

NEUROPSYCHOLOGICAL FUNCTIONING IN ACTIVE DUTY SOLDIERS WITH
PHYSICAL AND/OR PSYCHOLOGICAL TRAUMA

Robert S. Klein

Thesis Prepared for the Degree of
MASTER OF SCIENCE

UNIVERSITY OF NORTH TEXAS

December 2010

APPROVED:

Kenneth Sewell, Major Professor
Craig Neumann, Committee Member
Daniel Taylor, Committee Member
Vicki Campbell, Chair of the Department of
Psychology
James D. Meernik, Acting Dean of the Robert
B. Toulouse School of Graduate Studies

Klein, Robert S. Neuropsychological Functioning in Active Duty Soldiers with Physical and/or Psychological Trauma. Master of Science (Psychology), December 2010, 110 pp., appendices, references, 118 titles.

This quasi-experimental study investigates neuropsychological functioning differences between 63 active duty soldiers who were placed into three groups (MTBI, PTSD, control) to provide better information for differentiating PTSD and MTBI. The ANAM and MicroCog were utilized to measure psychomotor speed, memory, and attention. Participants with PTSD performed worse on most measures of psychomotor speed and attention, and endorsed more symptoms of depression and anxiety when compared to MTBI and control participants. Further, attention appears to be the best cognitive domain for differentiating PTSD from MTBI, whereas memory variables did not differentiate these groups. Clinical and research implications of these findings are discussed.

Copyright 2010

by

Robert S. Klein

TABLE OF CONTENTS

	Page
1. INTRODUCTION.....	1
Posttraumatic and Mild Traumatic Brain Injury	
Topic Outline	
Posttraumatic Stress Disorder	
Mild Traumatic Brain Injury	
Overlapping Symptoms	
Study Objectives	
2. METHOD.....	24
Participants	
Setting and Apparatus	
Instruments	
Procedure	
3. RESULTS.....	38
Data Examination	
Demographic Data	
Data Analytic Strategy	
Hypothesis Testing	
Psychomotor Speed	
Attention	
Memory	
Supplementary Analyses	
4. DISCUSSION.....	52
Clinical and Operational Implications	
Direction for Future Research	
Limitations	
Conclusion	
APPENDICES	70
A: ANAM TBI MILITARY TEST LIST AND ANAM AND MICROCOG INDICES PAIRED WITH PSYCHOLOGICAL CONSTRUCTS	
B: DEFINITIONS OF MTBI FOR WHO, CDC, ACRM, AND AAN	
C: DEMOGRAPHICS CHARACTERISTICS OF THE STUDY GROUPS, PERFORMANCE ON NEUROPSYCHOLOGICAL TESTS BY STUDY GROUPS, PERFORMANCE ON BEHAVIORAL MEASURES BY STUDY GROUPS, AND DEPENDENT VARIABLES FROM SUPPLEMENTARY ANALYSIS	
D: WILK'S LAMBDA AND CANONICAL CORRELATION AND STANDARDIZED DISCRIMINANT FUNCTION AND STRUCTURE COEFFICIENTS FOR ALL GROUPS AND MTBI AND PTSD GROUPS	
E: SUMMARY OF HYPOTHESES IN ASSOCIATION WITH ALL	

ANALYSES
F: SUMMARY OF NONPARAMETRIC TEST RESULTS
G: ABBREVIATIONS AND QUESTIONNAIRE

BIBLIOGRAPHY.....97

INTRODUCTION

Posttraumatic and Mild Traumatic Brain Injury

The Global War on Terrorism has brought to the forefront the issue of the relation between mild traumatic brain injury (MTBI) and combat-induced posttraumatic stress disorder (PTSD). The two are related because of the similarities in how service members incur MTBI and/or PTSD. Service members are increasingly being exposed to concussive blasts related to improvised explosive devices (IED). In and of itself, being exposed to an IED blast meets the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) criterion A1 for PTSD and can potentially cause MTBI due to concussive blast. Typically associated with a concussive blast is a loss of consciousness (LOC). Whether PTSD co-occurs with traumatic brain injury is controversial because of LOC. The controversy revolves around whether a service member has to be conscious during the traumatic event to form a memory. One of the major criteria for the diagnosis of PTSD is memory of the traumatic event. Researchers have reported 20% to 40% rates of PTSD in those that have received a TBI (Bryant & Harvey, 1995; Hickling et al., 1998; Ohry et al., 1996; Rattock & Ross, 1993).

Complicating the diagnostic process is the presence of postconcussive symptoms in those with MTBI. Postconcussional syndrome (PCS), as defined by the World Health Organization, is persistence of three or more of the following symptoms for at least 3 months post-head injury: headache, dizziness, fatigue, irritability, insomnia, concentration or memory difficulty (International Statistical Classification of Diseases and Related Health Problems, Tenth Edition, 2007). The persistence of symptoms is assumed to be due to metabolic and physiologic changes in the brain that have not returned to homeostasis (Iverson et al., 2004). Bazarian et al. (1999) showed that postconcussive symptoms are reported more by MTBI patients without positive

neurological or radiological findings than patients with moderate or severe TBI. Research suggests that a significant risk factor for the development of PCS is three or more prior concussions, which a service member can receive via multiple combat tours (Iverson et al., 2006; Iverson et al., 2004). The diagnosis of PCS is complicated by symptoms encompassed within the syndrome and a differential diagnosis that includes among others PTSD, depression, somatization, and chronic pain. Further, there is a debate on the etiology of postconcussional syndrome; either neurological or psychological. The neurological side of the debate suggests that postconcussional symptoms are attributed to neurological damage and the psychological camp suggests that symptoms are attributed to transient physiological disturbance and are maintained by psychological distress (Levin et al., 1987; Lishman, 1988; Rutherford, 1989).

Another factor in this relation is whether bodily injury is a risk factor for PTSD. Research has shown that soldiers with bodily injury are at greater risk for developing PTSD compared to non-injured soldiers (Koren, Norman, Cohen, Berman, & Klein, 2005). Moreover, research has shown that PTSD and TBI can cause impairment in executive functioning, which further complicates making an accurate diagnosis (Lux, 2007; Twamley, Hami, & Stein, 2004). Research has shown that postconcussional syndrome can develop as a result of whiplash injury and/or postconcussive symptoms are comorbid with a whiplash injury (Evans, 1992; Miller, 1998). In addition to physiological injuries, symptoms accompanying whiplash include: concentration and memory difficulties, headache, anxiety, and depression. These symptoms overlap with PTSD and PCS, but physiological injuries such as bruising to ligaments and soft tissues of the neck and head differentiate whiplash from PTSD (Harvey, Brewin, Jones, & Kopelman, 2003). Having overlapping symptoms presents a challenge for most military clinicians when trying to determine when a service member is suffering from PTSD or MTBI.

Most of the research and statistics tracking combat related concussive blast MTBI has been done by the Defense and Veterans Brain Injury Center (DVBIC). DVBIC was established by a mandate from Congress because of the prevalence of brain injury in service members. It is a collaborative effort between the Department of Defense and Department of Veterans Affairs that includes select facilities from both departments. For example, the DVBIC center at Fort Bragg, North Carolina primarily works with soldiers who received a traumatic brain injury (TBI) from conducting parachutist operations, and the Veterans Administration hospital in Tampa, Florida is the primary hospital at which veterans with severe brain injury receive treatment. The mission of the DVBIC is to serve active duty military, their dependents and veterans with TBI through ensuring state-of-the-art medical care, innovative clinical research initiatives and educational programs (www.dvbic.org). According to the September 2008 DVBIC statistics, over 32,977 service members have been wounded in action with over half being blast related. Thirty-three percent of service members who required medical evacuation for combat related injuries to Walter Reed Army Medical Center had a TBI. Of the reported cases of TBI over 90% of combat related TBIs are closed head injuries, with most service members sustaining MTBI.

As the war in Iraq progresses and warfare changes, so has the type of client that the neuropsychologist evaluates. During the early years of this war neuropsychologists at Fort Hood would evaluate returning service members who had received multiple concussions or had second impact syndrome. Now evaluating a service member with multiple concussions or second impact syndrome is a rarity. This shift has been due to advances in anti-IED technology such as the Warlock, shift in military tactics, the replacement of soft sided high mobility multi-wheeled vehicle (HMMWV) to up-armored HMMWVs and the current MRAP (mine resistant ambush protected) armored vehicle. Neuropsychologists at Fort Hood are currently being asked to

determine whether a service member with PTSD has any long standing cognitive issues due to concussive blast-based concussion received months to years after the concussive blast. A typical soldier referred to the TBI Clinic is identified at PDHRA or is going through a medical evaluation board (MEB) and is not typically self-referred.

Military neuropsychologists have to deal with factors specific to military culture, not typical in the civilian sector, such as duties or training cycle that can potentially impact neuropsychological functioning. Staff duty is an example of a duty that can impact testing results. This duty requires a soldier to stay awake for 24 hours and leads to sleep deprivation. Gunnery and night firing are examples of training events that can impact test results because they lend the soldier to experience sleep deprivation.

Topic Outline

First, an overview of research on neuropsychological functioning in people with PTSD is presented. Next, an overview of the research on neuropsychological functioning in MTBI is discussed. Then, an overview of the research on overlapping symptom between PTSD and MTBI will be presented. To conclude, a study is proposed to differentiate neuropsychological functioning in MTBI and acute PTSD.

Posttraumatic Stress Disorder

PTSD is amongst the most controversial diagnoses included in the *DSM-IV-TR* (Spitzer, First, & Wakefield, 2007; Gold, Marx, Soler-Baillo, & Sloan, 2005; Boals and Schuettler, 2008). The only other diagnosis that generates as much conjecture is dissociative identity disorder. The controversy with PTSD revolves around the boundaries of the disorder, diagnostic criteria, central assumptions, clinical utility, and prevalence in various populations (Spitzer, First, & Wakefield, 2007). Gold et al. (2005) and Boals and Schuettler (2008) arrived at conflicting

results when looking at the importance of criterion A1 and A2 in defining PTSD. Gold et al. (2005) reported that higher levels of PTSD symptoms were associated with non-traumatic events than traumatic events when scoring results were based on classification by coders. On the other hand, Boals and Schuettler (2008) found that PTSD symptoms were more associated with traumatic events than non-traumatic events when scoring results were based on participants' ratings. Further Boals and Schuettler (2008) reported that criterion A1 had a minimal relation to PTSD symptoms when A2 was considered. These two conflicting studies bring to light the validity of the role of criterion A1 and A2 in diagnosing PTSD. PTSD is an anxiety disorder with four major criteria: 1) exposure to or the witnessing of a traumatic event in which a person experiences an intense fear, 2) symptoms of reexperiencing, 3) avoidance of thoughts, feelings, or reminders of the trauma, and 4) increased arousal as denoted by hypervigilance, irritability or sleep disturbances. These symptoms must cause clinically significant impairment for at least one month. Beyond one month, PTSD can be labeled as acute if the symptoms persist for less than three months. If symptoms persist beyond three months the diagnosis is considered chronic. A final classification for PTSD is delayed onset. This occurs when the symptoms appear at least six months after the traumatic event. The present study involves soldiers diagnosed as having acute PTSD. This is to avoid potential confounds associated with psychopharmacological interventions and prolonged neuroendocrine responses to stress.

Research on neuropsychological functioning in service members with PTSD has shown deficits in executive functioning, processing speed, attention and attentional shifting, learning, and memory (Gil et al., 1990; Leskin & White, 2007; Samuelson et al., 2006). Factors to consider when determining neuropsychological functioning in people with PTSD are whether deficits are due to physical injury from combat, trauma exposure, PTSD symptomatology,

premorbid IQ, neuroendocrine functioning and associated neuroanatomy changes, and history of substance abuse. Koenen et al. (2002) in a Vietnam era study, provided common risk factors for PTSD that can be considered during the differential diagnosis process and when interpreting results of neuropsychological testing. Common risk factors were earlier age at first trauma, exposure to multiple traumas, paternal depression, less than high school education at entry into the military, service in Southeast Asia, and preexisting conduct disorder, panic disorder or generalized anxiety disorder, and major depression. Although the presence of these historical risk factors does not contraindicate MTBI, they still provide additional information regarding the possible original posttraumatic symptoms.

A physical injury due to combat has been shown to be a risk factor for PTSD. Koren, Norman, Cohen, Berman, and Klein (2005) compared combat injured soldiers and soldiers who took part in combat situations but were not injured to determine if injury was a risk factor for PTSD. About 17 percent of combat injured soldiers met diagnostic criteria for PTSD, whereas about three percent of non-injured soldier met criteria for PTSD. This study also showed that injured soldiers had higher scores on clinical scales for depression, anxiety, and dissociation than those not injured. Of note, neither the severity of injury nor severity of the trauma related to the presence of PTSD.

Neuroimaging (structural and functional) has become an important method for investigating neurobiological etiology of PTSD because the images can reveal dysfunction (hypo- or hyper-activity) in different brain regions associated with PTSD symptomatology. A magnetic resonance imaging (MRI) study by Andreasen et al. (1993) showed that WAIS-R full scale IQ and verbal IQ were significantly correlated with hippocampal volume. Morey, Petty, Cooper, LaBar, and McCarthy (2008) conducted research using functional MRI to investigate the

relation of executive and emotion processing regions of the brain with PTSD symptoms. They showed a link between behavioral symptoms of PTSD and neuroanatomy features. Furthermore, it showed that executive and emotional processing systems of the brain are affected differently by PTSD symptoms. Functional MRI scans from the Morey, Petty, Cooper, LaBar, and McCarthy (2008) demonstrated that activation associated with the presented emotional stimuli was positively correlated with level of PTSD symptoms in the frontolimbic regions (ventromedial prefrontal cortex, inferior frontal gyrus, and ventral anterior cingulated gyrus). Additionally, it showed that activation associated with the executive task was negatively correlated with PTSD symptoms in the dorsal executive network (middle frontal gyrus, dorsal anterior cingulated gyrus, and inferior parietal lobule). A fMRI study with a symptom provocation paradigm involving script-driven traumatic imagery show that PTSD participants showed decreased activation of the thalamus, anterior cingulated gyrus (brodmann's area 32), and the medial frontal gyrus (Brodmann's area 10/11) than did the comparison group (Lanius et al, 2001).

Research with animals and stress has provided a model for hypothesizing changes in humans. Prolonged exposure to stress has been shown to produce structural changes to the amygdala, hippocampus, and prefrontal cortex. The amygdala and hippocampus are structures in the limbic system key to understanding PTSD symptomology. The amygdala is involved in the regulation and expression of emotion and prolonged exposure to stress causes dendrite hypertrophy. The hippocampus is involved in forming, storing, and processing memory. Prolonged stress has been shown to alter the pyramidal cells of CA3 of the hippocampus. Discussion of the hippocampus volume as a risk factor for PTSD is controversial because research has suggested that some decrease in volume maybe due to premorbid factors whereas

other studies suggest it is a by-product of prolonged traumatic exposure (Gilbertson et al., 2002; Bremner et al., 1995; Gurvits et al., 1996). Bremner et al. (1995), in a MRI study, showed that service members with combat-related PTSD had an eight percent smaller right hippocampal volume relative to comparison subjects. Additionally, decreased hippocampal volume related to deficits in short-term verbal memory, as measured with the Wechsler Memory Scale. Finally, PTSD has been shown to be related to abnormalities in the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system. In a study by Yehuda (2001) it was discovered that higher corticotrophin releasing factor levels, lower catecholamine levels in the central nervous system, and variability in cortisol release are all neuroendocrine factors that may account for cognitive changes in PTSD.

Mild Traumatic Brain Injury

An estimated one million people in the United States sustain a traumatic brain injury and about 80 percent of these injuries are classified as mild” (Belanger, Vanderploeg, Curtiss, & Warden, 2007, p.5). The accuracy of this statistic is made problematic because there is not a single set of diagnostic criteria for MTBI, however there are some overlapping criteria amongst major organizations that research this topic: American Congress of Rehabilitation Medicine (ACRM), Center for Disease Control (CDC), and World Health Organization (WHO). A diagnosis of MTBI is typically defined using three criteria: 1) Glasgow Coma Scale (GCS) score, 2) quantity of loss of consciousness (LOC), and 3) quantity of retrograde amnesia. Further challenges are that there is rapid resolution of acute signs and symptoms, lack of guidelines for assessing specific diagnostic criteria, and typically an absence of objective evidence of injury on structural neuroimaging (Ruff et al, 2009).

The GCS is used for clinical decision-making during triage in prehospital settings or emergency departments because it is a means of quantifying level of consciousness in patients with traumatic brain injury (Stuke, Diaz-Arrastia, Gentilello, & Shafi, 2007). The GCS was originally designed to quantify level of consciousness for moderate to severe TBI and not mild. GCS has limited utility as a classification of MTBI due to ceiling effect and limited sensitivity (McCrae, 2008). A quantified level of consciousness is determined by scoring each of the three response areas; eye opening, verbal, and motor. Based on the cumulative GCS score the head injury is classified into three categories: severe is a score of 8 or less, moderate is a score of 9 to 12, and mild is a score of 13 to 15. In addition to providing a point of reference to level of consciousness it has been used to direct diagnostic and therapeutic decisions and as a predictor of outcome after traumatic brain injury. Research on whether the GCS is an effective outcome predictor is mixed. Balestreri et al. (2004) monitored participants over a 10 year period and found mixed results between the GCS and Glasgow Outcome Score. Results showed a significant correlation between the GCS and Glasgow Outcome Score during the first five year period and no correlations for the next five years. This shows that the GCS lost its predictive value after five years.

Ruff et al. (2009) suggest that GCS is not an effective tool for categorizing a TBI based on timeline of when the GCS is administered. This timeline is important because receiving a GCS score after 30 minutes is part of the CDC, ACRM, and WHO definition of MTBI. Further, the WHO recognizes the limited practicality of providing a GCS score 30 minutes post-injury (Holm, Cassidy, Carroll, & Borg, 2005). The following example provided by Ruff et al. (2009) clarifies the use of the GCS on a timeline and how it can be misleading when categorizing TBI:

This patient has a witnessed LOC of approximately 3-4min during which time he was unresponsive. Due to his unresponsiveness, his GCS would have been 10 or less had he

been evaluated. An ambulance crew arrived at approximately 10 min post-injury and assessed him with a GCS of 10. They reassessed him 20 min post-injury with GCS of 14. The patient's GCS was 14 at the hospital at 6 hr post-injury and a GCS of 15 was documented 12 hr post-injury. It would be a mistake in this case to assume that the patient had a moderate TBI based on the initial low GCS score of 10, obtained approximately 10 min post-injury, because the GCS was within the mild range (i.e., 13-15) within the first 30 min post-injury. Therefore, a mild TBI should be diagnosed (p. 8).

Beyond the GCS is Ommaya and Gennarelli's (1974) classification system that provides grades of concussions severity that can be used to operationally define MTBI and aid in differentiating mild and moderate TBI. This classification system is an improvement over the GCS because of its usage of multiple indicators from confusion to amnesia and unconsciousness which is intended to improve the sensitivity in the detection of MTBI (McCrae, 2008). Ommaya and Gennarelli (1974) define cerebral concussion as a graded set of clinical syndromes following head injury wherein increasing severity of disturbance in level and content of consciousness is caused by mechanically induced strains affecting the brain in a centripetal sequence of disruptive effect on brain functioning and structure. Based on the work of Ommaya and Gennarelli (1974), the American Academy of Neurology (AAN) developed a scale to define and grade the severity of MTBI. See Table 5 for AAN definition of a concussion and how each grade of concussion is defined.

