MILD TRAUMATIC BRAIN INJURY

Vs.

POST TRAUMATIC STRESS DISORDER:

A DIFFERENTIAL DIAGNOSIS

By

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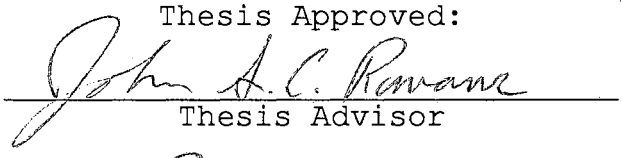

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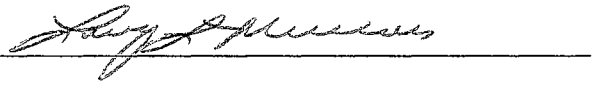

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This dissertation is dedicated to my mother, Lily Johnson, and my wife, Heather Johnson, two individuals who have had an irreversible, positive impact on my life.

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CHAPTER I

INTRODUCTION

Mental health professionals working within the medical field are frequently called upon to evaluate and diagnose individuals involved in various types of traumatic accidents (e.g. motor vehicle accidents, falls, sports related injuries, physical assaults, work accidents, etc.). Although many do not seek medical services, an estimated 7 million cases of head injury are thought to occur annually in the United States (Bennett & Raymond, 1997), with approximately 70% (Kraus & Nourjah, 1989) of these injuries accounted for by mild traumatic brain injury. Reports of the frequency of PTSD occurring in individuals involved in some type of accidental head injury vary from 10% (Mayou, Bryant, & Duthie, 1993), between 17% and 33% (Middelboe, Anderson, & Birket-Smith, 1991; Ohry, Rattock & Solomon, 1996; Rattock & Ross, 1993) to 39% (Blanchard, Hickling, & Taylor, 1995). Based on these numbers, a staggering 700,000 to 2,730,000 individuals a year may potentially present with the differential diagnostic dilemma of mild traumatic brain injury vs. post-traumatic stress disorder.

To tease out these constructs, clinicians must consider several variables in making a diagnosis. Numerous parallels have been drawn between the sequelae of mild traumatic brain injuries (MTBI) and post traumatic stress disorder (PTSD)(Hickling et al., 1998) which can lead to misdiagnoses, improper treatment recommendations, and ineffectual aftercare plans.

Purpose of Study

There is a long-standing controversy surrounding research of MTBI, reflecting in part, ambiguities in definition and variation in neurobehavioral assessment, as well as confusion between post-concussion symptoms (PCS) and cognitive weakness (Kibby & Long, 1996). More recently, the existence of PTSD occurring co-morbidly with MTBI has become a hotly contested issue (Bryant & Harvey, 1998). While some research contends that PTSD does not occur following MTBI (Sbordone & Liter, 1995), or at least very infrequently (Kay et al. 1992; Mayou, Bryant, & Duthie, 1993), other researchers claim PTSD can readily stem from MTBI (Middelboe, Anderson, & Birket-Smith, 1991; Ohry, Rattock & Solomon, 1996; Rattock & Ross, 1993).

Although limited in comparison to the investigation of more severe traumatic brain injury, several studies have researched the use of neuropsychological assessment in MTBI, as well as post-concussive symptoms. However, the neuropsychological assessment and comparison of PTSD in MTBI populations constitutes only a small collection of studies. Moreover, there is no known study that compares neuropsychological measures across both MTBI and PTSD populations, while taking into account time post injury. This study attempts to fill that gap in the literature.

Significance of Study

The significance of the current study lies in the patient's right to receive the most competent services available and overall quality of life issues. MTBI and PTSD stem from different etiologies and carry very different treatment recommendations. Misdiagnosis may potentially set the patient up for failure and can cause frustration, anxiety, depression, and loss of confidence in an already vulnerable individual. In addition, there is the potential to further refine assessment techniques within the field of neuropsychology by sorting out the "shades of gray" that differentiate MTBI from PTSD on cognitive measures.

Research Questions

The overall question addressed by this study is to what extent there is a significant difference in neuropsychological assessment of attention/concentration, processing speed, and memory between individuals diagnosed with MTBI vs. those diagnosed with PTSD. Should a significant differences arise, a secondary issue is to what extent a predictive equation can be formed, using the results of the neuropsychological measures, that would discriminate between these two diagnostic groups. The second question is the extent to which time post-injury differentially effects performance between and within the two groups on neuropsychological measures.

Assumptions: Assessment of MTBI and PTSD

The literature overwhelmingly cites cognitive deficits of memory, attention/concentration, and processing speed as the three primary cognitive sequelae following MTBI (Barth et al., 1983; Bohnen et al., 1992; Dikmen et al., 1986; Klonoff & Lamb, 1998; Leininger et al., 1990; Stuss et al., 1989; & Raskin et al., 1998). Similarly, memory,

attention/concentration, and processing speed difficulties are common complaints in most PTSD cases (Davidoff et al., 1988; Price, 1993; Sbordone & Liter, 1995; Vasterling et al., 1998; & Wolfe & Charney, 1991).

Specialized neuropsychological tests can serve as valuable measures in differentiating patient groups, as well as for assessing how certain types of information are selectively processed following trauma, providing quantitative and qualitative data on a number of high-level processing abilities (Wolfe & Charney, 1991). Neuropsychological testing has been documented to play an important role in the evaluation of mild traumatic brain injury (Cullum & Thompson, 1997; Leinigner et al., 1990; Harlage, 1997; Klonoff & Lamb, 1998; Raskin, Mateer, & Tweeten, 1998; Cicerone, 1997; Reitan & Wolfson, 1997; Trueblood & Schmidt, 1993), as well as post traumatic stress disorder (Everly & Horton, 1989; Hickling, Gillen, Blanchards et al., 1998; Vasterling et al. 1998; Wolfe & Charney, 1991).

Definitions of Related Terms

Mild head trauma and post-traumatic stress disorder have both been terms applied to brain injuries in which

there are little or no neurological findings present. Injuries of this type have evoked debate over the sequelae since the 1760's when the issue was first raised in the medical literature (Trimble, 1981, as cited in Davidoff et al., 1988). The differentiation possesses important implications for head trauma rehabilitation. The identification of mechanisms underlying deficient cognition is the first step in designing an effective rehabilitation treatment program (Davidoff et al., 1988).

Because of the traumatic cause of MTBI and the fact that many PTSD symptoms (such as sleep disorder, irritability, difficulty concentrating, etc.) overlap MTBI, it is not uncommon for clients to be given a diagnosis of PTSD after a MTBI in lieu of a diagnosis of brain injury (Kay et al., 1992). This "overlap" is demonstrated in Appendix A, which provides a physical, cognitive, and emotional/behavioral list of symptoms for both MTBI and PTSD.

Mild Traumatic Brain Injury

Traumatic brain injury (TBI) is a major health problem in the United States. Traumatic brain injury refers to a broad range of neurological, cognitive, and emotional

factors that result from the application of a mechanical force to the head. Mechanical force can be applied on a continuum from none to severe, with the extent of the brain injury related to the intensity at which this force is applied (Kibby & Long, 1996).

Head injury can be either open or closed. Open head injury indicates that the skull has been penetrated by a missile. Closed head injuries can be classified as acceleration or deceleration. Acceleration injuries involve the head being struck by a faster moving object, for example a flowerpot falling on the head. In deceleration injuries, the head is moving and hits a fixed and solid object, for example the head hitting the dashboard of a car (Price, 1993).

