CHAPTER 6

Who should retire after repeated concussions?

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Introduction

The decision to medically retire an athlete following repeated concussive injuries remains a complex and controversial area. For the most part, there are no evidence-based recommendations to guide the practitioner, and the published "guidelines" have little or no scientific validity.[1] This view has been reinforced by the recent Vienna and Prague Consensus conferences, which emphasized the need to individualize clinical management and avoid reliance on anecdotal recommendations.[2,3]

In situations in which the athlete has suffered a life-threatening severe brain injury, has persistent neurological symptoms, or has a residual neurologic deficit, the decision to retire is straightforward. At the end of the day, good clinical judgment and common sense remain the mainstay of management.

In the broadest sense, an athlete who is closely monitored with stable neuropsychological testing throughout his or her career, and who has no ongoing postconcussive symptoms, has little to be concerned about even after sustaining a number of concussive injuries.

Methodology

The relevant literature was searched by using MEDLINE (1966–2005) and SportDiscus (1975–2005) searches, hand searches of journals and reference lists, and discussions with experts and sporting organizations worldwide. In addition, a keyword search was carried out on the author's EndNote database of over 6000 articles on sport-related concussive injuries. The keywords and Medical Subject Headings (MeSH) terms used in all searches included concussion, brain injury, head injury, head trauma, brain trauma, sports injuries, and brain commotion.

Background

There is no scientific evidence that sustaining several concussions over a sporting career will necessarily result in permanent damage. Part of the neuromythology surrounding concussion is the "three-strike rule"—namely, if an athlete has three concussions, then he or she should be ruled out of competition for an arbitrary period of time. On occasions, the athlete's sports participation is permanently curtailed. This anecdotal approach was

originally attributed to Quigley in 1945 and subsequently adopted by Thorndike, who suggested that if any athlete suffered "three concussions, which involved loss of consciousness for any period of time, the athlete should be removed from contact sports for the remainder of the season."[4] This approach has no scientific validity, but it continues to be the anecdotal rationale underpinning most of the current return-to-play guidelines.

The unstated fear behind this approach is that an athlete suffering repeated concussions would suffer a gradual cognitive decline similar to the so-called "punch-drunk" syndrome or chronic traumatic encephalopathy seen in boxers.[5–7] On the basis of published evidence, this fear is largely unfounded, and recent developments suggest that the risk of traumatic encephalopathy in this setting may be largely genetically based, rather than being simply a manifestation of repeated concussive injury.[8]

The issue becomes further confused when well-known athletes suffering from recurrent head trauma appear in the media or lay press. In some cases, the injuries suffered by such athletes are more severe than the typical sports-related concussive injuries, but no distinction is made in the mind of the public. In such injuries, long-term symptoms are not wholly unexpected. In other cases, the so-called "postconcussive" symptoms experienced are mostly headache. This symptom is nonspecific and can be the result of a variety of causes other than concussion.[9]

Adding to the difficulty is the fact that when professional athletes suffer repeated concussions, they are not banned from sport, as may be the advice given to less elite athletes. Whilst it is true that many professional athletes are monitored more closely than other sporting participants, nevertheless the variation in management advice between elite and recreational athletes is often seen as hypocritical, resulting in a situation in which recreational athletes prematurely return to sport after concussion and as a result suffer ongoing problems.

Much of the concern in relation to the management of repeated concussive injury relates to the absence of consensus in the grading of the severity of concussion injury and the lack of scientifically valid return-to-play guidelines. Until these central issues are resolved, it is unlikely that a clear answer to the problem of retirement due to chronic symptoms will emerge.

