

The Clinical Management of Sports Concussion

Michael W. Collins, PhD, and Kristen L. Hawn, MA, ATC

Address

University of Pittsburgh Medical Center, Center for Sports Medicine,
Sports Concussion Program, 3200 South Water Street,
Pittsburgh, PA 15203, USA.

E-mail: collinsmw@msx.upmc.edu

Current Sports Medicine Reports 2002, 1:12–22

Current Science Inc. ISSN 1537-890x

Copyright © 2002 by Current Science Inc.

Concussion is among the hottest topics in sports medicine today. It is a highly individualized injury which oftentimes has a subtle presentation that is easily misdiagnosed, and therefore mishandled. When to return a concussed athlete to participation is a highly controversial topic that, to this point, has been primarily based on any one of 17 sets of guidelines. Neuropsychologic and cognitive testing has introduced a quantitative and sensitive measure that allows the clinician to verify an athlete's return to normal functioning levels. It is based on the firm belief that every concussion is a unique event, and must be handled as such. The realization that there is no one formula that can handle an injury this complex and multifaceted is perhaps the greatest breakthrough in mild traumatic brain injury research in the past decade.

Introduction

The American Academy of Neurology defines mild traumatic brain injury (MTBI), or concussion, as “a trauma-induced alteration in mental status that may or may not be accompanied by a loss of consciousness” [1]. Conservative numbers estimate that approximately 300,000 sports-related MTBIs occur every year [2]. In all likelihood, this is a gross underestimation, due to the profound variation in concussion definition and, therefore, diagnosis.

Concussion has gained recent national attention through the highly publicized cases of elite athletes, but those at greatest risk are likely to be high school and collegiate athletes, considering that a minimum of 1.5 million compete in football alone [3,4••]. There is typically less medical control over these populations, which makes the recognition and management of concussion difficult for a variety of reasons. First, athletes with concussion may present with a wide variety of symptoms, many of which are subtle, and may go unrecognized by an untrained professional, or even the athletes themselves. A

review of the common signs/symptoms of injury is presented in Table 1. A second concern is that many athletes may be reluctant to report concussive symptoms, due to the fear that they will be removed from the game, jeopardizing their status on the team, or their careers. Attempting to “play through” a concussion, however, could have long-term or even catastrophic consequences.

The focus of this review is the safe return of the head-injured athlete to full participation. At the offset, we review some basic concepts of concussion, as well as management concerns in the concussed athlete. We then briefly address the acute care of the athlete, and outline the current status of the field in making return-to-play decisions. Following this is a review of pertinent literature in several germane research areas that may help to elucidate the variable nature of this injury. Lastly, we discuss a more individualized protocol for concussion management, including neuropsychologic assessment and other ancillary quantitative methods. However, this approach to concussion management should not be misconstrued as yet another set of guidelines with broad application to all athletes and every concussion.

Three primary issues exist in the clinical management of the head-injured athlete. First, more serious intracranial pathology in the form of skull fracture, epidural hematoma, subdural hematoma, and parenchymal hematoma must be ruled out. Clinicians should be aware that major head injury may present with an initial lucid interval, followed by delayed neurologic deterioration [4••]. A rapid progression of headache, personality change, or mental status deterioration should alert the clinician to this concern, and facilitate emergent evaluation. A second management concern is the prevention of catastrophic outcome from second impact syndrome (SIS). This occurs when an athlete experiences a second concussive insult closely following a first. Both the initial as well as the second insult may be considered mild, with the second impact occurring up to 10 days after the first trauma. Most athletes who have succumbed to SIS have complained of discernable symptoms (headache, nausea, feeling slow) following the first insult and before the second [5]. The majority of documented cases have occurred in high school athletes, which may be secondary to specific neurodevelopmental issues, or possible selection bias (the largest athletic population is teenaged or younger). A final and much more prevalent management concern in the concussed athlete is the salient possibility of cumulative

Table 1. Frequently observed signs of concussion

Signs observed by medical staff
Athlete appears dazed
Vacant facial expression
Confusion about assignment
Athlete forgets plays
Disorientation to game, score, opposing team
Inappropriate emotional reaction (laughing, crying)
Athlete displays incoordination or clumsiness
Athlete is slow to answer questions
Loss of consciousness (even for seconds)
Any change in typical behavior or personality
Symptoms reported by athlete
Headache
Nausea
Balance problems or dizziness
Double or fuzzy vision
Sensitivity to light or noise
Feeling slowed down
Feeling "foggy" or "not sharp"
Change in sleep pattern
Concentration or memory problems
Irritability
Sadness
Feeling more emotional
Symptoms may worsen with exertion. Athlete should not return to play until symptom-free.

brain injury related to repeated traumas. Several neuropsychologic studies have identified the possibility of cognitive and neurobehavioral sequelae associated with two or more concussive events [6,7,8]. This has led to speculation that successive concussive impacts may lead to mild yet significant impairment of cognitive processes (eg, attention, memory), personality changes, and somatic disturbances (eg, sensitivity to light, dizziness). This collection of symptoms has been termed *postconcussion syndrome*, and can be quite incapacitating. It should be noted that discernable cognitive deficits have been identified in particular athletes from a single concussive event [9]. Significant risk factors have yet to be determined for these poor outcomes, although research efforts are currently underway.

In assessing traumatic brain injury, all clinicians should be aware that neurodiagnostic techniques such as computed tomography [CT] scans, magnetic resonance imaging [MRI] scans, and electroencephalogram [EEG], although invaluable in identifying more severe intracranial pathology, are typically insensitive to measuring the subtle neuronal aspects of MTBI [10]. Their false negatives often provide an errant basis on which return-to-play decisions are made. However, the advent of magnetization transfer imaging (MTR) [11], and the monitoring of brain electrophysiologic activity through event-related potentials (ERPs) [12,13], as well as the constant evolution of positron emission tomography (PET) scanning and functional (fMRI) [14••] technology, may be able to offer the future establishment of neurodiagnostic norms with the ability to accurately assess concussion severity [11,12].

