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Current Concepts

Concussion in Sports*

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From the AOSSM Concussion Workshop Group, Rosemont, Illinois

This is a special report of the findings of the Concussion Workshop, sponsored by the AOSSM in Chicago in December 1997. Here follows a listing of the members of the workshop: Julian Bailes, MD, American Association of Neurological Surgeons; Arthur Boland, MD, AOSSM; Charles Burke III, MD, National Hockey League; Robert Cantu, MD, American College of Sports Medicine; Letha "Etty" Griffin, MD, National Collegiate Athletic Association; David Hovda, PhD, Neuroscientist, UCLA School of Medicine; Mary Lloyd Ireland, MD, American Academy of Orthopaedic Surgeons; James Kelly, MD, American Academy of Neurology; Greg Landry, MD, American Academy of Pediatrics; Mark Lovell, PhD, Neuropsychology Specialist, Henry Ford Health Systems; James Mathews, MD, American College of Emergency Physicians; Michael McCrea, PhD, Neuropsychology Specialist, Waukesha Memorial Hospital; Douglas McKeag, MD, American Medical Society for Sports Medicine; Dennis Miller, ATC, National Athletic Trainers Association; Jeffrey Minkoff, MD, AOSSM; Stephen Papadopoulus, MD, Congress of Neurological Surgeons; Elliott Pellman, MD, National Football League; Richard Quincy, MS, PT, ATC, Sports Physical Therapy, El Pomar Sports Center; Herbert Ross, DO, American Osteopathic Academy of Sports Medicine; Bryan Smith, MD, National Collegiate Athletic Association; and Edward Wojtys, MD, Workshop Chairman, AOSSM.

The views in this report do not necessarily represent the views of the entire group comprising the Concussion Workshop Group.

One of the most challenging problems faced by medical personnel responsible for the health care of athletes is the recognition and management of concussions.[40,53] Concussions can be defined as any alteration in cerebral function caused by a direct or indirect (rotation) force transmitted to the head resulting in one or more of the following acute signs or symptoms: a brief loss of consciousness, lightheadedness, vertigo, cognitive and memory dysfunction, tinnitus, blurred vision, difficulty concentrating, amnesia, headache, nausea, vomiting, photophobia, or a balance disturbance. Delayed signs and symptoms may also include sleep irregularities, fatigue, personality changes, an inability to perform usual daily activities, depression, or lethargy. Although many concussions are mild, the range of injury is wide. Nevertheless, concussions are a form of traumatic brain injury.

In recent years, these injuries have captured many news headlines as several professional football and hockey players have retired because of the effects of concussions. Interestingly, depending on the nature of the sport and the type (for example, rotation) and degree of contact expected, these injuries are many times viewed as just "part of the game." While many of these injuries are minor, some can be quite serious, with long-term consequences. Therefore, early detection through a thorough knowledge of the signs and symptoms and specific documentation of the injury is critical to the management of concussion and the monitoring of the natural history of the injury.[36,40] Unfortunately, attempts to characterize and classify the spectrum of concussions by stratifying the signs and symptoms as indicators of relative severity have been difficult.[7,10] Yet the need to accurately diagnose the severity of these injuries is obvious, especially at the time of injury when the triage decision could be critical to the patient's future. Returning an injured athlete to competition when the brain needs time to recover is an obvious concern. One of the reasons for concern is the second-impact syndrome,[24,29,52,59] a rare but ominous consequence of an untimely blow to a vulnerable central nervous system. While recent reviews cast a shadow of doubt on the occurrence and frequency,[51] the catastrophic nature of these events requires its consideration in the evaluation and treatment of concussions. Also, the cumulative effects of repeated injuries, even mild injuries, over time remains a serious concern to those involved in sports medicine.[15] The fact that some athletes do not recover as

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expected from concussions and are hampered by persistent symptoms for weeks or months is troublesome. In 1999, a complete understanding of the pathobiology of cerebral concussion is still lacking,[66] as is an explanation as to why the brain of some athletes may become so vulnerable to secondary injury after a seemingly mild insult.

Because of these lingering concerns, an American Orthopaedic Society for Sports Medicine-sponsored Concussion Workshop was held in December 1997 to assemble representatives from the medical community who routinely diagnose and treat these injuries in athletes. Invited participants included health-care professionals who perform research on brain injuries, a variety of clinicians responsible for the care of the athlete, and representatives of organized contact sports (NFL, NHL, NCAA). These representatives met with the hope of defining areas of agreement and disagreement in the detection and management of concussion in sports.

Realizing that differences do exist among clinicians regarding the safety of return-to-play at various time points after concussion, defining areas of disagreement was also a goal of the concussion workshop so that these differences could be subjected to discussion and investigation. Lastly, participants focused on the key elements of the initial evaluation of concussion so that data collection, future studies, and follow-up reports could benefit from the use of common terminology and evaluation tools.

NEUROBIOLOGY OF CEREBRAL CONCUSSION: WHY IS THE BRAIN SO VULNERABLE AFTER A CONCUSSION?

Cerebral concussion has been and continues to be defined primarily in neurologic terms related to an altered state of consciousness and to neuropsychologic variables associated with transient (or lasting) deficits in cognition and various other symptoms. Unfortunately, at this time there are no objective neuroanatomic or physiologic measurements that can be used to determine if a patient has sustained a concussion or to assess the severity of the insult. Neurologic and neurophysiologic studies have gone to great lengths to garner measurement instruments through an understanding of traumatic brain injury unconsciousness and the corresponding cognitive disruption that is associated with concussion.[15,17,38,41,54] These studies have provided information regarding the clinical indications of the degree of severity while highlighting the incident rate of minor head injury in sporting activities. However, at present, we lack a complete understanding of the pathobiology of cerebral concussion and an explanation of why, after a seemingly mild insult, the brain may become so vulnerable to secondary injury in some patients.

