The investigation of a potential link between chronic traumatic encephalopathy and posttraumatic stress disorder

2012

Lucas Driskell
University of Central Florida

Find similar works at: http://stars.library.ucf.edu/honorstheses1990-2015

University of Central Florida Libraries http://library.ucf.edu

Part of the Psychology Commons

Recommended Citation

http://stars.library.ucf.edu/honorstheses1990-2015/1352

This Open Access is brought to you for free and open access by STARS. It has been accepted for inclusion in HIM 1990-2015 by an authorized administrator of STARS. For more information, please contact lee.dotson@ucf.edu.
THE INVESTIGATION OF A POTENTIAL LINK BETWEEN
CHRONIC TRAUMATIC ENCEPHALOPATHY AND
POSTTRAUMATIC STRESS DISORDER

by

LUCAS DRISKELL

A Thesis submitted in partial fulfillment of the requirements
for the Honors in the Major Program in Psychology
in the College of Science
and in The Burnett Honors College
at the University of Central Florida
Orlando, Florida

Fall Term 2012

Thesis Chair: Dr. Peter Hancock
ABSTRACT

With the advancement of protective gear and medical aid, soldiers are now surviving traumatic experiences that were once fatal. As a result, the prevalence of brain injury and posttraumatic stress disorder in military service members has grown. Those who have obtained brain injury are at risk of developing chronic traumatic encephalopathy, a neurodegenerative syndrome. To date, there is no cure, treatment, or diagnostic method (besides autopsy) for chronic traumatic encephalopathy. Because chronic traumatic encephalopathy and posttraumatic stress disorder present many of the same symptoms and have the possibility of deriving from the same traumatic experience, an investigation of a potential link is necessary. This study explores the possibility of chronic traumatic encephalopathy being misdiagnosed as posttraumatic stress disorder. This is done by analyzing the frequency of brain injury along with the comorbidity of posttraumatic stress disorder and brain injury. This thesis also proclaims the need for research that attempts to develop diagnostic criterion and treatment methods for chronic traumatic encephalopathy.
DEDICATION

For the brave soldiers fighting for our freedom,

For the victims of brain injury who are fighting for normality,

For the family members and loved ones of brain injury victims, who are trying their best to grasp their new reality,

For my mentors Peter Hancock, Deborah Beidel, and Joseph DiNapoli, for your time, patience, and willingness,

For Tracy Sanders, for guiding me through the world of research,

And especially, for Blair Gabler, my companion, support, and motivation.
ACKNOWLEDGEMENTS

I would like to individually thank everyone who has made my thesis possible. Tracy Sanders, thank you for being my personal mentor for the past 2 years, I have learned so much from you. Even with your stressful schedule you have always made time to help me. Dr. Hancock, Dr. Beidel, and Mr. DiNapoli, I truly appreciate the time, patience, and willingness that all three of you have given me through this process. To Dad, whether you realize it or not, you are a true inspiration to millions, especially to me. Watching your painful journey to a full recovery from severe traumatic brain injury has inspired me to dedicate my life to helping and healing those with brain injury. To my Stepmom, for coming into my life right at the perfect time. Without your guidance and help through college, I am not sure if I would of made it this far. To Mom, thank you for always being available to talk, especially when I needed a break from writing. You always have the right words to say to transform my stress into productivity. And to Blair, I could not have done this without you. Thank you for being my support, my fuel, and my de-stressor. Thank you for your patience through my whole thesis process. To all of my friends, thank you for being nothing but encouraging of my dreams and aspirations.
# TABLE OF CONTENTS

INTRODUCTION .......................................................................................................................... 1

LITERATURE REVIEW ............................................................................................................... 4

Brain Injury .................................................................................................................................. 4

Posttraumatic Stress Disorder (PTSD) ........................................................................................ 11

Chronic Traumatic Encephalopathy (CTE) .............................................................................. 14

Prior Studies Investigating the Relationship between PTSD and TBI ...................................... 16

SUMMARY .................................................................................................................................. 19

REFERENCES ............................................................................................................................. 22
LIST OF FIGURES

Figure 1: The processes of the development of chronic traumatic encephalopathy and posttraumatic stress disorder. Note how both derive from a traumatic event, and that numerous symptoms are shared..................................................................................................................... 20
INTRODUCTION