Recent research into the subtle metabolic effects of concussion has triggered burgeoning insights into the pathophysiology of injury, which has fostered a greater understanding of the acute presentation of concussion and its implications. Utilizing a rodent model, Hovda *et al.* [15] have described a metabolic dysfunction that occurs when cells immediately injured upon concussive insult are exposed to dramatic evolving changes in both their intracellular and extracellular environments. These changes are the result of excitatory amino acid (EAA)-induced ionic shifts with increased Na/K ATPase activation, and resultant hyperglycolysis [16]. This process is accompanied by a decrease in cerebral blood flow that is not well understood. One theory explains it as the result of an accumulation of endothelial Ca⁺⁺, which is thought to cause widespread cerebral neurovascular constriction [15]. The resulting imbalance between increased energy requirements and its decreased availability has been postulated to propagate a cellular vulnerability that is particularly susceptible to even minor changes in cerebral blood flow, increases in intracranial pressure, and apnea [15]. Animal models have indicated that this dysfunction can last up to 10 days, whereas cerebral hyperglycolysis has been observed in head-injured humans for as long as 2 weeks, with a subsequent metabolic depression that was present for over 1 year [15,16]. Extrapolating this theory of metabolic dysfunction to humans is still premature, but it raises many pertinent questions regarding the threat of vulnerability, how long it lasts, and if it is accompanied by identifiable markers of both injury and recovery.

Presently there are no curative medical treatments for concussion. This renders early recognition of postconcussive symptoms and the prevention of additional concussive injury a crucial precautionary step. However, tremendous controversy reigns among sports medicine professionals regarding concussion assessment, diagnosis, and management. To date, no consensus has been achieved.

Acute Management of Concussion

Appropriate acute care of the concussed athlete begins with an accurate assessment of the gravity of the situation. As with any serious injury assessment, the first priority is always to evaluate the athlete's level of consciousness and ABCs (airway, breathing, and circulation). The attending medical staff must always be prepared with an emergency action plan in the event that the evacuation of a critically head- or neck-injured athlete is necessary. This plan should be familiar to all staff, be well delineated, and frequently rehearsed.

Upon ruling out more severe injury, the acute evaluation continues with an assessment of concussion. The clinician should establish the presence of any loss of consciousness and amnesia, in addition to verifying orientation to person, place, and time. Acute assessment of the athlete's verbal memory (eg, three words at 0 and 5 minutes) will help to delineate the potential presence of anterograde amnesia.

Table 2. Mental status testing*

Cognitive testing
Orientation
Ask the athlete the following questions:
What stadium is this?
What city is this?
Who is the opposing team?
What month is it?
What day is it?
Anterograde amnesia
Ask the athlete to repeat the following words:
Girl, dog, green
Retrograde amnesia
Ask the athlete the following questions:
What happened in the prior quarter/period?
What do you remember just prior to the hit?
What was the score of the game prior to the hit?
Do you remember the hit?
Concentration
Ask the athlete to do the following:
Repeat the days of the week backward, starting with today
Repeat these numbers backward: 63, 419
Word list memory
Ask the athlete to repeat the three words from earlier (girl, dog, green)

Any failure should be considered abnormal. Consult a physician if the athlete exhibits any signs or symptoms.

*The University of Pittsburgh Medical Center Concussion Card is a brief mental status evaluation and symptom inventory intended for sideline and athletic training room use immediately following injury. Its intended purpose is to assist the clinician in determining the presence of concussive injury, and is not for use in making safe return to competition decisions.

Retrograde amnesia may be accurately assessed by asking about the athlete's memory of the traumatic event itself, and the events just preceding it (eg, score of the game, opponent, and a description of the incident itself). Anterograde amnesia is considered to be a much more common occurrence, and typically lasts much longer in duration than retrograde amnesia. Identifying the common signs and symptoms of concussion (Table 1), and performing appropriate mental status exams, such as those outlined in the University of Pittsburgh Medical Center Concussion Card (Table 2), may help to establish an accurate overall picture of acute concussion. However, its presentation is diverse and often subtle. Relying solely on the subjective response of the athlete can be dangerous, because athletes may be afraid to lose their position or endanger their career by admitting how poorly they are feeling. Any signs or symptoms reported by the athlete or observed by the clinician throughout the evaluation should preclude return to play, as well as signal the necessity of a more comprehensive examination. From this point on, serial review of the athlete, in addition to careful observance for latent signs and symptoms, is critical.

Another sideline assessment tool proven effective in diagnosing concussion is the Standardized Assessment of Concussion (SAC) [17]. This standardized mental status exam measures gross aspects of orientation and memory. It also includes a brief neurologic exam. These are abbreviated versions of more extensive exams, and although they

are useful in the initial diagnosis of concussion, they should not be used in place of more comprehensive testing when making return to participation decisions. As outlined by McCrea *et al.* [17], the SAC has been found to accurately separate concussed athletes and controls within 24 hours of injury. Such mental status testing may be useful in detecting overt cognitive deficits, and supply immediate information to athletic trainers and other medical personnel who are responsible for immediate, on-field clinical decision-making.

Current Grading Systems and Return-to-play Guidelines

Once the diagnosis of concussion is delineated, making the postinjury decision to return an athlete to participating is one of the major challenges of concussion management. How can you know when they have fully recovered and are ready to safely return? At present, there are at least 17 disparate concussion scales constructed for the purpose of grading the severity of concussion and ultimately determining return to play (Table 3). In addition to those in listed in Table 3, the American Orthopedic Society for Sports Medicine (AOSSM) has set forth generalized recommendations for concussion management, which are displayed in Table 4 [18•]. The contributions that have been made by the concussion guidelines, in terms of

increased awareness and the use of uniform terminology in describing signs and symptoms, has dramatically improved concussion management. However, their lack of an empiric foundation is too critical to overlook. There are no current prospective data available to validate any one existing guideline. Most were primarily developed by panels of experts in the field, and are based on popular belief or practice rather than empiric evidence [10,19•]. This has yielded widely variant guidelines that have no consensus, and assume the same standards for all age groups. Clearly, this lack of scientific evidence has resulted in these many disparate grading systems, as well as disjointed diagnosis and management of the injury.

