# When to Consider Retiring an Athlete After Sports-Related Concussion

Cara L. Sedney, MD[a,*], John Orphanos, MD[b], Julian E. Bailes, MD[b]

**KEYWORDS**

• Concussion • Mild traumatic brain injury • Athletes

The decision to retire an athlete due to sports-related concussion is often a controversial one, fraught with conflicting pressures from the athlete and others involved in the sport. Postconcussion syndrome has long been recognized as resulting from participating in sports and is discussed in sports medicine texts.[1,2] The retirement of several National Football League (NFL) players in the 1990s from chronic postconcussive effects has recently brought this issue into the public eye. Currently, the decision to retire an athlete after concussion is based on several basic principles of neurology, sports medicine, and concussion management. This knowledge in turn stems from the biology of concussion and traumatic brain injury and from increasing understanding of the effect of chronic brain trauma. Although concussion guidelines continue to play an important role in acute management of concussion, the decision to retire an athlete is a more complex one, dependent on myriad factors.

## SOCIAL AND LEGAL IMPLICATIONS IN THE DECISION TO RETIRE

Concussive injury has been observed to manifest in four stages (**Table 1**) with a distinct temporal association. In the acute concussive stage lasting up to 1 week after injury, physical symptoms, cognitive deficits, and emotional disturbances may be evident. This may be followed by a postconcussive stage consisting of persistent acute phase symptoms, which are nevertheless self-limited to approximately 6 weeks after mild traumatic brain injury (MTBI). A prolonged postconcussion syndrome lasting up to 6 months may be experienced and may be manifested by declining athletic, work, or school performance. Finally, a severe and rare form of chronic concussive injury,

[a] Department of Neurosurgery, West Virginia University, PO Box 9183, Robert C. Byrd Health Sciences Center, Morgantown, WV 26506, USA
[b] Department of Neurosurgery, West Virginia University, PO Box 9183, Robert C. Byrd Health Sciences Center, Morgantown, WV 26505, USA
* Corresponding author.
*E-mail address:* csedney@hsc.wvu.edu

Clin Sports Med 30 (2011) 189–200
doi:10.1016/j.csm.2010.08.005 **sportsmed.theclinics.com**
0278-5919/11/$ – see front matter 

**Table 1**
**Stages of concussive injury**

| Acute Concussion | Postconcussion Syndrome | Prolonged Postconcussion Syndrome | Chronic Traumatic Encephalopathy |
|---|---|---|---|
| Physical symptoms (headache, dizziness, hearing loss, balance difficulty, insomnia, nausea/vomiting, sensitivity to light or noise, diminished athletic performance)<br>Cognitive deficits (loss of short-term memory, difficulty with focus or concentration, decreased attention, diminished work or school performance)<br>Emotional disturbances (irritability, anger, fear, mood swings, decreased libido) | Persistent concussion symptoms<br>Usually lasting 1–6 weeks after MTBI<br>Self-limiting | Symptoms lasting over 6 months<br>Lowered concussion threshold<br>Diminished athletic performance<br>Diminished work or school performance | Latency period (usually 6–10 years)<br>Personality disturbances<br>Emotional lability<br>Marriage/ personal relationship failures<br>Depression<br>Alcohol/ substance abuse<br>Suicide attempt/ completion |

chronic traumatic encephalopathy (CTE), may manifest after a variable latency period and demonstrates profound personality changes and dysfunction in many areas of life.

## CONCUSSION PATHOPHYSIOLOGY

There is a greater understanding of concussion now than ever before. The pathophysiology of concussion is characterized by a variety of metabolic mechanisms, which are both acute and subacute in nature. On initial insult, a hypermetabolic state is created with excessive release of excitatory neurotransmitters, despite a decrease in cerebral blood flow.[3] This is characterized by ion movements, such as influx of calcium and efflux of potassium, causing extensive depolarization of neurons, along with decreased intracellular magnesium. Glycolysis is accelerated and lactate accumulates, causing neuronal membrane damage and resulting in altered blood-brain barrier permeability.[3] This initial hypermetabolic state is followed by a hypometabolic state of widespread neuronal suppression, called a spreading depression,[3] possibly due to the relative decrease in cerebral blood flow. Increased intracellular calcium leads to mitochondrial dysfunction but rarely leads to cell death in experimental concussion.[3] Neuronal dysfunction results from the decrease in magnesium, accumulation of lactate, and relative hypoxia. In addition, mechanical factors, such as stretching or tearing of neuronal axons, may further result in membrane disruption and ion fluxes.

