As many as 4 million athletes in the United States experience mild traumatic brain injury (TBI) each year, 300,000 of whom are diagnosed with the clinical syndrome of concussion.1 This estimate might account for a majority of identified concussions, with up to 92% going unrecognized and untreated.2 Defined by its absence of any structural injury on conventional neuroimaging, concussion is caused by an impulsive force to the cranium resulting in a constellation of neuropsychiatric manifestations including headaches, dysequilibrium, cognitive slowing, and fatigue.2 While many concussions do not produce clinically evident sequelae, an estimated 5.3 million Americans currently have TBI-related disability.1

HISTORY OF CHRONIC TRAUMATIC ENCEPHALOPATHY Given the various definitions of concussion and its related disease processes, such as mild TBI or chronic traumatic encephalopathy (CTE), it is nearly impossible to completely elucidate the history of CTE in an article of this length. The reviewer is referred to the astute summary by McCrory and Berkovic3 for a more comprehensive summary. In brief, the earliest known documentation of a clinical syndrome ultimately representing concussion dates back to the 10th century CE. A Persian physician known as Rhazes, perhaps one of the earliest neurologists, differentiated this transient clinical syndrome from severe TBI in his landmark text, Liber Continens.3 The chronic syndrome of dysequilibrium, gait instability, mental confusion, and eventual parkinsonism with significant cognitive deterioration would later be described among prize fighters in the early 20th century.4 This “punch-drunk” state is now clinically referred to as CTE and is thought to affect not only boxers but other athletes such as players of American football. Current research indicates this syndrome is characterized by 1 of 2 major clinical presentations: a behavior/mood variant characterized by bouts of extreme irritability and depression, and a cognitive variant that nearly always follows the behavior/mood variant.5

Over the last 30 years, world-class athletes and American football stars such as Al Toon (New York Jets) and Merrill Hoge (Pittsburgh Steelers, Chicago Bears) have been forced into early retirement (1992 and 1994, respectively) for reasons thought to be related to postconcussive syndrome.6 Experiencing cognitive and psychiatric disturbances, notably headaches, depression, and chronic fatigue, they were unable to continue participating in the highly revered American pastime. These and other symptoms are now recognized and termed postconcussive syndrome (PCS).7

In light of the chronic cognitive changes faced by these and other professional football retirees, then National Football League (NFL) Commissioner, Paul Tagliabue, founded the Mild Traumatic Brain Injury Committee in 1994 to galvanize research efforts into TBI and PCS.7 This committee, chaired by Dr. Elliot Pellman (rheumatology), was charged with the aims of collecting data from players and coaching staff, assessing equipment integrity and safety, and synergizing research efforts with equipment production. The safety of NFL players had long been a priority of this organization, and in fact, some of the earliest (and minor) legislative changes concerning equipment safety were implemented in the 1970s.8 That being said, the quality of the NFL’s own research and the standards set for helmet safety have been called into question. (For more than 30 years the goal of helmets was only the prevention of skull fracture!9) But it would take clear and irrefutable scientific evidence before the NFL would recognize the consequences of repeated TBI, without skull fracture or associated intracerebral hemorrhage, and view this condition more seriously.

The work of neuropathologist Omalu et al.10 published in 2005 demonstrated for the first time that CTE was an actual progressive tauopathy. Originally educated in Nigeria and board certified in anatomical, clinical, and forensic pathology and neuropathology, Dr. Omalu was working at the University of Pittsburgh when he published his landmark case report of an autopsy performed on an NFL player who was 12 years retired. The patient had displayed cognitive impairment, parkinsonism, and mood disorder in the years leading up to his death from coronary artery disease. He had had no family history of Alzheimer disease and was found to have an APOE genotype of ε3/ε3. Moreover, he had had no personal history of concussions outside of football. Omalu et al.10

From the Department of Neurology (J.E.S.), Hospital of the University of Pennsylvania, Philadelphia; and Department of Neurology (S.F.W.), Mount Sinai Beth Israel, New York, NY.

