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Boxing Lessons: An Historical Review of Chronic Head Trauma in Boxing and Football

Jason P. Shurley and Janice S. Todd

In recent years there has been a significant increase in the scrutiny of head trauma in football. This attention is due largely to a host of studies that have been highly publicized and linked the repetitive head trauma in football to late-life neurological impairment. Scientists and physicians familiar with boxing have been aware of such impairment, resulting from repeated head impacts, for more than 80 years. Few, however, made the connection between the similarity of head impacts in boxing and football until recent decades. This article examines the medical and scientific literature related to head trauma in both boxing and football, paying particular attention to the different emphases of that research. Further, the literature is used to trace the understanding of sport-related chronic head trauma as well as how that understanding has prompted reform efforts in each sport. Finally, in light of the current understanding of the long-term sequelae of repetitive head trauma, some consideration is given to what football administrators can learn from the reform efforts in boxing.

Keywords: chronic traumatic encephalopathy, concussion, punch drunk, dementia pugilistica

On June 7, 2012, a consolidated “mega-lawsuit” was filed in federal court in Philadelphia against the National Football League (NFL). The case brought together more than 80 separate lawsuits previously filed against the NFL linked to the long-term consequences of head injuries in football. The plaintiffs in the new suit include 2138 former players and an additional 1218 family members and other related parties (Wilner, 2012). They charge that the league “fraudulently concealed” the long-term effects of head trauma, claiming in their 88-page complaint that “The NFL’s response to the issue of brain injuries . . . has been, until very recently, a concerted effort of deception and denial” (Mihoces, 2012). The suit further charges that “The NFL, like the sport of boxing, was aware of the health risks associated with repetitive blows producing sub-concussive and concussive results and the fact that some members of the NFL player population were at significant risk of developing long-term brain damage and cognitive decline as a result” (Wilner, 2012).

This essay, originally drafted before the filing of the new consolidated NFL lawsuit, examines the history of chronic head injuries in both boxing and football. However, unlike the plaintiff’s attorneys in the NFL case, who wish to claim that the league was fully cognizant of the health risks produced by subconcussive and concussive head trauma, our review of the scientific literature

suggests that physicians and sport scientists studying head injuries in football have only recently begun to pay attention to the similar injury patterns of the two sports. Boxing, the so-called “sweet science of bruising” (Oates, 1995, p. 52), has been under medical scrutiny for its high incidence of head injuries and their long-term degenerative consequences for at least 80 years. However, and perhaps even ironically, for most of those same decades, as thousands of American football players suffered concussion after concussion, almost no one connected what was happening in football to the well-documented history of chronic head injuries in boxing. For example, in September 2010 an autopsy report on a football player from the University of Pennsylvania, Owen Thomas, who had taken his own life, concluded that his brain showed degeneration associated with chronic head trauma, specifically a condition now referred to as chronic traumatic encephalopathy (CTE). These morphologic changes to Thomas’ brain had occurred in spite of the fact that he had never been diagnosed with a concussion at any point during his football career. *New York Times* reporter Alan Schwarz, when commenting on the significance of the autopsy’s finding, wrote “The idea that CTE can stem from hits below the level of concussion . . . is relatively new” (Schwarz, 2010c). In a narrow sense, Schwarz is correct; the diagnosis of CTE in former football players is barely a decade old. However, the notion that the brain’s ability to properly function gradually declines because of the knockouts and subconcussive blows they suffer is definitely not news in the sport of boxing. The term “punch drunk” was used by Dr. Harrison Martland as early as 1928 to describe exactly the same condition (Martland, 1928).

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In this article we examine the evolution of the understanding of sport-related chronic head trauma. While Schwarz and others were surprised that CTE could develop in football players, this review suggests that such a result should have been anything but surprising, had football only paid more attention to boxing. It is sad, but since the time of Martland almost no one looked at the possibility that football could cause the same long-term brain deterioration that boxing did—until we entered the 21st century. In the 1990s and first decade of the 21st century, it appears that some of this lack of attention can be traced to the NFL's Committee on Mild Traumatic Brain Injuries, a committee which helped to deflect attention away from the obviously similar physiological and psychological symptoms of CTE seen in former players of both sports. As will be shown in a series of articles that are still frequently cited, the members of this important committee defended NFL policies that sent players with concussions back into the same game in which they had just been injured, and denied that chronic traumatic encephalopathy would develop in football players. The committee's loss of credibility, and our new understanding of the similar patterns of head trauma seen in these two sports, came about only because other scientists continued to ask good questions and to gather and analyze the evidence. Even so, our aim in this review is not to assign blame but simply to document what was known about chronic head trauma in these two sports and when such knowledge entered scientific discourse. The question of whether the administrators of football (or boxing) acted in the best interests of their athletes in light of such knowledge must now be left to the courts.

From “Punch Drunk” to “Chronic Traumatic Encephalopathy”— Repetitive Head Trauma in Boxing: 1928–1973

While Martland's article in the *Journal of the American Medical Association (JAMA)* was the first scientific article to use “punch drunk” to describe the cognitive impairment, loss of memory, slurred speech, balance disturbances, and abnormal movements seen in many older boxers, the term was well-known and the symptoms described by Martland had been observed by both laymen and boxing insiders well before the 1920s. In the boxing community, Martland explained, it was also referred to at times as “slug nutty” and “cutting paper dolls.” In his pioneering study of the condition, Martland examined five men who were referred to him by a boxing promoter who described them as “punch drunk.” Following examination, Martland concurred that the five displayed abnormal behaviors which he characterized as “punch drunk syndrome.” He further suggested that the condition probably occurred most frequently in poorly skilled boxers who were likely to sustain more blows to the head. In his article, Martland detailed one particular boxer as a case study. The subject was “a top notcher”

who had fought for seven years and had been retired for 15 at the time of examination. According to Martland, the former boxer, although apparently healthy in many respects, exhibited a staggering gait, tremors of the hands, and stammering. Martland reasoned that “there is a very definite brain injury due to single or repeated blows on the head or jaw which cause multiple concussion hemorrhages in the deeper portions of the cerebrum. . . . These hemorrhages are later replaced by a gliosis or a degenerative progressive lesion in the areas involved” (p. 1103). Martland speculated that the hemorrhages in those with the punch drunk condition were fewer in number or located in different regions of the brain than those in individuals who died as a result of their head injuries. These lesions, he speculated, were likely responsible for the physical manifestations of what he came to call punch drunk syndrome. While conceding that his theory “is quite unsusceptible of proof at the present time” (as he was dealing with living subjects), the author suggested that the condition could be observed in 50% of older fighters (p. 1103).