A study of the definitions of MTBI by the WHO Collaborative Center Task Force on Mild Traumatic Brain Injury identified discrepancies in definitions such as whether a GCS scores was incorporated and duration of LOC. Of those definitions that included a GCS score the range was 13-15, 14-15 or just 15 (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004). Due to the variance in what defines MTBI, this study used criteria outlined by the ACRM because military clinicians use this criterion to diagnose MTBI. See Table 5 to compare what criteria the WHO, CDC, ACRM, and AAN use to define MTBI (McCrae et al., 2008). The ACRM defines a

person with a mild traumatic brain injury as a ~~person~~ who has had a traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

1. any period of loss of consciousness, not to exceed 30 minutes.
2. any loss of memory for events immediately before or after the accident with posttraumatic amnesia not to exceed 24 hours.
3. any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, confused), with an initial GCS (after 30minutes) score of 13-15".(McCrea, et al., 2008).

Another point of contention in defining MTBI is the timeline for recovery; specifically, when symptoms and cognitive impairments resolve after the trauma. The timeline is broken down into two phases: acute-recovery phase and post-acute recovery phase. It is presumed that most symptoms and measureable cognitive deficits occur during acute recovery phase. Research has shown that ~~neurophysiologic~~ effects of MTBI follow a course of recovery consistent with the natural course of symptom and cognitive recovery as the brain returns to a normal physiologic state within days to weeks of injury" (McCrea, 2008, p.139). In the acute recovery phase, a majority of cases with measureable cognitive deficits resolve over a period of days to weeks. The DSM-IV-TR and ICD-10 concur that symptoms and cognitive impairments become clinically significant if they persist three month or more and refer to this condition as postconcussional disorder.

Belanger and Vanderploeg (2008) conducted a meta-analysis to assess the effect of concussion on indices of cognitive functioning when administered immediately post-injury (within 24 hours) and seven days post-injury and concluded that the acute effect of MTBI were greatest for delayed memory, memory acquisition, and global cognitive functioning. Additionally, delayed memory remained impaired at seven days. A meta-analysis by Binder, Rohling, and Larrabee (1997), which only included those who were not symptomatic and three months or more post injury, concluded that there was a relationship between MTBI and cognitive

deficits and that the cognitive domain of attention had the largest effect. Of note, in this study the severity of injury accounted for more variance than did specific neuropsychological domain.

However, a meta-analysis by Frenchman, Fox, and Mayberry (2005), which included acute and post-acute recovery phase MTBI participants, concluded that processing speed indices had the largest effect and time since injury was found to be a significant moderator variable. Beyond the post-acute recovery phase, a meta-analysis designed to assess the long-term neuropsychological impact of multiple MTBI revealed that this population had poorer performance on measures of delayed memory and executive functioning, but the overall effect of multiple MTBI on neuropsychological functioning was not significant (Belanger, Spiegel, & Vanderploeg, 2010).

An IED, an iconic part of the Global War on Terror, typically produces a blast injury. The explosion produced by an IED generates a wave of over-pressurized air, which can produce brain injury. As of now, it is not known whether the effects of blast injury on the brain are related to mechanical effects of the over-pressurized wave, the inertial effects of alternating high and low pressure, or some other mechanism. Furthermore, it is not known whether the diagnosis and assessment of TBI severity associated with a blast injury differs from the blunt force trauma associated with other injuries, such as motor vehicle accident. A fluid percussion model of brain injury, similar to an IED related concussive blast, has been studied in animals and used to hypothesize changes in people with MTBI. “Human blast injury studies in organs other than the brain have shown that at least two atmosphere percussion waves in the fluid media of the brain can produce MTBI findings similar to findings in animal studies” (Trudeau et al., 1998, p. 309). Over-pressure waves have been associated with producing diffuse axonal injury (DAI) via rapid acceleration and deceleration (coup-countercoup). DAI is associated with the shearing or damaging of axons that project from the brain stem. If the coup-countercoup action is severe

enough it can cause a loss of consciousness. When a LOC is experienced a soldier can further harm the brain by making significant contact with a physical object such as a weapon, vehicle structure, or the ground as he or she falls.

Arciniegas and Beresford (2001) studied brain-behavior relation and regional cortical vulnerability to TBI. They determined that dorsolateral prefrontal cortex, orbitofrontal cortex, anterior temporal cortex, amygdala, hippocampal-entorhinal complex, and ventral brainstem are the areas most commonly affected by a TBI. The dorsolateral prefrontal cortex is the primary center for executive functioning, which includes sustained and complex attention, memory retrieval, abstraction, judgment, insight, and problem solving. The orbitofrontal cortex is responsible for managing emotional and social responding. The anterior temporal cortex is responsible for memory retrieval and sensory-limbic integration. The amygdala is responsible for emotional learning and conditioning (fear and anxiety). The hippocampal-entorhinal complex is responsible for declarative (conscious) memory. The ventral portion of the brainstem is responsible for arousal, ascending activation of the diencephalic, subcortical, and cortical structures. The ascending cholinergic system of the diencephalic is key to understanding sleep-waking cycle, emotional arousal, and the initiation of defensive and alarm behaviors (Brudzynski, Kadishevitz, & Fu, 1998).

Neurocognitive and physical sequelae (post-concussive symptoms) are associated with MTBI, either persistent or temporary. Post-concussive symptoms usually resolve on their own in one to three months (Thompson, Scott, & Dubinsky, 2008). Arciniegas, Anderson, Topkoff, and McAllister (2005) suggest considering psychological and social stressors to understand the persistence of symptoms. Being in a high stress environment can prolong the recovery period due to the impact stress hormones have on the immune response. Ruff, Levin, and Marshall

(1986) suggested that post-concussional symptoms should be investigated on three dimensions; somatic, cognitive, and affective. Somatic symptoms include headache, dizziness, vision difficulty, and deficits in balance and motor functioning. Neurocognitive sequelae consist of attention/concentration, memory, cognitive processing speed, simple and complex reaction time (Bleiberg et al., 2004; Arciniegas, Anderson, Topkoff, & McAllister, 2005). Typical affective symptoms include anxiety, depression, irritability, and mood swings. Kennedy et al.(2007) showed that post concussive symptoms occur immediately after brain injury and can include cognitive deficits in memory, attention, and concentration; physical or somatic complaints of fatigue, disordered sleep, dizziness, and headache; and affective complaints or irritability, anxiety, and depression. Sequelae associated with MTBI can have an operational impact on military units that should be considered by military clinicians when deciding when a soldier should be returned to duty. Neurocognitive sequelae can interfere with a soldier's judgment which is important when determining the application of rules of engagement. DVBIC suggests further operational impacts such as alterations in attention/concentration, maneuverability/flexibility/judgment, and impulse control. In operational terms these deficits may adversely affect driving, handling firearms, establishing situational awareness, and may result in adverse outcomes such as friendly fire incidents.

Overlapping Symptoms

Service members are increasingly being exposed to concussive blasts related to IEDs. In and of itself, being exposed to an IED blast meets the DSM-IV criterion A1 and can potentially cause MTBI due to concussive blast. Research is mixed on whether PTSD occurs in conjunction with MTBI (Glaesser, Neuner, Lutgehetmann, Schmidt & Elbert, 2004; Kennedy, et al., 2007; King, 1997; Elbert & Schauer, 2002; Sbordone & Liter, 1995; Mayou, Bryant, & Duthie, 1993,

and Schwarzbald et al., 2008). These mixed research findings might be due to the overlap in diagnostic criteria and its interpretation. A lack of agreement in the research community regarding what PTSD and MTBI symptoms overlap further complicates diagnosis. DVBIC considers depression, anxiety, and attention difficulties as overlapping symptoms. Depression, anxiety, and sleep are non-neuropsychological overlapping symptoms of PCS and PTSD that the ICD-10 and DSM-IV-TR agree upon. The Veterans administration considers concentration difficulty, sleep difficulty, irritability, and social withdrawal as overlapping symptoms. Further complicating the differential diagnosis process is the overlapping symptoms between anxiety and major depression, which are common behavioral symptoms of MTBI and PTSD. These overlapping symptoms consist of problems with sleep, concentration, and fatigue as well as psychomotor/arousal symptoms (Stahl, 2008). Other research suggests that irritability, attentional dysfunction, difficulty concentrating, amnesia, decreased cognitive processing, and sleep disturbances as overlapping (Glaesser, Neuner, Lutgehetman, Schmidt, & Elbert, 2004; King, 1997; Trudeau et al., 1998). Kennedy et al. (2007) suggests that arriving at a differential diagnosis of PTSD requires understanding of etiology of the different symptoms seen in MTBI. To do this the researchers suggested conducting a thorough biopsychosocial assessment to account for the presenting neurological and psychological factors, which may suggest specific underlying mechanisms that can help in early detection, diagnosis, and treatment. Another factor confounding the diagnostic and research process is the necessity in the PTSD diagnostic criteria of experiencing intense affect associated with a traumatic event (A2). Experiencing intense affect is not universally accepted as demonstrated by not being a part of the criteria for PTSD in the ICD-10. Brewin et al. (1999b) found that intense emotions appear to be the norm, but that a

small portion of trauma victims did not report intense fear, helplessness, or horror but subsequently developed PTSD.

King (1997) proposed three factors that can lead to confusion in diagnosing MTBI versus PTSD. The first factor is LOC, which is thought to prevent some people from developing PTSD. The rationale here is that if you cannot remember the traumatic event you cannot be psychologically traumatized by it. LOC is the most controversial of the overlapping symptoms because if there is no memory whatsoever of the traumatic event, than this seems to preclude the cardinal PTSD feature of re-experiencing. Second is the existence of post-concussion symptoms, which appears to overlap with some PTSD symptoms: poor concentration, depression, anxiety, sleeps disturbance, and irritability. The third factor is the interpretation of the amnesia for the event. A lack of memory of a traumatic event in MTBI could be due to organic-based amnesia, which is a sign of head injury. The lack of memory associated with PTSD could be due to psychologically-induced amnesia, which can be interpreted as the PTSD symptom of avoidance. Additionally, King (1997) suggested three mechanisms in which PTSD and TBI might co-occur. The first condition is met when the period of anterograde and retrograde amnesia are small or non-existent. The second condition is met when non-declarative memory systems for the traumatic event are in operation. The third condition is a phenomenon referred to as “islands” of memory. Islands of memory or recollections of events are sometimes part of anterograde amnesia in MTBI and occur outside of continuous memory (King, 1997). Two alternate explanations for this phenomenon are that “false memory” (based on what the patient is told after the event) being inserted into the period of anterograde amnesia and an isolated memory being retained while recall of the surrounding events is lost due to psychogenic processes” (King,

1997, p. 83). However, King (1997) did not offer an explanation for those who may be traumatized by the context of the injury.

The coexistence of PTSD and TBI is considered to be a paradox and has fostered much research to clarify the clinical distinction. Harvey, Brewin, Jones and Kopelman (2003) proposed three theoretical arguments against the dual diagnosis with resolutions for each argument. Resolutions are based on ambiguity of PTSD criteria and accepting that TBI patients do experience similar symptoms to those diagnosed with PTSD, but that there are differences in symptoms content. The first argument is the lack of affect, which is associated with the DSM-IV-TR PTSD criteria A1 and A2. The crux of this argument is whether a person who suffers PTA or disturbed consciousness could have experienced an intense emotion to warrant a diagnosis. The first resolution to this argument postulates that a person with a TBI does not experience intense affect and it does not hinder the development of PTSD or ASD. This resolution is based on the questionable necessity of whether experiencing intense emotions is crucial to the development of ASD or PTSD. This is highlighted by the lack of this criterion in the ICD-10 and research showing that intense emotion is not observed in response to every traumatic event (Brewin, Andrews, & Rose, 1999b). The second resolution suggests that TBI victims do experience intense affect prior to or subsequent to a traumatic impact. Three rationales were provided for how a person with a TBI could experience intense affect: islands of memory, affect associated with the period immediately prior or subsequent to a traumatic impact, and information encountered after the trauma may lead to intense affect.

The second argument revolves around whether a person who experiences an altered state of consciousness (PTA or LOC) can re-experience a traumatic event. The rationale is that because there is an absence of memory of the traumatic event, than no intrusive re-experiencing

of the trauma is possible, which is necessary for a diagnosis of PTSD. The first resolution to this argument is that the lack of re-experiencing a trauma in those who have suffered a TBI is not a barrier to developing PTSD. The rationale given was that symptoms of re-experiencing, as outlined by the DSM-IV-TR criterion B, do not depend on having conscious memory of the traumatic event and therefore, a person can still experience these symptoms in the absence of memory of the traumatic event. The second resolution postulates that people that have experienced a TBI do re-experience the traumatic event. Four explanations were provided, of which two were previously discussed; islands of memories and memory of events prior and subsequent to the PTA, LOC or altered conscious state. The other two explanations involve miss representations of memories. This explanation is based on research that suggests that memories are malleable (Hyman & Loftus, 1998; Reynolds & Brewin, 1998).

The third argument revolves around whether there is a lack of avoidance related to the traumatic event; criterion C. The rationale for this argument is that if a person is amnesic for the event then there is no desire to avoid; therefore, a dual diagnosis is not possible. The first resolution for this argument postulates that people who have experienced a TBI and do not avoid reminders of the trauma should not be precluded from being diagnosed with PTSD, because they could experience emotional numbness despite showing minimal evidence of cognitive or behavioral avoidance. The second resolution suggests that avoidance symptoms can occur independently of memory for the event and may occur in the uncoupling between conscious memories of the event and fear-conditioned behavior.

The National Academy of Neuropsychology (NAN) published an educational paper in 2009 that could be used to help clarify overlapping symptoms. This paper provides recommendations for assessing LOC, PTA, and confusion and disorientation because the

organization recognized that post-traumatic confusion or amnesia may impact a person's capacity to accurately self-report MTBI diagnostic criteria. Thus, NAN intended to address the challenge faced by neuropsychologists who have to evaluate and diagnose MTBI weeks to months after an injury. Of note, it is not uncommon for neuropsychologists at Fort Hood to be asked to determine whether there is a neuropsychological deficit years after injury; due to the current operational tempo in Iraq and Afghanistan, it is very rare that these neuropsychologists see a soldier during the acute recovery phase. Most soldiers come to the Fort Hood TBI Clinic with a dual diagnosis, further complicating the issue of diagnosing a cognitive deficit related to MTBI.

To assess LOC, NAN recommends conducting collateral interviews with observers of the incident to reduce the likelihood of making two fundamental mistakes: 1) patients may assume they were unconscious when they were not, and 2) patients sometimes deny experiencing LOC when they did (Ruff et al., 2009). NAN recommended questions for the clinician to ask to determine the absence or presence of LOC: ~~“Has anyone told you that you were unconscious?”~~ and ~~“Who saw you unconscious?”~~ (Ruff et al., 2009, p. 6).

To assess retrograde amnesia and PTA, NAN has determined that it is essential to assess what the patient remembers versus what he or she has been told or has surmised (Ruff et al., 2009). In the civilian sector this means that these retrograde and anterograde amnesia gaps could be filled in by what the patient learned from talking to eyewitnesses or reading the police report. In the military these gaps can be filled by fellow soldiers well after the incident or by combat medics who provide an abridged version of the story from firsthand account or from the person who triaged the soldier at the point of injury. To assess anterograde amnesia and PTA, it is recommended that the clinician ask the client: ~~“What is the first event you remember after the~~

injury?” with a follow up question, “Can you describe in as much detail as possible what you can remember immediately after your injury?” (Ruff et al., 2009, p. 6). It was further recommended to assess whether the gap in memory may be due to medications or enduring severe pain. If retrograde amnesia occurs, there should be a gap between what the patient recalls of the last events before the accident and the impact. For PTA to occur, there should be a gap between the impact and what the patient first recalls after the injury. It is also recommended that the clinician assess for psychogenic amnesia because people with PTSD can have partial amnesia (Ruff et al., 2009). A study by Anderson and Levy (2009) has shown that people can control unwanted memories by stopping memory retrieval, which supports the existence of an active forgetting process and establishes a neurocognitive model for inquiry into motivated forgetting.

To assess confusion and disorientation, NAN recommends determining whether such manifestations are due to psychological or physiological origins. Psychological factors such as experiencing shock/being overwhelmed can produce confusion. NAN provides several questions to help in determining whether the state of confusion was caused by psychological or physiological factors: “Were you scared after the accident?”, “Did you feel stressed worked up, or overwhelmed?”, “Was your heart beating rapidly?”, and “Did you have an anxiety attack?” (Ruff et al., 2009, p. 7). Accurately identifying disorientation and confusion can be enhanced if the clinician carefully establishes a timeline for the experience. The confusion and disorientation should not follow the patient’s conscious awareness of what took place, rather must be directly linked to the presumed cause of the trauma to the brain. To understand the timeline, the NAN educational paper (2009) provides an example:

“If an individual fully recalls the accident and then describes a feeling of being dazed, confused, or even disoriented after he or she realizes that extent of the

bodily injuries or detrimental consequences to others, then this is likely not due to the mild TBI per se” (Ruff et al., 2009, p. 7).

These recommendations may shed some light onto King’s (1997) three factors that could lead to confusion in diagnosing MTBI versus PTSD.

Glaesser, Neuner, Lutgehetmann, Schmidt, and Elbert (2004) explored the role of LOC in the development of PTSD. This study was conducted using three different periods of LOC; more than 12 hours, less than one hour, and no LOC at all. It was determined that PTSD and TBI can still overlap, even for those who experienced an extended period of LOC. Further findings from this study showed that patients in the group without a LOC were more likely to be diagnosed with PTSD, have more intrusive memories, re-experiencing symptoms, and psychological distress and physiological reactivity to reminders of the traumatic event.

Quantitative electroencephalogram (qEEG) has been used in research to examine overlapping symptoms. qEEG measures electrical patterns (brainwaves) on the surface of the scalp using electrodes. A computer uses an algorithm to compare brainwave patterns from an afflicted person to a standardized sample of normal brainwave patterns. qEEG is not an independent measurement instrument for diagnosing a brain injury. Instead, it is typically recommended to serve as a point of confirmation in conjunction with standardized neuropsychological testing. Research has shown that qEEG is effective in identifying and measuring brain damage in people with MTBI (Nuwer, Hovda, Schrader, & Vespa, 2005).

Trudeau et al. (1998) examined qEEG in veterans with chronic PTSD that did and did not have a history of blast concussion. To compare participants he used the Thatcher’s normative database. Results of the study showed a statistically significant difference in Thatcher discriminant scores between those with and without blast concussion on attention. Attentional difficulties were more

prevalent in the blast group than in the no blast group. Such findings may be used to help differentiate MTBI and PTSD.