Definition of concussion

Until recently, there was no universal agreement on the standard definition or nature of sports concussion.[2,3] Historically, the term has been used to refer to a transient disturbance of neurological function caused by the "shaking" of the brain that accompanies low-velocity brain injuries.[10]

The definition published by the Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons was widely used to define concussive injuries until 2001. This definition states that concussion is "a clinical syndrome characterized 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."[11] Over time, however, it had become clear that this definition really did not adequately define the clinical entity, and as a result an expert consensus conference on sports concussion was held in Vienna in 2001, with a second meeting being held in Prague in 2004.[2,3] The Prague meeting redefined sports concussion as follows:

• Sports concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

— Concussion may be caused either by a direct blow to the head, face, neck, or elsewhere on the body, with an "impulsive" force transmitted to the head.

— Concussion typically results in the rapid onset of short-lived impairment of neurological function, which resolves spontaneously.

— Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.

— Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.

— Concussion is typically associated with grossly normal structural neuroimaging studies.

One of the key developments of the Prague Conference was the understanding that concussion may be categorized for management purposes as either *simple* or *complex*.[3] These categories were defined as follows:

• In *simple concussion*, an athlete suffers an injury that progressively resolves without complications over 5–10 days. In such cases, apart from limiting playing or training whilst the patient is symptomatic, no further intervention is required during the period of recovery, and the athlete typically resumes sport without further problem. This form of concussion represents the most common form of this injury, and can be easily managed by primary-care physicians or by certified athletic trainers working under medical supervision.

• *Complex concussion* encompasses cases in which athletes suffer persistent symptoms, specific sequelae, or prolonged cognitive impairment following the injury. This group may also include athletes who suffer multiple concussions over time, or in whom repeated concussions occur with progressively less impact force. In this group, there may be additional management considerations beyond simple return-to-play advice, and it is envisaged that such athletes would be managed in a multidisciplinary manner by physicians with specific expertise in the management of concussive injury, such as a sports neurologist or neurosurgeon.

Whilst this distinction remains a speculative and as yet scientifically unvalidated approach, it reflects the clinical situation, in which the majority of athletes recover quickly after concussion and return to sport without further difficulty; however, there is a small percentage of athletes who suffer persistent symptoms or persistent disability due to the injury. It was therefore recommended that the latter group should be managed more aggressively by concussion experts.

These proposed concussion subtypes may represent differences in individual clinical phenomenology (confusion, memory problems, loss of consciousness), anatomical localization (e.g., cerebral vs. brainstem), biomechanical impact (rotational vs. linear force), genetic phenotype (ApoE4-positive vs. ApoE4-negative), neuropathological change (structural injury vs. no structural injury), or an as yet undefined difference. These factors may operate independently or interact with each other. It is clear that the variations in clinical outcome with the same impact force require a more sophisticated approach to the understanding of this phenomenon than the approaches currently available.

Published guidelines for return to sport after concussion

The published guidelines recommending termination of all contact sport following three concussions during the course of an athletic season need to be considered by clinicians extremely carefully. In the absence of documented objective evidence of brain injury or clinical evidence of persistent postconcussive symptoms, there is no scientific support for this generalization, and the abandonment of such anecdotal approaches has been recommended by two international expert consensus conferences.[2,3]

It is also worth considering that athletes excluded from competition on such a basis may consider a medicolegal appeal that would be impossible to defend in a court of law.

The principal anecdotal guidelines are outlined below for discussion purposes, and—as mentioned above—these are not supported by published scientific evidence and should be considered management "opinions" at best.

The main guidelines on returning to sport after repeated concussive injury are those published by Cantu[12–14] and the Colorado Medical Society.[15] The American Academy of Neurology guidelines[16] are derivative from the latter (Tables 6.1, 6.2).

It can be seen that there are many superficial similarities between the two scale systems. Although the criteria for the severity of injury differ, the mandatory requirement is that two grade 3 injuries or three injuries of any grade should result in termination of the athlete's season. As a Cantu Grade 2 is equivalent to a Colorado Grade 3, it is evident that the scales give differing recommendations for the same injury.