Mild traumatic brain injured individuals often present with a variety of physical, cognitive, and behavioral-affective symptoms. Physical symptoms may include headache, fatigue, sleep disturbance, dizziness, sensory changes, and various pains and discomforts. Cognitive complaints can include attention and concentration difficulties, memory difficulties, diminished ability to process, learn, and retain new information, and solve complex problems. Behavioral-affective signs often are irritability,

diminished frustration tolerance, social isolation, interpersonal difficulties, diminished confidence and self-esteem, and depression (Sbordone & Liter, 1995).

Most of the cognitive, emotional, and physical symptoms typically show substantial amelioration over time, with the majority of MTBI patients experiencing complete recovery within a few months after injury (Barth, Macciocchi, & Giordani, 1983; Binder, 1986; Levin et al., 1987, as cited in Cullum & Thompson, 1997). However, post-concussive syndrome (PCS) is a term reserved for patients who have persisting subjective symptomatology following cerebral concussion (Binder, 1986). Common symptoms that lead to a diagnosis of PCS include the following: memory problems, headache, insomnia, blurred vision, concentration problems, tinnitus, irritability, anxiety, depression, dizziness, fatigue, diplopia, photophobia, phonophobia, and confusion (Shaw, 1998).

Post Traumatic Stress Disorder

Post traumatic stress disorder (PTSD) is defined in the DSM IV as a pervasive pattern of symptoms persisting for at least a month following personal exposure to a traumatic event in which the person was confronted with

actual or threatened physical integrity, serious injury, or death to self or others. In response to the event the individual had intense fear, helplessness, or horror (DSM-IV, p.427-428).

An additional criteria of PTSD is that the event is reexperienced by frequent, recurrent distressing recollections of the event, nightmares, intense psychological and/or physiological distress at exposure to internal or external cues that are associated with the event, or a feeling as though the event was recurring via illusions, hallucinations, flashbacks, or a sense of actually reliving the experience. Individuals will also demonstrate a persistent avoidance of any stimuli associated with the trauma by avoiding thoughts, feelings, conversations concerning the trauma, avoiding activities and places that remind them of the trauma, inability to recall importance aspects of trauma, lack of interest and participation in important activities, detachment from others, lack of range of emotions, and a pessimistic future out-look for their lives. Other behavioral indications include initial insomnia, irritability, difficulty concentrating, hypervigilance, and exaggerated startle response (DSM-IV, p.427-428).

Patients with PTSD frequently report cognitive complaints of memory, learning, attention, and concentration difficulties; as well as deficits in planning, organization, and judgement (Wolfe & Charney, 1991). Additional affective-behavioral symptoms can include sleep disturbances, exaggerated startle response, hypervigilance, and irritability; as well as anhedonia, social isolation, and a sense of a foreshortened future (DSM-IV, 1994 p.428).

Shaw (1998) provides a summary chart, helpful in looking at MTBI, PTSD, and PCS on a continuum of injury based on neurological findings, cognitive deficits, and emotional/behavioral sequelae (Appendix B).

CHAPTER II

REVIEW OF THE LITERATURE

Introduction

An understanding of the dynamics involved in a diagnosis of MTBI and PTSD is essential in teasing out the subtle differences between the two terms. Seeing how other researchers have approached the dilemma, neuropsychologically and otherwise, gives a glimpse of where the current study could contribute to the existing knowledge base.

MTBI Theory Review

Diagnosing MTBI is a point of controversy, as definitions of what entails a MTBI has varied for years. However, in October of 1990 the Head Injury Interdisciplinary Special Interest Group of The American Academy of Physical Medicine and Rehabilitation developed a definition which the majority of the research is now referencing as the formal definition of MTBI. The academy noted that a person with mild brain injury is a person who

has traumatically induced physiological disruption of brain function as manifested by at least one (not necessarily all) of the following:

> 1. Any period of loss of consciousness up to approximately 30 minute duration. Within 30 minutes after the injury, the person must have progressed to a GCS (Glasgow Coma Scale) of 13 to 15 (of a possible 15).
>
> 2. Any loss of memory for events immediately before or after the accident (retrograde or anterograde amnesia).
>
> 3. Any alteration of mental state at the time of accident (e.g., feeling dazed, disoriented, or confused).
>
> 4. Focal neurological deficit that may or may not be transient (in the past, a mild head injury was ruled out if focal signs persisted).
>
> 5. Posttraumatic amnesia not greater than 24 hours.
>
> (Kibby & Long, 1996)

Kibby and Long raise three difficulties with the formal definition of MTBI. First, the mild and moderate brain injured populations are combined. Early research in the field noted that moderate TBI as post traumatic amnesia ranging from 1 to 24 hours (Russell, 1932). This overlap could cause some confusion when comparing past research findings to present day data (Kibby & Long, 1996).

Secondly, the definition covers a vast range of severity. Rutherford et al.(1979) showed that the

difference between a TBI causing post-traumatic amnesia of a few seconds and one a TBI causing post-traumatic amnesia of 30 minutes or 24 hours is quite substantial in the group's outcome performance.

Lastly, the lower limits permit over inclusiveness. The definition of MTBI does not require a loss of consciousness or transient amnesia, inferring that one can simply have a momentary alteration in mental state at the time of the accident. Any individual who sustains a head blow (even if it was very mild and there is little evidence implicating the brain) would be included in this definition. The definition does not necessarily imply structural damage to the brain, but rather simply an impact to the head (Kibby & Long, 1996).

Approximately 42% of all mild brain injuries are the result of a motor vehicle accident (MVA)(Kraus & Nourjah, 1989). Bennett and Raymond provide a useful summation of what happens to the brain when a TBI results from an MVA. Two things are important to understand. First, the brain is soft tissue with a gelatinous consistency. Secondly, the inside of the skull is very rough, particularly around above the eyes and in the temporal regions. Due to these rough bony contours the design of the skull is less than optimal for protecting the brain from sudden forces applied

to the head. Damage is produced when the brain is set into motion within the skull and rubs over and collides with rough surfaces. As the 3 ½ pound mass rotates unevenly it stretches and disconnects axons that normally conduct information within the brain. This is referred to as the shear-strain phenomenon which is the result of rapid brain movement within the cranial vault, causing stretching of white matter fibers and eventual degeneration. This damage is diffuse and can occur by rapid acceleration or deceleration, without ever striking the head (Bennett & Raymond, 1997). This type of white matter injury is often related to processing speed and/or attentional deficits.

Neuropsychological assessment measures are designed to assess the ramifications of neurological injury or disease. This is based on the belief that neurobiological damage manifests itself via cognitive, behavioral, or affective deficits. The psychobiology of minor head injury was researched by Montgomery et al. (1991). They used 26 consecutive admission to an accident and emergency unit for minor head injury. Each patient had a neurological examination, post-traumatic symptom checklist, EEG power spectra analysis, auditory brain stem-evoked potential recordings, and a four-choice reaction time measurement. These measures were administered again at six weeks and six

months. Post-traumatic symptoms were persistent in half of all patients at six months, almost half of the patients had abnormal brain stem conduction at six weeks, and head injured patients still had prolonged reaction times at six months compared to healthy normal participants. These findings reflect both cortical and brain stem damage following minor head injury, with the brain stem damage being more persistent (Montgomery, Fenton, McClelland, MacFlynn, & Rutherford, 1991).

