The Million Dollar Question: When Should an Athlete Retire after Concussion?

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Abstract
Management of acute concussions is guided by consensus statement, and the return-to-play process begins when an athlete’s symptoms and examination return to baseline. This process may be relatively clear if symptoms resolve within the normal time frame following a first or second concussion. This decision-making process is more complicated in an athlete with prolonged unresolved symptoms, multiple concussions both with and without prolonged recovery, or a structural brain injury. In these situations, determining when to retire an athlete after concussion is a complex decision, without available evidence-based guidelines. This article will discuss absolute and relative contraindications to returning an athlete to contact sport following a concussion in three separate scenarios: following potentially life-threatening brain injury, persistent clinical symptoms or signs of prolonged postconcussion syndrome, and multiple concussions but without residual symptoms or signs.

Introduction
With an estimated 3.8 million sports-related concussion per year in the United States (20), proper treatment of an acute concussion is essential. Coaches, officials, parents, and athletes are receiving greater training in recognizing a concussion than ever before. The management of acute concussion has been addressed in a recent consensus statement (35); however there are no similar consensus statements or clear evidence-based guidelines that dictate when an athlete should be retired from contact sports following concussion. Given the complexities of the decision-making process when considering retirement, each athlete’s situation needs to be considered individually. Ideally the treatment team should consist of a multidisciplinary team with experience in both brain injury and sports medicine.

Variables to be considered include age, level of play, comorbid conditions, and concussion history (including force of the impact as well as number, severity, and duration of prior concussions). Recommendations are generally more conservative for youth athletes than for adults.

As defined by the most recent International Consensus Statement (35), a concussion is a complex pathophysiological process with generally short-lived neurological impairment that resolves spontaneously. It may be caused by a direct blow to the head or elsewhere on the body with force transmission to the head. Concussion reflects a largely functional disturbance rather than a structural injury, although some advanced imaging techniques have demonstrated findings that await further elucidation. Most injuries resolve in 7 to 10 d, although in approximately 15% of cases, recovery may be prolonged. Athletes suspected of having experienced a concussion must be removed from play immediately. There is no same-day return to play for any athlete that has experienced a concussion, regardless of skill level or age. Return to play is a medically supervised, stepwise process that begins only after an athlete’s symptoms and neurological and cognitive examinations have returned to baseline. Return to practice or competition is contraindicated in athletes with any of the following: abnormal neurological examination, unresolved postconcussion signs or symptoms present at rest or emerging with exertion, abnormalities on neuropsychological testing, or structural abnormality on standard computed tomography (CT) or magnetic resonance imaging (MRI) imaging (5).

Return to play is appropriate for athletes who recover within the anticipated time frame. When the recovery is prolonged or the athlete has experienced multiple concussions, it is not a question of when he or she will return to play but if return to play is wise. The brain is not yet fully developed in young and adolescent athletes, causing many to recommend more conservative management in these cases. Without clear evidence, the decision-making process is also complex at the professional level, where early retirement has significant financial ramifications for the athlete (11).
This article will discuss potential retirement considerations for athletes in three separate scenarios:

1. Following potentially life-threatening brain injury
2. Persistent clinical symptoms or signs of prolonged postconcussion syndrome
3. Multiple concussions but without residual symptoms or signs

Severe or Life-Threatening Brain Injury

Any type of structural injury to the skull or brain indicates that the athlete has experienced a more severe brain injury than a concussion. The care team in these scenarios must include disciplines with experience in managing moderate and severe brain injury, including neurosurgery, physiatry, neurology, neuropsychology, physical and occupational therapy, and speech pathology. We can expect that following these more severe brain injuries, there are more persistent sequelae.

Intracranial bleeding sometimes can complicate head injuries. An epidural hematoma (EDH) results from skull fracture with damage to the meningeal artery, without significant direct parenchymal injury. In contrast, an acute subdural hematoma (SDH) results from bleeding of the cortical vessels or bridging veins and is often associated with underlying parenchymal injury. It has been recommended that athletes with traumatically induced subarachnoid hemorrhage should not return to collision or contact sports (6,38). In the absence of underlying coagulopathy or brain lesion such as aneurysm or arteriovenous malformation, there is no thought of an increased risk of repeat hemorrhage or concussion following either an EDH or SDH (10). Return to play can only be considered once the athlete has returned to baseline on symptom score, neurological exam, and neuropsychological testing and has been cleared by a neurosurgeon (29).

