additional resources for academic accommodations should be developed for both clinicians and educators.[147] (C)

Return to Play

Return to play after concussion should be individualized, gradual, and progressive and should consider factors that may affect individual risk and outcome.[4,15,29,43,58,95,148] (C) The athlete should be free of concussion symptoms at rest as well as during and after exertion before returning to full participation. (C) The athlete should also have a normal neurologic exam including a normal cognitive and balance evaluation, ideally compared to a preinjury baseline. (C) Once the athlete is asymptomatic and has returned to their baseline measures (if available), a gradual and medically supervised incremental return to activity should be initiated and includes a step-wise increase in physical demands, sports-specific activities, and physical contact. This progression may take days to weeks to months depending on the individual responses and modifying circumstances. It is important to consider individual factors that are suspected to increase susceptibility and/or prolong recovery after concussive injury. The severity of a concussion is based on the nature, burden, and duration of symptoms, the frequency and past history of concussions, and the presence of prolonged symptoms. All should all be considered when determining the symptom-free interval required prior to starting the return-to-play progression. (C)

The return-to-play progression should be individualized, with symptoms and cognitive and balance exams used to track recovery. (C) A typical return-to-play progression for an uncomplicated concussion is shown in Table 5. If the athlete develops symptoms with an increase in activity level, the progression should be stopped and the athlete returned to the previous phase when symptom free again. (C) The return-to-play decision is individualized for the specific

TABLE 5. Graduated Return-to-Play Protocol

Rehabilitation Stage	Objective of Stage
No activity	Recovery
Light aerobic exercise	Increase heart rate
Sport specific exercise	Add movement
Non-contact training drills	Exercise, coordination, and cognitive load
Full-contact practice	Restore athlete's confidence; coaching staff assesses functional skills
Return to play	

circumstances of each concussion.[15,148] A final return-to-practice/play determination should occur with documented medical clearance from a licensed healthcare provider trained in the evaluation and management of concussion. (C)

Neuroimaging

The vast majority of athletes with sports-related concussion do not require neuroimaging.[149–154] Standard neuroimaging with computed tomography (CT) or magnetic resonance imaging (MRI) is negative in concussion but are used to evaluate for more serious brain injury.[4,95] Computer tomography is best used acutely for evaluating for bony fracture and for intracranial bleeding, contusion, mass effects, and/or brain stem herniation, whereas MRI is more sensitive for evaluating persistent or worsening symptoms or concern for underlying pathology (eg, headache or seizure disorder, arteriovenous malformation, Chiari malformation, etc).[155] Computed tomography exposes the brain to radiation and should be used judiciously. Magnetic resonance imaging technology has improved with stronger magnets and different techniques that can detect minor abnormalities after concussion, although the clinical relevance of the findings are unclear.

Other imaging techniques used primarily in concussion research include positron emission tomography (PET) and single-photon emission computed tomography (SPECT) imaging which measure cerebral glucose uptake and regional cerebral blood flow, respectively. Both PET and SPECT incorporate injected radioisotope, are time consuming, and have undetermined predictive value, which limits their clinical use.

Recent research is evaluating new neuroimaging techniques after sports-related concussion including functional MRI (fMRI), diffusion tensor imaging (DTI) and MR spectroscopy (MRS). Functional MRI demonstrates neuronal dysfunction by measuring regional changes in blood oxygenation patterns measured in response to a specific task that the individual performs while in the scanner. The information obtained is totally dependent on the tasks being performed (eg, working memory, sensorimotor coordination, or visuospatial memory). Abnormal patterns of activation have been noted in several studies of sports-related concussion.[155–163]

Diffusion tensor imaging provides structural images of white matter fiber tracts of the brain by measuring the movement of water within the brain. Fractional anisotropy is a measure of the relative directionality of water diffusion in axons, and in healthy individuals the direction is organized and in a specific direction (anisotropy). Both fractional anisotropy and mean diffusivity have been shown to change after sports-related concussion, though how these correlate with clinical measures of injury and recovery have not yet been elucidated.[164–173]

MR spectroscopy measures neurometabolites via proton magnetic resonance spectroscopy that are important in the neuropathology of concussive injury. MR spectroscopy typically evaluates N-acetylaspartate, creatinine, choline, myoinositol, and lactate, and preliminary research in sports-related concussions show changes in the ratio of these neurometabolites in different areas of the brain.[13,173,174] The research involving the use of MRS as well as other advanced neuroimaging techniques is evolving rapidly, but currently there are no definitive clinical correlations to make them applicable to patient care.

Short-term Risks Associated with Premature Return to Play

There are potential health risks of returning an athlete with persistent symptoms to play including the possibility of second impact syndrome (SIS) or diffuse cerebral swelling, and increased susceptibility to a recurrent or more severe concussion and prolonged duration of symptoms. Second impact syndrome is described as occurring when an individual sustains a second head injury before the symptoms associated with the initial injury have fully cleared.[175,176] The pathophysiology of SIS is thought to involve a loss of autoregulation of the brain's blood supply, leading to vascular engorgement, marked increase in intracranial pressure, brain herniation, and ultimately coma or death. There is significant debate as to whether SIS is related to a prior head injury or if it represents a separate pathophysiologic malignant brain edema, a form of diffuse cerebral swelling described in children.[177–179] The pathophysiology of diffuse cerebral swelling after first impact is described in laboratory animal models.[180,181] There are also limited cases of SIS reported in the literature, and while rare, SIS is reported as more common in boxers and athletes under the age of 18. The history and pathophysiology of death in some reported cases of SIS do not support the diagnosis. Whether or not a discrete entity of SIS exists, the association with concussion is a compelling reason why an athlete should not return to play before symptoms of their concussion have completely resolved.

Returning an athlete to play with persistent symptoms may predispose an athlete to a worse concussion. (B) Concussion decreases cognitive ability and reaction time,[16,51,103,125,182] which theoretically diminishes an athlete's ability to respond to the demands of the sport, increasing the risk of a second brain impact and injury to other body parts. In addition, early activity after concussion can prolong symptoms and neurocognitive recovery[183] on a pathophysiological basis; studies suggest there is an increased window of brain vulnerability after concussion secondary to impairment of cellular energy metabolism.[9] A second injury before the brain has recovered results in worsening cellular metabolic changes and more significant cognitive deficits in animal laboratory models.[9,11–14,16,184] In humans this may translate to increased susceptibility to concussion, increased morbidity from relatively lower forces, or prolonged symptom duration and neurocognitive recovery.

