

Evidence-Based Review of Sport-Related Concussion: Clinical Science

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Abstract: The clinical nature of sport-related concussion is discussed in this paper. Particularly highlighted are the difficulties with definition, injury severity grading, classification, and understanding of clinical symptoms. In addition, the well-recognized sequelae of concussion including the motor and

convulsive manifestations are discussed in detail. Where possible, an evidence-based approach is adopted to assist the understanding of the literature in this complex area.

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INTRODUCTION

The clinical manifestations of concussion as a transient neurologic syndrome without structural brain injury have been known since the 10th century A.D.¹ Despite over 1,000 years of conceptual development, the diagnosis and management of this condition remains controversial.

WHAT IS THE DEFINITION OF CONCUSSION?

There is no universal agreement on the standard definition or nature of concussion.^{2,3} The historical context of this injury refers to a transient disturbance of neurologic function caused by the “shaking” of the brain that accompanies low velocity brain injuries.^{1,4–6}

Following pioneering experimental work demonstrating the transient and functional nature of concussion by Denny-Brown and Russell, the term “acceleration concussion” was proposed as the generic descriptor that should be applied to all forms of traumatic brain injury.⁷ A variation on this view holds that concussion refers to the mechanism of injury and motion of the brain within the skull rather than any clinical symptoms or pathology.^{8–10}

In an attempt to resolve this confusion, the Committee on Head Injury Nomenclature of the Congress of Neurologic Surgeons proposed a “consensus” definition of concussion. The American Medical Association and the International Neurotraumatology Association subsequently endorsed this definition.^{11,12} This definition has now become accepted by most researchers in this field.

The Congress of Neurologic Surgeons definition states that concussion is

“a clinical syndrome characterised by the immediate and transient post-traumatic impairment of neural function such as alteration of consciousness, disturbance of vision or equilibrium etc. due to brainstem involvement.”

Developing in parallel with the term “concussion” has been the term “mild brain injury.” Jennet et al. proposed the Glasgow Coma Scale (GCS) as a prospectively validated prognostic scale for the assessment of traumatic brain injury.¹³ This scale distinguished mild, moderate, and severe brain injury on the basis of a standardized score at 6 hours following injury.

Because the GCS was designed to be applied 6 hours after brain injury, the full spectrum of brain injury must also encompass a “minimal” injury subset that falls below the threshold for a GCS mild injury (as measured at 6 hours). In clinical practice, the majority of sporting concussions fall into this group. Several common features that incorporate clinical, pathologic, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

1. Concussion may be caused either by a direct blow to the head or elsewhere on the body with an “impulsive” force transmitted to the head.
2. Concussion results in an immediate and short-lived impairment of neurologic function.
3. Concussion may result in neuropathologic changes; however, the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
4. Concussion may result in a graded set of clinical syndromes that may or may not involve loss of consciousness (LOC). Resolution of the clinical and cognitive symptoms typically follows a sequential course.

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Current definitions of concussion remain unsatisfactory from the sporting injury standpoint. It may be that with further research a more specific definition incorporating the factors listed above as well as a time-limited symptom categorization may be developed.

WHAT ARE THE CLINICAL FEATURES OF CONCUSSION?

The acute symptoms of concussion have been described in detail in many published studies. Prospectively validated signs and symptoms include amnesia, LOC, headache, dizziness, blurred vision, attentional deficit, and nausea.^{14,15} While the symptoms described are rather broad in nature, from a practical standpoint, the presence of an attentional deficit and/or amnesia should be essential for the diagnosis rather than say headache alone. By definition, concussive phenomena are immediate. This has the potential to cause confusion given that symptoms that are delayed (e.g., nausea) may be due to other causes such as trauma-triggered migraine rather than concussion.

The attentional deficit that may be detected by formal neuropsychologic testing often is loosely described by clinicians as "confusion" or "disorientation." While these terms are often seen as sine qua non of concussion, it is more scientifically correct to use the terminology of an attentional deficit or attentional dysfunction. This has important clinical relevance given that many of the standard clinical concussion assessment questions and/or neuropsychologic tests should only be administered after the attentional deficit has resolved.

