

Chapter 2

The Research

For years now the relationship between traumatic brain injuries (TBIs) and post-traumatic stress disorder (PTSD) has been a controversial issue that seems to leave many unanswered questions. The most salient issue is whether an individual with a TBI can develop PTSD if they have no memory of the incident. While they share many commonalities, such as symptomatology, and even more obvious ones, like the fact that they both stem from a traumatic event, not all traumatic events result in TBI or PTSD. Among individuals in the United States, approximately 61 % of men and 51 % of women will be exposed to trauma during their lifetime, but only about 5 % of men and 10 % of women will develop PTSD based on the National Comorbidity Survey (Kessler et al. 1995). It has been estimated that there are nearly 10 million TBI incidences annually, with almost 1.7 million emergency department visits yearly in the United States (Hyder et al. 2007).

Yurgil et al. (2014) found in a military sample that TBI during one's most recent deployment is the strongest predictor of post-deployment PTSD, even when accounting for pre-deployment symptoms, prior TBIs, and combat intensity. It has long been recognized that TBI and PTSD evidence many of the same symptoms, resulting from physiological, neurological, and psychological damage. Dating back to World War I, it has been noted that soldiers who were exposed to mortar attacks and grenade blasts began to experience psychological and neurological symptoms, which at the time was termed "Shell Shock". With the growth of research on these symptoms and their origin, we have now made distinctions between brain injury and PTSD; however, differentiation often still becomes grayed.

There can be difficulty when assessing someone who has received brain injury from a traumatic event because TBI and PTSD share numerous symptoms. There is always the possibility of PTSD being overlooked in someone who presents with mood or behavioral difficulties (McMillan et al. 2003). Similarly, TBI may be

overlooked if a person does not evidence any neurological symptoms and only presents with psychological complaints. While the identification of moderate and severe brain injuries tend to be much more straightforward, mild TBI and PTSD can present similar 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 comorbidity, Hoofien et al. (2001) tested 76 patients who received a TBI diagnosis an average of 14 years before the study and found that 14 % still met full diagnostic criteria for PTSD.

The ultimate purpose of a neuropsychological evaluation is to provide recommendations that will inform the patient what steps they should take to address their presenting problems. In order to give appropriate recommendations, the neuropsychologist must be accurate in their conceptualization and diagnostic capabilities; and, in the case of differentiating TBI from PTSD, they must have a deep working knowledge of the neurological and psychological underpinnings of both disorders. Thus, before we look at specific approaches to understanding a patient that presents with TBI/PTSD symptoms, it is first necessary to examine what research has shown about each disorder individually. To date, much of the epidemiological research on the relationship between TBI and PTSD has focused on the military population, due to the prevalence of exposure to physically and psychologically traumatic events. However, even with this limitation, there is still ample research on TBI and PTSD individually, providing the authors the opportunity to gather a selected sample of research to emphasize the important aspects of these two disorders.

Traumatic Brain Injury

Traumatic brain injury (TBI) is a form of acquired brain injury, typically caused by a sudden blow to skull or violent jerk of the head. TBIs often result in either: (1) direct damage to the neurons of the brain, and/or (2) shearing of neuronal axons that allow the brain to communicate within its self and with the rest of the body. Due to the intricate nature of the brain's organization, symptoms of TBI widely vary depending on severity, location, and duration of the damage. Having said that, some more common symptoms of TBI range from headaches, confusion, vomiting, sleep disturbances, depression, anxiety, impaired attention, fatigue, speech impairments, visual spatial deficits, vision impairments, memory deficits, personality changes, mood disorders, paralysis, to death. Approximately 57 million people worldwide have been hospitalized with one or more TBIs (Murray and Lopez 1996).

It was estimated that in 2009, 2.4 million hospital emergency department visits, hospitalizations, or deaths related to a TBI occurred in the United States (Faul and Coronado 2014). In 2006, approximately 5.3 million people were living with significant disabilities caused by TBI that inhibited their ability to return to prior levels of functioning (Langlois et al. 2006). According to the World Health Organization, TBI will surpass numerous diseases as the major cause of disability and death by the year 2020.

On a global scale, the primary cause of TBI is road traffic accidents (62 %), with violence (24 %) and falls (8 %) ranked as second and third (Hyder et al. 2007). These statistics are not incorporating the vast amount of those who receive some form of brain injury and do not seek treatment. Since every brain trauma incident is unique to its source of injury, the assessment and treatment of such disorders can be a complicated task. Thus, TBIs can be categorized into different classifications depending upon cause and severity in order to aid in specificity of diagnosis and treatment.

There are three overall severity classifications that TBIs can be placed into: mild, moderate, and severe. Now each level of severity will be discussed in further detail.

Mild Traumatic Brain Injury. Mild TBI is described as neurological damage ranging from minimal to no change of severity from a patient's usual cognition level (Bruns and Jagoda 2009). Prior research has found that mild TBIs substantially outnumber moderate and severe TBIs, accounting for an estimated 80 % of all TBIs (Elder et al. 2010; Hoge et al. 2008; Tanielian and Jaycox 2008). Bruns and Jagoda (2009) reported that only 1 % of mild TBIs will require neurosurgical intervention. While most people that receive a mild TBI recover relatively quickly and fully, this type of injury must not be overlooked. Mild TBI can still cause permanent neurological and neuropsychological dysfunction. Unfortunately, many people who receive mild TBI do not seek medical treatment because they are oblivious of the severity of their injury.

Currently, there is not one definitive definition of mild TBI because brain trauma is such an individualized-injury; however, the three most commonly used definitions of mild TBI were developed by: (1) the World Health Organization Collaborating Centre Task Force on Mild Traumatic Brain Injury in 2004, (2) the Center for Disease Control working group in 2003, and (3) the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine in 1993. While all three definitions are slightly different from one another, they correspond on the majority of criteria. When integrated, the salient criteria for mild TBI are the patient having received an injury to the head from an external force or acceleration/deceleration forces that resulted in *one or more* of the following: confusion, disorientation, loss of consciousness for less than 30 min, dysfunction of memory around the time of injury, or observable neurological or neuropsychological dysfunction such as seizures or focal deficits.

In addition to the three definitions that were just discussed, the Glasgow Coma Scale (GCS) is almost always used in order to assess the severity of brain injury. The GCS ranks patients upon a neurological scale ranging from 3 to 15. 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, and any score of 8 or lower would fall in the range of severe head trauma. The scale is broken up into three dimensions: (1) stimulus required for eye opening, with a possible score of 1–4, (2) best verbal response, with a possible score of 1–5, and (3) best motor response, with a possible score of 1–6.