Prolonged Concussive Symptoms/ Postconcussion Syndrome

While the majority of concussions resolve within 7 to 10 days, in some cases symptoms will persist for weeks, months, or years beyond the initial injury. The evolution of concussion injury to postconcussion syndrome is ill defined and poorly understood. It is difficult to define where concussion ends and postconcussion syndrome begins. The syndrome is simply defined as symptoms and signs of concussion that persist for weeks to months after the incident.[95,185–187] Symptoms of postconcussion syndrome can be subjective or objective and are often vague and nonspecific making the diagnosis difficult. Although any symptom of concussion can be involved, commonly reported symptoms include headache; dizziness; insomnia; exercise intolerance; cognitive intolerance; psychological symptoms such as depressed mood, irritability, and anxiety; cognitive problems involving memory loss, poor concentration, and problem solving; fatigue; or noise and light sensitivity.[81]

While there are many potential causes of postconcussion syndrome, none have been proven or accepted. There is, however, no proven correlation between the severity of the concussion injury and 1) the likelihood of developing persistent postconcussion symptoms, 2) any structural damage to the brain or disruption of neurotransmitter systems, or 3) the presence of psychological factors like depression, anxiety, or posttraumatic stress disorder.[88,143–145] Risk factors for postconcussion syndrome include increasing age, female sex (possibly because women are more likely to seek medical care when they have symptoms), or non–sports-related concussion (car collision, fall, or assault). Compared to other forms of concussion, sports-related concussions seem less likely to result in postconcussion syndrome.[88,143–145]

The foundation of postconcussion syndrome management is time. Recovery from postconcussive syndrome can be a long and slow process that is often frustrating for patients and removes them from their normal endeavors in school and sport. Management of postconcussion syndrome is ideally done by a team of providers who work with concussion on a regular basis. (C) Comprehensive neuropsychological evaluation by a trained neuropsychologist utilizing standardized paper and pencil testing can be helpful to define cognitive deficits and areas of potential neurocognitive rehabilitation. (C) Cognitive therapy,[188] integrated neurorehabiltation programs,[187] and supervised progressive exercise programs may improve recovery.[189] (B) Progressive exercise programs involve exercising to the onset of symptoms, then every other day exercise at 80% of the symptom threshold, with retesting and progression of activity as tolerated. The general return-to-exercise recommendation is to advance slowly as symptoms permit. Similar to the management of concussion, athletes with postconcussion syndrome will need academic and workplace accommodations to restrict or modify cognitive loads until learning processes have recovered. Treatment of coexistent or new mood and sleep disturbances may also be beneficial. (C)

Disqualification from Sport

There are no evidence-based guidelines for disqualifying or retiring an athlete from their sport after concussion. Several authors have proposed that a clinician consider disqualifying an athlete if any of the following are present: structural abnormality on neuroimaging; multiple lifetime concussions; persistent diminished academic or workplace performance; persistent postconcussive symptoms; prolonged recovery courses; and perceived reduced threshold of sustaining recurrent concussions.[190–195] There have been no agreed upon absolute number of concussions an individual can sustain before disqualification from contact sports is necessary. Each case should be carefully deliberated with all parties involved with an understanding of risks and unknowns. An individualized approach to determining disqualification is essential. (C)

Long-term Sequelae of Sports-related Concussion

There is increasing concern that head impact exposure and recurrent concussions contribute to long-term neurological sequelae including chronic traumatic encephalopathy (CTE) and chronic neurocognitive impairment (CNI). There is no known relationship between CNI and CTE.

Chronic traumatic encephalopathy is a neurodegenerative disease associated with repetitive brain trauma and characterized pathologically by the accumulation of tau protein in specific areas of the brain. Chronic traumatic encephalopathy results in executive dysfunction, memory impairment, depression, and poor impulse control.[196] Chronic traumatic encephalopathy is a diagnosis made only after death with confirmatory histopathology, and the prevalence of this condition is unknown. Recent studies have described the histopathological findings of CTE in the postmortem analysis of athletes' brain tissue.[197–201] The typical symptoms and behaviors preceding death in athletes with CTE suggest a link between neurobehavioral patterns and the neuroanatomical areas of the brain affected. Chronic traumatic encephalopathy is not a continuation of postconcussion syndrome or symptoms from an acute concussion, but rather develops decades after exposure. Not all athletes diagnosed with CTE postmortem reported concussions during play, raising the question if subconcussive blows may contribute to the development of CTE. Given the large number of athletes participating in contact or collision sports and the small number of cases described, it is likely that other factors such as genetic predisposition play a role in its development.[178,196]

Chronic neurocognitive impairment after head injury can present in postconcussive syndrome, but can also occur years after a symptom-free interval, and CNI symptoms and behaviors can be demonstrated by neuropsychological testing. Some studies have identified an association between prior concussions and chronic cognitive dysfunction,[37,85,202,203] but others have found no association.[204–207] Studies involving former American football players[42,208] and soccer players who were active at the time of the study[209–211] suggest an increased risk of CNI with increased exposure to concussions and subconcussive insults to the head. Guskiewicz showed a higher incidence of mild cognitive impairment and depression in former National Football League athletes with a history of concussion compared to those who did not have a history of concussion.[42,208] Matser showed decreased performance on neuropsychological testing in athletes who had a higher incidence of concussion and heading.[209–211] However, a community-based study of high school football players in Minnesota with long-term follow-up

(median 50.2 years) showed no increase in dementia, Parkinson's disease, or amyotrophic lateral sclerosis in football players compared to the general population.[212] While the potential for CNI and CTE is concerning, large-scale, epidemiological studies will be required to more clearly understand the causes and develop prevention strategies.

Education

Studies demonstrate that athletes, parents, and coaches lack the knowledge needed to make informed decisions about concussion.[213–215] Misconceptions and lack of understanding about symptoms of concussion, as well as the recommended treatment and return-to-play guidelines, still exist.[213,215] Administrators and coaches play a big part in the health and safety of the athlete and they are often the first source of knowledge for the athlete and parents on topics like concussion.[213]

Education of medical personnel, coaches, athletes, and parents can enhance the identification of concussions and improve treatment and prevention. (C) Preseason education, including symptoms and signs of concussion, proper fit and use of equipment, player respect, sport rules, sport-specific technique, symptom reporting and assessment, treatment and testing options, and return-to-play guidelines, will help all to better understand the significance and importance of this injury. The education method and medium should be tailored to the group to optimize the learning method.[216]

Currently, there are a variety of concussion educational materials available through governmental agencies, educational institutions, and private companies. Table 6 below lists some recommended concussion education resources.

Prevention of Sports-related Concussion

All sports-related concussion cannot be prevented, but several efforts may reduce the incidence and severity. (B) These changes will require a shift in attitude and expectation on the part of players, coaches, officials, administrators, parents, and fans, especially with respect to contact and collision sports.

The rules of play form the basis of safe conduct for the game. Stringent enforcement of the rules by coaches and officials[217] and strict adherence to the rules by players will reduce the incidence of concussion. The rules of the game set expectations for behavior on the field of play and define infractions for collisions with increased risk to the aggrieved player. Rigorous education and consistent modeling of the rules by coaches and officials reinforce the importance of clean play. Fair play rules were shown to reduce concussion injury in hockey tournaments[218] and may have similar benefits for other sports. Promoting fair play encourages respect for opponents and emphasizes safety precautions for athletes. Coaches, parents, and managers must role model fair play values for effectiveness. Rule changes based on epidemiologic data also have reduced concussion and neck injury in some sports: 1) banning "spear tackling" in American football, 2) enforcing no "checking from behind" in ice hockey,[219] and 3) limiting "elbow to head" contact in soccer.[217]

Limiting the number of contact practices will reduce exposures and subsequently the number of concussions. This concept is being tested in American college football, college lacrosse, and college soccer in some conferences, and if proven effective may also be beneficial in other sports such as ice hockey and rugby.[220,221] Similar restrictions in contact-collision for different age groups, such as prohibiting body checking in boys' lacrosse and ice hockey below the age of 13 to 15, also has the potential for preventing injury.

Player behavior plays an integral part in injury reduction. Athletes must be taught correct playing techniques and appropriate behavior by trained and qualified coaches. Any behavior that increases concussion risk should be eliminated, and this may require a culture change in players, coaches, officials, administrators, and fans. Sports-specific techniques like tackling, body checking, and heading the ball must be properly taught and demonstrated before they are used in game situations.