The physiology of concussion

At present, there is no existing animal model or other experimental model that accurately reflects a sporting concussive injury. It has been noted in experimental models of more severe injury that a complex cascade of biochemical, metabolic, and membrane gene-expression changes occur.[17] Whether similar metabolic changes occur in sports concussion, however, is still currently speculative.[18]

Table 6.1 Return-to-sport guidelines: Cantu system (adapted from Cantu 1986[12])

Severity grade	1st concussion	2nd concussion	3rd concussion
Grade 1 No LOC, PTA < 30 min	RTP after 1 week if asymptomatic	RTP in 2 weeks if asymptomatic for at least 1 week	Terminate season. RTP next season if asymptomatic
Grade 2 LOC < 5 min, PTA > 30 min	RTP after 1 week if asymptomatic for at least 1 week	Minimum of 1 month off sport. RTP if asymptomatic for at least 1 week. Consider terminating season	Terminate season. RTP next season if asymptomatic
Grade 3 LOC > 5 min, PTA > 24 h	Minimum of 1 month off sport. RTP if asymptomatic for at least 1 week	Terminate season. RTP next season if asymptomatic	

LOC, loss of consciousness; PTA, post-traumatic amnesia; RTP, return to play.

Table 6.2 Return-to-sport guidelines: the Colorado guidelines (adapted from Kelly *et al.* 1991[15])

Severity grade	1st concussion	2nd concussion	3rd concussion
Grade 1 No LOC, confusion, no amnesia	RTP after 20 min if asymptomatic	RTP if asymptomatic for at least 1 week	Terminate season. RTP next season if asymptomatic
Grade 2 No LOC, confusion, amnesia	RTP after a minimum of 1 week with no symptoms	RTP after a minimum of 1 month with no symptoms for at least 1 week	Terminate season. RTP next season if asymptomatic
Grade 3 LOC	RTP after a minimum of 2 weeks with no symptoms.	Terminate season. RTP next season if asymptomatic	Terminate season. RTP next season if asymptomatic

LOC, loss of consciousness; RTP, return to play.

Although experimental research has enhanced our understanding of the physiological changes to the brain following severe head trauma, there is still uncertainty as to what is happening to the human brain following minor concussive injuries, and in particular in sport-related concussion.

The neuropathology of concussion

The nature of transient loss of cerebral function following a blow to the head has excited much speculation over the centuries regarding whether microscopic neuropathological changes occur, or whether other cerebral pathophysiological processes manifest the clinical symptoms of concussion. At this stage, these important issues remain unresolved. In general terms, although it is well accepted that minor neuropathological changes may occur following concussive brain injury, the clinical symptoms are due to functional disturbance, presumably at the cell membrane level, rather than due to any underlying structural injury. This is supported by experimental evidence demonstrating that mechanical stress can produce a sudden neuronal depolarization followed by a period of nerve-cell transmission failure, in the absence of structural injury.[19]

Human models of concussion are necessarily limited, given that virtually all patients recover without detectable permanent sequelae. In the handful of case reports of individuals dying of other causes after brain injury, scattered neuronal cell death can be demonstrated. However, the findings are generally insufficient to explain the degree of clinical dysfunction, suggesting that the clinical symptoms become manifest through additional functional cell impairment.[20]

The neuropsychology of concussion

The application of neuropsychological testing in concussion has been shown to be of value, and it continues to contribute significant information to the evaluation of concussion.[21–24] It has been demonstrated that cognitive recovery may precede or follow clinical symptom resolution, suggesting that the assessment of cognitive function should be an important component in any return-to-play protocol.

It must be emphasized, however, that neuropsychological assessment should not be the sole basis of a return-to-play decision, but should rather be seen as an aid to clinical decision-making. Although neuropsychological screening may be carried out or interpreted by nonclinicians, the final return-to-play decision should remain a medical one.

It is only in the past few decades that there has been interest in studying the neuropsychological consequences of concussion, and particularly of those injuries seen in sport.[21,25–28] While there is now acceptance that there is an organic basis to the problems associated with concussion, controversy remains regarding the nature of the cognitive deficits, as well as the speed and extent of recovery from them.