Each scale listed in Table 3 relies heavily on loss of consciousness (LOC) and amnesia for classification of severity. Recent research has shown that there may be no discernable correlation between brief LOC and severity of concussion outcome [12,18•,20•,21,22]. For example, in a retrospective analysis of trauma patients, Lovell *et al.* [20•] found no significant differences between victims who had no LOC, definite LOC, and uncertain LOC. More recent work with athletes has found no differences in acute recovery from concussion in those experiencing brief LOC and no LOC [9]. Importantly, until further research is completed, more extended LOC (typically defined as > 1 minute) must be seen as pathognomonic and should warrant emergent neurologic evaluation. To date, no individual sign or symptom of concern (*eg*, headache, anterograde amnesia, retrograde amnesia, balance problems) has been proven to directly correlate with concussion severity. There is speculation that retrograde amnesia may be a better indicator of poor outcome, though as of yet this too is unproven.

In short, concussion management guidelines do not appear to have scientifically evolved to the extent that they can be relied upon to make accurate and safe return to participation decisions. Further, the grading procedures for concussion are too varied, and result in communication difficulties among clinicians. This lack of uniformity and resulting management confusion speaks clearly to the fact that concussion is not a unitary phenomenon. It may present differently across athletes due to a myriad of factors. Since the advent of neuropsychologic testing, and more individualized management of the injury, prospective data are emerging that begin to shed light on individual factors that may play a role in both the incidence and severity of concussion. Traditional neurologic literature and recent sports-specific literature highlights the variable nature of this injury.

Demographic and Premorbid Factors

Gender

The increased participation of women in organized athletics has raised the issue of whether there is a gender influence on concussion incidence and severity. To date, very little research has specifically examined gender differ-

ences in MTBI. The majority of published literature has focused on nonathletic populations (*eg*, accident victims) and rodents. For example, in their work with trauma victims, Clinchot *et al.* [23] report an increased likelihood that women with MTBI will report sleep disturbances and will headaches up to 1 year after injury. In addition, the authors noted that women may be less likely than men to be employed or in school 1 year after mild head injury [23]. Another nonathletic study reviewed the effects of MTBI on collegiate academic performance [24]. Findings showed that after MTBI, women suffered a significant decrease in grade point average as compared with controls, whereas there were no similar findings for men [24]. In a study examining patients seen in the emergency department following MTBI, Bazarian *et al.* [25] found that women were more likely than men to suffer postconcussive symptoms at 1, 3, and 6 months following their injury. However, these differences only reached statistical significance at 3 months, which may have been reflective of the differences in injury mechanism (most injuries in women were due to motor vehicle accidents, whereas most of the injuries in men were the result of sports activities). Even when controlling for demographic, premorbid, and event-related factors, most research to date has shown that women have worse outcomes than men after MTBI. A recent meta-analysis that included eight studies and 20 outcome variables revealed that across 85% of those variables, outcome was again worse in women [26].

Although very little research has examined gender differences in concussion among athletes, several recent studies have begun to emerge. In a sample of elite soccer players, Barnes *et al.* [27] retrospectively demonstrated that males suffer concussions of greater severity, and are subject to a higher incidence of injury. Boden *et al.* [28] performed a prospective study involving 15 National Collegiate Athletic Association men's and women's soccer teams over two seasons that yielded very similar results in terms of incidence, but did not delineate severity in terms of gender.

Even though most of the literature has reported increased symptoms and poorer outcome among women who have experienced head injury, recent animal models have suggested that female sex hormones may actually play a neuroprotective role in the brain after concussive injury [29,30]. Progesterone is thought to reduce cerebral edema and potentially facilitate cognitive recovery [29], although studies of estrogen influence have yielded mixed results. One study has shown estrogen to play a protective role in males, and increase mortality in females [31]. Other research has demonstrated that estrogen can assist in maintaining normal cerebral blood flow, and actually decrease mortality when administered acutely [30]. All of the aforementioned research suggests that gender may have some measure of clinical significance in the incidence and severity of MTBI. More research in this area is necessary to accurately describe the implications of such differences.

Table 3. Current concussion grading systems

Grade	0	1	2	3	4	5	6
Colorado	No LOC, confusion, no amnesia, RTP after 20 min if normal exam	No LOC, confusion, amnesia, no RTP that day	LOC, transport to medical facility emergently, neurosurgical evaluation	LOC > 5 min, transport to medical facility emergently, neurosurgical evaluation or PTA > 24 h	LOC (brief or prolonged)		
Cantu	Normal consciousness, no LOC or PTA < 30 min, observe on sidelines, RTP in selected circumstances	LOC < 5 min or PTA 30 min–24 h, no RTP, evaluate in medical facility	LOC > 5 min, transport to medical facility emergently, neurosurgical evaluation or PTA > 24 h	LOC (brief or prolonged)			
AAN	Transient confusion, no LOC, confusion symptoms or mental status change resolves in < 15 min	Transient confusion, no LOC, confusion symptoms or mental status change lasts > 15 min	LOC > 5 min, transport to medical facility emergently, neurosurgical evaluation or PTA > 24 h	LOC (brief or prolonged)			
Roberts	Bell ringer, no LOC, no PTA, RTP after 30 min if symptoms clear < 10 min	No LOC or PTA < 30 min, observe on sidelines, no RTP that day	LOC < 5 min or PTA 30 min–24 h, no RTP that day, often evaluate in medical facility	LOC > 5 min, transport to medical facility emergently, neurosurgical evaluation or PTA > 24 h			
Jordan	No LOC, confusion, no amnesia, RTP after 20 min of observation	No LOC, confusion with amnesia < 24 h, RTP when asymptomatic for 1 wk	LOC not exceeding 2–3 min, PTA > 24 h, RTP after 1 wk	LOC > 2–3 min, RTP not for 1 mo, RTP if asymptomatic for 2 wk			
Glasgow Scale	Minor head injury: 13–15 pts	Minor head injury: 13–15 pts	Moderate head injury: 9–12 pts	Moderate head injury: 9–12 pts; severe head injury: 8 pts or less	Severe head injury: 8 pts or less	Severe head injury: 8 pts or less	Severe head injury: 8 pts or less
Torg	Bell rung, short-term confusion, momentary LOC, dazed appearance, unsteady gait	Vertigo, PTA	Veritigo, PTA, retrograde amnesia	PTA, immediate transient LOC	Paralytic coma, cardiorespiratory arrest	Death	

AAN—American Academy of Neurology; c/o—complains of; LOC—loss of consciousness; PTA—post-traumatic amnesia; RTP—return to play; WNL—within normal limits. (From Johnson R, Personal correspondence. American Medical Society for Sports Medicine; with permission.)

