



# Research DIGEST

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## Guest Authors

Kevin M. Guskiewicz, PhD, ATC  
University of North Carolina  
at Chapel Hill

Ruben J. Echemendia, PhD  
Psychological and  
Neurobehavioral Associates,  
State College, PA

Robert Cantu, MD  
University of North Carolina  
at Chapel Hill  
Emerson Hospital, Concord,  
MA

Brigham and Women's  
Hospital, Boston, MA

## Co-editors

David Bassett, Jr., PhD  
University of Tennessee

Michael La Monte, PhD  
University of Buffalo  
The State University of  
New York

Diane Wiese-Bjornstal, PhD  
University of Minnesota

## Assessment and Return to Play Following Sports-Related Concussion

### Introduction

Cerebral concussion is perhaps the most puzzling and mismanaged neurological condition described in the sports medicine literature. While identifying and diagnosing the injury can be quite challenging, the greatest mystery lies in the pathophysiology and the brain's nonsystematic course of recovery. Concussive injuries are common and comprise nearly 10% of all athletic injuries (2, 77). Despite the fact that the number of publications on *sport concussion* and *mild traumatic brain injury* in sport published since the year 2000 is more than twice the total number of publications in the 30 years preceding the start of the 21st century, there is much debate surrounding the best management strategies. The old adage of "every question answered generates two new questions" could not be more true when dealing with our knowledge regarding the evaluation and management of sports-related concussion. The new knowledge in this area has led to significant discussions among international experts, consensus groups, and has resulted in several recent position papers on the topic of sport-related concussion (1, 6, 33, 61, 62).

The purpose of this paper is to review the current state-of-the-art and -science with respect to the assessment and management of athletes who are at risk for sustaining cerebral concussion. The assessment and management protocols are derived from the most recent scientific and clinical based literature on sport-related concussion.



*"Contemporary methods of concussion assessment, involving the use of symptom checklists, neuropsychological testing, and postural stability testing, are indicated for any athlete suspected of having sustained a concussion."*

## Defining the Injury

Cerebral concussion is best classified as a mild diffuse brain injury and is often referred to as mild traumatic brain injury (MTBI). MTBI is an injury that occurs as a result of a blow to the head or other part of the body causing an acceleration or deceleration of the brain inside the skull. Typical symptoms include: headache, nausea, vomiting, dizziness, balance problems, feeling “slowed down,” fatigue, trouble sleeping, drowsiness, sensitivity to light or noise, loss of consciousness (LOC), blurred vision, difficulty remembering, or difficulty concentrating (85). In 1966, the Congress of Neurological Surgeons proposed the following consensus definition of concussion: “Concussion is a clinical syndrome characterized by immediate and transient impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces” (22). Although the definition received widespread support in 1966, more contemporary opinion is that this definition fails to include many of the predominant clinical features of concussion, such as headache, nausea, amnesia, concentration difficulties, and balance deficits (1, 6, 33, 45, 61, 62, 84). It is often reported that there is no universal agreement on the definition; however, agreement does exist on several features that incorporate clinical, pathologic, and biomechanical factors associated with MTBI:

- (1) concussion is caused by a direct blow to the head or elsewhere on the body resulting in a sudden mechanical loading of the head that generates turbulent rotatory and other movements of the cerebral hemispheres.
- (2) these collisions or impacts between the cortex and bony walls of the skull typically cause an immediate and short-lived impairment of neurologic function involving a potpourri of symptoms; in some cases the symptomatology is long-lasting and results in a condition known as “post-concussion syndrome.”
- (3) concussion may cause neuropathologic changes or temporary deformation of tissue; however, the acute clinical symptoms primarily reflect a functional disturbance rather than a structural injury.
- (4) concussion may cause a gradient of clinical syndromes that may or may not involve loss of consciousness; resolution of the clinical and cognitive symptoms often follows a sequential course, but is dependent upon a number of factors including magnitude of the impact and the individual’s concussion history.
- (5) concussion is most often associated with normal results on conventional neuroimaging studies (6, 27, 33, 45, 74, 76, 84).

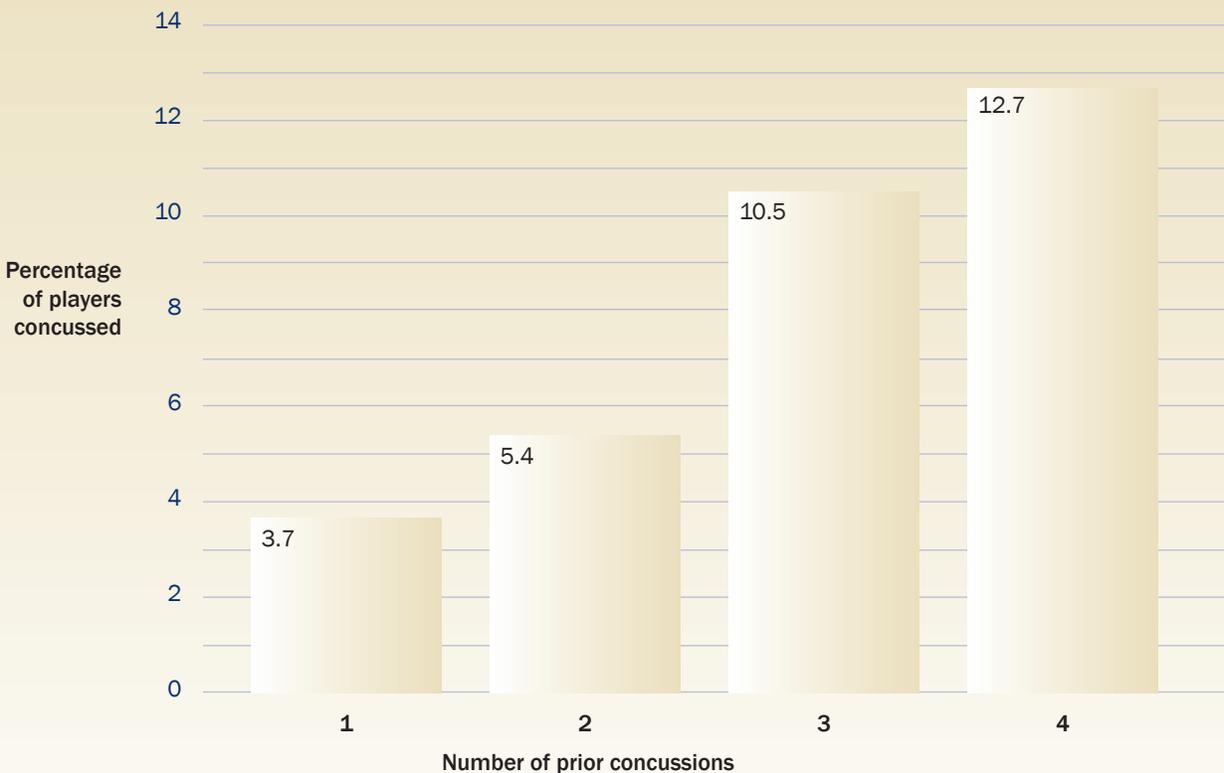
The colloquial term “ding” has too often been used when athletes sustain a blow to the head, resulting in a stunned and confused state that resolves within minutes. This term trivializes the initial confused state since the evolving signs and symptoms of a concussion are often not evident until several minutes to hours after the blow (33). The terms “ding” and “bell ringer” should not