Protective equipment has not yet shown a role in concussion reduction.[30,222,223] Helmets have played a significant role in reducing scalp lacerations, skull fractures, and intracranial bleeds, and their use in sports such as cycling, skiing, and snowboarding should be strongly supported. While headgear and helmets can reduce biomechanical forces associated with a blow to the head and could possibly reduce concussion severity, helmet use has not been shown to prevent or reduce concussion.[222] There is potential for improved helmet design and for sport-specific helmets to decrease concussion in sport. Mouth guards prevent peri-oral and dental injury, but there is little evidence that mouth guards reduce concussion rates.[95,131] Often overlooked is the possibility that use of protective equipment may change player behavior and secondarily contribute to an increase in concussion risk.

Neck strengthening may limit transmitted forces to the head and dampen impact to the brain. Differences in head-neck segment mass and girth have been demonstrated between women and men and may underlie the differences seen in reported concussion incidence between sexes.[60,224] From a theoretical standpoint, increasing the strength and rigidity of the head-neck segment could decrease the acceleration forces seen by the head when struck. However, neck strengthening programs have not yet, to date, been published that demonstrate a significant intervention effect. In addition, the benefits of stronger neck muscles are not effective when the athlete has no opportunity to 'prepare' for impact as often occurs in rule infractions.

Educating athletes, parents, coaches, administrators, athletic trainers, and physicians involved in contact and collision sports is important for athlete safety. (C) An understanding of the risks, detection and assessment techniques, and the principles of safe return to play should help improve concussion safety.

Legislation

Concussion-related legislation in many states requires education of athletes, parents or guardians, and coaches,

TABLE 6. Concussion Education Web Sites

NCAA Concussion program	NCAA.org/concussion
CDC Concussion Education/Head Up	www.cdc.gov/concussion/sports
NFL Health and Safety	NFLhealthandsafety.com

removal from play or practice at the time of a suspected concussion, and written clearance for return to practice or play by a licensed healthcare provider trained in the evaluation and management of concussion. Such laws provide a uniform standard for scholastic and nonscholastic sports organizations regarding concussion safety and management.[225] These efforts have arisen as safety measures to improve the care of this injury by standardizing the roles and responsibilities of athletes, parents or guardians, coaches, and healthcare providers.[225] Healthcare providers should be aware of the laws in the state where they practice. Over 80% of states have laws in place, and there are ongoing efforts to establish legislation in all 50 states.[226] These laws should be viewed as living documents to be modified as the best educational policies and practices are developed, the most effective strategies for implementing and measuring compliance of the laws are determined, and as new knowledge about sports concussions develops.

CONCLUSIONS

Concussion is a concerning and complicated problem in sport requiring a multifaceted approach to diagnosis and management. Athletes, coaches, officials, and parents need to be educated regarding signs and symptoms of concussion to recognize the injury on the field of play and to seek appropriate evaluation. It is critical that athletes are forthcoming regarding their symptoms as this is often the only detectable indication of concussion. A physician or other health professional trained in the evaluation and management of concussion who knows the athlete well is in the best position to correctly diagnose a concussed athlete. Standardized sideline tests provide a helpful, uniform approach for examination, but further studies are needed to delineate their accuracy. No athlete diagnosed with a concussion should return to play on the same day or while symptomatic. The return-to-play decision is a medical one. Additional research is needed to validate current assessment tools, further delineate the role of NP and balance testing, validate return-to-play guidelines, and improve identification of those at risk for prolonged concussive symptoms or other short- or long-term complications.

REFERENCES

1. Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the 1st International Conference on Concussion in Sport, Vienna 2001. Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries. *Br J Sports Med.* 2002;36:6–10.
2. McCrory P, Johnston K, Meeuwisse W, et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *Br J Sports Med.* 2005;39:196–204.
3. Guskiewicz KM, Bruce SL, Cantu RC, et al. National Athletic Trainers' Association position statement: management of sport-related concussion. *J Athl Train.* 2004;39:280–297.
4. Herring SA, Cantu RC, Guskiewicz KM, et al. Concussion (mild traumatic brain injury) and the team physician: a consensus statement—2011 update. *Med Sci Sports Exerc.* 2011;43:2412–2422.
5. McCrory P, Meeuwisse W, Johnston K, et al. Consensus statement on concussion in sport, 3rd International Conference on Concussion in Sport held in Zurich, November 2008. *Br J Sports Med.* 2009;43(suppl 1):i76–i90.
6. Meehan WP III, d'Hemecourt P, Comstock RD. High school concussions in the 2008-2009 academic year: mechanism, symptoms, and management. *Am J Sports Med.* 2010;38:2405–2409.
7. Ebell MH, Siwek J, Weiss BD, et al. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician.* 2004;69:548–556.
8. Prins ML, Hales A, Reger M, et al. Repeat traumatic brain injury in the juvenile rat is associated with increased axonal injury and cognitive impairments. *Dev Neurosci.* 2010;32:510–518.
9. Shrey DW, Griesbach GS, Giza CC. The pathophysiology of concussions in youth. *Phys Med Rehabil Clin N Am.* 2011;22:577–602, vii.
10. Barkhoudarian G, Hovda DA, Giza CC. The molecular pathophysiology of concussive brain injury. *Clin Sports Med.* 2011;30:33–48, vii–iii.
11. Vagnozzi R, Tavazzi B, Signoretti S, et al. Temporal window of metabolic brain vulnerability to concussions: mitochondrial-related impairment—part i. *Neurosurgery*. 2007;61:379–388; discussion, 388–379.
12. 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;133:3232–3242.
13. Vagnozzi R, Signoretti S, Tavazzi B, et al. Temporal window of metabolic brain vulnerability to concussion: a pilot 1h-magnetic resonance spectroscopic study in concussed athletes—part iii. *Neurosurgery*. 2008;62:1286–1295; discussion, 1295–1286.
14. Tavazzi B, Vagnozzi R, Signoretti S, et al. Temporal window of metabolic brain vulnerability to concussions: oxidative and nitrosative stresses—part ii. *Neurosurgery*. 2007;61:390–395; discussion, 395–396.
15. Esposito G, Van Horn JD, Weinberger DR, et al. Gender differences in cerebral blood flow as a function of cognitive state with pet. *J Nucl Med*. 1996;37:559–564.
16. Longhi L, Saatman KE, Fujimoto S, et al. Temporal window of vulnerability to repetitive experimental concussive brain injury. *Neurosurgery*. 2005;56:364–374; discussion, 364–374.
17. Lincoln AE, Hinton RY, Almquist JL, et al. Head, face, and eye injuries in scholastic and collegiate lacrosse: a 4-year prospective study. *Am J Sports Med.* 2007;35:207–215.
18. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train.* 2007;42:311–319.
19. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil.* 2006;21:375–378.
20. Gessel LM, Fields SK, Collins CL, et al. Concussions among United States high school and collegiate athletes. *J Athl Train.* 2007;42:495–503.
21. Powell JW, Barber-Foss KD. Traumatic brain injury in high school athletes. *JAMA*. 1999;282:958–963.
22. Bakhos LL, Lockhart GR, Myers R, et al. Emergency department visits for concussion in young child athletes. *Pediatrics*. 2010;126:e550–e556.
23. Lincoln AE, Caswell SV, Almquist JL, et al. Trends in concussion incidence in high school sports: a prospective 11-year study. *Am J Sports Med.* 2011;39:958–963.
24. Schulz MR, Marshall SW, Mueller FO, et al. Incidence and risk factors for concussion in high school athletes, North Carolina, 1996-1999. *Am J Epidemiol.* 2004;160:937–944.
25. Marar M, McIlvain NM, Fields SK, et al. Epidemiology of concussions among United States high school athletes in 20 sports. *Am J Sports Med.* 2012;40:747–755.
26. McCrea M, Hammeke T, Olsen G, et al. Unreported concussion in high school football players: implications for prevention. *Clin J Sport Med.* 2004;14:13–17.
27. Valovich McLeod TC, Bay RC, Heil J, et al. Identification of sport and recreational activity concussion history through the preparticipation screening and a symptom survey in young athletes. *Clin J Sport Med.* 2008;18:235–240.
28. Williamson IJ, Goodman D. Converging evidence for the underreporting of concussions in youth ice hockey. *Br J Sports Med.* 2006;40:128–132; discussion, 128–132.
29. Makdissi M, Darby D, Maruff P, et al. Natural history of concussion in sport: markers of severity and implications for management. *Am J Sports Med.* 2010;38:464–471.
30. Benson BW, Hamilton GM, Meeuwisse WH, et al. Is protective equipment useful in preventing concussion? A systematic review of the literature. *Br J Sports Med.* 2009;43(suppl 1):i56–i67.