These pathophysiologic changes usually occur over a specific time frame and in a specific sequence, accounting for the stepwise progression of symptoms after sports-related concussion, and also relate to the susceptibility of more severe injury after an initial insult. Although new pathologic methods have shown an anatomic basis for chronic concussive changes, overall, these effects have classically been thought of as physiologic rather than structural in nature and account for the majority of symptoms-based guidelines in the management of concussion. Ordinarily, brain imaging

studies are normal, although postconcussive positron emission tomography may reflect the decreased glucose metabolism, and certain immunohistochemical stains may demonstrate neuronal damage (**Fig. 1**).

## LONG-TERM EFFECTS OF CONCUSSION

The long-term pathophysiology of repeated head trauma is particularly important to the decision to retire an athlete after sports-related concussion. Despite the early recognition of symptomatology in retired boxers with early neurodegenerative changes, many chronic concussive effects are still being explored and are incompletely understood. The first report of chronic, permanent neurologic sequelae in career boxers was by Martland[4] in 1928, which he termed, *dementia pugilistica* (DP). This introduced the possibility of long-term effects from sport in the form of advanced parkinsonism, pyramidal tract dysfunction, ataxia, and behavioral abnormalities in a 38-year-old man. It is believed that DP is present in approximately 20% of retired boxers. Since then, persistent concussive symptoms, such as memory problems; decreased attention, focus, or concentration; and mood swings or personality changes have been recognized as having long-term effects on athletic, work or school, and interpersonal functioning. These consequences may present at any age and any level of sporting competition (**Fig. 2**).

More recently, the discovery of specific pathologic findings during the autopsies of several well-publicized deaths among retired athletes has solidified the association of repeated head trauma with another rare condition, CTE, which causes severe cognitive and behavioral disturbances.[5,6] Much of research on chronic effects of head injury is based on clinical and autopsy observations rather than laboratory investigation. Both macro- and microscopic pathologic changes have been described. Corsellis and colleagues[7] describe gross pathologic findings in the brains of men who had been boxers, including cavum septum pellucidum, scarring of cerebellar folia, and degeneration of the substantia nigra. Microstructural changes may be more pertinent to the pathophysiology of chronic concussion, however, and include accumulation of tau protein in the form of neurofibrillary tangles (NFTs) and neuritic threads (NTs) (**Fig. 3**).

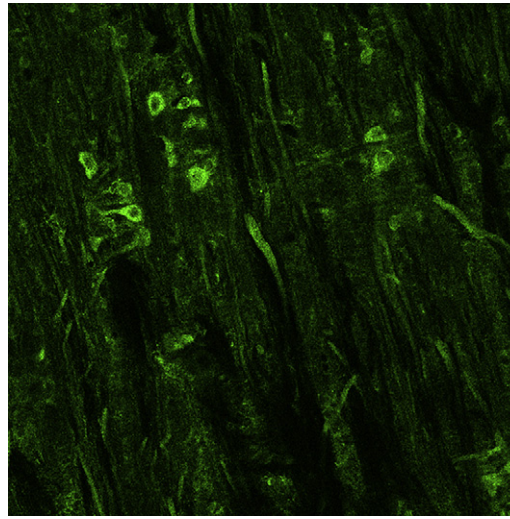

**Fig. 1.** Torn axons within the brainstem and corticospinal tract of the rat, shown via immunohistochemical staining of β-amyloid precursor protein after traumatic brain injury.