The next report in the medical literature on chronic head trauma in boxers was written in 1934 by Mayo Clinic physician Harry Parker. Parker described three men whom he had personally examined, all retired boxers, who complained of symptoms similar to those described by Martland. Echoing Martland, Parker also argued that neurological degeneration was based, at least in part, on the volume of trauma sustained. In his words, those afflicted tended to be “the less expert but courageous men who take considerable injury in the hope of wearing out their opponent” (Parker, 1934, p. 20). The case studies Parker provided were of fairly young men, between 24 and 30 years of age. The chief complaint of each man was of motor dysfunction, primarily a staggering gait and trembling of the hands. Decreased ability to concentrate, slurred speech, and problems with memory loss were also mentioned by each of the men. Parker hesitated to speculate on the morphological cause of the symptoms, noting that postmortem study was “lacking.” He believed, however, that the men's condition was related to their experience in the ring, noting “the frequency of occurrence of conditions of this kind as reported by . . . people who followed the profession of pugilism makes it seem very likely that [the patients'] profession led to their ultimate disablement” (p. 28).

Three years later, J.A. Millspaugh, a naval medical officer, sought to rename the condition described by Martland and Parker (Millspaugh, 1937). Millspaugh preferred the term “dementia pugilistica,” on the basis that the term “punch drunk” had a “derisive connotation.” The use of Latin also “medicalized” the condition and the new term enjoyed considerable cachet among doctors, the media, and the boxing community. It is, in fact, still occasionally used to describe damage to the brain as a result of repetitive injuries (Batteh-Freiha, 2010).

Millspaugh's article also described the symptoms of the disorder, although no case studies were provided and the symptoms enumerated by the author were, as he

described them, “composite impression[s].” Those symptoms included shuffling gait, amnesia, transient paralysis, confusion, delusion, and “various mannerisms such as scowling, snorting, blowing, grimacing, crouching, and squaring off” (p. 300). While providing little new information on the exact cause of the condition or diagnostic criteria, Millspaugh’s observations were quite prescient given the current debate over football, as he noted “The mental unbalance more commonly encountered among pugilists is also observed among other sports representatives who sustain considerable head trauma” (p. 297). Further, he wrote that “Repeated and frequent concussions . . . are, to say the least, not conducive to stabilized mental equilibrium” (p. 302).

British neurologist Macdonald Critchley published the first of two important studies on head trauma in boxers in 1949 (Critchley, 1949). In “Punch Drunk Syndrome: The Chronic Traumatic Encephalopathy of Boxers,” Critchley demonstrated that the condition was generally not observable until a number of years had passed since the onset of boxing. In his 21 subjects, the minimum for evidence of behavioral and neurological changes was six years; although in one individual it took 40 years before symptoms became manifest. The average time for the onset of the condition after retirement was 16 years (Critchley, 1949). In his second and more well-known 1957 article, Critchley dropped the term “punch drunk syndrome” in favor of “chronic progressive traumatic encephalopathy” (Critchley, 1957). Critchley, who claimed to have seen more than 60 patients suffering from boxing-related head trauma in his practice, detailed 11 case studies in the article published in the *British Medical Journal*. The symptoms generally described in those individuals included “progressive slowing of speech and thought . . . slowness of movement, and tremors” (p. 357).

Several important observations, incorporated into our current understanding of repetitive head trauma, are attributable to Critchley. Like Millspaugh, he observed that the symptoms previously mentioned were seen in men “notorious as being able to ‘take it’” (p. 358). Critchley explained that the affected boxers were generally able to endure tremendous beatings and still stay upright and continue the match. And, of special importance to our modern inclusion of depression as a symptom of CTE in football players, is Critchley’s observation that brain damage produced by repetitive trauma could lead to personality changes. Critchley’s fifth case study described a British seaman and recreational boxer who became increasingly distant and angry throughout the course of his naval career, which included his participation in more than 300 boxing matches. Critchley concluded that, in development of the condition, “The sum total of contests is important, as well as the number of occasions upon which the boxers have been rendered unconscious” (p. 359). This statement suggests that the effects of chronic head trauma are dependent on the volume of impacts as well as the magnitude of those events, and it should not be forgotten that in addition to absorbing blows to the head during actual bouts, boxers are also hit repeatedly in

sparring and practice sessions. Once the boxer had been subject to impacts sufficient to “establish” the disorder, Critchley noted, the condition was not reversible and progressed steadily in spite of the boxer’s retirement.

Critchley was not able to look inside his patient’s heads, however, and suggested in the conclusion of his 1957 study that it was imperative that doctors begin to do postmortem studies of the brains of boxers (p. 362). Up to that time, only one study had examined the morphological changes in the brain of an individual diagnosed with punch drunkenness. That study, published by Brandenburg and Hallervorden in 1954, described the presence of “senile plaques,” similar to those observed in Alzheimer’s patients, in the brain of a 31-year-old ex-boxer. The discovery of this particular kind of damage in the brain of such a young fighter was an important development in the understanding of the cause of the physical manifestations of punch drunkenness. Martland’s much earlier work on concussion hemorrhages had been done on patients who died as a result of their head injuries; he did not perform autopsies on individuals suspected of having the punch drunk condition who had lived for years after sustaining their head trauma (Martland, 1928).

As boxing rose in popularity with the American public thanks to the advent of regularly televised matches in the late 1940s and 1950s, medical investigators continued to try to determine the true nature and extent of the punch drunk condition (Roberts, 1969; Rodriguez, 2009, p. 38; Sercl and Jaros, 1962). However, it was difficult to assess the extent of this condition with no standard diagnostic criteria. In his initial description of punch drunk syndrome, Martland had speculated that the condition affects 50% of boxers who remain in the ring long enough to develop the condition. British physician A.H. Roberts tested this hypothesis and attempted to more clearly define the condition when he randomly examined 224 ex-boxers who had registered with the British Boxing Control Board between 1929 and 1955. Roberts had the men take intelligence and memory tests, submit to a full clinical exam, and complete a medical history. Roberts’ study, published in 1969, showed an overall prevalence of chronic brain damage in only 17% percent of the boxers he examined. However, he observed that individuals with longer careers were far more likely to suffer from “traumatic encephalopathy.” Like Critchley, Roberts concluded that the amount of exposure to trauma is a key variable in eliciting chronic brain damage, and he speculated that a small degree of brain damage may occur with every fight. Regarding the cause of the “traumatic encephalopathy,” Roberts believed it might be caused by “microscopic lesions” of the brain produced by the trauma. He noted, however, that postmortem “histological proof that such acute lesions do occur is still lacking” (p. 105).

