Concussion: How Bennet Omalu exposed the worst kept secret in football

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There is more than a little dramatic embellishment in *Concussion*, the truth-based story of how neuropathologist Bennet Omalu discovered a previously unrecognized brain disease in football players, and then fought a David and Goliath battle against the National Football League (NFL). The Goliath in the story is the NFL, its executives, and its doctors, cast as villains out to silence critics and stifle science.

The story begins with “Iron Mike” Webster (portrayed by David Morse), a former football player with the Pittsburgh Steelers. Webster died of a heart attack at age 50 after a protracted struggle with dementia, depression, and substance abuse. His body ends up, by chance, on the table of Dr. Bennett Omalu (Will Smith), a Nigeria-born pathologist working for the Allegheny County Coroner’s Office in Pittsburgh. It’s Omalu’s passion for brains – not football that – compels him to investigate Webster’s case, not to determine the immediate cause of death, which was clear, but to discover what caused the legendary Super Bowl champ’s infamous decline into homelessness, poverty, mental illness, and legal problems.

The (Re)discovery of CTE

What Omalu found looked like dementia pugilistica (DP) or “punch drunk syndrome,” the neurological disorder known to cause dementia and movement disorders in boxers, but never before seen in other athletes. Harrison Martland first described punch drunk syndrome in a paper in the *Journal of the American Medical Association* (Martland 1928). The name dementia pugilistica was coined a decade later by Millspaugh (Millspaugh 1937), while the modern term, Chronic Traumatic Encephalopathy (CTE) was first introduced by Critchley (Critchley 1949). (The movie inaccurately
credits Omalu with naming CTE, a case of gilding the lily, since his scientific contributions have been significant enough in their own right.) By the 1970s, the disorder had been well-documented and described in the medical literature (Corsellis et al 1973), but had only been seen in retired boxers.

Omalu’s neuropathological investigation fatefuly led him to conclude that Webster also suffered from CTE, the first known case of the disease in a former football player. It would not, of course, be the last. To date, there have been approximately 150 published case studies (Maroon et al 2015), and CTE has been found postmortem in former athletes in several contact and martial sports, in combat veterans, victims of domestic abuse, and others who experienced repetitive head trauma. Omalu’s case study of Webster, “Chronic Traumatic Encephalopathy in a National Football League Player” (Omalu et al 2005), was published in *Neurosurgery*. T’was then, as the story goes, that Omalu’s troubles began, and the main focus of *Concussion* is the good doctor’s ensuing fight with the NFL, and NFL scientists who questioned his findings, his scientific work, and his credibility. Three members of the NFL’s Mild Traumatic Brain Injury Committee wrote the editor of *Neurosurgery* asking that Omalu’s paper be retracted, claiming serious flaws with the science (Casson, Pellman & Viano 2006a). A war of words ensued in the letters pages of *Neurosurgery*, with Omalu and colleagues going head to head with Casson and colleagues (Casson, Pellman, and Viano 2006a and 2006b; Omalu et al 2006a). The journal declined to retract Omalu’s paper, and proceeded to publish his second CTE case study, “Chronic Traumatic Encephalopathy in a National Football League Player: Part II” (Omalu et al 2006b), again examining a former football player, Terry Long who played eight seasons with the Pittsburgh Steelers.

**Neuropathology of CTE**

CTE is marked by patchy distributions of Tau neurofibrillary tangles (NFTs) in the brain. The location of Tau in CTE corresponds to areas exposed to direct trauma, such as blows to the top and side of the head in football players, and the front of the head in boxers. Not surprisingly, clinical symptoms also apparently correspond to the locations of brain trauma. For example, slurred speech and balance problems (commonly seen in former boxers, giving rise to the term “punch drunk”) are associated with cerebellar scarring and nerve loss, while altered affect and memory are seen in individuals with frontal and temporal lobe damage, located at the front and sides of the head, respectively (Chin, Toshkezi & Cantu 2011). The precise pathological process that initiates Tau deposition remains unknown, but the delay in the appearance of symptoms suggests that the
process continues and the disease progresses even long after athletes cease playing. Microscopically, brains with CTE are unlike brains with other neurodegenerative diseases such as Alzheimer’s dementia. In advanced CTE, there are several typical gross pathological characteristics including reduced brain weight, enlargement of the ventricles, and cerebral atrophy (McKee et al 2016).