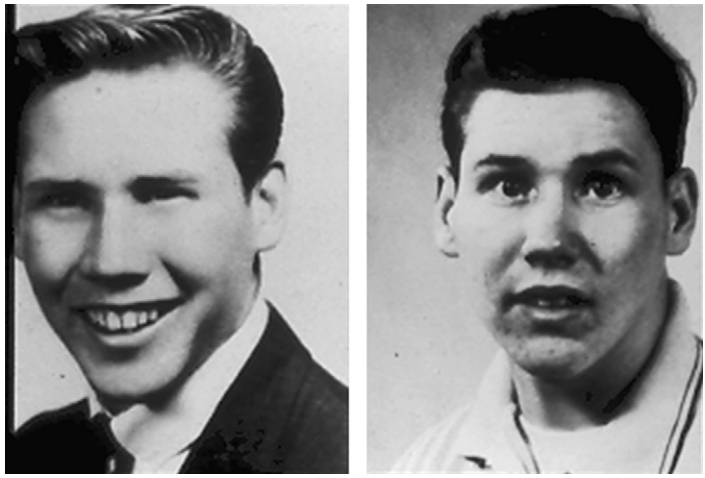

**Fig. 2.** Consecutive photographs of a high school football player before and after concussive injury sustained in sport. (*From* Schieder R. Head and neck injuries in football. 1st edition. Baltimore [MD]: Williams and Wilkins; 1973; with permission.)

Corsellis and colleagues[7] reported the first neurohistologic evidence of CTE on postmortem examination of boxers. Furthermore, recent case reports by Omalu and colleagues[5,6] illustrate histopathologic changes comprising a diffuse accumulation of tau proteins (NFTs and NTs) in patients after chronic, repeated exposure to brain impact during a sporting career. Their series of patients, including seven professional football players, two professional wrestlers, and one boxer, exhibited "diffuse, sparse to frequent" and "sparse to moderate" tau-immunoreactive NFTs and NTs.[6] The NFTs may take a variety of forms, including band-shaped, flame-shaped, and large and small perikaryal tangles. Both the NFTs and NTs are made up of helical, paired tau proteins.[8] Neuritic plaques may be found but were absent in several cases in Omalu and colleagues'[5] series, bearing resemblance to a rare type of dementia, known as tangle-only dementia.[9] The only direct confirmation of CTE remains direct postmortem examination of the brain with immunohistochemistry, because no neuroimaging study is capable of visualizing these changes.

CTE seems to affect the brain's emotional function, as reflected in the Papez circuit described by James Papez[10] in 1937. This limbic pathway plays a role in memory and emotion by connecting primitive areas of the brain, such as the hippocampal formation, mammillary bodies, entorhinal cortex, thalamus, and brainstem nuclei, and structures, such as the locus ceruleus.

The pathophysiology of CTE is believed due to microstructural changes within neurons because of the effects discussed previously, especially hyperphosphorylation of microtubule-associated tau protein.[11] It is found that the abnormal metabolism of injured neurons leads to accumulation of neuronal cytoskeletal and transmembrane proteins[12–14] and likely altered function of these neuronal elements. Further theories on the effect of protein accumulation include decreased efficacy of neuronal repair or increased susceptibility to reactive oxygen species.

## DECISION-MAKING PROCESS

The basic principles of sports medicine and acute concussion management deal with the implications of the pathophysiology (discussed previously), specifically, the

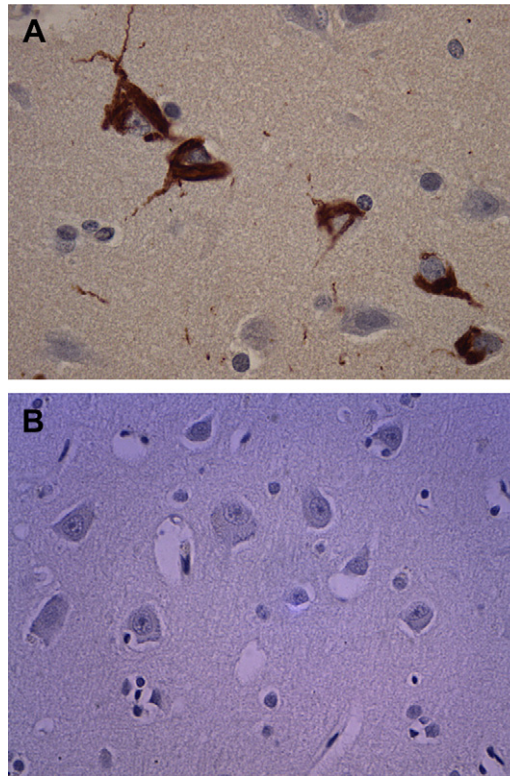


**Fig. 3.** (*A*) Tau antibody stain from the cerebral cortex of a professional football player showing typical changes of CTE: NFTs (large, globular intracellular inclusions) and NTs (whispy). (*B*) Normal control.

