Evidence-based Guideline Update: Evaluation and Management of Concussion in Sports


The guideline is endorsed by the National Football League Players Association, the Child Neurology Society, the National Association of Emergency Medical Service Physicians, the National Association of School Psychologists, the National Athletic Trainers Association, and the Neurocritical Care Society.

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**DISCLOSURES**

(1) Dr. Giza is a commissioner on the California State Athletic Commission, a member of the steering committee for the Sarah Jane Brain Project, a consultant for the National Hockey League Players’ Association (NHLPA), a member of the concussion committee for Major League Soccer, a member of the Advisory Board for the American Association for Multi-Sensory Environments (AAMSE), and a subcommittee chair for the Centers for Disease Control and Prevention (CDC) Pediatric Mild Traumatic Brain Injury Guideline Workgroup; has received funding for travel for invited lectures on traumatic brain injury (TBI)/concussion; has received royalties from Blackwell Publishing for “Neurological Differential Diagnosis”; has received honoraria for invited lectures on TBI/concussion; has received research support from the National Institute of Neurological Disorders and Stroke (NINDS)/National Institutes of Health (NIH), University of California, Department of Defense (DOD), NFL Charities, Thrasher Research Foundation, Today’s and Tomorrow’s Children Fund, and the Child Neurology Foundation/Winokur Family Foundation; and has given (and continues to give) expert testimony, has acted as a witness or consultant, or has prepared an affidavit for 2–4 legal cases per year.

(2) Dr. Kutcher receives authorship royalties from UpToDate.com; receives research support from ElMindA, Ltd.; is the Director of the National Basketball Association Concussion Program; is a consultant for the NHLPA; has received funding for travel and honoraria for lectures on sports concussion for professional organizations; and has given expert testimony on TBI cases.

(3) Dr. Ashwal serves on the medical advisory board for the Tuberous Sclerosis Association; serves as associate editor for *Pediatric Neurology*; has a patent pending for the use of HRS for imaging of stroke; receives royalties from publishing for *Pediatric Neurology: Principles and Practice* (coeditor for 6th edition, published in 2011); receives research support from NINDS grants for pediatric TBI and for use of advanced imaging for detecting neural stem cell migration after neonatal HII in a rat pup model; and has been called and continues to be called as treating physician once per year for children with nonaccidental trauma in legal proceedings.
(4) Dr. Barth has received funding for travel and honoraria for lectures on sports concussion for professional organizations, has given expert testimony on TBI cases, and occasionally is asked to testify on neurocognitive matters related to clinical practice.

(5) Mr. Getchius is a full-time employee of the American Academy of Neurology.

(6) Dr. Gioia has received funding for travel from Psychological Assessment Resources, Inc., and the Sarah Jane Brain Foundation; served in an editorial capacity for Psychological Assessment Resources, Inc.; receives royalties for publishing from Psychological Assessment Resources, Inc., and Immediate Post-Concussion Assessment and Cognitive Testing; has received honoraria from University of Miami Brain and Spinal Cord Conference and the State of Pennsylvania Department of Education; and has given expert testimony on 1 case of severe TBI.

(7) Dr. Gronseth serves as a member of the editorial advisory board of Neurology Now and serves as the American Academy of Neurology Evidence-based Medicine Methodologist.

(8) Dr. Guskiewicz serves on the editorial boards for the Journal of Athletic Training, Neurosurgery, and Exercise and Sport Science Reviews; serves as a member of concussion consensus writing committees for the National Athletic Trainers' Association (NATA), American Medical Society for Sports Medicine, and American College of Sports Medicine; serves on the National Collegiate Athletic Association’s (NCAA) Health and Safety Advisory Committee for Concussion, the National Football League’s (NFL) Head Neck and Spine Committee, and the NFL Players’ Association’s (NFLPA) Mackey-White Committee; has received funding for travel and honoraria for lectures on sports concussion for professional organizations; has given expert testimony on TBI/concussion cases; and has received research funding from the NIH, CDC, National Operating Committee for Standards in Athletic Equipment, NCAA, NFL Charities, NFLPA, USA Hockey, and NATA.

(9) Dr. Mandel reports no disclosures.
(10) Dr. Manley reports no disclosures.

(11) Dr. McKeag serves as Senior Associate Editor, *Clinical Journal of Sports Medicine*, and as Associate Editor, *Current Sports Medicine Reports*.

(12) Dr. Thurman reports no disclosures.

(13) Dr. Zafonte serves on editorial boards for *Physical Medicine & Rehabilitation* and *Journal of Neurotrauma*; receives royalties from Demos – Brain Injury Medicine Text; receives research support from the NIH, National Institute on Disability and Rehabilitation Research, DOD and has given expert testimony for an evaluation for the Department of Justice.

**ABBREVIATIONS**

AAN: American Academy of Neurology  
AE: Athlete exposure  
ANAM: Automated Neuropsychological Assessment Metrics  
BESS: Balance Error Scoring System  
CBI: Chronic Brain Injury  
CI: Confidence interval  
CLO: Clinician level of obligation  
COP: Center of pressure  
CR: Concussion rate  
CTE: Chronic traumatic encephalopathy  
GSC: Graded Symptom Checklist  
HITS: Head Impact Telemetry System  
ImPACT: Immediate Post-Concussion Assessment and Cognitive Testing  
IRR: Incidence rate ratio  
LHCP: licensed health care provider knowledgeable and skilled in sports concussions and practicing within the scope of his or her training and experience  
LOC: Loss of consciousness  
mTBI: Mild traumatic brain injury
NFL: National Football League
OR: Odds ratio
PCSS: Postconcussion Symptom Scores
PID: Postinjury day
PTA: Posttraumatic (anterograde) amnesia
RGA: Retrograde amnesia
RR: Relative risk
RTP: Return to play
SAC: Standardized Assessment of Concussion
SFWP: Symptom-free waiting period
SOT: Sensory Organizational Testing
SRC: Sport-related concussion
SSTET: Subsymptom threshold exercise training
ABSTRACT

Objective: To update the 1997 American Academy of Neurology practice parameter regarding the evaluation and management of sports concussion, with a focus on four questions: 1) For athletes what factors increase or decrease concussion risk? 2) For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those with concussion and those at increased risk for severe or prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment? 3) For athletes with concussion, what clinical factors are useful in identifying those at increased risk for severe or prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment? (4) For athletes with concussion, what interventions enhance recovery, reduce the risk of recurrent concussion, or diminish long-term sequelae?

Methods: A systematic review of the literature from 1955–June 2012 for pertinent evidence was performed. Evidence was assessed for quality and synthesized into conclusions by use of a modified form of the Grading of Recommendations Assessment, Development and Evaluation process. Recommendations were developed using a modified Delphi process.

Results: 1) Specific risk factors increase (type of sport – football, rugby) or decrease (type of sport – baseball, softball, volleyball, and gymnastics; rugby helmet use) the risk of concussion. 2) Diagnostic tools useful to identify those with concussion include graded symptom checklists, Standardized Assessment of Concussion, neuropsychological testing (paper-and-pencil and computerized), and the Balance Error Scoring System. 3) Ongoing clinical symptoms, history of prior concussions, and younger age identify those at risk for prolonged postconcussion impairments. Risk factors for recurrent concussion include having a history of multiple concussions and being within 10 days after an initial concussion. Risk factors for chronic neurobehavioral impairment include concussion exposure and apolipoprotein E epsilon4 genotype. 4) There is insufficient evidence to show that any intervention enhances recovery or diminishes long-term sequelae after a sports-related concussion. Nineteen evidence-based recommendations were developed in 3 categories: preparticipation counseling, assessment and management of suspected concussion, and management of diagnosed concussion.
INTRODUCTION

Concussion is recognized as a clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, which may involve loss of consciousness (LOC). For the most part, definitions of the terms concussion and mild traumatic brain injury (mTBI) overlap, as both terms represent the less-severe end of the traumatic brain injury (TBI) spectrum, where acute neurologic dysfunction generally recovers over time and occurs in the absence of significant macrostructural damage. For the purposes of this evidence-based review, the “reference standard” for article inclusion is a clinician-diagnosed concussion/mTBI. Whereas the definitions for a clinician-diagnosed concussion/mTBI are not identical throughout the existing literature, the vast majority of these cases are recognized as being sufficiently similar to allow for review and data extraction (see appendix 1).

Estimates of sports-related mTBI range from 1.6–3.8 million affected individuals annually in the United States, many of whom do not obtain immediate medical attention. Table 1 summarizes the currently available data for the overall concussion rate (CR) and the CRs for five commonly played high school and collegiate sports in males and females. As public awareness of sports concussion has increased substantially in recent years (Concussion issue of Sports Illustrated, New York Times articles, Congressional hearings), existing guidelines for recognition and clinical management remain largely consensus based. Medical care, when sought, is provided by a range of medical professionals who vary widely in their degree of expertise in evaluating and managing affected athletes suffering from sports concussions. This variability in care provider experience and training, coupled with an explosion of published reports related to sports concussion and mTBI, has led to some uncertainty and inconsistency in the management of these injuries. Legislative actions in many states have begun to mandate aspects of education and management of youth sports concussions. The impetus for developing this systematic, evidence-based multidisciplinary guideline derives from several factors: high-profile injuries in professional sports, questions about cumulative damage risk, concerns about increased vulnerability in youth, and ongoing extrapolation of mTBI guidelines from sports to other settings (such as military ‘blast’ TBI). As of this writing, 42 states have passed legislation pertinent to sports concussion management, making objective guidelines all the more important.
Since 1997, the guidelines predominantly used for management of sports concussions have been consensus based.\textsuperscript{e7-e9,e10} Over time there has been a move away from using an acute grading system in trying to predict concussion outcomes to using a more individualized approach based on risk factors for prolonged recovery. Current standard of practice is to remove from the field athletes suspected of sustaining a concussion until they are assessed by a medical professional to determine whether a concussion has occurred. Athletes with concussion are then assessed over time, and a gradual return to play (RTP) procedure is followed. In many states, recent legislation prohibits young athletes with concussion from returning to play the same day. On the basis of these factors, previous guidelines permitting same-day RTP in subsets of athletes with concussion are in need of updating.

This evidence-based guideline, which replaces the 1997 American Academy of Neurology (AAN) practice parameter on the management of sports concussion,\textsuperscript{e10} reviews the evidence published between 1955 and June 2012 regarding the evaluation and management of sports concussion in children, adolescents, and adults. In accordance with AAN criteria, Class IV evidence (case reports, case series, meta-analyses, systematic reviews) was excluded. A multidisciplinary panel was formed, including representatives from child and adult neurology, athletic training, child and adult neuropsychology, epidemiology and biostatistics, neurosurgery, physical medicine and rehabilitation, and sports medicine. For the purposes of this guideline, we evaluated studies that defined as their clinical outcome a disruption in brain function caused by biomechanical force regardless of whether they used the term \textit{concussion} or sports-related \textit{mild traumatic brain injury} (see appendix 1). We examined the evidence for risk or protective factors for concussion, including but not limited to age, gender, sport-specific variables, health habits, medical history, and protective gear use. We also carefully studied the efficacy of available diagnostic tools (e.g., standardized checklists, neuropsychological testing, and neuroimaging) and factors that might affect the diagnostic accuracy of those tools. We further assessed the evidence for factors that affect a range of short- and long-term outcomes and evidence for interventions to reduce recurrent concussion risk and long-term sequelae. This guideline addresses the following clinical questions:

1. For athletes what factors increase or decrease concussion risk?
2. For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those with concussion and those at increased risk for severe or prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment?

3. For athletes with concussion, what clinical factors are useful in identifying those at increased risk for severe or prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment?

4. For athletes with concussion, what interventions enhance recovery, reduce the risk of recurrent concussion, or diminish long-term sequelae?

**DESCRIPTION OF THE ANALYTIC PROCESS**

This guideline was developed according to the processes described in the 2004 and 2011 AAN guideline development process manuals. After review of potential panel members’ conflict of interest statements and curriculum vitae the AAN Guideline Development Subcommittee (appendices 2 and 3) selected a multidisciplinary panel of experts. Panel members came from multiple specialties, including neurology (adult: J.S.K., S.M.; child: S.A., C.C.G.), neuropsychology (adult: J.B.; child: G.A.G.), neurosurgery (G.M.), sports medicine (D.B.M.), athletic training (K.G.), neurorehabilitation (R.Z.), epidemiology/statistics (D.J.T.), and evidence-based methodology (G.G.). After an analytic framework was considered, questions answerable from the evidence were developed through informal consensus. A medical librarian assisted the panel in performing a comprehensive literature search of multiple databases to obtain the relevant studies. Appendix 4 presents the comprehensive search strategy, including terms, years, and databases searched.

Only papers relevant to sports concussion or sports-related mTBI published between 1955 and June 2012 were included. A study’s quality (risk of bias) as it pertains to the questions was measured by the AAN’s four-tiered classification of evidence schemes pertinent to diagnostic, prognostic, or therapeutic questions (appendix 5). Class I and II studies are discussed in the guideline text and documented in the evidence tables (appendix 6). Class III studies are included in the evidence tables but may not have been mentioned in the text if multiple studies with higher levels of evidence are available. As previously mentioned, Class IV studies such as case series or meta-analyses have been excluded. Characteristics influencing a study’s risk of bias and
generalizability were abstracted using a structured data collection form. Each article was selected for inclusion and study characteristics abstracted independently by two panel members. Disagreements were resolved by discussion between the two panel members. A third panel member adjudicated remaining disagreements. Evidence tables were constructed from the abstracted study characteristics.

Evidence was synthesized and conclusions developed using a modified form of the Grading of Recommendations Assessment, Development and Evaluation process. The confidence in evidence was anchored to the studies’ risk of bias according to the rules outlined in appendix 7. The overall confidence in the evidence pertinent to a question could be downgraded by one or more levels on the basis of five factors: consistency, precision, directness, publication bias, or biological plausibility. In addition, the overall confidence in the evidence pertinent to a question could be downgraded one or more levels or upgraded by one level on the basis of three factors: magnitude of effect, dose response relationship, or direction of bias. Two panel members working together completed an evidence summary table to determine the final confidence in the evidence (appendix 7). The confidence in the evidence is indicated by use of modal operators in conclusion statements in the guideline. “Highly likely” or “highly probable” corresponds to high confidence level, “likely” or “probable” corresponds to moderate confidence level, and “possibly” corresponds to low confidence level. Very low confidence is indicated by the term “insufficient evidence.”