While Roberts was examining human subjects, neurosurgeon Friedrich Unterharnscheidt was utilizing animal models to explore the mechanical aspects of head injuries (Unterharnscheidt, 1970). In his various research studies, monkeys, cats, and rabbits were

subjected to head blows by a “concussion gun.” The intent of these experiments was to determine the dose of impact that would cause a concussion, as well as the outcome of repetitive impacts. Concussion in the animals was defined as a loss of consciousness due to the traumatic impact. A variety of experimental groups were used, including subjects exposed to single or repetitive impacts. In the case of animals subjected to repeated impacts, the intervals were set at varying rates between five and twenty seconds in order to mimic the conditions of a boxing match. Animals were sacrificed and examined after receiving their specified number of impacts. Unterharnscheidt noted that there were no remarkable physical changes in the brains of animals who received only one subconcussive impact. Nor were there changes in the brains of animals receiving multiple subconcussive impacts, provided they were sacrificed immediately after the injury. The animals that survived for several days or weeks after their injuries did show histological changes of the brain. From this, Unterharnscheidt concluded that “impacts of lesser intensity (as employed in these experiments), when applied in close succession, cause brain damage of greater severity than that produced by the higher impact intensities applied at longer intervals (several days to one week)” (p. 441). It should also be noted that the most comprehensive examination of all aspects of the medical literature on boxing is contained in the 900-page *Boxing: Medical Aspects*, published by Freidrich Unterharnscheidt and his wife Julia Taylor-Unterharnscheidt in 2003.

Although a few researchers autopsied individual boxers and reported those results in the medical literature (Constantiniades and Tissot, 1967; Corsellis and Brierley, 1959; Grahamann and Ule, 1957), the proof sought by Roberts—that the physical manifestations of punch drunk syndrome were caused by microscopic brain lesions—was finally provided in a 1973 article by Corsellis, Bruton, and Freeman-Browne in the journal *Psychological Medicine* (Corsellis, Bruton, & Freeman-Browne, 1973). Corsellis and his colleagues autopsied the brains of 15 boxers who died decades after quitting the ring. Over half of the boxers investigated had participated in more than 300 fights and many had careers spanning longer than 15 years. In their examination of the brains of these former boxers, they saw consistent neurological changes that were atypical for men of their age. To confirm that the changes were consistent with chronic traumatic encephalopathy, Corsellis also interviewed the men’s wives and other family members about the physical presentation of the condition. The symptoms described during the interviews were similar to those mentioned by previous authors describing *dementia pugilistica* or CTE. Taken with the previous histological examinations, however, the authors concluded that a characteristic pattern of injury, including cerebral atrophy, could be shown in retired boxers. In describing this, the authors wrote, “septal anomalies have occurred suspiciously often. . . . Secondly, Parkinsonism as well as neuronal degeneration and loss of pigment in the *substantia nigra*

have been mentioned several times. Thirdly, a diagnosis of Alzheimer’s disease has sometimes been broached, although in this connection the most striking and more anomalous feature has been the tendency for neurofibrillary tangles to occur in the *absence* of senile plaques” (p. 300). Corsellis and his colleagues had definitively documented a degeneration of the brain resembling other “organic dementias” like Parkinson’s disease—but with its own unique injury pattern.

Contemporary Understanding of the Underlying Causes of CTE

One of the difficulties in reading the literature in this area is that the term *concussion* carries different meanings in scientific discourse. Paul McCrory and Simon Berkovic addressed these different meanings in an historical analysis of the term in 2001, published in *Neurology*. According to them a concussion is both the immediate and transient symptoms of a mild traumatic brain injury (McCrory & Berkovic, 2001). It is also, they explain, both a clinical state of brain function and the events that brought about that clinical state. The concussion occurs when the brain is shaken or jarred and the brain, which normally floats surrounded by cerebral fluid, hits the bony inner wall of the skull. Historically, the belief was that at this point of contact, bleeding or small hemorrhages would occur that subsequently resulted in a temporary loss of brain function and even unconsciousness. Some, however, argue that these sites resulted in permanent lesions and perhaps small-scale loss of neurological function. S.J. Peerless and N.B. Rewcastle, for example, noted in their 1967 study that despite the fact that most concussion patients appear to return to normal, we “should not exclude the possibility that a small number of neurons may have been permanently disconnected or have perished” (Peerless & Rewcastle, 1967, p. 582).

Recent research has clarified that the physiological aspects of head injuries are far more complicated. In a concussion, symptoms have generally been regarded as transient as it appears in most cases that the brain heals itself with time and returns to normal function. However, when the brain suffers a sudden acceleration or deceleration caused by a blow to the head—whether the brain actually hits the side of the skull or not—neurons and other brain tissues are pulled and twisted and often shear and tear as the brain swirls inside the skull. If the blow is severe enough and considerable damage is done, the result is a concussion in which brain function is impaired until the neurons have time to heal (Bartholet, 2012). However, as Peerless and Rewcastle pointed out back in 1967, some neurons may not return to full function (Peerless & Rewcastle, 1967). Further, it is now believed that even subconcussive impacts move the brain enough inside the skull to trigger morphological changes.

In recent years we’ve witnessed a new understanding of the true complexity of minor head injuries like concussions and the subconcussive blows we associate with

CTE. Researchers have demonstrated that mechanical stresses in the brain caused by sudden movements also alter the permeability of the neuron's membrane (Jenkins et al., 1989; Julian & Goldman, 1962; Pettus, Christman, Giebel, & Povlishock, 1994; Povlishock & Pettus, 1996). This change in membrane permeability appears to allow a significant efflux of potassium ions and influx of calcium ions (Fineman, Hovda, Smith, Yoshina, & Becker, 1993; Katayama, Becker, Tamura, & Hovda, 1990; Takahashi, Manaka, & Sano, 1981). By 2001, physicians Christopher Giza and David Hovda had compiled the results of this research into what they termed the "neurometabolic cascade of concussion" (Giza & Hovda, 2001). They argued that mechanical injury to the brain also resulted in ionic shifts within the brain's neurons. Neurons send electrical signals by creating an electrochemical gradient, keeping some ions like potassium inside the cell and others—such as calcium and sodium—outside the cell. By opening up special channels, the ions temporarily displace each other, causing a change in the charge of the cell that results in the conduction of an electrical signal. Mechanical injury to the cell causes an extended opening of the channels and allows for large-scale migration of ions, negating the cell's ability to continue to send an electrical signal. The end result is altered cognitive function.