Table 3. Current concussion grading systems (Continued)

Grade	0	1	2	3	4	5	6
Ommaya		Confusion, normal consciousness, no amnesia	Confusion, confusion & amnesia, normal consciousness, PTA amnesia only	Coma < 8 h, PTA amnesia, retrograde amnesia	Coma 6–24 h	Coma > 24 h	Coma, death within 24 h
Ommaya & Gannarelli		Confusion, normal consciousness, no amnesia	Confusion, confusion & amnesia, normal consciousness, PTA only	Normal consciousness, confusion & amnesia, PTA, retrograde amnesia	Coma (paralytic), awoken with confusion & amnesia	Coma > 24 h, persistent vegetative state	Coma, death
Hughenoltz & Richard Saal		Transient or no LOC, PTA < 1h	LOC < 5 min, PTA 1–24 h	LOC > 5 min, PTA > 24 h	LOC 5–10 min, retrograde & possible PTA, transport to medical facility, RTP after neurosurgical clearance	LOC not arousable, Transport to hospital emergently, notify hospital for neurosurgical care	
Nelson	Head struck or moved rapidly, not stunned, later c/o headache & difficulty concentrating	Normal consciousness, no LOC, mild headache, no amnesia, steady on feet, RTP if symptoms clear	Momentary LOC, unsteady gait, headache, no amnesia, RTP after 30 min if symptoms clear	3A: No or momentary LOC, no PTA, observe; 3B: brief LOC, retrograde & PTA, no play, RTP WNL	LOC > 1 min, not comatose, demonstrate grade 2 symptoms in recovery		
Kulund		Stunned, dazed, no confusion, dizziness, no nausea, visual disturbance, feels well after 1–2 min	LOC, mental confusion, tinnitus, dizziness, retrograde amnesia, skill recovery may be rapid	Longer LOC, headache, confusion, PTA amnesia, retrograde amnesia			
Gersoff		Confusion, normal consciousness, no amnesia	Confusion, PTA only, normal consciousness	Normal consciousness, confusion, PTA and retrograde amnesia	LOC, coma (paralytic), awoken with confusion & amnesia	Coma, possible cardiorespiratory collapse, possible persistent vegetative state, possible death	Coma, death
Wilberger & Maroon		Short-term confusion, minimal or no LOC, PTA 15–20 min	Confusion, PTA > 20 min, LOC < 5 min	LOC > 5 min, severe headache, PTA > 12 h			

AAN—American Academy of Neurology; c/o—complains of; LOC—loss of consciousness; PTA—post-traumatic amnesia; RTP—return to play; WNL—within normal limits. (From Johnson R, Personal correspondence. American Medical Society for Sports Medicine; with permission.)

Table 4. AOSSM return to play guidelines

Return to play (same day)	
Signs and symptoms cleared within 15 min or less, both at rest and exertion	
Normal neurologic evaluation	
No documented LOC	
Return to play (not the same day)	
Signs and symptoms did not clear in 15 minutes at rest or with exertion	
Documented LOC	
AOSSM—American Orthopedic Association for Sports Medicine; LOC—loss of consciousness.	

Age

Just as there has been an increase in female athletic participation in recent years, there has also been greater participation among younger populations. Again, it is noteworthy that current concussion guidelines assume identical return-to-play criteria for athletes regardless of age. Unfortunately, no systematic research has been conducted to specifically examine the recovery of function in the high school age and younger populations, even though they represent the majority of at-risk athletes. However, recent research by Collins [9] has shown that based on neuropsychologic test results, high school athletes may recover more slowly than their collegiate counterparts. The authors corroborate these results with previous studies in which significant postconcussion differences were shown between high school and college athletes on standard neuropsychologic tests [6•]. Age should be an element of particular concern in light of at least 17 documented deaths related to SIS, the majority of which occurred in athletes between the ages of 13 and 18 [5].

Although research specific to age-related differences in concussion outcome is scant, several theories have emerged. One such theory is that children may undergo more prolonged and diffuse cerebral swelling after MTBI, which suggests that they may be at an increased risk for secondary intracranial hypertension and ischemia. This may also lead to a longer recovery period, and could increase the likelihood of permanent or severe neurologic deficit should reinjury occur during the recovery period [32,33]. Another hypothesis is that the immature brain may be potentially 60 times more sensitive to glutamate mediated *N*-methyl-D-aspartate (NMDA) excitotoxic brain injury [34]. This hypersensitivity may render the child or adolescent more susceptible to the ischemic and injurious effects of EAA after MTBI. Alternatively, the popular concept of cortical plasticity suggests that younger athletes should make a better recovery than that of their older counterparts. There has been clinical evidence of marked synaptic excess in children, relative to adults, which allows for neural pathway rerouting during recovery and functional plasticity in the developing brain [35]. As time is not addressed in this theory, it is intuitive to assume that a more complete recovery is possible due to the described plasticity, although it may take a longer period of time. Lon-

gitudinal and prospective studies examining the effects of age on sports concussion outcome are currently underway and, in time, may elucidate this important clinical consideration.