The panel formulated a rationale for recommendations (appendix 8) based on the evidence systematically reviewed, on stipulated axiomatic principles of care, and, when evidence directly related to sports concussion was unavailable, on strong evidence derived from the non–sports-related TBI literature. This rationale is explained in a section labeled “Clinical Context” which precedes each set of recommendations. From this rationale, corresponding actionable recommendations were inferred. To reduce the risk of bias from the influences of “group think” and dominant personalities, the clinician level of obligation (CLO) of the recommendations was assigned using a modified Delphi process that considered the following prespecified domains: the confidence in the evidence systematically reviewed, the acceptability of axiomatic principles of care, the strength of indirect evidence, and the relative magnitude of benefit to harm.
Additional factors explicitly considered by the panel that could modify the CLO include judgments regarding the importance of outcomes, cost of compliance to the recommendation relative to benefit, the availability of the intervention, and anticipated variations in athletes’ preferences. The prespecified rules for determining the final CLO from these domains is indicated in appendix 8. The CLO is indicated using standard modal operators. “Must” corresponds to “Level A,” very strong recommendations; “should” to “Level B,” strong recommendations; and “might” to “Level C,” weak recommendations.

ANALYSIS OF EVIDENCE
For athletes what factors increase or decrease concussion risk?
Some athletes may be at greater risk of having a sports-related concussion (SRC) associated with different factors (e.g., age, gender, sport played, level of sport played, equipment used). Comparisons of the findings of such studies are difficult because of varying study populations, case definitions, methods of case reporting, and methods of analysis. Accordingly, we considered for analysis only those studies that directly compared the concussion risk between two or more of these variables when equivalent study populations, definitions, and methods were employed. Our literature search screened 7381 abstracts, the articles for 400 of which underwent detailed methodological review. Of these 400 articles, 132 underwent full-text review and had pertinent evidence extracted. Appendix 6, Q1, contains data related to this question from 27 Class I and 5 Class II studies. Table 1 summarizes the concussion incidence in commonly played high school and collegiate sports.

Age/level of competition. Four Class I studies that examined CRs by age or competition level were reviewed. Because these two parameters co-vary closely (e.g., high school athletes typically are aged 14–18), we evaluated together studies that addressed either variable, noting that other variables (e.g., greater strength and weight of more-mature athletes) are also closely related to age and competition level.

Two studies compared CRs in collegiate versus high school athletes; one found higher CRs in collegiate athletes in each of 9 sports,\textsuperscript{e14} and the other found higher CRs in high school football athletes relative to collegiate athletes.\textsuperscript{e15} A third study involved middle and high school students
engaged in taekwondo. The incidence of head blows and concussions was associated with young age and a lack of blocking skills, as calculated using a multinomial logistic model.\textsuperscript{e16} A fourth study, in ice hockey players, examined athletes in four age groups: 9–10 years, 11–12 years, 13–14 years, and 15–16 years.\textsuperscript{e17} When compared with those of the youngest age group, the relative risks (RRs) of concussion for the older groups were significantly higher - 3.4 (11–12 years), 4.04 (13–14 years), and 3.41 (15–16 years).

**Conclusion.** There is insufficient evidence to determine whether age or level of competition affects concussion risk overall, as findings are not consistent across all studies or in all sports examined (inconsistent Class I studies). Because of the greater number of participants in sports at the high school level, the total number of concussions may be greater in that age group.

**Gender.** Some investigators have suggested that male athletes may be at greater concussion risk than female athletes because of males’ greater body weight, speed, and tendency to play sports faster and more forcefully.\textsuperscript{e18,e19} Other hypotheses put forth suggest that female athletes might be at greater risk because of their smaller physical size and weaker neck muscle strength.\textsuperscript{e18,e19} Available data suggest that gender differences vary by sport. Appendix 6, Q1, summarizes data from four Class I and three Class II studies.

In a study of high school and collegiate sports played by both genders, separate comparisons indicated that females had significantly higher CRs than males in the sports of soccer (RR 1.68, 95% confidence interval [CI] =1.08–2.60, \( p=0.03 \), 183 total concussions) and basketball (RR 2.93, 95% CI 1.64–5.24, \( p<0.01 \), 138 total concussions).\textsuperscript{e14} A similar study of 5 collegiate sports, which compared CRs of males and females, found significantly higher CRs in females playing soccer (\( X^2=12.99, p<0.05 \)) and basketball (\( X^2=5.14, p<0.05 \)); comparisons of CRs between genders were not significant in lacrosse, softball/baseball, or gymnastics.\textsuperscript{e20} A third study of high school varsity athletes found that among sports played by both genders, CRs were somewhat higher among females for the sports of soccer and basketball, although the differences were nonsignificant.\textsuperscript{e21} The remaining Class I study reported on the sport of taekwondo and found a higher CR in males which was not significant.\textsuperscript{e16}
In a retrospective study of self-reported concussions and concussion symptoms among players of several collegiate sports, females reported slightly higher numbers of recognized concussions per year (nonsignificant), and males more frequently reported events with symptoms consistent with unrecognized concussions, suggesting that concussions may be unreported in a higher proportion of males than females. Two other Class II studies found that the concussion incidence in females was higher, describing an RR of 2.4 in soccer and an RR of 1.6 in lacrosse.

Conclusions. Because of the greater number of male participants in sports studied, the total number of concussions is greater for males than females for all sports combined. However, the relationship of concussion risk and gender varies among sports. It is highly probable that concussion risk is greater for female athletes participating in soccer or basketball (3 studies, Class I).

Type of sport. Among the numerous studies that described concussion incidence among athletes playing a specific sport, we found eight Class I studies that directly compared concussion risk between two or more sports (appendix 6, Q1).

One study compared concussion risk among collegiate athletes competing in football, basketball, ice hockey, soccer, wrestling, volleyball, gymnastics, or baseball/softball. The highest degree of risk was found for football, followed (in descending order) by hockey, wrestling, soccer, and lacrosse; volleyball and gymnastics had the lowest rates. Appendix 6, Q1, lists relative CRs (as compared with those for soccer or football) for both genders for the more-common sports.

Another group of investigators published a study in five separate parts that for this review was considered one Class I study. The authors reported concussion incidence for collegiate athletes competing in football, basketball, women’s field hockey, and lacrosse. During competitions or games, the CRs were highest for football, followed (in descending order) by men’s lacrosse, women’s lacrosse, women’s field hockey, and basketball.

A third Class I study examined CRs among high school and collegiate athletes playing American football, basketball, soccer, wrestling, baseball, softball, and volleyball. CRs were highest for football and soccer; appendix 6, Q1, lists relative CRs for some of these sports (at both the high
school and collegiate levels and by gender). Another Class I study of high school varsity athletes found that among 10 sports studied, football accounted for most (63%) of the mTBIs. The sports with highest rates of mTBI occurrence per 1000 game exposures were football followed by soccer; those with the lowest mTBI occurrence rates were baseball/softball and volleyball. A fifth study described concussion occurrence among collegiate football, basketball, ice hockey, soccer, and lacrosse players. This study found higher CRs among men’s football and women’s soccer and hockey players, although the differences were nonsignificant. CRs for some of these sports are tabulated in appendix 6, Q1. A sixth study compared pre–high school and high school Australian rugby and soccer players, finding that rugby union football was associated with a significantly higher injury rate than soccer. A seventh study described sports concussion incidence from 2005 to 2009 in US high school male athletes in football, soccer, basketball, wrestling, and softball, and in US high school female athletes in soccer, basketball, and softball. The study found the highest CRs in football (4.1/10,000 athletic exposures [AEs]), girls’ soccer (1.84/10,000 AEs), boys’ soccer (1.47/10,000 AEs), and girls’ basketball (1.19/10,000 AEs). A final study looked at sports concussion incidence prospectively from 1997 to 2007, again finding the highest CR in football (0.6/1000 AE) and the second-highest CR in girls’ soccer (0.35/1000 AEs).

Conclusion. For athletes it is highly likely that there is a greater concussion risk with American football and Australian rugby than with other sports analyzed here. Among the sports studied, it is highly likely that the risk is lowest for baseball, softball, volleyball, and gymnastics. For female athletes it is highly likely that soccer is the sport with the greatest concussion risk (multiple Class I studies).

Equipment. There were no studies of the extent of concussion risk reduction afforded by helmet use in American football, a reflection of the long-standing universal use of this equipment in organized competitions, which precludes comparative study with control groups not using helmets. There were no studies of soccer protective headgear that allowed quantification of concussion risk.
Mouth guards have been suggested to provide a protective effect against concussion by mitigating forces experienced by a blow to the jaw. There are three Class I studies addressing the use of headgear or mouth guards in reducing concussion, all focused on rugby. In one study, nonprofessional rugby players who reported always wearing headgear during games were 43% less likely to sustain an mTBI relative to players who never wore headgear (incidence rate ratio [IRR] 0.57 95% CI 0.40–0.82, \( p = 0.0024 \), multivariate analysis). Univariate analysis of mouth guard use suggested a similar protective effect on concussion incidence; however, this effect was nonsignificant in the multivariate analysis. The second study, examining professional rugby players, found significantly reduced concussion risk among players using headgear but only nonsignificant concussion risk reduction among players using mouth guards (RR 0.69 95% CI 0.41–1.17). The third study, in concussion risk among collegiate rugby players, showed that mouth guards had no protective effect (RR 1.24 95% CI 0.45–3.43).

**Conclusion.** It is highly probable that headgear use has a protective effect on concussion incidence in rugby (two Class I studies). There is no compelling evidence that mouth guards protect athletes from concussion (three Class I studies). Data are insufficient to support or refute the efficacy of protective soccer headgear. Data are insufficient to support or refute the superiority of one type of football helmet over another in preventing concussions.

**Position.** Concussion risks may vary relative to positions played by athletes on competitive sports teams. These risks must be considered by individual sport.

For professional rugby, two Class I studies found inconsistent and nonsignificant differences in concussion incidence between players in forward and back positions. One Class II study found a significantly greater concussion risk among players in forward positions.

For collegiate football, one Class I study found a weak association between position and concussion risk, with riskiest positions being (in descending order) linebacker (CR 0.99 95% CI 0.65–1.33), offensive lineman (CR 0.95 95% CI 0.66–1.24), defensive back (CR 0.88 95% CI 0.57–1.18), quarterback (CR 0.83 95% CI 0.34–1.31), tight end (CR 0.78 95% CI 0.30–1.26), special teams (CR 0.77 95% CI 0.27–1.28), defensive lineman (CR 0.76 95% CI 0.48–1.05),
running back (CR 0.71 95% CI 0.38–1.04), and receiver (CR 0.54 95% CI 0.27–0.81). In the same study, a combined position category of “linebacker/receiver” was also reported (CR 1.90 95% CI 1.0–3.4). A Class II football study provided data indicating greatest risk among defensive lineman and least risk among quarterbacks; the relative concussion risk between players of these positions was marginally significant.

For collegiate men’s ice hockey, one Class I study suggested a greater concussion risk among players in defensive or forward positions relative to that for goalies, but the results were nonsignificant. For collegiate men’s and women’s soccer, a Class II study found a modest, nonsignificant increased concussion risk among goalie and defensive positions.

Conclusion. Data are insufficient to characterize concussion risk by position in most major team sports. In collegiate football, concussion risk is probably greater among linebackers, offensive linemen, and defensive backs as compared with receivers (Class I and Class II studies).

**Body checking in ice hockey.** One Class I study investigated the relationship between body-checking experience in ice hockey and concussion incidence, finding a greater risk of SRC and a greater risk for severe SRC (time lost from playing ≥10 days) in peewee players in provinces where body checking was permitted (SRC IRR: 3.75, 95% CI 2.02–6.98; severe SRC IRR: 3.61, 95% CI 1.16–11.23). This same study found that prior SRC was a risk factor for sustaining a subsequent SRC (2.14, 95% CI 1.28–3.55) and having a severe SRC (2.76, 95% CI 1.10–6.91). This result was supported in a follow-up study of these players over a second consecutive year.

Conclusion. Data suggest with moderate confidence that prior exposure to body checking is a risk factor for SRC in ice hockey (two related Class I studies).

**Athlete-related factors.** Athlete-specific characteristics, such as body mass index (BMI) and number of hours spent training, were investigated in one Class I study. Having a BMI greater than 27 (1.77, \( p = 0.007 \)) and training less than 3 hours per week (1.48, \( p = 0.03 \)) were both found to be related to a higher SRC risk in community rugby union athletes.
Conclusion. Athlete-specific characteristics such as body mass index greater than 27 and time spent training less than 3 hours likely increase the risk of concussion (one Class I study).

Cumulative impacts. One Class I study investigated the association between the number of impacts experienced by high school American football athletes, as measured by in-helmet impact sensors, and concussion diagnosis. The authors found no association.\textsuperscript{e45}

Conclusion. Data are insufficient to characterize concussion risk by the cumulative number of impacts experienced during the course of an American football season in high school athletes.

Team record. In competitive youth ice hockey, team performance as measured by winning percentage was investigated as a risk factor for SRC in one Class I study. Whereas a winning record was associated with less injury in general, no effect was seen on SRC.\textsuperscript{e46}

Conclusion. Data are insufficient to characterize SRC risk by team performance as measured by winning percentage (one Class I study).

For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those with concussion? For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those at increased risk for severe or prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment?

Our literature search screened 1703 abstracts from which 409 were selected and the full-text articles obtained for detailed methodologic review. Of these 409 studies, 195 full-text publications were classified by evidence level. For this question, to maximize clinical utility we categorized the evidence for each tool as follows: diagnosis of concussion, severe or prolonged early postconcussion impairments, neurologic catastrophe, or chronic neurobehavioral impairment.
The vast majority of these studies involved high school or collegiate athletes. No studies specifically looking at age groups below high school age were found.