In addition, there are a wide variety of subjective findings that may be encountered in concussed athletes. The prognostic significance of these is unknown. These include descriptions of vacant stare, irritability, emotional lability, impaired coordination, sleep disturbance, noise/light intolerance, lethargy, behavioral disturbance, and altered sense of taste/smell. The time to resolution of these signs and symptoms is extremely variable.

Other recently described features of acute concussion include convulsive and motor phenomena.¹⁶⁻¹⁸ The pathophysiological mechanism of these events is unclear but speculated to involve brainstem reflex mechanisms stimulated by afferent facial connections.

DO CLINICAL SYMPTOMS PREDICT INJURY SEVERITY?

The presence and severity of clinical symptoms or neuropsychologic performance may have prognostic significance. While this area has not been rigorously examined, a number of points are evident.

Recent studies of sport-related concussion have noted that orientation questions (e.g., orientation in time, place, person) were less sensitive in discriminating concussed from nonconcussed football players when compared with questions of recently acquired memory.^{19,85} This has a number of clinical implications, given that the assessment of posttraumatic amnesia (PTA) is usually contingent on the use of items of orientation.²⁰

Retrograde amnesia or the period of loss of recall for events prior to brain injury has been extensively documented in traumatic brain injury.²¹⁻²⁵ Retrograde amnesia is a time-variable entity with a lengthening of the amnesic period in the first few minutes after injury²⁵ and then subsequently shrinkage²¹, leaving a small period of permanently lost retrograde amnesia. In general, although retrograde amnesia is considered to be a consistent feature of traumatic brain injury, neither its presence nor duration is considered a reliable indicator of injury severity or outcome.²⁶

The duration of PTA has been found to correlate with the severity and outcome of severe traumatic brain injury.^{8,27-30} Few studies have investigated the qualitative features of PTA in mild concussive injury. In those cases, PTA has not been found to be a prognostic marker.^{14,20,25,31-35}

While the presence of headache, dizziness, blurred vision, LOC, and nausea have been noted in prospective studies of concussion,^{14,15} their prognostic significance remains unclear. The presence of LOC as an isolated marker of injury severity has been examined in a number of studies and has not been found to reflect either injury severity or neuropsychologic performance.^{152,153,163}

WHAT IS THE BEST CLASSIFICATION OF INJURY SEVERITY?

The classification of severity of concussive injury is a source of controversy. More than 90% of all concussions that are sustained in sports are considered to be "mild," characterized by no LOC, transient confusion, and/or a brief duration of posttraumatic amnesia.^{14,15,36,37} At the present time, there are at least 25 different published injury-grading systems. Most of these are impractical for clinical use in sport.^{9,13,38-51} There have been no prospectively validated studies of any of the current grading systems in sport-related head injury.

Grading Scales

The 25 published sport-related concussion severity scales can be broken down into a number of broad groupings.

Surrogate head injury scales. Whereas the practical needs in the majority of concussions are for a scale that is biased toward distinguishing the marginally clinical injury from the mild-to-moderate injury, most of the scales are biased towards the higher severity injuries. In some cases these include coma, persistent vegetative state, and death, far beyond the historical definition of concussion outlined earlier.^{38,39,43,50} The aim of these types of injury scales is to avoid missing the more severe brain injuries (e.g., cerebral hemorrhages) that may mimic concussion in their early stages. There is no evidence that these scales achieve this goal (Table 1).

