

REGARDING POLICY IN CHRONIC TRAUMATIC ENCEPHALOPATHY AS A  
TRANSHISTORIC DISORDER

by

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## ABSTRACT

An individual historian can be categorized as belonging to one of two mutually exclusive and exhaustive groups: transhistoricists, those that believe in an object's existence independent of external forces and its ability to remain fundamentally unaffected across time, or culturalists, believing an object's quality or features are dependent on the time and place of its reference. Disease entities have been examined through both perspectives quite fruitfully, expanding the whole of academia's appreciation of the relationship between disease and history. However, chronic traumatic encephalopathy, or CTE, has recently been embroiled in a nationwide National Football League scandal wherein the livelihood of many affected retired players depends on the court's decision in the accompanying tort case to deal out appropriate justice. The nosological understanding of CTE is crucial in the debate – to include all affected players, despite dramatic revisions in our understanding of CTE as a disease across the 20<sup>th</sup>-century, all parties must recognize CTE, originating in a 1928 case study as “punch drunk syndrome,” as a timeless entity that has undergone progressive iterations in categorization. In this instance, the culturalist perspective would render the disease's history sufficiently fragmented and prevent a cohesive narrative that includes all manner of diagnostic varieties. Even if antithetical to the present state of the humanities, the transhistoric approach is the only satisfactory perspective to uphold justice in the case of suffering football players.

## CHAPTER ONE

## INTRODUCTION

In February 2011, Dave Duerson, a former player in the National Football League, died of a self-inflicted gunshot wound to the chest.<sup>1</sup> Having played eleven seasons of football, Duerson displayed remarkable insight into the emotional and psychological battle being waged inside his mind, heartbreakingly evidenced by his suicide note in which he asked to donate his brain to science.<sup>2</sup> The combination of Duerson reportedly suffering from “headaches, blurred vision, and a deteriorating memory,” the method of suicide that preserved his brain, and the note pleading, “Please, see that my brain is given to the N.F.L.’s brain bank” indicate that Duerson was acutely aware of a sinister evil corrupting his mental faculties.<sup>3</sup>

Duerson played as a member of the defense, in a position commonly referred to as a “safety.” The safety’s responsibility is to sit behind the line of scrimmage and watch as the play unfolds. Should the offense manage to get the ball past the defensive line, the safety acts as a fast-moving and hard-hitting obstacle to prevent further movement. Plays involving the safety position are often the most vicious and hardest hitting interactions to be found in the sport.

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<sup>1</sup>. Ryan Jaslow, “Dave Duerson Suicide: Was Brain Disease to Blame,” last modified May 2, 2011, <https://www.cbsnews.com/news/dave-duerson-suicide-was-brain-disease-to-blame/8>

<sup>2</sup>. Jaslow, “Dave Duerson Suicide.”

<sup>3</sup>. Alan Schwarz, “Duerson’s Brain Trauma Diagnosed,” last modified May 2, 2011, <https://www.nytimes.com/2011/05/03/sports/football/03duerson.html>

Upon the posthumous inspection of his brain, researchers found “moderately advanced” signs of chronic traumatic encephalopathy, or CTE.<sup>4</sup> Dr. Ann McKee of the Boston University’s Center for Traumatic Encephalopathy, a prominent medical researcher in the ongoing debate of the dangers of head trauma in the NFL, argued that it was likely “that if [Duerson] hadn’t had the CTE, he wouldn’t have developed those symptoms that he was experiencing at the end of his life and perhaps he wouldn’t have been compelled to end his life.”<sup>5</sup> Other researchers agree.<sup>6</sup> There is a growing consensus linking the physicality of football, CTE as a disease, and the emotional and physical manifestations of the disease suffered by the players.

Dr. Ann McKee has presided over several high-profile cases of suspected CTE. For example, Tyler Sash, a former New York Giants player, was found dead in his house after having overdosed on a combination of methadone and hydrocodone.<sup>7</sup> McKee’s examination revealed that on a scale of 0 to 4 (the traditional scale used to rate the severity of CTE), Sash’s CTE was a 2.<sup>8</sup> While Sash’s CTE could have technically been more pronounced, he was only twenty-five years old. This debilitating disease had infiltrated and corrupted the mind of an otherwise healthy individual. Had the overdose not occurred, Sash would have had very few years before the disease completely incapacitated him.

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<sup>4</sup>. Jaslow, “Dave Duerson Suicide.”

<sup>5</sup>. Jaslow, “Dave Duerson Suicide.”

<sup>6</sup>. Many contemporary medical researchers have weighed in on this topic, including: Ann McKee, Breton M. Asken, Sharon B. Shively, Carrie Esopenko, and Kimbra Kenney.

<sup>7</sup>. Jared Dubin, “Former Giants Safety Tyler Sash Found to Have Advanced Stages of CTE,” last modified January 27, 2016, <https://www.cbssports.com/nfl/news/former-giants-safety-tyler-sash-found-to-have-advanced-stages-of-cte/>

<sup>8</sup>. Dubin, “Former Giants Safety.”

One of the more poignant examples of violent acts related to brain trauma was that of Jovan Belcher. Belcher was a Kansas City Chiefs linebacker, again, only twenty-five years old, who shot and killed his girlfriend at home (leaving their three-month-old baby behind), drove to the Chiefs' training facility, and then killed himself in front of team officials.<sup>9</sup> Reportedly, Belcher “thanked his bosses for trying to help him fix his ailing relationship with his girlfriend” and asked the officials to “take care of [his] baby”<sup>10</sup> before firing a bullet into his brain. Belcher’s body was exhumed a year after his death and was found to be littered with “neurofibrillary tangles of tau protein,” which is indicative of repeated brain trauma over an extended period. In particular, Belcher’s signs of CTE were localized around the hippocampus, “an area of the brain involved with memory, learning, and emotion.”<sup>11</sup>

The emergence (or, more likely, the resurgence) of a number of high-profile cases regarding football players and CTE in the NFL has not been analyzed by historians. It is a relatively recent topic and the consequences of related events are still unfolding. This project is an attempt to preemptively frame subsequent discussions of the history of NFL related brain trauma within the framework of the transhistoric nature of disease and illness.

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<sup>9</sup>. Steve Delsohn, “OTL: Belcher’s Brain had CTE Signs,” last modified September 30, 2014, [http://www.espn.com/espn/otl/story/\\_/id/11612386/jovan-belcher-brain-showed-signs-cte-doctor-says-report](http://www.espn.com/espn/otl/story/_/id/11612386/jovan-belcher-brain-showed-signs-cte-doctor-says-report)

<sup>10</sup>. Lindsey H. Jones, “Belcher to Chiefs: I have hurt my girl; I can’t go back,” last modified June 18, 2013, <https://www.usatoday.com/story/sports/nfl/chiefs/2012/12/18/jovan-belcher-kansas-city-chiefs-kassandra-perkins-murder-suicide/1777359/>

<sup>11</sup>. Delsohn, “OTL: Belcher’s.”

Broadly speaking, historians have conceptualized diseases such as CTE in two ways. The first, with some trepidation, may be considered what I will term a “culturalist account.” This position emphasizes the centrality of culture in defining many significant aspects of who and what we as humans are. The circumstances specific to a given, and temporary, understanding of a disease from the culturalist perspective dictate the necessary and sufficient conditions of that disease to the human body. For instance, the specifics of diseases like chronic traumatic encephalopathy, post-traumatic stress disorder, and the human immunodeficiency virus are contingent and reflect the cultural or societal values at the time that the disease was diagnosed. Implicit in this position is an understanding and acceptance that those ostensibly essential characteristics of disease created by the culture will change in response to changes to the culture.

Accompanying the belief in the primacy of change as an identifying feature of disease is, in some cases, a complete refutation of the disease category’s state of existence. For example, culturalists may share an overlapping belief in the philosophical standpoint of anti-realism that, although there are many varieties, disagree with either the existence or independence of an object’s (in this case the disease-as-object) relation to our perception of it. It seems likely, if not probable, that a culturalist would claim that a disease is culturally constructed in such a manner that it either does not exist outside of the cultural construction or, at least, is not independent of it.

The second rough collection of methodologies that pertain to the historical understanding of disease holds that the latent entity under consideration manifests entirely within the biology of humans. Borrowing from the work of Richard J. McNally,

a Harvard psychologist studying anxiety disorders, a disease (in McNally's case PTSD) is understood as a "transhistoric entity" or "a timeless, psychological entity, a natural kind discovered by astute clinicians."<sup>12</sup> While McNally himself does not necessarily consider the term applicable to PTSD, the transhistoric nature of any disease is the implicit stance championed by most studying the biological nature of the human body. The "transhistoric account" argues for a materialist understanding of the human body with varying shades of progressivism and biological determinism. A transhistoric object is one that exists apart from our knowledge of it and has properties that are independent above and beyond our linguistic, philosophical, or conceptual perceptions of the object.

While each of these approaches to the historical understandings of disease have their own merits, the transhistoric account is inextricably enmeshed with the judicial system, especially regarding tort cases where specific "scientific" understandings become necessary to advance cases of wronged parties. It is important to ask whether the historical segmentation on behalf of the culturalist account would collapse the legal and scientific foundations that enable the pursuit of justice. The 2017 National Football League's tort cases involving CTE afflicted players are dependent on ninety-years' worth of historical, scientific progressivism that holds CTE as a disease within the transhistoric account. Adopting many varieties of the culturalist account, and criticizing the transhistoric account, may enable colorful, interesting, and corrective histories to be brought forth, but with the unforeseen consequence of collapsing the consistency and internal logic of related tort cases and their intended justice.

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<sup>12</sup>. Richard J. McNally, "Is PTSD a Transhistorical Phenomenon," in *Culture and PTSD*, ed. Devon Hinton (Philadelphia: University of Pennsylvania Press, 2015), 117.

In January 2010, Dr. Ira Casson, a “co-chairman of the NFL’s panel on head injuries,” claimed that, “There is not enough valid, reliable or objective scientific evidence at present to determine whether or not repeat head impacts in professional football result in long-term brain damage.”<sup>13</sup> Less than a year later, the league introduced new player policies aimed at reducing the number of head injuries, including a “ban on helmet-to-helmet hits,” and the inclusion of a mandatory poster in all teams’ locker rooms.<sup>14</sup> The poster entitled “CONCUSSION: A Must Read for NFL Players” outlines the facts, symptoms, and diagnostic avenues to pursue in the case of a suspected concussion. This rapid change in the league’s approach to head injuries likely followed from the realization that their lackadaisical, if not outright reprehensible, attitude towards their players’ health was catching up to them in the public and legal spheres.

The players and their families had been told by league officials that repeated head injuries throughout their athletic career would not culminate in any serious or deadly disease. The league’s official stance on the seriousness of repetitive brain injuries, to the anger of more than 4,500 plaintiffs seeking answers and compensation for either their own or loved one’s illness, flew in the face of the compounding scientific articles and evidence to the contrary.<sup>15</sup> The plaintiffs and their lawyers argued that the NFL knowingly deceived the public regarding the dangers of repetitive brain injuries. While Dr. Casson may point to a lack of evidence, even a cursory dive into the medical

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<sup>13</sup>. Associated Press, “Neurologist Denies Concussion-Disease Link,” last modified January 5, 2010, <https://www.cbsnews.com/news/neurologist-denies-concussion-disease-link/>

<sup>14</sup>. Paolo Bandini, “NFL Concussion Lawsuits Explained,” last modified August 29, 2013, <https://www.theguardian.com/sport/2013/aug/29/nfl-concussions-lawsuit-explained>

<sup>15</sup>. Bandini, “NFL Concussion.”

literature reveals nearly a century of case studies, experiments, and developments in the science of the brain that convincingly link brain trauma to long-term effects.

My argument will progress as follows. First, I will analyze the state that CTE currently holds within the medical literature to establish how contemporary medical researchers treat the disease. Second, survey how historians of medicine have treated the transhistoric nature (or lack thereof) of related, but ultimately distinct, instances of diseases in order to establish a precedent for how CTE may be understood historically. Third, starting in 1928, I will outline the distinctly transhistoric pedigree of CTE within the medical literature as researchers have revolutionized and subsequently developed previous conceptualizations of a latent biological entity. Finally, I will argue that, regarding the culturalist and transhistoric approaches, only the latter allows for a transmission of stable, even if inherently erroneous, information across time to create a foundation on which judicial justice can be structured.

Understanding the complex and multilayered concepts surrounding CTE can often be an uphill battle. From either the sciences or the humanities, the populations that suffer from the symptoms and the symptoms themselves often defy straightforward, matter-of-fact categorizations. There are only two necessary conditions to be met to encounter the debilitating symptoms of CTE: have a brain and subject that brain to abrupt changes in velocity. While the focus of this project is on athletes to provide an important case study arguing for or against certain historical methodologies regarding tort law, many sufferers of CTE include, among others, victims of domestic abuse and war participants.

In “Traumatic Brain Injury, Shell Shock, and Posttraumatic Stress Disorder in the Military – Past, Present, and Future,” (2012) Shively et al. state that, “15% to 20% of service members returning from Operation Iraqi Freedom experienced at least 1 TBI [traumatic brain injury].”<sup>16</sup> 75% of those TBIs are classified as “mTBIs,” or concussions.<sup>17</sup> Additionally, they speculate that the “protection of modern body armor and helmets” has shifted the disease outlook resulting from “explosive exposure” of servicemembers (including men and women) from probable death to “multiple exposures to high explosive with subsequent nonfatal brain injuries.”<sup>18</sup> The causal link between military service and repeated head injuries is only compounded through “repeated deployments.”<sup>19</sup>

In addition to the dangers faced by servicemembers, even a cursory glance at the medical literature reveals a population that has been alarmingly left out of the conversation. Survivors of domestic abuse or those suffering from intimate partner violence (IPV) are, in hindsight, some of the individuals most vulnerable to the conditions of CTE and related diseases. Often referred to as “invisible scars,” traumatic brain injuries defy the “stereotypical image of a domestic violence victim” as suffering from a “swollen, black eye” or “trying to cover up her bruises.”<sup>20</sup> The statistics, limited though they may be, are staggering: “one in three women and one in four men have been

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<sup>16</sup>. Sharon B. Shively et al., “Traumatic Brain Injury, Shell Shock, and Posttraumatic Stress Disorder in the Military – Past, Present, and Future,” *Journal of Head Trauma Rehabilitation* 27, no. 3 (2012): 234.

<sup>17</sup>. Shively, 234.

<sup>18</sup>. Shively, 234.

<sup>19</sup>. Shively, 234.

<sup>20</sup>. Heather Mongilio, “Invisible Scars: How Domestic Violence Victims Have Been Left Out of the Discussion on Traumatic Brain Injuries,” (Master’s thesis, American University, 2018): 3.

physical abused by an intimate partner at some point in their lives.”<sup>21</sup> In addition, “estimates of more than 50%-75% of abused women have been shown to sustain *repetitive* TBIs.”<sup>22</sup>

The severity of TBIs in this population is multiplied when considering that, historically, the set of symptoms reported by “women subjected to partner violence” has been “attributed to the stress of being in an abusive situation rather than the potential sequelae of TBI.”<sup>23</sup> In other words, the emotional and behavioral disturbances reported by sufferers of domestic violence is often dismissed as symptomatic of the abusive relationship and seldom linked to an established disease caused by the violence itself (perhaps indicative of a tendency to victim-blame survivors of domestic abuse). Moreover, though abused populations may suffer repetitive TBIs, medical attention is almost never sought for mild TBIs (mTBIs or “concussions”) despite being categorized within the larger designation of TBI.

While a focus on boxers and football athletes may suggest a masculine locality of this disease, the reality of CTE is far more complicated. CTE is not strictly a sport-related disease (despite the focus of this project). With these various reservations and caveats in mind, it is important to understand how medical and scientific fields currently understand the nature of CTE.

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<sup>21</sup>. G. Zieman et al., “Traumatic Brain Injury in Domestic Violence Victims: A Retrospective Study at the Barrow Neurological Institute,” *Journal of Neurotrauma* 34, no. 4 (2017): 876.

<sup>22</sup>. Eve M. Valera, “Increasing Our Understanding of an Overlooked Public Health Epidemic: Traumatic Brain Injuries in Women Subjected to Intimate Partner Violence,” *Journal of Women’s Health* 27, no. 6 (2018): 736.

<sup>23</sup>. Valera, 736.

Chronic traumatic encephalopathy is a “progressive neurodegenerative disease”<sup>24</sup> caused by protein inclusions thought to be brought on by exposure to chronic inflammation of brain tissue. These inclusions (referred to as neurofibrillary tangles [NFT] in the medical literature) are the result of a traumatic brain injury (TBI) disrupting the normal, stabilization function of proteins, in particular Tau proteins, causing a disconnect between the protein and corresponding “microtubule fibrils within axons.”<sup>25</sup> This disconnect provides the opportunity for proteins to cluster into the NFTs and inhibit the normal functioning of the brain. The size and location of NFTs may vary greatly – indeed, it is suspected that the outwardly manifested symptoms of CTE (such as emotional irregularity, depression, etc.) depend significantly on the location of NFTs within an individual’s brain.

There is a debate, however, within the medical community as to just how progressive the disease may be. The required amount of repetitive brain trauma (RBT) for the necessary conditions for CTE to gain serious diagnostic credibility is unclear.<sup>26</sup> Additionally, CTE (along with Alzheimer’s disease) cannot be definitively diagnosed prior to an autopsy. This culminates in a potent and insidious disease that, frustratingly, can only be posthumously confirmed even given the multi-faceted set of symptoms that, in hindsight, make it glaringly obvious. Moreover, even if technology provides a method of detecting CTE without a brain biopsy in the future, it is perhaps little consolation for a disease that remains untreatable.

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<sup>24</sup>. Breton M. Asken et al., “Research Gaps and Controversies in Chronic Traumatic Encephalopathy: A Review,” *JAMA Neurology* 74, no. 10 (2017):1257.

<sup>25</sup>. Asken, 1257.

<sup>26</sup>. Asken, 1257.

The recent attention paid to CTE-afflicted football players indicates a trend within the medical community and, perhaps, the audience that consumes football-related media. CTE, however, has not remained a static diagnostic category. Ann McKee, the researcher from Boston University's Center for Traumatic Encephalopathy, wrote that "repetitive brain trauma" has been known to cause "progressive neurological deterioration" since at least the 1920s (originating in the boxing community).<sup>27</sup> CTE was previously known, according to McKee, as *dementia pugilistica* (inspired by the Latin *pugil* meaning "boxer"). Furthermore, CTE itself is a specific condition found within the broader scope of TBIs that encapsulate an extensive range of potential diseases and disorders relating to the lasting trauma associated with physical impacts to the head and corresponding brain injuries.

CTE's treatment in the medical literature has an immediate and lasting impact on the players in the NFL. Before laying bare the history of this disease, it is pertinent to examine this treatment. The transhistoric understanding of CTE is the culmination of nearly a century's worth of work and analysis on a countless number of individuals who suffer and have suffered from lasting symptoms due to irreparable damage to the brain. McKee examined several cases of CTE and made an immediate link between *dementia pugilistica* and *chronic traumatic encephalopathy* as being two variants on a hidden, but ultimately knowable, biologically latent entity.

McKee and others assume that repeated physical impacts on the head, regardless of intensity, affect the brain; historical context may be interesting but is largely irrelevant

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<sup>27</sup>. Ann McKee et al., "Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy After Repetitive Head Injury," *Journal of Neuropathy and Experimental Neurology* 68, no. 7 (2009): 709.

regarding how one ought to understand an illness. For example, McKee states, “Neuropathologically, CTE is characterized by atrophy of the cerebral hemispheres, medial temporal lobe, thalamus, mammillary bodies, and brainstem, with ventricular dilatation and a fenestrated cavum septum pellucidum.”<sup>28</sup> McKee’s inherent, fundamental message is that biological facts inevitably determine a particular outcome. The boxer in 1920 and the football player in 2017, to medical professionals like McKee, are suffering from the same disease. The difference in diagnosing and treating this “same disease” only differs in virtue of the technology, education, and expertise available to the medical personnel (all of which may progressively improve over time).

