JOURNAL OF ATHLETIC TRAINING

VOLUME 36 • NUMBER 3 • JULY–SEPTEMBER 2001

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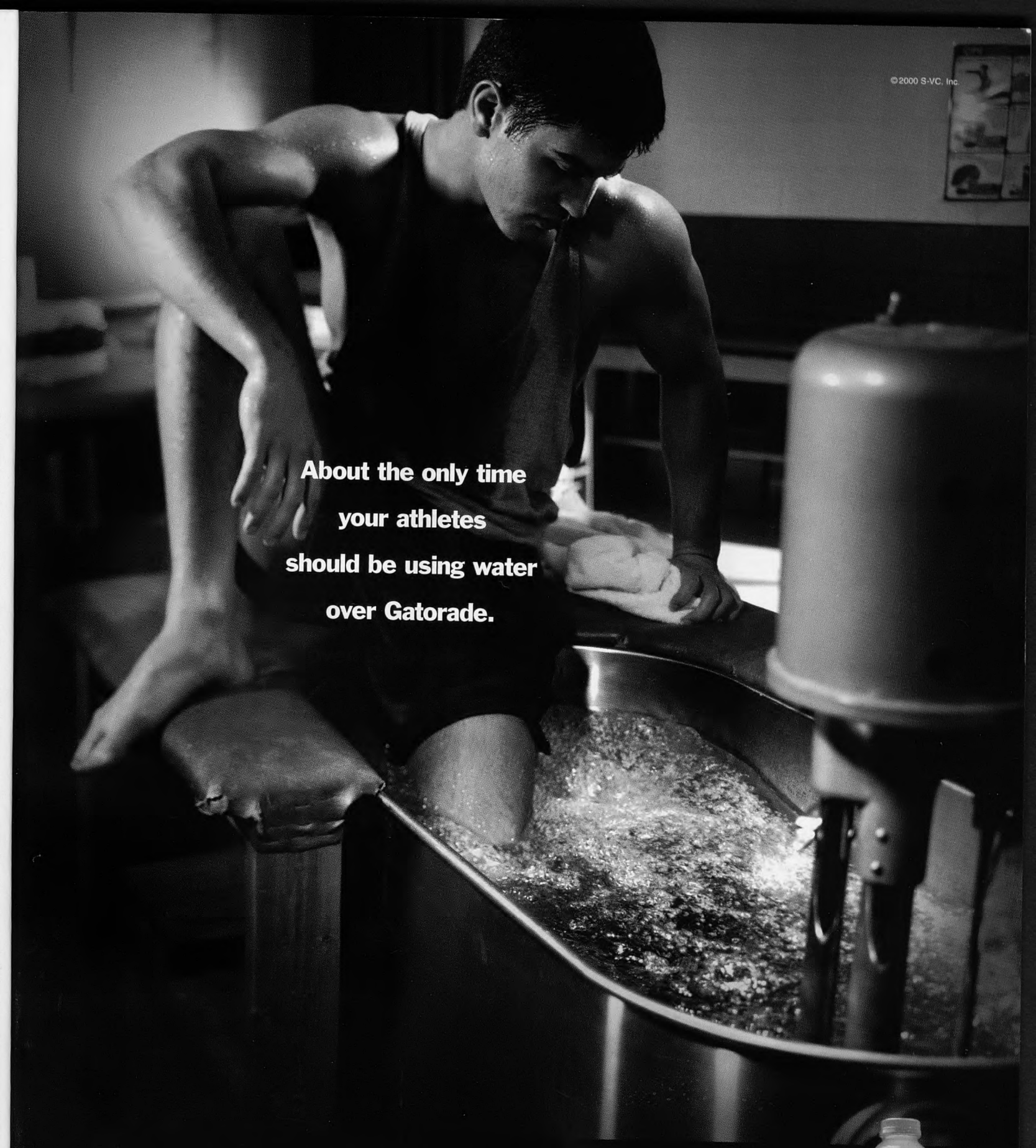

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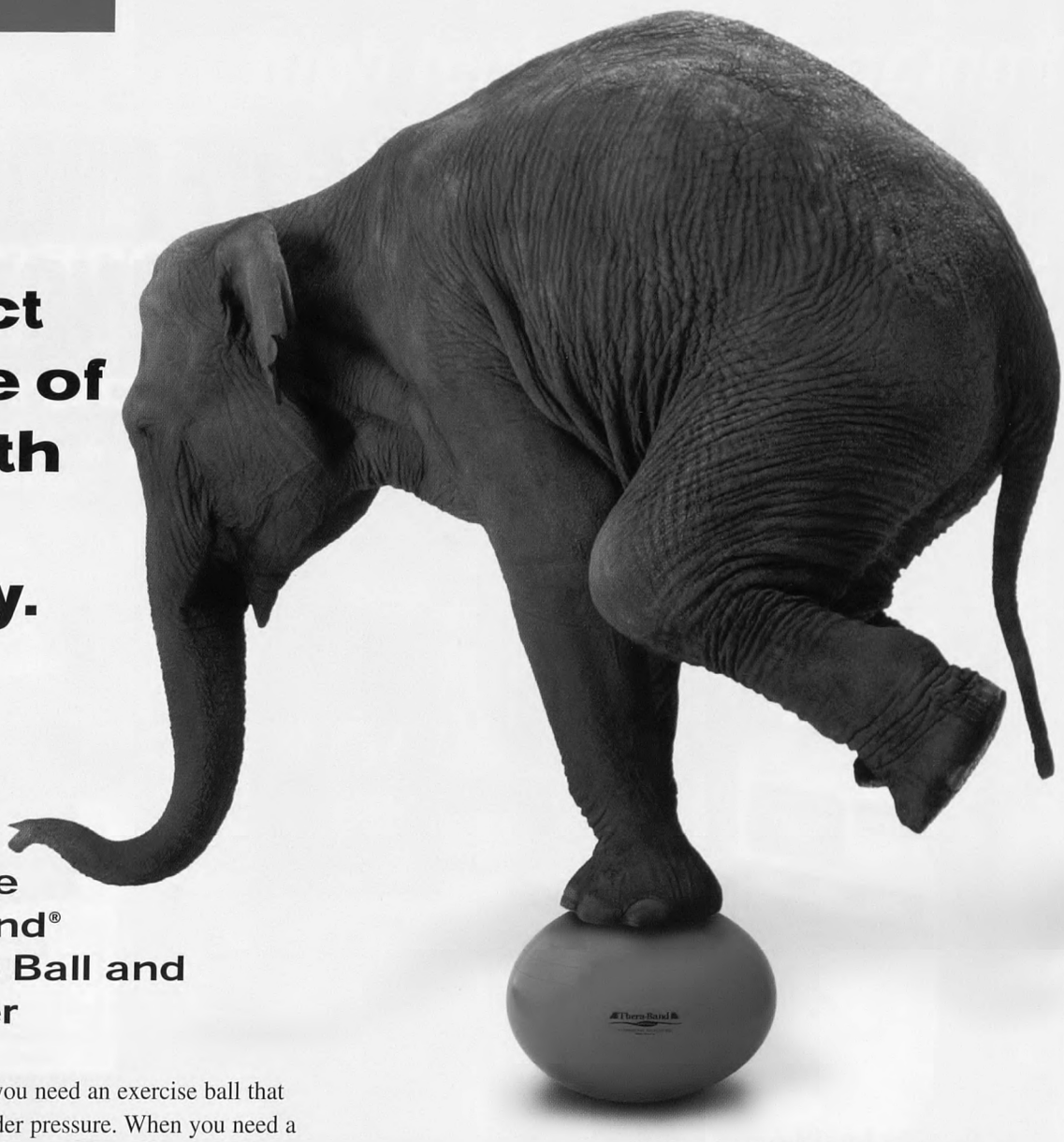

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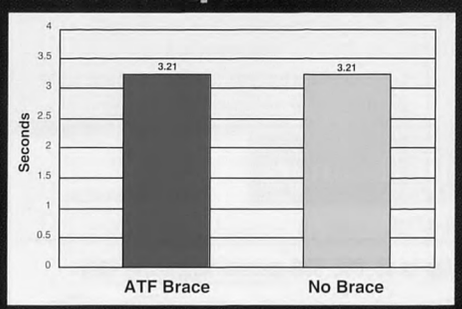

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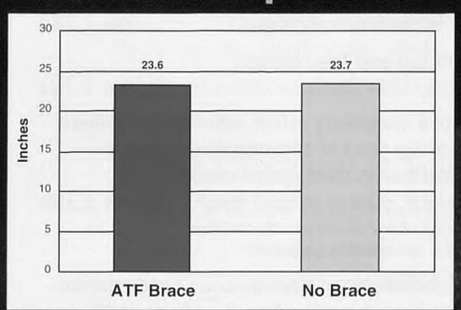

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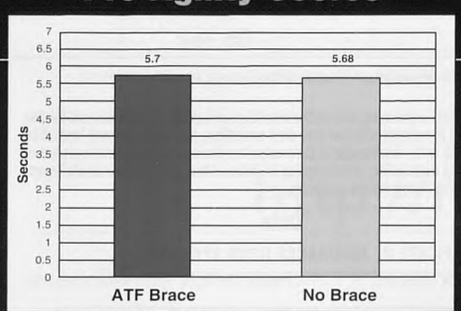

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Editorial

The Concussion Puzzle: 5 Compelling Questions

Kevin M. Guskiewicz

Sport-related concussion has received significant attention in recent years. Media coverage of multiple injuries to several high-profile athletes, some of whom were forced into retirement because of repeated concussions, has created a welcome awareness among sports medicine personnel and the general public. Many of the questions that researchers have failed to address in the past are now being studied. Despite the complexities associated with studying sport-related concussion, answers to some of the more intriguing questions are on the horizon.

1. Which concussion grading scale and return-to-play guideline is the best?

Currently, at least 16 different grading systems are proposed in the literature. Although most systems are considered safe, clinicians often question their practicality. Most are based on limited scientific data, if any, and therefore, none has emerged as the "gold standard." Much emphasis is placed on loss of consciousness and amnesia, when fewer than one third of all sport-related concussions involve either condition. Perhaps grading of the injury should take place after total symptom resolution, as length of symptoms (be it minutes, days, or weeks) is an important factor that is often overlooked. In any event, the sports medicine team is best equipped when a plan is agreed on by both the athletic trainer and the team physician. Once a plan is in place, consistent use of that plan should provide safe decision making.

2. After how many concussions should a career be ended?

We do not currently know the answer to this question, and researchers and clinicians should be reluctant to state a specific number. No two concussions are the same. A great deal of variability exists in the amount of transient impairment associated with these injuries, depending on the mechanism, location, forces applied to the brain, and resultant tissue damage. Some evidence suggests that if injury severity remains constant, total recovery time (ie, time until asymptomatic) increases with each successive injury. This rate of recovery is the telling factor and emphasizes the need for baseline neuropsychological and balance testing for measuring recovery after repeated injuries.

3. Which is the best battery or combination of concussion tests to conduct?

Clearly, the research community is making progress in this area. Assessment of concussion can be likened to putting together a puzzle with many pieces. Decisions should not be made based on the fitting of only one piece of the puzzle. Some neuropsychological tests are simply not sensitive enough to detect neurocognitive deficits in athletes suffering from concussion, while others are rising to the surface as more efficient tests. Computerized testing appears to be the wave of the future, saving significant time and resources while permitting group baseline testing; however, most of these programs have not yet been validated. Postural stability tests, whether performed on a forceplate or as more simple clinical tests, appear to offer valid information regarding the athlete's motor domain after injury. The most important piece of the puzzle must be symptom resolution. All comprehensive concussion assessment protocols should place symptom resolution and techniques for determining the status of an athlete's symptoms at the forefront.

4. Are long-term deficits associated with repeated concussion?

Again, more research is needed. The one thing that is obvious to most clinicians and researchers is that several pieces to the "concussion puzzle" dictate outcome. In some cases, poor outcome is the result of a severe injury, whereas in other cases, it is the result of an excessive number of previous injuries. Early research seems to suggest that recovery after recurrent injury is delayed when compared with an earlier injury. We also know that athletes with a history of concussion have a 3-fold increase in risk of further injury after 1 previous injury. Extrapolation of these findings could suggest that recurrent injury is associated with long-term deficits, but longitudinal studies will be needed to confirm this theory.

5. Are long-term cognitive deficits associated with soccer heading, and should headgear be made mandatory for youth soccer players?

There is no proof of neurocognitive impairment in collegiate soccer players with a long history of soccer participation or a history of concussion, or both. Furthermore, no compelling evidence suggests that headgear is necessary in youth soccer players at this time. Some investigators propose that headgear may even increase rotational loads and increase contact time with the head.

I believe that you will find that this special issue on sport-related concussion provides the answers to many of these very important questions. Answers to other questions that have yet to be answered are just on the horizon and will eventually provide athletic trainers and team physicians with answers that are likely to change the way in which this very complex injury is managed. I thank all of the authors, reviewers, editors, and editorial staff for their commitment to this project. I hope that this special issue on sport-related concussion will become your most useful and referenced holding for managing cases involving an athlete with concussion.

Editor's Note: Kevin M. Guskiewicz, PhD, ATC, is Associate Professor in the Department of Exercise and Sport Science and the Department of Orthopaedics, University of North Carolina at Chapel Hill, Chapel Hill, NC, and Guest Editor for this special issue of the Journal of Athletic Training.

Foreword

Robert C. Cantu

I am pleased and honored to provide some opening words for this major work on concussion edited by Drs Perrin and Guskiewicz. In these pages, they have accumulated the insight of more than 30 international experts on neurologic athletic mild head injury. The result is a thoroughly comprehensive and scientifically impeccable treatment of this crucial area of sports medicine.

Certainly, this is a timely undertaking. Organized sports participation and competition have increased worldwide at an unprecedented rate during the past 30 years. This phenomenon encompasses people of all ages, from children and adolescents through senior citizens. It also includes both sexes, as women are now literally making great strides in athletic endeavors off limits to them in previous generations, notably in contact and collision sports such as soccer, rugby, and wrestling. With such participation by women in collision sports has come an increased risk of concussion.

When I published guidelines for return to contact sports after a concussion more than 15 years ago,[1] I ended with multiple conclusions:

1. "Much more data on concussion are needed." Prospective studies of concussion had not yet been done, nor was the pathophysiology of concussion well understood. In this issue, particularly in the discussion by Giza and Hovda of concussion pathophysiology and in the report by Powell on epidemiology and the article by Mueller on catastrophic injuries, the voids in knowledge that I wrote about 15 years ago have been eliminated.
2. "More research is needed on the cumulative effects of concussion." This concern is addressed in the report by Barth et al on acceleration-deceleration sport-related concussion, in the description by Macciocchi et al of multiple concussion syndrome, and in the account by Erlanger et al of monitoring the resolution of postconcussion syndrome.
3. "This paper is meant to serve only as a guideline: deviation based upon the clinical judgment of the treating physician may be entirely appropriate." This conclusion is addressed in articles by Bailes and Hudson, Cantu, Kelly, Oliaro et al, Guskiewicz et al, McCrea, Randolph, and Barr that deal with the clinical management of concussion, including return-to-play issues. In addition, multiple ways of assessing concussion are covered, including the use of postural stability and the utility and limitations of neuropsychological testing.
4. "A continued aggressive educational effort directed at team physicians, athletic trainers, and coaches is needed." Although virtually all of the papers in this issue discuss this concern, Halstead and Winters specifically address new equipment that may be useful in the prevention of concussion.
5. "Lawyers read medical journals, too. . . regarding lawyers and lawsuits, I want to make it bluntly clear that. . . return to competition in contact sports after concussion. . . is a clinical judgment in every case." Unfortunately, now, more than ever before, no clinical treatise would be complete without a chapter on legal ramifications. This area is comprehensively covered by Osborne.

Again, I reiterate how excited I am to be a part of this effort. The reader of this issue will be treated to the most in-depth and up-to-date assessment of concussion that has ever been made available to the sports medicine profession. As such, it has a place in the library or on the bookshelf of every athletic trainer, researcher, and physician and will prove to be of particular benefit to all active practitioners of sports medicine.

Editor's Note: Robert C. Cantu, MA, MD, FACS, FACSM, is Chairman, Department of Surgery, and Chief, Neurosurgery Service, Emerson Hospital, Concord, MA.

Reference

1. Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Physician Sportsmed.* 1986;14(10):75–83.

Journal of Athletic Training 2001;36(3):228–235

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The Neurometabolic Cascade of Concussion

Christopher C. Giza; David A. Hovda

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Christopher C. Giza, MD, contributed to conception and design; acquisition and analysis and interpretation of the data; and drafting, critical revision, and final approval of the article. David A. Hovda, PhD, contributed to acquisition and analysis and interpretation of the data and critical revision and final approval of the article.

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Objective: To review the underlying pathophysiologic processes of concussive brain injury and relate these neurometabolic changes to clinical sports-related issues such as injury to the developing brain, overuse injury, and repeated concussion.

Data Sources: Over 100 articles from both basic science and clinical medical literature selected for relevance to concussive brain injury, postinjury pathophysiology, and recovery of function.

Data Synthesis: The primary elements of the pathophysiologic cascade following concussive brain injury include abrupt neuronal depolarization, release of excitatory neurotransmitters, ionic shifts, changes in glucose metabolism, altered cerebral blood flow, and impaired axonal function. These alterations can be correlated with periods of postconcussion vulnerability and with neurobehavioral abnormalities. While the time course of these changes is well understood in experimental animal models, it is only beginning to be characterized following human concussion.

Conclusions/Recommendations: Following concussion, cerebral pathophysiology can be adversely affected for days in animals and weeks in humans. Significant changes in cerebral glucose metabolism can exist even in head-injured patients with *normal* Glasgow Coma Scores, underscoring the need for in-depth clinical assessment in an effort to uncover neurocognitive correlates of altered cerebral physiology. Improved guidelines for clinical management of concussion may be formulated as the functional significance and duration of these postinjury neurometabolic derangements are better delineated.

Key Words: metabolism, physiology, repeated concussion, traumatic brain injury

Concussion is defined as any transient neurologic dysfunction resulting from a biomechanical force. Loss of consciousness is a clinical hallmark of concussion but is not required to make the diagnosis. Other symptoms include confusion, disorientation, unsteadiness, dizziness, headache, and visual disturbances. These postconcussive deficits occur with minimal detectable anatomic pathology and often resolve completely over time, suggesting that they are based on temporary neuronal dysfunction rather than cell death. Neuronal dysfunction can occur due to ionic shifts, altered metabolism, impaired connectivity, or changes in neurotransmission. Thus, a complete understanding of the phenomenon of concussion requires knowledge of the underlying pathophysiology of this injury. In this article, we will review the neurometabolic events following experimental concussive brain injury and then apply this knowledge to specific scenarios pertinent to sport-related concussion.

POSTCONCUSSIVE PATHOPHYSIOLOGY

An Overview of Concussion Pathophysiology

Immediately after biomechanical injury to the brain, abrupt, indiscriminant release of neurotransmitters and unchecked ionic fluxes occur. The binding of excitatory transmitters, such as glutamate, to the N-methyl-D-aspartate (NMDA) receptor leads to further neuronal depolarization with efflux of potassium and influx of calcium. These ionic shifts lead to acute and subacute changes in cellular physiology.

Acutely, in an effort to restore the neuronal membrane potential, the sodium-potassium (Na^+-K^+) pump works overtime. The Na^+-K^+ pump requires increasing amounts of adenosine triphosphate (ATP), triggering a dramatic jump in glucose metabolism. This "hypermetabolism" occurs in the setting of diminished cerebral blood flow, and the disparity between glucose supply and demand triggers a cellular energy crisis. The resulting energy crisis is a likely mechanism for postconcussive vulnerability, making the brain less able to respond adequately to a second injury and potentially leading to longer-lasting deficits.

Following the initial period of accelerated glucose utilization, the concussed brain goes into a period of depressed metabolism. Persistent increases in calcium may impair mitochondrial oxidative metabolism and worsen the energy crisis. Unchecked calcium accumulation can also directly activate pathways leading to cell death. Intra-axonal calcium flux has been shown to disrupt neurofilaments and microtubules, impairing posttraumatic neural connectivity.

This overview represents a simplified framework of the neurometabolic cascade (Figure 1). Other important components of posttraumatic cerebral pathophysiology include, but are not limited to, generation of lactic acid, decreased intracellular magnesium, free radical production, inflammatory responses, and altered neurotransmission. We will now discuss some of

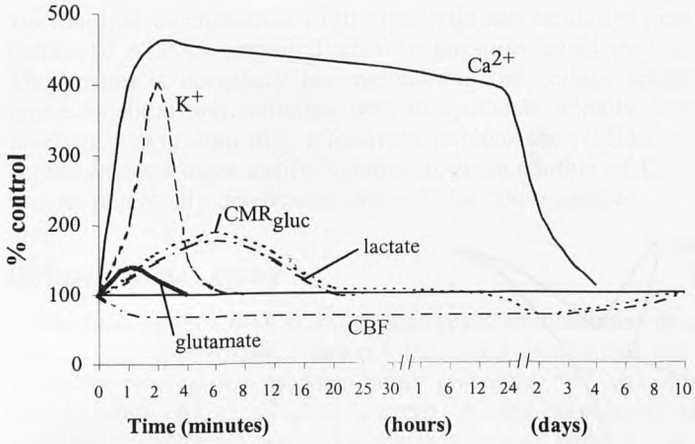

Figure 1. Neurometabolic cascade following experimental concussion. K^+, potassium; Ca^{2+}, calcium; CMRgluc, oxidative glucose metabolism; CBF, cerebral blood flow. (Reprinted with permission. Giza CC, Hovda DA. Ionic and metabolic consequences of concussion. In: Cantu RC, Cantu RI. *Neurologic Athletic and Spine Injuries.* St Louis, MO: WB Saunders Co; 2000:80–100.).

the pertinent details of postconcussive pathophysiology in both experimental animal models and in humans.

Acute Metabolic and Ionic Changes

Immediately after biomechanical injury to the brain, there is disruption of neuronal membranes, axonal stretching, and opening of voltage-dependent K^+ channels, which leads to a marked increase in extracellular K^+.[1–4] In addition, nonspecific depolarization leads to an early, indiscriminate release of the excitatory amino acid (EAA) glutamate, which exacerbates the K^+ flux by activating kainate, NMDA, and D-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid (AMPA) receptors (Figure 2, events 1 and 2). In rats, treatment with the EAA inhibitor kynurenic acid greatly diminishes the posttraumatic K^+ efflux.[3]

Normally, excessive extracellular K^+ is taken up by surrounding glial cells.[5–7] By this mechanism, the brain can maintain physiologic K^+ levels after mild perturbations; however, larger insults, such as brain trauma or ischemia, overcome this compensation.[8–11] As extracellular K^+ increases, neuronal depolarization is triggered, leading to further release of EAAs, opening of EAA receptor channels (NMDA, AMPA, kainate), and still greater K^+ flux (Figure 2, event 3). This massive excitation is then followed by a wave of relative neuronal suppression that has been termed *spreading depression.*[12–16] One important distinction between classic spreading depression and postconcussive K^+ fluxes is that after trauma, diffuse areas of the brain are affected simultaneously. Early loss of consciousness, amnesia, or other cognitive dysfunction may be manifestations of a posttraumatic spreading depression–like state.

In an effort to restore ionic homeostasis, energy-requiring membrane pumps are activated[17–19] and trigger an increase in glucose use (Figure 2, events 4 and 5).[20–22] This increase in glucose use occurs almost immediately after fluid percussion injury in rats and persists for up to 30 minutes in the ipsilateral cortex and hippocampus.[22] After more severe injury such as cortical contusion, increased glucose metabolism may last 4 hours in areas distant from the contusion core.[23] Because cerebral oxidative metabolism typically runs near its maximum, an abrupt increase in energy requirements is best met by an increase in glycolysis.[24,25]

Accelerated glycolysis leads to increased lactate production and is seen after both ischemic[26–28] and concussive[29–33] brain injury. In addition to hyperglycolysis, oxidative metabolism is also impaired after brain trauma.[34–36] This impairment of mitochondrial function can lead to reduced ATP production, which provides a second stimulus for increased glycolysis. Thus, lactate production by glycolysis increases concurrent with a decrease in lactate metabolism, resulting in lactate accumulation (Figure 2, event 6). Elevated lactate levels can result in neuronal dysfunction by inducing acidosis, membrane damage, altered blood brain barrier permeability, and cerebral edema.[37–41] Increased levels of lactate after traumatic brain injury (TBI) may leave neurons more vulnerable to a secondary ischemic injury,[42] but whether this is the case in repeated traumatic injury is not known. An intriguing hypothesis suggests that glial lactate production increases posttraumatically and that this excess lactate is actually transported into neurons for use as an alternate fuel.[43]

Cerebral Blood Flow–Glucose Metabolism Uncoupling

Under normal conditions, cerebral blood flow (CBF) is tightly coupled to neuronal activity and cerebral glucose metabolism. After experimental fluid percussion injury, however, CBF may be reduced to 50% of normal.[44–47] This posttraumatic decrease in CBF does not approach the 85% reduction seen in frank ischemia[48]; nonetheless, in a setting of increased glucose use (hyperglycolysis), this mismatch in supply and demand results in a potentially damaging energy crisis.

Calcium Influx, Mitochondrial Dysfunction, and Delayed Glucose Hypometabolism

Calcium accumulation is seen within hours of experimental concussion and may persist for 2 to 4 days.[49–52] The posttraumatic depolarization and K^+ efflux triggers the release of EAAs that, in turn, activates NMDA receptors.[3] Activated NMDA receptors form a pore through which calcium (Ca^{2+}) can enter the cell. A potent N-type calcium channel blocker, SNX-111, significantly reduces postconcussive Ca^{2+} accumulation,[53] presumably by reducing release of glutamate.[54] Results of treatment with NMDA receptor antagonists have been mixed, however. No reduction in Ca^{2+} accumulation was seen after weight-drop injury and pre-treatment with the NMDA receptor blocker MK-801,[55] but treatment with HU-211, a synthetic cannabinoid with a pharmacologic profile characteristic of an NMDA receptor antagonist, was associated with a reduction in post-TBI Ca^{2+} accumulation.[56]

Excess intracellular Ca^{2+} may also be sequestered in mitochondria,[34,36] resulting in impaired oxidative metabolism and, ultimately, energy failure (Figure 2, events 7 and 8). Cytochrome oxidase histochemistry, which is a measure of oxidative metabolism, shows a biphasic reduction after experimental concussion. In the ipsilateral cortex, a relative reduction on day 1 recovers by day 2, only to be reinstated on day 3, to bottom out on day 5, and to recover by 10 days postinjury. Smaller but more lasting changes are seen in the ipsilateral hippocampus, with decreases still evident at 10 days.[57]

After the initial period of hyperglycolysis, cerebral glucose

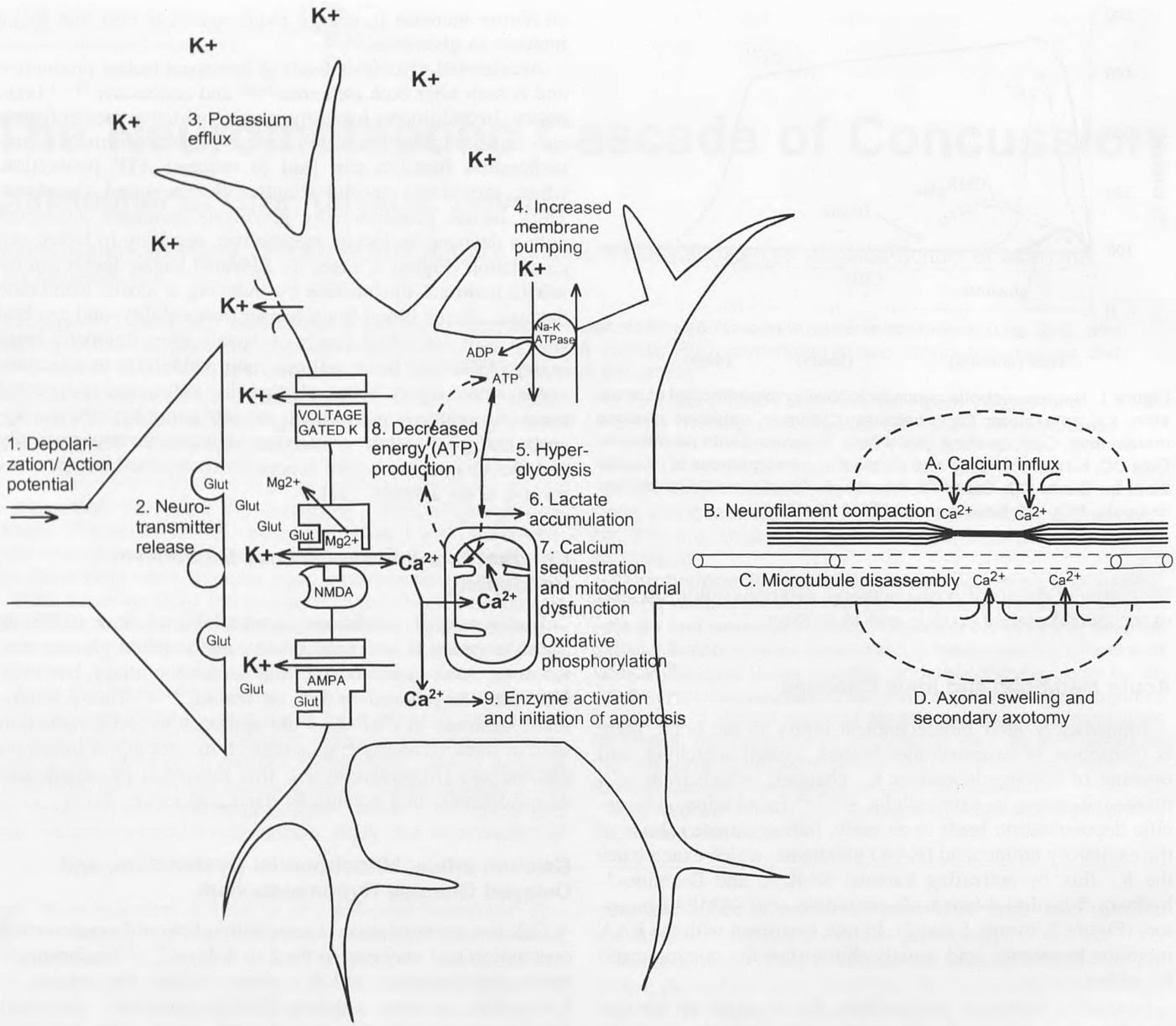

Figure 2. Neurometabolic cascade following traumatic injury. (1) Nonspecific depolarization and initiation of action potentials. (2) Release of excitatory neurotransmitters (EAAs). (3) Massive efflux of potassium. (4) Increased activity of membrane ionic pumps to restore homeostasis. (5) Hyperglycolysis to generate more adenosine triphosphate (ATP). (6) Lactate accumulation. (7) Calcium influx and sequestration in mitochondria leading to impaired oxidative metabolism. (8) Decreased energy (ATP) production. (9) Calpain activation and initiation of apoptosis. A, Axolemmal disruption and calcium influx. B, Neurofilament compaction via phosphorylation or sidearm cleavage. C, Microtubule disassembly and accumulation of axonally transported organelles. D, Axonal swelling and eventual axotomy. K^+, potassium; Na^+, sodium; Glut, glutamate; Mg^{2+}, magnesium; Ca^{2+}, calcium; NMDA, N-methyl-D-aspartate; AMPA, d-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid.

use is diminished by 24 hours postinjury and remains low for 5 to 10 days in experimental animals.[22] Positron emission tomography (PET) in humans shows similar decreases in global cerebral glucose metabolism that may last 2–4 weeks post-TBI.[58] In addition, this study found that post-injury hypometabolism did not correlate closely with level of consciousness as measured by Glasgow Coma Score (GCS). Depressed cerebral glucose metabolism was seen in comatose patients as well as in walking, talking patients, suggesting that significant neurometabolic abnormalities may occur in the absence of overt clinical symptoms.[58] What is not yet clear is whether this hypometabolism represents a period when the brain is relatively protected from secondary injury or if the brain is more vulnerable because it is unable to respond adequately to further energy demands. It is also unknown whether this depressed glucose metabolism is responsible for more subtle neurocognitive deficits seen after TBI.

Reductions in Magnesium

Intracellular magnesium levels are also immediately reduced after TBI and remain low for up to 4 days.[59–62] This reduction in magnesium has been correlated with postinjury neurologic deficits, and pretreatment to restore magnesium levels results in improved motor performance in experimental animals.[63] Decreased magnesium levels may lead to neuronal dysfunction

via multiple mechanisms. Both glycolytic and oxidative generation of ATP are impaired when magnesium levels are low. Magnesium is necessary for maintaining the cellular membrane potential and initiating protein synthesis. Finally, low levels of magnesium may effectively unblock the NMDA receptor channel more easily, leading to greater influx of Ca^{2+} and its potentially deleterious intracellular consequences.

Diffuse Axonal Injury

Mechanical stretching of axons may result in membrane disruption and even depolarization.[4] Increased axolemmal permeability persists for up to 6 hours postinjury[64,65] and can lead to influx of Ca^{2+} (Figure 2, event 9A) and mitochondrial swelling.[66,67] Neurofilament compaction occurs from 5 minutes to 6 hours postinjury (Figure 2, event 9B), either by phosphorylation, which alters neurofilament stability,[68–70] or by calpain-mediated proteolysis of sidearms, which can lead to neurofilament collapse.[71] Increased axonal Ca^{2+} levels have been shown to lead to microtubule breakdown from 6 to 24 hours after the initial injury (Figure 2, event 9C).[72,73]

Intra-axonal cytoskeletal abnormalities lead to accumulation of organelles at the site of axonal damage due to continued axonal transport along intact segments. These focal axonal swellings eventually develop constrictions that then lead to secondary axotomy (Figure 2, event 9D) and formation of axonal bulbs.[73,74] Signs of secondary axonal disconnection may be seen as soon as 4 hours postinjury but have been reported to persist over days and even weeks in brain-injured humans.[75]

Delayed Cell Death and Persistent Calcium Accumulation

Post-TBI increases in Ca^{2+} do not inevitably lead to cell death. As mentioned previously, elevated intracellular Ca^{2+} may certainly lead to impaired mitochondrial metabolism, but neurons may still survive. In fact, after moderate experimental concussion, Ca^{2+} accumulation peaks in 2 days and resolves without obvious morphologic damage by 4 days.[50] Animals experiencing more severe injury and demonstrating anatomic damage show persistent Ca^{2+} elevations at the injury site. In adult animals, a delayed rise (14 days postinjury) of Ca^{2+} is seen in distant structures (thalamus), which corresponds to neuronal death.[52]

Intracellular Ca^{2+} may trigger cell death by a variety of mechanisms (Figure 2, event 9), including overactivation of phospholipases,[76] plasmalogenase, calpains,[77,78] protein kinases,[79] nitric oxide synthase, and endonucleases. These alterations may then lead to free radical overproduction,[80] cytoskeletal reorganization,[81] and activation of apoptotic genetic signals.[82]

Neurotransmitter Alterations

Long-term deficits in memory and cognition in a setting of minimal anatomic change are often seen after concussion. These may result from dysfunctional excitatory neurotransmission. Postconcussive alterations have been reported in glutamatergic (NMDA),[83–85] adrenergic,[86,87] and cholinergic[88] systems. Long-term potentiation, an NMDA-dependent measure of plasticity, may be persistently impaired in the hippocampus after concussive brain injury.[89–91] Concussive brain injury also leads to early changes in choline acetyltransferase activity[88] and later loss of forebrain cholinergic neurons.[92] Impaired cholinergic neurotransmission leads to learning and spatial memory deficits in animals.[93,94]

Inhibitory neurotransmission is also altered after TBI. A loss of γ-aminobutyric acid-producing (GABAergic) hilar neurons can compromise normal inhibition of hippocampal dentate granule cells.[95] This loss of inhibitory neurons may predispose the traumatized brain to subsequent development of seizures.[96]

CONCERNS RELEVANT TO ATHLETIC CONCUSSION

Concussion in the Developing Brain

With increasing numbers of children and young adults participating in organized sports and sustaining head injuries, understanding the effects of TBI on the immature brain becomes more and more important. Clinical dogma has held that the younger the brain, the more resilient it is after injury. Recent studies of moderate fluid percussion in juvenile rats would seem to support this contention, demonstrating no obvious neurologic or pathologic deficits in these young animals.[97,98] Using a closed head injury weight-drop model, significant deficits are seen only at injury severities that result in very high mortality (75%).[99]

On the other hand, there is also evidence to support the idea of specific developmental periods when the young brain may be more vulnerable to injury. TBI in children results in higher mortality than in adolescents, perhaps due to an increased incidence of cerebral edema.[100–102] In the experimental model of developmental concussion, the youngest rats became hypotensive after even mild injuries and tended to have longer apnea times than adult rats. After severe fluid percussion injury, mortality in these immature animals approached 100%.[97]

It is reasonable to hypothesize that diffuse mechanical injury can have lasting effects on the complex sequence of neurochemical and anatomical events occurring during normal development. Indeed, long-term follow-up studies demonstrate persistent neurocognitive deficits after pediatric TBI.[103,104] However, it is also difficult to assess developmental deficits in children after mild brain injury, as signs of overt neurologic dysfunction may be lacking, and loss of developmental potential may only be demonstrable at a later time or under specific circumstances.

Environmental enrichment provides a useful experimental model with which to study developmental plasticity after brain injury. In an enriched environment, rats are reared communally in a large cage with multiple toys, tunnels, and objects that are changed regularly. When compared with animals reared in standard conditions, enriched rats have increases in cortical thickness, larger neurons, more glia, a greater number of synapses, and enhanced dendritic branching.[105–107] The enriched rats are also superior in cognitive testing using the Morris water maze.[108] However, when moderately concussed juvenile rats are reared in an enriched environment, they fail to develop the increased cortical thickness and enhanced cognitive performance seen in sham-injured enriched controls.[109] These results demonstrate that developmental brain injury, even without early behavioral deficits or significant later morphologic damage, can lead to impaired plasticity. Further studies must be done to determine whether this loss of experience-dependent plasticity is permanent or whether it represents a

window of impairment after which the capacity for neural reorganization recovers.

Overuse Injury

As demonstrated by the recent focus on concussion in football and hockey, both athletic trainers and athletes feel significant pressure to return athletes to practice and play as soon as possible after injury. Although returning to play may be delayed because of concerns about susceptibility to a second brain injury, returning to practice might seem like a reasonable means of maintaining physical conditioning while awaiting full recovery.

In animals, the importance of limb use in recovery of function after unilateral cortical lesions has been well demonstrated.[110] In fact, recovery of function was associated with increased dendritic growth in the homotopic region of the uninjured cortex, dependent on use of the intact forelimb. However, restraint of the uninjured forelimb, and thus forced overuse of the injured limb, resulted in a failure of dendritic enhancement in the intact cortex, an increase in the lesion size in the injured cortex, and a longer-lasting behavioral deficit.[111] There also appears to be a time window when the deleterious effects of forced overuse are mitigated to some degree. In the same model, when immobilization of the intact arm was delayed 1 week after the injury, the functional recovery was still delayed, but the increase in lesion size did not occur.[112] The results of these studies suggest that, at least after focal brain injury, it is possible to overstimulate the injured brain and that this excessive activation can lead to longer-lasting deficits.

Repeated Concussion

How soon to return to play after a head injury and the consequences of repeated concussions are two of the most important health-related issues in sports today. We have earlier reviewed what is known about the neurometabolic cascade of events that occurs after experimental brain injury (Figure 1). Acute abnormalities include ionic fluxes, indiscriminate glutamate release, hyperglycolysis, lactate accumulation, and axonal injury. Later steps in this physiologic cascade involve increased intracellular calcium, mitochondrial dysfunction, impaired oxidative metabolism, decreased glycolysis, diminished CBF, axonal disconnection, neurotransmitter disturbances, and delayed cell death. It is during this postinjury period, when cellular metabolism is already stretched to its limits, that the cell is more vulnerable to further insults.[13] Examining the time course of the post-TBI neurometabolic cascade may help us determine guidelines for vulnerability of the concussed brain to a second injury.

Several physiologic parameters indicate windows of potential vulnerability in the traumatized brain. First, consider the period of glucose metabolism-CBF uncoupling. This phenomenon is most dramatic during the hyperglycolytic phase, which, in the rat, begins at the time of injury and lasts for at least 30 minutes.[22] At this time, cerebral metabolism is already at its limit, and any further demand in energy (due to increased ionic flux) or reduction in energy (due to impaired blood flow or reduced ATP synthesis) may tip the scale in favor of irreversible neuronal injury. Thus, injured cells may be capable of recovering after an initial injury, but a second concussion during this energy crisis can lead to cell death. After the initial hyperglycolytic period, cerebral glucose metabolism is reduced, as is CBF, apparently resolving the mismatch in energy supply and demand. However, during this period, CBF may be unable to respond to a stimulus-induced increase in cerebral glucose metabolism, reinstating the metabolic crisis. An increase in glycolysis in this period may be due to excessive external stimulation or a second injury (concussion, ischemia, or seizure).

A second potential period of vulnerability centers on intracellular Ca^{2+} accumulation. Increased Ca^{2+} levels may impair mitochondrial metabolism at the time when the cell can least tolerate a reduction in ATP production. Additional Ca^{2+} influx, again due to increased physiologic stimulation or a second injury, may go on to activate proteases that initiate the march to programmed cell death. In the rat model, this period of acute Ca^{2+} accumulation is somewhat severity dependent and lasts 2 to 4 days.[50,52]

Another period of risk may be associated with impaired neurotransmission. Alterations in NMDA receptor composition can persist for up to 1 week after injury in developing rats,[84] and a second injury in this period can lead to further impairment of excitatory neurotransmission with a greater degree of cognitive dysfunction. Long-term potentiation, postulated as a mechanism for learning and memory, is impaired for up to 8 weeks after experimental brain injury[91] and may be another means by which altered excitatory neurotransmission results in neurobehavioral deficits. Diminished attention and cognition are particularly important in an athletic setting, when subtle impairments will likely increase the risk of recurrent head injury.

Post-TBI changes in inhibitory neurotransmission seen in rats[95,96] can leave neurons more susceptible to massive depolarization and EAA release after a recurrent concussion. Excessive excitation may then more easily lead to seizure activity, increased energy demand, and possibly further cell death.

As each of these physiologic parameters has its own time frame, and each head injury can be very different from the next, it is difficult to definitively state the true duration of vulnerability to a second injury. Preliminary studies using a double concussion model in rats revealed increased anatomic damage and prolonged hypometabolism when 2 concussions were separated by as much as 5 hours.[114] Double concussion also appears to increase immunostaining for glial fibrillary acidic protein (a marker for gliosis and scarring) and lead to greater cell loss when compared with a single injury.[115] Interestingly, in a recent report, multiple mild concussions preceding a more severe concussion by 3 to 5 days actually resulted in a better functional outcome than an isolated severe concussion.[116] However, the anatomic injury appeared unchanged. This finding raises the possibility that after TBI, there is a period of increased danger to a second injury, followed by a period when the brain may actually be more capable of recovering from a repeated injury.

Of course, translating these experimental time frames into time frames relevant to human concussion can be tricky. Posttraumatic derangements in glucose metabolism resolve within 7 to 10 days in rats, whereas in humans, persistent depression of glucose uptake has been reported 2 to 4 weeks later.[58] Evidence of secondary axotomy is seen as soon as 4 hours postinjury in animals, but evidence of ongoing axonal damage has been reported in human brain tissue even weeks after trauma.[75] In general, the time frame for events in rats is much shorter than for similar periods in humans, and it would not be unreasonable to assume that these periods of postinjury physio-

logic change are longer lasting in humans. In addition, differences in injury type and severity certainly affect the duration of these neurometabolic changes and must also be considered when determining back-to-play status.

SUMMARY

Cerebral concussion is followed by a complex cascade of ionic, metabolic, and physiologic events. The earliest changes are an indiscriminate release of EAAs and a massive efflux of K^+, triggering a brief period of hyperglycolysis. This is followed by more persistent Ca^{2+} influx, mitochondrial dysfunction with decreased oxidative metabolism, diminished cerebral glucose metabolism, reduced CBF, and axonal injury. Late events in the cascade include recovery of glucose metabolism and CBF, delayed cell death, chronic alterations in neurotransmission, and axonal disconnection. Clinical signs and symptoms of impaired coordination, attention, memory, and cognition are manifestations of underlying neuronal dysfunction, most likely due to some of the processes described above. It is difficult to match clinical signs with specific underlying physiologic derangements, and guidelines permiting return to play only after resolution of all motor and cognitive deficits are a minimal precaution. There is recent evidence that even relatively asymptomatic (GCS 13-15) patients may demonstrate depressed glucose metabolism on PET imaging following TBI, reinforcing the need for meticulous clinical assessment. Further study will reveal more details of the time course for this neurometabolic cascade. This will eventually permit improved clinical monitoring of posttraumatic pathophysiology in actual patients, including variables such as cerebral glucose metabolism, blood flow, neuronal activity, and even molecular changes.

Traumatic injury to the developing brain may lead to long-lasting changes in cognitive potential, perhaps even with little evidence of an initial deficit. Children and adolescents who sustain a concussive brain injury should be closely monitored over time for the later appearance of neurobehavioral abnormalities.

Repeated injury within a particular time frame can lead to a much larger anatomical or behavioral impairment than 2 isolated injuries. The second injury may be obvious, such as a repeated concussion, hypoxia, or seizure, or it may occur in the form of premature activation or overstimulation of the injured brain. An awareness and understanding of postconcussive pathophysiology will help in determining the best time course for return to practice and return to play.

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Classification of Sport-Related Head Trauma: A Spectrum of Mild to Severe Injury

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Objective: To identify the types of injuries the human brain incurs as a result of traumatic forces applied to the cranium. In athletic events and endeavors, the full spectrum of intracranial hemorrhages in various compartments, raised intracranial pressure, and diffuse nonhemorrhagic damage may be seen. In this review, we describe these serious injuries and the more common mild traumatic brain injury in their clinical presentations and relate concussion classification to the overall picture of traumatic brain injury.

Methods: Our cumulative experience with athletic injuries, both at the catastrophic and mild traumatic brain injury levels, has led us to a management paradigm that serves to guide us in the classification and treatment of these athletes.

Discussion: The occurrence of intracranial injuries in sports has now been well documented. Intracranial hematomas (epidural, subdural, and parenchymal) and cerebral contusions can result from head injury. Many patients sustain a diffuse brain injury, resulting in elevated intracranial pressures, without a blood clot or mass lesion. The classification of concussion and the use of concussion guidelines are not uniform. However, the major emphases are agreed upon: the close and careful scrutiny of the athlete, an expeditious but reliable neurologic examination, and proper on-field management. Return-to-play decisions are based on many factors that affect normal functioning, both on and off the playing field.

Conclusions: Sufficient knowledge now exists to allow us to carefully evaluate the injured athlete, to place him or her in the management scheme to minimize the potential for permanent cerebral dysfunction, and to know when the athlete can safely return to contact sport participation.

Key Words: diffuse brain injury, hematoma

As the number of participants in recreational and organized sporting events continues to increase, we are faced with an expanding interest in understanding the effects of mild traumatic brain injury (MTBI). Concussion, as MTBI is commonly termed, is a concern in every sport that has head impact or collision as a possible consequence. Paramount to an appreciation for the seriousness and potential consequences of concussion is the concept that it exists as a pathophysiologic entity along a spectrum of injury, ranging from mild concussion to severe, diffuse injuries.[1,2] Associated severe cognitive, memory, and motor deficits persist among survivors of severe, diffuse brain injury, and the mortality rate is greater than 50%.[3–5]

Diffuse brain injury differs from focal brain injury in that a global insult results in widespread neuronal dysfunction. Diffuse brain injury occurs along a continuum of diffuse axonal injury (DAI), represented by shearing of white-matter fiber tracts as they course from the cortex to the midbrain and brain stem, producing characteristic radiographic findings (Figures 1 and 2).[6] Rotational energy forces lead to disruption of axons along the neuroaxis, marked by small areas of hemorrhage.[7] Disruption of axon fibers projecting within the cerebrum and brain stem has been documented in autopsy studies in patients with MTBI.[3,4]

As kinetic energy is applied to the human cranium, both acceleration-deceleration and rotational mechanisms occur. Acceleration-deceleration injury, also considered translational (linear) impact, usually results when the subject's body and head are traveling at a particular speed and strike a solid object. Similarly, a head at rest may be struck by a moving object. The resultant injury causes linear, tensile, and compressive strains that disrupt the cerebral anatomy and cytoarchitecture. Rotational (angular) movements also take place, secondary to the fixation of the brain at the foramen magnum and craniospinal junction and the relative tethering of the midbrain as it passes through the tentorial hiatus. Energy directed to the head may cause transmission of force in a rotatory direction, often producing diffuse brain injury with shearing of the white-matter fiber tracts.[3]

Although it is possible for combinations of biomechanical factors to coexist during head injury, observation has shown that one mechanism is usually predominant. In wrestling, the takedown or activity associated with this maneuver usually causes MTBI.[8] Biomechanical studies concerning athletic injuries have suggested that angular head accelerations have a higher incidence of injury.[9] Acceleration-deceleration energy forces are more likely in contact sports, especially football, ice hockey, and boxing. These can happen within the framework

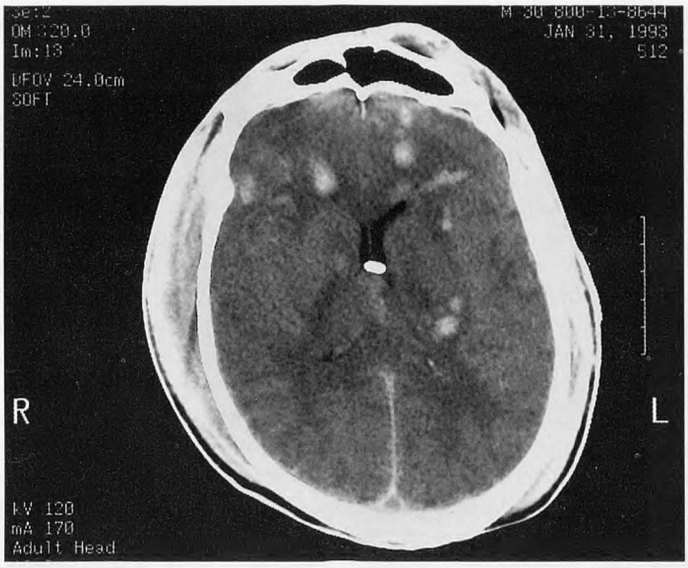

Figure 1. Computed tomography (CT) scan showing diffuse axonal injury in a 30-year-old male victim of a motor vehicle accident. Patients with diffuse axonal injury classically are unconscious from the moment of impact, and impairment is often worse than in patients with other primary injury (contusions, intracerebral hemorrhage, subdural hematoma, epidural hematoma). Diffuse axonal injury characteristically appears as multiple small, focal scattered lesions throughout the white matter, usually at gray-white junctions, sparing the cortex. CT and magnetic resonance imaging (MRI) underestimate the true extent of injury.

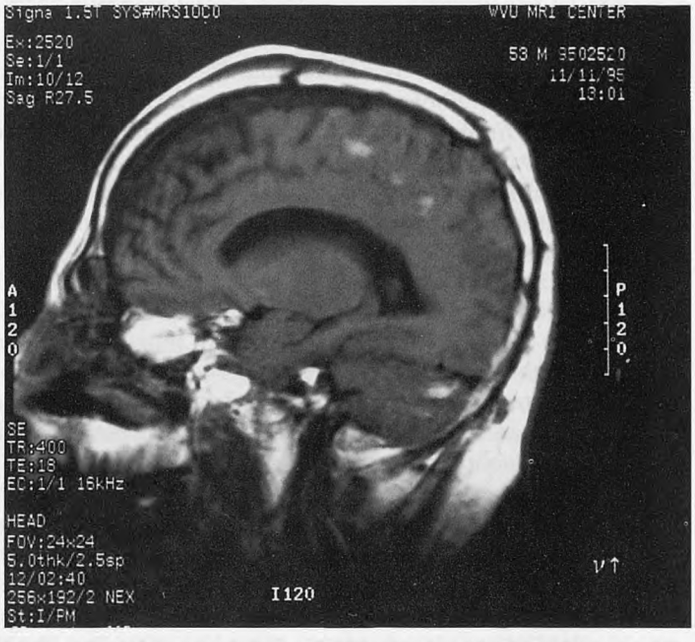

Figure 2. MRI scan in a comatose patient with closed head injury showing diffuse axonal injury changes. While large hematomas with mass effect are ordinarily not seen, diffuse axonal injury represents widespread shearing of axons and multiple areas of injury.

of the contest rules and often within a single event. Blocking and tackling, checking, jabs, cross-punches, and many other techniques predominately deliver acceleration-deceleration or linear energy vectors to the contestant. In contrast, mechanisms exemplified by the boxing hook punch impart rotatory forces to the mandible and cranium. The rotatory component of the cranial or mandibular impacts often contributes to the forces that cause loss of consciousness (LOC), providing the etiology for many athletic cerebral concussions.

In addition, forces applied to the head in contact sports have also been described as impact or impulsive loading. Resulting from a rapidly applied energy input, impact loading is a direct blow to the cranium occurring over less than 200 milliseconds. This can cause skull deformation and energy shock-wave propagation through the skull and brain, resulting in underlying cerebral injury. In contrast, when the head is placed in motion and suddenly accelerated or decelerated as a result of either an impact to another part of the body or as a secondary response to a direct impact, impulsive loading occurs. This mechanism causes compressive, shear, and tensile stresses to the brain, leading to more diffuse or remote injuries. It is more often seen with rotational energy inputs and is less effectively prevented by protective headgear, which is better able to dissipate impact-loading energy input.[10]

Presence of the normal state of consciousness signifies that the person is awake and alert with the ability to interact with the environment. A normal level of consciousness depends on a complex interaction of cortical, subcortical, and brain stem nuclei. Alteration of the state of consciousness occurs when the integrity of this neurophysiologic functional unit has been interrupted. The reticular activating system extending through the brain stem must interact with the hypothalamus and cerebral hemispheres in a normal feedback-loop mechanism for consciousness to be maintained. Any alteration of this circuitry and feedback, therefore, produces a change in the state of consciousness. The major focus of categorizing concussions in sports in the past has centered on the occurrence of LOC. We now appreciate that other and ongoing expressions of cerebral dysfunction, such as memory and cognitive dysfunction, are important and likely predictive. This is especially true when considering only the very brief period of LOC, which may not be as indicative of a serious athletic injury.[11,12]

In addition to the biomechanics, recent experimenters have elucidated the biomechanical abnormalities that represent concussion. Activation of the glycolytic process as a result of cellular requirements to maintain ionic gradients is believed to be present in concussion. Large increases occur in extracellular potassium concentrations through voltage-gated potassium channels.[13–15] Neurotransmitters, in particular glutamate, appear to play a significant role in opening ionic channels after even MTBI. A quantitative rise in glucose utilization, in an effort to correct ionic perturbations in transmembrane potentials, is seen in newer concussion models.[16,17] Metabolic dysfunction after concussive injury produces a period of neuronal vulnerability in which there is both higher demand for glucose to correct ionic movements and a paradoxical reduction in cerebral blood flow, the latter influenced by calcium movements. The rise in glycolytic energy requirements has been shown both experimentally and clinically to be present within the first several days after concussive brain injury.[17]

Experimental concussion models have demonstrated transient cerebral ischemia, edema, widespread neuronal depolarization from release of acetylcholine, and the shearing of neurons and nerve fibers (particularly in the brain stem) as potential explanations for alterations of mental status after closed head injury. While no gross neuropathologic changes are consistently demonstrated in experimental concussion, disruption of multiple neurons and their connections, along with scattered capillary damage in the brain stem reticular formation, has been reported.[17,18]

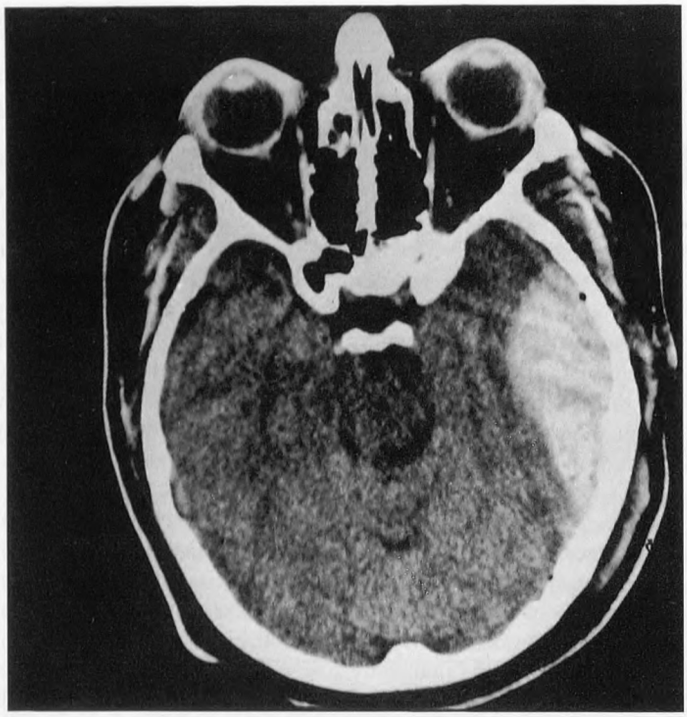

Figure 3. CT scan showing epidural hematoma in the left middle cranial fossa with significant mass effect. The lenticular or biconvex shape is typical, indicative of its extra-axial location.

TRAUMATIC INTRACRANIAL LESIONS

Epidural Hematoma

Epidural hematoma is an accumulation of blood between the dura and skull. The dura becomes detached and dissects to the point of dural attachment to the overlying cranium. Hemorrhage occurs beneath the skull and outside the dura, resulting in the classic computed tomography (CT) appearance of a biconvex or lenticular shape of the hematoma (Figure 3). Epidural hematoma is caused by head impact, usually of the acceleration-deceleration type, and can result in inward deformity, leading to dural detachment from the inner table of the skull. Most patients with an epidural hematoma have a skull fracture, which leads to laceration of the middle meningeal artery or vein. In addition, bleeding can occur from the actual bone fragments or the diploic space, leading to collection of blood in the epidural location.

Characteristic of epidural hematoma is an isolated injury to the skull, dura, and dural vessels that leads to hematoma formation. In most of these acceleration-deceleration injuries, the skull has sustained the major impact forces and absorbed the resultant kinetic energy. The heavy force delivered to the cranium and transmitted to the brain often disrupts the state of consciousness. In contrast with other injuries, such as subdural hematoma, in which the brain often sustains a primary and major injury, epidural hematoma is often not associated with primary brain injury.

Another important distinguishing feature of this clinical entity is a lucid interval. The lucid interval occurs when a substantial blow has been transmitted to the cranium and causes the person to lose consciousness. The patient may subsequently appear asymptomatic and have a normal neurologic examination. The problem arises when an injury to the skull or dural vessels or both leads to a slow accumulation of blood in the epidural space. This hematoma outside the brain may remain relatively asymptomatic until it reaches a critically large size and compresses the underlying brain.[19] The compression can be transmitted to the brain stem and rapidly progress to neurologic dysfunction, brain herniation, and possibly death.[20] Any patient or athlete who has sustained a significant head impact should be observed in the awake state and not allowed to retire for sleep until the longer lasting effects of the head impact are known. Any patient with a significant LOC (minutes) or neurologic abnormality should have a more thorough medical evaluation, including CT scanning. The clinical manifestations of epidural hematoma depend on the type and amount of energy transferred, the time course of the hematoma formation, and the presence of simultaneous brain injuries. Often, the size of the hematoma determines the clinical effects. In addition to a lucid interval, patients with an epidural hematoma may present with no LOC, persistent unconsciousness, or any variation of these features.[21]

Subdural Hematoma

This form of intracranial hemorrhage has been divided into acute subdural hematoma, which presents within 48 to 72 hours after injury, and chronic subdural hematoma, which occurs in a later time frame with more variable clinical manifestations. An acute subdural hematoma is the most common major head injury and is associated with severe neurologic disability and death in many patients. Acute subdural hematoma results from bleeding within the subdural space as a result of stretching and tearing of the subdural veins. These veins drain from the cerebral surface and connect to the dura or dural sinuses. In addition, the bone irregularities of the middle cranial fossa, sphenoid bone, and frontal fossa form a rough surface over which inferior cortical surface contusions can form, resulting in hemorrhage in the subdural space.