Moreover, the additional calcium inside the cell appears to damage the neural tissue in a variety of ways. When injured, pumps which normally work slowly and steadily to push calcium back out of the cell begin working at a frenetic pace. Because blood flow has been impaired, however, the cells have difficulty supplying the energy required to keep up with the demand. This creates what Giza and Hovda refer to as an "energy crisis" making "the brain less able to respond adequately to a second injury and potentially leading to longer lasting deficits" (p. 228).

Giza and Hovda also found that the additional intracellular calcium "disrupt[s] neurofilaments and microtubules" (p. 228). The microtubules are associated with a protein called tau, which has become the defining diagnostic indicator for CTE. Disruptions of the microtubules can cause tau proteins to clump together inside the cell, creating the neurofibrillary tangles described by Corsellis and ultimately leading to the cell's death. While neurons may be able to recover from some damage, repeated insults—before the cell has recovered—may lead to more diffuse damage and increased likelihood of cell death (Gavett, Stern, & McKee, 2011). Neuronal damage also causes the release of excitatory neurotransmitters such as glutamate, which can be toxic to neurons in high concentrations (McKee, et al., 2009). Writing in 2001, Giza and Hovda reminded readers that their findings confirm the fact that "significant metabolic abnormalities may occur in the absence of overt clinical symptoms" (Giza & Hovda, 230). Although there is much more work to be done in this area, it is now widely accepted that both concussions and subconcussive impacts are matters of grave concern for athletes and the administrators of sport.

"Dings," "Feeling Fuzzy," and the History of Head Trauma in Football

In October 1897, in a match between the University of Georgia and the University of Virginia, held in Atlanta, Georgia running back Richard Vonalbade ("Von") Gammon was hit by an opposing player, landed on his head, and lay unconscious on the field for several minutes. Gammon's well-intentioned teammates pulled him to his feet where he wobbled and immediately began to vomit ("From the gridiron to the grave," 1897). Two doctors in attendance at the game came from the stands to lend assistance to Gammon, who was diagnosed as having sustained a concussion and taken to nearby Grady Hospital. He died early the next morning and his death set off one of the first great debates on the safety of football in America (*Football victim*, 1897).

Von Gammon's death from a head injury was not unusual in this early era of American football, but what happened in Atlanta following his death was nothing short of extraordinary. On the morning after Gammon's death, the speaker of the Georgia House of Representatives was quoted in a front-page article in the *Atlanta Constitution* decrying the game's brutality. In that same article, another member of the House, Joe Mansfield, called for the abolishment of the game and compared the brutality of football to that of bullfighting. The state's Attorney General, Joseph M. Terrell, weighed in with a similar opinion, telling the reporter that football "is as brutal as it looks, it ought to be prohibited . . . it's a wonder that all of the players were not killed ("Death knell," 1897). In the week that followed Gammon's death, a bill made its way to the floor of the Georgia House of Representatives asking that football be banned in the state; it passed overwhelmingly by a vote of 91–3 less than a week later (*Anti-football bill*, 1897). The City of Atlanta, not be outdone by the state legislature, passed a ban on football inside the city limits that same week. And the three largest universities in the state—Gammon's alma mater, The University of Georgia; Georgia Tech; and Mercer University in Macon—announced they were disbanding their football teams ("Atlanta will have no football," 1897).

Georgia's spasm of football reform turned out to be short-lived. Despite the passage of the bill by the Georgia State Senate as well as the House of Representatives, Governor William Yates Atkinson refused to sign the bill because of a letter from Gammon's mother about how much her son had loved the game. The furor gradually died away, the three universities resumed play the next season, and except for the White House Conference on Football organized by President Theodore Roosevelt in 1905 (which ultimately led to the formation of the NCAA as a way to create standardized rules and limit violence in football), it would be more than 100 years before lawmakers again paid much attention to traumatic head injuries in football (Watterson, 2000).

During that intervening century, as football grew into America's favorite spectator sport and began being played in Pee-Wee, Pop Warner, junior high school, high

school, collegiate, and professional divisions, surprisingly little attention was paid to the head trauma that became part of the game. In 1933, M.A. Stevens and W.M. Phelps published one of the first manuals on injury treatment in football, but devoted only five pages of text out of 198 to concussions even though they wrote that “more concussions occur in football than are generally recognized” (p. 63). Beyond suggesting that players wear a helmet, Stevens and Phelps offered little advice about head injuries in football as they did not consider them particularly dangerous. “It is well to emphasize,” they wrote, “that concussions and fractured skulls per se are not particularly dangerous and do not have deleterious after-effects unless there has been brain (cortical) damage with sub-dural or extra-dural hemorrhage” (Stephens and Phelps, 1933, p. 63). Stevens and Burke optimistically concluded, “we look forward to the near future when vastly improved headgear will eliminate all serious head injuries” (p. 66).

Harvard University’s team doctor, Augustus Thorndike, took a slightly different position in 1952. In the *New England Journal of Medicine*, Thorndike described a variety of possible injuries sustained by athletes and suggested that patients who suffered more than three concussions in their lives, or those who had a concussion with “more than a momentary loss of consciousness,” should not continue to participate in sports in which they might expect to receive another blow to the head (Thorndike, 1952, p. 554). He then noted, “The college health authorities are conscious of the pathology of the ‘punch-drunk’ boxer. Just how much one should permit recurrence of cerebral concussion in college athletics is a matter of opinion” (p. 556). Thorndike goes no further with this analogy, unfortunately, and it does not appear to have been noticed by others writing about football in later decades.