Learning disability

Learning disability (LD) refers to a heterogeneous group of disorders revealed by difficulties in the acquisition and use of listening, speaking, writing, reading, reasoning, or mathematical abilities and which is traditionally diagnosed in early childhood [6•]. Prior history of LD has been linked to lower baseline cognitive performance within a large, multiuniversity sample of football players [6•]. In conjunction with multiple concussions, LD yielded lowered overall functioning in learning-disabled football players, suggesting a potential additive effect. Identifying these athletes prior to their participation may be important, because the diagnosis of concussion in a learning-disabled athlete is typically more difficult [4••]. As well, these athletes are often susceptible to having a diminished cognitive reserve that may lead to deleterious implications in returning these athletes to participation in collision sports.

Prior concussion history

Another important and obvious outcome consideration is the presence of prior concussive injury. There is a growing body of evidence that suggests there may be cumulative detrimental effects of multiple concussions [4,6–8]. This has typically been associated with the neuropsychologic impairment and neurologic abnormalities that have been primarily documented among boxers [8,36,37]. However, even when looking at a nonathletic population, Gronwall and Wrightson [7] found that patients presenting after their second or third MTBI were more severely affected on tests of information processing capacity than those without prior injury. Lately, the topic of cumulative effects of concussion has been of rising concern among athletic populations. In a study of almost 400 college football players, Collins *et al.* [6•] have shown long-term mild deficits in executive functioning and speed of information processing in athletes suffering two or more concussions. As well, Matser *et al.* [8] hypothesize that cumulative long-term consequences of repetitive blows to the head are being seen in professional soccer players. Currently, there are no good prospective studies of cumulative concussive injury. Clearly, prospective research is indicated in this area as well. At this point in time, there are simply no reliable data available to determine how many concussions should preclude return to participation.

Although many clinicians have begun to recognize the potential long-term deleterious effects of multiple concussions, there is less acceptance of the theory that a distinct subset of athletes appear to suffer particularly problematic responses to initial injury that worsen with each successive concussion. In clinical experience, my colleagues and I have seen many such cases, oftentimes resulting from seemingly minor incidents. Unfortunately, predicting which athletes are

susceptible to such outcomes is not yet possible. Many factors, such as demographics, environment, and premorbid functioning may attribute to individual outcome.

Recent research linking Alzheimer's disease (AD) and history of head injury has raised the question of whether there may be a genetically determined susceptibility to poor outcome. Given the similarities between chronic traumatic brain injury (CTBI) or dementia pugilistica and AD, Jordan [37] examined the question of a potential genetic predisposition to the effects of concussion in boxers. The authors considered the connection between boxers who possessed a higher CTBI score (measure of neurologic impairment) and presence of the apolipoprotein E (ApoE) $\epsilon 4$ genotype. Their findings suggest that the ApoE $\epsilon 4$ genotype may be linked to higher incidence of severe neurologic deficits in boxers with extensive exposure to their sport [38]. Friedman *et al.* [39] corroborated these findings in a nonathletic population, with results that indicate a strong association between the ApoE $\epsilon 4$ allele and poor clinical outcome in patients with MTBI, implying the potential for genetic predisposition to the effects of concussion [39]. Teasdale *et al.* [40] also conducted a study among nonathletes that showed significant genetic association with the ApoE $\epsilon 4$ allele and MTBI outcome, supporting the theory of a genetic influence.

In theory, presence of the ApoE $\epsilon 4$ allele discourages the growth and branching of neurites, and enhances aggregation of A β (the main component of neuritic plaques) [36,39,41]. The appearance of plaques is the most important determinant of susceptibility to AD, making possession of the ApoE $\epsilon 4$ allele in combination with a history of head injury a major potential risk factor in late-onset AD [36,40,41]. This raises the suggestion that there may be a potential genetic predisposition to poor concussion outcome. However, these findings must be interpreted with caution until further research is able to more precisely define the hypothesized relationship.

Approaches to Individualized Management

Neuropsychologic testing

There is no predictable response to head injury. The extensive variability in the profile of each concussed athlete makes uniform grading scales a potentially inadequate measure by which to determine readiness to return to participation. As such, a more individualized and data-driven approach to concussion management is likely to be more reasonable and prudent. At the forefront of such approaches is neuropsychologic testing.

Neurocognitive deficits resulting from concussion have been widely documented [6,8,42,43]. Many studies over the past 20 years have reinforced the ability of neuropsychologic evaluation to reveal ongoing and potentially cumulative difficulties commonly associated with MTBI [6,8,42–45]. Barth *et al.* [42] were among the first to use a large-scale, prospective neuropsychologic testing approach

among an athletic population. Over the course of the study, the authors retested approximately 200 primarily Ivy League football players suffering concussion who had received a baseline neuropsychologic evaluation. Results demonstrated deficits with attention and working memory that appeared to resolve around day 5 postinjury. Their results demonstrated that the effects of concussion can be aptly assessed by a neuropsychologic testing approach. In a similar prospective study (*ie*, baseline, postinjury evaluation), Collins *et al.* [6] revealed that cognitive deficits (*eg*, memory, concentration) persisted until at least day 5 postinjury, relative to noninjured controls, in a subset of concussed college football players. Macciocchi *et al.* [43], using data from the original Barth *et al.* [42] study, compared neuropsychologic testing and symptom scores of control subjects to those of 183 concussed collegiate football athletes. Their results showed impaired performance and increased symptomatology among the concussed athletes that resolved within 5 days for most participants. A retrospective study by Matser *et al.* [8] evaluated neuropsychologic test data for 33 amateur soccer players, and compared results with that of track and swimming athletes. Results from this study revealed moderate to severe impairment in both memory and planning in the soccer population, as compared with the control athletes. Taken together, these studies suggest that neuropsychologic evaluation is a sensitive tool that may be utilized to assess the often subtle and potentially debilitating effects of concussive injury. Neuropsychologic test data appear to provide objective, quantifiable, and individualized standards to better determine safe return to participation and overall management of the concussed athlete.