In September of 2010, the Department of Veterans Affairs reported a population of 22.7 million Veterans. Since 2001, there have been more than two million U.S. military personnel deployed to Iraq or Afghanistan. Serving in the military is a dangerous job that presents many opportunities for injury. While in combat areas, soldiers are at constant risk of encountering dangers such as, improvised explosive devices (IEDs), mortar attacks, enemy gun shots, missiles, and physical assaults. With the advancement of protective gear and medical aid, soldiers are surviving injuries that may have proven fatal in the past. Unfortunately due to the increase of survival of a life threatening experience, there is an increase of soldiers returning with psychological and physiological disorders. For soldiers, open and closed head injuries are a common trepidation that unfortunately becomes a reality for many. Traumatic brain injury (TBI) has commonly been referred to as the signature injury of Operation Enduring Freedom and Operation Iraqi Freedom due to its emerging prevalence. In 2008, approximately one quarter of deployed service members reported head and neck injury, including severe brain trauma (Hoge et al., 2008). Those who receive brain trauma are at risk of developing chronic traumatic encephalopathy (CTE). CTE is defined as, “a progressive neurodegenerative syndrome caused by single, episodic, or repetitive blunt force impacts to the head and transfer of acceleration-deceleration forces to the brain” (Omalu et al., 2011a). According to McKee et al. (2009), more than 17% of those who obtain repetitive concussions or mild TBI develop CTE. Some common
symptoms of CTE are lack of concentration, mood disorders, explosive behavior, paranoia, dysarthria, mental slowing, and irritability (Omalu et al., 2005).

In addition to physiological damage, troops also face psychological harm such as posttraumatic stress disorder (PTSD). PTSD is an anxiety disorder that is caused by experiencing or witnessing an event that was life threatening or physically harmful to oneself or to others (American Psychiatric Association, DSM-IV-TR, 2000). Some of the most common symptoms of PTSD are vivid flashbacks, nightmares, depression, anxiety, and irrational anger. Between 10% and 17% of troops deployed to combat zones have developed PTSD (Sundin, Fear, Iversen, Rona, & Wessely, 2010).

Prior research has extensively explored the similarities between PTSD and TBI (Belanger, Kretzmer, Vanderploeg, & French, 2009; Bryant & Harvey, 1998; Hoge et al., 2008; McMillan, Williams, & Bryant, 2003; Schneiderman, Braver, & Kang, 2008; Warden, 2006), yet there are still difficulties with treatment. For successful treatment one must be able to immerse themselves in the memory of the trauma. Unfortunately, many of those with TBI have cognitive deficits that challenge ones ability to rehearse the traumatic event along with sustaining the attention span to focus on the remembrance. Although there is substantial research on PTSD and TBI, few have explored the possibility of a relationship between PTSD and CTE (Omalu et al., 2011b). Further understanding of the potential link between CTE and PTSD promises to provide vital information for the development of future treatment methods for victims of brain injury.

With the understanding that CTE and PTSD share many symptoms, this paper intends to bring attention to the possibility of soldiers with CTE mistakenly being diagnosed with PTSD. Prior research has shed light upon patients who were being treated for PTSD with a history of
concussions and blast exposure, yet were never diagnosed with mild TBI (Trudeau et al., 1998). Besides for possible improper diagnosis, the creation of an assessment tool for CTE is crucial because of the high rate of suicides and parasuicides associated with this disorder (Stern et al., 2011; Omalu, Bailes, Hammers, & Fitzsimmons, 2010). The present research will investigate the potential link between CTE and PTSD by analyzing symptom similarities of TBI and PTSD. Exploring the relationship between TBI and PTSD is vital because, as noted before, brain injury is the primary cause of CTE. For the purpose of developing a full understanding of TBI, the present research will also investigate the commonality of brain injury, the different classifications of brain injury, and the ways in which a soldier may obtain brain injury. During the discussion of these topics, the unnerving reality of brain injury’s prevalence and symptoms will expose the possibility of a potential link between PTSD and CTE.
LITERATURE REVIEW

Brain Injury

It is estimated that in the United States alone, 1.7 million people are annually seen in the emergency room and hospital for TBI (Faul, Xu, Wald, Coronado, 2010). This is not incorporating the vast amount of those who receive mild TBI and do not seek treatment. Every brain trauma incident is unique to its source of injury, causing the assessment and treatment to be a complicated task. Although each case of brain injury may be unique due to its cause, all brain injuries derive from a sudden blow to or violent jerk of the head. Depending on severity, TBI symptoms range from headaches, confusion, vomiting, sleep disturbances, depression, anxiety, impaired attention, fatigue, a concussion, speech impairments, vision impairments, personality disorders, mood disorders, cognitive impairment, to death.