**Q2a: For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those with concussion?**

Appendix 6, Q2, lists the studies of tools relevant to the diagnosis of concussion. Whereas the reference standard by which these studies were conducted was a concussion diagnosed by a clinician (physician or certified athletic trainer), it should be noted that no studies were found that explicitly examined interrater reliability. The 1997 AAN concussion definition\(^{e10}\) was the most commonly utilized definition for the purposes of these studies (see appendix 1).

All studies employed a case-control design specifically including athletes on the basis of the presence or absence of concussion. Many of the studies also obtained baseline testing on a large cohort of athletes prior to concussion (i.e., a nested case-control design). All studies compared the performance on the putative diagnostic tool of athletes with concussion to that of athletes without concussion. For many studies it was not possible to determine whether the clinician diagnosis of concussion (the usual reference standard) was influenced by knowledge of the result of the putative diagnostic test. Thus, some degree of incorporation bias may have affected the results. All but one study failed to include athletes for whom there was the potential of diagnostic uncertainty relative to the presence of concussion—that is, the studies examined here included only athletes in whom concussion was definitely diagnosed by the reference standard and athletes in whom there was no suspicion of concussion. This introduced potential spectrum bias and led to the majority of these studies being rated Class III relative to the diagnostic accuracy question. One study reduced the potential of spectrum bias by including athletes without concussion who had musculoskeletal injuries.\(^{e47}\) This study was rated Class II.

**Post-Concussion Symptom Scale or Graded Symptom Checklist.** Nine Class III studies utilized either a Post-Concussion Symptom Scale (PCSS) or Graded Symptom Checklist (GSC) to assist in concussion diagnosis (appendix 6, Q2). These tools may be administered by a nonphysician or by self-report. Most studies\(^{e48-e54}\) involved a 14- to 22-item scale, with a 7-point scale to self-report symptom severity (“0” served as an anchor representing not present; “6” represented worst
The aggregate score of all possible symptoms was recorded and compared with baseline measures or noninjured matched control subjects, or both. In one study, symptom duration was factored in to the scoring,\textsuperscript{e55} and in another study the number of symptoms endorsed was the variable of interest.\textsuperscript{e56}

Clinical diagnosis by a physician or athletic trainer was generally the reference standard by which these scales were compared. Two studies reported on the same cohort of athletes with concussion,\textsuperscript{e48,e49} reporting a symptom score increase of 21 points (95% CI 16–26) acutely following concussion, with a sensitivity of 0.89 and specificity of 1.00 within 3 hours postinjury. The other study showed an overall highly significant increase in symptoms (approximately 20 points, with standard errors ± 4) in the concussed group versus in controls ($p=0.001$).\textsuperscript{e55} The remaining studies reported similar findings of elevated scores postinjury, with one study\textsuperscript{e54} also reporting sensitivity of 0.64 and specificity of 0.91.

**Conclusion.** With the reference standard being clinician diagnosis of concussion, it is likely that a GSC or PCSS will identify concussion in the proper clinical context with moderate diagnostic accuracy (sensitivity 64%–89%, specificity 91%–100%) (multiple Class III studies). Proper clinical context refers to use of these tools in athletes after an observed or suspected event during which biomechanical forces were imparted to the head. It should be emphasized that the sensitivity of these symptom checklist tools is insufficient to rule out a suspected concussion, as an important proportion of athletes with concussion (11%–36%) will perform normally on these tests.

**Standardized Assessment of Concussion.** The Standardized Assessment of Concussion (SAC) is an instrument designed for 6-minute administration to assess four neurocognitive domains—Orientation, Immediate Memory, Concentration, and Delayed Recall. SAC scores are considered sensitive to change when the instrument is administered following a concussion. The SAC is designed for use by nonphysicians on the sidelines of an athletic event, although physicians or neuropsychologists may use the instrument. Whereas it offers a quantifiable measure that is feasible for rapid, sideline evaluation and also may be used for follow-up, the SAC is not intended as a substitute for more thorough medical, neurologic, or neuropsychological
evaluation. Three alternate forms were designed to allow follow-up testing with minimal practice effects in order to track postconcussion recovery. Appendix 6, Q2, summarizes 7 studies — all Class III — examining the use of the SAC as a diagnostic tool to identify the presence of a concussion. Four studies demonstrate relatively high sensitivity (0.80–0.94) and specificity (0.76–0.91) with the SAC when the test is used on the sideline after suspected injury.

**Conclusion.** The current evidence supports the use of the SAC as a diagnostic tool that is likely to identify the presence of concussion in the early stages postinjury (sensitivity 80%–94%, specificity 76%–91%) (multiple Class III studies). The moderate to high sensitivity and specificity reported support this conclusion, including the elevated postinjury score (best within 48 hours postinjury) relative to baseline rates and rates of control subjects. It is emphasized that an important proportion of athletes with concussion (6%–20%) will not be identified by the SAC as having concussion.

**Neuropsychological testing.** Appendix 6, Q2, summarizes 33 studies—1 Class II and 32 Class III—examining the use of neuropsychological testing as a diagnostic tool to identify the presence of a concussion. All studies employed a case-control design comparing athletes who were concussed with athletes who were not concussed. Some studies compared performance with baseline (nested case-control design).

The test measures are divided into two types on the basis of their method of administration: paper-and-pencil and computer. Study outcomes/effects were categorized as follows for identifying the presence of concussion: provision of sensitivity/specificity classification rates, detection of concussion versus no-concussion group differences, and detection of change in neuropsychological performance over the course of recovery. The single Class II study compared the performance of athletes with concussion on the Automated Neuropsychological Assessment Metrics (ANAM) test battery with the performance of athletes without injuries and athletes with musculoskeletal injuries. The concussed group performed significantly worse than the noninjured group on several subtests of the ANAM. The group with musculoskeletal injuries performed intermediately between the concussed group and noninjured group.
Examples of Class III studies as regards paper-and-pencil administration include McCrea et al., \(^{e48}\) who report sensitivity of 0.88 and specificity of 0.93 at day 7 postinjury. Examples of Class III studies as regards computer-based administration include Erlanger et al., \(^{e62}\) Collie et al., \(^{e63}\) and Broglio et al., \(^{e64}\) who report significant differences in memory and response speed between athletes with concussion and controls without concussion. Macciocchi et al., \(^{e65}\) Collins et al., \(^{e66}\) and Echemendia et al., \(^{e67}\) use paper-and-pencil tests, whereas Parker et al., \(^{e68}\) and Broglio et al. \(^{e69}\) use computer-administered tests in demonstrating sensitivity to cognitive recovery over time with serial neuropsychological assessment designs. Notably, no study examined outcomes in preadolescent athletes or girls’ sports exclusively.

**Conclusion.** When the reference standard of clinician diagnosis of concussion is used, it is likely that neuropsychological testing of memory performance, reaction time, and speed of cognitive processing, regardless of whether administered by paper-and-pencil or computerized method, is useful in identifying the presence of concussion (sensitivity 71%–88% of athletes with concussion) (one Class II study, multiple Class III studies). Twelve percent to 29% of athletes with concussion will not be identified as having concussion by neuropsychological testing.

There is insufficient evidence to support conclusions about the use of neuropsychological testing in identifying concussion in preadolescent age groups. It is important for the practitioner to understand the utility and limitations of different neurocognitive test batteries and the proper clinical context in which they may be used most effectively. There were no studies directly comparing different brands of computerized neurocognitive testing and thus no evidence to support one particular computerized test over another.

**Balance Error Scoring System.** The Balance Error Scoring System (BESS) is a clinical balance battery that involves use of 3 stances (double leg, single leg, tandem) on 2 surfaces (firm, foam) and is designed to evaluate postural stability. Four Class III studies \(^{e48,e49,e70,e71}\) utilized the BESS to assist in the diagnosis of concussion (appendix 6, Q2). In these studies the BESS was utilized to confirm the physician diagnosis at the time of injury or within 24 hours following the injury—often in combination with other assessment tools (see “Diagnostic measures used in combination” section). For identifying the presence of concussion, the studies consistently
demonstrated elevated BESS scores (i.e., worse balance performance) at the time of injury and within the initial 24 hours postinjury relative to the athletes’ individualized baseline scores or those of matched control subjects, or both.

One study\textsuperscript{e49} reported that BESS scores in college football players with concussion changed from baseline by approximately 6 points when the athletes were measured at the time of injury. At one day postinjury, each player’s average BESS score was approximately 3 points greater than baseline. For most athletes, BESS performance returned to preseason baseline levels (average 12 errors) by 3–7 days postinjury. These modest changes in BESS performance, as well as rapid recovery of static balance, have been reported in other studies of athletes. Another study of collegiate football players\textsuperscript{e48} reported BESS scores signaling impairment in 36% of injured subjects immediately following concussion, relative to 5% in the control group. Twenty-four percent of injured subjects demonstrated impairment when retested with the BESS at 2 days postinjury, relative to 9% by day 7 postinjury. Sensitivity values for the BESS were highest at the time of injury (sensitivity = 0.34). Specificity values for this instrument ranged from 0.91–0.96 across postinjury days (PIDs) 1–7.

Guskiewicz et al.\textsuperscript{e70} and Riemann et al.\textsuperscript{e71} identified differences on the BESS in collegiate athletes when compared with age- and sport-matched control subjects. Differences ranged between 6–9 BESS points when athletes with concussion were compared with athletes without concussion. Deficits relative to baseline typically recover within 3–5 days postinjury.\textsuperscript{e49,e70} Although quite specific, the BESS is not highly sensitive in detecting concussion. However, the sensitivity is increased when the BESS is used in combination with a graded symptom checklist and the SAC.\textsuperscript{e48}

**Conclusion.** When the reference standard of clinician diagnosis of concussion is used, the BESS assessment tool is likely to identify concussion with low to moderate diagnostic accuracy (sensitivity 34%–64%, specificity 91%) (multiple Class III studies). The BESS alone is not likely to identify a high proportion of athletes with concussion (false-negative rates 36%–66%).
**Sensory Organization Test.** The Sensory Organization Test (SOT) uses a force plate to measure a subject’s ability to maintain equilibrium while it systematically alters orientation information available to the somatosensory or visual inputs (or both). Seven studies utilized the SOT to assist in the diagnosis of concussion. Appendix 6, Q2, summarizes the studies—all Class III—examining the use of the SOT to identify both the presence of a concussion and concussion-related complications (more-prolonged recovery/impairment). In all cases the SOT was utilized to confirm the physician diagnosis at the time of the injury or within 24 hours postinjury—often in combination with other assessment tools (see “Diagnostic measures used in combination” section). For identifying the presence of concussion the studies consistently demonstrated lower SOT scores within 24 hours postinjury ranging between 8–12 points relative to individualized baseline rates or those of matched control subjects.

The SOT has been shown to be sensitive to detect concussion and especially to detect sensory interaction and balance deficits following concussion. As with the studies involving the BESS, studies utilizing the SOT have identified deficits on average about 3 days postinjury. One study reported sensitivity of 0.61 for the SOT, and a later study by the same group reported sensitivity of 0.57 and specificity of 0.80 (at a 75% CI). A study comparing athletes with concussion and healthy controls reported no differences in center of pressure (COP) displacement amplitude but reported a difference in the pattern of COP oscillations, using approximate entropy techniques.

**Conclusion.** When the reference standard of clinician diagnosis of concussion is used, the SOT assessment tool is likely to identify concussion with low to moderate diagnostic accuracy (sensitivity 48%–61%, specificity 85%–90%) (multiple Class III studies). The SOT alone is not likely to identify a high proportion of athletes with concussion (39%–52%).

**King-Devick test.** The King-Devick test is a quick and relatively simple test that measures timed reading of a series of irregularly spaced numbers on a handheld card. One Class III study showed that times increased significantly immediately after a match both in boxers and in mixed martial arts fighters who sustained head trauma. Use of the test to predict clinically diagnosed SRC was not reported.
Conclusion. Data are insufficient to support or refute the use of the King-Devick test for diagnosis of SRC.

Gait stability/dual tasking. Gait stability/dual tasking involves analysis of gait (using reflective markers on the extremities captured by a multicamera array) while the subject simultaneously completes simple mental tasks. Seven Class III studies utilized the gait stability test or a dual (virtual reality) task to assist in identifying concussion (appendix 6, Q2). These relatively new concussion assessment tools measure concurrent performance of motor and cognitive tasks. Several studies\textsuperscript{e68,e76–e78} have identified slowed gait or altered weight distribution (13\%–26\% center of mass deviation) during gait stability testing using single task and dual tasks (cognitive and motor). Significant differences in results on the gait tests have been noted in the dual-task gait assessment at day 28 postinjury, although not in the single-task or neuropsychological assessment. Two studies\textsuperscript{e79,e80} using divided-attention tasks in virtual environments identified subtle abnormalities in balance and moderate residual visual–motor disintegration in a small sample of patients with concussion. None of these studies reported sensitivity or specificity.

Conclusion. When the reference standard of clinician diagnosis of concussion is used, gait stability assessment and dual-task testing in virtual environments are possibly useful for identifying concussion (multiple Class III studies).

Imaging and electrophysiology. Twelve Class III articles used various MRI techniques to compare abnormalities in athletes who have concussion with athletes who are not injured. The techniques studied include diffusion tensor imaging,\textsuperscript{e81,e82} spectroscopy,\textsuperscript{e83–e87} and functional MRI.\textsuperscript{e88,e89} Four Class III studies compared electrophysiological parameters, including QEEG,\textsuperscript{e90} motor evoked potentials,\textsuperscript{e91} event-related potentials,\textsuperscript{e92} and EEG Shannon entropy,\textsuperscript{e93} between athletes with concussion and noninjured controls. The majority of imaging and electrophysiology studies demonstrated subtle and significant differences between athletes with concussion and athletes without concussion (appendix 6, Q2).
**Conclusion.** When the reference standard of clinician diagnosis of concussion is used, specialized imaging and electrophysiologic techniques are possibly useful in identifying athletes with concussion (multiple Class III studies).

