

If You're Not Measuring, You're Guessing: The Advent of Objective Concussion Assessments

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Sport-related concussion remains one of the most complex injuries presented to sports medicine professionals. Although the injury has been recognized since ancient times, the concussion-assessment process has seen significant advances over the last 30 years. This review outlines the addition of objective measures to the clinical evaluation of the concussed athlete, beginning in the 1980s and continuing through the modern age. International and domestic organizations now describe standardized symptom reports, neurostatus and

neurocognitive-function evaluations, and postural-control measures as standards of medical care, a significant shift from a short time ago. Despite this progression, much about the injury remains unknown, including new clinical and research-based assessment techniques and how the injury may influence the athlete's cognitive health over the long term.

Key Words: evidence-based medicine, traumatic brain injury, evaluation

Concussions, the mildest form of traumatic brain injury, have been cited as a “silent epidemic” by the Centers for Disease Control and Prevention.¹ Awareness of concussive injuries is not new and was noted in the medical literature as far back as the 5th century BCE. The *Hippocratic Corpus* noted that “commotion of the brain” resulted in loss of speech, hearing, and sight.² Injury evaluation and management were not addressed in that early document, and little changed in the understanding of the injury through the Roman era.² Indeed, injury knowledge did not evolve until medieval times, when the physician Lanfrancus established that concussive symptoms were the result of a temporary loss of cerebral function that resolved quickly. These symptoms were contrasted against more serious injuries that resulted in physical damage to the brain tissue.² Further support for this concept came with the advent of the microscope in the 17th century, when the idea that concussion represented a clinical syndrome after insult was first presented.² Four hundred years later, the most sophisticated medical microscopes—magnetic resonance imaging and computed tomography imaging—have not been able to demonstrate a structural change to the cerebral tissue after concussion. Medical research has, however, advanced in other areas and provided clinicians with measures sensitive to postinjury clinical changes.

Various hypotheses on the mechanisms and underpinnings that result in the clinical signs and symptoms of concussion have been proposed over the centuries, but our current understanding is generally consistent with the original theory.² That is, the signs and symptoms of concussion are the clinical manifestations resulting from a change in the functional capacity of the cerebral tissue, and injury resolution represents the natural return to homeostasis of the impaired neurons.³ Recognizing that direct or

indirect forces to the brain may result in concussions,⁴ medical professionals are charged with rapidly identifying the injured athlete to reduce the risk for further injury. Before the 21st century, concussions were largely ignored and were described in terms that diminished the injury severity (eg, “clearing the cobwebs” or “getting your bell rung”). If an injury was suspected, the evaluation process was largely subjective and involved asking the athlete simple questions, such as “What is your name?”, “Are you OK?”, or “How many fingers am I holding up?”

Over the last 20 years, our understanding of concussion mechanics, including cellular-level pathogenesis, and injury assessment and management have increased dramatically. Concussions during sport and recreation are now thought to occur as often as 3.8 million times per year,⁵ resulting in up to 7 injuries per minute every day of the year in the United States. Although each patient requires individual management, 90% of concussed athletes recover by day 7 after injury⁶ with little to no intervention; evidence⁷ suggests that injured athletes who continue playing and sustain additional head impacts experience prolonged recoveries. To reduce this risk, most sports medicine organizations^{4,8,9} now endorse the use of a multifaceted examination to support the clinical evaluation with an assessment battery that includes measures of athlete-reported symptoms, neurocognitive status, and balance. This information, which provides important objective data to the clinician^{10,11} when making decisions, was first used in 1982, when Barth et al^{12,13} launched the seminal sport concussion work at the University of Virginia: the Sports as a Laboratory Assessment Model (SLAM). The intent of our literature review, therefore, is to summarize the evolution of the modern objective concus-

sion-assessment protocol relative to certified athletic trainers (ATs) and other sports medicine professionals.

THE SYMPTOM EVALUATION

The signs and symptoms of concussion were the first clinical aspects to be documented in uncovering the natural history of the injury. The authors of the SLAM study^{12,14} described increases in headache, memory problems, and dizziness after injury, with resolution over the next 10 days. Since that time, ATs have used symptom checklists as the primary evaluation tool for assessing the injury¹⁵; several symptom checklists have been developed and placed into clinical practice, with each intended for grading the presence and severity of signs or symptoms. The Post-Concussion Symptom Checklist was introduced in 1998 and proposed as a standardized assessment tool for the evaluation of concussion-related postinjury symptoms.¹⁶ This checklist included 16 items similar to those used by Barth et al.¹² The International Conference on Concussion in Sport (ICCS) group then suggested a standardized concussion-symptom scale that contained 19 items.¹⁷ A similar scale, the Graded Symptom Checklist, with 27 items, was endorsed by the National Athletic Trainers' Association (NATA) shortly thereafter.¹⁸ Modifications to the symptom list were made by the ICCS group in 2004,¹⁹ 2008,²⁰ and 2012⁴; the most current version contains 22 items that are graded on a scale from zero (*none*) to 6 (*severe*).