Essential to this discussion is the fact that during the minutes to few days after concussion injury, brain cells that are not irreversibly destroyed remain alive but exist in a vulnerable state. This concept of injury-induced vulnerability has been put forth to describe the fact that patients suffering from head injury are extremely vulnerable to the consequences of even minor changes in cerebral blood flow and/or increases in intracranial pressure and apnea.[13,24,53] In animal models of head injury, experiments have indicated that up to 3 days after either a concussion or a cerebral contusion, a reduction in cerebral blood flow, which would normally be well tolerated, now produces extensive neuronal cell loss.[29,30,42,43,63] Although not understood in terms of its underlying cellular mechanism, this concept of injury-induced vulnerability is a major concern in the management of all patients with head injuries and is not confined to second-impact syndrome.[13]

Experimental studies have identified metabolic dysfunction as the key postconcussion physiologic event that produces and maintains this state of vulnerability. This period of enhanced vulnerability is characterized by both an increase in the demand for glucose (fuel) and an inexplicable reduction in cerebral blood flow (fuel delivery).[58] The result is an inability of the neurovascular system to respond to increasing demands for energy to reestablish its normal chemical and ionic environments. This is dangerous because these altered environments can kill brain cells.

This initial injury-induced increase in the demand for glucose is primarily the response of cells activating sodium-potassium pumps.[1,33,72] This increased demand for energy occurs immediately on injury as brain cells are exposed to a massive ionic flux, including the resultant increased levels of extracellular potassium ($[K^+]_e$).[32] The $[K^+]_e$ elevation is linked to the stimulation of excitatory amino acid receptors since it can be drastically attenuated by blocking glutamatergic receptors before injury.[31] Furthermore, this injury-induced increase in $[K^+]_e$ activates ATP-dependent sodium-potassium pumps, resulting in a tremendous metabolic stress to already damaged neural tissue.[33,72]

The acute increase in glycolytic energy demand after traumatic brain injuries has been demonstrated experimentally, both in animals[2,25,27,33,71,72] and in severely injured patients.[5] In addition, this injury-induced increase in glucose metabolism has been shown to occur in the presence of low cerebral blood flow.[55,69,70,73] These findings provide compelling evidence that the ratio between the use of fuel (glucose) and fuel delivery (cerebral blood flow) is out of balance. Moreover, in studies using animals, this mismatch between glucose demand and fuel availability can be seen after mild concussion brain injury.[26,32] From these studies it appears that in virtually all types and severity of head injuries, the dramatic increase in glucose metabolism during the first minutes to days after injury is a fundamental part of a cellular pathophysiologic cascade seen after cerebral concussion. Correlating these cellular changes to clinical manifestations is a continuous challenge for researchers and clinicians alike.

The increase in cerebral glucose metabolism after human head injury has been demonstrated recently in severe head injuries.[5] Using positron-emission tomography for assessing the local cerebral metabolic rate for glucose (intracerebral metabolic rate glucose [ICMRglu], measured in milligrams per 100 grams per minute), the ICMRglu is markedly

increased during the first few days after head injury, with evidence of increases lasting up to 1 week. This is all part of a disturbance in metabolic autoregulation that includes the experimentally proven increase in glucose metabolism and the relative reduction in cerebral blood flow seen in some concussion patients. In an attempt to establish a theory of prognostication, it is of value to note that outcomes in animal studies vary with the rate of cerebral blood flow recovery. Unfortunately, the acute increase in glucose metabolism in some patients is associated with a relative reduction in cerebral blood flow.[60] However, as in animal studies, the patients with the greatest degree and rate of cerebral blood flow recovery achieve the best outcome. Given the experimental data described, it is not surprising that the duration of reduced cerebral blood flow is an important prognostic factor in the outcome of patients with head injury. Consequently, the sustained reduction in cerebral blood flow due to the loss of metabolic autoregulation can potentiate the adverse effects of subsequent injuries, leading to more pronounced neural degeneration and a worse outcome.

Interestingly, there appears to be a strong relationship between the concentration of Ca^{2+} and regional control of cerebral blood flow and, in fact, a close association has been shown between the regions of the brain exhibiting Ca^{2+} flux and the area of reduced cerebral blood flow after traumatic brain injury. To better understand the altered physiology present, autoradiographic images of $^{45}Ca^{2+}$ have been compared with those obtained via [^{14}C]iodoantipyrine for the assessment of cerebral blood flow.[57] In rats, cerebral blood flow was measured at various times after fluid percussion ("closed-head") and cortical impact ("open-head") brain injuries.[14,42] These results indicated that for all cortical, hippocampal, and basal forebrain structures assessed, there were immediate decreases in cerebral blood flow that gradually resolved over 3 days. The magnitude in the reduction of cerebral blood flow was correlated with the amount of Ca^{2+} influx near the site of injury. This finding, coupled with the well-documented effect that Ca^{2+} channel blockers (nimodipine and dextromethorphan) have on the ability to increase cerebral blood flow under various brain injury conditions,[28,44,45,62] lend strong support to the contention that the reduction in cerebral blood flow after traumatic brain injury is due to the effect that Ca^{2+} has on cerebral blood flow.