Neuropsychologic testing protocol

Neuropsychologic assessment has been rooted in more traditional neurologic populations (*eg*, AD, general TBI, seizure disorder) for decades. Standard evaluations for these populations involve a clinical interview and administration of paper and pencil tests by a trained neuropsychologist that may take up to 4 or 5 hours to complete. Because traditional neuropsychologic evaluation techniques are obviously impractical when faced with the testing of entire athletic teams, a screening approach is typically undertaken. This approach, described in detail elsewhere [10,46], is geared toward assessing those domains of cognitive functioning that are most likely to be affected by concussion, such as attention/concentration, memory, information processing speed, and motor speed or coordination. Traditional sports-specific neuropsychologic test batteries have been proven to be both valid and reliable measures to delineate the subtle effects of concussive injury [43,47,48]. Their resistance to practice effects are of particular importance, as testing is most often serial in nature [45].

Ideally, a neuropsychologic testing approach in athletic populations will involve pre- and postconcussion evalua-

tions. The preseason test, or baseline, provides a basis for comparison if the athlete is later concussed. This is important for several reasons. First, each athlete's level of performance will likely vary considerably on the test battery. Without assessing preinjury levels, it is difficult to determine whether postinjury deficits are truly attributable to concussion. As well, some athletes may perform more poorly due to individual discrepancies in cognitive function, learning disabilities, or related concerns. Lastly, prior concussions may mediate the effects of cognitive function, and cloud the assessment of postconcussive test results if a baseline test has not been obtained.

Once a concussion has occurred, follow-up testing should ideally be conducted within 24 to 48 hours of injury, and again at approximately 5 to 7 days postinjury (or subsequently in the more severely injured athlete). Unlike the previously discussed neurodiagnostic measures, which provide information on brain structure, neuropsychologic testing is able to provide clinicians with a measure of functional status. In combination with an individual preseason baseline, or when compared with a database of matched controls, postinjury neuropsychologic assessment may offer a tangible evidenced-based determination of when an athlete is able to return to participation [10,45,46].

Several paper and pencil neuropsychologic test batteries are currently being utilized in the National Football League, National Hockey League, and in several collegiate programs across the country. These systematic programs have been highly successful and, without exception, have made extensive contributions to the understanding of concussion assessment, diagnosis, and management. However, such paper and pencil testing protocols require a qualified neuropsychologist to administer both the preseason and follow-up exams. As well, athletes need to be tested individually for up to 30 minutes per evaluation. As a result, the widespread implementation of paper and pencil neuropsychologic testing has been somewhat limited, because the technology is not readily available to the general high school and collegiate sport populations.

Realizing these concerns, a major recent advancement in concussion diagnosis and management has been the development and implementation of computerized neuropsychologic assessment. At the forefront of this technology is the advent of ImpACT (Immediate Post-concussion Assessment and Cognitive Testing) [49,50], an approximate 25-minute computerized battery of neuropsychologic tests prepared specifically for use in athletic concussion. This technology has made neuropsychologic evaluation a practical option for athletes at all levels. Such an approach has addressed many of the drawbacks of traditional neuropsychologic testing for large groups of athletes, such as prohibitive costs, lengthy testing procedures, and practice or learning effects. Specifically, ImpACT can be effectively administered to groups of athletes (by networking the software into computer labora-

tories) by an athletic trainer or team physician who has received minimal instruction. Learning effects are significantly reduced by a test battery consisting of a nearly infinite number of alternate forms by randomly varying the stimulus array with each test module. ImpACT contains a self-report symptom scale, and is also able to measure reaction time reliably to one thousandth of a second across individual test modules. Consultation and interpretation by a qualified neuropsychologist is advised following postinjury evaluation, though is also streamlined, because data can easily be faxed or emailed. At the current time, ImpACT is being utilized for concussion management by approximately 200 high school, college, and professional athletic programs nationally and internationally.

Although neuropsychologic testing currently appears to be the gold standard in concussion management, there are other ancillary measures that can be beneficial in the diagnosis and evaluation of concussion. Guskiewicz *et al.* [51] utilized the SMART Balance Master (NeuroCom International, Clackamas, OR) to test for postural instability after mild head injury, in an attempt to set the precedence for establishing recovery curves based on objective data. Their study revealed that concussed athletes exhibited increased postural instability for the first 3 days following injury. They concluded that this was due to a sensory interaction problem that caused the injured athlete to fail to use their visual system correctly. Balance testing or postural stability has recently been a popular topic among some clinicians, but current research in this area has been conducted with small sample sizes, and has yet to be confirmed with larger groups of athletes [51–53].

Conclusions

This article has attempted to elucidate recent research in concussion etiology, presentation, outcome, and return to participation management parameters. Based upon existing studies, it is clear that the effects of concussion may be highly variable and dependent upon a myriad of factors. Again, the realization that there is currently no one formula or guideline that can safely manage an injury as complex and multifaceted as concussion is perhaps the greatest breakthrough in research over the past decade. It is highly unlikely that a single set of parameters will ever be able to accomplish such a task. Therefore, it is our desire to offer the clinician a more individualized and comprehensive approach to concussion management. To that end, we identify three phases of concussive injury management: preseason baseline neuropsychologic evaluation, on-field evaluation, and postinjury assessment.

An optimal systematic concussion management program begins with a baseline neuropsychologic evaluation prior to the athletic season for those athletes at high risk of sustaining concussion (*eg*, football, hockey, soccer). It should be noted that one baseline for a high school, college, or professional athlete will likely serve for the

athlete's tenure within that level of competition. Once contact participation begins, all athletes suspected of suffering a concussion should be administered simple sideline mental status examinations (*ie*, SAC, University of Pittsburgh Medical Center On-Field Concussion Evaluation) to identify overt cognitive deficits, or subtle signs/symptoms of injury. During the on-field examination, any overt neurologic/cognitive deficit or single sign or symptom of injury (*eg*, headache, confusion, balance problems, personality change) should clearly preclude return to participation for that contest. If there is resolution of all signs and symptoms on serial sideline exam, as well as on exertion, returning the athlete to active participation that day may be a viable option. Serial evaluation and assessment of the athlete's status during the contest is indicated, especially because the presentation of concussion sequelae may be an evolving process. Subsequently, if the athlete is withheld from competition and severe intracranial pathology is ruled out, postinjury assessment in the form of neuropsychologic evaluation may be prudent to help determine overall management and return to participation issues (even for those athletes who were initially cleared to play). At the current time, prevailing standards of care would suggest that an athlete must exhibit intact cognitive function in addition to a symptom free presentation before return to participation is considered. This status can be determined by comprehensive neuropsychologic evaluation (with a symptom inventory), and based upon return to baseline cognitive levels or intact functioning, as compared with normative values. If both conditions are adequately met, return to graded exertion prior to contact participation is likely prudent, as postconcussion difficulties may evolve with increased cerebral blood flow. Clearly, considerations according to prior history of concussion incidence and outcome may dictate return to participation and management directives.