Subtle differences exist among these scales, but the symptoms of headache, dizziness, difficulty concentrating, nausea, fatigue, trouble falling asleep, drowsiness, feeling slowed down, and feeling in a fog are common to all. In large studies²¹⁻²³ of postconcussion symptoms, 83% of concussed athletes reported headache, followed by dizziness (65%) and confusion (57%). These symptoms, however, are often nonspecific and are commonly reported among nonconcussed athletes.²⁴ Some researchers²⁵⁻²⁷ have therefore refined the symptom list to items that are most relevant to concussion while maintaining comparable psychometric properties. Pivotal to the advancement of concussion clinical care was the recognition that symptoms may be slow to develop in some concussed athletes.²⁸ Clinical outcomes are not associated with loss of consciousness,²⁹ which occurs in less than 10% of all patients.²¹

Evaluating and quantifying concussive signs and symptoms offer many advantages and have been shown to identify approximately 90% of concussed athletes.¹¹ In spite of the broad implementation of these processes in clinical care, athletes may be motivated to alter symptom reports at baseline or postinjury for a number of reasons. For example, anecdotal accounts suggest that some athletes may inflate baseline symptom reports to mask elevated postinjury scores. Others may not recognize concussive signs and symptoms or intentionally not report them once the injury occurs³⁰⁻³³ because of influence from teammates, friends, coaches, or parents. Furthermore, some athletes may be motivated to inflate symptom reports or continue reporting symptoms despite injury resolution as a means to leave a sport.

For these reasons and also because of the natural day-to-day presence of symptoms commonly associated with

concussion,^{34,35} some degree of symptom reporting is not unexpected. Clinicians, therefore, face the difficult task of interpreting which symptoms are directly related to concussion and which are related to other conditions. For example, a headache in an athlete who has sustained a blow to the head may be the result of a concussive injury but may also originate from a neck injury,³⁶ exercise, a migraine headache, or simply wearing a helmet.³⁷ These possibilities are supported by the findings of a large investigation³⁸ that showed some evidence of concussionlike symptoms in nonconcussed control populations. Others have demonstrated that returning symptomatic athletes who are far beyond the acute injury stage to exercise can reduce symptoms,^{39,40} suggesting that long-term symptoms may not be directly related to the concussion.⁴¹

Although symptom scales and checklists provide some objectivity to the examination, they rely heavily on athlete honesty and can be influenced by factors unrelated to concussion. As such, symptom scales alone cannot accurately identify all concussed athletes,¹⁰ nor can they accurately identify injury recovery.^{42,43} This gap thus requires more objective measures to complement and extend the evaluative process.

THE NEUROCOGNITIVE ASSESSMENT

Neurocognitive testing developed as a way to evaluate different domains of cognitive functioning (eg, memory, concentration, or attention). Pencil-and-paper tests were implemented largely to detect and evaluate gross cognitive functioning, and the SLAM study¹⁴ was the first to apply the tests to an athletic population. In that investigation, 2350 athletes from 10 universities completed the Paced Auditory Serial Addition Test, the Digit Span Test, the Trail Making Test, and a symptom evaluation. Each of the 195 athletes with a diagnosed concussion underwent postinjury evaluations at fixed time points (24 hours and days 5, 10, and 12); declines in cognitive functioning and increased symptom reports were present immediately postinjury and resolved over the next 10 days.¹⁴ This study was the first to implement a baseline-postinjury assessment in a sporting environment, which stands as the de facto assessment model today. The only change has been a move away from fixed assessment days to clinically derived assessment points, including when the athlete is asymptomatic and has been cleared to return to play after performing a graded exercise protocol.⁸

Although remarkably innovative, the SLAM model was also highly labor intensive, as the pencil-and-paper neurocognitive assessment battery required substantial time to administer on a one-on-one basis. Indeed, shortly after the initial study findings were published, a similar model was implemented by the Pittsburgh Steelers Organization (with other professional teams following) but was met with resistance, in part because of the testing duration. In response, Mark Lovell, PhD, and his colleagues began developing computer-based assessments that could be administered in a manner that was rapid, systematic, and repeatable.