The reduction in cerebral blood flow after traumatic brain injury is likely due to an increased vasoconstriction caused by endothelial accumulation of Ca^{2+}. Recently, several laboratories have reported that the elevation of extracellular Ca^{2+} potentiates the neurovascular constriction of central and peripheral arterioles.[19,22,64,65] These studies demonstrate that the twofold elevation of extracellular Ca^{2+} (from 2 to 4 mM) dramatically enhances the vasoconstriction induced by increased capillary pressure,[22] neuropeptide Y,[16] angiotensin II,[65] and endothelins.[65] Although the mechanism by which Ca^{2+} mediates vasoconstriction remains unknown, it appears to involve protein kinase C as an intermediate step.[65] Additional evidence derived from binding studies suggests that the levels of extracellular Ca^{2+} facilitate the extent of vasoconstriction. These studies revealed high levels of L-type calcium channels in arteriole branch points.[19]

Whatever the mechanism, it is now quite clear that although cerebral concussion may not, in and of itself, produce extensive neuroanatomic damage, the surviving cells are placed in a state of vulnerability. This vulnerability is perhaps best characterized in terms of a metabolic dysfunction. The cascade of events leading to this dysfunction is multidimensional, resulting initially in acute periods of hyperglycolysis, followed by a more chronic period of metabolic depression. However, in general terms, this dysfunction may be thought of as a breakdown in the harmony between energy demand, production, and delivery.

The fact that this energy crisis exists after severe head injury is now documented.[5] However, the critical question for sports medicine is, does this metabolic alteration occur with lesser degrees of injury in humans and, if so, how long does the crisis last? Only a few experiments have addressed this question.[12,23] Work with a cerebral concussion model in the rodent indicates that a state of metabolic dysfunction can last for as long as 10 days. Whether this period correlates with the period of cellular vulnerability in man or with the results of neuropsychologic testing is not yet known, nor is it clear that the time frame would be similar in man.

When comparing head injury-induced pathophysiology in animals to what is seen clinically, the time course of events appears to be longer in human patients. This is particularly true for changes in cerebral metabolism and alterations in extracellular neurochemistry. For example, cerebral hyperglycolysis has been observed for as long as 2 weeks after human head injury, with the subsequent metabolic depression being present for over 1 year. In contrast, experimental studies using animals rarely report hyperglycolysis after 6 hours, and the recovery from metabolic depression typically occurs within a few weeks. As already reviewed, these physiologic changes after cerebral concussion play an important role in terms of cellular vulnerability. Consequently, when trying to estimate the length of time after clinical concussion during which the brain is vulnerable, extrapolation from animal studies will most likely result in estimates that are too short.

INITIAL EVALUATION—ANTICIPATION, AWARENESS, PREPARATION

The goal of this workshop section on initial evaluation was to define and prioritize the steps that should be taken by medical personnel responding to an athlete who has sustained a potential concussion. The evaluation process has been subdivided into those measures that should be addressed on the playing field, when an athlete is down, and those that can be performed on the sideline after the player has either been removed from the playing surface or has come off the field independently.

On-the-Field Evaluation

The most important objective of on-the-field evaluation is to make an accurate and complete diagnosis of the level of

consciousness and to rule out the presence of significant associated injuries, especially to the cervical spine. Those responsible for the care of athletic teams must have a plan formulated in advance that should include a routine protocol for assessing athletes with head injuries. This should include the presence of adequately trained personnel, appropriate equipment, and an emergency back-up plan to evacuate a critically injured player safely and promptly, should it become necessary. Medical personnel must review these procedures before the season and be assured that all responsible persons understand the routine.

Medical personnel must understand the mechanisms of head injury, realizing that concussions may occur either by direct contact of the head against a hard surface or from sudden rotational or shear forces transmitted to the brain. Rapid acceleration or deceleration of the head and neck from a whiplash type of force can be as harmful as direct contact with a hard surface. Whereas a brief attempt to determine the mechanism of injury is advisable, prolonged questioning about the mechanism should not delay the initial assessment on the field. When approaching a player who is injured, observing the posture of the athlete and noting any spontaneous motion or verbalization from the player is the first step. Total lack of motion in the extremities should always alert those at the scene to the potential for a cervical spine injury. Incoherent speech would suggest a significant concussion. The player's helmet should not be removed initially unless a cervical spine injury can be ruled out. The face guard may require removal in emergent situations.

The ABCs of Evaluation. The respondents' initial obligation is to determine whether the injured player is breathing spontaneously, has an unobstructed airway, and has a pulse. Second, medical personnel should quickly determine whether further evaluation on the sidelines is appropriate or whether emergent transport to a hospital is needed. For the cardiovascular assessment, the carotid and radial pulses are usually the most accessible. If the patient has an adequate airway, respiration, and pulse, the initial assessment of the level of consciousness should be performed in the position in which the athlete lies. If the player is unconscious, one must assume that the athlete has an associated cervical spine injury until proven otherwise.

In the absence of a pulse and adequate respiration, the neck should be stabilized by an experienced person. With the assistance of two or three trained personnel, the athlete may then be log-rolled into a supine position so that cardiopulmonary resuscitation can be initiated effectively.

Athletes with closed-head injuries frequently have a blank expression, may appear confused, exhibit delayed verbal responses, and seem emotionally labile. The standard method of assessing the level of consciousness is by establishing a Glasgow Coma Scale[57] rating (Table 1). By observing the patient's eyes and motor and verbal responses, one can quantify the level of consciousness. A Glasgow Coma Scale of 11 or higher is usually associated with an excellent prognosis for recovery. On the other hand, a Glasgow Coma Scale of 7 or less is considered very serious.