A range of neuropsychological deficits has been reported after mild concussive injury. The major areas of deficit include:

- Disturbances of new learning and memory.[29–38]
- Planning and the ability to switch mental "set."[29,35,38,39]
- Reduced attention and reduced speed of information processing.[29,34,35,40–47]

There have also been isolated reports suggesting that impairments may be evident in tasks involving visuospatial constructional ability, language, and sensorimotor function.[29,35]

Recovery of neuropsychological function after concussion in sport

In general terms, there appears to be clear evidence of neuropsychological deficits during the first week after mild concussive injury, but variable findings tend to develop after that period.[1,27,28]

There are a number of methodological issues that may underlie the inconsistencies reported between studies—including test selection, different mechanisms of injury, and varying severities of injury. In the various studies, wide variations in the severity of injury have been included under the rubric of concussion, ranging from no loss of consciousness (LOC) through to LOC for 1 week or more, and mild stunning of the sensorium for a few seconds through to patients with post-traumatic amnesia lasting for 4 months.

In addition, concussive injuries may result from a number of different causes, such as motor-vehicle accidents, sporting injuries, falls, and domestic trauma. This heterogeneity may account for some of the differences between studies, since the magnitude of the head acceleration forces may differ considerably depending on the cause.

With regard to the various neuropsychological test instruments used in the different studies, a number of methodological issues arise, including test selection, lack of sensitivity of various tests, practice effects, inadequate identification of premorbid characteristics influencing test results, inconsistent time points for testing, lack of suitable control groups, small sample sizes, and compensation issues.[48–50]

The postconcussion syndrome

The issue of the constellation of physical and cognitive symptoms that have been labeled as "postconcussive syndrome" (PCS) is as controversial today as when it was first proposed in the 19th century.[51]

PCS may include symptoms such as headache, vertigo, dizziness, nausea, memory complaints, blurred vision, noise and light sensitivity, difficulty in concentrating, fatigue, depression, sleep disturbance, loss of appetite, anxiety, incoordination, and hallucinations.[36,52–55]

Table 6.3 Postconcussion symptoms scale (adapted from Lovell and Collins 1998[26])

	Rating						
	None	**Moderate**			**Severe**		
Headache	0	1	2	3	4	5	6
Nausea	0	1	2	3	4	5	6
Vomiting	0	1	2	3	4	5	6
Drowsines	0	1	2	3	4	5	6
Numbness or tingling	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sleeping more than usual	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
More emotional than usual	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervousness	0	1	2	3	4	5	6
Other	0	1	2	3	4	5	6

Various PCS scales are widely used in sports-concussion assessment (Table 6.3). Although debate continues regarding the relative contribution of organic versus psychological factors in the genesis of PCS, the critical factor that clinicians need to be aware of is that these symptoms are nonspecific in nature and are not confined to concussion. It has been demonstrated that up to 60% of uninjured individuals may report PCS symptoms, and similarly high scores have been demonstrated in various medical and psychological illnesses.[56]

The risk of repeat concussions in sport

It has become a widely held belief that after sustaining a concussive injury, one is then more prone to future concussive injury. The evidence for this is limited at best. In a widely quoted study by Gerberich *et al.*, which involved self-reported questionnaires concerning a history of head injury in high-school gridiron footballers, an increased risk of subsequent concussions was reported in players with a history of concussion.[57] This study is flawed by significant methodological problems. Not least is the fact that the authors included cases of catastrophic brain injury. Furthermore, the reliability of a self-diagnosis of concussion is questionable, given that only 33% of those with loss of consciousness and 12% of those with other symptoms were medically assessed. The majority of the diagnoses of "concussion" were made by the coach, other team-mates, or by the players themselves.