Brain injury falls into three severity classifications: mild, moderate, and severe. Mild TBI is described as damage ranging from minimal to no change of severity from a patient’s usual cognition level or mental health status (Bruns & Jagoda, 2009). Prior research has found that mild TBIs substantially outnumber moderate and severe TBIs (Elder, Mitsis, Ahlers, & Cristian, 2010; Hoge et al., 2008; Tanielian & Jaycox, 2008). Bruns and Jagoda (2009) reported that only 1% of mild TBIs will require neurosurgical intervention. Even though most mild TBIs do not require neurosurgical intervention, this type of injury must not be overlooked. Mild TBI can still produce neurological and neuropsychological dysfunction, and concussive symptoms. Unfortunately, many people who receive mild TBI do not seek medical treatment because they are oblivious of the severity of their injury.
Moderate TBI is similar to mild TBI in the sense that it may go undiagnosed because the victim does not seek medical assistance. Moderate TBI symptoms are usually not as obvious as those of severe TBI. Many of those with moderate TBI seek treatment weeks to months after the incident with the concern of not feeling quite like himself or herself (Zillmer & Spiers, 2001). Severe TBI results from acute injury to the brain. Those with severe TBI have a loss of consciousness lasting longer than 24 hours, and experiences amnesia for longer than 6 days. Moderate and severe TBI both present major complications with edema of the brain, intracranial bleeding, skull fractures, and brain herniation (Zillmer & Spiers, 2001). As noted before, each and every case of TBI is unique to its bearer, meaning that the recovery process is dependent upon the patient’s individual response. Although there is an established classification of severity, mild, moderate, and severe TBIs can all result in death.

Along with classifications of severity, there are also classifications of injury processes in the brain. Primary injury in TBI occurs at the moment of the trauma and is a direct result of the injury. Common primary brain injuries are hemorrhages, contusions, concussions, and axonal fiber ripping. Secondary brain injury is damage that may be caused by a primary injury. It is important to note that secondary brain injury is an indirect result of the primary injury. Secondary brain injuries may appear days, weeks, or months after the primary injury. Secondary brain injuries may present as edema, increased intracranial pressure, intracranial infection, or even epilepsy. When assessing the extent to which one with severe TBI will recover, the severity of primary brain injury and the development of secondary brain damage are the deciding factors.

As mentioned before, edema of the brain is common in moderate and severe brain injuries. Edema of the brain is the result of the brain swelling after an injury. The complication
with edema of the brain is that there is a limited amount of space provided for swelling because of the restrictions set by the skull. Subsequently, a trauma team installs an intracranial monitoring catheter into the ventricles of the brain to monitor the intracranial pressure. In moderate and severe brain injuries, severe intracranial pressure is the most common cause of death (Zillmer & Spiers, 2001).

One of the most widely used assessment tools for brain injury is the Glasgow Coma Scale (GCS). The GCS test patients upon a neurological scale ranging from zero to fifteen. A score of 13 or higher would classify as mild head trauma injury. A score ranging from 9 to 12 would classify as moderate head trauma injury. Any score of 8 or lower would fall in the range of severe head trauma. The scale is broken up into three dimensions, and the total points from the three equal the Glasgow Coma Score. The first dimension is referred to as eye response and rates from 1 to 4. The classification of one point in the eye response category is eyes not opening. For two points to be given, the patient’s eyes only open in response to pain. For three points, the patient’s eyes open to speech. For a full four points, the patient has full control over the opening and closing of their eyes. The second dimension is referred to as verbal response and rates from 1 to 5. In order for a patient to receive one point in the verbal response category, they must present no verbal response. For two points, the patient can moan but the sounds are not understandable. For three points, the patient produces exclamatory and/or random speech, but is unable to have a conversation. For a patient to receive four points, they must be capable of conversational speech, but present as slightly confused or disorientated. For a full five points, the patient is fully capable of conversational speech with coherency and absence of confusion. The third dimension is referred to as motor response and rates from 1 to 6. A patient who receives
one point in this category presents no motor response. For two points, the patient presents extension to pain. For three points, the patient reacts with abnormal flexion to pain. For four points, the patient presents both flexion and withdrawal to pain. The patient is capable of pulling a part of their body away from the source of pain. For five points, the patient is able to localize the pain source and presents purposeful movements towards and away from pain source. For the full six points, the patient successfully obeys physical commands.