31. Collins MW, Iverson GL, Lovell MR, et al. On-field predictors of neuropsychological and symptom deficit following sports-related concussion. *Clin J Sport Med.* 2003;13:222–229.
32. McCrea M, Barr WB, Guskiewicz K, et al. Standard regression-based methods for measuring recovery after sport-related concussion. *J Int Neuropsychol Soc.* 2005;11:58–69.
33. McCrory P. 2002 Refshauge lecture. When to retire after concussion? *J Sci Med Sport.* 2002;5:169–182.
34. Mansell JL, Tierney RT, Higgins M, et al. Concussive signs and symptoms following head impacts in collegiate athletes. *Brain Inj.* 2010;24: 1070–1074.
35. Jinguji TM, Bompadre V, Harmon KG, et al. Sport concussion assessment tool-2: baseline values for high school athletes. *Br J Sports Med.* 2012;46:365–370.
36. McLeod TC, Leach C. Psychometric properties of self-report concussion scales and checklists. *J Athl Train.* 2012;47:221–223.
37. Preiss-Farzanegan SJ, Chapman B, Wong TM, et al. The relationship between gender and postconcussion symptoms after sport-related mild traumatic brain injury. *PM R.* 2009;1:245–253.
38. Colvin AC, Mullen J, Lovell MR, et al. The role of concussion history and gender in recovery from soccer-related concussion. *Am J Sports Med.* 2009;37:1699–1704.
39. Frommer LJ, Gurka KK, Cross KM, et al. Sex differences in concussion symptoms of high school athletes. *J Athl Train.* 2011;46:76–84.
40. Lovell M, Collins M, Bradley J. Return to play following sports-related concussion. *Clin Sports Med.* 2004;23:421–441, ix.
41. Emery C, Kang J, Shrier I, et al. Risk of injury associated with body-checking experience among youth hockey players. *CMAJ.* 2011;183: 1249–1256.
42. Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. *Med Sci Sports Exerc.* 2007;39:903–909.
43. Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA concussion study. *JAMA.* 2003;290:2549–2555.
44. Guskiewicz KM, Weaver NL, Padua DA, et al. Epidemiology of concussion in collegiate and high school football players. *Am J Sports Med.* 2000;28:643–650.
45. Hollis SJ, Stevenson MR, McIntosh AS, et al. Incidence, risk, and protective factors of mild traumatic brain injury in a cohort of Australian nonprofessional male rugby players. *Am J Sports Med.* 2009;37:2328–2333.
46. Kristman VL, Tator CH, Kreiger N, et al. Does the apolipoprotein epsilon 4 allele predispose varsity athletes to concussion? A prospective cohort study. *Clin J Sport Med.* 2008;18:322–328.
47. Bruce JM, Echemendia RJ. Concussion history predicts self-reported symptoms before and following a concussive event. *Neurology.* 2004; 63:1516–1518.
48. Iverson GL, Gaetz M, Lovell MR, et al. Cumulative effects of concussion in amateur athletes. *Brain Inj.* 2004;18:433–443.
49. Schatz P, Moser RS, Covassin T, et al. Early indicators of enduring symptoms in high school athletes with multiple previous concussions. *Neurosurgery.* 2011;68:1562–1567; discussion, 1567.
50. Lau B, Lovell MR, Collins MW, et al. Neurocognitive and symptom predictors of recovery in high school athletes. *Clin J Sport Med.* 2009; 19:216–221.
51. Slobounov S, Slobounov E, Sebastianelli W, et al. Differential rate of recovery in athletes after first and second concussion episodes. *Neurosurgery.* 2007;61:338–344; discussion, 344.
52. Iverson GL, Gaetz M, Lovell MR, et al. Relation between subjective fogginess and neuropsychological testing following concussion. *J Int Neuropsychol Soc.* 2004;10:904–906.
53. Iverson G. Predicting slow recovery from sport-related concussion: the new simple-complex distinction. *Clin J Sport Med.* 2007;17:31–37.
54. Lau BC, Kontos AP, Collins MW, et al. Which on-field signs/symptoms predict protracted recovery from sport-related concussion among high school football players? *Am J Sports Med.* 2011;39:2311–2318.
55. Covassin T, Swanik CB, Sachs ML. Sex differences and the incidence of concussions among collegiate athletes. *J Athl Train.* 2003;38:238–244.
56. Dick RW. Is there a gender difference in concussion incidence and outcomes? *Br J Sports Med.* 2009;43(suppl 1):i46–i50.
57. Broshek DK, Kaushik T, Freeman JR, et al. Sex differences in outcome following sports-related concussion. *J Neurosurg.* 2005;102:856–863.
58. Kutcher JS, Eckner JT. At-risk populations in sports-related concussion. *Curr Sports Med Rep.* 2010;9:16–20.
59. Covassin T, Swanik CB, Sachs M, et al. Sex differences in baseline neuropsychological function and concussion symptoms of collegiate athletes. *Br J Sports Med.* 2006;40:923–927; discussion, 927.
60. Tierney RT, Sitler MR, Swanik CB, et al. Gender differences in head-neck segment dynamic stabilization during head acceleration. *Med Sci Sports Exerc.* 2005;37:272–279.
61. Gioia GA, Collins M, Isquith PK. Improving identification and diagnosis of mild traumatic brain injury with evidence: psychometric support for the acute concussion evaluation. *J Head Trauma Rehabil.* 2008;23: 230–242.
62. Daneshvar DH, Nowinski CJ, McKee AC, et al. The epidemiology of sport-related concussion. *Clin Sports Med.* 2011;30:1–17, vii.
63. Guskiewicz KM, Valovich McLeod TC. Pediatric sports-related concussion. *PM R.* 2011;3:353–364; quiz, 364.
64. Giza CC, Hovda DA. The neurometabolic cascade of concussion. *J Athl Train.* 2001;36:228–235.
65. Zuckerman SL, Odom M, Lee YM, et al. 145 sport-related concussion and age: number of days to neurocognitive baseline. *Neurosurgery.* 2012;71:E558.
66. Field M, Collins MW, Lovell MR, et al. Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. *J Pediatr.* 2003;142:546–553.
67. Moser RS, Schatz P, Jordan BD. Prolonged effects of concussion in high school athletes. *Neurosurgery.* 2005;57:300–306; discussion, 300–306.
68. Sim A, Terryberry-Spohr L, Wilson KR. Prolonged recovery of memory functioning after mild traumatic brain injury in adolescent athletes. *J Neurosurg.* 2008;108:511–516.
69. Bruce DA, Schut L, Sutton LN. Brain and cervical spine injuries occurring during organized sports activities in children and adolescents. *Prim Care.* 1984;11:175–194.
70. Kirkwood MW, Yeates KO, Wilson PE. Pediatric sport-related concussion: a review of the clinical management of an oft-neglected population. *Pediatrics.* 2006;117:1359–1371.
71. Snoek JW, Minderhoud JM, Wilmink JT. Delayed deterioration following mild head injury in children. *Brain.* 1984;107(pt 1):15–36.
72. Pellman EJ, Powell JW, Viano DC, et al. Concussion in professional football: epidemiological features of game injuries and review of the literature—part 3. *Neurosurgery.* 2004;54:81–94; discussion, 94–86.
73. Yard EE, Schroeder MJ, Fields SK, et al. The epidemiology of United States high school soccer injuries, 2005-2007. *Am J Sports Med.* 2008; 36:1930–1937.
74. Dick R, Putukian M, Agel J, et al. Descriptive epidemiology of collegiate women's soccer injuries: National Collegiate Athletic Association injury surveillance system, 1988-1989 through 2002-2003. *J Athl Train.* 2007;42:278–285.
75. Agel J, Evans TA, Dick R, et al. Descriptive epidemiology of collegiate men's soccer injuries: National Collegiate Athletic Association injury surveillance system, 1988-1989 through 2002-2003. *J Athl Train.* 2007; 42:270–277.
76. Boden BP, Kirkendall DT, Garrett WE Jr. Concussion incidence in elite college soccer players. *Am J Sports Med.* 1998;26:238–241.
77. Terrell TR, Bostick RM, Abramson R, et al. APOE, APOE promoter, and tau genotypes and risk for concussion in college athletes. *Clin J Sport Med.* 2008;18:10–17.
78. Tierney RT, Mansell JL, Higgins M, et al. Apolipoprotein E genotype and concussion in college athletes. *Clin J Sport Med.* 2010;20:464–468.
79. Terrell TR, Bostick RM, Barth JT, et al. Prospective cohort study of the association of genetic polymorphisms and concussion risk and postconcussion neurocognitive deficits in college athletes [abstract]. *Clin J Sport Med.* 2012;22:172.
80. Schaal K, Tafflet M, Nassif H, et al. Psychological balance in high level athletes: gender-based differences and sport-specific patterns. *PLoS One.* 2011;6:e19007.
81. Kontos AP, Covassin T, Elbin RJ, et al. Depression and neurocognitive performance after concussion among male and female high school and collegiate athletes. *Arch Phys Med Rehabil.* 2012;93:1751–1756.
82. Erlanger DM, Kutner KC, Barth JT, et al. Neuropsychology of sports-related head injury: dementia pugilistica to post concussion syndrome. *Clin Neuropsychol.* 1999;13:193–209.