This transhistoric assumption is evident not only in the medical literature, but also in recent litigation involving the work of a number of researchers, the coverup by the NFL, and the suffering, deaths, and suicides of football players spanning decades. The plaintiffs (all NFL football players who retired prior to July 7<sup>th</sup>, 2014) alleged that the “NFL Parties” were “aware of the evidence and risks associated with repetitive traumatic brain injuries” but failed to “warn and protect players against those long-term risks and ignoring and concealing this information from players.”<sup>29</sup> The case ended in a “No Fault” plea by the NFL which allows due payment without direct incrimination. Players (or their families) now receive fixed compensation corresponding to diagnoses of certain diseases by a board-certified physician up to the year of the decision (2017). For example, amyotrophic lateral sclerosis (ALS or Lou Gehrig’s disease) awards a \$5 million

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<sup>28</sup>. McKee, “Chronic Traumatic Encephalopathy in Athletes,” 709.

<sup>29</sup>. “Official NFL Concussion Settlement.” Accessed October 15, 2018.  
<https://www.nflconcussionsettlement.com/FAQDetails.aspx?q=154#154>

settlement, CTE \$4 million (although this would be awarded to the family of a player due to the difficult nature of confirming CTE in a living patient), and \$3.5 million for a diagnosis of Parkinson's or Alzheimer's.

The scientific understanding of disease referenced in the court proceedings and settlement is distinctly transhistoric. This transhistoric perspective operates under a very specific assumption of how objects across time relate to each other. They assume a progressive, additive, and linear position separated by relatively great distances. For example, in the "Injury Definitions" document provided to stipulate the terms of the case, one qualifying diagnosis for a monetary award is "evidence of a moderate to severe cognitive decline" "as determined by and in accordance with the standardized neuropsychological testing protocol."<sup>30</sup> Or, for a specific disease like CTE, a "post-mortem diagnosis" "made by a board-certified neuropathologist" is necessary.<sup>31</sup> These outlines for disease diagnosis operate squarely within the transhistoric approach: a biological truth is nestled in the brains of the afflicted players that, using McNally's words, can be uncovered by astute clinicians.

How should historians view McKee's apparent lack of historical nuance concerning CTE? Is it right to consider the historic events surrounding the understanding of CTE as mere blips on an upward-trending chart of inevitable scientific progress? To be certain, this is the perspective that is often taken by those who hold dearly to the fact-finding enterprise of science that is only inhibited by time and circumstance – science

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<sup>30</sup>. "Reference Guides." Accessed October 15, 2017.  
[https://www.nflconcussionsettlement.com/Reference\\_Guides.aspx](https://www.nflconcussionsettlement.com/Reference_Guides.aspx)

<sup>31</sup>. "Reference Guides." Accessed October 15, 2017.  
[https://www.nflconcussionsettlement.com/Reference\\_Guides.aspx](https://www.nflconcussionsettlement.com/Reference_Guides.aspx)

forever trudges forward due to the inextricable relationship between the scientific method and truth. Truth is continually crystallizing, through science, into beautiful structures that shed light on otherwise darkened aspects of our systems of knowledge. Medical researchers during the 1920s were on to “something,” and that something is far more pronounced and sophisticated in the current era and the cycle of sophistication will continue indefinitely.

This tongue-in-cheek depiction of scientific disciplines would likely offend both parties across the STEM divide but is meant to crudely preface an ongoing disagreement between the sciences and humanities on how to perceive objects across time. However, it is important to underscore that this view of the scientific method may be primarily limited to the public perception: many philosophers, historians, sociologists, and even scientists themselves acknowledge the complications involved with any truth-finding enterprise. Historian Timothy J. LeCain and other scholars blend the material and cultural understandings of history and reject such a strict dichotomy of perspectives.<sup>32</sup> Regardless, using several stark examples from the culturalist perspective, it becomes clear that both ends of the spectrum are still heavily populated by scholars producing work that often reinforces the divide. The differences between the two approaches can be exemplified using a disease – posttraumatic stress disorder (PTSD) – that has been studied extensively by both sides and may serve as a foil for the historical study of CTE.

The U.S. Department of Veterans Affairs defines PTSD as “a mental health problem that some people develop after experiencing or witnessing a life-threatening

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<sup>32</sup>. Timothy J. LeCain, *The Matter of History: How Things Create the Past* (United Kingdom: Cambridge University Press, 2017).

event, like combat, a natural disaster, a car accident, or sexual assault.”<sup>33</sup> It is usually understood to include the following symptoms: flashbacks, avoidance of certain situations, negative thoughts and feelings, and hyperarousal.<sup>34</sup> Although this set of symptoms and definitions outlined by the Department of Veterans Affairs is vague and generalized, even this generalized understanding does not fully encapsulate, or provide understanding for, all types of manifestations of the disorder. For example, PTSD develops in some individuals as a result of a life-long struggle with certain situations (e.g., abusive family members). This definitional misunderstanding has not gone unnoticed by the American Psychiatric Association (APA), the organization in charge of updating the Diagnostic and Statistical Manual (DSM). The DSM is an “authoritative volume that defines and classifies mental disorders in order to improve diagnoses, treatment, and research”<sup>35</sup> and is currently in its fifth iteration. Each update, as far as PTSD is concerned, tries to better situate the disorder within the larger societal and professional context (including as many varieties of the disorder without being too generalized that it becomes unwieldy). Using the DSM-5 and other information, it becomes possible to create a rough historical trajectory of PTSD as it has existed since the 1980s when it was introduced as a clinical diagnosis. However, this says nothing of diagnoses of proto-disorders that resemble PTSD in practice and whether any historical

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<sup>33</sup>. “What is PTSD,” *U.S. Department of Veterans Affairs*, accessed November 29, 2017. <https://www.ptsd.va.gov/public/PTSD-overview/basics/what-is-ptsd.asp>.

<sup>34</sup>. “What is PTSD,” *U.S. Department of Veterans Affairs*, accessed November 29, 2017.

<sup>35</sup>. “Diagnostic and Statistical Manual of Mental Disorders (DSM-5),” *American Psychiatric Association*, accessed November 29, 2017. <https://www.psychiatry.org/psychiatrists/practice/dsm>

connection exists between PTSD and earlier attempts to define and classify mental disorders that are similar in kind to PTSD.

With a basic idea of how PTSD exists currently, we may come back to the initial goal of examining a disorder from either the transhistorical or culturalist perspective. Concerning PTSD, the transhistoric position claims that as long as humans have been physiologically capable of being affected by traumatic events in adverse ways, we have been susceptible to a condition, a “something,” that is currently defined as “posttraumatic stress disorder.” In addition, the diagnosis of PTSD has developed, or progressed, through a long history of attempts to define and categorize a similar set of symptoms, albeit under different names or designations. “Soldier’s heart” in the American Civil War, “shell-shock” in WWI, “combat fatigue” in WWII, and “posttraumatic stress disorder” in the post-Vietnam War era is one such sequence of “names” that have attempted to categorize a section of the population suffering in localized, particular, and historically similar ways.

In *Culture and PTSD: Trauma in Global and Historical Perspective* (2016), editors Devon Hinton and Bryon Good have compiled a number of essays that illustrate the cultural prevalence and transcultural nature of PTSD. In the chapter “Is PTSD a Transhistorical Phenomenon,” Richard J. McNally discusses two prominent attitudes towards PTSD: is PTSD a transhistoric entity or is it a “socially constructed phenomenon arising in the wake of the Vietnam War?”<sup>36</sup> According to McNally, traumatologists (medical professionals who “study or treat the effects of psychological trauma”)

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<sup>36</sup>. McNally, “Is PTSD a Transhistorical Phenomenon,” 117.

generally favor the former view while historians and anthropologists lean toward the latter.<sup>37</sup> Essentially, the debate falls on a divide between ontological commitments to either nature or culture with regard to how PTSD is able to exist both within the minds and bodies of those afflicted but also, simultaneously, within the social imagination of the surrounding culture. To resolve this dispute, McNally believes, it becomes imperative to show a “biological basis for psychological trauma.”<sup>38</sup>

While *Culture and PTSD* tries to show a “transcultural character” of PTSD, McNally concerns himself with the transhistorical nature of the disorder.<sup>39</sup> How might one begin to show an underlying entity that has existed throughout history albeit under different diagnostic categories? One potential solution to the difficulties in transcending time to create a cohesive narrative is to adopt a realist approach towards disease and illness. In other words, although symptoms and descriptions of an illness are mere attempts to define and categorize, the true purpose of such ventures is to “reflect an underlying, latent entity” that is capable of being held within an objective, empirical understanding.<sup>40</sup>

This approach, as McNally notes, is not without potential problems. If two different historical designations for an entity (e.g. “shell-shock” and PTSD) are indicative of varying attempts by physicians to “point towards” an inherent truth within the biology and psychology of humans, we must be able to account for “wildly different

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<sup>37</sup>. McNally, “Is PTSD a Transhistorical Phenomenon,” 117.

<sup>38</sup>. McNally, 118.

<sup>39</sup>. McNally, 120.

<sup>40</sup>. McNally, 126.

presentations” throughout history.<sup>41</sup> If we cannot reconcile otherwise incompatible presentations of an underlying, transhistoric disorder, for example, how can we justify including all possible manifestations of traumatic experience under a single heading? Are “flashbacks” and “hysteria” similar enough in kind to automatically include them as historic representations of the disease we currently understand as PTSD?

While being convinced of PTSD as a transcultural phenomenon (i.e., seen across cultural boundaries with varying contextual backgrounds), McNally cannot, however, readily ascribe the disorder as simultaneously transhistorical. What prevents McNally, it would seem, from claiming PTSD as a transhistorical disorder is an inability of the symptoms and descriptions to accompany the potential underlying biological truth across time. The set of embodied symptoms in one era, according to McNally, are not indicative of a trend throughout time to another era. If a relationship between the felt experience of an illness or disease and a set historical period cannot hold, the entire enterprise of the transhistorical conception of disease collapses.

The rejection of the transhistoric position on PTSD can be seen more directly in the work of active historians in the field of the history of medicine. This rejection usually results from a relatively small set of justifications. First, transhistoric conceptions of disease often, intentionally or not, assign normative claims to scientific communities separated by large swaths of time (over a century concerning the set of symptoms currently described as “PTSD”). For example, if we assume a teleological progression of medical science, there is some sense that the science being performed by today’s medical

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<sup>41</sup>. McNally, “Is PTSD a Transhistorical Phenomenon,” 126-7.

community is inherently better (more accurate, effective, etc.) than the science in 1915 when “shell-shock” was introduced as a diagnostic category.<sup>42</sup>

Furthermore, scholars such as Tracey Loughran, author of *Shell-Shock and Medical Culture in First World War Britain* (2017), argue that what would otherwise be considered variations on a biological truth (i.e., transhistoric) are simply too varied to be compared across time.<sup>43</sup> Loughran argues that the “shell-shock” diagnosis was created from a number of medical disciplines, certainly not limited to the burgeoning psychological and psychiatric approaches to the human body circa 1915. Gastroenterologists, for example, had medical knowledge to contribute regarding what symptoms should be considered relevant to “shell-shock.”<sup>44</sup> What results is a disorder that, despite prima facie evaluations that resemble certain facets of current PTSD diagnoses, is a separate and distinct entity requiring its own historical consideration.

Loughran wastes very few pages before proclaiming, in absolutist terms, that, “‘Shell-shock’ is not PTSD.”<sup>45</sup> Despite the ostensible similarities between “shell-shock” and PTSD, Loughran’s argument bears some theoretical weight. If one were to accept a direct, historical connection between “shell-shock” and PTSD, Loughran argues, one would also be forced to accept a few unsettling and unforeseen consequences. For example, if PTSD and “shell-shock” are interchangeable (or equivalent disorders), that would mean that a consistent reaction to wartime events has existed across time and

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<sup>42</sup>. Tracey Loughran, *Shell-Shock and Medical Culture in First World War Britain* (Cambridge: University Printing Press, 2017), 10.

<sup>43</sup>. Loughran, 14.

<sup>44</sup>. Loughran, 6.

<sup>45</sup>. Loughran, 14.

cultures, remaining relatively unchanged.<sup>46</sup> Indeed, this perspective would ultimately claim that a set of subjective symptoms have been, with wavering accuracy, ascribed to an otherwise objective disorder. Combatants across time, according to Loughran's depiction of this presentist position, would be susceptible to the same physical and psychological reactions to war-related horrors. This approach, however, significantly interferes with the usefulness of culture as a tool for dissecting events in the past. If an argument cannot account for culture as influencing an individual's reaction to war (e.g., conceptions of masculinity, duty, etc.), perhaps that argument, Loughran and others believe, is fundamentally flawed.

Loughran also argues that the development of "shell-shock" is plainly laid out in the medical literature of the era and, with respect to the objectivity of medical diagnoses, approaching the history of a disease involves tackling important attitudes towards what it means to label a set of symptoms. As Loughran claims, "A medical diagnosis is never a purely objective statement of what happens in the body."<sup>47</sup> Medical diagnoses, for Loughran, involve consensus and disagreement, the creation of knowledge and lack thereof. Due to the considerable number of specialists contributing to the shared wealth of knowledge surrounding "shell-shock," how information is organized and distributed to relevant populations becomes important. A gastroenterologist would have a different approach to diagnosing and treating a soldier with "shell-shock" than a general surgeon. With this in mind, Loughran's treatment of "shell-shock" as an "unstable diagnosis made

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<sup>46</sup>. Loughran, *Shell-Shock and Medical Culture*, 14.

<sup>47</sup>. Loughran, 52.

up of other unstable diagnoses”<sup>48</sup> becomes easier to understand. “Shell-shock,” by virtue of the surrounding medical environment, became an umbrella term, a catchall, for any difficult-to-place diagnoses that inhabited the bodies and minds of people exposed to the war. “Hysteria,” “traumatic neurosis,” “concussion,” “epilepsy,” “insanity,” and “psychosis” were all relevant symptoms that enabled a medical practitioner to label, or diagnose, a soldier with “shell-shock.” The differences, then, between “shell-shock” and PTSD, at least regarding symptoms indicating a potential diagnosis, are sufficient to distinguish each as independent and historically contingent.

Expanding the inquiry beyond the realm of PTSD-related diseases, the transhistoric nature of disease is a problematic issue facing most historians of medicine. Take, for example, Keith Wailoo’s *Drawing Blood: Technology and Disease Identity in Twentieth-Century America* (1997). Wailoo places his analysis directly in the middle of the historical relationship between disease and technology. In particular, diagnostic techniques (such as examining blood in hematology) do not define and categorize disease but stand “in relation to disease phenomena” and affect social policy and the lived experiences of patients and medical practitioners.<sup>49</sup> Wailoo points out how technology (and its advancement) shapes the identity of otherwise inherent biological truths about the human body. From his perspective, the historical, cultural, and social context of a disease and surrounding technology create and maintain identities of disease and those suffering from a given disease. To illustrate the point, Wailoo poses an example in his introductory

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<sup>48</sup>. Loughran, *Shell-Shock and Medical Culture*, 52.

<sup>49</sup>. Keith Wailoo, *Drawing Blood: Technology and Disease Identity in Twentieth-Century America* (Baltimore: Johns Hopkins University Press, 1997), 2.

chapter: in an earlier era, would sufferers of HIV have been hemophiliacs or IV drug users? Wailoo argues emphatically no – without the technology of blood transfusions and IV needles, hemophiliacs and drug users would not be some of the highest risk individuals regarding the HIV disease.<sup>50</sup> In other words, HIV in the 19<sup>th</sup>-century would not have manifested itself in the same way it does in the modern era. Additionally, an individual suffering from “an identical human immunodeficiency virus” would have manifested differently, such as tuberculosis or pneumonia, and, furthermore, a medical practitioner who was given an HIV test from the future and directions on how to use it would place it on a shelf to collect dust because it had no use in that particular context.<sup>51</sup> Using a number of blood diseases that relied on blood testing, Wailoo shows that the context of the “disease” within the surrounding technological context was more important in the construction of conceptions of illness than the inherent biological truth found within the sickness of the human body.

This culturalist approach upheld by many historians, including Wailoo and Loughran, holds that it is not simply biology, but also context, which gives rise to conceptions, categorizations, and understandings of disease. Furthermore, the transhistoric nature of disease is abandoned in favor of nuanced approaches toward history that allow more complex, enriching, but decidedly anthropocentric stories to be told. These are laudable goals in theory but potentially disastrous for the plaintiffs in the NFL concussion cases.

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<sup>50</sup>. Wailoo, *Drawing Blood*, 14-15.

<sup>51</sup>. Wailoo, 16.

It is not always the subject matter of history that has problematic moral implications. Rather, sometimes it is also the manner in which that historical analysis is performed. While the illumination of the culturalist and transhistoric positions is not intended to be proscriptive in nature, it is the case that the methodology one employs while examining a historical object (such as CTE) may have unintended consequences concerning the object in the present day. It is possible that this historical segmentation on behalf of the culturalists would undermine the legal and scientific foundations that enable the pursuit of justice. How would such an iconic product of the cultural turn be expected to respond to such allegations? Either historians abandon previously held beliefs that are effectively the cornerstone of many sub-disciplines within the larger category of history itself, or historians sacrifice the pursuit of justice in the hopes of preserving such methodologically driven ideals that may resonate so destructively outside the academy.

## CHAPTER TWO

## MARTLAND &amp; PARKER: PUNCH DRUNK

What follows is a history of CTE that highlights athletes who have suffered from troubling symptoms related to head trauma ranging from pugilists in 1928 to football players in 2005. The manner in which this information ought to be held, transformed, and broadcast to the public should remain at the forefront of this history. While CTE itself has a history, the argument here claims that the type of examination or understanding (i.e., culturalist vs. transhistoric) that allows for a billion-dollar settlement may ought to primacy in the debate between historians advocating for a particular methodology. For example, a player from the 1980s (or their family) would be entitled to many millions if they were able to prove they were suffering from a concise list of diseases as we currently understand them (CTE, Alzheimer's, etc.) under the transhistoric perspective. However, the culturalist perspective, in order to remain consistent with the authors discussed thus far, would necessarily have to conclude that the 1980s disease and the 2018 disease are inherently different historical objects and, thus, ought to be considered as distinct entities and no compensation would be required (a position that would likely benefit the NFL). CTE has a straightforward existence, at least from the medical community's perspective, which follows logical, daresay predictable, transformations that culminate in 2017's legal battle with the NFL and begins in the 1920s with boxers suffering from an unknown medical condition.

In an article titled “Evolving Concepts of Chronic Traumatic Encephalopathy as a Neuropathological Entity,” published in 2017 in *Neuropathology and Applied Neurobiology*, authors H. Ling et al. begin their inquiry by introducing the history of the topic at hand:

In 1928, Martland described the clinical features of ‘punch drunk’ in boxers, a condition later known as ‘dementia pugilistica.’ ‘Chronic traumatic encephalopathy (CTE),’ first coined by Critchley, then became the preferred term which has been widely used in the recent surge in scientific and public interest in this neurodegenerative consequence linked with past exposure to subconcussive or concussive repetitive head impacts.<sup>1</sup>

Ling et al.’s structuring of CTE’s history is one example out of many that follow a similar pattern.