Neurosurgical scales. Traditional neurosurgical thinking is evident in the composition of each of these scales.^{41,42,44,45,47,52-54} As previously mentioned, the duration of LOC and PTA are important outcome predictors in severe brain injury. The extrapolation of such concepts to milder grades of brain injury remains specu-

TABLE 1. Surrogate head injury scales

Grade	Ommaya & Gennarelli, 1974	Ommaya, 1990	Torg, 1991	Gersoff, 1991
1	Confusion	Confusion, no LOC, no amnesia	Confusion, momentary LOC, dazed appearance, unsteady gait	Confusion, no LOC, no amnesia
2	Confusion and amnesia, PTA, no LOC	Confusion, no LOC, PTA	Vertigo, PTA	Confusion, PTA, no LOC
3	No LOC, Confusion and amnesia, PTA and RGA	Coma <6 hrs, PTA and RA	Vertigo, PTA, RA	No LOC, confusion, PTA and RA
4	Paralytic coma	Coma 6–24 hrs	PTA, immediate LOC	LOC, paralytic coma
5	Coma (>24 hrs) PVS	Coma >24 hrs	Paralytic coma, cardiorespiratory arrest	Coma
6	Coma, death	Coma, death <24 hrs	Death	Coma, death

LOC, loss of consciousness; PTA, posttraumatic amnesia; PVS, persistent vegetative state; PA, retrograde amnesia.

lative. In each of these scales, an arbitrary separation is made between different grades of concussive injury (Table 2).

Sport-specific scale. Sporting organizations throughout the world have responded to the needs of injury management within their given sport by the development of their own severity scales.^{55,56} In most cases, this has been performed by medical officials within the sport. As a result, these specific scales have much in common with Table 2 and share the same criticisms (Table 3).

Sporting injury scales. In these cases, the stated aim of the injury scales is to distinguish the trivial or marginally significant injuries from more severe injuries.^{9,40,49,57–60} In some cases, attempts have been made to amalgamate neurosurgical concepts within this framework, but in general these types of scales are largely symptom driven.

Often terminology is used loosely (e.g., “extended” LOC or “prolonged” retrograde amnesia), which makes interpretation of the arbitrary subcategories difficult. Although this approach is more in keeping with the typical management problems faced by sports medicine clinicians, nevertheless, no scientific validation has been attempted with any of these scales (Table 4A and B).

Unclassifiable scale. In this category, the scaling system proposed is more reflective of pathophysiological constructs rather than clinical management.⁶¹ (Table 5).

In summary, no perfect scale exists that satisfies the needs of the clinician to be both scientifically valid and practical (Table 6). It is for this reason that Cantu suggested that the final management decision following a concussion is a clinical judgment in every case, and it may be entirely appropriate to deviate from a particular

TABLE 2. Neurosurgical scales

	Maroon, 180	Hugenholz, 1982	Cantu, 1986	Wilberger, 1989	Colorado, 1991	Roberts, 1992	VNI (Polin, 1986)	AMSSM, 1998
0						Bell ringer, no LOC, no PTA		
1 mild	No LOC	Transient or no LOC, PTA <1 hr	No LOC, PTA <30 min	Minimal or no LOC, PTA <20 mins, Short-term confusion	No LOC, confusion, no amnesia	No LOC, PTA <30 mins	Momentary LOC, PTA <1 hr, GCS 15	No LOC, symptoms <5 mins
1a							Momentary LOC, PTA 1–24 hrs, GCS 15	
2 mod	LOC with RA	LOC <5 min, PTA 1–24 hrs	LOC <5 min, PTA >30 min	LOC <5 min, PTA >20 mins, confusion	No LOC, confusion, amnesia	LOC <5 mins, PTA 30 min–24 hrs	LOC <5 mins, PTA >24 hrs, GCS <15 for <5 minutes	LOC <1 min, symptoms 5 mins–24 hrs
3 severe	LOC >5 mins	LOC >5 min, PTA >24 hrs	LOC >5 min, PTA >24 hrs	LOC >5 min, PTA >12 hours, severe headache	LOC	LOC >5 mins, PTA >24 hrs	LOC <5 mins, PTA NA, 12 < GCS < 15 for <60 ins	LOC >1 min, symptoms >24 hrs
4							LOC 5–60 mins, PTA NA, GCS <12 for >5 minutes or <15 for >1 hour	

LOC, loss of consciousness; PTA, posttraumatic amnesia; PVS, persistent vegetative state; RA, retrograde amnesia; GCS, Glasgow Coma Scale; NA, not applicable.