A subdural hematoma may occur as an isolated collection of blood within the subdural space or as a more complicated hematoma associated with brain parenchymal injury. Many patients with complicated acute subdural hematomas sustain diffuse irreversible brain damage and do not improve after evacuation of the hematoma, the latter representing an epiphenomenon in the injury process. The outcome of subdural hematoma is thus often influenced by the extent of the concomitant parenchymal brain injury more than the subdural hematoma collection per se.[22]

The clinical presentation of any patient, including an athlete, with acute subdural hematoma can vary and includes those who are awake and alert with no focal neurologic deficits, but typically patients with any sizable acute subdural hematoma have a significant neurologic deficit (Figure 4). This may consist of alteration of consciousness, often to a state of coma or major focal neurologic deficit. Skull fracture is much less commonly associated with subdural hematoma than with epidural hematoma. Football players with 2 mild concussions without LOC, separated by 7 and 10 days, sustained acute subdural hematomas.[23]

A chronic subdural hematoma is defined as a hematoma present 3 weeks or more after a traumatic injury. The pathogenesis of chronic subdural hematoma involves an injury that results in bleeding into the subdural space. The initial hemorrhage may be a small amount that fails to generate significant brain compression. However, bleeding or oozing of blood into the subdural space may continue.[24] After 1 week, a chron-

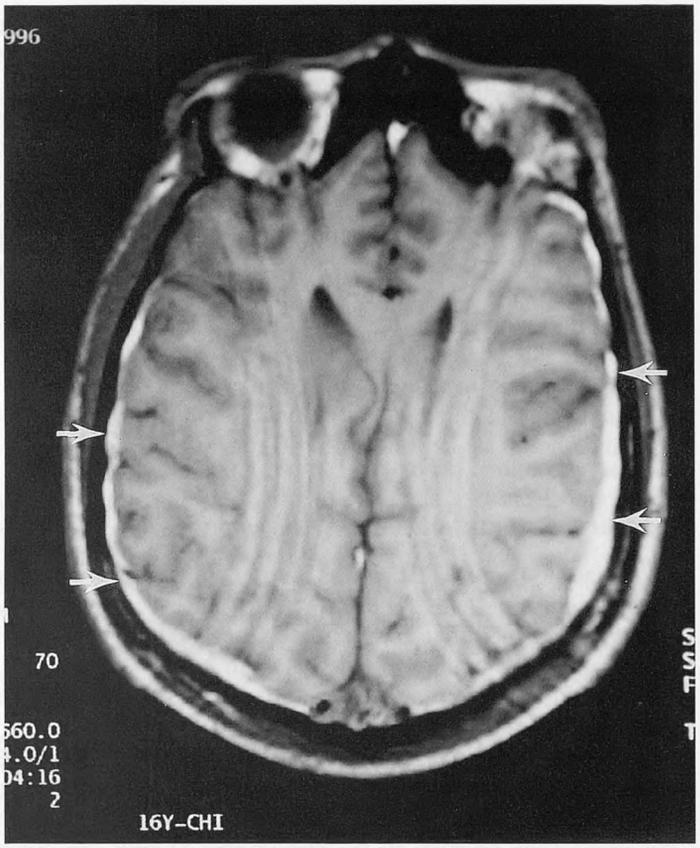

Figure 4. MRI of a high school football player with persistent headaches and poor school performance. Although the CT scan was normal, the MRI demonstrated small bilateral subdural hematomas, thus documenting the presence of an injury.

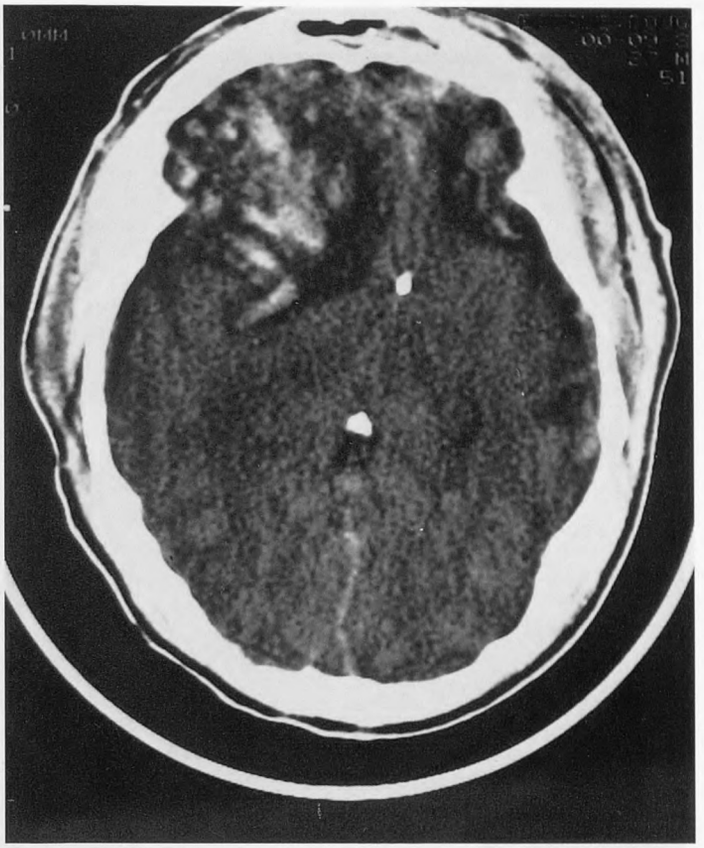

Figure 5. CT scan in a patient with bifrontal (right greater than left) hemorrhagic contusions. This patient presented in a coma after an acceleration-deceleration injury and did not undergo surgery but instead was managed for increased intracranial pressure.

ic subdural hematoma involves infiltration of fibroblasts to organize into an outer membrane. Subsequently, an inner membrane may form, and this encapsulated hematoma may become a dynamic osmotic membrane that interacts with the production and absorption of cerebrospinal fluid. Effusion of protein may occur, setting up an active process within the membrane.[25]

The diagnosis of chronic subdural hematoma is often difficult because of the protean clinical manifestations. Patients may have clinical symptoms suggestive of increased intracranial pressure, mental disturbance such as personality change or even dementia, symptoms with focal transient neurologic deficits similar to transient ischemic attacks, a meningeal syndrome with nuchal rigidity and photophobia, a clinical course with a slow progression of neurologic signs reminiscent of cerebral neoplasm, or a progressive and severe headache syndrome only.[26] Although not common in athletes, chronic subdural hematoma must always be the differential diagnosis, especially in those presenting with a remote history of head impact. The diagnosis is confirmed by CT scanning demonstrating the extra-axial low-density fluid collection in the subdural space.

Intracerebral Hemorrhage

A cerebral contusion is a heterogeneous zone of brain damage that consists of hemorrhage, cerebral infarction, necrosis, and edema. Cerebral contusion is a frequent sequela of head injury and in some studies represents the most common traumatic lesion of the brain visualized on radiographic evaluation. Contusions occur most often as a result of acceleration-deceleration mechanisms from the inward deformation of the skull at the impact site. This results in transient compression of the brain against the skull and the focal area of parenchymal injury. This energy is conducted to the underlying brain, resulting in cerebral contusion, the degree of which depends on the energy transmitted, the area of contact, the involved area of the cranium, and other factors (Figure 5).[27]

Contusions can vary from small, localized areas of injury to large, extensive areas of involvement. A cerebral contusion injury can evolve over hours and days after the injury. Multiple small areas of contusions may coalesce into a large area resembling a lesion, more accurately termed intraparenchymal hemorrhage. Injuries remote from the site of cranial impact may also occur. The direct, or coup, lesion results from injury at the impact site, and the remote, or contrecoup, lesion occurs as the opposite side of the brain rebounds against the skull or because of vacuum phenomena existing within the parenchyma at that location. The contrecoup lesion results in a hemorrhagic lesion in the cerebral tissue directly opposite the impact site, typically at the inferior surfaces of the frontal and temporal lobes. Contusions are often multiple and are frequently associated with other extra-axial and intra-axial hemorrhagic lesions.

The clinical course of patients with cerebral contusion varies greatly, depending on the location, number, and extent of the hemorrhagic contusion lesions. The patient may present with essentially normal function or may experience any type of neurologic deterioration, including coma. Frequently, behavioral or mental status changes exist due to involvement of the fron-

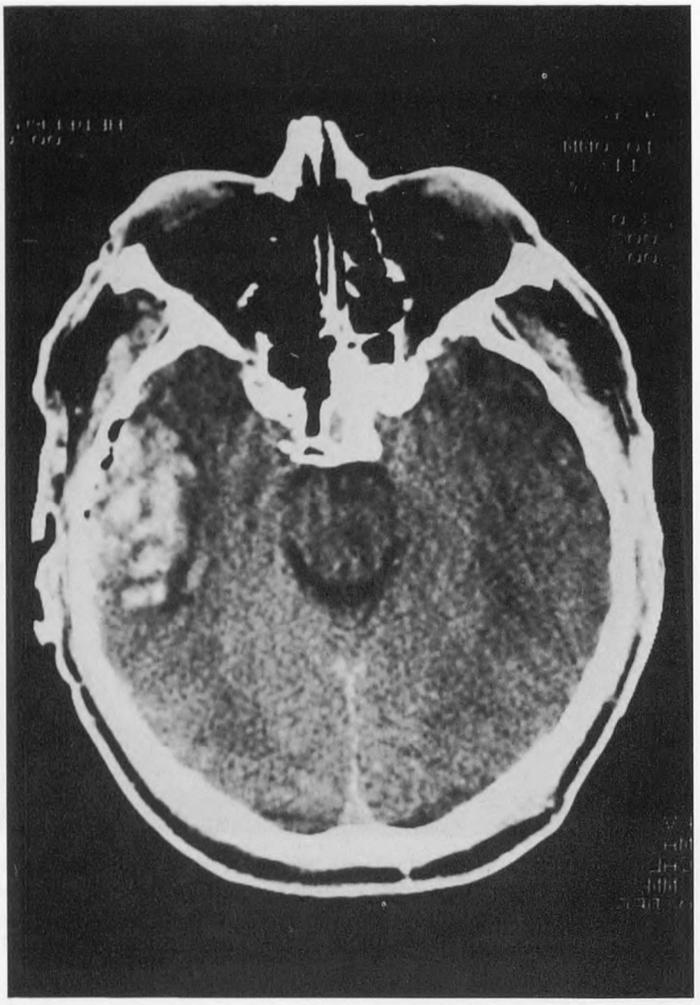

Figure 6. CT scan showing right temporal lobe contusions. These contusions tend to enlarge and lead to brain swelling.

tal or temporal lobes. The diagnosis of cerebral contusion is firmly established by CT scanning, which is also useful for following patients as the lesions evolve throughout their clinical course (Figure 6).

Intracerebral hematoma is a parenchymatous hemorrhage that is often similar in pathophysiology and radiographic appearance to a cerebral contusion. An intracerebral hematoma represents a localized collection of blood within the brain. The distinction between a hemorrhagic contusion and intraparenchymal hematoma depends upon the latter being recognized as a confluent area of homogeneous bleeding within the brain. Intracerebral hematomas usually present with a focal neurologic deficit but may progress to further neurologic deterioration, including coma and death resulting from brain herniation syndromes. This diagnosis is readily made by CT scanning, which shows a hyperdense, localized collection of blood. Intracerebral hematomas have been, along with subdural hematomas, the most common cause of sport-related lethal brain injuries.

Another entity, delayed traumatic intracerebral hematoma, is a clot that forms hours to days after the initial trauma. Although most frequently seen in the older population, it must always be borne in mind when evaluating and attending to any patient who has sustained a significant head impact. The athlete is also at risk because these hematomas are seen more commonly when there has been rotational head trauma. Delayed traumatic hematomas are believed to be due to later bleeding into an already contused region of the brain, to vascular injury, or to the development of a coagulopathy.

Finally, there are severe brain injuries in which the patients do not sustain any form of hematoma or mass effect but instead have a global or diffuse brain insult. This often results in widespread cerebral edema and elevated intracranial pressure (ICP), similar to the pathophysiologic response seen in the second-impact syndrome. In the absence of an operable, space-occupying lesion, the patient is often treated for increased ICP with online ICP monitoring. This is performed by placing a catheter in the brain parenchyma or ventricles, providing a constant measure of ICP. The cerebral perfusion pressure is defined as the mean systemic blood pressure minus the ICP and should be kept at 50 to 70 mm Hg or higher.

Cerebral Concussion

Concussion, or MTBI, is the most common form of head injury seen in athletes. The Centers for Disease Control[28] recently proclaimed that approximately 300 000 cases of MTBI occur annually in the United States from athletic activities. Powell and Barber-Foss[8] estimated 62 000 cases of concussion in American high school sports annually.

The classical cerebral concussion is defined as a posttraumatic state that results in LOC, with the patient regaining full return of consciousness within 24 hours. Clinically, concussion has often been referred to as physiologic without anatomical disruption in the brain. Newer research has shown both anatomic and biochemical markers of definitive injury secondary to MTBI. Concussion has also been defined as an immediate and transient impairment of neural function, such as alteration of consciousness, disturbance of vision or equilibrium, and other similar symptoms. We now appreciate that concussion symptoms are related to either cerebral cortical dysfunction, such as confusion, disorientation, memory, or information-processing abnormalities, and others, or to brain stem abnormalities. Examples of the latter include the unconscious state, visual or auditory symptoms, nausea and vomiting, ataxia, and incoordination. Some of these patient complaints may also relate to injury to and dysfunction of the inner ear or vestibular mechanism. Sleep disturbances are common and often contribute greatly to the symptom complex.

We have gained a greater appreciation for the importance of having accurate methods for classifying MTBI. This recognition has led to the development of several different classification schemes, unfortunately leading to some uncertainty at times over nomenclature. One must keep in mind that all scales thus far published were formulated based on experiential theory and not on scientifically proven research. No prospective, randomized clinical trial has established any particular concussion classification system as superior or proven. Rather, based on years of experience and observation, these guidelines have been employed as our best and most accurate means of currently categorizing athletic MTBI. The National Athletic Trainers' Association has not adapted or endorsed any one grading scale or set of return-to-play guidelines, nor have most organizations. The best grading scale and return-to-play guidelines will be based on scientific evidence, but most importantly, a systematic evaluation and management scheme must be followed.

During the last 2 decades, no fewer than 8 classification systems have been proposed for management of sport-related

concussions.[4,29–32] While disagreement and differences existed regarding terminology and symptom priority, these efforts have led to the recognition of the importance of standardization in the care of the concussed athlete. In recent years, 3 concussion classification schemes have been accepted in widespread application. In 1986, Cantu[29] proposed a concussion grading scale in which the persistence of memory disturbance, termed amnesia, is given greater emphasis. The Colorado[32] guidelines and the American Academy of Neurology (AAN)[31] practice parameters similarly place any athlete with any period of LOC, no matter how brief, within the most severe grade of concussion (Table).

CONCUSSION CLASSIFICATION

A mild concussion (grade 1) is most commonly seen in athletes. The Colorado guidelines define a grade 1 concussion as no LOC, with confusion being the hallmark sign.[32,33] The Cantu definition[29] of a mild concussion is one without LOC, with confusion alone or a brief (less than 30 minutes) period of amnesia. The AAN practice parameters[31] classify a grade 1 concussion as having no LOC and mental status abnormalities that resolve in less than 15 minutes. This type of concussion is not infrequent in football games, occurring in at least 1 player in nearly every game. If thoroughly searched for, a grade 1 concussion is often found. It may be stated by nonmedical personnel that the player was "dinged." The athlete, who is awake and alert, may be able to function unnoticed during the course of the athletic contest. If significant disorientation, confusion, memory disturbance, dizziness, headache, or any neurologic abnormality persists after the 15-minute observation period, the athlete has more than a mild concussion. We should remember that concussion may be present and significant without the person's sustaining a LOC. Ommaya and Gennarelli[4] showed in their animal model that 3 of 6 grades of concussion did not involve LOC. They postulated that, unless shearing forces reached the reticular activating system within the midbrain and brain stem, cortical and subcortical structures could be affected to produce amnesia and confusion but not LOC.

The Colorado guidelines classify a moderate or grade 2 concussion as associated with the development of amnesia either initially or during the period of observation; there is no LOC.[32] The athlete is removed from competition and not allowed to return. Cantu[29] defined the moderate concussion as less than 5 minutes of unconsciousness or posttraumatic amnesia for longer than 30 minutes but less than 24 hours' duration, while the AAN system[31] specified no LOC but only mental status changes lasting longer than 15 minutes.

A severe or grade 3 concussion is defined as a player having LOC by the Colorado and AAN guidelines.[31,32] Cantu[29] termed a severe or grade 3 concussion as one having greater than a 5-minute period of unconsciousness or 24 hours of posttraumatic amnesia. This patient may require emergent transport to the nearest hospital facility with CT scanning and, if indicated, consideration should be given for neurosurgical consultation. The possibility of concomitant cervical spine injury must always be considered in an unconscious patient and transport performed with cervical immobilization and maintenance of an adequate airway.

EVALUATION AND MANAGEMENT

The goals in evaluating the potentially head-injured athlete are threefold: (1) the fact that a head injury has potentially occurred must be recognized; (2) the athletes requiring transport to a medical facility for further workup and treatment must be accurately identified; and (3) a decision must be made regarding when the athlete may return to competition. Obviously, detection of a potentially life-threatening or neurologically devastating injury is of paramount importance. In light of the possibility of second-impact syndrome, those athletes with mild head injuries must be cautiously observed and returned to play only when they are absolutely symptom free.

Any athlete who receives a blow to the head or any significant acceleration-deceleration-type force to the head should be presumed to have a possible head injury and should be thoroughly evaluated. The athlete should be evaluated for level of consciousness, steadiness of gait, orientation, and posttraumatic amnesia. Those players with grade 1 concussions generally should be observed for 20 to 30 minutes. If the sensorium totally clears, the neurologic examination is normal, and no residual symptoms are present, the athlete can usually be allowed to return to the game. Any persistent symptoms, such as headache, dizziness, or confusion, necessitate removal from competition and evaluation by a physician. An athlete with a grade 2 concussion should be removed from the game. Evaluation by a physician should be performed in most cases.[17] In grade 3 concussion, the athlete's level of consciousness or orientation may be altered severely or for a prolonged time, or other neurologic deficits may be present. Transport to a hospital for emergency evaluation of the player by a neurosurgeon and diagnostic neuroimaging should be undertaken immediately if indicated. A cervical collar and long spine board should be used to prevent secondary injury to the player until spinal involvement can be definitely excluded. The unconscious athlete must be treated as any other unconscious patient. The airway must be firmly established and adequate breathing ensured. Airway protection may occasionally require emergent intubation. Hemodynamic monitoring is needed to ensure adequate circulation. Any focal neurologic changes, such as posturing or a dilated pupil, can be addressed with moderate hyperventilation.

The role of the certified athletic trainer during games and practices is to prevent injury and provide immediate first-aid care and triage. One of the more challenging aspects of game and practice coverage is the response to injuries involving the head and cervical spine. Knowledge concerning the clinical presentation and proper emergency care for these players with serious or catastrophic intracranial injuries is requisite for athletic trainers and medical personnel.

Before the season, the athletic training staff creates an emergency medical plan, instituting all procedures that must be followed during an emergency injury. The various entities making up the institutional medical team, the secondary support teams including local emergency medical services, ambulance units, and level 1 trauma centers (including helicopter transport), should be listed. Policies should go through the proper administrative protocols before being instituted. Also, all equipment to be used on a daily basis must be inspected and tested before each season, and in some instances, such equipment must be recalibrated annually.

The certified athletic trainer is trained in evaluation and screening skills and competencies in treating catastrophic head and spine injury. Initially, after a traumatic injury on the field, the athletic trainer performs an immediate basic life support evaluation to assess airway, breathing, and circulation. Then the secondary assessment, including neurologic and orthopae-

Concussion Management

Classification	Grade	Signs/Symptoms*	First Concussion	Second Concussion	Third Concussion
Colorado Medical Society Guidelines[32]†	1 (mild)	Confusion without amnesia; no loss of consciousness	May return to play if without symptoms for at least 20 minutes	Terminate contest or practice; may return to play if without symptoms for at least 1 week	Terminate season or may return to play in 3 months if without symptoms
	2 (moderate)	Confusion with amnesia; no loss of consciousness	Terminate contest or practice; may return to play if without symptoms for at least 1 week	Consider terminating season; may return to play in 1 month if without symptoms	Terminate season; may return to play next season if without symptoms
	3 (severe)	Loss of consciousness	Terminate contest or practice and transport to hospital; may return to play in 1 month, after 2 consecutive weeks without symptoms	Terminate season; may return to play next season if without symptoms	Terminate season; strongly discourage return to contact or collision sports
Cantu Grading System[29]‡	1 (mild)	No loss of consciousness; posttraumatic amnesia less than 30 minutes in duration	May return to play if asymptomatic for 1 week§; terminate season if CT or MRI abnormality	Return to play in 2 weeks if asymptomatic at the time for 1 week	Terminate season; may return to play next season if asymptomatic
	2 (moderate)	Loss of consciousness less than 5 minutes in duration or posttraumatic amnesia longer than 30 minutes but less than 24 hours in duration	Return to play after asymptomatic for 2 weeks§; terminate season if CT or MRI abnormality	Minimum of 1 month; may return to play then if asymptomatic for 1 week; consider terminating season	Terminate season; may return to play next season if asymptomatic
	3 (severe)	Loss of consciousness for more than 5 minutes or posttraumatic amnesia for more than 24 hours	Minimum of 1 month; may then return to play if asymptomatic for 1 week§	Terminate season; may return to play next season if asymptomatic	Consider no further contact sports
American Academy of Neurology Guidelines[31]‖	1 (mild)	Transient confusion; no loss of consciousness; symptoms or abnormalities resolve in less than 15 minutes	Remove from contest; may return to play if abnormalities or symptoms clear within 15 minutes	Terminate contest; may return to play after 1 week without symptoms at rest and with exercise	
	2 (moderate)	Transient confusion; no loss of consciousness; symptoms or abnormalities last more than 15 minutes	Terminate contest; may return to play after 1 full asymptomatic week at rest and with exertion§	Terminate contest; may return to play after at least 2 asymptomatic weeks at rest and with exertion; terminate season if any CT or MRI abnormality	
	3 (severe)	Any loss of consciousness, either brief (seconds) or prolonged (minutes)	Terminate contest; transport to hospital if unconscious or neurologic abnormality§; if concussion brief (seconds) may return in 1 week if no symptoms at rest and with exertion; if concussion prolonged (minutes) return in 2 weeks, no symptoms	Terminate contest; may return to play after minimum of 1 month asymptomatic†; terminate season if any CT or MRI abnormalities	

*Postconcussion symptoms, mental status abnormalities.
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‡Reprinted with permission of McGraw-Hill, Inc. Cantu RC. Guidelines for return to contact sports after a cerebral concussion. *Phys. Sportsmed.* 1986; 14(10):75–83.
§CT or MRI recommended if symptoms or signs persist.
‖Reprinted with permission of Lippincott Williams & Wilkins. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. *Neurology.* 1997; 48:575–580.
Table reprinted with permission from KM Guskiewicz, "Concussion in sport: the grading-system dilemma," *Athletic Therapy Today,* 2001:6(1); 18–27.

dic evaluation, takes place. In the conscious athlete, responses to questions allow for immediate information to be derived. The unconscious athlete creates a more complex and urgent evaluation.

The movement of an athlete from a prone or side-lying position requires teamwork among all members of the immediate and extended medical staff. The log-roll procedure remains the most accepted protocol in moving an athlete with a suspected head or cervical spine injury. The senior staff member should maintain the head position, while the other team members roll the athlete's head and body in unison to a supine position. The National Athletic Trainers' Association in conjunction with the Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete[34] recently recommended that the facemask be removed, regardless of the athlete's current respiratory status, and that the helmet remain on the athlete unless it does not allow for immobilization of the head, the facemask cannot be safely removed, or the helmet interferes with immobilization during transport. The task force also recommended that the medical staff have sufficient practice in all aspects of emergency care before an incident.

Thus, it is important for caregivers at athletic contests to be cognizant of the spectrum of injuries that occur in response to both minor and major brain trauma. In addition to life-threatening hematomas, diffuse brain injury is common and can range from mild to severe. However, the former, as we are increasingly appreciating, may have more implications in terms of numbers of athletes affected, difficulty with diagnosis and classification, potential for long-term symptoms, and complexity with regard to return-to-play decisions.

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Posttraumatic Retrograde and Anterograde Amnesia: Pathophysiology and Implications in Grading and Safe Return to Play

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Objective: The presence of posttraumatic amnesia (PTA) and loss of consciousness have been main factors used in a number of concussion guidelines. In this article, the focus is on using PTA (both retrograde and anterograde) as salient indicators of traumatic brain injury severity and the most reliable index of outcome prediction, even in mild cases.

Data Sources: A MEDLINE search for the years 1990–2000 using the key words *posttraumatic retrograde* and *anterograde amnesia*, *concussion* and *mild traumatic brain injury* was done.

Data Synthesis: On-the-field testing of PTA is a salient and integral component of the initial and follow-up neurologic assessments of the head-injured athlete.

Conclusions/Recommendations: Initial and follow-up assessments of PTA, anterograde and retrograde, are an essential part of the neurologic evaluation of the head-injured athlete. Increasingly, neuropsychological testing, including computer models, is being employed in this assessment. The importance of not just PTA but all postconcussion signs and symptoms being absent at rest and exertion before allowing the athlete to return to play is emphasized.

Key Words: concussion, mild traumatic brain injury, athletic injury

Concussion is derived from the Latin word *concussus*, which means to shake violently. Initially, it was thought to produce only a temporary disturbance of brain function due to neuronal, chemical, or neuroelectrical changes without gross structural change. We now know that structural damage with loss of brain cells does occur with some concussions. In the last several years, the neurobiology of cerebral concussion has been advanced predominantly in animal studies but also in studies in man as well. It has become clear that, in the minutes to days after concussive brain injury, brain cells that are not irreversibly destroyed remain alive but in a vulnerable state. These cells are particularly vulnerable to minor changes in cerebral blood flow, increases in intracranial pressure, and especially anoxia. Animal studies have shown that, during this period of vulnerability, which may last as long as a week with a minor head injury such as a concussion, a minor reduction in cerebral blood flow that would normally be well tolerated now produces extensive neuronal cell loss.[1–5] This vulnerability appears to be due to an uncoupling of the demand for glucose, which is increased after injury, with a relative reduction in cerebral blood flow. While the precise mechanisms of this dysfunction are still not fully understood, it is now clear that, although concussion in and of itself may not produce extensive neuronal damage, the surviving cells are in a state of vulnerability characterized by a metabolic dysfunction, which can be thought of as a breakdown between energy demand and production. Precisely how long this period of metabolic dysfunction lasts is not presently fully understood. Unfortunately, there are today no neuroanatomic or physiologic measurements that can be used to precisely determine the extent of injury in concussion or the severity of metabolic dysfunction or precisely when it has cleared. It is this fact that makes return-to-play decisions after a concussion a clinical judgment.

Team physicians, athletic trainers, and other medical personnel responsible for the medical care of athletes face no more challenging problem than the recognition and management of concussion. Indeed, such injuries have captured many headlines in recent years and have spurred studies within both the National Football League and the National Hockey League.

When discussing concussion, we must realize that there is no universal agreement on the definition and grading of concussion.[1,6–8] Tables 1–8 present different attempts at grading concussion, all focusing on loss or nonloss of consciousness and posttraumatic amnesia (PTA) as hallmarks in the grading schemes. Furthermore, they may not give enough attention to the other signs and symptoms of concussion. As we all know, a patient with concussion may display any combination of the following signs and symptoms: a feeling of being stunned or seeing bright lights, brief loss of consciousness, lightheadedness, vertigo, loss of balance, headaches, cognitive and memory dysfunction, tinnitus, blurred vision, difficulty concentrating, lethargy, fatigue, personality changes, inability to perform daily activities, sleep disturbances, and motor or sensory symptoms.

Presently, there is no universal agreement that PTA is a better

Table 1. Cantu Grading System for Concussion[9]*

Grade 1	No loss of consciousness; posttraumatic amnesia less than 30 minutes
Grade 2	Loss of consciousness less than 5 minutes in duration or posttraumatic amnesia lasting longer than 30 minutes but less than 24 hours in duration
Grade 3	Loss of consciousness for more than 5 minutes or posttraumatic amnesia for more than 24 hours

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Table 2. Colorado Medical Society Grading System for Concussion[10]

Grade 1	Confusion without amnesia; no loss of consciousness
Grade 2	Confusion with amnesia; no loss of consciousness
Grade 3	Loss of consciousness

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Table 3. AAN Practice Parameter (Kelly and Rosenberg) Grading System for Concussion[6]

Grade 1	Transient confusion; no loss of consciousness; concussion symptoms or mental status abnormalities on examination resolve in less than 15 minutes
Grade 2	Transient confusion; no loss of consciousness; concussion symptoms or mental status abnormalities on examination last more than 15 minutes
Grade 3	Any loss of consciousness, either brief (seconds) or prolonged (minutes)

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Table 4. Jordan Grading System for Concussion[11]

Grade 1	Confusion without amnesia; no loss of consciousness
Grade 2	Confusion with amnesia lasting less than 24 hours; no loss of consciousness
Grade 3	Loss of consciousness with an altered level of consciousness not exceeding 2 to 3 minutes; posttraumatic amnesia lasting more than 24 hours
Grade 4	Loss of consciousness with an altered level of consciousness exceeding 2 to 3 minutes

Table 5. Ommaya Grading System for Concussion[12]

Grade 1	Confusion without amnesia (stunned)
Grade 2	Amnesia without coma
Grade 3	Coma lasting less than 6 hours (includes classic cerebral concussion, minor and moderate head injuries)
Grade 4	Coma lasting 6 to 24 hours (severe head injuries)
Grade 5	Coma lasting more than 24 hours (severe head injuries)
Grade 6	Coma, death within 24 hours (fatal head injuries)

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Table 6. Nelson Grading System for Concussion[7]

Grade 0	Head struck or moved rapidly; not stunned or dazed initially; subsequently complains of headache and difficulty in concentrating
Grade 1	Stunned or dazed initially; no loss of consciousness or amnesia; sensorium clears in less than 1 minute
Grade 2	Headache; cloudy sensorium longer than 1 minute in duration; no loss of consciousness; may have tinnitus or amnesia; may be irritable, hyperexcitable, confused, or dizzy
Grade 3	Loss of consciousness less than 1 minute in duration; no coma (arousable with noxious stimuli); demonstrates grade 2 symptoms during recovery
Grade 4	Loss of consciousness for more than 1 minute; no coma; demonstrates grade 2 symptoms during recovery

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Table 7. Roberts Grading System for Concussion[8]

Bell ringer	No loss of consciousness; no posttraumatic amnesia; symptoms less than 10 minutes
Grade 1	No loss of consciousness; posttraumatic amnesia less than 30 minutes; symptoms greater than 10 minutes
Grade 2	Loss of consciousness less than 5 minutes; posttraumatic amnesia greater than 30 minutes
Grade 3	Loss of consciousness greater than 5 minutes; posttraumatic amnesia greater than 24 hours

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Table 8. Torg Grading System for Concussion[13]

Grade 1	"Bell rung"; short-term confusion; unsteady gait; dazed appearance; no amnesia
Grade 2	Posttraumatic amnesia only; vertigo; no loss of consciousness
Grade 3	Posttraumatic retrograde amnesia; vertigo; no loss of consciousness
Grade 4	Immediate transient loss of consciousness
Grade 5	Paralytic coma; cardiorespiratory arrest
Grade 6	Death

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or more sensitive predictor of outcome after traumatic brain injury than depth and duration of unconsciousness,[14–16] but many consider the duration of PTA the best indicator of traumatic brain injury severity[15,17] and the most dependable marker of outcome prediction,[18–25] even in mild cases.[16,26] While variously described by different investigators, PTA includes impaired orientation, that is, retrograde amnesia and anterograde amnesia.[22,27–29] Recently, some investigators[16,30–32] have suggested that PTA might better be called posttraumatic confusional state.

Posttraumatic amnesia may be divided into 2 types. The first type of PTA is retrograde, defined by Cartlidge and Shaw[23] as a "partial or total loss of the ability to recall events that have occurred during the period immediately preceding brain injury." The duration of retrograde amnesia usually progressively decreases. The second type of PTA is anterograde amnesia, a deficit in forming new memory after the accident, which may lead to decreased attention and inaccurate perception. Anterograde memory is frequently the last function to return after the recovery from loss of consciousness.[33]

Memory and new learning are believed to involve the cerebral cortex, subcortical projections, hippocampal formation (gyrus dentatus, hippocampus, and parahippocampal gyri), and the diencephalons, especially the medial portions of the dorsomedial and adjacent midline nuclei of the thalamus.[34] In addition, frontal lobe lesions may cause alterations in behavior, including irritability, aggressiveness, and loss of inhibition and judgment. Recently, evidence has been presented that the right frontal lobe plays a prominent role in sustained attention.[35]

The lack of a universal definition or grading scheme for

Table 9. Evidence-Based Cantu Grading System for Concussion

Grade 1 (mild)	No loss of consciousness; posttraumatic amnesia* or postconcussion signs or symptoms lasting less than 30 minutes
Grade 2 (moderate)	Loss of consciousness lasting less than 1 minute; posttraumatic amnesia* or postconcussion signs or symptoms lasting longer than 30 minutes but less than 24 hours
Grade 3 (severe)	Loss of consciousness lasting more than 1 minute or posttraumatic amnesia* lasting longer than 24 hours; postconcussion signs or symptoms lasting longer than 7 days

*Retrograde and anterograde.

concussion renders the evaluation of epidemiologic data extremely difficult. As a neurosurgeon and team physician, I have evaluated many football players who suffered a concussion. Most of these injuries were mild, not involving loss of consciousness, and were associated with PTA, which was helpful in making the diagnosis, especially in mild cases.

I developed a practical scheme for grading concussion severity based on the duration of unconsciousness or PTA (or both), which has worked well on the field and sideline (see Table 1). The most mild concussion (grade 1) occurs without loss of consciousness, and the only neurologic deficit is a brief period of posttraumatic confusion or PTA, which, by definition, lasts less than 30 minutes. The moderate (grade 2) concussion is usually associated with a brief period of unconsciousness, by definition, not exceeding 5 minutes. Less commonly, consciousness is not lost; the athlete instead experiences a protracted period of PTA lasting more than 30 minutes but less than 24 hours. A severe (grade 3) concussion occurs with a more protracted period of unconsciousness lasting longer than 5 minutes. Rarely, it may occur without a loss of consciousness or with a shorter period of unconsciousness but with a very protracted period of PTA lasting more than 24 hours. In reality, prospective studies over the last several years have shown that virtually all concussions are grade 3 by this guideline because of PTA lasting longer than 24 hours (D. Erlanger, unpublished data, 2000). A protracted period of unconsciousness lasting more than 5 minutes is almost never seen on athletic fields; most periods of unconsciousness last seconds to a minute. Prospective studies over the last 10 years have demonstrated a correlation between the duration of postconcussive symptoms and PTA and abnormal results on neuropsychological tests. Therefore, I present an evidence-based modification of the original Cantu guidelines[9] (Table 9).

When checking for orientation and retrograde amnesia on the field, asking the athlete the current quarter, the score, what happened, and the names of the current and last week's opponents is useful. When checking for attention or anterograde amnesia deficits, useful tests are repeating 4 words immediately and 2 minutes later, repeating 5 numbers forward and especially backward, and repeating months of the year backward.

Recently, computer-administered minineuropsychological tests have been proposed as a more feasible way to conduct group baseline assessments,[36,37] as well as a personal digital assistant version that can be connected to the Internet.[38,39]

Thus, while not yet the standard of care, neuropsychological tests (with a preseason baseline and serial postconcussion assessments) are assisting clinicians in concussion management, including return-to-play decisions.

Whether an athlete has been unconscious is, of course, important. It is generally believed that the degree of brain injury sustained is indicated by the depth and duration of coma.[40–42] However, the coma referred to by these authors is not the seconds to minutes usually seen on the athletic field but rather hours' or days' duration. Thus, while not diminishing the importance of being rendered unconscious, I find it illogical to grade a concussion that produces postconcussion symptoms lasting months or years without loss of consciousness as less severe than a concussion resulting in brief unconsciousness and resolution of all postconcussion symptoms within a few minutes or hours. Brett Lindros, Al Toon, Jim Miller, Steve Young, and Merrill Hodge are professional athletes whose careers were ended by concussions without loss of consciousness that produced sustained postconcussion symptoms. We know these athletes consider their concussions very severe.

RETURN TO COMPETITION AFTER CONCUSSION

A sobering realization is that the ability to process information may be reduced after a concussion, and the severity and duration of functional impairment may be greater with repeated concussions.[43–45] Studies clearly suggest that the damaging effects of the shearing injury to nerve fibers and neurons are proportional to the degree to which the head is accelerated and that these changes may be cumulative.[46–48] Once a player has incurred an initial cerebral concussion, his or her chances of incurring a second one are 3 to 6 times greater than for an athlete who has never sustained a concussion.[48–51]

Table 10 presents guidelines for return to play after a concussion, including termination of a season. Before an athlete returns to play, he or she must not only be free of PTA symptoms but also of all postconcussion symptoms at rest and exertion. All the guidelines agree on this salient point. Table 11 is a postconcussion signs and symptoms checklist I have found useful. Thus, while it is a clinical decision as to when to return an athlete to play after a concussion, to return an athlete with postconcussion symptoms risks not only cumulative brain in-

Table 10. Guidelines for Return to Play After Concussion*

	First Concussion	Second Concussion	Third Concussion
Grade 1 (mild)	May return to play if asymptomatic for 1 week	Return to play in 2 weeks if asymptomatic for 1 week	Terminate season; may return to play next season if asymptomatic
Grade 2 (moderate)	Return to play after asymptomatic for 1 week	Minimum of 1 month; may return to play then if asymptomatic for 1 week; consider terminating season	Terminate season; may return to play next season if asymptomatic
Grade 3 (severe)	Minimum of 1 month; may then return to play if asymptomatic for 1 week	Terminate season; may return to play next season if asymptomatic	

*Asymptomatic in all cases means no postconcussion symptoms, including retrograde or anterograde amnesia, at rest or with exertion.

Table 11. Postconcussion Signs and Symptoms Checklist

	(Please check all that apply)	
	Initial Symptoms	Symptoms Today
Depression		
Dizziness		
Drowsiness		
Excess sleep		
Fatigue		
Feel "in fog"		
Feel "slowed down"		
Headache		
Irritability		
Memory problems		
Nausea		
Nervousness		
Numbness/tingling		
Poor balance		
Poor concentration		
Ringing in ears		
Sadness		
Sensitive to light		
Sensitivity to noise		
Trouble falling asleep		
Vomiting		

jury but the second-impact syndrome and would be against the recommendations of all current guidelines.

CONCLUSION

There is no universal agreement on concussion grading and return-to-play criteria after a concussion. There is, however, unanimous agreement that an athlete still suffering postconcussion symptoms at rest and exertion should not return to contact or collision sports. In this article, I present the logic for using the duration of posttraumatic amnesia (retrograde and especially anterograde) as a criterion to be employed in the grading of concussion severity.

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Loss of Consciousness: Pathophysiology and Implications in Grading and Safe Return to Play

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James P. Kelly, MD, provided conception and design; analysis and interpretation of the data; and drafting, critical revision, and final approval of the article.

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Objective: To provide historical background and current concepts regarding the importance of loss of consciousness (LOC) in the evaluation of concussion and athletes.

Data Sources: A MEDLINE search identified scientific and clinical articles on sport concussion management published from 1966 to present. Discussions were held with authors of selected reports. Recent research findings reported at national meetings were reviewed.

Data Synthesis: The relative importance of LOC in the evaluation of concussion was reviewed in light of scientific and clinical evidence in the literature. Comments made by authors of concussion grading scales were considered in the development of expert consensus statements.

Conclusions/Recommendations: The observation of LOC at the time of concussion must be viewed as reflecting a potentially worrisome traumatic brain injury. LOC is followed by more severe acute mental status abnormalities and carries a greater risk of intracranial pathology than concussion without LOC. Prolonged LOC represents a neurologic emergency, which may require neurosurgical intervention. Lingering symptoms of concussion, even without LOC, should be monitored closely and managed according to established guidelines for safe return to play.

Key Words: concussion, mild traumatic brain injury, sport concussion guidelines

Does an athlete's loss of consciousness (LOC) affect the management of sport-related concussion? Increasing attention surrounds the issue of concussion in sports and recreation in the medical literature,[1] as evidenced by the devotion of this entire issue of the *Journal of Athletic Training* to the topic. I will attempt to address the question, "In mild traumatic brain injury (MTBI), or more specifically, sport-related cerebral concussion, what is the importance of LOC?"

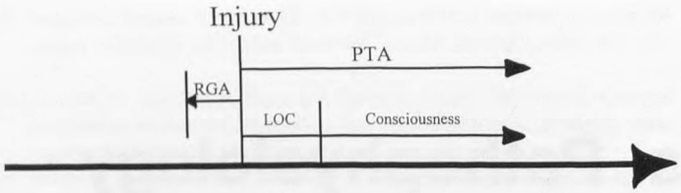
It is important to correct a common misconception that concussion only occurs if an individual is rendered unconscious. Health care professionals know that concussions often occur without LOC—especially in the world of athletics.[2] Temporary confusion or even a permanent gap in memory (posttraumatic amnesia or PTA) can occur without LOC.[3] This is true for concussion that occurs in any setting, not only in sports and recreation. While the focus of this article is concussion during athletic activity, any past medical history of concussion outside the sports arena must be considered when health care professionals make decisions regarding return to participation in sports.

When an individual sustains a concussion with amnesia, he or she often later assumes that there was a LOC; however, it is important for the health care professional to determine whether LOC actually occurred. An individual who has amnesia associated with the injury cannot observe himself or herself and is, by definition, an unreliable historian regarding the event itself. Anyone rendered unconscious also has an associated amnesia, both for the period of time while unconscious and also for some span of time thereafter, before an awareness of the surroundings and events returns (Figure). A brief period of memory loss or retrograde amnesia is common following LOC and sometimes occurs even without LOC.[4]

Concussion is diagnosed by determining what actually happened to the individual's brain function at the time of the injury.[2] This diagnosis is not made by neuroimaging studies, such as computed tomography (CT) scanning or magnetic resonance imaging (MRI) scanning, nor is it made by neuropsychological testing performed days or weeks later. The examination of the athlete at the time of the injury or detailed accounts of the signs and symptoms at the time of the injury are much more valuable in formulating a diagnosis of concussion. In the sport setting, eyewitness reports or videotape evidence of the injury are often available and helpful in the neurologic assessment of the athlete.

Pathophysiology of Loss of Consciousness

It is important to distinguish between unconsciousness from a neurologic cause and other uses of the term "unconsciousness" in psychology or philosophy. Whereas psychological unconsciousness refers to a state of unawareness or repressed ideas,[5] neurologic unconsciousness is paralytic coma.[6] This neurologic state represents a form of brain dysfunction involving either the hemispheres or the deep structures of the brain (including the reticular activating system, which governs sleep and wake cycles[7]), or both. In the neurologic uncon-



Time course of concussion. RGA = retrograde amnesia, LOC = loss of consciousness, PTA = posttraumatic amnesia.

Table 1. Scale of Severity of Traumatic Brain Injury Not Involving Loss of Consciousness

	Start	Progression To
I	Confusion	Normal consciousness without amnesia
II	Confusion	Normal consciousness with posttraumatic amnesia
III	Confusion	Normal consciousness with posttraumatic amnesia plus retrograde amnesia
IV	Coma (paralytic)	Level III: Normal consciousness with posttraumatic amnesia plus retrograde amnesia
V	Coma	Vegetation state or death
VI	Death	

Adapted with permission from Ommaya and Gennarelli.[6]

scious state, responses to the external world are primitive or reflexic and may be absent altogether. After severe traumatic brain injury, emergence from a coma into vegetative state does not change the fact that the individual is still unconscious, even though the eyes may be open. Only in the somewhat higher level of function known as the minimally conscious state do we see the beginning of neurologic consciousness and higher-level behaviors indicating an awareness of the external world.[8]

The Glasgow Coma Scale (GCS)[9] was created to assess the depth of coma from traumatic causes of unconsciousness. Early in the establishment of this scale and the associated Glasgow Outcome Scale (GOS),[10] it became clear that deeper levels of coma as measured by lower scores on the GCS typically carry a worse prognosis and poorer outcome on the GOS in adults[11] and children.[12] Lower GCS scores are associated with longer PTA,[13] and several studies have shown PTA to be the best single predictor of outcome from all severities of traumatic brain injury.[14,15]

From decades of animal experiments and human experience with traumatic brain injury, we know that concussion commonly occurs without LOC.[6,16,17] Periods of brief unconsciousness may be associated with concussion. Unconsciousness lasting longer than 30 minutes is thought to indicate a more serious form of brain injury than concussion.[18]

Important experimental animal work nearly 30 years ago by Ommaya and Gennarelli[6] demonstrated that 3 of 6 grades of traumatic brain injury severity could be determined with milder forms of brain injury not involving LOC (Table 1). The mildest form was that of transient confusion without LOC or amnesia. The second grade included confusion plus PTA without unconsciousness. The third grade included confusion and PTA plus retrograde amnesia, again without LOC. Grades IV through VI involve LOC associated with progressively worse neurologic outcomes. Within each of these grades, shearing strains on brain tissue produced lesions that were detected near the cortical surface in milder cases, while deeper lesions within the brain were associated with more severe biomechanical forces.

Neuroimaging studies such as MRI[19] and CT[20] scans are known to detect traumatic lesions within the brain, MRI scans being more sensitive than CT scans.[21,22] Individuals who are not rendered unconscious by head trauma are less likely to have subcortical contusions than those who are rendered unconscious.[20] The longer the period of unconsciousness, the deeper the location of the lesions detected by CT[23] and MRI scan.[20] In cases of relatively mild traumatic brain injury, focal lesions on CT or MRI scans predict worse outcome.[13,16] Brain lesions due to trauma have well-described neurobehavioral manifestations, some of which are seen during acute concussion as a result of diffuse damage primarily affecting frontal lobe functions, such as organization of thinking, information processing, decision making, planning, and carrying out complex tasks.[24]

External forces that affect head movement cause mechanical stress within the brain tissue, producing a sudden electric discharge or depolarization of nerve cells throughout the brain.[25] LOC can occur from this effect alone. This electric depolarization leads to an outpouring of neurotransmitters in the brain, and a cascade of neurochemical changes results in excitatory and damaging effects on the nerve cells. The subsequent metabolic rearrangement can be measured by sophisticated functional neuroimaging techniques such as positron emission tomography (PET) scanning.[26]

High-powered electron microscopes have been used to analyze changes in brain tissue after mild traumatic forces have been applied.[27] If these experimental animals are sacrificed immediately after the application of the traumatic force, the nerve cell projections known as axons appear normal; however, if the animal is allowed to survive the injury for several hours, axonal swelling and later degeneration occur. Neurochemical[28] and structural changes,[29,30] followed by the delayed effects of diffuse axonal injury,[27] are known to occur with mild traumatic brain injury. Even so, it is not clear whether the mildest form of concussion, such as that producing transient confusion without amnesia, leads to these anatomical changes.

Studies of cerebral glucose metabolism in human traumatic brain injury have failed to show a relationship between LOC and metabolic perturbations that follow the injury. Glucose metabolism of patients who had sustained concussion but are fully alert at the time of PET scanning is not significantly different than those who are comatose at the time of the testing.[26] However, we may be underestimating the amount of time the brain needs to recover from concussion because even after mild traumatic brain injury, metabolic derangements require about 10 days to resolve.[31]

Neuropsychological testing is our most sensitive measure of cognitive dysfunction after concussion, and it is the most useful method of determining readiness to return to play. Studies using neuropsychological tests after concussion have failed to show any difference between those who lost consciousness and those who did not.[32,33] These tests are typically conducted days after the concussion and usually not in a sport setting but rather in a medical office setting as a follow-up evaluation. The most powerful way of using these instruments is to obtain preinjury testing on each athlete, so that postinjury scores can be compared against individual baseline performance. Some amateur sports teams are using this technique, and such tests

Table 2. Changes in Standardized Assessment of Concussion Scores* After Concussion (n = 91)

	Immediately After Concussion	15 Minutes After Concussion	48 Hours After Concussion
No loss of consciousness, no posttraumatic amnesia (n = 76)	−3	−1	Back to baseline
Posttraumatic amnesia (n = 8)	−4.5	−3	Back to baseline
Loss of consciousness (n = 7)	−14	−13	Back to baseline

* Rounded to nearest 0.5 on 30-point scale.[37]

have become standard parts of concussion evaluation protocols for some college football programs[34] and the National Hockey League.[35] The National Football League has put a similar program in place. I have been involved in coordinating the neuropsychological testing program for the Chicago Bears for the last 7 years. However, if cognitive functions are not evaluated within minutes of the injury, the distinction between grades of concussion is lost.

Sideline mental status testing can be valid and useful in documenting the effects of concussion. We recently reported that neurocognitive deficits can be detected immediately after even mild concussions in football players with the Standardized Assessment of Concussion (SAC).[36] On the 30-point SAC scale, a drop of 3 points from preinjury baseline was seen in 76 athletes who experienced transient confusion without PTA or LOC. Eight players who had PTA without LOC dropped 4.5 points, and 7 players who had brief LOC dropped 14 points (Table 2). Only the mildest form of concussion was associated with rapid recovery of cognitive functions by retesting at 15 minutes after the injury. The group with PTA improved somewhat but did not regain the baseline level of performance. The LOC group demonstrated virtually no improvement in cognitive functions by 15 minutes postinjury.[37] This study is the first to show that disturbances in mental function can be measured immediately after concussion in order to determine the severity of the injury. The findings of this study are most consistent with the grading scale published by the Colorado Medical Society.[38,39]

Return to Play after LOC

Concussions with LOC are commonly associated with longer recovery time than concussions without LOC. That is the major reason why each of the most widely used sports concussion guidelines (Table 3) indicate that the appearance and length of LOC are associated with a more worrisome concussion.[38–41]

Table 4. Time to Return to Play After Removal From Contest*

Grade of Concussion	Time to Return to Play†
Grade 1	Within 15 minutes
Multiple grade 1 concussions	1 week
Grade 2	1 week
Multiple grade 2 concussions	2 weeks
Grade 3: brief loss of consciousness (seconds)	1 week
Grade 3: prolonged loss of consciousness (minutes)	2 weeks
Multiple grade 3 concussions	1 month or longer, based on clinical decision of evaluating physician

* Reprinted with permission from the Quality Standards Subcommittee of the American Academy of Neurology.[41]
† Only if asymptomatic with normal neurologic assessment at rest and with exercise.[41]

The consensus regarding return to play developed by the American Academy of Neurology[41] is presented in Table 4. Any athlete rendered unconscious (grade 3) for even a few seconds must not be allowed to return to that practice or game and must undergo neurologic examination by a physician. In fact, only those without amnesia whose symptoms clear within 15 minutes are allowed to return to play that day.

LOC lasting several minutes should be viewed as a potential neurosurgical emergency requiring urgent medical evaluation. A brief LOC is followed by 1 week off. Longer periods of LOC extend the time of recovery by an additional week or more. Two grade 3 concussions in the same season result in 1 month off. Other contributory factors, such as persistent headache, head pain from associated soft tissue injury, neck pain, and sleep disturbance adversely affect mental functions and can prolong recovery time. The athlete's physician may determine that a longer recovery time is required.

More than one member of a panel of sports medicine specialists[31] reported routinely returning players to the same practice or game in which they had been rendered unconscious. Such a practice is unsupported by neuroscience or expert consensus and puts the athlete in danger of permanent and possibly catastrophic neurologic harm.

Summary

It is rewarding to have the opportunity to present this information to the readership of the *Journal of Athletic Training* because the athletic trainer is the health care professional most

Table 3. Sports Concussion Grading Scales*

	Grade 1	Grade 2	Grade 3
Cantu (1986)[40]	No loss of consciousness, posttraumatic amnesia <30 minutes	Loss of consciousness <5 minutes or posttraumatic amnesia of 30 minutes to 24 hours	Loss of consciousness >5 minutes or posttraumatic amnesia >24 hours
Colorado (1991)[38,39]	Confusion without amnesia, no loss of consciousness	Confusion with amnesia, no loss of consciousness	Loss of consciousness
American Academy of Neurology (1997)[41]	Transient confusion, no loss of consciousness, concussion symptoms <15 minutes	Transient confusion, no loss of consciousness, concussion symptoms >15 minutes	Any loss of consciousness (brief or prolonged)

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likely to witness a concussion during a sport event and is most often in a position to initiate the evaluation and management of the injured player. A growing body of scientific knowledge supports the notion that concussion should be considered a serious medical condition that can have a long-lasting and even catastrophic outcome if misunderstood and mismanaged. I have reviewed the evidence regarding the role of LOC in the diagnosis and management of sport concussion. The evidence generally supports the contention that concussion with LOC represents a worse form of brain injury than concussion without LOC but that rates of recovery vary from individual to individual. Detailed neurologic evaluation, neuroimaging, neuropsychological assessment, and honest reporting of symptoms by the athlete each have very important roles in the safe management of concussion.

All return-to-play guidelines published in recent times emphasize the need for monitoring an athlete with mental status testing of some kind and insisting that he or she be symptom free for some prescribed period of time before returning to activity. Neuropsychological evaluation of the athlete who had been rendered unconscious should be done whenever possible before clearance to return to play. Again, these guidelines are based on the consensus of specialists selected for their expertise in the diagnosis and management of sport concussion. Recent evidence in medical science seems to support the observation that we have been underestimating the amount of time it takes to recover from concussion. The careful clinician errs on the side of caution in making return-to-play decisions after concussion in sports.

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Acceleration-Deceleration Sport-Related Concussion: The Gravity of It All

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Objective: To discuss a newtonian physics model for understanding and calculating acceleration-deceleration forces found in sport-related cerebral concussions and to describe potential applications of this formula, including (1) an attempt to measure the forces applied to the brain during acceleration-deceleration injuries, (2) a method of accruing objective data regarding these forces, and (3) use of these data to predict functional outcome, such as neurocognitive status, recovery curves, and return to play.

Background: Mild concussion in sports has gained considerable attention in the last decade. Athletic trainers and team physicians have attempted to limit negative outcomes by gaining a better understanding of the mechanisms and severity of mild head injuries and by developing meaningful return-to-play criteria. Mild head injury in sports has become an even greater area of focus and concern, given the negative neurobehavioral outcomes experienced by several recent high-profile professional athletes who sustained repeated concussions. Applying the principles of physics to characterize injury types, injury severity, and outcomes may further our development of better concussion management techniques and prevention strategies.

Description: We describe the search for models to explain neuronal injury secondary to concussion and provide an exploratory method for quantifying acceleration-deceleration forces and their relationship to severity of mild head injury. Implications for injury prevention and reduction of morbidity are also considered.

Key Words: mild head injury, physics, athletic injury, axonal injury, whiplash

It has been more than 20 years since the epidemic of mild head injury and the associated medical, social, psychological, and economic consequences were first documented in the scientific literature.[1] Before that time, mild head trauma was considered little more than an inconvenience or nuisance to the health care community. Poor outcomes from mild head injury were attributed to conversion disorders (wherein physical symptoms or deficits that imply a neurologic or medical problem have psychological factors as a basis), depression, or other psychological overreactions to an apparently minor, transient injury. This limited understanding of the mechanism and sequelae of mild head injury was challenged in the late 1970s and early 1980s by Rimel et al,[1] Barth et al,[2] and Gronwall and Wrightson.[3,4] Their research revealed that some of these mild injuries resulted in impaired neurocognitive functioning that persisted for 1 to 3 months after trauma and caused slower-than-expected return to work.

Concurrent with early findings of poor outcome after mild head injury, other investigators designed animal studies to detect the presence of gross neuropathologic and histologic indications of disrupted brain functioning. Gennarelli et al[5] developed and used an animal model of injury analogous to the whiplash-type injury experienced by patients with mild head injuries involved in automobile crashes. In their model, a mild cerebral injury could be administered to an animal without direct impact to the skull using acceleration-deceleration forces. Microscopic examination of brain tissue from primates exposed to this experimental model revealed axonal shear strain on autopsy. Shear strain injury is observed as the tearing or stretching of axons, which is frequently not detected in patients with mild head injuries using gross neuroimaging techniques, such as magnetic resonance imaging or computed tomographic scans. Although its focus was on the neuropathologic impact that resulted from linear forces on the brain, the research of Gennarelli et al[5] was instrumental in documenting cerebral injury from an apparently mild nonimpact head injury.

SPORTS AS A LABORATORY ASSESSMENT MODEL

The aforementioned studies suggested a link between mild head injury and poor cognitive, psychosocial, and neurologic outcomes in some patients, but they raised even more questions regarding mechanism of injury (impact versus nonimpact, linear versus rotational), associated neurophysiology, and individual vulnerability. Additionally, neurocognitive deficits associated with mild head injury are often subtle, and there are tremendous differences in individual abilities. Therefore, the need to control for preinjury functioning and ability became apparent as a way of determining who is most vulnerable to a poor outcome from mild head injury. In addition, questions arose about the length of the typical recovery curve for most people. In an attempt to answer these questions, Barth et al[6] and Macciocchi et al[7] at the University of Virginia developed the Sports as a Laboratory Assessment Model (SLAM)

and published the first studies of the neuropsychological sequelae of mild acceleration-deceleration cerebral concussion in college football players. In this model, entire sports teams undergo baseline preseason neuropsychological assessment, which addresses preinjury functioning. When a player sustains a concussion during the natural course of play, he or she is reassessed, along with a matched and uninjured player, to control for practice effects due to additional testing. Next, subsequent serial assessment allows for tracking of the recovery curve. The research of Barth et al[6] and Macciocchi et al[7] revealed that all football players who sustained a concussion recovered to the performance of uninjured controls within 5 to 10 days after injury.

Even though the aforementioned research and studies by Levin et al,[8] Dikmen et al,[9] Ruff et al,[10] and others focused on understanding the clinical consequences of mild head injury in the general public, the sports medicine community began to take notice of SLAM as a means of studying concussion in athletes. In conjunction with professional experience, opinion, and consensus, Cantu,[11] Kelly et al,[12] and others used these data as a point of reference in the development of guidelines for return to play in an attempt to protect athletes from possible catastrophic injury related to multiple subconcussive blows and second-impact syndrome.[13] These data served as the foundation for further explorations into objectively understanding and evaluating sport-related head injury.

As a result of athletes' strong desire to compete and return to play, there is a tendency within the sports community to minimize the seriousness of injuries. In this context, mild head trauma has long been viewed as inconsequential because the forces exerted on the brain were deemed insufficient to cause significant neurophysiologic damage. Even under conditions in which there is no overt impact, trauma to the brain is possible. Trauma can result from a rapid change in the head's velocity or change in vector speed over time. Change in velocity over time is defined as acceleration or deceleration. Significant force, in the absence of direct and visible impact to the head, can have a detrimental effect on brain tissue. The newtonian laws of physics yield a model for potentially understanding the "gravity" of these forces on the brain. Through greater understanding and application of physics (biomechanical) principles, we may eventually develop more objective and predictive models for evaluating the immediate and long-term effects of forces exerted on the brain in the sports arena.

LAWS OF MOTION AND MECHANICS OF INJURY

To explain the mechanism of acceleration with rapid deceleration in clinical aspects of mild head injury, Varney and Roberts[14] suggested applying fundamental newtonian formulas to the description of linear and rotational vector forces on the head and brain. These formulas can assist in calculating the stresses and energy displacement on neural fibers under various conditions, such as motor vehicle crashes. Severity of head injury, measured as the force of acceleration and deceleration, can be determined from such analyses. In turn, calculations can be made with regard to the potential for neurocognitive impairment. Barth et al[15] suggested that this newtonian physics approach be applied to the measurement of sport-related acceleration-deceleration head injury to add to our understanding of injury severity.