TABLE 1
Glasgow Coma Scale[a]

Response	Point/s	Action
Eye opening		
Spontaneously	4	Reticular activity system is intact; patient may not be aware
To verbal command	3	Opens eyes when told to do so
To pain	2	Opens eyes in response to pain
None	1	Does not open eyes to any stimuli
Verbal		
Oriented, converses	5	Relatively intact CNS; aware of self and environment
Disoriented, converses	4	Well articulated, organized, but disoriented
Inappropriate words	3	Random, exclamatory words
Incomprehensible	2	Moaning, no recognizable words
No response	1	No response or intubated
Motor		
Obeys verbal commands	6	Readily moves limbs when told to
Localizes to painful stimuli	5	Moves limb in an effort to avoid pain
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Extension	2	Decerebrate rigidity
No response	1	Hypotonic, flaccid: suggests loss of medullary function or concomitant cord injury

[a] Normal, 15.

The athlete's orientation to time, place, and person should be determined by asking the date, month, day of the week, the score, the period of the game, or the play in which he or she was injured. It is also important to establish the presence of retrograde amnesia, which is associated with a more significant injury. This can be done by asking about events earlier in the day, such as what was consumed for breakfast, how the athlete traveled to the game, or the location of the locker room. The presence of symptoms such as dizziness, blurring of vision, and head or neck pain should be noted before moving the patient.

When an associated cervical spine injury has been ruled out and the level of confusion and orientation has improved to the point where the athlete can understand and follow commands, the patient may be assisted into a sitting position. This position will often decrease intracranial pressure and help to relieve the patient's confusion and apprehension. Keep the patient in the sitting position until you are satisfied that the symptoms are improving and that the athlete has adequate strength, coordination, and orientation to follow instructions. At this point, the athlete may be assisted into the standing position with people on either side for support. If the athlete is unsteady in the upright position, it is safer to remove him or her

from the field seated in a motorized cart or on a stretcher. However, if the athlete does have adequate strength and coordination, he or she can be assisted from the field, being sure there are people on either side for assistance, if necessary.

On-the-Bench Evaluation

When a player with a head injury is brought to the sidelines, he or she should be thoroughly evaluated in a routine manner to further define the level of injury. This should include a review of symptoms, a careful neurologic examination, and neuropsychologic testing. Players with concussion are frequently confused, irritable, and, at times, even combative. They frequently ask to be left alone. It is preferable to take the player to a quiet spot on the sidelines near the end of the bench or into the locker room. The player should be questioned about the symptoms of dizziness, light-headedness, vertigo, blurring or double vision, photophobia, ringing in the ears, headache, nausea, and vomiting. Many of these symptoms may be present initially after an acute head injury, while headache, nausea, and vomiting may not become evident for several minutes after the precipitating trauma. Vomiting is not very common after athletic injuries, but when it is present, it suggests a significant injury with elevated intracranial pressure and should be cause for concern.

The initial clinical examination should also include careful inspection and palpation of the head and neck followed by a careful neurologic evaluation. A baseline evaluation is important to accurately appreciate any changing clinical signs and symptoms in a deteriorating situation. In all contact injuries to the head or facial region, particularly those in which a helmet is not worn, the scalp, skull, and facial bones should be palpated, in search of lacerations and tenderness. If there is a laceration, it should be cleansed and then inspected carefully with a sterile glove for crepitus, which is indicative of an underlying skull fracture. The periorbital, mandibular, and maxillary areas should be carefully palpated after blunt trauma. Having an athlete open and close the mouth and clench the teeth will often lead to detection of a malocclusion or pain secondary to a mandibular fracture. The nose should also be observed for deformity and palpated for crepitus and tenderness in facial injuries. The presence of clear fluid around the nose (rhinorrhea) is indicative of a skull fracture in the cribriform plate region.

The neurologic examination should include a careful eye examination. About 3% of the population has one pupil larger than the other (anisocoria). This should have been detected on a preparticipation physical examination and the information should be available in the athlete's record. A direct blow to the face can result in a unilateral dilation of the pupil due to sympathetic nerve response. Serious head injuries, such as a skull fracture or subdural hematoma, may damage the third cranial nerve (oculomotor), but this is generally evident later in the clinical course. It is, therefore, essential to have a baseline evaluation of the size and symmetry of the pupils to appreciate subsequent changes that may result from increasing intracranial pressure. Visual acuity (ability to read small print), visual fields, extraocular motion, the level of the eyes (asymmetric with infraorbital blow-out fracture), and the presence of nystagmus should be part of the initial assessment. Nystagmus may be seen after a sudden rotational or shearing injury to the brain stem. It may be transient and is most frequently detected by the initial observer. A baseline evaluation of the 7th cranial nerve (facial) is also essential because paralysis of the 7th nerve may be the result of a basilar skull fracture, resulting in increasing intracranial pressure. The tympanic membrane should be visualized while looking for a spinal fluid leak (otorrhea) from a fracture in the petrous region of the temporal bone. Bleeding behind the tympanic membrane may be seen with skull fractures. Ecchymosis posterior to the ear over the mastoid region (Battle's sign) is a subsequent finding indicative of skull fractures in the posterior region of the head.

The cervical spinous processes and the brachial plexus in the supraclavicular region should be palpated. Pain with movement or tenderness warrants further assessment. Even though neck pain is common after head injuries, radiographic examination of the cervical spine is indicated in the presence of pain and tenderness.

Upper extremity strength should be thoroughly assessed, including the rotator cuff muscles, biceps, triceps, deltoid, wrist extensors and flexors, and the intrinsic muscles. Sensation in the arms and legs should be tested, and a baseline Hoffman test performed. Functional lower extremity strength and coordination can be evaluated by observing the athlete while standing, toe and heel walking, and squatting. Coordination can be evaluated by the finger-nose test, tandem walking, and the Romberg test.