After Thorndike, football research in the middle years of the 20th century generally falls into three divisions: 1) studies of head and cervical cord injuries that resulted in death or quadriplegia (Alley, 1964; Murphy, 1987; Reid, Tarkinton, Epstein, & Odea, 1971; Schneider, Reifel, Crisler, & Oosterbaan, 1961; Torg, Vegso, Sennett, & Das, 1985); 2) studies related to the creation and testing of new models of football helmets (Levy, Ozgur, Berry, Aryan, & Apuzzo, 2004a; 2004b); and, 3) analyses of the physiological mechanisms and visible symptoms of concussions (Gronwall & Wrightson, 1974, 1975; Ommaya, 1974; Saunders & Harbaugh 1984; Yarnell & Lynch, 1973). Paul Yarnell and his research partner, Steve Lynch, for example, described several case studies of players who “got dinged” during football games. Quoting author and former football player Dave Meggyesey, they defined the “ding” as getting “hit in the head so hard that your memory is affected, although you can still walk around and sometimes even continue playing.” (Yarnell & Lynch, 1973, p. 196). Not surprisingly, no concern was expressed in this article about any possible long-term changes in brain function or cognition because of football. R.C. Schneider also commented on the practice

of getting football players back in the game as quickly possible. “The thoughtful neurosurgeon cannot help but think of the dichotomy in standards set for the average citizen and football player. In many hospitals the traffic accident victim is unequivocally hospitalized for 24 hours if there has been a blow to the head with unconsciousness.” Continuing, he wrote, “However, with football players, it is obviously oftentimes ‘necessary’ to return them to the game if they have only sustained a first or second degree concussion” (Schneider, 1973, p. 165).

In a study, published the following year, researchers associated with Northwestern University’s Medical School used radio telemetry and electroencephalography readings (EEGs) to study the actual impacts players received during a football game (Reid, Epstein, & Louis, 1974). A report on their research, published in *Physician and Sports Medicine*, explained that they measured more than 650 impacts sustained by one football player during a single season at Northwestern University and recorded his EEG readings, the force of the impacts, and shot video as he played and practiced to create a complete record of his head impacts during the season. The impacts measured between 150g and 450g. One of the hardest of these blows resulted in a concussion, marking the first time that researchers captured EEG readings while someone was actually concussed. Particularly relevant to the current debate over CTE was the group’s finding that “a concussion need not be the result of a single blow but may be the cumulative effect of a series of blows” (p. 34). Also worthy of note was their observation of the altered EEG patterns experienced after a series of intermediate-level impacts caused the subject to report that he “felt fuzzy.” In their discussion, Reid and his colleagues observed that “The helmet that is designed to absorb high intensity impacts may provide little protection from low or medium intensity blows, and the cumulative effect of these can be injurious to the brain.” (p. 34).

Two months later, *The Physician and Sports Medicine* published a nine-page round table discussion between the journal’s editor, Allan J. Ryan, Ayub Ommaya of the National Institutes of Health, and Chester Pierce and Thomas Quigley of Harvard University. Entitled “Concussion and Head Injuries,” the article explores the latest information on the cause and diagnosis of concussions in athletes of all kinds, discusses best medical practices for players who receive head injuries in football games, talks about the redesign of football helmets to limit concussions, and suggests that the practice of “spearing” or leading with the head when tackling an opponent be eliminated from the game. The most fascinating part of the article, however, comes at the end, when Ryan switches gears from a discussion of rugby—in which no helmets are worn and relatively few head injuries occur—to boxing. Although Ryan does not mention Corsellis’ 1973 boxing study by name, he introduces the subject of the new autopsy study on retired boxers and then asks the committee if boxing should be banned (Corsellis et al., 1973; Ryan, 1974a, p. 53). A lengthy discussion ensues about the safety of boxing

among the panel members, but at no time does anyone make the leap and compare boxing and football. Nor is there any discussion by these experts of possible long-term brain injury as a result of playing football. (p. 54).

In an editorial in that same issue, Ryan revisits the topic of long-term brain damage from sport participation: “But how many more are there (suffering undiagnosed head injuries) whose deficits remain subclinical or who in later years manifest the signs and symptoms of cerebral deterioration? How many prize fighters, for instance, have the punch-drunk syndrome late in their lives?” (Ryan, 1974b, p. 85)? Continuing, Ryan noted the recent survey of football injuries completed by Blyth and Mueller (1974a, 1974b), who had found that concussion without loss of consciousness was very common in football, and then asked, “What will 20-year follow-up on these players show? . . . What about the thousands of players who are ruled out of competition because of repeated concussions with serious temporary symptoms but no detectable permanent deficits?” (p. 85). Although Ryan suggests that the answers to these rhetorical questions will be found in the previously cited round table; as noted, the experts did not really address the question of CTE in football. Nor does Ryan take a hard stance on the question in 1979 when he returns to the topic in an editorial on concussion safety called “A Hit in the Head” (Ryan, 1979). Ryan opened it with a report on the 1977 research completed at Purdue University that compared the EEGs of freshmen football players following their first season of play with collegiate wrestlers. Only 8.7% of the wrestlers had had an unusual EEG, compared with 35% of the football players. Four years later, the Purdue researchers retested 28 of the football players and discovered that by then 57% of them showed abnormal EEGs (Hughes, Wilms, Adams, & Combs, 1977). Although Ryan discusses EEG changes and brain atrophy in boxers in his next paragraph, he, once again, does not question the need for hard hitting in football. He finishes his editorial by stating, “The possibility of injury and permanent disability is present in every sport. But the risk of lasting brain damage from concussion does not justify entirely eliminating contact and combative sports” (p. 51).

We pay particular attention to the articles in *Physician and Sports Medicine* during this era because the journal was written with coaches, physical educators, and sports physicians in mind—people who might not normally read medical and scientific journals. Begun in 1973, the peer-reviewed *Physician and Sports Medicine* used layperson’s terms to report scientific research on medical issues in sport, and it was an important conduit by which scientific and medical information reached practitioners in the early years of the emergence of the sport science profession. Allan Ryan, as its editor, had also served as president of the American College of Sports Medicine and was recognized as a leader in the field of sports medicine during this era (Berryman, 1995, pp. 77–87). His interest in, and understanding of, head injuries in boxing and football are, therefore, of importance in the history of the increasing awareness of CTE. Ryan’s

last article in the *Physician and Sports Medicine* on the question of football and head injuries appeared in 1987. Entitled “Brain Injuries in Football,” the editorial noted that there had been nine football fatalities the previous year because of head injuries, and that eight of them had been high school players. In talking about the concussive events that led to these deaths, Ryan called attention to the research of R.W. Rimel and his colleagues, who found that concussions often continued to cause neurological and cognitive disorders for months after the original incident, (Rimel, Giordani, Barth, Boll, & Jane, 1981). Ryan also cited the work of Gronwall and Wrightson, who found a similar slowness in returning to “normal” in their two studies in the previous decade (Gronwall & Wrightson 1974, 1975). In the end, Ryan guardedly wrote, “We must be more alert to the occurrence of concussion in a football player during practice or play. . . . Additionally, if we are to reduce and perhaps eliminate fatalities due to brain injury, we must make serious efforts to determine whether athletes who have sustained concussions may be susceptible to further, more serious brain injury, and whether they should continue to play football” (Ryan, 1987, p. 39).