There are no validated tests that can confirm CTE in a living individual, although ongoing research is focused on discovering diagnostic modalities, including brain imaging and biomarkers. McKee and colleagues recently published the first validated neuropathological criteria for diagnosing and staging CTE postmortem (McKee et al 2016). The four stages of CTE are associated with different neuropathological characteristics and progressive, accumulating clinical symptoms. In Stage I, Tau NFTs are localized, and clinical symptoms are nonspecific, including headache and loss of attention and concentration. In Stage II there is progression of NFTs, and more severe clinical and psychiatric symptoms, including depression and mood swings, disinhibition, impulsivity, explosivity, loss of attention and concentration, headache, and short-term memory loss. In Stage III, macroscopic neuropathological changes such as cerebral atrophy are seen, with cognitive impairment and memory loss, executive dysfunction, and visuospatial abnormalities. The symptoms of CTE accumulate and worsen, and individuals with Stage IV CTE are demented, with profound short-term memory loss, frequently exhibiting paranoia, in addition to the symptoms that develop in earlier stages of the disease (McKee et al 2013).

The actual incidence of CTE in athletic or other populations remains unknown. Researchers at Boston University and the Concussion Legacy Foundation (concussionfoundation.org), the research institution co-founded by Omalu and former wrestler Chris Nowinski, have identified CTE in 96 percent of the brains of former NFL football players examined, and in 79 percent of the brains from individuals who played football professionally, semi-professionally, in college, or in high school (Breslow 2015). While that is an astonishingly high number, there is selection bias in the sample since CTE can only be diagnosed postmortem, by examining brain tissue. Few people donate their brains for study after death. When brains are donated, it is often because the deceased exhibited clinical symptoms while alive. Thus, the actual incidence in both professional athletes and the general population is at this time unknown.
Clinical symptoms

One of the film’s most valuable contributions is not its heroic narrative of Omalu’s war with the NFL, but rather its brief but effective, and often moving dramatization of the emotional and psychological toll of CTE on those afflicted. Owing to the current inability to diagnose the condition in living subjects, CTE case studies to date have been retrospective, utilizing medical records and verbal autopsies from survivors (Stern et al 2013; McKee et al 2016). The progressive toll of CTE is devastating and distressing for both the afflicted individuals, and for their families and caregivers. The clinical symptoms of CTE are numerous and varied, and manifest as combinations of disordered memory and executive functioning, mood disorders, motor neuron disease, suicidality, substance abuse, violent and erratic behavior, impulsivity, personality and behavior changes, poor judgment (especially concerning financial matters), early dementia, and premature death, all of which significantly impact the quality of life of affected individuals and those close to them (Stern et al 2013). Problematically, the clinical symptoms of CTE are nonspecific and overlap with several other neurological disorders, including post-concussion syndrome, Alzheimer’s Disease, frontotemporal dementia, Parkinson’s Disease, Lewy Body Dementia, Amyotrophic Lateral Sclerosis, and Post-Traumatic Stress Disorder. Clinical symptoms in early stages overlap significantly with symptoms typical in concussion and blast concussive injuries, post-concussion syndrome, post-traumatic stress disorder (PTSD), and psychiatric illness. A further complication is that the populations affected by concussion, blast injury, CTE, and PTSD – athletes and military personnel – also frequently overlap (McKee et al 2013). The non-specificity of CTE symptoms complicates efforts to develop a confirmatory, symptom-based clinical diagnosis, particularly when co-morbidity with other conditions is possible, and in some populations, probable (Johnson, in press). The lack of clinical diagnosis can hamper efforts by those affected to obtain disability benefits and to understand the cause of symptoms that can significantly disrupt their lives and impair their ability to function. Both of these problems are depicted in Concussion, when the film turns from Omalu’s travails to those experienced by retired football players engaged in their own battles, including Webster’s struggle to understand and self-manage his devastating and debilitating symptoms, and the struggles of other retired players to obtain disability benefits during the NFL’s long period of CTE-denial (Fainaru & Fainaru-Wada 2012; Fenno 2016).

Two distinct phenotypic presentations of CTE have been identified among a small sample (n=33) of neuropathologically confirmed cases (Stern et al 2013): a behavioral/mood subgroup, and
a cognitive subgroup. The behavioral/mood subgroup initially exhibits behavioral/mood disturbance, including explosivity and physical/verbal violence, with minimal cognitive and motor symptoms. The behavioral/mood group are also younger at onset of symptoms, and die earlier. The cognition subgroup is older at onset of symptoms, demonstrates cognitive impairments such as memory loss and executive dysfunction, and is more likely to progress to dementia than the other subgroup. A third, smaller subgroup (n=3) is asymptomatic at the time of death; all have early stage I or II disease.