Neuroimaging (structural and functional) has become an important method for investigating neurobiological etiology of MTBI because the images can reveal dysfunction (hypo- or hyper-activity) in different brain regions associated with MTBI symptomatology. Structural imaging has been less predictive of outcome than functional imaging. It is common practice for emergency room physicians to order a CT scans to rule-out the need for neurosurgical intervention. This type of scan is not as effective as fMRI or SPECT in detecting underlying abnormalities associated with MTBI because abnormalities occur at the micro rather than macroscopic level (Flanagan, Cantor, & Ashman, 2008; McCrae, 2008). Further classification of MTBI can result from neuroimaging. If there is positive finding then the MTBI is classified as complicated and when there is negative finding the MTBI is referred to as uncomplicated. Research has shown those with positive neuroimaging take longer to recover than those with negative imaging (Iverson et al., 2006).

The Global War on Terrorism has provided researchers the opportunity to study neurophysiological and brain-behavior changes due to concussive blast. Prior to the Global War on Terrorism, theories about the affects of a concussive blast on human neuroanatomy and psychology were derived based largely on animal studies, blast injuries to other parts of the human anatomy, and from experience with blunt force trauma such as in a motor vehicle accident. This war has brought to the forefront the controversy of whether PTSD and MTBI co-occur. Overlapping symptoms between MTBI and PTSD have made it difficult to differentiate between them. Further complicating the process is that research evidence is mixed on whether they co-occur based on LOC and other factors.

Study Objectives

The present study investigates neuropsychological functioning differences between MTBI and PTSD in order to provide mental health clinicians a tool to assist in differentiating between MTBI and PTSD. Specifically, indicators of neuropsychological functioning were investigated.

It was hypothesized that MTBI participants will score lower on variables of attention than participants in the PTSD group and control group. The second hypothesis for this study was that PTSD participants will score lower on variables of memory than MTBI participants. The third hypothesis for this study is that PTSD participants will score lower on variables of memory than controls. The final hypothesis was that there will not be a statistically significant difference between groups on measures of psychomotor speed.

METHODS

Participants

According to the September 2008 statistics report kept by defense veterans brain injury center (DVBIC), the majority of service members receiving treatment for traumatic brain injury (TBI) are in the early 20's, white-males, junior enlisted, and in a combat arms job. The participant population will primarily be Caucasian males with combat experience, with smaller numbers of females and ethnic minorities. Participants will reside in a combat arms unit and serve in combat arms military occupation specialty (MOS). The primary combat arms MOSs in this study are armor, cavalry scout, and infantry. The majority of participants were in their 20s and 30s, hold at least a high school degree, and in good physical health. Control group participants were recruited from various combat arms units on Fort Hood. The clinical participants were recruited from service members who seek behavioral health services at Carl R. Darnall Army Medical Center (CRDAMC) at Fort Hood.

A quasi-experimental design was employed, with participants placed into groups based on diagnosis. Diagnostic groups for this study consisted of MTBI, acute PTSD, and controls who have experienced a combat situation involving a concussive blast and no diagnosis of PTSD or MTBI. The goal was to obtain at least 30 participants per group, for a total of 90 participants for the study. A floating ceiling of 30 was established based on experience with this population and testing location. From previous experience it has been determined that accumulating 30 MTBI participants will be a challenge based on the timeframe for this study and the deployment schedule for the combat Units on Fort Hood. It is projected that control and PTSD groups will reach 30 participants faster than MTBI group. Once MTBI enrollment has caught up with the 30 participant floating ceiling there will be an increase based on a one-for-one basis. For example,

when the 31st MTBI participant has been tested the 31st PTSD and control participant will be tested. The equality in group membership is being done for statically comparison. Limitations of generalizing research using such small sample sizes will be highlighted in the discussion section of the final report. For example, small sample size may not allow for sufficient power to detect group differences among variables and may lead to positive findings that may not be an accurate reflection of the population.

As described above, the present study followed McCrea et al. (2001) and ACRM criteria for traumatically induced disruption of brain function, as manifested by at least one of the following:

1. any period of loss of consciousness, not to exceed 30 minutes.
2. any loss of memory for events immediately before or after the accident with posttraumatic amnesia not to exceed 24 hours.
3. any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, confused), with an initial GCS (after 30 minutes) score of 13-15.

MTBI participants for this study consisted of those that are three month or more post-injury since the timeline for recovery is controversial and this is when a majority of soldiers are evaluated by neuropsychologists at Fort Hood. A diagnosis of MTBI (Cognitive Disorder NOS) will be given to a soldier by either a neuropsychologist or neurologist. Exclusion criteria for the MTBI group include: GCS <13, under the age of 18, those who received a MTBI by means other than concussive blast (e.g. motor vehicle accident), penetrating head injury, documented hearing impairment, deficits in vision that cannot be corrected by glasses, pre-trauma history of neurological disease, systemic disease (ex. AIDS, diabetes, hypertension), psychiatric disorder (ex. ADHD), substance abuse with AUDIT score of 20 or above on pre or post-trauma and/or DAST-10 score of 6 or above or pre-existing alcohol or drug abuse, developmental disorder,

sleep apnea, a prior history of concussion with LOC or hospitalization for previous head injury, positive neuroimaging (complicated MTBI), and sniper qualified.

PTSD participants were diagnosed according to the DSM-IV TR. This group only includes soldiers specified as Acute. Exclusion for this group includes: under the age of 18, history of TBI with LOC or hospitalization for previous head injury, documented hearing impairment, deficits in vision that cannot be corrected by glasses, pre-trauma history of neurological disease, systemic disease, co-morbid psychiatric disorder, substance abuse with AUDIT score of 20 or above on pre or post-trauma and/or DAST-10 score of 6 or above or pre-existing alcohol or drug abuse, developmental disorder, and sniper qualified.

Control group participants were recruited from various combat arms units on Fort Hood that have been exposed to an improvised explosive device (IED) and have not been diagnosed with MTBI or PTSD. The exclusion criterion for this group is consistent with the criteria outlined for MTBI and PTSD.

Setting and Apparatus

The present study was conducted in the TBI Clinic of the CRDAMC on Fort Hood, Texas. The TBI Clinic is a separate building from the hospital that provides outpatient neuropsychological and consultation services to maintain the mental health of active-duty personnel. It is staffed with active-duty and civilian professional staff including psychiatrists, psychiatric nurse practitioners, neuropsychologists, social workers, and psychology technicians.

Instruments

A questionnaire devised by the author was used to collect demographic data, information related to traumatic experience, and questions related to psychological constructs related to MTBI sequelae and PTSD symptomology. See Appendix A for questionnaire. Further, two

computer administered and scored neuropsychological instruments were utilized to measure aspects of attention, memory, and psychomotor speed. The usage of computer based instruments has pros and cons. A pro for using such tests is that it greatly decreases the amount of time required for testing, scoring, and interpreting. Both tests take about one hour to administer and score, whereas comparable traditional individually-administered paper-based neuropsychological tests could take as long as two days. This relates to a service member missing less work and therefore a unit can potentially be more productive. One con for these tests is that it limits the clinician's ability to observe verbal and non-verbal manifestations that could be important to interpreting test results. A second con is that test instructions assume that the examinee has a basic understanding of computer usage and terminology. For example, the instruction to "enter" your answer could be replaced with "type" your answer to facilitate understanding in those with minimal exposure to computers. A final downside to these particular tests is that they often do not allow the examinee to go back and change an answer.

The Automated Neuropsychological Assessment Metrics (ANAM) is a computer administered and scored neuropsychological screener originally developed for the Department of Defense and designed for repeated administrations. There are multiple versions of the ANAM that are employed by major federal agencies such as the Department of Defense, NASA, and the FAA. The development and management of the ANAM now falls under the responsibility of the center for the study of human operator performance at the University of Oklahoma. This study used the ANAM4 TBI Military Battery, which is designed to aid in the assessment of general cognitive function following a head injury. It is normed on a stratified sample of over 5,000 military personnel and the military reference group data was provided by DVBIC at Fort Bragg (Vincent et al., 2008). Research has shown that construct validity for ANAM subtests correlate

well with traditional neuropsychological tests (Bleiberg et al., 2000; Kabat et al., 2001; Short, 2007). It has been shown to correctly classify brain injured patient with 91% accuracy (Levinson & Reeves, 1997). In a study using traditional paper-based neuropsychological tests and the ANAM, it was determined that ANAM was better at detecting MTBI (Bleiberg, Kane, Reeves, Garmoe, & Halpern, 2000). It provides measures of neurocognitive functioning including response speed, attention/concentration, immediate and delayed memory, spatial processing, and decision processing speed and efficiency (Reeves, Winter, Bleiberg, & Kane, 2007). Some of the subtests are based on traditional neuropsychological tests while some are similar to existing psychological tests. See Table 2 for a list of tests in the ANAM4 TBI Military.

The ANAM4 TBI Military battery first administers several self report questionnaires related to demographics, TBI, sleep, and mood. The first self report section asks an examinee to provide demographic information on his or her age, gender, ethnicity, medical diagnosis, and medications. The next self-report is the TBI questionnaire, which is designed to assess injury history and related symptomology. Then the examinee answers a self-report questionnaire related to sleep, which provides a measure of state and trait assessment of energy-fatigue level. Finally, the examinee fills out a self-report related to mood, which provides measures of state and trait mood. The examinee is presented a series of adjectives and is instructed to select the box/number (similar to Likert scale) that best represents his or her current state with respect to the presented adjective.

The first neuropsychological subtest on the ANAM is Simple Reaction Time, a measure of visuo-motor response speed. To measure reaction time an examinee is instructed to press a button as quickly as possible each time an asterisk appears on the computer screen. The second subtest is Code Substitution-Learning, a measure of visual search, sustained attention, and

encoding. This subtest is somewhat similar to the WAIS-III digit-symbol search subtest. On this test an examinee is provided a digit-symbol key and must compare a presented digit symbol pair to decide whether the presented pair is associated with a pair in the key. The third subtest is Procedural Reaction Time, which is a measure of reaction time and processing efficiency. On this test an examinee is presented a series of single digit numbers (2, 3, 4, and 5) and is instructed to press a specific key when the numbers two or three are presented and a different key when the numbers four or five are presented. The fourth subtest is Matching-to-Sample, which is a measure of spatial processing and visuospatial working memory. On this test an examinee is presented a four by four grid pattern in which eight cells are shaded. The design is taken away and the examinee is presented two designs and asked to pick which of these two designs is similar to the previously presented design. The fifth subtest is Mathematical Processing, which is a measure of basic mathematical skills, concentration, and working memory. An examinee is presented a math problem that has three single numbers that involves two mathematical operations. The examinee presses a key based on whether the answer to the problem is less than or greater than five. The next subtest is Code Substitution-Delayed (Recognition), which is a measure of long-term memory. This test is similar to the Code Substitution-Learning test except that the examinee is only shown a digit-symbol pair. The examinee is asked to determine whether the presented pair is associated with the key from the Code Substitution-Learning test presented earlier. The final subtest of the ANAM is the Simple Reaction Time (Repeated). The execution and objective of this test is the same as the Simple Reaction Time as was presented earlier in the test.

Results of the ANAM are broken down into three sections: summary performance indicator, performance at a glance, and performance detail. The summary performance indicator

section provides an overall assessment of the soldier's performance for the test. Results are qualitative described as average or above, below average, and clearly below. The performance at a glance section provides information on the soldier's performance on each domain and performance is qualitatively described in the same manner as in the summary performance indicator section. The performance detail section provides three variables per domain: Throughput, percent correct (% Correct), and mean reaction time (Mean RT). Each of these variables has the soldier's raw score with its accompanying percentile and standard score. Even though this information is provided, the C-Shop developed the ANAM data extraction and presentation tool (ADEPT) to convert scores for statistical analysis. ADEPT Throughput (TP) score, as used in the statistical analysis, is defined as the number of correct responses per unit of available time. The TP variable is most often used for analysis because it combines speed (mean RT) and accuracy (percent correct) into a single variable. However, for Simple Reaction Time non-normality will most likely still be a problem (C-SHOP; ANAM, 2008). Qualitative descriptors of a Throughput score are as follows: below the 2nd percentile is clearly below average; between the 2nd and 9th percentile is below average; above the 9th percentile is considered average (A. Vincent, personal communication, February 10, 2010).

The MicroCog is a computer administered and scored neuropsychological battery originally developed for the Department of Defense and designed for repeated administration. It is intended as a screening tool for use as part of a traditional paper-based neuropsychological examination. Development and management of the MicroCog now falls to PsychCorp. The MicroCog is normed on over 800 non-military adults (18–89 years of age) representative of the 1988 census data (Lopez, Summerall, & Ryan, 2002). Exclusion criteria for the MicroCog's standardized sample included a head injury with a LOC, on antidepressant/

antianxiety/antipsychotic or beta-blocker medication, and diagnosis of a mental disorder. Neither the developers nor PsychCorp have reported MicroCog findings on people with MTBI or PTSD to provide normative information for these groups. In a review of the journal articles on the search engine PsychInfo, MedLine, and PubMed it appears that MicroCog has primarily been used in substance abuse and biomedical research to measure neurocognitive changes. It is based on established psychological tests that could be adapted to a multiple-choice response format for use on a computer. There are two versions of the MicroCog available: standard form and short form. The present study will be using this standard form, which consists of 18 subtests and takes about one hour to complete. Subtests on the MicroCog are similar to certain subtests on the WAIS-R, WMS-R, and other traditional psychological tests. For example, the MicroCog subtests Numbers Forward and Numbers Reversed are similar to the WAIS-R Digit Span Forward and Digit Span Backward.

Nine interrelated cognitive domains are derived from the 18 subtests: attention/mental control, memory, spatial processing, reasoning/calculation, reaction time, information processing accuracy, information processing speed, cognitive functioning, and cognitive proficiency. Of these nine, five are considered to be neurocognitive index scores: attention/mental control, memory, spatial processing, reasoning/calculation, and reaction time. The other four are considered higher order indices: information processing speed, information processing accuracy, general cognitive functioning, and general cognitive proficiency. Standard scores are calculated for each index and they share a common metric for evaluating performance. A score of 100 is the average score for each age group and education level, with a standard deviation of fifteen. Qualitative descriptors are as follows: 69 and below (below average), 70-84 (low average), 85-114 (average), and 115 and above (above average).

The attention/mental control domain is a measure of attention and concentration. The first two subtests in this domain are similar to the WAIS-R Digit Span Forward and Digit Span Backwards, in which the examinee is presented on the screen a series of digits and is expected to type the numbers in forwards or backwards. The third subtest in this domain is the Alphabet subtest, which is similar to auditory trials. It is a continuous performance task in which a series of letters, ranging from A to O, are presented on the screen in a random order. The participant is expected to respond to the presented letters by typing them in alphabetical order. The last two subtests (Wordlist 1 & 2) are similar to Boston Incidental Verbal Learning Test, which involves the presentation of a list of categorized words. During Wordlist 1 an examinee is expected to choose words from a list of words that belong to a category. For example, the examinee is told the category is clothing and is expected to choose words that relate to clothing from a list of 16 words that are presented on the screen for one second each. Wordlist 2 consists of a list of 36 words, including the 16 words from Wordlist 1. When a word is presented the examinee must indicate whether the word was previously presented. The memory domain measures short-term and long-term memory. Memory is tested using two short stories similar to those in the logical memory subtest of the WMS-R. For short term memory the examinee is asked multiple-choice questions immediately after the story is removed from the screen. According to the manual, long-term memory of the story is tested approximately 20-30 minutes after a filled delay. The reasoning/calculation domain is a measure of reasoning skills. Reasoning is tested using three subtests: analogies, object matching, and math calculation. The use of analogies is a measure of inductive reasoning and its format is similar to the Miller Analogies Test. The object match subtest is a measure of cognitive flexibility and is modeled after the Visual Verbal Test. Math calculation is the final subtest of this domain, which is a measure of ability to perform basic

mathematical operations and is modeled after the WAIS-R arithmetic subtest. The spatial processing domain is a measure of visuo-spatial processing. It is measured using three subtests: Tic Tac 1 & 2, and The Clock. The Tic Tac tests measure short term recall of the location of patterns presented in a 3x3 block matrix. Tic Tac 1 is a visual display of 3x3 block matrix in which three to five blocks contain a colored square. Immediately after presentation, the examinee reproduces that pattern using the numeric keypad based on visual memory. In Tic Tac 2 is similar to Tic Tac 2 but it presents 3x3 block matrix with different configurations later on in the test. The Clock subtest displays analog clock faces indicating the time with and without associated hour markings. The participant is presented with five digital choices from which the correct time is chosen. The final domain of Reaction Time measures the time between stimuli being presented and the examinee's key response. The MicroCog measures simple reaction time with two subtests: Timer 1 & 2. These subtests measure simple reaction time in auditory, visual, and auditory/visual modalities. Timer 1 is administered at the beginning of and Timer 2 is administered towards the end of test. On both of these tests an examinee will respond to a sound or imagine by pressing the enter key. A participant is presented a warning tone and then press the enter key when an image is presented. The examinee is instructed to respond as fast as possible and to keep his or her finger on the enter key.

Information processing speed (IPS) measures the time the individual takes to complete tasks, while accuracy is disregarded. Information processing accuracy (IPA) reflects the accuracy of performance while disregarding speed. General cognitive functioning (GCF) is a global measure of neurocognitive ability based on performance on the information processing speed and information processing accuracy indexes. No studies have been published to assess the concurrent validity of the GCF with another measure of intellectual ability such as the WAIS-IV,

but Pearson Corporation states that there is a .4 correlation (C. Friedeck, personal communication, February 2, 2010). General cognitive proficiency (GCP) is a measure of neurocognitive ability based on the average of subtest proficiency scores.

For each of the subtests the MicroCog manual provides a reliability coefficient per age group determined by utilizing a single administration, split-half internal consistency methodology. The Spearman-Brown formula was used for correlation and correction for the two halves. For the Attention/Mental Control domain: Number Forward (.61-.72), Numbers Reversed (.76-.84), Wordlist 1 (.84-.90), and Wordlist 2 (.88-.96). In the Memory domain reliability for each subtest is reported as: Story 1 & 2 (.53-.74) and Delayed Recall (.61-.83). For the Spatial Processing domain: Clock (.60-.81) and TicTac (.66-.79). The reliability coefficients for the Reasoning/Calculation domain: Analogies (.30-.73), Math (.60-.78), Object Match A (.45-.69), and Object Match B (.52-.63). For the Reaction Time domain the reliability ranges from .80-.96 for Timer 1 and .75-.94 for Timer 2. A study by Helmes & Miller (2006) compared the MicroCog to the WMS-III and found that general memory index of the WMS-III correlated moderately with the Memory (.53) and General Cognitive Functioning (.52) indexes of the MicroCog. In a study by Raymond, Hinton-Bayte, Radel, Ray, and Marsh (2006) the test-retest reliability coefficient for the five cognitive domains ranged from .49 to .84.

The Beck Anxiety Inventory was used to measure self-reported symptoms of anxiety which are minimally shared with those of depression (BAI; Beck & Steer, 1988). The BAI has 21 items related to anxiety symptoms that are rated on a 4 point scale from 0 (not at all) to 3 (severely; I could barely stand it). Cutoff scores for interpreting the intensity of self-reported anxiety are as follows: 0-7 (minimal); 8-15 (mild); 16-25 (moderate); 26-63 (severe). Internal consistency reliability coefficients range from .85 and .94.

