Paying the Piper: NFL’s concussion policy results in huge class action lawsuit

By Joseph M. Hanna

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The plight of NFL players suffering from concussion-related injuries has long been the subject of media coverage, scientific debate and fan interest. Still, recent events — such as the suicides of several NFL alumni, groundbreaking clinical studies, and a giant class-action lawsuit which threatens the livelihood of the league itself — have once again brought the topic to the forefront of national attention. Still, the question remains: will the NFL face liability for its arguably deficient efforts to inform players of the risks associated with football induced head trauma?

GAME-CHANGING SCIENCE

In 2005 clinical studies performed by independent scientists determined that multiple NFL concussions can cause problems such as depression and early-onset dementia. First, Dr. Bennett Omalu and Dr. Robert Cantu examined the brain tissue of three deceased NFL players (Mike Webster, Terry Long, and Andre Waters) that had suffered multiple concussions throughout their NFL careers. Prior to their premature deaths, all three had presented symptoms of sharply deteriorated cognitive function and psychiatric symptoms such as paranoia, panic attacks, and depression. After the brain tissue of all three revealed neurofibrillary tangles, neurotrophil threads, and cell dropout, Omalu concluded that Chronic Traumatic Encephalopathy (“CTE”) triggered by multiple NFL concussions represented a partial cause of their deaths.

CTE is a neurological disorder first discovered in athletes (such as boxers) who sustained multiple blows to the head. Initially, CTE presents through symptoms such as poor concentration/memory, dizziness, and
headaches. Later, CTE may progress into dementia or Parkinsonism, causing symptoms such as a general slowing in muscle movement, hesitancy in speech, and tremors of the hands.

In response, the NFL Concussion Committee (“NFL Committee”) denied a link between concussions and cognitive decline, claimed that more research was needed to reach a definitive conclusion, and later stated: “[w]e own this field. We are not going to bow to some no-name Nigerian with some bullshit theory.” Noting that (ironically) no committee members were neuropathologists, Omalu questioned the integrity of the NFL Committee, quipping “[h]ow can doctors who are not neuropathologists interpret neuropathological findings better than neuropathologists?”

Although additional studies (again in 2005 and later in 2008) reached similar conclusions, the NFL continued to ignore these findings. Instead, during its first Concussion Summit in June 2007, the league issued a warning pamphlet to players which simply stated that “there is no magic number for how many concussions is too many.” Dr. Ira Casson, the former NFL Committee’s Co-Chair, publicly discounted current research on the subject as unreliable and inconclusive.

CONGRESSIONAL HEARING

The concussion debate reached a boiling point in September 2009, when an NFL-commissioned University of Michigan study found that NFL alumni are diagnosed with Alzheimer’s disease (or similar) vastly more often than the national population. Shortly thereafter Congress announced that it would hold a hearing to discuss “legal issues relating to football head injuries.”

In October 2009, members of the House Judiciary Committee criticized the NFL’s concussion policy. Though NFL Commissioner Roger Goodell declined to comment on issues requiring medical expertise, the committee played an HBO Real Sports recording of Casson, the NFL’s medical voice on the issue, who denied all potential links between multiple head injuries and later-life cognitive decline. Notably, Representative Linda Sanchez of California analogized the NFL’s denial of a causal link between NFL concussions and cognitive decline to the tobacco industry’s denial of the link between cigarette consumption and ill health effects. Sanchez encouraged Goodell to get “ahead on this issue, if only to cover [the NFL] legally,” suggesting that the NFL might avoid liability if it simply issued adequate warning to NFL players.

REMEDIAL MEASURES

The NFL took several remedial measures after the 2009 hearing. First, Casson and fellow co-chair Dr. David Viano both resigned from their NFL Committee positions. To replace the old NFL committee, the NFL selected prominent neurologists Dr. H. Hunt Batjer and Dr. Richard G. Ellenbogen as co-chairs of the newly minted NFL Head, Neck and Spine Medical Committee (“HNSMC”).

Second, the NFL partnered with the Center for the Study of Traumatic Encephalopathy (“CSTE”) by pledging to donate $1 million to support its research. Third, NFL spokesperson Greg Aiello admitted: “[i]t’s quite obvious from the
medical research that’s been done that concussions . . . lead to long-term problems[].”

Fourth, each team was required to make an independent doctor available to examine players and determine whether a player should return to play after sustaining a concussion.