It would seem obvious that in any collision sport, the risk of concussion is directly proportional to the amount of time for which a participant plays the sport. In other words, the more games played, the greater the chance of an injury occurring. The likelihood of a repeat injury may therefore simply reflect the level of exposure to injury risk.

In addition, Gerberich acknowledges that the observed increased likelihood of concussion could also be explained by a player's style of play. The player's risk of injury may be increased by using dangerous game strategies and illegal tackling techniques. Similar criticisms can also be leveled at another retrospective study, in which it was reported that once an initial concussion was sustained, the probability of incurring a second concussion greatly increases.[58]

Does repeat concussion result in cumulative damage?

Apart from boxing-related head injuries, the most widely cited studies on the cumulative effects of concussion have included patients with injuries sustained in motor-vehicle accidents that were severe enough to warrant hospital treatment. Generally, concussive injuries suffered in collision sports such as football involve lesser degrees of acceleration–deceleration forces than those experienced in motor-vehicle accidents.[34,43,59–61]

The limitations of retrospective studies in concussion, such as the widely cited motor-vehicle accident studies by Gronwall and Wrightson, include diagnostic uncertainty, relying on both self-reported injury recall, as well as the lack of medically validated injury diagnosis. For example, some head injuries in the cited studies were retrospectively assessed up to 8 years after their occurrence.[34,43,59–61]

It is widely acknowledged that boxing carries a high risk of neurological injury. However, boxing should not be considered as a model for cumulative head injury seen in other sports, since it presents unique risks to the athlete in terms of the frequency of repetitive head trauma.[7,62] Recently, specific genetic abnormalities have been reported as the major risk factor for the development of traumatic encephalopathy.[63,64]

In another series of retrospective studies involving retired Scandinavian soccer players, cognitive deficits were noted.[65–68] In these studies, significant methodological problems flaw the results. The problems include a lack of pre-injury data, selection bias, lack of observer blinding, and inadequate control subjects. The authors conclude that the deficits noted in the former soccer players were explained by repetitive trauma such as that caused by heading the ball. However, the pattern of deficits is equally consistent with alcohol-related brain impairment, a confounding variable that was not controlled for. To date, there has been no replication of these findings by other independent groups.[69–74]

In other retrospective studies involving a wide range of traumatic brain injury, loss of consciousness was associated with evidence of permanent change in fine motor control.[75] The significance of this symptom in isolation from other cognitive domains is questionable. Other studies have suggested that this may be an effect of environmental factors rather than being due to the effect of injury.[76] More recent prospective studies have failed to find any adverse prognostic features in individuals who suffered a loss of consciousness with concussion in comparison with those who did not.[35,77,78]

In animal studies of experimental concussion, animals have been repeatedly concussed 20–35 times during the same day and within a 2-hour period. Despite these unusually high numbers of injuries, no residual or cumulative effect was demonstrated.[79]

Is there a genetic susceptibility to brain injury in sports?

Recent research in boxers has suggested that chronic traumatic encephalopathy, or the so-called "punch-drunk syndrome," in boxers may be associated with a particular genetic

predisposition. The apolipoprotein E epsilon-4 (ApoE) gene, a susceptibility gene for late-onset familial and sporadic Alzheimer's disease, may be associated with an increased risk of chronic traumatic encephalopathy in boxers.[63,80,81] In a nonboxing population, ApoE polymorphism was significantly associated with death and adverse outcomes following acute traumatic brain injury, as seen in a neurosurgical unit.[82] In a recent prospective study, ApoE genotypes were tested for their ability to predict days of unconsciousness and functional outcome after 6 months.[83] A strong association was demonstrated between the ApoE allele and a poor clinical outcome.

Furthermore, ApoE-deficient (knockout) mice have been shown to have memory deficits, neurochemical changes, and diminished recovery from closed head injury in comparison with controls.[84] It is suggested that ApoE plays an important role in both neuronal repair[85] and antioxidant activity,[84] resulting in ApoE knockout mice exhibiting an impaired ability to recover from closed head injury. Although we are only in the early stages of understanding these issues, an interaction between genetic and environmental factors may be critical in the development of postconcussive phenomena or concussive sequelae.