There were other studies, of course, that Ryan could also have cited in the 1980s. Susan Gerberich and her research team, for example, reported on a survey of more than 3000 high school players in Minnesota and documented that 69% of those who were knocked unconscious during a game were allowed to continue playing on the same day, and that some players were still experiencing concussion symptoms six to nine months after the initial incident (Gerberich, Priest, Boen, Straub, & Maxwell, 1983). In 1984, R.L. Saunders and R.E. Harbaugh looked at the particular problems associated with resuming play too soon after a concussion, which they described as the “second impact,” a term that would be embraced by athletic trainers and others in the years ahead (Saunders & Harbaugh, 1984; J. McQuillen, B. McQuillen, & Morrow, 1988; Kelly et al., 1991). Athletic training professor W.E. Buckley at Pennsylvania State University reported his eight-year statistical analysis of concussions in college football in 1986; William Alves and his colleagues at the University of Virginia reported on a four-year statistical analysis of “minor” head injuries sustained by 10 collegiate football teams; and Robert Cantu authored “When to Return to Contact Sports after a Cerebral Concussion” in 1988 (Buckley, W.E., 1986; Alves, Rime, & Nelson 1987; Cantu, 1988).

An article that might have helped Ryan think more seriously about the possibility of CTE outside the sport of boxing, however, did not appear until 1989; it dealt with soccer players, not American football players (Sortland & Tysvaer, 1989). Still, “Brain Damage in Former Association Football Players” was thought provoking. The Norwegian research team used cerebral computer tomography (CT) scans to evaluate the brains of 33 former members of the Norwegian national soccer team who ranged in age from 39 to 68 years of age. They found after taking several dozen measures of each man’s brain

that 33% had noticeable cerebral atrophy and a widening of the lateral ventricles. They also found evidence of cortical atrophy in 18% of their subjects but could not definitively link that to head trauma as age could also have been a factor for that change. Investigators Sortland and Tysvaer made no assessment of behavior changes, but did conclude their article by stating that the brain damage seen in the CT scans “is considered to be caused by multiple small head injuries” (p. 48).

Reform Efforts in Boxing Related to Chronic Head Trauma

During the 1980s, while most football researchers continued to be largely concerned with concussions, second impact syndrome, helmets, and other acute forms of football head trauma, the American Medical Association began raising strong questions about the ethics of boxing. Concerns about the safety of boxing were not new (Morrison, 1986), but in the 1980s the mounting scientific evidence of possible life-long impairment caused many to question the nature of the sport itself (Anderson, 2007). Following the particularly visible and tragic death of South Korean boxer Duk Koo Kim in a televised title-fight in Las Vegas, the American Medical Association debated the issue at its 1983 national convention and passed a resolution calling for the end of amateur boxing (Carp, 2007). A second resolution calling for a ban on all boxing—amateur and professional—was passed in December 1984 (Morrison, 1986). Leading this movement was *JAMA* editor George Lundberg, who published two scathing editorials in *JAMA* in the 1980s entitled “Boxing should be banned in civilized countries” (Lundberg, 1983) and “Boxing should be banned in civilized countries: Round two” (Lundberg, 1984). In response to the AMA’s charge that boxing was unsafe, the National Boxing Safety Center was founded to perform medical examinations on boxers and to promote research on boxing injuries (Nash, 1985). Other changes *proposed* as means to improve safety in this era included closer medical supervision and disqualification of fighters with an excessive number of knockouts; training referees to intervene more quickly; mandatory rest periods between knockouts; teaching boxers proper defensive fighting; mandatory use of headgear at all levels of boxing; elimination of blows to the head; and changes to the gloves (Nash, 1985). Not all the suggested reforms were accepted, of course. Protective head gear is still worn only in amateur boxing matches and blows to the head continued to be allowed in all levels of boxing. Boxing did tighten its policies regarding how soon after a knockout a boxer could return to the ring, however, and has set mechanisms in place to monitor how often boxers are involved in matches (Farber, 1994).

Ironically, the use of gloves has probably served to encourage the development of CTE in boxers. As observed by historian Elliot Gorn, blows to the head were rare in the bare-knuckle era because hitting a man’s head

with a bare fist can result in bruising and even broken bones (Gorn, 1986). The adoption of gloves in the late 19th century, however, allowed a man to hit his opponent innumerable times in the head without fear of fracturing his hand. Allan Ryan called for the elimination of gloves on this basis, stating that it would “make boxing a sport of jabs and defense . . . deemphasiz[ing] knockout punches” (Ryan, 1983, p. 49).

The NFL Finds the Committee on Mild Traumatic Brain Injury

In football, meanwhile, second impact syndrome and concerns about the proper treatment of concussed athletes dominated the scientific literature on athletic head trauma in the 1990s (Dikmen, McLean, & Temkin, 1986; Farber, 1994; Goldstein, 1990; Harrington, Malek, Cicerone, & Katz, 1993; Kelly et al., 1991; Ruchinskas, Francis, & Barth, 1997; Wilberger, 1993). Although the NFL then allowed individual teams to set their own policy on concussions and head injuries, the League decided in 1994 to begin its own investigations and NFL Commissioner Paul Tagliabue announced the formation of the Committee on Mild Traumatic Brain Injury, appointing New York Jets team physician Elliott Pellman as its chairman (Schwarz, 2007). The NFL’s choice as chairman of this important committee was curious. Pellman specialized in rheumatology, had no particular expertise in neurology, and had no record of research on concussions or head injuries. (Keating, 2007; Schwarz, 2007; Wilson, 2005).

Nonetheless, Pellman, biomechanist David Viano, and several other colleagues connected to the NFL committee began gathering data on head injuries and eventually published 16 papers—all in the scholarly journal *Neurology*, edited by Mike Apuzzo, the New York Giants’ neurosurgical consultant (Pellman, Lovell, Viano, & Casson, 2006; Pellman, Lovell, Viano, Casson, & Tucker, 2004, 2005; Pellman et al., 2004a; Pellman & Viano, 2006; Pellman, Viano, Casson, Arfken, & Powell, 2004; Pellman et al., 2004b; Pellman, Viano, Tucker, & Casson, 2003; Pellman, Viano, Tucker, Casson, & Waeckerle, 2003; Pellman, Viano, Withnall et al., 2006; Viano, Casson, & Pellman, 2007; Viano et al., 2005a; Viano et al., 2005b; Viano, Hamberger, Bolouri, & Saljo, 2009; Hamberger, Viano, Saljo, & Bolouri, 2009; Viano & Pellman, 2005; Viano, Pellman, Withnall, & Shewchenko, 2006). As a body of work, the articles have been controversial (Keating, 2006; Laskas, 2009). *ESPN: The Magazine* reporter Peter Keating observed in 2006 that the NFL Committee’s studies contradict the research and experiences of “many other doctors who treat sport concussions, not to mention the players who have suffered them” (Keating, 2006).