By 2010, the NFL had finally conceded that head injuries can cause severe cognitive health problems. Though this admission was overdue, the NFL deserves credit for finally embracing the findings of independent scientists. The league later took other proactive measures as well. In February 2011, the HNSMC announced that team medical personnel would implement a standardized sideline concussion assessment protocol, consisting of a limited neurological/cognitive examination and a balance assessment. Following a December 2011 incident, when Cleveland Browns quarterback Colt McCoy suffered a concussion after an illegal hit but was returned to the game after two plays, the NFL issued a memo stating that third-party athletic trainers would be placed in each stadium to help monitor player concussions. Later, in July 2012, Commissioner Goodell hinted that the league was in the process of developing a test for a tablet (or iPad) which, when used on the sideline, could help determine whether or not a player had suffered a concussion.

Still, most of these measures were taken after the class-action suits against the NFL had been filed, and not everyone is truly convinced of the league’s newfound concern for its players. Terry Bradshaw noted, “[t]hey’re forced to care now because it’s politically correct to care. Lawsuits make you care. I think the PR makes you care.”

TRAGIC NEWS

On Dec. 17, 2009, Cincinnati Bengals receiver Chris Henry, 26, died after falling (or jumping) out of the back of a pickup truck. When Omalu and Dr. Julian Bailes performed a postmortem study on Henry’s brain tissue, they discovered trademark signs of CTE. Notably, these signs were not caused by the accident, as signs of CTE develop slowly over time. Further, Henry, the twenty-second professional football player to be diagnosed with CTE, died while still active in the NFL, having developed CTE by his mid-20s.

Sadly, Henry’s death was no isolated incident. In February 2011 former Chicago Bears defenseman Dave Duerson shot himself fatally in the chest after experiencing deteriorating cognitive symptoms that he believed were linked to CTE. Before his death, Duerson left specific instructions to his family: “Please, see that my brain is given to the N.F.L.’s brain bank,” presumably to confirm his self-diagnosed suspicions. In May 2011, the CSTE confirmed that Duerson had “indisputable” evidence of CTE in his brain tissue samples, also noting that there was “no evidence” of any other mental disorder.

In April of 2012, former Atlanta Falcon Ray Easterling, also committed suicide, dying of a self-inflicted gunshot wound at his home in Virginia. Prior to his death, Easterling had experienced a variety of classic CTE symptoms, suffering from memory loss, hand tremors, and eventually dementia. Notably, Easterling had been the lead plaintiff in the first class-action lawsuit filed against the NFL, previously alleging that the league ignored and concealed the dangers of concussions for years.
Just two weeks after Easterling’s death, Junior Seau, a 20-year veteran of the NFL, also committed suicide by a self-inflicted gunshot wound to the chest. Prior to his death, Seau had struggled with depression and other personal problems. His family has agreed to donate his brain to researchers to look for signs of trauma and CTE.

Disturbingly, it appears that the NFL concussion problem extends further than the current media coverage. For example, though the recent suicides and current litigation have brought the issue to the forefront of national attention, the progressive nature of the disease and the societal stigma toward mental illness has undoubtedly resulted in the under-reporting of concussion-related afflictions. One study of retired NFL players by the Center for Brain Health at the University of Texas revealed that these individuals suffered higher instances of cognitive defects and depression compared to the control subjects. While this somewhat mirrors Dr. Omalu’s findings, it is significant because many of the players were clinically depressed — i.e., exhibited symptoms such as difficulty sleeping, weight gain/loss, decreased energy levels — and had no idea. As concussion-associated depression has no mood component, affected players won’t necessarily experience the emotional volatility traditionally associated with the disorder. In effect, many CTE sufferers could be unaware that a problem exists until the disease has progressed into its intermediate/advanced stages.

SCIENTIFIC RESEARCH TAKES OFF

Fortunately, the recent media coverage has garnered significant attention for CTE throughout the scientific community. One study of over 100 active and retired NFL players strongly indicated that these athletes face a significantly higher risk of incurring permanent brain damage, including a susceptibility to dementia much higher than the national average. Another study conducted at the Albert Einstein College of Medicine has made progress in the area of diagnosing concussion-related traumatic brain injuries. Using a technology known as diffuse tensor imaging (DTI), researchers could detect unique abnormalities in the brains of those who had a concussion, where other methods of detection had failed to do so. This study also found that the micro-structural integrity of brain tissue found in those who had suffered concussions was abnormally low compared to the micro-structural integrity of the brain tissue in control groups (concussion-free participants).