Despite negative neurologic and neuroradiologic findings, a consistent pattern of subjective complaints by MTBI survivors is reported, with many of these complaints substantiated by formal neuropsychometrics findings. The three primary problem areas are fluctuations in attention and concentration, recent memory problems, and impaired processing speed (Bennett & Raymond, 1997).

Attention Review

One of the most widely used models of attention is by Posner and Rothbart (1992). In this model attention has three identified networks. The posterior attention network is involved in orienting to and locating sensory stimuli in space. The structures involved are the parietal cortex, pulvinar and reticular thalamic nuclei, and superior colliculus. The second is the anterior attention network

that appears to be responsible for the detection and selection target stimuli and inhibition of responses to irrelevant stimuli. The neural structures related to this network are the anterior cingulate gyrus and supplementary motor area. The vigilance network is the third identified attention network. It involves the locus coeruleus and brainstem reticular connections with cerebral cortex, predominantly lateral frontal cortex (Cicerone, 1997).

In 1991 a principle component analysis was conducted by Mirsky et al. on eight measures of attention with a mixed neuropsychological and normal control sample. A four factor solution emerged which reflected the abilities required to focus attention (Trail Making Test Part A and B), sustain attention (Continuous Performance Test), shift attention (Wisconsin Card Sorting Test), and numeric encoding (Digit Span and Arithmetic)(as cited in Cicerone, 1997).

Shum et al.(1990) likewise conducted a principal component analysis on eight tests of attention, in both a sample of normal control participants, and patients with a closed head injury. A three factor solution was arrived at which included measures of visual-motor scanning (Trail Making Test, Part A and B), sustained-selective attention

(Stroop Interference and serial 7 subtractions), and attention span (Digit Span)(as cited in Cicerone, 1997).

In Cicerone's own study (1997), he used Digit Span forwards and backwards, Trail Making Test Part A and B, Continuous Performance Test of Attention (CPTA), and Paced Auditory Serial Addition Test (PASAT). Fifty-seven MTBI subjects were used along with 40 control subjects from the community with no previous history of brain injury. Each group was administered the attention measures. Results indicated that neuropsychological measures of attention may be sensitive to the subtle cognitive dysfunction experienced after MTBI. There was a range of sensitivity with respect to the four measures, with the PASAT and CPTA being more sensitive than Digit Span or Trails. One explanation was that the measures assess different aspects of attention (Cecerone, 1997).

Memory Review

Memory begins with the registration of information in cortical input modules. Environmental events are picked up by the input modules, which are responsible for decoding and classifying the data at a perceptual, pre-semantic level. The output is relayed to the central system

structures, where meaning is assigned to the output (Squire, 1992).

Output is consciously apprehended by virtue of its being delivered to working memory where it is picked up by the hippocampal component. The hippocampal component binds the information it receives with the modules and central systems, and in turn gives rise to the conscious experience. This is process is referred to as a memory trace, and is encoded as file entry within the hippocampus. This whole process is referred to as memory consolidation (Squire, 1992). Conscious recollection of an event occurs when a cue (internal or external) gains access to working memory (consciousness), activates the hippocampal index, and interacts with a memory trace. The result of that interaction is then delivered back to working memory or consciousness for use (Squire, 1992).

Neuropsychological investigation of MTBI has consistently corroborated the subjective complaints of memory loss by patients. In a 1992 study, 11 experimental participants who had experienced an MTBI and 11 age matched samples with no history of MTBI were evaluated to determine if measures of qualitative aspects of memory functioning, such as utilization of semantic strategies might better reveal memory disturbances characteristic of the MTBI

population. A main effect of group membership (MTBI or Control) was found only when the delay was considered. The results indicated statistically significant effects for delayed recall, as MTBI group was found to perform significantly worse that the control group (Zappala & Trexler, 1992).

Kay et al. (1992) found decreased speed and ease of information processing to be the primary deficit after MTBI. Deficits in learning and memory reportedly appeared more related to the encoding and registration of new information than to a primary deficit in retrieval. Furthermore, it was proposed that measures of speed and attention such as Digit Symbol form the WAIS-R may be normal and that attentional deficits often appear only under conditions of complex attention.

Neuropsychological Assessment of MTBI

Effects of head injury on mental functions have usually been studied in patients with severe trauma. Studies on the consequences of mild injury are much more rare, in spite of the fact that these patients frequently complain of difficulties in concentration and memory and poor intellectual efficiency (Gentilini et al., 1985).

Rimal et al. (1981) analyzed data from 538 MTBI patients. In the study, MTBI was defined as loss of consciousness for less than 20 minutes, Glascow Coma Scale values ranging from 13 - 15, and length of hospitalization of less than 48 hours. Three months after the injury occurred, 424 patients were brought in for a follow-up assessment. Of these 424: 79% complained of persistent headaches, 59% had memory loss, and 34% had not resumed occupational activities.

In response to the finding by Rimal et al. One study, compared 50 mild brain injured individuals to 50 subjects. The purpose of the study was to evaluate neuropsychological deficits in a population of patients whose head injuries were defined as mild compared with those of a control group matched for age, educational level, and socioeconomic status with the MTBI group. Six measures to assess concentration, memory, and intelligence were used: Selective Attention Test, Digits Forward Test, Word Recognition, Buschke's Test, Working Memory Test, and Raven Progressive Matrices. Results indicate that the mean differences between the MTBI and control group were not significantly different. The authors concluded that in spite of a general trend toward lower performances, the MTBI group did not exhibit impairment, and if there was

structural damage following MTBI, patients generally experienced a recovery from a neuropsychological stand point within one month following the incident (Gentilini et al., 1985).

In a similarly designed study, Dikmen, McLean, and Temkin (1986) compared 20 MTBI subjects to 20 control group individuals at 1 and 12 months following injury. Both groups were administered an extensive neuropsychological battery as well as psychological measures. Results indicated that brain injured individuals performed worse at one month on their ability to remember newly acquired information after a delayed recall measure, concentration, and discrimination between rhythmic patterns. There was also a 7 second mean difference between the two groups on Trail Making Test Part B (MTBI 58 seconds vs. Control 51 seconds). At 12 months none of the psychological measures evidenced significant differences. The psychological measure used the Sickness Impact Scale (SIS). Scores are based on the subjects' perception of the extent to which their injuries and/or other health problems have affected their day to day functioning: sleep and rest, emotional behavior, body care, home management, mobility, social interaction, ambulation, alertness, communication, recreation, eating, and work. On every subscale, but

eating, the head injury group reported significantly greater health related difficulties than did the uninjured subjects. At one month few MTBI patients (4 out of 20) had resumed major roles (work, school, home management) or leisure activities. At 12 moths 15 of the 20 patients had resumed the major roles in their occupation.

Barth et al.(1983) attempted to document the relationship between cognitive, emotional, and behavioral sequelae associated with minor head trauma. The study randomly selected 70 patients for neuropsychological evaluation. In the sample, 97% had been rendered unconscious from various events including MVA, falls, and sports injuries. Results indicated that age, education, rapid visuomotor problem solving (Trails), and memory impairment (immediate and delayed deficits) seem highly correlated with cognitive functioning after minor head trauma. In addition, 32% of the patients referred for neuropsychological evaluation had at least one elevated (T 70) clinical scale on the Minnesota Multiphasic Personality Inventory (MMPI). Depression was significantly correlated with the Halstead Impairment Index ($p < .01$).