TABLE 3. Sport-specific scales

	Rugby football league (UK)	Federation Internationale de Ski (FIS)	Amateur Boxing Association	Auto Cycle Union (UK)	The Jockey Club (UK)	Rugby Union (Aust)	Rugby League (Australia)
Mild	No LOC	Transient concussion	Immediate recovery	LOC <5 mins	No LOC	No LOC, confusion and disorientation, double vision, giddiness, unsteadiness	<i>Grade 0</i> No LOC, not stunned or dazed, subsequent headache or difficulty in concentration <i>Grade 1</i> No LOC, stunned or dazed, no amnesia, sensorium clears <1 min
Mod	LOC <2 mins	LOC <60 secs	Complete recovery within 2 mins	LOC 5–60 mins	LOC <60 secs or any degree of PTA or if rider sent to hospital	LOC <4 mins, vomiting	<i>Grade 2</i> No LOC, cloudy sensorium >1 min, headache, may have amnesia, tinnitus, or dizziness
Severe	LOC >2 mins	LOC >60 secs	Complete recovery delayed for >2 mins	LOC >60 mins	LOC >60 secs	LOC >4 mins	<i>Grade 3</i> LOC <1 minute, not comatose, Grade 2 symptoms during recovery <i>Grade 4</i> LOC >1 minute, not comatose, Grade 2 symptoms during recovery

LOC, loss of consciousness; PTA, posttraumatic amnesia.

set of guidelines depending on the circumstances surrounding the injury.⁴⁷

WHAT ARE THE NEUROPSYCHOLOGIC FINDINGS IN CONCUSSION?

Only in the past few decades has there been interest in studying the neuropsychologic consequences of concussion, particularly those injuries seen in sport. A range of neuropsychological (NP) deficits has been reported after mild concussive injury. The major areas of deficit include disturbances of new learning and memory,^{24,62–70} planning and the ability to switch mental “set”,^{62,65,67,71} and reduced attention and speed of information processing.^{15,62,69,72–81}

There have also been isolated reports suggesting that impairments may be evident on tasks involving visuospatial constructional ability, language, and sensorimotor function.^{62,65} One area of concern that has not been studied in detail is the proposal that, following recovery, deficits may still be evident when assessed under conditions of physiological stress.⁶⁴

The use of preinjury or baseline testing of NP function is important since it addresses the potential methodological problems inherent in a number of published studies where nonsport controls or reference values are used for comparison of results. A number of groups have developed strategies using baseline neuropsychologic testing, suggesting that such techniques are feasible in the clinical situation. Studies in both American and Australian football dating from the mid-1980s are testament to the usefulness of this approach.^{15,75,82,83}

WHAT IS THE ROLE OF NEUROPSYCHOLOGIC TESTING IN SPORT?

The development of sport-related neuropsychologic testing occurred concurrently in both America and Australia in the 1980s. In professional Australian football, a standardized neuropsychologic test battery was developed and commenced use in 1985.^{75,84–86} In the United States, the earliest studies were performed at the University of Virginia, and have spread to other Universities and high schools.^{15,66} Subsequently, interest has developed in professional American football⁸³ as well as the National Hockey League.

The application of simple validated neuropsychologic tests in the clinical setting of sport-related concussion to measure memory function has created much interest. The standardized questions below have been determined as being sensitive in discriminating between concussed and nonconcussed individuals.¹⁴

Which ground [field] are we at?
Which team are we playing today?
Who is your opponent at present?
Which quarter is it?
How far into the quarter is it?
Which side scored the last goal?
Which team did we play last week?
Did we win last week?