Unfortunately, diagnostic research does little to propose a solution or rectify past harms. In the coming years, the issue(s) will be not whether concussions are linked to football-induced head trauma, but what can be done to reduce player susceptibility to CTE, and whether an adequate warning would have made a real difference.

LEGAL IMPLICATIONS/CLASS-ACTION LAWSUIT

Since at least 2005, the NFL has been on notice of medical studies linking football head injuries with later-life cognitive decline. Accordingly, over 2,400 former NFL alumni took legal action by filing several suits against the league, alleging in part that the league “deliberately and fraudulently concealed from its players the link between football-related head impacts and long-term neurological injuries.” The football helmet manufacture Riddell, Inc. was also named as a defendant, undoubtedly because of advertisements stating that Riddell helmets reduced the risk
These suits have since been collapsed into one “master complaint,” in the Eastern District of Pennsylvania. While the NFL has been ordered to file a motion to dismiss by August 9, 2012, new plaintiffs continue to file suit, and more will likely join the fray in the coming months.

POTENTIAL DEFENSES

Still, the NFL has several defenses that it may use to skirt liability here. First, the league may seek dismissal of the suit by arguing that it is preempted by the NFL players’ collective bargaining agreement and the NFL Constitution and Bylaws (the “CBAs”) under § 301 the Labor Management Relations Act. Arguably, if the league has breached its duty to minimize the risk of concussion-related harm to NFL players, and the CBAs outline the obligations of the NFL regarding the issuance of warnings and player safety – i.e., the resolution of state law claims requires interpretation of a collective bargaining agreement – this is a labor dispute. Therefore, because the CBAs stipulated to arbitration proceedings in the event of a dispute, the matter must go to arbitration. Previously, courts have supported this defense in similar suits against the NFL. Still, the success of this request for arbitration will depend on whether the court accepts that premise that the plaintiffs’ claims “arise under” the CBAs.

Second, the NFL might argue that, absent some special relationship, mere awareness of independent studies did not by itself impose a legal duty to warn players about the cognitive consequences of concussions. Courts have suggested that NFL players are employees of their respective teams, not the league. Consequently, the NFL might argue that there is no special relationship stemming from employment that would trigger an affirmative duty to warn NFL players about the long-term risks associated with concussions.

Third, the NFL could raise the defense of contributory negligence – i.e. there can be no recovery for negligence if the injured person, by his own negligence, proximately contributed to the injury – on behalf of the players, asserting that these individuals contributed to their own injury by: (i) failing to report their concussive conditions; and (ii) returning to play before their concussion symptoms completely disappeared. The NFL instructs players to self-report their concussion symptoms, indicating that concussion symptoms should be immediately reported to team medical personnel, and that players should be asymptomatic before returning to play.

Thirty of 160 NFL players surveyed by the AP in November of 2009 replied that they either failed to report or underreported concussion symptoms, and admitting returning to play despite them. The NFL would argue that players negligently contributed to their own cognitive injuries by failing to report these concussion symptoms and returning to play before becoming symptom free. Players could respond by arguing that the NFL’s contractual scheme incentivizes them to conceal their concussion symptoms. NFL player contracts do not guarantee player payment beyond the season in which an injury occurs. This structure maximizes the risk of players incurring permanent cognitive problems because it incentivizes players to withhold their concussion symptoms and play through head injuries.
These are only three of the NFL’s possible defenses: the league could pursue other tactics such as apportioning the blame via comparative negligence, or arguing that the statute of limitations has run on individual plaintiffs’ claims. Still, given the sheer scope of this litigation, it seems likely that the NFL and Riddell might be inclined to settle the case to avoid potentially catastrophic, Big Tobacco-like liabilities.

Not everyone is convinced that comparisons to the Big Tobacco litigation are appropriate, however. For one thing, unlike tobacco use, the effect of individual concussions on a football player remains unclear. Further, the NFL retains medical personnel who are employed specifically to detect and prevent player injuries, whereas smoker plaintiffs were given no such attention. Lastly, because NFL players could have sustained permanent mental injuries at any point in their career (high school, college, etc.), proving the causal chain — i.e., that the NFL’s failure to warn resulted in injury — is difficult at best.