The study of sports concussion and management is rapidly evolving. The realization that the same seemingly minor mechanism of injury may result in one athlete being out for 2 days and another for 2 or more months, underscores the importance of an individualized, evidence-based approach to concussion management. In light of these issues, utilization of the outlined neuropsychologic testing approach can provide the clinician with an individual basis for comparison, and produce evidence of an athlete's level of cognitive functioning. This is currently the clearest verification for when an athlete can safely resume participation. Continued refinement of this and other post-concussion management approaches, in light of evolving research, is the next step in the evolutionary process of sports-concussion management.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
 - Of major importance
1. Quality Standards Subcommittee, American Academy of Neurology: **Practice Parameter: the management of concussion in sports.** *Neurology* 1997, 48:581–585.
 2. Collins MW, Grindel SH, McKeag DB: **Concussion in US football players.** *The "Head Injured" Athlete. American Medical Society for Sports Medicine. XXVI FIMS World Congress of Sports Medicine.* Orlando, FL; June 3, 1998.
 3. Lovell MR, Collins MW: **Neuropsychological assessment of the head-injured professional athlete.** In *Neurological Sports Medicine.* Edited by Bailes JE, Day AL. Rolling Meadows: American Association of Neurological Surgeons; 2001:169–179.
 4. •• Bailes JE, Cantu RC: **Head injury in athletes.** *Neurosurgery.* 2001, 48:26–46.

This may be the most comprehensive and well-written review of concussion in the literature.

5. Cantu R, Voy R: **Second impact syndrome: a risk in any sport.** *Physician Sportsmed* 1995, 23:27–36.
6. • Collins M, Grindel S, Lovell M, *et al.*: **Relationship between concussion and neuropsychological performance in college football players.** *JAMA* 1999, 282:964–970.

This is important prospective research involving the pre- and post-neuropsychologic testing of concussed collegiate football players. It highlights a potential link between concussion and learning disability in reduced cognitive functioning.

7. Gronwall D, Wrightson P: **Cumulative effects of concussion.** *Lancet* 1975, 2:995–997.
8. Matser E, Kessels A, Lezak M, *et al.*: **Neuropsychological impairment in amateur soccer players.** *JAMA* 1999, 282:971–973.
9. Collins MW: **Concussion assessment in high school and college athletes: the ImPACT System.** *Proceedings from New Developments in Sports-Related Concussion.* Pittsburgh, PA; July 27–29, 2001.
10. Echemendia RJ, Putukian M, Mackin RS, *et al.*: **Neuropsychological test performance prior to and following sports-related mild traumatic brain injury.** *Clin J Sport Med* 2001, 11:23–31.
11. McGowan JC, Yang JH, Plotkin RC, *et al.*: **Magnetization transfer imaging in the detection of injury associated with mild head trauma.** *Am J Neuroradiol* 2000, 21:875–880.
12. Johnston KM, Lassonde M, Pfito A: **A contemporary neurosurgical approach to sport-related head injury: the McGill Concussion Protocol.** *J Am Coll Surg* 2001, 192:515–524.
13. Dupuis F, Johnston KM, Lavoie M, *et al.*: **Concussions in athletes produce brain dysfunction as revealed by event-related potentials.** *Neuroreport* 2000, 11:4087–4092.
14. •• McAllister TW, Saykin AJ, Flashman LA, *et al.*: **Brain activation during working memory 1 month after mild traumatic brain injury.** *Neurology* 1999, 53:130.

This is groundbreaking research utilizing fMRI in the assessment of regional patterns of brain activity that occur in response to varying degrees of working memory demands, shortly after mild traumatic brain injury.

15. Hovda DA, Prins M, Becker DP, *et al.*: **Neurobiology of concussion.** In *Sports-related Concussion.* Edited by Bailes JE, Lovell MR, Maroon JC. St. Louis: Quality Medical Publishing; 1999:12–51.
16. Bergsneider M, Hovda DA, Shalmon E, *et al.*: **Cerebral hyperglycolysis following severe human traumatic brain injury: a positron emission tomography study.** *J Neurosurg* 1997, 86:241–251.
17. McCrea M, Kelly JP, Randolph C, *et al.*: **Standardized assessment of concussion (SAC): on-site mental and status evaluation of the athlete.** *J Head Trauma Rehab* 1998, 13:27–35.

