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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.

Key Words: Concussion—Traumatic brain injury—Sport.
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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 amnestic period in the first few minutes after injury[25] and then subsequently shrinkage[21], 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	*Grade 0* No LOC, not stunned or dazed, subsequent headache or difficulty in concentration *Grade 1* 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	*Grade 2* 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	*Grade 3* LOC <1 minute, not comatose, Grade 2 symptoms during recovery *Grade 4* 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 football[83] 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 consensus 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.

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

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 psychologic 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

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 SAC[19] or the Maddocks questions[14] 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.

REFERENCES

1. Rhazes A. *Opera Medica Varia*. Venice: Bonetus Locctellus, 1497.
2. Shetter A, Demakis J. The pathophysiology of concussion: a review. *Adv Neurol* 1979;22:5–14.
3. McCrory PR. Were you knocked out? A team physician's approach to initial concussion management. *Med Sci Sports Exerc* 1997;29(7 Suppl):S207–212.
4. Lanfrancus. *A Most Excellent and Learned Worke of Chirurgery Called Chirurgia Por Va Lanfranci*. London: T Marshe, 1565.
5. da Carpi B. *Isogogae Brevis*. Bononie: Benedictum Hectorus, 1523.
6. Bell B. *A System of Surgery*. Edinburgh: Charles Elliott, 1786.
7. Denny-Brown D, Russell WR. Experimental cerebral concussion. *Brain* 1941;64:93–163.
8. Russell WR, Smith A. Post traumatic amnesia in closed head injury. *Arch Neurol* 1961;5:4–17.
9. Nelson WE, Jane JA, Gieck JH. Minor head injury in sports: a new system of classification and management. *Phys Sportsmed* 1984;12:103–107.
10. Rutherford W. Post concussional symptoms: relationship to acute neurological indices, individual differences and circumstances of injury. In: Levin H, Eisenberg H, Benton A, eds. *Mild Head Injury*. New York: Oxford University Press, 1989:217–228.
11. Congress of Neurological Surgeons. Committee on Head Injury Nomenclature: glossary of head ijury. *Clin Neurosurg* 1966;12: 386–394.
12. American Medical Association. Subcommittee on classification of sports injuries: standard nomenclature of athletic injuries. Chicago: American Medical Association, 1966.
13. Jennett B, Bond M. Assessment of outcome after severe brain damage: a practical scale. *Lancet* 1975;1:480–484.
14. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. *Clin J Sport Med* 1995; 5:32–35.
15. Barth JT, Alves WM, Ryan TV, et al. Mild head injury in sports: neuropsychological sequelae and recovery of function. In: Levin HS, Eisenberg HM, Benton AL, eds. *Mild Head Injury*. New York: Oxford University Press, 1989:257–275.
16. McCrory P, Bladin P, Berkovic S. Retrospective study of concussive convulsions in elite Australian rules and rugby league footballers: phenomenology, aetiology and outcome. *Br Med J* 1997;314:171–174.
17. McCrory P, Berkovic S. Concussive convulsions: Incidence in sport and treatment recommendations. *Sports Med* 1998;25: 131–136.
18. McCrory P. Videoanalysis of the acute clinical manifestations of concussion in Australian rules football. In: *SMA/ACSP Annual Scientific Conference, 1996.* Canberra: SMA, 1996:214–215.
19. McCrea M, Kelly J, Randolph C, et al. Standardised assessment

of concussion (SAC): On site mental status evaluation of the athlete. *J Head Trauma Rehab* 1998;13:27–36.
20. Shores A, Marosszeky J, Sandanam J, et al. Preliminary validation of a clinical scale for measuring the duration of post traumatic amnesia. *Med J Aust* 1986;144:569–572.
21. Benson DF, Geschwind N. Shrinking retrograde amnesia. *J Neurol Neurosurg Psychiat* 1967;30:539–544.
22. Levin HS, Grossman RG, Kelly PJ. Short term recognition memory in relation to severity of head injury. *Cortex* 1976;12: 175–182.