predictable, stepwise nature of symptoms, the susceptibility to further injury after initial insult, and the prevalence of physiologic rather than structural abnormalities. The statement of the First International Conference on Concussion in Sport, held in Vienna in 2001, succinctly outlined these principles.[15] These basic principles have led to the establishment of a variety of guidelines on concussion management. The Colorado Medical Society system, the Cantu grading system, and the American Academy of Neurology system are all currently in use. The Colorado and Cantu systems both include the recommendation that retirement should be considered after three or more concussions.

The decision to retire an athlete, however, is rarely due to one, or even several, incidents, but rather multiple concussions and subconcussive injuries over an extended period of time, many of which may not be diagnosed or recognized. Often the concussion guidelines do not apply in these cases. Furthermore, current research suggests that the effects of concussion may be cumulative, with new manifestations of this being seen in research and in emerging cases. Therefore, the decision to retire an athlete due to concussion must be made after extensive study of the patient's history and neurologic examination as well as advanced methods, such as neuropsychological testing and neuroimaging. **Box 1** lists several decisive factors for retirement, either from the season or from contact sports entirely.

**Box 1**
**Decisive factors for retirement[a]**

*Season Ending*

- Prolonged post concussion syndrome
- 3 or more concussions in single season
- 2 or more major concussions[b] in single season
- Diminished academic performance
- Diminished athletic performance
- CT or MRI brain scan abnormality

*Career Ending*

- Chiari malformation
- Intracranial hemorrhage
- Diminished academic performance or cognitive abilities
- Persistent prolonged post concussion syndrome
- Lowering of threshold for concussion (as judged by physicians, athletes, coaches, certified athletic trainers)
- 3 or more major concussions
- CT or MRI scan documentation of structural brain injury
- Nonresolving functional MRI scan deficits
- CTE symptoms

[a] All return to play and retirement decisions are individualized. Some features are relative contraindications for return to play.
[b] Major concussion: symptomatic for greater than 1 week.

The basis for retirement from contact sports is complex, and the many factors may be encountered in any stage of an athlete's career, from high school to professional levels. Retirement after concussion generally reflects the underlying brain injury and falls into one of the four categories (discussed previously) (see **Table 1**), depending on the timing of concussion. Some athletes may choose to retire due to intractable acute symptoms after concussion. This may include symptoms that interfere with play or acute catastrophic injury. Others may choose to retire due to postconcussive syndrome, which usually consists of self-limited sequelae of concussion, such as dizziness, headaches, and declining academic or athletic performance. Thirdly, more prolonged postconcussive effects are evident thereafter and may result in a decision to retire due to changes in performance, motivation, or personality, which again may manifest in declining athletic or academic abilities. Finally, in unique subsets of patients, displaying signs or symptoms of CTE may prompt a decision to retire.

### *History*

Exposure to MTBI is the primary historical information used in a decision to retire an athlete. The number, symptoms, and time to recovery after concussion are all important factors, as is a history of returning to play while still symptomatic. Although rare, a history of postconcussive seizures should be sought. The mechanism of concussion, if known, as well as the type of protective equipment, if any, being worn is also important. Information regarding current postconcussive symptomatology is important, and this is best

gleaned from the patient, family, athletic trainers, and others. The frequency and severity of headaches should be recorded. Mood swings, irritability, insomnia, lack of concentration, or impaired memory are pertinent. Personality changes may be pervasive. Any persistent postconcussive symptoms or permanent neurologic symptoms from a concussion, such as organic dementia, hemiplegia, and homonymous hemianopsia, should prompt investigation into retiring an athlete.[16] A history of declining school or athletic performance may be a sensitive indicator of both early and chronic changes after concussion and this is particularly important to elicit if present.