Many medical and psychological professionals recognize two subtypes of mild TBI: complicated and uncomplicated. Complicated mild TBI is diagnosed when the

patient meets criteria for a mild TBI and has a brain abnormality (e.g., edema, hematoma, or contusion) visible on neuroimaging on the day of the injury (Iverson and Lange 2011). Conversely, uncomplicated mild TBI is diagnosed when the patient meets criteria for mild TBI, but does not evidence any damage via neuroimaging.

During the first few days after a mild TBI, many individuals report experiencing headaches, drowsiness, difficulty with concentration and attention, dizziness, and feeling mentally cloudy. These symptoms often last for days to weeks. There has been much discussion on determining factors that can predict neuropsychological outcome in patients with mild TBI. The two most researched factors are duration of loss of consciousness (LOC) and duration of posttraumatic amnesia (PTA). The term loss of consciousness is typically defined as a sleep-like state of being. Posttraumatic amnesia refers to the patient's inability to remember things that have happened immediately after the head trauma. As discussed by Iverson and Lange (2011), numerous researchers have reported that while there is no clear association between brief LOC and neuropsychological functioning (Leininger et al. 1990; Lovell et al. 1999), there appears to be a relationship between the presence and duration of PTA and worse immediate outcome and recovery (Collins et al. 2003; McCrea et al. 2002). In regards to neuropsychological performance after mild TBI, impairment in processing speed, working memory, verbal fluency, executive functioning, new learning, and memory are most commonly seen (Alexander 1995; Barrow et al. 2006; Belanger et al. 2005; McAllister et al. 2006).

Moderate and Severe Traumatic Brain Injury. While mild TBIs account for the majority of brain injuries (80 %), moderate (10 %) and severe (10 %) brain injuries are estimated to evenly comprise the rest of the distribution. Similarly to mild TBI, there is no one definitive definition for moderate or severe TBIs; thus, the GCS, duration of LOC, and duration of PTA are most often used for differentiation and diagnosis. For moderate TBI, many abide by the criteria of a GCS ranging from 9 to 12, duration of LOC of 30 min to 24 h, and duration of PTA of 1–7 days. For severe TBI GCS of 3–8, duration of LOC of more than 24 h, and duration of PTA greater than 7 days is most commonly used.

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 sometimes 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 and Spiers 2001). A common complaint of both moderate and severe TBI is memory disruption. As already mentioned, many individuals experience PTA (also known as anterograde amnesia) and have difficulty remembering events that have occurred after their head trauma. Depending on numerous factors, symptoms of PTA can last from minutes to months. On the contrary, the inability to remember events that occurred before a head trauma is commonly referred to as retrograde amnesia. Similarly to PTA, retrograde amnesia ranges in duration of memory impairment and the date to which the individual can remember (e.g., whether one week or three years prior to the head trauma).

Along with classifications of severity, there are also classifications of injury processes in the brain. Moderate and severe TBI can both present with major

complications such as edema of the brain, intracranial bleeding, skull fractures, and brain herniation. 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, necrosis, apoptosis, or 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 crucial deciding factors.

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 fractures the skull and damages specific regions of the brain. The resulting symptoms are dependent upon the localization of damage and complications with infections or hemorrhaging. In some cases, the fracturing of the skull can actually protect the brain by absorbing the force of the blow and not transmitting it into the brain itself as seen in closed head injuries.

Closed head injuries are the result of the brain undergoing acceleration and/or deceleration. When the brain endures acceleration, the head rapidly changes from stationary to moving causing the stationary brain to smash into the moving cranium. An example of acceleration would be a person's head being hit by an object such as a tree limb or baseball bat. Deceleration of the brain would occur when the head is moving at a constant speed, but then is stopped abruptly. An example would be an individual riding in a car that is forced to slam on its brakes, causing the person to fly forward and slam their head upon the windshield. Although the person's head would immediately stop once it hit the windshield, the brain floating in cerebral spinal fluid would slam into the front of the skull close to the same speed the car was originally going. 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 some cases, closed head injuries result in a coup countercoup injury. The coup injury is the result of either the primary acceleration or deceleration, causing the brain to collide with the skull. The countercoup occurs after the brain bounces off the skull from the first collision, and then hits the opposing side of the skull. Coup and countercoup injuries can result in both focal and diffuse injuries, contusions, concussions, and the tearing of neuronal fibers.

There are several caveats to these classifications. In some cases, a closed-head injury may cause focal damage because of a vascular tear or rupture which causes focal bleeding in the brain. Such bleeding can result in hematomas, often in the subdural area of the brain. In these cases, the hematoma will grow and become a

mass which acts like a space occupying lesion. If treated quickly or if it resolves spontaneously, such hematomas while sounding scary may have no impact. However, when not treated they can continue to grow to a size where the internal pressure of the brain is raised, causing damage to tissue and even cutting off blood flow to the brain (as the heart cannot pump strongly enough to overcome the increased pressure) leading to anoxia or hypoxia and significant cognitive impairment or even death. While such disorders are more likely as we age, they can occur in anyone at any age.

A second but similar issue occurs when the bleedings occurs not in the meninges but within the grain itself as a result of a rupture of a blood vessel which may be related to the presence of a preexisting malformation or aneurysm. In such cases, bleeding may damage brain tissue and create a focal injury similar to that seen in penetrating injuries. Severity of the problems can range from mild to severe (even causing death) depending on many individual factors. Bleeding in the brain may of course also occur in penetrating head injuries.

Blast-Related Brain Injuries. Blast-related brain injuries become increasingly recognized by the military (rather than dismissing such disorders as emotional as has been done throughout history) as well as by the public after well publicized terrorist blast effects. It has been reported that the most common cause of war injuries are from explosions and blasts (Warden 2006). 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 blast 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 the individual), and the fourth is quaternary (caused from burns, asphyxia, and toxin exposure) (DePalma et al. 2005).

Primary. Primary blast injuries consist of damage to the brain caused by the change of atmospheric pressure after an 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. 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 vehicles driving above. 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. Researchers 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 et al. (1998) reported finding a subgroup of patients with PTSD who, although they had a history of mild concussion on exposure to explosions, had never been diagnosed with brain injury. There is still little known about the neuropsychological ramifications of blast induced brain trauma, making the differentiation between PTSD and TBI more difficult to determine.

Quaternary. Quaternary effects are caused by indirect effects caused from burns, respiratory difficulties causing hypoxia and anoxia, cardiac arrest, exposure to toxins, excessive blood lost, and injuries to other bodily systems. This is clearly difficult to define as the possibilities are nearly endless and depend on the exact factor in each individual situation. In many cases, the individual will die or suffer such extreme disabilities that neuropsychological testing will never take place, but in other cases these factors can cause extreme cognitive and emotional problems arising from brain damage or secondary injury, as well as the emotional effects of such events.