23. Russell WT. *The Traumatic Amnesias*. London: Oxford University Press, 1971.
24. Yarnell P, Lynch S. Retrograde amnesia immediately after concussion. *Lancet* 1970;i:863–864.
25. Yarnell P, Lynch S. The "ding": amnestic state in football trauma. *Neurology* 1973;23:196–197.
26. Long CJ, Webb WL, Jr. Psychological sequelae of head trauma. *Psychiatr Med* 1983;1:35–77.
27. Russell WR. The after effects of head injury. *Edinburgh Med J* 1934;41:129–144.
28. Russell WR, Nathan P. Traumatic amnesia. *Brain* 1946;69: 280–300.
29. Sciarra D. Head injury. In: Rowland L, ed. *Merrit's Textbook of Neurology. 7th Ed*. Philadelphia: Lea & Febiger, 1984:277–279.
30. Smith A. Duration of impaired consciousness as an index of severity in closed head injury: a review. *Dis Nerv Sys* 1961;22: 69–74.
31. Fisher CM. Concussion amnesia. *Neurology* 1966;16:826–830.
32. Gronwall D, Wrighson P. Duration of post-traumatic amnesia after mild head injury. *J Clin Neuropsychol* 1980;2:51–60.
33. Levin HS, O'Donnell V, Grossman R. The Galveston orientation and amnesia test: a practical scale to assess cognition after head injury. *J Nerv Ment Dis* 1979;167:675–684.
34. Levin H, Benton A, Grossman A, eds. *Neurobehavioural Consequences of Closed Head Injury*. New York: Oxford University Press, 1982.
35. Richardson JTE. *Clinical and Neuropsychological Aspects of Closed Head Injury*. London: Taylor and Francis Ltd., 1990.
36. Cantu RC. Reflections on head injuries in sport and the concussion controversy [Editorial]. *Clin J Sport Med* 1997;7:83–84.
37. Ryan AJ. Protecting the sportsman's brain (concussion in sport). Annual guest lecture, 1990, London Sports Med Institute. *Br J Sports Med* 1991;25:81–86.
38. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. *Brain* 1974;97:633–654.
39. Ommaya A. Biomechanical aspects of head injuries in sports. In: Jordan B, Tsaris P, Warren R, eds. *Sports Neurology*. Maryland: Aspen Publishers, Inc., 1990.
40. Kulund D. Athletic injuries to the head, neck and face. In: Kulund D, ed. *The Injured Athlete*. Philadelphia: J.B. Lippincott, 1982:225–257.
41. Hugenholtz H, Richard MT. Return to athletic competition following concussion. *Can Med Assoc J* 1982;127:827–829.
42. Kelly JP, Nichols JS, Filley CM, et al. Concussion in sports. Guidelines for the prevention of catastrophic outcome. *JAMA* 1991;266:2867–2869.
43. Torg JF, ed. *Athletic Injuries To the Head, Neck and Face. 2nd Ed*. St. Louis: Mosby Year Book, 1991.
44. Wilberger JE, Maroon JC. Head injuries in athletes. *Clin Sports Med* 1989;8:1–9.
45. Roberts W. Who plays? Who sits? Managing concussion on the sidelines. *Phys Sportsmed* 1992;20:66–72.
46. Cantu RC. Cerebral concussion in sport. Management and prevention. *Sports Med* 1992;14:64–74.
47. Cantu RC. Guidelines for return to contact sports after cerebral concussion. Phys Sportsmed 1986;14:75–83
48. Society CM. *Report of the Sports Medicine Committee: Guidelines for the Management of Concussions in Sport (Revised)*. Denver: Colorado Medical Society, 1991.
49. Saal J. Common American football injuries. *Sports Med* 1991; 12:132–147.
50. Gersoff W. Head and neck injuries. In: Reider B, ed. *Sports Med: The School Age Athlete*. Philadelphia: W.B. Saunders, 1991.
51. Maroon JC, Steele PB, Berlin R. Football head and neck injuries—an update. *Clin Neurosurg* 1980;27:414–429.
52. Maroon JC, Bailes J, Yates A, et al. Assessing closed head injuries. *Phys Sports Med* 1992;20:37–44.
53. Polin R, Alves W, Jane J. Sports and head injuries. In: Evans R, ed. *Neurology and Trauma*. Philadelphia: W.B. Saunders and Co., 1996:166–185.
54. American Medical Society for Sports Medicine. *Position Statement on Head Injury*: AMSSM, 1997.
55. National Health & Medical Research Council. *Football Injuries of the Head and Neck*. Canberra: NH&MRC of Australia, 1995.
56. Turner M. Concussion and head injuries in sport. In: Turner M, ed. *The Jockey Club Conference on Head Injury in Sport, 1998.* London: The Jockey Club of England, 1998.