83. Bailey CM, Samples HL, Broshek DK, et al. The relationship between psychological distress and baseline sports-related concussion testing. *Clin J Sport Med.* 2010;20:272–277.
84. Covassin T, Elbin RJ III, Larson E, et al. Sex and age differences in depression and baseline sport-related concussion neurocognitive performance and symptoms. *Clin J Sport Med.* 2012;22:98–104.
85. Collins MW, Grindel SH, Lovell MR, et al. Relationship between concussion and neuropsychological performance in college football players. *JAMA*. 1999;282:964–970.
86. Lau BC, Collins MW, Lovell MR. Sensitivity and specificity of subacute computerized neurocognitive testing and symptom evaluation in predicting outcomes after sports-related concussion. *Am J Sports Med.* 2011;39:1209–1216.
87. Lau BC, Collins MW, Lovell MR. Cutoff scores in neurocognitive testing and symptom clusters that predict protracted recovery from concussions in high school athletes. *Neurosurgery*. 2012;70:371–379; discussion, 379.
88. Solomon GS, Haase RF. Biopsychosocial characteristics and neurocognitive test performance in National Football League players: an initial assessment. *Arch Clin Neuropsychol.* 2008;23:563–577.
89. Kinart CM, Cuppett MM, Berg K. Prevalence of migraines in NCAA Division I male and female basketball players. National Collegiate Athletic Association. *Headache*. 2002;42:620–629.
90. Lipton RB, Pan J. Is migraine a progressive brain disease? *JAMA*. 2004; 291:493–494.
91. Robbins MS, Lipton RB. The epidemiology of primary headache disorders. *Semin Neurol.* 2010;30:107–119.
92. Gordon KE, Dooley JM, Wood EP. Is migraine a risk factor for the development of concussion? *Br J Sports Med.* 2006;40:184–185.
93. Mihalik JP, Stump JE, Collins MW, et al. Posttraumatic migraine characteristics in athletes following sports-related concussion. *J Neurosurg.* 2005;102:850–855.
94. Bernhardt D, Roberts W. *Preparticipation Physical Evaluation Monograph*. 2010. American Academy of Pediatrics. Pediatric Care Online. https://www.pediatriccareonline.org/pco/ub/view/Pediatric-Care-Updates/261108/0/preparticipation_physical_evaluation?amod=aapea&login=true&nfstatus=401&nftoken=00000000-0000-0000-0000-000000000000&nfstatusdescription=ERROR%3a+No+local+token. Accessed October 19, 2012.
95. McCrory P. Sport concussion assessment tool 2. *Scand J Med Sci Sports.* 2009;19:452.
96. Collie A, Darby D, Maruff P. Computerised cognitive assessment of athletes with sports related head injury. *Br J Sports Med.* 2001;35:297–302.
97. Echemendia RJ, Bruce JM, Bailey CM, et al. The utility of postconcussion neuropsychological data in identifying cognitive change following sports-related MTBI in the absence of baseline data. *Clin Neuropsychol.* 2012;26:107–1091.
98. Hunt TN, Ferrara MS. Age-related differences in neuropsychological testing among high school athletes. *J Athl Train.* 2009;44:405–409.
99. Greenwald BD, Cifu DX, Marwitz JH, et al. Factors associated with balance deficits on admission to rehabilitation after traumatic brain injury: a multicenter analysis. *J Head Trauma Rehabil.* 2001;16:238–252.
100. Rinne MB, Pasanen ME, Vartiainen MV, et al. Motor performance in physically well-recovered men with traumatic brain injury. *J Rehabil Med.* 2006;38:224–229.
101. Kaufman KR, Brey RH, Chou LS, et al. Comparison of subjective and objective measurements of balance disorders following traumatic brain injury. *Med Eng Phys.* 2006;28:234–239.
102. Geurts AC, Ribbers GM, Knoop JA, et al. Identification of static and dynamic postural instability following traumatic brain injury. *Arch Phys Med Rehabil.* 1996;77:639–644.
103. 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;290:2556–2563.
104. Riemann BL, Guskiewicz K, Shields EW. Relationship between clinical and forceplate measures of postural stability. *J Sport Rehabil.* 1999;8: 71–82.
105. Peterson CL, Ferrara MS, Mrazik M, et al. Evaluation of neuropsychological domain scores and postural stability following cerebral concussion in sports. *Clin J Sport Med.* 2003;13:230–237.
106. Guskiewicz K, Ross SE, Marshall SW. Postural stability and neuropsychological deficits after concussion in collegiate athletes. *J Athl Train.* 2001;36:263–273.
107. Riemann BL, Guskiewicz KM. Effects of mild head injury on postural stability as measured through clinical balance testing. *J Athl Train.* 2000;35:19–25.
108. Finnoff JT, Peterson VJ, Hollman JH, et al. Intrarater and interrater reliability of the Balance Error Scoring System (BESS). *PM R*. 2009; 1:50–54.
109. Mulligan I, Boland M, Payette J. Prevalence of neurocognitive and balance deficits in collegiate aged football players without clinically diagnosed concussion. *J Orthop Sports Phys Ther.* 2012;42:625–632.
110. Broglio SP, Puetz TW. The effect of sport concussion on neurocognitive function, self-report symptoms and postural control: a meta-analysis. *Sports Med.* 2008;38:53–67.
111. Register-Mihalik JK, Mihalik JP, Guskiewicz KM. Balance deficits after sports-related concussion in individuals reporting posttraumatic headache. *Neurosurgery*. 2008;63:76–80; discussion, 80–72.
112. Koehler DM, Shipman J, Davidson MA, et al. Is early venous thromboembolism prophylaxis safe in trauma patients with intracranial hemorrhage. *J Trauma.* 2011;70:324–329.
113. Depew AJ, Hu CK, Nguyen AC, et al. Thromboembolic prophylaxis in blunt traumatic intracranial hemorrhage: a retrospective review. *Am Surg.* 2008;74:906–911.
114. Norwood SH, Berne JD, Rowe SA, et al. Early venous thromboembolism prophylaxis with enoxaparin in patients with blunt traumatic brain injury. *J Trauma.* 2008;65:1021–1026; discussion, 1026–1027.
115. Echemendia RJ, Herring S, Bailes J. Who should conduct and interpret the neuropsychological assessment in sports-related concussion? *Br J Sports Med.* 2009;43(suppl 1):i32–i35.
116. Johnson EW, Kegel NE, Collins MW. Neuropsychological assessment of sport-related concussion. *Clin Sports Med.* 2011;30:73–88, viii–ix.
117. Ellemberg D, Henry LC, Macciocchi SN, et al. Advances in sport concussion assessment: from behavioral to brain imaging measures. *J Neurotrauma.* 2009;26:2365–2382.
118. Fazio VC, Lovell MR, Pardini JE, et al. The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *NeuroRehabilitation*. 2007;22:207–216.
119. Van Kampen DA, Lovell MR, Pardini JE, et al. The "value added" of neurocognitive testing after sports-related concussion. *Am J Sports Med.* 2006;34:1630–1635.
120. Iverson GL, Lovell MR, Collins MW. Interpreting change on impact following sport concussion. *Clin Neuropsychol.* 2003;17:460–467.
121. Randolph C, McCrea M, Barr WB. Is neuropsychological testing useful in the management of sport-related concussion? *J Athl Train.* 2005;40: 139–152.
122. McCrory P. Sports concussion and the risk of chronic neurological impairment. *Clin J Sport Med.* 2011;21:6–12.
123. Randolph C, Lovell M, Laker SR. Neuropsychological testing point/counterpoint. *PM R*. 2011;3:S433–S439.
124. Collie A, Maruff P, Makdissi M, et al. Statistical procedures for determining the extent of cognitive change following concussion. *Br J Sports Med.* 2004;38:273–278.
125. Sullivan SJ, Bourne L, Choie S, et al. Understanding of sport concussion by the parents of young rugby players: a pilot study. *Clin J Sport Med.* 2009;19:228–230.
126. Broglio SP, Macciocchi SN, Ferrara MS. Sensitivity of the concussion assessment battery. *Neurosurgery*. 2007;60:1050–1057; discussion, 1057–1058.
127. McCrory PR, Berkovic SF. Video analysis of acute motor and convulsive manifestations in sport-related concussion. *Neurology*. 2000;54: 1488–1491.
128. McCrory P. Using a sledgehammer to crack a walnut: the modern management of concussion. *Inj Prev.* 2007;13:364–365.
129. Schmidt JD, Register-Mihalik JK, Mihalik JP, et al. Identifying impairments after concussion: normative data versus individualized baselines. *Med Sci Sports Exerc.* 2012;44:1621–1628.
130. McCrory P. Future advances and areas of future focus in the treatment of sport-related concussion. *Clin Sports Med.* 2011;30:201–208, xi-ii.
131. McCrory P. Do mouthguards prevent concussion? *Br J Sports Med.* 2001;35:81–82.
132. Register-Mihalik JK, Guskiewicz KM, Mihalik JP, et al. Reliable change, sensitivity, and specificity of a multidimensional concussion assessment battery: implications for caution in clinical practice [published online ahead of print June 9, 2012]. *J Head Trauma Rehabil.*