Following an EDH, the athlete may return to play once the hematoma is fully resolved and the fracture fully healed (10). Following an SDH, decisions on return to play depend on whether there is underlying parenchymal injury and whether the athlete is treated with either a burr hole or craniotomy. If there is parenchymal injury, the athlete should likely not return to contact or collision sports (10,34,38). If there is no underlying parenchymal injury and no surgery was required, the athlete may return to play once the hematoma has resolved, the brain has reexpanded to fill the space, and there is no residual subdural hygroma (10). This will generally take several months to a year.

A burr hole will heal more quickly than a craniotomy, with much less concern over the capacity of the skull to withstand a future impact. In cases that required craniotomy, in addition to those mentioned, the bone flap must be fully healed before return. Bony union on CT generally takes 12 months (10,38). There is a theoretical risk that after surgery for an intracranial bleed, the cerebrospinal fluid pathways are altered, which may decrease buoyancy of the brain and increase susceptibility to future concussion in these patients (38).

Return to collision or contact sport following craniotomy is assessed on a case by case basis, taking into account the demands of the sport and the ability to protect the craniotomy site with protective gear (10). Decisions should be made with input from a multidisciplinary team including a neurosurgeon, neuroradiologist, neuropsychologist, and sports medicine physician (29).

Second impact syndrome (SIS) is a rare but life-threatening event that has been described as occurring when an athlete sustains a second head injury while still experiencing symptoms from a previous head injury. The second blow is often relatively minor, but usually within minutes the athlete collapses in a semicomatose state. SIS is thought to result from altered cerebral autoregulation with resultant vascular congestion and massive brain swelling. This leads to increased intracranial pressure and brainstem herniation, resulting in death or severe morbidity (5). In the unlikely event that an athlete recovers fully following SIS, a history of SIS is an absolute contraindication for return to contact or collision sports (8).

Persistent Symptoms Following Concussion

The vast majority of athletes recover from a concussion within 7 to 10 d; however approximately 10% to 15% may have symptoms that extend beyond the expected recovery period (23). Of these with prolonged symptoms, a small proportion will develop postconcussion syndrome (PCS), defined by symptoms of greater than 3 months in duration. This persistence of symptoms has been ascribed to biological effects of the injury, psychological factors, social factors, or a combination (24,25).

As the symptoms reported with PCS can be rather nonfocal and nonspecific, and may overlap with depression, it can be difficult to differentiate between ongoing concussion symptoms and a mood disorder. This overlap in presentation complicates the decision-making process. The athlete with true persistent symptoms postconcussion should be retired from contact sports. On the other hand, an athlete with a mood disorder or other psychological issues may be able to return to sport with proper treatment.

Anxiety and depression can be acute symptoms associated with concussion, but they also may have other etiologies. Mood disorders and other psychological conditions can predate the concussion, be associated with the concussion, or occur after the concussion has resolved. One example revolves around the happiness and pleasure many athletes derive from sports. Removing athletes from their activities certainly places them at risk for mood disorders. In addition, sports may be a primary source of social interaction for athletes, and prolonged removal from these situations also may place them at increased risk for mood disorder. As such, an athlete may experience secondary mood disorders that are not directly due to ongoing brain injury. In a professional athlete faced with loss of livelihood due to mild traumatic brain injury (mTBI), depression or anxiety may be at the forefront of their symptom profile.

Furthermore, in the work of Ponsford et al. (39), comparing trauma patients with and without mTBI, he found that individuals in either group who reported psychological factors such as anxiety, posttraumatic stress disorder (PTSD), or other life stressors had a higher likelihood of reporting unresolved symptoms at 3 months. In either group, higher anxiety levels at 1 wk also were associated with higher likelihood of PCS at 3 months (39). McCauley et al. (32) found
that postconcussion symptoms 3 months postinjury were associated with concomitant depression or PTSD, and even in individuals with resolution of cognitive symptoms post-mTBI, depressive symptoms persisted (32). In addition, as demonstrated by Whittaker et al. (42), PCS may be influenced by negative illness perception, meaning that the expectation of prolonged symptoms and poor recovery may be a self-fulfilling prophecy.