Computer-based testing offered the promise of more precise reaction-time measures, decreased practice effects and administration time, and ease of administration. Reaction times are recorded to the thousandth of a second,

and computer programming automates the implementation of alternate forms. Internal validity checks identify athletes who may have intentionally performed poorly at baseline,⁴⁴ although 10% may evade detection.⁴⁵ Perhaps the key features that resulted in the rapid adoption of computer-based testing were the time and ease of administration. Indeed, a complete pencil-and-paper neurocognitive evaluation of an athlete may have taken up to 4 hours, although modified one-on-one batteries typically lasted 45 minutes. Conversely, the time to complete computer-based tests can be 20 to 30 minutes, and multiple athletes can be tested concurrently under appropriate conditions.⁴⁶ Injury sensitivity improved from 23% to 44%^{10,11} on pencil-and-paper tests to 63% to 82%^{10,47} on the cognitive components of computer-based tests, but concerns about reliability and false-positive findings hampered recommendations for their isolated use.⁴⁸⁻⁵¹

Ultimately, given the time pressures of sports medicine, practitioners were quick to adopt computer-based testing. Indeed, in 2001, only 15% of ATs in all settings were using any form of neurocognitive testing in the management of patients with concussive injuries.⁵² The rates of pencil-and-paper versus computer-based assessment were not provided, but the latter was just becoming available in the marketplace at that time, suggesting that the former was still the instrument of choice. Four years later, approximately 5% of surveyed ATs were using pencil-and-paper tests, and an additional 15% reported using computer-based assessments.⁵³ Computer-based testing rose again to 33% in 2009.¹⁵ The current standard for the clinical management of concussions by ATs is to include a neurocognitive evaluation.⁸

Neurocognitive assessments have vastly improved the concussion-assessment process, but their use is limited in the athletic environment (eg, sideline). In recognition of this limitation, the Standardized Assessment of Concussion (SAC) was developed in 1997 as a neurostatus screening tool for use in the field immediately postinjury.⁵⁴ The SAC outlined a standard clinical evaluation that included both cognitive and physical assessments, and since its inception, it has been widely disseminated and adopted for both clinical^{4,8} and research³⁸ use. Although the test has a high level of sensitivity when administered immediately after injury,¹¹ its use beyond the 48-hour mark is less impressive. Despite this, the SAC fills a large gap in the concussion-management process left by neurocognitive testing and provides the clinician with additional objective information when administered immediately postinjury.

THE BALANCE ASSESSMENT

The addition of cognitive functioning to the clinical assessment battery in the 1980s represented a movement away from relying solely on subjective measures (ie, symptoms) to the inclusion of objective measures that supported the clinical examination. This movement continued with the addition of balance assessment in the next decade. The earliest balance assessment used to evaluate the functional abilities of the brain after injury was the Romberg test.⁵⁵ With the patient's feet together and eyes closed, the examiner subjectively evaluates irregular sway patterns. However, the test results are highly subjective; the

examiner notes only if the sway pattern is normal or not based on his or her clinical experience.

By implementing a modified version of the Romberg test,⁵⁶ Ingersoll and Armstrong⁵⁷ were the first to identify the importance of quantifying balance in relation to the concussion-assessment protocol. They used force-plate technology to track center-of-pressure movement and objectively demonstrated increased anterior-posterior sway during postinjury assessments of the concussed participants. Including multiple stable and unstable support surfaces in a follow-up investigation demonstrated impaired balance up to 3 days postinjury.⁵⁸ These findings were replicated using advanced postural-control measures,⁵⁹⁻⁶¹ and the authors⁵⁹ concluded that the Romberg test was insensitive to the balance deficits brought about by concussive injuries.

Although these fundamental investigations forever changed the concussion-assessment process, force-plate technology was limited to research and hospital settings that could afford this sophisticated equipment. The Balance Error Scoring System (BESS) was therefore designed as a low-cost objective measure of balance. The test implemented components of the Shumway-Cook balance test⁵⁶ (ie, firm and foam surfaces), modified the stances, and added a procedure for objectively quantifying errors.⁶²⁻⁶⁴ The BESS error scores tracked closely with increased center-of-pressure sway.⁶⁵ As a result, the test was soon included in large-scale clinical investigations of concussion^{38,61} and became a standard of care for ATs.^{8,18} The addition of an objective postural-control measure to the concussion-assessment paradigm significantly strengthened the evaluative process, with immediate postinjury (ie, sideline) sensitivity increasing to 94% when combined with a brief neurocognitive examination and symptom report.¹¹ Similar to the SAC and all concussion assessments, however, the sensitivity of the BESS declines rapidly in the days postinjury¹¹ as recovery is typically swift and spontaneous.⁶⁶

POSITION AND CONSENSUS STATEMENTS

The development of objective concussion-assessment tools represents the integration of research into clinical practice, with science verifying what can be applied in a clinical setting. Ultimately, individual research projects help to identify and validate assessment tools, but position and consensus statements make these tools the standard for clinical care. Several organizations have disseminated statements over the years; none have been as influential as those coming from the ICCS and the NATA.

In 2001, the first ICCS meeting was held in Vienna, Austria. The document that emerged from that meeting had a broad influence on concussion management, starting with a new injury definition.¹⁷ The "complex pathophysiological process brought about by biomechanical forces" reiterated the well-established concept that the injury results from a direct or indirect force to the head, the clinical signs and symptoms represent a functional (not structural) change to the cerebral tissue as demonstrated by normal imaging, and the clinical impairments are typically short lived. New at the time of publication, however, was a statement that loss of consciousness was not an injury requirement and that grading scales should be abandoned in guiding the return-

to-play process. These changes ushered in the era of individualized return-to-play management, with neurocognitive testing (by computer or pencil and paper) emphasized as the cornerstone technique for tracking recovery. Balance testing was not included in the document, but the seminal papers^{59,61} in this area had just been released. This was also the first time a graduated return-to-play progression was outlined, although the timing between steps and cognitive rest was not addressed.