For a subset of patients with a long history of exposure, symptoms suggestive of CTE should be sought, such as explosive behavior, excessive jealousy, mood disorders, and paranoia. These symptoms often demonstrate latency from initial exposure and are progressive. Omalu and colleagues[5] performed an extensive postmortem psychological history during their series of 5 patients and unearthed a variety of common historical clues pointing to chronic cognitive and neuropsychological decline. These included drug and alcohol abuse, increasing religiosity, suicidal ideations or attempts, insomnia, hyperactivity, breakdown of intimate or family relationships, exaggerated responses to stressors, poor business or financial management, bankruptcy, and others.[5] Although the latency period of symptoms in CTE is not known, these should be investigated when interviewing an athlete and family for possible retirement after concussion.

### *Documented Brain Injury*

Specific, potentially catastrophic events after concussion, such as second impact syndrome and surgically treated brain injuries (eg, subdural hematoma), would ordinarily disqualify an athlete from further participation in contact sports. As discussed by Cantu,[16] any athlete surviving a second impact syndrome should retire from contact sports. In addition, any athlete requiring surgery for evacuation of an intracranial hemorrhage should be considered for retirement, due to multiple factors, including changes in cerebrospinal fluid dynamics[16] and the decrease in structural integrity of the skull, although some investigators have reported that, rarely, an athlete may be considered to return to play after craniotomy after healing of bony defect.[17] Non–sports-related concussions during an athlete's life, including childhood injuries, may also contribute to the magnitude of injury and these must be documented and considered.

### *Outcome of Examinations*

The proper management of an athlete after acute concussion requires accurate examination and testing to guide decision making regarding play. Several guidelines have been proposed to assist caregivers in determining an initial grade of concussion followed by a delayed concussion grading to improve accuracy in diagnosis and management. These principles assist in making a decision to retire an athlete.

The physical examination should center on any abnormal neurologic findings. A complete and detailed neurologic examination should be performed, searching for any focal abnormalities. Visual field testing should be completed. Tests of balance and coordination should be performed to assess for ataxia. Examination of reflexes for hyperreflexia as well as other long-tract findings should be documented. Patients should be observed for signs of parkinsonism. Mental status examination is an important clinical prelude to further neuropsychological testing. Even with extensive neurologic examination, findings may be normal in a large percentage of patients with recurrent concussion.

Neuropsychological testing has undergone an evolution in its sports applications, and the neurocognitive and neuropsychological features may be measured with

current methods. Neuropsychological impairment has been related to concussive and subconcussive injury in boxing, football, and soccer players, and neuropsychological testing is crucial in the determining the consequences of concussive injury. The number of concussive events has been shown by Guskiewicz and colleagues[18,19] to be significantly related to lifetime risk of depression as well as late-life cognitive impairment, with a suggestion that increasing number of concussions leads to higher prevalence. Several recent studies show the subtle cognitive effects of concussive injury and suggest that specialized testing of attention and information processing be used for assessment of postconcussive effects.[20] These discoveries have lead to a consensus statement among the participants of the most recent International Conference on Concussion in Sport held in Zurich, which emphasizes the importance of neuropsychological testing.[21] It has been recommended by Echemendia and colleagues[22] that clinical neuropsychologists, rather than athletic trainers or others, administer the complex psychometric tests to evaluate for these subtle abnormalities. Furthermore, it has been suggested that specifically developed computer-based tests be used, such as ImPACT, Cogsport, ANAM, and others.[23]

The advent of these computerized platforms, with access to the Internet and qualified neuropsychological opinion, make neuropsychological testing all the more practical and available for a larger population. For accurate assessment of effects of concussive injury on athletes before making a decision to end an athlete's career, it is imperative that these tests be administered. It has been shown by multiple sources that simple tests of intellectual and mental function do not reveal the true extent of cognitive decline. Furthermore, baseline neuropsychological examinations are important and preferred for comparison and more sensitive detection of decline. Although there are many nuances of correctly applying neuropsychological testing and interpreting their results, both traditional and computer-based instruments have proved invaluable in athletic concussion management. Therefore, neuropsychological testing, either in consultation with a neuropsychologist or computer based, is objective data that greatly aid sports medicine clinicians when deciding if retirement from sport should be pursued.