Deceleration, which must necessarily follow acceleration, is the key issue when discussing the forces applied in mild concussion. Deceleration can be viewed as negative acceleration or decreasing velocity over time. The formula for calculating acceleration or deceleration is as follows:

$$a = (v^2 - v_o^2)/2sg$$

In this formula, a is acceleration or deceleration, v_o is initial speed in a given direction before deceleration starts, v is the directional speed at the end of deceleration, and s is the distance traveled during deceleration. The use of g in this formula allows for the expression of results in terms of multiples of acceleration due to gravity or g force. One g force is equivalent to 9.812 m/s^2 (10.73 yd/s^2). Since v in a sports acceleration-deceleration model is generally calculated as 0, because the player is presumably brought to a halt, the formula can be simplified to the following:

$$a = -v_o^2/2sg$$

A real example of the application of this formula could be gleaned from game film of any contact sport, including football, soccer, lacrosse, wrestling, and equestrian sports (eg, contact with the ground, a branch, or a fence post) having high prevalence of mild traumatic brain injury. Using these films, velocity (directional speed) and stopping distances can be calculated. For instance, if a running back is traveling at 3.658 m/s (4 yd/s) and his head is brought to a stop in a distance of 0.152 m (6 in or 0.167 yd) (both of which are realistic and, in fact, conservative), the following deceleration would be calculated:

$$a = (-4)^2/(2)(0.167)(10.73) = 4.46g$$

In this hypothetical yet realistic case, the formula yields the player's velocity change over time as 4.46g, or more than 4 times the normal acceleration due to gravity, which is 1g. The force on any part of the player's mass (m), which experiences an acceleration of magnitude (a) (regardless of whether a is positive, reflecting acceleration, or negative, reflecting deceleration) is given by the Newton Second Law of Motion:

$$F = ma$$

If a is nothing but the acceleration of gravity, 1g, or, for example, a player falling to the ground with no other forces affecting him or her, the Newton Law gives the following:

$$F = mg$$

For example, if the player experiences an acceleration of 10g, the force on any element of mass, for example the brain, is $F = (m)(10g)$. Therefore, the body element experiences 10 times the force of what it would experience from gravity. Just how much g force on the brain would cause irreparable damage depends on many additional factors, as we discuss throughout this article (the study of these issues is referred to as biomechanics). Although acceleration of 30g or greater is frequently calculated in motor vehicle crashes that cause irreparable brain injury, what remains to be established is whether repeated exposure to forces of magnitude around 10g is cumulative and ultimately leads to permanent brain damage.

If the earlier value of the acceleration is inserted into the Newton Law, the Law then reads as follows:

$$F = mv^2/2s$$

This equation highlights the fact that if several different collisions occur all with the same initial speed (v), then the smaller the stopping distance (s), the larger the resulting force on the brain. Thus, if a player should crash into an almost immovable object, such as a goalpost or the ground, the value of s would be very small and the potential injury more severe.

We have yet to confirm what magnitude of force has significant adverse effects on the brain. An additional complication is that there are often numerous directions, or vectors, of force that might influence outcome. The simplest cases involve linear deceleration, commonly consisting of head-on or angled impacts. In the head-on variety, both players quickly experience deceleration, particularly if they are running at the same speed and have approximately the same mass. If they collide helmet to helmet or shoulder to shoulder, they are likely to decelerate very rapidly; hence, greater force is applied, and the probability of neurologic injury is higher. In this same situation, if one player's upper body collides with the other player's lower body, both athletes have longer deceleration distances and times, reducing the applied force on the brain. In the case of the angled impacts, deceleration distances and times are usually longer; thus, injury severity will likely be less. It is important to note, however, that angular impacts can cause rotational forces on the brain, which, if severe enough, can result in several rapid changes in velocity (directional speed) over short distances, periods, or both.

Countless scenarios exist for acceleration-deceleration injuries in sports. The aforementioned scenarios assume that both players are anticipating the collision and are prepared. If unaware of an impending impact, players may fail to appropriately align their bodies or tense their neck muscles. In such cases, players may experience a whiplash-type force. This creates torque, seen as rotation of the head either in or out of its original plane. When changes in velocity (acceleration) are dramatic and occur over short distances, the outcomes are more negative than those in injuries that result from linear impacts.

Acceleration-deceleration, by definition, implies a particular direction or vector. Changes in the vector of acceleration or deceleration (ie, rotational or twisting forces) further complicate the computation of the sum of forces brought to bear on the brain. In other words, whether the brain is "torqued" in a rotational fashion has considerable influence on functional outcome. Consider "clotheslining," which can occur as a result of player-to-player contact in some contact sports. In this instance, the head does not merely decelerate in unidirectional fashion but is actually decelerating in the original vector and accelerating in a new vector, usually rotating backward and downward. Multiple vectors of acceleration and deceleration in response to forces applied to the brain likely account for the greatest histokinetic changes, or axonal injuries, in mild head injury. These likely lead to the greatest impairments in neurobehavioral outcome.

It is also important to note that the brain is at risk for damage at numerous points. In the linear case, sufficient force in the opposite velocity vector may cause the brain to strike against the inner skull in the direction it was initially traveling (coup injury). Additionally, the brain may "rebound" from the direction of the deceleration and strike the inner lining of the skull in the opposite direction (contrecoup injury). With rotational force, the sites in which the brain may contact or scrape the inner lining of the skull become manifold. Although no true coup or contrecoup injury may exist, the magnitude of tissue alteration (ie, shear strain injury and diffuse axonal injury) can be significantly larger when significant rotational forces are applied to the brain.

USING NEWTON TO PROTECT THE ATHLETE

Physics formulas for calculating acceleration-deceleration and forces applied to the head also have implications for the prevention of and protection against serious injury. Both the time and the distance over which changes in velocity occur influence outcome. For instance, the cushioning effect of helmets increases the distance of deceleration and reduces the forces associated with these injuries. Helmets also increase the surface area across which the blow, or force, is absorbed. This is evident in another newtonian formula, wherein P refers to pressure, F indicates the force applied, and A is the area to which the force is applied: $P = F/A$. By distributing the applied force to the helmet from an impact with another helmet, body part, or the ground, the pressure exerted on the head is actually decreased as a function of the area of the helmet.

Winters[16] reports on the value of properly fitted mouthguards, which may reduce the severity and incidence of cerebral concussion for specific mechanisms of injury. Using the physics model, the cushioning effects of a properly fitted mouthguard, particularly during a linear impact that involves the mandible, increase the time and distance of deceleration and likely offer cerebral protection. Enforcement of rules against spearing (using the head to tackle) is another clear strategy that also helps to increase deceleration distance. When a player's first contact is against the body of an opponent, the head has more distance for any changes in velocity. Proper training to prepare for contact on the sports field is also essential, since unexpected blows or changes in velocity of the head can produce the greatest forces on the brain. In soccer, properly tensing the muscles of the back and neck in preparation to head a ball disperses the area across which the force is applied. The head, neck, and upper torso are, therefore, used in unison to absorb the impact of the ball on the head, resulting in decreased velocity change for the head itself. This principle is easily extended to training athletes in the rules and techniques of tackling or checking, specifically, how to absorb these blows through anticipation and preparation of the entire body.

FUTURE DIRECTIONS

Use of the aforementioned formulas provides a good conceptual basis for understanding the mechanics of forces applied to the brain during sport-related concussion. Additionally, these formulas have pragmatic uses as well. Today's video technology allows for minute discriminations between distances and times. In the sport setting, analysis of game films thus permits computation of player velocity before impact and the deceleration value. Factoring in player mass, computing an estimate of the force of impact is then a reasonable endeavor. A database that tracks mechanism of injury (eg, head to head, head to body, head to ground, head to goalpost), estimated force of impact, and resultant functional outcome mea-

sures (eg, loss of consciousness, altered consciousness, neurologic and neuropsychological signs and symptoms) is then attainable. A history of head injury and the estimated magnitude of the force involved are also important factors that allow us to begin to examine the effect of repeated exposures to small force impacts.

Clearly, the analysis of the force applied specifically to the head is more complicated than we have suggested herein, since it necessitates consideration of all vectors involved in the impact. However, moviegoers will note that technologies can now create a freeze-frame rotation around a particular scene. Although few cameras are used, computer-generated images are inserted to fill the gaps. Applying this technique to the game or practice setting may enable coaches, athletic trainers, and other medical personnel to analyze game films and examine the direction of the forces applied to the head. Although such an evaluation is seemingly complex, perhaps it will not be too far in the future when such images will be both generated and analyzed by these programs, yielding more refined measurement of these forces and enhancing our understanding of the mechanics of mild head injury in sports.

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Management of Cerebral Concussion in Sports: The Athletic Trainer's Perspective

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Objective: To present a new approach in the evaluation and management of concussion from the athletic trainer's perspective.

Background: The evaluation and management of concussion continues to be a controversial topic among sports medicine professionals. Inconsistent management, lack of objective data, and confusion concerning assessment techniques may lead to inappropriate decisions regarding when to return an athlete to competition after concussion. In this article, we provide recommendations and considerations for the certified athletic trainer in the management of concussion. We also present a quantifiable assessment technique that provides more information on which return-to-play decisions can be made; this technique can be used during the initial sideline examination as well as during subsequent follow-up examinations.

Recommendations: Certified athletic trainers and team physicians should consistently use appropriate grading scales. Assessment of concussion should include a symptom checklist, the Balance Error Scoring System, and the Standardized Assessment of Concussion, and the results should be compared with the athlete's normal baseline scores. Follow-up neuropsychological and postural stability testing is recommended. Return-to-play decisions should be based on the grade of concussion, scores on objective tests, and presence of concussive symptoms during exertional activities.

Key Words: mild head injury, neuropsychological testing, postural stability testing, grading scales

The evaluation and management of concussions continues to be a controversial topic among certified athletic trainers (ATCs) and other sports medicine professionals. Choices about assessment protocol and return-to-play decisions have been based on poorly validated guidelines and clinical judgment. With an incidence of 300 000 sport-related concussions per year[1] and the potential for catastrophic outcomes,[2] better guidelines for managing cerebral concussion in sport are needed.

The complexity of this topic may stem from a lack of agreement in the sports medicine community concerning the definition of, and the critical symptoms relating to, a cerebral concussion. The 1966 Committee on Head Injury Nomenclature[3] defined a cerebral concussion as a clinical syndrome characterized by "immediate and transient impairment of neural functions, such as an alteration of consciousness, disturbance of vision and equilibrium due to brain stem involvement." It is important to note that an athlete sustaining a cerebral concussion does not have to lose consciousness. In fact, loss of consciousness (LOC) is reported in only 8.9% of those sustaining a concussion.[4] Because more than 90% of concussions do not result in LOC, many of these injuries go unreported or are not reported until the end of the practice or competition.[5] For this reason, it is important for the ATC and team physician to be aware of other signs and symptoms associated with concussion. These symptoms may include but are not limited to headache, confusion, tinnitus, dizziness, balance disturbance, blurred vision, nausea, and posttraumatic amnesia.[4,6]

The difficulty in using the current grading scales for evaluating concussion is that most are based on loss of consciousness and the presence of posttraumatic amnesia, which occur very infrequently, while essentially ignoring multiple symptoms that present in a variable pattern within this group of athletes. The result is that ATCs and physicians have difficulty distinguishing among the various degrees of concussion. The confusion and frustration with the current scales have resulted in many sports medicine professionals not using the grading scales for evaluation purposes. In a recent study, up to 63% of ATCs reported not using any scale for evaluating concussion.[7] The ramifications of not using a grading scale are compounded by the lack of a biological marker for making return-to-play decisions. Instead, decisions to return an athlete to competition are based largely on the initial evaluation and subsequent follow-up evaluations.

Our main purpose is to present the perspective of 3 ATCs on a more comprehensive approach for evaluating and managing concussion. In describing this new approach to managing concussion, we present a model for making return-to-play decisions that are not based solely on a guideline but rather on the athlete's symptoms and performance on objective tests such as the Balance Error Scoring System (BESS) and the Standardized Assessment of Concussion (SAC). This model is much like the functional performance model of injury management that ATCs are accustomed to using with musculoskeletal injuries.

A SYSTEMATIC APPROACH

A systematic approach to management of concussion requires that all sports medicine personnel involved in treating

athletes must be familiar with instruments, procedures, and recommendations for evaluation, follow-up care, and return-to-play decisions. Physicians are present at a relatively small percentage of practices and contests. Thus, the initial evaluation and management often is completed by an ATC. Physicians and ATCs should agree on definitions, procedures, and referral guidelines. Clear communication among sports medicine personnel is necessary to provide consistent and proper care to the athlete.

A systematic evaluation begins with baseline testing in the preparticipation physical examination. The baseline scores provide an individualized "normal" to be used for comparison should the athlete sustain a concussion. Obtaining baseline scores on a series of cognitive and postural stability tests is paramount, considering that, unlike orthopaedic injuries, there is no contralateral limb to use for comparison. Baseline scores are obtained for the BESS and the SAC, both of which can easily be readministered on the sideline or in the locker room after injury. A brief battery of neuropsychological tests and a more sophisticated balance test performed on a computerized forceplate can also be administered if these resources are available.[8,9]

Baseline testing should be conducted with the cooperation of the team physician and coaching staff. The neuropsychological and balance testing can be administered as part of the preparticipation physical examination or during the summer sessions if the athletes are available.

Recognition and Evaluation

Acute injury assessment begins with the observation and recognition that a concussion may have occurred. Loss of consciousness, although relatively infrequent, is the first consideration in concussion management. When the athlete sustains a concussion that results in LOC, a primary survey must be performed to determine if the airway or circulation has been compromised, causing a critical situation. Unconscious athletes must be treated as if they have also sustained a cervical spine injury. These athletes must be placed on a spine board and transported to the hospital for further evaluation. The athlete with an altered state of consciousness who is unable to communicate with the ATC regarding his or her symptoms should also be placed on a spine board and sent for evaluation of possible intracranial pathology.

Certified athletic trainers are most often presented with an athlete who has sustained a concussion but has not been rendered unconscious. Athletes commonly believe that having "their bell rung" is a natural part of the game and do not report receiving a concussion. Unlike ankle or knee injuries that result in obvious impairment, those athletes sustaining concussions are not always distinguishable.

Once the ATC has recognized a concussion and performed a primary survey to rule out life-threatening injuries, a thorough history must be obtained regarding any LOC, confusion, or amnesia. Because ATCs observe the occurrence of a concussion from the sideline, it is sometimes necessary to obtain information about the athlete from those who were on the field. This can include asking officials or teammates for information. Testing for amnesia and orientation is best done by asking questions of recent memory and not items such as name, date of birth, or specific date.[10] Most often, testing occurs on the sideline and should be performed at the end of the bench area, away from teammates and other distractions. Occasionally, it is necessary to perform the tests in the athletic training room or locker room because of the distraction from fans, teammates, or other sideline personnel. Test results are not environmentally sensitive, which allows for sideline scores to be compared with baseline test scores administered in other laboratory or classroom settings.[11]

Table 1. Postconcussion Symptom Scale (0–6)*

Symptom	Preseason Baseline	Time of Injury	2 to 3 Hours Postinjury
Headache			
Nausea			
Vomiting			
Dizziness			
Poor balance			
Sensitivity to noise			
Ringing in ears			
Sensitivity to light			
Blurred vision			
Poor concentration			
Memory problems			
Trouble sleeping			
Drowsiness			
Fatigue			
Sadness/depression			
Irritability			
Neck pain			
Total score			

*0 = none, 6 = severe.

Postconcussive Symptoms

Testing continues with the presentation of a postconcussion symptom scale (Table 1), which, in addition to being administered at preseason baseline, time of injury, and 2 to 3 hours postinjury, should also be administered daily until the athlete is asymptomatic. The athlete is asked to rate the severity of each concussive symptom on the list from 0 (none) to 6 (severe). This allows the clinician a more objective measure of symptom severity, and changes in symptoms can be quantified for better comparison. It is recommended that the athlete remain out of competition until symptom scores return to 0 or to baseline scores.

Cranial Nerve Testing

After reviewing the athlete's symptoms, assess the function of the cranial nerves. The cranial nerves can be tested quickly: sense of smell (I), eye tracking and pupil reactivity (III, IV, V), facial expressions (VII), biting down (V), swallowing (X), protrusion of the tongue (XII), and shoulder shrugs (XI).[12] Alterations of the cranial nerve responses may indicate a more severe head injury and the need for immediate referral of the athlete to a physician or transferal to the hospital.

Balance Error Scoring System

Once cranial nerve function is determined to be intact, the ATC should test equilibrium and balance on the BESS.[9] The BESS is a quantifiable version of a modified Romberg test for balance, consisting of 3 tests lasting 20 seconds each, performed on firm and foam surfaces. The athlete first stands with the feet narrowly together, the hands on the iliac crests, and

Table 2. Balance Error Scoring System (BESS)

Scorecard (No. of errors)	Firm Surface	Foam Surface
Double-leg stance		
Single-leg stance		
Tandem stance		
Total errors		
Total score		

the eyes closed. The athlete holds this stance for 20 seconds while the ATC records the number of balance errors. A balance error is operationally defined as opening the eyes, hands coming off the hips, taking a step, moving the hips into 30° or more abduction, lifting the forefoot or heel, or remaining out of testing position for more than 5 seconds. The test is repeated with a single-leg stance using the nondominant foot and again using a heel-toe stance with the nondominant foot in the rear. All 3 tests are performed on a firm surface (grass, turf, court) and again on a piece of medium-density foam. A piece of foam can easily be carried in a travel trunk or equipment bag for road games. The number of errors on each of the 6 tests are added for a total BESS score (Table 2).

Standardized Assessment of Concussion

Neuropsychological testing and testing of mental status are performed using the SAC. The SAC measures the immediate neurocognitive effects of concussion and is designed to assess orientation, immediate memory, concentration, and delayed memory.[13] Deficits in these cognitive functions are often associated with athletes sustaining a concussion.[14] Administration of the SAC takes 5 minutes; orientation, immediate memory, neurologic function, concentration, delayed recall, and symptoms during exertional testing are assessed.[13]

The scores obtained from these tests should be compared with the athlete's baseline scores obtained in the preseason screening process or physical examination. If a physician is present, the ATC should provide the physician with the scores and refer the athlete to the physician for definitive evaluation and classification of the severity of the concussion.

A classification guide that includes concentration, coordination, and cranial nerve assessment (3 Cs), along with the traditional landmarks of consciousness and amnesia, can be very useful to the ATC and team physician (Table 3). By using the BESS to identify problems with coordination and the SAC to detect impaired concentration, the ATC and team physician may have a more objective means of assessing an athlete's concussion.

MANAGEMENT

Once the athlete has been properly evaluated, the ATC and physician must decide on the best course of management for the athlete. This decision is based not only on the evaluation but also on any previous history of concussion and the potential for further injury. If the athlete was rendered unconscious for 1 minute or longer and continues to have disturbed sensorium, he or she should be transported to the hospital for further neurologic testing and imaging. The use of neuroimaging (magnetic resonance imaging and computerized tomography scanning) to assist in the evaluation and management of concussion has been debated. Magnetic resonance imaging has been reported effective in demonstrating contusions and larger cranial lesions, but often these neuroimaging devices are not sensitive enough to detect axonal damage resulting from a concussive blow.[15]

The goals in managing an athlete with a concussion should be to prevent a catastrophic outcome and to return the athlete to competition in a manner that minimizes both the time away from competition and, more importantly, the possibility of second-impact syndrome or more severe head injury. Obviously, this is a delicate balance and not an easy goal to achieve. The more objective information obtained during the decision process, the more comfortable the decision being rendered as to an athlete's ability to safely return to play.

The ATC is an invaluable member of the health care team, particularly when an athlete suffers a concussion. The ATC often has daily interaction and the opportunity to establish a trusting relationship with the athletes. This trust is a vital part of the management process. Athletes who do not report injuries or who underreport symptoms may be placing themselves at risk for a catastrophic outcome. Athletes may be more com-

Table 3. University of North Carolina Classification of Cerebral Concussion

Grade	Level of Consciousness	Cranial Nerves, Cognition,* and Coordination (3 Cs)	Headache
0 (mild)	No LOC†	Mild confusion but asymptomatic in 10 minutes; passes functional tests without recurrence of signs and symptoms	Possibly develops later
1 (mild)	No LOC	At least 1 of the following is present: 1. Abnormal cranial nerve function lasting <1 hour 2. Abnormal cognition lasting <1 hour 3. Abnormal coordination lasting < 3 days	Probable; lasts from 10 minutes to as long as 2 days
2 (moderate)	Brief LOC from 10 seconds to 1 minute or altered consciousness lasting <2 minutes	At least 1 of the following is present: 1. Abnormal cranial nerve function lasting >1 hour 2. Abnormal cognition lasting >1 hour 3. Abnormal coordination lasting longer than 3 days	Probable; lasts 24 hours to 4 days
3 (severe)	LOC >1 minute or altered consciousness lasting >2 minutes	2 of 3 Cs are abnormal for more than 24 hours	Likely; lasts longer than 4 days

*Cognition includes orientation, memory, concentration, and attention.
†LOC indicates loss of consciousness.

fortable reporting symptoms to an ATC, whom they see on a daily basis, rather than to a physician they do not see regularly. The ATC may also be better able to identify subtle signs that an athlete is suffering from a concussion by knowing the athlete's usual behavior and demeanor. It is paramount for the ATC to educate the athlete about the signs and symptoms of concussion and the dangers of second-impact syndrome, which can result from not reporting concussion symptoms to the ATC.

Return-to-Play Decisions

Like grading scales, a number of guidelines have been proposed for returning an athlete to competition after sustaining a concussion.[2,16–18] These guidelines are very conservative and are based less on scientific data and more on clinical experiences and educated estimates of time needed for the complications of concussion to resolve. Evidence suggests that athletes are frequently being returned to competition on a more aggressive timetable than the guidelines recommend.[4] A great deal of variation exists among the guidelines, but the consensus is that return to competition should not occur until the athlete is asymptomatic, both at rest and during exertion. The guidelines in Table 4 are based not only on severity of concussion but also on recent research addressing the sequelae of concussion (K. M. Guskiewicz, PhD, ATC, unpublished data, 2000).[19] It is important to remember that these are just guidelines for returning an athlete to competition. Each case should be reviewed on an individual basis to determine whether the individual is able to return safely to sport. Several factors should be considered when making this decision. The first is the athlete's prior concussion history. An athlete with a previous concussion during the season is at 3 to 4 times greater risk for suffering a second concussion.[4,20] The risk of concussion increases further after each subsequent concussion. Another factor to consider is the sport the athlete is playing. An athlete participating in a noncontact, low-risk sport will most likely be returned to competition sooner than an athlete returning to a high-risk, collision sport, such as football or wrestling. Early assessment and daily follow-up examinations allow the ATC and team physician to determine when the athlete is asymptomatic based on comparisons with baseline neuropsychological and postural stability tests. More importantly, they also allow clinicians to observe an athlete whose status is deteriorating due to possible intracranial pathology. These athletes must be identified and treated emergently.

Table 4. University of North Carolina Return-to-Play Guidelines After Concussion

Grade	Suggested Action
0	Remove the athlete from contest. Examine immediately for abnormal cranial nerve function, cognition, or coordination or other postconcussive symptoms at rest and with exertion. May return to contest if examination is normal and asymptomatic for 20 minutes. If any symptoms develop within 20 minutes, return that day is not permitted.
1	If athlete is removed from contest after developing symptoms, daily follow-up evaluations are necessary. May begin restricted participation when asymptomatic at rest and after exertional tests for 2 days. Unrestricted participation allowed if asymptomatic for 1 additional day and neuropsychological and balance testing normal.
2	Remove the athlete from contest and prohibit return that day. Examine immediately and at 5-minute intervals for evolving intracranial pathology. Reexamine daily. May return to restricted participation when athletic trainer and physician are assured the athlete has been asymptomatic at rest and with exertional testing for 4 days. Unrestricted participation if asymptomatic for an additional 2 days and performing restricted activities normally and comfortably.
3	Treat the athlete on field or court as if cervical spine injury has occurred. Immediate examination and reevaluation at 5-minute intervals for signs of intracranial pathology. Reexamine daily. Return is based on resolution of symptoms: 1. If symptoms totally resolve within first week, return to restricted participation when the athlete has been asymptomatic at rest and with exertion for 10 days. If asymptomatic for an additional 3 days of restricted activity, the athlete may return to full participation. 2. If symptoms do not resolve within the first week, the athlete may return to restricted participation when asymptomatic at rest and with exertion for 17 days. Return to unrestricted participation if asymptomatic an additional 3 days.

Note: If the athlete suffers a second concussion within 3 months of the first concussion, the athlete must be removed for twice the maximum time for the respective grade of concussion.

Establishing Protocol

The establishment of a specific protocol in managing concussion is necessary to maintain the consistency of care and ensures that proper procedures are followed when making decisions about returning athletes to competition. The protocol should cover procedures for the most mild injuries, in which the athlete is permitted to return to the game, to the most severe injuries, involving the presence of a variety of symptoms for days or weeks.

An athlete sustaining a grade 0 concussion who has successfully passed all tests on the sideline or in the locker room and has been asymptomatic for at least 15 minutes should perform exertional testing, such as sprints and push-ups, as well as sport-specific functional tests. Recurrent symptoms preclude the athlete from returning to participation. Postconcussive symptoms may manifest during these procedures due to increased intracranial pressure, indicating unresolved pathology. Athletes whose symptoms linger or return during testing should be removed from competition and reevaluated. Athletes sustaining concussions graded 1 through 3 need to undergo further, more sensitive testing at regulated intervals.

Neuropsychological and Postural Stability Testing

A more comprehensive neuropsychological test battery and more sophisticated postural stability testing can be performed in the clinic or athletic training room on subsequent postinjury days. The Neurocom Smart Balance Master System (Neurocom International, Inc, Clackamas, OR) has been used to quantify balance deficits and sensory organization problems resulting from concussion. Recovery curves have also been established that allow the clinician to determine if improved scores occur from learning or from an alleviation of concussive symptoms. Clinicians in settings without sophisticated forceplate systems should perform postural stability testing using the BESS as previously described. Researchers have de-

Table 5. Neuropsychological Test Battery

Neuropsychological Test	Function(s) Assessed
Stroop Color World Part 3	Mental flexibility and attention
Hopkins Verbal Learning	Verbal memory
Symbol Digit Modalities	Visual scanning and attention
Trail-Making B	Visual scanning, mental flexibility, and attention
Control Oral Word Association	Verbal fluency
Wechsler Digit Span Forward and Backward	Attention span

scribed a strong correlation between the BESS and more expensive platform balance systems.[21]

A suggested battery of neuropsychological tests (Table 5) should be given 24 hours after injury and again on the initial postinjury days until the athlete has returned to baseline on all tests. Along with the neuropsychological tests, the concussion symptom checklist should be administered to track changes in symptoms.

Using the Information

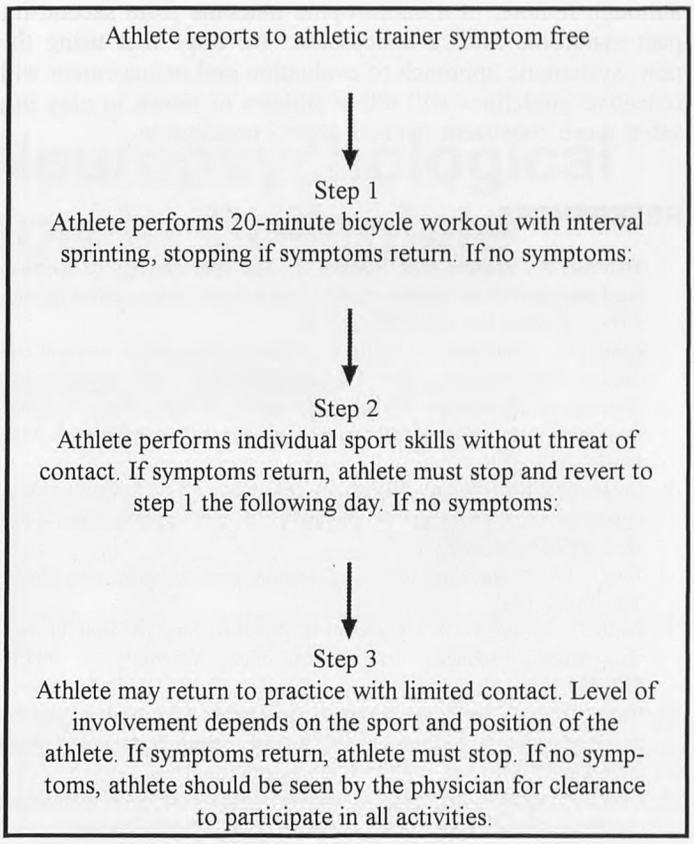
The testing protocol provides the clinician with objective, quantifiable data on the athlete from the establishment of a baseline "normal" to time of injury until full recovery. The frequent testing allows the clinician to make daily recommendations concerning recovery rather than holding an athlete out longer than necessary or returning the player to sport before recovery has occurred. Although guidelines are used to assist us with decision making, return-to-play decisions are based on the athlete's symptoms, test scores, and ability to perform exertional activities without an increase in symptoms.

Once an athlete is free of postconcussive symptoms, he or she is not immediately placed back into competition. Rather, the athlete is progressed back into activities with the ATC documenting any change in symptoms (Figure).

Athletes who do not experience recurrent symptoms and have returned to 95% of baseline scores on the cognitive and balance tests are good candidates for return-to-participation clearance by the physician. The methods used for returning the athlete to participation are similar to those employed by ATCs in deciding when to return an athlete after suffering a musculoskeletal injury. A window of protection is followed by successive performance-based criteria that, when achieved, advance the athlete to the next level of participation. A contact athlete may be able to participate in drills and activities wearing a jersey designating "no contact." The athlete can work on skills and timing without the threat of contact and recurrent concussion. It is important that the coaching staff supports and complies with the noncontact activity, understanding that it may allow for a quicker and safer return to play for that athlete.

PREVENTION

Unlike musculoskeletal injuries, few strength and conditioning methods will help prevent further concussions. However, that does not mean no preventive measures can be taken to minimize recurrence of concussions and other head injuries. Again, athletes sustaining a concussion are at a 3-fold increased risk for future concussions, and that risk increases with each successive injury.[4,19]



Returning an athlete to competition after concussion.

In order to prevent recurrence, it is necessary to determine how the concussive incident occurred. Reviewing game or practice films may provide an opportunity to see the mechanism of injury: a direct blow, a blow from hitting the ground, or even a rotational component. More importantly, the film may help reveal poor techniques, such as leading with the head to tackle or block or heading a soccer ball incorrectly. Reviewing the tape with the athlete and the coach may be useful in improving the athlete's technique or changing the coach's teaching methods. Preventing another concussion may be the difference in an athlete's ability to return to participation during the season.

CONCLUSIONS

Recognition and proper assessment of concussion in athletes continues to be a difficult problem in the field of sports medicine. The difficulty stems from the inconsistent use of terms and guidelines available for evaluation. This confusion is made more problematic by a lack of objective information used in the assessment and follow-up care of athletes with concussions.

We have presented a systematic approach to evaluating and managing concussions based on objective, quantifiable data. Evaluation guidelines for grading concussions will allow for improved communication among caregivers and more consistency of care. Guidelines for return-to-play decisions after a concussion are based on quantifiable data and a model similar to the protocol for returning athletes to competition after musculoskeletal injury. As with any injury, the goal in managing players with concussions is to decrease the chance of recurrence. This is especially important considering the possibility,

although remote, of a catastrophic outcome from second-impact syndrome after a concussion. We hope that using this new, systematic approach to evaluation and management with objective guidelines will allow athletes to return to play in a safer, more consistent manner after a concussion.

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Postural Stability and Neuropsychological Deficits After Concussion in Collegiate Athletes

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Objective: Postural stability and neuropsychological testing are gradually becoming integral parts of postconcussion assessment in athletes. Clinicians, however, sometimes question the viability of instituting preseason baseline testing and the value of these results in making return-to-play decisions. Our purpose was to examine the course of recovery on various postural stability and neuropsychological measures after sport-related concussion. A secondary goal was to determine if loss of consciousness and amnesia, both of which are heavily weighted in most of the concussion classification systems, affect the rate of recovery.

Design and Setting: All subjects underwent a battery of baseline postural stability and neuropsychological tests before the start of their respective seasons. Any athletes subsequently injured were followed up at postinjury days 1, 3, and 5. Matched control subjects were assessed using the same test battery at the same time intervals.

Subjects: We studied 36 Division I collegiate athletes who sustained a concussion and 36 matched control subjects.

Measurements: We assessed postural stability using the Sensory Organization Test on the NeuroCom Smart Balance Master System and the Balance Error Scoring System. Neurocognitive functioning was measured with several neuropsychological tests: Trail-Making Test, Wechsler Digit Span Test, Stroop Color Word Test, and Hopkins Verbal Learning Test.

Results: Injured subjects demonstrated postural stability deficits, as measured on both the Sensory Organization Test and Balance Error Scoring System. These deficits were significantly worse than both preseason scores and matched control subjects' scores on postinjury day 1. Only the results on the Trail-Making Test B and Wechsler Digit Span Test Backward resulted in a logical recovery curve that could explain lowered neuropsychological performance due to concussive injury. Significant differences were revealed between the control and injured groups at day 1 postinjury, but a significant decline between baseline and postinjury scores was not demonstrated. Loss of consciousness and amnesia were not associated with increased deficits or slowed recovery on measures of postural stability or neurocognitive functioning.

Conclusions: Athletes with cerebral concussion demonstrated acute balance deficits, which are likely the result of not using information from the vestibular and visual systems effectively. Neurocognitive deficits are more difficult to identify in the acute stages of concussion, although concentration, working memory, immediate memory recall, and rapid visual processing appear to be mildly affected. More research is necessary to determine the best neuropsychological test battery for assessing sport-related concussion.

Key Words: mild head injury, balance, neurocognitive function

The use of neuropsychological and postural stability testing for the management of sport-related cerebral concussion is gradually becoming more commonplace among sports medicine clinicians. Recent research suggests that the use of a comprehensive approach may assist the athletic trainer and team physician in identifying signs of a concussion not easily detected during a routine clinical examination.[1–3] Similarly, the use of these tests can eliminate some of the guesswork from the return-to-play (RTP) decision after concussion, as the subjective nature of symptoms associated with the injury make this assessment uniquely challenging. Despite the potentially catastrophic consequences of an athlete's premature return to competition after concussion, RTP decisions are often based on speculation rather than certainty. The life-threatening consequences of second-impact syndrome are well documented in the literature[4–8] and should be a legitimate concern for all sports medicine personnel.

Awareness is clearly increased regarding the dangers of sport-related concussion compared with 10 years ago. Although football is generally recognized as the sport most often associated with concussion, moderate to high incidences of concussion have been noted in basketball, softball, soccer, baseball, boxing, rugby, and ice hockey.[9–11] Despite rule changes and equipment modifications aimed at reducing concussion in sport, these injuries still occur frequently. The incidence of recurrent injury (15% to 20%) is higher than that of the initial injury (5% to 10%) in most sports,[12–15] thereby suggesting the need to validate objective assessment techniques that enable the clinician to grade the initial injury and minimize the risk of a second or third such injury. Reports of

the cumulative effects of multiple head injuries, as well as multiple head impacts, on long-term cognitive functioning are causing clinicians to rethink their approach to managing concussion in sport.[16–20] Athletes sustaining concussion have displayed deficiencies in neurocognitive functioning such as attention, memory, concentration, and information processing as a result of cerebral concussion.[16,21–31] Additionally, the areas of the brain disrupted as a result of concussion or traumatic brain injury have been reported to be responsible for the maintenance of postural equilibrium.[1–3,32–41] As a result of these findings, neurocognitive and postural stability measures have been proposed as means by which concussion can be objectively assessed. Traditionally, clinicians have used the Romberg test for assessing disequilibrium in head-injured athletes, but only recently has computerized posturography been available to offer a more objective, challenging, and quantifiable assessment.

Although published reports have contributed significantly to our understanding of injury mechanisms, high-risk sports and positions, and symptoms associated with concussion, they have been limited in their ability to help us substantiate the recommended concussion grading scales and RTP guidelines. Most experts would agree that the nearly 20 proposed grading scales and RTP guidelines are very safe when adhered to closely; however, clinicians often question their practicality. While these scales and guidelines may be safe, most are based on a collection of clinical observations rather than experimentally based research findings. Much of the disagreement surrounding these grading scales and RTP guidelines stems from a dearth of scientific data to support them, which has resulted in the lack of a gold standard in the management of sport-related concussion. Most of the grading scales place significant weight on loss of consciousness (LOC) and amnesia, yet these have not proven to be predictive of neurocognitive decline, motor insufficiency, or long-term disability.

Our purpose was to investigate the effect of concussion on postural stability and neurocognitive function in athletes and furthermore to determine if injured players experiencing LOC and amnesia had a slower recovery than those who did not experience LOC or amnesia. The findings may provide insight into a more comprehensive approach for obtaining objective information with which clinicians can assess sport-related concussion.

METHODS

Subjects consisted of 36 collegiate athletes who sustained a concussion during either practice or competition and 36 recruited matched control subjects. Injured players who had received preseason baseline neuropsychological and postural stability testing were assessed on days 1, 3, and 5 postinjury. The matched control subjects were athletes of the same approximate age, height, and weight as their injured counterparts who had played approximately the same amount of time on the day of their matched counterparts' injuries. They were assessed according to the same schedule as the injured subjects. Control subjects who had sustained a concussion within 6 months of testing or who presented with a vestibular deficit or an acute musculoskeletal injury that affected postural equilibrium were excluded from the study.

Concussion was defined as injury to the brain caused by a sudden acceleration or deceleration of the head that resulted in any immediate, but temporary, alteration in brain functions, such as loss of consciousness, blurred vision, dizziness, amnesia, or memory impairment. All injured athletes were referred to the Sports Medicine Research Laboratory after being evaluated by a certified athletic trainer and team physician. All subjects were informed of the procedures and inherent risks of the investigation. They read and signed an informed consent form in accordance with the University of North Carolina's Academic Affairs Institutional Review Board, which approved the study. In addition to the postural stability and cognitive assessments, any current signs and symptoms associated with concussion were recorded at the time of the assessment.

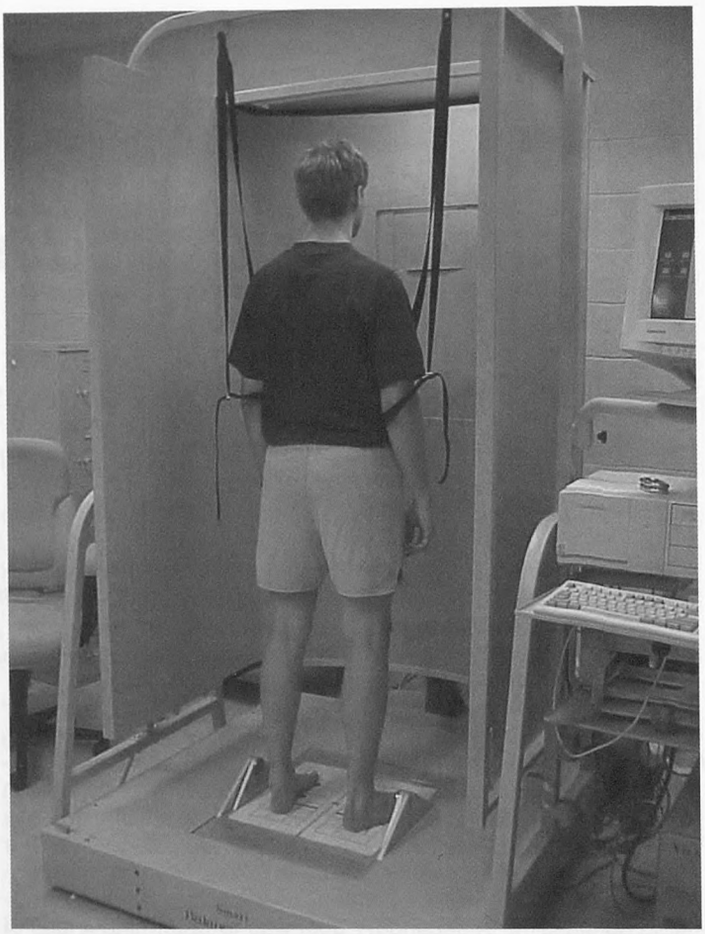

Figure 1. Smart Balance Master System.

Postural Stability Assessment

We took 2 measures of postural stability during each assessment. The first measure was the Sensory Organization Test (SOT) administered on the NeuroCom Smart Balance Master System (NeuroCom International, Inc, Clackamas, OR) (Figure 1). This forceplate system measures vertical ground reaction forces produced by the body's center of gravity moving around a fixed base of support. The SOT is designed to systematically disrupt the sensory selection process by altering available somatosensory or visual information or both while measuring a subject's ability to minimize postural sway.

The test protocol consists of 18 total trials (20 seconds each), in which the subject is asked to stand as motionless as possible with the feet shoulder-width apart. Three trials are completed for each of the 6 conditions presented in Figure 2, in which 3 different visual conditions (eyes open, eyes closed, sway-referenced visual surround) are crossed with 2 different

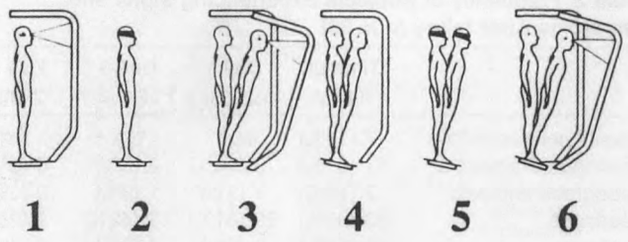

Figure 2. Six testing conditions for Sensory Organization Test used with NeuroCom's Smart Balance Master System.

surface conditions (fixed, sway referenced). The term *sway referencing* involves the tilting of the support surface or visual surround (or both) to directly follow the athlete's center-of-gravity (COG) sway. During sway-referenced support-surface conditions (4–6), the forceplate tilts synchronously with the subject's anterior-posterior (A-P) COG sway. Similarly, during sway-referenced visual-surround conditions (3, 6), the visual surround tilts synchronously with A-P COG sway. Sway referencing causes orientation of the support surface or surround to remain constant relative to body position. The SOT can assess the subject's ability to ignore the inaccurate information from the sway-referenced sense(s). A composite equilibrium score describing a person's overall level of performance during all of the trials in the SOT is calculated, with higher scores indicating better balance performance. The composite score is a weighted average of the equilibrium scores from the 18 trials (3 for each of the 6 conditions); the scores from conditions 1 and 2 are weighted slightly less than those of conditions 3 through 6. The equilibrium scores from each of the trials represent a nondimensional percentage comparing the subject's peak amplitude of A-P sway with the theoretical A-P limit of stability. The theoretical limit of stability is based on the individual's height and size of the base of support. It represents an angle at which the person can lean in any direction before the COG would move beyond a point that allows him or her to remain upright (ie, point of falling). Lower percentages result in a higher (better) composite score. As part of the SOT, relative differences between the equilibrium scores of various conditions are calculated using ratios to reveal specific information about each of the sensory modalities involved with maintaining balance. These ratios are useful in identifying sensory integration problems, as lower ratios indicate an inability to compensate for disruptions in selected sensory inputs. The vestibular ratio is computed by using scores obtained in condition 5 (eyes closed, sway-referenced platform) and condition 1 (eyes open, fixed platform). This ratio indicates the relative reduction in postural stability when visual and somatosensory inputs are simultaneously disrupted. The visual ratio is obtained by comparing condition 4 with condition 1, and the somatosensory ratio compares condition 2 with condition 1.

The second test of postural stability was the Balance Error Scoring System (BESS) and served as a clinical evaluation measure independent of the forceplate measure. Three different stances (double, single, and tandem) were completed twice, once while on a firm surface and once while on a 10-cm-thick piece of medium-density foam (thickness 45 cm^2 × 13 cm, density 60 kg/m^3, load deflection 80–90 kg) for a total of 6 20-second trials. Subjects were instructed to assume the required stance by placing their hands on their iliac crests and were informed that when they closed their eyes, the test would

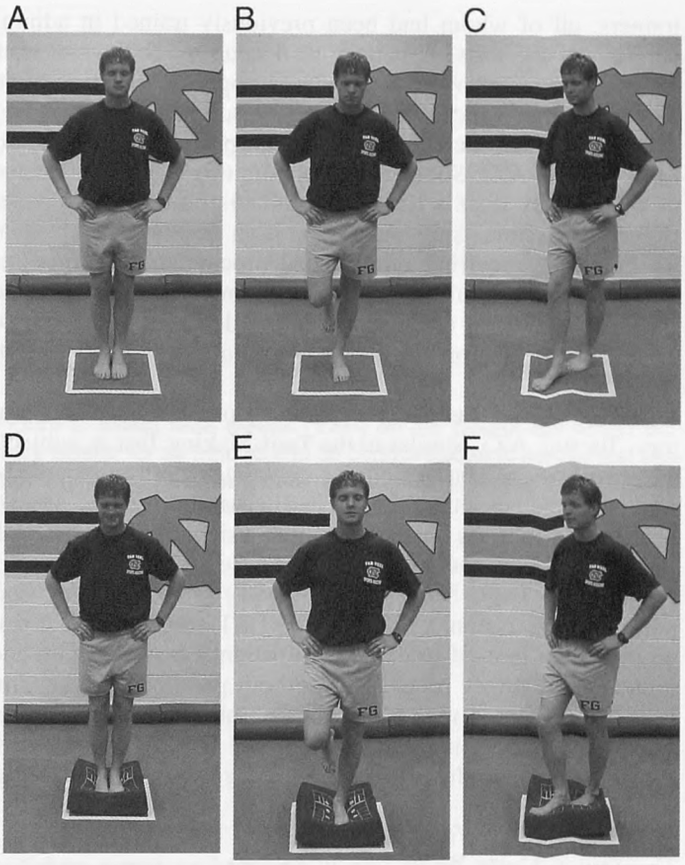

Figure 3. Balance Error Scoring System (BESS) performed on the firm surface (top, A–C) and foam surface (bottom, D–F).

Table 1. Balance Error Scoring System

Errors
Hands lifted off iliac crests
Opening eyes
Step, stumble, or fall
Moving hip into more than 30° of flexion or abduction
Lifting forefoot or heel
Remaining out of testing position for more than 5 seconds

BESS score calculated by adding 1 error point for each error committed.

begin. During the single-limb stance trials, subjects were asked to maintain the contralateral limb in 20° of hip flexion and 45° of knee flexion. Additionally, we asked subjects to stand quietly and as motionless as possible in the stance position, keeping their hands on their iliac crests and their eyes closed (Figure 3). We demonstrated the 6 types of errors (Table 1) before testing and instructed the subjects to minimize the number of errors during the test trials. They were further told to make any necessary adjustments in the event that they lost their balance and to return to the testing position as quickly as possible. Performance was scored by adding 1 error point for each error committed. This method of testing has been previously described in detail and has been shown to be both valid and reliable using normal subjects.[42]

Neurocognitive Assessment

We assessed neurocognitive function using standardized administration and scoring procedures in a quiet, controlled environment. The tests were administered by certified athletic

trainers, all of whom had been previously trained in administration of the tests. A battery of 5 neuropsychological tests was used to assess various aspects of cognitive function often depressed after concussion. The test descriptions follow.

Trail-Making Test A (Reitan Neuropsychological Laboratory, Tucson, AZ). Subjects completing this test are instructed to sequentially trace a list of 25 numbers on a piece of paper as fast as possible using a pen. This is an attentional and visual tracking task requiring rapid visual processing. The time required for successful completion is recorded, adding 1 second for each sequential error committed. Adding of 1 second for each sequential error is a modification to the standard administration.

Trail-Making Test B (Reitan Neuropsychological Laboratory, Tucson, AZ). Similar to the Trail-Making Test A, subjects are instructed to connect circles containing both numbers (1–13) and alphabet letters (A–L) in alternating numeric and alphabetic fashion as fast as possible using a pen. This task assesses working memory and rapid visual processing.

Wechsler Digit Span Test (WDST) (Psychological Corporation, San Antonio, TX). The WDST consists of a 2-part protocol and is used to examine a patient's concentration and immediate memory recall. During both parts of the test, subjects are presented with a series of numbers and asked to repeat the digits in either the same order (Digits Forward) for the first part or in the reverse order (Digits Backward) for the second part.

Stroop Color Word Test (Stoelting Co, Wood Dale, IL). The Stroop Color Word Test is designed to assess cognitive flexibility, attention, and response inhibition by examining a subject's ability to separate word and color-naming stimuli through the use of 3 separate subtests. Only subtest 3 was analyzed in this study. The words RED, BLUE, and GREEN are randomly listed in 5 columns of 20 items. Subjects are instructed to read aloud the color of the print for each item (possibly the word RED printed in green ink). Subjects are given 45 seconds to complete as many items as possible.

Hopkins Verbal Learning Test (HVLT) (Johns Hopkins University, Baltimore, MD). Each form of the HVLT consists of a 12-item word list composed of 4 words from 3 semantic categories used for assessing verbal memory and learning. The subject is instructed to listen carefully and memorize the word list. The subject then recalls as many words as possible in any order. The examiner records the number of correct responses, and the same procedure is repeated for 2 more trials. The numbers of correct responses on the trials are added for a total immediate-memory recall score. After the third trial, the subject is read 24 words and asked to identify words contained in the original list. The number of incorrect responses is subtracted from the overall delayed recognition score.

DATA ANALYSIS

We calculated separate mixed-model (1 between, 1 within), repeated-measures analyses of variance (ANOVAs) for the SOT composite score, each of the 3 SOT ratio scores, the BESS score, and each of the neuropsychological test scores using SPSS 10.0 statistical software (SPSS, Inc, Chicago, IL). These analyses determined if significant differences existed between groups (injured and control) and across postinjury days for each of the dependent variables. An additional mixed-model repeated-measures ANOVA performed only on the injured group compared those subjects with LOC or amnesia or both against those subjects without LOC or amnesia across the same postinjury days. Tukey post hoc analyses were performed for all significant interactions. Level of significance ($P < .05$) was set a priori for all statistical analyses.

Table 2. Frequency of Subjects Experiencing Signs and Symptoms After Injury (n = 36)

	Time of Injury	Day 1 Postinjury	Day 3 Postinjury	Day 5 Postinjury
Loss of consciousness	7 (19%)	NA*	NA	NA
Anterograde amnesia	11 (31%)	5 (14%)	2 (6%)	0 (0%)
Retrograde amnesia	7 (19%)	1 (3%)	1 (3%)	0 (0%)
Headache	33 (92%)	29 (81%)	15 (42%)	9 (25%)
Confusion	20 (56%)	2 (6%)	2 (6%)	0 (0%)
Disorientation	21 (58%)	2 (6%)	0 (0%)	0 (0%)
Blurred vision	24 (67%)	6 (17%)	4 (11%)	2 (6%)
Photophobia	11 (31%)	6 (17%)	4 (11%)	1 (3%)
Dizziness	26 (72%)	6 (17%)	3 (8%)	0 (0%)
Disequilibrium	16 (44%)	3 (8%)	0 (0%)	0 (0%)
Fatigue	9 (25%)	7 (19%)	3 (8%)	4 (11%)
Sleepiness	10 (28%)	5 (14%)	6 (17%)	4 (11%)
Nausea/vomiting	14 (39%)	2 (6%)	2 (6%)	2 (6%)
Irritability	6 (17%)	3 (8%)	3 (8%)	2 (6%)
Neck pain	11 (31%)	12 (33%)	7 (19%)	5 (14%)

*NA indicates not applicable.

Table 3. Frequency Distribution of Injured Subjects by Sport (n= 36)

Team	No. of Subjects
Cheerleading	3
Crew (women's)	1
Basketball (men's)	3
Field hockey	1
Football	10
Lacrosse (men's)	5
Lacrosse (women's)	5
Rugby (women's)	1
Soccer (men's)	1
Soccer (women's)	1
Softball	1
Track (men's)	1
Wrestling	3

RESULTS

Seventy-two subjects participated in this study (50 males and 22 females). The injured group consisted of 36 Division I collegiate athletes (age = 19.5 ± 1.34 years, height = 180.34 ± 11.81 cm, weight = 83.43 ± 19.80 kg) who had sustained a concussion during either practice or competition. The control group consisted of 36 recreational and collegiate athletes (age = 20 ± 2.36 years, height = 179.07 ± 10.47 cm, weight = 81.50 ± 20.45 kg) who were matched with injured subjects for sex, age, height, weight, and activity level. The number of subjects experiencing signs and symptoms associated with concussion is presented in Table 2, and the sample is further described by sport in Table 3. All of the injured subjects were symptomatic at the time of injury, and all but 2 subjects were symptomatic on day 1 postinjury. Twenty-two subjects reported symptoms on day 3 postinjury, and only 12 subjects remained symptomatic beyond day 3 postinjury.

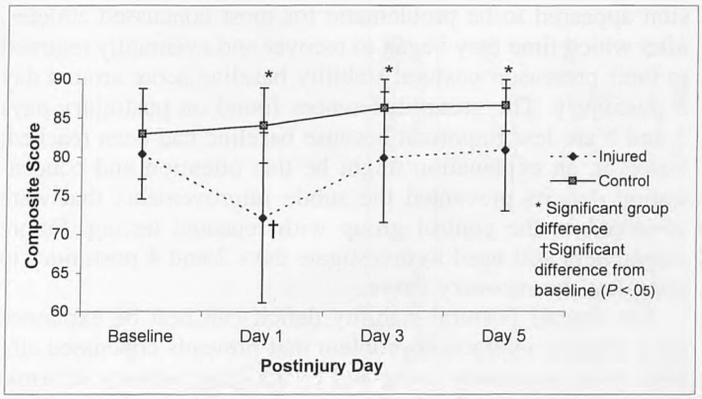

Figure 4. Composite Score means (±SD) on the NeuroCom Smart Balance Master for 36 injured and 36 control subjects across test sessions (preseason through day 5 postinjury). Higher scores represent better performance.

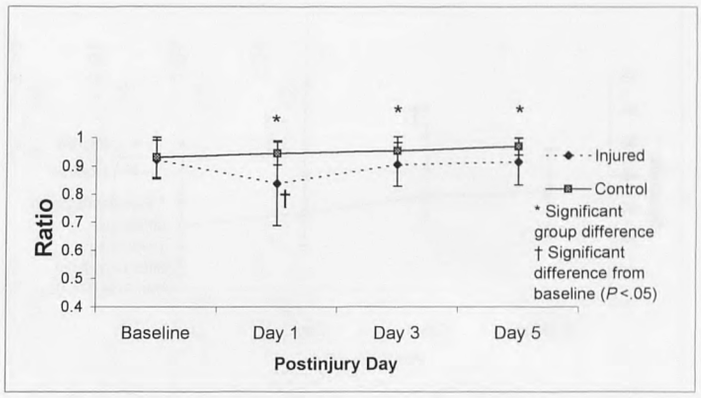

Figure 6. Visual ratio means (±SD) on the NeuroCom Smart Balance Master for 36 injured and 36 control subjects across test sessions (preseason through day 5 postinjury). Higher scores represent better performance.

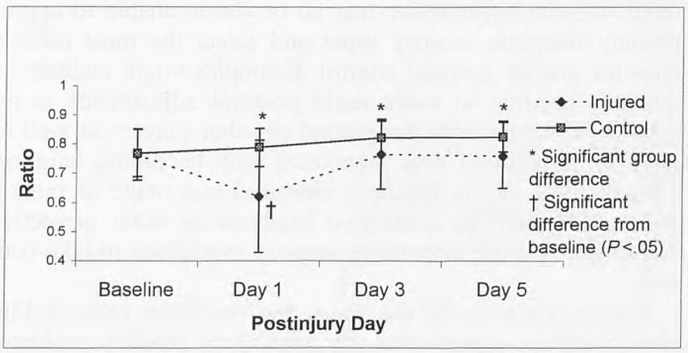

Figure 5. Vestibular ratio means (±SD) on the NeuroCom Smart Balance Master System for 36 injured and 36 control subjects across test sessions (preseason through day 5 postinjury). Higher scores represent better performance.

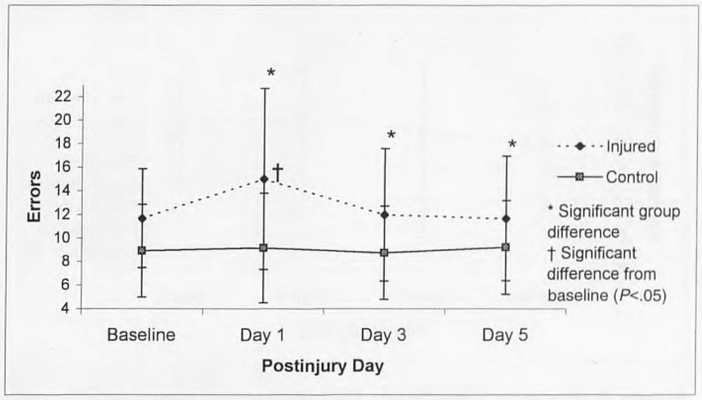

Figure 7. Balance Error Scoring System means (±SD) (combined errors on all 6 trials) for 36 injured and 36 control subjects across test sessions (preseason through day 5 postinjury). Lower scores represent better performance.

Postural Stability Recovery

Repeated-measures ANOVA for SOT composite scores on the Smart Balance Master System revealed a significant group-by-day interaction ($F_{3,210} = 10.17$, $P < .01$). Tukey post hoc analysis revealed that injured subjects demonstrated decreased postural stability on day 1 postinjury in comparison with their baseline as well as in relation to the control subjects' postinjury day 1, 3, and 5 scores, respectively (Figure 4). While differences between control subjects and injured subjects were statistically significant on postinjury days 3 and 5, recovery back to baseline occurred between postinjury days 1 and 3 for the injured subjects. Additional analyses of the ratio scores (visual, vestibular, and somatosensory) revealed significant group-by-day interactions for the visual ratio ($F_{3,210} = 13.57$, $P < .01$) and vestibular ratio ($F_{3,210} = 6.48$, $P < .01$), suggesting that postural stability deficits observed in athletes with concussions could be linked to a sensory interaction problem. Mean scores for these ratios across the baseline and 3 postinjury test sessions are presented in Figures 5 and 6. Post hoc analyses again revealed low vestibular and visual ratios on day 1 postinjury in injured subjects compared with their baseline and postinjury day 3 as well as the control subjects' postinjury day 1, 3, and 5 ratio scores.

The BESS results revealed a similar trend to those of the SOT, as the repeated-measures ANOVA showed a significant group-by-day interaction ($F_{3,210} = 2.68$, $P < .05$). Tukey post hoc analysis again demonstrated that injured subjects had decreased postural stability on day 1 postinjury in comparison with baseline and day 3 postinjury scores as well as the control subjects' postinjury day 1, 3, and 5 scores (Figure 7).

Neurocognitive Recovery

Repeated-measures ANOVA revealed significant group-by-day interactions for 3 of the neuropsychological tests; however, only the Trail-Making Test B (Figure 8) and Digit Span Test Backward (Figure 9) scores resulted in a recovery curve that could be logically associated with concussion and subsequent lowered neuropsychological performance. Results from these 2 tests revealed significant differences between the control group and injured group at postinjury days 1 (Trail-Making Test B and WDST) as well as postinjury days 3 and 5 (Trail-Making Test B) but failed to show a statistically significant decline in performance between baseline and postinjury scores. Despite the absence of this significant difference across days for the injured group, there is still a trend in the opposite direction from control subjects' scores at postinjury day 1, suggesting that injured subjects may have mild cognitive deficits during the initial days after injury on these 2 tests. Group comparisons for all neuropsychological test scores are presented in Table 4.

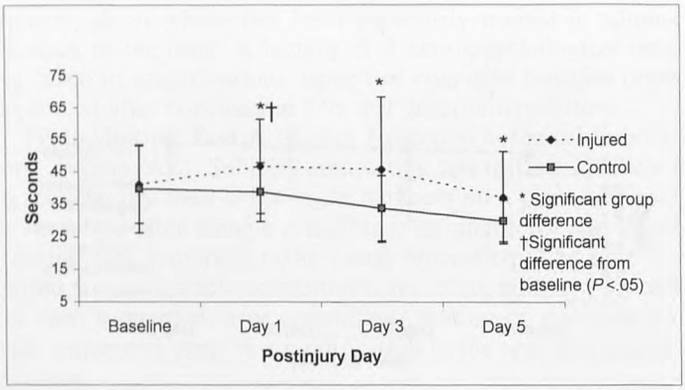

Figure 8. Trail-Making Test B means (±SD) for 18 injured and 23 control subjects across test sessions (preseason through day 5 postinjury). Lower scores represent better performance.

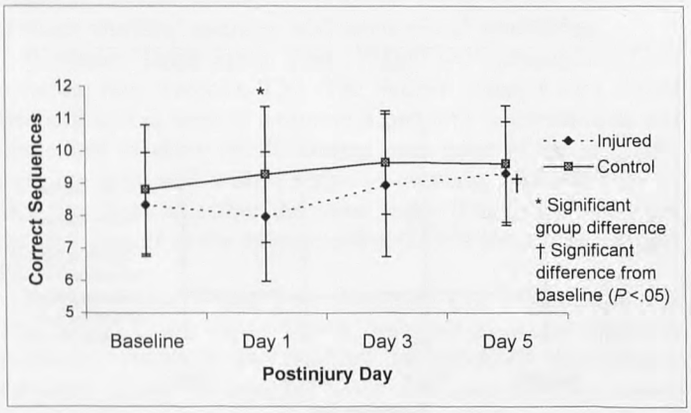

Figure 9. Wechsler Digit Span Test Backward means (±SD) (combined errors on all 6 trials) for 36 injured and 36 control subjects across test sessions (preseason through day 5 postinjury). Higher scores represent better performance.