For example, authors Pischuitta et al. (2017), in the journal of *Experimental Neurology*, state, “Originally described in clinical studies of boxers as the ‘punch-drunk’ syndrome, the associated neuropathology later became recognized as dementia pugilistica, more recently CTE.”<sup>2</sup> Similarly, in “Research Gaps and Controversies in Chronic Traumatic Encephalopathy: A Review (2017),” authors Asken et al. claim the following: “Medical literature on the long-term effects of repetitive brain trauma (RBT)...began at least a century ago with the description by Martland of a “punch drunk” syndrome in retired boxers. References to dementia pugilistica and chronic progressive traumatic encephalopathy of boxers appeared in the following decades.”<sup>3</sup> Asken and his

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<sup>1</sup>. H. Ling, “Evolving Concepts of Chronic Traumatic Encephalopathy as a Neuropathological Entity,” *Neuropathology and Applied Neurobiology* 43, (2017): 467.

<sup>2</sup>. Francesca Pischuitta et al., “Single Severe Traumatic Brain Injury Produces Progressive Pathology with Ongoing Contralateral White Matter Damage One Year after Injury,” *Experimental Neurology*, 300, (November 2017): 167.

<sup>3</sup>. Asken, “Research Gaps,” 1256.

colleagues, however, go beyond the usual fare of introductory notes and include an unabashed “Timeline of Notable Publications” in the history of repetitive brain trauma research.<sup>4</sup> This timeline, unsurprisingly, does not include unexpected turns or abrupt shifts in historical understanding – on the contrary, it is literally a straight line that presents the history of a particular disease beginning in the 1920s and ending with the current state of research in the “Present,” or 2017 when the article was published.

This timeline presents a unique insight to the mind of a medical researcher who has an interest in the historical context of the research surrounding a particular disease. If the goal is to present the linear history of CTE as a tool for an argument over ethical implications of historical methodology, it may behoove this investigation to resist reinventing the wheel and utilize the perspective offered directly by the medical researchers. This timeline has an inherent explanatory value for the history of disease from the perspective of those who directly manipulate and research the disease as an object. A dissection of this timeline as a stand-in for the underlying presumptions of how we ought to understand disease would be appropriate. To that end, the handful of recent publications in scientific journals mentioned thus far highlight a surprising attention to the history of a disease that has little connection to the research currently being performed. Current medical researchers must handle, research, and manipulate the disease-as-object that exists independently of the history behind it.

Although, without a doubt, there is a precedent to introduce a topic by spending a few, brief sentences showing where, when, and why previous research had been

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<sup>4</sup>. Asken, “Research Gaps,” 1256.

performed, it is worth noting a common theme throughout the articles presented: the history of CTE, as a disease, begins approximately in 1928 with the work of Harrison S. Martland. Martland's article, published in the *Journal of the American Medical Association*, begins with a description of an unknown "condition" afflicting "prize fighters" that is described, among the fighters themselves, using the slang "punch drunk."<sup>5</sup> Beyond the terminology adopted by the fighters, Martland points out that fans that attend boxing matches also recognize symptoms of this mysterious disease: "cuckoo," "goofy," "cutting paper dolls," or "slug nutty" are all terms used to describe the fighter, in-action, either resisting or succumbing to whatever ails him.<sup>6</sup>

As early as the second paragraph of Martland's expository account of this disease, the term "punch drunk" is given free reign, shed of its scare quotes, and is adopted as a legitimate approximation of the condition. Martland notes that punch drunk is most commonly found in the fighters "of the slugging type, who are usually poor boxers and who take considerable head punishment" and in "second rate fighters used for training purposes" that often suffer multiple knock-downs a day.<sup>7</sup> Martland appears to be describing a situation in which both the severity and quantity of hits contribute, perhaps in an additive effect, to the punch drunk syndrome.

Martland continues in greater detail of the symptoms: "There may be only an occasional and very slight flopping of one foot or leg in walking"; "In some cases periods of slight mental confusion may occur as well as distinct slowing of muscular action"; "a

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<sup>5</sup>. Harrison S. Martland, "Punch Drunk," *Journal of the American Medical Association*, 91, no. 15 (1928): 1103.

<sup>6</sup>. Martland, 1103.

<sup>7</sup>. Martland, 1103.

peculiar tilting of the head . . . a staggering, propulsive gait with the facial characteristics of parkinsonian syndrome [*sic*], or a backward swaying of the body, tremors, vertigo and deafness. Finally, marked mental deterioration may set in necessitating commitment to an asylum.”<sup>8</sup>

Martland believes that punch drunk syndrome affects both physical and mental faculties. Whether an unusual gait, “parkinsonian” symptoms, or mental deterioration, the symptoms of punch drunk are at once serious and ubiquitous. However, the origin of the description of symptoms is not entirely clear. Martland makes several allusions to slang on behalf of fighters and even comments that “[t]he early symptoms of punch drunk are well known to fight fans, and the gallery gods often shout “Cuckoo” at a fighter,” presumably to goad a fighter that has begun to show more serious symptoms of continued head trauma.

Evidence of his serious approach to differentiating punch drunk from other disorders, Martland mentioned the overlap that punch drunk has with other diseases and the implications found therein. He states:

Of course the symptoms produced by the late manifestations of epidemic encephalitis, by the juvenile and presenile [relating to old age] types of paralysis agitans [i.e. Parkinson’s disease], by syphilis, brain tumors and other forms of cerebral injury may so closely resemble those of the condition punch drunk as to be differentiated only with extreme difficulty or not at all.<sup>9</sup>

Martland, however, assures his audience that “the occurrence of the symptoms in almost 50 per-cent of fighters who develop this condition in mild or severe form, if they keep at the game long enough” is an accurate indication that some brain injury does or will

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<sup>8</sup>. Martland, “Punch Drunk,” 1103.

<sup>9</sup>. Martland, 1103.

exist in the future (due to their occupation).”<sup>10</sup> In other words, punch drunk is prevalent in such a specific population as to suggest a commonality between patients deserving of a unique medical designation.

Martland ends his introductory section by claiming that, “As far as I know this condition has practically not been described in medical literature” and that, “I realize that this theory, while alluring, is quite unsusceptible of proof at the present time, but I am so convinced from my former studies on post-traumatic encephalitis that this is the logical deduction that I feel it my duty to report this condition.”<sup>11</sup> Martland believes that he is on the cutting edge of medical research pertaining to the head injuries suffered by athletes, or at least a subsection of athletes, and we see the first action in what will eventually be a slew of research, revisions, theoretical upheavals, and semantic arguments: Martland identifies and categorizes a disease. While this theory, by Martland’s own admission, is unsubstantiated and based on relatively simple logical deductions (e.g., repeated blows to the head cause lasting symptoms), the infantile modern mechanisms of scientific advancement, lacking standardization and strict regulations, is evident.

Asken et al.’s 2017 article references, slightly past Martland, that “Parker” published a paper titled “Traumatic Encephalopathy (‘Punch Drunk’) of Professional Pugilists.” We can immediately make two deductions, before even cracking the article itself: first, there appears to be an effort to either rename, further categorize, or simply clarify punch drunk as a disease, evidenced by Parker’s scare quotes surrounding “punch

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<sup>10</sup>. Martland, “Punch Drunk,” 1103.

<sup>11</sup>. Martland, 1103.

drunk;” second, Parker’s subject still appears to be firmly situated in the examination of boxers (professional pugilists).

Harry L. Parker, reading before the Chicago Neurologic Society on April 19<sup>th</sup>, 1934, makes a quick mess of complicating, through the use of a variety of patients, how the study of “repeated injuries to the brain of a professional boxer” should be performed.<sup>12</sup> Namely, Parker splits the whole of professional boxers that suffer head injuries into two categories: those who receive serious enough injuries to die either during the bout, “or a few hours later,” and those “which more by their repetition than by their severity lead to slower development of disability during the fighter’s career.”<sup>13</sup> Parker’s paper is chiefly concerned with the latter category.

Parker begins by alluding to his predecessor and the previous medical research: “Martland, in 1928, drew attention to the various mental and physical states encountered in examination of former pugilists, and under the title of ‘punch drunk’ outlined some interesting clinical syndromes.”<sup>14</sup> Some of the syndromes, or symptoms, that Parker believes to be interesting include “occasional clumsiness of one foot,” “walking to the corner of the ring,” and “periods of confusion.”<sup>15</sup>

Parker’s work takes an interesting turn, however, by shifting the focus from all fighters, regardless of skill, to “[a]ggressive, hardy fighters who like to give the crowd an exhibition of blows” and “less expert but courageous men who take considerable injury in

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<sup>12</sup>. Harry L. Parker, “Traumatic Encephalopathy (‘Punch Drunk’) of Professional Pugilists” (presentation, The Chicago Neurologic Society, Chicago, Illinois, April 19, 1943): 20.

<sup>13</sup>. Parker, “Traumatic Encephalopathy,” 20.

<sup>14</sup>. Parker, 20.

<sup>15</sup>. Parker, 20.

the hope of wearing out their opponents”<sup>16</sup> as the ones most likely to be afflicted by this relatively unknown disorder. Additionally, “quick, agile, clever boxers who guard themselves well and take little punishment seem to escape” the horrors of punch drunkenness later in life. Parker’s division of the fighting groups, while not seemingly relevant for the development of “punch drunk” as a diagnostic category, is indeed interesting. It appears as if the skills and professionalism of the boxers have become paramount to understanding the nature of the disease.

Before presenting the case studies that he believes supports the narrative of repeated head injuries causing lasting damage, Parker quickly acknowledges the importance of both his and Martland’s work. Parker states:

The point that Martland urged, however, was that the high frequency with which professional pugilists develop crippling disease of the central nervous system of one sort or another suggest a result of the repeated injuries to the brain that they received while carrying on the activities of their profession.<sup>17</sup>

Parker, by referring to a previously published work on the history of this particular disease, has created a precedent and the conditions necessary to establish the transhistoric timeline that will be heavily relied upon in the future, especially concerning the illnesses of NFL players.

To elucidate the problem of connecting “repeated blows to the head” and the clinical symptoms outlined by Martland and himself, Parker intends to present some new material “as an argument for the existence of such a condition as ‘punch drunk’ or traumatic encephalopathy” in the form of a few, select case studies. Note that Parker has no qualms

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<sup>16</sup>. Parker, “Traumatic Encephalopathy,” 20.

<sup>17</sup>. Parker, 21.

with the naming scheme of the disease at hand – punch drunk and traumatic encephalopathy appear to both be acceptable designations for the mysterious collection of symptoms.

In “Case I,” Parker examines a young man, age 24, that visited the Mayo Clinic on February 9, 1934, suffering from “weakness of the legs and tremor of the hands.”<sup>18</sup> Parker does his due diligence and lists a brief medical history of this man: no family history of similar symptoms, he had influenza twelve years prior, and he had “commenced to box” as an amateur at the age of fifteen.

In 1926, the subject of “Case I” had progressed through the ranks of boxers in the area and was now considered a “professional pugilist.”<sup>19</sup> That year, at sixteen years old, he received a “blow over the left temple” during a boxing match which “had knocked him down, and blood had run from his left ear.”<sup>20</sup> Parker notes that the five years after this event “Case I” had “averaged about two fights a month” but had made “no great headway in his profession, for he had lost as often as he had won and he had not improved his technique greatly.”<sup>21</sup> Again, Parker is noting the seeming important connection between clinical symptoms and the skill of the boxer. He remarks that “[h]e was not a very good boxer,” evidenced by a “flattened nose and two ‘cauliflower ears’” indicating that “the patient usually made little effort to guard himself.”<sup>22</sup> After a particularly brutal fight that witnessed numerous hits to the head and knockdowns, the patient vomited periodically throughout

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<sup>18</sup>. Parker, “Traumatic Encephalopathy,” 21.

<sup>19</sup>. Parker, 21.

<sup>20</sup>. Parker, 21.

<sup>21</sup>. Parker, 21.

<sup>22</sup>. Parker, 21.

the night, staggered home “as if drunk,” and slept poorly due to nausea.<sup>23</sup> The patient sought medical help (from a different physician than Parker) and was prescribed a three-day rest in bed, plagued by constant nausea and diplopia [double vision].<sup>24</sup>

Two years later, by the time “Case I” and Parker had crossed paths, the symptoms and their severity had changed very little. The patient still had trouble walking and constantly dragged his feet. This symptom was so severe that Parker noted the patient “wore away the toes of his shoes.”<sup>25</sup> The patient’s mother, possibly accompanying her son to visit the Mayo Clinic, complained that the subject was experiencing intense nervousness since his last fight, had become forgetful regarding “names and details,” and that his hands shook.<sup>26</sup>

Parker’s physical examination of “Case I” included several tests aimed at surmising the severity of the symptoms. For example, Parker had the subject close his eyes while standing upright and watched for a loss of balance. When the subject failed to remain upright, or a positive “Romberg’s sign,” and a “slight, horizontal nystagmus was present,” or involuntary twitching of the eyes, Parker made a diagnostic note. Furthermore, Parker tested for “Babinski’s sign,” a reflex exhibited primarily by infants when stimulating the sole of the foot.<sup>27</sup> While the subject’s “tendon reflexes were greatly exaggerated,” the Babinski test was negative – a positive test in adults is strongly associated with damage to the nervous system or brain. Was Parker perplexed at his subject suffering from some

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<sup>23</sup>. Parker, “Traumatic Encephalopathy,” 22.

<sup>24</sup>. Parker, 22.

<sup>25</sup>. Parker, 22.

<sup>26</sup>. Parker, 22.

<sup>27</sup>. Parker, 22.

neurological disorders but no other, perhaps more common, ones? Doubtless, he was left wondering how to tie all these loose diagnostic ends together. “Case I” clearly exhibited severe neurological issues that Parker suspected was due to a vicious amateur boxing career. Or, as Parker explains, from one fight in particular.

After disclosing the diagnostic peculiarities of “Case I,” Parker engages in a brief, but authoritative, “Comment” section with, presumably, the intention of highlighting the burgeoning importance of continuing to investigate the “high frequency with which professional pugilists develop crippling disease of the central nervous system.”<sup>28</sup> Parker begins, “There is but little doubt that the severe beating received by this pugilist in his last fight was responsible for the chain of circumstances which followed.”<sup>29</sup> Or, “Case I’s” particularly brutal fight on April 1932, two years before the meeting with Parker at the Mayo Clinic, created a separation of diagnostic and medical timelines – that of before and after the April fight. Interestingly, he concedes that,

It might also be suspected that his previous experiences [i.e., fights before the April fight] had altered him so that he was an easy victim in his last fight, but of this there is no evidence, and he attributed all his difficulties to his last and futile appearance in the ring.<sup>30</sup>

Parker seems to be increasingly relying on the testimony of “Case I” regarding the manifestation of this unknown collection of syndromes. Parker’s subject-sourced reasoning here may be two-fold. First, Parker, in his introductory notes, outlines the possible existence of a strange disease, first sketched by Martland four years prior. His intended contribution to Martland’s work was the hope of “further elucidating the problem” through

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<sup>28</sup>. Parker, “Traumatic Encephalopathy,” 21.

<sup>29</sup>. Parker, 22.

<sup>30</sup>. Parker, 23.

the use of “new material” that illustrated this potential disease’s “very variable clinical features.”<sup>31</sup> In other words, it was not Parker’s goal to venture into nosology but to simultaneously support Martland’s previous work on ‘punch drunk’ and to anticipate future medical investigations into the lives of professional pugilists. A couple of additional case studies added to the medical literature may have been the best possible avenue for Parker to take in this regard.

A second explanation for Parker’s reasoning exists, however. Without much fanfare or verbose critical analysis, he simply may not have known. While we may assume that Parker’s nosological deviation would have been minor in an effort to connect his work to Martland’s, the existing framework simply could not have supported much more than slight historical references to his direct predecessor. Parker supports this explanation with some concluding remarks on his subject:

Clinically, the picture was a mixed one, suggesting multiple lesions involving the cerebellar, corticospinal, and basal ganglia systems of motor control. *No specific nervous syndrome appeared*, such as Parkinson’s disease, but rather a medley of scattered and incomplete lesions of the brain.<sup>32</sup>

Parker’s take on the myriad of symptoms presented by “Case I” seems straightforward: there simply was not the medical categories to support the creation, simplification, or elucidation beyond the presentation of a few select case studies by Parker himself. While Martland’s astute observations revealed a clinical enigma, Parker’s work was, in a sense, a continuation of those original observations, neither revealing or obfuscating the true nature of this disease. He concludes his 1934 article with a plea to the surrounding medical

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<sup>31</sup>. Parker, “Traumatic Encephalopathy,” 21.

<sup>32</sup>. Parker, 23.

literature. He starts, “. . . the frequency of occurrence of conditions of this kind as reported by others among people who followed the profession of pugilism makes it seem very likely that their profession led to their ultimate disablement.”<sup>33</sup> For Parker, there was no doubt that boxing was the direct cause of his subjects’ ailments, even if the cause was, as stated, found in the consequences of a single, albeit particularly brutal, match. He continues, “It is to be hoped that in the future more statistical data, backed up by pathological studies, may put these conclusions on a firm basis.”<sup>34</sup> Parker, along with Martland, can conceive of a mysterious disease, caused by a brutal sport, that is only mysterious due to a lack of fact-based analysis of a broader population. Parker’s dearly held belief is that the continued march of scientific innovation and prudent analysis will reveal the intricacies of a disease that, at the moment, he could not.

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<sup>33</sup>. Parker, “Traumatic Encephalopathy,” 28.

<sup>34</sup>. Parker, 28.

## CHAPTER THREE

## MILLSPAUGH: DEMENTIA PUGILISTICA

In the 35<sup>th</sup> volume of the *United States Navy Medical Bulletin*, J. A. Millspaugh, a lieutenant in the Medical Corps of the Navy, published a brief, but altogether revelatory, analysis of the nosology surrounding the collection of symptoms and syndromes found within the boxing community. Described in the same breath as introducing “less derisive” terminology and “meandering,” Millspaugh’s 1937 article “Dementia Pugilistica” marks a turning point in diagnostic credibility regarding this disease.<sup>1</sup>

Millspaugh begins with a casual discussion of the disease at hand. He admits that the common categorization of this disease, “punch drunk,” is “colorful,” “long in lay use,” and rarely requires “elucidation.”<sup>2</sup> In other words, it appears as if Millspaugh recognizes the utility of informal medical designations. It is easy to imagine the causes and symptoms of a disease addressed as “punch drunk” to varying levels of medical knowledge and training. He continues by listing “intended synonymous professional terms” that were often used to describe a similar syndrome: “concussion of the brain, post traumatic neurosis or psychosis, traumatic encephalitis and traumatic encephalopathy.”<sup>3</sup> Ultimately, Millspaugh explains, these terms or diagnoses fail to be “distinctive” in accordance with the strange and peculiar nature of this enigmatic disease. Millspaugh then alludes to the title of the article, “Dementia Pugilistica,” as a proposed term to rule out “related but nevertheless

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<sup>1</sup>. Rudy Castellani, “Dementia Pugilistica Revisited,” *Journal of Alzheimer’s Disease* 60, no. 4 (2017): 1209.

<sup>2</sup>. J. A. Millspaugh, “Dementia Pugilistica,” *United States Navy Medical Bulletin*, 35 (1937):297.

<sup>3</sup>. Millspaugh, 297.

distinct traumatic brain injuries” while also supplying “a distinctive term for a definite condition.”<sup>4</sup>

“Punch drunk,” for Millspaugh, “implies a derisive connotation” among “collegiates” and, also, “hardened professionals” often resent the associated “implication.”<sup>5</sup> While Millspaugh is not forthcoming with his sources regarding the negative connotations surrounding “punch drunk,” perhaps indicating an anecdotal source for these sentiments, the association is not hard to imagine. Professionals of both the boxing and medical world collectively strive for a sense of legitimacy and integrity. As seen with Martland and Parker’s work on punch drunk and expanded on by Millspaugh, the groups most closely associated with the set of symptoms at hand are the “less expert boxers” and the “most typical examples of this disorder” are found in boxers less concerned with “defensive ingenuity.”<sup>6</sup> No matter how skilled the boxer during in the late 1930s may have been, a diagnosis of punch drunk or dementia pugilistica was likely both socially and professionally embarrassing as well as a direct threat to one’s livelihood if that boxer depended on the winnings to pay the bills.