In another study, three neuropsychological tests were used in the analysis of traumatic brain injury. The Trail Making Test, Auditory Short Term Memory Test, and Paced

Auditory Serial-Addition Test(PASAT). Two groups were constructed. Group one consisted of 26 outpatients previously hospitalized with varying degrees of severity. Group two composed 22 non-hospitalized mildly concussed patients. Results indicated that both Trails Part A and Part B times were significantly slower than the control group. The short-term memory test revealed significant group differences with the brain injured group remembering less than controls, with impairment worsening with greater delay. The PASAT results paralleled the later two test results. (Stuss, Stethem, Hugenholtz, & Richard, 1989).

In a recent study by Raskin, Mateer, and Tweeten (1998) they assessed 148 subjects who met the criteria for MTBI with a comprehensive neuropsychological battery, including a measure of personality. Results indicated that MTBI subjects performed significantly worse than normative data on tests of time dependent attention (i.e. Symbol Digit Modalities Test, Trail Making Test part B), and tests of verbal memory (i.e. WMS-R Paired Associates, WMS-R Logical Memory). Demographic variables gender and age were related to cognitive performance; however, education, educational status, length of loss of consciousness, and time post injury were not related. Furthermore, performance

of cognitive neuropsychological measures were not related to any emotional or personality variable.

The primary interest of Leininger et al. (1990) was to explore whether injuries accompanied by a brief loss of consciousness result in poorer neuropsychological outcome than injuries not accompanied by a loss of consciousness. Analysis compared the neuropsychological performance of concussion and mild concussion patients and revealed no evidence that injuries associated with a brief traumatic loss of consciousness were more debilitating than injuries associated with dazing or confusion, but no formal loss of consciousness.

Postconcussive Syndrome Review

The etiology of PCS remains controversial with possible causes ranging from organic injuries to preexisting psychological problems. In addition, age, education, alcohol abuse, prior head injury, multiple trauma, psychological reactions to injury, and malingering have also been suggested causes of PCS (Klonoff & Lamb, 1998). Due to methodological and design issues the reported prevalence of PCS varies widely. Research findings suggest

that 23-90% of individuals with MTBI have PCS at one month post injury (Kibby & Long, 1996).

Researchers differ on many of the nuances associated with PCS involvement in MTBI. One camp views PCS as a separate diagnosis from MTBI. One particular researcher makes the connection by stating that in MTBI most of the cognitive, emotional, and physical symptoms typically show substantial amelioration with time, with most of the clients demonstrating almost complete recovery within a few months after injury. However, in patients with more enduring deficits, a diagnosis of postconcussive syndrome is often made (Evens, 1992).

Additional authors note that neither minor head injury nor mild traumatic brain injury should be equated with postconcussion syndrome, which is often used to signify dysfunction out of proportion to injury, regardless of whether there is brain damage or not (Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992). In support of this notion it has been noted that although PCS and MTBI share many of the same symptoms they present in different fashion. In PCS the symptoms are likely to worsen with time, severity of symptoms will be in excess of what would be expected, depression and anxiety are antecedent to cognitive or physical deficits, and the person's premorbid make-up is

important. The descriptors involved in defining PCS are somewhat in contrast to those presented by a definitive neurological injury, such as MTBI, in which case symptoms usually improve with time. Likewise, in MTBI the severity of symptoms are consistent with or less than expected, given the injury, and depression and anxiety are consequent to cognitive or physical deficits (Shaw, 1998).

The other camp views PCS as simply an extension of MTBI, or a way of describing the symptoms of MTBI. One researcher indicates that recovery from PCS is clearly the norm following MTBI, and most make a rather complete recovery within months; however, a small subgroup of individuals seem to experience more persistent symptoms (Cullum & Thompson, 1997). Similarly, Sbordone and Liter (1995)indicate that PCS is a term that people frequently ascribe to the complex list of symptoms that follow a minor or mild traumatic brain injury which he describes as a cerebral concussion produced by acceleration or deceleration forces acting on the brain to produce a period of confusion and/or amnesia.

One study tested the hypothesis that patients with postconcussive symptoms 6 months after mild head injury have cognitive dysfunction, as compared with matched, symptom free mild head injury patients and healthy controls

subjects. Patients with PCS (n=9), patients without PCS (n=9), and healthy controls (n=9) were administered the Auditory Verbal Learning Test (memory task), Stroop Color Word Interference Test (selective attention), and Computerized Divided Attention Test. Multivariate analysis yielded a significant group effect. In separate analysis, the verbal learning test failed to reach significance between groups. The Stroop test showed that the patients with PCS had significantly higher interference times than both control groups. The divided attention task revealed an overall significant group effect as patients with PCS were significantly slower than the both control groups (Bohnen, Jolles, & Twijnstra, 1992).

Couch (1995) conducted a review of the literature on post-trauma syndrome. He relates that post-trauma syndrome usually includes the following elements headache, dizziness, depression, increased irritability, impaired concentration, decreased ability to learn, personality change, intolerance to alcohol, decreased libido (Guttman, 1943; Brenner, Friedman, Merritt, & Denny-Brown, 1944, Miller, 1961). Speed (1989) expanded the list by including depression related symptoms, vestibular-related symptoms, impaired ability to think or concentrate, change in personality, and diminished libido.

Mittenberg designed at study to determine whether symptoms of mild cerebral trauma could be related to what patients believe to be the likely symptoms that occur after head injury. Results indicated that common expectations of postconcussive headache, anxiety, depression, concentration difficulty, vertigo, diplopia, confusion, irritability, fatigue, photophobia, and memory difficulties differed little from the observed incidence of these same symptoms in patients with head injuries. Furthermore, patients with head injuries consistently underestimated the normal prevalence of their symptoms in their retrospective accounts, compared to base rate reported by normal control subjects. This suggested that patients may reattribute benign emotional, physiological, and memory symptoms to their head injury. The researches concluded that if an imaginary concussion will reliably elicit expectations of a coherent cluster of symptoms virtually identical to PCS, than the expectation of symptomatology may share almost as much variance with the syndrome as head injury itself. "A causative role is suggested" (Mittenberg et al., 1992).

In support of this assertion, Dikmen, McLean, and Temkin (1986) studied 20 head-injured patients comparing them to a 20 member normal control group on percent of endorsement on the Head Injury Symptom Checklist (HISC).

Results indicated that on the symptoms listed: headaches, fatigue, dizziness, blurred vision, bothered by noise, bothered by light, insomnia, difficulty concentrating, irritability, loss of temper easily, memory difficulties, and anxiety; only "bothered by noise," insomnia, and memory difficulties were significantly different for the two groups one month post-injury.

Results of another study supported the findings of Mittenberg et al., as the uninjured, control group subjects, endorsed many of the same problems as the injured group. The authors note that significant disruption of psychosocial functions has been observed following head injury, especially during the first initial month. The reason for this disruption is not solely related to the head injury itself. Other system injuries, such as orthopaedic problems, lacerations, etc; which are often sustained as part of the same accident and appear to be somewhat responsible for the psychosocial disruption associated with MTBI (Dikmen, McLean, & Temkin, 1986).