Loss of Consciousness and Amnesia

We compared injured subjects with (n = 12) and without (n = 24) amnesia and with (n = 7) and without (n = 29) LOC and found no significant group-by-day interactions or group main effects for either SOT or BESS scores ($P > .05$). The same analyses performed on neuropsychological test variables also revealed no significant interactions or main effects ($P > .05$) (Table 5). These findings indicate that injured athletes who experienced LOC or amnesia (or both) performed no worse than injured athletes who did not experience LOC or amnesia (or both) on any of the postural stability or neuropsychological tests.

DISCUSSION

The use of both neuropsychological screening and postural stability (balance) testing is gradually becoming standard practice in the management of sport-related concussion. However, much discussion continues among researchers and clinicians regarding the best test battery and test sequencing. Additionally, debate surrounds the emphasis placed on LOC and amnesia in the grading of concussion. Perhaps the most important finding in our investigation was that athletes recovering from cerebral concussion demonstrated postural stability deficits most likely linked to a sensory interaction problem during the immediate postinjury period. The initial 2 days after concussion appeared to be problematic for most concussed athletes, after which time they began to recover and eventually returned to their preseason postural stability baseline score around day 3 postinjury. The group differences found on postinjury days 3 and 5 are less important because baseline had been reached; however, an explanation might be that attention and concentration deficits prevented the subtle improvements that were observed in the control group with repeated testing. Future researchers will need to investigate days 2 and 4 postinjury to complete the recovery curve.

The overall postural stability deficit can best be explained by a sensory interaction problem that prevents concussed athletes from accurately using and exchanging sensory information from the visual, vestibular, and somatosensory systems. The integration of vestibular and visual information is essential for the maintenance of equilibrium under certain altered conditions similar to those performed during the SOT.[43–47] If a subject has difficulty balancing under conditions in which environmental or sensory conditions (or both) have been altered, we can hypothesize that he or she is unable to appropriately integrate sensory input and select the most reliable cues for precise postural control. Examples might include the athlete's needing to make rapid postural adjustments in response to impact with the ground or other players as well as changes in reaction time associated with becoming fatigued. Occasionally, vision becomes obscured and range of motion becomes limited by protective headgear or other protective padding, requiring alternative sensory modalities to take control.

Recovery curves for the visual and vestibular ratios in Figures 5 and 6 suggest that the postinjury stability problems occurred primarily under the altered sensory conditions involving unstable (sway-referenced) surface conditions and either normal or absent vision (conditions 4 and 5). These results affirm our earlier findings of significant differences between concussed athletes and control subjects on day 1 postinjury compared with preseason or subsequent tests, or both, using the Chattecx Balance System[1] (Chattanooga Group, Hixson, TN) and the NeuroCom Equitest.[2] The current findings also concur with other recent investigations of patients with mild head or neck injuries. Such studies have revealed significantly higher magnitudes of postural instability during inaccurate visual conditions or altered surface conditions.[40,41] Our findings on the BESS are also promising, given that they are consistent with an earlier finding of a significant relationship between SOT and BESS scores in a group of 16 concussed athletes.[3] Based on our findings, the BESS is a practical, valid, and cost-effective method of objectively assessing postural stability in athletes suffering from concussion.

Much speculation exists about the underlying neural deficits causing postural instability after cerebral concussion. Some authors have suggested that the observed balance problems are diffuse and rarely related to pure vestibular and cerebellar dysfunction,[37,38] while others claim that concussed individuals may need longer processing time to readjust balance because of slow subcortical activity and spatiotemporal disruption of postural responses.[41] Minor axonal dysfunction at the level of the brainstem or cerebellum has also been proposed as the potential cause of postural instability after concussive injury.[48] Although this may appear unlikely in sport-related concussion, the sudden deceleration of the brain causes shearing forces, which in turn cause transport failures in the axon.[49] In other words, disruption of any axons responsible for transmitting

Table 4. Neuropsychological Test Results in Injured and Control Subjects: Mean (Standard Deviation)

Test	Preseason Baseline	Day 1 Postinjury	Day 3 Postinjury	Day 5 Postinjury	Group Main Effect	Group × Day Interaction
Hopkins Verbal Learning Test, Immediate Memory						
Injured	30.62 (3.70)	29.17 (4.21)	30.19 (4.94)	29.09 (4.78)	$F_{1,70} = 1.75$	$F_{3,210} = 3.67$
Control	30.70 (3.35)	30.89 (2.86)	29.86 (6.20)	31.78 (3.24)*	$P = .19$	$P = .01$
Hopkins Verbal Learning Test, Recognition						
Injured	11.66 (0.62)	11.14 (1.48)	11.50 (1.28)	11.65 (0.63)	$F_{1,70} = 2.19$	$F_{3,210} = 2.40$
Control	11.79 (0.52)	11.83 (0.39)	11.40 (1.65)	11.74 (0.50)	$P = .14$	$P = .07$
Wechsler Digit Span Test Forward						
Injured	10.26 (1.32)	9.67 (1.59)	10.50 (1.40)	10.43 (1.17)	$F_{1,70} = 3.35$	$F_{3,210} = 1.17$
Control	10.58 (1.08)	10.39 (1.25)	10.67 (1.67)	10.93 (1.14)	$P = .07$	$P = .32$
Wechsler Digit Span Test Backward						
Injured	8.34 (1.58)	7.97 (1.99)	8.94 (2.23)	9.30 (1.64)†	$F_{1,70} = 3.39$	$F_{3,210} = 2.58$
Control	8.81 (1.99)	9.28 (2.10)*	9.65 (1.64)	9.59 (1.82)	$P = .07$	$P = .05$
Stroop Color Word Test 3						
Injured	48.99 (10.93)	49.21 (8.62)	56.50 (11.59)	57.89 (13.07)	$F_{1,70} = 5.68$	$F_{3,210} = 0.91$
Control	55.02 (10.08)	56.06 (10.95)	59.98 (12.49)	63.11 (10.34)	$P = .02$	$P = .44$
Trail-Making Test A						
Injured	19.99 (6.37)	22.34 (7.60)	20.56 (8.29)	17.51 (6.86)	$F_{1,57} = 0.99$	$F_{3,171} = 1.37$
Control	19.90 (5.31)	21.01 (5.60)	17.58 (4.88)	16.59 (3.67)	$P = .32$	$P = .27$
Trail-Making Test B						
Injured	41.05 (11.96)	46.71 (14.62)†	45.82 (22.54)	37.13 (14.27)	$F_{1,38} = 4.14$	$F_{3,114} = 2.74$
Control	39.73 (13.63)	38.74 (9.12)*	33.80 (10.01)*†	29.95 (6.42)*†	$P = .05$	$P = .05$

Higher scores represent better performance on all tests except Trail-Making Tests A and B.
*Significant group difference (group × day interaction).
†Significant difference from baseline (group × day interaction).

Table 5. Postural Stability and Neuropsychological Test Results in Athletes With and Without Loss of Consciousness (LOC) and Amnesia: Mean (Standard Deviation)*

Test	Preseason Baseline	Day 1 Postinjury	Day 3 Postinjury	Day 5 Postinjury	Group Main Effect	Group × Day Interaction
Sensory Organization Test						
LOC	84.86 (2.95)	70.41 (10.06)	79.57 (12.94)	79.24 (12.07)	$F_{1,34} = 0.01$	$F_{3,102} = 1.74$
No LOC	79.29 (4.69)	72.49 (11.73)	79.90 (7.33)	81.23 (6.81)	$P = .91$	$P = .16$
Amnesia	79.48 (5.01)	71.55 (12.52)	78.65 (11.59)	78.93 (11.49)	$F_{1,34} = 0.58$	$F_{3,102} = 0.140$
No amnesia	80.82 (4.90)	72.35 (10.94)	80.43 (6.61)	81.80 (5.92)	$P = .45$	$P = .94$
Balance Error Scoring System						
LOC	12.36 (2.32)	15.86 (5.58)	12.00 (4.16)	16.14 (5.93)	$F_{1,34} = 1.16$	$F_{3,102} = 1.58$
No LOC	11.48 (4.59)	14.79 (8.21)	11.93 (5.94)	10.58 (4.70)	$P = .29$	$P = .198$
Amnesia	9.13 (4.89)	12.25 (6.02)	11.33 (7.80)	10.75 (6.02)	$F_{1,34} = 3.15$	$F_{3,102} = 0.96$
No amnesia	12.92 (3.30)	16.38 (8.20)	12.25 (4.25)	12.12 (5.07)	$P = .09$	$P = .41$
Hopkins Verbal Learning Test, Immediate Memory						
LOC	32.21 (3.49)	29.86 (5.46)	32.86 (5.24)	30.86 (4.84)	$F_{1,34} = 2.04$	$F_{3,102} = 0.57$
No LOC	30.24 (3.70)	29.00 (3.95)	29.55 (4.74)	28.66 (4.74)	$P = .162$	$P = .63$
Amnesia	29.96 (4.64)	29.08 (4.56)	29.50 (5.28)	29.17 (5.29)	$F_{1,34} = 0.17$	$F_{3,102} = 0.29$
No amnesia	30.96 (3.18)	29.21 (4.13)	30.54 (4.84)	29.05 (4.61)	$P = .69$	$P = .82$
Hopkins Verbal Learning Test, Recognition						
LOC	11.55 (0.52)	11.42 (0.53)	11.71 (0.76)	11.71 (0.49)	$F_{1,34} = 0.26$	$F_{3,102} = 0.28$
No LOC	11.69 (0.65)	11.10 (1.62)	11.45 (1.38)	11.64 (0.67)	$P = .62$	$P = .84$
Amnesia	11.53 (0.78)	11.50 (0.90)	11.83 (0.39)	11.75 (0.45)	$F_{1,34} = 1.15$	$F_{3,102} = 1.00$
No amnesia	11.73 (0.53)	10.96 (1.68)	11.33 (1.52)	11.61 (0.71)	$P = .29$	$P = .40$
Wechsler Digit Span Test Forward						
LOC	10.18 (1.07)	10.00 (0.82)	10.71 (1.49)	10.71 (0.95)	$F_{1,34} = 0.34$	$F_{3,102} = 0.33$
No LOC	10.27 (1.39)	9.59 (1.72)	10.44 (1.40)	10.25 (1.21)	$P = .56$	$P = .80$
Amnesia	10.36 (1.07)	9.42 (1.83)	10.25 (1.48)	10.25 (1.42)	$F_{1,34} = 0.24$	$F_{3,102} = 0.47$
No amnesia	10.21 (1.44)	9.79 (1.47)	10.62 (1.38)	10.39 (1.05)	$P = .63$	$P = .71$
Wechsler Digit Span Test Backward						
LOC	8.77 (1.47)	8.43 (2.30)	9.86 (2.12)	9.86 (1.86)	$F_{1,34} = 1.30$	$F_{3,102} = 0.27$
No LOC	8.24 (1.62)	7.86 (1.94)	8.72 (2.23)	9.17 (1.58)	$P = .26$	$P = .85$
Amnesia	8.44 (1.44)	8.45 (1.44)	9.25 (2.45)	9.33 (2.23)	$F_{1,34} = 0.07$	$F_{3,102} = 0.26$
No amnesia	8.29 (1.68)	8.29 (1.68)	8.79 (2.15)	9.29 (1.30)	$P = .79$	$P = .86$
Stroop Color Word Test 3						
LOC	49.93 (10.48)	45.29 (11.10)	56.14 (13.20)	60.43 (7.81)	$F_{1,32} = 0.01$	$F_{3,102} = 1.36$
No LOC	48.76 (11.20)	50.16 (7.86)	56.59 (11.42)	57.28 (14.09)	$P = .95$	$P = .26$
Amnesia	48.04 (11.41)	47.58 (10.15)	52.67 (14.66)	54.92 (15.34)	$F_{1,34} = 1.13$	$F_{3,102} = 0.62$
No amnesia	49.99 (10.93)	50.03 (7.86)	58.42 (9.49)	59.38 (11.86)	$P = .30$	$P = .605$
Trail-Making Test B						
Amnesia	37.76 (6.41)	43.11 (12.49)	40.78 (19.27)	35.67 (12.86)	$F_{1,15} = 1.07$	$F_{3,45} = 0.46$
No amnesia	44.75 (15.83)	50.75 (16.58)	51.50 (25.84)	38.78 (16.43)	$P = .32$	$P = .72$

*Higher scores represent better performance on all tests except the Sensory Organization Test and Trail-Making Test B. Insufficient data available for Trail-Making Test B (LOC versus no LOC) and Trail-Making Test A (LOC versus no LOC, amnesia versus no amnesia).

information to centers responsible for maintenance of balance can lead to postural instability. Another possible explanation for postural stability or neurocognitive deficits after concussion could center on the biochemical cascade models that have been developed in animals. Hovda et al[50] have described a neurochemical and metabolic cascade that occurs after cerebral concussion. Within the first hour of the concussion and up to several days postinjury, the brain is thought to be in a vulnerable state due to increased glucose metabolism and diminished cerebral blood flow. Although only speculative at this time, the imbalance between glucose needs and available supply during the first few days after injury could partially explain the deficits observed in our study.

Because the SOT cannot identify a specific lesion or pathologic condition, the source of concussion-related balance deficits is unknown. We know it will involve either peripheral sensory receptors responsible for detecting motion or central structures, primarily the cerebellum, cerebral cortex, and brain stem, which are involved in the perception and integration of sensory information. Some authors have reported that concussive trauma to the head or neck can result in changes to the normal weighing of sensory cues and create a reorganization pattern of the remaining sensory cues underlying postural control.[51,52] The depressed visual and vestibular ratios found in our study may best be explained by this phenomenon.

The primary purposes of the human vestibular system are to (1) maintain the eyes fixed on a stationary target in the presence of head and body movement and (2) maintain balance in conjunction with additional information from visual and somatosensory inputs. To accomplish the first, the semicircular canals of the vestibular labyrinth sense angular acceleration of the head, converting it to velocity information and sending it through the vestibulo-ocular reflex pathways to the ocular muscles. Second, balance is maintained by central integration of vestibular, visual, and somatosensory orientation information. The vestibular system provides angular information from the semicircular canals and linear acceleration information (including gravity) from the utricles and saccules of the inner ear and transmits it via the vestibulospinal spinal tract to the spinal and lower extremity muscles. Under normal conditions, visual and somatosensory information is adequate for maintenance of balance; however, in populations with known vestibular deficits, the inner ear's sense of balance is essential when visual and somatosensory inputs are disrupted or provide conflicting information.

Two mechanisms are possible for vestibular involvement after cerebral concussion: (1) the peripheral receptors themselves may be damaged and provide inaccurate senses of motion or (2) the brain centers responsible for central integration of vestibular, visual, and somatosensory information may be impaired. Mallinson and Longridge[53] found evidence of central integration balance deficits and subtle peripheral vestibular deficits when comparing SOT and electronystagmography results in patients with mild head injury from an associated whiplash injury. These findings suggest that various combinations of peripheral and central deficits may be the cause of balance deficits in athletes with concussion. An additional factor that surfaced from our research is the possibility that concentration and attention impairments identified on day 1 postinjury could be contributing factors to decreased postural stability. Future researchers should focus on this potential relationship.

Our findings did not substantiate the use of a long neuropsychological test battery for identifying underlying pathology in concussed athletes during the acute stage of injury. The injured athletes in this study demonstrated significantly poorer performance on selected tests of concentration and immediate memory recall (WDST Backward) and rapid visual processing and working memory (Trail-Making Test B). These 2 neuropsychological tests were the only ones to reveal significant differences relative to the control group at day 1 postinjury. Scores on the Trail-Making Test B also revealed group differences at postinjury days 3 and 5: the injured subjects did not appear to be learning the task as efficiently as the control subjects over the repeated tests. Therefore, understanding the learning curve may be as important as having a baseline measurement in cases in which improvement should be expected with repeated testing. Our research findings leave us with further questions surrounding the efficacy of certain neuropsychological testing during the acute stage of injury in athletes with relatively mild injuries. Is there really a cognitive deficit, or are these tests not sensitive enough to detect cognitive decline after this type of injury? While selected tests may be sensitive to the injury, others (such as the Trail-Making Test A, HVLT, Stroop Color Word Test 3, and WDST Forward) may not be as sensitive. In our study, the efficacy of the HVLT may be questioned in an athletic population given the sudden spreading of scores (between injured and control subjects) at day 5 postinjury. We propose that the motivation levels of many injured players by day 5 postinjury may explain the dropoff in HVLT scores. Several researchers have reported significant neuropsychological deficits during the acute stage of concussion,[21–27] but the literature has failed to establish a consensus on which tests are most sensitive or which areas of cognition are most affected. Based on our findings, working memory, immediate memory recall, concentration, and rapid visual processing abilities appear to be affected during the initial stage of injury recovery. Interestingly, a previous study by our group found that the WDST and Trail-Making Test B were the best predictors of symptom severity at day 3 postinjury in collegiate football players.[54]

Some researchers suggest that neuropsychological testing may play a more important role in identifying undetected pathologic conditions once the athlete appears asymptomatic or in cases of postconcussion syndrome, when the athlete experiences lingering signs and symptoms.[25,55,56] In analyzing our control subjects more closely, we found a very subtle learning curve between baseline and day 1 postinjury on the Stroop Color Word Test 3, the Trail Making Test B, and the WDST Backward, similar to that reported by Oliaro et al.[57] As noted previously, the injured subjects in our study demonstrated a deviation from this learning curve, as they did not learn at the same rate as control subjects on these 2 tests. Injured subjects also had heightened variability among subjects (larger standard deviations) on several of the neuropsychological tests on day 1 postinjury, further supporting our claim that they probably had mild cognitive deficits. Echemendia et al[28] reported similar learning trends and suggested that a return to preseason baseline score may not be a sufficient indicator of "normal" functioning. Our findings support their claim that injured players should probably be expected to exceed baseline scores on measures with known practice effects before they are rendered "normal."

The recovery for signs and symptoms (Table 2) appears to follow the recovery for postural stability and selected tests of neurocognitive function. With the exception of headache, most

signs and symptoms had resolved by day 3 postinjury. However, 42% of the athletes still reported a headache on day 3 postinjury, and 25% reported a headache on day 5 postinjury. While our purpose was not to study RTP decision making, most players had returned to restricted participation by day 5 postinjury. Only those with lingering symptomatology were withheld beyond day 10 postinjury. When the injured subjects were stratified by LOC and amnesia, no significant interactions or group main effects (Table 5) were seen for any of the postural stability or neuropsychological measures. This finding suggests that neither LOC nor amnesia was associated with course of recovery or was a predictor of postural stability or neuropsychological test performance during the acute stage of injury. Our findings are consistent with those of Lovell et al,[58] who reported no neuropsychological deficits in patients with traumatic LOC compared with those without LOC or those who were uncertain about LOC. Clinicians should, therefore, be cautious about overemphasizing LOC and amnesia while potentially ignoring other important signs and symptoms.

CONCLUSIONS

No two concussions are created equal. Some injuries may involve the cognitive centers of the brain, while others involve the balance centers. The motor domain of neurologic functioning should, therefore, be assessed along with the cognitive domain after all concussions. Our findings reveal that postural stability deficits were present in most athletes sustaining cerebral concussion, and often they do not resolve until 3 days postinjury. It appears that sensory feedback from the visual, vestibular, and somatosensory systems in athletes with concussion is not properly processed during the first few days after injury. Although neuropsychological testing has become popular in recent years for assessing the cognitive domain of neurologic functioning, more research is necessary to establish the most sensitive, practical, and useful battery of tests. Of the tests we used in this study, the WDST Backward and the Trail-Making Test B were most sensitive for tracking recovery after cerebral concussion in athletes. Memory and concentration deficits during the immediate recovery period can be detected using these tests. One of the limitations of our study is that it involved subjects with relatively mild concussions (grades 1 and 2), most of whom had not experienced previous concussions. Future researchers should attempt to compare the neuropsychological and postural stability recovery curves in athletes with more serious and recurrent episodes of concussion.

In the absence of sophisticated forceplate systems, the BESS is an efficient and cost-effective alternative for objectively assessing postural stability. Similarly, the clinician who does not have access to traditional neuropsychological assessment tools should at the very least find simple and easy-to-use alternatives, such as the Standardized Assessment of Concussion.[30,31] Once validated in the athletic setting, computerized neuropsychological assessment tools may become a more useful option for the sports medicine clinician. Finally, we recommend that athletes sustaining a concussion never be permitted to return to activity until all postconcussive symptoms have resolved. Based on our findings, athletes whose symptoms resolve quickly after injury should, at the very least, be held from competition for 3 days after any episode in which they might have sustained a concussion. Clinicians should seriously consider whether or not they might be placing athletes at risk by returning them earlier than 3 days postinjury. Clinicians should also realize that postural stability and neuropsychological testing are only 2 pieces of a very large puzzle in the assessment of concussion. Concussive injury may not necessarily affect the postural control system or neurocognitive areas of the brain in every patient. Furthermore, the presence of LOC or amnesia or both may have little to do with the recovery rate, and therefore, these conditions should not be overemphasized in the management of concussion. The most comprehensive concussion assessment will involve a sound clinical examination with close monitoring of all symptoms while including objective measurements such as postural stability testing and neuropsychological testing.

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Standardized Mental Status Testing on the Sideline After Sport-Related Concussion

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Objective: The effects of concussion on mental status are often difficult to assess on routine clinical examination. I investigated the efficacy of standardized mental status testing on the sport sideline to detect abnormalities that result from concussion and provide an objective measure of postinjury cognitive recovery.

Design and Setting: All subjects underwent a standardized preseason baseline mental status evaluation. Standardized testing of injured and uninjured control subjects was repeated on the sideline immediately after concussion and 48 hours after injury.

Subjects: Sixty-three high school and collegiate football players with concussion and 55 uninjured control subjects were studied.

Measurements: The Standardized Assessment of Concussion (SAC) was administered to evaluate neurocognitive functioning and neurologic status.

Results: Immediately after concussion, injured subjects performed significantly below preinjury baseline and below uninjured controls on the SAC. Measurable deficits in orientation, concentration, and memory were evident immediately after concussion. A decline in SAC score at time of injury was 95% sensitive and 76% specific in accurately classifying injured and uninjured subjects on the sideline. Injured subjects demonstrated significant improvements in SAC score 48 hours after injury.

Conclusions: Standardized mental status testing can be a valuable tool to assist the sports medicine clinician in detecting the immediate effects of concussion on mental status, tracking resolution of immediate postconcussive mental status abnormalities, and making more informed decisions on return to play after injury.

Key Words: athletic injuries, neuropsychological tests

The sports medicine clinician is faced with many challenges in providing care for young athletes, but perhaps none is more complex than the diagnosis and management of concussion.[1] Detecting concussion is relatively clear-cut when the subject is rendered unconscious or obviously amnestic and disoriented, but more than 90% of sport-related head injuries result in no observable loss of consciousness (LOC) or amnesia and only slight disorientation.[2,3] Milder deficits in neurocognitive functioning (eg, memory, concentration) are more common after an athlete experiences a concussion.[4,5] Unfortunately, the effects of concussion on mental status are usually more subtle than obvious, often making them difficult to identify and fully characterize on routine clinical examination.

Recent efforts have focused on the development of brief, standardized methods of concussion assessment for use on the sport sideline, including measures to evaluate neurocognitive status,[6–8] postural stability,[9,10] and postconcussive symptoms.[11,12] The aim of standardized screening instruments is to reduce the amount of "guesswork" often encountered by the sports medicine clinician in assessing concussion on the sideline. Researchers[4,5] have emphasized the value of standardized mental status testing on the sideline after concussion to clarify the acute effects of injury and establish an index of severity against which to track recovery. More accurate injury assessment is also intended to reduce the risks of recurrent injury, cumulative neuropsychological impairment, and catastrophic outcome associated with sport-related concussion. Certified athletic trainers have made clear their impression that the addition of standardized concussion assessment methods results in a more clinically informative and accurate injury evaluation than a routine clinical examination alone.[13]

The Standardized Assessment of Concussion (SAC)[6,8] was developed to provide clinicians with a more objective and standardized method of immediately assessing an injured athlete's mental status on the sport sideline within minutes of having sustained a concussion. The instrument is intended as a supplement to other methods of concussion assessment (eg, neuropsychological evaluation, postural stability testing) but not meant to be a stand-alone measure to determine the severity of injury or an athlete's readiness to resume participation after concussion. Previous investigators[4,5,14] have documented the psychometric properties of the SAC and supported the sensitivity and clinical validity of the instrument in detecting sport-related concussion and providing a gross measure of postinjury cognitive recovery. The interpretation of validity and reliability data from earlier studies was limited, however, by the lack of a uninjured control group to undergo serial testing on the SAC under the same conditions as the injured subjects. The current study's design included a control group to assess the validity of the SAC in differentiating injured and uninjured subjects after sport-related concussion and provide an objective measure of postinjury neurocognitive recovery.

Table 1. Sample Characteristics and Standardized Assessment of Concussion (SAC) Scores for Injured and Control Subjects

	Uninjured Controls (n = 55)*	Injured Subjects (n = 63)*	Statistic
Age, y	18.22 (1.96)	18.19 (2.24)	$t = -0.07$, $P = .94$
Education, y	13.09 (1.75)	12.89 (2.00)	$t = -0.58$, $P = .56$
Baseline SAC scores			
Total score	26.64 (2.12)	27.14 (1.81)	$t = 1.39$, $P = .16$
Orientation	4.95 (0.23)	4.84 (0.37)	$t = -1.81$, $P = .07$
Immediate memory	14.45 (0.88)	14.56 (0.82)	$t = 0.65$, $P = .52$
Concentration	3.35 (1.36)	3.59 (1.19)	$t = 1.03$, $P = .31$
Delayed recall	3.89 (1.24)	4.16 (0.92)	$t = 1.34$, $P = .18$
Concussion SAC scores			
Total score	26.87 (2.05)	22.46 (3.61)	$t = -8.01$, $P < .001$
Orientation	4.78 (0.42)	4.17 (1.16)	$t = -3.69$, $P < .001$
Immediate memory	14.29 (0.90)	12.92 (2.06)	$t = -4.57$, $P < .001$
Concentration	3.67 (1.14)	2.60 (1.12)	$t = -5.15$, $P < .001$
Delayed recall	4.13 (0.82)	2.76 (1.28)	$t = -6.80$, $P < .001$
48-Hour SAC scores			
Total score	27.53 (1.71)	27.19 (2.26)	$t = -0.90$, $P = .37$
Orientation	4.98 (0.13)	4.87 (0.34)	$t = -2.25$, $P = .04$
Immediate memory	14.73 (0.56)	14.19 (1.00)	$t = -3.53$, $P = .001$
Concentration	3.67 (1.04)	3.97 (0.92)	$t = 1.64$, $P = .10$
Delayed recall	4.15 (0.97)	4.16 (1.04)	$t = 0.07$, $P = .94$

*Mean (SD).

METHODS

Subjects and Design

A total of 1325 high school and collegiate football players were studied during the 1998 and 1999 football seasons. The sample consisted of 714 athletes from 16 high schools (mean age, 16.26 ± 0.83 years) and 611 players from 8 colleges and universities (mean age, 19.84 ± 1.30 years). All subjects underwent baseline testing on the SAC[6,8] before the start of the football season. This study was approved by the institutional review board at the host institution. All subjects granted written, informed consent. Baseline testing was conducted by certified athletic trainers during preseason fitness and weight training or noncontact football drills and included conditions of exertion as prescribed in the SAC to control for fatigue and other factors often encountered after concussion during a sporting event.

Sixty-three injuries (4.75% of total sample) were documented during the study, including 30 high school (4.20% of high school participants) and 33 collegiate players (5.40% of college participants). Three injured subjects (4.76% of all injuries) were noted to have any observed or reported LOC, with a maximum duration of several seconds. Four injured athletes (6.35%) without LOC exhibited measurable retrograde amnesia (inability to recall events preceding injury) or posttraumatic amnesia (PTA) (inability to recall events after injury), ranging from several seconds to minutes surrounding the injury. No subjects experienced recurrent concussion, and there were no neurosurgical complications, cases of second-impact syndrome,[15,16] or catastrophic outcomes encountered in this study.

Concussion was defined according to the American Academy of Neurology practice parameter[17] (ie, trauma-induced alteration in mental status with or without LOC). Criteria contributing to the identification of an injured player included mechanism of injury (eg, acceleration or rotational forces applied to the head), symptoms reported or signs exhibited (eg, alteration in mental status, confusion, headache, dizziness, memory problems)[17,18] by the player, and reports by teammates and other witnesses regarding the injured player's condition. The occurrence and duration of LOC and PTA were documented immediately after injury by the certified athletic trainer who examined the player.

Injured subjects identified by the team's certified athletic trainer as having sustained a concussion were tested with the SAC on the sideline immediately following injury and again 48 hours after injury. This immediate assessment was conducted within 5 minutes of the injury in all cases. Fifty-five uninjured control subjects, including 24 high school (mean age, 16.42 ± 0.65 years) and 31 college players (mean age, 19.61 ± 1.41 years), were randomly selected from the pool of varsity players and reexamined on the SAC according to the same protocol as the injured subjects. All case-matched uninjured control subjects underwent SAC testing on the sideline after their respective matched subjects sustained a concussion and 48 hours after injury under the same conditions as injured athletes. In most cases, control subjects were platoon or special teams players tested immediately after exiting the field of play to control for fatigue and exertion. Table 1 provides a comparison of demographic variables and injury assessment data for control and injured subjects. No data were missing for the control or injured groups at the baseline, time of injury, or 48-hour assessment points.

Evaluation Measure

The SAC[6,8] is a brief screening instrument designed for the neurocognitive assessment of concussion by a nonneuropsychologist with no prior expertise in psychometric testing. The SAC requires approximately 5 minutes to administer and includes measures of orientation, immediate memory, concentration, and delayed recall, summing to a total composite score of 30 points (Table 2). A standard neurologic screening is included in the SAC to assess deficits in strength, sensation, and coordination that result from concussion. The occurrence and duration of LOC, retrograde amnesia, and PTA also are re-

Table 2. Standardized Assessment of Concussion (SAC)

1) Orientation:

Month: _____	0	1
Date: _____	0	1
Day of week: _____	0	1
Year: _____	0	1
Time (within 1 hr): _____	0	1
Orientation Total Score _____	/	5

2) Immediate Memory: (all 3 trials are completed regardless of score on trial 1 & 2; total score equals sum across all 3 trials)

List	Trial 1		Trial 2		Trial 3	
Word 1	0	1	0	1	0	1
Word 2	0	1	0	1	0	1
Word 3	0	1	0	1	0	1
Word 4	0	1	0	1	0	1
Word 5	0	1	0	1	0	1
Total						

Immediate Memory Total Score _____ / 15

(Note: Subject is not informed of delayed recall testing of memory)

Neurologic Screening:

Loss of Consciousness: (occurrence, duration)

Retrograde & Posttraumatic Amnesia:
(recollection of events pre- and post-injury)

Strength:

Sensation:

Coordination:

3) Concentration:

Digits Backward (If correct, go to next string length. If incorrect, read trial 2. Stop after incorrect on both trials.)

4-9-3	6-2-9	_____	0	1
3-8-1-4	3-2-7-9	_____	0	1
6-2-9-7-1	1-5-2-8-6	_____	0	1
7-1-8-4-6-2	5-3-9-1-4-8	_____	0	1

Months in Reverse Order: (entire sequence correct for 1 point)

Dec-Nov-Oct-Sep-Aug-Jul
Jun-May-Apr-Mar-Feb-Jan _____ 0 1

Concentration Total Score _____ / 5

Exertional Maneuvers
(when appropriate):

5 jumping jacks — 5 push-ups
5 sit-ups — 5 knee bends

4) Delayed Recall:

Word 1		0	1
Word 2		0	1
Word 3		0	1
Word 4		0	1
Word 5		0	1
Delayed Recall Total Score	_____	/	5

Summary of Total Scores:

Orientation	_____	/	5
Immediate Memory	_____	/	15
Concentration	_____	/	5
Delayed Recall	_____	/	5
Overall Total Score	_____	/	30

corded on the SAC. Three equivalent alternate forms of the test were used in this study to minimize practice effects from additional administration.[4,5] The SAC is printed on pocket-sized cards for convenient use by athletic trainers and medical personnel who examine athletes on the sideline.

Data Analysis

Repeated-measures analyses of variance (ANOVAs) were conducted to analyze SAC scores, with group (injured versus control) as a between-subjects factor and assessment point (baseline, concussion, 48 hours) as a within-subjects factor. Level of competition (high school versus college) was included as a covariate in the repeated-measures ANOVAs. Statistical significance of $P < .05$ was set a priori for the ANOVAs. Post hoc comparisons of group differences at each assessment interval were evaluated using t tests. Paired t tests were used to analyze any significant differences in SAC scores within groups at baseline, time of injury, and 48 hours after injury. To minimize error rates associated with multiple comparisons, the .01 level of probability was used for post hoc comparisons.[19] Bivariate (Pearson) correlational analyses were computed to examine the test-retest reliability of SAC scores for injured and control subjects across administrations.

The SAC change scores were derived based on the difference between SAC total score at baseline and immediately after injury. A frequency distribution of observed change scores for injured and control subjects was generated to examine corresponding levels of sensitivity and specificity in accurately identifying injured and uninjured subjects. Documentation of concussion by the certified athletic trainer and inclusion of an injured subject in this research protocol, independent of performance on the SAC, was used as the standard on which to base sensitivity and specificity calculations. Sensitivity refers to the probability that a subject with a certain diagnosis (eg, concussion) will be correctly identified by a specific test result (eg, SAC change score). Specificity refers to the probability that an individual without the condition will be correctly classified as not having the condition. The sum of sensitivity and specificity calculations often provides the greatest overall classification value, but the relative importance of sensitivity and specificity varies, depending on the clinical issue under study. With respect to the diagnosis and management of concussion, one may argue that accurately detecting

the greatest proportion of injured subjects (ie, sensitivity) is more critical than precisely identifying those subjects without an injury. Data were analyzed with SPSS 10.0 statistical software (SPSS Inc, Chicago, IL).[20]

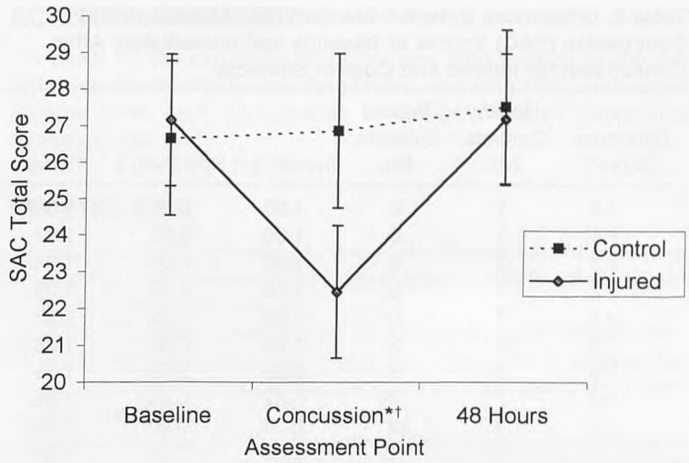
Preliminary Analysis

The preseason baseline SAC total score mean for subjects who were eventually injured during the study was higher for college players than high school subjects ($t_{1,61} = -2.51$, $P = .02$), but there were no significant differences in SAC total scores for the 2 groups immediately after concussion ($t_{1,61} = -0.403$, $P = .69$) or 48 hours after injury ($t_{1,61} = -1.55$, $P = .13$). There was also no significant difference in the average change from preseason baseline SAC total score by high school and collegiate subjects immediately after injury ($t_{1,61} = -0.86$, $P = .40$). Additionally, the interaction (level × assessment point) ($F_{2,230} = 0.47$, $P = .63$) effect did not reach statistical significance for level of competition (high school versus college) as a covariate in the repeated-measures ANOVA that looked at the effects of group (injured versus controls) on SAC performance across time. Therefore, injury and control data for high school and collegiate subjects were combined for formal analysis of the study's main findings. There was no significant difference in SAC total score for injured and control subjects on preseason baseline testing ($t_{1,116} = 1.39$, $P = .16$). There were also no significant differences in SAC total score by those injured subjects administered different forms of the SAC (A, B, C) immediately after concussion ($F_{2,60} = 0.45$, $P = .77$).

RESULTS

Repeated-measures ANOVA revealed a significant interaction effect (group × assessment point) for SAC total score ($F_{2,232} = 64.38$, $P < .001$). Main effects for group ($F_{1,116} = 16.71$, $P < .001$) and assessment point ($F_{2,232} = 76.98$, $P < .001$) on SAC total score were also significant. The pattern of mean SAC total scores for injured and control subjects at baseline, time of injury, and 48 hours after injury is illustrated in the Figure. Significant interaction effects were also revealed for the Orientation ($F_{2,232} = 8.00$, $P < .001$), Immediate Memory ($F_{2,232} = 15.02$, $P < .001$), Concentration ($F_{2,232} = 26.52$, $P < .001$), and Delayed Recall ($F_{2,232} = 24.99$, $P < .001$) subtests of the SAC. Main effects for group were demonstrated on Orientation ($F_{1,116} = 18.85$, $P < .001$), Immediate Memory ($F_{1,116} = 16.79$, $P < .001$), and Delayed Recall ($F_{1,116} = 7.41$, $P = .007$) subtests but not for the Concentration section ($F_{1,116} = 1.10$, $P = .30$). Main effects for assessment point were significant for the Orientation ($F_{2,232} = 23.95$, $P < .001$), Immediate Memory ($F_{2,232} = 28.27$, $P < .001$), Concentration ($F_{2,232} = 20.68$, $P < .001$), and Delayed Recall ($F_{2,232} = 18.41$, $P < .001$) subtests.

Paired t tests indicated that injured subjects immediately after concussion performed significantly below their preseason baseline SAC total score ($t_{1,62} = 11.03$, $P < .001$), but uninjured control subjects did not ($t_{1,54} = -1.03$, $P = .31$). Injured subjects also scored significantly below preinjury baseline immediately after concussion on the Orientation ($t_{1,62} = 4.25$, $P < .001$), Immediate Memory ($t_{1,62} = 6.43$, $P < .001$), Concentration ($t_{1,62} = 6.12$, $P < .001$), and Delayed Recall ($t_{1,62} = 7.74$, $P < .001$) sections of the SAC. Control subjects performed slightly higher than their baseline performance on the SAC Concentration subtest ($t_{1,54} = -2.10$, $P = .04$) on the sideline, but there were no other significant differences between baseline and time of injury scores for controls on the other SAC subtests.



Standardized Assessment of Concussion (SAC) total scores for injured subjects (n = 63) and uninjured controls (n = 55) at baseline, immediately after concussion, and 48 hours after injury. *Injured group was significantly different from control group ($P < .001$). †Injured group was significantly different from baseline score ($P < .001$). Error bars indicate 1 SD above and below group mean.

The SAC total score for injured subjects was significantly lower than uninjured controls immediately after injury ($t_{1,116} = -8.01$, $P < .001$). Scores for injured subjects immediately after concussion were also significantly lower than controls on the Orientation ($t_{1,116} = -3.69$, $P < .001$), Immediate Memory ($t_{1,116} = -4.57$, $P < .001$), Concentration ($t_{1,116} = -5.15$, $P < .001$), and Delayed Recall ($t_{1,116} = -6.80$, $P < .001$) sections of the SAC.

There was no significant difference between SAC total score for the injured group at baseline and 48 hours after injury ($t_{1,62} = -0.23$, $P = .82$). The only significant deficit relative to preseason baseline for injured subjects at the 48-hour assessment point was on the Immediate Memory section ($t_{1,62} = 2.98$, $P = .004$). A practice effect for injured subjects from baseline to 48 hours was detected on the Concentration section ($t_{1,62} = -2.87$, $P = .006$), and there were no significant differences on the Orientation ($t_{1,62} = -0.50$, $P = .62$) or Delayed Recall subtests ($t_{1,62} = 0.00$, $P > .99$).

Uninjured controls demonstrated a slight improvement from baseline to 48 hours that reached statistical significance for SAC total score ($t_{1,54} = -3.32$, $P = .002$) and the Immediate Memory ($t_{1,54} = -2.76$, $P = .008$) section. There were no significant differences in scores for uninjured controls at baseline and 48 hours on the Orientation ($t_{1,54} = -1.00$, $P = .32$) Concentration ($t_{1,54} = -1.90$, $P = .06$), and Delayed Recall ($t_{1,54} = -1.31$, $P = .20$) subtests. There was no significant difference in SAC total score for injured and uninjured subjects at the 48-hour assessment point ($t_{1,116} = -0.90$, $P = .37$). Injured subjects did, however, score significantly lower than uninjured control subjects on the Immediate Memory section of the SAC 48 hours after injury ($t_{1,116} = -3.53$, $P = .001$). There were no significant group differences on the other SAC subtests at the 48-hour assessment point.

As would be expected, the level of test-retest (baseline to time of injury) correlation was higher for the uninjured control

Table 3. Differences Between Standardized Assessment of Concussion (SAC) Scores at Baseline and Immediately After Concussion for Injured and Control Subjects

Difference Score*	Healthy Controls, No.	Injured Subjects, No.	Sensitivity†	Specificity‡	Sum§
+5	1	0	1.00	0.00	1.00
+4	1	0	1.00	0.02	1.02
+3	3	0	1.00	0.04	1.04
+2	7	0	1.00	0.09	1.09
+1	7	0	1.00	0.22	1.22
0	23	3	1.00	0.35	1.35
−1	3	5	0.95	0.76	1.71
−2	7	6	0.87	0.82	1.69
−3	3	12	0.78	0.95	1.73
−4	0	11	0.59	1.00	1.59
−5	0	7	0.41	1.00	1.41
−6	0	5	0.30	1.00	1.30
−7	0	6	0.22	1.00	1.22
−8	0	2	0.13	1.00	1.13
≤−9	0	6	0.10	1.00	1.10

*Baseline SAC score minus the concussion SAC score.
†Proportion of true positives identified as positive on the test.
‡Proportion of true negatives identified as negative on the test.
§Sensitivity plus specificity.

group ($r = 0.66$, $P < .001$) than the injured group ($r = 0.38$, $P = .002$). The mean difference in SAC total score from baseline to time of injury was −4.68 (3.37) for the injured group and +0.24 (1.71) for the control group. Test-retest reliability comparing SAC total score at baseline and 48 hours after injury was significant for both the injured ($r = 0.69$, $P < .001$) and control groups ($r = 0.48$, $P < .001$). Ninety-five percent of injured subjects demonstrated a drop in SAC total score by at least 1 point immediately after concussion, compared with 24% of uninjured controls on test-retest. The distribution of observed change in SAC scores, with sensitivity and specificity values, for the injured and control groups is shown in Table 3. A case-by-case qualitative review of SAC scores immediately after injury indicated that subjects who had sustained LOC or PTA were more severely impaired than those who did not, but sample sizes were not sufficient to support formal statistical analysis.

DISCUSSION

The approach to mental status testing on the sport sideline after a suspected concussion has historically been qualitative and subjective, sometimes limited to the clinician's impression that "something's off" with respect to the injured athlete's presentation. It is now widely recognized that stereotypic questions such as "how many fingers am I holding up?" and "where are you?" are of little diagnostic value to the sports medicine clinician in determining whether an athlete has sustained a concussion and, if so, grading the severity of the injury. Most clinicians are now aware of the importance of systematically assessing the injured athlete's mental status and neurocognitive functioning to detect deficits in orientation, memory, and concentration after a concussion but simply lack an objective and systematic method for doing so.

The results of this study are consistent with earlier reports[4,5,14] that support the value of standardized mental status assessment measures to aid the clinician in the diagnosis and management of concussion in athletes on the sport sideline. Standardized cognitive testing in this study was sensitive to subtle deficits in orientation, memory, and concentration in injured subjects who were otherwise not displaying signs of frank disorientation, amnesia in the classic sense, or gross neurologic dysfunction. Injured athletes who sustained a concussion as defined by the American Academy of Neurology guidelines[17] exhibited, on average, a decrease of more than 4 points on the SAC immediately after concussion, whereas uninjured controls retested on the sideline showed an average increase of less than 1 point above their baseline. A drop of 1 point or more from preseason baseline score on the SAC was 95% sensitive and 76% specific in correctly classifying injured and uninjured subjects. These findings also suggest that the decline in SAC score by injured subjects immediately after concussion represents the direct effect of injury on cognitive functioning and is not due to other extraneous factors often encountered on the sport sideline (eg, fatigue, crowd noise, distractibility). Clinicians should, however, consider the potential impact of other postconcussive symptoms (eg, headache, nausea) on a player's performance on mental status testing.

The clinician's diagnosis is perhaps most difficult to make when subtle signs and symptoms raise the suspicion of concussion, but classic indicators of injury (eg, LOC, amnesia, focal neurologic abnormalities) are not manifested. Results from this study indicate that significant neurocognitive changes can be detected on standardized testing after concussion without LOC, PTA, or focal neurologic abnormalities. Approximately 90% of all injured subjects in this study experienced no LOC, PTA, or changes in gross neurologic status but exhibited measurable deficits in orientation, memory, or concentration function on standardized mental status testing immediately after injury. Although the infrequent occurrence of LOC and PTA in the current study did not allow formal statistical analysis of the effect of these phenomena on SAC scores, qualitative review suggests that LOC and PTA were accompanied by more severe cognitive impairment immediately after concussion. Further study is required to clarify the relative importance of LOC, PTA, and other factors in predicting recovery after sport-related concussion.

After determining that an athlete has indeed sustained a concussion, the clinician's next challenge is tracking recovery and determining whether an athlete is fit to return to action in the same contest. If not, the question remains as to *when* it is safe for a player to return to play after a period of recovery. The current findings suggest that standardized mental status testing may be helpful in establishing a quantifiable index against which to track resolution of acute, postconcussive, cognitive sequelae, and return to baseline cognitive functioning. Follow-up testing for residual deficits in new learning and memory appears to be especially critical, based on the current findings. These data can be used by the clinician in combination with information from other screening instruments, physical examination, more extensive neuropsychological testing, and the player's self-report on postconcussive symptoms to determine the level of recovery and the readiness to return to competition. Preseason baseline testing of all athletes is the recommended model for use of concussion screening instruments in sports, because comparison with an athlete's preinjury performance on a given measure provides the most accurate indicator of postinjury recovery.

A multidimensional approach to sideline assessment of concussion is strongly recommended. Assessment of one symptom domain is not sufficient, since the effects of concussion may

manifest differently across individuals. Clinicians now have available several brief, standardized methods for assessment of mental status,[6–8] postural stability,[9,10] and postconcussive symptoms[11,12] appropriate for use in a sport setting. Neuropsychological testing is also recognized as a valuable tool to objectively measure the effects of sports concussion and track postinjury recovery[11,21–25] but is not very feasible for use on the sideline during sporting events. The combination of brief, standardized measures to quantify the immediate effects and follow-up neuropsychological testing of the injured athlete likely represents the best model for measuring the neurocognitive effects of injury, tracking recovery of function, and assisting the clinician in decision making on the athlete's return to competition.

The clinician should be aware of various limitations related to the use of brief concussion screening tools and have a full command of guidelines for the administration, scoring, and interpretation of a screening instrument before applying it in a clinical situation. These measures provide the clinician with objective information on the presence and severity of concussion but are not intended as a substitute for formal neurologic evaluation, neuropsychological testing, or medical follow-up of the injured subject. Brief assessment tools do not allow an exhaustive evaluation of all cognitive domains (eg, reaction time, information processing speed) sensitive to change after concussion. Screening instruments also do not represent a stand-alone method for declaring full recovery or clearing an athlete to return to play after concussion. All clinical information should be considered to ensure that a player is completely symptom free for a period before being released to return to competition after experiencing a head injury of any severity. More empirical data are required to address the debate on how long an athlete should be withheld from competition after concussion.

Current ongoing studies include SAC testing of injured subjects and matched controls at more points after injury to further clarify the course of early cognitive recovery after concussion. These studies will also correlate SAC findings with results from neuropsychological testing and other concussion assessment methods. A new line of research is under way that correlates objective clinical methods (eg, SAC, neuropsychological testing, postural stability testing, symptom reports) and findings on functional magnetic resonance imaging as a more direct measure of neurophysiological recovery after sport-related concussion. The specific aim of these studies is to elucidate the period of cerebral vulnerability after concussion, which may influence guidelines for the recommended period an athlete should be withheld from competition after injury.

CONCLUSION

The use of standardized measures can improve the accuracy of concussion assessment on the sport sideline. The ultimate goals of these methods are to provide the clinician with a more systematic framework for examining an injured athlete, to allow implementation of proper injury management strategies, and to permit more informed decisions on return to play. Early and accurate diagnosis of concussion is also critical to reducing the potential risks of recurrent injury, cumulative neuropsychological impairment, and catastrophic outcomes associated with second-impact syndrome. Screening instruments are valuable tools to assist the sports medicine clinician in the assessment and management of concussion but should not be used as a replacement for medical evaluation or as the sole determinant of an injured athlete's readiness to return to play after concussion.

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Monitoring Resolution of Postconcussion Symptoms in Athletes: Preliminary Results of a Web-Based Neuropsychological Test Protocol

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Dr Erlanger was an author of the Concussion Resolution Index and has a proprietary interest in HeadMinder, Inc.

Objective: A new Web-based neuropsychological test was field tested to determine usefulness in detecting and monitoring resolution of symptoms after sport-related concussions and in providing objective information for return-to-play decisions.

Design and Setting: We obtained neuropsychological baseline data on all subjects. After concussion, subjects were administered alternate, equivalent follow-up tests until symptoms resolved. Follow-up testing typically occurred at 1- to 2-day intervals after the concussion.

Subjects: Baseline testing was obtained for 834 athletes as part of ongoing field trials. Subsequently, 26 athletes sustained concussions and were studied.

Measurements: We administered The Concussion Resolution Index (CRI) at baseline and alternate forms posttrauma. Follow-up tests included a self-report inventory of neurophysiologic symptoms.

Results: A total of 88% of patients were identified as symptomatic on initial postconcussion testing. The CRI appeared relatively resistant to retest effects, and multiple administrations tracked resolution of symptoms over short and extended time periods.

Conclusions: Although the CRI is still in field trials, preliminary data indicate that the CRI may be a useful method for athletic trainers and other professionals to expeditiously track resolution of symptoms after sport-related concussion.

Key Words: brain injury, return-to-play guidelines, computerized testing, Internet testing

Team physicians and athletic trainers increasingly are recognizing the importance of concussion management in athletes. Although consensus has not been reached regarding the specifics of return-to-play guidelines after a concussion, experts uniformly agree that athletes should not return to play until all symptoms have resolved. Concussive injuries require immediate evaluation in all athletes, typically by means of a short neurologic screening and mental status test.[1,2] After the initial assessment and characterization of an injury, subsequent follow-up tests may show persistence of symptoms for 3 to 10 days in uncomplicated cases of single concussion.[3]

Neuropsychological evaluation using comparison of preinjury baseline and postconcussion performances is considered the most sensitive objective method of detecting the presence and resolution of cognitive postconcussion symptoms.[4] Typically, mild changes in cognitive functions such as attention and concentration, memory, information processing, and motor speed are identified.[5] In practice, however, implementing traditional neuropsychological protocols is time consuming, inefficient, and expensive. Athletes must be tested individually in a face-to face format; testing requires 30 minutes to 2 hours per athlete; administration, scoring, and statistical analysis must be carried out by trained clinicians; and, in the event of a concussion, follow-up tests must be scheduled, administered, scored, and statistically analyzed. All of these factors impede the athletic trainer's ability to use the results in a timely fashion.

Another well-known and longstanding problem in neuropsychology is related to statistical analysis of serial assessments. How test-retest reliability, practice effects, and their interaction should be most accurately identified and accounted for in the interpretation of serial test results is not entirely

clear. Test-retest reliability is rarely provided for many tests used in sport concussion assessment, and reported reliability estimates are derived from relatively long between-test time intervals. Echemendia et al[6] reported test-retest reliabilities for the following commonly used tests: Controlled Oral Word Association Tests, r_{12} = .77; Symbol Digit Modalities Test, r_{12} = .70; Trail-Making Test Part A, r_{12} =.43; Trail-Making Test Part B, r_{12} = .54; VIGIL 1, r_{12} = .49; Digit Span, r_{12} = .52. While no "gold standards" in the field characterize short interval test-retest reliability, investigators recently examined test-retest changes and described reliability estimates from .70 to the low .90s as "generally good."[7] These estimates are consistent with standards usually applied in the field.

In addition, knowledge of the management of practice effects and their relationship to test-retest reliability in group analyses in particular is limited. Significant practice effects have been documented in frequently used tools for assessment of concussion, including the Paced Auditory Serial Addition Test, the Stroop Color Word Test, and the Trail-Making Test Parts A and B.[8] Furthermore, practice effects seem to vary across tests, across test-retest intervals, and across subjects.[7,9] Although alternate test forms can mitigate these problems to a certain extent, such forms are not available for many frequently administered tests, and little has been published on the use of alternate forms for follow-up assessment of postconcussion cognitive symptoms.

These challenges have complicated the implementation of the baseline-posttrauma assessment model of sport concussion. In order to address and more precisely manage limitations due to test-retest reliability and assess the practice effects, a number of researchers have employed the Reliable Change Index (RCI).[6,10] This statistical technique allows for direct comparison of an athlete's postconcussion performance with his or her baseline performance while incorporating test reliability and practice effects into statistical computations and results. The RCI was designed specifically to assess intraindividual differences across time. In group analyses, on the other hand, means and their standard deviations are typically employed for comparison, and no built-in mechanism directly accounts for low reliability coefficients or practice effects. The development of computerized measures has been proposed in order to tackle many of the practical, psychometric, and statistical challenges in sport concussion assessment.[5,10,11] Recently, several computerized measures have been used in sports, including the Automated Neuro-Psychological Assessment Metrics (ANAM, Office of Military Performance Assessment Technology, Walter Reed Army Institute of Research, Washington, DC),[11] Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT, Henry Ford Research Center, Detroit, MI),[5] and Vigil Continuous Performance Test (VIGIL, The Psychological Corporation, San Antonio, TX).[6] However, limited information is available regarding their suitability for baseline and posttrauma analyses.

The Concussion Resolution Index (CRI) (HeadMinder, Inc, New York, NY)[12] is a Web-based computerized neuropsychological assessment battery designed for athletic trainers and other professionals who manage and monitor resolution of symptoms due to sport-related concussion. The CRI was developed to address many concerns regarding the use of current assessment techniques, including issues of test-retest effects, practice effects, the need for alternate forms, ease of administration, time efficiency, and cost.

METHODS

Participants and Design

Participants were athletes recruited from 14 teams and institutions. Institutional review board approval was obtained for 12 institutions, including all high schools. Two institutions for adult athletes did not have an institutional review board, nor did they request such review. Informed consent was obtained from all participants; parental consent was obtained for all high school students. Baseline CRI evaluations were performed on 834 athletes. Assessments were conducted in institutional computer laboratories, with athletes in groups ranging in size from 4 to 20. Trained administrators supervised all assessments. Subsequently, athletes who sustained a concussion, as identified by their treating professional, received follow-up tests until all symptoms resolved. These follow-up tests were administered according to clinical judgment, typically at 1- to 2-day intervals. The test findings were not used as a return-to-play criterion.

Evaluation Measure

The CRI is a Web-based computerized neuropsychological assessment instrument that was developed specifically to compare an athlete's postconcussion performance with his or her own pretrauma baseline performance. The CRI can be administered from any computer with an Internet connection. The administrator views test results and reports from any Internet-connected computer using a secure password. CRI baseline assessments take less than 25 minutes, and postconcussion assessments take approximately 20 minutes. Statistical analyses of injured athletes' test performances are adjusted for test-retest reliability and for practice effects, and reports are generated automatically after the posttrauma test is completed. Alternate forms are available so that multiple follow-up assessments can be administered to track resolution of cognitive symptoms. Self-reported neurophysiologic symptoms are also incorporated into the results along with pertinent medical background and concussion history. This information is included to assist the treating professional in gathering a comprehensive assessment of postconcussion symptom pattern and resolution. All records, including injury and medical history, are secure, confidential, and available only to authorized professionals with a password.

The CRI includes measures of cognitive functions associated with postconcussion syndrome, such as memory, reaction time, speed of decision making, and speed of information processing.[10,13,14] Six subtests are administered at baseline and again at each posttrauma evaluation. These 6 subtests constitute 3 speeded test indices and 2 error scores (Figure 1). Animal Decoding, during which athletes are instructed to type in numbers keyed to pictures of animals, and Symbol Scanning, during which athletes are instructed to rapidly determine whether identified sets of symbols are present among a set of distractors, comprise the Processing Speed index. Reaction Time, during which athletes press the spacebar when a target shape appears on the screen, and Cued Reaction Time, during which athletes press the spacebar when a target shape appears immediately after a "cue" shape, comprise the Simple Reaction Time index. An error index is also calculated based on total false-positives and false-negatives in these 2 tests. Visual Recognition 1 and Visual Recognition 2 present series of pic-

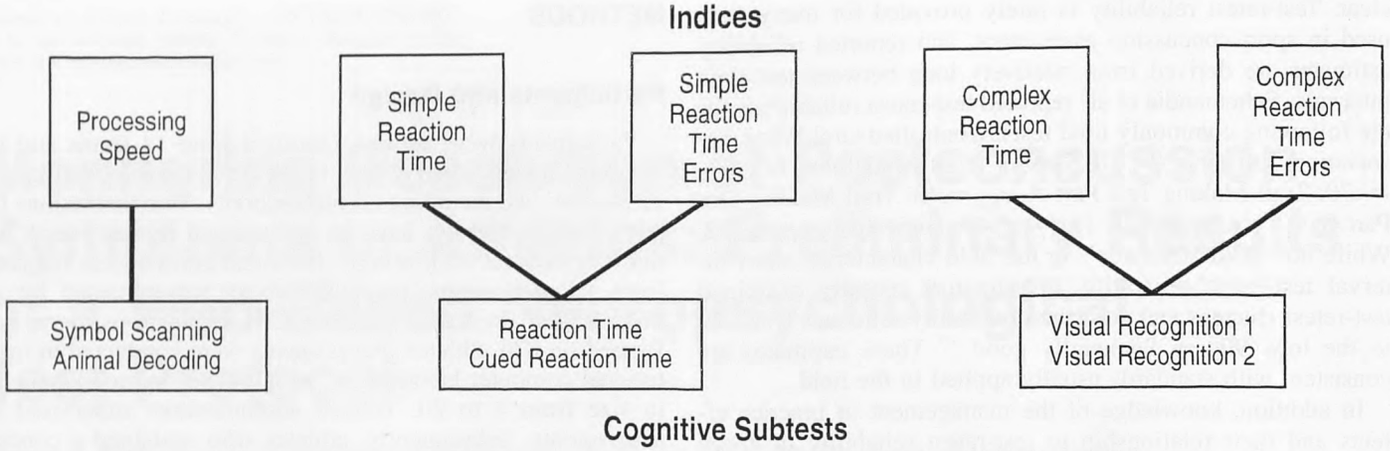

Figure 1. Derivation of indices.

tures, some of which are repeated. Athletes are instructed to press the spacebar as quickly as possible whenever they recognize a picture from an earlier exposure. The response latency is measured. An error index is calculated based on total false-positive and false-negative responses on these 2 tests.

Because the CRI is a new instrument, we describe its principal psychometric characteristics. Norms were derived from performance data collected from 414 athletes in high school, college, and club settings during the initial test development phase.[15] Of these, 216 were male (52%) and 198 female (48%). Roughly equal numbers of participants younger and older than 18 years were recruited to provide normative data suitable for use with both high school and adult-aged populations. Most (58%) were white, 12% were African American, 6% were Asian American, and 4% were Hispanic. Six percent identified their ethnicity as "other," and 14% chose not to identify an ethnic group.

Concurrent validity analysis reveals that CRI indices and subtests are correlated with traditional neuropsychological measures. Correlation of the CRI Processing Speed Index with other measures of processing speed was 0.66, 0.60, 0.57, and 0.58 for the Symbol Digit Modality Test, the Grooved Pegboard Test (dominant and nondominant hands), and the WAIS-III Symbol Search subtest, respectively. The CRI Simple Reaction Time Index correlated with Grooved Pegboard performance at 0.46 and 0.60 for the dominant and nondominant hands, respectively, and with the Trail-Making Test Part A at 0.56. Correlations of the CRI Complex Reaction Time Index with Grooved Pegboard performance were 0.59 and 0.70 for the dominant and nondominant hands, respectively. These correlations are mostly in the moderate range[16] and indicate that the CRI indices measure similar constructs in normative subjects. However, it is not clear at this time, given the differences in the interface between computerized and noncomputerized instruments, whether they actually measure identical constructs. Moreover, although 2 measures may be correlated in healthy subjects, we may not be able to assume that this is the case in a sample of concussed individuals. For instance, the Digit Symbol and Symbol Digit Modalities Tests have a correlation of 0.73 in normal subjects. However, reports of the clinical sensitivity of the Symbol Digit Modalities Test to sport-related concussion outnumber those for the Digit Symbol Test.[4] One purpose of our study was to explore the clinical validity of the CRI indices.