The reality for medical researchers and boxers during the late 1930s was that athletes with particularly poor athletic skill were more likely to suffer from the punch-drunk syndrome. Again, the logical progression is easily understood, if not altogether alluring: if getting hit in the head produces immediate and severe consequences, and unskilled boxers get hit more often than skilled boxers, we would expect unskilled boxers

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4. Millspaugh, “Dementia Pugilistica,” 297.

5. Millspaugh, 297.

6. Millspaugh, 297.

to suffer from the severe consequences more often than skilled boxers. However, if a surprising number of boxers across all levels of skill begin to exhibit symptoms indicative of punch-drunkenness, we could expect at least two pressures to begin weighing on the boxing and medical community alike.

First, medical researchers would need to close the gap between observed populations and predicted populations. Inherent in the disease title of “punch drunk” is an understood proclivity towards manifesting in certain, specific populations. As evidenced above, most medical researchers anticipate, perhaps even with a significant bias, to diagnose or associate amateur boxers, particularly those with a rough professional career, with the umbrella of symptoms known to the 1930s medical community as “punch drunk.” This fact, possibly understood by previous researchers such as Martland and Parker, but certainly acted upon by Millsbaugh, pointlessly limits the diagnostic and accompanying therapeutic power of the disease category. What would a medical researcher, for example, do with a patient exhibiting obvious signs of “punch drunkenness” without ever having participated in a boxing match (i.e., been on the receiving end of a punch)? Consequently, given ample time to immerse itself within the surrounding medical community, the “colorful descriptive term” of punch drunk leaves medical professionals, such as Millsbaugh, wanting. However, this sensitivity to nosology is not limited to the medical community.

In addition to medical researchers reacting to the pressures brought about by a limited, even obtuse, disease category, boxing professionals also resented the use of “punch drunk.” It is easy to demean or invalidate an individual’s boxing accomplishments under

the shadow of associated connotations of suffering punch drunk, especially if, as mentioned, the diagnosis is dependent on receiving many hard hits throughout a boxer's career. Martland's 1928 article briefly mentions the audience's reaction to the onset of these debilitating symptoms: the "gallery gods" often hoot, holler, and throw accusations of "cuckoo" around with all the fervor of a high stakes match. Without indulging too much in speculation, it is also possible to imagine the dualistic nature of suffering from "punch drunk" from the perspective of a boxer. As evidenced by Parker's "Case I," instances of punch drunk seem, from the perspective of our 1930s medical researchers, to be quasi-permanent with little hope for recovery. Once an individual has descended to the horrors of losing their memory, an inability to function physically, and lost their previous source of income, there was no experiment or case study to point to regarding quality of life studies of ex-boxers. The physical and medical reality of their conditions was, however, separated from the social and cultural baggage associated with being a broken, down-and-out boxer with a condition most commonly linked to poor fighting skills.

Millspaugh, then, injects himself within this muted conversation with a potential solution: find a better name for the disease. Millspaugh justifies this endeavor as follows: first, he questions, "Is each occupational calling to receive a name for disability pertinent thereto;" second, Millspaugh states, "The mental unbalance more commonly encountered among pugilists is also observed among other sports representatives who sustain considerable head trauma."<sup>7</sup>

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<sup>7</sup>. Millspaugh, "Dementia Pugilistica," 297.

Millspaugh's first objection, pertaining to the "occupational calling," appears to be a warning to a pre-standardized notion of medical research that may balloon out of control. While not explicitly stated by Millspaugh, we can wonder at the consequences of such an approach. Does an individual's profession take precedence regarding nosology or is there an underlying collection of truths that can be utilized to more accurately diagnose and treat patients? If a pilot suffers from "aeroneurosis" and a boxer from "pugilneurosis" yet the diagnostic criteria and symptomatic manifestation is identical, what is holding medical researchers beholden to one diagnosis over the other? Embedded in this conceptual breakdown of nosological principles provided by Millspaugh is an appreciation of biology over profession. He recognizes that the commonalities across professional divides far outweigh the importance of the profession-to-disease relationship. In other words, the pilot and the boxer, despite differing professions, clearly share a significant aspect of medical determinants – namely, both the boxer and pilot share the same biology and suffer alike. This recognition resulted in an appeal by Millspaugh to avoid "unnecessarily encumber[ing] the nosological nomenclature."<sup>8</sup>

Millspaugh's second motivating factor for creating a discussion regarding the nosology of punch drunk, related to the first, is relatively simple: the "mental unbalance" seen in pugilists is "also observed among other sports representatives who sustain considerable head trauma."<sup>9</sup> Again, this would limit the diagnostic potential of a mysterious disease to a specific sport for relatively unknown reasons. In addition, this limitation would occur despite existing evidence that other athletes (who are not getting

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<sup>8</sup>. Millspaugh, "Dementia Pugilistica," 297.

<sup>9</sup>. Millspaugh, 297.

punched) suffer from similar symptoms and would be a travesty to medical and scientific integrity. Millspaugh intends to outline a more appropriate nosological approach.

In a five-page account, Millspaugh proposes the following: first, replace “punch drunk,” “a condition long recognized but accorded little medical cognizance,” with the term “dementia pugilistica;” second, present a “physical-physic syndrone [*sic*] characterizing the condition;” finally, urge the “intervention of medical officers” in boxing matches in the interest of “mental hygiene.”<sup>10</sup> Millspaugh’s discussion of replacing punch drunk with dementia pugilistica is largely limited to the above discussion of nomenclature – a brief transitional interlude compared to what follows.

Millspaugh’s second objective, outlining a physical-psychic syndrome of the condition, involves, unsurprisingly, summarizing the work of previous medical researchers. Millspaugh nods to Martland’s “23 fighters” said to be “punch drunk” and how “four of these were known to be in asylums.”<sup>11</sup> He then explains the distinction between “gross accidental brain injuries” with “macroscopic hemorrhage” and “those due to repeated or less severe trauma to the central nervous system” and “microscopic” hemorrhages.<sup>12</sup> The impact of this distinction is meant to be obvious – macroscopic damage to the brain, at least to medical researchers like Millspaugh, carry very little mystique when compared to the enigmatic dementia pugilistica at hand. Millspaugh follows an examination of Martland with a brief reference to Parker’s work mentioned above: “No specific nervous syndrome appeared, such as Parkinsons’ [*sic*] disease, but rather a medley

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<sup>10</sup>. Millspaugh, “Dementia Pugilistica,” 302.

<sup>11</sup>. Millspaugh, 298.

<sup>12</sup>. Millspaugh, 298.

of scattered and incomplete lesions of the brain.”<sup>13</sup> However, Millspaugh expands beyond Parker’s relatively limited predictions of what groups suffer and to what degree. Specifically, Millspaugh mentions that “many veterans, amateur, semiprofessional, and professional prize fighters” suffer from an “altered visage and mind” that is “as authentic in its manifestations as are some of the more classical examples.”<sup>14</sup> In other words, Millspaugh recognizes, and states within the medical literature, that populations outside the case studies mentioned previously of brutish, uncoordinated amateurs suffer from a similar set of symptoms. Millspaugh is apparently suggesting that one’s skill in boxing is not the only factor in determining or predicting the onset of dementia pugilistica.

Either from a lack of availability or means, Millspaugh opts to recount some interpersonal encounters with alleged instances of the disease (as opposed to Parker’s reliance on clinically examining ex-boxers). One morning, Millspaugh recalls, while “awaiting a boat aboard a cruiser,” he noticed a fellow officer exhibiting some strange behavior.<sup>15</sup> This “boisterous” individual gave the impression he was “directing a deadly engagement” by yelling, dancing about, and pantomiming, or extravagant expression through gesture. Millspaugh asked around – this individual was a “well-known athlete while at the Naval Academy.”<sup>16</sup> A year later, Millspaugh and five medical officers were assembled in a room when this same individual burst in. Sitting on a windowsill, this man “spouted forth concerning his ailment and abruptly departed in a dash.”<sup>17</sup> Millspaugh

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<sup>13</sup>. Millspaugh, “Dementia Pugilistica,” 298.

<sup>14</sup>. Millspaugh, 299.

<sup>15</sup>. Millspaugh, 300.

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<sup>17</sup>. Millspaugh, 300.

recalls the doctors then shaking their heads, smiling lightly, regarding one another with a “questioning gaze,” and murmuring, “Punch drunk.”<sup>18</sup>

Millspaugh continues with a rather compassionate analysis of the origin of this disease – namely, the punishment dealt by greedy or thoughtless individuals. Rather than blame the fighting prowess of affected fighters (again, note that Millspaugh deviates from Parker and Martland by claiming that nearly all fighters suffer from “dementia pugilistica,” not just those lacking in skill), Millspaugh takes aim at “thoughtless” managers and trainers that, through greed, match fighters with “opponents far out of their class in experience, condition, strength, or weight.”<sup>19</sup> Additionally, Millspaugh adds a financial dimension to the already nebulous punch drunk pseudonym. “Quasi-professional” fighters, or those who earn the majority of their livelihood through other means than fighting, will, according to Millspaugh, participate in a bout “attracted only by the financial reward.”<sup>20</sup>

With the history of the disease laid bare, albeit brief, Millspaugh continues with the final project and, indeed, the bulk of the paper: analyzing the rules of boxing and the role of medical officers therein. The true scope of Millspaugh’s investigation becomes clear once he engages with the intricate relationship between boxing and boxing officials. Publishing in the *Navy Medical Bulletin* and expressing a concern over whether medical officers from the U.S. Navy are present to “examine boxers and attend bouts,” we see that Millspaugh’s experience with dementia pugilistica is largely limited to the boxing matches hosted, either officially or unofficially, by members of the United States Navy.<sup>21</sup> That being

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<sup>18</sup>. Millspaugh, “Dementia Pugilistica,” 300.

<sup>19</sup>. Millspaugh, 301.

<sup>20</sup>. Millspaugh, 301.

<sup>21</sup>. Millspaugh, 299.

said, Millspaugh acknowledges that the naval services follows the same set of rules and regulations supplied by the National Collegiate Athletic Association, particularly those rules concerning the use of medical examiners. Rule 6, Sec. 1, for example, of the National Collegiate Athletic Association states, “The Officials [for the boxing bout] shall be a referee, two judges, two timekeepers, and one medical officer.”<sup>22</sup> Additionally, Millspaugh notes that Rule 7, Sec. 1 specifically calls for contestants to present themselves for examination before a medical officer at the appointed time.<sup>23</sup> Millspaugh’s concern over the official-to-boxer relationship seems to reside in the boxing official’s immediate apprehension of problematic symptoms arising from the nature of the sport. In an effort to alleviate, prevent, or, in the case of 1930s medical research, understand a mysterious disease, Millspaugh argues that bouts of boxing ought to stringently adhere to the rules and regulations set forth by the relevant associations. In other words, the individual with the greatest access to the manifestations of symptoms in boxers is undoubtedly the officials themselves and, Millspaugh argues, they are at the forefront of the burgeoning medical knowledge surrounding dementia pugilistica.

Millspaugh describes the onset of dementia pugilistica, presumably to an audience comprised of boxing officials, following his plea of rigid observance of boxing regulations. Millspaugh warns that the “physical-psychic syndrome” is accompanied by both alterations in the boxer’s “visage and mind.”<sup>24</sup> Dyslalia, or difficulties in speech, “modified breathing habits” resulting from clogged upper respiratory passages, and a declining “psychic”

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<sup>22</sup>. Millspaugh, 299.

<sup>23</sup>. Millspaugh, 299.

<sup>24</sup>. Millspaugh, 299.

attitude often characterized as “belligerent” or “vindictive” are all potential consequences of a boxer suffering from dementia pugilistica.

Millspaugh continues with what seem to be more immediate indications of an underlying disease. Included are “various mannerisms” from “scowling” to “grimacing,” presumably, although not explicitly stated, during the boxing bout. Interestingly, Millspaugh notes that certain sounds, “similar in tone to the ring bell,” “illicit [*sic*]” conditioned reflexes such as “offensive attitudes” in experienced boxers. Generalized “confusion,” however, Millspaugh argues, “requires but little observation.”<sup>25</sup> Particularly brutal matches between boxing opponents was nearly universally followed by what Millspaugh describes as “transient directional orientation” or, in other words, stumbling and trouble finding their corner. Unsurprisingly, Millspaugh also recounts that many fighters suffer from a daze or “stupor” after recovering from a knockout.<sup>26</sup> In addition to dyslalia, dysbasia (the “swaying, shuffling, staggering, and later a propulsive or festinating gait”) is also common. Millspaugh notes the expression “walking on his heels” is applied quite often to boxers about this presentation of illness. Lastly, he describes amnesia that always accompanies “knockout punches affecting the head” and that many fighters attest to “amnesia of some degree” as a “consequence of severe beatings,” regardless of knockout.<sup>27</sup>

Millspaugh, when compared to medical researchers like Martland, blurs the line between the professional and the informal, especially concerning the colloquial language

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<sup>25</sup>. Millspaugh, “Dementia Pugilistica,” 300.

<sup>26</sup>. Millspaugh, 300.

<sup>27</sup>. Millspaugh, 300.

attributed to the suffering fighters. The symptomatic expression, for example, offered by Millspaugh makes no attempt to distinguish between immediate and lasting effects supposedly brought on by dementia pugilistica. Millspaugh understands the underlying disease to be accurately described by the aforementioned constellation of symptoms but, despite an apparent compassion for the fighters themselves, it becomes difficult to separate the disease from the profession. Vindictiveness, scowling, conditioned reflexes, and “mild delusionary trends” aimed at magnifying a fighter’s “former prowess” are all described as a result of the disease itself yet is easily imagined as a consequence of a type of person drawn to combative sports. Millspaugh, as stated in his conclusion, intended to present a “physical-psychic syndrome” that more accurately characterizes the punch drunk/dementia pugilistica disease, yet one might wonder at the delicate distinction between introduced complication and holistic representation.

Millspaugh describes the origin of his expertise as decidedly a spectator of the sport. It is likely that Millspaugh’s observations were gathered from personal interactions with the fighters in the capacity as a spectator, not a medical professional. Indeed, Millspaugh admits that “no individual cases are presented in detail” and that his observations “are gleaned rather from reflection” and form a “more or less composite impression of many individuals who presented the various signs and symptoms delineated.”<sup>28</sup> Millspaugh’s chosen method of investigation and reporting distinctly results in “tall tales and amusing anecdotes” but, he assures the reader, the “erratic behavior of fighters” may be observed “wherever the coterie of the ring foregather.” Regardless of

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<sup>28</sup>. Millspaugh, “Dementia Pugilistica,” 302.

potential amplification of facts, Millspaugh reminds his audience that “all exaggeration is based on facts.”<sup>29</sup>

The history of this disease took one step further toward greater illumination regarding cause, symptomatic expression, and possible treatments. While Martland created a dialogue and Parker presented specific cases, Millspaugh’s contribution appears to have been multiple insights to the nature and culture of boxing. Whether the personality of fighters or the nuances of officiating boxing bouts, Millspaugh encourages his audience to be cognizant of the unavoidable repercussions of a lifelong career in boxing, however complex or innumerable those repercussions may be. With Millspaugh’s innovative, if not haphazard, approach towards the research of dementia pugilistica, the medical community has a clearer picture of the cultural aspects of boxing, if not any greater elucidation on the disease-as-object.

As it relates to the plight of affected NFL players, however, Millspaugh’s medical research started a conversation regarding who and what are authoritative voices when it comes to diagnosing the deteriorating health of athletes. In particular, people in unique positions to observe the actions of athletes both in and out of their given athletic arena, Millspaugh argues, ought to be given a voice. An inclusive discourse is for the overall betterment of, as Millspaugh argues, the nosology of dementia pugilistica and, as I argue, the positive health outcomes of future players (especially from the transhistoric perspective). When a large, amorphous organization such as the National Collegiate Athletic Association (under Millspaugh’s consideration) engages in a reciprocal discourse

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<sup>29</sup>. Millspaugh, “Dementia Pugilistica,” 302.

with boxing officials witnessing the negative consequences of boxing, the chances of establishing corrective or preventative policies in the future increases dramatically. Moreover, while the responses given to medical personnel by afflicted athletes were considered inextricable from their social background, the possibility of including boxing officials in the discussion regarding the symptomology of dementia pugilistica would prove invaluable for lessening the restrictions on who is allowed to authoritatively contribute to the discussion.

## CHAPTER FOUR

## BOWMAN AND BLAU: TRAUMATIC ENCEPHALOPATHY OF PUGILISTS

If individuals like Millspaugh expanded the systems of knowledge surrounding dementia pugilistica by relatively small leaps in cultural knowledge, Samuel Brock's edited volume *Injuries of the Brain and Spinal Cord and their Coverings: Neuro-Psychiatric, Surgical, and Medico-Legal Aspects* situated the disease within the greater medical context of the 1940s.<sup>1</sup> The persistent physical and psychiatric symptoms that follow a retired boxer throughout the rest of their life is given, in some sense, scientific and medical legitimacy: although through an invariably evolving nosological framework, dementia pugilistica is presented alongside chapters covering topics such as "Concussion and Contusion of the Brain and Their Sequelae," "General Pathological Considerations in Brain Injury," and "The Medio-Legal Aspects of Injuries of the Brain and Spinal Cord and their Coverings."<sup>2</sup> In other words, the diagnostic significance of a variety of diseases related to brain trauma are situated within the greater context of the medical industry.

While the entire volume contains valuable insights into the historical development of brain-related trauma, Karl Bowman and Abram Blau's chapter titled "Psychotic States Following Head and Brain Injury in Adults and Children" is a groundbreaking analysis of the various methodological and diagnostic approaches used both historically and contemporaneously for the 1940s. Bowman, a professor of psychiatry out of the

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<sup>1</sup>. *Injuries of the Brain and Spinal Cord and their Coverings: Neuro-Psychiatric, Surgical, and Medico-Legal Aspects*, ed. Samuel Brock (Baltimore: The Williams & Wilkins Company, 1943).

<sup>2</sup>. Chapter 4 by Sir Charles Symonds, Chapter 2 by George B. Hassin, M.D., and Chapter 23 by Moses Keschner, M.D., LL.B., respectively.

University of California Medical School and the Superintendent of the Langley Porter Clinic in San Francisco, and Blau, an assistant clinical professor of psychiatry out of the New York University College of Medicine, introduce the topic as follows: “The relation between head injury and mental disease is a highly controversial subject.”<sup>3</sup> The controversial nature of head trauma is at the center of their chapter.

With the reader primed for a contentious debate with regard to head trauma, Bowman and Blau forcefully state that the lay and casual connection between head injury and subsequent disease, at least in the broader public, is often overemphasized.<sup>4</sup> They admit, however, that this emphasis has, at least upon a prima facie evaluation, certain merit: first, head injury is a “simple and intelligible basis for an otherwise mysterious mental disorder;” second, the stigma of a hereditary basis for “mental disease” is removed if the disease itself is “obviously due to an outside agency such as head trauma;” finally, a complication of what Bowman and Blau call the “post hoc ergo propter hoc” fallacy, or the natural assumption that if an event follows another then the second event was caused by the first.<sup>5</sup> Bowman and Blau assure their readers that such “a conclusion is often not justified” concerning the relationship between head trauma and subsequent mental disorders.<sup>6</sup>

Bowman and Blau’s stated goals are multi-faceted, including etiology, industrial compensation, and “large sums of money” resulting in a need to reevaluate the

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<sup>3</sup>. Karl Bowman and Abram Blau, “Psychotic States Following Head and Brain Injury in Adults and Children,” in *Injuries of the Brain and Spinal Cord and their Coverings: Neuro-Psychiatric, Surgical, and Medico-Legal Aspects*, ed. Samuel Brock (Baltimore: The Williams and Wilkins Company, 1943): 342.