In a military setting, combat activities, noncombat activities, and training procedures present a myriad of opportunities for head injury. Because of the many different ways one can receive brain trauma, along with the necessity of immediate attention to flesh wounds, brain damage is not always noticed. Soon after soldiers starting returning from Operation Iraqi Freedom and Operation Enduring Freedom, the Veterans Affairs hospitals discovered many soldiers with mild TBI that were not acknowledged during discharge. It seems unbelievable for damage to a vital organ such as the brain to go unnoticed, but it happens more often then fathomable. Some soldiers even pass the GCS scale with a 15 (no brain injury), yet when given a CT scan the results clearly provide evidence of brain damage (Bochicchio et al., 2008). With the estimate that 30% of troops may receive mild TBI during their time spent in service, there are hundreds of thousands of soldiers at risk of receiving unnoticed brain injury (Hoge et al., 2008).

Understanding all aspects of brain injury is vital to investigating a potential link between CTE and PTSD. By examining brain injury, PTSD, and CTE each individually, a possible comorbidity will arise by the coexistence of symptoms. Although, to the extent in which CTE and PTSD can arguably be linked is the purpose of this research, a full understanding of brain injury is necessary.
**Closed and Penetrating Head Injuries.** Physical damage to the brain can result from two methods of injury, either an object penetrating the skull and damaging the brain, or the rapid acceleration and/or deceleration of the head causing the brain to hit the insides of the skull. These mechanisms of physical brain injury are separated into two classifications, penetrating, and closed head injuries. Penetrating head injuries occur when a foreign object enters through the skull and damages specific regions of the brain. The resulting symptoms are dependent upon the focalization of damage, and complications with infections and hemorrhaging. If the penetrating head injury does not result in death, the victim will likely suffer neurological deficits. Closed head injuries are the result of the brain undergoing acceleration and/or deceleration. When the brain endures acceleration, the brain rapidly changes from stationary to moving. An example would be a person’s head being hit by a weapon or object such as a soldier’s baton. Deceleration of the brain would occur when the head is moving at a constant speed, but then is stopped abruptly. An example would be a soldier riding in a Humvee that is forced to slam on its brakes, causing the soldier to fly forward and slam their head upon the windshield. Both acceleration and deceleration can cause massive damage to the brain by ripping neuronal fibers, and bruising the brain from impact against the skull. Contusions can become very dangerous, resulting in hemorrhage and edema of the brain.

In many cases, closed head injuries result in a coup contrecoup injury. The coup injury is the result of either the primary acceleration or deceleration, causing the brain to collide with the skull. The contrecoup occurs after the brain bounces off the skull from the first collision, and then hits the opposing side of the skull. Coup and contrecoup injuries can result in both focal and diffuse injuries, contusions, concussions, and the tearing of neuronal fibers. Due to the
prevalence of IEDs and mortar attacks in Operation Enduring Freedom and Operation Iraqi Freedom, soldiers are at high risk of both penetrating and closed head injuries. Taber, Warden, and Hurley (2006) discuss a finding of 88% of TBIs being due to closed head injuries.

**Blast Related Brain Injuries.** It has been reported that the most common cause of war injuries are from explosions and blasts (Warden, 2006). When a soldier is exposed to a blast, the assessment for brain injury is extremely important. At the Walter Reed Army Medical Center, 59% of patients who were tested for brain injury due to blast exposure were diagnosed with TBI (Okie, 2005). Explosions pose as a serious threat to soldiers because of the many ways in which they can cause harm. There are four categories of blasts effects that are designated by the way a blast can cause injury. The first is primary (caused from pressure change), second is secondary (caused from projectiles), third is tertiary (caused from wind propelling body), and the fourth is quaternary (caused from burns, asphyxia, and toxin exposure) (DePalma, Burris, Champion, & Hodgson, 2005). For the purpose of this paper, and the lack of quaternary induced brain injury research, only primary, secondary, and tertiary brain injuries will be discussed.