18. • Wojtyś ED, Hovda D, Landry G, *et al.*: **Concussion in sports.** *Am J Sports Med* 1999, 27:676–686.
This is a comprehensive work detailing all aspects of concussion management resulting from the Concussion Workshop, a gathering of experts in all areas of sports medicine and neurohealth, sponsored by the American Orthopedic Society for Sports Medicine.
19. • Collins M, Lovell M, McKeag D: **Current issues in managing sports-related concussion.** *JAMA* 1999, 282:2283–2285.
This paper thoroughly reviews the current status of the field while making a strong case, with the support of case examples, for necessary changes toward the use of a more individualized concussion-management approach.
20. • Lovell MR, Iverson GL, Collins MW, *et al.*: **Does loss of consciousness predict neuropsychological decrements after concussion?** *Clin J Sports Med* 1999, 9:193–198.
This research with trauma patients utilizes neuropsychologic testing in examining the well-accepted but unproved notion that loss of consciousness is the most salient indicator of concussion outcome.
21. Yarnell P, Lynch S: **Retrograde memory immediately after concussion.** *Lancet*. 1970, 1:863–864.
22. Hanlon RE, Demery JA, Martinovich Z, Kelly JP: **Effects of acute injury characteristics on neurophysical status and vocational outcome following mild traumatic brain injury.** *Brain Inj* 1999, 13:873–887.
23. Clinchot D, Bogner JA, Mysiw WJ, *et al.*: **Defining sleep disturbances after brain injury.** *Am J Phys Med Rehabil* 1998, 77:291–295.
24. Gerberich SG, Gibson RW, Fife D, *et al.*: **Effects of brain injury on college academic performance.** *Neuroepidemiology* 1997, 16:1–14.
25. Bazarian J, Wong T, Harris M, *et al.*: **Epidemiology and predictors of post-concussive syndrome after minor head injury.** *Brain Inj* 1995, 13:173–189.
26. Farace E, Alves W: **Do women fare worse: a meta-analysis of gender differences in traumatic brain injury outcome.** *J Neurosurg* 2000, 93:539–545.
27. Barnes BC, Cooper K, Kirkendall DT, *et al.*: **Concussion history in elite male and female soccer players.** *Am J Sports Med* 1998, 26:433–438.
28. Boden BP, Kirkendall DT, Garrett WE: **Concussion incidence in elite college soccer players.** *Am J Sports Med* 1998, 26:238–241.
29. Roof RL, Duvdevani R, Stein DG: **Gender influences outcome of brain injury: progesterone plays a protective role.** *Brain Res* 1993, 607:333–336.
30. Roof RL, Hall ED: **Estrogen-related gender differences in survival rate and cortical blood flow after impact acceleration head injury in rats.** *J Neurotrauma* 2000, 17:367–388.
31. Emerson CS, Headrick JP, Vink R: **Estrogen improves biochemical and neurologic outcome following traumatic brain injury in male rats, but not in females.** *Brain Res* 1993, 8:95–100.
32. Bruce DA, Alavi A, Bilaniuk L, *et al.*: **Diffuse cerebral swelling following head injuries in children: the syndrome of malignant brain edema.** *J Neurosurg* 1981, 54:170–178.
33. Pickles W: **Acute general edema of the brain in children with head injuries.** *New Engl J Med* 1950, 242:607–611.
34. McDonald JW, Johnston MV: **Physiological and pathophysiological roles of excitatory amino acids during central nervous system development.** *Brain Res Brain Res Rev* 1990, 15:41–70.
35. Huttenlocher PR: **Synaptic density in human frontal cortex: developmental changes and effects of aging.** *Brain Res* 1979, 163:195–205.
36. Roberts GW, Allsop D, Bruton C: **The occult aftermath of boxing.** *J Neurol Neurosurg Psychiatry* 1990, 53:373–378.
37. Jordan BD: **Neurologic aspects of boxing.** *Arch Neurol* 1987, 44:453–459.
38. Jordan BD, Relkin NR, Ravdin LD, *et al.*: **Apolipoprotein E e4 associated with chronic traumatic brain injury in boxing.** *JAMA* 1997, 278:136–140.
39. Friedman G, Froom P, Sazbon L, *et al.*: **Apolipoprotein E e4 genotype predicts a poor outcome in survivors of traumatic brain injury.** *Neurology* 1999, 52:244–248.
40. Teasdale G, Nicoll J, Murray G, Fiddes M: **Association of apolipoprotein E polymorphism with outcome after head injury.** *Lancet* 1997, 350:1069–1071.
41. Mayeux R, Ottman R, Tang M-X, *et al.*: **Genetic susceptibility and head injury as risk factors for Alzheimer's disease among community-dwelling elderly persons and their first-degree relatives.** *Ann Neurol* 1993, 33:494–499.
42. Barth J, Alves W, Ryan T, *et al.*: **Mild head injury in sports: neuropsychological sequelae and recovery of function.** In *Mild Head Injury*. Edited by Levin HS, Eisenberg HM, Benton AL. New York: Oxford University Press; 1989:257–275.
43. Macciocchi S, Barth J, Alves W, *et al.*: **Neuropsychological functioning and recovery after mild head injury in collegiate athletes.** *Neurosurgery* 1996, 39:510–514.
44. Rimel R, Giordani B, Barth J, *et al.*: **Disability caused by minor head injury.** *Neurosurgery* 1981, 9:221–228.
45. Hinton-Bayre AD, Geffen GM, Geffen LB, *et al.*: **Concussion in contact sports: reliable change indices of impairment and recovery.** *J Clin Exp Neuropsychol* 1999, 21:70–86.
46. Lovell MR, Collins MW: **Neuropsychological assessment of the college football player.** *J Head Trauma Rehab* 1998, 13:9–26.
47. Dikmen S, Mclean A, Terukin N: **Neuropsychological and psychosocial consequences of minor head injury.** *J Neurol Neurosurg Psychiatry* 1986, 49:1227–1232.
48. Hugenholtz H, Stuss D, Stethem L, Richard M: **How long does it take to recover from a mild concussion?** *Neurosurgery* 1988, 22:853–858.
49. • Maroon JC, Lovell M, Norwig J, *et al.*: **Cerebral concussions in athletes: evaluation and neuropsychological testing.** *Neurosurgery* 2000, 47:659–671.
This is an important paper addressing all pertinent questions surrounding concussion management, including a comprehensive overview of the relevance and significance of a neuropsychologic testing approach.
50. Lovell M, Collins M, Podell K, Maroon J: **The comparison of computerized versus traditional neuropsychological testing in developing objective criteria for return to play following concussion in high school athletes.** *27th Annual Meeting of the National Academy of Neuropsychology*. Orlando, FL; October 15–18, 2000.
51. 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:S213–221.
52. Mrazik M, Ferrara MS, Peterson CL, *et al.*: **Injury severity and neuropsychological and balance outcomes of four college athletes.** *Brain Inj* 2000, 14:921–931.
53. Guskiewicz KM, Perrin DH, Bansneder BM: **Effect of mild head injury on postural stability in athletes.** *J Athlet Train* 1996, 31:300–306.