be used to describe a concussive injury; likewise, as per the Zurich guidelines, the term “simple concussion” should be discouraged (62). While it is most important for athletes, parents, coaches, and clinicians to recognize a concussive injury, it is less important that the injury be graded or classified as mild, moderate, or severe. Concussion education is paramount, and efforts by organizations such as the Centers for Disease Control and Prevention, National Football League, National Hockey League, National Collegiate Athletic Association, National Federation of State High School Athletic Associations are helping athletes to understand the signs and symptoms of a concussion, as well as the potential negative consequences (i.e., second-impact syndrome, predisposition to future concussions, post-concussion syndrome, etc.) of not reporting a concussive episode. Once the athlete understands the injury, they can provide a more accurate report of factors associated with the concussion. Creating this awareness is vitally important in the global management of the injury. Sports medicine clinicians should take an active role in the education of athletes, coaches, parents, other medical personnel, and the media.

One point of widespread agreement is that the immediate management of the concussed athlete varies according to the nature and severity of the injury. It is important for clinicians to recognize that concussions are rarely associated with macroscopic abnormalities on neuroimaging because MTBI most often results in select neurons being rendered temporarily dysfunctional but not destroyed. Although concussion is often referred to as a “diffuse axonal injury” (DAI), MTBI does not usually appear to result in “shearing” of axons, but rather in a stretching, twisting, and in some cases a separation of dendritic branches (16, 74, 75, 86). This process typically occurs in a very small number of axons within the region of insult, with the vast majority of axons initially affected recovering over a short period of time. The pathophysiologic sequence that occurs following traumatic brain injury, even in its mildest form, is best described as a process and not an event (16, 28, 29, 45, 74, 75, 86). This process has significant clinical relevance when considering an athlete’s risk for recurrent injury.

Once an athlete has suffered a concussion, he or she is at increased risk for subsequent head injuries. Guskiewicz and colleagues (35, 40) found that collegiate athletes had a 3-fold greater risk of suffering a concussion if they had sustained 3 or more previous concussions in a 7-year period and that players with 2 or more previous concussions required a longer time for total symptom resolution after subsequent injuries (Figure 1). Players also had a 3-fold greater risk for subsequent concussions in the same season, whereas recurrent, in-season injuries occurred within 10 days of the initial injury 92% of the time (35). Another similar study of high school athletes (20), found that athletes with 3 or more prior concussions were at an increased risk of experiencing LOC (8-fold greater risk), anterograde amnesia (5.5-fold greater risk), and confusion (5.1-fold greater risk) after subsequent concussion.

**Figure 1. Percentage of players concussed during the study period, stratified by number of prior concussions (0, 1, 2, or 3+) (35)**



$\chi^2 = 30.11$ ,  $df=3$ ,  $P < .001$

From: Guskiewicz, K. M., M. McCrea, S. W. Marshall, R. C. Cantu, C. Randolph, W. Barr, J. A. Onate and J. P. Kelly. Cumulative effects associated with recurrent concussion in collegiate football players: The NCAA Concussion Study. *JAMA* 290:2549-2555, 2003.

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Despite the increasing body of literature, debate still surrounds the question of how many concussions are enough to recommend ending the player's career. Some research suggests that 3 concussions in a career may be enough to permanently end a player's participation in sport (20, 34, 35). However, other studies suggest that the total number of concussions is not related to decreased neurocognitive functioning (17, 60). In light of these contradictory findings, an individualized approach to concussion management appears to be the most appropriate because concussions present in varying degrees of severity, and all athletes do not respond in the same way to concussive insults. Emerging human as well as animal data suggest that an athlete's age is important factor to consider in the management of concussed athletes. Consistent with the animal literature (63) regarding the immature brain, younger athletes tend to have more severe symptoms that last longer than older athletes (26, 53, 66).

The increased risk of recurrent injury, as well as the slower recovery often seen following subsequent concussions, may be

indicative of increased neuronal vulnerability following multiple concussive injuries. Animal studies have described an acute neuro-metabolic cascade involving accelerated glycolysis and increased lactate production immediately following concussion (29, 64, 71). The increased lactate is believed to leave neurons more vulnerable to secondary ischemic injury and has been considered a possible predisposition to repeat injury (29). Later steps in this physiologic cascade involve increased intracellular calcium, mitochondrial dysfunction, impaired oxidative metabolism, decreased glycolysis, axonal disconnection, neurotransmitter disturbances, and delayed cell death. Decreased cerebral blood flow has been reported to last approximately 10 days following concussive injuries in animal models (29) which is consistent with the finding of an apparent 7-10 day period of increased susceptibility to recurrent injury. The disrupted cellular metabolism described in animal models that creates increased cellular vulnerability (29, 43, 78) requires further investigation in human models.

## Clinical Evaluation

A thorough clinical examination conducted on the field of play is critical for the identification and management of sports concussions. This initial evaluation (or series of evaluations) should include a thorough *history* (including number and severity of previous head injuries), *observation* (including pupil responses), *palpation*, and *special tests* (including simple tests of memory, concentration, motor coordination and cranial-nerve functioning). The primary goal of the on-field assessment is to identify any life threatening conditions such as a developing intracranial bleed. In cases where the athlete's symptomatology is worsening, especially if there is a deterioration to a stuporous, semi-comatose, or comatose state of consciousness, the situation must be treated as a medical emergency.

In most cases, the athlete will be medically stable and the assessment progresses to a secondary level with the intent of determining the developing signs and symptoms of the concussion. An emerging model of sport concussion assessment involves the use of brief screening tools to evaluate post-concussion signs and symptoms, cognitive functioning, and postural stability on the sideline immediately after a concussion, as well as more sophisticated neuropsychological evaluation to track recovery further out from the time of injury. Ultimately, the results from the neuropsychological tests, coupled with the physical examination and other aspects of the injury evaluation, assist the clinician and other sports medicine professionals in making a safe return to play decision.