<sup>4</sup>. Bowman, 342.

<sup>5</sup>. Bowman, 342.

<sup>6</sup>. Bowman, 342.

“relationship of head trauma to the development of mental disorders.”<sup>7</sup> Interestingly, Bowman and Blau note that an increase in industrial accidents and laws dictating when and where to place responsibility for injuries has prompted medical experts to weigh in regarding the connection between brain trauma and subsequent, permanent brain illness. In other words, while the understanding behind brain trauma is relatively limited, the surrounding commercial-industrial complex has necessitated a significant jump in the diagnostic and nosological capabilities of medical culture. This motivation resulted in two significant goals for the summarization provided by Bowman and Blau: show how, if at all, medical experts can distinguish between “pre-existing factors” that would mischievously “improve chances of compensation” and genuine illness resulting from work-related injuries and, additionally, separate “psychogenic symptoms” from “true organic syndrome[s].”<sup>8</sup> In other words, what characteristics of brain trauma are sufficiently unique from other factors when determining the health of an individual after an accident?

The distinction between psychogenic symptoms and organic syndromes is at the heart of Bowman and Blau’s concern over the developing traumatic frameworks concerning brain injuries. Indeed, Bowman and Blau state the growing concern that, quite frequently, “psychogenic symptoms will be superimposed on a true organic syndrome.”<sup>9</sup> For example, if we take some of the anecdotal evidence provided by Parker’s more rigid case study, Bowman and Blau are indicating that it is exceedingly difficult to differentiate

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7. Bowman, “Psychotic States,” 342-3.

8. Bowman, 343.

9. Bowman, 343.

between individuals that “naturally” display these characteristics (e.g., through developmental disorders), those that have legitimate, lasting brain damage from traumatic injuries, and those willing to feign traumatic injury to the brain for greater compensation from their employer. The latter category is especially concerning for Bowman and Blau considering that disease resulting from brain injury is mysterious, temporally imprecise, and is often suffered without external evidence. This is, of course, exacerbated, at least from Bowman and Blau’s perspective, by a wide spectrum of possible “reactions” to organic disease, including exaggeration and minimization. For example, they believe some individuals exist that are willing to “exaggerate consciously a condition which is truly organic.”<sup>10</sup>

To combat the exaggeration of disease, Bowman and Blau introduce, albeit briefly, the concept of “simulation or malingering of post-traumatic psychoses.”<sup>11</sup> Again, Bowman and Blau preface the discussion with a warning of controversy and considerable disagreement among medical researchers but provide the following definition of “malingerer:”

[T]he individual becomes a malingerer only when he consciously and purposely, in order to deceive, to evade responsibility, or to derive gain, feigns illness and voluntarily tries to reproduce signs and symptoms which he really does not have, or extravagantly exaggerates minor ones which he has.<sup>12</sup>

According to the framework laid thus far, Bowman and Blau present this definition of malingerer to differentiate it from the “unconscious mechanisms of the neuroses” and, in some cases, the “severe exaggeration of symptoms” that eclipses

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<sup>10</sup>. Bowman, “Psychotic States,” 343.

<sup>11</sup>. Bowman, 343.

<sup>12</sup>. Bowman, 343.

nefarious financial motivations to such a degree that it could only occur in the “truly sick.”<sup>13</sup> While the authors intend to separate genuine illness from the reprehensible actors willing to falsely portray sickness for monetary gain, it would appear that even those condemnable actors occur, at least in Bowman and Blau’s estimation, in degrees of severity. Bowman and Blau conclude their preface with a reassurance that “experienced observers” may always find, given ample effort, obvious failings in those individuals attempting to defraud the complicated system of relationships between commercial entities, suffering employees, and the medical experts opining on suspected illness.

Rather than explicating the nature of malingerers more than necessary, Bowman and Blau present the concept of fraudulent sufferers for the purposes of contextualizing the subsequent discussion of the classification of brain trauma – with the implicit understanding that differentiating between malingerers and genuine “organic syndromes” is only supplemented by further understanding the nature of the syndromes themselves. However, Bowman and Blau refer to the “standard classified nomenclature of diseases” agreed upon by the American Medical Association, the American Neurological Association, and the American Psychiatric Association as “inadequate.”<sup>14</sup> The inadequacy of the “Section on the Classification of Mental Disorders,” Bowman and Blau argue, is due to the lack of “traumatic neurosis” or “traumatic psychoneurosis” designations, resulting in an inability to create fruitful discussions regarding the topic.<sup>15</sup> What results is a new classification scheme, offered by Bowman and Blau, not

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<sup>13</sup>. Bowman, “Psychotic States,” 344.

<sup>14</sup>. Bowman, 344.

<sup>15</sup>. Bowman, 344.

necessarily concerned with different “disease entities,” but categorizing the numerous and varied “mental syndromes” that are known to follow a head injury.<sup>16</sup> Their classification is structured around two main concepts: primary and secondary traumatic mental disorders. The former has a “fairly clear” relationship between the traumatic event and the mental symptoms and the latter appreciates the trauma as a “variable part in the production of the pathology” but allows for other “diffuse” influences such as “constitutional, psychogenic, or organic factors” in disease manifestation.<sup>17</sup>

While mostly ignoring the secondary traumatic mental disorders of Bowman and Blau’s presented scheme (defined as “mental disorders associated with, or precipitated or complicated by, but not due primarily to head trauma”) as tangential to the central argument, there are at least two noteworthy designations presented in this “updated” classification system.<sup>18</sup> First, under primary traumatic mental disorders, Bowman and Blau list “Acute and Subacute” as a subcategory containing, among others, “Concussion syndrome.” Second, under the “Chronic” subcategory, they list “Traumatic encephalopathy of pugilists (punch drunk).”<sup>19</sup> Referring back to Asken et. al’s 2017, the contemporary article providing the linear structure for the nosological development of CTE, the authors note that Bowman and Blau’s 1940 chapter in the edited volume is the “first use of ‘chronic traumatic encephalopathy’ describing persistent symptoms in a retired boxer.”<sup>20</sup> While it seems evident that Asken et. al took some liberties with that

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<sup>16</sup>. Bowman, “Psychotic States,” 344.

<sup>17</sup>. Bowman, 344-5.

<sup>18</sup>. Bowman, 345.

<sup>19</sup>. Bowman, 345.

<sup>20</sup>. Asken, “Research Gaps,” 1256.

claim as the string of words “chronic,” “traumatic,” and “encephalopathy” never occur in that exact order except in a footnote describing a case study intended to support their claims, Bowman and Blau’s work deserves to be examined in greater depth to fully understand this moment in 20<sup>th</sup>-century medical history with regard to the development of CTE as a diagnostic category.<sup>21</sup>

Bowman and Blau define primary traumatic psychoses as those resulting exclusively from traumatic injury to the brain caused by “direct or indirect physical force to the head.” Additionally, the “mental condition” is related to “organic alterations” within the brain caused by outside, physical sources – or, in other words, organic matter of the brain has been negatively affected. Bowman and Blau inform the reader that “older writers” had been handicapped by the “limitations of neurological and psychiatric knowledge of their time” resulting in a relatively limited historical background to subsequently inform their research.

However, despite a lack of historical grounding, the authors also note that primary traumatic psychoses, including but not limited to punch drunk, is, historically speaking, relatively rare. Bowman and Blau refer to only thirteen cases of psychoses following the 8,985 head injuries in the Franco-Prussian War and that “insanity” among soldiers during WWI was not higher than the “civil population.”<sup>22</sup> Additionally, a 1922 survey concluded that only 0.3% out of 70,987 admissions to mental hospitals in the United States were due to “traumatic psychoses.”<sup>23</sup> While not explicitly addressed by Bowman

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<sup>21</sup>. Bowman, “Psychotic States,” 375.

<sup>22</sup>. Bowman, 347.

<sup>23</sup>. James V. May, *Mental Diseases: A Public Health Problem* (Boston: R.G. Badger, 1922):457.

and Blau, the conflict at hand appears to be a frustrating environment to begin or continue medical research concerning traumatic brain injury. If the medical community, as evidenced by Bowman and Blau's argument, is woefully uninformed in the 1940s regarding brain trauma, the larger societal norms dictating individual responses to suspected injuries to the brain is to usually avoid professional help (and leads to underreporting). Also, given that the nosology of brain injury has continued with a frenetic pace since at least 1928, the scientific framework behind traumatic brain injuries cannot properly support continued, sophisticated research. This is, in general, the conversation that Bowman and Blau intend to place their restructured disease classification.

Before presenting the first subcategory of primary traumatic psychoses, Bowman and Blau speak briefly about symptomatology. They claim that primary traumatic psychoses have "no specific symptoms" and that their picture is the same as "the whole group of organic reaction types."<sup>24</sup> In other words, the "syndromes" following head trauma are often indistinguishable from illness as the result of other causes. Toxins are one example: whether endogenous, having an internal origin, or exogenous, an external one, disorders caused by toxins are similar enough in nature to make discerning their symptomatic origins difficult. Eclampsia (convulsions in pregnant women) and alcohol-related illness, especially given the nascent state of medical knowledge of Bowman and Blau's 1940s, are two examples of illnesses caused by toxins that result in symptoms concerningly similar to those presumably caused by physical trauma to the brain.

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<sup>24</sup>. Bowman, "Psychotic States," 350.

Bowman and Blau, in addition to toxins, include infections (e.g., syphilis), “degeneration” (e.g., “senility”), brain disease (e.g., tumors), and “metabolic and general diseases” (e.g., pellagra or myxedema) as possible complications to a differential diagnosis.<sup>25</sup> In summation, Bowman and Blau state, “It is very hard to estimate the effect of head injury in a person already suffering from an organic brain disease.”<sup>26</sup>

The first subcategory of primary traumatic mental disorders includes both “acute” and “subacute” classifications. While Bowman and Blau list traumatic coma, traumatic delirium, and others under this category, the most noteworthy inclusion is that of “concussion syndrome.” In general, the occurrence of acute primary traumatic psychoses, Bowman and Blau argue, is included in “all head traumata” with severity depending on the “degree of injury.” Bowman and Blau present their first subcategory in an extraordinarily inclusive manner: ranging from immediate death following a severe cranial injury to a relatively inconsequential daze that lasts a few minutes at most, the authors believe all immediate symptoms affecting the brain resulting from physical trauma may be considered acute psychoses. The result is, between the two stated extremes, a “great variety of acute and subacute conditions, in short, confused states, severe deliria and protracted Korsakoff [memory disorder] psychoses.”<sup>27</sup>

One of the most fascinating immediate symptoms resulting from acute traumatic injury to the brain is that of concussion syndrome. Bowman and Blau note that “no detailed study of acute traumatic psychoses has appeared in the American and English

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<sup>25</sup>. Bowman, “Psychotic States,” 350.

<sup>26</sup>. Bowman, 350.

<sup>27</sup>. Bowman, 351.

literature.”<sup>28</sup> This is an interesting contention especially considering the treatment of Martland, Parker, and Millspaugh offered here. It would appear as if Bowman and Blau have positioned their analysis of concussion syndrome specifically around a “psychiatric orientation,” preventing otherwise natural connections between our previous authors’ investigations surrounding the consequences of boxing. Martland, for example, while ostensibly investigating the same symptomatic manifestation as Bowman and Blau, had not orientated his research around the same topic of psychiatry. It is difficult to conclude, then, whether Bowman and Blau believe their work has much overlap with Martland’s previous analysis. Despite their belief that acute and subacute primary traumatic psychoses originate from “serious microscopic alterations in the cerebral tissue,” a similar conclusion reached by our previous authors, Bowman and Blau either failed to recognize the similarity in diagnoses or, through nosological conventions, disregarded it entirely. Indeed, the similarities with hindsight between punch drunk and concussion syndrome may have been irrevocably shadowed by interpretive differences, even if those differences were as simple as naming conventions.

Bowman and Blau’s description of concussion syndromes deserves proper analysis here with the goal of creating a singular nosological trajectory persisting well into the 21<sup>st</sup>-century. Bowman and Blau describe concussion syndrome as periods of “light forms of confusion” with an absence of more “pronounced” mental symptoms.<sup>29</sup> However, the confusion is often preceded by “a loss of consciousness” or a “slight clouding,” such as, although not explicitly stated, knockout punches seen in boxing.

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<sup>28</sup>. Bowman, “Psychotic States,” 351.

<sup>29</sup>. Bowman, 352.

Additionally, emotional irregularity, such as “euphoria or irritability,” is common. An important factor in concussion syndromes, however, is a favorable prognosis and, as Bowman and Blau claim, a complete recovery in a relatively short time.<sup>30</sup> To conclude their symptomology, Bowman and Blau claim, “However it must be emphasized that occasionally chronic mental symptoms may be noted later,” effectively creating chaos regarding understanding the temporal aspects of this disease. Additionally, the symptoms Bowman and Blau suggest accompany concussion syndrome must also occupy a space between a lack “more pronounced mental symptoms” and “chronic mental symptoms” – a space that is altogether unclear. It may seem commonsense that lasting, or chronic, symptoms resulting from an acute injury would classify as “pronounced,” albeit with an understanding that time is an important factor in realizing that these symptoms exist in the first place.

Perhaps Bowman and Blau mean to reserve the possibility of a chronic traumatic psychosis having a causal relationship to a concussion syndrome. In other words, the concussion syndrome is the precursor to a chronic disease – an altogether realistic and probable connection recognized today. However, it is important to resist unduly forcing the progressive advancement of medical understanding beyond what our 1940s authors meant. It would be expected for such a connection to plausibly exist within the subtext of such historical material but left for future medical researchers to “correctly” or appropriately make the connection themselves. Regardless, Bowman and Blau seem to

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<sup>30</sup>. Bowman, “Psychotic States,” 352.

realize, if not make readily apparent to the reader, an important relationship between the concussion syndrome and subsequent, and direr, diagnoses.

In addition to the “simple” concussion syndrome, Bowman and Blau continue into the “more severe” post-traumatic psychoses that, by definition, persist for “weeks or months.”<sup>31</sup> The acute traumas that persist for weeks occur, according to our authors, in three distinct stages: first, the individual suffers from a coma or unconsciousness; second, the period of unconsciousness is followed by “delirium,” “stupor,” or “apathy;” finally, amnesia or a generalized memory disorder.<sup>32</sup> Bowman and Blau note that there is no specific transition from one stage to another and may entirely depend on the individual in question. Here, our authors attempt to explain the physiological mechanisms at play with each stage. The first stage, or unconsciousness, results from a “paralysis of function,” presumably from the impact itself. The delirium, stupor, or apathy, the second stage, are “acute symptoms” resulting from “irritation” of the brain tissue. The final stage of memory disorders, Bowman and Blau claim, is the result of “basic defects in higher cerebral activities” that are known to “recover more slowly.”<sup>33</sup>

It is not clear whether Bowman and Blau claim that cases of amnesia occur in response to the first and second stages or if amnesiac symptoms are ever-present but are diagnostically shadowed by the “shock” and “irritation” of previous stages. While Bowman and Blau are certainly making a great effort to explicate the nuances of varying categories of trauma-related disorders, the differences between subcategories of diseases

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<sup>31</sup>. Bowman, “Psychotic States,” 352.

<sup>32</sup>. Bowman, 352.

<sup>33</sup>. Bowman, 352.

are not always clearly explained. While Bowman and Blau's treatment of head injuries is certainly presented in greater detail than any of our previous authors, it would be inappropriate to expect a comprehensive account at this point.

Bowman and Blau spend the next few pages outlining the intricacies of each of these stages and possible deviations in symptomology and diagnostic realities. Ranging from a discussion of length of expected unconsciousness, departing briefly to contemplate the existence and prognosis of acute traumatic psychosis in children, and ending with a description of the emotional variance of patients suffering from acute traumatic psychosis, Bowman and Blau leave the subcategory of acute and subacute primary traumatic psychoses (that includes concussion syndrome) with few stones unturned. The prognosis of such psychoses, in general, is positive: Bowman and Blau claim that, while dependent on the length of the "initial disturbance of consciousness," recovery from such injuries should be nearly complete from months to mere days.<sup>34</sup>

Although less than categorical, the outline presented is sufficient to mark a temporal separation from the subsequent category, chronic primary traumatic psychoses. A hypothetical boxer, then, that suffers a particularly brutal knockout punch, languishes for a week or so, but recovers with the only negative repercussion being a hazy account of the fight (or some other shade of amnesiac symptomology) could be confidently placed within the acute traumatic psychosis category without a concern for lasting symptoms. The same boxer may suffer from multiple instances of acute traumatic psychosis, say through a less-than-illustrious amateur career, without necessarily

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<sup>34</sup>. Bowman, "Psychotic States," 359.

implicating his well-being into the category of chronic psychoses. This imagined boxer's plight would, of course, exist entirely within the categorization and understanding provided by Bowman and Blau.

Following acute primary traumatic psychosis, Bowman and Blau outline the second subcategory of traumatic brain injury: those leaving patients "chronically incapacitated."<sup>35</sup> The authors admit their clear lack of foundational knowledge surrounding the category in a series of statements that encapsulate a surprisingly wide range of possible necessary conditions to be considered "chronic primary traumatic psychoses." While the foremost condition is that "residual neurological and mental disturbances" persist after the aforementioned acute symptoms recede, Bowman and Blau state that "chronic residual symptoms" may improve over time, but some cases remain chronically incapacitated. Additionally, while a chronic psychosis does not necessarily need to be preceded by a "distinct acute course," or a predictable pathological progression, when and if it does there is no way to determine the "latent period" between acute and chronic psychosis.<sup>36</sup> Indeed, even the nature of the chronic psychosis is highly debatable. Bowman and Blau concede that, relative to the injury-to-acute-psychosis causal relationship, chronic psychosis, especially those without preceding acute symptoms, can be nearly impossible to definitively prove. This is especially relevant with our authors' initial motivation for this analysis in mind: to further develop the legal and judicial position of traumatic brain injuries regarding commercial accidents. If nothing else, this point illustrates Bowman and Blau's interest in engaging with larger social and

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<sup>35</sup>. Bowman, "Psychotic States," 361.

<sup>36</sup>. Bowman, 361.

cultural structures that may have previously inhibited due process in a court of law. As an aside, it is not apparent whether Bowman and Blau have vested financial interests in the court's opinion of traumatic brain injury.

Bowman and Blau make an important distinction between two types of chronic primary traumatic mental disorders: between what they term "post-traumatic personality disorders" and "post-traumatic defect conditions."<sup>37</sup> In short, the former category is meant to characterize syndromes that have a "predominance of psychiatric rather than neurological symptoms" while the latter is chiefly concerned with "cerebral abnormalities" that lead to sensory or motor defects. For example, an individual diagnosed with a post-traumatic personality disorder would have emotional disturbances, "defects in mental functioning," and, in more severe cases, changes to their actual character.<sup>38</sup> These personality changes would be understood to have occurred, although the mechanism is not entirely clear, from a traumatic brain injury (after the initial acute symptoms had receded). However, an individual diagnosed with post-traumatic defect conditions would suffer from "gross lesions" of the brain including, but not limited to, losing brain tissue, problematic scarring, and destruction of glial cells (those responsible for protecting and nourishing neurons).<sup>39</sup> The symptomology of such physical destruction to the brain, according to Bowman and Blau, would include catatonic-like states, "lack of verbal or motor volition," and all types of "paraphasia," or speech disorders where the individual speaks in a jumbled manner.<sup>40</sup> It is important to understand Bowman and

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<sup>37</sup>. Bowman, "Psychotic States," 345.