**Diagnostic measures used in combination.** Eight Class III studies\(^{e48,e49,e54,e55,e94-e97}\) (appendix 6, Q2) have examined the contribution of multiple diagnostic methods (e.g., symptom report, neuropsychological testing, balance assessment) to the prediction of concussion diagnosis. Three studies examined the sensitivity and specificity of a multimodal assessment battery, reporting on the improvement in classification rates with this approach. Several studies examined the contribution of neuropsychological testing, symptom report, and balance assessment, reporting on the contribution of each method independently to the diagnostic prediction of concussion and to length of recovery. For example, Van Kampen et al.\(^{e54}\) reported sensitivity of the PCSS to be 64% in identifying athletes with concussion, whereas the combination of neurocognitive testing using Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) with the PCSS increased sensitivity to 83%. McCrea et al.\(^{e48,e49}\) exemplifies studies that demonstrate the contribution of each separate diagnostic measure in discriminating diagnostic groups and tracking recovery with the BESS, SAC, and paper-and-pencil neuropsychological testing.

**Conclusion.** A combination of diagnostic tests as compared with individual tests is likely to improve diagnostic accuracy of concussion (multiple Class III studies). Currently, however, there is insufficient evidence to determine the best combination of specific measures to improve identification of concussion.

**Q2b. For athletes suspected of having sustained concussion, what diagnostic tools are useful in identifying those at increased risk for severe or prolonged early impairments, neurologic catastrophe, or chronic neurobehavioral impairment?**

In addition to use for confirmation of the presence of concussion, diagnostic tools may potentially be used to identify athletes with severe or prolonged concussion-related early impairments, sports-related neurologic catastrophes (e.g., subdural hematoma), or chronic neurobehavioral impairments. No studies were found relevant to prediction of sports-related
neurologic catastrophe or chronic neurobehavioral impairment. Studies relevant to the identification of concussion-related early impairments are summarized in appendix 6, Q2.

**Postconcussion Symptom Scale or Graded Symptom Checklist.** Appendix 6, Q2, also summarizes the use of symptom scales to identify greater postconcussion impairments (6 studies: 1 Class I, 2 Class II, 3 Class III) in athletes. For identifying concussion-related impairments, one Class I study reported correlations between elevated symptom scores and persistent cognitive deficits. One Class II study showed that postconcussive headache symptoms were associated with worse computerized cognitive test performance on PID 7. Lavoie et al. demonstrated impaired reaction time and attention on a modified visual oddball paradigm in athletes with symptomatic concussion relative to that of controls or athletes with asymptomatic concussion. Three Class III studies utilized the PCSS or GSC to identify associations between elevated symptoms and LOC at the time of injury and persistent headaches or protracted recovery in athletes.

**Conclusion.** It is likely that elevated postconcussive symptoms are associated with more-severe or prolonged early postconcussive cognitive impairments (6 studies: 1 Class I, 2 Class II, 3 Class III).

**Standardized Assessment of Concussion.** Two Class 1 studies demonstrated that the SAC detects significant differences between athletes with concussion relative to their baseline function and relative to the baseline function of noninjured controls. These differences were most notable within 48 hours postinjury.

**Conclusion.** It is likely that lower SAC scores are associated with more-severe or prolonged early postconcussive impairments (2 Class I studies).

**Neuropsychological testing.** Six studies were found that examined identification of prolonged concussion-related impairments. One Class I study and 1 Class II study used paper-and-pencil testing. Two Class I and 2 Class II studies reported on computerized neuropsychological testing. A Class II study reported that paper-and-pencil
neuropsychological testing (Repeatable Battery for the Assessment of Neuropsychological Status) demonstrates subclinical cognitive impairment in athletes, whereas another Class II study found that a slower recovery course was 18x more likely with three low scores on ImPACT, a computerized neuropsychological test.

**Conclusion.** It is likely that neuropsychological testing predicts delayed postconcussion recovery (three Class I and three Class II studies).

**Balance Error Scoring System.** One Class I study identified a prolonged recovery curve for BESS, with increased error scores seen as late as 10 days postconcussion in a subset of individuals.

**Conclusion.** It is possible that BESS identifies athletes with early postconcussion impairments (one Class I study).

**Sensory Organization Test.** One Class I study and one Class II study identified a prolonged recovery curve for SOT, with increased error scores seen as late as 10 days postconcussion in small subsets of individuals.

**Conclusion.** It is likely that SOT identifies athletes with early postconcussion impairments (one Class I study, one Class II study).

**Gait stability/dual tasking.** Two studies identified prolonged recovery after concussion as measured using gait testing (Class I) and divided-attention (Class III) tasks but were limited by very small sample sizes.

**Conclusion.** It is possible that gait stability dual tasking testing identifies athletes with early postconcussion impairments (one Class I study, one Class III study).

**Diagnostic measures used in combination.** Nine studies (5 Class I, 3 Class II, and 1 Class III) analyzed the use of combined measures for predicting early postconcussion impairments
With respect to predicting length of recovery, Lau et al., using discriminant function analysis, report the classification rates of short or long recovery using neurocognitive testing and symptom assessment separately (PCSS 63.21%, 4 ImPACT composites 65.38%) and in combination (73.53%). Iverson reported that athletes with complex concussions (recovery time >10 days) performed more poorly on neuropsychological testing and reported more symptoms than those with simple concussions (recovery time <10 days).

**Conclusion.** A combination of diagnostic tests as compared with individual tests is highly likely to improve the prediction of length of recovery (9 studies: 5 Class I, 3 Class II, and 1 Class III). At the time of this writing, however, there is insufficient evidence to determine the best combination of specific measures to improve prediction of prolonged recovery.

**Clinical context:** Most studies support the use of some tools (PCSS, GSC) for tracking recovery beyond the initial assessment and utilizing the scores for a more-informed RTP decision. However, analysis of the data supporting such use is beyond the scope of this question.

**For athletes with concussion, what clinical factors are useful in identifying those at increased risk for severe or prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions, or chronic neurobehavioral impairment?**

For this question, we grouped the evidence by the following 4 risk factor types to maximize clinical utility: severe or prolonged early postconcussion impairments, neurologic catastrophe, recurrent concussions and chronic neurobehavioral impairment. Our literature search screened 3523 abstracts, the articles for 769 of which were reviewed in detail. Of these 769 articles, 235 publications were reviewed and pertinent evidence extracted. We found 28 Class I, 25 Class II, and 16 Class III studies to be relevant (appendix 6, Q3).

**Severe or prolonged early postconcussion impairments.** We found 32 studies relevant to predictors of more-severe or prolonged early postconcussion symptoms, some of which also measured cognitive impairment; of these there were 15 Class I, 8 Class II, and 9 Class III studies (appendix 6, Q3). The vast majority of these studies were of high school and collegiate athletes,
although one Class I study evaluated peewee ice hockey players (aged 11–12 years) and reported an annual IRR of 2.76 (95% CIs 1.1–6.9) for severe concussion (>10 playing days lost) in those with a previous history of any concussion relative to those with no prior concussions. 

**Acute symptoms.** Fifteen studies addressed whether very early concussion symptoms could help predict the severity or duration of postconcussive impairments (4 Class I, 6 Class II, and 5 Class III studies).

One study of Australian-rules footballers tested within 11 days after a concussion and when still symptomatic relative to their individual baselines on a computerized cognitive battery (CogSport) showed significantly slowed reaction times and no improvement on the Digit Symbol Substitution Task or Trails B relative to players with concussion who were asymptomatic at testing time. This study was limited in that it did not indicate the specific times postconcussion when the tests were administered. Furthermore, the concussed group that was symptomatic at testing also had a significantly greater number of symptoms at the time of injury than the asymptomatic group (4.9±2.0 versus 3.3±1.1, \( p=0.002 \)), tended to have more prior concussions (3.1 versus 2.4, \( p=0.12 \)) and had a higher percentage who lost consciousness at the time of injury (32% versus 19.4%, \( p=0.27 \)). Nonetheless, this study supports the position that clinical symptoms are associated with objective measures of cognitive impairment. Another Class I study prospectively followed National Hockey League players, measuring 559 concussions over 7 seasons. Significant predictors for time lost from playing include postconcussive headache (\( p<0.001 \)), fatigue (\( p=0.01 \)), amnesia (\( p=0.02 \)), and an abnormal neurologic examination (\( p=0.01 \)). Risk factors for missing >10 days of playing time were headache (odds ratio [OR] 2.17 [95% CI 1.33–3.54]) and fatigue (OR 1.72 [95% CI 1.04–2.85]).

The third Class I study showed no significant effect of LOC (\( p=0.09 \)) or amnesia (\( p=0.13 \)) on predicting prolonged recovery. The final Class I study looked at biomechanical predictors of worse early concussion impairments in high school football players using the Head Impact Telemetry System (HITS). Parameters studied include time from session start until injury, time from previous impact, peak linear acceleration, peak rotational acceleration, and HIT severity profile. Nineteen players sustained 20 SRCs over 4 years; however, none of the
biomechanical parameters measured showed an association with postconcussion symptoms or cognitive dysfunction.

Six studies also demonstrated a relationship between specific acute symptoms or early cognitive test results and greater “severity” of concussion as determined by longer duration of symptoms or cognitive impairment (or both) or delayed RTP. One study showed that athletes complaining of headaches 7 days postinjury were more likely to have had on-field anterograde amnesia (OR 2.67, 95% CI 1.03–6.92), 3 or 4 abnormal on-field markers (LOC, retrograde amnesia [RGA], posttraumatic [anterograde] amnesia [PTA], disorientation; OR 4.07, 95% CI 1.25–13.23) or >5 minutes of mental status change (OR 4.89, 95% CI 1.74–13.78). Furthermore, relative to athletes with concussion who had no headaches at PID 7, those with headaches showed significant impairments in reaction time and memory scores (ImPACT) and a greater number of total symptoms.\textsuperscript{e98}

A separate article from the same center studied 108 high school football players and divided them into 2 groups on the basis of whether they met any of the following four criteria for “complex” concussion: concussive convulsion, LOC >1 min, history of multiple prior concussions, or nonrecovery by 10 days postinjury. Those individuals meeting at least one of the four criteria were classified as having complex concussions; those meeting none of the criteria were considered as having simple concussions. Standardized symptom scores and computerized cognitive tests (ImPACT) were obtained on average 2.2 days postinjury. Significant differences between simple and complex concussions were found in symptom clusters related to migraine (\(p=0.001\)), cognitive function (\(p=0.02\)), and sleep disturbances (\(p=0.01\)), and for cognitive measures of impaired visual memory (\(p=0.01\)) and prolonged processing speed (\(p=0.007\)). No significant differences between groups were detected for the neuropsychiatric symptom cluster, verbal memory, or reaction time.\textsuperscript{e51}

The third Class II study used medical clearance for RTP in Australian-rules footballers as a concussion severity marker.\textsuperscript{e103} With use of a Cox proportional hazard model, symptoms associated with prolonged RTP include headache >60 hours, fatigue/tiredness, “fogginess,” or >3 symptoms at initial presentation. Headache <24 hours was associated with a more-rapid RTP. In
this study, deficits on paper-and-pencil cognitive testing (Digit Symbol Substitution Test and Trails B) paralleled symptom recovery. Deficits on a computerized test battery (CogSport) showed a 2- to 3-day recovery lag, which was interpreted as being a more-sensitive indicator of incomplete recovery. This study was limited in that the cognitive test results were available to the team physicians and potentially could influence the outcome measure (RTP).

Another Class II study was a preliminary report\textsuperscript{104} using methodology similar to that reported by Collins and colleagues\textsuperscript{98}; however, subjects were stratified not by headache symptoms but rather by the presence or absence of “fogginess” as part of standardized symptom and computerized cognitive assessment testing (ImPACT) conducted on average 6.8 days postinjury. Significant differences in total symptom scores (foggy 35.5±22.9 versus not foggy 3.7±6.5, \(p<0.0001\)), composite reaction time (\(p<0.002\)), composite processing speed (\(p<0.004\)), and composite memory (\(p<0.01\)) were detected. Effect sizes in this study were notably greater than in the earlier study of postconcussive headache.\textsuperscript{98} In a Class II study of 247 high school and collegiate athletes, those with baseline headache were more likely to have suffered \(\geq 3\) prior concussions and to endorse postconcussive symptoms. The presence and severity of posttraumatic headache were associated with a greater number of posttraumatic symptoms (\(p<0.02\)), decreased balance scores (\(p=0.05\)), and reduced cognitive test scores on PID 1 (\(p<0.05\)).\textsuperscript{105} When a numerical threshold approach was used, migraine symptoms, cognitive symptoms, visual memory, and processing speed could be used to distinguish between short (\(\leq 14\) days) and long (>14 days) recovery.\textsuperscript{106} However, in this study, over one-third of the athletes (69/177) did not have follow-up. Furthermore, even when thresholds with 80% sensitivity were used, none of the parameters just mentioned showed a specificity greater than 16%.

Prior traumatic brain injury/concussion. Six studies reported the relationship of prior TBI/concussions on the severity or duration of postconcussion recovery (4 Class I studies,\textsuperscript{15,39,42,101} 2 Class III studies\textsuperscript{80,107}). In professional hockey players, time missed from play increased by 2.25x (95% CI 1.41–3.62) for each recurrent concussion.\textsuperscript{101} In junior hockey players, a history of prior concussions carried a greater risk (IRR=2.76 [95% CI 1.10–6.91]) for a concussion that took >10 days for recovery.\textsuperscript{42} Duration of recovery was related to the number of prior concussions (\(p=0.03\)) in collegiate football players.\textsuperscript{39} Number of symptoms was associated
with prior concussion. These 4 studies are Class I. One Class III study showed higher rates of early symptoms (LOC, PTA, confusion, 5+ minutes of mental status change) in athletes with a history of 3+ concussions when compared with those with no concussion history.

**Gender.** Five Class I studies investigated gender differences in the severity of early postconcussion symptoms and neurocognitive testing, with inconsistent results. Females had worse reaction times (Concussion Resolution Index, ImPACT) and worse visual memory on computerized testing (ImPACT). These studies had conflicting data on the severity of postconcussive symptoms, with females having more symptoms in 2 of the 3 studies just mentioned. In the third study males reported more symptoms of sadness and vomiting than females. In a study of symptoms, males endorsed more amnesia and confusion, whereas females reported drowsiness and phonophobia more often. Finally, no significant gender difference was reported in postconcussive depression.