Return to sport after life-threatening head injury

The return to sport following a severe or potentially life-threatening brain injury is controversial, and there are few guidelines for the clinician to follow. There are some situations in which the athlete might place himself or herself at an unacceptably high risk of sustaining further injury and hence should be counseled against participating in collision sports. In such situations, common sense should prevail.

Although sports physicians should keep an open mind when assessing neurological recovery from severe brain injuries, it is nevertheless recommended that at least 12 months should pass before such a decision is contemplated.

Thoughtful deliberation and analysis of all the available medical evidence should occur when such decisions are being taken. It is also recommended that the advice of a neurologist or neurosurgeon experienced in the management of sports head injuries should be sought. This is an important point, because a number of individuals who suffer a moderate to severe traumatic brain injury may be left with a lack of insight and impaired judgment over and above their other neurological injuries. This in turn may make such an individual unreliable in gauging recovery. The use of neuropsychological assessment, as well as information from family and friends, may assist the clinician in his or her deliberation. The assessment of cognitive performance and/or clinical symptoms when the patient is fatigued is often useful.

Returning to collision sport is relatively contraindicated in almost any situation in which a surgical craniotomy is performed. In such situations, the subarachnoid space is traumatized, thus setting up scarring of the pia–arachnoid of the brain to the dura, with both loss of the normal cushioning effect of the cerebrospinal fluid and vascular adhesions that may subsequently bleed if torn during head impact. Even if neurologic recovery is complete, a craniotomy for anything other than an extradural hematoma effectively precludes a return to collision sport (Table 6.4).

When there is an epidural hematoma without brain injury or other condition in which surgery is not required, a return to sport may be contemplated in selected patients, as per the discussion above, after a minimum of 12 months—assuming that neurologic recovery is complete.

Table 6.4 Conditions contraindicating a return to contact sport (adapted from Cantu 1998[13])

- Persistent postconcussional or post-injury symptoms
- Permanent neurological sequelae—hemiplegia, visual deficit, dementia or cognitive impairment
- Hydrocephalus with or without shunting
- Spontaneous subarachnoid hemorrhage from any cause
- Symptomatic neurologic or pain-producing abnormalities around the foramen magnum
- Craniotomy for evacuation of intracerebral or subdural hematoma

Conclusions

Who should retire after recurrent concussive injury? It appears self-evident that athletes with persistent cognitive or neurological symptoms should be withheld from collision sports until such time as their symptoms fully resolve. Following more severe brain injury, persistent neurological deficit, or symptoms, a history of craniotomy or intracranial surgery and spontaneous subarachnoid hemorrhage should preclude further participation.

In a setting of repeated uncomplicated concussive injury with full recovery after each episode, the situation is more difficult. Although published guidelines exist, they do not have any scientific validity and should be seen only as anecdotal suggestions for the clinician. It is the author's practice in professional sport to perform neuropsychological testing routinely on all athletes before the season and serially after concussive injury. More importantly, no athlete returns to sport until he is symptom-free and has returned to his or her neuropsychological baseline performance.

The central issue relates to the nature of the injury. Whilst there is no doubt that severe concussion with persistent symptoms occurs (the "complex concussion" in the Prague guidelines), athletes with typical concussive injuries recover quickly and return to sport without difficulty. In this setting, the scientific evidence that sustaining a number of concussions over the course of a season, or over a career, causes chronic neurological dysfunction is nonexistent. Clinicians should be aware of the neuromythology surrounding this issue and should manage their patients using evidence-based guidelines—or, if guidelines are lacking, then using good common sense.

Key messages

• No evidence-based guidelines exist in relation to the return to sport after repeated concussions.