The Center for Epidemiologic Studies Depression Scale was used to measure self-reported symptoms of depression (CES-D; Radloff, 1977). The CES-D has 20 items related to depressive symptomatology that are rated on a 4-point scale from (Rarely or none of the time, less than 1 day) to 3 (Most or all of the time, 5-7 days). Scores range from 0 to 60 and a score of 16 or greater on the CES-D is interpreted as suggestive of clinically significant depression. The CES-D has demonstrated good psychometric properties and its use as a screener for depression among adults has been supported (Radloff, 1977; Boutin-Foster, 2008).

The Epworth Sleepiness Scale was used to measure self-reported sleepiness. The ESS is an 8-item self-report measure designed to assess the overall level of daytime sleepiness (Johns; ESS, 1991). Each item uses a 4-point Likert scale (0 = would never to 3 = high chance). The items scores sum to produce a total score (range 0 – 24). Scores greater than 10 are considered to be suggestive of significant daytime sleepiness; scores greater 15 have been associated with pathological sleepiness that may be due to conditions such as obstructive sleep apnea or narcolepsy. Internal consistency for the ESS was demonstrated in a sample of medical students and patients with sleep disorders. Cronbach's alpha ranged from 0.73 to 0.88. Test-retest reliability was 0.82 for medical students and 0.91 for sleep apnea patients. ESS scores were inversely correlated with sleep latencies determined by overnight polysomnographic studies ($r = -.38$) and by daytime multiple sleep latency test ($r = -.51$), which was expected given that short sleep latencies and low multiple sleep latency tests scores are laboratory indications of daytime sleepiness.

The Alcohol Use Disorders Identification Test (AUDIT) was used to screen for alcohol problems. The AUDIT is a self-report measure developed by the World Health Organization to identify potentially harmful or hazardous drinking patterns in the past year (Babor, del la Fuente,

Sander & Grant, 1992). Internal consistency estimates have ranged from .75 to .94 in a variety of psychiatric populations (Allen, Litten, Fertig, & Babor, 1997; Dawe, Seinen, & Kavanagh, 2000). It contains 10-items that are rated on a 5-point scale (0 = never to 4 = daily or almost daily). AUDIT scores range from 0 to 40. Research demonstrates that it is sensitive and specific (with a cut-off of ≥ 8) in samples of patients with psychiatric disorders (Dawe, Seinen, & Kavanagh, 2000; Maisto, Carey, Carey, Gleason, & Gordon, 2000).

The short Drug Abuse Screen Test (DAST-10) was used to screen for behaviors related to drug abuse. The DAST-10 is a brief self-report screen that contains 10 of the original 20 items designed to assess drug use behavior in the last year (Skinner, 1982). Research has shown that the DAST-10 is internally consistent ($\alpha = .86$), temporally stable ($ICC = .71$), and able to discriminate between psychiatric outpatients with and without current drug abuse/dependence diagnoses (Cocco & Carey, 1998). A score of 3 or above suggests the likelihood of a drug use disorder. Research suggests that sensitivity and specificity with psychiatric population are optimized with a score of 3 or above (Maisto, Carey, Carey, Gleason, & Gordon, 2000).

Procedure

Clinicians at the TBI clinic, R&R center, and Department of Neurology were briefed individually or at a staff meeting on the main aspects of the research and the voluntary nature of participation. Clinicians were asked to briefly announce the research project to soldiers who appear to meet the criteria for participation. When a soldier volunteers, the clinician informed the Fort Hood principle investigator who in-turn informed the research principle investigator (PI). From this point, the research PI contacts the soldier to discuss the research project. Once the soldier has verbally agreed to participation, the PI scheduled the participant for the full informed consent process and testing. Upon arrival in the laboratory, all participants were given two

copies of the informed consent form and briefed on all aspects of the research project by the research PI. Only those who volunteer and sign the consent form participation. Informed consent documents were stored separately from all other measures and documents. Participant's confidentiality was maintained by assigning an identification code used on all assessment records.

Control group participants were recruited using the chain of command. The PI briefed select combat arms unit commanders on the aspects of the study in order for the commander to determine if the PI will be authorized to brief soldiers during daily mass formations. Once authorized, the PI attended daily scheduled mass formations to brief soldiers on the study. Soldiers who volunteered met with the PI after the formation to receive an in-depth briefing on the study and then schedule a time for the formal consent process and testing. Soldiers informed their chain of command of when they were being tested to confirm that there is not a work schedule conflict. Informed consent documents are stored separately from all other measures and documents. Participant confidentiality was maintained by assigning participants and identification code used on all assessment records.

After completing the consent process and filling out the researcher-developed questionnaire, the participants were placed in front of a computer that has the first test loaded. At this point the instructions on the ANAM or MicroCog guided the participant through the test battery. To account for ordering effects such as testing-fatigue, odd numbered participants in each group were administered the MicroCog first and then the ANAM and even numbered participants in each group were administered the test in the reverse order. As another measure taken to counter testing-fatigue, participants were given the option to take at least a 10 minute break before starting the second test.

RESULTS

Data Examination

Prior to analysis, all data were entered into SPSS 17.0 for Windows and examined for missing values, extreme values, and overall accuracy. Relevant variables were converted into a histogram to visually confirm normality. Particular attention was given to the statistical assumption of homogeneity of variance due to differences in cell sizes and the assumption of normality based on small cell sizes. The assumption of homogeneity of variance was assessed via the Levene's test and normality was assessed via the Shapiro-Wilks test. The following variables did not meet the assumption of normality: simple reaction for mild traumatic brain injury (MTBI), simple reaction (repeated) for posttraumatic stress disorder (PTSD) and MTBI, MicroCog reaction time for MTBI, CES-D for control, and BAI for control. A critical significance alpha level of .05 was used for all statistical analyses.

Demographic Data

Sixty three combat arms active duty soldiers from the First Cavalry Division and the 3rd Armored Cavalry Regiment on Fort Hood, Texas participated in this study. Descriptive statistics are presented for all demographic variables in Table 8, including participants' years of education, number of deployments, age, ethnicity, years of service, pay grade, time since concussion to assessment, gender, and history of concussion. Demographics for participants in each group were consistent with the September 2008 statistics report kept by DVBIC; the majority of participants were in their early 20's, white males, junior enlisted (\leq E-6), and in a combat arms job. Participants in the current sample ranged in age from 18 to 29 years ($M = 23.0$, $SD = 3.07$). Planned comparison ANOVAs were conducted in order to determine whether PTSD, MTBI, and control participants differ demographically on variables of age, number of deployments, years of

education, pay grade, and years of service. Results of these analyses indicated a significant age difference between PTSD and control participants. The omnibus F test for age was significant, $F(2,60) = 3.21, p = .047$. The linear contrast comparing PTSD and control participants was significant, $t(34.94) = -2.48, p = .02$ (two-tailed). An examination of the means demonstrates that PTSD participants were oldest, followed by MTBI then control. No other demographic variables were statistically significant between groups.

Data Analytic Strategy

The present study investigated four primary a priori research hypotheses with the purpose of evaluating the difference between MTBI, PTSD, and control. First, it was hypothesized that MTBI participants would score lower on variables of attention than participants in the PTSD group and control group. The second hypothesis for this study was that PTSD participants would score lower on variables of memory than MTBI participants. The third hypothesis for this study was that PTSD participants would score lower on variables of memory than controls. The final hypothesis was that there would not be a statistically significant difference between groups on measures of psychomotor speed. See Table 16 for executive summary of analyses.

First, hypotheses were tested for each dependent variable (DV) with separate univariate one-way ANOVAs with Group (PTSD, MTBI, and Control) as the independent (IV). Univariate ANOVAs are reported per domain: psychomotor speed, memory, and attention. See Tables 9 for means and standard deviations for each variable by domain. ANOVAs with planned contrasts were applied to hypotheses with either Games-Howell or Duncan's multiple range test applied as the selected follow-up test. The Games-Howell post hoc test was applied to analyses that did not meet homogeneity of variance due to unequal sample sizes; it has good power, and tight control over Type I error rate, but can be liberal when sample sizes are small (Field, 2005). Duncan's

multiple range test was applied to analyses that met the assumption of homogeneity of variance. If the Levene's test was less than .05 then the Welch procedure was employed and reported. The Welch procedure was selected because it has considerable advantages both in power and protection against Type I error (Howell, 2007). For comparison purposes, the non-parametric Kruskal-Wallis and Kolmogorov-Smirnov Z follow-up test and parametric ANOVA were reported for analyses containing variables that did not meet the assumption of normality. Kolmogorov-Smirnov Z is similar to the Mann-Whitney test but has better power than the Mann-Whitney test when sample sizes are less than about 25 per group (Field, 2005).

Next, two descriptive discriminant function analyses (DFA) were calculated to further clarify group differences. The first DFA included all three groups and the second DFA only included MTBI and PTSD groups. The PTSD and MTBI groups DFA was calculated to simplify the most clinically meaningful comparison.

Caution should be taken when interpreting multivariate analyses due to small cell sizes. Specifically, the power to detect true differences is greatly attenuated when there are fewer than 30 subjects per group in a multivariate analysis (Stevens, 1996). Tabachnick and Fidell (2001) state that a large sample size is necessary for multivariate statistics because of correlations used to calculate these statistics are not very stable when based on small samples. A small sample size may result in an unacceptable Type II error rate. This occurs because unstable correlations tend to provide less reliable estimates of the degree of relation among variables.

Hypothesis Testing

Psychomotor Speed

Three linear contrasts were computed on the MicroCog's reaction time and the ANAM's simple reaction time, simple reaction time (repeated), and procedural reaction time to examine

the hypothesis that there would not be a statistically significant difference between groups on measures of psychomotor speed.

The omnibus F test for the MicroCog's reaction time was significant, Welch $F(2,35.76) = 7.61, p = .003$. The linear contrast between PTSD and MTBI groups for the MicroCog's reaction time was significant, $t(33.8) = 2.38, p = .02$ (two-tailed). The linear contrast between PTSD and control groups was significant, $t(26) = 3.62, p = .001$ (two-tailed). The linear contrast between MTBI and control groups was not significant, $t(25.3) = 2.76, p = .13$ (two-tailed). Based on the Games-Howell post hoc test, PTSD had the slowest reaction time, followed by MTBI then controls with the latter two not significantly different from each other (see Table 9).

Nonparametric analyses were computed given that the control group MicroCog reaction time variable was not normally distributed (see Table 17). The omnibus H test for reaction time was significant, $H(2) = 9.05, p = .01$. Based on the Kolmogorov-Smirnov Z test, PTSD has significantly slower reaction time, followed by MTBI then controls, with the only significant difference between PTSD and control groups (see Table 17).

The omnibus F test for the ANAM's simple reaction was significant, Welch $F(2,36.53) = 6.88, p = .002$. The linear contrast between PTSD and MTBI groups for the ANAM's simple reaction time was significant, $t(32.8) = 2.59, p = .01$ (two-tailed). The linear contrast between PTSD and control groups was significant, $t(26.5) = 3.84, p = .01$ (two-tailed). The linear contrast between MTBI and control groups was not significant, $t(34.1) = -1.67, p = .10$ (two-tailed). Based on the Games-Howell post hoc test, PTSD had the slowest reaction time, followed by MTBI then controls with the latter two not significantly different from each other (see Table 9). Nonparametric analyses were computed given that the control group ANAM simple reaction variable was not normally distributed (see Table 17). The omnibus H test for reaction time was

significant, $H(2) = 9.49, p = .01$. Based on the Kolmogorov-Smirnov Z test, PTSD has significantly slower reaction time, followed by MTBI then controls with the only significant difference between PTSD and control groups (see Table 17).

The omnibus F test for the ANAM's simple reaction (repeated) was significant, Welch $F(2,38.0) = 4.62, p = .02$. The linear contrast between PTSD and MTBI groups for the ANAM's simple reaction (repeated) time was significant, $t(38.5) = 2.74, p = .01$ (two-tailed). The linear contrast between PTSD and control groups was significant, $t(31.2) = 2.92, p = .01$ (two-tailed). The linear contrast between MTBI and control groups was not significant, $t(35.1) = .148, p = .88$ (two-tailed). Based on the Games-Howell post hoc test, PTSD had the slowest reaction time, followed by control then MTBI with the latter two not significantly different from each other (see Table 9). Nonparametric analyses were computed given that the control group ANAM simple reaction (repeated) variable was not normally distributed. The omnibus H test for reaction time was significant, $H(2) = 11.20, p = .004$. Based on the Kolmogorov-Smirnov Z test, PTSD has significantly slower reaction time, followed by control then MTBI with the latter two not significantly different from each other (see Table 17).

The omnibus F test for the ANAM's procedural reaction was significant, Welch $F(2,38.81) = 5.55, p = .01$. The linear contrast between PTSD and MTBI groups for the ANAM's procedural reaction time was significant, $t(32.7) = 2.96, p = .01$ (two-tailed). The linear contrast between PTSD and control groups was significant, $t(32.7) = 3.29, p = .002$ (two-tailed). The linear contrast between MTBI and control groups was not significant, $t(39) = -.486, p = .63$ (two-tailed). Based on the Games-Howell post hoc test, PTSD had the worst processing speed capacity, followed by MTBI then controls with the latter two not significantly different from each other (see Table 9).

The homogeneity of variance assumption was not met for the DFA that compared all groups (Box's $M-F(20, 12756.61) = 2.45, p = .000$) on the psychomotor speed indices indicating that the covariance matrices cannot be pooled for this analysis. There was a notable canonical correlation (.52) on Function 1 with an effect size of $R^2_c = .27$ (see Table 12). There was a second canonical correlation (.21) on Function 2 with an effect size of $R^2_c = .04$ (see Table 12). The full model test of Functions 1 to 2 was statistically significant at $p < .01$. The test of Function 2 was not statistically significant ($p = .45$) and was thus excluded from consideration. Standardized discriminant function coefficients and structure coefficients were examined to determine what variables contributed to the group difference (see Table 13). For Function 1 procedural reaction time, simple reaction time, and reaction time were responsible for group differences. Regarding the group centroids, it appears that on Function 1 PTSD differed from MTBI and control groups. Classification results indicate that this discriminant function correctly classified 59% of original grouped cases. The discriminant function was better at predicting control (65%), followed by MTBI (57%) then PTSD (55%).

It was determined that the homogeneity of variance assumption was met for the DFA that compared MTBI and PTSD groups (Box's $M-F(10, 7993) = 1.58, p = .11$) on the psychomotor speed indices indicating that the covariance matrices can be pooled for this analysis. In examining the canonical discriminate functions, there was notable canonical correlation (.44) on Function 1 with an effect size of $R^2_c = .19$ (see Table 14). The test of Functions 1 was not statistically significant and was thus excluded from consideration.

Attention

A linear contrast was calculated on the MicroCog's attention/mental control variable and the ANAM's code substitution-learning variable to evaluate the hypothesis that MTBI

participants would score lower on variables of attention than participants in the PTSD and control groups. The contrast compared MTBI to PTSD and controls.

The omnibus F test for the MicroCog's attention/mental control variable was significant, Welch $F(2,38.81) = 6.75, p = .002$. The linear contrast that compared MTBI to PTSD and controls groups on the MicroCog's attention/mental control was not significant, $t(46) = -1.60, p = .06$ (one-tailed). Based on the Games-Howell post hoc test, PTSD has significantly lower attention scores, followed by MTBI then controls with the later two not significantly different from each other (see Table 9).

The omnibus F test for the ANAM's code substitution-learning was statistically significant, $F(2,60) = 4.72, p = .01$. The linear contrast for the ANAM's code substitution-learning was not significant, $t(46) = -1.55, p = .01$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower learn/sustained attention scores, followed by MTBI then controls with the later two not significantly different from each other (see Table 9).

It was determined that the homogeneity of variance assumption was met for the DFA that compared all groups (Box's $M—F(6, 86028.81) = 2.05, p = .06$) on the attention indices indicating that the covariance matrices can be pooled for this analysis. In examining the canonical discriminate functions, there was notable canonical correlation (.49) on Function 1 with an effect size of $R^2_c = .24$ (see Table 12). The canonical correlation (.07) on Function 2 showed effect size of $R^2_c = .005$ (see Table 12). The full model test of Functions 1 to 2 was statistically significant at $p < .002$. The test of Function 2 was not statistically significant ($p = .59$) and was thus excluded from consideration. Standardized discriminant function coefficients and structure coefficients were examined to determine what variables contributed to the group difference (see Table 13). For Function 1 MicroCog's attention was primarily responsible for

group differentiation and to a lesser extent ANAM's code substitution-learning. Regarding the group centroids, it appears that on Function 1 PTSD differed from MTBI and control groups. Classification results indicate that this discriminant function correctly classified 48% of original grouped cases. The discriminant function was better at predicting PTSD (64%), followed by control (45%) then MTBI (33%).

It was determined that the homogeneity of variance assumption was met for the DFA that compared MTBI and PTSD groups (Box's $M-F(3, 326983.70) = 2.28, p = .08$) on the attention indices indicating that the covariance matrices can be pooled for this analysis. In examining the canonical discriminate functions, there was notable canonical correlation (.45) on Function 1 with an effect size of $R^2_c = .20$ (see Table 14). The test of Function 1 was statistically significant ($p = .01$). Standardized discriminant function coefficients and structure coefficients were examined to determine what variables contributed to the group difference (see Table 15). For Function 1 MicroCog's attention was primarily responsible for group differences and to a lesser extent ANAM's code substitution-learning. Classification results indicate that this discriminant function correctly classified 65% of original grouped cases. The discriminant function was slightly better at predicting PTSD (68%) than predicting MTBI (62%).

Memory

A linear contrast was calculated on the MicroCog's reasoning/calculation, memory, and spatial processing variables and the ANAM's code substitution-delay (recognition), math processing, and match-to-sample variables to examine the study hypothesis that PTSD participants will score lower on variables of memory than MTBI participants. The contrast compared MTBI to PTSD.

The omnibus F test for MicroCog's reasoning/calculation was statistically significant, $F(2,60) = 3.84, p = .01$. The linear contrast between MTBI and PTSD for MicroCog's reasoning/calculation was significant, $t(60) = -1.74, p = .04$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower reasoning (working memory) scores than controls with no significant differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

The omnibus F test for MicroCog's memory was statistically significant, Welch $F(2,37.88) = 5.64, p = .004$. The linear contrast between MTBI and PTSD for MicroCog's memory was not significant, $t(36.61) = -1.60, p = .06$ (one-tailed). Based on the Games-Howell post hoc test, PTSD has significantly lower memory scores than controls with no significant differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

The omnibus F test for MicroCog's spatial processing was statistically significant, $F(2,60) = 4.55, p = .01$. The linear contrast between MTBI and PTSD for MicroCog's spatial processing was not significant, $t(60) = -.949, p = .17$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD and MTBI groups have significantly lower spatial processing scores than controls with no difference between PTSD and MTBI (see Table 9).

The omnibus F test for ANAM's match-to-sample was statistically significant, $F(2,60) = 3.25, p = .02$. The linear contrast between MTBI and PTSD for ANAM's match-to-sample was not significant, $t(60) = -1.53, p = .07$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower spatial memory scores than controls with no significant differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

The omnibus F test for the ANAM's code substitution-delay (recognition) was not statistically significant, $F(2,60) = .802, p = .23$. The linear contrast between MTBI and PTSD for the ANAM's code substitution-delay (recognition) was not significant, $t(60) = .579, p = .28$ (one-tailed) (see Table 9).