### Post-concussion Symptom Assessment

Self-reported symptoms are among the more obvious and recognizable ways to assess the effects of concussion. Typical self-reported symptoms after a concussion include but are not limited to headache, dizziness, nausea, and vomiting. Other common symptoms are listed in Table 1 (6). Self-reported symptoms are referenced by many of the concussion grading scales (14, 21, 24, 29, 50). A number of concussion symptom checklists (14, 21, 24, 39) and scales (53, 54, 58, 73) have been used in both research and clinical settings. A symptom checklist, which is comprised of a list of concussion-related symptoms, allows the athlete to report whether the symptom is present or not by responding either "yes" (experiencing the symptom) or "no" (not experiencing the symptom). A symptom scale is a summative measure that allows the athlete to describe the extent to which he or she is experiencing the symptom. These instruments commonly incorporate a Likert-type scale that allows the player to rate presence and severity of post-concussion symptoms. These scores are then summed to form a composite score that yields a quantitative measure of overall injury severity and benchmark against which to track post-injury symptom recovery. Initial evidence has been provided for the structural validity for a self-

report concussion symptom scale (73). The symptoms contained in these checklists are not specific to concussion, as most people have experienced headache, fatigue, irritability, trouble sleeping, etc. at some point in their lives. Thus, obtaining a baseline symptom score is helpful to establish the type and frequency of any preexisting symptoms that are attributable to factors other than the head injury (e.g., illness, fatigue, travel, and/or menstruation). Although very useful in assessing and managing concussive injuries, it is important to underscore that athletes are motivated to return to play and may consciously or unconsciously minimize or omit their symptoms in order to return to the playing field (22, 61).

### Use of objective concussion assessment tools

Comprehensive assessment of concussion is best accomplished within the context of a multi-disciplinary sports medicine team that has access to a variety of assessment techniques including sideline assessment, medical evaluation, neuropsychological testing and the assessment of postural stability.

### Neuropsychological Assessment

Neuropsychological testing in sports has become very popular over the last 15 years for the assessment of concussion and identification of an athlete's functional and cognitive limitations following injury. Since much of the neurocognitive impairment that occurs following MTBI is a result of abnormal neurochemical cascades that begin within hours and minutes of the concussive blow (42, 85), only those techniques that assess functional status are effective in detecting the injury. Neuroimaging techniques, such as fMRI (functional magnetic resonance imaging) that assess the functional aspects of the brain injury, are useful in research applications and show promise for clinical utility (8).

In order for neuropsychologists to infer the presence of pathology through deficit measurement, post-injury test scores must be compared to an estimate of "pre-morbid" or pre-injury measurement. Unlike most other populations, athletes can be evaluated prior to sustaining a MTBI, which creates "baseline" data than can be used to accurately compare pre and post-injury functioning. Barth et al. (7) at the University of Virginia are widely credited with initiating this baseline testing paradigm in the mid-1980's. Using baseline testing among college football players, they demonstrated that neurocognitive deficits were apparent in athletes as soon as 24 hours post- and as long as 5 days post-concussion. Cognitive functioning gradually improved over a period of time with most, but not all athletes showing complete recovery by about 10 days post-injury. Research using this baseline- serial post-injury evaluation paradigm has grown exponentially since the early studies by Barth et al. (7).

Echemendia et al. (24) examined a multi-sport college population and found that a limited battery of neuropsychological measures

**Table 1. Graded Symptom Checklist**

**Graded Symptom Checklist (GSC)**

Symptom	Time of Injury	2-3 Hours Post-injury	24 Hours Post-injury	48 Hours Post-injury	72 Hours Post-injury
Blurred vision					
Dizziness					
Drowsiness					
Excess sleep					
Easily distracted					
Fatigue					
Feel "in a fog"					
Feel "slowed down"					
Headache					
Inappropriate emotions					
Irritability					
Loss of consciousness					
Loss of orientation					
Memory problems					
Nausea					
Nervousness					
Personality change					
Poor balance/coordination					
Poor concentration					
Ringing in ears					
Sadness					
Seeing stars					
Sensitivity to light					
Sensitivity to noise					
Sleep disturbance					
Vacant stare/glassy eyed					
Vomiting					

NOTE: The GSC should be used not only for the initial evaluation but for each subsequent follow-up assessment until all signs and symptoms have cleared at rest and during physical exertion. In lieu of simply checking each symptom present, the certified athletic trainer can ask the athlete to grade or score the severity of the symptom on a scale of 0-6, where 0 = not present, 1 = mild, 3 = moderate, and 6 = most severe.

could reliably differentiate concussed athletes from uninjured athletes as soon as 2 hours post-injury. They found that the concussed athletes scored significantly lower than controls at 2 hrs and 48 hours following injury, and that group differences were also evident at one week following injury. No differences were found between the groups at one month post-injury. This study underscored the dynamic nature of recovery following concussion because concussed athletes' neuropsychological performance *declined* from 2 hours to 48 hours post-injury whereas the control group *improved* during the same time frame. Other groups have since been replicated this finding using different neuropsychological techniques (10, 39, 57). More importantly, Echemendia et al. (24) found that while neuropsychological test scores could statistically differentiate between concussed and non-concussed athletes at 48 hours, post-concussion symptoms as measured by the standardized Post-Concussion Symptom Scale could not distinguish the two groups. This finding is noteworthy because it helped to expose the problems of relying exclusively on symptoms to determine return to play.

McCrea et al. (59) used a sideline cognitive screening instrument (Standardized Assessment of Concussion; SAC) in combination with selected traditional neuropsychological measures with college students and found that concussed athletes' SAC scores were significantly lower than baseline when compared to non-concussed athletes. In contrast to studies using more comprehensive neuropsychological measures that showed typical recovery by 10 days post-injury, scores on the SAC returned to baseline within 48 hours of injury. These findings highlight the complimentary nature of brief screening instruments and the more comprehensive batteries. Brief screening instruments such as the SAC have proven to be very useful on the sideline and during the acute phase of recovery (initial 48 hours), whereas neuropsychological test batteries are more effective in identifying enduring neurocognitive deficits (57).