Common Psychological Outcomes of Traumatic Brain Injuries. Traumatic brain injuries often result in psychological symptoms and disorders. One of the most famous examples of the brain's role in personality is the case of Phineas Gage, a railroad worker who survived an accident during which an iron rod went straight through his left frontal lobe. Before his accident Phineas Gage was described as a hard working, responsible, and pleasant man; however, after the accident he was seen to be fitful, impulsive, and disrespectful. The case of Phineas Gage ignited the field of research on the relationship between the brain and psychological disorders, and while leaps and bounds have been made since his accident, there is still much to discover.

Some of the most common psychological disorders associated with TBIs are major depression, generalized anxiety disorder, PTSD, panic disorder, obsessive-compulsive disorder, substance abuse, and specific phobia (Deb et al. 1999; Federoff et al. 1992; Hibbard et al. 1998; Jorge et al. 1993; Van Reekum et al. 1996). Within the literature there are vast differences in reported post-TBI rates of psychological

disorders. In regards to major depression, the prevalence rate ranges from 14–77 % depending on the study at which one looks (Deb et al. 1999; Fann et al. 1995; Federoff et al. 1992; Hibbard et al. 1998; Jorge et al. 1993; Van Reekum et al. 1996; Varney et al. 1987). Various studies report rates of 3–28 % for generalized anxiety disorder (Fann et al. 1995; Hibbard et al. 1998; Jorge et al. 1993; Van Reekum et al. 1996), and 3–27 % for PTSD (Bryant et al. 2000; Deb et al. 1999; Hibbard et al. 1998). Moreover, 4–17 % receives a diagnosis of panic disorder, 2–15 % receive obsessive–compulsive disorder, and 1–10 % received phobic disorder diagnosis, while 5–28 % receive a diagnosis of substance abuse (Deb et al. 1999; Hibbard et al. 1998; Van Reekum et al. 1996).

In attempt to determine the long-term effects of TBI on psychological health, Koponen et al. (2002) evaluated 60 patients on an average of 30 years after their TBI. The Schedules for Clinical Assessment in Neuropsychiatry was used to help assess the Axis I disorders, while the Structured Clinical Interview for DSM-III-R Personality Disorders was utilized for the Axis II disorders. The researchers found that 61.7 % of patients had an Axis I disorder during their lifetimes, and 40 % had an Axis I disorder at the time of evaluation. Of the 60 patients, 48 % had an Axis I disorder develop after the TBI, while 22 % had an Axis I disorder before their TBI. The most common Axis I disorder found post-TBI was major depression, occurring in 27 % of patients at some point after the TBI, and 10 % at the time of assessment. Panic disorder was diagnosed in 8 % of patients at some point after the TBI, and 7 % still met criteria for panic disorder. 12 % of the male patients met criteria for a substance abuse disorder after their TBI, while 8 % had the disorder at the time of assessment. 23 % of the patients had at least one personality disorder after their TBI, 15 % were avoidant, 8 % were paranoid, and 7 % were schizoid. The findings of this study suggests that, not only can TBI cause psychiatric disorders, but the effects of TBI on psychological health can be long-lasting, in many cases lasting longer than 30 years. In particular, TBI seems to be a major risk factor for disorders, such as major depression, substance abuse, and the development of various personality disorders.

While there is a plethora of research on psychological outcomes of TBI, there tends to be numerous problems that researchers run into when attempting to study this phenomenon. First, a common methodological problem is that most studies do not take into account is that individuals with TBI tend to have difficulty with retrospective reporting of issues before their TBI. Being that one of the strongest predictors of psychological illness is prior psychological illness, this leads one to question the validity in results of psychological illnesses resulting from TBI. Second, some studies only look at the presenting disorder at the time of the study, which may be 1–30 years after the TBI, rather than looking at the whole history of psychological problems. This large range in time also makes it difficult to understand a timeline and progression of psychological problems after a TBI.

In order to address some of these concerns, Ashman et al. (2004) conducted a longitudinal study and a simultaneous cross-sectional study to examine the frequency of Axis I disorders in persons with TBI during the first 6 years post-injury. At the Research and Training Center in the Department of Rehabilitation and Medicine at

Mount Sinai School of Medicine in New York City, 188 participants that had received a TBI within the previous four years were recruited. Participants completed either two or three assessments, each one-year apart from each other. The semi-structured clinical interview called the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition was utilized in order to assist clinicians in diagnostic accuracy. Of the 188 participants, 29 % have mild TBI, 62 % had moderate or severe TBI, and 9 % had loss of consciousness of unknown duration. One important finding from this study was that there were few cross-sectional differences in age; thus, age at the time of injury had little impact on Axis I diagnoses. In regards to gender, significantly more women met criteria for PTSD, depression, and anxiety disorder after their TBI than men. However, significantly more men met criteria for a substance abuse disorder. Also, the researchers found that psychological disorders pre-injury significantly predicted the presence of post-injury diagnosis. When controlling for this factor there was still a significant frequency of depression, PTSD, and anxiety post-TBI. Overall, the results of the study indicated that: (1) there is a high frequency of individuals that develop an Axis I disorder after TBI, and (2) there is an inverse relationship between odds of developing an Axis I disorder after TBI and time since injury, meaning your chances of having an Axis I disorder after a TBI declines over time.

Posttraumatic Stress Disorder

The Diagnostic and Statistical Manual, fifth edition (DSM-5) characterizes Post-traumatic Stress Disorder (PTSD) by the development of distinct symptoms after exposure to one or more traumatic events. Exposure can consist of directly experiencing the event, witnessing a traumatic event, learning about traumatic events that have happened to loved ones, and being exposed to the aftermath of traumatic events. Another feature of PTSD is the presence of intrusive symptoms, such as nightmares, flashbacks, or marked physiological reactions to internal or external cues that remind the person of the trauma. Persistent avoidance of such cues and familiar stimuli, as well as marked changes in cognition and arousal are typically present. Changes in cognition may present as difficulty with memory, distortions about the cause or consequences of the traumatic event, fear, horror, anger, diminished interests, and inability to experience positive emotions (American Psychiatric Association 2013). Alterations in arousal and reactivity often present as irritability, anger outburst, recklessness, hypervigilance, problems with concentration, and sleep disturbances (American Psychiatric Association 2013).

Although this is just one disorder, the clinical presentation can vary. While some individuals with PTSD present predominately with a depressed mood and negative cognitions, others are characterized by a more fear-based, behavioral and emotional reaction (American Psychiatric Association 2013). In others, hypervigilance and arousal are predominant, while in some a more dissociative reaction is present (American Psychiatric Association 2013). Neuropsychologically speaking, PTSD

has been shown to cause significant impairments in memory, learning, attention, and executive functioning (Johnsen and Asbjørnsen 2008; Vasterling et al. 1998; Yehuda et al. 2004).