The omnibus F test for the ANAM's math processing was statistically significant, $F(2,60) = .261, p = .04$. The linear contrast between MTBI and PTSD for the ANAM's math processing was not significant, $t(46) = -.746, p = .23$ (one-tailed). PTSD has significantly lower math processing (working memory) scores than control with no significant differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

A linear contrast was calculated on the MicroCog's reasoning/calculation, memory, and spatial processing variables and the ANAM's code substitution-delay (recognition), math processing, and match-to-sample variables to evaluate the hypothesis that PTSD participants will score lower on variables of memory than controls. The contrast compared PTSD to controls.

The omnibus F test for MicroCog's reasoning/calculation was statistically significant, $F(2,60) = 3.84, p = .01$. The linear contrast between PTSD and controls for MicroCog's reasoning/calculation was significant, $t(60) = 2.73, p = .004$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower reasoning (working memory) scores than controls with no significant differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

The omnibus F test for MicroCog's memory was statistically significant, Welch $F(2,37.88) = 5.64, p = .004$. The linear contrast between PTSD and controls for MicroCog's memory was significant, $t(30.06) = 3.18, p = .02$ (one-tailed). Based on the Games-Howell post hoc test, PTSD has significantly lower memory scores than controls with no significant

differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

The omnibus F test for MicroCog's spatial processing was statistically significant, $F(2,60) = 4.55, p = .01$. The linear contrast between PTSD and controls for MicroCog's spatial processing was significant, $t(60) = 2.97, p = .002$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD and MTBI groups have significantly lower spatial processing scores than controls with no difference between PTSD and MTBI (see Table 9).

The omnibus F test for ANAM's match-to-sample was statistically significant, $F(2,60) = 3.25, p = .023$. The linear contrast between PTSD and controls for ANAM's match-to-sample was significant, $t(60) = 2.53, p = .01$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower spatial memory scores than controls with no significant differences between PTSD and MTBI groups or between MTBI and control groups (see Table 9).

The omnibus F test for the ANAM's code substitution-delay (recognition) was not statistically significant, $F(2,60) = .802, p = .23$. The linear contrast between PTSD and controls for the ANAM's code substitution-delay (recognition) was not significant, $t(46) = .579, p = .28$ (one-tailed) (see Table 9).

The omnibus F test for the ANAM's math processing was significant, $F(2,60) = .261, p = .04$. The linear contrast between PTSD and controls for the ANAM's math processing was significant, $t(60) = 2.25, p = .01$ (one-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower math processing (working memory) scores than control with no differences between PTSD and MTBI groups or MTBI and control groups (see Table 9).

It was determined that the homogeneity of variance assumption was met for the DFA that compared all groups (Box's $M—F(42, 10558.32) = .99, p = .50$) on the memory indices indicating that the covariance matrices can be pooled for this analysis. In examining the canonical discriminate functions, there was notable canonical correlation (.50) on Function 1 with an effect size of $R^2_c = .25$ (see Table 12). The second canonical correlation (.20) on Function 2 showed an effect size of $R^2_c = .04$ (see Table 12). The full model test of Functions 1 to 2 and Function 2 were not statistically significant ($p < .09$ and $p < .79$, respectively); thus they were excluded from consideration.

It was determined that the homogeneity of variance assumption was met for the DFA that compared MTBI and PTSD groups (Box's $M—F(21, 6152.12) = 1.09, p = .35$) on the memory indices indicating that the covariance matrices can be pooled for this analysis. The canonical correlation (.32) on Function 1 (effect size of $R^2_c = .10$, see Table 14) was not statistically significant.

Supplementary Analyses

A linear contrast was calculated on the BAI, CES-D, ESS and MicroCog variables of General Cognitive Functioning (GCF), General Cognitive Proficiency (GCP), and Information Processing Accuracy (IPA) to determine whether there were differences between PTSD and MTBI on these indices.

The omnibus F test for the BAI was statistically significant, Welch $F(2,39.52) = 14.94, p = .000$. The linear contrast for the BAI was significant, $t(37.34) = -3.97, p = .000$ (two-tailed). Based on the Games-Howell post hoc test, PTSD has significantly higher anxiety scores, followed by MTBI then controls with the later two not significantly different from each other. Nonparametric analyses were computed given that the control group BAI variable was not

normally distributed (see Table 17). Results of this nonparametric analysis confirm the parametric findings with the omnibus H test for the BAI being significant, $H(2) = 22.02, p = .000$. Based on the Kolmogorov-Smirnov Z test, PTSD has significantly higher anxiety scores, followed by MTBI then controls with the later two not significantly different from each other.

The omnibus F test for the CES-D was significant, $F(2,60) = 13.06, p = .000$. The linear contrast for the CES-D was significant, $t(60) = -4.46, p = .000$ (two-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly higher depression scores, followed by MTBI then controls with the later two not significantly different from each other (see Table 10). Nonparametric analyses were computed given that the control group CES-D variable was not normally distributed (see Table 17). Results of this nonparametric analysis confirm the parametric findings with the omnibus H test for the CES-D being significant, $H(2) = 19.30, p = .000$. Based on the Kolmogorov-Smirnov Z test, PTSD has significantly higher depression scores, followed by MTBI then controls with the later two not significantly different from each other.

The omnibus F test for the ESS was not significant, $F(2,60) = .89, p = .42$. The linear contrast for the CES-D was not significant, $t(60) = -1.22, p = .23$ (two-tailed) (see Table 10).

The omnibus F test for the GCF was significant, $F(2,60) = 11.06, p = .000$. The linear contrast for the GCF was significant, $t(60) = 2.35, p = .02$ (two-tailed). Based on the Duncan's multiple range test, PTSD has significantly lower general cognitive functioning scores, followed by MTBI then controls with all three group being significantly different from each other (see Table 11).

The omnibus F test for the GCP was significant, $F(2,60) = 9.63, p = .000$. The linear contrast for the GCP was significant, $t(60) = 2.31, p = .03$ (two-tailed). Based on the Duncan's

multiple range test, PTSD has significantly lower general cognitive proficiency scores, followed by MTBI then controls with all three group being significantly different from each other (see Table 11).

The omnibus F test for the IPA was significant, $F(2,60) = 8.07, p = .001$. The linear contrast for the IPA was significant, $t(60) = 2.58, p = .01$ (two-tailed). Based on the Duncan's multiple range post hoc test, PTSD has significantly lower processing accuracy scores, followed by MTBI then controls with the later two not significantly different from each other (see Table 11).

Analyses of covariance (ANCOVA) were calculated to evaluate the effects of CES-D and BAI on the observed differences on attention variables between MTBI and PTSD. None of these differences were affected by introducing the covariates; PTSD subjects still showed significantly worse attention, even when controlling for depression and anxiety. This finding was replicated when covariates of CES-D and BAI were included in one ANCOVA.

DISCUSSION

The present study investigates neuropsychological functioning differences between MTBI and PTSD in order to provide mental health professionals information to assist in differentiating MTBI and PTSD. Of the four hypotheses examined in this study, the only hypothesis that was strongly supported related to PTSD participants scoring lower on variables of memory than controls.

The hypothesis that MTBI participants would score lower on variables of attention than participants in the PTSD and control groups was not supported. At the multivariate level of analyses PTSD differed from MTBI and controls, but MTBI and control groups were not significantly different from each other. This finding was replicated at the univariate level of analysis.

Previous literature supports the finding that PTSD participants performed worse on measures of sustained attention and learning in comparison to non-PTSD control (Vasterling, Brailey, Constans, Constans, & Sutker, 1998; Vasterling et al., 2002; Yehuda et al., 1995). The finding that MTBI in the post-acute recovery phase does not differ from control on a measure of sustained attention was also supported by the literature (Vanderploeg, Curtiss, & Belanger, 2005). Additionally, a MTBI study that tracked cognitive functioning from immediately post-injury, at one month, and 3 month post-injury demonstrated that sustained attention remain significantly poorer over the 3 month period as compared to control (Kwok, Lee, Leung, & Poon, 2008). Overall, the finding that PTSD performed significantly worse on measures of attention than MTBI and control groups may best be accounted for by the hyperactive noradrenergic function and dopaminergic sensitization found in PTSD, which impacts functioning of the medial prefrontal cortex (mPFC). The mPFC has been has been implicated in

the regulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis and modulating working memory and attention (Radley, Williams, & Sawchenko, 2008).

The hypothesis that there would not be a statistically significant difference between groups on measures of psychomotor speed was not supported. Results of a DFA that compared all groups indicated that PTSD significantly worse than MTBI followed by control. However, results of a DFA that compared PTSD and MTBI did not support the hypothesis. Results of univariate analysis showed various group differences, depending on what type of analysis was calculated: parametric or nonparametric. Parametric results indicate that PTSD differed from MTBI and control groups. Nonparametric results indicated that PTSD differed from MTBI and control groups on MicroCog Reaction Time and ANAM Simple Reaction Time and MTBI differed from PTSD and control groups on ANAM Simple Reaction Time (R).

The finding from this study that PTSD performed significantly worse than MTBI and control groups was not supported by previous literature. Previous literature suggests that there was not a significant difference between PTSD and control groups (Crowell, Kieffer, Siders, & Vanderploeg, 2002; Samuelson et al. 2006). A study by Brenner et al. (2010) demonstrated that there is not a significant difference between post-acute recovery phase MTBI and control on a measure of processing speed. Knowing that PTSD performed significantly worse than MTBI and control groups on most measures of psychomotor speed may suggest to neuropsychologists that when assessing clients diagnosed with PTSD with a history of blast-MTBI, that the decreased functioning is most likely due to enduring cognitive symptoms related to PTSD than a by-product of blast injury. Deficits in psychomotor speed can be accounted for by dysfunction of the dopaminergic system. Dopaminergic dysfunction has also been linked to PTSD symptoms of hypervigilance and exaggerated startle (Charney, Deutch, Krystal, Southwick, & Davis, 1993).

Pharmacological treatment using methylphenidate has been shown to improve speed of processing, via D2 receptor (Warden, et al., 2006).

The hypothesis that PTSD participants would score lower on variables of memory than MTBI participants was not supported. Results of the DFA that compared all groups and the DFA that compared MTBI and PTSD indicated that no groups were significantly different from each other. At the univariate level of analysis it appears that neither group differed on any index of memory.

No literature was found to substantiate this finding because literature only compared MTBI or PTSD to a control group. This may be due to the experimental nature of postconcussive symptoms following MTBI. One plausible reason for this finding is that it may suggest that those with MTBI do not have enough cognitive reserve to make up for a deficit in memory. Another plausible reason for this finding is that soldiers in these two groups may share a common deficit in overall cognitive functioning which does not allow the soldier to compensate for a deficit.

The final hypothesis that PTSD participants would score lower on variables of memory than controls was supported at the univariate level; depending on the type of memory measured. Results of the DFA that compared all groups and the DFA that compared MTBI and PTSD indicated that no groups were significantly different from each other. At the univariate level of analysis five of the six memory indices indicated that PTSD differed from control. From the 5 indices that differentiated PTSD from control it appears that PTSD performed the worst followed by MTBI then control. Code Substitution-Delay (long-term memory) was the only indice that did not differentiate groups.

A study by Vasterling, Brailey, Constans, and Sutker (1998) confirmed that PTSD performed significantly worse than controls on a measure of short term memory. However, this study also demonstrated that PTSD differed from control on a measure of long-term memory, which is incongruent with the current study. The finding that PTSD had worse working memory than controls was confirmed by Vasterling et al. (2002). Worse memory functioning in PTSD participants may be related to enduring cognitive symptoms. This is useful information when conducting a neuropsychological evaluation of a soldier diagnosed with PTSD with a history of blast-MTBI.

Supplementary analyses revealed that PTSD endorsed significantly more depressive and anxiety symptoms followed by MTBI then control with the latter two not significantly different from each other. Analyses to assess the relative contribution of depressive and anxiety symptoms on the difference between PTSD and MTBI indicated that PTSD alone was associated with cognitive impairment.

There is no previous literature to substantiate this finding because literature has been dedicated to differentiating MTBI or PTSD from a control group. One plausible explanation for this finding is that anxiety and depression are enduring behavioral symptoms of PTSD. Related to this finding is the rate of PCS symptoms endorsed in those who sustained MTBI. Studies show that the reporting of PCS symptoms is influenced by factors other than head injury, suggesting that PCS symptoms are not specific to mild traumatic brain injury. Iverson (2006) determined postconcussion-like symptoms were common in depressed people. This suggests that depression should be considered in the differential diagnosis process. Further, Suhr and Gunstad (2002) demonstrated that depressive symptoms accounted for elevations in PCS symptoms and no cluster of PCS symptoms were specific to head injury. In a study consisting of

active duty service members and veterans, Belanger, Kretzmer, Vanderploeg, and French (2010) determined that after controlling for PTSD symptom severity that there was not a significant difference between MTBI and moderate to severe TBI groups on rate of postconcussion symptom endorsement.

Another finding from supplementary analyses was that there was not a significant difference in subjective sleepiness between groups. It would seem intuitive that those diagnosed with PTSD would have worse sleep, particularly given that sleep difficulty is a diagnostic criterion (D1) for PTSD. However, this finding may best be accounted for by the under reporting behavior of participants. This under reporting behavior may be a cultural phenomenon and most likely not an effort to present oneself in a more positive light. A study by Linn (1946) looked at whether a psychological test based on civilian norms was able to measure the same underlying construct in military personnel. The objective of this study was to determine whether there was a difference between military personnel and the civilians. It was discovered that responses given by normal soldiers were different from norms based on responses of normal civilians. The difference was hypothesized to be due to personality changes that have been produced by military indoctrination. Linn (1946) advised that this is a normal process in the course of adaptation to army life and must be taken into account in the military use of any personality tests based on civilian standards. This study showed that there is a difference between military personnel and civilians that should be taken into account by military psychologists that conduct behavioral assessment.

Neuroimaging studies have even demonstrated that there are some shared neuroanatomical structures between those that modulate sleep and those that are implicated in fear response and PTSD. Germain, Buysse, and Nofzinger (2008) have postulated that the

hyperactivity amygdala and impaired medial frontal cortex associated with PTSD also influence the regulation and/or expression of rapid eye movement (REM) and non-rapid eye movement (NREM) sleep. Further, a meta-analysis of polysomnographic studies comparing sleep in people with and without PTSD showed that PTSD participant had more stage 1 sleep, less slow wave sleep, and greater rapid-eye movement density compared to participants without PTSD (Kobayashi, Boarts, & Delahanty, 2007).

Clinical and Operational Implications

The accurate identification and effective treatment of active duty soldiers who have received a diagnosis of PTSD and have received a concussive blast MTBI is one of the primary goals in military psychology today. However, the clinical distinction between PTSD and postconcussive syndrome is complicated by overlapping symptomatology. According to DVBIC, attention difficulties, depression and anxiety are overlapping symptoms. Further complicating the diagnostic process is that there are no definitive biological markers for establishing the diagnosis of PTSD or MTBI and there is no neuroimaging method to differentiate PTSD and post-concussive syndrome.

Results of this study demonstrated that attention indices are the strongest differentiator of those diagnosed with PTSD without a history of concussive MTBI than those that received a concussive blast MTBI without a diagnosis of PTSD. This finding should be taken into consideration by military neuropsychologists who are trying to determine whether neurocognitive sequelae are associated with MTBI or PTSD. Two cautionary statements about these findings need to be made. First, as with every diagnosis, clinicians should use multiple indices of neurocognitive sequelae when arriving at a diagnosis. Second, caution should be taken when interpreting results of this study based on findings being based on a small sample size.

In operational terms, combat arms MOS leaders may observe decreased performance in soldiers diagnosed with PTSD on tasks that demand a high level of attention and psychomotor speed such as gunnery. A high degree of attention and psychomotor speed is essential in MOSs such as armor and mechanized infantry because a fight is usually won by the crew that gets the round out first. Leaders should operationally evaluate the extent to which neurocognitive deficits may impact a soldier's performance and determine whether the soldier is a fit for their current duty position or even whether there is a need to re-class a soldier to a MOS that does not place a premium on attention and psychomotor speed.

Direction for future research

There are two camps on the post concussional syndrome (PCS) debate: those who think it is psychological and those who think it is physiological (McCrae, 2008). It is plausible that a third position exists, attributing PCS to a mixture of both physiological and psychological components. It could be possible for a soldier to score within normal limits on traditional paper based test, but have positive QEEG and/or functional imaging and still have cognitive and emotional issues associated with the trauma. Currently, there are no definitive biological markers for establishing a clinical diagnosis of MTBI or PTSD and there is no imaging method for identifying or distinguishing PCS from PTSD.

Future research should be conducted to determine how and if physiological measures such as QEEG and neuroimaging could be used in an assessment battery to differentiate between PTSD and MTBI. One step would be to determine if traditional paper-based and computer-based neuropsychological measures correlate with physiological markers such as EEG and neuroimaging. Current research has found that different physiological measures correlate with each other. Thatcher et al. (2001) found that qEEG measures correlate with qMRI. Kirov et al.

(2007) in a proton magnetic resonance spectroscopy (H-MRS) study, found that changes in metabolic levels in the thalamus in those who sustained MTBI could be a characteristic of mildness. This finding suggests that H-MRS could serve as an objective laboratory indicator for differentiating mild from severe categories of head-trauma, regardless of the presence or lack of current clinical symptoms. Miles et al. (2008) concluded that diffuse tensor imaging (DTI) may provide short term predictive markers of cognitive functioning in patients with MTBI. Changes in DTI have been shown to be present in early and late post injury, which suggests a role as an early indicator and potential prognostic measures of permanent brain damage. (Miles et al. 2008). A neuroimaging study by Huang (2009) used magnetoencephalography (MEG) and diffusion tensor imaging (DTI) to evaluate their utility in diagnosing MTBI. Results of that study provided findings that could potentially be used to differentiate PTSD, MTBI, and those with post-concussive sequelae. One finding was that the integrated approach with MEG and DTI was more sensitive than conventional CT and MRI in detecting subtle neuronal injury in MTBI. This finding is important because emergency room physicians typically used structural imaging techniques such as CT or MRI to rule-out the necessity of neurosurgical intervention. A second finding was that this integrated imaging approach provided evidence for neurocognitive post-concussive symptoms. A third finding was that in some cases, abnormal MEG delta waves were observed in subjects without DTI abnormality, indicating that MEG may be more sensitive than DTI in diagnosing MTBI. Employing a multi-method multi-trait approach should increase the validity and reliability of the neuropsychologist diagnosis.

Second, research should look at the utility of the AAN's concussion Grade scale. Two areas to focus on are: 1) whether there is any physiological and paper-based testing evidence for the different grade scale scores; and 2) whether the grade scales are predictive of recovery. If

there is no physiological evidence for a grade scale than this may indicate that there should only be an overarching definition of MTBI instead of scaling the damage. Research on determining the predictive capacity of the AAN grade scale is necessary because, if validated, it could replace GCS and GOS in predicting recovery from MTBI. It could also guide MTBI research. Based on these findings, the DoD should investigate the utility of incorporating the AAN's concussion grade scale into basic medical training and expert field medical training. If combat medics are able to triage at the time of injury this data might be helpful for researchers and predicting recovery.