The development of computerized test platforms provided a paradigm shift for neuropsychological assessment in sports. Several computerized neuropsychological test platforms have

been developed, but only four have consistently appeared in the sports concussion literature: ImPACT (Immediate Post-Concussion Assessment and Cognitive Testing) (52), CogSport (18), HeadMinder Concussion Resolution Index (48), and the Automated Neuropsychological Assessment Metrics Sports Medicine Battery (ANAM-SMB) (9). These batteries allow for groups of athletes to be assessed in a standardized manner, with better measurement of reaction time, and virtually immediate access to test scores (82, 83). Although different in their content, each of these batteries allows for a thorough assessment of simple and complex information processing speed, which has been shown to be a key deficit following concussion. While these batteries have extended the use of neuropsychological measurement to a much larger number of athletes in a cost-effective manner, they have drawbacks as well: they do not fully assess memory functioning since they are only capable of examining recognition memory; they minimize the interaction between the athlete and the neuropsychologist thereby reducing qualitative observations of performance; player motivation and effort is less effectively assessed and managed using group administration formats; and they limit the ability to examine the *process* by which injured athletes solve problems, learn information, and remember information (12). Hybrid concussion management programs exist and employ a combination of paper and pencil assessment and computer-based assessment.

Computer-based assessment has generated a rich literature in the neuropsychological study of concussion. Using ImPACT, Collins et al. (19) examined on-field predictors of neuropsychological functioning in a large sample of concussed high school and college athletes. At three days post-injury, athletes with "poor" outcomes were 10-times more likely to have had on-field retrograde amnesia and four times more likely have had any on-field traumatic amnesia. No effect was found for loss of consciousness. Erlanger et al. (25) employed the Headminder CRI computer battery and found that cognitive impairment at initial post-concussion assessment (when compared to baseline) is a significant predictor of duration of post-concussion symptoms. Using the ANAM-

**Figure 2. Balance Error Scoring System**



SMB, Bleiberg et al. (11) replicated the findings by Echemendia et al. (24) showing that impairment in cognitive functioning was apparent on the day of injury and 1-2 days post-injury, with recovery over a 3-7 day period. They noted that the differences between the groups were not necessarily due to a decrement in functioning in the concussed group but rather an absent or restricted practice effect in the concussed group when compared to controls (10). Interestingly, the same pattern was observed when concussed athletes with a history of concussion were compared to those with no history of concussion. Control subjects and injured athletes with no history of concussion revealed practice effects whereas concussed athletes with a history or previous concussions did not reveal practice effects (9).

Researchers have provided the medical community with a wealth of knowledge regarding neuropsychological assessment in recent years. Nearly everyone agrees that neuropsychological testing is an integral part of the assessment plan following concussion. However, there is still some disagreement as to the most appropriate timing and frequency of these post-injury assessments. Many clinical research protocols (21, 24, 25, 38, 39, 53, 57, 61) have utilized daily serial testing, regardless of the presence of symptoms, while others have recommended testing only once an athlete is symptom-free (56, 61).

### Balance Assessment

Clinical balance testing following concussion has proven to be useful (39). Disruption of specific brain areas following head injury affects postural equilibrium (4, 5, 32, 39, 55, 65, 81). Concussion diminishes cerebral reserve despite apparent recovery and places individuals at risk for more prolonged disability after a second concussion (30, 31). For years, clinicians have evaluated head injury and balance with Romberg's tests of sensory modality function. This is an easy and effective sideline test, but there is more to posture control than just balance and sensory modality (67-70), especially when assessing head injured subjects (5, 36, 37, 44). Therefore, more refined testing instruments have the potential to improve clinical decision-making.

Advancements in technology have provided the medical community with systems such as the Smart Balance Master or EquiTest (NeuroCom International, Inc., Clackamas, OR), which provide an easy and practical method of quantitatively assessing functional postural stability. Thus, the potential exists to assess concussed athletes, as well as identify possible abnormalities that might be associated with concussion, and develop a recovery curve based on quantitative measures for determining readiness to return to activity. While these force platform systems have gained acceptance for objectively assessing balance following concussion, they are expensive and therefore not readily available to many clinicians in the collegiate and high school settings. Therefore, the use of a clinical balance test, the Balance Error Scoring System (BESS), was developed as a valid and reliable low-tech assessment tool for the management of sport-related concussion (39, 79, 80).

Several studies have identified balance deficits during the initial 3-5 days following concussion, however, there is limited explanation as to why some concussed athletes demonstrate these deficits, and others do not (32, 36, 38, 39). Guskiewicz and colleagues (39) identified balance deficits lasting up to three days post-injury in 36 collegiate athletes recovering from MHI when assessed using both Sensory Organization Test (SOT) on the NeuroCom Smart Balance Master and the BESS. While these deficits could be due to a sensory interaction problem preventing concussed athletes from accurately utilizing and exchanging sensory information from the visual, vestibular, and somatosensory systems, more scientific study is needed.

The BESS is recommended over the standard Romberg test, which for years was used as a subjective tool for the assessment of balance. The BESS was developed to provide clinicians with a rapid, cost-effective method of objectively assessing postural stability in athletes on the sports sideline or training room after a concussion. Three different stances (double, single and tandem) are completed twice, once while on a firm surface and once while on a piece of medium density foam (Airex, Inc.), for a total of six trials (Figure 2). The test has been described in detail by Guskiewicz et al. (39, 57, 79, 80).

**Table 2. Cantu evidence-based grading system for concussion (15)**

Grade 1 (mild)	No LOC PTA < 30 minutes, PCSS < 24 hours
Grade 2 (moderate)	LOC < 1 minute or PTA > 30 minutes < 24 hours or PCSS > 24 hours < 7 days
Grade 3 (severe)	LOC ≥ 1 minute or PTA ≥ 24 hours or PCSS > 7 days

LOC = loss of consciousness; PTA = post-traumatic amnesia (anterograde/retrograde); PCSS = post-concussion signs/symptoms other than amnesia

A multifaceted approach to concussion assessment is strongly recommended for serial evaluations following concussion. The inclusion of objective balance testing in the assessment of concussion is recommended in combination with a graded symptom checklist and neuropsychological testing.

### **Determining Injury Severity**

As research has expanded our understanding of the complexity of concussive injuries, less emphasis has been placed on the practice of grading for injury severity. One concern has been that nearly all of the concussion severity scales placed far too much emphasis the presence or absence of loss of consciousness and amnesia. It is important to monitor all post-concussion symptoms that the athlete may experience so that a final grading of the severity of concussion can be deferred until all symptoms have cleared. It is this belief that gave rise to the grading scale shown in Table 2 (14).