In 2004, the NATA published its first position statement on the management of sport-related concussion.¹⁸ This document called for the abandonment of colloquial terms (eg, “ding,” “bell rung”) and for baseline and postinjury assessments of high-risk athletes that included measures of symptoms, cognitive functioning, and postural control (ie, BESS). Our understanding of the injury has since changed: at the time, it was considered safe for concussed athletes to return to play on the same day as injury if symptoms resolved within 20 minutes and there was no loss of consciousness or amnesia. Similar to the International Sport Concussion Group, a progressive exercise protocol was outlined for when the athlete became asymptomatic, but no timeline was provided.

In that same year, ICCS met in Prague, Czech Republic, to update its original statement. The revised document¹⁹ reversed the earlier version in presenting the concussion grades of simple and complex. *Simple concussions* adhered to a typical recovery curve (7–10 days) and required no intervention or neurocognitive testing. *Complex injuries* lasted longer than 10 days, presented with loss of consciousness lasting more than 1 minute, or resulted in prolonged impairment. The guidelines prescribed that complex injuries be directly managed using neurocognitive testing but did not indicate how the clinician was to differentiate between simple and complex at the time of injury. Neurocognitive testing remained the key feature of the management process. However, the Standardized Concussion Assessment Tool (SCAT) was introduced for sideline use and included a neurologic screening tool, a signs and symptoms evaluation, Maddocks questions, and an abbreviated version of the SAC. Perhaps the most important advances were that for the first time no same-day return to play for concussed athletes was proposed, and the concept of cognitive rest was introduced.

Four years later in Zurich, the ICCS met to update its consensus recommendations.²⁰ This version abandoned the controversial simple-complex grading system and identified the clinical examination as the key component of the concussion diagnosis. The examination was to be supported by neurocognitive testing, which was not to be used in isolation but rather in conjunction with other assessment measures. This was reflected in the SCAT2, which included a modified version of the BESS (excluding foam-surface testing), a graded symptom checklist, Maddocks questions, and the complete SAC. Another key change was allowing a same-day return-to-play recommendation for adult athletes who showed complete symptom resolution.

The most recent ICCS meeting in 2012 resulted in few substantial changes to the 2008 recommendations and guidelines.⁴ The concussion diagnosis continued to be based on the clinical examination but was supported by other assessment modalities, and only the scoring, not the content, of the SCAT changed. The total or composite

SCAT score was removed, allowing each component to be scored independently and weighted equally. Additionally, use of the full BESS (ie, inclusion of the foam conditions) was recommended when possible to improve injury sensitivity.^{4,67} Overall, the 2012 consensus group introduced stricter guidelines regarding same-day return to play: no athlete should be returned, regardless of age or recovery status. Furthermore, the guidelines surrounding injury management were clarified. That is, absolute physical and cognitive rest was made less stringent; activities that did not provoke symptoms were allowed. Also, a 24-hour interval was instituted between return-to-play progression steps, with most athletes taking approximately 1 week to return to full participation once they become asymptomatic.

Most recently, the NATA updated its position statement on concussion management in 2014 to reflect the scientific advances that had occurred over the previous decade.⁸ The new document placed the onus on ATs to educate their athletes about the concussion-management process and to document all aspects of injury management. Similar to the 2004 NATA statement,¹⁸ all athletes at high risk for injury should undergo a baseline evaluation that includes a clinical history with a symptom assessment, physical and neurologic evaluation, and assessments for motor control and neurocognitive function. In the event of an injury, all of these tools should be implemented and used to support the clinical examination, which stands as the criterion standard for injury diagnosis. Once an injury is suspected or identified, no athlete should be returned to play on the same day. Concussed athletes should be removed from sport after injury, but physical and mental exertion that does not exacerbate symptoms is allowed. Unrestricted return to play, however, is not permitted until the athlete resumes baseline levels of performance and completes a 6-step exercise progression.

WHERE DO WE GO FROM HERE?

The science of diagnosing and managing concussion in sport is still evolving, and although many questions have been answered, much is still to be learned. In the last 15 years, published papers addressing concussions outnumber those published in the entire century prior, and ATs are at the forefront of much of this research. The most pressing question for clinicians and researchers remains: “Is there a biomarker that can identify and diagnose concussion and indicate complete metabolic recovery?” Next, we need a more definitive answer to “What are the long-term effects of injury?”