The DSM-5 reports that the lifetime risk of developing PTSD in the United States is 8.7 %, and the 12-month prevalence among adults is 3.5 % (2013). Not surprisingly so, an estimated one-third to more than one-half of those who are survivors of rape, military combat and captivity, and political or cultural internment and genocide develop PTSD. This disorder appears to be less prevalent in young children and older adults who are exposed to a traumatic event.

Acute Stress Disorder. While the main focus of this book is PTSD and TBI, an explanation of acute stress disorder is warranted due to its strong predictive power of PTSD. Acute stress disorder is essentially the same disorder with the same symptom presentation as PTSD, however, the key difference is the timeline. Acute stress disorder is diagnosed when the symptoms are present 3 days to 1 month after exposure to the traumatic event(s), whereas PTSD is diagnosed when the symptoms persist for more than 1 month. In order to investigate the relationship between acute stress disorder and PTSD, Harvey and Bryant (1998) assessed 92 motor vehicle accident survivors for acute stress disorder within 1 month of their trauma, and again at 6 months post-trauma for PTSD. After the first round of assessments within 1 month, 13 % of participants were diagnosed with acute stress disorder and 21 % had subclinical levels. At the 6-month follow-up, 78 % of the acute stress disorder patients and 60 % of the subclinical patients met criteria for PTSD. Specifically, the symptoms that had the strongest predictive power were acute numbing, depersonalization, sense of reliving the trauma, and motor restlessness. Countless studies since Harvey and Bryant's has supported the strong relationship between acute stress disorder and PTSD, and with the changes to both disorders in the latest DSM-5, the relationship appears to be stronger than before.

Neurocircuitry of Posttraumatic Stress Disorder. A unique feature of PTSD in comparison to most other psychiatric disorders is that the etiology is almost always well defined. Having such a specific cause helps neuroanatomical and neuropathological research, allowing researchers over the past few decades to use neuroimaging to test neurocircuitry hypotheses. To date, the strongest neurocircuitry model for PTSD is the fear-conditioning model. This model is based off of the three types of symptoms that characterize PTSD: (1) reexperiencing (flashbacks, nightmares, and physical pains), (2) avoidance (avoiding things that are reminders of the trauma, feeling numb, and losing interests in people and activities), and (3) hyperarousal (hypervigilance, easily startled, tension, emotionally labile, and difficulty sleeping). By connecting these symptoms with what is known about specific regions of the brain, it was determined that the limbic system, a region that plays a large role in emotional processing, appears to be involved in PTSD. Specifically within the limbic system, the brain structures implicated in PTSD are the prefrontal cortex (PFC), amygdala, and the hippocampus. The PFC is considered to be the brain region responsible for decision-making, personality, complex behavior, and social behavior. The amygdala, the control center for the fight-or-flight response, plays a key role in the learning and memory of fear responses. The hippocampus is best

known as the region of the brain for short-term and long-term memory storage. After exposure to trauma, those with PTSD evidence reduced activation in the PFC and hippocampus, allowing the amygdala to over-respond to any potentially fearful events. The hyperresponsivity of the amygdala causes the strong emotional tie with the memory of the traumatic event, the under-activation of the PFC prevents the suppression of attention to trauma-related stimuli, and reduced hippocampal functioning causes the difficulties with the identification of safe stimuli and accompanying explicit memory difficulties (Bremner et al. 1995; Rauch et al. 2006).

Relationship Between TBI and PTSD

The acknowledgment that there is some form of relationship between TBI and PTSD, whether intentional or not, has been noted throughout history. Dating back to World War I, soldiers who were frequently exposed to mortar attacks and grenade blasts while fighting in the trenches were often diagnosed as having “Shell Shock”. Shell Shock was a disorder characterized by amnesia, headaches, dizziness, tremors, and hypersensitivity. While such symptoms would typically be seen after a mild TBI, these soldiers evidenced no visual signs of head injuries. At the time, due to a lack of knowledge, doctors from all over disagreed on the cause of these symptoms. Some doctors posed that the soldiers had a hidden brain injury caused by the blast waves, while others argued the symptoms were due to carbon monoxide poisoning formed by the explosions. However, slowly overtime, doctors started to see soldiers with Shell Shock symptoms that were never exposed to explosions or mortar attacks; thus, the idea of a psychological cause was formed.

With the growth of research we have now made many distinctions between brain injury and psychological damage. However, the prevalence of comorbidity, as well as the difficulty of distinction between the correct origins of symptoms denotes the necessity for deeper understanding of the brain-behavior relationship in individuals with such disorders.

One of the first articles written to describe the occurrence of PTSD after a TBI was done so by McMillan (1991), in which he described the case of an 18-year-old female who was involved in a car wreck that resulted in a severe brain trauma and the death of her passenger. It was reported that she lost consciousness for at least three days. Initially she suffered from mild right hemiparesis, mild dysphasia, euphoria, memory difficulties, and little insight. However, with rehabilitation, she made a strong recovery and returned to work after seven months. Fourteen months after the accident she returned complaining of fatigue, difficulty with concentration and coping at work, and some dizziness and severe headaches. Additionally, she expressed feelings of depression, failure, loss of interests, poor appetite, and hopelessness, obtaining a score of 27 (moderately severe range) on the Beck Depression Inventory (BDI). She was described by her mother to be irritable, verbally aggressive, and moody.

The patient reported having frequent intrusive thoughts of her dead friend throughout the day, as well as survivor guilt and strong anxiety when she thought about the wreck or when she entered a hospital. Along with other symptoms, she met full criteria for PTSD, while having a moderate degree of general impairment evidenced by neuropsychological testing 14 months after the wreck. After 4 months of therapy her BDI score fell to a 9 (not depressed), and her symptoms improved dramatically. This article serves as one of the first case studies to report in-depth that PTSD can develop despite experiencing a loss of consciousness. Moreover, that treatment for PTSD symptoms in an individual with TBI can prove to be efficacious.

In controversial study conducted by Sbordone and Liter (1995), the authors stated that it is highly unlikely that mild TBI patients actually develop PTSD symptoms. They examined 70 patients who had a previous diagnosis of either PTSD or mild TBI, and asked them to, in the most detail as possible, describe the traumatic event and the symptoms they developed from said event. The researchers found that while all of the patients with PTSD could provide a very detailed and emotionally charged recollection, none of those with mild TBI could. Moreover, none of the mild TBI patients reported any symptoms of intrusive thoughts, nightmares, hypervigilance, or startle reactions, nor did they become upset while talking about their traumatic event.