With the numerous types of PCS complaints and the frequency rates ranging widely, the etiology of the PCS cluster symptoms are starting to be investigated. Recently, research investigating the relationship between stress, coping, and postconcussive symptoms was conducted. In the

first study, 179 undergraduates were administered the Symptom Rating Scale (SRS), Daily Stress Inventory (DSI), and Perceived Stress Scale (PSC). In the second study a separate sample of 55 undergraduate students completed the SRS, DSI, and PSC on two occasions 28 days apart. Results suggested that individual's perception of stress, independent of the frequency of the stressful events, is significantly related to the level of symptom complaint. No significant relationship was found between frequency of stressful event and level of symptom complaint. In addition, individual ratings of perceived stress were extremely stable over a one-month time interval, with perceived stress varying between individuals, but stable within individuals. The authors suggest that such individual differences, and the strong correlation between perceived stress and level of symptom complaint, would potentially explain the wide variance in individual's symptom complaints following MTBI (Machulda, Bergquist, Ito, and Chew, 1998).

PTSD Review

PTSD was first called "schreckneurose," coined by Emil Kraeplin (1886), regarded by some as the father of modern

psychiatric diagnosis. "Schreckneurose" referred to a condition, "composed of multiple nervous and psychiatric phenomena arising as a result of severe emotional upheaval or sudden fright, which would build up great anxiety. It can therefore be observed after serious accidents and injuries, particularly fires, railway derailments, or collisions" (as cited in Price, 1993).

Today PTSD is known as a psycho-biologic syndrome involving re-experiencing phenomena, avoidance behavior, and heightened autonomic responses following a severe stressor (Warden et al., 1997). Dikmem and Levin (1993) note that accidents and injuries, even mild, are traumatic and may be perceived at the time as life threatening, and may in fact, have involved serious injury or death to others. Recovery from an accident may be complicated by PTSD. The symptom cluster described as part of the disorder includes irritability, anger outbursts, difficulty sleeping, problems concentrating and is often associated with memory difficulties, emotional lability, physical symptoms (headaches and vertigo), depression, and anxiety. "These symptoms are strikingly similar to symptoms reported as a result of head injury."

The main objective of the Vasterling et al. (1998) study was to examine attention, learning, and memory

functions in a community-recruited sample of traumatized individuals. They selected the three areas of cognitive functioning (attention, learning, and memory) thought to be vulnerable to disruption after stress exposure, and would allow for a comparison of arousal-based theories with those that stress the role of the hippocampus. In regards to attention, the Mirsky et al.(1991) attention model was used: ability to sustain (Continuous Performance Test, Stroop Test Interference scores), ability to shift (Wisconsin Card Sorting Test), and ability to encode (Digit Span and Arithmetic). Learning and memory measures were assessed by the Rey Auditory Verbal Learning Test (AVLT) and the Continuous Visual Memory Test (CVMT). Results suggested the existence of impaired attention and mnemonic processes in PTSD. The PTSD group displayed deficits in sustained attention and mental manipulation of information, but not on measures of selective attention or flexibility in shifting attention. Memory measures yielded relative weaknesses for the PTSD groups ability to initially acquire information, but no differences in memory savings.

Everly and Horton (1989) conducted a pilot study using 14 patients diagnosed by DSM-III-R criteria as having PTSD. The study was specifically focussed upon symptoms of memory

dysfunction in PTSD patients. Patients were administered the Four-word Short-term Memory. Administration involves the examiner reading four unrelated words, followed by a three digit number. The patient counts backwards by 3's from the three digit number for an interval of 15 or 30 seconds. Results suggested possible neuropsychological impairment in this sample of PTSD patients, as 12 of 14 patients were impaired on one of the two criteria. For the 15 second interval 9 of 14 patients appeared impaired. For the 30 second interval 11 of 14 patients appeared impaired. The authors proposed this study as supporting evidence that the PTSD is a condition of limbic system hypersensitivity and instability.

MTBI and PTSD

Hickling et al. (1998) attempted to answer two questions linked to the diagnostic controversy of MTBI and PTSD. The study included 107 motor vehicle accident (MVA) victims. The first question was to determine whether MVA survivors who report a loss of consciousness as a result of their MVA (and therefore have no or limited recall of the traumatic event), actually have lower rates of PTSD than individuals with no loss of consciousness during the MVA.

The second question was to compare the neuropsychological functioning of MVA victims who meet diagnostic criteria for PTSD with those who do not meet criteria on a battery of cognitive tests sensitive to the effects of concussion. The results indicated that the MVA victims with the greatest evidence of having suffered a TBI (i.e. those who suffered a loss of consciousness), tended to perform most poorly on certain neuropsychological tasks (i.e. Stroop Color Word Test & Trail Making Test). Subjects with no reported injury to the head and subjects who lost consciousness displayed poorer delayed recall for verbal material on list learning material (Rey Auditory-Verbal Learning Test (RAVLT). In regards to the second question, PTSD diagnosed by CAPS (assessment measure based on 16 DSM-IV criteria) did occur in 40% of those injured severely enough to lose consciousness. However, the researchers note that one explanation is that due to the very short duration of unconsciousness (1-15 min) it is possible that enough frightening memories from both immediately prior to the impact and immediately after the impact existed to provide the development of PTSD. This is an example of sequential PTSD.

Fuller, Monna, David, and Sanderlin (1991) designed a study with the purpose of comparing the neuropsychological

functioning of chronic PTSD patients (combat veterans) with and without a history of TBI. The sample groups were divided based on reported history of TBI, defined as loss of consciousness greater than one hour. The neuropsychological battery included measures of general intelligence (Shipley), attention/concentration (Stroop, Trails), memory (WMS-R), and abstract reasoning (WAIS-R Similarities). Results indicated that both groups differ significantly from expected values based on previously published normative information.

Acute stress disorder (ASD) is used in the DSM-IV to describe posttraumatic stress in the initial month following a trauma. The criteria include avoidance, arousal symptoms, dissociative, and re-experiencing. ASD is believed to be a precursor to posttraumatic stress disorder (Koopman, Classen, & Spiegel, 1994). In a study designed to investigate the predictors of acute trauma response following MTBI, researchers used 48 adult MVA admissions to a major trauma hospital sustaining a MTBI. ASD was diagnosed in 7 of the patients (14.6%) and two patients (4.2%) met all but one criteria regarding them as a sub-syndromal diagnosis. The variables which accounted for the most variance in the prediction of acute stress severity were age, Beck Depression Inventory, avoidance coping,

neuroticism, Dissasociative Experiences Scale, psychiatric history, and history of PTSD (Harvey & Bryant, 1998).

One scale suitable for assessment with TBI and PTSD is a self-report scale based on the DSM-III-R criteria (PTSD Inventory). The inventory measures the intensity based on the number of symptoms of the syndrome and consists of 17 statements which correspond to the 17 PTSD symptoms listed in the DSM-III-R. Symptoms include four intrusive, seven avoidant, and six hyperarousal symptoms. To be diagnosed as PTSD, respondents have to endorse at least 1 intrusive, 3 avoidant, and 2 hyperarousal symptoms. The scale was found to have a high convergent validity when compared with diagnoses based on structured clinical interviews (Soloman, Bebenishty, Neria, et al., 1993 as cited in Ohry, Rattok, & Solomon, 1996).

Co-Morbidity Debate

The degree to which the conceptualization of MTBI and PTSD varies is testimony to the continuum of views that researchers have on the topic. This leads into the debate over the co-morbidity verses the incompatibility of MTBI and PTSD. Mental health professionals differ on whether PTSD and MTBI are mutually exclusive, or if those persons

who sustain MTBI or cerebral concussion can develop a concomitant post traumatic stress disorder. Part of the controversy stems from a good deal of variation in the definition and conceptualization of MTBI, PCS, and PTSD.