A number of research groups and private companies are searching for a 100% accurate diagnostic marker that can be deployed on the sideline within seconds of injury. Low-cost vision-based tests that can be easily implemented on the sideline or in a clinical setting have been investigated as a diagnostic tool but with inconclusive results.^{68,69} Salivary and serum samples have been the targets for many investigators, but no single marker or combination has been borne out.⁷⁰ Conversely, advanced imaging techniques, such as functional magnetic resonance imaging, diffusion tensor imaging, magnetic resonance spectroscopy, and event-related potentials, have shed light on many of the brain’s inner workings. With respect to concussion, it is reasonable to assume that brain-activation patterns after concussion may remain altered despite normal performance

on our best clinical-assessment tools.^{71,72} In addition, metabolic changes in the brain may persist beyond the time of clinical recovery.^{73–76} Measurements such as these are costly and time consuming to obtain, yet they may be the first step along the path to a diagnostic tool.

There is also considerable speculation that concussive injuries, or even repetitive head impacts in the absence of concussion, may propagate brain degeneration.⁷⁷ Extreme caution is warranted in interpreting these oft-publicized results. These findings are limited by a lack of high-level scientific designs, selection bias, lack of controls, and failure to address other physiological and psychological conditions that may have produced a similar outcome. Additionally, numerous publications counter the speculation of widespread long-term declines.^{78–86} Indeed, contact- and collision-sport athletes live longer than the general population; have no increased risk for dementia, Parkinson disease, or amyotrophic lateral sclerosis⁸⁷; and have a suicide rate that is half the expected rate in the general population.⁸⁸ Despite these findings, science continues to catch up with the misinformation communicated through the media regarding possible relationships. These results do not diminish our need to better understand the injury in the long term and to emphasize that prospective trials implementing a broad swathe of clinical assessments, advanced imaging techniques, and genotyping will answer these questions. Clinicians must therefore educate their athletes as to what has been clearly demonstrated in the literature and what remains to be proven.

CONCLUSIONS

No sports medicine topic is more polarizing than concussion, and today's standard of care supersedes where we were just a decade or two ago. In the era of evidence-based medicine, medical organizations are including objective measures as part of their clinical care for patients with concussive injuries. With the addition of each validated measure, more and more of the guesswork is being removed from the process, which is being quantified in a clinically meaningful way. Measures of symptoms, neurocognitive functioning, and postural control (ie, balance) provide ample objective information that clinicians can successfully integrate into their decision making. Injury management continues to be coordinated by the AT, who now manages a multidisciplinary team of medical professionals. Yet many questions persist about more sophisticated injury diagnostic measures, postinjury rehabilitation, and the long-term effects of injury. In the meantime, the responsible clinician must remain up to date on this topic and balance between what is known and what is reasonable and prudent for those patients in his or her clinical care.

REFERENCES

- Langlois JA, Rutland-Brown W, Thomas KE. *Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations, and Deaths*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2004.
- McCrorry PR, Berkovic SF. Concussion: the history of clinical and pathophysiological concepts and misconceptions. *Neurology*. 2001; 57(12):2283–2289.
- Giza CC, Hovda DA. The new neurometabolic cascade of concussion. *Neurosurgery*. 2014;75(suppl 4):S24–S33.
- McCrorry P, Meeuwisse WH, Aubry M, et al. Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport held in Zurich, November 2012. *Br J Sports Med*. 2013; 47(5):250–258.
- Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: a brief overview. *J Head Trauma Rehabil*. 2006;21(5):375–378.
- McCrea M, Guskiewicz KM, Randolph C, et al. Incidence, clinical course, and predictors of prolonged recovery time following sport-related concussion in high school and college athletes. *J Int Neuropsychol Soc*. 2013;19(1):22–33.
- Terwilliger V, Pratson L, Vaughan C, Gioia G. Additional post-concussion impact exposure may affect recovery in adolescent athletes. *J Neurotrauma*. 2016;33(8):761–765.
- Broglio SP, Cantu RC, Gioia GA, et al. National Athletic Trainers' Association position statement: management of sport concussion. *J Athl Train*. 2014;49(2):245–265.
- Harmon KG, Drezner J, Gammons M, et al. American Medical Society for Sports Medicine position statement: concussion in sport. *Clin J Sport Med*. 2013;23(1):1–18.
- Broglio SP, Macciocchi SN, Ferrara MS. Sensitivity of the concussion assessment battery. *Neurosurgery*. 2007;60(6):1050–1057.
- McCrea M, Barr WB, Guskiewicz KM, et al. Standard regression-based methods for measuring recovery after sport-related concussion. *J Int Neuropsychol Soc*. 2005;11(1):58–69.
- Barth JT, Alves W, Ryan T. Mild head injury in sports: neuropsychological sequelae and recovery of function. In: Levin H, Eisenberg HA, Benton A, eds. *Mild Head Injury*. New York, NY: Oxford University Press; 1989.
- Barth JT, Freeman JR, Broshek DK. Mild head injury. In: Ramachandran V, ed. *Encyclopedia of the Human Brain*. San Diego, CA: Academic Press; 2002:81–92.
- Macciocchi SN, Barth JT, Alves W, Rimel RW, Jane JA. Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery*. 1996;39(3):510–514.
- Covassin T, Elbin R III, Stiller-Ostrowski JL. Current sport-related concussion teaching and clinical practices of sports medicine professionals. *J Athl Train*. 2009;44(4):400–404.
- Lovell MR, Collins MW. Neuropsychological assessment of the college football player. *J Head Trauma Rehabil*. 1998;13(2):9–26.
- Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. *Br J Sports Med*. 2002;36(1):6–10.
- Guskiewicz KM, Bruce SL, Cantu RC, et al. National Athletic Trainers' Association position statement: management of sport-related concussion. *J Athl Train*. 2004;29(3):280–297.
- McCrorry P, Johnston K, Meeuwisse W, et al. Summary and agreement statement of the second International Conference on Concussion in Sport, Prague 2004. *Br J Sports Med*. 2005;39(4):196–204.
- McCrorry P, Meeuwisse W, Johnston K, et al. Consensus statement on concussion in sport, 3rd International Conference on Concussion in Sport held in Zurich, November 2008. *Br J Sports Med*. 2009; 43(suppl 1):i76–i90.
- Guskiewicz KM, McCrea M, Marshall SW, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. *JAMA*. 2003;290(19):2549–2555.
- Guskiewicz KM, Weaver NL, Padua DA, Garrett WE. Epidemiology of concussion in collegiate and high school football players. *Am J Sports Med*. 2000;28(5):643–650.
- Delaney JS, Lacroix VJ, Leclerc S, Johnston KM. Concussions among university football and soccer players. *Clin J Sport Med*. 2002;12(6):331–338.
- Iverson GL, Lange RT. Examination of “postconcussion-like” symptoms in a healthy sample. *Appl Neuropsychol*. 2003;10(3): 137–144.
- Randolph C, Millis S, Barr WB, et al. Concussion symptom inventory: an empirically derived scale for monitoring resolution of