For athletes in college or adult club teams, 2-week test-retest reliabilities for the CRI Indices were 0.90 for the Processing Speed Index, 0.73 for the Simple Reaction Time Index, and 0.72 for the Complex Reaction Time Index. For high school students, 2-week test-retest reliabilities were 0.79 for the Processing Speed Index, 0.72 for the Simple Reaction Time Index, and 0.65 for the Complex Reaction Time Index. These test-retest reliabilities compare reasonably well with those noted above for tests typically administered in the assessment of sport-related concussion.

Statistical Analysis

For statistical analyses, performances on the posttrauma tests are compared with baseline scores for each of the 3 indices and the 2 error scores. For the indices, which comprise normally distributed scores, the RCI[17] is used. The RCI yields a standard score (z score) as follows: $x_2 - x_1 / s_{diff}$, where $s_{diff} = \sqrt{2(SE^2)}$, and SE is the standard error of measurement. The RCI is used to determine if changes in scores from baseline to posttrauma retest are significant. As stated above, this is a useful means of comparing preinjury and postinjury scores derived from traditional face-to-face neuropsychological measures.[10] Analysis of normative data revealed a uniform significant practice effect on the Processing Speed Index. Therefore, in accordance with the Temkin et al[7,9] model, injured players' trauma scores were adjusted by −0.17 seconds on the Processing Speed Index. For the Simple Reaction Time and Complex Reaction Time Indices, mean practice effects of 0.004 and −0.001 seconds, respectively, were identified. Adjustments for these effects were not included due to their size. In keeping with previous use of the reliable change index in a sport setting,[9] the CRI classifies *P* values of less than .05 (1-tailed test) as indicative of significantly worsened (ie, slowed) performance when compared with baseline scores. *P* values ranging from .05 to .15 are described as suggestive of borderline worsened performance due to concussion. Analyses of error scores, which are not normally distributed, are based on the frequency of additional errors on the postinjury test compared with the baseline levels. Cutoffs used for classification of performance are based on additional error values (rather than absolute number of errors) found in fewer than 15% and 5% of the normative sample, representing borderline and significant increases in errors, respectively.

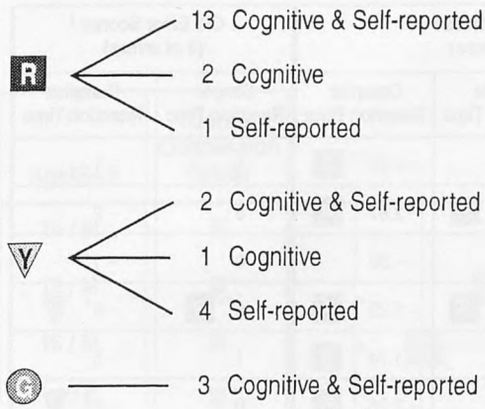

Figure 2. First follow-up after trauma.

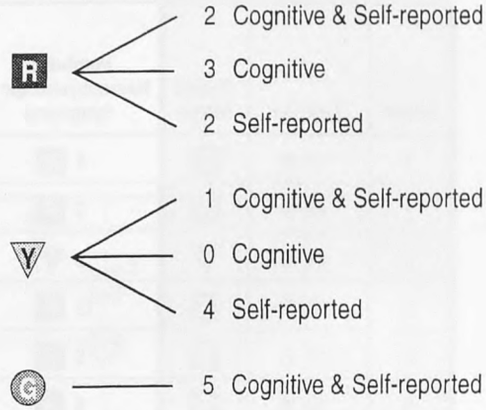

Figure 3. Second follow-up after trauma.

Classification of Test Results

The overall performance of athletes on each posttrauma CRI administration is depicted visually by a traffic light and classified as red, yellow, or green. Red lights reflect statistically significant cognitive test results or 3 or more neurophysiologic symptoms, or both, and indicate that symptoms do not appear to have resolved. Yellow lights indicate borderline cognitive test results or 1 or 2 neurophysiologic symptoms, or both, signaling a need to examine results more closely in view of other factors. Green lights indicate nonsignificant cognitive test findings and no self-reported ongoing neurophysiologic symptoms. If the athlete reports any neurophysiologic symptoms, even if all postconcussion cognitive test scores are within normal limits, performance is classified as either yellow or red, depending on the number of symptoms. All results are identified as statistical test findings or self-reported test findings, or both; no interpretation accompanies these reports. Clinicians are urged to consider a range of factors not assessed by the CRI in any return-to-play decision.

RESULTS

Twenty-six athletes sustained concussions. Fourteen (54%) of these athletes underwent periodic follow-up CRI evaluations until all cognitive and all self-reported neurophysiologic symptoms resolved. Four athletes (15%) underwent follow-up CRI evaluations until cognitive symptoms resolved, but they remained classified as yellow or red due to ongoing self-reported symptoms only. Four other athletes (15%) underwent at least 2 postconcussion CRI exams and were identified with cognitive symptoms at the final posttrauma test. Four athletes (15%) underwent only one postconcussion evaluation with significant cognitive findings and no further CRI follow-up.

Twenty-three of the 26 concussed athletes (88%) were classified as either red (n=16) or yellow (n=7) on the first follow-up test (Figure 2). Three of these athletes (12%) had positive findings (2 red, 1 yellow) due to decreased cognitive performance alone, in comparison with their own baseline performances. These athletes would not have been identified as symptomatic based solely on self-report. At the second postconcussion test (Figure 3), 12 athletes still showed evidence of ongoing cognitive or neurophysiologic symptoms, or both, with 7 classified as red and 5 as yellow. Twenty-five percent of these athletes were identified as symptomatic based on their performance on cognitive measurements alone.

Each athlete's performance on the indices contributed to the initial overall CRI classification status of red (impaired), yellow (borderline), or green (asymptomatic) (Table). Self-reported neurophysiologic symptoms were present in 20 of the 26 (77%) patients. In 18 (69%) of these subjects, at least one cognitive test index indicated borderline or significant decreases in cognitive test performance. Eleven of the athletes (42%) had more than one significant cognitive test finding. Fifteen athletes (58%) manifested both neurophysiologic and cognitive symptoms. Three (12%) had cognitive symptoms without neurophysiologic complaints, and 5 (19%) reported neurophysiologic symptoms but had no significant cognitive findings.

The most sensitive CRI cognitive index, Complex Reaction Time, was significant in 13 (50%) of the athletes. The mean effect size for this index was a z score of −1.44 and ranged from −5.76 to 2.13. The next most sensitive was the Simple Reaction Time Index, which showed results in the borderline to significant range in 11 (42%) of the subjects. The mean effect size for this index was a z score of −1.39 and ranged from −7.04 to 2.04. Finally, the Processing Speed Index showed significant results in 4 (15%) of the cases. The mean effect size for this index was a z score of 0.06 and ranged from −2.78 to 2.90. These large ranges are consistent with the variability seen in postconcussion studies, especially in light of evidence that symptoms may wax and wane over time.[14,19]

One athlete (15), who tested within normal limits on follow up, obtained cognitive test scores below baseline on all 3 indices. Although this finding could be due to regression to the mean or other factors, it underscores the importance of interpretation and clinical supervision of any neuropsychological test results.

Figure 4 depicts the initial severity and course of symptom resolution for each athlete according to the time of test administration. The Cantu grading scale[18] is used here because the severity and duration of symptoms are of primary interest. Accordingly, 12 concussions were grade 1 (46%), 6 (23%) were grade 2, and 8 (31%) were grade 3. All patients with concussions who were followed until asymptomatic had resolved their symptoms by day 15, with the grade 3 injuries generally requiring more time for symptom resolution.

The CRI appeared to be relatively resistant to retest effects, even when tests were administered at close intervals. Indeed, 3 athletes (14, 19, and 22) consistently showed borderline or impaired results across 3 or more tests within a 30-hour period. More generally, resolution of symptoms appeared to progress linearly for some of the athletes. However, others evidenced a

Athlete	Age/Sex	Overall Status	Number of Neurophysiologic Symptoms	CRI Indices [1] (z-scores)			CRI Error Scores [1] (# of errors)	
				Processing Speed	Simple Reaction Time	Complex Reaction Time	Simple Reaction Time	Complex Reaction Time
1	17 M	R	6 R	−.10	−.90	−4.27** R	1	−1
2	19 M	R	5 R	−2.78** R	−2.76** R	−2.87** R	6	−2
3	21 M	Y	2 Y	−1.18* Y	−.12	−.30	0	−1
4	16 F	R	12 R	−1.01	−7.04** R	−5.22** R	−6** R	−9* Y
5	15 F	R	5 R	.75	−.82	−1.74** R	1	−5
6	16 M	R	6 R	2.54	.77	−2.26** R	0	−6* Y
7	16 F	R	9 R	.04	−3.47** R	−2.00** R	1	−2
8	19 F	R	3 R	.20	.02	2.13	−1	0
9	19 F	R	0	.42	−.62	−1.90** R	0	−1
10	19 M	R	1 Y	−1.62* Y	2.04	−2.50** R	0	−6* Y
11	19 F	R	2 Y	−.21	−1.92** R	−2.21** R	2	−9* Y
12	18 M	R	4 R	2.90	−2.75** R	N/A	0	N/A
13	18 F	R	2 Y	−.21	−1.91** R	−2.21** R	2	−9* Y
14	19 M	Y	2 Y	−.81	−.77	−.76	0	−3
15	21 M	G	0	−.95	−.67	−.78	0	2
16	20 M	R	0	−.84	−4.26** R	−3.60** R	−4** R	−12* Y
17	21 M	Y	1 Y	.45	−.93	.60	1	−3
18	19 M	Y	2 Y	−.37	.08	.03	1	4
19	20 M	R	1 Y	−.25	−4.25** R	−5.76** R	2	−1
20	17 F	R	1 Y	2.00	−1.02	−2.78** R	−2* Y	3
21	18 M	Y	1 Y	−1.59* Y	−1.21* Y	.26	1	0
22	18 M	Y	2 Y	1.32	−.15	1.32	0	1
23	18 M	R	4 R	1.22	−1.25* Y	.69	0	11
24	21 M	Y	0	.45	−1.45* Y	−.91	0	−3
25	19 F	G	0	.23	−.15	.60	0	−1
26	21 M	G	0	.94	−.88	.45	−1	−1

Initial postconcussion symptom breakdown.

***$P < .15$.**

***P < .05.**

[1]Negative numbers indicate worse performance.

waxing and waning course. For example, 2 athletes' status changed from nonsignificant to significant (6 and 10). When athlete 10 was first tested 2 days postinjury, he was symptomatic. Four days postinjury, all symptoms had resolved; however, he later developed neurophysiologic symptoms (headaches) and was reclassified as red. Ten days postinjury, he was tested again and classified as green. Similarly, when tested 2 days postinjury, athlete 6 had both neurophysiologic and cognitive symptoms. Four days postinjury, all symptoms had resolved; however, he later developed neurophysiologic symptoms (fatigue, difficulty sleeping, and memory problems) and was retested, displaying borderline performance on one of the cognitive indices.

DISCUSSION

Preliminary results indicate that the CRI is sensitive to the sequelae of concussion in athletes. Postconcussion symptoms were detected in 23 of 26 subjects (88%) studied on the initial testing. Of the 26 concussed athletes in our study, 5 athletes

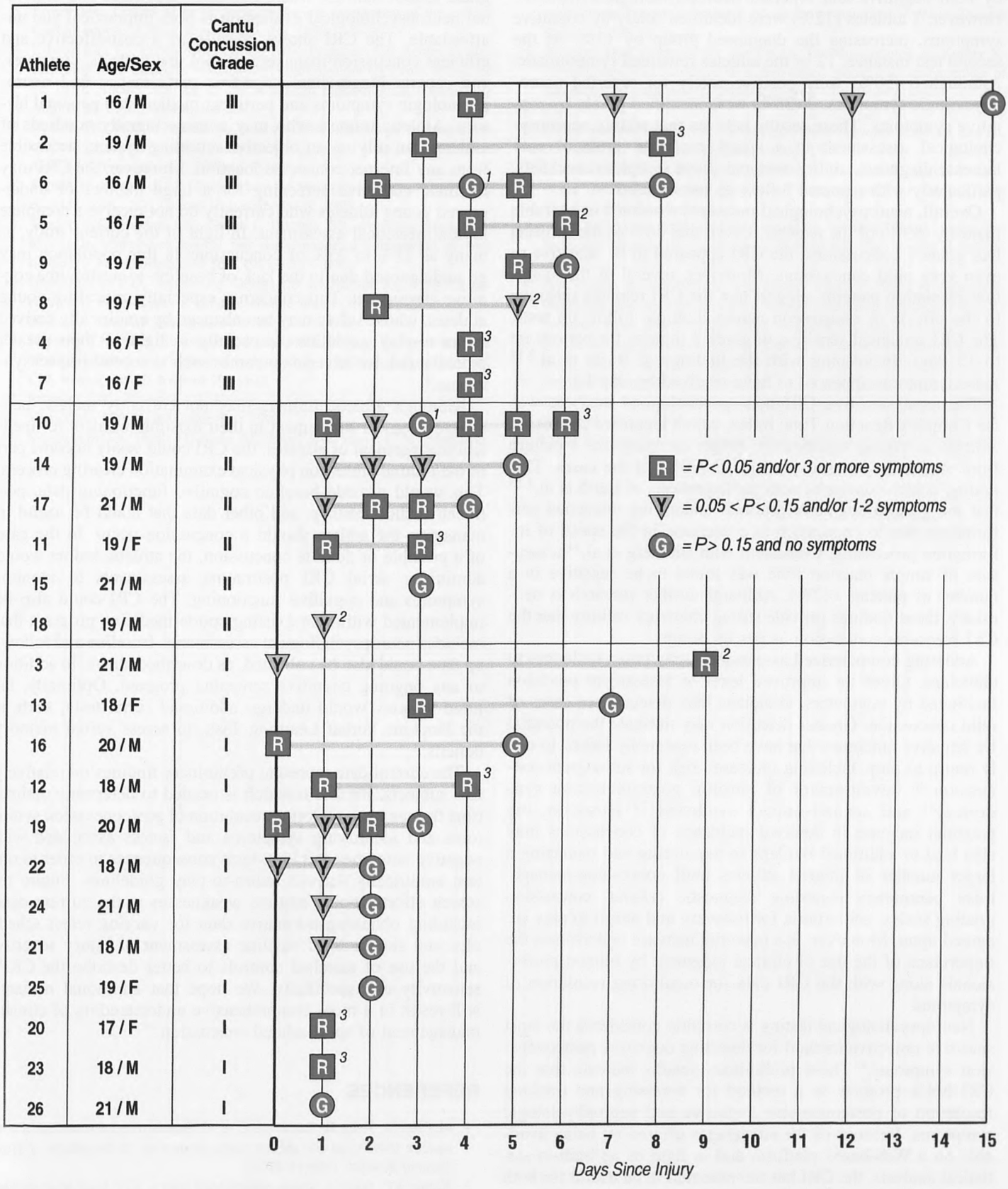

Figure 4. Course of Symptom Resolution

[1]Developed neurophysiologic symptoms and was retested.

[2]Due to neurophysiologic symptoms only. All cognitive test deficits resolved/athlete not retested.

[3]Follow-up not obtained.

(19%) would have been identified by reported neurophysiologic symptoms alone, and 15 athletes (58%) were identified by both cognitive and reported neurophysiologic symptoms. However, 3 athletes (12%) were identified solely by cognitive symptoms, increasing the diagnosed group by 13%. At the second test instance, 12 of the athletes remained symptomatic. Although 6 (50%) were positive solely for reported neurophysiologic symptoms, 3 (25%) were identified purely by cognitive symptoms. These results indicate that adding neuropsychological assessment to a sports medicine protocol may increase diagnostic ability over and above symptom checklists, particularly with repeated follow-up assessments.

Overall, neuropsychological measures worsened in a sizable majority (69%) of the patients. Given that 46% of the subjects had grade 1 concussions, the CRI appeared to be sensitive to even very mild concussions. Moreover, several of the symptom resolution patterns suggest that the CRI remains sensitive to the effects of concussion across multiple follow-up tests. The CRI remained sensitive to grade 3 injuries for periods up to 15 days. In keeping with the findings of Barth et al,[3,13] most symptoms appeared to have resolved by day 10.

The most sensitive CRI neuropsychological measure was the Complex Reaction Time Index, which identified concussed athletes as taking significantly longer to recognize a picture from an earlier exposure trial in 13 (50%) of the cases. This finding is also consistent with the hypothesis of Barth et al,[3,13] that the general cognitive problem underlying worsened performance due to concussion is a decrease in the speed of information processing. Consistent with Bleiberg et al,[14] a measure of simple reaction time was found to be sensitive in a number of patients (42%). Although further research is necessary, these findings provide strong construct validity that the CRI measures concussion as per its design.

Adopting computerized assessments may pose challenges to clinicians. Given an improved level of assessment precision facilitated by computers, clinicians may detect more cases of mild concussion. Greater detection may mitigate the potential for negative outcomes that have been repeatedly linked to early return to play, including increased risk for subsequent concussion,[20] development of chronic postconcussion syndrome,[21] and second-impact syndrome.[22] However, the potential increase in detected incidence of concussions may also lead to additional burdens in monitoring and managing a larger number of injured athletes until concussion-management parameters regarding diagnostic criteria, concussion grading scales, and criteria for recovery and return to play are agreed upon. Moreover, this potential increase underscores the importance of the use of clinical judgment by trained professionals along with the CRI data for monitoring resolution of symptoms.

Neuropsychological testing is currently considered the most sensitive objective method for detecting cognitive postconcussion symptoms.[4] These preliminary results indicate that the CRI holds promise as a method for assessing and tracking resolution of postconcussive cognitive and neurophysiologic symptoms. Because of the advantages offered by being available on a Web-based platform and in light of its built-in statistical analysis, the CRI has the potential to be useful for both research and time-efficient management of sport-related concussion.

Many professional and university sports teams have already integrated neuropsychological assessment into their assessment protocols as their resources support professional neuropsychological consultation.[5] Unfortunately, the greatest number of at-risk athletes remain in the college, high school, and grade school settings, where resources are scarce and individual neuropsychological evaluation is both impractical and unaffordable. The CRI shows promise as a cost-effective and efficient concussion-management tool that collects, compares, and reports longitudinal cognitive performance and neurophysiologic symptoms and pertinent medical and personal history. Athletic trainers who may oversee literally hundreds of athletes can rely on an objective reporting system, accessible from any Internet-connected location. Moreover, the CRI may facilitate cognitive screening for a large number of underserved young athletes who currently do not receive a complete multidimensional assessment. In light of the current study, as many as 11% to 25% of concussions in this population may go undiagnosed due to the lack of baseline-to-posttrauma cognitive assessment. This concern is especially critical for young athletes, whose safety may be enhanced by empirically derived return-to-play guidelines, especially in light of their greater potential risk for adverse outcomes such as second-impact syndrome.[4]

Although athletic trainers may not currently include neuropsychological assessment in their existing protocol for medical management of athletes, the CRI could easily become part of the routine preseason physical examination clearing process. This would provide baseline cognitive functioning data, pertinent medical history, and other data that could be useful in managing the athlete should a concussion occur. In the case of a possible or definite concussion, the athletic trainer would administer serial CRI posttrauma assessments to monitor symptoms and cognitive functioning. The CRI could also be implemented within an existing sports medicine program that includes neuropsychological assessments. Baseline and follow-up data could also be collected, as described above, in addition to any ongoing cognitive screening program. Optimally, injured athletes would undergo additional serial tests, such as the Hopkins Verbal Learning Task, to assess verbal memory deficits.

The current data represent preliminary findings on relatively few subjects. Further research is needed to determine optimal time frames for monitoring resolution of postconcussion symptoms and identifying symptoms and factors associated with negative outcomes and long-term consequences in order to obtain empirically derived return-to-play guidelines. Future research efforts should address weaknesses in the current data, including obtaining normative data for varying retest schedules and standardized sideline assessment of injury severity, and the use of matched controls to better describe the CRI's sensitivity and specificity. We hope that additional research will result in a more comprehensive understanding of clinical management of sport-related concussion.

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Implementation of Neuropsychological Testing Models for the High School, Collegiate, and Professional Sport Settings

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Christopher Randolph, PhD, provided conception and design; analysis and interpretation of the data; and drafting, critical revision, and final approval of the article.

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Objective: To review models for the use of neuropsychological testing in the management of sport-related concussion at various levels of competition.

Background: As we come to understand the natural history of sport-related concussive brain injury, it is increasingly evident that significant neurologic risks are associated with this type of injury. These risks include (1) acute intracranial pathology, (2) catastrophic brain swelling from second-impact syndrome, and (3) the potential risk for markedly prolonged recovery or permanent cognitive dysfunction associated with multiple concussions.

Description: Neuropsychological testing has proved to be a useful tool in the medical management of sport-related concussion. In this paper, I describe a systematic model for the implementation of neuropsychological assessment of athletes at various levels of competition.

Clinical Advantages: The systematic model was designed to incorporate state-of-the-art techniques for the detection and tracking of neurocognitive deficits associated with concussion into recently formulated guidelines for the medical management of sport-related concussion. Current applications of the model are discussed, as well as ongoing studies designed to elaborate the empirical underpinnings of the model and refine clinical decision making in this area.

Key Words: concussion, sports, brain injury

Mild traumatic brain injury (concussion) suffered by athletes engaged in organized sports has become the focus of increased attention by medical personnel engaged in the care of athletes, sport administrative bodies, the news media, and the players themselves. The growing number of medical reports and lay press articles devoted to the topic of sport-related concussion is a testament to the significance and complexity of this problem.

Concussion is typically the result of trauma to the head in contact sports, but it can occur in noncontact sports as well, usually as a result of falls. Concussion can also occur without a direct blow to the head if sufficient rotational forces are applied to the brain (eg, a whiplash injury).[1] Kelly et al[2] defined concussion as a "trauma-induced alteration in mental status that may or may not involve loss of consciousness." This trauma-induced alteration in mental status can range in severity from a brief feeling of being dazed after the injury to an immediate loss of consciousness.

Traumatic brain injury has been recognized as a serious hazard for athletes since at least the turn of the century. President Theodore Roosevelt's concern over the 19 athletes who were killed or paralyzed by football injuries in 1904 led to the formation of the National Collegiate Athletic Association as a governing body to establish rules for safer competition.[3] Although rules have been changed to improve player safety and protective equipment continues to evolve, concussive brain injury remains common in football. Approximately 63 000 incidents per year are estimated to occur in high school football alone in the United States.[4]

Football is not the only organized sport that carries a significant risk of concussion. Ice hockey has been reported to have even higher rates of concussion, and soccer has only slightly less risk.[5] Even sports such as field hockey, wrestling, and lacrosse carry a substantial risk of concussive brain injury.[4] Obviously, there is an inherent risk of physical injury (including concussion) associated with any sport, and changes in rules and improvements in equipment can only reduce these risks to a point. This is where the medical management of concussion becomes essential. A sophisticated medical management system for sport-related concussion is important for 3 main reasons:

1. Diagnosis and appropriate management of acute concussion. The appropriate management of the athlete at the time of the injury includes evaluating the severity of the concussion and identifying any potential neurosurgical emergencies (eg, epidural, subdural, or intracerebral hemorrhages) that would require immediate intervention. This initial evaluation and subsequent monitoring are of primary importance in cases of more severe injury, and the critical importance of appropriate medical intervention in such cases is obvious.
2. Prevention of second-impact syndrome. This potentially fatal syndrome is thought to result from the effects of a second concussion that occurs while the player is still

symptomatic from an earlier concussion.[2,6–9] In second-impact syndrome, cerebrovascular autoregulation is apparently disrupted, resulting in cerebrovascular congestion and malignant brain swelling with markedly elevated intracranial pressure. Reports of this syndrome have been limited thus far to adolescents and young adults, with a number of documented fatalities. Although controversy exists regarding the actual mechanism associated with this syndrome,[10] the potential consequences are obviously catastrophic and argue for the development of sensitive techniques to ensure complete recovery from concussion before exposing a player to the risk of another injury.[11]

3. Monitoring of athletes to prevent prolonged recovery or permanent disability due to multiple concussions. The natural history of mild traumatic brain injury remains poorly understood. To date, virtually no prospective neuropsychological studies of an unselected (ie, non–self-referred) series of consecutive patients with concussion exist. As a result, no empirically determined parameters for the expected rate and degree of recovery from concussions of varying levels of severity have been established. It has been hypothesized for a number of years, however, that a previous history of concussion may result in the slowing of recovery or less complete recovery (or both) that is observed in individuals after a first concussion.[12] It has been my experience that young adults typically recover rapidly and completely after a single concussion with brief or no associated loss of consciousness. This observation has also been made by others.[13] The rate and ultimate degree of recovery in individuals who have suffered multiple concussions is less clear but may be slowed or reduced, particularly when the concussions are closely spaced.[14]

POTENTIAL MECHANISMS AND OUTCOMES OF PERMANENT DISABILITY FROM MULTIPLE CONCUSSIVE BRAIN INJURIES

Some evidence, primarily derived from animal studies, suggests that the rotational forces on the brain that appear to be responsible for producing concussion can result in scattered axonal injuries resulting from shearing forces.[15] In milder cases, these pathologic changes are detectable only at the microscopic level. The rapid and apparently complete neuropsychological recovery that we typically observe in humans who have experienced injuries of comparable severity (in terms of momentum of impact) to these animal models is probably explicable by the concept of functional *reserve*; that is, we can compensate for mild, traumatically induced neuronal loss as a result of inherent redundancies in brain structures and systems. Although a certain degree of functional (ie, synaptic) reorganization may also take place after such injuries, this requires a longer period of time and, therefore, probably does not contribute to the rapid clinical recovery we typically observe.

Adhering to this theoretical model, each subsequent insult to the brain, however trivial, results in further depletion of this reserve capacity, eventually limiting the rate and perhaps the degree to which functional recovery can occur. This depletion could have 2 potential effects. The first is a direct, permanent loss of some neurocognitive functions as a result of repeated trauma. The second is a potentially increased sensitivity to the effects of normal aging or other disease states on the brain, for example, the premature expression of such age-related degenerative conditions as Parkinson disease or Alzheimer disease.

A prior history of head injury has been reported to be a risk factor for the diagnosis of Alzheimer disease[16,17] and Parkinson disease.[18] This may be due to prior neuronal loss from traumatic brain injury lowering the threshold for the clinical expression of these disorders. This model is based on the observation that the neuropathologic changes associated with each of these disorders are also observed in the brains of normal older individuals and that neither disease becomes clinically manifest until a certain degree of neuronal loss is reached. It is also well established that certain domains of cognitive functioning (eg, memory) decline with normal aging; it is conceivable that prior neuronal loss due to repeated head injury could accelerate this normal decline or make it clinically apparent at an earlier age.

Not only do all of these issues demand empirical investigation, they also underscore the need for a program to monitor the neurocognitive status of the athlete with a history of concussion, to minimize or avoid the possibility of permanent disability.

THE DEVELOPMENT OF A MODEL FOR THE MANAGEMENT OF CONCUSSION IN PROFESSIONAL SPORTS

Only recently have medical guidelines for the management of sport-related concussion been formulated and published. In 1986, Cantu[19] published "Guidelines for return to contact sports after a cerebral concussion." In 1991, the Colorado Medical Society[20] published *Guidelines for the Management of Concussion in Sports.* Recently, the American Academy of Neurology[21] adopted a revised version of these guidelines. Although these guidelines differ slightly in specifics, they each represent an attempt to develop a standardized approach to the assessment and clinical management of sport-related concussion. They are largely determined by clinical judgment rather than empirical evidence and are subject to revision as we learn more about the natural history of concussion.[22]

The goal of this paper is to describe the model that my colleagues and I have developed for the management of sport-related concussion, with a particular focus on the role of neuropsychological testing. The general model was designed primarily for implementation in contact sports, in which the incidence of concussion is sufficiently high for each player to be at a substantial level of risk. Neuropsychological testing is used within this model to provide us with a sensitive index of higher-level brain functioning, by measuring functions such as memory, attention, and speed and flexibility of cognitive processing. These specific functions have been demonstrated to be particularly sensitive to impairment as a result of mild traumatic brain injury. In contrast, a variety of other cognitive domains are much less sensitive to concussion (eg, language, simple motor functions, and visuospatial abilities), and we generally do not include these in brief test batteries designed for this purpose.

An important point to be made prior to further discussion of this model is that neuropsychological test data are only one source of information regarding the effects of a concussion. Obtaining such data does not preclude the need to carefully screen for postconcussive symptoms (eg, headache, lightheadedness, nausea), directly evaluate neurologic status, and (in some cases) obtain neuroimaging studies. Abnormalities in

Table 1. Baseline Neurocognitive Battery*

Test	Description
Hopkins Verbal Learning Test	A memory test in which players are read a list of 12 words for immediate recall. Four learning trials are given.
Trail-Making Test Part B	An attentional task requiring rapid visual processing and working memory.
Letter-Number Sequencing Task	A working memory task, with increasing levels of difficulty. Players are given a random string of letters and numbers and have to sort them out mentally and recite them in order.
Stroop Color Word Test	An attentional test that requires speeded processing as well as response inhibition.
Controlled Oral Word Association Test	A verbal fluency test that requires the subject to rapidly retrieve words starting with a particular letter.
WAIS-3 Digit Symbol Test†	A coding test requiring rapid sensorimotor and processing speed and memory.
Hopkins Verbal Learning Test, Delayed Free Recall	Delayed free recall of the 12-word list learned earlier.
Hopkins Verbal Learning Test, Delayed Recognition	Delayed recognition testing of the 12-word list learned earlier.

*We use 3 alternate forms of this battery to minimize practice effects. Total time required for the battery is 20 to 25 minutes.
†WAIS indicates Wechsler Adult Intelligence Scale.

any one of these domains can be taken as evidence of residual effects from a concussion. Performing normally on neuropsychological examination does not necessarily rule out the possibility that a player may be otherwise symptomatic or have neurologic abnormalities or exhibit direct evidence of brain trauma on magnetic resonance imaging.

Neuropsychological testing is, however, clearly one of the most sensitive techniques for detecting abnormal brain functioning after concussion, and as such, it plays an important role in the medical management of sport-related concussion. There are three basic types of neuropsychological test batteries used in the management of sport-related concussion. These include the sideline examination, the baseline neurocognitive examination, and the comprehensive neuropsychological examination.

LEVELS OF NEUROPSYCHOLOGICAL ASSESSMENT

The Sideline Examination

There is a role for brief neurocognitive testing in the sideline evaluation of athletes shortly postconcussion. All of the guidelines for the management of sport-related concussion rely on the assessment of cognitive status immediately postconcussion in distinguishing between grade 1 and grade 2 concussions, and this assessment determines whether an athlete returns to play in the ongoing game. The most widely used and best validated instrument for this purpose is the Standardized Assessment of Concussion (SAC).[23,24] The SAC consists of a 30-point scale that measures orientation, attention, and anterograde memory. It was designed for use by athletic trainers, coaches, and sideline medical personnel and takes approximately 5 minutes to administer. The SAC is a very brief scale constructed for the sole purpose of detecting and quantifying the acute neurocognitive effects of concussion. The sideline examination consists of a brief neurologic screening with the SAC and exertional maneuvers designed to raise intracranial pressure in a controlled fashion.

The Baseline Neurocognitive Examination

The baseline examination consists of a somewhat more detailed and difficult set of neurocognitive tests, typically lasting 20 to 30 minutes. Tests are targeted at those neurocognitive functions most sensitive to impairment from concussion (memory, attention, and speed and flexibility of cognitive processing). This type of battery was originally developed to be administered to professional football and hockey players before the start of preseason training. This procedure, as the name implies, is done to obtain baseline neurocognitive data on all players, so that potentially subtle changes in neurocognitive status postconcussion can be detected. The need for individual baseline examinations arises from the recognition that substantial interindividual differences exist in preinjury cognitive functions such as memory and attention and that subtle deficits in a particular individual might be overlooked because of these differences. Baseline neurocognitive testing is also being conducted with greater frequency at the collegiate level, although the constituent subtests vary from site to site. A number of ongoing projects are designed to transfer baseline testing to a fully computerized test platform, but these are currently in a development phase and still lack sufficient clinical validity to support their implementation on a wide scale.

Our baseline battery of tests (Table 1) was modeled on a battery that was originally composed by Lovell and Collins[25] for this purpose in their work with the Pittsburgh Steelers. We have refined this battery to take advantage of newer tests, as well as to eliminate constituent subtests that were found to lack sensitivity to concussion. We constructed 3 alternate forms of this battery (A, B, and C) to minimize practice effects on repeated testing. Our battery requires approximately 20 minutes to complete.

The Comprehensive Neuropsychological Examination

A standard clinical neuropsychological examination consists of comprehensive testing of multiple neurocognitive domains (eg, motor, attention, memory, language, visuospatial functions, executive and problem-solving functions) and assessment of psychological and emotional functioning. This typically requires 3 to 6 hours of testing for an adult. When the referring concern is traumatic brain injury, this type of examination is usually reserved for patients in whom there is some question of permanent impairment. In most cases of sport-related concussion, we expect complete recovery, and

therefore, this type of examination is unnecessary. There are cases, however, in which an athlete has suffered multiple concussions or has had the baseline examination repeated to the point that the results are difficult to interpret. In those athletes, a comprehensive neuropsychological examination can be very informative with respect to quantifying impairment and informing decision making.

MANAGEMENT MODELS FOR HIGH SCHOOL, COLLEGIATE, AND PROFESSIONAL COMPETITION

The resources available to athletic trainers at various levels of competition are obviously markedly different, as are the consequences for withholding a player from return to play. At the National Football League or National Hockey League level, a decision to keep a key player from returning to play for one or more games could potentially result in tremendous financial losses for the team. Conversely, sending a player back too quickly could result in a second concussion, which might require a much more protracted recovery and potential loss of the player for the entire season. At the high school level, the consequences of this decision-making process are not as momentous, and it may be somewhat easier to adopt a conservative management approach. In terms of resources, professional teams also have the resources to retain experienced neurologists and neuropsychologists to manage athletes with concussive brain injuries in consultation with team physicians and athletic trainers. Collegiate teams are typically less well equipped for such purposes (although there are some exceptions), and high schools cannot be expected to adopt such a model. As a result, it is appropriate to design different models for high school, collegiate, and professional sport settings.

In the models detailed (Tables 2 through 4), the American Academy of Neurology guidelines[21] for the management of concussion in sports are used to grade concussion level and determine return to play. Although no data suggest that one set of guidelines is inherently superior to another for this purpose, the American Academy of Neurology guidelines have the most objective criteria for differentiating between a grade 1 and grade 2 concussion (provided that a formal sideline evaluation, such as the SAC, is conducted). Differentiating a grade 1 from a grade 2 concussion is important because this provides an objective and standardized measure of the appropriateness of allowing a player to return to an ongoing game. With the use of the SAC, this differentiation can be made objectively. The models below are suggested as guidelines for implementing the various levels of neuropsychological testing (sideline, baseline, and comprehensive evaluations).

The Model for High School–Level Concussion Management

At the high school level, obtaining baseline neuropsychological testing is probably unrealistic from a practical standpoint, at least until well-validated computerized batteries are available. The need to obtain baseline scores on the SAC is also debatable. While this practice may slightly improve the sensitivity and specificity of the SAC,[26] a reasonable alternative would be to set a fairly conservative cut-off level for impairment. For example, in our total sample of 91 concussed and 1189 normal high school and collegiate athletes, an SAC score of 25 or less as a cut-off for impairment correctly identified 80% of the injured players and misidentified fewer than 30% of the normal players. This is a reasonably conservative approach that would seem to be appropriate for the high school level (Table 2). A concussion symptom checklist[25] can also be useful to monitor postconcussive symptoms in a standardized way over time.

The Model for Collegiate-Level Concussion Management

Many collegiate athletic programs use a brief neurocognitive battery such as the one detailed in Table 1 to obtain baseline measures of participants in sports with a high risk for concussion. The consequences of removing key players from competition are more significant at the collegiate level than at the high school level, which also argues for a somewhat more sophisticated approach to concussion management. Most larger schools have graduate-level psychology training programs with faculty and students who can serve as a resource for facilitating baseline testing. The suggested collegiate-level model is detailed in Table 3.

The Model for Professional-Level Concussion Management

Currently, all National Hockey League and most National Football League players undergo baseline neurocognitive testing as part of concussion management programs. All of these teams can easily obtain quality neurologic and neuropsychological consultation to help athletic trainers and team physicians with decision making regarding return-to-play status for players who have experienced concussions. Our program with the Chicago Bears was the first (to our knowledge) to involve baseline testing of an entire professional sports team, and this program has been ongoing for several years. The suggested professional-level model is detailed in Table 4.

Overall, the 3 models differ only with respect to the extent to which baseline testing is implemented in the preseason period and during the follow-up management. Obviously, these are guidelines and not practice standards. Individual athletic trainers, team physicians, and consulting neurologists and neuropsychologists may elect to deviate from these guidelines based on the circumstances of a particular case and their clinical judgment. Athletic trainers are likely to be the individuals who are responsible for coordinating the preseason, sideline, and postconcussion management protocols, and they should use these models as a starting point to determine the most appropriate protocol for their teams.

CURRENT LIMITATIONS TO THE USE OF NEUROPSYCHOLOGICAL TESTING

Although neurocognitive testing clearly has a role in the management of sport-related concussion, some factors limit the utility of this approach in detecting subtle impairments of brain function after concussion. These factors primarily involve the baseline examination, rather than the sideline examination or full-scale neuropsychological assessment. Sideline examinations have limitations due to time constraints and availability of personnel for administration of the examination; however, the SAC has proven utility in identifying neurocognitive impairments in players who are otherwise asymptomatic. Full-scale neuropsychological assessments are detailed clinical tools that should be used only for situations in which a player exhibits long-standing symptoms.

Table 2. Algorithm for Implementation of High School–Level Model*

Preseason Testing	Concussion Grade/Description	Sideline Management	Follow-Up Management
Players with no concussion history: consider obtaining SAC preseason baselines; if not obtained, set SAC cutoff score at conservative level.	Grade 1: Transient confusion, no loss of consciousness, all symptoms and cognitive abnormalities as measured by the SAC resolve within 15 min.	Clinical evaluation, including SAC; if player is asymptomatic and performs in normal range within 15 min, he or she is cleared to return to play. Multiple grade 1 concussions, however, require removal from game.	Player is monitored for postconcussive symptoms. Multiple grade 1 concussions suggest an increased risk of future concussion. In these cases, when player is several weeks postconcussion, consider obtain baseline neuropsychological testing for future concussion management.
Players with a history of multiple grade 1 or any grade 2 or 3 concussions should undergo baseline neuropsychological testing.	Grade 2: Transient confusion, no loss of consciousness, symptoms and/or cognitive abnormalities as measured by the SAC fail to resolve within 15 min.	Clinical evaluation, including SAC; by definition for this stage of concussion, player is symptomatic and/or performance has remained below normal for >15 min. Remove from game and monitor frequently for signs of evolving intracranial pathology.	Formal neurologic evaluation the next day if postconcussive symptoms persist. If baseline testing exists, repeat the testing once the player becomes neurologically asymptomatic. Player should be symptom free (including lack of identified neuropsychological impairments) for 1 week before return to play after a single grade 2 concussion, 2 weeks after multiple grade 2 concussions. Once player is several weeks postconcussion, obtain baseline neurocognitive testing for future concussion management.
	Grade 3: Any loss of consciousness. (a) brief (seconds) (b) prolonged (minutes)	Transport from field to hospital by ambulance if still unconscious or worrisome signs detected. If player regains consciousness before transport, he or she should be removed from game and undergo a thorough neurologic examination, including the SAC, and frequent subsequent monitoring for signs of evolving intracranial pathology.	Formal neurologic evaluation the next day. If baseline testing exists, repeat the testing once the player becomes neurologically asymptomatic. Player should be symptom free (including lack of identified neurocognitive impairments) for 1 week before return to play if loss of consciousness was brief, 2 weeks if prolonged, and 4 or more weeks for multiple grade 3 concussions. Once player is several weeks postconcussion, obtain baseline neuropsychological testing for future concussion management.

*SAC indicates Standardized Assessment of Concussion.[23,24]

Table 3. Algorithm for Implementation of Collegiate-Level Model*

Preseason Testing	Concussion Grade/Description	Sideline Management	Follow-Up Management
Players with no concussion history: obtain baseline neurocognitive testing if resources available. Consider obtaining SAC preseason baselines.	Grade 1: Transient confusion, no loss of consciousness, all symptoms and cognitive abnormalities as measured by the SAC resolve within 15 min.	Clinical evaluation, including SAC; if player is asymptomatic and performs in normal range within 15 min, he or she is cleared to return to play. Multiple grade 1 concussions, however, require removal from game.	Player is monitored for postconcussive symptoms. Multiple grade 1 concussions suggest an increased risk of future concussion. In these cases, when player is several weeks postconcussion, obtain baseline neuropsychological testing for future concussion management.
Players with a history of multiple grade 1 or any grade 2 or 3 concussions should definitely undergo baseline neuropsychological testing.	Grade 2: Transient confusion, no loss of consciousness, symptoms and/or cognitive abnormalities as measured by the SAC fail to resolve within 15 min.	Clinical evaluation, including SAC; by definition for this stage of concussion, player is symptomatic and/or performance has remained below normal for >15 min. Remove from game and monitor frequently for signs of evolving intracranial pathology.	Formal neurologic evaluation the next day if symptoms persist. If baseline testing exists, repeat once the player becomes neurologically asymptomatic. Player should be symptom free (including lack of neurocognitive impairments) for 1 week before return to play after single grade 2 concussion, 2 weeks after multiple grade 2 concussions. If player did not undergo baseline testing, once player is several weeks postconcussion, obtain baseline neurocognitive testing for future concussion management.
	Grade 3: Any loss of consciousness. (a) brief (seconds) (b) prolonged (minutes)	Transport from field to hospital by ambulance if still unconscious or worrisome signs detected. If player regains consciousness before transport, he or she should be removed from game and undergo a thorough neurologic examination, including the SAC, and frequent subsequent monitoring for signs of evolving intracranial pathology.	Formal neurologic evaluation the next day. If baseline testing exists, repeat once the player becomes neurologically asymptomatic. Player should be symptom free (including lack of neurocognitive impairments) for 1 week before return to play if loss of consciousness was brief, 2 weeks if prolonged, and 4 or more weeks for multiple grade 3 concussions. If player did not undergo baseline testing, once player is several weeks postconcussion, obtain baseline neurocognitive testing for future concussion management.

*SAC indicates Standardized Assessment of Concussion.[23,24]

Table 4. Algorithm for Implementation of Professional-Level Model*

Preseason Testing	Concussion Grade/Description	Sideline Management	Follow-Up Management
All players undergo baseline neurocognitive testing.	Grade 1: Transient confusion, no loss of consciousness, all symptoms and cognitive abnormalities as measured by the SAC resolve within 15 min.	Clinical evaluation, including SAC; if player is asymptomatic and performs in normal range within 15 min, he or she is cleared to return to play. Multiple grade 1 concussions, however, require removal from game.	Player is monitored for postconcussive symptoms. Multiple grade 1 concussions suggest the need for follow-up neurologic and neurocognitive evaluations.
	Grade 2: Transient confusion, no loss of consciousness, symptoms and/or cognitive abnormalities as measured by the SAC fail to resolve within 15 min.	Clinical evaluation, including SAC; by definition for this stage of concussion, player is symptomatic and/or performance has remained below normal for >15 min. Remove from game and monitor frequently for signs of evolving intracranial pathology.	Formal neurologic evaluation the next day if symptoms persist. Once player is neurologically asymptomatic, repeat baseline neurocognitive testing. Player should be symptom free (including lack of neurocognitive impairments) for 1 week before return to play after a single grade 2 concussion, 2 weeks after multiple grade 2 concussions.
	Grade 3: Any loss of consciousness. (a) brief (seconds) (b) prolonged (minutes)	Transport from field to hospital by ambulance if still unconscious or worrisome signs detected. If player regains consciousness before transport, he or she should be removed from game and undergo a thorough neurologic examination, including the SAC, and frequent subsequent monitoring for signs of evolving intracranial pathology.	Formal neurologic evaluation the next day. Once player is neurologically asymptomatic, repeat baseline neurocognitive testing. Player should be symptom free (including lack of neurocognitive impairments) for 1 week before return to play if loss of consciousness was brief, 2 weeks if prolonged, and 4 or more weeks for multiple grade 3 concussions.

*SAC indicates Standardized Assessment of Concussion.[23,24]

The baseline battery is arguably the most important tool in the decision-making process in managing sport-related concussion. The consulting neuropsychologist is usually called on to make a recommendation based solely on the results of this battery. In a typical scenario, the data from a postconcussive examination are compared with the data from a player's preseason (or earlier) baseline testing and reviewed for evidence of significant decline that would indicate that the player is still experiencing the effects of the concussion. This comparison is complicated by the following facts:

1. A certain degree of measurement error is associated with all psychological tests, such that scores may fluctuate somewhat on retesting.
2. The degree of this error term varies in magnitude depending on the nature of the specific test.
3. Practice effects associated with most cognitive tasks result in some improvement in performance from one test session to the next.
4. The magnitude of the practice effect can vary as a function of the individual test and as a function of the interval between testings.

The neuropsychologists involved in this interpretive process must weigh all of these factors in clinical decision making. Unfortunately, adequate empirical evidence to guide this decision making is currently lacking. Although we are usually aware of short-term (ie, days or weeks) retest reliability and practice effects for individual tests, longer-term data (months or years) are usually lacking, as are data on the effects of administering the battery multiple times over a series of weeks (which is not uncommon). As a result, a neuropsychologist is often forced to rely more heavily on clinical judgment than would be ideal in such settings.[27] One approach to minimize this problem is to routinely postpone neurocognitive testing after a concussion until a player is otherwise completely asymptomatic. The rationale for this is basic: if a player is symptomatic, no further documentation of the fact that he or she has not yet recovered from a concussion is necessary. As a matter of practice, however, players at the professional level are routinely tested while still symptomatic. This almost invariably leads to one more testing session after the symptoms have resolved and obviously complicates the interpretation of the additional test session, which may be requested only a few days after the first postconcussive examination. Reserving the neurocognitive testing until players are otherwise asymptomatic is an important measure that can be taken to minimize uncontrolled variance in the test data. Studies establishing the parameters of test-retest stability and practice effects over time with baseline[25] are another.

FUTURE DIRECTIONS IN THE REFINEMENT OF THESE MODELS

As indicated previously, the empirical underpinnings of the SAC examination are sufficiently firm to support its implementation as a standard tool for the sideline evaluation of concussion in a variety of settings. The management guidelines of our models follow a strategy for which we have moderate clinical certainty. The use of this type of standardized approach with objective neurocognitive measurement will enable the accumulation of evidence for evaluation of needed modifications over time. Our ultimate goal is to obtain sufficient empirical evidence to establish an accepted standard of practice.

One important route to that goal will be the refinement of our interpretation of the baseline neurocognitive test battery. A number of groups are currently researching this issue. Many investigators agree that a fully computerized battery may be the most workable approach. This approach will have the effect of making test administration and scoring fully objective, simplifying interpretation, and facilitating the exchange of baseline data for players as they move through different levels of competition or from one team to another. It will also simplify the practical aspects of obtaining baseline and follow-up testing, as a neuropsychologist will not need to be on site for the testing. Some preliminary data suggest that such an approach is feasible,[28] but the clinical validity studies necessary to support the implementation of a specific battery are still lacking. Investigations of test-retest data for different time intervals, clinical validity with injured players, and criterion validity data for comparison with existing individually administered batteries will be needed to establish the sensitivity and validity of any computerized approach.

CONCLUSIONS

A number of publications over the last several years have focused on the consequences and management of sport-related concussion. In many ways, the development of various scales, guidelines, and management models have far outpaced our scientific progress in this area, and we continue to lack an empirical basis for most of our interventions. It is clear, however, that sport-related concussion has specific, short-term consequences and potential long-term effects and that neuropsychological testing has a role in the evaluation and management of players who suffer such injuries. The management models presented above are predicated on our current understanding of the short-term consequences and recovery from sport-related concussion and incorporate the most well-validated neuropsychological tools for detecting residual effects of concussion. The data that continue to accumulate across centers using these models should advance our understanding of the natural history of mild traumatic brain injury and thereby more clearly elucidate the risks involved and the most appropriate interventions for athletes who sustain concussions at all ages and levels of competition.

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Methodologic Issues in Neuropsychological Testing

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Objective: To familiarize athletic trainers with methodologic issues regarding the development and implementation of neuropsychological tests used in programs for monitoring sport-related cerebral concussion.

Data Sources: Knowledge base and MEDLINE and PsychLit searches from 1980–2000 using the terms *sports, athletes, concussion,* and *brain.*

Data Synthesis: Neuropsychological testing is a proven method for evaluating symptoms of concussion that results from a variety of different causes. These tests have been shown to be effective in evaluating symptoms of subtle cognitive dysfunction in a number of patient groups. Applying these tests in an athletic population has required some procedural modifications, including the use of brief test batteries, collection of preseason baseline data, and evaluation of subtle postconcussive changes in test scores over time. New methods are now being used for improved evaluation of the reliability and validity of neuropsychological tests in athletes. Proper scientific analysis of the psychometric properties of neuropsychological tests and the ultimate value of their use in the sport setting will require years of detailed study on large numbers of athletes with and without symptoms of concussion.

Conclusions/Recommendations: Athletic trainers and related personnel need to be aware of the training and methodologic issues associated with neuropsychological testing. Knowledge of the scientific properties of these tests, their advantages, and current limitations will ultimately enhance the athletic trainer's ability to use information from neuropsychological testing in an effective manner.

Key Words: concussion, head injury, assessment, psychometrics

Symptoms of sport-related cerebral concussion are, by nature, subjective and vaguely defined. People who evaluate head injuries in the athlete need objective means to determine the pattern and severity of symptoms. The field of sports medicine has turned to neuropsychology, the scientific study of brain-behavior relationships, to provide methods that can be used on the sideline and in the athletic training room for assessment of postconcussive changes in orientation, concentration, and memory. In this article, I will review many of the methodologic issues involved in using neuropsychological tests and the advantages they offer for assessment of athletes with symptoms of sport-related cerebral concussion.

WHAT IS CLINICAL NEUROPSYCHOLOGY?

Clinical neuropsychology is a relatively new professional field that has evolved over the years from advancements in both clinical and experimental psychology to a distinct specialty. Individuals who practice clinical neuropsychology are doctoral-level professionals who specialize in the evaluation of diseases that affect the brain. Clinical neuropsychologists complete graduate training in psychology plus additional training at the internship and postdoctoral levels in the assessment and treatment of brain disorders. The practice of neuropsychology requires state licensure as a psychologist. Many practitioners also receive board certification in the field. Most neuropsychologists work in medical and mental health facilities, whereas others work in private practices or rehabilitation settings.

Assessment of concussion is by no means new to the field of neuropsychology. Many years of experience have gone into developing tests for evaluating impairments in attention, memory, and higher-order executive functions that may be exhibited by those with concussion secondary to various causes, including motor vehicle crashes, work injuries, or violent crimes.

Neuropsychologists are very much aware that no single test is effective in diagnosing the presence or absence of concussion. The tests that are used are not effective when used in isolation, and they are not designed for that purpose. Neuropsychological tests are administered most appropriately as groups, otherwise termed *test batteries.*[1] The purpose of using a test battery is to look for consistencies in symptoms as exhibited in variations among a number of different test scores. In a typical clinical setting, assessment of a patient with a concussion often requires a long test battery that may take from 4 to 8 hours to administer. Neuropsychologists are trained to evaluate the patient's motivational and emotional states, in addition to measuring the pattern and severity of cognitive impairment. They also consider to what degree an individual's age, educational background, or a multitude of other factors may influence test performance. Interpretation of these tests

takes years of clinical training supplemented by extensive clinical experience. These are among the many reasons why neuropsychological tests need to be administered and interpreted under the immediate supervision of a doctoral-level psychologist who has obtained the requisite training.

NEUROPSYCHOLOGY IN THE SPORT SETTING

Moving from the clinical laboratory to the sport setting has provided new challenges and opportunities for the field of clinical neuropsychology.[2–4] Sport-related concussion occurs on the practice or playing field, where methods for an immediate assessment of symptoms are needed. Many teams do not have the financial resources to send their athletes for extensive neuropsychological evaluations. Athletes rarely have the time to undergo a 4- to 8-hour examination in the midst of the season. The ages of athletes participating in organized sports range from childhood to mid adulthood, and participants come from a variety of cultural and educational backgrounds. Previous studies[5] have demonstrated that learning disabilities are prevalent and effective in influencing neuropsychological test performance. In contrast to a typical patient with concussion whose self-reports of symptoms brings him or her to medical attention, athletes are often known to minimize the severity of their symptoms to remain on the active roster. The emotional and motivational factors that influence neuropsychological test performance in athletes are markedly different from those seen in other individuals with concussion.

Neuropsychology has made many recent advances to meet the demands of the sport setting. A neuropsychological method for evaluating symptoms immediately following concussion has been developed by McCrea et al.[6,7] The Standardized Assessment of Concussion (SAC) is a brief (5-minute), standardized measure of orientation, concentration, and memory that can be given at the sideline by athletic trainers and other team officials who have received training in its administration and interpretation. The SAC comes in alternate forms, devised to avoid the presence of "learning effects," which are liable to occur when tests are administered on more than one occasion. In its typical use, the SAC is administered to all players at the beginning of the athletic season to obtain a baseline of their performance. Athletes with suspected concussion are administered an alternate form of the SAC to determine if there are any changes in scores that would represent the effects of concussion. This information is used with other clinical criteria to make decisions about return to play and the need for a more extensive neurodiagnostic workup, including a neurologic consultation and more detailed neuropsychological testing.

Models of assessment using more extensive batteries of neuropsychological tests have been developed for use with athletes during the past 10 to 15 years.[2,3] These batteries are being implemented by an increasing number of teams in the high school, college, and professional ranks to assess more persistent symptoms that may result from concussion. The approach consists of a brief (20- to 30-minute) test battery that is used to obtain baseline information on the athlete before the athletic season. Athletes who receive a concussion during the season are sent to a neuropsychological consultant for additional testing within 24 to 48 hours after the injury to determine the presence or absence of changes from the initial test performance. Objective information regarding the player's cognitive functioning is then provided to the team's medical staff to help them make determinations about return to play. Athletes who exhibit clear features of cognitive disturbance attributable to concussion are then followed up with additional testing until their test scores return to baseline levels.

The demands of the sport setting make it necessary for neuropsychologists to adopt test batteries that are shorter and more portable than those used in a typical clinical or hospital setting. The availability of baseline results is crucial to these models of testing. Obtaining baseline information on a given player enables the neuropsychologist to shorten the neuropsychological test battery by avoiding the use of long and arduous tests for evaluating overall intelligence and estimating premorbid level of functioning. The use of serial testing enables the neuropsychologist to control for many pre-existing factors, including intelligence, cultural factors, and a medical history, including effects of previous concussions, that might be influencing the athlete's test scores. With baseline test results available, the neuropsychologist can make informed decisions about the presence or absence of changes in cognitive functioning over time by using the athlete's previous functioning as a starting point.

Although the use of brief test methods administered during several periods provides many advantages, a number of methodologic challenges exist for the neuropsychologist working in the sport setting. Many of these are reviewed herein.

PSYCHOMETRIC PROPERTIES OF NEUROPSYCHOLOGICAL TESTS

A clinical examination is a systematic evaluation designed to elicit pathologic signs or symptoms. In the past, sideline evaluation of an athlete with suspected concussion might have included a number of haphazard questions about the current location, date, or score of the game. Many of the same questions would then be repeated during follow-up examinations. Neuropsychological tests offer the advantage of being standardized in their administration and use. This means that the procedures, materials, and scoring of the tests are fixed so that the same precise methods can be followed by others at different times and places. This type of testing offers a means of observing symptoms in a controlled manner, with the use of a numeric scale that can be compared with previous test scores or with scores obtained from other individuals who have been administered the same procedure. Using these methods, officials from one team can compare their athletes' test results with those obtained from another team or evaluate how an athlete's performance compares with published norms taken from a large sample of athletes.

The development and standardization of a neuropsychological test take many steps. These steps include deciding on the psychological construct or symptom to be evaluated, establishing the format of questions or procedures to be administered, and performing a series of scientific experiments to determine the quality of the information the test provides.

Individuals experiencing a concussion often report feeling "slowed down" or "foggy."[8] They also report difficulties with processing and retaining information. Clinicians have known for years that disturbances in concentration and memory are among the most prominent and most persisting complaints. Neuropsychologists who investigate the underlying features of these cognitive disturbances have developed a number of paper-and-pencil tests of sustained attention, learning, and recall to be used as objective measures of these often vaguely defined symptoms. Years of work have gone into the develop-

ment of these measures. Scientific studies have been performed to determine whether the tests are able to properly assess the process in question and whether they are able to accurately identify impairments in patients with verified brain dysfunction.[9]

Neuropsychologists turn to the field of psychometrics, the study of psychological tests and measurements, to evaluate the scientific quality of their measures. In this context, the terms *reliability* and *validity* are often used. Reliability refers to the consistency or stability of test scores when they are obtained for an individual after repeated observations or under different testing conditions.[10] When discussing reliability, one needs to consider both the "true score," which would be the value obtained if the test were a perfect measure of the ability in question, and the "test error," which represents the effects that other factors may impose on the measurement process. Neuropsychologists are well aware that there is no error-free test. Thus, one can assume that a test score reflects some reality of the measurement in question. However, one must also consider other factors that clearly can contribute to the overall test score. These may include random influences, such as motivational state or factors specific to the testing conditions, such as the presence of extraneous noise in the room. Other factors may be attributed to inherent inaccuracies in the test itself. Much of a test's value lies in its ability to maximize its assessment of a given ability while minimizing the effects of these extraneous error factors.

Validity is a concept that refers to how well an instrument measures what it is supposed to be measuring.[10] This is an extremely important issue in the field of neuropsychology. Neuropsychologists consider many aspects of validity when a decision is made whether or not to use a particular test. One of these concerns whether the content of the test provides an accurate representation of the symptom in question. Another factor to consider is how performance on the test in question compares with other tests that have been established for evaluating the same symptom. Finally, one must know the demonstrated accuracy of the test in making a clinical decision. When using tests to measure symptoms of brain dysfunction, the neuropsychologist needs to know how successfully the tests have been used to distinguish between those with and without independently verified features of brain impairment.

Performing a validation study on a neuropsychological test requires sampling an adequate number of subjects to demonstrate the efficacy of the measure. Samples sizes of 35 or more subjects with independently diagnosed conditions are needed to perform the statistical analyses necessary to study the condition in question. Some suggest that sample sizes exceeding 400 subjects may be required.[11] This presents a challenge when evaluating a condition such as sport-related cerebral concussion, for which the expected rate of occurrence in a sample of high school football players is less than 4%.[12] To properly determine the validity of a test in evaluating symptoms in this population, one would minimally need to study 875 athletes at baseline, follow up the athletes closely throughout the season, and identify all the subjects who develop symptoms of concussion. To establish more precise validity statistics, samples sizes of 10 000 need to be obtained. The time and effort required to perform an acceptable validation study in athletes is certainly not negligible.

Reliability and validity provide the foundation of all forms of scientific measurement, including neuropsychological testing. Accurate interpretation of the neuropsychological test findings is based on the premise that the test is reasonably free of measurement error or random influence and that the test can be used to support a specific clinical inference, such as the presence or absence of cognitive dysfunction secondary to brain injury. In basic terms, the psychometric properties of a given test instrument, combined with the experience of using it, directly affect the neuropsychologist's ability to trust that a given score may represent a "true" performance accurately measuring the symptom in question. An example of the steps required for proper development and implementation of a neuropsychological test is provided herein.

DEVELOPMENT OF THE SYMBOL DIGIT MODALITIES TEST

Developing a neuropsychological test requires an extensive amount of work. This will be illustrated with a discussion of the Symbol Digit Modalities Test (SDMT), a measure that is frequently used for assessment of sport-related concussion. Most of the teams in the National Hockey League and the National Football League are now using the written version of this test. The test is also now included in test batteries used by the increasing number of concussion monitoring programs developed for high school and collegiate athletes.