<sup>38</sup>. Bowman, 362.

<sup>39</sup>. Bowman, 369.

<sup>40</sup>. Bowman, 370.

Blau's distinction of these two subcategories of chronic psychoses for the purposes of illustrating the last type of chronic disorders: those of a "mixed" nature including characteristics and symptoms of both.

"Chronic post-traumatic disorders of mixed types" is the final, and arguably most important, category presented by Bowman and Blau for the purposes of understanding the transhistoric nature of punch drunk, dementia pugilistica, and the like. The first disorder included under this heading, while lengthy, is quite telling: "*Traumatic Encephalopathy of Pugilists or The 'Punch Drunk' State*." Bowman and Blau present this encephalopathic disease as a "mental deterioration" occurring in individuals that participate in certain sports, namely pugilists and football, due to "repeated frequent head injuries."<sup>41</sup> The authors make a brief nod to the medical research history by noting that this disease was long known to the "lay public" but not "recognized by medical men as an authentic traumatic sequel until described by Martland."<sup>42</sup> The lay public's knowledge is presumably a reference to Martland's investigation and conclusion that spectators, officials, and athletes alike knew about potential long-term health repercussions involved with a boxing career – albeit, as mentioned by Bowman and Blau, without an official medical designation.

Here, Bowman and Blau reveal much about the enterprise of medical research during the 1940s, perhaps even without intending to. It may seem strange that an awareness of a causal effect, even if concerned parties lack a medical degree, between repeated hits to the head and long-term health consequences would be dismissed as

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<sup>41</sup>. Bowman, "Psychotic States," 373.

<sup>42</sup>. Bowman, 373.

inauthentic based on the source of that information alone. While we may consider a boxer suffering from an unknown condition or a boxing spectator that has seen numerous fights as informed, if unrefined, authorities, Bowman and Blau seem outright hesitant to give that cultural understanding much credence. This perspective treats the inclusion of a specific, named medical disorder into the larger medical lexicon as uniquely authoritative with regard to the understanding of that disorder. In other words, while Bowman and Blau are certainly not presenting some bastardization of an antirealist position, there is something to be said about the distinction between the “lay” public’s perception of a disease and that of a medical researcher. Undoubtedly, the disease referred to here as traumatic encephalopathy of pugilists, or ‘punch drunk,’ certainly existed as a real, definable biological entity prior to Martland’s work in the late 1920s. However, Bowman and Blau treat Martland’s seminal 1928 article as a paradigmatic shift between all time before 1928 where punch drunk did not exist as an identifiable disease and the twelve years between Martland’s article and Bowman and Blau’s chapter where the disease entity, or rather the variable nosological designation, underwent significant refinement, development, and progression. Indeed, the only relevant connection between the pre-1928 system of knowledge surrounding punch drunk and the period under Bowman and Blau’s purview is, as they state, Martland’s decision to keep the designation of “punch drunk” as the “most appropriate and descriptive of this condition.”<sup>43</sup> This historical connection, however, is only used by Bowman and Blau to keep the chain of progressive iterations alive and signify the previous, antiquated missteps in medical research.

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<sup>43</sup>. Bowman, “Psychotic States,” 373.

Bowman and Blau continue with their description of the punch drunk state by informing the reader that “no pathological studies have been presented” and that most information about punch drunk “has been accumulated from professional sportsmen.”<sup>44</sup> While presented without much fanfare, this information suggests that Bowman and Blau perceive punch drunk’s status as a strange amalgamation of folk-knowledge and limited professional research. Bowman and Blau are also presumably referring to Martland, Parker, and Millspaugh’s small sampling of case studies to help illustrate this otherwise mysterious disease – case studies being interesting but altogether inconclusive. And, again, Bowman and Blau’s stated objective with this chapter is to help illuminate the various types of traumatic brain injuries with regard to their status in tort cases nationwide. This objective has an overwhelming need to have its study of disease conclusive and authoritative.

To further contextualize punch drunk for the reader, Bowman and Blau return to previous talking points that present the disorder as localized within populations lacking skill in their respective sport. Boxers, for example, who use “their speed, coordination and skill defensively” are “exempt” from the condition.<sup>45</sup> Furthermore, one can readily expect “second rate fighters” who are used to train other skillful boxers to have a high occurrence rate of punch drunk. However, deviating from previous medical researchers, Bowman and Blau do estimate that 60% of all men that fight within the professional ranks for at least five years can expect to have at least a mild form of punch drunk,

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<sup>44</sup>. Bowman, “Psychotic States,” 373.

<sup>45</sup>. Bowman, 373.

regardless of proclaimed skill. About 5% will have “fairly advanced conditions” and an even smaller population will decline to the point of requiring institutionalization.<sup>46</sup>

Continuing to expand on the relationship between sports and related chronic injuries, Bowman and Blau estimate that the earliest signs of punch drunk manifest approximately four years after entering the professional leagues or within a range of 30 to 60 bouts (whichever comes first).<sup>47</sup> However, these early signs are limited to a “diminished tolerance” for blows to the head and a “less effective” defensive ability resulting in otherwise purely superficial physical changes (such as a flattened nose or “cauliflower” ears). Indeed, Bowman and Blau seem to consider such changes indicative of an early onset punch drunk syndrome. While certainly lacking sophistication, the logical leaps are easy to grasp: if particular physical deformities are caused by hard punches, and hard hits to the head cause punch drunk, then those physical deformities may be helpful visual cues in the process of diagnosing an individual. Bowman and Blau continue with an off-the-cuff examination of a hypothetical fighter’s psyche. Namely, one should expect, when examining a suspected fighter of suffering from a mixed type of chronic post-traumatic disorder, to encounter a boxer with an undiminished boastful pride without the record to match – or, in other words, punch drunk boxers will claim to be quite capable but begin “to lose engagements which previously he could have won with ease.”<sup>48</sup> Additionally, those around the boxer will begin to notice “mental changes,” including a deteriorated attention or memory, states of “reverie,” an aptness to become

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<sup>46</sup>. Bowman, “Psychotic States,” 373.

<sup>47</sup>. Bowman, 374.

<sup>48</sup>. Bowman, 374.

“too sociable and voluble,” and, of course, a tendency to “continuously resemble a person who is partially drunk.”<sup>49</sup>

While listing potential symptoms, Bowman and Blau also present a punch drunk designation built on a series of stages, each with increasingly severe symptoms. Or, a disease symptomology that is far more nuanced than previously understood. For example, Bowman and Blau explicate a far more severe constellation of symptoms as the disease progresses. Distinct from issues dealing with memory or a “partially drunk state,” punch drunk may also present itself with an individual’s voice becoming “guttural [*sic*]” “as though something is held in the mouth,” tremors, Parkinsonian-type characteristics, and deafness.<sup>50</sup> Interestingly, the authors note that headaches, dizziness, and tinnitus, “which one might expect as symptoms,” are only present for a short time after a bout and “rarely persist as a part of the syndrome.”<sup>51</sup>

These symptoms are followed in disease severity by “mental and personality” changes and, in some cases, a constant state of confusion. Emotional instability, failing the “simplest intellectual activities,” a complete lack of insight “regarding their mental state or abnormal conduct” and, additionally, “resent any application of this stigma to themselves.”<sup>52</sup> Although previously mentioned by Martland and other medical researchers, Bowman and Blau make no effort to further explain these last two symptoms. First, a lack of “insight” is an inherently difficult condition to characterize. If, as previously mentioned by Bowman and Blau, much of this condition’s knowledge has

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<sup>49</sup>. Bowman, “Psychotic States,” 374.

<sup>50</sup>. Bowman, 374.

<sup>51</sup>. Bowman, 374.

<sup>52</sup>. Bowman, 374.

“been accumulated from professional sportsmen,” it would likely be safe to assume those same sportsmen are sufficiently insightful into their own condition to relay that information to medical researchers. However, based on previous comments by Bowman and Blau, it may also be safe to assume that the authors fail to give a certain respect to these accounts, at least to satisfy the degree of insightfulness expected. Bowman and Blau simply do not expand on these characteristics (of either the disease or the patient) much beyond speculation – indeed, their intention may have been to call an awareness to the possible degree of severity one may suffer from punch drunk. In other words, a boxer’s mental state may deteriorate to the point to no longer recognize their own conditions.

Second, although it is interesting to note that professional pugilists supposedly resent the stigma associated with punch drunk (a sentiment shared previously by other medical researchers), it is altogether unclear whether Bowman and Blau intend this characteristic to be a symptom of punch drunk or symptomatic of a larger culture in which the boxers reside. Again, if boxers depend on the livelihood provided by professional pugilism, a label of punch drunk could indeed be disastrous. However, Bowman and Blau’s analysis leaves much to the imagination regarding this connection. Is this resentment related to the boxer’s mental deterioration? Would one only expect to find this resentment in the more severe stages of punch drunk? Bowman and Blau may be making a quick reference to previous studies of punch drunk but their treatment, in both this section and elsewhere, lacks a satisfying and substantive analysis.

The authors quickly conclude their inclusion of punch drunk as a chronic primary traumatic psychosis of a mixed type by mentioning an action plan “intelligent pugilists” ought to follow. First, the more intellectual pugilists, according to Bowman and Blau, will recognize “[punch drunk’s] early manifestations” and plan to retire from boxing “before ominous signs and symptoms appear.”<sup>53</sup> This advice is apparently offered with the intent to stall the degenerative nature of punch drunk in affected individuals – that is, the authors believe the degeneration of punch drunk will only continue in the presence of continued hits to the head. This is an interesting insight into the authors’ conceptualization of the disease entity itself. Whereas some researchers might believe punch drunk to be an internally-driven sickness, prompted by external events but degenerating continuously through internal processes, Bowman and Blau believe the opposite: punch drunk will only progress in the presence of head trauma and eliminating the head trauma will halt the progress of the disease.

In addition to offering advice to the boxing community, Bowman and Blau continue their admonishment of your typical boxing professional: “[T]he majority of boxers do not show this foresight and continue to fight until they become so ineffective that they cannot obtain further engagements.”<sup>54</sup> This lack of foresight is indubitably followed, from “economic necessity,” by the fighters changing their position within the sport without a complete exit. For example, some may enlist as sparring partners for other professional fighters – a career choice that will ensure continued punishment. Then, Bowman and Blau drive their point home: “This type of traumatic encephalopathy, which

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<sup>53</sup>. Bowman, “Psychotic States,” 374.

<sup>54</sup>. Bowman, 375.

is self-limited and not progressive may therefore show various degrees of deterioration depending on the extent and degree of the traumata.” Punch drunk, or this chronic variety of traumatic encephalopathy, has ascended the ranks from a “lay public” concern over amateurish boxers to an established medical designation, created and agreed upon by “medical men.” Bowman and Blau understand punch drunk as an intersection between “personality disorders” and “defect conditions” that include a wide array of possible symptoms, all resulting from vicious and continued hits to the head.

Although it is tempting to disregard the authors’ rebuke of the boxing community as implicit in their own condition as specious, it is important to remember that Bowman and Blau’s analysis of the boxer-as-subject is not historically unique. Martland, Parker, Millspaugh, and now the combined Bowman and Blau all regard the punch drunk condition as inherent to the boxing activity and thus the boxer as well. As far as the presented chain of medical researchers are aware, punch drunk, dementia pugilistica, and chronic varieties of traumatic encephalopathy are all inextricably tied to the boxing community and, extrapolated, unlikely to be found outside that same community. The treatment of boxers aside, Bowman and Blau’s major contribution to the transhistoric perspective of punch drunk was its inclusion in the presented constellation of primary and secondary traumatic mental disorders as a legitimate, albeit limited by several factors (career, class, etc.), medical designation worthy of continued research and care by future “medical men.”

## CHAPTER FIVE

CRITCHLEY: CHRONIC PROGRESSIVE TRAUMATIC  
ENCEPHALOPATHY

Bowman and Blau's contribution to the transhistoric history of CTE as a diagnostic category was to categorize the existing strands of medical research surrounding traumatic head injuries into digestible classifications (for the purposes of tort cases). Included in these classifications was punch drunk – determined to be a chronic, primary traumatic mental disorder of a mixed type between a personality disorder and a defect condition. In other words, chronic traumatic encephalopathy of pugilists (and all other nosological variations) was introduced into the medical lexicon as a legitimate medical designation. Referring to Asken et. al's 2017 timetable outlining the history of CTE, Bowman and Blau's milestone of "describing persistent symptoms in a retired boxer" using "chronic traumatic encephalopathy" is followed by a notable publication by Macdonald Critchley, a neurologist at King's College Hospital and a general physician at the National Hospital in London. In particular, Critchley's 1957 article "Medical Aspects of Boxing. Particularly From a Neurological Standpoint," originally read as an opening paper for a meeting of the Chelsea Clinical Society on November 13, 1956, is responsible for, among other things, suggesting that the medical community at-large formally abandon "punch drunk" and adopt "chronic progressive traumatic encephalopathy of boxers" as the official medical designation for this strange, but slightly less mystifying, disease.

Critchley introduces his topic with two compelling opening statements. First, Critchley indicates that “traumata” that occur in boxing bouts or throughout a boxer’s career are “diverse in character.”<sup>1</sup> Critchley is aware of the complex nature of traumatic head injuries, especially those in boxing, and intends his audience to share that awareness. Second, Critchley addresses a concern that presides over the 1957 discussion of traumata in sports – although some studies by his peers indicate that “boxing *per se* causes no more injuries or fatalities than some other sporting events,” boxing is unique in that the goal, either explicit or implied, is to “render the opponent *hors de combat*,” or incapacitated due to injury. While other sports may have injury as an accidental consequence of athleticism, boxing’s relationship to injury is rather intentional and purposeful. This relationship, Critchley believes, encourages his own investigation to “enumerate” some of boxing’s “principal ill effects” to better inform the broader public regarding the potential consequences of a lifelong pursuit of prize fighting. However, Critchley makes “no plea either for or against pugilism” and will let others decide “[w]hether the benefits of amateur boxing outweigh the drawbacks.”<sup>2</sup>

Critchley, before discussing traumatic brain injury, spends an inordinate amount of space dedicated to “The Knock-Out.” Considered “so dramatic a spectacle,” Critchley’s interest in the knockout seems to revolve around the complex mechanisms involved with a powerful punch to the chin, a relatively brief period of unconsciousness, and a “comparatively complete” recovery from all related symptoms.<sup>3</sup> Presumably,

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<sup>1</sup>. Macdonald Critchley, “Medical Aspects of Boxing, Particularly from a Neurological Standpoint,” *British Medical Journal* 1, no. 5015 (1957):357.

<sup>2</sup>. Critchley, 357.

<sup>3</sup>. Critchley, 357.

Critchley's attention to the knockout is its intimate relationship with brain injury – or, being knocked out is possibly a traumatic brain injury in and of itself. Included in this analysis of the knockout may be an implicit plea to reexamine the boxing and medical community's understanding of a knockout's complete recovery.

Critchley and other neurologists' primary concern is in "experienced boxers," either professional or amateur, that have "undergone gradual physical and mental deterioration as a direct accumulated result of their careers."<sup>4</sup> The popular term for this deterioration, Critchley notes, is "punch-drunkenness." As with Bowman and Blau previously, Critchley briefly mentions punch drunk's likely origin or probable coiner as a "layman" but being introduced into the medical literature by Martland's 1928 paper. Since that paper, Critchley argues, punch-drunkenness has become "well known to the laity," developed "certain pejorative – almost derisory – connotations," and become synonymous with "goofy" and "slug-nutty." It is difficult to know whether Critchley's analysis is being performed concurrently with the 1950s or with Martland's 1928, however. Martland also noted many of these negative sentiments towards boxers that develop the punch drunk syndrome and discussed, albeit briefly, the larger cultural perception of the mysterious disease. Is Critchley simply echoing Martland's decades previous concerns or have those same sentiments persisted through time (especially in the boxing community)?

Evidently motivated by these negative connotations, the disease itself, Critchley notes, has gone through multiple nosological iterations since Martland's publication.

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<sup>4</sup>. Critchley, "Medical Aspects of Boxing," 358.

These “more esoteric and less offensive expressions” include “traumatic encephalopathy (Parker),” “dementia pugilistica (Millsbaugh),” and other variations not previously mentioned.<sup>5</sup> However, Critchley continues, “If we really require a suitable scientific alternative to ‘punch-drunkenness’ we may perhaps adopt the term ‘chronic progressive traumatic encephalopathy of boxers.’”<sup>6</sup> No explanation is given for Critchley’s suggested alternative. A lack of explanation, however, does not preclude an inherent historical significance – there is certainly some interesting historical analysis hanging off this nosological convention. For example, Bowman and Blau previously introduced “chronic traumatic encephalopathy” through a categorization of known traumatic brain injuries and a single case study’s diagnosis. Critchley’s dependence on Bowman and Blau’s work is simply the continuance of a medical and historical precedent. “Of boxers,” however, is an interesting inclusion. To date, every medical researcher dealing with punch drunk or its variants have indicated their acute awareness of the disease’s localized nature within the boxing community. Indeed, previous researchers have even stated that being a boxer is a necessary condition to acquire the disease – any other occupation or recreational activity simply will not suffice. The added modifier to the disease, then, at least without further input from Critchley, is unusually redundant. Finally, and perhaps the most significant suggestion by Critchley’s nosology, is the potential “progressive” nature of the disease. Bowman and Blau specifically noted chronic traumatic encephalopathy’s lack of continued degeneration, or progression, if one were to stop the offending activity

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<sup>5</sup>. Critchley, “Medical Aspects of Boxing,” 358.

<sup>6</sup>. Critchley, 359.

(in this case, boxing). Critchley's analysis or addition to the surrounding medical knowledge may indicate otherwise.

Critchley assures his reader, and the larger medical community, that his opinion, regarding both the nature of the disease and the naming conventions, is indeed expert: “. . . my own neurological experience has comprised a series of 69 cases of chronic neurological disease in boxers” and that the “great majority” of those cases ought to be considered examples of “punch-drunkenness.”<sup>7</sup> To accompany this nosological change is an account, presumably based on his many case studies, of the symptomology of chronic progressive traumatic encephalopathy of boxers, some of which contrasts harshly with previous accounts. For example, Critchley claims that punch-drunkenness is “much commoner among professionals than among amateurs” and that the disease is “very rare in coloured boxers.”<sup>8</sup>

The former statement, regarding professional boxers, is in direct conflict with nearly every account of punch drunk thus far. What compels Critchley to argue the occurrence of punch drunk in professional boxers as significantly higher is not clear – perhaps the majority of his own 69 accounts conform to this analysis. While speculative, it may be the case that the nature of professional bouts is more conducive to the “knockouts” mentioned previously by Critchley. If Critchley's understands punch drunk to have an important causal relationship to the particular trauma caused by knockout hits, as opposed to any type of impact trauma regardless of a loss of consciousness, then the type of spectacle found in prize-winning, professional bouts may seem a natural, if

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<sup>7</sup>. Critchley, “Medical Aspects of Boxing,” 359.

<sup>8</sup>. Critchley, 359.

slightly biased, inclusion in his theory. The latter statement, however, regarding the occurrence of punch drunk in non-white populations is mystifying. No evidence is offered in support of this claim. Furthermore, it is not entirely clear whether “coloured boxers” were not a representative group in Critchley’s body of work (leading him to this conclusion), if Critchley is arguing that punch drunk, as a disease entity, cannot physically occur in the brains of non-white athletes, or if some other unmentioned factor influences the occurrence of punch drunk in these populations (economic, cultural, etc.,).