133. Schatz P. Long-term test-retest reliability of baseline cognitive assessments using impact. *Am J Sports Med.* 2010;38:47–53.
134. Erdal K. Neuropsychological testing for sports-related concussion: how athletes can sandbag their baseline testing without detection. *Arch Clin Neuropsychol.* 2012;27:473–479.
135. Register-Mihalik JK, Kontos DL, Guskiewicz KM, et al. Age-related differences and reliability on computerized and paper-and-pencil neurocognitive assessment batteries. *J Athl Train.* 2012;47:297–305.
136. Schatz P, Moser RS, Solomon GS, et al. Prevalence of invalid computerized baseline neurocognitive test results in high school and collegiate athletes. *J Athl Train.* 2012;47:289–296.
137. Covassin T, Elbin RJ, Harris W, et al. The role of age and sex in symptoms, neurocognitive performance, and postural stability in athletes after concussion. *Am J Sports Med.* 2012;40:1303–1312.
138. Hutchison M, Comper P, Mainwaring L, et al. The influence of musculoskeletal injury on cognition: implications for concussion research. *Am J Sports Med.* 2011;39:2331–2337.
139. Jones NJ, Young C, Walter KD. Effect of education and language on baseline concussion screening tests in professional baseball players [abstract]. *Clin J Sport Med.* 2011;21:156.
140. Seifert TD, Evans RW. Posttraumatic headache: a review. *Curr Pain Headache Rep.* 2010;14:292–298.
141. Schreiber S, Barkai G, Gur-Hartman T, et al. Long-lasting sleep patterns of adult patients with minor traumatic brain injury (MTBI) and non-MTBI subjects. *Sleep Med.* 2008;9:481–487.
142. Busch CR, Alpern HP. Depression after mild traumatic brain injury: a review of current research. *Neuropsychol Rev.* 1998;8:95–108.
143. Reddy CC, Collins M, Lovell M, et al. Efficacy of amantadine treatment on symptoms and neurocognitive performance among adolescents following sports-related concussion [published online ahead of print May 18, 2012]. *J Head Trauma Rehabil.*
144. Strupp M, Thurtell MJ, Shaikh AG, et al. Pharmacotherapy of vestibular and ocular motor disorders, including nystagmus. *J Neurol.* 2011;258:1207–1222.
145. Alsalaheen BA, Mucha A, Morris LO, et al. Vestibular rehabilitation for dizziness and balance disorders after concussion. *J Neurol Phys Ther.* 2010;34:87–93.
146. McGrath N. Supporting the student-athlete's return to the classroom after a sport-related concussion. *J Athl Train.* 2010;45:492–498.
147. Schneiders AG, Sullivan SJ, Handcock P, et al. Sports concussion assessment: the effect of exercise on dynamic and static balance. *Scand J Med Sci Sports.* 2012;22:85–90.
148. Putukian M, Aubry M, McCrory P. Return to play after sports concussion in elite and non-elite athletes? *Br J Sports Med.* 2009;43(suppl 1):i28–i31.
149. Davis GA, Iverson GL, Guskiewicz KM, et al. Contributions of neuroimaging, balance testing, electrophysiology and blood markers to the assessment of sport-related concussion. *Br J Sports Med.* 2009;43(suppl 1):i36–i45.
150. Smits M, Houston GC, Dippel DW, et al. Microstructural brain injury in postconcussion syndrome after minor head injury. *Neuroradiology.* 2011;53:553–563.
151. Pulsipher DT, Campbell RA, Thoma R, et al. A critical review of neuroimaging applications in sports concussion. *Curr Sports Med Rep.* 2011;10:14–20.
152. Prabhu SP. The role of neuroimaging in sport-related concussion. *Clin Sports Med.* 2011;30:103–114, ix.
153. Echemendia RJ, Putukian M, Mackin RS, et al. Neuropsychological test performance prior to and following sports-related mild traumatic brain injury. *Clin J Sport Med.* 2001;11:23–31.
154. Amen DG, Trujillo M, Newberg A, et al. Brain SPECT imaging in complex psychiatric cases: an evidence-based, underutilized tool. *Open Neuroimag J.* 2011;5:40–48.
155. Kelly AB, Zimmerman RD, Snow RB, et al. Head trauma: comparison of MR and CT—experience in 100 patients. *AJNR Am J Neuroradiol.* 1988;9:699–708.
156. Slobounov SM, Zhang K, Pennell D, et al. Functional abnormalities in normally appearing athletes following mild traumatic brain injury: a functional MRI study. *Exp Brain Res.* 2010;202:341–354.
157. Talavage TM, Nauman E, Breedlove EL, et al. Functionally-detected cognitive impairment in high school football players without clinically-diagnosed concussion [published online ahead of print October 1, 2010]. *J Neurotrauma.* doi: 10.1089/neu.2010.1512.