While it is certainly possible to manage concussions without grading severity, the grade (given once symptoms have cleared and the athlete is preparing to return to activity) may be more meaningful in regards to how future concussions are managed. In other words, it can provide a concise descriptor of concussion history when managing subsequent concussions. For example, a more conservative approach would seem appropriate for someone with multiple severe concussions with signs and symptoms lasting weeks as compared to someone with multiple minor concussions with signs and symptoms lasting less than 24 hours.

### **Return to Play: The Management of Concussion**

It has been well established that players who have sustained one concussion are at a significantly higher risk for concussion than their non-concussed counterparts (35, 40) which emphasizes the need for accurate and empirically based return to play (RTP) decisions. Many clinicians believe the RTP guidelines are too conservative and, therefore, choose to base decisions on clinical judgment of individual cases rather than on general recommendations. It has been reported that 30% of all high school and collegiate football players sustaining concussions return to competition on the same day of injury; the remaining 70% average 4 days of rest before returning to participation. (35) Many RTP guidelines call for the athlete to be asymptomatic for at least 7 days before returning to participation after a grade 1 or grade 2 concussion (3, 13, 46, 47, 50). Although many clinicians deviate from these recommendations and are more liberal in making RTP decisions, studies (35, 57) suggest that perhaps the 7-day waiting period can minimize the risk of recurrent injury. On average, athletes required 7 days to fully recover after concussion. Same-season repeat injuries typically take place within a short window of time, 7 to 10 days after the first concussion (35), supporting the notion that there may be increased neuronal vulnerability or

blood-flow changes during that time, similar to those in animal models (29, 42, 43).

Sports medicine clinicians now have an array of techniques and measurement instruments that can and should be used in the management of concussion. Whether one uses post-concussion signs and symptoms, screening batteries (e.g., SAC), balance testing (37, 38), or neuropsychological testing, the temptation exists to select one indicator of functioning and use that indicator as the sole basis for return to play. These pieces all contribute to the concussion puzzle and no one piece should be used to the exclusion of others. Although differences exist, several influential papers (6, 33, 61, 62) have arrived at a consensus that emphasizes an individualized, graded return to play following a return to baseline of post-concussion signs and symptoms. All signs and symptoms should be evaluated and documented using a graded symptom scale or checklist when performing follow-up assessments and should be evaluated both at rest and after exertional maneuvers such as biking, jogging, sit-ups, and push-ups. As mentioned above, baseline measurements of neuropsychological and balance are strongly recommended so that they may be compared with post-injury measurements. Balance and neurocognitive function should return to baseline levels, allowing for changes due to practice effects and measurement error. The Prague (61) and Zurich (62) consensus statements emphasized the need for cognitive rest in addition to physical rest. In general, a graded progression of physical and cognitive exertion should be implemented following a period of being asymptomatic (usually 24-48 hours). Light aerobic exercise should be followed by sport-specific training and noncontact training drills, and any new or recurrent symptoms following these activities should be tracked. Documentation of symptoms is very important.

For the basketball player, this may include shooting baskets or participating in walk-throughs, and for the soccer player, this may include dribbling or shooting drills (but no heading) or other sport-specific activities. These activities are followed by full contact drills, and then return to play. Progression from one level of exertion to the next is predicated on the absence of post-concussion signs and symptoms at the previous level. At no time prior to release for full contact should a player be placed in a situation where they may sustain a head injury. In soccer, a graded progression of heading training is recommended as outlined by Kissick (51). The amount of time spent at each level of activity may vary depending on player history of concussion. For example, a player with a history of multiple concussions or a particularly severe concussion can be held at a level for 72 hours, rather than just 24 hours. Conversely, a player with a relatively minor concussion may be progressed more rapidly.

The individualized model replaces previous approaches to RTP that were based on grading concussion severity and applying uniform periods of inactivity based on concussion grade.

Although very useful and practical for college athletes and professional athletes who have access to a sports medicine team that is well versed in concussion management, this individualized model may prove less useful for high school and younger athletes who rely on their family physician or emergency room physician to guide their return to play. These physicians often do not have the expertise or the time to provide the careful monitoring and guidance that is needed for individualized progression. Physicians in these situations will likely resort to the use of previous guidelines that provide guidance for the amount of time needed for recovery prior to returning an athlete to sport.

It is strongly recommended that after recurrent injury, especially within-season repeat injuries, the athlete be withheld for an extended period of time (approximately 7 additional days) after symptoms have resolved.

## When to Disqualify an Athlete from Participation

The current consensus among most experts is that no athlete should return to participation on the same day after sustaining a concussion (62). This recommendation from the international consensus panel has been adopted by many of the sporting organizing bodies, such as NFSHSAA (National Federation of State High School Athletic Associations), NCAA (National Collegiate Athletic Association), and NFL (National Football League) in recent years, and most certainly should be followed when managing a suspected concussion in a youth or adolescent athlete. The exertional activities previously mentioned are useful in helping to determine readiness to return on serial testing in the days following the concussion. In making these decisions, several factors should be considered, including the athlete's age, concussion history, and sport-type. If the athlete is younger than 18 years old or has suffered a prior concussion, they should be managed with extra caution and the risk of subsequent concussions, Second Impact Syndrome, post-concussion syndrome, or other later-life consequences should be prominent considerations.

While the presence of LOC and/or amnesia have not been identified as good isolated predictors of long-term outcome following concussion, it is usually best to monitor these cases very carefully and to withhold activities for longer periods of time to observe for additional late-onset symptoms or worsening of existing symptoms. The decision to disqualify from further participation in a given season should be based on a comprehensive physical examination and serial assessment of symptoms, neurocognitive functioning, and balance. Additionally, other functional impairments (academic challenges, sleep disturbance, vertigo, etc.) and the athlete's past history of concussions should weigh into this decision.

Athletes with a history of prior concussions are at increased risk for sustaining subsequent concussions as well as for slowed recovery of self-reported post-concussion signs and symptoms, cognitive dysfunction, and balance (35, 40, 49, 57). In athletes with a history of three or more concussions and experiencing slowed recovery, a pattern of increased symptom duration, or the occurrence of concussive symptoms with less and less provocation, temporary or permanent disqualification from contact sports should be considered.

Though not based on prospective studies, many clinicians have operated under the belief that three mild concussions in one season should terminate participation in that season. Similarly, some believe that two moderate or severe concussions should also terminate a season. Although we cannot make definitive recommendations due to the lack of empirical evidence, clinical experience suggests that terminating a player's season after two concussions in a given season is most responsible, especially in the case of a younger athlete. If the season is terminated it is recommended that a sufficient amount of time (e.g., 3 months) with the player free of symptoms at rest and following exertion should elapse before RTP. Retirement from contact or collision sports participation should be seriously considered if a player's neurological examination has not returned to normal, or if they still have any post-concussion signs or symptoms at rest or exertion. Additional criteria that might well preclude return to competition would be a neuropsychological test battery that has not at least returned to baseline or imaging studies that show a lesion placing the athlete at increased risk of future head injury.