CONCLUSION

Studies performed by the nation’s scientists confirm a causal link between multiple NFL concussions and later-life cognitive decline. Further, the NFL Committee has been aware of these causal studies since at least 2005, and failed to issue adequate warning to league players. Additionally, current efforts to combat CTE cannot rectify the past harms suffered by many severely injured players. As a result, NFL alumni have targeted the league with failure to warn claims to recover for their cognitive injuries. Still, the NFL has a number of potentially exonerating defenses at its disposal. In any event, the next few months will determine the NFL’s ultimate liability for its actions and the financial future of the league.

Notes


3 See Cantu, supra note 2, at 223.

4 See id.

5 See id. These are several trademark symptoms of CTE.

6 See Cantu, supra note 2, at 223-24.
A 2005 clinical study conducted by Dr. Kevin Guskiewicz surveyed over 2,550 former NFL athletes and found that retired NFL players who had sustained three or more concussions in the league had a fivefold prevalence of Mild Cognitive Impairment (“MCI”) diagnosis compared to NFL alumni with no history of concussions. See Kevin M. Guskiewicz et al., Association between Recurrent Concussion and Late-Life Cognitive Impairment in Retired Professional Football Players, 57 NEUROSURGERY 722 (2005).

Dr. Ann McKee of Boston University studied the brain tissue of deceased NFL alumni John Grimsely and Tom McHale, finding that both exhibited distinct signs of CTE. See McKee et al., Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy After Repetitive Head Injury, 68 J Neuropathology Exp Neurol. 709, 732 (2009). McKee believed that decreasing the number of concussion would decrease the incidence of athlete CTE, stating “[t]here is overwhelming evidence that [CTE]is the result or repeated sublethal brain trauma.” See id.

Dr. Ira R. Casson) (“there is not enough valid, reliable or objective scientific evidence at present to determine whether . . . repeat head impacts in professional football result in long-term brain damage.”).

This rate was found to be 19x the normal rate of affliction for men ages 30-49.

See, e.g., Alan Schwarz, Concussion Expert’s Removal is Sought, N.Y. TIMES (Nov. 20, 2009), available at http://query.nytimes.com/gst/fullpage.html?res=9D04E6D81E3FF933A15752C1A96F9C8B63 (explaining that the NFLPA called for the removal of Dr. Casson as co-chair of the NFL Committee due to his efforts to discredit independent and league-sponsored evidence linking N.F.L. careers with heightened risk for dementia and cognitive decline); see also Alan Schwarz, N.F.L. Head Injury Study Leaders Quit, N.Y. TIMES (Nov. 25, 2009), at B11.

See Alan Schwarz, NFL Picks New Chairmen for Panel on Concussions, N.Y. TIMES (Mar. 17, 2010), at B1 (Batjer was the chairman of neurological surgery at Northwestern Memorial Hospital).

Seeid. (Ellenbogen was the chief of neurological surgery at Harborview Medical Center).

See Alan Schwarz, N.F.L. Picks New Chairmen for Panel on Concussions, N.Y. TIMES (Mar. 17, 2010), at B11.

See, e.g., Alan Schwarz, N.F.L. Acknowledges Long-Term Concussion Effects, N.Y. TIMES (Dec. 21, 2009), at D1 [hereinafter N.F.L. Acknowledges Long-Term Effects] (noting that league spokesman Greg Aiello communicated that the NFL could donate $1 million or more to CSTE); see also, Alan Schwarz, N.F.L. Gives $1 Million to Brain Researchers, N.Y. TIMES (Apr. 21, 2009), at B14 (confirming the league’s official donation of $1 million to further CSTE’s research efforts). As of the fall of 2011, the CSTE has begun recruiting participants for the DETECT (Diagnosing and Evaluating Traumatic Encephalopathy Using Clinical Tests) study, which will include 150 former NFL players, ages 40 to 69, and 50 same-age athlete control participants, to develop methods for diagnosing CTE during life through a variety of medical procedures. See Clinical Studies, BOSTON UNIVERSITY CENTER FOR THE STUDY OF TRAUMATIC ENCEPHALOPATHY, http://www.bu.edu/cste/our-research/clinical-studies/ (last visited June 20, 2012).