One of the first major studies to look at the neuropsychological relationship between PTSD and TBI was conducted by Hickling et al. (1998). Fueled by the desire to clear up the controversy as to whether one can actually develop PTSD after experiencing a TBI with loss of consciousness, the researchers attempted to answer two questions. First, they sought to determine whether motor vehicle accident (MVA) survivors who reported a loss of consciousness during their accident actually have lower rates of PTSD than those with no loss of consciousness. Second, the researchers posed if what is being called PTSD actually is due to brain injury, then those who meet criteria for PTSD should perform more poorly on neuropsychological testing; thus, they wanted to examine if those diagnosed with PTSD have greater neuropsychological dysfunction than those without PTSD in a brain-injured population. Of the 107 MVA survivors, 38 were diagnosed with PTSD. The researchers found that 40 % of those injured badly enough to lose consciousness met criteria for PTSD. Additionally, there were no differences found on neuropsychological testing between those who met criteria for PTSD and those who did not. Thus, this study suggests that many symptoms that are often attributed to PTSD may actually reflect the effects of TBI.

Bryant and Harvey (1998) conducted a study to determine if the occurrence of acute stress disorder following a mild TBI could be used to predict the development of PTSD. The researchers recruited 79 motor vehicle accident patients that sustained mild TBIs and tracked them for 6 months. Within 1 month of their injury patients were assessed for acute stress disorder, and after 6 months were assessed for PTSD using the PTSD module of the Composite International Diagnostic Interview. Acute stress disorder was diagnosed in 14 % of patients at 1 month, and at the 6-month follow-up 24 % satisfied criteria for PTSD. Of those diagnosed with acute stress disorder, 82 % were ultimately diagnosed with PTSD. Interestingly though, PTSD

was diagnosed in 11 % of those who had not been diagnosed with acute stress disorder. This study provided two important findings, (1) PTSD after mild TBI is definitely a concern that should be addressed, and (2) acute stress disorder, although a strong predictor, does not always precede PTSD. In addition to these findings the authors discussed two important topics. First, diagnosing acute stress disorder after TBI could possibly be problematic because of the similarity and overlap of symptoms with postconcussive symptoms. Both acute stress disorder and postconcussive symptoms can present as derealization, depersonalization, and amnesia. Second, the authors point out that their frequency of PTSD with a TBI (24 %) after a motor vehicle accident is consistent to another study's finding of PTSD after a motor vehicle accident with no TBI (39 %; Blanchard et al. 1996), supporting that TBI does not impact the formation of PTSD.

Two years after their motor vehicle accident, Harvey and Bryant (2000) attempted to contact the original 79 patients for a follow-up evaluation, at which time 50 patients were willing to participate in the study. At the 2-year assessment, 22 % of the patients met criteria for PTSD. It was found that 80 % of the patients originally diagnosed with acute stress disorder met criteria for PTSD after 2 years. Interestingly, of those who were originally not diagnosed with acute stress disorder, 8 % met criteria for PTSD.

After investigating if PTSD could develop after mild TBI, Bryant et al. (2000) sought to determine if PTSD could occur after severe TBI. They utilized the theory that postulates the conditioned fear of trauma is mediated in subcortical regions of the brain rather than in higher cortical processes, suggesting that even when severe brain injury (which is typically cortical) occurs, one is still able to reexperience the trauma. Bryant and colleagues predicted that those who develop PTSD after severe TBI would have trauma reexperiencing in the form of emotional and physiological reactivity instead of intrusive memories.

The researchers assessed 96 severely brain-injured patients 6 months after their injury and found that 27 % met criteria for PTSD. Upon further analysis they found that only 19.2 % of the patients with PTSD reported intrusive memories of the trauma, while 96.2 % reported emotional reactivity and 50 % reported physiological reactivity. Specifically, symptoms, such as intrusive memories, nightmares, and emotional reactivity, were found to have very strong positive predictive powers for the development of PTSD. These findings support their theory that first, PTSD can develop after severe brain injury, and second, trauma reexperiencing can be mediated by fear conditioning or mental representations rather than explicit memories.

Williams et al. (2002) also investigated the prevalence of PTSD symptoms after severe TBI. The authors utilized a community sample of 66 individuals, 51 of which had been involved in road accidents (30 as drivers, 11 as passengers, 7 as pedestrians, 3 as cyclists), 12 suffered falls, 2 were physically assaulted, and 1 was involved in a bomb explosion. The sample varied significantly with a range of 1–26 years since their traumatic event, age range of 17–70 years of age, and an education range of 9–19 years. Duration of loss of consciousness and posttraumatic amnesia were used to determine TBI severity level. The overall finding was that 18 % of their community sample had PTSD symptoms, of which 6 % had severe symptoms. It is important to

note that this finding is lower than what was found by Hickling et al. (1998) in individuals with mild TBI, suggesting that more severe the brain injury is, the less likely one is to develop PTSD afterwards.

While it was becoming supported that TBI and PTSD can co-occur, Van Reekum et al. (2000) sought to determine if there is a causative relationship between TBI and psychiatric disorders. The authors point out that if a causative relationship is found, it will have major implications for preventative measures after TBI, as well as litigation outcomes. Often it is the case that neuropsychologists are determining if someone's post-TBI difficulties are due to their TBI or due to a psychiatric disorder, as if they are separate. However, if there were a causative relationship, then one's problems would be secondary to psychiatric disorder, which is secondary to the TBI. Reekum and colleagues conducted a literature review on 42 articles, looking at disorders such as Depression, Bipolar, Generalized Anxiety Disorder, Obsessive–Compulsive Disorder, Panic Disorder, PTSD, Schizophrenia, Substance Abuse, and Personality Disorders. While there was strong evidence that TBI frequently caused some psychiatric disorders (Depression, Bipolar, Anxiety Disorders), there was no evidence that TBI caused PTSD. Actually, the findings suggested an inverse relationship between TBI and PTSD, in that PTSD is more common amongst mild TBI than it is amongst moderate or severe TBI, supporting the statement made by Williams et al. (2002). The authors raise the point that more severe TBI may be a protective factor for some psychiatric disorders due to sequelae such as reduced insight.

Bombardier et al. (2006) recognized that while numerous studies have looked at the prevalence rate for PTSD after TBI, very few have investigated if factors found to be predictive of PTSD in other patient populations increase the risk of developing PTSD in a TBI population. Predictors such as being female, little education, history of anxiety or depression, less severe brain injury, being assaulted, strong emotional reactions to the incident, and being under the influence of stimulant drugs. Another main question to their study was to what extent is meeting symptom criteria for PTSD associated with other current or past psychiatric disorders. Patients were recruited from a hospital in Seattle, Washington, and were determined to have a TBI by either radiological evidence of acute brain abnormality or a GCS score less than or equal to 12 within the first 24 h of admission. Over the course of 6 months, 125 participants were administered the 17-item PTSD Checklist-Civilian Version (PCL-C), the depression, panic, and anxiety modules of the Patient Health Questionnaire (PHQ), the one-item General Health Scale from the SF-36, as well as a interview inquiring about history, demographic data, and medical variables.