Imaging studies are integral for a full evaluation and should be performed. Although a CT scan may be performed to rule out any obvious abnormalities (eg, intracranial hemorrhage), the standard imaging method is MRI of the brain for further anatomic signs of trauma. Diffuse cerebral atrophy or ventriculomegaly may be encountered as well as other signs of chronic injury (**Fig. 4**). Imaging studies may reveal further, nontraumatic anatomic abnormalities and although these aspects may not be directly related to concussion, they too may prompt decision to retire an athlete. Discovery of any symptomatic abnormalities of the foramen magnum, such as a Chiari I malformation (**Fig. 5**), should prompt consideration of retirement, especially when combined with syringomyelia, obliteration of subarachnoid space, or indentation of the anterior medulla.[24] This has also been suggested for discovery of hydrocephalus or the incidence of spontaneous subarachnoid hemorrhage from any cause although no guidelines are currently in place.[16] More recently, MRI diffusion tensor imaging has demonstrated a correlation of increased fractional anisotropy and decreased radial diffusivity with increasing severity of postconcussive symptoms in adolescents after concussion.[25] This is currently increasing the ability to visualize and characterize postconcussive effects.

### *A Role for Future Genetic Testing*

Early research in CTE and other neurodegenerative diseases has found a trend of developing these diseases with certain genetic traits. Specifically, the ApoE4 and the ApoE3 alleles, in both homozygous and heterozygous forms, have been implicated

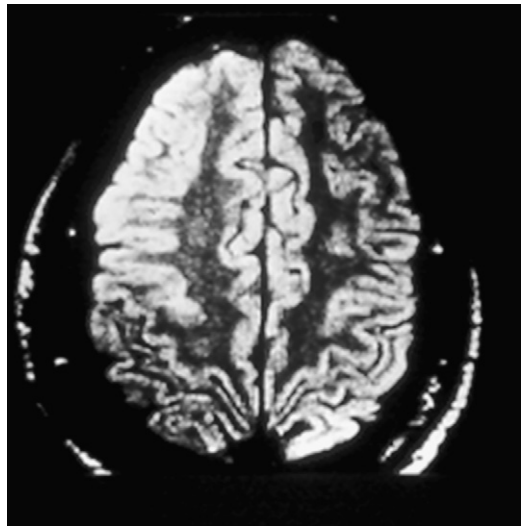

**Fig. 4.** MRI of a 24-year-old professional football player with career-ending postconcussion syndrome and a high-intensity lesion of the centrum semiovale.

in developing Alzheimer disease and CTE after brain trauma.[26,27] This was first discovered in a population of 30 career boxers and has since been confirmed in other populations with neurotrauma.[28] These proteins are various alleles of the apolipoprotein E, which is important for lipid transport and widely produced in the brain. Although this clinical science is still in its infancy, it may be possible to someday predict who will develop long-term problems from repeated head trauma in sports. Genetic testing, although raising ethical questions, may be a valuable tool for athletes who wish to know their risk for sustaining repeated concussive injury and may play a role in the decision to retire athletes in the future. Furthermore, although presence of hetero- or homozygous ApoE4 or ApoE3 confers a three- to ninefold increased risk of developing various forms of dementia after traumatic brain injury, the etiology is most likely multifactorial.

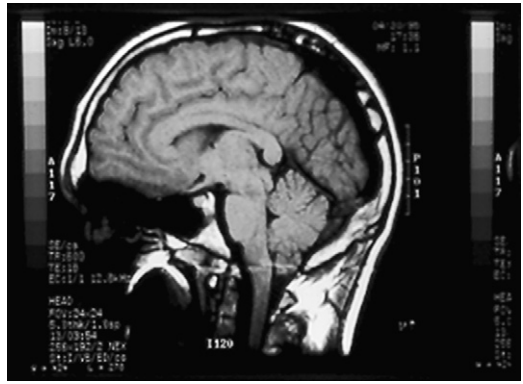

**Fig. 5.** Chiari malformation in a high school athlete who retired due to persistent postconcussion syndrome; a relative contraindication to continued contact sports participation.