158. Ptito A, Chen JK, Johnston KM. Contributions of functional magnetic resonance imaging (fMRI) to sport concussion evaluation. *NeuroRehabilitation.* 2007;22:217–227.
159. Gosselin N, Bottari C, Chen JK, et al. Electrophysiology and functional MRI in postacute mild traumatic brain injury. *J Neurotrauma.* 2011;28:329–341.
160. Keightley ML, Chiew KS, Winocur G, et al. Age-related differences in brain activity underlying identification of emotional expressions in faces. *Soc Cogn Affect Neurosci.* 2007;2:292–302.
161. Jantzen KJ, Oullier O, Marshall M, et al. A parametric fMRI investigation of context effects in sensorimotor timing and coordination. *Neuropsychologia.* 2007;45:673–684.
162. Chen JK, Johnston KM, Petrides M, et al. Neural substrates of symptoms of depression following concussion in male athletes with persisting postconcussion symptoms. *Arch Gen Psychiatry.* 2008;65:81–89.
163. Chen JK, Johnston KM, Frey S, et al. Functional abnormalities in symptomatic concussed athletes: an fMRI study. *Neuroimage.* 2004;22:68–82.
164. Zhang K, Johnson B, Pennell D, et al. Are functional deficits in concussed individuals consistent with white matter structural alterations: combined fMRI & DTI study. *Exp Brain Res.* 2010;204:57–70.
165. Schaefer PW, Huisman TA, Sorensen AG, et al. Diffusion-weighted MR imaging in closed head injury: high correlation with initial Glasgow Coma Scale score and score on modified Rankin Scale at discharge. *Radiology.* 2004;233:58–66.
166. Oni MB, Wilde EA, Bigler ED, et al. Diffusion tensor imaging analysis of frontal lobes in pediatric traumatic brain injury. *J Child Neurol.* 2010;25:976–984.
167. Wozniak JR, Krach L, Ward E, et al. Neurocognitive and neuroimaging correlates of pediatric traumatic brain injury: a diffusion tensor imaging (DTI) study. *Arch Clin Neuropsychol.* 2007;22:555–568.
168. Niogi SN, Mukherjee P, Ghajar J, et al. Extent of microstructural white matter injury in postconcussive syndrome correlates with impaired cognitive reaction time: a 3T diffusion tensor imaging study of mild traumatic brain injury. *AJNR Am J Neuroradiol.* 2008;29:967–973.
169. Levin HS, Wilde E, Troyanskaya M, et al. Diffusion tensor imaging of mild to moderate blast-related traumatic brain injury and its sequelae. *J Neurotrauma.* 2010;27:683–694.
170. Kraus MF, Susmaras T, Caughlin BP, et al. White matter integrity and cognition in chronic traumatic brain injury: a diffusion tensor imaging study. *Brain.* 2007;130:2508–2519.
171. Cubon VA, Putukian M, Boyer C, et al. A diffusion tensor imaging study on the white matter skeleton in individuals with sports-related concussion. *J Neurotrauma.* 2011;28:189–201.
172. Huisman TA. Diffusion-weighted imaging: basic concepts and application in cerebral stroke and head trauma. *Eur Radiol.* 2003;13:2283–2297.
173. Henry LC, Tremblay S, Boulanger Y, et al. Neurometabolic changes in the acute phase after sports concussions correlate with symptom severity. *J Neurotrauma.* 2010;27:65–76.
174. Yeo RA, Gasparovic C, Merideth F, et al. A longitudinal proton magnetic resonance spectroscopy study of mild traumatic brain injury. *J Neurotrauma.* 2011;28:1–11.
175. Cantu RC. Second-impact syndrome. *Clin Sports Med.* 1998;17:37–44.
176. Cantu RC, Gean AD. Second-impact syndrome and a small subdural hematoma: an uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. *J Neurotrauma.* 2010;27:1557–1564.
177. McCrory P. Does second impact syndrome exist? *Clin J Sport Med.* 2001;11:144–149.
178. McCrory P, Davis G, Makdissi M. Second impact syndrome or cerebral swelling after sporting head injury. *Curr Sports Med Rep.* 2012;11:21–23.
179. Kors EE, Terwindt GM, Vermeulen FL, et al. Delayed cerebral edema and fatal coma after minor head trauma: role of the CACNA1A calcium channel subunit gene and relationship with familial hemiplegic migraine. *Ann Neurol.* 2001;49:753–760.
180. Atkinson JL, Anderson RE, Murray MJ. The early critical phase of severe head injury: importance of apnea and dysfunctional respiration. *J Trauma.* 1998;45:941–945.
181. Engelborghs K, Verlooy J, Van Reempts J, et al. Temporal changes in intracranial pressure in a modified experimental model of closed head injury. *J Neurosurg.* 1998;89:796–806.
182. Lovell MR, Collins MW. Neuropsychological assessment of the college football player. *J Head Trauma Rehabil.* 1998;13:9–26.