Third, research should focus on determining what pharmaceutical therapies are effective in treating cognitive and psychological sequelae of MTBI. At present, no medication has received approval from the United States Food and Drug Administration (FDA) for the treatment of any behavioral or cognitive consequence of TBI. Arciniegas, Anderson, Topkoff, and McAllister (2005) suggest that in the absence of published studies to guide treatment, the selection of pharmacologic agents should be modeled after the approach used to select such agents for patients with cognitive, emotional, or somatic symptoms arising from other neurological or primary psychiatric conditions. Warden et al. (2006) went a step further and convened a panel of experts to review current literature on pharmacological treatment for TBI sequelae and found that the majority of evidence did not support any treatment standards and few guidelines due to a number of recurrent methodological problems. Further, the working group provided evidence-based guidelines that should guide the pharmacological treatment of neurobehavioral sequelae. The working group also determined that there is a need for well-designed randomized controlled trials to establish definitive treatment standards for those who suffer a TBI. Some research has shown that donepezil improves general functioning and

improves attentional skills in patients with chronic TBI sequelae (Khateb, Ammann, Annoni, & Diserens, 2005).

Fourth, the field of psychology should collaborate with AAN to clarify existing neuropsychological measures as per their recommendations for future research, ~~development~~ of a standardized, neuropsychological test battery designed to detect impairment associated with concussion” (AAN Practice parameters, p.5).

Fifth, researchers should develop a measure of perceived cognitive effort or perception of attention and memory difficulties. A MTBI soldier may show normal functioning on tests results, but still complain of working hard to remember or using calendars or the phone more frequently than pre-injury to compensate for deficits. Reynolds (2001) refers to the difference between within-normal-limits neuropsychological test results and patient perception as ~~functional reserve.~~” The concept of functional reserve suggests that a person may compensate for mild traumatically induced neuronal loss as a result of inherent redundancies in brain structures and systems. It was also suggested that additional brain trauma may result in a depleted reserve capacity and limit the rate and degree to which functional recovery can occur. An analogy for the functional reserve is that you are a V8 truck towing a boat at 55mph and then you get a concussion. The concussion is like losing two cylinders. You can still tow the boat at 55mph as a V6, but you have to spray more gas to do it. This may be what a MTBI soldier could be experiencing. Perhaps a baseline fMRI would help in distinguishing this perception. There is also a need for the development of tests that help clinicians distinguish between psychogenic amnesia, vestibular imbalance, whiplash, and actual concussion. These all can produce symptoms similar to PCS from a MTBI (McCrea, 2008). Developing these measures may shed

some light onto the PCS debate and/or help clinicians determine whether they should refer for a neuroimaging or physiological assessment.

Sixth, as an extension of this study, researchers should study how soldiers diagnosed with PTSD and Cognitive disorder NOS (based on the perception that cognitive deficits are due to a concussive blast injury) differ from those soldiers diagnosed with PTSD without a blast injury to further aid in the understanding of how MTBI and PTSD groups differ. A study by Brenner et al. (2010) compared post-blast MTBI soldiers that either had enduring MTBI symptoms, no enduring MTBI symptoms, and those that sustained MTBI and where diagnosed with PTSD. Results suggested that the presence of MTBI symptoms did not impact test performance. Further, there were no differences between soldiers with and without PTSD. The finding of no difference between those with and without PTSD is incongruent with the findings of the present study and is an indicator that a large sample study is required to determine whether is actually a difference between these groups.

Seventh, the Department of Defense should conduct research into the utility of using the AAN's sideline evaluation and research into the AAN's concussion management strategy following the first event and when to return to play after removal from contest guidelines. Researchers should determine whether these evaluation and management strategies can be feasibly employed in the military decision-making model for those who receive MTBI. One focus should be on how these standards could possibly impact a unit's readiness and effectiveness. There is a delicate balance between impacting a unit's effectiveness and a soldier's long-term neurologic health, such as the early onset of Alzheimer's disease. For example, the need to rotate which soldier is on point during a patrol may come into question. At the platoon level, for mechanized infantry and tank units, there is a prescribed doctrinal vehicular

marching order in which the platoon leader's wingman leads, then the platoon leader, followed by the platoon sergeant, and finally the platoon sergeant's wingman. This order is ingrained in the doctrinal responses (battle drills) to enemy contact. In battle drills each vehicle in the order performs a specific function. During the deployment training cycle a unit employs this doctrine so that each vehicle knows how to react when in contact and it is typical that combat commanders do not train soldiers to serve in the different roles. Typically the vehicle on point gets hit the most and therefore these soldiers receive multiple concussions throughout the tour.

Further, the DoD should develop a military version of AAN's sideline evaluation for employment with front line medical technicians such as the combat medic or combat lifesaver. For example, instead of asking about name of team in a prior contest, the combat medic could ask who their team/squad/ platoon leader was on their last mission. It is vital to develop this version for frontline providers because a soldier is more likely to discuss symptoms with these technicians than a primary care manager due to perceived repercussion such as being pulled off the line away from their unit.

Eighth, research should be conducted to determine whether a combat arms soldier diagnosed with PTSD performs worse than controls on tasks that place heavy demands on attention and psychomotor speed, such as Abrams or Bradley gunnery. On these tasks, a high premium is placed on these domains because the person who acquires the enemy first is typically able to get the first round on target; hence, increasing survivability of crew and equipment. In using Abrams and Bradley gunnery, a researcher can see if there is a difference between day and night performances, especially at night when sleep deprivation sets in. If combat arms soldiers diagnosed with PTSD perform worse on these tasks, it will be an indicator to combat arms

commanders and leaders that these soldiers may need to be re-classed to a MOS that does not place a premium on psychomotor speed and attention for survival.

Finally, the Veterans Administration (VA) needs to prepare for the possibility that this wave of combat veterans will have early onset of neurodegenerative disease such as Alzheimer's disease due to repeated concussions. Particular focus should be placed on identifying and tracking soldiers who served on Task Force Iron Claw because their job of route clearance typically involved receiving multiple mild concussions throughout their tour. According to Randolph (2001), the depletion of the reserve capacity via repeated brain trauma could have two potential effects. The first is a permanent loss of some neurocognitive functions, and the second is a potentially increased sensitivity to the effects of normal aging or other disease states on the brain (for example, the premature expression of age-related degeneration). In preparation for this generation's veterans with dementia-related disorders, the VA should start tracking Vietnam veterans with Alzheimer's disease to search for combat-related predictors.

Limitations

A major limitation of this study is its small sample size, which limits its generalization to the overall military population. Given the small sample size, the present study's findings need to be replicated to determine whether results can be generalized. The reasons for the small sample consist of the establishment of stability in Iraq, combat rotation cycle, and training cycle. At the onset of this study, President George W. Bush's "Surge" in Iraq was wrapping up and the objective of stability was met. A by-product of the surge was that there were fewer combat situations to produce MTBI or PTSD; therefore limiting the number of participants. Other by-products of the successful surge were the movement of forward operation bases out of Iraqi cities and decreased combat patrol, which relates to decreased chances of injury or being exposure to

life threatening situations. Second, participants for this study could only be pulled from soldiers who have served in Iraq based on Fort Hood combat arms units because these units only deployed to Iraq. Combat arms units on Fort Hood are categorized as heavy units, which mean that they consist of armored vehicles like 70-ton tanks and 40-ton Bradley fighting vehicles. The mountainous terrain in Afghanistan is not conducive to the employment of armored warfare. The finally factor revolves around scheduling participants while they are training for deployment and returning from deployment. Training for deployment for a heavy combat arms unit typically consists of 30 days of train up in the field at Fort Hood followed by a 30 day training center rotation, amongst other shorter field events. During these two blocks of 30 days training, the soldier does not go home and averages a 20 hour work day. Upon completion of these 30 day training events, it typically takes soldiers several days to a week to readjust. During this adjustment period, it is not advised to test soldiers because of the effects of sleep deprivation and rowdy spirit, amongst other psychological factors, that could affect testing results. Other military factors to consider are coming in to test from railhead operations, or coming in for testing after working staff duty (24 hour duty).

Limitations of the ANAM were observed in the wording of the TBI questionnaire which appears to be written at a higher reading level than the average participants' reading level. Participants were particularly challenged by words such as exertion and fidgety. It is recommended that the developer assess the reading level of the questionnaire and make adjustments according to the population this test is designed to assess. Another issue was observed during the Reaction Time subtest in which soldiers appeared to pick up on the pattern of presentation; when the pattern changes it caused a delayed reaction to the following asterisk. This behavior causes a large standard deviation which is not an accurate reflection a soldier's

reaction time. Code Substitute Delay is another subtest that clinicians need to cautiously interpret because there is a 50% chance of guessing correctly, which can produce a false negative. To decrease the chance of correctly guessing and provide a more accurate assessment of delayed recall, the developer could have four items presented at the same time and have the soldier click on the item he or she truly remembers. Taking this approach may produce a negligible increase in overall administration time, but still keeps with the spirit of being a screening measure. A third limitation of the ANAM is that it only has one mode of measuring reaction time (visual) whereas the MicroCog has three (visual, auditory, and auditory/visual). The MicroCog's measure of reaction time best mimics what a Bradley or Tank crewman goes through when operating the vehicle. The following discussion will elaborate why the MicroCog has better utility in assessing an armored vehicle crewman's functioning than the ANAM, particularly for gunners. There are three reactions that a gunner must go through: 1) the gunner identifies the target and follows the vehicle commander's commands, 2) the vehicle commander identifies the target and moves the turret to the target, which makes the gunner visually acquire the target and go through the rest of the firing sequence, and 3) the driver or loader gives the distance and direction and target type and the gunner has to move the turret and visually acquire the target. Further complicating this reaction is that the gunner has to know who is talking and has to know what azimuth the hull is in (turret vs. hull orientation). For example, a gunner scans for a target and listens for the tank commander's fire command. When the gunner visually acquires a target he goes through a sequence that involves ranging the target and selecting the correct ammunition button while still visually tracking the target. Once this has been done the gunner waits for the tank commander to give the command to fire. To be ecologically valid, multimodal reaction time testing seems most appropriate.

A limitation of the MicroCog is that participants were confused on a task that required them to replicate a design they previously saw using the number keys. The number keys correspond to a certain box in the design. For example, in a 3x3 box the upper left hand square corresponds to the 7 key, the lower right hand square corresponds to the 3 key, and the middle of the box corresponds to the 5 key. Further, the number pad is laid out like the design the participant has to replicate. Of note, a significant portion of participants preferred to use the non-number key pad numbers even though they were previously instructed to use the number key pad. Participants who did not use the number key pad had to experiment to figure out what number was associated with which box when using the number keys that are in a row, typically at the top of the keyboard.

Limitations of the author-developed questionnaire were based on wording of questions 17, 18, 21, and 22, which required the participant to remember information that occurred over multiple tours. In discussion with soldiers about questions 17 and 18, a significant portion of participants stated that they were unable to accurately report due to multiple combat tours and not remembering what happened during each. Another factor impacting the accuracy of reporting for these questions was examinees' definition of "wounded." A significant portion of soldiers did not consider receiving a concussion as a wound or as an injury, and tended to define a wound as an incident that led to observable physiological manifestation such as bleeding or breaking a bone. This notion appears to extend to Army regulations for reception of the Purple Heart. According to the regulation, a soldier is not authorized to receive the Purple Heart for receiving a combat-induced MTBI or having combat-induced PTSD (Paragraph 2-8, Army Regulation 600-8-22). In discussion with neurofeedback clinicians, the tendency not to interpret a closed head injury as a wound or injury is also seen in a non-military population (C. Fisher &

M. Johnson, personal communication, February 12, 2010). A second limitation to questions 21 and 22 was that it did not ask about the number of fire-fights in which a soldier was involved, which could be considered traumatic events.

Another significant limitation of this study is the reliability of the information on self-reports and some computer test results. It was common for soldiers to fall asleep during this study while taking a computer-based test. These soldiers typically self-report a 4 on the ANAM sleep scale, which is not indicative of falling asleep in a chair while taking a test. Those who fell asleep during the administration of a computer-based test were excluded from the study because their results did not accurately reflect their functioning. According to AUDIT results, 20% of participants drank (likely a dramatic underestimate of the base rate of drinking in a military sample). This under-reporting behavior occurred even with assurance that their information would not go into their medical records or be viewed by their chain of command. This suggests that caution should be taken when interpreting results of other self-reports in the study or that the more interactive paper-based tests may be a more accurate reflection of a soldier's true score.

Conclusion

Overall, the present study indicates that attention is the strongest differentiator of MTBI from PTSD, whereas memory indices do not appear to differentiate these groups. Further examination of results indicates that PTSD performed worst on most indices of attention, psychomotor speed, and memory than MTBI and control groups. However, most indices were unable to differentiate MTBI from control. Of note, MicroCog's spatial processing was the only variable that differentiated MTBI from control and ANAM's code substitution-delay (long-term memory) did not differentiate any groups. Deficits in cognitive functioning found in PTSD are most likely due to enduring cognitive symptoms, which is best accounted for by neuroanatomical

abnormalities caused by prolonged exposure to glucocorticoids and neurochemical systems that regulation neuropsychological functioning. In order to more accurately differentiate MTBI and PTSD it is recommended that neuropsychologists incorporate physiological measure such as neuroimaging and EEG into their evaluation. Finally, more research needs to be done on active duty military personnel so that military psychologists do not have to rely solely on research conducted on veterans to develop hypotheses about neurocognitive functioning. These are two unique populations and research should be dedicated to determining whether it is appropriate to draw similar conclusions based on the different populations.

APPENDIX A

ANAM TBI MILITARY TEST LIST AND ANAM AND MICROCOG INDICES PAIRED
WITH PSYCHOLOGICAL CONSTRUCTS

Table A.1

ANAM TBI Military Test List

Test List	Domain/Function
Demographics	User Profile
TBI Questionnaire	TBI History
Sleepiness Scale	Fatigue
Mood Scale	Mood State
Simple Reaction Time	Basic neural processing (speed/efficiency)(Emphasis on motor activity)
Code Substitution – Learning	Associative Learning (speed/efficiency)
Procedural Reaction Time	Processing Speed (choice RT/rule adherence)
Mathematical Processing	Working Memory
Matching to Sample	Visual Spatial Memory
Code Substitution – Delayed(Recognition)	Memory (delayed)
Simple Reaction Time (R)	Basic neural processing (speed/efficiency)

Table A.2

ANAM and MicroCog Indices Paired with Psychological Constructs

paired domains for MANOVA analysis		
Domain	ANAM domains	MicroCog indexes
Psychomotor Speed	Simple Reaction, Simple Reaction (R), Procedural Reaction time	Reaction Time
Memory	Math Processing, Matching to Sample, Code Substitution-Delay	Memory, Reasoning/Calculation, Spatial processing
Attention	Code Substitution-Learning	Attn/Mental Control

APPENDIX B

DEFINITIONS OF MTBI FOR WHO, CDC, ACRM, AND AAN

World Health Organization (WHO) MTBI is an acute brain injury resulting from mechanical energy to the head from external forces. Operational criteria for clinical identification include:

A) One or more of the following:

- . Confusion or disorientation.
- . Loss of consciousness for 30 minutes or less.
- . Post-traumatic amnesia for less than 24 hours.
- . Other transient neurological abnormalities such as focal signs, seizure, intracranial lesion not requiring surgery.

B) Glasgow Coma Scale score of 13–15 after 30 minutes post-injury or later upon presentation for healthcare.

C) These manifestations of MTBI must not be:

- . Due to drugs, alcohol, medication.
- . Caused by other injuries or treatment for other injuries (e.g., systemic injuries, facial injuries, or intubation).
- . Caused by other problems (e.g., psychological trauma, language barrier, or coexisting medical conditions).
- . Caused by penetrating craniocerebral injury.

Centers for disease control (CDC) conceptual definition of MTBI: A case of MTBI is an occurrence of injury to the head resulting from blunt trauma or acceleration or deceleration forces with one or more of the following conditions attributable to the head injury during the surveillance period:

- . Any period of observed or self-reported transient confusion, disorientation, or impaired consciousness.
 - . Any period of observed or self-reported dysfunction of memory (amnesia) around the time of injury.
 - . Observed signs of other neurological or neuropsychological dysfunction, such as:
 - Seizures acutely following head injury;
 - Among infants and very young children: irritability, lethargy, or vomiting following head injury;
 - Symptoms among older children and adults such as headache, dizziness, irritability, fatigue, or poor concentration, when identified soon after injury, can be used to support the diagnosis of mild TBI, but cannot be used to make the diagnosis in the absence of loss of consciousness or altered consciousness. Further research may provide additional guidance in this area.
 - . Any period of observed or self-reported loss of consciousness lasting 30 minutes or less.
- More severe brain injuries were excluded from the definition of MTBI and include one or more of the following conditions attributable to the injury:
- . Loss of consciousness lasting longer than 30 minutes.
 - . Post-traumatic amnesia lasting longer than 24 hours.
 - . Penetrating craniocerebral injury.

American Congress of Rehabilitation Medicine (ACRM) states that a patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

1. Any period of loss of consciousness.
2. Any loss of memory for events immediately before or after the accident.
3. Any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, confused).
4. Focal neurological deficit(s) that may or may not be transient.

But where the severity of the injury does not exceed the following:

1. Loss of consciousness (LOC) of 30 minutes.
2. After 30 minutes, an initial Glasgow Coma Scale (GCS) score of 13–15.
3. Post-traumatic amnesia (PTA) not greater than 24 hours.

Reference McCrae et al. 2008

American Academy of Neurology (AAN) defines a concussion as a trauma induced alteration in mental status that may or may not involve loss of consciousness. Confusion and amnesia are the hallmark of concussion. Frequently observed features of concussion are listed below:

- Vacant stare (befuddled facial expression)
- Delayed verbal and motor responses (slow to answer questions or follow instructions)
- Confusion and inability to focus attention (easily distracted and unable to follow through with normal activities)
- Disorientation (walking in the wrong direction, unaware of time, date, and place)
- Slurred or incoherent speech (making disjointed or incomprehensible statements)
- Gross observable incoordination (stumbling, inability to walk tandem/straight line)
- Emotions out of proportion to circumstances (distraught, crying for no apparent reason)
- Memory deficits (exhibited by the athlete repeatedly asking the same question that has already been answered, or inability to memorize and recall 3 of 3 words or 3 of 3 objects in 5 minutes)
- Any period of loss of consciousness (paralytic coma, unresponsiveness to arousal)

AAN practice parameter presents the following grading scale arrived at by a consensus of experts who reviewed all existing scales, including the recommendations in the Colorado Medical Society Guidelines.

Grade 1

1. Transient confusion
2. No loss of consciousness
3. Concussion symptoms or mental status abnormalities on examination resolve in *less* than 15 minutes.

Grade 1 concussion is the most common yet the most difficult form to recognize. The athlete is not rendered unconscious and suffers only momentary confusion (e.g., inattention, poor concentration, inability to process information or sequence tasks) or mental status alterations. Players commonly refer to this state as having been "dinged" or having their "bell rung."