- symptoms following sport-related concussion. *Arch Clin Neuropsychol*. 2009;24(3):219–229.
26. Piland SG, Motl RW, Guskiewicz KM, McCrea M, Ferrara MS. Structural validity of a self-report concussion-related symptom scale. *Med Sci Sports Exerc*. 2006;38(1):27–32.
 27. Piland SG, Motl RW, Ferrara MS, Peterson CL. Evidence for the factorial and construct validity of a self-report concussion symptoms scale. *J Athl Train*. 2003;38(2):104–112.
 28. Morgan CD, Zuckerman SL, Lee YM, et al. Predictors of postconcussion syndrome after sports-related concussion in young athletes: a matched case-control study. *J Neurosurg Pediatr*. 2015;15(6):589–598.
 29. Lovell MR, Iverson GL, Collins MW, McKeag DB, Maroon JC. Does loss of consciousness predict neuropsychological decrements after concussion? *Clin J Sport Med*. 1999;9(4):193–198.
 30. Lovell MR, Collins MW, Maroon JC, et al. Inaccuracy of symptom reporting following concussion in athletes. *Med Sci Sports Exerc*. 2002;34(5):S298.
 31. LaRoche AA, Nelson LD, Connelly PK, Walter KD, McCrea MA. Sport-related concussion reporting and state legislative effects. *Clin J Sport Med*. 2016;26(1):33–39.
 32. McCrea M, Hammeke T, Olsen G, Leo P, Guskiewicz K. Unreported concussion in high school football players: implications for prevention. *Clin J Sport Med*. 2004;14(1):13–17.
 33. Fedor A, Gunstad J. Limited knowledge of concussion symptoms in college athletes. *Appl Neuropsychol Adult*. 2015;22(2):108–113.
 34. Valovich McLeod TC, Bay RC, Lam KC, Chhabra A. Representative baseline values on the Sport Concussion Assessment Tool 2 (SCAT2) in adolescent athletes vary by gender, grade, and concussion history. *Am J Sports Med*. 2012;40(4):927–933.
 35. Piland SG, Ferrara MS, Macciocchi SN, Broglio SP, Gouteyron JF. Investigation of baseline self-report concussion symptom scores. *J Athl Train*. 2010;45(3):273–278.
 36. Ellis MJ, Leddy JJ, Willer B. Physiological, vestibulo-ocular and cervicogenic post-concussion disorders: an evidence-based classification system with directions for treatment. *Brain Inj*. 2015;29(2):238–248.
 37. Seifert T. Headache in sports. *Curr Pain Headache Rep*. 2014;18(9):448.
 38. McCrea M, Guskiewicz KM, Marshall SW, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. *JAMA*. 2003;290(19):2556–2563.
 39. Baker JG, Freitas MS, Leddy JJ, Kozlowski KF, Willer BS. Return to full functioning after graded exercise assessment and progressive exercise treatment of postconcussion syndrome. *Rehab Res Pract*. 2012;2012:705309.
 40. Leddy JJ, Kozlowski K, Donnelly JP, Pendergast DR, Epstein LH, Willer B. A preliminary study of subsymptom threshold exercise training for refractory post-concussion syndrome. *Clin J Sport Med*. 2010;20(1):21–27.
 41. Silverberg ND, Iverson GL. Is rest after concussion “the best medicine?”: recommendations for activity resumption following concussion in athletes, civilians, and military service members. *J Head Trauma Rehabil*. 2013;28(4):250–259.
 42. Broglio SP, Macciocchi SN, Ferrara MS. Neurocognitive performance of concussed athletes when symptom free. *J Athl Train*. 2007;42(4):504–508.
 43. Fazio VC, Lovell MR, Pardini JE, Collins MW. The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *NeuroRehabilitation*. 2007;22(3):207–216.
 44. Schatz P, Glatts C. “Sandbagging” baseline test performance on ImPACT, without detection, is more difficult than it appears. *Arch Clin Neuropsychol*. 2013;28(3):236–244.
 45. Erdal K. Neuropsychological testing for sports-related concussion: how athletes can sandbag their baseline testing without detection. *Arch Clin Neuropsychol*. 2012;27(5):473–479.
 46. Moser RS, Schatz P, Neidzwski K, Ott SD. Group versus individual administration affects baseline neurocognitive test performance. *Am J Sports Med*. 2011;39(11):2325–2330.