Headaches are the most common postconcussion symptom (19), and in athletes with persisting symptoms, headaches are the most common ongoing symptom (33). In addition, a concussion may exacerbate a premorbid headache disorder. One pitfall in the management of concussion is to assign etiology of all posttraumatic symptoms to unresolved concussion, while the symptoms instead may represent a concomitant headache disorder. Headaches can cause difficulty with cognitive activities, and this can cause confusion as to whether additional symptoms, such as slow reaction time or poor verbal memory, are truly related to ongoing pathology related to the mTBI versus consequences of the headache itself.

According to the International Classification of Headache Disorders, headaches occurring after a concussion are classified as secondary headaches and are further classified as chronic if they last longer than 3 months posttrauma. These can be migrainous in nature, which may respond to abortive or prophylactic medication. Less commonly, they also can be cervicogenic, related to a whiplash-type injury sustained at the time of impact (31). Typically these headaches are associated with neck pain and may respond to physical therapy and mobilization. Tension-type headaches and medication rebound headaches also may be seen following concussion.

In general, prior to return to play, athletes should have resolution of posttraumatic headaches without medications. In those athletes without headache resolution, it is important to determine whether they have recovered from the concussion and have developed instead an independent headache syndrome. This can be very difficult to determine and should be evaluated by a multidisciplinary team, including a headache specialist well versed in the management of concussion. In this case, return to play may be possible under the supervision of this headache specialist and the multidisciplinary team.

Multiple Concussions

Perhaps the most challenging scenario regarding retirement decisions is that of the athlete who has experienced numerous concussions but does not appear to have experienced any long-lasting sequelae. In this instance, it is especially important to understand the potential long-term risks associated with multiple concussions and whether repeated concussions result in permanent neurological injury.

Cantu et al. (5,8) assert that there is no specific number of concussions after which one must definitively retire. Previously published guidelines (6,7) recommended termination of a season after three concussions, with at least a 3-month symptom-free period before return to contact sports but without mention of permanent retirement. In adolescent and young athletes it is prudent to be more conservative and consider terminating a season after two concussions, even with apparent rapid resolution of symptoms following each concussion. In addition, athletes who experience increasing duration or severity of symptoms with subsequent concussions or who require less of an impact to sustain a concussion may warrant retirement (8).

The current literature regarding long-term cognitive effects following multiple concussions is not definitive, with several studies showing no negative effect (3,4,9,14,26), while others show possible small long-term deficits in memory or executive functioning (2,27). Athletes with a history of prior concussion may be at increased risk for future concussions (18). Following a concussion, they may report increased immediate symptoms (28), with increased symptom duration of greater than 1 wk (18). In a retrospective study of retired professional American football players, those athletes with self-reported recall of multiple concussions had a higher rate of self-reported cognitive difficulty (16) and a higher rate of depression diagnoses (17).

Retired National Football League (NFL) players appeared to have earlier onset of Alzheimer’s dementia than the general population, but no correlation was found with the number of previous concussions (16). In a separate retrospective study, NFL players were found to have lower all-cause mortality than the general population but higher mortality from neurodegenerative causes (30). There was no mention of the athletes’ concussion history in this study. These two studies contrast with a retrospective study of high school football players that did not find evidence for increased risk of dementia, Parkinson’s disease, or amyotrophic lateral sclerosis (40). In addition to conflicting results, it is not clear how generalizable these findings are to athletes in other sports and to nonprofessional athletes.

In recent years, there has been growing concern that numerous concussions or subconcussive impacts may lead to chronic traumatic encephalopathy (CTE). CTE is a distinct tauopathy that can be diagnosed only at autopsy (37). It is a rare progressive neurological disorder that results in cognitive, mood and behavioral, and neurological symptoms (1,13). Cognitive changes include deficits in memory and executive functioning with eventual dementia. Mood changes include apathy, depression, and even suicidality. Behavioral changes include impulsiveness, substance abuse, and violence. Neurological symptoms can manifest as parkinsonian symptoms or dysarthric speech.