The known case studies of athletes in the behavioral/mood subgroup include individuals like Chris Henry, a football player for the Cincinnati Bengals. Henry died at the age of 26, after sustaining a traumatic brain injury in a fall from a moving vehicle. He was the first active NFL player to be diagnosed with CTE. Notably, he had no documented history of concussion during his college and professional football careers. Also notable is that he had frequently been in trouble with the law, for criminal behavior and substance abuse, and was known as a “troublemaker” in the five years before his death. His family and friends, however, described him as quiet and introverted by nature (Roth 2013). There has long been debate about the supposed tendency towards bad behavior among professional athletes, and whether a culture of violence and entitlement in some sports creates criminals. Cases like Henry’s, however, give rise to disquieting questions about the possibility that repetitive brain injuries, some of which are simply unavoidable in certain sports, might be partly or wholly to blame, particularly when clinical symptoms like explosivity, impulsivity, and aggression are common in the behavioral/mood CTE subgroup.

There would be important legal implications if CTE can result in affected individuals not being fully in control of, or responsible for their behaviors. Indeed, there is research suggesting that neurological disease is a mitigating factor in some criminal behavior (Sfera et al 2014; Mendez 2010). For example, behavioral variant fronto-temporal dementia (bvFTD) is associated with antisocial and criminal behavior (Mendez 2010). Legally, individuals with bvFTD are often deemed competent to stand trial for criminal behavior, and are able to appreciate the wrongfulness of their actions, but might nonetheless be “organically incapable of regulating their behavior” (Sfera et al 2014:3). This is a particularly vexing problem when criminal law (as in the United States and Canada) predicates culpability not on the ability to control one’s behavior, but rather on the ability to appreciate the moral and/or legal wrongness of one’s actions (Sfera et al 2014).
Looking forward

In his battle with the NFL, Omalu was eventually vindicated. In March 2016, NFL executive vice president Jeff Miller publicly admitted to the worst kept secret in sports: that there is a link between playing professional football, and the development of CTE. Since his 2005 paper, Omalu has published many more, including case studies of athletes in other sports, and combat veterans (Omalu et al 2010a; Omalu et al 2011), as well as studies of the alarming number of suicides and parasuicides among individuals with CTE (Omalu et al 2010b). In the years since Omalu “rediscovered” CTE, there has been significant growth in scientific interest and research into concussions and CTE, and growing public concern about sport-related neurotrauma. However, research on CTE is still in its infancy, and a number of important questions remain unanswered, including questions about the actual incidence of the disease, the neuropathological mechanism(s) that initiate Tau deposition, why some athletes appear unaffected by the disease, as well as possibilities for diagnosis, treatment, and prevention. The gaps in knowledge about CTE have significant implications across a range of social, legal, and ethical domains. To date, much interest has been directed at the impact of CTE on professional athletes, who are an extremely small and atypical subset of the population affected by repetitive neurotrauma. Sport-related concussion is already recognized as a significant public health concern (CDC 2016; Canada 2016), but evidence suggests CTE is caused not just by concussions but also by routine, subclinical neurotrauma. With millions of children and teenagers worldwide playing neurotraumatic sports, including football, hockey, soccer, rugby, and lacrosse, the CTE problem may be orders of magnitude larger than the concussion problem, given the potential size of the affected population. As yet, there have been no large, longitudinal, epidemiological studies that might help determine the prevalence and real burden of CTE, so the full scope of the problem remains unknown.

The development of a symptom-based clinical diagnosis of CTE, similar to the clinical diagnosis of Alzheimer’s Disease, would be an important advance, potentially leading to more opportunities for prospective studies, therapeutic research, and enhancement of the physical and psychological well-being of affected individuals. Omalu’s postmortem diagnosis of Webster was the result of a chance encounter between a broken, defeated hero who, in his final moments of life, could not get the help he needed, and an unlikely champion of the dead, a man who had never seen a football game, but who claims that speaking for the departed is his calling. It came too little, too late for “Iron Mike,” just as it has for untold numbers of affected individuals.
CTE may, as it turns out, be an unavoidable hazard of the job for some, including professional athletes and military combat personnel. For them, finding effective treatments for the devastating symptoms of CTE, destigmatizing the neuropsychological symptoms, and acknowledging CTE as a compensable disability could provide improved quality of life. For others, however, including youth athletes, preventing sport-related neurotrauma is currently the best (and only) medicine (Johnson 2012; Johnson 2011), and will undoubtedly continue to be, even as the science of concussion and CTE progresses. Omalu himself has come to that conclusion (Omalu 2015), a realization Concussion hints at in the film’s final minutes, as Omalu watches children playing football, in a scene evocatively set at twilight to signal, perhaps, that this is a time of transition for sports, for athletes, and for the societies that worship both.

Two worthwhile documentaries are recommended for those interested in a less dramatically embroidered look at the problem of sport-related neurotrauma. Omalu’s work, and the subsequent fallout, are already dramatic enough, as seen in League of Denial: The NFL’s Concussion Crisis (Aronson & Kirk 2013), in which Omalu himself appears. Head Games features former wrestler-turned-concussion-crusader Chris Nowinski, and considers the impact of sport-related neurotrauma in both professional and youth football.

References

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