57. American Academy of Practice parameter: the management of concussion in sports (summary statement). *Neurology* 1997;48: 581–585.
58. Kelly J, Rosenberg J. Diagnosis and management of concussion in sports. *Neurology* 1997;48:575–580.
59. Schneider RC. *Head and Neck Injuries in Football: Mechanisms, Treatment and Prevention*. Baltimore: Williams & Wilkins, 1973.
60. Kolb J. Cerebral concussion. *Sports Train Med Rehab* 1989;1: 165–171.
61. Parkinson D. Concussion. *Mayo Clin Proc* 1977;52:492–499.
62. Barth JT, Macciocchi SN, Giordani B, et al. Neuropsychological sequelae of minor head injury. *Neurosurg* 1983;13:529–533.
63. Dikmen S, McLean A, Temkin N. Neuropsychological and psychological consequences of minor head injury. *J Neurol Neurosurg Psych* 1986;49:1227–1232.
64. Ewing R, McCarthy D, Gronwall D, et al. Persisting effects of minor head injury observable during hypoxic stress. *J Clin Neuropsych* 1980;2:147–155.
65. Leninger B, Gramling S, Farrell A, et al. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *J Neurol Neurosurg Psych* 1990;53: 293–296.
66. Levin HS, Mattis S, Ruff R, et al. Neurobehavioural outcome following minor head injury: a three centre study. *J Neurosurg* 1987;66:234–243.
67. Yarnell P, Rossie G. Minor whiplash head injury with major debilitation. *Brain Inj* 1988;2:255–258.
68. Gronwall D, Sampson H. *The Psychological Effects of Concussion*. Auckland: Oxford University Press, 1974.
69. Gronwall D, Wrightson P. Delayed recovery of intellectual function following minor head injury. *Lancet* 1974;ii:605–609.
70. Gronwall D, Wrighson P. Memory and information processing capacity after closed head injury. *J Neurol Neurosurg Psych* 1981;44:889–895.
71. Rimel RW, Giordani B, Barth JT. Moderate head injury: completing the clinical spectrum of brain trauma. *Neurosurg* 1982; 11:344–351.
72. Gentilini M, Nichelli P, Schoenhuber R, et al. Neuropsychological evaluation of mild head injury. *J Neurol Neurosurg Psych* 1985;48:137–140.
73. Gronwall D, Wrightson P. Cumulative effects of concussion. *Lancet* 1975;ii:995–997.
74. Levin H, Eisenberg HM, Benton AL, eds. *Mild Head Injury*. Oxford: Oxford University Press, 1989.
75. Maddocks D, Dicker G. An objective measure of recovery from concussion in Australian rules footballers. *Sport Health* 1989;7(Suppl):6–7.
76. Ruesch J, Moore B. Measurement of intellectual functions in the acute stages of head injury. *Arch Neurol Psychiatr* 1943;50: 165–170.
77. Ruesch J. Intellectual impairment in head injuries. *Am J Psychiatr* 1944;100:480–496.
78. Dencker SJ, Löfving B. A psychometric study of identical twins discordant for closed head injury. *Acta Psychiatrica Neurologica Scand* 1958;33(Suppl 22):1–17.
79. MacFlyn G, Montgomery E, Ferlin G, et al. Measurement of

reaction time following minor head injury. *J Neurol Neurosurg Psych* 1984;47:1313–1326.
80. van Zomeren AH. *Reaction Time and Attention After Closed Head Injury*. Liesse: Swets & Zeitlinger, 1981.
81. van Zomeren AH, Deelman BG. Differential effects of simple and choice reaction after closed head injury. *Clin Neurol Neurosurg* 1976;79:81–90.
82. Shuttleworth-Jordan AB, Balarin E, Pulchert J. Mild head injury effects in rugby: is the game really worth the cost. In: *International Neuropsychological Society 16th European Conference, 1993*. Madiera, Portugal: International Neuropsychological Society, 1993.
83. Lovell MR, Collins MW. Neuropsychological assessment of the college football player. *J Head Trauma Rehabil* 1998;13:9–26.
84. Dicker GD, McColl D, Sali A. The incidence and nature of Australian rules football injuries. *Aust Fam Phys* 1986;15:455–459.
85. Maddocks D, Saling M. Neuropsychological sequelae following concussion in Australian rules footballers. *J Clin Exp Neuropsychol* 1991;13:439–441.