Primary blast injuries consist of damage to the brain caused by the change of atmospheric pressure after a explosion. Once the explosion has occurred, there is a dramatic increase in atmospheric pressure caused by the oscillation of the blast waves. This rapid push of air from the explosion (increase of pressure) subsequently causes a vacuum effect, making the atmospheric pressure less than the norm. Then the second wave hits, causing the atmospheric pressure to increase slightly above the norm, before it then returns to a balanced pressure. For many years, this pressure change was believed to only harm the lungs, gastrointestinal tract, and the
eardrums. However recently it has been argued that, “Primary blast injuries to the brain include concussion as well as barotrauma caused by acute gas embolism” (DePalma et al., 2005). Although still controversial, primary blasts are believed by many to also harm the central nervous system.

Secondary and tertiary blast injuries are the injuries most commonly thought of when one thinks of explosions. Blast waves propel shrapnel, foreign objects, and in many cases soldiers, in all directions. As a result everyone in the vicinity becomes a target. Secondary blast injuries are those obtained by soldiers due to the undirected projection of foreign objects and shrapnel. In regards to the brain injury, secondary blast injuries can consist of both closed head and penetrating head injuries. Depending on how close someone is to the explosion, if they are wearing a helmet, the speed of the object being flung, and the shape of the object, dictates whether the injury will be closed head or penetrating. Tertiary blast injuries are sustained from the soldier being projected as an object due to the immense force of the blast wind. Soldiers are at high risk of both closed and penetrating head injuries when hurled by blast winds. In both secondary and tertiary blast injuries, the rapid acceleration and/or deceleration of the head can cause neuronal fiber tears, concussions, and contusions.

With the advancement of technology, IEDs and mortars have become extremely sophisticated. IEDs can be set off with a remote detonation, rigged for timed explosion, and even ignited by pressure sensors from above driving Humvee’s. The power of explosion devices has also increased, making these weapons more dangerous than ever. In many cases, with the combination of bodily injury and psychological trauma caused by an explosion, many soldiers are unaware of the brain injury they received. Neurologists believe that more than 30% of troops
who serve in active combat zones for four months or longer will receive neurological damage from IED and mortar blast waves, while presenting no surface damage (Glasser, 2007). Trudeau and Colleagues (1998) “discovered a subgroup of PTSD patients who had a history of mild concussion on exposure to explosions and who had never been diagnosed with brain injury”. Blast induced brain trauma has played a major role in Operation Enduring Freedom and Operation Iraqi Freedom, and will continue to be important field of study for researchers.