One of the first studies published by Pellman’s group addressed the question of possible chronic encephalopathy in football as part of a larger survey of concussions. In their concluding comments, Pellman claimed that they found no signs of CTE. “This was

not a surprising result,” they wrote, “because chronic traumatic encephalopathy has been reported only in boxers and a few steeplechase jockeys.” Continuing, he added, “numerous studies have shown that the occurrence of chronic encephalopathy of boxers is related to length of career and number of professional bouts, not to the number of knockouts (concussions) sustained. It is well documented that chronic encephalopathy of boxers results from the accumulation of damage from multiple subconcussive blows to the head over a prolonged period of time, not the number of concussions sustained” (Pellman et al., 2004b, p. 870). Pellman’s inability to admit that football was also a sport in which athletes received “multiple subconcussive blows to the head over a prolonged period of time” indicates either a gross lack of understanding of the nature of the sport itself or an attempt at deception. In 2007, Pellman resigned as chairman of the committee following a scathing article in *ESPN The Magazine* which dubbed him “Doctor Yes” and raised significant questions about the impartiality of his committee’s research (Keating, 2006). Even so, Pellman stayed on the committee for several years while it was cochaired by David Viano and neurologist Ira Casson, both of whom had been part of his original research team (Schwarz, 2007). Finally, in March 2010, Dr. H. Hunt Batjer and Dr. Richard G. Ellenbogen were named as cochairs of a renamed NFL Neck, Head, and Spine Committee (Schwarz, 2010a; NFL Health and Safety, 2012). Batjer and Ellenbogen, both eminent neurologists, reportedly have no previous ties to NFL teams and in their public remarks have been critical of the Pellman era in the NFL, stating that the research from that era “would not be used in any way moving forward” (Schwarz, 2010a).

Proof of CTE in Football: 2000–2012

Although considerable progress has been made during the past decade, our current understanding of the exact cause of CTE is still quite limited. The most recent diagnoses have been based on morphological findings similar to those described by Corsellis in which tau protein pathology is found in different distributions than is seen in Alzheimer’s and occurs “without neuritic plaques” (McCrory, 2011, p. 7). As previously discussed, it is theorized that the condition is related to the temporary derangement of neuron function. This derangement can initiate “multiple pathological cascades,” with the extent of the neurodegeneration being dependent on the nature of the original injuries and the length of survival after the triggering events (McKee et al., 2009, p. 732). Whether an individual is more likely to develop CTE as a result of a handful of concussive impacts which produce observable symptoms, or as a result of multiple subconcussive impacts, is unknown (McCrory, 2011). It is also important to note that some of the professional football players and boxers who have been autopsied in recent years also had issues with abuse of alcohol and/or illegal drugs and the effect of this is not clear (Gavett, Stern, & McKee, 2011;

McCrory, 2011). Nor do we know what impact possible anabolic steroid use may have had in the medical histories of any of these men.

However, during exactly the same years that Pellman and his colleagues were publishing work that defended the NFL—work that denied the possibility of CTE in football players—other researchers were working to help us understand the nature and etiology of CTE. The first true evidence of CTE in a professional football player arrived in 2002 with the autopsy of the Hall of Fame center, Mike Webster, by forensic pathologist Bennet Omalu (Omalu, DeKosky, Minster, Hamilton, & Wecht, 2005). Omalu also found evidence of CTE in subsequent years in the brains of Webster’s Pittsburgh Steelers teammates, Terry Long and Justin Strzelczyk, and in the brain of Philadelphia Eagles defensive back Andre Waters (Cantu, 2007; Laskas, 2009, Omalu, Bailes, Hamilton, Kamboh, Hammers, Case, & Fitzsimmons., 2011). By 2011, Omalu and his research team had documented CTE in seven professional football players and one high school player (Omalu, et al., 2011). They also, in 2010, reported the first case of CTE in an active NFL player. Chris Henry, a Cincinnati Bengals receiver, was only 26 when he died after falling (or jumping) out of a moving truck being driven by his fiancée. In the autopsy performed on his brain by Omalu, evidence of CTE was found despite his young age and the fact that he had no record of ever having had a concussion in the NFL, in college, or in high school (Schwarz, 2010b).

The other leading researcher in the area of neuropathology is Ann McKee, who became interested in CTE after discovering tau protein in the brain of a retired boxer she’d autopsied for the Framingham Heart Study (Gladwell, 2009). Her interest led her to a partnership with head-injury activist and former football player Chris Nowinski, director of the Sport Legacy Institute, who helps her acquire the brains of former NFL players for examination. McKee now directs the Center for the Study of Traumatic Encephalopathy at Boston University, which receives funding from the NFL (Willyard, 2011; Bartholet, 2012). McKee’s pathological examinations have demonstrated that CTE does develop in football players. In a paper published in 2011, McKee and her colleagues reported that they had examined the brains of 12 former NFL players, all of whom showed evidence of CTE (Stern, Riley, Daneshvar, Nowinsky, Cantu, & McKee, 2011). In 2009, she definitively wrote, “There is overwhelming evidence that the condition [CTE] is the result of repeated sublethal brain trauma that often occurs well before the development of clinical manifestations.” (McKee, Cantu, Nowinski et al., 2009, p. 734.) In 2011, in an article in *USA Today*, McKee told a reporter that she had now found evidence of CTE in more than 50 former athletes and was starting a large-scale investigation of living former football players. The longitudinal study, called DETECT, is being funded by the National Institutes of Health and will allow McKee to follow 50 former football players and 50 former athletes

from noncontact sports over the course of three years (Cohen, 2011).