86. Maddocks DL, Saling MM, Dicker GD. A note on the normative data for a test sensitive to concussion in Australian rules footballers. *Aus Psychologist* 1995;30:125–127.
87. McCrea M, Kelly JP, Kluge J, et al. Standardized assessment of concussion in football players. *Neurology* 1997;48:586–588.
88. Levin HS, Grafman J, Eisenberg HM, eds. *Neurobehavioral Recovery from Head Injury. 1st Ed*. Oxford: Oxford University Press, 1987.
89. Maddocks DL. Neuropsychological recovery after concussion in Australian rules footballers [PhD thesis]. Melbourne: University of Melbourne, 1995.
90. Dikmen S, Reitan RM. Psychological deficits and recovery of functions after head injury. *Trans Am Neurol Assoc* 1976;101: 72–79.
91. Hugenholz H, Stuss D, Stethem L, et al. How long does it take to recover from a mild concussion? *Neurosurg* 1988;22:853–858.
92. Bohnen N, Jolles J, Twijnstra A, et al. Late neurobehavioural symptoms after mild head injury. *Brain Inj* 1995;9:27–33.
93. Mateer C. Systems of care for the post-concussive syndrome. In: Horn L, Zasler N, eds. *Rehabilitation of Post-concussive Disorders*. Philadelphia: Hanley & Belfus Publishers, 1992:143–160.
94. Courville CB. *Commotio Cerebri: Cerebral Concussion and the Post-Concussion Syndrome and Their Medical and Legal Aspects*. Los Angeles: San Luca Publishers, 1953.
95. Levin HS, Amparo E, Eiseberg HM, et al. Magnetic resonance imaging and computerised tomography in relation to the neurobehavioural sequelae of mild and moderate head injuries. *J Neurosurg* 1987;66:706–713.
96. Rutherford WH, Merret JD, McDonald JR. Symptoms at one year following concussion from minor head injuries. *Injury* 1979;10: 225–230.
97. Rutherford WH, Merret JD, McDonald JR. Sequelae of concussion caused by minor head injuries. *Lancet* 1977;i:1–4.
98. Binder L. Persisting symptoms after mild head injury: a review of the post concussive syndrome. *J Clin Exp Neuropsychol* 1986;8: 323–346.
99. Binder L, Rohling M. Money matters: A meta-analytic review of the effects of financial incentives on recovery after closed head injury. *Am J Psychiatr* 1996;153:7–10.
100. Miller H. Accident neurosis: lecture 1. *Br Med J* 1961;i:919–925.
101. Miller H. Accident neurosis: lecture 2. *Br Med J* 1961;i:928–938.
102. Miller H. Mental after effects of head injury. *Proc Roy Soc Med* 1966;59:257–261.
103. Lishman W. Physiogenesis and psychogenesis in the post concussional syndrome. *Br J Psychiatr* 1988;153:460–469.
104. Jacobsen R. The post concussional syndrome: physiogenesis and malingering. An integrative model. *J Psychosomatic Res* 1995; 39:675–693.
105. Andersen S. Post concussional disorder and loss of consciousness. *Bull Am Acad Psychiatr Law* 1996;24:493–504.
106. Karzmark P, Hall K, Englander J. Late onset post concussion symptoms after mild brain injury: the role of pre-morbid, injury-related, environmental and personality factors. *Brain Inj* 1995;9: 21–26.
107. Bakay L, Glasauer FE. *Head Injury*. Boston: Little Brown & Co., 1980.
108. Masters SJ, McClean PM, Arcarese JS, et al. Skull x-ray examination after head trauma: recommendations by a multidisciplinary panel and validation study. *N Eng J Med* 1987;316:84–91.
109. Kelly A, Zimmerman R, Show R. Head trauma comparison of MR and CT—experience in 100 patients. *AJNR* 1988;9:699–704.
110. Stringer W, Balsiero J, Fidler R. Advances in traumatic brain neuroimaging techniques. *Neurorehab* 1991;1:11–30.
111. Haglund Y, Bergstrand G. Does Swedish amateur boxing lead to chronic brain damage? 2. A retrospective study with CT and MRI. *Acta Neurol Scand* 1990;82:297–302.
112. Jordan BD, Zimmerman RD. Magnetic resonance imaging in amateur boxers. *Arch Neurol* 1988;45:1207–1208.
113. Jordan BD, Zimmerman RD. Computed tomography and magnetic resonance imaging comparisons in boxers [see comments]. *JAMA* 1990;263:1670–1674.