On the sidelines, neuropsychologic testing can be performed to document defects in orientation, concentration, and memory. Orientation and retrograde amnesia are usually evaluated on the field. If the player has come off the field under his or her own power and was not examined on the field, these functions should be assessed immediately. Memory can be tested by asking the player to recall three words or three objects at 0 and 5 minutes. Detailed concentration can be evaluated by asking the player to repeat three, four, and five digits backward, to recite the months of the year in reverse order, or to do serial 7s. Knowledge of the player's capabilities through preseason testing is usually necessary in evaluating cognitive performance.

A player should be initially observed for a minimum of 15 minutes on the sidelines and reevaluated as needed. If any symptoms develop, the athlete should not return to competition that day. If the player has not lost consciousness, is oriented, and is asymptomatic, provocative testing should be performed next to determine whether symptoms will occur with physical stress. A 40-yard dash, five sit-ups, five push-ups, or five deep knee bends are usually adequate to increase intracranial pressure. Having the patient recline supine with the feet elevated for several seconds may also increase intracranial pressure sufficiently to cause symptoms. If there are any symptoms after these maneuvers, the player should not be allowed to return to play.

If a player is asymptomatic and returns to the game, it is essential that the athlete be reevaluated repeatedly during the contest to detect any change in clinical course. These subsequent evaluations are preferably performed by the same person who performed the initial examination. It is also helpful to communicate to the player the importance of being extremely honest about symptoms, realizing that many players will deny symptoms to be able to return to competition. The seriousness of the second-impact syndrome and postconcussion syndrome should be explained to the player before he or she is allowed to return to competition. A conservative approach would be to not allow the athlete who has had a head injury back into the game because of the potential risk.

NEUROPSYCHOLOGIC ASSESSMENT OF THE ATHLETE

Although the majority of athletes who experience a concussion are likely to recover completely, an unknown number of these athletes may experience chronic cognitive sequelae. In some cases, these difficulties can be permanent and disabling. At the current time, there are no curative medical treatments for concussion and the best approach to management of concussion emphasizes early recognition of postconcussion symptoms and prevention of additional concussion injury. While most clinicians are aware of the fact that suffering a second blow to the head while symptomatic from a previous concussion can have severe consequences, as in the case of second-impact syndrome,[34] many may not realize that concussions can lead to impairment of cognitive processes, mood, and behavior.[46]

If preinjury evaluations have been performed, neuropsychologic testing may be the most sensitive method of detecting postconcussive dysfunction. These test instruments are sensitive to even subtle changes in attention, concentration, memory, information processing, and motor speed or coordination.[46] Unlike other neurodiagnostic procedures that provide information on brain structure, such as computed tomography or magnetic resonance imaging, neuropsychologic testing provides information on the athlete's functional status. Unfortunately, traditional comprehensive approaches to neuropsychologic assessment that involve hours of testing are not applicable to the evaluation of large groups of athletes and are cost-prohibitive for most teams.

However, the usefulness of neuropsychologic assessment in clinical decision-making should not be shortchanged. The use of shortened neuropsychologic assessment procedures in organized athletics may lead to the resolution of a number of important clinical issues. In particular, the long-term impact of multiple concussions in athletes needs further clarification and may be successfully investigated through the longitudinal systematic neuropsychologic evaluation of athletes. The study of athletes who participate in contact sports provides an excellent opportunity to create baselines of cognitive function and to longitudinally investigate the single or cumulative effects of mild concussions. These studies may eventually lead to the development of more sensitive evaluation strategies and more effective treatment programs.

Baseline Neuropsychologic Assessment of the Athlete

The approach used by the Pittsburgh Steelers since 1993 is based on the University of Virginia study[47] and involves the systematic testing of the athlete at set times before and after a suspected concussion. This approach involves the formal evaluation of each player before the beginning of the season to provide the basis for comparison, in the event of a concussion during the season. Testing is then repeated within 24 hours after a suspected concussion, and again approximately 5 days after the injury.

If neuropsychologic testing is going to be used to evaluate the athlete with concussion, preseason baseline evaluation of the athletes is recommended whenever possible for several reasons. First, individual players vary tremendously with regard to their level of performance on tests of memory, attention, concentration, mental processing speed, and motor speed. Therefore, without the benefit of knowing how players perform before suffering a concussion, it is very difficult to determine whether any testing deficits are due to the effects of that concussion or are secondary to other factors that have nothing to do with the concussion. Some players may perform poorly on the more demanding tests because of preinjury learning disabilities, attention deficit disorder, or other factors such as test-taking anxiety. Since similar patterns of cognitive difficulties may be observed after a concussion, if the player has not been evaluated before the concussion, there is no sure way of determining whether the deficits predated the injury. Additionally, if the player has a history of previous concussions, it may not be clear whether the postconcussion assessment is identifying cognitive difficulties that are secondary to a recent concussion or to a previous event.[3]

Important Characteristics of the Neuropsychologic Evaluation

It is very important to emphasize that the neuropsychologic tests that are selected for the evaluation of athletes must be carefully constructed and researched before their actual clinical use. Each test should be thoroughly researched with regard to the reliability (the extent to which the test produces uniform results) and validity (the extent to which the test accurately measures what it is supposed to measure). Whenever possible, tests should be used that have multiple equivalent forms to limit the impact of practice effects due to repeated exposure to the tests.