CTE is associated with a history of prior brain trauma, but as all current literature is retrospective, causality has not been proven (36). It has been identified in contact or collision sport athletes as well as victims of physical abuse and members of the military (37). While an exposure to concussive or subconcussive impacts seems to be required, it is not known yet what other risk factors may lead to the development of CTE. These may include the total number of concussions or subconcussive impacts; substance use or abuse; medical comorbidities, especially those linked to chronic inflammation; psychiatric comorbidities; and genetic risk factors (13). The true incidence and prevalence of CTE also are unknown.

Cases of cognitive and neurological decline in former athletes do not always represent development of CTE and instead may represent Parkinson’s disease, Alzheimer’s dementia, vascular dementia, or other causes (22). In studies
of Alzheimer’s dementia, clinical presentation does not always match the neuropathology seen at autopsy, and indeed, some cases with pathologic changes at autopsy were cognitively intact clinically (12). The same variable relationship between clinical signs and underlying histopathology at autopsy is seen in the largest case study of CTE to date (37). Given this variability, it is challenging to correlate clinical features with pathology in modern cases of CTE.

While a discussion of CTE may be reasonable, particularly in adult professional collision sport athletes with a history of multiple concussions, the clinician must acknowledge that there is currently more that is unknown than known about this condition. At this time, it is difficult to make a retirement recommendation based solely on the possibility of developing CTE in the future.

The decision to retire an athlete after multiple concussions is complex and should be undertaken after careful consideration, with the input of a multidisciplinary team whenever possible. Members of the team should have experience in sports medicine and brain injury. The issue of informed consent, or signing a waiver, is likely not sufficient from a legal standpoint, as many players (especially at the professional level) will sign any document if it allows them to continue to play. Instead, for those athletes that are allowed to continue their sport of choice, a frank discussion of the risks, both known and unknown, must occur with the athlete and his or her family, and this should be well documented. Decisions must be made based on the best available evidence.

**Areas for Further Study**

Standard neuroimaging is generally normal following a concussion. However more advanced imaging methods such as functional magnetic resonance imaging, magnetic resonance spectroscopy, and diffusion tensor imaging continue to be studied for their potential use in identifying acute or ongoing changes following a concussion (21). Future research may help these imaging modalities move from a strictly research setting into clinical patient care, and this eventually may help to identify athletes with permanent neurological sequelae that cannot currently be assessed on standard imaging. While the existence of CTE is certainly concerning, further research into the risk factors for its development, as well as its true incidence and prevalence, is needed to help predict who may develop CTE and how we can help to prevent or treat it in the future.

Genetic risk factors, such as the APOE E4 gene, eventually may be used to determine an athlete’s risk, but studies are not yet conclusive (38). Genetic testing to prevent participation also carries several ethical and legal ramifications.

**Conclusion**

While it is likely better to be conservative than cavalier in managing an athlete after a concussion, premature retirement based on poorly supported decisions can have several downstream consequences. In the immediate setting, retirement may lead to depression or other mood symptoms from a loss of identity and peer group interactions. In this era of an obesity epidemic, it is also clear that participation in sports is beneficial for long-term health, as well as for character development and social bonds. Financial consequences exist not only for the professional athlete but also for the college or high school athlete training for an athletic scholarship. The sports medicine physician must therefore take great care when determining that an athlete should no longer participate in contact sports following concussion.

Absolute contraindications for return to play to contact or collision sports, based on expert opinion and clinical judgment, are provided in the Table. Unresolved deficits on functional MRI or other advanced imaging may become a contraindication for return to sport, but currently, such advanced imaging is used only in the research setting (15).

Relative contraindications for return to play include prolonged (greater than three months) PCS (5), increasing symptom duration or severity with subsequent concussions, especially if the athlete has experienced three or more concussions (7), and increasing susceptibility to concussion with decreasing impacts (8).

Situations that require evaluation on a case by case basis and involvement of a multidisciplinary team include the following: craniotomy, athletes with multiple concussions without evidence of structural abnormality, and athletes with persistent headache or mood disorder but without other complaints or abnormalities on testing. Recommendations may be more conservative in the young or adolescent athlete (5).

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**References**


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* Edema, hemorrhage, hydrocephalus, and arachnoid cyst.

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