**Age/Level of play.** Two Class I studies described an association between younger age/lower level of play and greater severity or duration of postconcussive symptoms or cognitive impairment. In a comparison of high school athletes and collegiate athletes, the former had symptoms and cognitive impairments of longer duration. In addition, cognitive impairments were still detectable by PID 7, a point at which postconcussive symptoms had resolved. A second study compared postconcussive performance using computerized cognitive testing (ImPACT) between National Football League (NFL) and high school football players. Whereas the assessment times were not matched (NFL athletes were tested generally 1–3 days earlier postinjury than high school athletes), high school athletes showed greater cognitive deficits at follow-up 1 and tended to have lower scores at follow-up 2, although there were no significant differences at time 2.

**Sport-related factors.** Body checking was associated with an increased risk for severe concussion in a Class I study of peewee ice hockey. A higher rate of Cantu grade II concussions was reported in a Class I study of injuries sustained on artificial turf (22%) relative to those sustained on natural grass (9%).

In professional football, quarterbacks were more likely to sustain a “severe” concussion, resulting in >7 days out of play (Class III study). Another Class III study of ice hockey
showed less time lost postconcussion in players wearing full-face shields rather than half-face shields.\textsuperscript{e115}

\textbf{Athlete-related factors.} Biomechanical measures were obtained from helmet-based accelerometers in a Class I study and found not to be predictive of suffering an SRC nor of having more-severe symptoms when an SRC is experienced.\textsuperscript{e102} A Class II study reported that athletes with preexisting headaches had more symptoms and lower neurocognitive scores (ANAM) after concussions.\textsuperscript{e105}

\textbf{Conclusions.} It is highly probable that ongoing clinical symptoms are associated with persistent neurocognitive impairments demonstrated on objective testing (1 Class I study,\textsuperscript{e63} 2 Class II studies\textsuperscript{e51,e98}). There is also a high likelihood that history of concussion (4 Class I studies,\textsuperscript{e15,e39,e42,e101} 2 Class III studies\textsuperscript{e80,e107}) is associated with more-severe/longer duration of symptoms and cognitive deficits. Probable risk factors for persistent neurocognitive problems or prolonged RTP include early posttraumatic headache (1 Class I study,\textsuperscript{e101} 5 Class II studies\textsuperscript{e51,e98,e103,e105,e106}); fatigue/fogginess (1 Class I study,\textsuperscript{e101} 2 Class II studies\textsuperscript{e103,e104}); and early amnesia, alteration in mental status, or disorientation (1 Class I study,\textsuperscript{e101} 1 Class II study,\textsuperscript{e98} 2 Class III studies\textsuperscript{e52,e98}). It is also probable that younger age/level of play (2 Class I\textsuperscript{e99,e113} is a risk factor for prolonged recovery. In peewee hockey, body checking is likely to be a risk factor for more-severe concussions as measured by prolonged RTP (1 Class I study\textsuperscript{e42}). Possible risk factors for persistent neurocognitive problems include prior history of headaches (1 Class II study\textsuperscript{e105}). Possible risk factors for more-prolonged RTP include having symptoms of dizziness (1 Class III study\textsuperscript{e116}), playing the quarterback position in football (1 Class III study\textsuperscript{e114}), and wearing a half-face shield in hockey (relative to wearing full-face shields, 1 Class III study\textsuperscript{e115}). In football, playing on artificial turf is possibly a risk factor for more-severe concussions (1 Class I study,\textsuperscript{e15} but small numbers of repeat concussions). There is conflicting evidence as to whether female gender or male gender is a risk factor for more postconcussive symptoms, so no conclusion could be drawn.

\textbf{Neurologic catastrophe.} We found no studies that measured the incidence or risk of severe TBI or intracranial complications after SRCs. Evidence pertaining to second-impact syndrome is
limited to case reports or series (Class IV\textsuperscript{e117}) and is excluded in accordance with AAN criteria. There is some controversy regarding the existence of this syndrome.\textsuperscript{e118,e119}

**Conclusion.** Data are insufficient to identify specific risk factors for catastrophic outcome after SRCs were found (although studies exist for mTBI in general).

**Recurrent concussions.** Ten studies were identified that had relevance to risk factors for recurrent concussion. Eight are Class I,\textsuperscript{e15,e34,e39,e40,e42,e66,e120,e121} one is Class II,\textsuperscript{e122} and one is Class III.\textsuperscript{e123}

**Prior concussion.** Six Class I studies\textsuperscript{e15,e34,e39,e40,e42,e120} and one Class II study\textsuperscript{e122} reported prior concussion as a risk factor for recurrent concussion (appendix 6, Q3). Emery et al.\textsuperscript{e76} showed a concussion IRR (relative rate) of 2.14 (95% CI 1.28–3.55) for peewee ice hockey players with a history of concussion relative to those with no prior concussion. A prior concussion was associated with a 1.6x–3x increased risk of concussion in multiple studies.\textsuperscript{e15,e34,e40,e120} One study (Class I) showed a "dose response" (risk of recurrence increases with number of concussions: after one concussion 1.5x, two concussions 2.8x, and three or more prior concussions 3.4x),\textsuperscript{e39} whereas another (Class I) did not.\textsuperscript{e120} Another study (Class II) of sport-related head injuries presenting to an emergency department reported a hazard ratio of 2.6x (95% CI 2.2–3.1) after one head injury and 5.9x (95% CI 3.4–10.3) after 2 head injuries.\textsuperscript{e122}

**Athlete-related factors.** A relationship between total years participating in football and total number of concussions was reported in high school players ($r=0.15$, $p<0.02$; Class I\textsuperscript{e66}). In this study of high school players, quarterbacks and tight ends had the highest rates of prior concussion, and running backs and kickers had the lowest rates. In a different study of professional football players, quarterbacks were at greatest risk (OR=1.92, 95% CI 0.99–3.74, $p<0.1$), and offensive linemen were at the least risk (OR=0.54, 95% CI 0.27–1.08, $p<0.1$) for repeat concussions, although neither of these positional analyses achieved significance (Class III\textsuperscript{e123}).
Time since previous concussion. In a study determining whether a symptom-free waiting period (SFWP) after concussion affected outcome, almost 80% of repeat concussions occurred within 10 days of the initial injury (Class I). Curiously, although the rate of repeat concussion was higher in the SFWP group than in the non-SFWP group, those in the SFWP group with repeat injuries returned to play 3.55 days sooner ($p<0.05$) than those with no repeat concussions. A separate study of college athletes found 92% of repeat concussions occurred within 10 days after the first concussion (Class I). Both studies had relatively small numbers of repeat concussions (24 and 12, respectively), but the timing results were consistent.

Conclusions. A history of concussion is a highly probable risk factor for recurrent concussion (6 Class I studies, 1 Class II study). It is also highly likely that there is an increased risk for repeat concussion in the first 10 days after an initial concussion (2 Class I studies), an observation supported by pathophysiologic studies. Probable risk factors for recurrent concussion include longer length of participation (one Class I study) and quarterback position played in football (one Class I study, one Class III study).

Chronic neurobehavioral impairment. Thirty-four studies (11 Class I, 16 Class II, 7 Class III) investigated risk factors for chronic neurobehavioral impairment (appendix 6, Q3). Nine studies include professional athletes, and 23 studies include amateur athletes. One study includes both professional and amateur athletes, and one study of soccer players did not specify the level of play. Evidence related specifically to chronic traumatic encephalopathy (CTE) was limited to case reports and series (Class IV) and did not meet AAN criteria for evidence-based recommendations. This level of evidence does not permit identification of incidence rates or risk factors.

Prior concussion in professional athletes. There were 10 studies (2 Class I, 7 Class II, 1 Class III) that used various forms of neuropsychological testing in professional athletes to examine the relationship between prior TBI and the development of chronic impairments. Studies in football players, boxers, soccer players, and licensed jockeys described an increased risk of chronic neurocognitive impairments with a greater exposure to
prior concussions. Because history of TBI/concussion was generally diagnosed retrospectively, these studies do not specify how the prior concussions were managed.

Both studies reported associations between prior concussion/exposure and neurocognitive impairment (1 study in rugby players showed chronic impairments as compared with noncontact athletes\textsuperscript{124}). A Class I study of jockeys found chronic neurocognitive deficits in those with a history of concussion and a relationship between multiple concussions and greater impairments.\textsuperscript{126}

Seven Class II studies detected an association between prior TBI and chronic neurobehavioral deficits; only one study did not show that result.\textsuperscript{139} A study of 30 professional boxers (Jordan et al.)\textsuperscript{133} reported an association between apoE4 genotype, high exposure to TBI (>12 professional bouts), and a clinical diagnosis of chronic TBI ($p<0.001$, Class II). ApoE4 genotype, particularly in conjunction with increasing age (as a surrogate for exposure to repeated mTBI), was also associated with greater cognitive impairment in professional football players (Class II\textsuperscript{136}). From results of a retrospective health questionnaire obtained from 2552 retired professional football athletes, an association was found between recurrent concussions and a clinical diagnosis of minimal cognitive impairment ($p=0.02$) and self-reported memory problems ($p=0.001$; Class II\textsuperscript{134}). Whereas this study did not detect a direct association with Alzheimer disease (AD), an earlier onset of AD in the NFL retiree population was shown relative to that in the general adult male population (age-adjusted prevalence ratio for AD = 1.37 [95% CI 0.98–1.56]). In another study using the same health survey, a relationship was also reported between recurrent concussions and a lifetime risk of depression ($p<0.005$, Class II\textsuperscript{135}). Players with 1–2 prior concussions (1.5x) and those with $\geq 3$ concussions (3x) were more likely to be diagnosed with depression relative to retired players without concussion history. Two studies\textsuperscript{137,138} with possibly overlapping cohorts showed neurocognitive impairments in professional soccer players as compared with control (swimming and track) athletes and a dose relationship between headers, concussions, and cognitive impairments. A third, larger study showed no such relationship.\textsuperscript{139}
Prior concussion in amateur athletes. Nine Class I, and 3 Class III studies examined the relationship between prior mTBI and the presence of neurobehavioral impairments in nonprofessional athletes (appendix 6, Q3). The sports studied include rugby, football, and soccer; the majority of studies included multiple sports.

Four Class I studies described an association between prior concussions and chronic cognitive dysfunction; five Class I studies did not show that result.

Six Class II studies supported a relationship between prior concussions and neurobehavioral deficits; two Class II studies did not show that relationship. One Class II study showed mixed results.

One Class III study supported an association. A second preliminary study did also; however, when a larger follow-up study from the same group was completed, no relationship between prior SRC and cognition was found.

Gender. Four studies (2 Class I, 1 Class II, 1 Class III) reported on gender with regard to chronic health effects.

One Class I study found slower reaction times, more symptoms, and lower neurocognitive scores (ImPACT) among female athletes. In another study involving 260 youth, high school and collegiate athletes, chronic impairments as measured by Rivermead Post Concussion Questionnaire were more frequently reported in female adults but not in female minors.

In a Class II study of 188 high school and collegiate athletes tested with ImPACT, females with a history of 2–3 concussions performed better than males with ≥2 concussions, with specific differences observed in visual memory, motor-processing speed, and reaction time.

Age. Three Class I studies reported on age in relationship to chronic problems. In a Class I study of 698 subjects that involved paper-and-pencil neuropsychological testing, prior
TBI and younger age were associated with reliable decrements on neurocognitive performance.\textsuperscript{e126} In a study of 111 rugby players, clinical symptoms and complaints of memory impairment were associated with exposure in retired and older recreational rugby players but not in active (younger) rugby players.\textsuperscript{e132} In a study of 40 patients examining the role of “heading” in elite national team soccer players developing chronic cognitive dysfunction, the authors noted no association with age, symptoms, or MRI findings and concluded that the reported head injury symptoms appeared to be related to acute head injuries rather than “heading” behaviors.\textsuperscript{e125}

One Class II study examined age and history of concussions in regard to neurocognitive function and event-related potentials.\textsuperscript{e92} Adolescents with a history of concussion scored lower on 1 of 10 cognitive tests administered; no age effects were seen for electrophysiologic measures.

\textit{Sports-related factors.} Sports-related factors that affect neurocognitive function were examined in 4 Class I (rugby position\textsuperscript{e124}; heading\textsuperscript{e125,e127,e130}) and 3 Class II (heading\textsuperscript{e137–e139}) studies. Six of these were related to heading in soccer.

In a Class I study of neurocognitive performance among 226 athletes with prior TBI, rugby players were found to perform worse on visuomotor speed as compared with noncontact control athletes.\textsuperscript{e124} Of note the rugby players had a higher percentage of individuals with \( \geq 2 \) concussions than the noncontact athletic controls, but rugby position did not significantly contribute to cognitive performance. A study of 254 collegiate athletes examining whether prior TBI and sport were neurocognitive risk factors found no evidence of sport-specific deficits on cognitive testing.\textsuperscript{e127} No association was found between heading in soccer and deficits on neuropsychological paper-and-pencil testing\textsuperscript{e130} or MRI findings.\textsuperscript{e125}

Conflicting data exist for cognitive impairments in relation to heading in soccer players. On the basis of computerized neuropsychological testing of professional soccer players, it was noted that no long-standing neuropsychological deficits were associated with heading or previous concussion.\textsuperscript{e139} Another Class II study of collegiate students (soccer athletes, nonsoccer athletes, and controls) found no association between participation in soccer and neurocognitive deficits, although heading was not specifically studied.\textsuperscript{e144} However, two other Class II studies reported
different findings with heading. One study found an association between heading and neurocognitive deficits. In a second study from the same group of investigators that involved 84 athletes, an association between a greater number of headers and attention and verbal memory dysfunction was observed.\textsuperscript{137,138}

\textit{Athlete-related factors.} Two Class II studies\textsuperscript{133,136} examined the relationship between ApoE genotype and chronic neurologic deficits. In a study of 30 boxers, high-exposure boxers with an ApoE epsilon4 allele had lower scores on the clinical Chronic Brain Injury (CBI) rating scale.\textsuperscript{133} A second study, examining professional football players, reported that those with the ApoE epsilon4 allele had lower cognitive test scores than players without this genotype; there was a relationship between cognitive dysfunction and ApoE epsilon4 and increasing age.\textsuperscript{136} No player had sustained TBI or concussion within 9 months of cognitive assessment. Neither of these studies reported a difference between heterozygotes and homozygotes. One Class I study found the combination of prior diagnosis of learning disability and history of multiple concussions to be associated with lower neurocognitive test results.\textsuperscript{66}

\textit{Conclusions.} Prior concussion exposure is highly likely to be a risk factor for chronic neurobehavioral impairment across a broad range of professional sports, and there appears to be a relationship with increasing exposure (2 Class I studies, 6 Class II studies, in football, soccer, boxing, and horseracing). Evidence is insufficient to determine if there is a relationship between chronic cognitive impairment and heading in professional soccer (inconsistent Class II studies). Data are insufficient to determine whether prior concussion exposure is associated with chronic cognitive impairment in amateur athletes. Likewise, data are insufficient to determine whether the number of heading incidents is associated with neurobehavioral impairments in amateur soccer. ApoE4 genotype is likely to be associated with chronic cognitive impairment after concussion exposure (2 Class II studies), and preexisting learning disability may be a risk factor (1 Class I study). Data are insufficient to conclude whether gender and age are risk factors for chronic postconcussive problems.