At the heart of the "mutually exclusive argument" (if a person has a MTBI then they can not have PTSD) is the idea that for one to have vivid and intrusive recollections of a trauma, one must be able to remember the trauma. The re-experiencing of the traumatic event is the central clinical feature of posttraumatic stress disorder. This may be manifested in recurrent disturbing dreams, which wake the individual with feelings of fright and terror, or through intrusive recollections of the event, which include vivid images of the traumatic situation (Mendelson, 1987). This poses a problem when the majority of MTBI's include retrograde amnesia (RA) and/or anterograde or post trauma amnesia (PTA). This stance is based on the unitary memory theory, which supports the concept that if the event is not available to memory, it cannot be argued that subsequent psychiatric symptoms derive from memory of the trauma (Layton & Wardi-Zonna, 1995).

One particular study, focussing on memory systems, looked at the co-occurrence of neurogenic amnesia and PTSD. Research indicates that establishing the etiology of

retrograde amnesia is more important than post-trauma amnesia to the discussion of PTSD in MTBI patients. This is based on the belief that it is the retrograde amnesia which erases the memories of the period prior to the traumatic event and the event itself; these are the memories which are expected to induce PTSD in patients (Layton & Wardi-Zonna, 1995). The study gave two case studies in which both patients had a history of retrograde and anterograde amnesia. In each case the inability to recall the trauma, diminished activities, difficulty concentrating, and physiologic reactivity, were endorsed. In addition, one subject had no intrusive thoughts or recollections and one endorsed both items. It should be noted however, that the details of the recollection were not evaluated for accuracy.

Layton and Wardi-Zonna support this assertion by citing three research studies as evidence. First, the occurrence of RA is correlated with duration of PTA (Russell & Smith, 1961). Secondly, RA does not resolve with pentothal interview (Russell & Nathan, 1946). Third, RA occurs in response to insults to the CNS that are not associated with emotional trauma (Squire, Slater, & Chace, 1975).

Support for the co-morbidity of PTSD and MTBI stems from the multiple memory theory in which declarative and non-declarative memory systems operate independently. Declarative memory refers to stored experiences that are actually or potentially accessible to conscious recollection. This is the memory system that is disrupted in amnesia (Layton & Wardi-Zonna, 1995). Non-declarative memory systems, mediate a variety of phenomena, none of which are accessible to consciousness. Hypothetically, these include motor skills, priming, classical conditioning, and habituation/sensitization. The proposed hypothesis is that when the declarative memory system is interrupted by trauma to the head, the non-declarative memory systems take over. Non-declarative memory is not disrupted in amnesia and able to change behavior as a function of prior knowledge rather than by subjective recollection or recognition. This theory is bolstered by several studies which find that although the percentage is relatively small, some individuals can have PTSD from an event in which they also incurred a MTBI (Layton & Wardi-Zonna, 1995).

The majority of the research argues for the potential or possibility of co-morbidity/concomitance of MTBI and PTSD. There are several studies which look at examples of

PTSD following mild closed head injury in groups ranging in size from 2 to 312. One study looked at 24 outpatients with diagnosed head injuries following various traumas. Each participant was asked to fill out standardized questionnaires assessing post-traumatic residuals. Thirty three percent of these participants met criteria for PTSD (Ohry, Rottok, & Solomon, 1996). The most highly endorsed symptoms were difficulty remembering aspects of the event, difficulty in concentrating, startled responses, and physiological activity. The least endorsed item criteria were recurrent dreams/nightmares about the event, and reliving the event

Another study looked at the same topic using 10 patients with closed head injuries (three of which met the criteria for MTPI) who carried a diagnosis of PTSD. These clients were drawn from a series of 312 closed head cases seen over 6 years. Results indicated that "in some closed head injury cases, whether minor or severe, the window of experience is sufficiently traumatic to result in the development of PTSD symptomotology, and a continuum of experience is not essential" (McMillan, 1996).

Larger groups have also been studied. The psychiatric consequences of road traffic accidents in 188 consecutive cases admitted to hospitals was investigated. All were MTBI

cases, as those with longer than 15 minutes loss of consciousness were excluded. PTSD criteria was met for 11% of the participants; however, none of the 11% PTSD sample had any loss of consciousness. Researchers concluded that risk of PTSD was inversely related to loss of consciousness and that acute distress was found to be a risk factor associated with the development of psychological difficulties such as phobias to external stimuli and PTSD (Mayou, Bryant, & Duthie, 1993).

Harvey and Bryant investigated 76 individuals who had car accidents. Thirty-eight had definite MTBI on the basis of post traumatic amnesia of less than 24 hours, and the presence of both retrograde and anterograde amnesia. The other 38 individuals did not sustain any head injury. The researchers found that 27% of the MTBI individuals and 42% of the non-MTBI participants met most diagnostic criteria for PTSD (Bryant & Harvey, 1995).

One of the most staunch proponents against the co-morbidity of PTSD and MTBI is Sbordone. Although his previous pieces were primarily discussion or theory papers, his latest study provides empirical support for his position (Sbordone & Liter, 1995). In the study he examined 70 patients who had previously been diagnosed as either having PTSD or MTBI. Each participant filled out a detailed

chronological history of the events that preceded, followed, and occurred during the traumatic experience. In addition, subjects were asked to indicate whether or not they were rendered unconscious or had amnesia, as well as describe the symptoms which they experienced secondary to the traumatic event. An interesting point was brought out, suggesting that PTSD patients will initially claim that they cannot recall the accident and/or experienced a loss of consciousness. However, when given a safe therapeutic environment to describe the trauma, the PTSD clients were able to relate in high detail and emotional/agitated state the events of the trauma. Sbordone purpose was to determine the relationship between the participant's recollection of the events immediately prior, during, and following the traumatic event, and the various symptoms the individuals' developed following the trauma.

Sbordone's results show that none of the PTSD patients reported a loss of consciousness compared to 85.7 of the MTBI/PCS patients. In addition, 71.4% of the MTBI/PCS participants reported some recall of the events which occurred within 15 minutes of the traumatic event, none of the MTBI/PCS clients could provide highly detailed recollection of the event. Conversely, 100% of the PTSD patients were able to not only recall some portion of the

traumatic event, but also were able to go into minute detailed recollection of the events preceding the trauma. When relating the recollections, the 100% of the PTSD patients were emotional and anxious compared to none of the MTBI/PCS participants. Interestingly enough, in frequency of cognitive, emotional, and physical complaints MTBI/PCS and PTSD patients were significantly different at the $p<0.01$ level on 18 of the 30 items. Items in which there was no significant difference were memory difficulties, word-finding difficulties, social-interpersonal difficulties, problem-solving difficulties, fatigue, distractibility, speech problems, decline in libido, need for excessive sleep, and photophobia (Sbordone & Liter, 1995).

Support for Sbordone comes from Corwin Boake (1996) who notes that he was able to find fewer than 10 case reports published in the English-language literature of patients who suffered from both disorders. In addition, the author breaks down the case reports. Two were patients with severe head injury, and in one of these the posttraumatic syndrome included a strong element of guilt about the death of a close friend (McMillan, 1991; Layton & Wardi-Zonna, 1995). The remaining cases involved patients with mild head injuries. One of the cases seemed more like an adjustment

disorder than PTSD (Horton, 1993). The severity of the mildly injured patients was not adequately documented. For example, the patients reported being partially amnesic for the trauma, but no medical records to document the evidence of a TBI. Dr. Boake concludes by saying clinicians should screen mildly head injured patients for PTSD, bearing in mind that the overall risk of PTSD is low (Bontke, 1996).