**Posttraumatic Stress Disorder (PTSD)**

Protective gear, armor, and medical aid have advanced to such an extent that many soldiers are surviving horrific events that in the past would have been fatal. Upon arriving home from war, some soldiers are haunted by vivid flashbacks and nightmares of an event that jeopardized the wellbeing of their life or a fellow soldier’s life. Research has consistently shown that soldiers who are exposed to combat have an increased risk of developing PTSD (Sundin et al., 2010). PTSD is an anxiety disorder that is caused by experiencing or witnessing an event that was life threatening or physically harmful to oneself or to others (American Psychiatric Association, DSM-IV-TR, 2000). The primary diagnostic criterion used for the diagnosis of PTSD is the DSM-IV-TR clinical criteria for PTSD diagnosis. The current DSM-IV-TR clinical criteria for PTSD is categorized into 6 sections titled A through F. The criteria for section A is as follows: The person must of been exposed to a traumatic event in which they experienced, witnessed, or were confronted with an event that was life threatening, or threatening to the physical integrity of self or others and also created a strong response of fear, helplessness or horror (American Psychiatric Association, DSM-IV-TR, 2000). The criterion for section B
requires that the traumatic event is re-experienced in one or more of the following ways: The event is reoccurring through images, thoughts, and/or perceptions; having dreams of the event; reliving the event whether through hallucinations, flashbacks, or illusions; Intense psychological distress when exposed to a cue that resembles the traumatic event; lastly, physiological reaction to cues that resemble the traumatic event (American Psychiatric Association, DSM-IV-TR, 2000). The criterion for section C requires that patient experiences numbing of responsiveness and avoidance of stimuli that associates with traumatic event in three or more of the following ways: avoids thoughts, feelings, or conversations related to traumatic event; avoids social interactions (activities and people) and places that associate with traumatic event; possess inability to recall substantial aspects of trauma; apparent diminished interest in participating in important activities; feel detached from others; limited range of affect; feelings of foreshortened future (American Psychiatric Association, DSM-IV-TR, 2000). Criterion for section D requires new increased arousal that presents in two or more of the following ways: difficulty with sleeping; irritability or explosive anger; concentration difficulties; hyper-vigilance; excessive response to being startled (American Psychiatric Association, DSM-IV-TR, 2000). Criterion for section E requires that symptoms in section B, C, and D, last longer than 1 month. Criterion for section F requires that the traumatic event cause clinically significant distress in social, occupational or other important aspects of the patient’s life (American Psychiatric Association, DSM-IV-TR, 2000). Upon diagnoses, the PTSD is distinguished between acute, chronic, or with delayed onset. Acute is used for a patient presenting symptoms for less than 3 months. Chronic is used if the duration of symptoms last 3 months or more. With delayed onset is used for those whose symptoms begin at least 6 months after the traumatic event.
The Clinician Administered PTSD Scale (CAPS) is the primary instrument used to assess PTSD. The Structured Clinical Interview for DSM (SCID) assesses PTSD in a less detailed fashion. The CAPS collects data on the frequency and severity of PTSD symptoms, whereas the SCID assesses a range of mental health disorders in addition to PTSD. Approximately 80% of those with lifetime PTSD suffer co-morbid psychiatric disorders such as depression, anxiety, or addiction (Foa, Keane, & Friedman, 2000). PTSD prevalence tends to increase months after post-deployment; Sundin et al. (2010) believes the increase in PTSD is due to the initial joy of being home overshadowing the actual mental difficulties. For some soldiers, once they get back into the normal routines of life, their PTSD symptoms start to become noticeable. Currently, the most efficacious treatment for PTSD is Cognitive-Behavioral Therapy. The three most common forms of Cognitive-Behavioral Therapy are cognitive therapy, exposure therapy, and group therapy. Cognitive therapy consists of meeting with a therapist and analyzing the traumatic flashbacks. By identifying the persistent bad thoughts, the therapist can teach the veteran to replace and control the thoughts. The therapist also helps the veteran understand that the traumatic event was not their fault. Exposure therapy consists of purposely replaying the traumatic event in ones head. Exposure therapy is based on the idea that consistent re-exposure causes habituation. When a veteran repeatedly relives the traumatic event, eventually they will habituate and not react with fear. With the guidance of a therapist, exposure therapy can be used by having the veteran verbally recall the traumatic event, or by the assistance of virtual reality and scents. Lastly, group therapy is commonly used, allowing veterans to meet up and share their personal stories. Group therapy helps the veterans to not feel alone, while also providing comfort by knowing that others have experienced the same traumas. Many veterans decide to use
multiple treatment methods rather than limiting themselves to one. Beidel, Frueh, Uhde, Wong, & Mentrikoski (2011) found that multi-component Cognitive-Behavioral Therapy is more effective than only exposure therapy in improvement of social functioning, specifically social engagement and interpersonal functioning.

**Chronic Traumatic Encephalopathy (CTE)**

Although CTE is new in the field of research, the knowledge that brain injury causes cognitive and motor deficits has heavily been discussed. In a 1928 paper by Martland, the effects of brain trauma were first discussed by his observations of boxers being repeatedly punched, in which he referred to it as “punch drunk”. Millspaugh took the research even further by outlining the cognitive and motor deficits that brain injury can cause, he termed the injury as “dementia pugilistica” (1937). With the advancement in technology, Corsellis, Bruton, and Freeman-Browne (1973) were able to describe the neuropathology of dementia pugilistica in boxers, and distinguish it from other neurodegenerative disorders.