Grade 2

1. Transient confusion
2. No loss of consciousness
3. Concussion symptoms or mental status abnormalities on examination last *more* than 15 minutes

Grade 3

1. Any loss of consciousness, either brief (seconds) or prolonged (minutes)

*The above is taken from the American Academy of Neurology. Practice parameters: the management of concussion in sports (summary statement)

APPENDIX C

DEMOGRAPHICS CHARACTERISTICS OF THE STUDY GROUPS, PERFORMANCE ON
NEUROPSYCHOLOGICAL TESTS BY STUDY GROUPS, PERFORMANCE ON
BEHAVIORAL MEASURES BY STUDY GROUPS, AND DEPENDENT VARIABLES
FROM SUPPLEMENTARY ANALYSIS

Table C.1

Demographics Characteristics of the Study Groups

Variable	MTBI	PTSD	Control
	N= 21	N= 22	N= 20
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Age	23.67 (3.01)	25.10 (2.51)	22.80 (3.37)
Years of Education	12.05 (.60)	12.32(.84)	12.30 (1.10)
Years of Service	3.83 (2.30)	5.05 (1.95)	3.45 (2.44)
Pay Grade	4.10 (1.14)	4.45 (.80)	3.75 (1.25)
Number of Deployments	1.62 (1.02)	1.91 (1.15)	1.35 (.81)
Time post concussion to assessment (months)	6.60 (2.51)	n/a	n/a
	Percent	Percent	Percent
Male	100	100	100
Combat Arms MOS			
Armor	47.6	40.9	10.0
Cavalry Scout	23.8	13.6	55.0
Infantry	28.6	45.5	35.0
Caucasian	71.4	81.8	75.0
History of Concussion	47.6	50.0	50.0

Table C.2

Performance on Neuropsychological Tests by Study Groups

Neuropsychological tests by domain	MTBI N= 21 <i>M (SD)</i>	PTSD N= 22 <i>M (SD)</i>	Control N= 20 <i>M (SD)</i>
Memory			
ANAM Math Processing	20.70 (5.05)	19.22 (7.17)	23.76 (7.14)
ANAM Matching to Sample	34.51 (12.19)	29.39 (11.45)	38.00 (9.00)
ANAM Code Substitution-Delay	46.88 (18.71)	40.52 (13.72)	43.46 (16.72)
MicroCog Memory	103.48 (11.80)	96.14 (17.81)	109.55 (8.17)
MicroCog Reasoning/Calculation	98.90 (12.75)	91.64 (15.48)	103.20 (12.57)
MicroCog Spatial Processing	96.24 (16.15)	91.55 (17.42)	106.40 (14.80)
Attention			
ANAM Code Substitution-Learning	57.02 (11.83)	47.39 (12.99)	56.88 (10.24)
MicroCog Attention/Mental Control	101.95 (10.36)	89.73 (16.48)	104.50 (8.54)
Psychomotor Speed			
ANAM Simple Reaction	212.65 (32.55)	175.02 (59.52)	226.83 (20.74)
ANAM Simple Reaction (R)	217.49 (39.45)	178.25 (53.83)	215.94 (26.49)
ANAM Procedural Reaction	96.51 (12.72)	79.62 (23.42)	98.42 (12.49)
MicroCog Reaction Time	99.38 (10.90)	88.23 (18.96)	103.70 (6.28)

Table C.3

Performance on Behavioral Measures by Study Groups

Subjective measure	MTBI N = 21 <i>M (SD)</i>	PTSD N = 22 <i>M (SD)</i>	Control N = 20 <i>M (SD)</i>
CES-D	13.33 (9.58)	28.63 (11.29)	13.55 (12.74)
BAI	10.67 (8.05)	22.77 (11.71)	6.10 (7.69)
ESS	8.24 (4.52)	10.00 (5.29)	8.45 (4.27)
AUDIT	5.98 (5.45)	6.50 (6.47)	7.60 (6.29)
DAST-10	.43 (.87)	.32 (.89)	.30 (.73)

Table C.4

Dependent Variables from Supplementary Analysis

Variable	MTBI N = 21 <i>M (SD)</i>	PTSD N = 22 <i>M (SD)</i>	Control N = 20 <i>M (SD)</i>
GCF	96.29 (10.88)	87.64 (15.49)	105.20 (8.49)
GCP	96.43 (10.54)	87.91 (14.97)	104.30 (9.91)
IPA	88.21 (15.97)	78.95 (16.93)	96.30 (11.87)

APPENDIX D

WILK'S LAMBDA AND CANONICAL CORRELATION AND STANDARDIZED
DISCRIMINANT FUNCTION AND STRUCTURE COEFFICIENTS FOR ALL GROUPS
AND MTBI AND PTSD GROUPS

Table D.1

Wilk's Lambda and Canonical Correlation for All Groups per Domain

Psychomotor speed

Function	Wilk's Lambda	x^2	df	p	R_c	R_c^2
1-2	.70	20.99	8	.007	.52	.27
2	.96	2.64	3	.45	.21	.04

Attention

Function	Wilk's Lambda	x^2	df	p	R_c	R_c^2
1-2	.75	16.95	4	.002	.49	.24
2	.99	.29	1	.59	.07	.005

Memory

Function	Wilk's Lambda	x^2	df	p	R_c	R_c^2
1-2	.72	18.82	12	.09	.50	.25
2	.04	2.40	5	.79	.20	.04

Table D.2

Standardized Discriminant Function and Structure Coefficients for All Groups

Psychomotor speed

Variable	Coefficient	r_s	r_s^2
Function 1			
Reaction time	.39	.83	.69
Simple reaction time	.41	.89	.79
Simple reaction time (repeat)	-.06	.72	.52
Procedural reaction time	.44	.83	.69
Function 2			
Reaction time	-.49	-.13	.02
Simple reaction time	-1.01	-.12	.01
Simple reaction time (repeat)	1.34	.52	.27
Procedural reaction time	.42	.30	.09

Attention

Variable	Coefficient	r_s	r_s^2
Function 1			
Attention	.79	.94	.88
Code-Substitution-Learning	.36	.69	.48
Function 2			
Attention	-.76	-.33	.11
Code-Substitution-Learning	1.04	.72	.52

Memory

Variable	Coefficient	r_s	r_s^2
Function 1			
Reasoning/Calculation	.39	.60	.36
Memory	.21	.72	.52
Spatial memory	.52	.67	.45
Code Substitution- Delayed	-.27	.11	.01
Math processing	.18	.51	.26
Match to Sample	.36	.56	.31
Function 2			
Reasoning/Calculation	.37	.41	.17
Memory	.31	.30	.09
Spatial memory	-.61	-.22	.05
Code Substitution- Delayed	-.69	.73	.53
Math processing	-.41	-.15	.02
Match to Sample	.17	.32	.10

Table D.3

Wilk's Lambda and Canonical Correlation for MTBI and PTSD Groups per Domain

Psychomotor speed

Function	Wilk's Lambda	X^2	df	p	R_c	R_c^2
1	.80	8.58	4	.07	.44	.19

Attention

Function	Wilk's Lambda	X^2	df	p	R_c	R_c^2
1	.80	8.91	2	.01	.45	.20

Memory

Function	Wilk's Lambda	X^2	df	p	R_c	R_c^2
1	.90	4.07	6	.67	.32	.10

Table D.4

Standardized Discriminant Function and Structure Coefficients for MTBI and PTSD Groups

Psychomotor speed

Variable	Coefficient	r_s	r_s^2
Function 1			
Reaction time	.04	.74	.55
Simple reaction time	.07	.80	.64
Simple reaction time (repeat)	.42	.86	.74
Procedural reaction time	.60	.92	.85

Attention

Variable	Coefficient	r_s	r_s^2
Function 1			
Attention	.69	.91	.83
Code Substitution-Learning	.48	.79	.62

Memory

Variable	Coefficient	r_s	r_s^2
Function 1			
Reasoning/Calculation	.54	.78	.61
Memory	.24	.74	.55
Spatial memory	.11	.43	.18
Code Substitution- Delayed	.26	.59	.35
Math processing	-.06	.36	.13
Match to Sample	.34	.66	.44

APPENDIX E

SUMMARY OF HYPOTHESES IN ASSOCIATION WITH ALL ANALYSES

Hypotheses	Type of Analysis			Conclusion
	All group DFA	MTBI/PTSD DFA	Univariate	
MTBI participants will score lower on variables of attention than participants in the PTSD group and control group.	PTSD differs from MTBI and Control; MTBI = Control	PTSD differs from MTBI	both variables indicate PTSD differs from MTBI and Control. PTSD performed the worst followed by MTBI then Control PTSD < Control=MTBI	Not supported at univariate or multivariate level
There is not a statistically significant different between groups on measures of psychomotor speed.	PTSD differs from MTBI and Control; MTBI = Control	PTSD = MTBI	<i>Parametric:</i> PTSD differs from MTBI and Control (PTSD performed the worst followed by MTBI then control). <i>Nonparametric:</i> PTSD differs from control on MicroCog Reaction Time and ANAM Simple Reaction Time. MTBI differs from PTSD on ANAM Simple Reaction Time (R) PTSD performed worst followed by MTBI then control on Simple Reaction Time and Reaction Time. PTSD performed worst followed by control then MTBI on Simple Reaction Time (R)	<i>Supported</i> with DFA comparing MTBI and PTSD. <i>Not supported</i> with all groups DFA or univariate level.

<p>PTSD participants will score lower on variables of memory than MTBI participants.</p>	<p>PTSD = MTBI = Control</p>	<p>PTSD = MTBI</p>	<p>MTBI = PTSD on all variables</p> <p>On Code Substitution-Delay PTSD performed worse followed by control then MTBI.</p> <p>All other indices: PTSD performed worst followed by MTBI then control.</p>	<p>Not supported</p>
<p>PTSD participants will score lower on variables of memory than controls.</p>	<p>PTSD = MTBI = Control</p>	<p>N/A</p>	<p>PTSD differed from Control on 5 of 6 variables.</p> <p>On the 5 variables: PTSD performed worst followed by MTBI, then control.</p> <p>Code Substitution-Delay: No difference. PTSD performed worst followed by control then MTBI.</p>	<p>Supported at univariate level by 5 of 6 variables.</p> <p>Not supported at multivariate level</p>

APPENDIX F
SUMMARY OF NONPARAMETRIC TEST RESULTS

Variable	<i>Kruskal-Wallis</i>		<u>Mean Ranks</u>		
	<i>(H)</i>	<i>p</i>	MTBI	PTSD	Control
Simple Reaction Time	9.49	.009	34.74	22.64	39.43
Simple Reaction Time (Repeat)	11.20	.004	39.31	21.64	35.73
Reaction Time	9.05	.01	33.55	23.23	40.03
CES-D	19.29	.000	25.26	45.82	23.88
BAI	22.02	.000	29.33	45.77	19.65

	<u>K-S(Z)</u>	<u><i>p</i></u>
MTBI v. PTSD		
Simple Reaction Time	1.19	.12
Simple Reaction Time (Repeat)	1.90	.001
Reaction Time	1.31	.07
CES-D	1.93	.001
BAI	1.63	.01
PTSD v. Control		
Simple Reaction Time	1.74	.005
Simple Reaction Time (Repeat)	1.34	.06
Reaction Time	1.56	.02
CES-D	1.82	.003
BAI	2.13	.000
MTBI v. Control		
Simple Reaction Time	.75	.62
Simple Reaction Time (Repeat)	.83	.50
Reaction Time	.68	.75
CES-D	.71	.70
BAI	1.31	.06

APPENDIX G

ABBREVIATIONS AND QUESTIONNAIRE

Table G.1

Abbreviations

AAN = American Academy of Neurology
ACRM = American Academy of Rehabilitation Medicine
ANAM = Automated Neuropsychological Assessment Metrics
BAI = Beck Anxiety Inventory
CDC = Center for Disease Control
CES-D = Center for Epidemiologic Studies Depression Scale
CRDAMC = Carl R. Darnall Army Medical Center
DTI = Diffuse Tensor Imaging
DVBIC = Defense Veterans Brain Injury Center
fMRI = functional Magnetic Resonance Imaging
GCS = Glasgow Coma Scale Score
HMMWV = High Mobility Multipurpose Wheeled Vehicle
IED = Improved Explosive Device
LOC = Loss of Consciousness
MEB = Medical Evaluation Board
MRAP = Mine Resistant Ambush Protected armored vehicle
MRI = Magnetic Resonance Imaging
MTBI = Mild Traumatic Brain Injury
NAN = National Academy Neuropsychology
PCS = Postconcussional syndrome
PDHRA = Post Deployment Health and Risk Assessment
PTA = Posttraumatic Amnesia
PTSD = Posttraumatic Stress Disorder
TBI = Traumatic Brain Injury
qEEG = Quantitative electroencephalogram
WHO = World Health Organization

Table G.2

Questionnaire

QUESTIONNAIRE

- 1) Diagnosis (circle one): MTBI PTSD none Other: _____
- 2) Age: _____
- 3) Ethnicity (Circle one) Caucasian, African-American, Hispanic, Asian-American, Pacific Islander, Bi-racial, other
- 4) Gender (circle one): Male Female
- 5) Rank: E-____, O-____, W____
- 6) MOS (circle one): Infantry Armor Cavalry Scout Other
- 7) Years of education: _____
- 8) High school and/or college GPA: _____
- 9) ASVAB score(s): _____
- 10) What month and year did you take the ASVAB? _____
- 11) GT score(s): _____
- 12) How many years of service do you have? _____
- 13) Number of deployments (circle one): 1 2 3 4 5 more than 5
- 14) How long have you been back since your last deployment? _____
- 15) (*MTBI personnel only*) How long has it been since your last concussion? _____
- 16) How long has it been since your last combat exposure? _____

17) Indicate the number of times you have been involved in each of the following types of events/situations and **NOT** sustained an injury: (Ex. in a convoy where an IED went off and you did not get injured)

- a. Fragment _____
- b. Vehicular _____
- c. Blast _____
- d. Fall _____

18) Indicate the number of times you have sustained each of the following types of injuries: (Ex. injured by a potato masher, HMMWV/MRAP rollover, some type of IED)

- a. Fragment _____
- b. Vehicular _____
- c. Blast _____
- d. Fall _____

19) If you indicated you were injured by an event in question above: Was there a loss of consciousness or being dazed following event (circle one): Yes No

- a. If yes, how long (circle one): 5 min or less 6-10 min. Over 20 min.

20) Prior history of concussion (for example, getting ~~knocked out~~ as a child, or ~~getting your bell rung~~ in high school football):

- Never
- Once
- Two times or more

21) At the platoon level and for all deployments, how many of your fellow Soldiers were wounded? _____

22) At the platoon level and for all deployments, how many of your fellow Soldiers died? _____

REFERENCE LIST

- Allen J., Litten R., Fertig J., & Babor T. (1997). A review of research on the Alcohol Use Disorders Identification Test (AUDIT). *Alcoholism: Clinical and Experimental Research*, 21, 613-619.
- American Academy of Neurology (1997). Practice parameter the management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee. *Neurology*, 48(3), 581-585. http://www.aan.com/professionals/practice/guidelines/pda/Concussion_sports.pdf.
- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders*, Fourth Edition, Text Revision. Washington, DC, American Psychiatric Association.
- Anderson, M. & Levy, B. (2009). Suppressing unwanted memories. *Current Direction in Psychological Science*, 18(4), 189-194.
- Andreasen, N., Flaum, M., Swayze, V., O'Leary, D., Alliger, R., Cohen, G., Ehrhardt, J., & Yuh, W. (1993). Intelligence and brain structure in normal individuals. *American Journal of Psychiatry*, 150(1), 130-134.
- Arciniegas, D., Anderson, C., Topkoff, J., & McAllister, T. (2005). Mild traumatic brain injury: A neuropsychiatric approach to diagnosis, evaluation, and treatment. *Neuropsychiatric Disease and Treatment*, 1(4), 311-327.
- Babor, T., de la Fuente, J., Saunders, J., Grant, M. (1992). AUDIT The Alcohol Use Disorders Identification Test: Guidelines for use in primary health care. Geneva. World Health Organization.

- Belanger, H., Spiegel, E., & Vanderploeg, R. (2010). Neuropsychological performance following a history of multiple self-reported concussions: A meta-analysis. *Journal of the International Neuropsychological Society, 16*(2), 262-267.
- Balestreri, M., Czosnyka, M., Chatfield, D., Steiner, L., Schmidt, E., Smielewski, P., Matta, B., & Pickard, J. (2004). Predictive value of Glasgow Coma Scale after brain trauma: Change in trend over the past ten years. *Journal of Neurology, Neurosurgery, and Psychiatry, 75*, 161-162.
- Barrett, D., Green, M., Morris, R., Giles, W. & Croft, J. (1996). Cognitive functioning and posttraumatic stress disorder. *American Journal of Psychiatry, 153*, 1492-1494.
- Bazarian, J., Wong, T., Harris, M., Leahey, N., Mookerjee, S., & Dombovy, M. (1999). Epidemiology and predictors of postconcussive syndrome after minor head injury in an emergency population. *Brain Injury, 12*, 173-189.
- Beck, A. & Steer, R. (1988). *Beck Anxiety Inventory manual*. San Antonio, TX: The Psychological Corporation.
- Belanger, H., Kretzmer, T., Yoash-Gantz, R., Pickett, T., & Tupler, L (2009). Cognitive sequelae of blast-related versus other mechanisms of brain trauma. *Journal of the International Neuropsychological Society, 15*, 1-8.
- Belanger, H., & Vanderploeg, R. (2005). The neuropsychological impact of sports-related concussion: A meta-analysis. *Journal of the International Neuropsychological Society, 11*(4), 345-357.
- Belanger, H., Vanderploeg, R., Curtiss, G., & Warden, D. (2007). Recent neuroimaging techniques in mild traumatic brain injury. *Journal of Neuropsychiatry Clinical Neuroscience, 19*(1), 5-20.

- Binder, L., Rohling, M., & Larrabee, G. (1997). A review of mild head trauma: I. Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, *19*(3), 421-431.
- Bleiberg, J., Cernich, A., Cameron, K., Sun, W., Peck, K., Ecklund, J., Reeves, D., Uhorchak, J., Sparling, M., & Warden, D. (2004). Duration of cognitive impairment following sports concussion. *Neurosurgery*, *54*(5), 1073-78.
- Bleiberg, J., Kane, R., Reeves, D., Garmoe, W., & Halpern, E. (2000). Factor analysis of computerized and traditional tests used in mild traumatic brain injury research. *The Clinical Neuropsychologist*, *14*, 287-294.
- Boals, A & Schuettler, D. (2008). PTSD symptoms in response to traumatic and non-traumatic events: The role of respondent perception and A2 criterion. *Journal of Anxiety Disorders*, *22*, 458-462.
- Boutin-Foste, C. (2008). An item-level analysis of the Center for Epidemiologic Studies Depression Scale (CES-D) by race and ethnicity in patients with coronary artery disease. *International Journal of Geriatric Psychiatry*, *23*(10), 1034-1039.
- Bremner, J., Randall, P., Scott, T., Bronen, R., Seibyl, J., southwick, S., Delany, R., McCarthy, G., Charney, D., & Innis, R. (1995). MRI based measurement of hippocampal volume in patients with combat related posttraumatic stress disorder. *American Journal of Psychiatry*, *152*, 973-981.
- Brenner, L., Terrio, H., Homaifar, B., Gutierrez, P., Staves, P., Harwood, J., Reeves, D., Adler, L., Ivins, B., Helmick, K., & Warden, D. (2010). Neuropsychological test performance in soldiers with blast-related mild TBI. *Neuropsychology*, *24*(2), 160-167.