\textbf{For athletes with concussion, what interventions enhance recovery, reduce the risk of recurrent concussion, or diminish long-term sequelae?}
Our literature search screened 892 abstracts; the full-text articles of 116 of those abstracts were reviewed in detail. Of these 116 articles, 15 publications were reviewed and pertinent evidence extracted. These studies are summarized in appendix 6, Q4.

Three Class III studies addressed interventions to enhance concussion recovery or mitigate postconcussive complications. One retrospective study of 95 athletes who presented to a university-based sports concussion clinic graded self-reported postconcussion activity levels (range: 0 [no activity] to 4 [full academic and athletic activity]) and then compared symptom checklists and computerized cognitive testing between groups. The moderate-activity group (level 2 = school activity and sports practice) performed better on visual memory ($p=0.003$) and reaction time ($p=0.0005$) testing, and trended toward fewer symptoms ($p=0.08$) relative to those with higher (or lower) postinjury exertion levels.

Another study combined prospectively acquired data from 3 parallel, multicenter studies to investigate 635 athletes with concussion. These athletes were separated into those who underwent a symptom-free waiting period (SFWP) of any duration (60.3%) and those without SFWP (39.7%). All underwent a battery of testing that included GSC, SAC, BESS, and a global neuropsychological test score. Baseline scores were compared with scores on the same measures taken during the first postconcussion week and with a final set of testing conducted 45–90 days postinjury; no differences were found between SFWP and non-SFWP groups for either acute injury or chronic injury test scores. Of the 24 athletes who sustained a second concussion during the same season, only 2 were in the non-SFWP group. The remaining 22 athletes (all in the SFWP group) showed significantly shorter duration of SFWP (2.96 days) and RTP (6.2 days) than the athletes with SFWP who did not sustain second concussions that season (5.78 days and 9.75 days, respectively).

The third study investigated a group of 12 patients with refractory postconcussion symptoms and measured the effects of controlled exercise, specifically subsymptom threshold exercise training (SSTET). The results showed that training could be conducted safely, and that after SSTET, subjects could exercise longer (pretraining exercise duration 9.8 minutes, posttraining 18.7
minutes), with higher peak heart rate (147 pretraining versus 179 posttraining) and systolic blood pressure (142 pretraining versus 156 posttraining) but without symptom exacerbation.

One Class III open-label study used amantadine in athletes who had not recovered by 21 days postinjury as compared with untreated controls. This study reported more-rapid symptom recovery and modest group differences in verbal memory and reaction time. However, there was no blinding or placebo control, and the two groups had neurocognitive differences at baseline.159

**Conclusion.** Each of these studies addresses a different aspect of postconcussion intervention, providing evidence that was graded as very low to low. On the basis of the available evidence, no conclusions can be drawn regarding the effect of postconcussive activity level on the recovery from concussion or the likelihood of developing chronic postconcussion complications. There was no randomized, controlled evidence to support the use of specific medications or nutritional supplements to enhance recovery from SRC.

**RECOMMENDATIONS**
For this guideline, recommendations have each been categorized as one of three types: 1) preparticipation counseling recommendations; 2) recommendations related to assessment, diagnosis, and management of suspected concussion; and 3) recommendations for management of diagnosed concussion (including acute management, RTP, and retirement). In this section, the term experienced licensed health care provider (LHCP) refers to an individual who has acquired knowledge and skills relevant to evaluation and management of sports concussions and is practicing within the scope of his or her training and experience. The role of the LHCP can generally be characterized in 1 of 2 ways: sideline (at the sporting event) or clinical (at an outpatient clinic or emergency room). The clinical contextual profiles transparently indicating the panel’s judgments used to formulate the recommendations are indicated in appendix 9.

**Preparticipation counseling**

**Clinical context: Preparticipation counseling**

Our review indicates that there are a number of significant risk factors for experiencing a concussion or a recurrent concussion in a sports-related setting. It is accepted that individuals
should be informed of activities that place them at increased risk for adverse health consequences.

**Practice recommendation: Preparticipation counseling**

1. School-based professionals should be educated by experienced LHCPs designated by their organization/institution to understand the risks of experiencing a concussion so that they may provide accurate information to parents and athletes (Level B).
2. To foster informed decision making, LHCPs should inform athletes (and where appropriate, the athletes’ families) of evidence concerning the concussion risk factors as listed below. Accurate information regarding concussion risks also should be disseminated to school systems and sports authorities (Level B).
   A. *Age or competition level.* There is insufficient evidence to make any recommendation as to whether age or competition level affects the athlete’s overall concussion risk.
   B. *Type of sport.* Among commonly played team sports with data available for systematic review, there is strong evidence that concussion risk is greatest in football, rugby, hockey, and soccer.
   C. *Gender.* Clear differences in concussion risk between male and female athletes have not been demonstrated for many sports; however, in soccer and basketball there is strong evidence that concussion risk appears to be greater for female athletes.
   D. *Equipment.* There is moderate evidence indicating that use of a helmet (when well fitted, with approved design) effectively reduces, but does not eliminate, risk of concussion and more-serious head trauma in hockey and rugby; similar effectiveness is inferred for football. There is no evidence to support greater efficacy of one particular type of football helmet, nor is there evidence to demonstrate efficacy of soft head protectors in sports such as soccer or basketball.
   E. *Position.* Data are insufficient to support any recommendation as to whether position increases concussion risk in most major team sports.
   F. *Prior concussion.* There is strong evidence indicating that a history of concussion/mTBI is a significant risk factor for additional concussions. There is
moderate evidence indicating that a recurrent concussion is more likely to occur within 10 days after a prior concussion.

Suspected concussion

**Clinical context: Use of checklists and screening tools for suspected concussion**

The diagnosis of an SRC is a clinical diagnosis based on salient features from the history and examination. Although different tests are used to evaluate an athlete with suspected concussion initially, no single test score can be the basis of a concussion diagnosis. There is moderate evidence that standardized symptom checklists (PCSS/GSC) and the SAC when administered early after a suspected concussion have moderate to high sensitivity and specificity in identifying sports concussions relative to those of the reference standard of a clinician-diagnosed concussion. There is low-moderate evidence that the BESS has low to moderate sensitivity and moderate to high specificity in identifying sports concussions. Generally, physicians with expertise in concussion are not present when the concussion is sustained, and the initial assessment of an injured athlete is done by a team’s athletic trainer, a school nurse, or, in amateur sports in the absence of other personnel, the coach. These tools can be implemented by nonphysicians who are often present on the sidelines. Proper use of these tests/tools requires training. Postinjury scores on these concussion assessment tools may be compared with age-matched normal values or with an individual’s preinjury baseline. Physicians are formally trained to do neurologic and general medical assessments and to recognize signs and symptoms of concussion and of more-severe TBI.

**Practice recommendations: Use of checklists and screening tools for suspected concussion**

1. Inexperienced LHCPs should be instructed in the proper administration of standardized validated sideline assessment tools. This instruction should emphasize that these tools are only an adjunct to the evaluation of the athlete with suspected concussion and cannot be used alone to diagnose concussion (Level B). These providers should be instructed by experienced individuals (LHCPs) who themselves are licensed, knowledgeable about sports concussion, and practicing within the scope of their training and experience, designated by their organization/institution in the proper administration of the standardized validated sideline assessment tools (Level B).
2. In individuals with suspected concussion, these tools should be utilized by sideline LHCPs and the results made available to clinical LHCPs who will be evaluating the injured athlete (Level B).

3. LHCPs caring for athletes might utilize individual baseline scores on concussion assessment tools, especially in younger athletes, those with prior concussions, or those with preexisting learning disabilities/ADHD, as doing so fosters better interpretation of postinjury scores (Level C).

4. Team personnel (e.g., coaching, athletic training staff, sideline LHCPs) should immediately remove from play any athlete suspected of having sustained a concussion, in order to minimize the risk of further injury (Level B).

5. Team personnel should not permit the athlete to return to play until the athlete has been assessed by an experienced LHCP with training both in the diagnosis and management of concussion and in the recognition of more-severe TBI (Level B).

**Clinical context: Neuroimaging for suspected concussion**

No specific imaging parameters currently exist for suspected SRC, but there is strong evidence to support guidelines for selected use of acute CT scanning in pediatric and adult patients presenting to emergency departments with mTBI. In general, CT imaging guidelines for mTBI were developed to detect clinically significant structural injuries and not concussion.\(^{e160,e161}\)

**Practice recommendation:**

CT imaging should not be used to diagnose SRC but might be obtained to rule out more serious TBI such as an intracranial hemorrhage in athletes with a suspected concussion who have LOC, posttraumatic amnesia, persistently altered mental status (Glasgow Coma Scale <15), focal neurologic deficit, evidence of skull fracture on examination, or signs of clinical deterioration (Level C).

**Diagnosed concussion**

**Clinical context: RTP – risk of recurrent concussion**

There is moderate to strong evidence that ongoing symptoms are associated with ongoing cognitive dysfunction and slowed reaction time after sports concussions. Given that postinjury
cognitive slowing and delayed reaction time can have a negative effect on an athlete’s ability to play safely and effectively, it is likely that these symptoms place an athlete at greater risk for a recurrence of concussion. There is weak evidence from human studies to support the conclusion that ongoing concussion signs and symptoms are risk factors for more-severe acute concussion, postconcussion syndrome, or chronic neurobehavioral impairment. Medications may frequently mask or mitigate postinjury symptoms (e.g., analgesic use for headache).

*Practice recommendations: RTP – risk of recurrent concussion*

1. In order to diminish the risk of recurrent injury, individuals supervising athletes should prohibit an athlete with concussion from returning to play/practice (contact-risk activity) until an LHCP has judged that the concussion has resolved (Level B).
2. In order to diminish the risk of recurrent injury, individuals supervising athletes should prohibit an athlete with concussion from returning to play/practice (contact-risk activity) until the athlete is asymptomatic off medication (Level B).

*Clinical context: RTP – age effects*

Comparative studies have shown moderate evidence that early postconcussive symptoms and cognitive impairments are longer lasting in younger athletes relative to older athletes. In these studies it is not possible to isolate the effects of age from possible effects of level of play, and there are no comparative studies looking at postconcussive impairments below the high school level. It is accepted that minors in particular should be protected from significant potential risks resulting from elective participation in contact sports. It is also recognized that most ancillary concussion assessment tools (e.g., GSC, SAC, BESS) currently in use either have not been validated or are incompletely validated in children of preteen age or younger.

*Practice recommendations: RTP – age effects*

1. Individuals supervising athletes of high school age or younger with diagnosed concussion should manage them more conservatively regarding RTP than they manage older athletes (Level B).
2. Individuals using concussion assessment tools for the evaluation of athletes of preteen age or younger should ensure that these tools demonstrate appropriate psychometric properties of reliability and validity (Level B).

**Clinical context: RTP – concussion resolution**

There is no single diagnostic test to determine resolution of concussion. Thus, we conclude that concussion resolution is also predominantly a clinical determination made on the basis of a comprehensive neurologic history, neurologic examination, and cognitive assessment. There is moderate evidence that tests such as symptom checklists, neurocognitive testing, and balance testing are helpful in monitoring recovery from concussion.

**Practice recommendation: RTP – concussion resolution**

Clinical LHCPs might use supplemental information, such as neurocognitive testing or other tools, to assist in determining concussion resolution. This may include but is not limited to resolution of symptoms as determined by standardized checklists and return to age-matched normative values or an individual’s preinjury baseline performance on validated neurocognitive testing (Level C).

**Clinical context: RTP – graded physical activity**

Limited data exist to support conclusions regarding implementation of a graded physical activity program designed to assist the athlete to recover from a concussion. Preliminary evidence suggests that a return to moderate activity is possibly associated with better performance on visual memory and reaction time tests, with a trend toward lower symptom scores as compared with scores for no-activity or high-activity groups. Preliminary evidence also exists to suggest that a program of progressive physical activity may possibly be helpful for athletes with prolonged postconcussive symptomatology. There are insufficient data to support specific recommendations for implementing a graded activity program to normalize physical, cognitive, and academic functional impairments. It is accepted that levels of activity that exacerbate underlying symptoms or cognitive impairments should be avoided.

**Practice recommendation: RTP – graded physical activity**
LHCPs might develop individualized graded plans for return to physical and cognitive activity, guided by a carefully monitored, clinically based approach to minimize exacerbation of early postconcussive impairments (Level C).