The authors found that in their sample of complicated mild to severe TBI, 11.3 % met PTSD symptom criteria. They also found that those with more severe TBI had a lower incidence of PTSD than those with milder TBI. The authors point out that the incidence of PTSD after TBI from a motor vehicle accident is much lower than PTSD after a motor vehicle accident with no TBI, which is at least 34 % (Blanchard et al. 1995; Ursano et al. 1999). In regards to factors that contribute to the diagnosis of PTSD, the researchers found that people with less than a high school education were at a higher risk than those with more education. Also, those who recall feeling

terrified or helpless, as well as those that were assaulted, were more likely to meet criteria for PTSD. Lastly, those who had used stimulant drugs (such as cocaine or amphetamine) around the time of trauma were more likely to develop PTSD. Interestingly, while meeting criteria for PTSD was significantly related to greater psychosocial impairment, it was not related to poorer subjective health ratings. However, the authors only using one question to measure subjective health may have limited this. Probably the most salient issue raised by this study is the necessity of assessing past and current psychological history. The authors reported that 29 % of those who met PTSD symptom criteria reported a history of PTSD before the accident. Thus, PTSD symptoms after a TBI may really just be a continuation or exacerbation of the individual's previous diagnoses. Additionally, it was remarkable that 79 % of those who met PTSD symptom criteria also reported symptoms consistent with major depressive disorder. Moreover, 71 % of those that met PTSD symptom criteria reported having major depressive disorder before their injury. Thus it is suggested that depression may play a large role as a risk factor for PTSD after TBI.

At this point, almost all studies looking at the relationship between PTSD and TBI were conducted in adults. Consequently, Mather et al. (2003) explored the relationship between PTSD and presence of mild TBI in children following road traffic accidents. Criteria used by the researchers were children had an age between 6 and 16 years old, currently enrolled in school, and if they had received a mild TBI there was witnessed loss of consciousness and an initial GCS of 13–15 that returned to a full GCS within 24 h of injury. The average age of the 43 participants was 9.7 years, and the sample was comprised of 20 males and 23 females. Twenty of the children were passengers in motor vehicle accidents, 17 were hit as pedestrians, and 6 were on a motorcycle or bicycle. Of the sample, 14 sustained mild TBI and the remaining 29 were classified as not brain injured.

The Children's Posttraumatic Stress Reaction Index (CPTS-RI) was used to measure PTSD symptomatology. The children were also administered the Revised Children's Manifest Anxiety Scale and the Children's Depression Inventory for self-reported anxiety and depression levels, respectively. Parents completed the PTSD module of the Anxiety Disorders Interview Schedule-Children Version to assess their report of their child's PTSD symptomatology, as well as the Child Behavior Checklist (CBCL) to assess internalizing and externalizing behaviors displayed by their children.

Overall, the researchers found that 74 % of children evidenced significant PTSD symptomatology roughly 6 weeks after their accident. There was not a significant difference between those who sustained a mild TBI and those that did not. 86 % of the children with mild TBIs, and 69 % of the children with no brain injury were classified as experiencing significant levels of PTSD symptomatology. This finding is interesting because previous studies with adults suggests that the presence of brain injury decreases the chances of developing PTSD after a traumatic event, however, these results suggest the opposite in children. In regard to comorbidity, children with PTSD were significantly more likely to have higher levels of anxiety and depression.

While the majority of the children had a reduction in PTSD symptomatology over time, one child that initially had no PTSD-like symptoms evidenced severe PTSD

at the follow-up assessment. Interestingly, two of this child's siblings that were also involved in the same accident evidenced severe PTSD initially, suggesting that being around their siblings may have caused this child to develop PTSD. Another important factor this study highlights is the accuracy of parental report, which may be detrimental to proper assessment. The researchers found that while 74 % of children endorsed some level of PTSD symptomatology, only 42 % of parents reported significant PTSD symptoms in their children. The authors note that some of this discrepancy may have been due to the difference between the parent report and child report questionnaires, however, it seems that this still only highlights the necessity for careful and thorough evaluations in children.

Military Posttraumatic Stress Disorder and Traumatic Brain Injury. While many researchers still study the relationship between TBI and PTSD, the focus of population has heavily changed. Until the early 2000's most studies were on individuals who received TBIs and PTSD from motor vehicle accidents, assaults, or falling. However, over the past 15 years the focus has changed as a result of the September 11, 2001 terrorists attack on the World Trade Center and the Pentagon. The relationship between PTSD and TBI has become more publicized and discussed now than ever before, with a strong focus on military population. In October, 2001, Operation Enduring Freedom (OEF) was launched, followed by Operation Iraqi Freedom (OIF) in March, 2003. Three additional smaller operations, Operation New Dawn, Operation Inherent Resolve, and Operation Freedom's Sentinel have also been conducted. An estimated 2.7 million military service members have been deployed to war zones since 2001, and more than half of them have been deployed more than once. At least 970,000 veterans have some degree of disability as a result of the wars, and countless live day-to-day with unrecognized physical and psychological scars.

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 gunshots, 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. Due to the increase of survival from 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). Between 10 and 17 % of troops deployed to combat zones have developed PTSD (Sundin et al. 2010). Hoge et al. (2008) found that 43.9 % of soldiers who reported loss of consciousness during battle injury met the requirements for PTSD. With such a high rate of exposure to physically and psychologically traumatic events, exploring the literature on TBI and PTSD in a military population is crucial to understanding these disorders.

Hoge et al. (2008) conducted one of the most prominent studies on mild TBI in returning U.S. soldiers to date. The focus of their study was on the prevalence and significance of self-reported history of combat-related mild TBI among soldiers after a year long deployment to Iraq. They sought to provide information that would further the literature on prevention and treatment strategies. In-depth questionnaires were sent to 4618 U.S. Army soldiers, from which 2525 soldiers were ultimately included in the study. The questionnaire asked whether or not the soldiers had been injured during deployment, what they were injured by, whether they received a mild TBI from the accident, and immediate symptoms of their accident (loss of consciousness, seeing stars, confusion, etc.). Combat intensity was measured using 17 of the 18 questions from the Combat Experiences Scale. Soldiers were asked to rate their overall health, and also completed the Patient Health Questionnaire 15-item somatic symptom severity scale (PHQ-15). An additional five questions were asked regarding post-concussive symptoms about memory, balance, concentration, ringing in the ears, and irritability. Depression and PTSD were assessed by using the 9-item depression assessment module of the PHQ and the 17-item National Center for PTSD Checklist, respectively.