- Brewin, C., Andrew, B., & Rose, S. (2000). Fear, helplessness, and horror in posttraumatic stress disorder: Investigating DSM-IV criterion A2 in victims of violent crimes. *Journal of Traumatic Stress, 13*(3), 499-509.
- Brudzynski S., Kadishevitz, L., & Fu, X. (1998). Mesolimbic component of the ascending cholinergic pathways: electrophysiological-pharmacological study. *Journal of Neurophysiology, 79*(4), 1675-1686.
- Bryant, R. & Harvey, A. (1995). Acute stress response: A comparison of head injured and non-head injured patients. *Psychological Medicine, 25*, 869-874.
- Carroll, L., Cassidy, J., Holm, L., Kraus, J., & Coronado, V. (2004). Methodological issues and research recommendations for mild traumatic brain injury: The WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 43*, 113-125.
- Charney, D., Deutch, A., Krystal, J., Southwick, S., & Davis, M. (1993). Psychobiological mechanisms of posttraumatic stress disorder. *Archive of General Psychiatry, 50*, 294-305.
- C - SHOP (2007). *ANAM4 TBI: User manual*. Norman, OK. Center for the Study of Human Operator Performance.
- Cocco, K., & Carey, K. (1998). Psychometric properties of the Drug Abuse Screening Test in psychiatric outpatients. *Psychological Assessment, 10*, 408-414.
- Collins, M., Field, M., Lovell, M., Iverson, G., Johnston, K., Maroon, J. & Fu, F. (2003). Relationship between post concussion headache and neuropsychological test performance in high school athletes. *American Journal of Sports Medicine, 31*, 168-173.

- Crowell, T., Kieffer, K., Siders, C., & Vanderploeg, R. (2002). Neuropsychological findings in combat-related posttraumatic stress disorder. *The Clinical Neuropsychologist, 16*(3), 310-321.
- Dalton, J., Pederson, S., & Ryan, J. (1989). Effects of posttraumatic stress disorder on neuropsychological test performance. *International Journal of Clinical Neuropsychology, 11*, 121-124.
- Dawe, S., Seinen, A., & Kavanagh, D. (2000). An examination of the utility of the AUDIT in people with schizophrenia. *Journal of Studies on Alcohol, 61*, 744–750.
- Elbert, T; Schauer, M. (2002). Psychological trauma: burnt into memory. *Nature, 419*:883.
- Evans, R. (1992). Some observations on whiplash injuries. *Neurology Clinics, 10*, 975-997.
- Fields, A. (2005). *Discovering Statistics Using SPSS*. Thousand Oaks: Sage.
- Flanagan, S. Cantor, J. & Ashman, T. (2008). Traumatic brain injury: future assessment tools and treatment prospects. *Neuropsychiatric Disease and Treatment, 4*(5), 877-892.
- Frencham, K., Fox, A., & Mayberry, M. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology, 27*, 334–351.
- Frueh, B.C., Gold, P.B., & de Arellano, M.A. (1997). Symptom overreporting in combat veterans evaluated for PTSD: Differentiation on the basis of compensation seeking status. *Journal of Personality Assessment, 68*, 369-384.
- Germain, A., Buysse, D., & Nofzinger, E. (2008). Sleep-specific Mechanisms Underlying Posttraumatic Stress Disorder: Integrative Review and Neurobiological Hypotheses. *Sleep Medicine Review, 12*(3), 185–195.

- Gil, T., Calev, A., Greenberg, D., Kugelmass, S., & Lerer, B. (1990). Cognitive functioning in post-traumatic stress disorder. *Journal of Traumatic Stress, 3*, 29-45.
- Gilbert, M., Shenton, M., Ciszewski, A., Kasai, K., Lasko, N. Orr, S., & Pitman, R. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience, 5*, 1242-1247.
- Glaesser, J., Neuner, F., Lutgehetman, R., Schmidt, R., & Elbert, T. (2004). Posttraumatic stress disorder in patients with traumatic brain injury. *Biomedical Central Psychiatry, 4*(5), 121-127.
- Gold, S., Marx, B., Soler-Baillo, J., & Sloan, D. (2005). Is life stress more traumatic than traumatic stress? *Journal of Anxiety Disorders, 19*, 687-698.
- Gurvits, T., Lasko, N., Schachter, S., Kuhne, A., Orr, S., & Pittman, R. (1993). Neurological status of Vietnam veterans with chronic posttraumatic stress disorder. *Journal of Neuropsychiatry and Clinical Neuroscience, 5*, 183-188.
- Gurvits, T., Shenton, M., Hokama, H., Ohta, H., Lasko, N., Gilbertson, M., Orr, S., Kikinis, R., Jolesz, F., McCarley, R., & Pitman, R. (1996). Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biological Psychiatry, 40*, 1091-1099.
- Harvey, A., Brewin, C., Jones, C., & Kopelman, M. (2003). Coexistence of posttraumatic stress disorder and traumatic brain injury: Towards a resolution of the paradox. *Journal of the International Neuropsychological Society, 9*, 663-676.
- Helmes, E., & Miler, M. (2006). A comparison of Microcog and the Wechsler Memory Scale (3rd ed.) in older adults. *Applied Neuropsychology, 13*(1), 28-33.

- Hickling, E., Gillen, R., Blanchard, E., Buckey, T. & Taylor, A. (1998). Traumatic brain injury and posttraumatic stress disorder. A preliminary investigation of neuropsychological test results in PTSD secondary to motor vehicle accidents. *Brain Injury, 12*, 265-274.
- Holm, L., Cassidy, J.D., Carroll, L., & Borg, J. (2005). Summary of the WHO collaborating centre for neurotrauma task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 37*, 137-141.
- Howell, D. (2007). *Statistical Methods for Psychology*. (6th Ed.). California: Thomsom Wadsworth.
- Huang, M., Theilmann, R., Robb, A., Angeles, A., Nichols, S, Drake, A. et al. (2009). Integrated Imaging Approach with MEG and DTI to Detect Mild Traumatic Brain Injury in Military and Civilian Patients. *Journal of Neurotrauma, 26*(8), 1213-1226.
- Hyman, I. & Loftus, E. (1998). Errors in autobiographical memory. *Clinical Psychology, 18*, 933-947.
- Iverson, G., Brooks, B., Collins, M. & Lovell, M. (2006). Tracking neuropsychological recovery following concussion in sports, *Brain Injury, 20*, 245–252.
- Iverson, G., Brooks, B., Lovell, M., & Collins, M. (2006). No cumulative effects for one or two previous concussions, *British Journal of Sports Medicine, 40*, 72–75.
- Iverson, G., Gaetz, M., Lovell, M., & Collins, M. (2004). Cumulative effects of concussion in amateur athletes, *Brain Injury, 18*, 101-112.
- Johns, M. (1991). A new method of measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep, 14*: 540–545.

- Jokic-Begic, N. & Begic, D. (2003). Quantitative electroencephalogram (qEEG) in combat veterans with post-traumatic stress disorder (PTSD). *Nordic Journal of Psychiatry*, 57(5), 351-355.
- Kabat, M., Kane, R., Jefferson, A., DiPino, R. (2001). Construct validity of selected automated neuropsychological assessment metrics (anam) battery measures. *Clinical Neuropsychologist*, 15, 498-507.
- Kane R.L., Roebuck-Spencer, T., Short, P., Kabat, M., & Wilken, J. (1997). Identifying and monitoring cognitive deficits in clinical populations using automated neuropsychological assessment metrics (ANAM) tests. *Archives of Clinical Neuropsychology*, 22(S1), S115-S126.
- Khateb, A., Ammann, J., Annoni, J., & Diserens, K. (2005). Cognition-enhancing effects of donepezil in traumatic brain injury. *European Neurology*, 54, 39–45.
- King, N. (1997). Post-traumatic stress disorder and head injury as a dual diagnosis: “Island” of memory as a mechanism. *Journal of Neurology, Neurosurgery, and Psychiatry*, 62, 82-84.
- Kirov, I, Fleysler, L., Babb, J., Silver, J., Grossman, R., & Gonen, O. (2007). Characterizing ‘mild’ in traumatic brain injury with proton MR spectroscopy in the thalamus: Initial finding. *Brain Injury*, 21(11), 1147-1154.
- Kobayashi, I., Boarts, J., & Delahanty, D. (2007). Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology*, 22, 660-669.
- Koenen, K., Harley, R., Lyon, M., Wolfe, J., Simpson, J., Goldberg, J., Eisen, S., & Tsuang, M. (2002). A twin registry study of familial and individual risk factors for trauma exposure and posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, 190, 209-218.

- Koren, D., Norman, D., Cohen, A., Berman, J., & Klein, E. (2005) Increased PTSD risk with combat-related injury: A match comparison study of injured and uninjured soldiers experiencing the same combat events. *American Journal of Psychiatry*, *162*, 276-282.
- Lanius, R., Williamson, P., Densmore, M., Boksman, K., Gupta, M., Neufeld, R., Gati, J., & Menon, R. (2001). Neural correlates of traumatic memories in posttraumatic stress disorder: A functional MRI investigation. *American Journal of Psychiatry*, *158*, 1920-1922.
- Leskin, L. & White, P. (2007). Attentional network reveal executive function deficits in posttraumatic stress disorder. *Neuropsychology*, *21*(3), 275-284.
- Levin, H., Amparo, E., Eisenberg, H., Williams, P., High, W., McArdle, C. & Weiner, R. (1987). Magnetic resonance imaging and computerized tomography in relation to the neurobehavioural sequale of mild and moderate head injury. *Journal of Neurosurgery*, *66*, 706-713.
- Levinson, D., & Reeves, D. (1997). Monitoring recovery from traumatic brain injury using automated neuropsychological assessment metrics (anam v10). *Archives of Clinical Neuropsychology*, *12*(2), 155–166.
- Linn, L. (1946). The Rorschach test in the evaluation of military personnel. *Rorschach Research Exchange*, *10*, 20-27.
- Lishman, W. (1988). Physiogenesis and psychogenesis in the “post-concussional syndrome.” *British Journal of Psychiatry*, *153*, 706-713.
- Lopez, S., Summerall, S., & Ryan, J. (2002). Factor structure of microcog in a clinical sample. *Applied Neuropsychology*, *(9)*3, 183-186.

- Lux, W. (2007). A neuropsychiatric perspective on traumatic brain injury. *Journal of Rehabilitation Research & Development*, 44(7), 951-962.
- Maisto S., Carey M., Carey K., Gleason J., & Gordon C. (2002). Use of the AUDIT and the DAST-10 to identify alcohol and drug use disorders among adults with a severe and persistent mental illness. *Psychological Assessment*, 12, 186-192.
- Mayou, R; Bryant, B; Duthie, R. (1993). Psychiatric consequences of road traffic accidents. *British Medical Journal*, 307, 647-651.
- McAllister, T. (2008). Neurobehavioral sequelae of traumatic brain injury: Evaluation and management. *World Psychiatry*, 7(3), 3-10.
- McCrae, M. (2008). *Mild traumatic brain injury and postconcussion syndrome: The evidence base for diagnosis and treatment*. New York: Oxford University Press.
- McHugh, P & Treisman, G. (2007). PTSD: A problematic diagnostic construct. *Journal of Anxiety Disorders*, 21, 192-200.
- McNally, R., & Shin, L. (1995). Association of intelligence with severity of posttraumatic stress disorder symptoms in vietnam combat veterans. *American Journal of Psychiatry*, 152, 936-938.
- Miller, L. (1998). Motor vehicle accidents: Clinical, neuropsychological and forensic considerations. *Journal of Cognitive Rehabilitation*, 16, 10-12.
- Morey, R., Petty, C., Cooper, D., LaBar, K., & McCarthy, G. (2008). Neural systems for executive and emotional processing are modulated by symptoms of posttraumatic stress disorder in iraq war veterans. *Psychiatric Clinics of North America*, 162(1), 59-72.
- Nuwer, M., Hovda, D., Schrader, L., & Vespa, P. (2005) Routine and quantitative EEG in mild traumatic brain injury. *Clinical Neurophysiology*, 116, 1-25.

- Ohry, A., Rattock, J., & Solomon, Z. (1996). Post-traumatic stress disorder in brain injury patients. *Brain Injury, 10*, 687-695.
- Ommaya, A. & Gennarelli, T. (1974). Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. *Brain, 97*(4), 633-654.
- Radley, J., Williams, B., & Sawchenko, P. (2008). Noradrenergic innervations of the dorsal medial prefrontal cortex modulates hypothalamo-pituitary-adrenal responses to acute emotional stress. *Journal of Neuroscience, 28*(22), 5806-5816,
- Radloff, L. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*(3), 385-401.
- Randolph, C. (2001). Implementation of neuropsychological testing models for the high school, collegiate, and professional sport settings. *Journal of Athletic Training, 36*(3), 288–296
- Rattock, J. & Ross, B. (1993). Post traumatic stress disorder in the traumatically head injured. *Journal of Clinical and Experimental Neuropsychology, 6*, 243.
- Raymond, P., Hinton-Bayre, A., Radel, M., & Ray, A. (2006). Test-retest norms and reliable change indices for the microcog battery in a healthy community population over 50 years of age. *The Clinical Neuropsychologist, 20*, 261-270.
- Reeves, D., Winter, K., Bleiberg, J. & Kane, R.(2007). ANAM genogram: Historical perspective, description, and current endeavor. *Archives of Clinical Neuropsychology, 22*S: S15-S37.
- Reynolds, M. & Brewin, C. (1998). Intrusive cognition, coping strategies and emotional responses in depression, posttraumatic stress disorder and a non-clinical population. *Behavior Research and Therapy, 36*, 135-148.

- Roebuck-Spencer, T., Sun, W., Cernich, A., & Farmer, K. (2007). Assessing change with the automated neuropsychological assessment (anam): issues and challenges. *Archives of Clinical Neuropsychology*, 22, S79-S87.
- Ruff, R., Iverson, G., Barth, J., Bush, S., Broshek, D., and the NAN Policy and Planning Committee. (2009). Recommendations for diagnosing mild traumatic brain injury: a national academy of neuropsychology education paper. *Archives of Clinical Neuropsychology*, 24, 3-10.
- Ruff, R., Levin, H., & Marshall, L. (1986). Neurobehavioral methods of assessment and the study of outcome in minor head injury. *The Journal of Head Trauma Rehabilitation*, 1(2), 43-53.
- Samuelson, K., Metzler, T., Rothlind, J., Choucroun, G., Neylan, T., Lenoci, M., Henn-Haase, C., Weiner, M., & Marmar, C. (2006). Neuropsychological functioning in posttraumatic stress disorder and alcohol abuse. *Neuropsychology*, 20(6), 716-726.
- Sbordone, R. & Liter, J. (1995). Mild traumatic brain injury does not produce post-traumatic stress disorder. *Brain Injury*, 9, 405-412.
- Schretlen, D., & Shapiro, A. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, 15, 341-349.
- Schwarzbold, M., Diaz, A., Martins, E., Rufino, A., Amante, L., Thais, M., Quevedo, J., Hohl, A., Linhares, M. & Walz, R. (2008). Psychiatric disorders and traumatic brain injury. *Neuropsychiatric Disease and Treatment*, 4(4), 797-816.
- Short, P., Cernich, A., Kabat, M., Wilken, J., & Kane, R. (2007). Initial construct validation of frequently employed anam measures through structural equation modeling. *Archives of Clinical Neuropsychology*, 22(S), S63-S77.

- Skinner, H. (1982). The Drug Abuse Screening Test. *Addictive Behaviors*, 7, 363–371.
- Spitzer, R., First, M., & Wakefie, J. (2007). Saving PTSD from itself in DSM-V. *Journal of Anxiety Disorders*, 21, 233–241.
- Stahl, M. (2008). *Stahl's essential psychopharmacology: neuroscientific basis and practical application* 3rd. ed. New York, Cambridge University Press.
- Stevens, J. (1996). *Applied multivariate statistics*. Mahway, NJ: Earlbaum
- Stuke, L., Diaz-Arrastia, R., Gentilello, L., & Shafi, S. (2007). Effect of alcohol on glasgow coma scale in head-injured patients. *Annals of Surgery*, 245(4), 651-655.
- Tabachnick, B. & Fidell, L. (2001). *Using multivariate statistics* (4th ed.). Boston: Allyn and Bacon.
- Thatcher, R., Biver, C., Gomez, J., North, D., Curtin, R. Walker, R., & Salazar, A. (2001). Estimation of the EEG power spectrum using MRI T2 relaxation time in traumatic brain injury. *Clinical Neurophysiology*, 112, 1729-1745.
- Thompson, J., Scott, K., & Dubinsky, L. (2008). Battlefield brain: Unexplained symptoms and blast-related mild traumatic brain injury. *Canadian Family Physician*, 54(11), 1549-1551.
- Thorton, K. & Carmody, D. (2009). Traumatic brain injury rehabilitation: QEEG biofeedback treatment protocols. *Applied Psychophysiology and Biofeedback*, 34, 59-68.
- Trudeau, D., Anderson, J., Hansen, L., Shagalov, D., Schmoller, J., Nugent, S., & Barton, S. (1998). Finding of mild traumatic brain injury in combat veterans with PTSD and a history of blast concussion. *Journal of Neuropsychiatry and Clinical Neuroscience*, 10, 308-313.
- Twamley, E., Hami, S., & Stein, M. (2004). Neuropsychological function in college students with and without posttraumatic stress disorder. *Psychiatry Research*, 126, 265-274.

- Vanderploeg, R., Curtiss, G., Belanger, H. (2005). Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 11*, 228-236.
- Vasterling, J., Brailey, J., Constans, J. Constans, K. & Sutker, P. (1998). Attention and Memory Dysfunction in Posttraumatic Stress Disorder, *Neuropsychology, 12*(1), 125-133.
- Vasterling, J., Duke, M., Brailey, K., Constans, J., Allain, A., & Sutker, P. (2002). Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. *Neuropsychology, 16*, 5–14.
- Vincent, A., Bleiberg, J., Yan, S., Ivins, B., Reeves, D. Schwab, K., et al. (2008). Reference data from the automated neuropsychological assessment metrics for use in traumatic brain injury in an active duty sample. *Military Medicine, 173*(9), 836-852.
- Warden, D., Gordon, B., McAllister, T., Silver, J., Barth, J., Bruns, J., Drake, A., Gentry, T., Jagoda, A., Katz, D., Kraus, J., Labbate, L., Ryan, L., Sparling, M., Waters, B., Whyte, J., Zapata, A., and Zitney, G. (2006). Guidelines for the pharmacological treatment of neuro behavioral sequelae of traumatic brain injury. *Journal of Neurotrauma, 23*(10), 1468-1501.
- World Health Organization. *International Statistical Classification of Diseases and Related Health Problems*, 10th Ed. (ICD-10), Geneva, Switzerland, 2007.
- Yehuda, R. (2001). Biology of posttraumatic stress disorder. *Journal of Clinical Psychiatry, 62*, 41-46.