CHRONIC TRAUMATIC ENCEPHALOPATHY IN THE NATIONAL FOOTBALL LEAGUE

When Andre Waters, a hard hitting National Football League (NFL) player known to have died as a result of chronic traumatic encephalopathy (CTE) attributed to the multiple concussions he experienced while playing in the NFL, made the front page of the New York Times on Thursday, January 18, 2007, he became the third NFL player to experience significant memory problems and five times more likely to develop earlier onset of Alzheimer’s disease (3). A study published this year by the same authors found a similar relationship between three or more concussions and clinical depression (4).

The NFL’s own publications in this journal on concussion state that they had seen no cases of CTE in the NFL (8–10). That finding is not a surprise as the NFL study included only active players in their 20s and 30s during a short 6-year window from 1996 to 2001.

Other significant limitations of the NFL studies include the following:

1) History of concussion: previous concussions either in professional football or during their playing careers in high school, college, or other levels of football were not included.

2) The population of NFL players changes from year to year: new players enter the league, older players leave the league, and we do not know the number of players who constituted the 1996 population who are still in the league in subsequent years.

3) There was difficulty collecting data on loss of consciousness; the initial data collection sheet did not ask for data regarding loss of consciousness.

4) This was a multisite study with numerous different examiners; there was no uniform method of evaluation of concussion in this study.

5) Return to play data were collected on players with initial and repeat concussion: there are many other factors that go into the decision of whether or not the player should return to play, including the importance of the player to the team; the importance of the upcoming game to the team; and pressure from owners, players, and their families, coaches, agents, and media may certainly influence the final decision on when the player returns to play.

ARE THE FINDINGS OF CTE IN THE BRAINS OF WEBSTER, LONG, AND WATERS A SURPRISE?

Certainly the Waters finding was no surprise to Julian Bailes, M.D., medical director, and Kevin Guskiewicz, Ph.D., the Director of the Study of Retired Athletes at the University of North Carolina at Chapel Hill. Their study of retired NFL players published in this journal found that those who had sustained three or more concussions were three times more likely to experience significant memory problems and five times more likely to develop earlier onset of Alzheimer’s disease (3). A study published this year by the same authors found a similar relationship between three or more concussions and clinical depression (4).
6) The results apply to mainly NFL-level players: extrapolation to younger players has not been demonstrated.

Should we be surprised that CTE has been reported in former NFL football players? I would echo Dr. Bailes and say “absolutely not,” but for additional reasons. Before I enumerate, let us first go back to the definition of CTE.

CTE, or dementia pugilistica, was first described by Harrison S. Martland in his landmark Journal of the American Medical Association article published in 1928 (5) as being characteristic of boxers “who take considerable head punishment seeking only to land a knock out blow.” It was also “common in second rate fighters used for training purposes.” The early symptoms he described were a “slight mental confusion, a general slowing in muscular movement, hesitancy in speech, and tremors of the hands.” Later, marked truncal ataxia, Parkinsonian syndrome, and marked mental deterioration may set in, necessitating commitment to an asylum” (5, p 1103).

Although Martland first described the clinical syndrome of CTE and Roberts (11) echoed the dangers of chronic brain damage in boxers in 1969, it was Corsellis et al. who first identified the neuropathology of this syndrome in the brains of 15 deceased boxers, eight of whom were either world or national champions (1).

Table I summarizes his findings of the four main components of this entity, areas of the brain damaged, and resultant signs and symptoms. It is critical to understand that although Corsellis pointed out four different areas of the brain and the resultant signs and symptoms, he did not state that all four areas needed to be involved for the diagnosis to be made. In fact, only eight out of 15 brains studied had all four areas of pathology present (2).

It was Corsellis who also reported CTE not only in boxers but other sports with a high risk of head injury, including those in which head injury occurred in declining frequency; among these were jockeys (especially steeplechasers), professional wrestlers, parachutists, and even a case of battered wife syndrome. With this history, it is no surprise to have cases from NFL football.

<table>
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<tr>
<th>Area damaged</th>
<th>Clinical symptoms/signs</th>
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<tr>
<td>Septum pellucidum, adjacent periventricular grey matter, frontal and temporal lobes</td>
<td>Altered affect (euphoria, emotional ability and memory)</td>
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<tr>
<td>Degeneration of the substantia nigra</td>
<td>Parkinson’s syndrome of tremor, rigidity, and brachykinnesia</td>
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<tr>
<td>Cerebellar scarring and nerve cell loss</td>
<td>Slurred speech, loss of balance and coordination</td>
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<tr>
<td>Diffuse neuronal loss</td>
<td>Loss of intellect, Alzheimer’s syndrome</td>
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SO WHAT ARE THE QUESTIONS TO BE ANSWERED?

The most pressing question to be answered concerns the prevalence of the problem. The Waters case came to light only because of Chris Nowinski, a former All-Ivy defensive tackle at Harvard and World Wrestling Entertainment professional wrestler who, after being forced to retire because of repeated concussions and postconcussion syndrome, researched and wrote a book on the subject of athletic concussions. Hearing of Waters’ suicide and suspecting CTE, Nowinski convinced the Waters family to send a portion of Waters’ brain to Dr. Omalu for neuropathological examination.

Since the Ted Johnson publicity in which this former New England Patriots star middle linebacker said, “I don’t want anyone to end up like me,” I have personally examined and spoken with a number of retired NFL players with postconcussion/CTE symptoms. Only an immediate prospective study will determine the true incidence of this problem. Although this study could be funded by the NFL charities, the NFL should refrain from introducing potential bias with regard to the team of neurosurgeons, neurologists, neuropsychiatrists, and neuropathologists with athletic head injury expertise chosen to carry out the study.

I also commend the fact that the brains of Webster, Long, and Waters have now been examined by other neuropathologists who concur with Omalu’s findings. Obtaining second opinions on such a high profile issue is just common sense.

Finally, it is clear that not all players with long concussion histories have met premature and horrific ends to their lives. However, as the list of NFL players retired as a result of postconcussion symptoms (e.g., Harry Carson, Al Toon, Merrill Hodge, Troy Aikman, Steve Young, Ted Johnson, Wayne Chrebet) grows and as the number of documented CTE cases increases, I believe the time for independent study of the problem as well as NFL recognition that there is a problem is now. Recognizing that soldiers were having problems with blast-related closed head injury, I have been a part of a team convened by the Department of Defense to write management algorithms and protocols in the past year. I believe the NFL would be prudent to assemble such an independent impeccably qualified “dream team” to tackle their problem with concussion and resultant CTE head on. Just as the National Association for Stock Car Auto Racing recently faced a problem, solved it, and became even more popular, I believe the NFL will lift this dark cloud if they confront the problem directly and honestly.

REFERENCES