Another scientist studying the course of head trauma in the living is Kevin Guskiewicz of the University of North Carolina at Chapel Hill. In 2005, Guskiewicz released the results of an ambitious survey demonstrating that chronic head trauma associated with lengthy football careers and/or multiple concussions sustained during a career was strongly associated with a host of psychological conditions that generally manifested later in life, including cognitive impairment, Alzheimer's disease, and depression (Guskiewicz et al., 2005, p. 723; Gladwell, 2009). In his survey of 2550 former NFL players, Guskiewicz found that those who had had at least three concussions during their career were five times more likely to suffer in later years from "Mild Cognitive Impairment" than were those who were never concussed. Repetitive subconcussive impacts *without* the observable signs associated with a concussion were also, in his opinion, a "risk-factor in late-life memory impairment, mild cognitive impairment, and Alzheimer's disease" (Guskiewicz et al., 2005, p. 723; Gladwell, 2009). Guskiewicz, who received a MacArthur Fellowship in 2011 to help support his research, has played a leading role in the fight against head injuries in football (Guskiewicz et al., 2007; Guskiewicz et al., 2003). In October 2009, when the government once again turned its attention to the violence of the sport and the potential health risks it imposed, Guskiewicz played a lead role in the United States' House Judiciary Committee hearings (Gladwell, 2009).

Guskiewicz has also worked to more fully understand the volume and intensity of the forces involved in head-to-head contact in football, as has Joseph J. Crisco of Brown University and Thomas Talavage of Purdue University. In separate studies using accelerometers to quantify the volume and magnitude of head impacts sustained by collegiate and high school football players, it is now estimated that players receive approximately 950 subconcussive impacts in a single season, with some individuals recording as many as 1600 (Crisco et al., 2010; Guskiewicz & Mihalik, 2011; Talavage et al., 2010). In their measurements of the impact of the blows, the researchers found that the players frequently sustained impacts between 80–100g without "sustaining a diagnosed concussion" (Guskiewicz & Mihalik, 2011, p. 10; Talavage et al., 2010). These forces are higher than the rates normally considered necessary for concussions, a fact that suggests that many players do not report when they are being hit for fear of being removed from the game (Talavage, et al., 2010).

Reforming Football

While the American Medical Association has not yet taken the strong stance that they did in the mid-1980s regarding the banning of boxing (a stand they renewed by vote in 2002), discourse among coaching and medical groups have already proposed a variety of methods

to theoretically make football less likely to cause head injuries (Elliott, 2002). The NFL, in response to the public embarrassment it suffered in the 2009 Congressional hearing, has taken several important steps during the past few years. First, the NFL's new medical committee passed a rule requiring all NFL teams to have an independent doctor available during games to make determinations as to whether a player should reenter the game. The committee followed that rule with advice that any player who suffers a concussion should not return to play or practice on the same day if he shows any signs or symptoms of a concussion. Posters explaining the new NFL return-to-play policy were put in all NFL locker rooms in 2009. NFL Commissioner Roger Goodell stated after putting the policy in place, "This new return-to-play statement reinforces our commitment to advancing player safety. Along with improved equipment, better education, and rules changes designed to reduce impacts to the head, it will make our game safer for the men who play it, and set an important example for players at all levels of play" (NFL.com, 2009). A much more important policy was put in place in 2011 as part of a new collective bargaining agreement negotiated by the players. They bargained with the NFL, and ultimately won, on an agreement to limit the number of "full contact" practices for players (Maske, 2011).

Other changes seen in recent years include the fact that officials have been increasingly diligent in penalizing players making head-first contact, and rules have been added to penalize targeting the head or neck of a defenseless player (Redding, 2010). The NCAA, for example, adopted Rule 9–6 in 2009 that allows conference officials to review game films after a game is completed to examine particularly dangerous plays and to impose sanctions if it is deemed that a "flagrant foul" occurred, even if the foul was not called during the game (Whiteside, 2009). The NFL has similarly stepped up enforcement of rules against helmet-to-helmet contact, introducing suspensions in addition to fines for breaking the rule (Layden, 2010). Other aspects of the game have also been amended or are under consideration for amendment. The "wedge" block on kickoff returns is no longer permitted, for example, and some experts argue for the elimination of kickoffs altogether (Cooper, 2010). Elimination of the "three point stance" has also been proposed as a means to limit helmet-to-helmet contact, particularly the type experienced on every play by offensive and defensive linemen (Gavett, Stern, & McKee, 2011). Researchers at the Purdue Acute Neural Injury Consortium, led by Talavage and Nauman, are also working on a new type of football helmet that they hope will reduce the cumulative effect of impacts (Purdue Newsroom, 2010). When such a product might be available and, most importantly, whether it will work, is unknown. While these steps are well intentioned, football as it is currently played is still a game of significant and numerous head impacts and it is embedded in a culture in which being seen as tough, manly, and "able to take it" is a large part of the attraction of the sport.

Boxing Lessons

One of the most significant lessons that can be learned from boxing is the need for a unified approach to imposing safety standards throughout the sport. Boxing, which has never had a unified national governing body, took no real action on head injuries for decades because each state had its own boxing commission and set its own safety standards. While professional football is somewhat regulated by the NFL, and college football falls under the jurisdiction of the NCAA, high school football is in many ways no different than boxing. Each state has its own educational agency to monitor high school sports, and although there is a National Association of State High School Associations, that agency makes recommendations to states and cannot mandate policy (Mueller & Colgate, 2009). In 2011, for example, the National Federation of State High School Associations issued a three-page “Point of Emphasis” related to the issue of concussions and head trauma in football. The report recommended, among other actions, that state agencies take steps to ensure that helmets are properly fitted, that referees watch for helmet-to-helmet contact, that coaches teach “heads up” hitting techniques, and that they encourage players to not try to get back into the game too quickly after a blow to the head (Colgate, 2011). However, all of these are merely “suggestions” and, moreover, none of them go as far as the collegiate Ivy League Conference’s decision in the summer of 2011 to disallow full-contact practices several days each week to minimize the quantity of potential blows (Belson, 2011).

In closing, we should contemplate the number 35,623,701. This is the number of American boys who played high school football between 1972 and 2007. We should also think about 1,929,069—the number of young men who played college football during that same quarter century (Daneshver, Nowinsky, McKee, & Cantu, 2011). As these men age, how many of them will begin to display some of the symptoms of this condition? Is there a true epidemic in our future? We can only hope that as the mechanisms causing CTE are more fully understood—and as we learn more about the brain’s ability to heal itself—the various divisions of football will come together to create effective safety policies so that the sport will not be faced, as boxing has been, and is, with the specter of so many of its greatest champions, as well as its journeyman athletes, diminished not by age but by the head trauma they received while participating in the sport they love.

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