Other assessment tools include the Standardised Assessment of Concussion (SAC)^{19,87}, which has been proposed as a tool for athletic trainers to diagnose concussion in a sport setting. Although a useful addition to the

TABLE 4. *Sporting injury scales*

	Schneider, 1973	Kulund, 1982	Kolb, 1989	AAN, 1998
Mild	No LOC, slight confusion, nil or transient amnesia, mild tinnitus/dizziness, no unsteadiness, rapid recovery	Briefly stunned, no confusion, headache, nausea, visual disturbance, incoordination, or unsteadiness; promptly regains awareness of surroundings	No LOC, stunned or dazed, PTA <30 mins, mild headache	Transient confusion, no LOC, concussion, symptoms or mental status abnormalities resolve >15 minutes
Moderate	LOC <5 minutes, momentary confusion, definitive retrograde amnesia, moderate tinnitus, dizziness, or unsteadiness; recovery <5 minutes	Loss of consciousness, some confusion, and RA; Tinnitus, dizziness, and unsteadiness	LOC 10 sec–5 mins, confusion, disorientation, PTA <30 mins, RA	Transient confusion, no LOC, concussion, symptoms or mental status abnormalities resolve >15 minutes
Severe	LOC >5 minutes, confusion >5 minutes, prolonged retrograde amnesia, severe tinnitus, dizziness, or unsteadiness, recovery >5 minutes	Extended LOC, headache, dizziness, tinnitus; confused and unsteady, extended RA	LOC >5 mins, PTA >24 hrs, confusion, headache, dizziness	Any LOC
Severity grade	Saal, 1991		Nelson, 1984	
Grade 0			Head struck but not immediately dazed or stunned; subsequent headache or difficulty concentrating	
Grade 1	No LOC, no amnesia, mild headache, unsteadiness		Stunned or dazed, no LOC or amnesia, bell rung, sensorium clears <1 in	
Grade 2	Momentary LOC, no amnesia, headache, unsteadiness		Headache, cloudy sensorium >1 min, may have tinnitus, amnesia, may be irritable, confused, dizzy, hyperexcitable	
Grade 3A	Momentary LOC, no PTA		LOC <1 min, not comatose (rousable with noxious stimuli), demonstrates grade 2 symptoms during recovery	
Grade 3B	Momentary LOC, RA and PTA			
Grade 4	LOC 5–10 mins, RA and possible PTA		LOC >1 min, not comatose, demonstrates grade 2 symptoms during recovery	
Grade 5	LOC: not rousable			

LOC, loss of consciousness; PTA, posttraumatic amnesia; RA, retrograde amnesia.

clinical armamentarium, it does not enable trainers to exclude a more significant intracranial injury, which may masquerade as concussion in the early stages. Similarly it is not validated as a return-to-play assessment tool. Its value over the Maddocks' questions described above is dubious and may be unnecessarily complex for its given role.

WHAT IS THE TYPICAL NEUROPSYCHOLOGIC RECOVERY PERIOD?

In general terms there appears to be clear evidence of the presence of NP deficits during the first week following mild concussive injury.^{15,41,69,73,75,79,82,88,89} Beyond this time variable, findings are reported with no consen-

sus of recovery time frames.^{15,63,69,72,75,79,88,90–93} Few studies, however, have had prolonged (>3 month) follow-up of subjects.

WHAT IS THE POSTCONCUSSION SYNDROME?

Descriptions of myriad physical and cognitive symptoms labeled as the postconcussion syndrome (PCS) remain as controversial today as when first proposed in the 19th century.⁹⁴ These symptoms include headache, vertigo, dizziness, nausea, memory complaints, blurred vision, noise and light sensitivity, difficulty concentrating, fatigue, depression, sleep disturbance, loss of appetite, anxiety, incoordination, and hallucinations.^{66,95–97} Two distinct schools of thought have arisen regarding the pathophysiology of this condition; the first proposes that the symptoms associated with PCS are a direct consequence of brain injury,^{98,99} while the second proposes that the symptoms are functional and represent psychological or emotional sequelae of the brain injury. The issue of malingering and compensatory litigation also is often proposed as a mechanism for symptom prolongation.^{100–106}

At this time, the relative contribution of these two

TABLE 5. *Unclassifiable scales*

Grade	Parkinson, 1977
Stage 1	Normal somatic mobility with impaired performance
Stage 2	Normal visceral mobility with impaired somatic mobility
Stage 3	Return of irregular visceral mobility with continuing somatic immobility
Stage 4	Visceral (respiratory) and somatic immobility

mechanisms remains unclear. In general, PCS may be under-reported in collision and contact sports, although relatively few studies have followed sporting populations for significant lengths of time.^{155–162}

WHAT IS THE ROLE OF INVESTIGATIONS FOLLOWING CONCUSSION?