The SDMT was developed as a measure of sustained attention and concentration by researchers at the University of Michigan in the late 1960s for use in screening for cerebral dysfunction in adults and children.[13] The test requires rapid transcription of meaningless designs into numeric responses. The final score reflects the number of correct transcriptions performed in 90 seconds. Standardized instructions for administration and methods for scoring and interpreting the test are provided in the test manual.[13]

Normative data for the SDMT were obtained from studies performed on a total of 1307 adult volunteers and 3680 children.[13] Evidence for the reliability of the test was initially obtained in a study of 80 adults administered the test in 2 test sessions throughout approximately 30 days. Comparisons of test scores obtained at times 1 and 2 resulted in a test-retest correlation of 0.80. Individuals tested at baseline obtained a score of 56.79 $\pm$ 9.84. Scores increased an average of 3.67 points to 60.46 $\pm$ 11.16 at retesting. The increase in scores at time 2 reflects a "practice effect," a feature that is commonly seen in test-retest situations.

The data, taken together, indicate that approximately 64% of the variance can be accounted for by a feature that is intrinsic to the SDMT. The other 36% of the variance is thus attributable to random error, including these observed practice effects. Although this figure may seem high, it is similar to the degree of error observed in similar tests of these abilities. This finding illustrates the fact that neuropsychological measures do not provide error-free indexes of complex abilities such as cognitive functioning. However, these methods are currently the best measures that science has to offer.

Research has shown that scores obtained from SDMT correlate with other measures of attention and concentration. Investigators comparing scores from the SDMT and the Digit Symbol subtest from various versions of the Wechsler Adult Intelligence Scale have found correlation coefficients ranging from 0.73 to 0.79.[1] In one study, the pooled correlation coefficient with a group of other commonly used test measures, including the Stroop Color Word Test, Paced Auditory Serial Addition Test, and measures of simple and choice reaction

time, was 0.58.[14] Early validity studies were performed on individuals with and without brain dysfunction. Scores obtained from 36 individuals with brain trauma were lower than those observed in the controls.[13] The SDMT has been shown effective in discriminating between subjects with head injuries and controls.[14] Its sensitivity to detecting symptoms of concussion in a sample drawn from an initial total of 2350 athletes has also been demonstrated.[2]

DEVELOPMENTS IN EVALUATION OF TEST RELIABILITY

Assessment of clinical change is essential to the use of neuropsychological testing in the management of concussion in the sport setting. The major premise is that decreases from baseline test scores at retesting signal the presence of cognitive impairment secondary to concussion. Increasing scores in subsequent testing reflect improvement in functioning, with an eventual return to the baseline level. Accurate detection of changes in test scores is a critical component to detection of the often subtle changes in behavior after concussion.

As mentioned herein, understanding the reliability of a given test is important when examining changes in test scores over time. In many applications, psychological tests can provide highly reliable scores, particularly when stable traits, such as verbal skills or personality characteristics, are assessed.[10] Unfortunately, this is not the case when assessing symptoms of concentration disturbance in conditions, including concussion. In studies of healthy individuals, it is well known that scores on tests of attention, processing speed, and memory are among the most unreliable indexes, since they happen to be most prone to the influence of "normal" confounding factors, such as anxiety level and fatigue.[9] The situation becomes even more complicated when evaluating individuals who have sustained a concussion, when the goal is to assess changes attributable to mild brain dysfunction. In these patients, one must consider not only the effects of normal variations in test scores but also those attributed to the added influence of other factors, including pain and possible side effects of medications.[8] Those evaluating changes in neuropsychological functioning that result from concussion are thus faced with the difficult challenge of measuring subtle changes in symptoms having inherently low levels of reliability in the presence of a multitude of other factors that may cause confounding changes in test scores.

Development of newer tests with increasing levels of reliability might help to address some of the issues encountered when evaluating changes in attention and concentration test scores over time. Given the length of time needed to develop and study the use of new tests, this is not likely to provide any immediate solution to the problem. In the meantime, neuropsychologists are evaluating new ways to interpret changes in test scores in those measures currently in use.

Some investigators have studied the use of Reliable Change Indices (RCIs) as a method of evaluating "true" changes in test scores over time.[15,16] An RCI is a statistical method for developing cutoff scores that are useful for evaluating meaningful changes in test scores independent of psychometric issues, such as practice effects and other sources of variance.

Computation of an RCI can be performed on any neuropsychological test measure that has been studied through a test-retest paradigm with the population of interest. The standard error of the difference is derived and standardized according to the normal distribution. In its typical use, the RCI results in a difference score, representing changes that occur in less than 5% of the study sample. Change scores that exceed this level thus represent the effects of "true" changes attributable to the effects of concussion or recovery. Recent applications of the RCI paradigm in the sport setting have been conducted on studies of Australian National Rugby League professionals[17]; these authors have determined that reliable changes can be detected with a 6-point difference on the SDMT. Thus, players retested after injury with decreases of 6 points or more on the SDMT are likely to be showing impairments in processing speed attributable to the effects of concussion. Similar studies are currently under way to derive RCIs for other tests in current use with athlete populations.

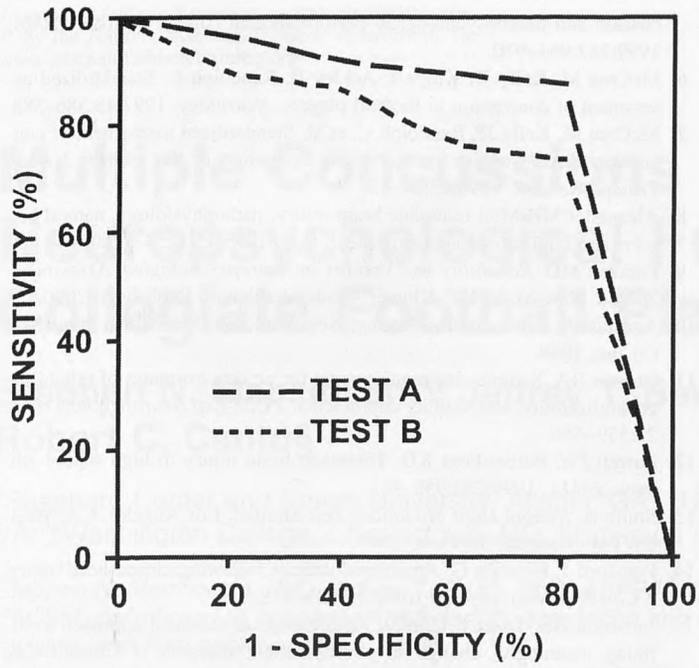
DEVELOPMENTS IN EVALUATION OF TEST VALIDITY

Those contemplating using neuropsychological testing as part of their concussion management program often ask, "How accurate are these tests in evaluating the effects of concussion?" This is a question about the validity of the tests. As mentioned herein, most of the neuropsychological tests currently used in sport settings have been validated in previous studies of concussion. Further validation through more specific studies of athletes with concussion is currently under way.

Assessing the validity of neuropsychological tests is typically limited to the analysis of group means. For example, if patients with traumatic brain injury are found to have significantly lower scores on a given memory test than healthy controls, then that memory test is usually considered to provide a valid measure of memory impairment associated with brain injury. What is not assessed by this type of analysis, however, is what proportion of patients can be correctly classified into brain-injured and non–brain-injured groups by a given score on the test. Analyses of classification rates and the diagnostic accuracy of clinical test data are often overlooked in neuropsychological studies of test validity.

Interest is now turning to studying the ability to predict the outcome of an individual case. To address this issue, an increasing number of investigators are assessing the accuracy of neuropsychological tests through the use of signal detection statistics and computation of receiver operating characteristic (ROC) curves.[18,19] Signal detection statistics provide indexes of the ability to make a clinical decision when faced with a given criterion or test score. The sensitivity of a measure refers to the proportion of individuals with the condition in question whose scores exceed that criterion. Specificity of a measure refers to the number of individuals without the condition classified accurately as not achieving that criterion. The ROC curves provide graphic representations of the tradeoff between true-positive and false-positive rates of classification. Calculation of the area under the ROC curve provides an empirical method for determining the diagnostic accuracy of a given test or helping to determine which of 2 tests is more accurate for diagnosing a certain condition. An example of ROC curves for 2 hypothetical tests is provided in the Figure.

Only a few researchers have addressed the diagnostic accuracy of using neuropsychological tests for assessment of concussion in a sport setting. In a recent study using the SAC, a decline of 1 point or more from baseline was observed in 94% of a sample of 50 high school and collegiate football players assessed within 15 minutes of experiencing a concussion.[20] This level of decline was observed in only 24% of a



Receiver operating characteristic curve for hypothetical tests A and B developed for assessment of concentration disturbance secondary to sport-related concussion. Sensitivity and specificity values for classification of athletes with and without concussion are represented graphically for both tests. The larger area under the curve indicates that test A provides more accurate classification of these 2 athlete groups. This finding can be demonstrated empirically through statistical comparison of area-under-the-curve values for both tests. Such information would be used to establish that test A is a more valid and sensitive measure for assessing the effects of concussion than test B.

sample of uninjured athletes undergoing routine retesting as part of a larger study. These results provide further validation of the use of the SAC as a measure of the immediate effects of concussion in athletes. The study also provides the clinician with information that athletes who exhibit a decline from their baseline score on this measure after injury will have more than a 90% chance of exhibiting the effects of concussion as opposed to a less than 25% chance of a false-positive error. Information of this nature is of obvious benefit to the clinician, who will use this information, with his or her experience and other relevant information, to make decisions about return to play. Similar statistics regarding sensitivity and specificity will be available for other neuropsychological tests once a number of ongoing studies with athletes have been completed.

DISCUSSION

Neuropsychological testing provides a scientific method for evaluating symptoms of cognitive dysfunction that result from sport-related cerebral concussion. The ultimate goal in using these tests is to provide the athletic trainer and team physician with objective information to help them arrive at a decision about return to play or the need for obtaining additional workup. Neuropsychological test results should not be used in isolation but rather in the context of a full postinjury evaluation, including all appropriate and available methods.

Many athletic trainers inquire about the requirements for implementing neuropsychological testing programs for their athletes. One must first consider the fact that the practice of neuropsychological assessment requires years of specific preparation, consisting of graduate school course work and clinical training. Most states require a license in psychology to purchase and use these instruments in a clinical setting. Thus, by law, the clinical application of neuropsychological test batteries in a sport setting should be conducted under the direct supervision of a licensed psychologist who has received training in the use of these instruments. Athletic training staff and trainees should be encouraged to receive instruction in the use of more portable screening instruments, such as the SAC, for immediate assessment of athletes on the sideline. One must remember that these screening measures have been developed for evaluation of the immediate effects of sport-related concussion and provide the team medical staff with information regarding the need for more detailed medical evaluation, including neuropsychological assessment.

The development and validation of neuropsychological tests require years of work and studies of large numbers of subjects. Test batteries currently used for assessing athletes represent an adaptation of methods developed for use in a hospital or clinical setting. The potential to obtain baseline test results on an "at-risk" athletic population represents a relative advantage over the typical clinical situation, in which information regarding the subject's uninjured state is rarely available. In athletes, time constraints require the use of brief neuropsychological test batteries that can be completed in less than ½ hour. Emphasis on the analysis of changes from baseline to postinjury scores requires detailed knowledge about the performance of the tests when given on multiple occasions. The relative brevity of the battery places more attention on the ability of each test to detect subtle changes that result from brain injury. Much more work needs to be done to further our understanding about the reliability and validity of neuropsychological tests when used with an athletic population.

Scientific advances are now being made to provide more precise information about the use of neuropsychological testing with athletes. Information including RCIs to determine changes in scores and signal detection statistics cannot be used blindly by sports medicine clinicians in their care of athletes with known or suspected concussion. There will never be a neuropsychology "cookbook" that spells out all the decision rules regarding the use of these test scores. Although the tests may yield a plethora of numbers, the ultimate use of this statistical information remains with the decision-making team, with much room for interpretation. For example, if neuropsychological test scores yield a "conservative" estimate of an 80% chance that a particular athlete has experienced a concussion, parents and educators might want the athlete to remain out of competition. Coaches or other team officials, however, might view the same numbers as indicating a 20% chance of no symptoms and recommend an immediate return to play. It should be made clear that the cutoff points used in any statistical decision are ultimately arbitrary and need to be set by the clinician, depending on experience and the question at hand.

Neuropsychological testing has been used in the sport setting increasingly during the past 5 years. Although the tests have shown promise in the management of sport-related cerebral concussion, their limitations should be realized. These measures provide the scientific "state of the art" for assessment of cognitive dysfunction secondary to a wide range of medical causes, although they are by no means perfect. The field of clinical neuropsychology is known for being more crit-

ical of its methods than other health-related disciplines. This ultimately results in continued studies of ways to improve methods. Continued use of neuropsychological tests is recommended despite their inherent flaws, although athletic trainers and team medical personnel have a right to suspend judgment about their ultimate value in a sport setting until more information about their use in athletes has been obtained.

Scientific data regarding the reliability and validity of the most commonly used paper-and-pencil tests are now emerging and are appearing increasingly in the professional literature. These data are the results of many years of hard work testing many thousands of individual athletes. New computerized methods for assessing neuropsychological functioning in athletes are now in development. Athletic trainers should remain skeptical about these more automated methods until their psychometric properties, including reliability and validity, have been demonstrated in an empirical manner on adequate numbers of athletes, both with and without symptoms of concussion. Keeping in mind the methodologic issues involved in the development and use of these methods will ultimately make the athletic trainer an informed consumer of neuropsychological testing services, both in the present and in the future.

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Multiple Concussions and Neuropsychological Functioning in Collegiate Football Players

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Stephen N. Macciocchi, PhD, Jeffrey T. Barth, PhD, ABPP/ICN, Lauren Littlefield, PhD, and Robert C. Cantu, MA, MD, FACS, FACSM, contributed to conception and design; acquisition and analysis and interpretation of the data; and drafting, critical revision, and final approval of the article.

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Objective: To document neurocognitive and neurobehavioral consequences of 1 versus 2 concussions.

Design and Setting: Nonequivalent, pretest-posttest cohort design with multiple dependent measures. Participants were selected from a large sample of athletes who participated in a comprehensive, multiuniversity study of football-related concussion.

Subjects: College football players who sustained 1 and 2 grade 1 concussive injuries were matched for age, education, and duration of competitive football.

Measurements: Neuropsychological tests and symptoms checklists.

Results: Multivariate analysis of variance did not show a statistically significant difference in test performance between players with 1 or 2 concussions. Chi square analyses revealed that concussions significantly increased the number of symptom complaints, but symptoms returned to baseline by 10 days postinjury. The effects of 2 injuries did not appear to be significantly greater than that of a single injury. Differences in response to concussion were observed.

Conclusions: Neurocognitive and neurobehavioral consequences of 2 concussions did not appear to be significantly different from those of 1 concussion, but methodologic issues place limitations on data interpretation. Additional studies are needed to clarify the neuropsychological consequences of multiple concussions.

Key Words: sports injuries, neuropsychological tests, symptoms

Over the past 15 years, research pertaining to concussive injuries sustained during athletic endeavors has increased substantially.[1] Findings from these studies have been generally consistent and suggest that concussive injuries in competitive American football can cause time-limited neuropsychological and neurobehavioral problems.[2–4] Although 1 concussion does not appear to result in significant morbidity, the effect of multiple concussions is less clear. In a recent study,[4] a posttest-only control group design was used to compare athletes who had a history of 1 concussion with athletes who had a history of 2 or more concussions. The authors found that athletes who sustained 2 or more concussions reported more neurobehavioral symptoms and had more impairment on selected neuropsychological tests than athletes who had a history of a single concussion. Despite the differences on some neuropsychological measures, athletes with a history of 1 versus 2 or more concussions did not differ on tests of auditory attention, verbal fluency, verbal learning, verbal memory, or fine motor dexterity.[4] In addition, players with 2 or more concussions were aggregated, and the effect of different numbers of concussions was not specified.[4]

Most studies investigating the effects of single or multiple concussions have been retrospective investigations using posttest-only designs.[1] Accordingly, we designed our investigation to prospectively examine the neurobehavioral and neuropsychological consequences of 2 concussive injuries. Players who sustained 2 concussions were compared with players who sustained 1 concussion using a nonequivalent, pretest-posttest comparison design. Players who sustained 1 concussion were used as controls in order to contrast the effects of 1 versus 2 concussions. As mentioned previously, several studies have shown that a single concussion is associated with time-limited neurocognitive impairment. As such, identifying neurocognitive impairment was not the primary focus of our investigation. The primary goal was to determine if a second concussion produced identifiable cognitive deficits above and beyond those observed after a single injury. Based on prior investigations, we hypothesized that players who sustained 2 concussions would evidence significantly greater neurocognitive dysfunction and postconcussive symptoms compared with players who sustained a single injury.

METHODS

Subjects

Participants in this study (n = 24) were selected from a larger sample of athletes who participated in a comprehensive

study of concussive injury in Division I-A collegiate football players. In the initial study, 2300 players were prospectively examined and followed for 4 years to determine the neuropsychological consequences of concussive injuries. During the study period, 195 players sustained grade 1 concussions based on contemporary classification guidelines.[1] Six percent of all players with documented concussions sustained 2 injuries (n = 12). Five of these athletes sustained concussions in the same year (mean separation, 33 days; range, 14 to 70 days), while 7 players sustained concussions in consecutive years (mean separation, 532 days; range, 364 to 686 days). Players who sustained 2 concussions (T_2) were compared with a selected cohort of players who sustained a single concussion (S). Players sustaining 1 concussion were selectively matched with players sustaining 2 concussions based on age, education, years in competitive football, and prior concussion history (none). Players with a single concussion (S) had a mean age of 19.5 years and a mean 8.4 years of experience in competitive football, and players with 2 concussions (T_2) had a mean age of 19.1 years and a mean 9.1 years of experience in competitive football.

METHODS

All players (n = 24) were assessed preseason to establish baseline functioning. In addition to completing a physical examination, players completed several neuropsychological measures, including the Paced Auditory Serial Addition Task (Brainmetric Software, Marlton, NJ),[5] the Trail-Making Tests A and B from the Halstead-Reitan Neuropsychological Test Battery (Reitan Neuropsychological Laboratory, Tucson, AZ),[6] and the Symbol Digit Test (Psychological Assessment Resources, Inc, Odessa FL).[7] These tests were designed to measure various aspects of visual and auditory attention as well as information processing speed. Psychometrics of these instruments can be obtained from various sources.[8] Players also completed a history questionnaire and a symptom checklist.

Players who were suspected of sustaining head injuries during practices or games were examined by certified athletic trainers and physicians using standardized medical and mental status procedures. Players' temporal and spatial orientation and short-term memory were systematically assessed after injury. Players failing items requiring intact orientation and memory were considered to have sustained a concussion and were continuously assessed until resolution of posttraumatic confusion. No players in our study experienced a documented loss of consciousness or posttraumatic confusion lasting longer than 30 minutes, which is consistent with a grade 1 concussion using the American Academy of Neurology and Virginia Neurologic Institute Standards.[1] Players who failed the mental status examination were then assessed at 24 hours, 5 days, and 10 days postinjury using the neuropsychological measures administered during the baseline assessment. Neuropsychological tests were administered by research staff trained in test administration.

RESULTS

Neuropsychological test scores and self-reported symptoms of players who sustained 2 injuries (T_2) were compared with test scores and symptoms of players who sustained a single injury (S) using a between-subjects multivariate analysis of variance (MANOVA). In addition, a within-subjects MANOVA was used to compare scores of players experiencing 2 injuries after their first (T_1) and second (T_2) injuries to determine if a second concussion produced a change in cognitive functioning. Additionally, players who were injured twice in close temporal proximity (mean separation, 33 days) were compared with players who sustained 2 injuries over 2 or more seasons (mean separation, 532 days). Finally, preseason and postseason scores of players with 2 injuries were compared using a within-subjects MANOVA to examine changes over time.

Mean test scores for each group are presented in Table 1. The MANOVA analysis revealed that the test results of players with a single injury (S) did not differ significantly from those of players who sustained 2 injuries, either at the time of their first injury (T_1: $F = 4.2$, $P < .06$) or second injury (T_2: $F = 1.09$, $P < .386$). Within-subjects comparison of players who sustained 2 injuries after their first injury (T_1) and second injury (T_2) revealed no significant differences in test performance ($F = 0.858$, $P < .514$). Comparison of players' preinjury test scores with postseason performance after their second injury revealed a trend toward improved performance ($F = 3.27$, $P < .108$). When the group sustaining 2 concussions was analyzed separately, no differences were noted in test performance between players who sustained injuries in close proximity or in successive seasons ($F = 1.12$, $P < .351$).

Players' self-reported symptoms (headache, dizziness, and memory loss) were summed before completing the analyses (Table 2). Statistical examination of the total number of symptoms using χ^2 analyses revealed a significant effect for time. Both groups (S and T_1) had a statistically significant increase in the number of players with symptoms (headache, dizziness, and memory loss) at 24 hours postinjury ($\chi^2_4 = 22$, $P < .001$) and 5 days postinjury ($\chi^2_4 = 40$, $P < .001$). In contrast, the number of players with symptoms at 10 days postinjury was not significantly different from the number with symptoms preseason ($\chi^2_4 = 0.20$, $P < .50$). Analyses of symptoms with respect to groups revealed significant differences in symptom reports (headache, dizziness, and memory loss) between group S (single injury) and group T_1 after their first injury ($\chi^2_2 = 10.6$, $P < .005$). Players who sustained 2 injuries did not evidence statistically significant differences in symptom reports after first injuries (T_1) and second injuries (T_2) ($\chi^2_2 = 1.41$, $P < .50$). The proportion of patients reporting symptoms also did not differ for players sustaining injuries in close proximity and players sustaining more remote injuries.

DISCUSSION

Our analyses suggest that 2 grade 1 concussive injuries sustained at least 2 weeks apart during competitive American football did not result in significantly more neurocognitive impairment than a single concussive injury. Compared with players who sustained a single injury, players who sustained 2 injuries performed as well as or better on all neuropsychological tests after their first and second concussions. In addition, after a second concussion, there was no evidence of a decrement in test performance relative to the performance observed after players' first concussions. Furthermore, players who sustained 2 concussions performed better on postseason assessments than on preseason examinations.

Analyses of self-reported symptoms revealed a significant effect for time after injury. The number of players reporting symptoms increased significantly after 1 or 2 injuries, but

Table 1. Test Scores in Players With 1 versus 2 Mild Head Injuries*

Test	Time				
	Preseason	24 h Postinjury	5 d Postinjury	10 d Postinjury	Postseason
Trail-Making A					
S	21	22	17.9	17	NA†
T_1	22.8	21.7	18.8	18.6	NA
T_2	22.8	17.6	16.9	15.9	16.6
Trail-Making B					
S	46.8	39	39.8	34.5	NA
T_1	50.5	40.1	35.2	36.1	NA
T_2	50.5	37.4	30.3	29.9	32.9
Symbol Digit					
S	55.7	57.8	62.2	61.3	NA
T_1	62.5	59.2	65.9	70.0	NA
T_2	62.5	61.5	68.8	71.4	71.4
Paced Auditory Serial Addition Task 3					
S	77	81.9	96	88.4	NA
T_1	82.5	86.7	94.4	93.8	NA
T_2	82.5	92.1	96.0	94.6	94.3
Paced Auditory Serial Addition Task 4					
S	65	62	78.5	88.1	NA
T_1	72.4	77.1	90.8	88.1	NA
T_2	72.4	86.6	90.2	93	88.4

*S indicates 1 concussion (control); T_1, 2 concussions (first injury); and T_2, 2 concussions (second injury).
†Not available.

Table 2. Number of Players Reporting Postconcussive Symptoms*

Time	No. Reporting Headache			No. Reporting Dizziness			No. Reporting Memory Loss		
	S	T_1	T_2	S	T_1	T_2	S	T_1	T_2
Preseason	4	3	3	1	1	1	1	1	1
24 h postinjury	8	4	5	2	3	4	2	2	4
5 d postinjury	4	8	7	0	4	5	2	3	3
10 d postinjury	4	2	4	0	2	2	2	1	0

*S indicates 1 concussion (control); T_1, 2 concussions (first injury), and T_2, 2 concussions (second injury).

symptom reports essentially returned to baseline by 10 days postinjury in both groups. The most commonly reported symptom in both groups was headache, but players who sustained 2 concussions reported more symptoms after their first and second concussions when compared with players who sustained a single concussion. Despite the presence of a differential response to the first injury, the frequency of players' symptoms after first and second injuries revealed no statistically significant increase in symptoms after a second injury, whether this injury occurred in close proximity to the first injury or at a more remote time. In other words, even though one group of players experienced more symptoms after their first injury, the responses to their first and second concussions were remarkably similar. Although interesting, the significance of these findings is not entirely clear. Differences in symptom reports could be due to normal variations in injury response accentuated by selective matching. In the future, variability in symptom reports after injury can be examined to assess whether players who experience prominent self-reported symptoms after a concussion are at greater risk for a second concussion.

Despite our findings, several methodologic issues merit discussion. First, the base rate of documented multiple injuries in our sample was quite low (6%).[3] As such, our data are based on a small sample of players who may not adequately represent the population of players who typically sustain multiple injuries. Second, all of our players sustained grade 1 concussions by contemporary classification standards.[9] Although the effect of injury severity is generally consistent across players, the cumulative effects of more severe injuries are unknown. Third, the timing of injuries in our study was variable. For example, only 2 players experienced a second injury within 2 weeks of their first injury. In fact, 7 players did not even sustain both injuries in the same year but rather within 12 to 24 months. Because neurocognitive impairment and neurobehavioral symptoms after 1 concussion resolve rather rapidly,[2–4] the extended time between injuries may have limited the in-

teraction between the first and second injuries. Most importantly, even though we observed no differences between players with proximal versus remote injuries, our sample was too small to definitively answer questions about injury proximity. Finally, none of our players sustained more than 2 concussions, which limits direct comparison with studies assessing players with as many as 10 concussions.[4]

In addition to sample size, injury frequency, and the timing of injuries, test sensitivity issues require comment. For example, a number of neuropsychological tests are susceptible to practice effects.[10] In our study, players who were injured twice were exposed to all tests on at least 7 occasions. Actually, despite being injured, players evidenced improved performance over time regardless of testing time (24 hours, 5 days, 10 days) or injury status (1 or 2 concussions). As such, the genuine neurocognitive consequences of concussions may be obscured by considerable exposure to tests. Of course, an injury with serious neuropsychological consequences would most likely reduce the influence of practice effects, but there was no evidence of a significant decline in neuropsychological test performance for any player in our sample.

A final issue deserving attention is the effect of group research on individual responses to concussion. For example, neurocognitive test data and symptom reports document variability in response to concussions with apparently equivalent clinical features such as duration of posttraumatic amnesia. In other words, the group that experienced 2 concussions did report more symptoms after their first injury, and this reporting continued after their second concussion. Consequently, group studies using aggregated data may obscure differential responses to and recovery from injury. In order to address this issue, investigators have recently recommended using reliable change indexes (RCIs) when conducting research.[11] RCIs are calculated using preinjury and postinjury scores, with mathematical consideration given to the standard error measurement and test reliability. In essence, RCI is a type of effect size. Calculating effect sizes of injuries for individual players may yield information that would be lost when summing group data. For example, players with large injury effects can be examined independently for relationships among injury severity, neurocognitive functioning, and neurobehavioral symptoms.

In spite of the study's limitations, our data suggest that 2 concussions do not result in a statistically or clinically significant increase in neurocognitive deficits relative to a single concussion. There is also no compelling evidence that self-reported symptoms are more common or severe after a second injury. Unfortunately, methodologic limitations do not permit generalization of these data to populations in whom injuries may be more frequent, may occur in closer temporal proximity, or may be more severe. Nonetheless, as documented by other studies, our data do suggest that self-reported symptoms may be sensitive indicators of postinjury neuropsychological impairment.[2] As such, the presence of symptoms should be given serious consideration in return-to-play decisions, regardless of neuropsychological test performance.[1] In any case, further research is needed to more closely examine the effect of multiple concussions on neuropsychological function. Until then, we can have modest confidence in the fact that, although undesirable, 2 grade 1 concussions occurring at least 2 weeks apart did not appear to produce significantly greater impairment than a single injury, at least in this population of collegiate football players.

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Cerebral Concussion: Causes, Effects, and Risks in Sports

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Objective: To characterize the causes, effects, and risks associated with concussion in sports.

Background: Concussion is an injury associated with sports and is most often identified with boxing, football, ice hockey, and the martial arts. In addition, recent research has shown that concussion occurs in many different sports. In the decade of the 1990s, concussion became a primary issue for discussion among the media, sports sponsors, sports medicine professionals, and athletes.

Description: Concussion is an injury that results from a wide variety of mechanisms and has numerous signs and symptoms that are common to different types of injury. Continued improvement in prevention and management strategies for concussion requires a strong body of research from a variety of different disciplines. It is essential that research efforts focus on both prevention and management and that researchers and clinicians work closely toward their common goals.

Conclusions/Recommendations: Until the research community is able to provide sound recommendations for the prevention and management of the concussion, the care of the injured player falls squarely on the clinician. It is important for sports medicine professionals to continue to stay up to date on the advances in understanding concussions and how to care individually for each player who sustains a concussion.

Key Words: mild traumatic brain injury, head injury, injury prevention

In today's competitive sports environment, large numbers of athletes participate in a wide variety of youth, high school, collegiate, professional, and recreational sports. Whereas some sports (eg, football) have maintained a consistent number of participants, others have increasing participation.[1] For the younger participants, the sport experience provides an environment in which they can grow and develop physically, mentally, and socially. For college and professional athletes, sports offer an opportunity for personal success and future employment. For recreational athletes, sports provide opportunities for maintaining a healthier lifestyle and an outlet for relieving the tensions of modern life. As sports programs continue to flourish, it is the responsibility of the sponsors of these programs to provide an environment that minimizes the risk of injury.

Risk of injury is inherent in sports participation. This risk stems from the nature of the game and the specific activities of the participants, both during their participation and during events that surround their participation. For example, collision sports such as football and ice hockey characteristically have more acute traumatic injuries than sports such as swimming and track. Boxing has more head-related trauma because of the focus of the sport. Within each sport, a general injury pattern and specific types of injury are unique to the sport. One type of injury that can occur in any sport or physical activity is concussion. This injury represents the most common type of acute brain injury in sports and is most often associated with boxing, football, ice hockey, and martial arts.[2] As with other types of injury, the frequency and severity of concussion that is associated with a sport is a function of the nature of the game, specifically, the rules and regulations, the specific physical activities of participants, and the environmental conditions associated with the game.

HISTORICAL PERSPECTIVE

Concussion has been associated with a range of definitions that generally focus on the nature of the medical signs and symptoms present at the time of injury. Concussion has been described as a clinical syndrome characterized by immediate and transient posttraumatic impairment of neural function, such as alteration of consciousness or disturbance of vision or equilibrium, and other signs and symptoms due to brain stem involvement.[3] This approach includes the classic "ding" associated with head injury in sports. Some authors have indicated that concussion must be associated with a loss of consciousness, either short term or long term. Concussion has been defined as a "trauma-induced alteration in mental status that may or may not involve a loss of consciousness."[4,5] If surveyed, the public would probably associate concussion with boxing, football, and ice hockey and would not consider the injury a real problem for other sports. This perception is most likely related to the visibility of boxing, football, and ice hockey at the professional level. The current thinking among sports medicine clinicians is that concussion occurs in all sports but with varying frequencies. In reality, concussions have always been a part of competitive athletics.

The study of Gerberich et al[6] published in 1983 was one of the first widely cited articles to deal with concussions in high

school football and spanned the years 1978–1982. They found that 20% of the reported injuries in high school football were concussions and that 14% of the high school football players included in their study reported a history of concussion associated with a loss of consciousness. From this work, other authors have projected a frequency of 200 000 concussions per year in high school football. It is important to consider the era for the data collection in this article. In the early 1970s, emphasis was on the use of the head and the face mask as the initial points of contact for blocking and tackling. Risks associated with this technique were documented, and in 1976, the National Federation of State High School Associations Football Rules Committee banned the use of the face mask as the initial point of contact. The players surveyed by Gerberich et al[6] had participated before the ban. In addition, football players in the 1970s wore a variety of helmets that are no longer in use or manufactured. Therefore, these data may not accurately reflect the magnitude of the problem for today's high school football player.

In the early 1980s, the discussion of concussion took a giant leap forward with the work of Rimel et al[7] and Barth et al.[8] Their research efforts identified neuropsychological effects associated with the injury. During the decade that followed, a great deal of discussion flourished among members of the neuroscience community regarding the description, classification, and management of concussions and guidelines for the return to competition after concussion.[4,5,9–12] As the medical community learned more about the natural history of concussion, the importance of the injury, regardless of the sport, was recognized.

The discussion of concussions related to sports received very little attention until the early 1990s. The media and fans fostered a heightened awareness for sport concussion as they learned of high-profile professional athletes who attributed their retirements to repetitive concussions. Additionally, postconcussion syndrome was identified in players who retired for other reasons in the months and years after their injuries. As a result, the current research concerned with the identification, management, and long-term effects of concussion is adding new and exciting information to the professional body of knowledge required to reduce the risk of injury.

In 1994, the National Athletic Trainers' Association (NATA) Research and Education Foundation conducted the Mild Brain Injury Summit.[13] The program brought together professionals from neurosurgery, neuropsychology, neurology, sports medicine, and athletic training. The objective was to examine the current knowledge regarding the risk of concussion in sports. Panel members discussed the definition of concussion and the various types of programs designed to provide medical care and management for concussion, examined the status of existing research, and made recommendations for future research.[13]

During the past few years, a plethora of articles, papers, and symposia have discussed the concussion. Much of the discussion has focused on refinements of the grading and management systems identified during the 1980s and early 1990s.[5,10,14] Many in the medical professions and media have encouraged the use of the consensus practice option identified by the American Academy of Neurology, but there is still no consensus on which concussion grading system is the best.[4] Another issue that has received much discussion and been the focus of numerous papers and several research projects is the role of neuropsychological screening and follow-up as a tool for managing concussion. Within the many articles are a number of common points as well as continuing points of disagreement, especially regarding the grading and management of the injury. It is important for us to be able to integrate the new knowledge regarding concussion with the past knowledge to move forward in the area of prevention.

One of the most challenging issues facing medical and paramedical professionals is the identification of concussions. The main problem with identification is the variety of signs and symptoms that may or may not be present. For example, headache, dizziness, nausea, or memory alterations may be symptoms of concussion, or they may be symptoms of other injury. The injury may result in short-term or long-term unconsciousness or no loss of consciousness. The signs and symptoms present at the time of injury may disappear very quickly, or they may linger for long periods. In some rare cases, the initial signs and symptoms may disappear and then reappear with dramatic consequences. Some of the common symptoms result from different types of injury; for example, dizziness may result from brain injury or from vestibular injury (W. Meeuwisse, oral communication, 2000).

Once a concussion has been identified in a player, it warrants professional evaluation by clinicians who are trained and up to date on the management of concussion. Team physicians and athletic trainers must realize that concussions occur in every sport and that, although the injury may have occurred in volleyball, its proper management is just as important as if the concussion had occurred in football.

After the identification and management of concussion, the question of return to participation becomes most important. How long should the athlete wait to return to collision sports? How long should he or she wait to return to noncollision sports? What is the potential for the player to sustain a second concussion? And, does this second injury create more significant damage than the first one? How can the player be sure that the brain has truly "returned to normal"? Current research is focusing directly on the answers to these questions. Until that research is complete, the clinician should treat the players who sustain concussions on an individual basis. Decisions regarding return to play should be based on the signs and symptoms associated with the player's injury and not simply grouped into one of the many current classification and injury management systems. Among the new tools being placed in the hands of clinicians is the ability to compare baseline brain function parameters with the same parameters after a concussion. The research in this area is most promising and will be an asset to the management of concussions.[15–18]

The sponsors of sports programs have challenges of their own to face. They must design and maintain injury prevention programs that minimize the risk of injury, including concussion. They must take into consideration the nature of the sport and the activities of the players as they make decisions that affect the injury risk pattern. Specific areas that require attention are facilities and equipment, player protective equipment, and competition rules and regulations. Combining and using information from medicine, program sponsors, athletic trainers, coaches, and players is essential to improving player safety.

RESEARCH ISSUES

As concussion became the sports injury "issue" of the 1990s, numerous researchers began projects regarding concussions. It became apparent from the findings of the earlier pro-

jects that a variety of different methods of identifying a concussion exist. Some clinicians described a concussion as a loss of consciousness. Others identified a concussion only if memory problems were associated with the injury. Still other clinicians considered a very minor impact to the head, often called a "ding," to be a concussion. The confusion over the definition created problems for multicenter research programs. To be comfortable with the consistency of data coming from different locations, researchers' projects began to identify mild traumatic brain injury (MTBI) as a synonym for concussion.[19] MTBI represents an injury that meets specific criteria regarding the presence of signs and symptoms, and when these minimum conditions exist, the injury is considered reportable for the research project.[19] In general, the MTBI approach to injury identification encompasses all of the qualities that have been associated with concussion. The use of an operational definition of a reportable MTBI allows researchers and clinicians to begin their analysis from a common reference point for injury.

EPIDEMIOLOGY OF CONCUSSION

Among a wide variety of sports, the potential for concussion is related to the number of opportunities within the sport for activities that produce collisions. For example, in football, the number of collisions involving the head is very high. Some players on the field experience a head impact on every play. In other sports such as ice hockey, impacts with the head are expected but not inherent in the design of the sport. Sports such as tennis and swimming have little potential for collision, although falls on the court or collisions with walls may occur. The frequency of collisions associated with a specific sport is a function of the opportunity for collision to occur within the context of the sport. The number of collisions in a practice or game is directly related to the potential for concussion.

To evaluate the potential for injury, we must have a general understanding of the likelihood that injury will occur. In the case of concussion, the likelihood of injury is a function of the number of times a player's head sustains an impact within the context of participation. These impacts may be incidental (unintentional) and occur as a result of the nature of the game, or they may be impacts that result from intentional acts (eg, fighting). There may be head impacts from objects associated with the game, such as sticks, surfaces, boundary obstructions, or game operations equipment. These impacts may be frequent and considered a part of the game, as in football, or very unusual, as in tennis. The important consideration is that the concussion can occur in any activity, regardless of the nature of the activity, and that when the injury occurs, it has the potential for a lasting effect on the player. Since high school football has the largest number of participants and is most often associated with concussion, an estimate of the number of head impacts would provide perspective on the risk of injury.

CONCUSSION IN HIGH SCHOOL SPORTS

The NATA conducted a study of the frequency, type, and severity of injury in high school football for the 1995 through 1997 seasons. The purpose of the study was to examine the frequency patterns associated with participation in 10 high school sports: football, boys' and girls' basketball, boys' and girls' soccer, wrestling, field hockey, baseball, softball, and girls' volleyball.[19]

The subjects in the NATA study were athletes on the varsity sports rosters at the study schools. Athletic trainers certified by the NATA Board of Certification recorded data from 236 high schools over the 3-year study period. Before the project began, the operational definitions and reporting requirements were included in a user's manual and distributed to all data recorders. The definition of *reportable injury* included injuries necessitating removal of athletes from participation for the remainder of the current practice or game or longer. Additionally, all fractures, dental injuries, and MTBIs were reportable, regardless of time lost. Because of the variations in the definition, classification, and management of concussion that exist among the different classification systems, it was important that the study employ a definition for concussion that could be used by all study participants. Rather than ask the athletic trainers to report a "concussion," the study identified a definition for a reportable MTBI. An MTBI represented an event identified by the athletic trainer as an incident that required the cessation of a player's participation for initial observation and evaluation of the injury signs and symptoms before returning to play, either in the current session or subsequent sessions.[19]

In the NATA study, data were collected for 23 566 reportable injuries in 3 years, of which 1291 (5.5%) were MTBIs.[19] The injury rates per 100 player-seasons for each sport were 3.66 for football, 1.58 for wrestling, 1.14 for girls' soccer, 0.92 for boys' soccer, 1.04 for girls' basketball, 0.75 for boys' basketball, 0.46 for softball, 0.23 for baseball, 0.46 for field hockey, and 0.14 for volleyball. Among the players with MTBI, 76.1% missed fewer than 8 days, with a median time lost for all MTBIs of 3 days. Rates of MTBI were higher in a game than in a practice for all sports except volleyball.

Based on this study, the rates of MTBI varied among sports, and no sport was without the occurrence of an MTBI. The prevention of MTBI, given its close association with a variety of different types of collisions, may be most successful using interventions aimed at controlling the participation environment. Decision makers in sports safety should focus their prevention efforts on programs that minimize the potential for head impacts from collisions, both intentional and unintentional. The continued cooperation of sports sponsors, researchers, medical professionals, and sports participants is essential to help minimize the risk of concussion.

CONCUSSION PREVENTION

The foundation for developing a sports injury prevention program is that regardless of the preventive steps taken to avert sports injuries, including concussion, some players will continue to be injured. It becomes the task of the injury prevention team to work toward limiting the numbers of injuries by using regulatory controls, educating participants, designing specialized protective products, and monitoring the injury frequency patterns through ongoing surveillance programs.[20] First, the program must emphasize prevention by focusing on decisions relevant to the rules and regulations of the games. Continual review of this area allows the governing agency to require that the sport meet the current standards of injury prevention. Second, the coaches must continue to review the up-to-date techniques for teaching appropriate skills that facilitate player performance and, at the same time, provide consistent protection from injury. In addition, coaches need to be sensitive to both general and sport-specific conditioning to properly prepare the

Countermeasures: Concussion in Sports[21]

Factors	Phase		
	Pre-event	Event	Postevent
Host (player)	Preparticipation evaluation of concussion history	Properly designed and maintained player head protection, when appropriate	Recognition and management of players evaluated for concussion
Agent (sport)	Rules and regulations relating to minimizing the risk of injury	Maintenance of standards related to the safety issues for the sport (eg, rule enforcement)	Evaluation of injury incidents to establish procedures for preventing future injury
Environment (ambient conditions)	Playing facilities free of inherent hazards (eg, padded sideline equipment for collision games)	Resources (personnel) immediately available to evaluate and manage the injured	Availability of emergency medical services and medical support for injury management

athletes for the rigors of competition. Continuing investigation of the products that are used to protect players from the inherent hazards associated with the sport should occur. The continued monitoring of the injury patterns associated with sports will go a long way in providing the safety decision makers with up-to-date information regarding the status of injury prevention programs.

The early recognition of concussion provides for the implementation of proper management for even the least severe injury. This early identification and management minimizes the risk of a negative outcome and maximizes the probability of the player's returning to competition without an increased risk of reinjury. The process of monitoring concussion in players should include player-reported symptoms, apparent signs of incomplete recovery, and the development of protocols for assessing brain function (ie, neuropsychological assessment).

A DECISION MODEL FOR PREVENTION

Injuries in sports occur in a specific moment when a wide variety of internal (player-related) and external (sport-related) risk factors converge. To begin the process of developing an injury prevention or injury control program, including programs specifically aimed at concussion, the decision maker must bring order to the variables that exist at the time of injury. William Haddon Jr[21] developed a model for categorizing some of the complexity and diversity of the variables that exist at the time of an injury. His model addresses the conditions that are present before the injury occurs (pre-event), at the time that the injury occurs (event), and after the injury occurs (postevent).[21]

In Haddon's model, the 3 time phases are coupled with the variables associated with the host (player), agent (sport), and environment (ambient conditions) to produce a matrix for planning intervention strategies.[21] For example, a model for developing intervention programs for preventing sports concussion might resemble the model shown in the Table. The items in this table represent a general approach to concussion. Because of the varying nature of each sport, a specific approach that considers the qualities and conditions for the sport must be developed.

RECOMMENDATIONS FOR CONCUSSION MANAGEMENT

The management and prevention of concussion have become paramount issues among sports medicine professionals. Until we have a better understanding of exactly what constitutes a concussion, especially regarding the amount of force associated with specific levels of injury, it is difficult to focus on the most important strategies for preventing the initial injury. We must focus our attention on preventing reinjury by enhancing our management of injured players. Clinicians associated with sports may have a variety of ideas regarding the prevention and management of concussion; the management recommendations identified by the NATA summit[13] seem to capture the essence of most of the more common ideas:

1. The injured player should be managed as an individual case.
2. Clinicians should familiarize themselves with current standards for the evaluation of concussion on the sideline as well as in the office.
3. Consistent and routine follow-up procedures should be implemented to monitor the individual's progress.
4. Clinicians should provide accurate and consistent information to the patient's "supporters" regarding danger signals associated with concussion.
5. Accurate documentation of the injury event, findings on evaluation, and decisions to return to participation should be encouraged.

Another important area examined by the NATA summit panel members[13] was the question of the current state of knowledge regarding the research that must be done to better understand the risks of concussion both in the short term and in the long term. The following recommendations provide direction for the research programs associated with concussion:

1. Research requires a multidisciplinary team of professionals. The team represents the neuroscience community, the rehabilitation professions, and sports medicine team physicians and athletic trainers.
2. Important areas for consideration are the effects of multiple injuries and the relative risks associated with continued participation.
3. Emphasis should be placed on developing procedures for acquiring neuropsychological baselines to evaluate the effect of concussion over time.
4. Research efforts that focus on pharmacologic intervention for prevention and management are encouraged.
5. Specific programs must address the recovery time for concussion and its relationship to reinjury and the long-term effects.

SUMMARY

The problem of concussion in sports is one that has moved to the forefront in the past few years. The retirement of high-

profile professional athletes as a result of repetitive concussion and postconcussion syndrome has heightened the awareness of the sports community to the importance of these injuries. The potential for serious effects of brain injury on the individual player's physical and mental status is generally accepted. The ability to provide objective information regarding the exact nature of the effects of concussion, both in the short term and over time, has been lacking. The unpredictability of concussion and the inability to identify cases in the general population have made large-scale research projects impossible. Thus, the focus of research and education regarding brain injury has been centered on the more serious cases. Recently, the research community has begun to implement programs for the in-depth study of concussion in the sports arena. Under these conditions, head injury risks can be identified and patients with concussion can be followed to assess long-term effects. Today's computer technology has made the uniform documentation of injuries among multiple institutions a reality. The ability to coordinate information from multiple sites, multiple professions, and a wide variety of athletes will provide the foundation for developing intervention programs for preventing and managing cases of concussion, both for the athlete and the nonathlete. The potential is bright for the future success of programs that will minimize the risk of concussion and techniques to manage the concussions that continue to occur. The intensity of the light cast depends directly on the continued cooperation of the sponsors of sports programs, the sports medicine community, the coaching community, and the consumers of the sport, the players.

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Catastrophic Head Injuries in High School and Collegiate Sports

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Objective: To describe the incidence of catastrophic head injuries in a variety of high school and college sports.

Design and Setting: Data on catastrophic head injuries were compiled in a national surveillance system maintained by the National Center for Catastrophic Sports Injury Research. The data were compiled with the assistance of coaches, athletic trainers, athletic directors, executive officers of state and national athletic organizations, a national newspaper clipping service, professional associates of the researchers, and national sport organizations.

Subjects: Data included all high school and college athletic programs in the United States.

Measurements: Background information on the athlete (age, height, weight, experience, previous injury, etc), accident information, immediate and postaccident medical care, type of injury, and equipment involved. Autopsy reports were used when available.

Results: A football-related fatality has occurred every year from 1945 through 1999, except for 1990. Head-related deaths accounted for 69% of football fatalities, cervical spinal injuries for 16.3%, and other injuries for 14.7%. High school football produced the greatest number of football head-related deaths. From 1984 through 1999, 69 football head-related injuries resulted in permanent disability. Sixty-three of the injuries were associated with high school football and 6 with college football. Although football has received the most attention, other sports have also been associated with head-related deaths and permanent disability injuries. From 1982 through 1999, 20 deaths and 19 permanent disability injuries occurred in a variety of sports. Track and field, baseball, and cheerleading had the highest incidence of these catastrophic injuries. Three deaths and 3 injuries resulting in permanent disability have occurred in female participants.

Conclusions/Recommendations: Reliable data collection systems and continual analysis of the data can help us to reduce the number of catastrophic head-related injuries. I include additional recommendations for injury prevention.

Key Words: fatality, brain injury

Head trauma results in more fatalities than any other sport injury and is the cause of most football fatalities, but it can also be associated with other sports.[1,2] Fatal head injuries have been reported in soccer, wrestling, track, baseball, and a number of other sports. The morbidity and mortality associated with traumatic brain injury have been labeled a silent epidemic because they have received remarkably little attention compared with other neurologic illnesses.[3] Traumatic brain injury is common in contact sports, with an estimated 250 000 concussions occurring every year in football alone.[4] Repeated concussions occurring within a short period can be fatal.[5,6]

Second-impact syndrome, or rapid brain swelling and herniation after a second head injury, is more common than previous reports in the medical literature have suggested.[7] Between 1980 and 1991, the National Center for Catastrophic Sports Injury Research at the University of North Carolina at Chapel Hill identified 29 probable cases among football players alone.[8,9] Schneider[10] first described second-impact syndrome in 1973. The syndrome occurs when an athlete who sustained a head injury—often a concussion or a worse injury, such as a cerebral contusion—sustains a second head injury before symptoms associated with the first have cleared. For a catastrophic condition that has a mortality rate approaching 50% and a morbidity rate nearing 100%, prevention takes on the utmost importance.[7]

Guskiewicz et al[11] found that football players who sustained 1 concussion in a season were 3 times more likely to sustain a second concussion in the same season compared with uninjured players. Recent statistics suggest that approximately 300 000 sport-related traumatic brain injuries or head injuries occur annually in the United States. The Centers for Disease Control and Prevention[12] reported a high incidence of repeated head injuries in several sports and warned that the likelihood of serious sequelae increases with repeated head injury.

METHODS

The collection of football fatality data began in 1931, when the American Football Coaches Association initiated the First Annual Survey of Football Fatalities.[8] The research has been carried out on a national level every year except for 1942 and has been conducted at the University of North Carolina at Chapel Hill since 1965. The title of the survey was changed in 1980 to the Annual Survey of Football Injury Research. The primary purpose of the research was, and still is, to make the game of football a safer and, therefore, a more enjoyable sport. As a result of these surveys, the game of football has

realized many benefits with regard to rules changes and improved equipment, medical care, and coaching techniques. The survey was expanded in 1977 to include neck injuries with permanent disability and again in 1984 to include brain injuries with permanent disability. The annual survey is today known as the Annual Survey of Catastrophic Football Injuries.[9] Data collection was expanded again in 1982 to include all sports at the high school and collegiate levels, and a National Center for Catastrophic Sports Injury Research was established. This expansion was related to the lack of catastrophic injury data in sports other than football, the expansion of female sports and female participation due to Title IX, and the dearth of female sport injury data.

The term *catastrophic injury* is defined as any severe injury incurred during participation in a school- or college-sponsored sport. Catastrophic injuries are categorized as follows:

- Fatal
- Nonfatal: permanent, severe functional disability
- Serious: no permanent functional disability but severe injury; for example, a fractured cervical vertebra without paralysis

Sport-related injuries are also considered direct or indirect.

- Direct: injuries resulting directly from participation in the skills of the sport
- Indirect: injuries caused by systemic failure as a result of exertion while participating in a sport activity or by a complication secondary to a nonfatal injury

Only direct injuries will be discussed in this paper.

Data were compiled with the assistance of coaches, athletic trainers, athletic directors, executive officers of state and national athletic organizations, a national newspaper clipping service, and professional associates of the researchers. The National Collegiate Athletic Association, National Federation of State High School Associations, and American Football Coaches Association also assisted in data collection. On receiving information concerning a possible catastrophic sports injury, contact by telephone, personal letter, and questionnaire was made with the injured player's coach or athletic director. Data collected included background information on the athlete (age, height, weight, experience, previous injury, etc), accident information, immediate and postaccident medical care, type of injury, and equipment involved. Autopsy reports were used when available. Data were collected as close to the time of injury as possible; however, as with any surveillance system, some cases were studied much later. Occasionally, a catastrophic injury is not detected until several years after the event. Furthermore, it is impossible to guarantee that every case is identified by the catastrophic surveillance system; infrequently, a case may be missed. However, every effort is made to identify injured athletes. The system is publicized within the sports medicine community and is supported by the National Collegiate Athletic Association, the National Federation of State High School Associations, and the American Football Coaches Association. The use of a national press clipping service provides a timely and economic mechanism for case identification.

In 1987, a joint endeavor was initiated with the Section on Sports Medicine of the American Association of Neurological Surgeons. The purpose of this collaboration was to enhance the collection of medical data. Dr Robert C. Cantu, Chairman of the Department of Surgery and Chief of the Neurosurgery Service at Emerson Hospital in Concord, MA, has been responsible for contacting the physician involved in each case and collecting the medical data. Dr Cantu is also a past president of the American College of Sports Medicine.

The remainder of this paper will be divided into 3 areas: (1) football head injuries that resulted in fatalities, (2) football head injuries that resulted in permanent disability or recovery, and (3) head injuries in sports other than football that resulted in fatalities, permanent disability, or recovery.

Table 1. Football Fatalities, 1945–1999*

Body Part	No.	%
Head	491	69.0
Cervical spine	116	16.3
Other	105	14.7
Total	712	100.0

*Includes high school, collegiate, professional, and recreational athletes.

Table 2. Football Head-Related Fatalities by Level of Play, 1945–1999*

Level of Play	No.	%
High school	368	75.0
College	34	6.9
Sandlot	76	15.5
Professional	13	2.6
Total	491	100.0

*Based on 1 500 000 junior and senior high school players, 75 000 collegiate players, approximately 220 000 sandlot players, and 5000 professional players.

RESULTS

Football Fatalities

Football fatality data have been collected since 1931, but due to different data collection techniques, I will report football head fatality data from the 1945 through 1999 football seasons. Each year from 1945 through 1999, except for 1990, has produced a fatal head injury. Fatal head injuries include subdural hematomas, brain injury, fractures, and aneurysms. The year 1990 was the only year since 1931 without any record of a football fatality in all of football. There were 712 total football fatalities from 1945 through 1999. As shown in Table 1, head injuries accounted for 491 (69%), cervical spine injuries for 116 (16.3%), and other injuries for 105 (14.7%) of these fatalities. High school football produced the greatest number of football fatal head injuries when compared with college football (Table 2). It must be emphasized that there are now approximately 1 500 000 high school and middle school football players and only 75 000 college players.

As expected, most of the fatal head injuries happened while the participant was tackling or being tackled in a game. Approximately 75% of the fatal head injuries from 1945 through 1999 were subdural hematoma injuries (Table 3), and 32 fatalities were listed as other brain injuries. If the subdural hematoma fatalities were combined with the brain fatalities, they would be responsible for more than 80% of the total number of fatal head injuries. It is plausible that a large number of the

Table 3. Football Head-Related Fatalities by Type of Injury, 1945–1999*

Type of Injury	No.	%
Subdural hematoma	366	74.5
Brain injury	32	6.5
Fracture	18	3.7
Aneurysm	7	1.4
Unknown	68	13.8
Total	491	99.9

*Because of rounding, percentages do not total 100.

Table 4. Football Head-Related Fatalities by Decade, 1945–1999*

Years	No.	%
1945–1954	87	17.7
1955–1964	115	23.4
1965–1974	162	33.0
1975–1984	69	14.1
1985–1994	32	6.6
1995–1999	26	5.1
Total	491	99.9

*Because of rounding, percentages do not total 100.

Table 5. Incidence of Football Fatalities per 100 000 Participants

Year	High School	College
1960	1.78	1.53
1968	2.60	6.60
1970	1.92	4.00
1980	0.69	0.00
1990	0.00	0.00
1999	0.27	1.33

fatal head injuries listed as unknown were some type of fatal brain injury.

The number of fatalities increased to an all-time high in the decade 1965–1974, then steadily declined through 1985–1994 (Table 4). The decade from 1995 through 2004 is obviously not yet completed, but the preliminary findings suggest a declining trend. The increase in fatal head injuries that began in the early 1960s and continued into the early 1970s can be directly related to the skills of tackling and blocking that were being taught during those years. This period in football became known for tactics such as spearing, butt blocking, face to the numbers, and face in the chest. Players were being taught to make initial contact with the head and face into the opponent's chest. Not only did the rate of fatal head injuries increase, but a similar increase was seen in the rate of fatal cervical spine injuries. The 1976 rule change prohibiting initial contact with the head or face resulted in a dramatic decrease in both fatal head and cervical spine injuries. The number of direct football fatalities per 100 000 participants was low, except for 1968 (Table 5).

Football Head Injuries Resulting in Permanent Disability or Recovery

In 1977, the National Collegiate Athletic Association initiated funding for the First Annual Survey of Catastrophic Football Injuries under the direction of the National Center for Catastrophic Sports Injury Research. Catastrophic injuries were defined as football injuries resulting in brain or spinal cord injury or head or spine fractures. All cases involved some disability at the time of the injury. Early data collection involved only spinal cord injuries, but in 1984, head injuries were included. From 1984 through 1999, there were 63 high school and 6 collegiate catastrophic head injuries (Table 6). All 69 of these injuries involved incomplete recovery. Using the previously mentioned participation numbers, the catastrophic head injury rate from 1984 through 1999 was 0.27 injuries per 100 000 high school participants. The rate for college players was 0.5 injuries per 100 000 participants. In addition to the injuries with incomplete recovery, a number of serious head injuries each year resulted in complete recovery. For comparison, during this same time span (1984 through 1999), there were 104 spine injuries in high school participants and 15 in collegiate participants. All 119 resulted in incomplete recovery.

Table 6. Catastrophic Football-Related Head Injuries, 1984–1999*

Level of Play	No.	%
High school	63	91.3
College	6	8.7
Total	69	100.0

*Based on 150 000 junior and senior high school players and 75 000 college players.

Catastrophic Head Injuries in Other Sports

For obvious reasons, football has received the most attention in the discussion of catastrophic athletic injury data collection. Until the formation of the National Center for Catastrophic Sports Injury Research in 1982, football was the only sport for which catastrophic injury data was collected. In terms of other sports, track and field and baseball stand out as being associated with the greatest number of catastrophic head injuries (Table 7). The pole vault was associated with most of the catastrophic track and field injuries and posed a serious concern after 3 deaths in 1983. In addition to the pole-vaulting accidents, a number of catastrophic head injuries involved participants being struck by a thrown discus, shot put, or javelin. Most of the baseball injuries have been associated with the head-first slide, player collisions, or being struck by a pitched or thrown ball.

Catastrophic injuries have not been limited to male participants. Cheerleading, gymnastics, softball, field hockey, and volleyball have all been associated with fatalities or permanently disabling injuries in female participants in recent years. Cheerleading accounted for more than 50% of catastrophic injuries to female participants over the past 17 years.

DISCUSSION

Recommendations for Prevention

Catastrophic head injuries may never be totally eliminated from sport, but with reliable injury data collection systems and constant attention to prevention measures, the frequency of these injuries can be dramatically reduced. Several factors are essential for preventing these injuries: (1) Preparticipation medical examinations and medical histories must be mandatory for participants at all levels of competition. (2) Every school and college that participates in athletics should hire an athletic trainer certified by the National Athletic Trainers'

Table 7. Catastrophic Head Injuries in Other High School and College Sports, 1982–1999*

Sport	No. of High School Participants	No. of College Participants	No. Resulting in Fatalities	No. Resulting in Disability	No. Resulting in Recovery
Track and field	850 000	55 000	10	8	11
Baseball	450 000	20 000	3	5	8
Soccer	550 000	23 000	2	1	4
Wrestling	230 000	7000	1	0	0
Swimming	200 000	16 000	0	1	0
Basketball	1 000 000	25 000	1	0	4
Gymnastics	25 000	2500	1	0	0
Softball	330 000	10 000	0	0	1
Ice hockey	30 000	4000	0	1	2
Cheerleading	75 000	NA	2	3	12
Total			20	19	42

*Participants include both male and female participants. NA indicates not available.

Association Board of Certification, and this individual should be a regular member of the school's faculty or organization. (3) Game rules must be strictly enforced by officials, coaches, and school officials. Coaches should know and be able to teach the proper fundamental skills of the sport. Players should be taught not to make initial contact with their head or face while blocking or tackling. (4) An athlete who has experienced or shown signs of head trauma (loss of consciousness, visual disturbance, headache, dizziness, obvious disorientation, memory loss) should receive immediate medical attention and should only return to participation after first being cleared by a physician or certified athletic trainer. (5) Athletes, parents, and coaches should be educated about the signs and symptoms associated with head injury, as well as the dangers of recurrent injury and second-impact syndrome. (6) The team's certified athletic trainer must always be prepared for a possible catastrophic head injury. Everyone involved must know what to do before the injury takes place. Being prepared and knowing what to do may be the difference that prevents permanent disability. A written emergency plan should be distributed to all relevant personnel and should include an evacuation plan, available transportation, portable and open communication, and game and practice schedule awareness in local hospital emergency departments. These recommendations will not prevent injuries from happening, but they will possibly prevent serious injuries from resulting in permanent disability.