### *Social and Legal Implications in the Decision to Retire*

One of the difficulties in a decision to retire an athlete based on concussive injury is noted by Bailes and Cantu,[29] who recognize that athletes generally desire to continue play, and thus continue their exposure to potential concussions. Although the decision to retire an athlete is a difficult one because of the many factors to be considered, it is made further contentious because of the many social factors involved. Not only the athlete but also the coach, team members, agents, and athlete's family have considerable stake in the decision to retire. Often athletes are under considerable financial stress to continue in their sport; and this can extend to all levels of competition, from high school athletes hoping for a college scholarship to professional athletes who may have limited other marketable skills and are supporting a family. These aspects are further confirmed by Gerberich and colleagues,[30] who noted that athletes are often reticent to describe their full symptomatology after concussive injury. This phenomenon may have less of an impact during a decision to retire an athlete compared with a return to play decision, because these debates are often initiated only after firm evidence of postconcussive injury is apparent to all involved. Nevertheless, social and financial factors may have a significant impact on this decision.

Despite the many complexities of a retirement decision, clearly the athlete must be involved in this life-changing decision. Ideally, this decision should be made with the athlete and family in agreement. At times, however, the very cognitive and neuropsychological decline that may prompt a decision to retire interferes with the executive functioning and judgment of the athlete. In this case, neuropsychological testing must be performed and documented regarding the extent of a patient's ability to participate in the retirement decision.

The decision to retire an athlete after concussion may be compounded by complicating factors. Goldberg[31] notes that frequently team-employed physicians have been cited as downplaying injury and encouraging return to play. He makes the case that sports-related health decisions should be made by independent physicians as in workers compensation claims. To avoid this complicating factor, some have advised for retirement assessments to be performed by an independent physician or to encourage a second opinion from one. This is currently being implemented by the NFL to provide local neurosurgeons and neurologists for "second opinion" consultations of concussion.

Finally, the decision to allow or disallow return to play when a question of retirement arises has several legal implications for treating physicians. The decision to retire is usually symptom driven. Furthermore, much has still to be learned about the potential for chronic brain injury and CTE. In spite of the uncertainty of the impact of concussive injury on long-term functioning, however, physicians nevertheless must weigh carefully the benefits and risks of return to play and counsel athletes accordingly; some have advocated a conservative approach.[32] Furthermore, physicians have a responsibility to provide athletes with full information about their medical condition and possible consequences of return to play so that their own decision is informed. In making this decision, the duty of a physician is to protect the health and safety of the athlete, regardless if he or she wants to return to play.

In summary, the effects of concussion may lead to a variety of short- and long-term effects, which may lead to the decision to retire from contact sports. These effects follow a recognizable progression and may cause an athlete to opt out of play at any point along this progression. To elucidate the effect of concussion or MTBI and weigh in on a decision to retire, the treating physician needs to take into account the history, neurologic examination, brain imaging, and neuropsychological testing.

In addition, myriad social factors surrounding play must be taken into consideration. As always, neurologic sports medicine requires an individualized approach.