Finally, Guskiewicz et al. (34) studied the long-term consequences of repeated concussions, and reported that retired professional football players with 3 or more reported concussions had a 5-fold prevalence of mild cognitive impairment (MCI) diagnosis. Additionally, retired players with 3 or more concussions had a 3-fold prevalence of reported significant memory problems compared to retirees without a history of concussion. Although there was not an association between recurrent concussion and Alzheimer's Disease (AD), the researchers observed an earlier onset of AD in the retirees than the general U.S. male population. A later study involving a similar cohort of retired NFL players suggested a link between recurrent concussion and clinical depression in retired professional football players (41). These findings suggest that the histological features of dementia-related syndromes, as well as depression, may be initiated by repetitive cerebral concussions in professional football players, and that these findings may suggest early disqualification for those with recurrent concussion.

## Conclusions

There has been quite an evolution with respect to concussion management over the last 15 years. This evolution has brought technology and objective testing methods to the forefront of concussion management; the emphasis on education and awareness has certainly played a major part in helping to manage these injuries. Perhaps the greatest influence that clinicians can have on preventing these injuries, or at least preventing catastrophic outcome, is to educate athletes, coaches, and parents about the dangers of playing while symptomatic following a concussion. Reports of the cumulative effects of multiple concussions presented in this paper, as well as multiple head impacts on long-term cognitive functioning, should lead clinicians to re-think their approach to managing sport concussion. Contemporary methods of concussion assessment, involving the use of symptom checklists, neuropsychological testing, and postural stability testing, are indicated for any athlete suspected of having sustained a concussion. Clinicians working with high-risk sports should conduct baseline assessments, including neuropsychological and postural stability tests, prior to the start of their season. Testing should also be conducted following exertional activities that are typically performed prior to a full return to activity. Most importantly, clinicians must recognize that the recovery and RTP considerations involve many factors.

## References

1. Concussion (mild traumatic brain injury) and the team physician: A consensus statement. *Med Sci Sports Exerc.* 38:395-399, 2006.
2. Centers for Disease Control and Prevention. Nonfatal sports and recreation-related injuries treated in emergency departments—United States, July 2000-2001. *MMWR* 51:736-740, 2002.
3. Practice parameter: The management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology.* 48:581-585, 1997.
4. Adams, J. The neuropathology of head injuries. In: *Handbook of Clinical Neurology: Injuries of the Brain and Skull.* P. Vinken and G. Bruyn (Eds.). Amsterdam: North-Holland Publishing Co., pp. 35-65, 1975.
5. Arcan, M., M. Brull, T. Najenson, and P. Solzi. FGP assessment of postural disorders during the process of rehabilitation. *Scandinavian Journal of Rehabilitative Medicine.* 9:165-168, 1977.
6. Aubry, M., R. Cantu, J. Dvorak, et al. Summary and agreement statement of the First International Conference on Concussion in Sport, Vienna, 2001: recommendations for the improvement of safety and health of athletes who suffer concussive injuries. *British Journal of Sports Medicine.* 36:6-10, 2002.
7. Barth, J. T., W. M. Alves, T. V. Ryan, S. N. Macciocchi, R. W. Rimel, J. A. Jane, et al. (1989). Mild Head Injury in Sports: Neuropsychological Sequelae and Recovery of Function. *Mild Head Injury* (1st ed., pp. 274-293). New York: Oxford University Press.
8. Bigler, E. D., and W. W. Orrison. Neuroimaging in sports-related brain injury. In: *Traumatic brain injury in sports: An international neuropsychological perspective.* M. Lovell, Echemendia, R., Barth, J., Collins, M. (Ed.). Lisse: Psychology Press, 2004.
9. Bleiberg, J., A. Cernich, and D. Reeves. Sports concussion applications of the automated neuropsychological assessment metrics sports medicine battery. In: *Sports Neuropsychology: Assessment and management of traumatic brain injury.* R. J. Echemendia (Ed.). New York: Guilford, 2006.
10. Bleiberg, J., A. N. Cernich, K. Cameron, et al. Duration of cognitive impairment after sports concussion. *Neurosurgery.* 54:1073-1080, 2004.
11. Bleiberg, J., N. N. Cernich, K. L. Cameron, W. Sun, K. Peck, J. Uhorchak, et al. Duration of cognitive impairment following sports concussion. *Neurosurgery.* 54:1-6, 2004.
12. Bruce, J. M. An examination of the memory strategies employed by concussed athletes in a verbal recall test. Master's Thesis, Pennsylvania State University, 2001.
13. Cantu, R. Guidelines for return to contact sports after a cerebral concussion. *Phys Sports Med.* 14:75-83, 1986.
14. Cantu, R. C. Posttraumatic retrograde and anterograde amnesia: Pathophysiology and implications in grading and safe return to play. *J Athl Train.* 36:244-248, 2001.
15. Cantu, R. C. Return to play guidelines after a head injury. *Clin Sports Med.* 17:45-60, 1998.
16. Christman, C. W., M. S. Grady, S. A. Walker, K. L. Holloway, and J. T. Povlishock. Ultrastructural studies of diffuse axonal injury in humans. *J Neurotrauma.* 11:173-186, 1994.
17. Collie, A., P. McCrory, and M. Makdissi. Does history of concussion affect current cognitive status? *Br J Sports Med.* 40:550-551, 2006.
18. Collie A., P. Maruff, M. Makdissi, et al. CogSport: Reliability and correlation with conventional cognitive tests used in postconcussion medical evaluations. *Clinical Journal of Sport Medicine.* 13:28-32, 2003.