Regardless of Critchley’s brief racialized tangent, he concludes that, ultimately, two numbers are of utmost importance regarding the development of the disease: the number of total contests and the number of times knocked unconscious.<sup>9</sup> Given an ample amount of both, one would expect to find several “clinical features which underlie all the diverse patterns of the disease.”<sup>10</sup> While generalized as “evolution of mental and physical anomalies” marking the “onset of encephalopathy,” some specific symptoms include “euphoric dementia,” little to no “insight into [their] deterioration,” decline of speech and thought, and truculent, “uninhibited violent behavior.”<sup>11</sup> Critchley, unlike Bowman and Blau, expand on the unique, but supposedly ubiquitous, characteristic of a punch drunk diagnosis: the “victim,” or patient’s, inability to communicate their symptoms to medical professionals. Critchley’s “clinical findings” suggest that the patient “may spontaneously admit to but few disabilities, if any at all.” Furthermore, this inability to report their own

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<sup>9</sup>. Critchley, “Medical Aspects of Boxing,” 359.

<sup>10</sup>. Critchley, 359.

<sup>11</sup>. Critchley, 359.

mental or physical ailments suggests an “insidious development” and a “lack of awareness so characteristic of the dementing patient.”<sup>12</sup>

While Bowman and Blau presented this interesting element of the punch drunk diagnosis as an insight into the personality of those who tend to box in the first place (i.e., uneducated and brutish), Critchley suggests a lack of adequate communication in response to medical inquiries as, perhaps, a mental deficiency caused by the trauma, not other extenuating circumstances. This would give a considerable amount of due respect to the boxing community’s apprehension of their own physical and mental health – something that, prior to Critchley’s analysis, had not been afforded to them. It is important to consider, again, the financial boon that prize fighting was to some people. Combined with the enduring connotations surrounding punch drunk, the physical and mental deterioration proving an otherwise capable fighter as inept would be infuriating, perhaps even hopeless. A lack of motivation to be forthcoming would certainly be expected given those feelings combined with the nosy investigations of outside medical researchers intending to publicize their results. While Critchley’s comments are limited to an appreciation of the relationship between mental health and a willingness to cooperate with medical researchers, it is certainly a significant change in tone when compared to his predecessors.

Critchley then presents eleven case studies with the purpose of arriving at his treatment of “Traumatic Encephalopathy,” especially in conjunction with punch-drunkness. Or, the medically and professionally acceptable alternative to punch-

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<sup>12</sup>. Critchley, “Medical Aspects of Boxing,” 359.

drunkenness. Note, however, that the disease itself has changed very little in meaningful terms. Indeed, Critchley's nosology is presented as an aside to accompany the main thrust of his contribution – expanding on our understanding, rather than fundamentally changing, the traumatic encephalopathy in the wake of knockouts.

Perhaps one of the most enigmatic features of chronic traumatic encephalopathy, at least for Critchley, is the frustrating dormancy of the disease. While Critchley is vaguely aware that the disease takes a “matter of years” to manifest, a previous study conducted by himself revealed, out of “21 punch-drunk naval patients,” the period between the start of a boxing career and the onset of neurological symptoms averaged sixteen years.<sup>13</sup> However, the range of those periods could be as little as six years and as many as forty. It is certainly difficult to establish definitive characteristics of a disease with such a lengthy incubation period, only exacerbated by the seemingly countless agitators towards complete incapacitation.

In addition to a less-than-complete picture of chronic traumatic encephalopathy's expected manifestation in patients, Critchley also notes that “traumatic encephalopathy is a progressive condition” and that “once established it not only does not permit of reversibility, but it ordinarily advances steadily.”<sup>14</sup> The context of Critchley's comments here seem to indicate that the disease itself has a propensity to progressively worsen as opposed to previous accounts that linked a worsening disease to continued head trauma. The staggering implication of such a significant change to the disease outlook cannot be overstated, even if Critchley only devotes a paragraph to it. Before Critchley, chronic

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<sup>13</sup>. Critchley, “Medical Aspects of Boxing,” 360.

<sup>14</sup>. Critchley, 360.

traumatic encephalopathy, or punch drunk, was exclusive to the boxing community – not only did the individual need to be a boxer to develop the disease, the progression of the disease was dependent on continued experience in the boxing arena. While Critchley’s opinion does not deviate from the assumption that more boxing experience necessarily leads to a worsened state of punch-drunkenness, to allow chronic traumatic encephalopathy to worsen without further environmental input from external forces opens the proverbial floodgates for who, or what community, can suffer from this disease. For example, a one-off traumatic injury to the head early in life, by Critchley’s own analysis, could develop, under the right conditions, into a full-blown chronic traumatic encephalopathic diagnosis. In other words, Critchley has taken a boxing disease and made it a human disease found most often in occupations with the most traumatic head injuries.

Critchley concludes rather anti-climatically with an appeal to “practising [*sic*] neurologist[s]” that there is much interest in the sport of boxing. Particularly, the “phenomenon of groggy states” that occur after a boxing bout and “the condition known as traumatic progressive encephalopathy (or punch-drunkenness).”<sup>15</sup> However, Critchley admits that “pathological data” is scarce regarding the condition and any medical researcher willing to investigate could prove a boon to the entire medical enterprise. Or, in other words, Critchley wraps up his investigation and nosological analysis with a plea to both contemporary and future researchers: take what work has been done by Martland,

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<sup>15</sup>. Critchley, “Medical Aspects of Boxing,” 362.

Parker, Millspaugh, Bowman and Blau, himself, and a myriad of other medical researchers and continue to improve.

While who and what Critchley decides to reference in his own medical analysis of chronic traumatic encephalopathy is not necessarily noteworthy in and of itself, Critchley's cognizance of the surrounding medical literature certainly seems to inform many of the nosological changes he advocates for. For example, while Critchley references the nosology of Parker, Millspaugh, and others, he fails to reference, or even mention, the medical research done by authors that presumably underlays their respective designations. Parker's "traumatic encephalopathy" is included as a "more esoteric" attempt to fruitfully and fairly describe both the ailment and those who ail from it but Parker's "Case I," and, indeed, his entire collection of medical knowledge from the 1934 article, is suspiciously absent. It is difficult to say whether this is indicative of the state of medical research during Critchley's decade or informs the reader more of Critchley's individual approach towards the continuation of a transhistoric entity. Or, we might ask why Critchley determined Parker's nosological contribution, something that has been presented time and time again as simple alternatives, more important than his research into the symptomology of a patient. Referring to Critchley's entire article in the *British Medical Journal*, only Martland's 1928 article is used, albeit for context and introductory notes, to establish a "history" of medical practice. However, nearly every author presented here was used by Critchley to establish a transhistoric understanding of 20<sup>th</sup>-century medical research, especially that concerning chronic traumatic encephalopathy.

## CHAPTER SIX

## CORSELLIS, ROBERTS, AND OMALU: CTE AND FOOTBALL

Before explicating the relationship between the transhistoric entity of CTE and tort law, it is important to detail a few additional case studies that take the nosology of CTE from Critchley's 1957 to the present. Although the medical designation, in the "post-Critchley" era, is relatively stable, significant changes are still taking place in the late 20<sup>th</sup>-century that fundamentally change our understanding of the disease. While not examined here, it should be noted that the technological innovations occurring in the medical field during this period eclipse nosological considerations regarding uncovering the mysteries of this still relatively unknown disease entity. Medical researchers are beginning to extend their investigation beyond case studies through increasingly sophisticated techniques and technologies. While Martland, Millsbaugh, and Parker's case studies greatly informed their opinions and medical designations, we begin to see the physicality of the human brain inform medical research more than the first-hand account of individual patients. While Martland was concerned with the "slug nutty" behavior of boxers, researchers in the 1970s and beyond begin to weigh the deceased's brain, measure the dilation of ventricles (or a "Naked-Eye Appearance of Brain"), and encounter unknown, but suspicious, "tangles" in the grey matter of suspected cases of CTE.<sup>1</sup>

John Corsellis et al.'s "The Aftermath of Boxing" (1973) is a significantly formative publication in the history of CTE. Corsellis' study is "not concerned with the

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<sup>1</sup>. J. A. N. Corsellis et al., "The Aftermath of Boxing," *Psychological Medicine*, 3, (1973): 272.

sudden lethal catastrophe that happens rarely during a boxing contest” but with “an inquiry into the nature of any abnormalities” in the “brains of boxers who have died after retiring from the ring.”<sup>2</sup> Corsellis mentions the transhistorical legacy presented here with a brief nod to Martland’s 1928 introduction of “punch-drunk into the medical literature.”

Corsellis references Martland’s work and, specifically, the “controversial question” regarding “the possibility that lasting cerebral damage might occur” with repeated blows to the head, an unavoidable consequence of a career in boxing. Corsellis’ contention is that the nature and existence of “structural changes” to the brain is dependent on a “pattern of damage” brought forth in relevant studies – studies that, Corsellis admits, are rare.<sup>3</sup> The state of research and understanding in 1973, Corsellis argues, regarding the “vulnerability” of the brain, “are still based more on supposition than on fact.”<sup>4</sup> Furthermore, Corsellis remarks at the bifurcation of the “punch-drunk syndrome”: a sizable contingent of so-called authorities have argued that boxing has no “appreciable risk to the brain” while most medical experts, according to Corsellis, “express their opinions more cautiously.”<sup>5</sup>

To illustrate this division in disease understanding, Corsellis includes some statements from fellow medical researchers to grant a clearer perspective of the field. For example, Corsellis includes the following quote from I. A. McCown, another expert investigating punch drunk: “The acute cerebral trauma and its sequelae, the chronic post-traumatic encephalopathy, have long been considered the most frequent serious

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<sup>2</sup>. Corsellis, “The Aftermath of Boxing,” 270.

<sup>3</sup>. Corsellis, 270.

<sup>4</sup>. Corsellis, 270.

<sup>5</sup>. Corsellis, 297.

complication of boxing.”<sup>6</sup> Corsellis also includes McCown’s opinion that “punch-drunk syndrome has become symbolic of brain injury” and, in essence, is emblematic of the entire sport. However, not all researchers are as direct as McCown, an issue Corsellis is acutely aware of. In addition to McCown’s statements, A. D. Wright’s professional statements are used to represent a more conservative approach towards the punch-drunk-to-boxing relationship. Wright is quoted as believing that there is “some danger” in boxing but there is no justification for “doing away with the sport” entirely.<sup>7</sup> As Wright argues, many, if not all sports, could be “eliminated” on the basis of a concern for the athletes’ health. Corsellis’ proposed solution to this bifurcation is to neuropathologically examine the brains of fifteen ex-boxers that were collected from 1957 to 1973 in order to catalog potential structural changes resulting from a career in boxing.

The conclusion for Corsellis was incontrovertible: those fifteen brains, all with a varying degree of structural damage, established a “solid foundation” to claim that “some experienced boxers develop a clinical disorder, the greater part of which has a neuropathological basis.”<sup>8</sup> While Corsellis was concerned that other factors may have influenced the state of his subjects’ brain, such as alcohol, old age, and other non-boxing related injuries, the background of the patients was such that the “paramount reason for the insidious neurological and psychological deterioration” of the patients was the “brain damage incurred while boxing.”<sup>9</sup> However, Corsellis could not provide an explanation for mechanisms that manifested more severe symptoms. For example, structural changes to

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<sup>6</sup>. Corsellis, “The Aftermath of Boxing,” 297.

<sup>7</sup>. Corsellis, 297.

<sup>8</sup>. Corsellis, 301.

<sup>9</sup>. Corsellis, 299.

the brain resulting from a lifetime of boxing provided a plausible explanation for a myriad of symptoms, ranging from “mild clumsiness” and “loss of memory” to debilitating “dement.”<sup>10</sup> Beyond mystification at the underlying mechanisms of symptomatic manifestation, Corsellis remarks that, “A single punch . . . to the head need not visibly alter the structure of the brain but there is still the danger that, at an unpredictable moment and for an unknown reason, one or more blows will leave their mark.”<sup>11</sup> As much as Corsellis engaged with the surrounding medical literature and knowingly built upon the history of this disease, he ought to be understood in the context of, while revolutionary, being reworked and contributed to as the transhistoric nature of this latent entity is passed along in the relay race of science.

Progressing beyond Corsellis’ publication, with a title that pays homage to that study, Gareth W. Roberts et al.’s “The Occult Aftermath of Boxing” (1990) refers to a “classic study” that describes the “neuropathology of the condition” related to the “trauma to the head experienced by boxers.”<sup>12</sup> The bibliographical reference for this “classic study” is, unsurprisingly, Corsellis et al.’s “The Aftermath of Boxing.”<sup>13</sup> Moreover, the first sentence of Roberts’ article references Martland’s 1928 publication but refers to the disease itself as “dementia pugilistica,” abbreviated “DP.”<sup>14</sup> A large portion of the article is focused on the symptomatic similarities between dementia pugilistica and Alzheimer’s disease –

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<sup>10</sup>. Corsellis, “The Aftermath of Boxing,” 301.

<sup>11</sup>. Corsellis, 301.

<sup>12</sup>. Gareth W. Roberts et al., “The Occult Aftermath of Boxing,” *Journal of Neurology, Neurosurgery, and Psychiatry*, 53, (1990): 373.

<sup>13</sup>. Roberts, 378.

<sup>14</sup>. Roberts, 373.

two diseases that Roberts suspects are intriguingly similar and understanding one may lead to innovations in the pathology of the other.

Roberts, unlike our previous authors, has no qualms with describing DP as progressive. Like previous authors, however, Roberts also creates a causal link between the severity of the disease and either career length or “total number of bouts.”<sup>15</sup> Given a significant length of career or number of bouts, the disease can manifest in three stages of “clinical deterioration:” first, the patient is afflicted with “affective disturbances and psychotic symptoms;” second, the patient becomes socially unstable and a number of psychiatric symptoms, including memory loss and signs of “Parkinsonism;” finally, an abrupt drop in “cognitive functioning” including inevitable dementia.<sup>16</sup> These three stages are presented as both progressive and temporally unstable. Or, while the disease will progressively get worse without external influences (although continuing to participate in the sport will exacerbate the condition), the worsening of DP occurs in distinct stages of severity.

The major contribution of Roberts’ analysis is the examination of “neurofibrillary tangles” that occur in the brain of patients suffering from a number of diseases. While Alzheimer’s disease (AD) is the most common disease with such tangles, Roberts includes the remarkable similarity between tangles in Alzheimer’s patients and those boxers with DP. Indeed, Roberts’ analysis concludes that his DP brains have tangles that are “structurally and immunologically indistinguishable from the tangles which are thought to

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<sup>15</sup>. Roberts, “The Occult Aftermath of Boxing,” 373.

<sup>16</sup>. Roberts, 373.

be central to the disease process in AD.”<sup>17</sup> It is not too far a stretch, then, to assume that the aetiology for one disease may prove insightful for the other. For the first time in nearly 60 years of transhistorical development of CTE as a diagnostic category, the tissue of the brain has become paramount to understanding the pathology and disease outlook of this disease. While the brain has been an important object for the study of punch drunk, DP, CTE, etc., for the entire history of the disease, Corsellis’ investigation prompted the investigation of the changes to the structure of the brain and Roberts et al. have now created the precedent to investigate the microscopic changes to the brain in the event of repeated head trauma.

Now that Roberts et al.’s analysis opened the diagnostic possibility of an important, and biologically similar, link between Alzheimer’s disease and dementia pugilistica, many medical researchers post-Roberts attempted to solidify the link. J.F. Geddes et al., for example, in the article “Neuronal Cytoskeletal Changes are an Early Consequence of Repetitive Head Injury,” published 1999 in *Acta Neuropathol*, attempts to establish the “early histological changes caused by the repetitive trauma that eventually produces dementia pugilistica” to further understand DP as a disease entity.<sup>18</sup> Geddes’ introductory notes include a reference to previous medical studies, such as those performed by Roberts et al., stating, “Detailed post mortem studies of elderly retired boxers suffering from dementia pugilistica have shown a neuropathological picture very like that of Alzheimer’s disease.”<sup>19</sup> In particular, Geddes is interested in the “widespread neocortical neurofibrillary

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<sup>17</sup>. Roberts, “The Occult Aftermath of Boxing,” 373.

<sup>18</sup>. J. F. Geddes, “Neuronal Cytoskeletal Changes are an Early Consequence of Repetitive Head Injury,” *Acta Neuropathol* 98, no. 2 (1999): 171.

<sup>19</sup>. Geddes, 171.

tangles” that the brains of dementia pugilistica patients have been found to have, a phenomenon very similar to that of the brains of Alzheimer’s patients.

Without much fanfare, Geddes sheds much of the historical methodology that his medical predecessors were beholden to and drastically widens the scope of dementia pugilistica, or Geddes’ interchangeable use of “chronic encephalopathy.” Not only is chronic traumatic encephalopathy neuropathologically similar to Alzheimer’s, Geddes reports that clinical studies have been performed on participants in other sports that see “repeated mild head injury” and concludes that chronic traumatic encephalopathy is not a disease exclusive to boxing. Additionally, Geddes notes a recent case study in which a “demented woman” who had been suffered a lifelong history of physical abuse had “pathology identical to that seen in boxers.”<sup>20</sup> Geddes’ main point is that while the “morphological similarity” between AD and CTE is such to warrant serious comparative studies, and that the “pathogenic mechanisms” must be similar between the diseases given the similar tangles found in the brains of both types of patients, the “progression” of changes caused by repeated hits to the head is not well understood (unlike CTE’s counterpart, AD, which is well understood).<sup>21</sup>

While not necessarily apropos to the transhistorical understanding of CTE, Geddes’ chosen case studies deserve a brief mention. While most of our previous medical researchers, ranging back to Martland in 1928, have used current and previous boxers to illustrate their points, Geddes et al. use a wide range of subjects to potentially illustrate the wide-ranging and devastating effect that CTE can have. Indeed, while certainly destructive

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<sup>20</sup>. Geddes, “Neuronal Cytoskeletal Changes,” 171.

<sup>21</sup>. Geddes, 171.

within the boxing community, being limited solely to the that community understates the shared biological truth inherent in all humans – we all possess a brain equally susceptible to trauma caused by physical impacts. Geddes' first case is a professional boxer, age 23, who died unexpectedly after his first and only severe head injury after four years of professional bouts. Likewise, the second case is a former boxer, age 28, who had been an amateur boxer since leaving school at sixteen. Dying of a grand mal seizure after spending many years in and out of psychiatric facilities, Geddes notes that he suffered a "history of psychotic illness."<sup>22</sup> Unlike the first two cases, however, the third case focused on a "mentally subnormal man," age 28, who had a "long history of head banging" that died from complications related to jumping from a first floor window.<sup>23</sup> Additionally, the fourth case was a "mentally retarded" man that suffered from a lifetime of seizures, including subsequently dropping "heavily to the ground" many times a week.<sup>24</sup> Lastly, Geddes' fifth case, was a 23-year-old soccer player who "regularly headed the ball while playing" and died of brain swelling after sustaining a head injury during a football match.<sup>25</sup> Geddes' included case studies are surprisingly wide ranging and inclusive – although none of the presented case studies are women, the deceased patients' history included a wide variety of sources for repeated head trauma including, but not limited to, boxing.

Geddes concludes confidently: the five case studies presented, after extensive posthumous examinations of the brain, show that "chronic mild repetitive head injury" leads to an "early age" of "definable pathological changes," seen most notably as

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<sup>22</sup>. Geddes, "Neuronal Cytoskeletal Changes," 172.

<sup>23</sup>. Geddes, 172.

<sup>24</sup>. Geddes, 172.