**Clinical context: Cognitive restructuring**

Patients with mTBI/concussion may underestimate their preinjury symptoms, including many symptoms that are known to occur in individuals without concussion, such as headache, inattention, memory lapses, and fatigue. After injury there is a tendency to ascribe any symptoms to a suspected mTBI/concussion. Patients with chronic postconcussion symptoms utilize more medical resources, namely, repeat physician visits and additional diagnostic tests. Cognitive restructuring is a form of brief psychological counseling that consists of education, reassurance, and reattribution of symptoms and often utilizes both verbal and written information. Whereas there are no specific studies using cognitive restructuring specifically in sports concussions, multiple studies\textsuperscript{e162–e169} using this intervention for mTBI have been conducted and have shown benefit in both adults and children by reducing symptoms and decreasing the proportion of individuals who ultimately develop chronic postconcussion syndrome.

**Practice recommendation: Cognitive restructuring**

LHCPs might provide cognitive restructuring counseling to all athletes with concussion to shorten the duration of subjective symptoms and diminish the likelihood of development of chronic postconcussion syndrome (Level C).

**Clinical context: Retirement from play after multiple concussions – assessment**

In amateur athletes, the relationship between multiple concussions and chronic neurobehavioral impairments is uncertain. In professional athletes, there is strong evidence for a relationship between multiple recurrent concussions and chronic neurobehavioral impairments. A subjective history of persistent neurobehavioral impairments can be measured more objectively with formal neurologic/cognitive assessments that include a neurologic examination and neuropsychological testing.

**Practice recommendation: Retirement from play after multiple concussions – assessment**
1. LHCPs might refer professional athletes with a history of multiple concussions and subjective persistent neurobehavioral impairments for neurologic and neuropsychological assessment (Level C).

2. LCHPs caring for amateur athletes with a history of multiple concussions and subjective persistent neurobehavioral impairments might use formal neurologic/cognitive assessment to help guide retirement-from-play decisions (Level C).

**Clinical context: Retirement from play – counseling**

Other risk factors for persistent or chronic cognitive impairment include longer duration of contact sport participation and preexisting learning disability. In professional athletes, data also support ApoE4 genotype as a risk factor for chronic cognitive impairment. The only modifiable risk factor currently identified is exposure to future concussions or contact sports.

**Practice recommendations: Retirement from play – counseling**

1. LHCPs should counsel athletes with a history of multiple concussions and subjective persistent neurobehavioral impairment about the risk factors for developing permanent or lasting neurobehavioral or cognitive impairments (Level B).

2. LHCPs caring for professional contact sport athletes who show objective evidence for chronic/persistent neurologic/cognitive deficits (such as seen on formal neuropsychological testing) should recommend retirement from the contact sport to minimize risk for and severity of chronic neurobehavioral impairments (Level B).

**RECOMMENDATIONS FOR FUTURE RESEARCH**

1. Additional investigations into factors affecting concussion risk, natural history, and outcome are warranted.

2. Given the number of youth sports participants, extending concussion assessment, natural history, and recovery studies into younger ages (pre–high school) is important, including comparative studies with older age groups. Also needed are development and validation of assessment tools for use in the younger age ranges.
3. Clinical trials of different postconcussion management strategies and RTP protocols are needed to provide a foundation for evidence-based interventions.

4. Research also is needed in the area of gait stability testing with dual tasks or divided-attention task performance for concussion diagnosis, as well as whether these tasks have more utility in determining readiness for RTP in athletes with protracted symptom recovery.

5. Additional studies to determine the efficacy of sideline tools (PCSS, SAC, BESS, King-Devick, clinical reaction time testing) to help diagnose concussion are needed. These studies should include both athletes with SRC and those without SRC; in addition, however, these studies should include, in particular, athletes suspected of having sustained SRC but who ultimately are not diagnosed with SRC, or athletes who are injured but not diagnosed with concussion, or both.

6. The use of neuroimaging, and in particular advanced (microstructural—diffusion tensor imaging or functional—fMRI, MRS) imaging, shows promise in the setting of SRC, but further studies are needed to determine the utility of these modalities in the management of individual SRCs.

7. Management of chronic postconcussion symptoms and impairment likely involves different pathophysiologic and psychological processes that should respond to different interventions. More clinical studies of treatment for chronic postconcussive problems should be conducted.

8. Further definition, investigations, or registries (or a combination of these) of significant postconcussive complications such as second-impact syndrome and CTE are necessary to determine the actual risks and risk factors for these conditions.
Table 1. Concussion incidence in high school and collegiate competitions among commonly played sports

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<tr>
<th>Sport</th>
<th>Rate/1000 games</th>
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<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
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<tr>
<td>Football(^{14})</td>
<td></td>
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<tr>
<td>High school</td>
<td>1.55</td>
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</tr>
<tr>
<td>College</td>
<td>3.02</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>Ice hockey(^{24})</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school</td>
<td>–</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>College</td>
<td>1.96</td>
<td>–</td>
<td></td>
</tr>
<tr>
<td>Soccer(^{14})</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school</td>
<td>0.59</td>
<td>0.97</td>
<td></td>
</tr>
<tr>
<td>College</td>
<td>1.38</td>
<td>1.80</td>
<td></td>
</tr>
<tr>
<td>Basketball(^{14})</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school</td>
<td>0.11</td>
<td>0.60</td>
<td></td>
</tr>
<tr>
<td>College</td>
<td>0.45</td>
<td>0.85</td>
<td></td>
</tr>
<tr>
<td>Baseball/softball(^{14,a})</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school</td>
<td>0.08</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>College</td>
<td>0.23</td>
<td>0.37</td>
<td></td>
</tr>
<tr>
<td>Summary of 9 sports(^{14,b})</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High school</td>
<td>0.61</td>
<td>0.42</td>
<td></td>
</tr>
<tr>
<td>College</td>
<td>1.26</td>
<td>0.74</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) Assumes that competitive high school and collegiate baseball players were mainly male and softball players were mainly female.

\(^b\) Sports include football, boys’ and girls’ soccer, volleyball, boys’ and girls’ basketball, wrestling, baseball, and softball.
Appendix 1: Definition of terms

The use of rigid definitions in the area of sports concussion poses an existential challenge to any attempt to quantify the evidence for their management. Whereas this author panel concurred that the distinctions between concussion and mild traumatic brain injury (mTBI) are not well established, there was also a practical consideration that the vast majority of cases of sports-related concussion (SRC) and sport-related mTBI describe similar entities. Thus for this guideline, we included studies that described either SRCs or sports-related mTBI. In general, SRC/mTBI required a clinician (physician, certified athletic trainer) diagnosis, if one follows definitions set forth by the American Academy of Neurology (1997), Colorado Medical Society, the Cantu scale, or the American Congress of Rehabilitation Medicine (1993). It is also recognized that the setting (date, location) of a given study might affect specifics of the definition—for example, older studies are more likely to require loss of consciousness (LOC) for a concussion diagnosis, or studies based in the emergency room are likely to include individuals with greater symptoms or impairments than sideline-based studies. In citing the literature, these distinctions are considered and listed in the text and summary evidence tables.

**Concussion** – pathophysiologic disturbance in neurologic function characterized by clinical symptoms induced by biomechanical forces, occurring with or without LOC. Standard structural neuroimaging is normal, and symptoms typically resolve over time.

**Mild traumatic brain injury** – historically, this has referred to biomechanically induced brain injury with a Glasgow Coma Score of 13–15. Concussions may be included in this categorization.

**Subconcussive injury** – a theoretical, very mild, biomechanically induced brain injury that may occur in the absence of overt clinical symptoms of concussion. Recent concern has been raised regarding the existence of this entity on the basis of two predominant lines of evidence: very sensitive neuroimaging and electrophysiologic measures showing group differences between individuals exposed to contact sports as compared with non–contact sport controls, and an apparent dose response between contact sport exposure and chronic cumulative neurocognitive impairments.
**Early postconcussion impairment** – subjective symptoms or objective neurocognitive deficits induced by a concussion during the acute/subacute phase (days–weeks).

**Late postconcussion neurobehavioral impairment** – subjective symptoms or objective neurocognitive or behavioral abnormalities seen chronically (months–years) following (a) concussion(s).

**Postconcussion neurologic catastrophe** – any more-severe form of traumatic brain injury or other traumatically induced intracranial complication after a concussion requiring urgent/emergent intervention (e.g., epidural hematoma, cerebral edema).

**Simple concussion** – proposed by the Concussion in Sport Group (CISG) in 2005\(^{e173}\) as a designation for concussions wherein symptoms resolve over 7–10 days without complication. Subsequently abandoned in the 2009 CISG consensus document.\(^e7\)

**Complex concussion** – proposed by the CISG in 2005\(^{e173}\) as a designation for concussions wherein symptoms or cognitive impairments are persistent, and for cases associated with convulsions/seizure, LOC >1 minute, or history of multiple concussions. Also withdrawn in the 2009 CISG updated consensus document.\(^e7\)
Appendix 2: 2011–2013 Guideline Development Subcommittee (GDS) members
John D. England, MD, FAAN (Chair); Cynthia Harden, MD (Vice-Chair); Melissa Armstrong, MD; Eric Ashman, MD; Misha-Miroslav Backonja, MD; Richard L. Barbano, MD, PhD, FAAN; Diane Donley, MD; Terry Fife, MD, FAAN; David Gloss, MD; John J. Halperin, MD, FAAN; Cheryl Jaigobin, MD; Andres M. Kanner, MD; Jason Lazarou, MD; Steven R. Messé, MD, FAAN; David Michelson, MD; Pushpa Narayanaswami, MD, MBBS; Anne Louise Oaklander, MD, PhD, FAAN; Tamara Pringsheim, MD; Alexander Rae-Grant, MD; Michael Shevell, MD, FAAN; Theresa A. Zesiewicz, MD, FAAN; Jonathan P. Hosey, MD, FAAN (Ex-Officio); Stephen Ashwal, MD, FAAN (Ex-Officio); Deborah Hirtz, MD, FAAN (Ex-Officio)
Appendix 3: Mission Statement of GDS

The mission of the GDS is to prioritize, develop, and publish evidence-based guidelines related to the diagnosis, treatment, and prognosis of neurological disorders.

The GDS is committed to using the most rigorous methods available within our budget, in collaboration with other available AAN resources, to most efficiently accomplish this mission.
Appendix 4: Search strategy

See PDF labeled “Sports concussion appendix 4 search strategy” at the Neurology® website at www.neurology.org.
Appendix 5: AAN rules for classification of evidence for risk of bias

For questions related to diagnostic accuracy

Class I

- Study is a cohort survey with prospective data collection.
- Study includes a broad spectrum of persons suspected of having the disease.
- Disease status determination is objective or made without knowledge of diagnostic test result.
- The following also are required:
  a. Inclusion criteria are defined.
  b. At least 80% of enrolled subjects have both the diagnostic test and disease status measured.

Class II

- Study is a cohort study with retrospective data collection or is a case-control study. Study meets criteria a–b.
- Study includes a broad spectrum of persons with the disease and persons without the disease.
- The diagnostic test result and disease status are determined objectively or without knowledge of one or the other.

Class III

- Study is a cohort or case-control study.
- Study includes a narrow spectrum of persons with or without the disease.
- The diagnostic test result and disease status are determined objectively, without knowledge of one or the other, or by different investigators.

Class IV

- The study does not include persons suspected of the disease.
- The study does not include patients with the disease and patients without the disease.
- The study uses an undefined or unaccepted independent reference standard.
- No measures of diagnostic accuracy or statistical precision are presented or calculable.
For questions related to prognostic accuracy

Class I
- The study is a cohort survey with prospective data collection.
- The study includes a broad spectrum of persons at risk for developing the outcome.
- Outcome measurement is objective or determined without knowledge of risk factor status.
- The following also are required:
  a. Inclusion criteria are defined.
  b. At least 80% of enrolled subjects have both the risk factor and outcome measured.

Class II
- The study is a cohort study with retrospective data collection or is a case control study. Study meets criteria a–b.
- The study includes a broad spectrum of persons with the risk factor and outcome and persons without the risk factor and outcome.
- The presence of the risk factor and outcome are determined objectively or without knowledge of one or the other of these variables.

Class III
- The study is a cohort or case-control study.
- The study includes a narrow spectrum of persons with or without the disease.
- The presence of the risk factor and outcome are determined objectively, without knowledge of the one or the other, or by different investigators.

Class IV
- The study does not include persons at risk for the outcome.
- The study does not include patients with the risk factor and patients without the risk factor.
- The study uses undefined or unaccepted measures of risk factor or outcomes.
- No measures of association or statistical precision are presented or calculable.
**For questions related to therapeutic intervention**

**Class I**
- The study is a randomized clinical trial.
- All relevant baseline characteristics are presented and substantially equivalent between treatment groups or there is appropriate statistical adjustment for differences.
- Outcome measurement is objective or determined without knowledge of treatment status.
- The following also are required:
  a. The primary outcome(s) is/are defined.
  b. The inclusion criteria are defined.
  c. There is accounting of dropouts and crossovers (with at least 80% of enrolled subjects completing the study).
  d. There is concealed allocation.

**Class II**
- The study is a cohort study meeting criteria a–c above or is a randomized, controlled trial that lacks one or two criteria a–d.
- All relevant baseline characteristics are presented and substantially equivalent among treatment groups, or there is appropriate statistical adjustment for differences.
- There is masked or objective outcome assessment.

**Class III**
- The study is a controlled study (including well-defined natural history controls or patients serving as their own controls).
- The study includes a description of major confounding differences between treatment groups that could affect outcome.
- Outcome assessment is masked, objective, or performed by someone who is not a member of the treatment team.