Timeline for the Evaluation of the Athlete with Concussion

The initial evaluation of the athlete with concussion begins on the playing field and should continue until symptoms have completely resolved. When formal neuropsychologic evaluations of the athlete are performed, they should take place within 24 hours of the suspected concussion, whenever possible. Many athletes who have suf-

fered very mild concussions may appear to be symptom-free by this time, but a neuropsychologic evaluation to determine more subtle aspects of cognitive functioning can be very revealing, even if the player denies persistent difficulties. If the athlete displays any deficits on neuropsychologic testing, a follow-up evaluation should be undertaken within 48 hours. The interval of 5 days represents a useful and practical follow-up interval. Currently, research is underway that should better clarify the expected recovery curve for athletes with different severity levels.

Neuropsychologic Assessment Instruments

A large number of neuropsychologic test instruments have been successfully employed for the assessment of sports-related brain injuries. The selection of these test instruments has generally followed the previous application of these tests with brain-injured nonathletes. Limitations regarding the time that is typically available for the evaluation of athletes has also been an important factor in the selection and use of neuropsychologic assessment with athletes.

The tests listed here have been found to be useful in the assessment of sports-related head injury. This is a limited list of tests that are currently in use at both the professional and college levels that have been shown to have predictive use. Computerized versions of neuropsychologic tests are now under development and should result in increased access to testing by sports medicine physicians in the near future.

The current test procedures are the Trail making test, Parts A and B[56]; Stroop test[18]; Digit span from the Wechsler Memory Scale, Revised[68]; Symbol Digit modalities test[61]; Controlled oral word association test[4]; Hopkins verbal learning test[6]; Letter and number sequencing from Wechsler Memory Scale—III[67]; Grooved pegboard test[39]; and Ruff's Figural Fluency test.[47]

STANDARDIZED ASSESSMENT OF CONCUSSION: SIDELINE EVALUATION OF THE ATHLETE

The Standardized Assessment of Concussion (SAC) (Appendix 1) was developed to establish a valid, standardized, systematic sideline evaluation for the immediate assessment of concussion in athletes.[36] An objective quantifiable initial assessment of the injury is essential to evaluating a player's readiness to return to competition.[54] Close observation and reliable clinical assessment of the injured athlete are thought to be critical to the prevention of a more serious or catastrophic brain injury,[35,53,59] second-impact syndrome,[8] or cumulative neuropsychologic impairment.[20,31] Clinicians point out that it is often difficult to assess athletes without objective test measures because of the subtlety of concussion symptoms and a tendency on the part of the injured player to deny symptoms to be able to return to play.[40] The importance of objectively assessing orientation, concentration, and memory as part of the on-field mental status examination of athletes suspected of having suffered concussion has been emphasized.[11,36]

The Standardized Assessment of Concussion

The SAC[50] was developed in line with the neuropsychologic research documenting the impairment that occurs with concussions.[9] The SAC includes measures of orientation, immediate memory, concentration, and delayed recall. The SAC was intended to be a standardized means of objectively documenting the presence and severity of neurocognitive impairment associated with concussion to provide immediate information to athletic trainers and other medical personnel responsible for clinical decision-making in the care of athletes. The SAC is not, however, intended as a substitute for formal clinical or neuropsychologic evaluation of the injured athlete, although correlations will be drawn between the result of the SAC and the latter.

The SAC takes approximately 5 minutes to administer and is designed for use by a nonneuropsychologist with no prior expertise in psychometric testing. Three alternate forms (A, B, and C) of the SAC were designed to allow for follow-up testing of injured players with minimal practice effects to track postconcussion recovery. The SAC is printed on pocket-sized cards for convenient use by athletic trainers and other medical personnel on the sideline.

Orientation is assessed by asking the subject to provide the day of the week, month, year, and time of day to within 1 hour. A five-word list is used to measure immediate memory; the list is read to the subject for immediate recall and the procedure is repeated for three trials. Concentration is tested by having the subject repeat, in reverse order, strings of digits that increase in length from three to six numbers. Reciting the months of the year in reverse order is also used to assess concentration. Delayed recall of the original five-word list is also assessed. A composite total score is computed to derive an index of the subject's overall level of impairment after concussion.

Research efforts have focused on use of the SAC with high school and college football players because of the relatively high incidence rate of concussion at these levels (Ref. 37; J. Powell, personal communication, 1998). The results of separate studies in 1995[48] and 1996[49] support the clinical use of the SAC in the evaluation of concussion in football players. In those studies, athletic trainers administered the SAC to 568 normal, noninjured high school and college players before the start of the football season and immediately after concussion of any player during the 1995 and 1996 football seasons. Research findings revealed that 33 players suspected of having sustained a concussion scored statistically significantly below the group of normal, noninjured players on the SAC. Further analysis revealed that players with concussion, as a group, also scored significantly below their own normal baseline in terms of the SAC total score. On average, players with concussion dropped 3.5 points (maximum of 30) from preinjury baseline, which falls 1.48 SDs below their own mean and 1.58 SDs below the normal mean for the control group, indicating that the SAC total score

appears to be sensitive in detecting cognitive defects in the injured players that were tested. These injuries were initially classified by athletic trainers as mild concussions without observable evidence of significant neurologic dysfunction. The SAC demonstrated a preliminary, but useful, trend in tracking recovery from concussion. Follow-up testing on 28 of the 33 injured players indicated that all players had returned to baseline on all SAC measures within 48 hours.