CTE presents as a mixture of cognitive, mood, and behavioral neuropsychological and neuropsychiatric changes (Baugh et al., 2012). Cognitive changes typically include memory, executive functioning deficits, language difficulties, and eventually dementia (Stern et al., 2011; Omalu et al., 2005; Gavett, Stern, & McKee, 2011). Mood changes present symptoms such as irritability, lack of impulse control, depression, apathy, suicidal ideation, and paranoia (Baugh et al., 2012; Stern et al., 2011; Omalu et al., 2005). Behavioral changes in victims of CTE include impulse control problems, disinhibition, development of substance abuse and addiction problems, and aggression and increased violence (Baugh et al., 2012). Microscopically, CTE is
distinguished by its abundance of neurofibrillary inclusions, presented as neurofibrillary tangles, neuropil threads, and glial tangles (Gavett et al., 2011). Neuropathologically, there is a degeneration of the cerebral hemispheres, medial temporal lobe, thalamus, mammillary bodies, brain stem, microglial activation, and parenchymal histiocytes (McKee et al., 2009; Omalu et al., 2011b). McKee and Colleagues (2009) report that what separates CTE from other tauopathies is its preference of the superficial cortical layers, irregular, patchy distribution in the frontal and temporal cortices, prominent perivascular, periventricular and subpial distribution, tendency for sulcal depths, and marked accumulation of tauimmunoreactive astrocytes. Although most of the confirmed cases of CTE have been found in athletes, CTE can develop in anyone who has received head trauma. CTE has been most commonly found in athletes of sports such as American football, professional hockey, and professional wrestling, but CTE has also been discovered in a Iraqi veteran, circus clown, an epileptic, and two victims of abuse (one self-inflicted) (Gavett et al., 2011; Geddes, Vowles, Nicoll, & Revesz, 1999; McKee et al., 2009; Omalu et al., 2005; Omalu et al., 2006; Omalu et al., 2010; Omalu et al., 2011a; Omalu et al., 2011b; Roberts, Whitwell, Acland, & Bruton 1990; Stern et al., 2011). CTE tends to have a period of latency until exposure. For many, it begins with personality changes such as irrational anger, and depression. Soon after, cognition is altered causing memory and executive functioning difficulties (Gavett et al., 2011). Symptoms worsen over time into dementia, accompanied by speech and gait abnormalities (McKee et al., 2009). Unfortunately, there is no clinical diagnostic criterion for CTE, leaving post-mortem biopsy as the only certain method of diagnosis. Given that millions of soldiers are fighting overseas for our freedom, further research for development of a diagnostic criterion and biomarkers of CTE is urgent.
Prior Studies Investigating the Relationship between PTSD and TBI

Both PTSD and TBI result from a traumatic event. In combat and blast related TBI the traumatic event is both physically and psychologically damaging, causing the two to coexist (Belanger et al., 2009). The acknowledgment that TBI and PTSD present many of the same symptoms has been noted throughout American history. Dating back to World War I, the soldiers who fought in the trenches were frequently exposed to mortar attacks and grenade blasts. As a result, many received both psychological and neurological damage, which was termed then as “Shell Shock.” With the growth of research, we have now made distinctions between brain injury and psychological damage (now known as PTSD).

Combat activities, noncombat activities, and training procedures all present opportunities for both physical and psychological head trauma. Hoge, et al. (2008) found that 43.9% of soldiers who reported loss of consciousness during battle injury met the requirements for PTSD. Several studies (Belanger et al., 2009; Hoge et al., 2008; Vanderploeg, Belanger, & Curtiss, 2009) found that PTSD symptoms are strongly associated with mild TBI. Schneiderman and others (2008) found that high PTSD scores were found most frequently amongst a few select groups, one of which were those diagnosed with level 1 and level 2 mild TBI. These studies suggest that TBI and PTSD may have a correlation.

There can be difficulty when assessing someone who has received brain injury from a traumatic event because TBI and PTSD have some similar symptoms. There is always the possibility of PTSD being overlooked in someone who presents mood or behavioral difficulties (McMillan et al., 2003). Both TBI and PTSD can present symptoms such as irritability, sleep disturbance, memory disturbance, personality and mood changes, shortened patience, depression,
hostility, and anxiety. To test the extent of PTSD and TBI co-morbidity, Hoofein, Gilboa, Vakil, and Donovick, (2001) tested 76 patients who received a TBI diagnostic an average of 14 years before the study and found that 14% still meet full diagnostic criteria for PTSD.