See Alan Schwarz, N.F.L. Asserts Greater Risks of Head Injury, N.Y. TIMES (July 27, 2010), at A1; see also Press Release, National Football League, Concussion: A Must Read for NFL Players (July. 26, 2010) [emphases added]. (“‘[T]raumatic brain injury can cause a wide range of short- or long-term changes affecting thinking, sensation, language, or emotions.’ These changes may lead to problems with memory and communication, personality changes, as well as depression and the early onset of dementia. Concussions and conditions resulting from repeated brain injury can change your life and your family’s life forever.”).


See Alan Schwarz, Former Bengal Henry Found to Have Had Brain Damage, N.Y. TIMES (June 29, 2010), at B10.

Dr. Bailes is the chairman of the department of neurosurgery at West Virginia University. Id.

Henry’s brain sample demonstrated brown discolorations, a tau protein buildup, inflammation, and white matter changes. See Madison Park, Young player had brain damage more often seen in NFL veterans, CNN.COM, http://www.cnn.com/2010/HEALTH/07/02/brain.damage.henry/index.html. In healthy brain tissue, virtually no protein tangles, which show up as brown spots, are visible. Id.

Like many of the other players found to have had CTE after their deaths, Henry had behavioral problems in his final years that might have been at least partly a result of the disease, which is linked to depression, poor decision-making and substance abuse. Id. He was arrested five times in a 28-month stretch for incidents involving assault, driving under the influence of alcohol and marijuana possession. Id. The league suspended him several times for violating its personal-conduct policy. Id.


See id.


See Mike Tearney, Former Player’s Suicide Won’t End his Widows Fight, N.Y. TIMES (May 4, 2012), at B9, available at http://www.nytimes.com/2012/05/04/sports/ray-easterlings-widow-to-keep-fighting-for-retired-nfl-players-with-head-injuries.html?pagewanted=all. At times, Easterling would go for a job and become disoriented, prompting his wife to initiate one-woman search parties in the early hours of the morning. See id.


See Pilon, supra note 45.

See id.

See id.


Traditional methods of brain scanning (i.e., CT scans or MRIs) were not capable of detecting these distinctions. See id.

See Concussion Victims have Unique Spatial Patterns of Brain Abnormalities that Change over Time, NEWS MEDICAL (June 8, 2012), http://www.news-medical.net/news/20120608/Concussion-victims-have-unique-spatial-patterns-of-brain-abnormalities-that-change-over-time.aspx (discussing the results of the Albert Einstein School of Medicine Study). Worse still, the study revealed that these abnormal regions of brain tissue could retain this reduced level of structural integrity for up to an entire year following the concussive injury. See id.


See Farmer, supra note 55.


See id.


Only one such suit has targeted individual teams for liability so far; still, this is likely because worker’s compensation exclusive remedy laws bar employees from suing employers for work-related injuries. See Paul Anderson, Latest Concussion Lawsuit Targets Teams, NFL CONCUSSION LITIGATION (June 27, 2012), http://nflconcussionlitigation.com/.

62 See 488 AM. JUR. 2d Labor and Labor Relations § 2428 ("A final adjustment by a method agreed upon by the parties is the desirable method for settlement of grievance disputes over the application or interpretation of an existing collective-bargaining agreement. If a party sidesteps contractual-grievance machinery by suing in federal court without having attempted to invoke the grievance mechanism, the claim must be dismissed.").

63 See id. (noting that failure to exhaust contractual remedies may bar suit).


65 See, e.g., *Restatement (Second) of Torts § 314 (1965).*

66 See, e.g., *N. Am. Soccer League v. NFL*, 670 F.2d 1249, 1252 (2d. Cir. 1982).

67 Notably, if the league took the stance that it owed no duty to warn the players, it would have to rebut the argument the NFL’s voluntary creation of an internal Concussion Committee created a duty on the part of the league to exercise reasonable care. See Restatement (Third) of Torts § 42 (2005) ("Once an actor begins to render voluntary assistance to a third party, the actor undertakes a duty to proceed with reasonable care when such third party relies on the actor’s assistance.").

68 See 38 AM. JUR. 2d Negligence § 174.

69 See Press Release, National Football League, *supra* note 13 (The NFL’s August 14, 2007 informational player pamphlet contained these instructions)).

70 Id.


72 Id.

73 See NFL CBA (2006) app. c, § 9 at 251.


76 See id.

77 Id.