**REFERENCES**


1. Schneider R. Head and neck injuries in football. 1st edition. Baltimore (MD): Williams and Wilkins; 1973.
2. Torg J. Athletic injuries to the head, neck, and face. 2nd edition. St Louis (MO): Mosby; 1991.
3. Giza C, Hovda D. The neurometabolic cascade of concussion. J Athl Train 2001; 36:228–35.
4. Martland H. Punch drunk. J Am Med Assoc 1928;91:1103–7.
5. Omalu B, Bailes J, Hammers J, et al. Chronic traumatic encephalopathy, suicides, and parasuicides in professional American athletes: the role of the forensic pathologist. Am J Forensic Med Pathol 2010;31:130–2.
6. Omalu B, Bailes J, Hamilton R, et al. Emerging histomophologic subtypes of Chronic Traumatic Encephalopathy (CTE) in American athletes. J Neurosurg, in press.
7. Corsellis J, Bruton C, Freeman-Browne C. The aftermath of boxing. Psychol Med 1973;3:270–303.
8. Omalu B, Fitzsimmons R, Hammers J, et al. Chronic traumatic encephalopathy in a professional American wrestler. J Forensic Nurs 2010;6:130–6.
9. Omalu B, Hamilton R, Kamboh M, et al. Chronic Traumatic Encephalopathy (CTE) in a National Football League Player: case report and emerging medicolegal practice questions. J Forensic Nurs 2010;6:40–6.
10. Papez J. A proposed mechanism of emotion. Arch Neurol Psychiatry 1937;38: 725–43.
11. Smith D, Chen X, Nonaka M, et al. Accumulation of amyloid beta and tau in the formation of neurofilament inclusions following diffuse brain injury in the pig. J Neuropathol Exp Neurol 1999;58:982–92.
12. DeKosky S, Abrahamson E, Ciallella J, et al. Association of increased cortical soluble abeta42 levels with diffuse plaques after severe brain injury in humans. Arch Neurol 2007;64:541–4.
13. Gabbita S, Scheff S, Menard R, et al. Cleaved-tau: a biomarker of neuronal damage after traumatic brain injury. J Neurotrauma 2005;22:83–94.
14. Ilkonomovic M, Uryu K, Abrahamson E, et al. Alzheimer's pathology in human temporal cortex surgically excised after severe brain injury. Exp Neurol 2004; 190:192–203.
15. Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. Br J Sports Med 2002;36:6–10.
16. Cantu R. Head injuries in sport. Br J Sports Med 1996;30:289–96.
17. Miele V, Bailes J, Martin N. Participation in contact or collision sports in athletes with epilepsy, genetic risk factors, structural brain lesions, or history of craniotomy. Neurosurg Focus 2006;21:E9.
18. Guskiewicz K, Marshall S, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. Med Sci Sports Exerc 2007; 39:903–9.
19. Guskiewicz K, Marshal S, Bailes J, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. Neurosurgery 2005;57:719–26.

20. Collins M, Grindel S, Lovell M, et al. Relationship between concussion and neuropsychological performance in college football players. JAMA 1999;282:964–70.
21. McCrory P, Meeuwisse W, Johnston K, et al. Consensus statement on concussion in Sport: the 3rd international conference on concussion in sport held in Zurich in November 2008. Br J Sports Med 2009;43(Suppl 1):i76–90.
22. Echemendia R, Herring S, Bailes J. Who should conduct and interpret the neuropsycholocial assessment in sports-related concussion? Br J Sports Med 2009; 43(Suppl 1):i32–5.
23. Cantu R. An overview of concussion consensus statements since 2000. Neurosurg Focus 2006;21:E3.
24. Callaway G, O'Brien S, Tehrany A. Chiari I malformation and spinal cord injury: cause for concern in contact athletes? Med Sci Sports Exerc 1996;28:1218–20.
25. Wilde E, McCauley S, Hunter J, et al. Diffusion tensor imaging of acute mild traumatic brain injury in adolescents. Neurology 2008;70:948–55.
26. Omalu B, Dekosky S, Minster R, et al. Chronic Traumatic Encephalopathy in a national football league player. Neurosurgery 2005;57:128–34.
27. Geddes J, vowles G, Robinson S, et al. Neurofibrillary tangles, but not Alzheimer-type pathology, in a young boxer. Neuropathol Appl Neurobiol 1996;22:12–6.
28. Jordan B, Relkin N, Ravdin L, et al. Apolipoprotein E4 associated with chronic traumatic brain injury in boxing. JAMA 1997;278:136–40.
29. Bailes J, Cantu R. Head injury in athletes. Neurosurgery 2001;48:26–45.
30. Gerberich S, Priest J, Boen J, et al. Concussion incidences and severity in secondary school varsity football players. Am J Public Health 1983;73:1370–5.
31. Goldberg D. Concussions, professional sports, and conflicts of interest: why the national football league's current policies are bad for its (players') health. HEC Forum 2008;20:337–55.
32. Osborne B. Principles of liability for athletic trainers: managing sport-related concussion. J Athl Train 2001;36:316–21.