Overall, 4.9 % of soldiers reported an injury with loss of consciousness, while 10.3 % endorsed an injury with an altered mental status without loss of consciousness. Hoge et al. (2008) found that soldiers who endorsed mild TBI were significantly more likely to report a blast mechanism of injury, exposure to more than one explosion, high combat intensity, and hospitalization. As already noted, 43.9 % of soldiers who reported loss of consciousness during battle injury met the requirements for PTSD. 27 % of those with an altered mental status but no loss of consciousness met criteria for PTSD. It was found that loss of consciousness and combat intensity were the only two factors significantly associated with PTSD symptomatology. Consistent with literature from civilian population, injury with loss of consciousness was significantly related to the development of major depressive disorder, as well as poorer general health. So overall, soldiers with mild TBI reported significantly higher rates of physical and mental health problems, and injuries with loss of consciousness resulted in a much greater risk of health problems.

Although numerous studies show that PTSD and TBI have a high comorbidity rate, very few truly take a look at the accuracy and best method of assessment for these disorders. There is currently no definitive method for determining which symptoms are due TBI and which are due to PTSD. While some symptoms are more clear-cut than others, there are numerous common symptoms that could go either way. It has been suggested that one method of segregating PTSD from TBI symptoms would be conducting PTSD or TBI specific treatment to see which symptoms subside and which remain. Although initially this seems like a possible solution, various researchers argue that due to the “biological interface” that suggests a physiological correlation between PTSD and TBI, treatment may alleviate both TBI and PTSD symptoms, in turn, providing inconclusive results (Church and Palmer-Hoffman 2014; Kennedy et al. 2007).

On the other hand, Church and Palmer-Hoffman (2014) raise the point that the results of such treatment may in actually just highlight the difficulties we have in

distinguishing between such disorders and the lack of knowledge we have in the treatment capabilities for TBI and PTSD individually. Church and Palmer-Hoffman (2014) sought to examine whether etiology (PTSD or TBI) was important in terms of treatment outcomes by providing emotional freedom techniques (EFT) coaching to 59 veterans with PTSD, to determine whether the resolution of PTSD symptoms would correlate with a reduction in TBI symptoms. Emotional freedom technique is a brief exposure therapy with somatic and cognitive components. During this treatment method, patients are asked to pair the memory of a traumatic event with a statement of self-acceptance, while simultaneously stimulating 12 different acupressure points with finger tips. The researchers noted that while EFT has been shown to meet APA's Division 12 criteria for empirically supported treatments as a "well-established treatment" for PTSD, little is known of the impact it may have on TBI symptomatology.

Of the 59 veterans, 30 comprised the EFT group while 29 made up the wait-list control group. Participants completed assessments at baseline, after three sessions, after six sessions, and at 3- and 6-month follow-ups. Posttraumatic stress disorder symptoms were screened for by using the global severity index and positive symptom total on the Symptom Assessment-45, while the PCL-M (PTSD Checklist-Military version) was used at each assessment. The authors indicated that because there is no generally accepted brief TBI screener, nine items from the Patient Health Questionnaire somatoform module of the Primary Care Evaluation of Mental Disorders (PRIME-MD), along with a list of 17 TBI symptoms were used to assess for TBI. After isolating TBI and somatoform symptoms, analyses indicated a significant reduction in TBI symptoms after three EFT sessions, and further reductions were shown after six sessions. The reductions in symptoms were maintained after 3-months and 6-months. Many individuals who have sustained a mild TBI still report experiencing postconcussive symptoms (headache, fatigue, memory difficulties) years after their injury. However, in Church and Palmer-Hoffman's (2014) sample, both somatoform symptoms and TBI symptoms were significantly reduced. While there are certainly limitations to their study, the results still shed light on just how little we still know about the relationship between TBI and PTSD, as well as our ability to differentiate etiology of symptomatology.

Screeners and questionnaires are often used in medical and private practice settings due to their time efficiency and low cost, allowing clinicians to quickly and relatively cheaply gain insight into a client on multiple domains. While presenting and brief history of symptoms are crucial to an evaluation, these components are only pieces to the puzzle. In addition to understanding all of the present symptoms, a clinician must take a detailed history of the client and their traumatic event. Lange et al. (2014) sought to identify factors that are predictive of the endorsement of PTSD and postconcussive symptoms after a TBI in a military population. The researchers looked at a total of 22 factors related to demographic variables, injury circumstances, injury severity, treatment/evaluation, and psychological/physical symptoms.

Participants of the study were 1600 U.S. service members who sustained a mild to moderate TBI and were evaluated by the Defense and Veterans Brain Injury Center. Diagnosis and classification of TBI severity was primarily conducted by a

Physician's Assistant or Nurse who were trained to evaluate the presence and severity of TBI. The medical professionals determined severity and presence by conducting a comprehensive clinical screening that consisted of a patient interview, a comprehensive medical chart review, case conferencing, and a family interview and collection of other collateral information. Loss of consciousness (LOC), post-traumatic amnesia (PTA), and alteration of consciousness (AOC) were used to classify TBI severity. The authors reported that GCS scores were not available.

For a classification of moderate TBI one must have had a LOC for longer than 30 min to 24 h, PTA for 1–7 days, and the presence or absence of intracranial abnormality. Complicated mild TBI was classified as a LOC for less than or equal to 30 min, PTA for less than 24 h, and the presence of intracranial abnormality. Uncomplicated mild TBI had the same criteria, except for the need of an absence of intracranial abnormality. Equivocal mild TBI was classified by having no PTA or LOC, with a present AOC. Additionally, the Neurobehavioral Symptom Inventory (NSI), a 22-item measure that evaluates self-reported postconcussive symptoms, was utilized for assessing the presence and severity of TBI. The PCL-C Version, a 17-item self-report measure, was used for evaluating PTSD symptoms.

Overall, the authors found four factors to be statistically related to postconcussive symptom endorsement. The four factors were as follows: low bodily injury severity, posttraumatic stress symptoms, depression, and being wounded during a military operation related to the Global War on Terrorism (GWOT), with depression and posttraumatic stress symptoms as the most strongly associated with clinical elevations in postconcussive symptoms accounting for 41.5 % of the variance. Interestingly, brain injury severity was not associated with symptom reporting following TBI.

This study supports the findings of other studies that suggest PTSD and depression largely explain the relation between a history of TBI and postconcussion symptoms reporting. Lange et al. (2013) that clinically meaningful postconcussion symptom reporting occurs only 5.6 % of the time when there is an absence of these four factors: (1) symptom exaggeration, (2) poor cognitive effort, (3) depression, and (4) traumatic stress. If anything, the work of Lange and colleagues shed light on the numerous factors that must be taken into consideration when evaluating individuals that present with TBI.