 47. Schatz P, Pardini JE, Lovell MR, Collins MW, Podell K. Sensitivity and specificity of the ImPACT Test Battery for concussion in athletes. *Arch Clin Neuropsychol*. 2006;21(1):91–99.
 48. Resch J, Driscoll A, McCaffrey N, et al. ImPact test-retest reliability: reliably unreliable? *J Athl Train*. 2013;48(4):506–511.
 49. Elbin RJ, Schatz P, Covassin T. One-year test-retest reliability of the online version of ImPACT in high school athletes. *Am J Sports Med*. 2011;39(11):2319–2324.
 50. Broglio SP, Ferrara MS, Macciocchi SN, Baumgartner TA, Elliott R. Test-retest reliability of computerized concussion assessment programs. *J Athl Train*. 2007;42(4):509–514.
 51. Alsalaheen B, Stockdale K, Pechumer D, Broglio SP. Measurement error in the Immediate Postconcussion Assessment and Cognitive Testing (ImPACT): systematic review. *J Head Trauma Rehabil*. 2016;31(4):242–251.
 52. Ferrara MS, McCrea M, Peterson CL, Guskiewicz KM. A survey of practice patterns in concussion assessment and management. *J Athl Train*. 2001;36(2):145–149.
 53. Notebaert AJ, Guskiewicz KM. Current trends in athletic training practice for concussion assessment and management. *J Athl Train*. 2005;40(4):320–325.
 54. McCrea M, Kelly JP, Kluge J, Ackley B, Randolph C. Standardized assessment of concussion in football players. *Neurology*. 1997;48(3):586–588.
 55. Romberg MH. *A Manual of the Nervous Disease of Man*. London, England: Sydenham Society; 1853.
 56. Shumway-Cook A, Horak FB. Assessing the influence of sensory interaction on balance. *Phys Ther*. 1986;66(10):1548–1550.
 57. Ingersoll CD, Armstrong CW. The effects of closed-head injury on postural sway. *Med Sci Sports Exerc*. 1992;24(7):739–743.
 58. Guskiewicz KM, Perrin DH, Gansneder BM. Effects of mild head injury on postural stability in athletes. *J Athl Train*. 1996;31(4):300–306.
 59. Guskiewicz KM, Riemann BL, Perrin DH, Nashner LM. Alternative approaches to the assessment of mild head injury in athletes. *Med Sci Sports Exerc*. 1997;29(suppl 7):S213–S221.
 60. Peterson CL, Ferrara MS, Mrazik M, Piland SG, Elliot R. Evaluation of neuropsychological domain scores and postural stability following cerebral concussion in sports. *Clin J Sport Med*. 2003;13(4):230–237.
 61. Guskiewicz KM, Ross SE, Marshall SW. Postural stability and neuropsychological deficits after concussion in collegiate athletes. *J Athl Train*. 2001;36(3):263–273.
 62. Riemann BL, Caggiano NA, Lephart SM. Examination of a clinical method of assessing postural control during a functional performance task. *J Sport Rehabil*. 1999;8(3):171–183.
 63. Riemann BL, Guskiewicz KM. Objective mild head injury evaluation through a battery of clinical postural stability tests. *J Athl Train*. 1998;33(2):S18.
 64. Riemann BL, Guskiewicz KM. Assessment of mild head injury using measures of balance and cognition: a case study. *J Sport Rehabil*. 1997;6:283–289.
 65. Riemann BL, Guskiewicz KM, Shields EW. Relationship between clinical and forceplate measures of postural stability. *J Sport Rehabil*. 1999;8(2):71–82.
 66. Belanger HG, Vanderploeg RD. The neuropsychological impact of sports-related concussion: a meta-analysis. *J Int Neuropsychol Soc*. 2005;11(4):345–357.
 67. Guskiewicz KM, Register-Mihalik J, McCrory P, et al. Evidence-based approach to revising the SCAT2: introducing the SCAT3. *Br J Sports Med*. 2013;47(5):289–293.
 68. Mucha A, Collins MW, Elbin RJ, et al. A brief Vestibular/Ocular Motor Screening (VOMS) assessment to evaluate concussions: preliminary findings. *Am J Sports Med*. 2014;42(10):2479–2486.
 69. Galetta KM, Morganroth J, Moehringer N, et al. Adding vision to concussion testing: a prospective study of sideline testing in youth and collegiate athletes. *J Neuroophthalmol*. 2015;35(3):235–241.
 70. Zetterberg H, Blennow K. Fluid markers of traumatic brain injury. *Mol Cell Neurosci*. 2015;66(pt B):99–102.