19. Collins, M. W., G. L. Iverson, M. R. Lovell, et al. On-field predictors of neuropsychological and symptoms deficit following sports-related concussion. *Clinical Journal of Sports Medicine*. 222-229, 2003.
20. Collins, M. W., M. R. Lovell, G. L. Iverson, R. C. Cantu, J. C. Maroon, and M. Field. Cumulative effects of concussion in high school athletes. *Neurosurgery*. 51:1175-1179; discussion 1180-1171, 2002.
21. Collins, M. W., S. H. Grindel, M. R. Lovell, D. E. Dede, D. J. Moser, B. R. Phalin, S. Nogle, M. Wasik, D. Cordry, M. K. Daugherty, S. F. Sears, G. Nicolette, P. Indelicato, D. B. McKeag. Relationship between concussion and neuropsychological performance in college football players. *Journal of the American Medical Association*. 282:964-970, 1999.
22. Echemendia, R. J. (Ed.). *Sports neuropsychology: Assessment and management of traumatic brain injury*. New York: Guilford Publications, 2006.
23. Echemendia, R. J. and R. C. Cantu. Return to play following sports-related mild traumatic brain injury: The role for neuropsychology. *Appl Neuropsychol*. 10:48-55, 2003.
24. Echemendia, R. J., M. Putukian, R. S. Mackin, et al. Neuropsychological test performance prior to and following sports-related mild traumatic brain injury. *Clinical Journal of Sport Medicine*. 11:23-31, 2001.
25. Erlanger, D., T. Kaushik, R. Cantu, J. T. Barth, D. K. Broshek, J. R. Freeman, and F. M. Webbe. Symptom-based assessment of the severity of a concussion. *J Neurosurg*. 98:477-484, 2003.
26. Field, M., M. W. Collins, M. R. Lovell, and J. Maroon. Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. *Journal of Pediatrics*. 546-553, 2003.
27. Gennarelli, T. A. Head injury in man and experimental animals: Clinical aspects. *Acta Neurochir Suppl (Wien)*. 32:1-13, 1983.
28. Gennarelli, T. A. and D. I. Graham. Neuropathology of the head injuries. *Semin Clin Neuropsychiatry*. 3:160-175, 1998.
29. Giza, C. C. and D. A. Hovda. The neurometabolic cascade of concussion. *J Athl Train*. 36:228-235, 2001.
30. Gronwall, D. and P. Wrightson. Cumulative effect of concussion. *The Lancet*. 306(7943):995-997, 1975.
31. Gronwall, D. and P. Wrightson. Delayed recovery of intellectual function after minor head injury. *The Lancet*. 304(7881):605-609, 1974.
32. Guskiewicz, K. M. Postural stability assessment following concussion: one piece of the puzzle. *Clinical Journal of Sport Medicine*. 11:182-189, 2001.
33. Guskiewicz, K. M., S. L. Bruce, R. C. Cantu, et al. National Athletic Trainer's Association position statement: Management of sport-related concussion. *Journal of Athletic Training*. 39:280-297, 2004.
34. Guskiewicz, K. M., S. W. Marshall, J. Bailes, M. McCrea, R. C. Cantu, C. Randolph, and B. D. Jordan. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurgery*. 57:719-726; discussion 719-726, 2005.
35. Guskiewicz, K. M., M. McCrea, S. W. Marshall, R. C. Cantu, C. Randolph, W. Barr, J. A. Onate, and J. P. Kelly. Cumulative effects associated with recurrent concussion in collegiate football players: The NCAA Concussion Study. *JAMA*, 290:2549-2555, 2003.
36. Guskiewicz, K. M. and D. Perrin. Effect of mild head injury on cognition and postural stability. *Journal of Athletic Training*. 33:S8, 1998.
37. Guskiewicz, K. M., D. H. Perrin, and B. M. Gansneder. Effect of mild head injury on postural stability in athletes. *Journal of Athletic Training*. 31:300-306, 1996.
38. Guskiewicz, K. M., D. Riemann, D. H. Perrin, and L. M. Nashner. Alternative approaches to the assessment of mild head injury in athletes. *Medicine and Science in Sports & Exercise*. 29:213-221, 1997.
39. Guskiewicz, K. M., S. E. Ross, and S. W. Marshall. Postural stability and neuropsychological deficits following concussion in collegiate athletes. *Journal of Athletic Training*. 36:263-273, 2001.
40. Guskiewicz, K. M., N. L. Weaver, D. A. Padua, and W. E. Garrett, Jr. Epidemiology of concussion in collegiate and high school football players. *Am J Sports Med*. 28:643-650, 2000.
41. Guskiewicz, K. M., S. W. Marshall, J. Bailes, M. McCrea, H. P. Harding, Jr., A. Matthews, J. R. M. Mihalik, and R. Cantu. Recurrent concussion and risk of depression in retired professional football players. *Medicine & Science in Sport & Exercise*. 39(6), 903-909, 2007.
42. Hodva, D. A., S. M. Lee, and M. L. Smith. The neurochemical and metabolic cascade following brain injury: Moving from animal models to man. *Journal of Neurotrauma*. 12:903-906, 1995.
43. Hovda, D. A., A. Yoshino, T. Kawamata, Y. Katayama, and D. P. Becker. Diffuse prolonged depression of cerebral oxidative metabolism following concussive brain injury in the rat: A cytochrome oxidase histochemistry study. *Brain Res*. 567:1-10, 1991.
44. Ingersoll, C. and C. Armstrong. The effects of closed-head injury on postural sway. *Medicine and Science in Sports and Exercise*. 24:739-742, 1992.
45. Iverson, G. L. Outcome from mild traumatic brain injury. *Curr Opin Psychiatry*. 18:301-317, 2005.
46. Jordan, B. D. Head injuries in sport. In: *Sports Neurology*. B. D. Jordan, P. Tsans, and R. Warren (Eds.). Rockville: Aspen Publishers, 1989.
47. Jordan, B. D. Sports Injuries. In *Mild Brain Injury Summit*. Dallas: NATA, Inc., pp. 43-46, 1994.
48. Kaushik, T. E. and D. M. Erlanger. The HeadMinder Concussion Resolution Index. In: *Sports Neuropsychology: Assessment and management of traumatic brain injury*. R. J. Echemendia (Ed.). New York: Guilford, 2006.
49. Kaut, K. P., R. DePompei, J. Kerr, and J. Congeni. Reports of head injury and symptom knowledge among college athletes: Implications for assessment and educational intervention. *Clin J Sport Med*. 13:213-221, 2003.
50. Kelly, J. P. Loss of Consciousness: Pathophysiology and implications in grading and safe return to play. *J Athl Train*. 36:249-252, 2001.
51. Kissick, J. and K. M. Johnston. Return to play after concussion: Principles and practice. *Clin J Sport Med*. 15:426-431, 2005.
52. Lovell, M. The IMPACT neuropsychological Test Battery. In: *Sports Neuropsychology: Assessment and management of traumatic brain injury*. R. J. Echemendia (Ed.). New York: Guilford Publications, 2006.
53. Lovell, M. R., M. W. Collins, G. L. Iverson, M. Field, J.C. Maroon, R. Cantu, K. Podell, J. W. Powell, M. Belza, and F. H. Fu. Recovery from mild concussion in high school athletes. *Journal of Neurosurgery*. 296-301, 2003.
54. Maroon, J. C., M. R. Lovell, J. Norwig, K. Podell, J. W. Powell, and R. Hartl. Cerebral concussion in athletes: Evaluation and neuropsychological testing. *Neurosurgery*. 47:659-669; discussion 669-672, 2000.
55. Mauritz, K., J. Dichgans, and A. Hufschmidt. Quantitative analysis of stance in late cortical cerebellar atrophy of the anterior lobe and other forms of cerebellar ataxia. *Brain*. 102:461-482, 1979.