114. Levin HS, Lippold SC, Goldman A, et al. Neurobehavioural functioning and magnetic resonance imaging in young boxers. *J Neurosurg* 1987;67:657–667.
115. Ogawa T, Sekino H, Uzura M, et al. Comparative study of magnetic resonance and CT scan imaging in cases of severe head injury. *Acta Neurochir* 1992;55(Suppl):8–10.
116. Wilberger JE, Deeb Z, Rothfus W. Magnetic resonance imaging in cases of severe head injury. *Neurosurg* 1987;20:571–576.
117. Yokota H, Kurokawa A, Otsuka T, et al. Significance of magnetic resonance imaging in acute head injury. *J Trauma* 1991;31: 351–357.
118. Hayes RG, Nagle CE. Diagnostic imaging of intracranial trauma. *Phys Sportsmed* 1990;18:69–79.
119. Cabanis EA, Perez G, Tamraz JC. Cephalic magnetic resonance imaging of boxers. *Acta Radiologica* 1986;369(Suppl):365–366.
120. Holzgraefe M, Lemme W, Funke W, et al. The significance of diagnostic imaging in acute and chronic brain damage in boxing. *Int J Sports Med* 1992;13:616–621.
121. Abdel-Dayem HM, Sadek SA, Kouros K, et al. Changes in cerebral perfusion after acute head injury: comparison of CT with Tc 99m HMPAO SPECT. *Radiology* 1987;165:221–226.
122. Abdel-Dayem HM, Masdeu J, O'Connell R, et al. Brain perfusion abnormalities following minor/moderate closed head injury: comparison between early and late imaging in two groups of patients. *Eur J Nucl Med* 1994;21(Suppl):S109.
123. Roper SN, Mena I, King WA, et al. An analysis of cerebral blood flow in acute closed head injury using technicium 99m HMPAO SPECT and computed tomography. *J Nucl Med* 1991;32: 1684–1687.
124. Nedd K, Sfakianakis G, Ganz W, et al. 99mTc HMPAO SPECT of the brain in mild to moderate traumatic brain injury patients: compared with CT—a prospective study. *Brain Inj* 1993;7: 469–479.
125. Jacobs A, Put E, Ingels M, et al. Prospective evaluation of technicium 99m HMPAO SPECT in mild and moderate traumatic brain injury. *J Nucl Med* 1994;35:942–947.
126. Torigoe R, Hayashi T, Anegawa K, et al. Evaluation of SPECT with 123 I-IMP and 99Tc HMPAO in cerebral concussion patients. *No To Shinke* 1991;43:530–535.
127. Ruff RM, Crouch JA, Tröster AI, et al. Selected cases of poor outcome following a minor brain trauma: comparing neuropsychological and positron emission tomography assessment. *Brain Inj* 1994;8:297–308.
128. Jansen H, van der Naalt J, van Zomeren A, et al. Cobalt-55 positron emission tomography in traumatic brain injury: a pilot study. *J Neurol Neurosurg Psych* 1996;60:221–224.
129. Lorenzoni E. Electroencephalographic studies before and after head injuries. *Electroenceph Clin Neurophysiol* 1970;28: 216–218.
130. Tysvaer A, Storli O, Bachen N. Soccer injuries to the brain: a neurologic and encephalographic study of former players. *Acta Neurol Scand* 1989;80:151–156.
131. Tysvaer AT, Storli O. Soccer injuries to the brain. A neurologic and encephalographic study of active football players. *Am J Sports Med* 1989;17:573–578.
132. Schoenhuber R, Gentilini M, Orlando A. Prognostic value of

auditory brain-stem responses for late postconcussion symptoms following minor head injury. *J Neurosurg* 1988;68:742–744.
133. Pratap-Chand R, Sinniah M, Salem FA. Cognitive evoked potential (P300): a metric for cerebral concussion. *Acta Neurologica Scand* 1988;78:185–189.
134. Cremona-Meytard SL, Geffen GM. Visuospatial attention deficits following mild head injury in Australian rules football players. In: Hendy J, Caine D, Pfaff A, Hannan E, eds. *The Life Cycle: Development, Maturation, Senescence. Proceedings of the 16th Annual Brain Impairment Conference, 1993.* Sydney: Australian Academic Press, 1993:137–147.
135. Cremona-Meytard SL, Clark CR, Wright MJ, et al. Covert orientation of visual attention after closed head injury. *Neuropsychologica* 1992;30:123–132.