Catherine Bontke, MD, System Director for Rehabilitation Services at Rehabilitation Hospital of Connecticut notes, notes that she has only seen 1 patient out of 2,000 in the past 9 years with the duel diagnosis of PTSD and a mild traumatic brain injury. She goes on to note that the lack of cases is most likely due to several causes. One being that the development of PTSD in non-war related events is apparently low (Helzer, Robins, & McEvoy, 1987 as cited in Bontke, 1996). Secondly, developing post-traumatic anxiety syndromes consisting of one or two PTSD symptoms following an serious accident is only 43 out of 2,985 patients (Davidson et al., 1991). Lastly, the diagnosis of MTBI is often overlooked as the two syndromes can share many of the same symptoms and it takes a practitioner familiar with both syndromes to make an accurate diagnosis (Bontke, 1996).

Price (1993) is a strong supporter of the mutual exclusive argument. He states, "it should be clear that an injury that is accompanied by concussion cannot be followed by a posttraumatic stress disorder. The hallmark of PTSD is preoccupation with the trauma. If individuals have no memory of the of the events preceding their injury, they can neither ruminate over them nor have flashbacks of them." PTSD and PCS have some symptoms in common, as well as several diagnostic difficulties. They are however, two distinct entities and are mutually exclusive.

In an additional study, involving 47 active duty service members with moderate traumatic brain injury with neurogenic amnesia for the event evidence was found to support the mutually exclusive viewpoint. The results indicate that none of the patients met the DSM-III-R PTSD criteria at 24 months post injury. The authors suggest that because of the posttraumatic amnesia occurring in all of these patients, no declarative memory was able to be established for the events or the injury (Warden et al., 1997).

Conclusion

In conclusion, the review of the literature clearly shows the diagnostic and methodological difficulties

inherent in dealing with MTBI and PTSD. Evidenced by the lack of a cohesive, universally accepted definition and diagnostic criteria for each of the three terms.

Several studies have used neuropsychological assessment measures of attention, processing speed, and memory to evaluate MTBI and PTSD separately, and co-morbidly, with little success at obtaining clear group separation along objective measurement lines. There has been no research that compares neuropsychological measures across both MTBI and PTSD populations, while taking into account time post injury. In addition, most studies have used only one measure of attention/concentration, processing speed, and memory, the current study will use 11 measures that will assess both visual and verbally mediated aspects of attention/concentration, processing speed, and memory.

Discussing the relationship of MTBI and PTSD is a process of sorting through several "gray" areas. Because of the ambiguous nature of the topic and the overlapping symptomatology a diagnosis should be approached with care. Wolfe and Charney state "traumatic brain injury may mimic, rather than cause symptoms of PTSD," and that in instances of organic amnesia, cases of apparent PTSD may represent organically derived depression, psychological responses, or

both, constituting secondary reactions to loss of behavioral function (Wolfe & Charney, 1991).

CHAPTER III

METHODOLOGY AND DESIGN

Participants

This study is comprised of archival data from 137 individuals who were referred to a board certified neuropsychologist for evaluation for mild traumatic brain injury from the years 1993 to 1999. The patient cases consist of 65 individuals who received a diagnosis of PTSD and 72 individuals who received a diagnosis of MTBI. These individuals were selected from approximately 1200 archival files. Patients with a past history of alcohol/substance abuse, neurodegenerative disorder, sequential PTSD following MTBI, pre-existing PTSD, learning disability, previous head injury, extensive soft-tissue damage associated with the injury, and inpatient psychiatric treatment, as well as, those who experienced secondary hydrocephalus, hematoma, or other neurological complication following MTBI were excluded from the study. Patients who failed response bias testing were also excluded from the study. Participants were referred by emergency room staff, neuropsychologists, psychiatrists, speech language

pathologists, workers compensation courts, insurance case managers, psychologists, and attorneys.

The MTBI group was comprised of 69% males (n=50) and 31% females (n=22), consistent with the epidemiological findings in the literature, which notes the rate of brain injury in men to be 2 or 3 times greater than in women (Bennett & Raymond, 1997). Gender was equally represented in the PTSD group, demonstrated by 51% males (n=33) and 49% females (n=32). This was surprising, given that research indicates that women are twice as likely to develop PTSD following a traumatic accident than men (Van der Kolk, 1996). Individuals less than 6 ½ months post injury comprised 44% of the sample size (n=51), while 25% were 6 ½ to 12 ½ months post injury (n=29), 16% were 12 ½ to 24 ½ months post injury (n=18), and 15% were more than 24 ½ months post injury (n=17.

Materials

An interview was conducted with each patient to determine the history of the presenting injury, pre-morbid history, and current complaints. Whenever possible, this history was verified by immediate family members. Medical records detailing the injury or accident and subsequent

treatment leading up to the neuropsychological referral were also reviewed. Variables used in establishing a diagnosis of MTBI or PTSD were obtained from this information and included: length of post-traumatic amnesia, duration of loss of consciousness, Glasgow Coma Score rating, subjective reports of any altered mental state (feeling dazed, disoriented, confused, etc.), time post-injury and endorsements of PTSD clinical symptomatology based on DSM IV criteria for PTSD.

Following the interview and medical record review, all participants were administered a basic neuropsychological battery. This battery consisted of tests that assess basic domains such as attention, sensation, perception, motor, perceptual motor, memory/learning, language, executive function, and abstraction. Where appropriate, the battery was extended by additions of certain tests to address the individual needs of each patient (Lezak, 1995).

For purposes of this study only relevant portions of the data collected, based on the review of the literature, were analyzed. Tests scores included in the analysis consisted of Logical Memory I & II, Visual Reproduction I & II, and Paired Associates I & II (WMS-R; Wechsler, 1987), Stroop Color Word Interference Test (Stroop, 1935), Trails B (TMT; Army Individual Test Battery, 1944), Knox Cube

(KCT; Arthur, 1947), Digit Span forwards and backwards and Digit Symbol (WAIS-R; Wechsler, 1981).

The literature overwhelmingly cites cognitive deficits of memory, attention/concentration, and processing speed as the three primary cognitive sequelae following MTBI (Barth et al., 1983; Bohnen et al., 1992; Dikmen et al., 1986; Klonoff & Lamb, 1998; Leininger et al., 1990; Raskin et al., 1998; Stuss et al., 1989). Similarly, memory, attention/concentration, and processing speed difficulties are common complaints in most PTSD cases (Davidoff & Laibstain, 1988; Price, 1993; Sbordone & Liter, 1995; Vasterling et al., 1998; Wolfe & Charney, 1991).

Memory/learning was assessed by the Wechsler Memory Scale - Revised (WMS-R). The Wechsler Memory Scale-Revised (WMS-R) contains nine tests. The three included in this study were Logical Memory, Visual Reproduction, and Paired Associates. Both the immediate and delayed scores from Logical Memory, Visual Reproduction, and Paired Associates were used.

Logical Memory I employs a free recall immediately following auditory presentation in which the examinee listens to two short stories, and immediately after hearing each of the stories is asked to retell it from memory. After the examinee has recalled as much as possible from

both stories, the examinee relates that later on they will be asked again to tell the two stories (Wechsler, 1987). Logical Memory II takes place 30 minutes after the initial presentation of the stories in Logical Memory I. The participant is asked to tell the stories again one at a time with as much detail as possible (Wechsler, 1987).