71. Jantzen KJ, Anderson B, Steinberg FL, Kelso JA. A prospective functional MR imaging study of mild traumatic brain injury in college football players. *Am J Neuroradiol.* 2004;25(5):738–745.
72. McAllister TW, Sparling MB, Flashman LA, Guerin SJ, Mamourian AC, Saykin AJ. Differential working memory load effects after mild traumatic brain injury. *Neuroimage.* 2001;14(5):1004–1012.
73. Vagnozzi R, Signoretti S, Cristofori L, et al. Assessment of metabolic brain damage and recovery following mild traumatic brain injury: a multicentre, proton magnetic resonance spectroscopic study in concussed patients. *Brain.* 2010;133(11):3232–3242.
74. Vagnozzi R, Signoretti S, Tavazzi B, et al. Temporal window of metabolic brain vulnerability to concussion: a pilot H-magnetic resonance spectroscopy study in concussed athletes. Part III. *Neurosurgery.* 2008;62(6):1286–1295.
75. Henry LC, Tremblay S, Leclerc S, et al. Metabolic changes in concussed American football players during the acute and chronic post-injury phases. *BMC Neurol.* 2011;11:105.
76. Cimatti M. Assessment of metabolic cerebral damage using proton magnetic resonance spectroscopy in mild traumatic brain injury. *J Neurosurg Sci.* 2006;50(4):83–88.
77. McKee AC, Stern RA, Nowinski CJ, et al. The spectrum of disease in chronic traumatic encephalopathy. *Brain.* 2013;136(pt 1):43–64.
78. Blosler F. NFL mortality study. Washington, DC: US Department of Health and Human Services, National Institute for Occupational Safety and Health; 1994.
79. Davis GA, Castellani RJ, McCrory P. Neurodegeneration and sport. *Neurosurgery.* 2015;76(6):643–655.
80. Maroon JC, Winkelman R, Bost J, Amos A, Mathyssek C, Miele V. Chronic traumatic encephalopathy in contact sports: a systematic review of all reported pathological cases. *PLoS One.* 2015;10(2):e0117338.
81. Meehan W III, Mannix R, Zafonte R, Pascual-Leone A. Chronic traumatic encephalopathy and athletes. *Neurology.* 2015;85(17):1504–1511.
82. Castellani RJ, Perry G, Iverson GL. Chronic effects of mild neurotrauma: putting the cart before the horse? *J Neuropathol Exp Neurol.* 2015;74(6):493–499.
83. Iverson GL, Gardner AJ, McCrory P, Zafonte R, Castellani RJ. A critical review of chronic traumatic encephalopathy. *Neurosci Biobehav Rev.* 2015;56:276–293.
84. Gardner A, Iverson GL, McCrory P. Chronic traumatic encephalopathy in sport: a systematic review. *Br J Sports Med.* 2014;48(2):84–90.
85. McCrory PR, Meeuwisse WH, Kutcher JS, Jordan BD, Gardner A. What is the evidence for chronic concussion-related changes in retired athletes: behavioural, pathological and clinical outcomes? *Br J Sports Med.* 2013;47(5):327–330.
86. Solomon GS, Zuckerman SL. Chronic traumatic encephalopathy in professional sports: retrospective and prospective views. *Brain Inj.* 2015;29(2):164–170.
87. Savica R, Parisi JE, Wold LE, Josephs KA, Ahlskog JE. High school football and risk of neurodegeneration: a community-based study. *Mayo Clin Proc.* 2012;87(4):335–340.
88. Baron SL, Hein MJ, Lehman E, Gersic CM. Body mass index, playing position, race, and the cardiovascular mortality of retired professional football players. *Am J Cardiol.* 2012;109(6):889–896.

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