<sup>25</sup>. Geddes, 172.

neurofibrillary tangles (NFTs). While the pathology of CTE is remarkably similar to that of AD, Geddes remarks that the mechanism behind the manifestation of the NFTs is unknown. There is, in other words, some hidden reaction happening in the brain between head trauma and the creation of NFTs that needs to be better understood both for the prevention of CTE and the treatment of those afflicted. Additionally, Geddes cannot adequately remark on the possibility of “cytoskeletal changes” caused by a “single episode of severe head trauma” (as opposed to the repetitive nature of head trauma discussed in his analysis). Or, while most of the historical discussion regarding CTE has focused on the repetitive and temporal nature of brain trauma (possibly motivated by the sport of boxing), there is a possibility that CTE has a knowable relationship between a single, severe impact and disease. While the disease certainly takes time to develop and occurs in the presence of trauma to the brain, the sufficient amount of trauma required to provoke the disease is relatively unknown.

The last important body of evidence to observe is that of Bennet Omalu’s work on CTE and, more generally, concussion. While Omalu’s work spans many decades, most of the controversy surrounding the coverup by the NFL was sparked by a 2005 article, published in *Neurosurgery*, titled “Chronic Traumatic Encephalopathy in a National Football League Player.”<sup>26</sup> Omalu et al.’s objective in this study was to examine the brain of a single retired professional football player for signs of long-term traumatic injury.

Omalu introduces the topic by referring to the numerous NFL players who have prematurely retired due to “postconcussion syndrome,” or a disease caused by repeated

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<sup>26</sup>. Bennet Omalu, “Chronic Traumatic Encephalopathy in a National Football League Player,” *Neurosurgery* 57, no. 1 (2005).

concussions.<sup>27</sup> While Omalu notes that the NFL's "Committee on Mild Traumatic Brain Injury," established in 1994 to study the trend of players leaving the league from concussion-related injuries, is ostensibly looking out for the players' best interests, the results leave many in the medical field wanting. The Committee replaced the postconcussion syndrome with "mild traumatic brain injury," or MTBI for short, but the symptomology remains largely the same: MTBI presents with "alterations of awareness or consciousness" and includes symptoms such as persistent headaches, loss of balance, unsteadiness, cognitive dysfunction, and others.<sup>28</sup> Despite the Committee's recommendation, made in 1995, for the NFL to fund independent research regarding the causes of MTBI, Omalu notes that the "long-term cognitive and neurodegenerative" qualities of the disease are "little understood."<sup>29</sup> Omalu et. al's 2005 study presents the "first documented case of long-term neurodegenerative changes in a retired professional NFL player consistent with chronic traumatic encephalopathy."<sup>30</sup>

The patient, a 50-year-old ex-NFL player, had been retired from play for approximately twelve years, suffered from a deficient memory and "parkinsonian symptoms," and died suddenly of a myocardial infarction (heart attack).<sup>31</sup> Upon the completion of an autopsy, Omalu found evidence of NFTs to confidently confirm the CTE diagnosis. He offers a potential explanation for the mechanism behind CTE: the "pathological mechanisms for these delayed posttraumatic changes are though to be

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<sup>27</sup>. Omalu, "Chronic Traumatic Encephalopathy,"128.

<sup>28</sup>. Omalu, 128.

<sup>29</sup>. Omalu, 129.

<sup>30</sup>. Omalu, 129.

<sup>31</sup>. Omalu, 129.

biochemical cascades that are induced by cumulative effects of repeated low-grade concussive brain injury.”<sup>32</sup> In other words, trauma to the brain interrupts the normal functioning of brain cells and a progressive decay takes place, rendering affected areas of the brain permanently altered.

Omalu notes, however, that the majority of studies regarding brain injury in athletes has taken place either while the athlete is a participant in the sport, or relatively quickly after retiring (either from a growing list of symptoms or from death). What has not been studied, and indeed Omalu is attempting to address, is the effect of repetitive head trauma over a length of time not previously considered. While the case study presented here is a mere twelve years after retirement, Omalu and other medical researchers are concerned that the life-long effects of repetitive head trauma will cascade into a national epidemic once players (or any individual with a lifestyle involving potential brain injuries) start living longer and longer lives. However, Omalu concludes, despite the seemingly obvious link, that there is no “causal link between professional football and CTE.”<sup>33</sup> Omalu’s case study is intended to be a signal to other medical researchers and institutions that a “comprehensive cognitive and autopsy-based research” effort is needed on “long-term postneurotraumatic sequelae of professional American football.”<sup>34</sup> Indeed, Omalu laments the woefully sparse body of medical knowledge informing the relationship between CTE and NFL players.

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<sup>32</sup>. Omalu, “Chronic Traumatic Encephalopathy,”131.

<sup>33</sup>. Omalu, 132.

<sup>34</sup>. Omalu, 132.

While the sequence of medical researchers from Martland to Corsellis have emphasized the researcher-to-researcher transhistorical link, the post-Corsellis age of punch drunk, dementia pugilistica, and CTE have emphasized the empirical, “autopsy-based” sourcing of information to continuously improve our understanding of the disease. However, none of the modern medical researchers have been able to completely or satisfactorily disregard the effort of their predecessors. The work of Omalu, for example, cannot be presented to the reader without an interlude, albeit brief, into the historical context of the disease. The reason for this continuously reinforced relationship is simple: although Martland, Parker, Corsellis, Omalu, and others referred to, and sometimes debated, the nosological significance of the disease, each researcher inherently understood that their object of study was the same and that their research would ultimately continue on an upward, progressive trajectory towards complete mastery of this biological phenomenon. Omalu’s contribution was to take this “boxing” disease and remind the medical community that any individual subjected to sufficient trauma is susceptible, on a long enough time scale, to the insidiousness of CTE. To point, the community of retired NFL players, growing ever older by the day, are starting to present concerning symptoms and a call to action is necessary, on behalf of the medical community, to rectify the gap in knowledge. While Martland’s “slug nutty” boxers and Omalu’s retired football player are separated by time, culture, nosological conventions, and drastically different paradigms in scientific and medical understanding, they both suffer from, at least to medical researchers like Omalu, the same disease.

## CHAPTER SEVEN

## CONCLUSION

In response to the National Institute of Neurological Disorders and Stroke and the National Institute of Biomedical Imaging and Bioengineering (NINDS and NIBIB, respectively) conducting a series of panels to “define the neuropathological criteria for CTE,” Ann McKee published a medical consensus that publicly outlined the research progress of the disease. With an “Introduction” that touches on Martland’s 1928 article, mentions the largely semantic change to “dementia pugilistica,” and the “seminal 1973 monograph” “of dementia pugilistica in 15 former male boxers” by Corsellis et al., McKee races through the history and varying “histological techniques” related to CTE with astounding ease.<sup>1</sup> It is, after all, a story with a familiar beginning that has been told many times. The difference with this story, however, is that the criteria created by the NINDS/NIBIB is the “beginning of the process to fully characterize the pathology of CTE,” presumably as a distinct entity.<sup>2</sup> These criteria are at the foundation of the aforementioned “Injury Definitions” provided by the NFL settlement. Again, a monetary payment per the conditions of the case is dependent on a “post-mortem diagnosis” “made by a board-certified neuropathologist.”<sup>3</sup> A neuropathologist is beholden to the consensus of the medical community, a consensus created by scholars like McKee and organizations

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<sup>1</sup>. Ann C. McKee et al., “The First NINDS/NIBIB Consensus Meeting to Define Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy,” *Acta Neuropathologica*, 131, (2016): 76.

<sup>2</sup>. McKee, 83.

<sup>3</sup>. “Reference Guides.” Accessed October 15, 2017.

[https://www.nflconcussionsettlement.com/Reference\\_Guides.aspx](https://www.nflconcussionsettlement.com/Reference_Guides.aspx)

like the NINDS that, as shown, rely on the transhistoric conception of disease. Martland, Parker, Corsellis, McKee and all other researchers with findings relevant to the development of CTE as a diagnostic category fit in to the narrative of the transhistoric conception of disease and their methodology has justified the rulings dealt by the courts.

A culturalist position would not functionally be able to draw the same connections. Even if the stratified constructions of historical relationships are inherently an important change to the discipline of history, it is obvious that a causal and progressive chain of events is necessary to conceive of a consistent set of demands by the court. The set of afflicted players do not necessarily have the luxury of being stricken during the last few years – the range of dates involved stretches back in to the 1980s when player safety was not of the same concern as is currently held. As shown, however, the 1980s understanding of disease does not neatly relate to the 2017 understanding of disease from the culturalist approach. If, for example, Loughran’s approach to PTSD is adopted in the case of CTE, it becomes clear that “dementia pugilistica” and CTE exist as separate diagnostic categories. This would dictate, per the strict conditions of the settlement, that a player diagnosed with dementia pugilistica would not receive a payout, regardless of the biological truth of that historical conception of disease’s relationship to our modern understanding of CTE.

A potential solution to this practical problem of incorporating the culturalist position in the court proceedings would be to include all historically relevant nosological variants of the disease entity in the court’s decision. Or, the “Injury Definitions” document that establishes the guidelines for distributing payment per disease diagnosis would include subcategories for dementia pugilistica, punch drunk, and others. This approach, however,

has two significant problems. First, the relationship between CTE and the historically related diagnoses would still need to be explained. Including the diagnostic categories required of the culturalist approach would not be sufficient to distinguish itself from the transhistoric approach if the culturally-informed categories were only included to further contextualize the CTE diagnosis. Punch drunk, for example, would need to be a separate diagnosis that a board-certified physician, per the conditions of the settlement, would be willing to apply to a patient. As shown, however, the motivation to distinguish between punch drunk and CTE in a modern context does not exist in the medical community – they are the same disease with one being the product of a greater degree of sophistication.

A second inescapable issue of simply including all possible nosological variations of CTE is that the culturalist approach is not a monolithic perspective regarding what is considered a unique disease entity. While Loughran and Wailoo take relatively conservative culturalist approaches, limiting their investigation to single instances of historically complicated diseases, other theorists implicated in the culturalist designation do not. Annemarie Mol, author of *The Body Multiple*, for example, argues that for every instance of a new laboratory, researcher, or generalized apparatus that investigates atherosclerosis, a new disease entity is created in the process.<sup>4</sup> Two different medical personnel then, ostensibly researching the same disease, actually create two separate varieties of atherosclerosis through the process of ordinary research. Applied to the NINDS criteria for CTE's use in subsequent legal battles, we can see a dire situation emerge. There would potentially be no theoretical limit to the number of CTEs, to say nothing of all the

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<sup>4</sup>. Annemarie Mol, *The Body Multiple: Ontology in Medical Practice* (Duke University Press, 2002).

historically relevant diagnoses discussed here. Using some varied examples from the culturalist perspective, we see that the attempt to provide social justice for maligned parties would fail if the culturalist position was included in the framework of the tort case. It would appear as if the culturalist approach, especially within the history of medicine, has dire and unforeseen consequences that require further contemplation.

The relationship between each researcher on the transhistoric timeline presented here is underlaid by a mutual understanding that each iteration of disease designation is fundamentally and inherently incomplete – that is, only so much work can be done in a given monograph or scholarly article and it becomes the responsibility of future researchers to fill in otherwise mysterious aspects of a given disease. Martland’s efforts to elucidate a unique aspect of the consequences seen by the sport of boxing during the 1920s was not limited to Martland himself. In the century-long relay race of medical research pertaining to CTE, each individual on the chain of effect understood the impact of their work both on previous and future researchers. For example, while many medical experts during the mid to late 20<sup>th</sup>-century appreciated the work done by Martland (at least in the professional tone of an article in a medical journal), very few researchers took the opportunity to laud Martland’s nosology. “Punch drunk,” while an effective and creative designation sourced directly from the community directly related to the disease’s manifestation, it lacked a certain *je ne sais quoi* that left Martland’s predecessors searching for a more scientific, if not more professional, term. However, never, at any point during the nosological revolutions throughout the many years post-Martland, did a medical researcher attempt to refute the disease-as-object initially “discovered” and named punch drunk. In other words,

the idea that punch drunk, dementia pugilistica, chronic encephalopathy of boxers, or chronic traumatic encephalopathy could ever be different disease entities, despite the differing names, never crossed the minds of medical researchers.

The singular and transhistoric conception of CTE throughout the 20<sup>th</sup>-century, and up to the present, enables the transmission of disease categorization from the medical community into the judicial system. While the culturalist position certainly has merit, and leaving the possibility that the transhistoric position is dangerously reductionistic and potentially erroneous, the tort case that enabled, and is still supporting, suffering families to claim lump sums from the nefarious schemes of the NFL is not supported by the culturalist conception of disease. The settlement explicitly mentioned a few select disease entities known to plague the retired football player populations: ALS, CTE, and Alzheimer's are a few of those diseases. While some studies presented here have remarked on the pathological similarities between the diseases, the manifestation and presentation of those diseases are different enough to warrant distinct diagnoses. For example, while AD and CTE may be difficult to distinguish in the posthumous biopsy of a brain, the symptomatic expressions of each are wildly different. The difference between CTE and dementia pugilistica, however, is in naming convention alone.

To adopt the culturalist position, at least the one offered by such historians as Loughran, Wailoo, and others, is to presuppose a significant difference, especially across time, in disease categorization based on factors such as, to name one example, culture. If the culture that created dementia pugilistica, a nosological determination used up to at least the early 2000s, is significantly different from the committees that determined the nuances

of a CTE diagnosis, the two naming conventions, despite the identical nature of both, becomes increasingly at risk of bifurcating into different disease categorizations. If we recall Loughran's treatment of shell-shock, we see this reality is not entirely hypothetical. Again, Loughran argued that the medical apparatus that saw the birth of shell-shock was a morass of varying approaches towards human health, psychiatry being only one of many. Gastroenterology, for example, had an equal say in the diagnosing of an individual suspected of shell-shock as did the burgeoning field of psychiatry during WWI. PTSD, however, the disease with the ostensible nosological link to shell-shock, was the creation of a far more sophisticated psychiatric enterprise and was the diagnostic product of a vastly different scientific paradigm. The argument goes, then, that shell-shock and PTSD are different diseases, even if they attempt to categorize similar symptoms.

While Loughran's argument, and others like hers, are certainly excellent, introspective investigations into the nuances of the history of medicine, it is only allowed to be so given its largely irrelevant link to the present – in other words, there is no nationally publicized tort case involving hundreds of suffering individuals and reprehensible business practices involving the distinction, or lack thereof, between shell-shock and PTSD. PTSD, then, is not actively dependent on the transhistoric conception of disease to link patients across large swaths of time for the betterment of parties. CTE, and its transhistoric variant punch drunk, is.

There is certainly a large, punch drunk elephant in the room that demands to be dealt with presently: has the culturalist position been used against the common good before and is it even possible? Would a culturalist perspective ever see the light of day in a court

room? It is difficult to say for certain. The settlement between the NFL and concerned parties was not without certain restrictions being placed on the allocation of funds. The cutoff date, for example, for eligible players to receive compensation were all those that retired on or before the year of the settlement, 2017. A player that retires in 2018 and starts to see symptoms of CTE in 2028 is unfortunately without recourse. Would the NFL and its team of lawyers be excited at the prospect of further limiting the eligible player count, effectively saving them money? It would seem likely. Take, then, the hypothetical argument offered by a hypothetical NFL lawyer: “We’ll agree to payouts to those players diagnosed with CTE. However, a player that previous had been diagnosed with dementia pugilistica is not included in that medical designation (see culturalist account A, B, etc., for justification). Therefore, they will not be entitled to a payout.” It is altogether too easy to imagine a malicious lawyer salivating at the possibilities. We should count our blessings that, even if unlikely to spread, certain varieties of the culturalist position are confined to the humanities, left only to compel those without much control over the necessary functions of a stable society.

There is hope, however. Allan V. Horwitz’s recent *PTSD: A Short History* attempts to bridge the gap between transhistoric and culturalist attitudes toward disease. In particular, Horwitz examines the cultural “desirability” of a PTSD diagnosis in the post-Vietnam era. While awfully cynical, Horwitz presents an interesting problem: if a PTSD diagnosis is accompanied by cultural, financial, and societal support, or “favorable consequences,” and the disease thus becomes desirable to have, will that change the

historical interpretation of the disease's development?<sup>5</sup> Regardless of PTSD's supposed desirability, Horwitz's attempt to reconcile the PTSD's cultural antagonisms with its biological truisms is the following argument: "Even if PTSD has a neurological basis, history shows that any biological substructure must have an interpretative superstructure that shapes the manifest presentations of the constantly changing symptoms of this condition."<sup>6</sup> In vaguely Marxist terminology, Horwitz suggests interpreting a disease entity as containing an unchanging biological foundation but represented throughout society on a contingent basis. Or, while PTSD may have a biological and physical reality, the manner in which the disease manifests may change over time.

While Horwitz's argument attempts to take a middle ground regarding the transhistoric and culturalist debate, it falls theoretically flat. Horwitz intends to represent both the substructure and superstructure as equal parts in disease formation without sufficiently distinguishing the superstructure's existence as separate from the substructure. If we accept Horwitz's position that the biological substructure informs the manifestation of the superstructure, has our understanding of the disease escaped the dualistic perspective offered by choosing between transhistoricism or culturalism? Certainly not. The progressive chain of nosological iterations presented here has touched upon the superstructure of a CTE diagnosis throughout – including issues of image, race, finance, class, etc. However, the manifestation of CTE's constantly changing symptoms, or potentially causes, has ultimately not significantly impacted the important relationship between nosology and the courts. Horwitz has not escaped the essential transhistoric

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<sup>5</sup>. Alan V. Horwitz, *PTSD: A Short History* (Baltimore: Johns Hopkins University Press, 2018): 173.

<sup>6</sup>. Horwitz, 183.

perspective, only elucidated one of its potential forms – Horwitz’s interpretive superstructure is dependent on and entirely informed by the substructure. The difficulty lies in figuring out how to give the culturalist position enough theoretical allowance to escape the transhistoric underpinnings, or biological substructure, without destabilizing the entire enterprise of medical research. While Horwitz’s attempt to characterize PTSD as the offspring between a transhistoric and culturalist account falls short of convincing, his effort should be lauded.

What should we say, then, of the culturalist position as a whole? What use does it serve? The entirety of this analysis has presumed the importance of providing social justice through lump sums of the settlement. However, the transhistorical analysis of CTE that begins with Martland and ends approximately with the turn of the 21<sup>st</sup>-century has completely excluded African-Americans from the nosological developments of the 20<sup>th</sup>-century. As stated, Critchley even remarked that punch drunkenness was rare in non-white boxers, if not biologically unique to white athletes. Indeed, the transhistoric approach is a simplistic tool that tends to create exclusionary historical narratives. If the relationship between the tort case and the transhistoric perspective was limited to only transhistoricism, there would still be no need to consider the African-American experience as it relates to repetitive brain injuries. The presented medical progression of CTE witnessed from our researchers, at least in the early to mid-20<sup>th</sup>-century, saw no need to reconcile its racist beliefs with a universally increasing understanding of human biology. Yet, African-American ex-football players were equally, if not more so, represented in the collective group that sued the NFL. An awareness of this discrepancy is undoubtedly the influence of

a ubiquitous culturalist perspective that positions the exclusionary nature of western, modern society at the forefront of its analysis. Perhaps the account presented here condemns the modern framework of court proceedings to a greater degree than it ever could of the culturalist position.

Regardless of the undeniable social good provided by the culturalist position, it seems clear that it cannot be reconciled with the current mechanisms inherent in both modern medical systems of diagnosing disease and the ability for the courts to use tort cases for further social good. The contention between transhistoricist and culturalism is an aspect of academia's reality. Yet, the permanence of that contention is an issue for future historians and medical researchers to continue to debate.

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