183. Majerske CW, Mihalik JP, Ren D, et al. Concussion in sports: postconcussive activity levels, symptoms, and neurocognitive performance. *J Athl Train.* 2008;43:265–274.
184. Kissick J, Johnston KM. Return to play after concussion: principles and practice. *Clin J Sport Med.* 2005;15:426–431.
185. Ryan LM, Warden DL. Post concussion syndrome. *Int Rev Psychiatry.* 2003;15:310–316.
186. Jotwani V, Harmon KG. Postconcussion syndrome in athletes. *Curr Sports Med Rep.* 2010;9:21–26.
187. McAllister TW, Arciniegas D. Evaluation and treatment of postconcussive symptoms. *NeuroRehabilitation*. 2002;17:265–283.
188. Iverson GL. Outcome from mild traumatic brain injury. *Curr Opin Psychiatry.* 2005;18:301–317.
189. Leddy JJ, Kozlowski K, Donnelly JP, et al. A preliminary study of subsymptom threshold exercise training for refractory postconcussion syndrome. *Clin J Sport Med.* 2010;20:21–27.
190. Sedney CL, Orphanos J, Bailes JE. When to consider retiring an athlete after sports-related concussion. *Clin Sports Med.* 2011;30:189–200, xi.
191. De Beaumont L, Mongeon D, Tremblay S, et al. Persistent motor system abnormalities in formerly concussed athletes. *J Athl Train.* 2011;46:234–240.
192. De Beaumont L, Tremblay S, Poirier J, et al. Altered bidirectional plasticity and reduced implicit motor learning in concussed athletes. *Cereb Cortex.* 2012;22:112–121.
193. 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;132:695–708.
194. Cantu RC. Recurrent athletic head injury: risks and when to retire. *Clin Sports Med.* 2003;22:593–603, x.
195. McCrory P. What advice should we give to athletes postconcussion? *Br J Sports Med.* 2002;36:316–318.
196. Baugh CM, Stamm JM, Riley DO, et al. Chronic traumatic encephalopathy: neurodegeneration following repetitive concussive and subconcussive brain trauma. *Brain Imaging Behav.* 2012;6:244–254.
197. McKee AC, Cantu RC, Nowinski CJ, et al. Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. *J Neuropathol Exp Neurol.* 2009;68:709–735.
198. Omalu BI, Bailes J, Hammers JL, et al. Chronic traumatic encephalopathy, suicides and parasuicides in professional American athletes: the role of the forensic pathologist. *Am J Forensic Med Pathol.* 2010;31:130–132.
199. Omalu BI, DeKosky ST, Minster RL, et al. Chronic traumatic encephalopathy in a national football league player. *Neurosurgery*. 2005;57:128–134; discussion, 128–134.
200. Omalu BI, Fitzsimmons RP, Hammers J, et al. Chronic traumatic encephalopathy in a professional American wrestler. *J Forensic Nurs.* 2010;6:130–136.
201. Omalu BI, Hamilton RL, Kamboh MI, et al. Chronic traumatic encephalopathy (CTE) in a National Football League player: case report and emerging medicolegal practice questions. *J Forensic Nurs.* 2010;6:40–46.
202. Shuttleworth-Edwards AB, Smith I, Radloff SE. Neurocognitive vulnerability amongst university rugby players versus noncontact sport controls. *J Clin Exp Neuropsychol.* 2008;30:870–884.
203. Thornton AE, Cox DN, Whitfield K, et al. Cumulative concussion exposure in rugby players: neurocognitive and symptomatic outcomes. *J Clin Exp Neuropsyhol.* 2008;30:398–409.
204. Bruce JM, Echemendia RJ. History of multiple self-reported concussions is not associated with reduced cognitive abilities. *Neurosurgery*. 2009;64:100–106; discussion, 106.
205. Collie A, McCrory P, Makdissi M. Does history of concussion affect current cognitive status? *Br J Sports Med.* 2006;40:550–551.
206. Rutherford A, Stephens R, Fernie G, et al. Do UK university football club players suffer neuropsychological impairment as a consequence of their football (soccer) play? *J Clin Exp Neuropsychol.* 2009;31:664–681.
207. McAllister TW, Flashman LA, Maerlender A, et al. Cognitive effects of one season of head impacts in a cohort of collegiate contact sport athletes. *Neurology*. 2012;78:1777–1784.
208. Guskiewicz KM, Marshall SW, Bailes J, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurgery*. 2005;57:719–726.
209. Matser JT, Kessels AG, Lezak MD, et al. A dose-response relation of headers and concussions with cognitive impairment in professional soccer players. *J Clin Exp Neuropsychol.* 2001;23:770–774.
210. Matser JT, Kessels AG, Jordan BD, et al. Chronic traumatic brain injury in professional soccer players. *Neurology*. 1998;51:791–796.
211. Matser EJ, Kessels AG, Lezak MD, et al. Neuropsychological impairment in amateur soccer players. *JAMA*. 1999;282:971–973.
212. Savica R, Parisi JE, Wold LE, et al. High school football and risk of neurodegeneration: a community-based study. *Mayo Clin Proc.* 2012;87:335–340.
213. Sye G, Sullivan SJ, McCrory P. High school rugby players' understanding of concussion and return to play guidelines. *Br J Sports Med.* 2006;40:1003–1005.
214. Kaut KP, DePompei R, Kerr J, et al. Reports of head injury and symptom knowledge among college athletes: implications for assessment and educational intervention. *Clin J Sport Med.* 2003;13:213–221.
215. Valovich McLeod TC, Schwartz C, Bay RC. Sport-related concussion misunderstandings among youth coaches. *Clin J Sport Med.* 2007;17:140–142.
216. Provvidenza CF, Johnston KM. Knowledge transfer principles as applied to sport concussion education. *Br J Sports Med.* 2009;43(suppl 1):i68–i75.
217. Anderson SD. Bodychecking in hockey. *CMAJ*. 2004;170:16; author reply, 16, 18.
218. Roberts WO, Brust JD, Leonard B, et al. Fair-play rules and injury reduction in ice hockey. *Arch Pediatr Adolesc Med.* 1996;150:140–145.
219. Grindel SH, Lovell MR, Collins MW. The assessment of sport-related concussion: the evidence behind neuropsychological testing and management. *Clin J Sport Med.* 2001;11:134–143.
220. Ivy League presidents approve concussion recommendations for lacrosse and soccer [press release]. The IVY Leage Web Site; July 16, 2012. http://www.ivyleaguesports.com/information/gen-releases/2012-13/releases/Ivy_League_Presidents_Approve_Concussion_Recommendations_for_Lacrosse_and_Soccer. Accessed October 28, 2012.
221. Ivy League presidents approve concussion-curbing measures for football [press release]. The IVY League Web site; July 20, 2011. http://www.ivyleaguesports.com/sports/fball/2011-12/releases/Ivy_League_Presidents_Approve_Concussion-Curbing_Measures_for_Football. Accessed October 28, 2012.
222. Daneshvar DH, Baugh CM, Nowinski CJ, et al. Helmets and mouth guards: the role of personal equipment in preventing sport-related concussions. *Clin Sports Med.* 2011;30:145–163, x.
223. Navarro RR. Protective equipment and the prevention of concussion—what is the evidence? *Curr Sports Med Rep.* 2011;10:27–31.
224. Tierney RT, Higgins M, Caswell SV, et al. Sex differences in head acceleration during heading while wearing soccer headgear. *J Athl Train.* 2008;43:578–584.
225. Adler RH, Herring SA. Changing the culture of concussion: education meets legislation. *PM R*. 2011;3:S468–S470.
226. The Zachery Lystedt law. NFL Evolution Web site. http://www.nflevolution.com/article/The-Zackery-Lystedt-Law?ref=270. Accessed October 14, 2012.