Go to Neurology.org for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.
found diffuse neocortical amyloid deposits and neurofibrillary tangles with tau-positive neuritic threads in the absence of atrophy and in the presence of substantia nigra pallor.

In 2006, Omalu et al. presented their second case report of another retired NFL player whose premorbid clinical syndrome was characterized primarily by a profound major depressive disorder leading to suicide. The postmortem showed diffuse tau-positive neurofibrillary tangles, neutrophil threads, and pallor of the substantia nigra in the absence of both atrophy and amyloid plaques.8 Omalu et al. published in 2010 a third and similar autopsy-confirmed case of CTE in a retired NFL player who also had had neuropsychiatric sequelae, using the paper also to demonstrate the role of neuropathic findings in confirming diagnosis for medicolegal purposes.9 While the findings of Omalu et al. were initially met with marked resistance

**Figure** Timeline of events

- **October**: UNITE investigations collaborate to study deceased patients who suffered recurrent head trauma
- **July**: NFHS Concussion Summit Task Force recommends limiting “live” contact in high school football practices
- **July**: Pac-12 college football division limits “live” contact practices
- **September**: NFL donates $30M to NIH for TBI research
- **July**: Ivy League becomes first Division I college football league to limit “live” contact practices
- **August**: New NFL rule prohibits “helmet-first” hits to heads and necks between players
- **December**: Film “Concussion” released in theaters
- **2014**: NFHS Concussion Summit Task Force recommends limiting “live” contact in high school football practices
- **2013**: August: NFL pays settlement of $765M to retired players and families affected by CTE
- **2012**: July: Pac-12 college football division limits “live” contact practices
- **2011**: July: Ivy League becomes first Division I college football league to limit “live” contact practices
- **2010**: September: Owen Thomas (Penn football player) hangs himself; pathologic diagnosis of CTE made
- **2005**: July: First pathologic description of CTE in NFL player by Omalu and colleagues
- **1994**: NFL Committee on Mild Traumatic Brain Injury founded
- **1973**: First safety standards for football helmets established by the National Operating Committee on Standards for Athletic Equipment
- **1937**: “Dementia Pugilistica” coined by Millspaugh
- **1928**: “Punch-drunk” state among prize fighters described by Maritan
- **1915**: October: UNITE investigations collaborate to study deceased patients who suffered recurrent head trauma
- **2015**: December: Film “Concussion” released in theaters

CTE = chronic traumatic encephalopathy; NFHS = National Federation of High Schools; NFL = National Football League; TBI = traumatic brain injury; UNITE = Understanding Neurological Injury and Traumatic Encephalopathy.15 *For a comprehensive list of updated NFL rules pertaining to hits to the head, see reference 8.
from the professional sports community, in 2013, the NFL paid settlements to affected retired players and families in the sum total of $765 million (see the figure for a timeline of major events).

Since Dr. Omalu’s original histopathologic work, there have been several subsequent studies to characterize further the histomorphology of CTE as well as attempts to use PET imaging to identify potential premorbid patterns and provide diagnostic clues in the absence of tissue. The mechanism of the underlying injury is still a subject of much research.

**CONCUSSION (FILM)** Despite the American media’s recent bombardment with stories about the dangers of concussions, screenwriters of the film Concussion (which opened in theaters on December 25, 2015, and was released on video March 28, 2016) still pretend that knowledge of traumatic head injury in football is a foreign concept.

The curtains open to a black screen and sound effects of a football practice with helmets cracking and whistles blowing. For a movie about traumatic head injury in football, more of these sound bytes were expected, but they are few and far between. There is something uniquely visceral about the similarity in sound effect between helmets clashing and bones breaking that can be both exhilarating and deeply disturbing.