To date, most studies of concussion report computerized axial tomography or CT scanning. This imaging modality has proven of significant benefit in severe brain injury or where an intracranial hematoma is suspected; however, such abnormalities are rarely associated with mild brain trauma.^{71,107–110} Most experienced clinicians, however, would not advocate routine imaging of uncomplicated concussion.³

In recent times, magnetic resonance imaging (MR) has become the preferred technique to image acute brain trauma.^{95,111–118} MR studies in sport-related concussion have been inconsistent. In some studies, focal abnormalities have been demonstrated, whereas other studies are normal.^{109,117} In boxers, a number of studies have been performed following concussive injury or “knock-outs” that show no abnormalities.^{95,111–113,119,120}

Newer MR modalities, such as diffusion-weighted imaging, may offer scope for detection of physiological changes following concussion. A recent paper studying severe closed-head injury noted widespread diffusion weighted imaging changes following injury.¹⁵⁴ Unpublished hyperacute studies of diffusion, perfusion, and magnetic spectroscopy by one of the authors (P.M.) on a population of concussed football players did not reveal any abnormal changes.

Single photon emission computerized tomography (SPECT) imaging to measure blood flow patterns in neurologic diseases has been available for more than a decade. In acute head trauma, a variety of SPECT abnormalities have been reported^{121–124} and may be a determinant of outcome.^{125,126} No studies have been performed on sport-related concussion.

Positron emission tomography (PET) is a form of functional imaging that has the ability to quantitatively document cerebral blood flow, regional metabolism, and receptor function. Preliminary studies in mild brain injury suggest metabolic abnormalities in the anterior and posterior frontal and temporal lobes in the presence of normal CT and MR imaging.^{110,127,128} The significance of this is unknown. No PET studies have been reported in sport-related concussion.

Electroencephalography (EEG) may provide an index of irreversible brain damage in severe brain injury; however, concussion is usually associated with normal or nonspecific EEG findings.^{107,129} One sporting study reported EEG abnormalities in a cohort of retired soccer players as evidence of chronic brain injury; however, methodological problems flaw this study.^{130,131}

Evoked potentials (EP) have been studied following concussive injuries, and the findings have been inconsistent.¹³² Similarly cognitive evoked potentials such as P300 responses have attracted some research interest,

and studies have suggested that impaired ability to direct attention was a persistent finding in sports-related concussion and may represent cerebral pathology.^{133–135} The significance of these findings is unclear.

WHAT IS THE MANAGEMENT OF CONCUSSION?

Since the first reported case of a brain concussion in sport,¹³⁶ medical practitioners have debated over the best method of managing these injuries.^{3,137} In such a controversial area without hard scientific data, good clinical judgement and individualized care often override published management guidelines.⁴⁷

Initial Management

Aside from the basic principles of resuscitation, several important management points commonly expressed in the concussion literature warrant specific attention.³

In this setting, the importance of excluding a cervical spine injury is stressed.^{3,138} Following this step, a thorough neurologic examination should exclude any potentially catastrophic brain injury.^{139,140} It is also important to continue serial examination of a brain-injured athlete.³ The use of validated neuropsychologic screening tools such as the SAC¹⁹ or the Maddocks questions¹⁴ are important clinical tools to assist in concussion diagnosis.