CONCLUSIONS

Data from the National Center for Catastrophic Sports Injury Research have played a major role in helping to reduce the incidence of these catastrophic sport injuries. National governing bodies have used the data extensively to identify problem areas within their sports and to implement changes. Our continued collection of data on catastrophic injuries has also allowed these changes to be evaluated. Ongoing surveillance is important because sports are continuously evolving and changing, and inexperienced coaches continue to enter the coaching profession. Catastrophic injuries are rare, but the health care professional needs to be trained and prepared to deal with these potentially serious situations. To this end, every school should strive to have routine and regular access to the services of an athletic trainer certified by the National Athletic Trainers' Association Board of Certification.

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Principles of Liability for Athletic Trainers: Managing Sport-Related Concussion

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Objective: To provide an overview of the general legal principles of negligence for sports medicine professionals and apply these principles to situations involving athletes with head injury.

Data Sources: Case law dating back to 1976 and recent studies of sport-related concussion.

Summary: One of the most difficult problems facing athletic trainers and team physicians is the recognition and treatment of sport-related concussion. Providing medical clearance for sports participation and treatment of athletic injuries involves legal as well as medical issues. The threat of lawsuits exists for the sports medicine professional, whether the athlete is allowed to play or not. In general, established medical malpractice principles govern claims by athletes for injury or death caused by improper treatment by health care providers. The elements of negligence are examined, as well as the primary defenses an athletic trainer would use in court and risk management techniques to avoid litigation.

Conclusions/Recommendations: Athletic trainers may protect themselves from liability by including standardized cognitive or postural stability testing in preparticipation examinations, using objective tests rather than subjective judgement to evaluate athletes who have sport-related concussion, working closely with physicians, and keeping excellent records.

Key Words: negligence, duty, breach, causation, damage, reasonable person standard

One of the most difficult problems facing athletic trainers and team physicians is the recognition and treatment of sport-related concussion. Cerebral concussion involves a violent jarring or shaking of the brain caused by a sudden change in the momentum of the head. More than 300 000 sport-related traumatic brain or head injuries occur annually in the United States.[1] Moderate to high incidences of concussion have been reported in football, basketball, softball, soccer, baseball, boxing, rugby, and ice hockey.[1] Repeated head injury can result in permanent brain disability or death.[1]

Providing medical clearance for sports participation and treatment of athletic injuries involves legal as well as medical issues. The threat of lawsuits exists for the sports medicine professional whether the athlete is allowed to play or not. Athletic trainers and team physicians have been sued for prematurely clearing athletes, and surprisingly, have also been sued for not permitting athletes to play.[2–5]

Until recently, little was known about the medical consequences of head injury. This dearth of medical information has contributed to a lack of legal precedent, as only a few cases establish precedent when dealing with sport-related concussion. More cases dealing with negligence or medical malpractice involve a physician and an athlete; the medical condition generating the most media attention involves death as a result of a cardiac condition. The published cases involving severe head injuries are nearly all related to boxing, and most of those cases have been settled on issues unrelated to the actual diagnosis or treatment of the athlete.

In general, established medical malpractice principles govern claims by athletes for injury or death caused by improper treatment by health care providers.[6] In this paper, I provide an overview of the general legal principles of negligence for sports medicine professionals and apply these principles to situations involving athletes with head injury. Recommendations are made for athletic trainers and team physicians to protect themselves from liability.

NEGLIGENCE

In law, a tort is a private wrong or injury suffered by an individual as the result of another individual's conduct. The law provides injured individuals the right to be compensated through the recovery of damages. Torts may be intentional, meaning that the individual intended to act, or unintentional, in that the individual did not mentally intend to cause harm. Negligence is an unintentional tort.

Negligence law was founded on the principle that those who are harmed as the result of others' carelessness or failure to carry out responsibilities properly must be compensated. The person who was harmed has the burden of proving that the 4 legal elements of negligence are satisfied. The first element of negligence is to prove that there is a *duty* of care owed as a result of a relationship that exists between the parties. The second step is to prove that the defendant *breached* the duty owed to the injured party. Third, there must be proof that the breach of the duty is the *cause* of the harm to the plaintiff. Fourth, there must be *actual harm,* not just potential for harm to have occurred. All 4 elements of negligence must be proven in order for the plaintiff to be compensated by the defendant for damages.[7]

APPLICATION OF LEGAL PRINCIPLES TO ATHLETES WITH CONCUSSION

Duty

Although relatively few judicial opinions have concerned litigation between athletic trainers and competitive athletes, the courts have recognized that a duty exists between the parties.[7] The athletic trainer's responsibility, like that of the team physician, is to protect the health and safety of the athletes. In the case of an athletic trainer who is treating an athlete with a concussion, several legal duties exist as a result of the athletic trainer–athlete relationship. There are several possible recognized legal duties:

- Duty to properly assess the athlete's condition
- Duty to provide or obtain proper medical treatment
- Duty to provide clearance to participate
- Duty to inform the athlete of the risks of athletic participation given the particular medical condition

The case of *Pinson v State*[8] focused on the duty of the athletic trainer to a student-athlete. Michael Ray Pinson suffered a blow to the head during a collegiate football practice. He walked to the sidelines, said that he had been "kicked in the head," and collapsed unconscious. The athletic trainer examined Pinson while he was unconscious and noted a palsy on the left side of Pinson's face, an absence of control of the left side of his body, unequal pupils, and an absence of response to pain, sound, or movement. He also noted that Pinson remained unconscious for 10 minutes. The athletic trainer instructed a student athletic trainer to accompany Pinson to the hospital but failed to give instructions to forward information that should be given to the attending physician regarding the athlete's initial condition. The athletic trainer never spoke with the physician regarding the symptoms that he observed on the field after Pinson collapsed. Pinson was admitted to the hospital for observation. A skull radiograph was obtained, and all neurologic checks were normal. The attending physician telephoned the athletic trainer with instructions that Pinson should not participate in football practice for a week and that if he had any further symptoms, he should return to the hospital. The athletic trainer picked Pinson up from the hospital on his release, at which time Pinson reported that he had a headache. The next day, he complained of headaches again, and the athletic trainer gave him Empirin 4 (Glaxo Wellcome, Research Triangle Park, NC), a buffered aspirin. Pinson continued to complain to the athletic trainer that he had headaches for the next several days. One week after Pinson returned from the hospital, the athletic trainer called the team physician and reported that Pinson was asymptomatic for a concussion. The team physician did not examine Pinson and, relying on the report of the athletic trainer that Pinson was asymptomatic, cleared him to return to practice. Pinson practiced, traveled, and played in at least 2 games over the next 3 weeks. He complained of headaches, dizziness, nausea, and blurred vision throughout this time period, but the athletic trainer did not report any of the symptoms to either the team physician or the original attending physician. Exactly 1 month after his initial head injury, Pinson collapsed on the sideline during football practice. He was taken to the hospital and underwent brain surgery for a chronic subdural hematoma of several hundred cubic centimeters, an acute subdural hematoma of approximately 25 to 30 cubic centimeters, and a midline shift of almost 1.5 centimeters. Pinson remained in a coma for several weeks and suffered severe and permanent neurologic damage. At the time of the trial, Pinson was hemiparetic and had no use of his left arm and little use of his left leg. He had a shunt to drain excess fluid from his brain, suffered from severe cognitive problems, and experienced frequent seizures. The trial was conducted before a Tennessee Claims Commissioner, who held that the athletic trainer had a duty to report Pinson's initial neurologic signs and subsequent headaches to a medical physician.[8]

Breach

Whether a legal duty has been breached is assessed from the athletic trainer's adherence to accepted sports medicine practice. Known as the "reasonable person standard," an athletic trainer is expected to act as a reasonable athletic trainer would under the same or similar circumstance. The law recognizes that not all athletic trainers practice in the same settings, with equal access to resources, staff, facilities, or equipment, so the level of reasonable care changes according to the circumstances. Because an athletic trainer is a sports medicine professional, he or she would be held to the level of care that a reasonable sports medicine professional would be held to in the same situation.[9] This standard of care is usually established by expert testimony[9] based on national athletic training certification boards, standardized training programs, certification programs, and state licensing requirements. The certified athletic trainer must act with the skill and knowledge that is reasonable within the profession.

One of the difficulties of determining the legal standard of care for athletic trainers in dealing with athletes with concussion is the lack of a universally accepted standard for proper assessment and prescribed treatment of the injury. Several concussion grading scales[10–13] and return-to-play guidelines have been proposed in the literature; however, none has emerged as a "gold standard." Most scales use concussive symptoms to grade the injury severity, which determines how long an athlete should be kept from returning to play. These concussive symptoms include memory loss, dizziness, headache, difficulty concentrating, amnesia, nausea, ringing in the ears, visual problems, aphasia, eye twitches, and dysequilibrium.

One of the primary criticisms of the grading scales is that the grading is often based solely on loss of consciousness and amnesia, when most concussions involve neither of these symptoms.[1] Another problem is that the scales rely heavily on anecdotal clinical evidence and limited scientific data. Symptoms are measured based on the athlete's input, and athletes often underreport symptoms because of a strong desire to return to play. Having said this, the proposed grading scales are considered very safe by most of the sports medicine community.

Although sports medicine researchers do not universally endorse any specific grading scale, the scales do provide legal guidelines for a reasonable standard of care to be used by athletic trainers. However, research indicates that most athletic trainers do not even use these guidelines when assessing athletes' head injuries.[1] From a legal perspective, it is important to note that the athletic trainer's actions are not being compared with what the average athletic trainer would do in the same or similar circumstance. What an average athletic trainer does is sometimes difficult to determine, may be unduly deferential to an older or outdated procedure, and is unlikely to produce optimal

sports medicine care.[3] Just because most athletic trainers evaluate athletes' head injuries on a hunch does not mean that it is legally reasonable to do so. A recent study[1] indicated that almost one third of football players who experienced concussion were held out of play for only 14 minutes, although the grading scales generally recommend that an athlete who has suffered a minor head injury remain on the sidelines for at least 20 minutes after the injury before an evaluation is made about return to play. It could be argued that, based on the scientific knowledge available, this behavior is certainly not reasonable. A jury may consider this behavior even less reasonable if evidence is presented that 14% of these athletes suffered a grade 2 concussion according to the Cantu scale.[14] Recommendations for treating a grade 2 concussion would require an athlete without symptoms to rest for at least 1 week, which is far longer than 14 minutes!

Generally, 3 serious conditions can occur after an athlete has suffered a head injury: subdural hematoma, second-impact syndrome, and chronic postconcussion syndrome. Subdural hematoma is the most common cause of death in athletes.[13] It can occur after even a mild head injury and may develop slowly over several days to a week. Second-impact syndrome is characterized by brain swelling and intracranial pressure that occur when an athlete sustains a second concussion while still symptomatic from an initial head injury. Although rare, this second injury almost always results in permanent brain injury or death. Athletes with chronic postconcussion syndrome may experience blurred vision, headaches, and lack of concentration and balance for months after the initial head injury. Because of the seriousness of these conditions, sports medicine researchers have sought more objective means to assess the athlete's condition after head injury and make reasonable decisions regarding when it is safe for that athlete to return to play.

Although the sports medicine community has not come to a consensus, 2 methods of objective testing may satisfy the reasonableness test: neuropsychological testing and postural stability testing. Neuropsychological testing measures the athlete's cognitive flexibility, attention span, orientation, concentration, visual-spatial capacity, distractibility, immediate memory recall, and problem-solving abilities.[15] These tests directly measure the cognitive qualities that are affected by head injury and allow athletic trainers to objectively evaluate the athlete's condition.[15] Although administration of these tests has generally occurred in a clinical setting, recent research indicates that athletic trainers can also administer neuropsychological tests on the sidelines and achieve valid results.[16,17] The National Football League and National Hockey League currently use neuropsychological testing to assess professional athletes' cognitive abilities, establishing that it is reasonable to employ these tests as a standard for assessing, treating, and making return-to-play decisions.

Similarly, researchers have established that postural stability tests are reasonable to use in determining when symptoms of concussion cease.[18,19] These objective tests use sophisticated forceplate systems to challenge sensory systems involved in balance by altering visual and support surface conditions.[18] Although it may not be reasonable to expect the average athletic trainer to have access to this type of equipment, research indicates that there is a significant correlation between the results of simple tests that the athletic trainer can conduct on the sideline and the results of sophisticated postural stability tests.[19] The Balance Error Scoring System[19] is another user-friendly, cost-effective objective testing method. Athletic trainers can objectively measure the athlete's performance on the single-leg, double-leg, and tandem stances on firm and foam surfaces on the sidelines without needing computerized equipment to determine if there are any lasting effects of the head injury. The reasonableness standard related to breach of duty is measured "under the same or similar circumstances."[9] Because athletic trainers must make field decisions about whether the athlete should be allowed to play after experiencing a head injury, a jury could find that it is reasonable to expect an athletic trainer to perform simple sideline tests as a standard for assessing the athlete, treating the athlete, and making those decisions.

Perhaps one of the reasons that athletic trainers have relied so heavily on subjective measures and personal intuition in evaluating an athlete with a head injury is that they have nothing for comparison. Athletic trainers and team physicians routinely conduct preparticipation examinations to determine if an athlete has a condition that would preclude participation in sports. Although reported legal decisions provide little guidance regarding the appropriate nature and scope of a standard preparticipation examination, many of the filed lawsuits allege that the sports medicine professional did not discover a medical condition that later resulted in injury or death.[3] Case law indicates that physicians who conduct a thorough preparticipation examination in conformity with accepted standards of practice are not found to be liable for the athlete's injuries that occurred postexamination.[20,21] It is foreseeable that athletes who compete in contact sports may experience head injury; therefore, including neuropsychological and postural stability testing in preparticipation examinations seems reasonable. These tests provide athletic trainers with objective baseline data, providing a basis for comparison of cognitive function while also taking into account the individual differences of each athlete. In *Speed v State,*[22] a physician was found negligent in failing to order appropriate tests necessary to diagnose the nature of an athlete's condition. Similarly, an athletic trainer or team physician who fails to use prescribed subjective tests to assess the severity of head injury may also be negligent.

Deciding when an athlete who has suffered a concussion can safely return to play is one of the greatest challenges facing athletic trainers and team physicians. Sports medicine professionals must consider the intensity and physical demands of the athlete's sport, all objective clinical evidence, and the probability and severity of harm from athletic participation given the athlete's condition. Although the court decided the case of *Classen v Izquierdo*[23] on other grounds, the opinion indicated that a physician has a duty to conform to good and accepted standards of medical care in determining whether an athlete should continue participating in a sport. In this case, a ringside physician refused to stop a boxing match in which a participant received several blows to the head. The boxer ultimately died from the multiple head injuries he sustained, and the court indicated in the opinion that the failure of the physician to keep the athlete from competing may have constituted malpractice. In the case of an athlete with a head injury, there is uncertain potential for permanent disability or death. Given the extreme risks, it seems reasonable to err on the side of caution.[24]

When the decision has been made to let the athlete return to play after head injury, the athletic trainer or team physician has a duty to fully disclose information about the athlete's

medical condition to the athlete.[6] Failure to provide an athlete with full disclosure of material information about playing a sport with a medical condition or the potential consequences creates liability for negligence.[25] This duty to disclose relevant information relates to the issue of informed consent.

Informed consent is technically a defense for the intentional torts of assault and battery, but modern courts have translated this concept into negligence terminology.[26] As a legal principle, informed consent comes from the public policy that a competent adult has the legal right to determine what to do with his or her body. As such, adults may provide consent, but minors require consent by a parent or guardian.[27] To satisfy legal requirements, consent must represent an informed decision regarding the risks of treatment and participation. For an athlete's decision to be informed, the sports medicine professional must clearly warn of all material, short-term, and long-term medical risks of continued athletic participation under the circumstances. Material information is defined in *Canterbury v Spence*[25]: "a risk is thus material when a reasonable person, in what the physician knows or should know to be the patient's position, would be likely to attach significance to the risk or cluster of risks in deciding whether or not to forego the proposed therapy." The athletic trainer must explain all of the potential risks in plain and simple language that the athlete can comprehend.[28] Unless the medical risks are fully explained, the athlete has a claim against the athletic trainer if he or she can prove that he or she would not have played if informed of the material risks of doing so.[29]

Causation

In a legal case of negligence, the athlete must prove by a preponderance of the evidence that the breach was in fact the legal cause of the injury.[30] Actual cause is established if the athlete can prove that the athletic trainer's actions were a considerable determining factor in the damage claimed. When treating an athlete with a concussion, actual cause can be an act, such as the act of clearing an athlete to participate, or an omission of an act, such as a failure to conduct reasonable objective tests to assess the athlete's condition. If the athlete cannot prove actual cause, he or she must prove proximate cause. Proximate cause occurs when the action of the athletic trainer foreseeably leads to harm or injury to the athlete. Athletic trainers and team physicians can share liability if more than 1 person, other than the athlete, contributed to the injury.[7]

Pinson v State[8] also addressed the issue of causation. The athletic trainer argued that his failure to report Pinson's headaches and other symptoms to the team physician was not the cause of Pinson's injuries. In this case, the court determined that the failure to report this information was a substantial factor in bringing about the permanent damage Pinson suffered because it was foreseeable that Pinson's first injury would have been properly diagnosed and treated if the athletic trainer had reported the symptoms. Additionally, had the athletic trainer reported Pinson's headaches and other symptoms to a physician after his release from the hospital, additional tests such as a neurologic consultation and a computed tomography scan would have been ordered. The court concluded that but for the athletic trainer's failure to do his duty, Pinson would likely have had little or no permanent neurologic deficit.

Damage

The fourth element of negligence is damage, and the athlete has the burden of proving that actual damage exists. In a negligence action, the injured party typically seeks damages in any or all of the following areas: past, present, and future pain and suffering; past, present, and future medical expenses; and past, present, and future diminution of earning capacity.[31] In *Pinson v State,*[8] the court awarded Pinson $1.5 million dollars in damages, including $450 000 from the athletic trainer.

DEFENSES AGAINST NEGLIGENCE AND RISK MANAGEMENT

Working in an environment in which both the medical standards and legal requirements are uncertain, the athletic trainer or team physician should understand the potential defenses against negligence claims. The most complete defense is to prove that 1 of the 4 elements of negligence is not proven by a preponderance of the evidence. In *Pinson v State,*[8] the athletic trainer attempted unsuccessfully to prove that 3 of the elements were not present: (1) that he did not owe a duty to disclose information to the physician, (2) that he did not breach his duty because he acted as a reasonable athletic trainer under the circumstances, and (3) that his actions (omission) did not cause Pinson's injuries.

Another defense commonly used in athlete injury cases is the assumption of risk doctrine. An athlete can legally assume the risk of harm by opting to play with a known medical condition or injury, thereby removing the liability of the athletic trainer. Assumption of risk is based on the legal principle that no harm is done to one who consents. Because this is a defense, the athletic trainer has the burden to prove the following points:

- The athlete knew of the risk (or that a reasonable athlete should have known)
- The athlete agreed to accept the risk either expressly (orally or in writing) or by implied consent (participating)

Competitive athletes are often willing to assume health risks to engage in sport. For legal purposes, a competitive athlete is defined as one who participates in an organized team or individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires vigorous training in a systematic fashion.[3] Competitive athletes may exist at the youth, interscholastic, intercollegiate, and professional or master's levels of sports. These athletes accept some risk of injury just by engaging in competitive sports. When bodies collide, it is foreseeable that muscles may be bruised, connective tissue may be strained or torn, bones may be broken, and catastrophic injury or death may even occur. Athletes may be internally compelled to play by their love of the game, a need for affiliation, or the pursuit of excellence or prestige. They may want to play because of external forces such as peer pressure, living up to the expectations of coaches or parents, or potential economic gain. Whatever the reasons, athletes are often willing to play regardless of the health consequences.

The athletic trainer or team physician cannot abdicate his or her duty because the athlete wants to play. The duty of the sports medicine professional is to always protect the health and safety of the athlete. The athletic trainer must make participation decisions based on the best objective data available. Tremendous pressure may be placed on the athletic trainer to return the athlete to play as soon as possible by the coaching staff, administrators, other team members, alumnae and fans,

Common Sense Risk Management Techniques

Build relationships
A little goes a long way; athletes and their parents are much less likely to sue an athletic trainer who they think truly cares about them.

Obtain a written contract
Insist on a written contract that includes a detailed job description. This provides documentation as to the scope of the athletic trainer's employment.

Obtain a preparticipation examination
Neuropsychological or postural stability testing (or both) are recommended to establish a basis for comparison should head injury occur.

Obtain informed consent
Informed consent is a contract in which the participant acknowledges acceptance of the risks of engaging in the activity in exchange for the opportunity to participate. Written documentation reduces the chance of litigation. Note that informed consent contracts provide minimal protection against suits by those participants under 18 years of age, as parents may not sign away the rights of a minor, and minors may not legally enter into contracts. Obtain approval by legal counsel for proper language and structure.

Keep records
An accurate paper trail provides documentation that the athletic trainer satisfies the "reasonable person" standard. Medical records should report only facts and not the sports medicine professional's opinions. These documents are always admissible as evidence in a trial, whereas opinions are not necessarily admissible. However, if the opinion is included in the medical record, then the opinion is admitted. Remember to maintain the confidentiality of the athlete's medical record.

Participate in continuing education
This risk management policy is tied to the standard of care (ie, what a reasonable athletic trainer knew or should have known). The field of sports medicine is continually evolving, and athletic trainers or team physicians cannot rely on the fact that something has "always" been done a certain way.

and even the athlete. The athletic trainer cannot be influenced by the team's need for the player or even by the athlete's desire to play. Even if the athlete begs to be allowed to play, the athlete does not assume the risk of the athletic trainer's negligence.[30]

Another legal caveat is that the athlete must know and appreciate the specific risk of harm of participating. It is very important to note that an athlete with a head injury may be physically unable to appreciate the health risks of playing after a head injury. Does an athlete with a brain injury have the legal capacity to make this type of decision? To preserve this defense as an option, the athletic trainer could carefully assess the athlete's cognitive function to determine whether the athlete has the capacity to understand the risks. Athletic trainers and team physicians should always inform participants either orally or, preferably, in writing of the risks involved in the activity. For special events or one-time activities, the sports medicine professional may create a contract that fully describes the risks in plain and simple language. Words in the contract expressly state that by signing the document, the participant acknowledges the risks and accepts them.

Another defense related to assumption of risk is the doctrine of contributory negligence. If the athlete in any way contributed to his or her own harm, the sports medicine profession is not liable. As a defense, the athletic trainer or team physician bears the burden of proving that the plaintiff was in some way responsible for the injuries incurred. An athlete may be contributorily negligent if he or she voluntarily takes part in a sport and the decision to participate is unreasonable, or if the athlete deliberately disregards a warning or instruction not to participate. In *Jarreau v Orleans Parish School Board,*[28] a high school football player was found contributorily negligent for continuing to play while injured. The athlete may also be found to have contributed to his or her own harm by lying about a condition to the sports medicine professional. Contributory negligence is a complete defense, so the sports medicine professional who can prove that the athlete contributed to his or her harm in any way, no matter how small, is not liable for any damages.

As a public policy, it seems unfair that a professional who is negligent is freed from the obligation of paying damages because the plaintiff in some way contributed to the injury. Many states no longer allow assumption of risk or contributory negligence as a complete defense but mitigate damages by comparative negligence principles. Comparative negligence recognizes that damages should be paid by the one who caused the harm, but damages are decreased in proportion to the degree of damage contributed by the athlete. Instead of the plaintiff's receiving nothing because he or she contributed in part to the injury, the athlete recovers a lesser amount.

Many of the decisions in medical malpractice cases that were in favor of the sports medicine professional were not decided on the merits of whether the athletic trainer or team physician acted prudently but rather because the claim was dismissed based on the doctrine of governmental immunity.[10,20,32] In *Lennon v Peterson,*[32] a complaint against the athletic trainer at a public university was dismissed. Governmental immunity is granted to the state, preventing legal action for damages against the government and its political subdivisions. Public educational institutions are covered, but private institutions are not. Governmental immunity is not a total defense in most states but generally caps the amount that the government would be required to pay if a state actor is negligent. Governmental immunity statutes generally protect state employees who act within the scope of their employment but will not protect against acts of gross negligence, recklessness, or intentional torts. The case of *Gardner v Holifield*[10] was dismissed because the physician was the director of a public university's student health center and was acting within the scope of his employment under the state immunity statute.

Risk management procedures will not help sports medicine professionals defend themselves in a lawsuit but should help prevent litigious situations from occurring.

To develop a comprehensive risk management program, sports medicine professionals must take several actions:

- Identify the risks present in the program
- Estimate the extent of the risks, taking into consideration the seriousness of the injuries that may occur and the likelihood that the injury will occur
- Evaluate the options that could be taken to reduce risk
- Implement the risk reduction policies and procedures

As risk management procedures are established, it is important that whatever is done is measured against the standard of care that a reasonably prudent professional would give in the same or similar circumstance. Suggestions for risk management techniques are included in the Table.

CONCLUSIONS

Many sports medicine issues relating to head injury are medically and legally unresolved. Adherence to outdated sports medicine guidelines should not be a recognized defense. Standards should be updated and modified periodically as the practice of sports medicine evolves to promote the health and safety of athletes. Giving legal effect only to guidelines consistent with the medical state of the art provides an incentive to medical organizations to revise the guidelines to stay current with advances in sports medicine research.

It is imperative for sports medicine researchers to establish an evidence-based medical guideline for making return-to-play decisions for athletes after concussion. This standard would inform athletic trainers and other sports medicine professionals as to what the law expects of them and would prevent retrospective second guessing by lay jurors as to whether the practitioner's conduct was reasonable. Until a legal standard of care is established, athletic trainers and team physicians must work together to safeguard the athlete's health after head injury by relying on objective testing as well as subjective measures to evaluate athletes in return-to-play situations.

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Performance Testing Updates in Head, Face, and Eye Protection

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Objective: To describe the evolution and implementation of standards for head, face, and eye protection in sports.

Background: Recent changes in testing standards for head, face, and eye protection include the development of new equipment, the mandating of tougher standards, and the coverage of additional products by these standards, all in an effort to improve athletes' safety and reduce their risk of injury. The person selecting equipment needs to understand these standards, how they are developed for each piece of equipment, and which standards the piece of equipment is purported to meet.

Conclusions/Recommendations: The sports medicine clinician must recommend only the use of personal protective equipment that meets a current standard; must ensure that the equipment is maintained in its original form and that all parts and labels are present; and must ascertain that equipment is refurbished by a qualified reconditioner. By following these guidelines, we improve sport safety for our athletes and lessen their risk of injury.

Key Words: helmet, headgear, face protector, NOCSAE, ASTM, eyewear

Over the last several years, a number of changes have occurred with regard to the standards for testing headgear and eyewear. New equipment has been developed and used, tougher requirements mandated, and additional products covered by standards. Consequently, today's marketplace provides a much wider variety of athletic equipment than was available 20 years ago. Some of today's equipment might meet only one of the current standards, whereas other equipment might meet multiple standards. Unfortunately some equipment in the sports arena today might meet no standard! Most importantly, the person selecting equipment today needs to understand what these standards are, how they are written to ensure specific characteristics in a piece of equipment, and which standards a particular piece of equipment is purported to meet. It is also essential to note that, in many instances, only the manufacturer claims that the product meets a standard. With few exceptions, such as the Consumer Product Safety Commission (CPSC), the Snell Memorial Foundation (SNELL), the Safety Equipment Institute (SEI), and the Hockey Equipment Certification Council (HECC), no independent body is checking on the manufacturers to be sure that their claims are factual.

STANDARDS-SETTING PROCESS

Different types of helmets and eyewear are tested to different standards established by different standards-setting bodies. These standards, as well as those for most other protective equipment in North America, can usually be described as falling into one of several gross categories. Government standards, such as those mandated by the CPSC, and voluntary, or industry, standards are the most common. Among the voluntary standards, those mandated by a particular governing body seem to be the most effective. For example, if a piece of personal protective equipment is required for some type of play, such as an HECC-certified helmet is required by USA Hockey, then players will likely wear such equipment because they cannot participate without doing so. Governing bodies, however, rarely write or create such standards because that is not in their area of expertise. Rather, they rely upon other organizations, such as the American Society for Testing and Materials (ASTM) or the National Operating Committee on Standards for Athletic Equipment (NOCSAE) to arrive at a viable and effective standard. The ASTM is a premier standards-setting organization with more than 30 000 members that is well respected for its consensus standards. Other organizations, such as NOCSAE, operate using a smaller format. This more limited group is capable of setting standards much more quickly than the consensus approach allows. It could be argued that this second approach involves less input from concerned parties, but a "public" review period before actual implementation eliminates most of this criticism.

Governing bodies such as the National Collegiate Athletic Association (NCAA) and the National Federation of State High School Associations (NFHSA) can and do mandate that certain equipment worn or used by players in their programs meet a particular standard. In the case of helmets (football, baseball batter, and lacrosse), these governing bodies require that the helmets meet NOCSAE standards, and further, that all participants playing within the control of the governing body wear equipment clearly identified as being in compliance. In the preceding example, helmets must meet the NOCSAE stan-

dard for that particular activity and must clearly display the appropriate NOCSAE logo certifying compliance.

EVOLUTION OF STANDARDS

Early headgear standards were typically a force-transmission, measurement-based protocol. In this test, the headgear was usually mounted to a rigid (or semirigid) headform. A standard, malleable material (often a specific grade of aluminum) was placed between the headform and the inner surface of the helmet. An indenter of some type (usually a ball bearing) was placed so as to indent the malleable material on impact. Typically, a falling object—frequently a heavy block of wood with a specified mass and surface area—generated the impact energy. After impact, the depth of the dent was measured and used to determine the helmet's effectiveness. One example of this method is an early Canadian Standards Association (CSA) standard for hockey headgear issued in 1975 and revised in 1983.[1] Although these methods are not without merit, the injury-producing accelerations resulting from head impact were not measured.

Today's headgear standards attempt to measure these accelerations and set performance limits for that headgear. Two basic approaches to this type of testing exist. One method uses anthropometric (humanlike) headforms, triaxial acceleration transducers, and a mathematical integration to measure helmet energy attenuation (NOCSAE). The other approach uses a simple, skull-shaped metallic headform with a single-axis accelerometer to provide acceleration data (ASTM, SNELL, CPSC).

In addition to creating new standards, the standards-setting bodies are constantly reviewing standards with an eye toward making them more stringent but still practical to meet. As an example, in 1993, NOCSAE commissioned research to improve the consistency of test results obtained with the NOCSAE Drop System.[2] This was ultimately accomplished with several modifications, including changing the type of guide wires used by the system to reduce friction, improving the steps to calibrate the NOCSAE headform, and improving the carriage that holds the test helmet for impact.[3,4] At the same time, NOCSAE tightened the pass-fail criterion by 20% (even though the revised method resulted in up to a 5% impact energy gain) because it was believed that manufacturers could produce helmets that met this tougher standard.[5] Since that time, NOCSAE has continued standards improvement with the publication of important document revisions in 1998,[6] and further revisions are currently under review. NOCSAE testing methods[7] and performance requirements for batters' and catchers'[8] headgear have followed a similar evolution. NOCSAE standards for baseballs and softballs are currently in the review phase, with publication of new criteria anticipated in late 2001.

The NOCSAE test equipment includes an anthropometric headform that is very humanoid in its mechanical properties and appearance compared with other headforms used for helmet testing; this headform was first developed in the 1970s. The headforms used with other types of standards (ASTM, CPSC, etc) are typically a solid piece of metal cast or milled to look like a portion of the human head and skull. In 1999, NOCSAE decided to gather more comprehensive anthropometric data and to redesign the NOCSAE headforms to make them even more humanoid than the earlier version. This work is nearing completion at the time of this writing, with some of the new headforms being beta tested at several sites. Along with improved facial anthropometry (a key to better testing of eye and facial protective products), increased biofidelity with decreased frangibility are the goals of these changes.

Similarly, ASTM standards are constantly undergoing modification. For example, the ASTM test method for testing many types of headgear has undergone at least 7 important revisions in the last decade.[9] ASTM standards for hockey headgear,[10] baseball face protectors,[11] football helmets,[12,13] and equipment for several other sports have also undergone revision to improve either test repeatability or product performance.

DETAILS OF KEY HEADGEAR STANDARDS

Both the current NOCSAE and ASTM headgear standards are based on the helmet's falling in a guided free fall. In each case, the helmet is positioned on a headform instrumented to provide a measure of the helmet's ability to attenuate the kinetic energy imparted during the test. The energy is based on the drop velocity and the mass of the headform. In each test procedure, the drop mass and velocity are similar but vary with the type of headgear being tested. In some cases, the impact surface is different to allow testing of specific potential hazards that a particular type of headgear might encounter when in use. For example, in addition to falling onto other anvils, bicycling helmets fall onto a curbstone anvil that is shaped very much like a section of street curbing, as the name implies.[14] The measurement is usually plotted as acceleration in relation to time.

Both approaches measure linear acceleration only. Both standards require subject helmets to be "conditioned" (hot or cold, wet, etc) during some impacts. Conditioning environments are also variable depending on the type of headgear being tested. Despite these similarities, the tests have important differences. The most basic are outlined subsequently.

National Operating Committee on Standards for Athletic Equipment

From a technical standpoint, the NOCSAE standard is unique. The headform is anthropometric and complex in function, which allows for a wider area of impact locations, as the head is complete in its shape. Because of this feature, some impacts are not aligned exactly with the center of gravity (CG) of the system. In addition, data are gathered via a triaxial accelerometer to compensate for these geometric complexities. This method is similar to the way in which one would instrument a cadaver head for impact testing, using all 3 data channels and summing the signals, then using the square root of the summed squares for the resultant. The headform, like a human head, attenuates a large amount of energy during the test. The results are reported in severity index (SI) units, in which $SI = \int_0^t a^{2.5} dt$. The standard requires a series of impacts in specific locations and then the application of impacts to a random location, which can be any location within the protective area of the helmet. NOCSAE standards call for aggressive quality control measures from manufacturers claiming certification to the standard. These quality assurance requirements were clarified further in the 1996 and later revisions of the standards. Manufacturers claiming such certification must submit their testing facility to routine audits. NOCSAE does not police manufacturers for compliance but reviews laboratory competence and offers technical assistance to licensees. Football equipment (uniforms, helmets pads, etc) is typically re-

conditioned at the end of every season. The reconditioner then recertifies that the reconditioned football helmets meet the NOCSAE standard. In 1997, NOCSAE created highly specialized software for recertifiers in an effort to continue quality improvement. A second generation of this software is currently being developed for introduction in 2001.

American Society for Testing and Materials

The ASTM Committee F08 on Sports and Recreation establishes the ASTM Standards for Headgear used in sports; subcommittee F08.53 for headgear leads this effort. The ASTM criterion for most headgear uses the International Standards Organization (ISO) headform as the basis for the testing. This headform is typically made of a magnesium alloy with an outer shape similar to that of the skull. The headform comprises only a partial head, as it does not reach much below the basic plane (an imaginary plane drawn from the lowest part of the eye socket through the ear hole). Therefore, some impacts along the lower edge of a helmet's protective area may not be accurately tested. The headform has no biofidelity beyond its skull-like shape. Data are acquired via a uniaxial accelerometer mounted at the CG of the headform. Since all impacts are on the CG, only a single-channel accelerometer is needed.

Data output is usually provided in peak g levels, such that $A_{max} < N$ (maximum acceleration allowed is less than some N, which is usually 300 *g*). The pass-fail criterion may vary with headgear type but is normally 300 *g* or below. The standard closely relates to most other (non-NOCSAE) types of helmet testing protocols in use around the world today.

APPLICATION OF THE HEADGEAR STANDARDS

Both the ASTM and NOCSAE standards have a useful application. The ASTM standard seems a good choice for quality control and process control work, as it is highly repeatable. The NOCSAE standard is perhaps better suited to research, development, and the potential prediction of serious injury onset. When each organization has a standard published on a particular type of headgear, it is recommended that those responsible for selecting headgear verify that the product or brand selected meets both requirements, as they are not typically mutually inclusive.

EFFECT OF HEADGEAR STANDARDS

Cantu and Cantu[15] reported the incidence of deaths resulting from subdural hematoma in football for each decade in the period of 1945–1994. Incidence rates can be derived for each of those decades by relying on the data provided by the National Federation of High Schools and the NCAA as reported by Dr Frederick O. Mueller. While much of the injury reduction can be attributed to rule changes, improved coaching techniques, and better medical care, the positive effect on head injury reduction can be attributed in large part to the establishment of testing standards for headgear.

MECHANISMS OF HEAD INJURIES

Rather than attempt to review the available data on head injury mechanisms, I provide a brief description to serve as a simple foundation for understanding the rudiments of the primary head injury mechanisms seen in sports. Basically, there are 2 mechanisms: linear acceleration and rotational acceleration.

Linear Acceleration

As the name implies, linear acceleration is acceleration along a line and is the acceleration typically used for helmet testing at this time. It is reasonably well understood, and helmets in use today seem to tolerate very well the linear accelerations to which they are subjected in the standard test methods. Threshold limit values have been established, and helmets that meet those values have worked well in the field.

Rotational Acceleration

As the name implies, rotational, or angular, acceleration is nonlinear and can have many vector components. The injuries caused by angular acceleration are not well understood. Many of the remaining head injuries that occur on the field today may have rotational acceleration as the primary injury mechanism. Threshold limit values have not been established, although many levels have been suggested, and some data exist; no recognized test methods are in use. Helmets would not prevent these injuries, as they can occur without any head impact.

Combined Mechanisms

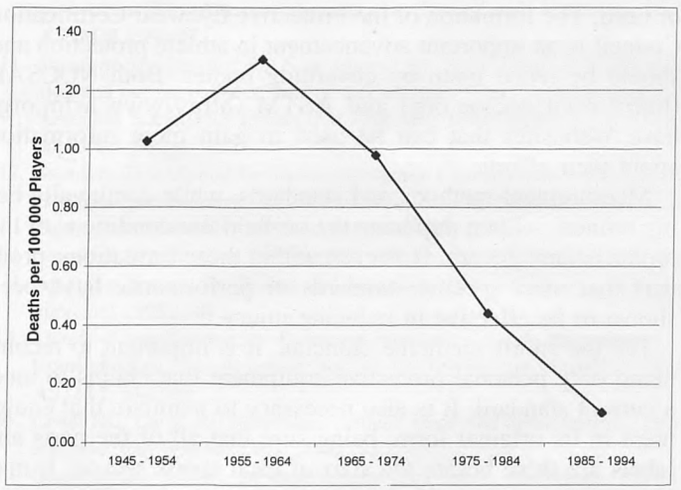
Most injuries on the playing field today are likely the result of combinations of linear and rotational acceleration, or they may even involve some unknown mechanics. Helmets may contribute to the management of these types of head injuries to some degree by managing linear accelerations and not unnecessarily exacerbating other motions. New test methods are under development that may allow products to be tested for both forms of acceleration simultaneously. It should also be noted that concussion is not addressed in any standard to date. Concussion is the threshold injury involving the brain. Ongoing research suggests several potential actions that may be implicated in concussion. Any investigation into that area requires an understanding of these mechanics on a molecular level. For our purposes, the basic mechanics involve tissue stretch.[16–18] This type of tissue strain may not be preventable by headgear, as it is not necessary to receive a blow to the head to incur such injury. Work is under way on a device to measure the combined effects of head impact in both linear and rotational acceleration, which may allow standards to one day soon address concussion. It appears that SIs as low as 250 and rotational accelerations of 4500 to 9000 $rad \cdot s^{-2}$ may be potential thresholds. A combined system of measure expressed as power with limits in the area of 12.8 kW may also be of value.[19]

Over the years, the decline in football players' deaths due to subdural hematomas has been significant and sustained (Figure). Although we might like to attribute this decline totally to improved equipment that met the new standards and changes in rules, as they surely had some effect, perhaps the most likely cause for this trend is improved medical treatment.

STANDARDS FOR EYEWEAR, GOGGLES, AND FACE PROTECTORS

In the area of eye and facial protection standards for athletes in nonmotorized sports, the development of standards has fol-

	1945 - 1954	1955 - 1964	1965 - 1974	1975 - 1984	1985 - 1994
Deaths	70	89	118	60	15
Incidence	1.03	1.31	0.98	0.44	0.10



Incidence of deaths in football due to subdural hematomas between 1945 and 1994.[15,28]

lowed a similar evolution. One important development was the American National Standards Institute (ANSI) Z87.1 standard for eye and face protectors.[20] This standard (and later versions) is likely to be the one used most often in industrial safety eyewear. Although this is often viewed as a good standard for nonindustrial applications (such as sports), more appropriate standards, including ASTM F 803-97,[21] are much more demanding and preferable for sports protection. For example, although you may trust your safety glasses in the hobby shop (where an avoided injury perhaps demonstrated the value of protective eyewear), those same safety glasses would be a poor choice for racquetball. The mass and energy of most sport-related, impact-based ocular threats are significantly higher than those specified by the typical industrial impact requirements.

The Protective Eyewear Certification Council certifies many types of sports eyewear, and its logo is an indicator that the selected eyewear is equal to the particular task. The council is a relatively new organization, very similar in concept to the HECC. Protective eyewear performance capabilities can be very deceiving, which is why certification to a standard is important. In addition to the obvious impact protective requirements, most eyewear standards include specific optical requirements. When using protective eyewear not in compliance with these optical requirements, the wearer may experience unanticipated visual distortion or interruption, vertigo, and other visual distractions that increase the potential for injury.

Although NOCSAE has no eyewear standards per se, NOCSAE standards exist for face protectors used by football players, baseball catchers, and lacrosse athletes. While these are also very demanding standards, devices that meet these requirements may not prevent objects such as fingers from entering the eye. If an athlete is playing with an eye injury or limited vision in one eye, the clinician should consult with the treating ophthalmologist to develop a protective plan that is likely to include eye protection that meets ASTM F 803 and a face protector that meets NOCSAE standards. The visors that some players attach to football face protectors are not covered by any standards at this time, although reputable manufacturers make several of these add-on visors and test them to their own internal performance requirements. Although faceguard-mounted visors offer some benefit, particularly when vision is already compromised, these devices do not offer the protective capability of the combat-style goggle eyewear that meets ASTM F 803.

In addition to the optical requirements, sports protective eyewear and facial protectors are also subjected to dynamic impact testing. This testing can include high-velocity impacts from hockey pucks[22] and baseballs[11] and low-velocity, high-mass impacts generated in football.[23] Unlike the previously discussed helmet testing protocols, the impact testing done for facial and eye protection products is typically limited to a contact–no contact, pass-fail criterion. Although sophisticated electronic methods can be used to determine contact, simple methods (such as a paste transfer) have long been used with good results. In the case of facial protectors that are not clear or see through (ie, wire-based product or molded gridlike components), optical clarity requirements may not be used. However, other testing to rule out the presence of scotoma (blind spots), particularly bilateral scotoma, may be used. This testing helps ensure that the wearer's field of view is not blocked in both eyes simultaneously.

Face protectors are often supplied with varying levels of coverage. Many styles of football faceguards are available, but most are variations on 1 of 3 basic types: an oral protector that covers only the mouth, leaving the nose and lower jaw exposed; a jaw protector that covers the mouth down to the jaw; and a nose protector with a bar running vertically down the middle to protect the nose. Obviously, the more facial area covered, the more protection the player is afforded. Nowhere is this effect more pronounced than in ice hockey, in which full-face protectors have a 100% success rate in preventing eye loss. Half shields, or visors, while better than nothing, can still allow enough penetration to cause a blinding injury. Recent and ongoing activities include standards for face and eye protectors used in paintball sports, as well as specialty additions to ASTM F 803-97.

EFFECT OF EYEWEAR STANDARDS

Pashby[24] identified how eye injuries in hockey were reduced in youths by requiring them to wear face protectors. The incidence of eye injury in Canadian amateur hockey players declined sharply, from 257 cases in the 1974–1975 season to 124 cases in the 1983–1984 season, since face protectors certified by the CSA and attached to CSA-certified helmets became mandatory for minor hockey players in this country. None of the reported cases in 1983–1984 occurred in players wearing such equipment. The average age of the injured players was 14 years in 1974–1975 but 24 years in 1983–1984; that is, younger players were now protected, but older players were not. Blows from hockey sticks and pucks were the top causes of eye injury in both seasons, but hyphema became the most common type of injury in 1983–1984, surpassing soft tissue damage. The frequency of legal blindness as the visual outcome fell from 19% in 1974–1975 to 11% in 1983–1984.

STANDARDS IMPLEMENTATION

When a standards-setting body establishes a new standard for a particular piece of athletic equipment, that action by itself does not mean that available equipment (or equipment made

available after the standard's effective date) will meet the new standard. Usually, reputable manufacturers honor the new standard and produce products in compliance with the standard, even if no governing body has yet mandated the use of equipment that meets that standard. In many cases, products are self-certified to meet the appropriate standards. Typically, the manufacturer's testing of the product in its own facility accomplishes this objective. In many cases, the manufacturer will, from time to time, use an outside independent laboratory to verify certification. Although this sounds like the honor system, and to a degree it is, competitive climates and possible litigation usually cause the self-certifier to be vigilant. Later, a governing body (NCAA, NFHSA, etc) may eventually mandate that athletes wear only products that meet the new standard, but this all takes time. As an example, NOCSAE introduced a standard for baseball and softball catcher's helmets[25] effective January 1, 1999. The NFHSA plans on making the use of a headgear certified to meet the NOCSAE catcher's helmet standard mandatory on January 1, 2003; the NCAA has no plans yet to make it mandatory. Even so, many manufacturers are introducing products that meet this new standard.

Another example is the implementation of standards for skiing and snowboarding helmets. ASTM finalized a standard for helmets used in all recreational snow sports in the spring of 2000.[26] No governing body has mandated the use of helmets that meet this standard at the time of this writing.

On the other hand, the CPSC's nationwide mandatory standard for bicycle helmets was issued in early 1998 and made effective immediately.[27] When standards are government mandated, the added inspection by government entities to verify compliance and the threat of criminal action if noncompliance is discovered are strong incentives for manufacturers' compliance.

SUMMARY

Many of the standards that have been created to judge the performance of protective equipment for athletic activities routinely undergo revision. It can be misleading to simply state that a particular standard is *x* number of years old. It is likely that the standards-setting body has had a standard in effect on that product for that period of time, but it is also very likely that the document in place today is quite different from the original of the same name. Typically, the latest revision date is prominently displayed on the document or as part of the title. For both NOCSAE and ASTM standards, the standard's numeric identification is followed by a dash or hyphen, followed by the year, as in NOCSAE DOC 001-98. When a midyear revision is developed, the designation is alphabetically added as a suffix to the year, as in ASTM F1446-95a. This designation means that after the 1995 revisions but before the end of 1995, the document was revised and reissued in 1995 as 95a.

Recent standards updates include the changes made to NOCSAE standards for headgear used in football, baseball, and lacrosse. These changes made clear demands on manufacturers' quality control while also increasing performance requirements. In addition to improving existing standards in areas of impact performance, helmet stability, and the like, the ASTM has introduced new headgear standards for skiing, downhill mountain biking, speed skating, roller and inline skating, and infant and toddler bicycling, while several others remain under development. This same ASTM committee has made similar strides in eye and face protection by revising and improving existing standards and creating new ones in areas of need. The formation of the Protective Eyewear Certification Council is an important advancement in athlete protection and should be relied upon by governing bodies. Both NOCSAE (http://www.nocsae.org) and ASTM (http://www.astm.org) have Web sites that can be used to gain more information about their efforts.

Measurement methods and standards, while continually being refined, seldom duplicate the on-field use conditions of the products they govern. However, within those limitations, products that meet specific standards of performance have been shown to be effective in reducing injury.[28]

For the sports medicine clinician, it is important to recommend only personal protective equipment that claims to meet a current standard. It is also necessary to maintain that equipment in its original form, being sure that all of the parts and labels are there before the start of each sports season. It may be wise to use a qualified sports equipment reconditioner (for example, a member of the National Athletic Equipment Reconditioners Association) to have the equipment refurbished periodically. Clinicians should not hesitate to ask the manufacturer claiming to meet a standard to supply independent proof of compliance. Clinicians can participate in the NOCSAE standard process by contacting the National Athletic Trainers' Association member who serves on the NOCSAE board to be sure that the group has the latest information and input. Clinicians can also become members of the ASTM. Participation in ASTM task groups does not require ASTM membership, but membership entitles the member to vote on any ASTM standard action.

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Heading in Soccer: Integral Skill or Grounds for Cognitive Dysfunction?

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Objective: To critically review the literature concerning the effect of purposeful heading of a soccer ball and head injuries on reported cognitive dysfunction in soccer players.

Data Sources: We searched MEDLINE (1965–2001) and SPORTDiscus (1975–2001) for refereed articles in English combining key words for soccer (eg, *soccer, football, association football*) with key words for head injuries (eg, *concussion, head injury*). In addition, literature on cognition and head injuries was obtained. We reviewed reference lists of current literature for pertinent citations that might not have been found in the search procedures.

Data Synthesis: The fact that soccer players (and other athletes) have selected cognitive deficits is not questioned, and the popular press is quick to publicize results of questionable validity. The reasons for such deficits are many. Much of the early data implied that heading was the culprit; however, subsequent research has suggested that other interpretations and factors may be potential explanations for these deficits. The current focus is on concussions, a known factor in cognitive dysfunction and a common head injury in soccer.

Conclusions/Recommendations: It is difficult to blame purposeful heading for the reported cognitive deficits when actual heading exposure and details of the nature of head-ball impact are unknown. Concussions are a common head injury in soccer (mostly from head-head or head-ground impact) and a factor in cognitive deficits and are probably the mechanism of the reported dysfunction.

Key Words: concussion, mild traumatic brain injury

Soccer is one of the oldest games. References to "kick ball" can be found as far back as 200 BC in China and around 4 BC in Greece. Although various versions of games that used a ball propelled by the feet emerged throughout the centuries, the first set of formal rules was set down by Cambridge University in 1848. The Football Association of England was founded in 1863 and further defined the rules of the game. The international governing body, Federation Internationale du Football, was founded in 1904 and today has more member nations than the United Nations.

Soccer is unique among sports because of the purposeful use of the unprotected head to control and advance the ball. Obviously, this skill places the head in a vulnerable position for injury. The recent death of Algeria's top scorer, Hocine Gacemi, from complications of a skull fracture after he "crashed head first into the ground after a clash of heads,"[1] along with parental questions on the safety of heading have brought this skill to the attention of the media. Also, the use of helmets in soccer has been debated.[2] The US Consumer Product Safety Commission met in May 2000 to discuss head injuries and protective equipment.

From attempted heading of the ball to falls, soccer provides many opportunities for head contact with the ball, ground, opponent, goalposts, and off-the-field objects. In this brief review, we will examine the available data on head injuries in soccer and their mechanisms and consequences. Much is still to be learned, and the critical reader is asked to carefully examine the data on cognitive deficits in soccer players. Are these deficits due to purposeful heading of the ball, or are the deficits a result of mild traumatic brain injury from head impacts other than routine heading of the ball?

MECHANICS OF IMPACT

The mechanics of impact are based on the relationship $F = ma$, where F is the force of impact, m is the mass of the object, and a is the acceleration of the object at any instant in time. Soccer balls come in 3 sizes and weights (Table 1). Before the 1970s, the ball was leather and could absorb considerable amounts of water when used on wet ground. In the mid 1970s, the leather ball was modified with a water-resistant coating, but it could still gain mass from water. The modern ball is synthetic and resistant to water absorption. When interpreting the literature, it is important to pay attention to the probable dates when soccer players might have competed using a leather ball.

Nearly every professional article and media presentation mentions that a kicked ball can travel more than 100 km/h. The implication is that these ball velocities occur across ages and both sexes. Yet, players rarely head that kind of shot voluntarily, even though accidental impacts are possible. The highest-velocity ball a player might voluntarily head would be from a punt (approximately 70 km/h), drop kick (approximately 85 km/h), or goal kick (also approximately 85 km/h). Maximum ball velocities according to age and sex have not been reported. Levendusky et al[3] suggested that most oppor-

Table 1. Dimensions of the Soccer Ball

	Size 3	Size 4	Size 5
Mass, g (oz)	312–340 (11–12)	312–369 (11–13)	396–453 (14–16)
Circumference, cm (in)	56–59 (23–24)	61–64 (24–25)	68–71 (27–28)
Age group, y	6–9	10–13	≥14

tunities for heading occur at ball velocities less than 65 km/h. A ball traveling 65 km/h hits a solid object for 10.23 milliseconds at a force of 850 to 912 N with an acceleration of 30 to 55 *g* (gravitational) forces.[2,4] In American football, impacts last for 200 to 350 milliseconds at *g* forces ranging from 150 to 450. In boxing, impact duration ranges from 14 to 18 milliseconds at 6000 N and *g* forces of 100. Schneider et al[5] estimated that the necessary concussive forces are about 22 $N \cdot s^{-1}$. The estimated impact of a soccer ball (at 64.37 km/h) is between 12.4 and 13.7 $N \cdot s^{-1}$, well below concussive levels.[3]

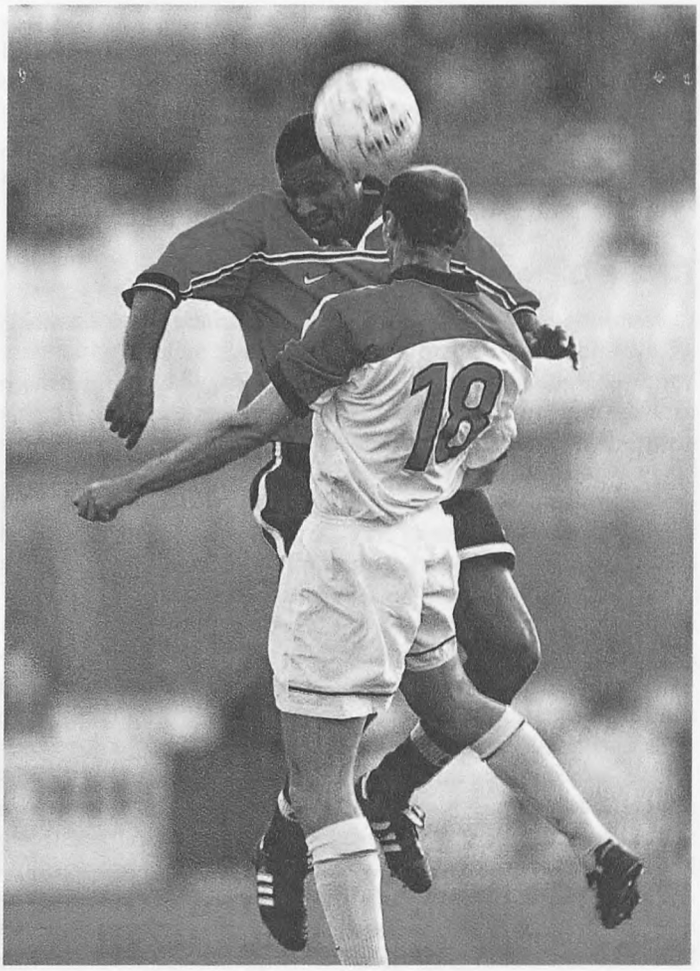
The head can withstand greater linear forces, whereas rotational impacts cause the most injury.[6] Purposeful heading is largely linear. Boxing impacts are both linear (from "jabs") and rotational (from "hooks"). In soccer, head injury from ball contact is mostly due to rotational impacts from accidental contact of the ball to the head. Head-ball contacts on the side of the head of an unprepared player can obviously lead to rotational impacts. Preparation of the head cannot be understated: strike an unprepared player on the forehead, and linear forces (pushing the head posteriorly) can be coupled with transverse rotation (as in a whiplash injury).

MECHANICS OF HEADING

Heading a soccer ball is a difficult task, which is further complicated by having to perform the skill during other activities (eg, standing, walking, running forward or backward, jumping, being challenged by an opponent, diving). Although coaching books describe teaching progressions, most do not discuss heading until the players are about 12 years or older. Younger players spend a modest amount of time practicing heading and even less time heading a ball in a game (making it more of a novelty than a polished skill). In youngsters, most heading opportunities occur after the ball has bounced. Any opportunity for heading requires the use of an age-appropriate ball (Table 1).

The ball is to be contacted on the forehead at or near the hairline (a coach will say to hit the ball on the forehead, where you put your hand to feel for a fever). Heading is active. Again, coaches will say "hit the ball, don't let the ball hit you." The trunk is hyperextended and the chin tucked in to the chest. The more trunk extension, the faster the forward velocity of the trunk and the harder the impact on the ball. The arms are usually extended for balance and protection. When the athlete jumps for a head ball, the legs are somewhat extended at the hip and flexed at the knee in preparation for striking the ball. Most of these positions are seen when 2 players compete for a head ball (Figure).

Successful heading is an exercise in precision timing. Flexing the hips brings the trunk and head toward the ball, while the arms are pulled back. Contracting the neck muscles fixes the head, making a firm surface for contact and effectively increasing mass of the contact surface. To contact the ball at the top of a jump (vertical velocity = 0), the trunk must be flexed to transfer force to the ball. Most ball-related injuries are due to the ball hitting an unprepared head (ie, the head and neck are not stabilized). This increases the ball mass-contact mass ratio and increases the risk of injury,[7] because the force of the ball hitting the head can accelerate the head backward.



When 2 players compete for a head ball, the arms and legs are extended and the knees are flexed (photograph courtesy of *U.S. Soccer*).

MECHANISMS OF HEAD INJURY IN SOCCER

Limited work is available on the mechanisms of head injury in soccer, and no reliable and valid statements exist on heading exposure in any level of soccer other than European (mostly English) professionals, where there is an average of 6 to 7 headers per game.[8] The number of headers occurring during training is largely unknown.

Boden et al[9] studied all Atlantic Coast Conference soccer teams for 2 years to determine concussion incidence. The most frequent mechanism of injury was head-to-head contact, followed by head to ground and then head to other body parts (eg, foot, knee, elbow). Importantly, purposeful heading was never a mechanism of injury, but injuries did occur when the player was accidentally struck by the ball. Although not discussed by Boden et al,[9] a concussion is possible without contact. If the impulse of contact is strong enough to cause significant rotation of the head, mild traumatic brain injury is possible.

The experienced coach or observer of the game can direct

us to important factors in heading and head injuries. The penalty area (where players compete for a cross-corner kick) and the collision of an onrushing forward and goalkeeper are the most likely circumstances for a head injury. Goalpost collisions are possible but not common. Near the midfield line, where players compete for airballs (eg, punts, goal kicks), is another troublesome place on the field.

RATES OF HEAD INJURY IN SOCCER

Head injuries have been reported to account for 4% to 20% of all injuries in soccer.[10] These statistics include nasal fractures, contusions, lacerations, concussions, and eye injuries. It is important to separate out the concussive injuries for this discussion. The added difficulty of grading a concussion is dealt with elsewhere in this issue.

Minimal published data are available on the frequency of head injuries in youths (12 to 14 years and younger). Powell and Barber-Foss[11] reported that mild traumatic brain injuries accounted for 3.9% and 4.3% of all injuries in boys' and girls' scholastic soccer, respectively. These injuries almost always occurred during games, and 80% or more of the injured athletes sat out for 1 week or less (median, 3 days). Powell and Barber-Foss[11] estimated 0.22 and 0.26 mild traumatic brain injuries per soccer team per season for boys and girls, respectively. Our ongoing survey of high-level youth soccer players (ages 12 to 18 years) in North Carolina shows that about 15% of all injuries were to the head (all head injuries, not solely concussions) and were due to player-player or player-ground contact. The survey of Boden et al[9] of college-aged players (18 to 22 years old) demonstrated that each team could expect about 1 concussion per season. The reported concussions were largely game related and not due to purposeful heading of the ball. Barnes et al[12] surveyed soccer participants (n = 137) at a US Olympic Sports Festival. More than half the men and one third of the women had a concussion history. In comparison, Gerberich et al[13] noted that 19% of secondary school football players had a concussion history, yet just fewer than half the Canadian Football League players (in 1997) reported a concussion.[14] Powell and Barber-Foss's[11] survey of secondary school sports indicated that mild traumatic brain injury accounted for 7.3% of all injuries in football, and each football team could expect nearly 2 concussions a season.