56. McCrea, M., W. B. Barr, K. Guskiewicz, C. Randolph, S. W. Marshall, R. Cantu, J. A. Onate, and J. P. Kelly. Standard regression-based methods for measuring recovery after sport-related concussion. *J Int Neuropsychol Soc.* 11:58-69, 2005.
57. McCrea, M., K. M. Guskiewicz, S. W. Marshall, W. Barr, C. Randolph, R. C. Cantu, J. A. Onate, J. Yang, and J. P. Kelly. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. *JAMA.* 290:2556-2563, 2003.
58. McCrea, M., J. P. Kelly, and C. Randolph. Standardized Assessment of Concussion (SAC): *Manual for Administration, Scoring and Interpretation.* 2nd ed. Waukesha, WI, 2000
59. McCrea, M., J. P. Kelly, C. Randolph, et al. Immediate neurocognitive effects of concussion. *Neurosurgery.* 50:1032-1042, 2002.
60. McCrory, P. The eighth wonder of the world: The mythology of concussion management. *Br J Sports Med.* 33:136-137, 1999.
61. McCrory, P., K. Johnston, W. Meeuwisse, M. Aubry, R. Cantu, J. Dvorak, T. Graf-Baumann, M. Lovell, and P. Schamasch. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *British Journal of Sports Medicine.* 39:196-204, 2005.
62. McCrory, P., W. Meeuwisse, K. Johnston, J. Dvorak, M. Aubry, M. Molloy, R. Cantu. Consensus statement on concussion in sport 3rd International Conference on Concussion in Sport, Zurich 2008. *Clinical Journal of Sport Medicine.* 19(3):185-200, 2009.
63. McDonald, J. W. and M. V. Johnston. Physiological pathophysiological roles of excitatory amino acids during central nervous system development. *Brain Research Review.* 15:41-70, 1990.
64. Meyer, J. S., A. Kondo, F. Nomura, K. Sakamoto, and T. Teraura. Cerebral hemodynamics and metabolism following experimental head injury. *J Neurosurg.* 32:304-319, 1970.
65. Mitchell, D. and J. Adams. Primary focal impact damage to the brainstem in blunt head injuries. *The Lancet.* 2(7823):215-218, 1973.
66. Moser, R. S. and P. Schatz. Enduring effects of concussion in youth athletes. *Archives of Clinical Neuropsychology.* 17(1):91-100, 2002.
67. Nashner, L. Adaptation of human movement to altered environments. *Trends in Neuroscience.* 5:358-361, 1982.
68. Nashner, L. Adapting reflexes controlling the human posture. *Exploring Brain Research.* 26:59-72, 1976.
69. Nashner, L. and A. Berthoz. Visual contribution to rapid motor responses during postural control. *Brain Research.* 150:403-407, 1978.
70. Nashner, L., F. Black, and C. Wall. Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. *The Journal of Neuroscience.* 2:536-544, 1982.
71. Nilsson, B. and C. H. Nordstrom. Rate of cerebral energy consumption in concussive head injury in the rat. *J Neurosurg.* 47:274-281, 1977.
72. Pellman, E. J., D. C. Viano, I. R. Casson, C. Arfken, and J. Powell. Concussion in professional football: Injuries involving 7 or more days out—Part 5. *Neurosurgery.* 55:1100-1119, 2004.
73. Piland, S. G., R. W. Motl, M. S. Ferrara, and C. L. Peterson. Evidence for the factorial and construct validity of a Self-Report Concussion Symptoms Scale. *J Athl Train.* 38:104-112, 2003.
74. Povlishock, J. T., D. P. Becker, C. L. Cheng, and G. W. Vaughan. Axonal change in minor head injury. *J Neuropathol Exp Neurol.* 42:225-242, 1983.
75. Povlishock, J. T. and H. A. Kontos. Continuing axonal and vascular change following experimental brain trauma. *Cent Nerv Syst Trauma.* 2:285-298, 1985.
76. Povlishock, J. T. and H. A. Kontos. The role of oxygen radicals in the pathobiology of traumatic brain injury. *Hum Cell.* 5:345-353, 1992.
77. Powell, J. W. and K. D. Barber-Foss. Traumatic brain injury in high school athletes. *JAMA.* 282:958-963, 1999.
78. Prince, D. A., H. D. Lux, and E. Neher. Measurement of extracellular potassium activity in cat cortex. *Brain Res.* 50:489-495, 1973.
79. Riemann, B. and K. Guskiewicz. Objective assessment of mild head injury using a clinical battery of postural stability tests. *Journal of Athletic Training.* 35:19-25, 2000.
80. Riemann, B. L., K. M. Guskiewicz, and E. W. Shields. Relationship between clinical and forceplate measures of postural stability. *J Sport Rehabil.* 8:71-82, 1999.
81. Saunders, R. and R. Harbaugh. The second impact in catastrophic contact-sports head injury. *JAMA.* 252:538-539, 1984.
82. Schatz, P. and J. Browndyke. Applications of computer-based neuropsychological assessment. *Journal of Head Trauma Rehabilitation.* 17(5):395-410, 2002.
83. Schatz, P. and E. A. Zillmer. Computer-based assessment of sports-related concussion. *Applied Neuropsychology.* 10(1):42-47, 2003.
84. Shaw, N. A. The neurophysiology of concussion. *Prog Neurobiol.* 67:281-344, 2002.
85. Webbe, F. Definition, physiology, and severity of cerebral concussion. In: *Sports neuropsychology: Assessment and management of traumatic brain injury.* R. J. Echemendia (Ed.). New York: Guilford Publications, 2006.
86. Yaghai, A. and J. Povlishock. Traumatically induced reactive change as visualized through the use of monoclonal antibodies targeted to neurofilament subunits. *J Neuropathol Exp Neurol.* 51:158-176, 1992.