In the same manner in which Mos Def portrayed Vivien Thomas in Joseph Sargent’s 2004 historical drama, Something the Lord Made, Will Smith epitomizes humble upbringings and pure intentions as the protagonist Dr. Bennet Omalu in Concussion. An ordinary man with an extraordinary background, Omalu comes upon the case of Mike Webster, a Pittsburgh Hall-of-Famer whose life ends tragically due to the psychological consequences of CTE. Half a dozen other NFL athletes will ultimately take their own lives before the film’s conclusion; however, each of these deaths chronologically followed Webster’s. A striking minority, these former football players probably experienced the extreme-most symptoms of CTE. No attention in the film is afforded to prior NFL players such as Toon or Hoge who fell into early retirement, battled with memory impairment, and coped with long-standing depression, men who likely represent the overwhelming majority of ex-NFL players with CTE. That being said, one could surmise that much of the evidence of earlier cases of CTE is ignored by the film because of a lack of awareness and limited clinical data before Omalu’s histopathologic studies. Now that clinicians, health care personnel, and professional organizations are recognizing the diagnosis and consequence of CTE, this illness is becoming more popularized in the news and other media (including films such as League of Denial), and more aggressive reforms are being demanded.11–13

Focused on the war between brain science and 21st century American football, Concussion delivers a relatively accurate portrayal of the events that followed the first histopathologic description of CTE in an NFL player15; however, the film deliberately ignores the NFL’s first set of safety standards for helmets, established more than 30 years before Omalu’s landmark publication. Painting the NFL as a macabre, deceptive, and dishonorable villain, the film elaborates this multibillion-dollar organization’s indifference, even neglect, toward this so-called safety revolution. (For a more thorough review on the NFL’s denial of postconcussive cognitive disorders, the reader is directed to the summary by Kain.14) Moreover, the film indicates that the NFL’s Mild Traumatic Brain Injury Committee (1994) was completely disregarded by Omalu et al., although this committee would later serve as the channel through which the NFL would allocate funding toward TBI research. Yes, this committee was chaired by a rheumatologist, and yes, the funding was relatively trivial at $1 million, but it represents the NFL’s first major response to losses of players such as Toon and Hoge. We can agree that the NFL has much more it can do to protect players, and one cannot put a price on human health, not even $765 million, but at least the NFL has made some progress in responding to the cultural and medical mandate to protect its participants. In all, the background events described in the film were correctly described, but the lens through which the film was produced was clouded by the bias of the protagonist.

As an above-average film with a compelling story, Concussion is worth the financial investment but not the emotional one. We are certainly not going to feel guilty for enjoying our Sunday afternoon football. The most dissatisfying aspect of the film was its complete omission of the historical safety attempts by the NFL before the 2000s, which undermines the reliability of all elements of the historical context for the film. That being said, Concussion has certainly catalyzed public awareness and action regarding head injury in contact sports. It is fully obvious that research into better safety and therapeutic interventions should be explored if we are to continue enjoying this great American pastime. We look forward to the results of the UNITE study15 and other ongoing investigations. With growing medical16,17 and societal recognition of football-related CTE, especially since release of Concussion,11–13 the NFL may be hard-pressed to continue doing so little for so many.

**AUTHOR CONTRIBUTIONS**
Dr. James E. Steigler: conception of the idea of the manuscript, drafting of the manuscript, and critical revisions to the manuscript for important intellectual content. Dr. Sarah F. Wesley: drafting of the manuscript and critical revisions to the manuscript for important intellectual content.

**STUDY FUNDING**
No targeted funding reported.
REFERENCES

Right Brain: Concussion (film): Review and historical context
James E. Siegler and Sarah Flanagan Wesley
Neurology 2016;87:e155-e158
DOI 10.1212/WNL.0000000000003174

This information is current as of October 3, 2016

Updated Information & Services
including high resolution figures, can be found at:
http://www.neurology.org/content/87/14/e155.full.html

References
This article cites 12 articles, 2 of which you can access for free at:
http://www.neurology.org/content/87/14/e155.full.html#ref-list-1

Subspecialty Collections
This article, along with others on similar topics, appears in the following collection(s):
Brain trauma
http://www.neurology.org/cgi/collection/brain_trauma

Permissions & Licensing
Information about reproducing this article in parts (figures,tables) or in its entirety can be found online at:
http://www.neurology.org/misc/about.xhtml#permissions

Reprints
Information about ordering reprints can be found online:
http://www.neurology.org/misc/addir.xhtml#reprintsus