One fact does appear evident: the higher and more competitive the play, the more frequent the incidence of concussion. More research at the recreational and professional levels, using many leagues and a consistent definition of concussion, is needed.

NEUROLOGIC AND NEUROPSYCHOLOGICAL FINDINGS IN SOCCER PLAYERS

The Norwegian Studies

The earliest description of heading injury was by Matthews,[15] who diagnosed "footballer's migraine" in the days when a wet ball could increase in mass by 20%. Tysvaer et al,[16–20] who examined neurologic and neuropsychological sequelae in active and retired soccer players, directed the first projects on neurologic deficiencies in soccer players.

Their first report[17] was a 1975 survey sent to 192 players in the 12 first-division Norwegian professional league teams. The subjects had a mean age of 25 years and had participated in an average of 100 professional games, with nearly half having international experience. A total of 128 players met the inclusion criteria. Of those, 77 had full neurologic examinations; half of the players reported symptoms related to heading. The primary symptoms were disorientation, headache, and nausea. Seven players were rendered unconscious, and 3 were hospitalized. The mechanisms of head injury in this study were not reported. Given the publication date and the age and experience of these players, one would have to assume that many played in an era when water retention by the ball was a factor in impact.

Tysvaer et al[16,18,19] published 3 articles in 1989 on neurologic and electroencephalographic (EEG) data on active and retired soccer players. The 69 active professional players (15 to 35 years of age and 128 professional games) were matched with a nonathletic, age-appropriate control group. All players with a history of head injury (4 concussions) reported acute symptoms. Eight players had persistent symptoms, and 2 had permanent complaints, but the neurologic examination results were normal in all players. Normal EEGs were obtained in 65% of the players and 87% of controls ($P < .001$). In addition, EEG abnormalities were most often detected in the youngest players. Forty-four percent of the players younger than 25 years had either a "slightly abnormal" or "abnormal" EEG, whereas 26% of the players older than 25 years had similar findings.

The 44 retired players (age range, 35 to 64 years; average length of retirement, 14 years) were compared with 37 men "in the same age range."[18] The EEGs were read in a blinded manner. Seventy percent of the retired players had a history of head trauma (5 concussions). The neurologist's interviews with the players revealed prolonged symptoms from heading in 11 players, and 8 had permanent complaints. A variety of neurologic symptoms were reported in the interviews (nystagmus, n = 5; impaired coordination, n = 2; reflex disturbance, n = 1; impaired hearing, n = 2; unsteadiness, n = 1; reduced cervical spine motion, n = 15). No statistical differences were found in EEG readings for players with and without complaints or for players categorized as "headers" and "nonheaders." "No clear differences" were noted when the different age groups of players were compared with the control subjects. Unlike the previous study, age was not a factor in EEG disturbances.

Anatomical evidence of brain damage in a subset of the retired players was assessed by computed tomography[16] in 33 of the retired professional players (age range, 39 to 60 years; range of retirement, 8 to 39 years). No control group was studied, which makes blinding a concern. The scans were read by one of the authors. Widened ventricles were found in 27% of the players assessed, indicative of central cerebral atrophy, and 18% had cortical atrophy. Players identified as "headers" had a significantly higher frequency of cortical atrophy. Only 7 players had a history of head injuries (with unconsciousness or amnesia), and they had no greater frequency of atrophy than those without a history of head injury. These findings of atrophy may or may not be associated with either reversible (eg, steroid therapy or starvation) or irreversible (eg, brain injury or aging) decline in intellectual function.

Finally, neuropsychological evaluation of these same retired players was reported in 1991.[20] The players were administered the Wechsler Adult Intelligence Scale, the Trail-Making Test Parts A and B, a modification of the Malstead-Wepman-Reitan aphasia screening test, tests of sensory-perceptual functions,

Table 2. The CAGE* Questionnaire of Alcohol Abuse

1. Have you ever *CUT* down on your drinking?
2. Have people *ANNOYED* you by criticizing your drinking?
3. Have you ever felt badly or *GUILTY* about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (ie, an *EYE OPENER*)?

*Acronym taken from the capitalized word in each question. Reprinted with permission from the *Journal of the American Medical Association* (1984;252:1905–1907). Copyright 1984, American Medical Association.

motor tests, tests of hemisphere dominance, and the Benton Visual Retention Test form C. For comparison, the authors chose 20 hospitalized patients with no history of head or neck injuries or evidence of brain damage. Group differences were most obvious in the Trail-Making Tests ($P < .01$). Impairment was severe to gross in 1 player, severe in 3 players, moderate in 14, mild in 12, and none in 7 compared with controls. Impairment was unrelated to subject age. Forty percent of the controls demonstrated mild impairment. The scores for nearly one third of the soccer players were low enough (both parts of the Trail-Making Test) to suggest physical damage to the brain. Throughout the full series of studies, the focus was on heading as a contributor to the findings.

Other Studies

The Norwegian studies brought attention to the problem and encouraged others to work in this area. Haglund and Eriksson[21] compared amateur boxers with active or former professional soccer players (25 to 44 years of age) and track athletes. After clinical studies (medical and neurologic studies), personality trait assessment (Karolinska Scales of Personality), magnetic resonance imaging (MRI), and neurophysiologic and neuropsychological examinations, they "failed to demonstrate any signs of chronic brain damage in soccer players who were known to be frequent headers." On one neuropsychological test (finger tapping), performance by the soccer players was lower than other athletes, but these results were still within normal limits.

Jordan et al[22] compared the 20 players on the US men's 1994 World Cup team with 20 track athletes and focused on symptoms of previous head injury and on MRI. Players were categorized as low- or high-frequency headers (player's opinion), and the authors attempted to form a heading exposure index based on years of experience and level and location of competition. Alcohol abuse is a known factor in organic and neurologic brain damage, and, therefore, each player was screened with the CAGE scale for alcohol use (Table 2).[23,24] No differences in symptoms were found between the 2 groups. Further, they "found no evidence that this exposure correlated with symptoms that might be attributed to chronic encephalopathy. In addition, increased heading exposure did not correlate with abnormalities on brain MRIs." Symptoms were correlated with history of head injury, not with any playing parameter or their index of heading exposure.

Recently, Dutch researchers[25] performed comprehensive neurologic assessments on amateur and professional soccer players while controlling for heading exposure, prior head injury, and history of alcohol intake. "Regional-level" amateurs were compared with a control group of swimming and track athletes. These amateur players showed impaired planning ability ($P < .001$, Wisconsin Card-Sorting Task, Complex Figure Test), memory ($P < .004$, Complex Figure Tests, Wechsler Memory Scale, 15-Word Learning Test), and motor ability ($P < .01$, fine motor behavior, nondominant hand puncture test). The 53 active professional soccer players were compared with a similar control group. The professional players scored lower on memory tests, planning, and visuoperceptual processing. Deficits in both studies were correlated with the head injury history. Matser et al[25] stated that "concussions incurred in amateur soccer may play a fundamental role in the development of cognitive impairment observed in these (soccer) players" and continued with "the neurological effects of amateur soccer appear to be related to soccer-related concussions caused by contact trauma."

Matser et al[25,26] grouped their subjects into controls and players. The player group actually contained 2 subgroups: players with a history of head injury and players without a history of head injury. Had the authors separated out these subgroups, some interesting findings would have been possible. If the results for the uninjured player group were similar to the control group, then purposeful heading would not have appeared to be a factor because they had a long history in the game without measurable deficits. However, if the uninjured player group was similar to the group with head injury history, then purposeful heading might indeed be a factor in the deficits. Grouping collegiate players this way does not support purposeful heading as a factor in cognitive deficits (K. M. Guskiewicz, unpublished data, 2000).

Short-term exposure to heading was studied in a prospective project on collegiate players.[27] One hundred male and female athletes were administered neuropsychological tests before and after 2 training sessions (1 with heading and 1 without heading). The tests included the Alphabet Backwards Test, Trail-Making Tests (Parts A and B), Stroop Color Word Test, and VIGIL/W. No significant differences were noted on any test when comparing the control (no-heading) condition with the experimental (heading) condition.

The parent of the very young soccer player should be relieved by the comments of Bijur et al,[28] who concluded, "there is no evidence. . . that multiple head injuries between birth and age 10 years have a deleterious effect on a global measure of intelligence or on academic achievement measured at 10 years of age." If they showed no deficits after many concussions, then deficits from infrequent subconcussive events, such as the occasional heading by the youngest of players, are unlikely. However, more longitudinal work is necessary in this area. Differences in the findings on the young people[29] and adults[16–20,25,26] may result from recall bias.

ASSIMILATING THE LITERATURE

The findings of the Norwegian studies are frequently cited as proof that heading is the cause of cognitive deficiencies. Other European and American investigators who mentioned decrements were not able to say that purposeful heading was the cause. Of interest is the low frequency of concussion in the Norwegian studies. According to Barnes et al[12] and Boden et al,[9] the number of concussions seems quite small. Were there really so few concussions, or were concussions underreported due to definition difficulties? Some methodologic problems and other confounding factors must be considered before we blame purposeful heading for any negative findings.

First, consider the inclusion criteria for the Norwegian studies, especially the studies of retired players. The Norwegian studies began in the early 1980s, so the players likely were

participating in the 1960s and 1970s, before the advent of the water-resistant ball. Most players of that era freely admit that heading a wet, heavy ball could lead to the reported symptoms.

Second, other causes of impaired cognitive function were not considered in the earlier studies. Alcohol abuse and malnutrition are known to lead to cognitive deficiencies.[23,24] Simply mentioning alcohol intake is not a valid way to explore alcohol intake or abuse.

Third, a previous head injury is a well-known cause of cognitive dysfunction. In the Norwegian studies, players with non–game-related head injuries were excluded, but the game-related head injuries were not described. Consciousness or unconsciousness was mentioned; however, few other details about concussion history or other head injuries were mentioned. Barnes et al[12] noted that 54% of the 72 men surveyed had a history of a diagnosed concussion, and 33% had multiple concussions. The authors estimated 50% odds of a male athlete playing for 10 years at this level sustaining at least one concussion. These results were for high-level amateur players, but we might be safe in assuming that professionals are more aggressive and may experience concussions at least at the same frequency, perhaps even more. Thus, concussion history is underreported in the Norwegian studies. Remember that the active professionals in the studies by Tysvaer et al[16–20] reported only 4 concussions in 69 players who averaged 128 professional games.

Part of the problem may be the definition of what constitutes a concussion. A common response by parents and players when questioned about a head injury is "there was no concussion—wasn't knocked out." Many physicians in Europe and South America believe concussions are not a problem, implying that concussions might be underdiagnosed.

Unconsciousness is not a requirement for cognitive dysfunction. Lovell et al[30] showed that loss of consciousness (LOC) was a poor predictor of cognitive deficits in patients with mild head trauma. The patients who met their inclusion criteria were divided into 3 groups: no LOC, undetermined LOC, or documented LOC. All patients with mild head injury showed "mildly decreased performance" on a variety of standard neuropsychological assessments, but LOC was not a factor in cognitive performance, because all 3 patient groups performed similarly. In addition, the effects of mild concussion can last well beyond the time when a patient might return to his or her regular activities. Macciocchi et al[31] showed that concussed collegiate American football players improved during the succeeding 10 days but still were not fully recovered. In addition, more than half of the patients studied by Rutherford[32] were still symptomatic up to 6 weeks after injury. Hugenholtz et al[33] showed that cognitive deficits from a "mild concussion" (grades 1 and 2) could still be documented up to 3 months after injury. A report by Rutherford et al[34] showed that nearly 15% of patients with "mild concussion" had symptoms for 1 year after injury. To ignore concussion history in soccer when reporting cognitive deficits could well bias conclusions and lead to the assumption that the neuropsychological deficits were due to purposeful heading.

A possible fourth factor is learning disabilities.[35] Children with dyslexia[35] and attention-deficit/hyperactivity disorders[29] tested poorly when compared with healthy controls. College students with learning disorders or mild brain injury performed below healthy students.[36]

Another factor is that the overwhelming majority of research on mild traumatic brain injury and soccer is cross-sectional. A longitudinal design with careful control of confounding factors would probably answer many of the questions raised by both the public and the medical community.

It is obvious that there are many factors to consider before attributing diminished cognitive function in soccer players to the subconcussive impacts of purposeful heading. The clinical significance of any reported dysfunction has yet to be clarified. Even though there are documented deficits, whether these deficits have any effect on activities of daily living is unknown.

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Concussion in Rugby: The Hidden Epidemic

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Objective: To determine the incidence of concussion in high school rugby players and compare the findings with the relevant published literature.

Design and Setting: Prospective data collection in one US high school rugby program.

Subjects: Two teams followed for 3 years.

Measurements: Injury rate, injury severity, and time withheld from competition.

Results: Seventeen concussions were recorded, accounting for 25% of all reported injuries. The incidence rate for concussion was 3.8 per 1000 athlete-exposures (95% confidence interval, 2.0–5.7) or 11.3 per 100 player-seasons (95% confidence interval, 5.9–16.7). Of the 17 concussions, 14 were Cantu grade 1, 2 were grade 2, and 1 was grade 3. Concussions accounted for 25% of all days lost from rugby participation due to injury.

Conclusions: The incidence of concussion in this study was higher than previously reported in other studies. Methodologic limitations and administrative rules that required suspension for injured players may have suppressed reporting in previous epidemiologic studies. The incidence of concussion in rugby is probably much higher than previously suggested.

Key Words: injury, epidemiology

Rugby union is a ball-carrying game somewhat similar to North American football in that both are full-body contact sports in which 2 teams compete. In rugby, however, both offensive and defensive teams take the field at the same time, the ball cannot be thrown forward, and it is illegal to block a player who is not in possession of the football. Rugby is popular in many countries, including New Zealand, Australia, Fiji, Japan, South Africa, Argentina, England, Wales, Scotland, Ireland, and France. In the United States, the game is largely played at the high school and club levels; the total playing population is estimated to be about 50 000.[1] Rugby has traditionally been a game for men, but recent years have seen an explosion in the numbers of women playing the game.

Concussion is of particular concern in rugby. Participants are largely unshielded from collision forces, and the cranium is subjected to violent acceleration-deceleration and rotational forces. Mouthguards and soft-shell head protection are used by some players, but their effectiveness in preventing concussion in rugby is open to question.[2,3] To complicate matters, much of the world's rugby is played in situations in which medical personnel are frequently not present. Those medical personnel who are available are often not well trained in recognizing and managing concussions. An administrative barrier also affects concussion identification and management. Under the rules of the game, as promulgated by the International Rugby Board, any player who self-reports or is diagnosed as having a concussion is subject to an automatic 3-week suspension from all competitions and team practices. This mandatory 3-week "stand-down" period is supposed to apply even when a player sustains a very mild injury and returns to a normal level of functioning within minutes. As a result of these factors, we came to suspect that many rugby concussions go unreported.

As a first step toward determining the true incidence of concussion in rugby players, we analyzed data from a Utah high school rugby program that has applied standardized and current criteria for the diagnosis and management of concussion. This database is an important source of information on high school rugby injury because the primary data collector is a certified athletic trainer (ATC), and ATCs are not widely available in high school rugby programs. We then consider the published epidemiologic data on concussion in light of the Utah data and argue that previous studies have consistently underestimated the true incidence of concussion in rugby.

METHODS

One of us (R.J.S.) is the ATC for Highland Rugby in Utah, a well-organized rugby program that has competed successfully in the United States and abroad. The program maintains a first and second team in varsity competition and averages about 16 games per season per team. Injuries that result in time lost from games or team practices and all fractures and concussions are documented in a computer database. This database is maintained exclusively by one individual (R.J.S.). Exposure data on games and team practices are also recorded in the computer database. Data from the 1998, 1999, and 2000 seasons were analyzed.

The Highland rugby medical staff strives to ensure that all concussions are identified and that these injuries are managed in accordance with current clinical recommendations. Concussion injuries are closely monitored and are frequently reviewed

by a team physician. Concussions are graded using the Cantu scale.[4]

To compare the Highland data with previously published estimates of the incidence of concussion in rugby, one of us (S.W.M.) searched for scientific articles reporting on the incidence of rugby injury. The MEDLINE and SPORTDiscus databases were searched for any article with the strings *rugby* and *injur** in the title, abstract, or subject header. The search was limited to articles written in English (to avoid translation costs), published since 1980, and presenting population-based data on the incidence of rugby injury and concussion. Nine such articles were identified.[5–13] The reference lists of these 9 articles were examined for additional publications that met our criteria but were not listed in MEDLINE or SPORTDiscus; one additional article was located.[14]

Each article was reviewed and the following data extracted: information on the study population, the number of injuries, the number of concussions, the definition of injury used in the study, the number of player-seasons of follow-up, and the methods used to collect injury data. The ways in which injury rates were reported in the literature varied widely. Some authors reported rates using player-hours as the denominator, whereas others used player-games or player-seasons. Player-seasons is the number of athletes per team multiplied by the number of seasons for which the team was followed; player-games is the number of athletes per team multiplied by the number of games for which each team was followed; and player-hours is the number of athletes per team multiplied by the number of competitions followed per team multiplied by the average length of the competition in hours. All of these exposure measures are variants on the underlying epidemiologic concept of person-time at risk of injury.

To compare the various studies, the concussion rate per 100 player-seasons was calculated using the data presented in each article. Player-seasons was used as the measure of exposure because this was the only measure that could be calculated for every study. For one study,[9] the number of players at risk was an approximate number only; in another[7] the approximate number of players at risk had to be estimated based on information in the article. One study[14] presented exposure data for only a defined subset of subjects; the concussion incidence in this subgroup had to be estimated using other information in the article.

RESULTS

Incidence and Description of Concussion Injuries in Highland Rugby

Seventeen concussions were recorded during the 3 seasons of follow-up. For comparison, the total number of injuries (concussion and nonconcussion combined) was 69. Concussions, therefore, accounted for 24.6% of all reported injuries. The incidence rate for concussion was 3.8 per 1000 athlete-exposures (95% confidence interval [CI], 2.0–5.7). Twelve of the injuries occurred in games, for a rate of 11.1 per 1000 player-games (95% CI, 4.8–17.4), and 5 occurred in practices (1.5 per 1000 player-practices; 95% CI, 0.2–2.8). Eight of the concussion injuries were to backs, and 9 were to forwards. Concussions accounted for 25.3% of all days lost from rugby participation due to injury.

Fourteen observed concussions were Cantu grade 1, 2 were grade 2, and 1 was grade 3. The grade 3 event was characterized by a 5- to 6-minute loss of consciousness, accompanied by disorientation and mild convulsions. The athlete recovered relatively quickly and returned to competition within 2 weeks. A grade 2 concussion resulted in the greatest time lost from participation. The athlete experienced dizziness, amnesia, disorientation, and headache and was withheld from competition for 71 days.

The rate for all injuries overall (concussion and nonconcussion combined) was 1.5 per 1000 athlete-exposures (95% CI, 1.2–1.9). To facilitate comparison with other studies, we also calculated rates using player-seasons as the unit of exposure. Using this measure, the incidence rate for all injury was 46.0 per 100 player-seasons (95% CI, 35.1–56.9), and for all injuries excluding concussions, the incidence rate was 34.7 (95% CI, 25.2–44.1). The concussion rate was 11.3 per 100 player-seasons (95% CI, 5.9–16.7).

Comparison With Published Studies Reporting Concussion Incidence Rates

We compared our results with the published studies to determine the degree of underreporting of concussion, if any, in previously published data. Estimates of the incidence of concussion varied considerably among studies. The proportion of overall rugby injury that was classified as concussion ranged from 1% to 22%, and the incidence rate for concussion ranged from 0.1 to 8.3 per 100 player-seasons (Table).

Under the assumption that the Highland data presented in this article are an estimate of the true incidence of concussion in these various playing populations, we constructed an index of concussion ascertainment in each study, defined as follows:

$$\phi_i = 100 \times \frac{\text{observed concussion rate in study } i}{\text{concussion rate in Highland study}}$$

Higher values of ϕ_i indicate greater success at identifying and collecting data on concussions; ϕ_i ranged from 1% (indicating that 99% of concussions were missed) to 86% (indicating that 14% were missed).

DISCUSSION

The Highland results demonstrate conclusively that it is possible, at the high school level, to collect high-quality data on rugby concussions. An ATC's involvement is the key to the success of the data collection system. Although ATCs receive instruction in the diagnosis and management of concussions, ATCs rarely work closely with a high school rugby program. The Highland data, therefore, afford us a unique insight into the incidence of concussion at this level of rugby.

Review of Previous Studies

The 10 studies reviewed herein were conducted in several different countries and covered both high school and adult playing populations. Nevertheless, the pattern is consistent, with all 10 studies producing estimates of concussion incidence well below those seen in Highland rugby. We present 2 explanations for this finding. First, the style of rugby played at Highland involves a greater risk of head impact or higher forces of the type needed to generate concussions, or both, resulting in a greater incidence of concussive injury. Second,

Estimates of the Incidence of Concussion in Rugby

Study	Year	Country	Population	Definition of Reported Injury	Data Collection Method	No. of Reported Concussions	No. of Reported Injuries Overall	Concussions as a Percentage of All Injuries	Incidence Rate per 100 Player-Seasons		Concussion Ascertainment Index, %
									All Injuries	Concussions	
Nathan et al[5]	1983	South Africa	High school males	Prevented play >7 days	Interview players who missed games	17	79	21.5	16.99	3.66	32.3
Sugerman[14]	1983	Australia	High school males	Prevented play or school	Teacher or coach report	22	258	8.5	114.67	9.78	86.3
Roux et al[6]	1987	South Africa	High school males	Prevented play >7 days and all concussions	Teacher or coach report; some personal visits	74	495	14.9	10.31	1.54	13.6
Davidson[7]	1987	Australia	High school males	Required medical care	School physician or nurse in central location	16	1444	1.1	10.56	0.12	1.0
Clark et al[8]	1990	South Africa	Adult males	Prevented play >7 days or medical care	Self-administered form; contact at club	10	114	8.8	95.00	8.33	73.6
Dalley et al[9]	1992	New Zealand	Mixed ages and both sexes	Required medical care	Club and team contacts	74	921	8.0	8.37	0.67	5.9
Hughes and Fricker[10]	1994	Australia	Adult males	Prevented play or required medical care	Team athletic trainer	5	133	3.8	110.83	4.17	36.8
Garraway and Macleod[11]	1995	Scotland	Adult males	Prevented play	Club contact; volunteer observers	20	358	5.6	29.44	1.64	14.5
Lee and Garraway[12]	1996	Scotland	High school males	Prevented play	School contact	18	210	8.6	12.32	1.06	9.3
Bird et al[13]	1998	New Zealand	Mixed ages and both sexes	Prevented play or required medical care	Weekly telephone follow-up of all players	23	569	4.0	159.83	6.46	57.0

previous researchers have underestimated the true incidence of concussion by varying degrees.

Specific characteristics of the Highland style of play are distinctive. More than half of the players have a background in North American football, which results in more emphasis on the "body-check" style of tackling. However, it seems implausible that differences in style of play could explain why rates of concussion in a Utah high school are 100 times greater than those reported in an Australian high school.[7] Methodologic differences must account for most of the variation in concussion incidence among the various studies. Assuming that the Highland data represent a valid estimate of the true incidence of concussion in the playing populations detailed in the Table, the previous epidemiologic studies[5–13] have probably underestimated the true incidence of concussion in rugby, in some cases by a substantial margin.

Possible Reasons for Underreporting in Previous Studies

A variety of methods were used in the 10 studies, ranging from weekly telephone contact with all players, both injured and uninjured (high-intensity follow-up), to recording all injuries reported to a central clinic (low-intensity follow-up). The definition of injury also varied among studies. We suggest that the observed concussion rate in each study is an artifact of 3 factors: (1) the actual rate of concussion in the population studied, (2) the definition of injury used in the study, and (3) the method used to collect injury data: in particular, the intensity of follow-up to ascertain injuries.

Variations in the true rate of concussion among these playing populations are probably minimal, since the basic characteristics of the game (running, tackling, passing) are similar for all populations. Intuitively, one would expect that variations in the definition of injury would influence the observed incidence rate. In fact, intensity of follow-up appears to be a stronger determinant of the observed incidence rate than the definition of injury, given that the investigators with the most restrictive injury definitions reported the highest rates for both concussion[5,6,8] and overall injury.[8]

There is also empirical evidence of systematic underreporting of concussion injuries in rugby. In one study,[6] the authors divided the participating high schools into 2 groups. The low-intensity follow-up group self-reported the injuries with little or no prompting, whereas the high-intensity follow-up group received regular personal visits as a prompt to reporting. Athletes at the high-intensity schools were 4 times more likely to report a concussion injury than those at the low-intensity schools, leading the authors to conclude that concussion in rugby is highly susceptible to underreporting.[6]

Providing well-trained medical professionals is essential to the identification and successful management of concussion. Unfortunately, much of the world's rugby is still played in situations in which medical personnel skilled in recognizing concussions are absent. Administrative barriers to the accurate reporting of concussion in rugby also exist. Any player diagnosed as having a concussion is subject to an automatic 3-week suspension from all competition. Originally designed to protect the player, this "stand-down" rule has probably put players at greater risk by discouraging athletes from seeking treatment and suppressing the reporting of symptoms associated with concussion. Sadly, some health care professionals have colluded in the process of discouraging the accurate reporting of concussions, as evidenced by the use of nonsensical diagnoses such as "traumatic migraine" or "loss of consciousness without concussion." Mindful of the situation, the International Rugby Board recently modified its rules to permit an adult athlete to return to play if medically cleared for competition by a neurologist.

We believe that increasing the number of qualified health care professionals who serve rugby teams and who are trained in current techniques regarding concussion is an essential step toward improving the recognition and management of these injuries. As a first step toward improved recognition of concussions on the rugby field, we need better data on the incidence of concussion in rugby. Further epidemiologic studies that provide accurate data on concussion incidence are required.

CONCLUSIONS

Published estimates of the incidence of concussion in rugby vary widely. Using data from a large, high school–based rugby program in the United States, we have demonstrated that the incidence of concussion in rugby is probably much higher than previously suggested.

Much of the world's rugby is played in countries where there is a shortage of medical personnel well trained in recognizing and managing concussion. In addition, until recently, the International Rugby Board ruled that any player with a concussion must be suspended from active competition for 3 weeks (even for a grade 1 injury that resolved within minutes), and the effect of this rule has probably been to dampen reporting of symptoms by concussed players.

As a result of these 2 factors, previous epidemiologic studies of rugby injury have probably underestimated the incidence of concussion in rugby, in some cases by a wide margin. Efforts to better prevent, recognize, and manage these injuries need to be implemented in the game of rugby. Developing accurate incidence data will assist us in raising awareness of the problem of concussion in rugby.

ACKNOWLEDGMENTS

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Commentary: Role of Properly Fitted Mouthguards in Prevention of Sport-Related Concussion

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Jackson E. Winters, Sr, DDS, provided conception and design, analysis and interpretation of the data, and drafting, critical revision, and final approval of the article.

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For many years, some experts have speculated that if athletes wear mouthguards in contact sports, many sport-related cerebral concussions might be prevented. The key reference for this position was a 1964 article in the *Journal of the American Dental Association* by Stenger et al.[1] Many other positions on the subject were established, most significantly by Hickey et al[2] in 1967 and by Chapman[3,4] in 1985 and 1990. Although no controlled experimental study has confirmed or refuted the idea that a properly fitted mouthguard reduces the likelihood of sustaining a concussive injury, evidence is mounting that the device could play a significant role in this critical area of player safety.

As sports and our culture evolve, the techniques of playing and coaching the activities improve. Today, there are more participants, both men and women, in recreational and organized sports. Players are bigger, stronger, and faster than their predecessors. The desire to win at all levels of competition, the number of college scholarships available, and the money professional athletes are making have created an environment more conducive for athletic injuries. With the new awareness and media attention being paid to such injuries, sport-related concussion is surfacing as a major concern.

In addition to education, we can make the playing of sports safer for participants by changing the rules of the game and by improving or changing the equipment. It is to the improvement of the equipment that this paper is directed.

My perspective comes from two unique positions. As a pediatric dentist over the years, I have used many different types of mouthguards with my patients. As a college football referee in the Mid-American Conference for 18 years, I have on-field observations of the types and quality of the mandatory mouthguards the players have been wearing. The four types of guards most commonly used are (1) stock over the counter, not mouth formed, (2) boil and bite, well worn, mouth formed, (3) cut-off, no posterior tooth coverage, and (4) custom fabricated over a model of the upper dental arch.[5] Through my involvement with the Academy for Sports Dentistry, I have discussed concussion prevention with many colleagues, including Dr John Stenger, and I have been able to establish the type of custom mouthguards constructed for the Notre Dame athletes in the 1964 study.[1] Over the past several football seasons, I have been directly involved with a series of ongoing studies investigating the role that properly fitted, pressure-laminated mouthguards can play in reducing concussive forces applied to the brain.

The methods used in our studies follow the Academy for Sports Dentistry position statement for properly fitted mouthguards.[6] The materials used were polyvinyl acetate copolymer (Dreve-Dentamid, Unna, Germany). Impressions were taken of the players' upper dental arches, and the protectors were fabricated over the models. The materials were formed with heat and pressure using a Drufomat machine (Westone Labs, Inc, Colorado Springs, CO). The posterior thickness varied from 3 to 4 millimeters in thickness after the final fitting (Figure 1).

Concussions result from the sudden acceleration or deceleration of the brain because of either direct or indirect (eg, rotational) forces. The strength of this force and its effect on the brain are directly related to the absolute value of the acceleration. From newtonian physics, acceleration refers to change in velocity, which is expressed in terms of units of time over units of distance:

$$a = (v^2 - v_0^2)/2s$$

In this formula, the letter a refers to acceleration, v_0 to initial velocity before the start of acceleration, v to the speed at the end of acceleration, and s to the distance traveled during acceleration. Consider that newtonian physics also supply the formula

$$F = ma,$$

where F signifies the force applied, m the mass of the object involved in the contact, and a the acceleration. As such,[5]

$$F = m\ (v^2 - v_0^2)/2s.$$

The unproven theory applied in this study, and essentially in the 1964 study by Stenger et al,[1] is that the properly fitted mouthguard effectively increases the time and distance involved in acceleration when the mechanism of injury is an upward blow to the head through the mandible. Note the separation in the pictures, with and without a properly fitted mouthguard in position, as well as the head of the condyle in relation to the base of the skull (Figures 2, 3).

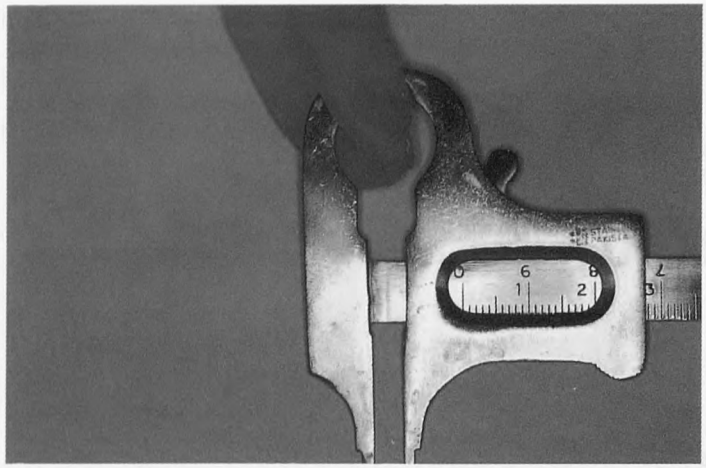

Figure 1. Properly fitted, pressure-laminated mouthguard with 3 to 4 mm of thickness posteriorly.

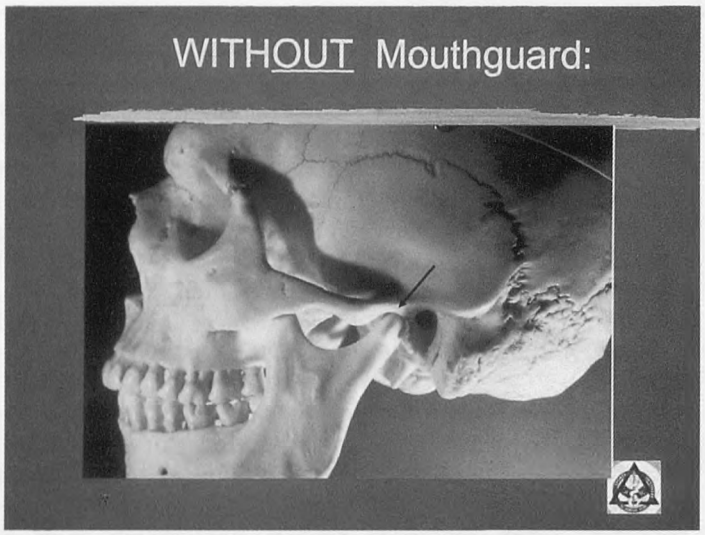

Figure 2. Note head of condyle and base of the skull without a properly fitted mouthguard. Figure courtesy of the Academy for Sports Dentistry.

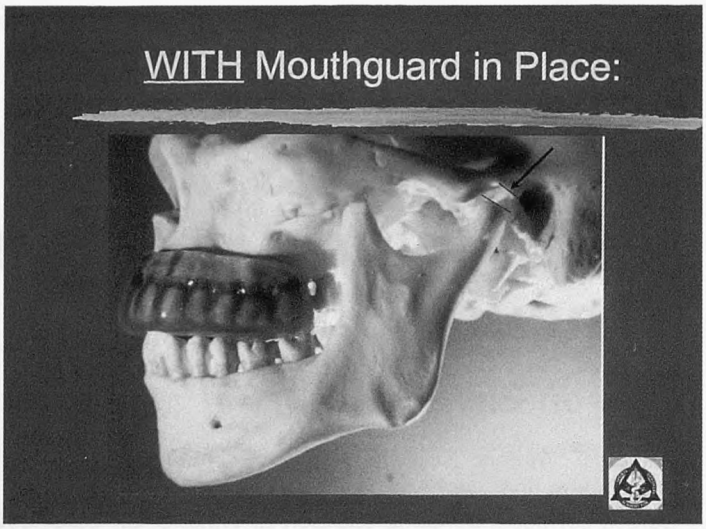

Figure 3. Note separation in space between the head of the condyle to the base of the skull with a properly fitted mouthguard in place. Figure courtesy of the Academy for Sports Dentistry.

The brain is a very difficult organ to study, and one must realize that concussions will likely always occur in collision and contact sports, such as football. Many factors are involved in reducing athletes' vulnerability to concussion, including genetics, anatomy, force of the blows, mass of the players, other equipment and rule changes, playing surfaces, coaching techniques, players being in harm's way when a force is present, and increasing knowledge about what constitutes a concussion.

At a time when clinicians and researchers are looking for answers to this very intriguing problem of sport-related concussion, it is important not to overlook some very obvious solutions. Although the techniques of fabrication and the materials have changed, the wheels of thought are returning 360 degrees to the 1960s and the study by Stenger et al.[1] Plastic face masks attached to helmets have reduced the incidence of dental and facial injuries in football; thus, the importance of wearing a mouthguard for concussion prevention may have been ignored. For example, mouthguards are not required equipment for National Football League players.[7] My contention is that over the past 35 years, quality has been sacrificed in exchange for a quick fix, ie, low-cost, ill-fitting mouthguards. These mouthguards do not hold their shape and fit so poorly that athletes sometimes alter them for speaking and comfort, forgoing the posterior thickness that might provide needed protection if a blow was delivered to the mandibular complex.[5]

Are properly fitted, pressure-laminated mouthguards an answer? Time will tell. Some of the preliminary work comparing the 1997 through 1999 NCAA college football seasons suggests that they have made a difference. The laminated mouthguards do not break down with use, and players do not tend to abuse or alter them. If a problem develops, correcting or reconstructing the mouthguard on the basis of the original model can easily be accomplished. In my opinion, and that of many other Academy for Sports Dentistry members, the theory makes sense. A posterior separation of 3 to 4 mm relates nicely to the newtonian laws of physics. This thickness converts to time and distance, over which acceleration occurs if a blow is delivered to the madibular complex.

CONCLUSIONS

From this theory and some of our early findings, it would appear that greater emphasis should be placed on the thickness in the posterior occlusal areas, especially in those high-contact sports requiring the use of mouthguards. Education of all those involved is the key. Team physicians, dentists, athletic trainers, and coaches must take into consideration both the athlete's previous medical history and the sport. Our emphasis must be on improving the quality of mouthguards for player safety as one way of attempting to reduce the incidence of concussion in athletes.

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NATA Research & Education Foundation

REQUEST FOR PROPOSALS

EXERCISE BY CHILDREN AND ADOLESCENTS IN WARM AND HOT ENVIRONMENTS

Background

Physiological responses to exercise in stressful environments may vary among participants. This is particularly true for hot environments in which impaired motor and mental function can result from hyperthermia. Dehydration of as little as 1% to 2% of body weight negatively affects physiologic function and athletic performance, while dehydration beyond 3% of body weight increases an athlete's risk of developing exertion-induced heat illness (NATA Position Statement: Fluid Replacement for Athletes, 2000). The effects of prolonged exercise under conditions of heat stress in adults have been studied extensively (ACSM Position Stand: Exercise and Fluid Replacement, 1996) Considerably less is known about how children and adolescents tolerate exercise in warm and hot environments.

The incidence of participation of children in sports has increased dramatically in the past decade, and children represent the largest group of individuals engaged in competitive sports in the United States. This growth in participation has outpaced efforts to clearly understand the childhood consequences of physical activity, of varying intensities, occurring under climatic stress. Children and adolescents possess a number of morphologic and functional characteristics that make them less efficient thermoregulators than adults. These include a larger surface area per unit body mass, higher O_2 cost of walking and running, lower cardiac output at a given O_2 uptake, and lower sweating rate. Furthermore, children appear to acclimatize to heat more slowly than do adults. The age-related differences in response to exercise in hot environments and their health consequences require further study.

Objectives

The NATA Research and Education Foundation encourages submission of high-quality research proposals that examine the medically and clinically relevant effects of prolonged exercise in hot environments on children and adolescents. Areas of interest may include but are not limited to: the physiological mechanisms underlying differences between children and adults in acclimating to exercising in warm and hot environments; hormonal factors affect sweating and other thermoregulatory mechanisms during puberty; gender-related differences in thermoregulation and heat acclimatization in pre-pubertal children; and the effects of repeated dehydration on growth and health in the child sports participant.

Procedure

Pre-proposal Submission - The NATA Foundation now requires that investigators interested in submitting a grant application to the NATA Foundation first submit a "Pre-proposal". The purpose of the Pre-proposal is to optimize the time invested by both the NATA Foundation Research Committee and the investigators in grant proposals submitted to the NATA Foundation. The Pre-proposal will allow the NATA Foundation Research Committee to evaluate whether or not the proposed research project is of interest to the NATA Foundation. The NATA Foundation Research Committee will evaluate the Pre-proposal both for subject matter (topic and hypotheses) and for research design/methodology. Based upon this evaluation, the committee will then either invite the submission of a full proposal or indicate that the proposed project is not of interest to the NATA Foundation. An invitation to submit a full proposal does not imply a commitment to funding. It does indicate that the topic is of potential interest to the NATA Foundation and that the general research design seems reasonable based on the information given in the Pre-proposal. A commitment to funding may occur only after a detailed review of the full proposal by the NATA Foundation Research Committee.

Instructions for Submission - The pre-proposal must be submitted in both hardcopy (2-page limit, single-spaced) and 3.5″ diskette. The applicant will receive results of the review within 6 weeks after the pre-proposal is received. Submission deadlines for full proposals are March 1 and September 1. The principal investigator must be explicit and concise in providing the following information:

1. Name, Credentials, Address, Phone, Fax, E-mail, Sponsoring Institution, Title of Proposal

2. Statement of the Problem. This section should contain a brief statement of the problem and should state explicitly how the project relates to athletic training and/or the healthcare of the physically active.

3. Specific Aims and Hypotheses. This section should present the specific questions to be addressed and the specific hypotheses that will be tested in the project. It is often helpful to present numbered specific aims accompanied by the associated hypotheses.

4. Experimental Design and General Methods. This section should contain a general outline of the research design of the proposed study, and should indicate what methods will be used to collect key data. There is no need to provide detailed descriptions of the methods.

Mail Completed Pre-proposal to:

Michael R. Sitler, EdD, ATC
Chair, NATA Foundation Research Committee
Department of Kinesiology, 114 Pearson Hall
Temple University, Philadelphia, PA 19122

NATA Research & Education Foundation

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PROCESS

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Please read all instructions before preparing the abstract. Individuals may submit only one abstract or clinical case report as primary (presenting) author, but may submit unlimited abstracts or clinical case reports as a co-author. All abstracts will undergo blind review. Authors may request a preference for oral or poster presentation of their abstracts. All presentations must be original (not previously presented). This restriction includes internet and worldwide web postings. Exceptions to this restriction are limited to state and district meetings of athletic training organizations.

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Specific Content Requirements

Free Communications abstracts must include the purpose of the study or hypothesis, a description of the subjects, the experimental methods and materials, the type(s) of data analysis, the results of the study, and the conclusion(s).

Instructions for Preparing the Abstract

1. Provide all information requested on the Abstract Author Information Form. Abstracts should be typed or word processed using a letter-quality printer with no smaller than elite (12 dpi) or 10-point typeface. Do not use a dot matrix printer.

2. Top, bottom, right, and left margins should be set at 1.5" using a standard 8.5" x 11" sheet of paper. Type the title of the paper or project starting at the left margin.

3. On the next line, indent 3 spaces and type the names of all authors, with the author who will make the presentation listed first. Type the last name, then initials (without periods), followed by a comma; continue with the other authors (if any), ending with a colon.

4. Indicate the institution (including the city and state) where the research or case report was conducted on the same line following the name(s) of the author(s).

5. Double space and begin typing the text of the abstract flush left in a single paragraph with no indentions. The text must be non-structured (i.e., no headings). Do not justify the right margin. **Do not include tables or figures.**

6. The abstract **must not exceed 400 words.**

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Clinical Case Report abstracts involve the presentation of unique individual athletic injury cases of general interest to our membership. No form is provided so that authors may use their own word-processing software to format and submit a clinical case report abstract **using a 700 word limit.** A maximum of one paragraph should be presented for each of the following required content area headings:

1) Personal data
2) Physical signs and symptoms
3) Differential diagnosis
4) Results of diagnostic imaging/laboratory tests
5) Clinical course
6) Deviation from the expected

Instructions for Preparing the Abstract

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2. Top, bottom, right, and left margins should be set at 1.5" using a standard 8.5" x 11" sheet of paper. Type the title of the paper or project starting at the left margin.

3. Provide all information requested on the Abstract Author Information Form. Please note that the institution (including the city and state) where the clinical case occurred should be cited, not the current address of the author(s), if different.

4. The title of the clinical case report should not contain information that may reveal the identity of the individual nor the specific nature of the medical problem to the reader. An example of a proper title for a clinical case report is "Chronic Shoulder Pain in a Collegiate Wrestler."

5. Complete the six different categories of information as required for a clinical case report abstract. These categories are:

   a. Personal Data/Pertinent Medical history (age, sex, sport/occupation of individual, primary complaint, and pertinent aspects of his/her medical history)
   b. Physical Signs and Symptoms (a brief summary of the physical findings)
   c. Differential Diagnosis (array of possible injuries/conditions)
   d. Results of Diagnostic Imaging/Laboratory Tests
   e. Clinical Course (e.g., diagnosis, treatment, surgical technique, rehabilitation program, final outcome)
   f. Deviation From the Expected (a brief description of what makes this case unique)

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(either Free Communications or Clinical Case Reports)

Complete the form and mail it, the original abstract, two photocopies of the original abstract, six (6) blind copies (showing no information about the authors or institution) of the abstract and a labeled 3.5" DISKETTE copy (preferably in WordPerfect or ASCII format; if you must send it in Macintosh format, please provide a text or rich text file, and use a high-density diskette) of your abstract to:

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ABSTRACTS POSTMARKED AFTER JANUARY 4, 2002, WILL NOT BE ACCEPTED.

NOTE: The NATA Foundation instituted a $25.00 Abstract Fee for each submission for presentation in 2001 and subsequent years. Please include a check payable to NATA Foundation with your abstract submission.

ABSTRACT AUTHOR INFORMATION FORM *(Please type)*

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Authors' Guide

(Revised January 2001)

The mission of the *Journal of Athletic Training* is to enhance communication among professionals interested in the quality of health care for the physically active through education and research in prevention, evaluation, management, and rehabilitation of injuries.

SUBMISSION POLICIES

1. Submit 5 copies of the entire manuscript (including tables and figures) to *Journal of Athletic Training* Submissions, Hughston Sports Medicine Foundation, Inc, 6262 Veterans Parkway, PO Box 9517, Columbus, GA 31908–9517. The term "figure" refers to items that are not editable, either halftones (photographs) or line art (charts, graphs, tracings, schematic drawings), or combinations of the two. A table is an editable item that needs to be typeset.
2. All manuscripts must be accompanied by a letter signed by each author and must contain the following statements: "This manuscript 1) contains original unpublished material that has been submitted solely to the *Journal of Athletic Training,* 2) is not under simultaneous review by any other publication, and 3) will not be submitted elsewhere until a decision has been made concerning its suitability for publication by the *Journal of Athletic Training.* In consideration of the NATA's taking action in reviewing and editing my submission, I the undersigned author hereby transfer, assign, or otherwise convey all copyright ownership to the NATA, in the event that such work is published by the NATA. Further, I verify that I have contributed substantially to this manuscript as outlined in item #3 of the current Authors' Guide." By signing the letter, the authors agree to comply with all statements. Manuscripts that are not accompanied by such a letter will not be reviewed. Accepted manuscripts become the property of the NATA. Authors agree to accept any minor corrections of the manuscript made by the editors.
3. Beginning with volume 36, the contribution of each author will be specifically identified in the published manuscript, in accordance with the Uniform Requirements for Manuscripts Submitted to Biomedical Journals: "Authorship credit should be based only on 1) substantial contributions to conception and design, or acquisition of data, or analysis and interpretation of data; 2) drafting the article or revising it critically for important intellectual content; and 3) final approval of the version to be published. Conditions 1, 2, and 3 must all be met. Acquisition of funding, the collection of data, or general supervision of the research group, by themselves, do not constitute authorship." For additional information, please visit the Uniform Requirements website: http://www.icmje.org/index.html.
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4. Financial support or provision of supplies used in the study must be acknowledged. Grant or contract numbers should be included whenever possible. The complete name of the funding institution or agency should be given, along with the city and state in which it is located. If individual authors were the recipients of funds, their names should be listed parenthetically.
5. Authors must specify whether they have any commercial or proprietary interest in any device, equipment, instrument, or drug that is the subject of the article in question. Authors must also reveal if they have any financial interest (as a consultant, reviewer, or evaluator) in a drug or device described in the article.
6. For experimental investigations of human or animal subjects, state in the Methods section of the manuscript that an appropriate institutional review board approved the project. For those investigators who do not have formal ethics review committees (institutional or regional), the principles outlined in the Declaration of Helsinki should be followed (41st World Medical Assembly. Declaration of Helsinki: recommendations guiding physicians in biomedical research involving human subjects. *Bull Pan Am Health Organ.* 1990;24:606–609). For investigations of human subjects, state in the Methods section the manner in which informed consent was obtained from the subjects. (Reprinted with permission of *JAMA* 1997;278:68, copyright 1997, American Medical Association.)
7. Signed releases are required to verify permission for the *Journal of Athletic Training* to 1) reproduce materials taken from other sources, including text, figures, or tables; 2) reproduce photographs of individuals; and 3) publish a Case Report. A Case Report cannot be reviewed without a release signed by the individual being discussed in the Case Report. Release forms can be obtained from the Editorial Office and from the *JAT* web page, or authors may use their own forms.
8. The *Journal of Athletic Training* uses a double-blind review process. Authors should not be identified in any way except on the title page.
9. Manuscripts are edited to improve the effectiveness of communication between author and readers and to aid the author in presenting a work that is compatible with the style policies found in the *AMA Manual of Style,* 9th ed. (Williams & Wilkins), 1998. Page proofs are sent to the author for proofreading when the article is typeset for publication. It is important that they be returned within 48 hours. Important changes are permitted, but authors will be charged for excessive alterations.
10. Published manuscripts and accompanying work cannot be returned. Unused manuscripts will be returned if submitted with a stamped, self-addressed envelope.

STYLE POLICIES

11. Each page must be printed on 1 side of 8½-by-11-inch paper, double spaced, with 1-inch margins in a font no smaller than 10 points. Each page should include line counts to facilitate the review process. Do not right justify pages.
12. Manuscripts should contain the following, organized in the order listed below, with each section beginning on a separate page:
    a. Title page
    b. Acknowledgments
    c. Abstract and Key Words (first numbered page)
    d. Text (body of manuscript)
    e. References
    f. Tables (each on a separate page)
    g. Legends to figures
    h. Figures
13. Begin numbering the pages of your manuscript with the abstract page as #1; then, consecutively number all successive pages.
14. Units of measurement shall be recorded as SI units, as specified in the *AMA Manual of Style,* except for angular displacement, which should be measured in degrees rather than radians. Examples include mass in kilograms (kg), height in centimeters (cm), velocity in meters per second ($m \cdot s^{-1}$ or m/s), angular velocity in degrees per second ($^\circ \cdot s^{-1}$), force in Newtons (N), and complex rates (mL/kg per minute).
15. Titles should be brief within descriptive limits (a 16-word maximum is recommended). If a disability is the relevant factor in an article, the name of the disability should be included in the title. If a technique is the principal reason for the report, it should be in the title. Often both should appear.
16. The title page should also include the name, title, and affiliation of each author, and the name, address, phone number, fax number, and e-mail address of the author to whom correspondence is to be directed. No more than 3 credentials should be listed for each author.
17. A structured abstract of no more than 250 words must accompany all manuscripts. Type the complete title (but not the authors' names) at the top, skip 2 lines, and begin the abstract. Items that are needed differ by type of article. **Literature Reviews**: Objective, Data Sources, Data Synthesis, Conclusions/Recommendations, and Key Words; **Original Research** articles: Objective, Design and Setting, Subjects, Measurements, Results, Conclusions, and Key Words**; Case Reports**: Objective, Background, Differential Diagnosis, Treatment, Uniqueness, Conclusions, and Key Words; **Clinical Techniques**: Objective, Background, Description, Clinical Advantages, and Key Words. For the Key Words entry, use 3 to 5 words that do not appear in the title.
18. Begin the text of the manuscript with an introductory paragraph or two in which the purpose or hypothesis of the article is clearly stated and developed. Tell why the study needed to be done or the article written and end with a statement of the problem (or controversy). Highlights of the most prominent works of others as related to your subject are often appropriate for the introduction, but a detailed review of the literature should be reserved for the discussion section. In a 1- to 2-paragraph review of the literature, identify and develop the magnitude and significance of the controversy, pointing out differences among others' results, conclusions, and/or opinions. The introduction is not the place for great detail; state the facts in *brief,* specific statements and reference them. The detail belongs in the discussion. Also, an overview of the manuscript is part of the abstract, not the introduction. Writing should be in the active voice (for example, instead of "Subjects were selected," use "We selected subjects") and in the first person (for example, instead of "The results of this study showed," use "Our results showed").
19. The body or main part of the manuscript varies

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according to the type of article (examples follow); however, the body should include a discussion section in which the importance of the material presented is discussed and related to other pertinent literature. When appropriate, a discussion subheading on the clinical relevance of the findings is recommended. Liberal use of headings and subheadings, charts, graphs, and figures is recommended.

a. The body of an **Original Research** article consists of a methods section, a presentation of the results, and a discussion of the results. The methods section should contain sufficient detail concerning the methods, procedures, and apparatus employed so that others can reproduce the results. The results should be summarized using descriptive and inferential statistics and a few well-planned and carefully constructed illustrations.
b. The body of a **Literature Review** article should be organized into subsections in which related thoughts of others are presented, summarized, and referenced. Each subsection should have a heading and brief summary, possibly one sentence. Sections must be arranged so that they progressively focus on the problem or question posed in the introduction.
c. The body of a **Case Report** should include the following components: personal data (age, sex, race, marital status, and occupation when relevant—not name), chief complaint, history of present complaint (including symptoms), results of physical examination (example: "Physical findings relevant to the rehabilitation program were . . . "), medical history (surgery, laboratory results, examination, etc), diagnosis, treatment and clinical course (rehabilitation until and after return to competition), criteria for return to competition, and deviation from expectations (what makes this case unique).
d. The body of a **Clinical Techniques** article should include both the *how* and *why* of the technique: a step-by-step explanation of how to perform the technique, supplemented by photographs or illustrations, and an explanation of why the technique should be used. The discussion concerning the *why* of the technique should review similar techniques, point out how the new technique differs, and explain the advantages and disadvantages of the technique in comparison with other techniques.

20. Percentages should be accompanied by the numbers used to calculate them. When reporting nonsignificant results, a power analysis should be provided.
21. **Communications** articles, including official Position Statements and Policy Statements from the NATA Pronouncements Committee; technical notes on such topics as research design and statistics; and articles on other professional issues of interest to the readership are solicited by the *Journal.* An author who has a suggestion for such a paper is advised to contact the Editorial Office for instructions.
22. The manuscript should not have a separate summary section—the abstract serves as a summary. It is appropriate, however, to tie the article together with a summary paragraph or list of conclusions at the end of the discussion section.
23. References should be numbered consecutively, using superscripted arabic numerals, in the order in which they are cited in the text. References should be used liberally. It is unethical to present others' ideas as your own. Also, use references so that readers who desire further information on the topic can benefit from your scholarship.
24. References to articles or books, published or accepted for publication, or to papers presented at professional meetings are listed in numerical order at the end of the manuscript. Journal title abbreviations conform to *Index Medicus* style. Examples of references are illustrated below. See the *AMA Manual of Style* for other examples.

    Journals:
    1. van Dyke JR III, Von Trapp JT Jr, Smith BC Sr. Arthroscopic management of postoperative arthrofibrosis of the knee joint: indication, technique, and results. *J Bone Joint Surg Br.* 1995;19:517–525.
    2. Council on Scientific Affairs. Scientific issues in drug testing. *JAMA.*1987;257: 3110–3114.

    Books:
    1. Fischer DH, Jones RT. *Growing Old in America.* New York, NY: Oxford University Press Inc; 1977:210–216.
    2. Spencer JT, Brown QC. Immunology of influenza. In: Kilbourne ED, Gray JB, eds. *The Influenza Viruses and Influenza.* 3rd ed. Orlando, FL: Academic Press Inc; 1975:373–393.

    Presentations:
    1. Stone JA. Swiss ball rehabilitation exercises. Presented at: 47th Annual Meeting and Clinical Symposia of the National Athletic Trainers' Association; June 12, 1996; Orlando, FL.

    Internet Sources:
    1. Knight KL, Ingersoll CD. Structure of a scholarly manuscript: 66 tips for what goes where. Available at http://www.journalofathletictraining.org/jat/66tips.html. Accessed January 1, 1999.
    2. National Athletic Trainers' Association. NATA blood borne pathogens guidelines for athletic trainers. Available at http://www.journalofathletictraining.org. Accessed January 1, 1999.
25. Table Style: 1) Title is bold; body and column headings are roman type; 2) units are set above rules in parentheses; 3) numbers are aligned in columns by decimal; 4) footnotes are indicated by symbols (order of symbols: *, †, ‡, §, ‖, ¶); 5) capitalize the first letter of each major word in titles; for each column or row entry, capitalize the first word only. See a current issue of the *Journal* for examples.
26. All black-and-white line art should be submitted in camera-ready form. Line art should be of good quality; should be clearly presented on white paper with black ink, sans serif typeface, and no box; and should be printed on a laser printer—no dot matrix. Figures that require reduction for publication must remain readable at their final size (either 1 column or 2 columns wide). Photographs should be glossy black and white prints. Do not use paper clips, write on photographs, or attach photographs to sheets of paper. On the reverse of each figure attach a write-on label with the figure number, name of the author, and an arrow indicating the top. (Note: Prepare the label before affixing it to the figure.) Authors should submit 1 original of each figure and 4 copies for review.
27. Authors must request color reproduction in a cover letter with the submitted manuscript. Authors will be notified of the additional cost of color reproduction and must confirm acceptance of the charges in writing.
28. Legends to figures are numbered with arabic numerals in order of appearance in the text. Legends should be printed on separate pages at the end of the manuscript.
29. The *Journal of Athletic Training* follows the redundant publication guidelines of the Council of Science Editors, Inc (*CBE Views.* 1996; 19:76–77; also available on the *JAT* web site at http://www.journalofathletictraining.org). Authors found in violation of redundant publication will have sanctions invoked by the Journal Committee of the National Athletic Trainers' Association, Inc.

PUBLICATION POLICIES

30. Original Research manuscripts will be categorized under the following table of contents subheadings: clinical studies, basic science, educational studies, epidemiologic studies, and observational/informational studies.
31. Only Case Reports and Clinical Techniques that define and establish the optimal standard of care or the practice of athletic training will be considered for publication in *JAT.* All other Case Reports and Clinical Techniques will be considered for publication in the *NATA News.*
32. Media Reviews will appear in the *NATA News.*

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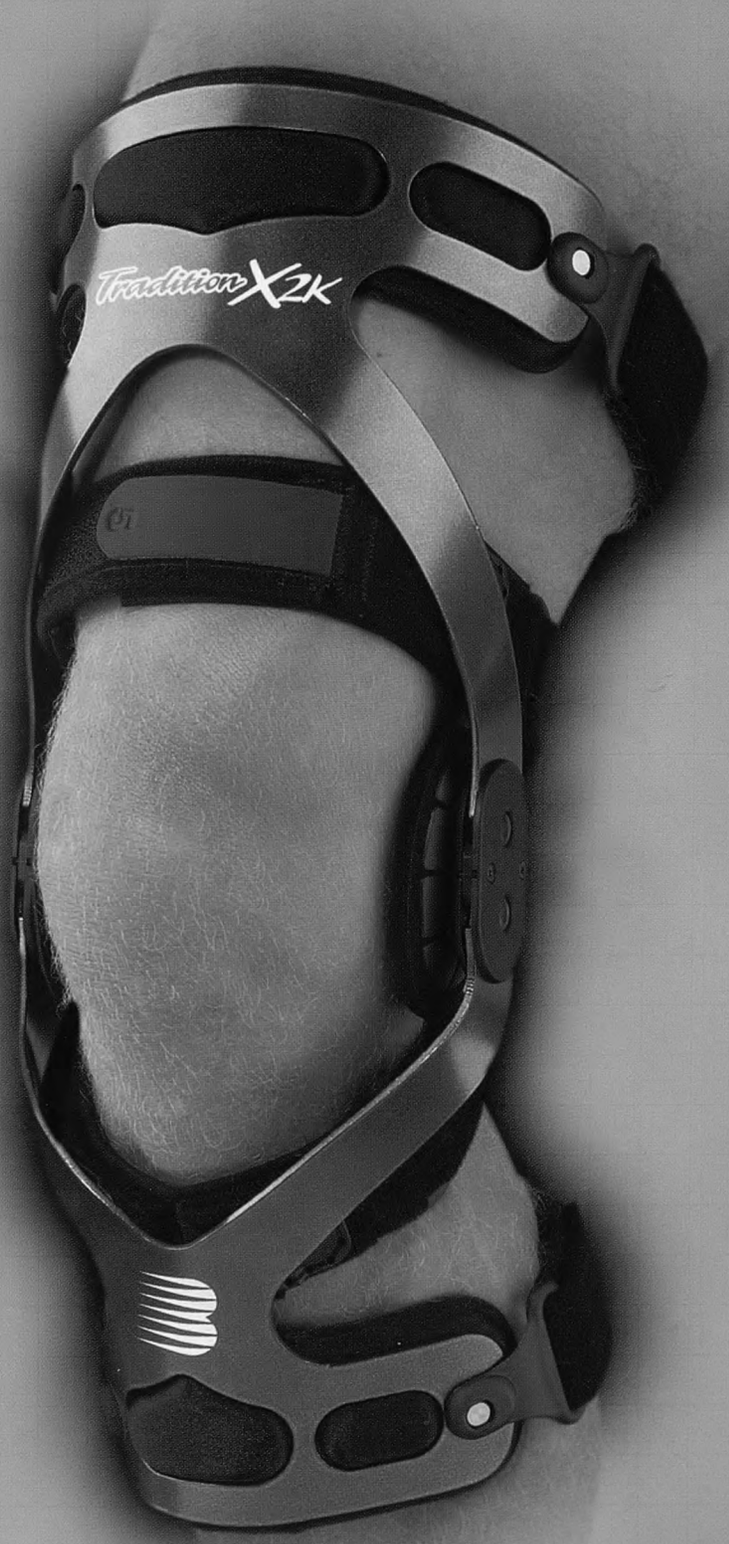

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