In general terms, an urgent medical assessment is warranted for all cases of concussion, principally to exclude a coexistent intracranial injury. The role of neuroimaging in this setting is controversial; however, most physicians experienced in concussion management do not routinely administer scans for uncomplicated concussion.³ Whether a concussed patient should be admitted to hospital is a decision for the treating doctor and guidelines vary in this regard. Recommendations for admission of a concussed individual to the hospital have been proposed.^{3,141} These include:

- Deterioration in the level of consciousness following injury
- Skull fracture
- Penetrating skull trauma
- Focal neurologic symptoms or signs
- Loss of consciousness greater than 5 minutes
- Persistent vomiting or headache after injury
- Difficulty in assessing the patient due to alcohol, drugs, epilepsy, etc.
- Other high-risk medical conditions (e.g., hemophilia)
- The lack of a responsible adult to supervise the athlete postinjury
- More than one concussion in a match or training session
- Head injuries in children

Return-to-Play Recommendations

After a diagnosis of concussion has been made, there are many factors that may influence the return-to-play decision. These include injury severity, past history of the athlete, demands of the chosen sport, the presence of postconcussive symptoms, and the speed of resolution of the acute symptoms.^{41,45,91,142}

While scientifically validated return-to-play guidelines do not yet exist, the consensus of experts in this field would suggest that complete resolution of concussion symptoms (both at rest and with exercise) would be mandatory prior to the resumption of training or playing. The speed of symptom resolution varies from individual to individual, and the use of arbitrary exclusion periods has limited value particularly where medical assessment is available. The development of step-wise concussion rehabilitation programs may help address this dilemma in the future.

Similarly guidelines recommending termination of all contact sport following repeated concussions during the course of an athletic season needs to be considered carefully. In the absence of documented objective evidence of structural brain injury or of adverse long-term problems, there is no scientific support for this generalization.

HOW MAY CONCUSSION BE PREVENTED?

There are relatively few methods by which concussive brain injury may be minimized in sport. Unlike musculoskeletal injuries, the brain is not an organ that can be conditioned to withstand injury. Thus extrinsic mechanisms of injury prevention must be sought.

Helmets have been proposed as a means of protecting the head and theoretically reducing the risk of brain injury. In sports where high-speed collisions are possible, or with the potential for missile injuries (e.g., baseball) or falls onto hard surfaces (e.g., gridiron, ice hockey), there is published evidence for the effectiveness of sport-specific helmets to be of benefit in reducing head injuries.¹⁴³⁻¹⁴⁵ For sports such as soccer, Australian football, and rugby, no sport-specific helmets have been shown to be of proven benefit in reducing rates of head injury. In fact, most commercially available soft helmets fail to meet impact testing criteria that would be typical of sport-related concussion.¹⁶⁴

Consideration of rule changes to reduce the head injury rate may be appropriate where a clear-cut mechanism is implicated in a particular sport. There is demonstrable evidence of the effect of rule changes, such as banning spear tackles in American football, thus reducing the incidence of catastrophic head and neck injury.¹⁴⁶ Similarly, rule enforcement is a critical aspect of such approaches. Interestingly, although anecdotal observation suggested illegal play as a common mechanism of injury, a formal video-analysis of injuries in Australian football failed to demonstrate this mechanism as contributing significantly to the overall concussion rate.¹⁵¹

Undoubtedly the use of correctly fitting mouth-guards can reduce the rate of dental orofacial and mandibular injuries. The evidence that they reduce cerebral injuries is largely theoretical,¹⁴⁷⁻¹⁴⁹ and the limited clinical evidence for a beneficial effect in reducing concussion rates has not been prospectively tested.

Neck muscle conditioning may be of value in reducing impact forces transmitted to the brain.⁴⁶ Biomechanical concepts dictate that the energy from an impacting object is dispersed over the greater mass of an athlete if the head

is held rigidly. In animal studies, cervical fixation increased the concussion threshold.¹⁵⁰ Studies have also demonstrated that the force required to concuss a "fixed" head were almost twice that of a moving head.⁷ Although attractive from a theoretical standpoint, there is little scientific support for this viewpoint. Video-analysis of concussive injury seen in Australian football, rugby, and soccer demonstrates that approximately 95% of concussive impacts are an accidental part of play, and the players concerned were unaware of impending impact, and hence were unable to tense their neck muscles in an attempt to withstand the impact.¹⁵¹

Arguably, the most important aspect of prevention is the education of team physicians and others involved in athletic care. On-field recognition of concussive injury remains a priority, as are the application of appropriate validated guidelines in returning athletes safely to sport.

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