• Persistent neurological symptoms or cognitive impairment should preclude a return to sport, but once these have resolved, there is no evidence that an athlete is at risk of long-term sequelae from concussive injury.

Sample examination questions

Multiple-choice questions (answers on p. 602)

1 In athletes, the presence of an ApoE4 phenotype (4/4) has been demonstrated to:
   A Confer a worse prognosis following traumatic brain injury
   B Be associated with chronic traumatic encephalopathy ("punch-drunk syndrome") in boxers

C Be associated with a poorer neuropsychological performance on postinjury assessment
D Be associated with persistent postconcussive symptoms

2 Contraindications to a return to sport after severe traumatic brain injury include:
A Persistent postconcussional or post-injury symptoms
B Permanent neurological sequelae—hemiplegia, visual deficit, dementia, or cognitive impairment
C Craniotomy for evacuation of intracerebral or subdural hematoma
D Spontaneous subarachnoid hemorrhage from any cause
E Symptomatic abnormalities about the foramen magnum

3 The *common* neuropsychological deficits noted following acute concussive injury in sport include:
A Disturbances of new learning and memory
B Reduced ability to switch mental "set"
C Reduced speed of information processing
D Impairment in visuospatial constructional ability
E Language disturbance

4 Are these statements regarding simple concussion from the Prague guidelines true or false?
A In simple concussion, concussion symptoms lasts less than 7–10 days
B Simple concussion can be managed by primary-care physicians
C Simple concussion includes cases in which a concussive convulsion has occurred
D Approximately 95% of concussions fall into this category

Essay questions

1 A 30-year-old professional American football quarterback suffers the 10th concussion of his career during a midseason game. His team is due to make the play-offs, and his presence is crucial for the success of the team. How would you monitor his recovery and determine whether he should return to play?

2 A rugby player suffers a severe head injury in a fight at a club one evening. As a result, he is taken to the regional neurosurgical center, where a craniotomy for intracranial pressure control is required. He recovers, and the skull defect is closed successfully. He comes to see you for advice on return to play. His Glasgow Coma Scale (GCS) score is 15 and he has no focal neurological signs. How do you approach the problem, and what advice would you give?

3 A 24-year-old professional soccer player sees you because of persistent headaches from "heading" the ball. He is worried that repeated heading may cause him to be "punch-drunk" in later life. What advice do you give him? Are there any tests that could assist you in advising him?

Case study 6.1

An Australian-Rules footballer gives a history of sustaining one or two episodes of concussion with loss of consciousness, as well as having four or five minor (no LOC) concussions per season. Despite this, he has no ongoing symptoms or neurological signs. Following each episode, he is withheld from sport until he is symptom-free and his neuropsychological tests have returned to baseline. During his 8-year professional career, no decrement in cognitive performance has been noted. His neuroimaging studies are normal, and his

ApoE4 status is negative (i.e., heterozygous allele). Despite the history of multiple concussions, there is no evidence of ongoing or permanent neurological injury.

Summarizing the evidence

Guidelines	Results	Level of evidence*
Definition	2 published papers	C
Injury severity guidelines	42 published guidelines	C
Return-to-play recommendations	5 published guidelines	C/D
Vienna/Prague guidelines	2 published papers	C
Retirement	3 published guidelines	C
Injury prevention		
Helmets	2 RCTs—helmets not protective	A1
Mouthguards	1 RCT—mouthguards not protective	A3
Rule change	2 published papers	C

* A1: evidence from large randomized controlled trials (RCTs) or systematic review (including meta-analysis).†
A2: evidence from at least one high-quality cohort.
A3: evidence from at least one moderate-sized RCT or systematic review.†
A4: evidence from at least one RCT.
B: evidence from at least one high-quality study of nonrandomized cohorts.
C: expert opinions.
D: case series.
† Arbitrarily, the following cut-off points have been used: large study size: ≥ 100 patients per intervention group; moderate study size ≥ 50 patients per intervention group.

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