As discussed earlier in this chapter, understanding the ramifications of being exposed to blasts are still in its infancy stages. Lippa et al. (2010) conducted profile analyses to explore the differences in self-reported postconcussive symptoms in 339 veterans reporting mild TBI dependent upon their mechanism of injury (blast only, nonblast only, or both blast and nonblast), distance from the blast, and number of blast injuries. The criteria used for mild TBI in this study were a self-reported LOC of 30 min or less, or disorientation for 24 h or less, following a credible injury mechanism. The NSI was used to measure postconcussive symptoms, and symptoms of PTSD were measured using the National Center for PTSD 17-item checklist (PCL). The PCL was developed to correspond with the Diagnostic and Statistical Manual-Fourth Edition (DSM-IV 1994). Similarly to Lange et al. (2014), the authors found that PTSD symptoms accounted for a considerable portion of variance in

postconcussive symptom report. Additionally, it was discovered that PTSD is more common in those with histories of blast-related TBIs than those with nonblast-related TBIs. However, neither the number of blast injuries nor the distance from the blast was correlated to total PTSD symptoms reported.

Neuroanatomy of PTSD with TBI

The advancement of technology has allowed researchers to explore the brain in a whole new way. Some studies have used functional magnetic resonance imaging (fMRI) and found that individuals with PTSD and mild TBI share abnormalities in the frontal lobes, more specifically the dorsolateral prefrontal, orbitofrontal, medial frontal, and the anterior cingulate cortex (Shu et al. 2014; Simmons and Matthews 2011; Stein and McAllister 2009). Individually, patients with PTSD tend to have hyperactivity in the medial frontal and anterior cingulate areas (Carrion et al. 2008; Matthews et al. 2011; Swick et al. 2012), while the neuroanatomical differences in those with mild TBI only vary case by case. Shu et al. (2014) utilized electroencephalography (EEG) to test whether those with PTSD and TBI share abnormal activation in various frontal regions, specifically the anterior cingulate cortex.

The researchers believe that PTSD symptomatology may particularly mediated by the anterior cingulate cortex, and this difference may be apparent during cognitive control tasks that require response inhibition. Participants were composed of 32 combat veterans, 17 with a mild TBI and PTSD, 15 with a mild TBI and no PTSD. A stop task was performed by each participant during EEG monitoring, requiring the inhibition of initiated motor responses. Interestingly, Shu et al. (2014) found that those with PTSD and mild TBI had a greater inhibitory processing event related potential (ERP) in the dorsal anterior cingulate. The researchers concluded that in veterans with mild TBI, larger ERPs in the dorsal anterior cingulate are associated with higher PTSD symptom endorsement. They continued to explain that this relationship is likely related to complications with controlling ongoing brain processes, including thoughts and consequently feelings about their trauma.

Yeh et al. (2014) investigated the differences in white matter between blast and impact injury, along with the impact of postconcussion and PTSD symptoms. Participants were 37 US service members, comprising of 29 with mild, 7 with moderate, and 1 with severe TBI; 17 experienced blast trauma and 20 were considered nonblast. TBI evaluations included a patient interview, a comprehensive medical chart review, case conferencing, and a family interview for collateral information. The diagnosis of TBI was based on the presence of duration of LOC, PTA, AOC, and neuroimaging. Mild TBI was considered as AOC or LOC for 30 min or less, or PTA for less than 24 h and no radiological abnormalities. Moderate TBI criteria were comprised of positive neuroimaging findings, PTA for more than 24 h, or LOC for more than 30 min. Finally, severe TBI was diagnosed for those with PTA for longer than one week or LOC for more than 1 day. Postconcussion was assessed for by using the

NSI, and the PCL-C was used for PTSD. Diffusion tensor imaging (DTI) was used to assess the neurocircuitry by fiber tracking and tract-specific analysis, along with region of interest analysis. Overall, for both blast and nonblast patients, the most common white matter injury was in the fronto-striatal and fronto-limbic circuits, along with the fronto-parieto-occipital association fibers.

The researchers reported finding significant differences between the blast and nonblast groups in subcortical tracks. Specifically, subcortical superior–inferiorly oriented tracks were more susceptible to blast injury, while anterior–posteriorly oriented tracks were more impacted by direct force trauma. In regards to the influence of PTSD and subconcussive symptoms, the tractography revealed higher endorsement of both PTSD and subconcussive symptoms was associated with low fractional anisotropy in the major nodes of compromised cortico-striatal-thalamic-cerebellar-cortical (CSTCC) network.

TBI, PTSD, and Alzheimer’s Disease

Numerous studies have linked TBI to an increased chance of developing Alzheimer’s disease, as well as causing an earlier onset for Alzheimer’s disease (Bilbul and Schipper 2011; Jellinger 2004; Johnson et al. 2010; Lye and Shores 2000). Likewise, some studies have found a correlation between presence of PTSD and development of dementia (Qureshi et al. 2010; Yaffe et al. 2010). Yaffe et al. (2010) found that those diagnosed as having PTSD were at almost double the risk of developing dementia compared with those without PTSD. They posed that PTSD might be involved in accelerating the aging of the brain, being that PTSD often last late into life and has been found to cause hypothalamic–pituitary–adrenal axis dysfunction. These researchers also discussed how some have found that veterans with PTSD have smaller hippocampal volumes, which have been shown to correlate with deficits in short-term memory performance. Since smaller hippocampal volumes are associated with poor cognitive function and increased risk of dementia in healthy elderly people, it may be argued that PTSD causes hippocampal atrophy, which in turn increases risk of cognitive decline and dementia. Yaffe et al. (2010) also points out that it is also possible a smaller hippocampus is a predisposition factor for both PTSD and dementia.

Weiner et al. (2014) are currently conducting a investigating the relationship between PTSD, TBI, and Alzheimer’s disease. Since both PTSD and TBI have been independently associated with Alzheimer’s disease, the present researchers hypothesize that TBI and/or PTSD reduce cognitive reserve, causing greater cognitive impairment after adjusting for age, education, prewar cognitive functioning, brain amyloid load, and hippocampal volume; and that there will be significant relationships between severity of PTSD and TBI and greater cognitive impairment. All participants will be administered the Clinician Administered PTSD Scale (CAPS) to identify PTSD symptomatology, along with a full battery of neuropsychological tests comprised of the Montreal Cognitive Assessment, everyday cognition, the

Mini-Mental State Examination, the Alzheimer's Disease Assessment Scale-Cognitive 13, the Logical Memory Test I and II, the Boston Naming Test, the Category Fluency Test, the Clock Drawing Test, the American National Adult Reading Test, the Auditory Verbal Learning Test, the Trail Making Test Parts A and B, the Clinical Dementia Rating, the Activities of Daily Living/Functional Assessment Questionnaire, the Neuropsychiatric Inventory, and the Geriatric Depression Scale. Additionally, cerebrospinal fluid (CSF) will be obtained at baseline using a lumbar puncture. Both amyloid PET images and MRI will be performed. Currently, participants are still being recruited; however, to date, this appears to be the largest study to look in-depth at the neurological and neuropsychological relationship between PTSD, TBI, and Alzheimer's disease.

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in Neuropsychological Evaluations

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