**Class IV**
- The study does not include patients with the disease.
- The study does not include patients receiving different interventions.
- The study uses undefined or unaccepted interventions or outcome measures.
- No measures of effectiveness or statistical precision are presented or calculable.
Appendix 6: Summary evidence tables

See Word document labeled “Sports concussion appendix 6 sum evid tables” at the Neurology® website at www.neurology.org.
Appendix 7: Rules for determining confidence in evidence

- Modal modifiers used to indicate the final confidence in evidence in the conclusions
  - High confidence: highly likely or highly probable
  - Moderate confidence: likely or probable
  - Low confidence: possibly
  - Very low confidence: insufficient evidence

- Initial rating of confidence in the evidence for each intervention outcome pair
  - High: requires two or more Class I studies
  - Moderate: requires one Class I study or two or more Class II studies
  - Low: requires one Class II study or two or more Class III studies
  - Very low: requires only one Class III study or one or more Class IV studies

- Factors that could result in downgrading confidence by one or more levels
  - Consistency
  - Precision
  - Directness
  - Publication bias
  - Biologic plausibility

- Factors that could result in downgrading confidence by one or more levels or upgrading confidence by one level
  - Magnitude of effect
  - Dose response relationship
  - Direction of bias
**Summary evidence table template**

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Outcome(s)</th>
<th>Number &amp; Class of Studies</th>
<th>Effect</th>
<th>Precision</th>
<th>Consistent</th>
<th>Directness</th>
<th>Plausible</th>
<th>Reporting Bias</th>
<th>Magnitude of Effect</th>
<th>Dose Response</th>
<th>Direction of Bias</th>
<th>Comment</th>
<th>Confidence in Evidence</th>
</tr>
</thead>
</table>
Appendix 8: Steps and rules for formulating recommendations

Constructing the recommendation and its rationale

Rationale for recommendation summarized in the Clinical Context includes three categories of premises

- Evidence-based conclusions for the systematic review
- Stipulated axiomatic principles of care
- Strong evidence from related conditions not systematically reviewed

Actionable recommendations include the following mandatory elements

- The patient population that is the subject of the recommendation
- The person performing the action of the recommendation statement
- The specific action to be performed
- The expected outcome to be attained

Assigning a clinician level of obligation

Modal modifiers used to indicate the final clinician level of obligation (CLO)

- Level A: “Must”
- Level B: “Should”
- Level C: “Might”
- Level U: No recommendation supported

CLO assigned by eliciting panel members' judgments regarding multiple domains, using a modified Delphi process. Goal is to attain consensus after a maximum of three rounds of voting. Consensus is defined by:

- ≥80% agreement on dichotomous judgments
- ≥80% agreement, within one point for ordinal judgments
- If consensus obtained, CLO assigned at the median. If not obtained, CLO assigned at the 10th percentile

Three steps used to assign final CLO

1. Initial CLO determined by the cogency of the deductive inference supporting the recommendation on the basis of ratings within four domains. Initial CLO anchored to lowest CLO supported by any domain.
• Confidence in evidence. CLO anchored to confidence in evidence determined by modified form of the Grading of Recommendations Assessment, Development and Evaluation process\(^{13}\)
  • Level A: High confidence
  • Level B: Moderate confidence
  • Level C: Low confidence
  • Level U: Very low confidence

• Soundness of inference assuming all premises are true. CLO anchored to proportion of panel members convinced of soundness of the inference
  • Level A: 100%
  • Level B: ≥80% to <100%
  • Level C: ≥50% to <80%
  • Level U or R: <50%

• Acceptance of axiomatic principles: CLO anchored to proportion of panel members who accept principles
  • Level A: 100%
  • Level B: ≥80% to <100%
  • Level C: ≥50% to <80%
  • Level U or R: <50%

• Belief that evidence cited from rerated conditions is strong: CLO anchored to proportion of panel members who believe the related evidence is strong
  • Level B: ≥80% to 100% (recommendations dependent on inferences from nonsystematically reviewed evidence cannot be anchored to a Level A CLO)
  • Level C: ≥50% to <80%
  • Level U or R: <50%

2. CLO is modified mandatorily on the basis of the judged magnitude of benefit relative to harm expected to be derived from complying with the recommendation
  • Magnitude relative to harm rated on 4-point ordinal scale
• Large benefit relative to harm: benefit judged large, harm judged none
• Moderate benefit relative to harm: benefit judged large, harm judged minimal; or benefit judged moderate, harm judged none
• Small benefit relative to harm: benefit judged large, harm judged moderate; or benefit judged moderate, harm judged minimal; or benefit judged small, harm judged none
• Benefit to harm judged too close to call: benefit and harm judged to be the same

  ▪ Regardless of cogency of the recommendation the CLO can be no higher than that supported by the rating of the magnitude of benefit relative to harm
    • Level A: Large benefit relative to harm
    • Level B: Moderate benefit relative to harm
    • Level C: Small benefit relative to harm
    • Level U: Too close to call

  ▪ CLO can be increased by one grade if CLO corresponding to benefit relative to harm greater than CLO corresponding to the cogency of the recommendation

3. CLO optionally downgraded on the basis of the following domains
  ▪ Importance of the outcome: critical, important, mildly important, not important
  ▪ Expected variation in patient preferences: none, minimal, moderate, large
  ▪ Financial burden relative to benefit expected: none, minimal, moderate, large
  ▪ Availability of intervention: universal, usually, sometimes, limited

The Clinical Contextual Profile shown below summarizes the results of panel ratings for each domain described above. The profile also indicates the corresponding assigned CLO. The last column indicates whether consensus was obtained for that domain.
Appendix 9: Clinical contextual profiles for recommendations

For an explanation of domains and rules for assigning CLOs to recommendations please refer to appendix 7. The clinical contextual profile corresponding to a recommendation or a set of recommendations follows the recommendation(s).

**Recommendations for preparticipation counseling**

1. Sideline LHCPs and school-based professionals should be educated by experienced individuals designated by their organization/institution to understand the risks of experiencing a concussion so that they may provide accurate information to parents and athletes (Level B).

2. To foster informed decision making, LHCPs should inform athletes (and where appropriate, the athletes’ families) of evidence concerning the concussion risk factors. Accurate information regarding concussion risks also should be disseminated to school systems and sports authorities (Level B).

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**Recommendations for suspected concussion**

**Use of checklists and screening tools for suspected concussion**

1. LHCPs should be instructed in the proper administration of standardized, validated sideline assessment tools. This instruction should emphasize that these tools are only an adjunct to the evaluation of the athlete with suspected concussion and cannot be used alone to diagnose concussion (Level B). These providers should be instructed by experienced individuals who themselves are licensed, knowledgeable about sports
conclusion, and practicing within the scope of their training and experience, designated by their organization/institution in proper administration of standardized validated sideline assessment tools (Level B).

2. In individuals with suspected concussion, these tools should be utilized by sideline LHCPs and the results made available to clinical LHCPs who will be evaluating the injured athlete (Level B).

3. LHCPs caring for athletes might utilize individual baseline scores on concussion assessment tools, especially in younger athletes, those with prior concussions, or those with preexisting learning disabilities/ADHD, as doing so fosters better interpretation of postinjury scores (Level C).

*Panel members chose not to downgrade CLO from Level B to Level C based on variation in preferences, financial burden, or availability*
4. Team personnel (e.g., coaching, athletic training staff, sideline LHCPs) should immediately remove from play any athlete suspected of having sustained a concussion, in order to minimize the risk of further injury (Level B).

5. Team personnel should not permit the athlete to return to play until the athlete has been assessed by an experienced clinical LHCP with training both in the diagnosis and management of concussion and in the recognition of more-severe TBI (Level B).

*NPanel members chose not to downgrade CLO from Level B to Level C on the basis of variation in preferences or availability

**Neuroimaging for suspected concussion**

CT imaging should not be used to diagnose SRC but might be obtained to rule out more serious TBI such as an intracranial hemorrhage in athletes with a suspected concussion who have LOC, posttraumatic amnesia, persistently altered mental status (Glasgow Coma Scale <15), focal neurologic deficit, evidence of skull fracture on examination, or signs of clinical deterioration (Level C).
**Recommendations for diagnosed concussion**

**RTP—risk of recurrent concussion**

1. In order to diminish the risk of recurrent injury, individuals supervising athletes should prohibit an athlete with concussion from returning to play/practice (contact-risk activity) until an LHCP has judged that the concussion has resolved (Level B).

<table>
<thead>
<tr>
<th>Level of obligation</th>
<th>C</th>
<th>May</th>
<th>Consensus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Availability</td>
<td>Limited 0</td>
<td>Sometimes 0</td>
<td>Usually 5</td>
</tr>
<tr>
<td>Financial burden</td>
<td>Prohibitive 0</td>
<td>Moderate 0</td>
<td>Minimal 5</td>
</tr>
<tr>
<td>Variation in preferences</td>
<td>Large 0</td>
<td>Moderate 6</td>
<td>Small 4</td>
</tr>
<tr>
<td>Importance of outcomes</td>
<td>Not important</td>
<td>Mildly Important</td>
<td>Important</td>
</tr>
<tr>
<td>Benefit relative to Harm</td>
<td>Too close to call</td>
<td>Small</td>
<td>Moderate</td>
</tr>
<tr>
<td>Magnitude of Harm</td>
<td>Large 0</td>
<td>Moderate 0</td>
<td>Minimal 7</td>
</tr>
<tr>
<td>Magnitude of Benefit</td>
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<td>Minimal 0</td>
<td>Moderate 4</td>
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<td>Weakly Cogent</td>
<td>Moderately Cogent</td>
</tr>
<tr>
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<td>&gt;50% to &lt; 80%</td>
<td>≥80% to 100%</td>
</tr>
<tr>
<td>Confidence in evidence</td>
<td>Very low 0</td>
<td>Low 1</td>
<td>Moderate 7</td>
</tr>
<tr>
<td>Acceptance of Principles</td>
<td>&lt;50%</td>
<td>&gt;50% to &lt; 80%</td>
<td>&gt;80% to &lt; 100%</td>
</tr>
<tr>
<td>Sound inference</td>
<td>&lt;50%</td>
<td>&gt;50% to &lt; 80%</td>
<td>≥ 80% to &lt; 100%</td>
</tr>
</tbody>
</table>

*Panel members chose not to downgrade CLO from Level B to Level C on the basis of variation in preferences or availability*

2. In order to diminish the risk of recurrent injury, individuals supervising athletes should prohibit an athlete with concussion from returning to play/practice (contact-risk activity) until the athlete is asymptomatic off medication (Level B).

**RTP – age effects**
1. Individuals supervising athletes of high school age or younger with diagnosed concussion should manage them more conservatively regarding RTP than they manage older athletes (Level B).

2. Individuals using concussion assessment tools for the evaluation of athletes of preteen age or younger should ensure that these tools demonstrate appropriate psychometric properties of reliability and validity (Level B).

RTP – concussion resolution

Clinical LHCPs might use supplemental information, such as neurocognitive testing or other tools, to assist in determining concussion resolution. This may include but is not limited to resolution of symptoms as determined by standardized checklists and return to age-matched normative values or an individual’s preinjury baseline performance on validated neurocognitive testing (Level C).
RTP – graded physical activity

LHCPs might develop individualized graded plans for return to physical and cognitive activity, guided by a carefully monitored, clinically based approach to minimize exacerbation of early postconcussive impairments (Level C).

<table>
<thead>
<tr>
<th>Level of obligation</th>
<th>C May</th>
<th>Consensus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Availability</td>
<td>Limited 0</td>
<td>Sometimes 0</td>
</tr>
<tr>
<td>Financial burden</td>
<td>Prohibitive 0</td>
<td>Moderate 2</td>
</tr>
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<td>Variation in preferences</td>
<td>Large 0</td>
<td>Moderate 10</td>
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<tr>
<td>Importance of outcomes</td>
<td>Not important</td>
<td>Mildly Important</td>
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<tr>
<td>Benefit relative to Harm</td>
<td>Too close to call</td>
<td>Small</td>
</tr>
<tr>
<td>Magnitude of Harm</td>
<td>Large 0</td>
<td>Moderate 0</td>
</tr>
<tr>
<td>Magnitude of Benefit</td>
<td>None 0</td>
<td>Minimal 1</td>
</tr>
</tbody>
</table>

Cogency of recommendation

| Strong evidence other cond | <50% | >50% to < 80% | >80% to 100% | X | Yes |
| Confidence in evidence    | Very low | Low 6 | Moderate 3 | High 2 | No |
| Acceptance of Principles  | <50% | >50% to < 80% | >80% to < 100% | 100% | Yes |
| Sound inference           | <50% | >50% to < 80% | ≥ 80% to < 100% | 100% | Yes |

Cogency of recommendation

| Strong evidence other cond | <50% | >50% to < 80% | >80% to 100% | X | Yes |
| Confidence in evidence    | Very low | Low 3 | Moderate 4 | High 1 | No |
| Acceptance of Principles  | <50% | >50% to < 80% | >80% to < 100% | 100% | Yes |
| Sound inference           | <50% | >50% to < 80% | ≥ 80% to < 100% | 100% | No |

Cognitive restructuring

LHCPs might provide cognitive restructuring counseling to all athletes with concussion to shorten the duration of subjective symptoms and diminish the likelihood of development of chronic postconcussion syndrome (Level C).

Retirement from play after multiple concussions – assessment

1. LHCPs might refer professional athletes with a history of multiple concussions and subjective persistent neurobehavioral impairments for neurologic and neuropsychological assessment (Level C).
2. LHCPs caring for amateur athletes with a history of multiple concussions and subjective persistent neurobehavioral impairments might use formal neurologic/cognitive assessment to help guide retirement-from-play decisions (Level C).

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**Retirement from play – counseling**

1. LHCPs should counsel athletes with a history of multiple concussions and subjective persistent neurobehavioral impairment about the risk factors for developing permanent or lasting neurobehavioral or cognitive impairments (Level B).

2. LHCPs caring for professional contact sport athletes who show objective evidence for chronic/persistent neurologic/cognitive deficits (such as seen on formal neuropsychological testing) should recommend retirement from the contact sport to minimize risk for and severity of chronic neurobehavioral impairments (Level B).
Panel members chose not to downgrade CLO from Level B to Level C on the basis of variation in preferences or financial burden.
REFERENCES

e6. Hearing Before the Committee on Commerce, Science, and Transportation, 112th Con, 1st Sess (2011) (statement of Jeffrey S. Kutcher, MD, associate professor, University of Michigan, Department of Neurology; Director, Michigan NeuroSport; Chair, Sports Neurology Section, American Academy of Neurology).
e124. Shuttleworth-Edwards AB, Radloff SE. Compromised visuomotor processing speed in players of Rugby Union from school through to the national adult level. Archives of Clinical Neuropsychology 2008;23:511-520.