Even though many symptoms of PTSD and TBI are the similar, each disorder has symptoms that remain unique. When assessing a patient, the clinician must be aware of whether the patient is presenting more organic symptoms such as difficulty of balance, headaches, vision impairment, or symptoms more distinct to PTSD such as re-occurring flashbacks, hyper-arousal, and nightmares. Many patients of TBI develop either temporary or permanent retrograde amnesia preventing flashbacks, which is a key symptom of PTSD. It has been argued by some (Rattok, Boake, & Bontke, 1996; Sbordone & Liter, 1992) that because of the coma associated with TBI, it is not possible for PTSD to develop. They believe a clear, specific remembrance is necessary for a true diagnosis of PTSD. Sbordone and Liter (1992) found that 100% of their PTSD patients were capable of providing a highly detailed recollection of the events that occurred within 15 minutes of the traumatic event, whereas none of the patients with mild TBI could do so. Although none were capable of giving highly detailed recollections, 71% of the same mild TBI patients were capable of recalling the event.

Since TBI is the primary cause of CTE, investigating the relationship between PTSD and TBI is very important. If there is a link between PTSD and TBI, it could provide evidence for the link between PTSD and CTE. Currently, evidence for a possible link between PTSD and CTE is in the case of a 27-year old Iraqi war veteran on whom Dr. Omalu et al. (2011b) performed an autopsy. Dr. Omalu et al. (2011b) conducted a gross neuropathological, histochemical, and immunohistochemical analysis of the subject’s brain, and was able to confirm a diagnosis of
CTE. The subject experienced many possible concussive incidents, such as exposure to mortar and IED blasts, losing consciousness during a car wreck, and receiving a hit during a football game that caused headaches and memory issues. The subject was diagnosed as having PTSD with hyper-arousal and numbing, as well as alcohol abuse. Two months before his suicide the patient reported continuous PTSD symptoms.
SUMMARY

Serving in the military is an extremely dangerous job that requires the most brave. During service and training, soldiers are at a very high risk of experiencing events that are both psychologically and physically traumatic. Brenner, et al (2010) found that 26% of mild TBI patients screened positive for PTSD during postdeployment. Due to exposure of traumatic events, an unfavorable amount of soldiers are coming home with serious mental health issues, whether derived from TBI, PTSD, or possibly CTE.

There are obvious distinctions between PTSD and CTE that cannot be ignored. The purpose of this paper is not to argue that they one in the same, as it is known that you can have one without the other. Although the development of PTSD in a victim does not always originate from a brain injury, receiving brain injury is always a traumatic event that has the possibility of causing PTSD. In Figure 1, the methods in which someone receives CTE and PTSD are displayed, along with the symptoms each share and individually possess. The Figure shows that CTE and PTSD both derive from different results of a traumatic event. Even though a traumatic event may result in only psychological damage (PTSD), or only physical brain damage, every traumatic event has the potential to result in both PTSD and brain damage. This potential comorbidity adds to the difficulty and necessity of a future assessment tool for CTE. It is noteworthy that Figure 1 displays the many shared symptoms, and the mutual origin of damage. Only because of these commonalities, is the possibility of misdiagnoses possible.

The purpose of this paper is to acknowledge that CTE and PTSD do share many symptoms, and with that understanding, explore the possibility of soldiers with brain injury
mistakenly receiving a diagnosis of PTSD rather than CTE. This is done best by exploring the relationship between TBI and PTSD because TBI is the primary cause of CTE. Both TBI and PTSD are obtained from a traumatic experience, which already presents as a sign for the possibility of a link. Research can provide multiple cases of soldiers who have obtained brain injury via concussion or blast exposure yet received a diagnosis of only PTSD (Trudeau et al., 1998; Omalu et al., 2011b). TBI may negatively affect the therapy outcome for a PTSD patient
causing difficulties with attention and memory. PTSD treatment cost the federal government millions of dollars each year in disability payments and treatment cost (Trudeau et al., 1998). It is inefficient and wasteful to give a veteran PTSD treatment if the root of their symptoms is actually from their undiagnosed brain injury. The case study presented by Omalu et al. (2011b) introduced a soldier with CTE who was only diagnosed with PTSD. This soldier committing suicide, leaving behind his wife and two children, could have been prevented with an assessment tool or other method of diagnosis for CTE. The high rate of suicide and parasuicides associated with CTE is one of the most crucial realities that support the necessity of an assessment tool, treatment plan, and intervention plan. The continuation of this research is important because the more knowledge about CTE we gain, the greater the possibility of development of a diagnostic tool, intervention plan, and treatment regimens; meaning less suicides, and a increased quality of life for victims of CTE.
REFERENCES


McKee, A. C., Cantu, R. C., Nowinski, C. J., Hedley-Whyte, E. T., Gavett, B. E., Budson A. E.,