In addition to SAC total score, each of the individual domains (orientation, immediate memory, concentration, and delayed recall) assessed as part of the SAC also yielded useful clinical information in recognizing the concussion, which may further our understanding of the immediate effects of mild forms of brain injury. Players with concussion scored significantly below normal controls on the orientation, immediate memory, concentration, and delayed recall sections of the SAC, despite the fact that none of the injured players were obviously disoriented or neurologically impaired. This finding suggests that the SAC may be able to detect subtle cognitive changes associated with concussion in the absence of other symptoms. Because all of the subjects with concussion in these studies[48,49] suffered what appeared to be mild injuries, further research is required to identify which SAC domain scores are most sensitive to change during injury and to characterize how orientation, concentration, and memory are affected in more severe forms of concussion.

An evaluation of the psychometric properties of the SAC showed no significant differences between scores of high school and college players, suggesting that age and education within the population studied have minimal effects on performance. There were no meaningful differences between forms A, B, and C of the SAC, thereby allowing for the reassessment of mental status and tracking recovery with minimal practice effects. There were no significant differences between examinations conducted during games or practice, suggesting that the emotion of athletic competition does not significantly confound test performance.

The important implication here is that baseline testing can be conducted during the off-season or preseason to establish a valid and reliable marker against which change associated with concussion can be detected. The finding that the average score for normal subjects fell 1.60 SDs below the ceiling and that only 7% of controls managed a perfect score of 30 on the SAC suggests that the instrument is reasonably free of significant ceiling effects. Collection of normal test-retest data on the SAC is underway to clarify the presence of any practice effects. The issue of interrater reliability is also being addressed by additional research, but is difficult to empirically assess. A number of factors are often encountered by brief screening instruments, including a limited range in scores by controls and the need for clinical data. These factors present problems because of the dynamic and unpredictable nature of concussion.

An important practical finding from research on the SAC is that athletic trainers thought that the instrument was convenient for use on the sideline during sporting events. A survey of several athletic trainers indicated that the SAC was easy to administer and score.

Research thus far on the SAC has involved comparing an injured player's score with his or her own preinjury baseline performance to detect change that is likely indicative of concussion. Further research is underway regarding the sensitivity, specificity, validity, and reliability of the SAC in detecting concussion when baseline data are not available for comparison. Sufficient data are not available at this point to determine if the SAC can be used clinically if baseline data are not available. Additional research is also being conducted on the clinical application of the SAC in assessing concussion in sports other than football, including soccer, hockey, lacrosse, and wrestling.

Empirical research data support the further testing of the SAC by medical personnel as an objective and quantifiable measure of the immediate neurocognitive effects of concussion. The SAC appears to be sensitive in evaluating athletes with concussion immediately after the injury and may be helpful in making decisions as to a player's readiness to return to play. The SAC is not, however, meant as a substitute for formal neurologic or neuropsychologic examination of the injured athlete. Rather, the SAC is intended to detect cognitive defects immediately so that further evaluation and proper management techniques can be implemented, if needed. Use of an objective, standardized mental status examination such as the SAC, in combination with a thorough clinical examination, may represent the most sensitive and informative approach to the sideline assessment of concussion. However, for the SAC to become an integral and unconditional component of the evaluation of an athlete with concussion, further statistical validation is necessary.

RECOMMENDATIONS FOR CONCUSSION WORK-UP AND RETURN TO PLAY

In general, if an athlete has any symptoms on the field that are related to a concussion, the athlete should not be allowed to continue to play. Additionally, athletes with concussions should always be evaluated by a physician before return to athletic play. Parameters for return to activity in the asymptomatic athlete should be the same for all sports, regardless of the degree of contact or use of protective equipment such as helmets. A small number of symptomatic athletes may require subsequent evaluation by a neurosurgeon or a neurologist because of persistent symptoms. Caution should always be exercised by the medical staff responsible for making return-to-play decisions because the athlete's motivation as well as peer or coaching pressure may be significant factors.

Most importantly, any athlete who is symptomatic after a concussion requires serial neurologic evaluations. These examinations should be performed, as needed, for as long as symptoms persist to determine if the athlete's condition is deteriorating. If a neurologic evaluation at any time reveals any deterioration in mental status or a loss of consciousness after a concussion, immediate transport to an appropriate emergency facility is indicated where a neurosurgeon or neurologist and diagnostic neuroimaging

are available. No other abnormalities on the neurologic examination would be needed to warrant such emergent treatment.

When a concussion occurs, the athlete should be observed and evaluated for a minimum of 15 minutes. The medical personnel at the competition may allow the athlete to return to play if there was no loss of consciousness and all signs and symptoms are normal. If the athlete's symptoms do not abate during the initial 15 to 20 minutes of observation, the athlete should be disqualified from that day's competition. Only when the athlete is totally asymptomatic, passes memory and concentration tests, and has no symptoms after provocative testing, may the athlete be returned to play. Once the athlete has returned to competition, medical personnel should continue to observe and reexamine the athlete carefully for any signs that the athlete is not 100% recovered. The increased stress of competition may produce signs and symptoms that are not produced by the provocative maneuvers off the field. If the athlete is not 100% recovered, the athlete should be disqualified. This is especially important in sports where breaks in the action are infrequent and frequent reevaluations off the field are not possible.

Several clinical rules are important to keep in mind when evaluating athletes with concussion. Any observed period of unconsciousness is significant and should always preclude return to play. Even brief episodes of loss of consciousness are usually associated with other symptoms that will preclude play. While a brief loss of consciousness is only one factor to consider in the clinical evaluation, it should be evaluated in context with other signs and symptoms. As with any other injury, careful serial follow-up examinations are always recommended.

Return-To-Play Classifications

Return to Play (Same Day).

1. Signs and symptoms cleared within 15 minutes or less both at rest and exertion
2. Normal neurologic evaluation
3. No documented loss of consciousness

Delayed Return to Play (Not the Same Day).