136. Pott P. *Observations On the Nature and Consequences of Those Injuries To Which the Head Is Liable From External Violence*. London: L Hawes, W Clarke, R Collins, 1760.
137. Dicker GD. A sports doctors dilemma in concussion. *Sports Train Med Rehab* 1991;2:203–209.
138. Dicker G, Maddocks D. Clinical management of concussion [see comments]. *Aust Fam Physician* 1993;22:750–753.
139. LeBlanc KE. Concussions in athletics: guidelines for return to sport. *J LA State Med Soc* 1998;150:312–317.
140. Leblanc KE. Concussion in sport: diagnosis, management, return to competition. *Compr Ther* 1999;25:39–44;discussion 45.
141. Teasdale G. Head injuries. In: *Oxford Textbook of Medicine*. Oxford: Oxford University Press, 1996:4044–4050.
142. Cantu RC. Return to play guidelines after a head injury. *Clin Sports Med* 1998;17:45–60.
143. Thomas S, Acton C, Nixon J, et al. Effectiveness of bicycle helmets in preventing head injury in children: a case control study. *Br Med J* 1994;308:173–176.
144. Mills NJ. Protective capability of bicycle helmets. *Br J Sports Med* 1990;24:55–60.
145. Fekete JF. Severe brain injury and death following minor hockey accidents: the effectiveness of the safety helmets of amateur hockey players. *Can Med Assoc J* 1968;99:1234–1239.
146. Cantu RC, Mueller FO. Catastrophic sports injuries in football 1977–1989. *J Spinal Disord* 1990;3:227–231.
147. Hickey J, Morris A, Carlson L, et al. The relation of mouth protectors to cranial pressure and deformation. *J Am Dent Assoc* 1967;74:735–740.
148. Chapman P. Concussion in contact sports and importance of mouthguards in protection. *Aust J Sci Med Sport* 1985;3:170–174.
149. Stenger J, Lawson E, Wright J, Ricketts J. Mouthguards, protection against shock to the head, neck and teeth. *J Am Dent Assoc* 1964;69:273–281.
150. Ommaya A, Rockoff S, Baldwin M. Experimental concussion. *J Neurosurg* 1964;21:241–265.
151. McCrory P, Berkovic S. Videoanalysis of the motor and convulsive manifestations of concussion in acute sport related head injury. *Neurology* 2000;54:1488–1491.
152. Leninger B, Gramling S, Farrell A, et al. Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *J Neurol Neurosurg Psych* 1990;53:293–296.
153. Lovell M, Iverson G, Collins M, et al. Does loss of consciousness predict neuropsychological decrements after concussion. *Clin J Sports Med* 1999;9:193–199.
154. Weishmann U, Symms M, Clark C, et al. Blunt head trauma associated with widespread water-diffusion changes. *Lancet* 1999;353:1242–1243.
155. Lishman W. Physiogenesis and psychogenesis in the post concussional syndrome. *Br J Psychiatr* 1988;153:460–469.
156. Jacobsen R. The post concussional syndrome: physiogenesis and malingering. An integrative model. *J Psychosomatic Res* 1995;39:675–693.
157. Binder L. Persisting symptoms after mild head injury: a review of the post concussive syndrome. *J Clin Exp Neuropsychol* 1986;8:323–346.
158. Barth JT, Macciocchi SN, Giordani B, et al. Neuropsychological sequelae of minor head injury. *Neurosurg* 1983;13:529–533.
159. Levin HS, Grafman J, Eisenberg HM, eds. *Neurobehavioural Recovery From Head Injury*. Oxford: Oxford University Press, 1987.
160. Dikmen S, McLean A, Temkin N. Neuropsychological and psychological consequences of minor head injury. *J Neurol Neurosurg Psych* 1986;49:1227–1232.
161. Andersen S. Post concussional disorder and loss of consciousness. *Bull Am Acad Psychiatr Law* 1996;24:493–504.
162. Karzmark P, Hall K, Englander J. Late onset post concussion symptoms after mild brain injury: the role of pre-morbid, injury-related, environmental and personality factors. *Brain Inj* 1995;9:21–26.
163. McCrory PR, Ariens MF, Berkovic SF. The nature and duration of acute concussive symptoms in Australian football. *Clin J Sports Med* 2000;10:235–239.
164. McIntosh AS, McCrory PR. Impact energy attenuation performance of football headgear. *Br J Sports Med* 2000;34:337–342.