1. Signs and symptoms did not clear in 15 minutes at rest or with exertion
2. Documented loss of consciousness

Any new headache in the first 48 to 72 hours after a concussion or an unusual headache should be considered a significant symptom and should preclude play; either is also an indication that further medical evaluation is needed. Caution should always be exercised in the younger athlete with headache, particularly a unilateral headache.

The other symptoms that should preclude play at any time are dizziness, slowness in responding to questions, evidence of difficulty concentrating, physical sluggishness, and memory loss, especially if there is a loss of memory of events before the injury (retrograde amnesia). Athletes who experience retrograde amnesia do not usually recover during the athletic contest. If the player has had any symptoms or difficulty with concentration tests, that player should not return to play. While a loss of consciousness usually receives a lot of attention by those attending an injured athlete, a brief loss of consciousness, such as a matter of a couple of seconds during the time it takes medical personnel to reach the athlete on the playing surface, may not be as significant as other symptoms that do not clear in the first 15 minutes.

While some concussion scenarios present challenges to the clinician, there is no question about a symptomatic athlete's status: the athlete should not return to play. However, the clinical decisions become more difficult when symptoms clear after 20 to 30 minutes, after the game, or the next day. All of these situations should be classified as prolonged symptoms and are cause for concern. Unfortunately, at the present time, it is not known if neurocognitive function returns to normal when symptoms subside in humans. Therefore, it cannot be assumed that an athlete is normal when he or she "feels fine." The return to play for a young (for example, high school) athlete who experienced symptoms for longer than 15 minutes continues to be a difficult decision and represents a gray zone in the medical literature. Current medical knowledge does not adequately address this situation. While some athletes may benefit from 5 to 7 days of rest after experiencing initial symptoms in excess of 15 minutes,[59] others may be able to safely return to play much sooner.

Until neuropsychologic testing can be done on enough asymptomatic athletes in the first 48 hours after symptoms resolve, correlation between the absence of symptoms and neurocognitive function in humans cannot be drawn, and even if it could, it may still not mean the player is safe to return to play. Presently, the NHL is performing neuropsychologic testing on all players after concussion. The relationship between neurofunction and symptoms in this group of athletes may soon be known. However, these same correlative studies, between symptoms and cognitive function, will have to be repeated in all age ranges and athletic groups to determine the safety of a return to play. It is very important not to generalize the results of these correlative studies. What is medically acceptable in adults may not be safe in teenagers or adolescents. Further studies in the various age groups and sports will be needed to answer these clinical problems safely. Unfortunately, as far as we know, there are no ongoing studies in child or adolescent athletes such as those being conducted by the NHL and the NFL.

Recommendations

1. Every athlete with concussion should be evaluated by a physician.
2. Loss of consciousness precludes return to play that day.
3. Persistence of (longer than 15 minutes) or delayed onset of any symptoms such as headache, dizziness, malaise, slowness to respond mentally or physically at rest, or with provocation (supine with legs elevated) or with exercise precludes return to play that day.

4. Any deterioration in physical or mental status after the initial trauma, such as increasing headache, dizziness, or nausea, warrants immediate transport to an emergency facility where neurologic or neurosurgical consultation and neuroimaging are available.

5. When prolonged symptoms (greater than 15 minutes) are experienced after a concussion, great care must be exercised in returning an asymptomatic athlete to practice or competition. Without at least 5 to 7 days of rest, neurofunction may not yet be normal. Further research is needed to demonstrate the association, or lack of association, between symptoms, neurocognitive function, and injury susceptibility. Until this age-specific information is available, such decisions must be approached with great concern. Repeated examinations of the athlete are needed during a gradual increase in physical exertion to determine if these stresses trigger symptoms. If symptoms recur, the athlete is not ready to return to play. Current neuroscience knowledge in humans does not give a safe, firm timetable for return to play after concussion in most circumstances. Therefore, each athlete with prolonged symptoms (more than 15 minutes) must be evaluated individually. Repeated and thorough evaluations, preferably by the same clinician, are most helpful in determining readiness to play.

6. Newer tools, such as balance testing,[21] cannot be recommended for clinical decision-making after concussion at this time. However, their use for further data collection is encouraged. The balance test may prove to be a useful tool for identifying impairment associated with concussion.

7. We recommend further study of the SAC[48–50] as part of the initial evaluation of an athlete with concussion to gain experience with its use. Furthermore, wide-scale examination of this instrument is needed at all levels of competition and in different athletic groups. While recognizing its clinical potential, we believe it is premature to recommend its generalized use as the sole determinant of clinical decisions after concussion. We do recommend continued wide-scale clinical testing of this instrument.

8. We recognize the need for continued clinical and basic science research of sports-induced concussions. The clinical use of neuropsychologic assessments in the study of athletes has been limited by a current lack of research studies that have specifically investigated the use of these assessments in sports. We recommend the establishment of cooperative studies across athletic organizations at the junior, high school, college, and professional levels that would promote the longitudinal study of large groups of athletes.

9. We specifically promote the establishment of databases on all athletes with concussions. If similar neuropsychologic instruments are used at all levels, longitudinal analysis of test results for specific athletes will be possible as the athlete progresses from one level to the next. This type of information would be particularly useful to athletes, their families, and physicians to assess the risk of future injury and further difficulties.

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Appendix 1 The SAC form for evaluating concussion.

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)

NEUROLOGICAL SCREENING:

Recollection if injury (pre- or post-traumatic amnesia)

Strength:

Sensation:

Coordination:

Loss of Consciousness:

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