In Visual Reproduction I, the patient is allowed to sequentially examine four cards that contain a geometric design. The cards are presented one at a time. The examinee looks at the geometric design for ten seconds, the design card is removed and the client is asked to draw the design from memory (Wechsler, 1987). Thirty minutes later, during Visual Reproduction II, the examinee is once again asked to draw all four geometric figures from memory.

Verbal Paired Associates I consists of eight word pairs which are read to the examinee one at a time. After the entire list is read, the first word of each pair is given to the client, who is asked to supply the second part of the word pair. There are four "hard" word pairs, and four "easy" word pairs to be learned. The subtest is discontinued when the examinee reaches criterion by correctly answering all eight items. The minimal number of trials is 3 and the maximum number of presentations is 6. Thirty minutes after the Verbal Paired Associates I is

administered the examinee is given the first word of each word pair and asked to supply the examiner with the word from the earlier list that went with it (Wechsler, 1987).

The WMS-R subtests were chosen because they function well in the identification of neuropsychological deficits caused by closed head injury (Reid & Kelly, 1993). Constructs of particular interest in neuropsychological assessment of memory in TBI are verbally mediated tasks of immediate span (Logical Memory and Paired Associates), free recall/long delay (Logical Memory), cued recall/long delay (Paired Associates), learning over repeated trials (Paired Associates), as well as visuo-perceptual mediated tasks of immediate span (Visual Reproduction) and free recall/long delay (Visual Reproduction)(Adams et al., 1996). Visual Reproduction has been shown to be sensitive to the effects of head trauma, correlating significantly with ventricular enlargement (Cullum & Bigler, 1986), and able to distinguish a group of MTBI patients from a control group (Stuss, Ely, et al., 1985). Significant and consistently lower scores on delayed recall of Verbal Paired Associates distinguished control subjects from MTBI patients who had apparently "recovered" from mild head injuries (Stuss, Ely, et al., 1985 as cited in Lezak p. 453).

For Logical Memory and Visual Reproduction subtests, each participant's performance was reflected by an age-corrected percentile. For Paired Associates a retention rate was calculated from immediate and delayed performances, in accordance with literature suggesting this as the most effective use of the measure for MTBI/PTSD purposes.

Attention/concentration and processing speed were measured by the Stroop Color/Word Test, Trails B, Knox Cube Test, Digit Span (Forwards and Backwards), and Digit Symbol Coding. The Stroop Color Word Interference test is based on the finding that it takes longer to name colors than to read words that represent colors. It takes even longer to name colors of printed words if the prink ink is a color different than the name of the word. A three trial protocol is used in which the examinee has 45 seconds on each trial to perform the required task. The stimulus changes with each trial. In the first trial the client is asked to read printed words of colors. The second trial consists of the client naming the color of ink used to print a series of "X's" as quickly as possible. In the third trial the word and the color in which it is printed differ, the client is asked to name the color of the ink while ignoring the word that is printed (Lezak, 1991).

The Stroop technique was selected due to it's sensitivity to the effects of closed head trauma, as even patients with "good recovery" continue to perform abnormally slow five months or more after the injury (Stuss, Ely, et al., 1985 as cited in Lezak (1995) p. 375). The Color/Word trial of the Stroop was used, with performances represented by an age-corrected percentile.

The Trail Making Test (TMT) has had wide use as a test of visual conceptual and visuo-motor tracking, since originally constructed as part of the Army Individual Test Battery in 1944. The scoring method that is most commonly used is the Reitan method. TMT is part of the Halstead-Reitan test battery. The test is given in two parts, "A" and "B." In part "A" the subject is first asked to draw lines to connect consecutively numbered circles on one work sheet. Part "B" consists of the participant being asked to alternatingly consecutive numbers and letters circles. The subject is told to connect the circles as fast as possible without lifting the pencil from the paper.

The use of the TMT was indicated by research that found the performances by patients with mild head injury to be slower than those of control subjects, with a linear relationship between slowing and severity of damage (Leininger et al., 1990, as cited in Lezak (1995) p. 383).

The TMT is also valued as an assessment of a patient's ability to respond to a complex visual array, to follow a sequence mentally, and deal with more than one stimulus or thought at a time (Eson et al., 1978; as cited in Lezak (1995) p. 384). In addition, it has proven to be an indicator of flexibility in shifting the course of an ongoing activity (Pontius & Yudowitz, 1980; as cited in Lezak (1995)p. 384). The TMT appears as an age-corrected percentile score.

Knox Cube Test (KCT) consists of four blocks affixed in a row on a strip of wood. The examiner taps the cubes in prearranged sequences of increasing length and complexity, and the participant's task is to reproduce the tapping pattern exactly. The KCT was specifically used because it tests immediate visuo-spatial attention span with the addition of a sequencing component (Shum et al., 1990). Due to score conversion limitations of archival data and original measure design, The KCT is scored as an age-equivalent level.

Digit Span (forwards and backwards) is a performance sub-test of the Wechsler Adult Intelligence Scale-Revised. In Digits forward, the examinee is instructed to listen carefully and repeat a series of numbers in the identical order immediately after presentation by the examiner. The

digits are given at a rate of one per second Digits backwards is analogous to Digits Forward with the exception being the examinee is asked to repeat the digit series in the reverse order of presentation (Wechsler, 1981).

Digit Span Forward was chosen because it appears to primarily be a measure of attention and has been shown to fall below normal limits in the first months following head trauma. Likewise, Digit Span Backwards tends to be sensitive to many kinds of brain damage, with a linear relationship between severity of lesion and number of reversed digits recalled (Lezak, 1995). Performance on Digit Span subtests is represented by age-corrected percentiles.

Digit Symbol is a performance sub-test of the Weschsler Adult Intelligence Scale - Revised. The subject is required to rapidly copy a series of symbols matched with corresponding numbers, as quickly as possible within 90 seconds.

Digit Symbol was indicated by the literature, suggesting it is a test of psychomotor performance that is relatively unaffected by intellectual prowess, memory, or learning (Erber et al., 1995). This test is consistently more sensitive to brain damage than other Wechsler Intelligence Scale subtests in that it's score is more

likely to be depressed even when damage is minimal, and is generally most depressed when other tests are affected as well (Lezak, 1995 p.378). Digit Symbol performance was reflected by age-corrected percentiles.

In addition to the neuropsychological cognitive measures, each individual's gender, age, education level, and time post-injury was reviewed. Age was the participant's chronological age at time of evaluation. Education was represented by the highest corresponding grade level the individual has completed at time of evaluation. Time post-injury was calculated as the number of months since injury as follows: Group One: less than 6 ½ months, Group Two: 6 ½ - 12 ½ months, Group Three: 12 ½ - 24 ½ months, and Group Four: more than 24 ½ months.

Procedure

All participants were tested in a quiet room using the standard protocol for each of the measures. Administration of all neuropsychological batteries was supervised by a board certified neuropsychologist. This is in keeping with the assertion that neuropsychological examination by a qualified neuropsychologist experienced with mild head injury can reasonably be expected to provide answers to

questions involving whether or not a brain injury has occurred and if so, how severe the injury is and the affect on a specific individual (Hartlage, 1997). Test selection, administration, scoring, and interpretation was conducted by a three-person assessment team, which included a board certified neuropsychologists and two advanced doctoral level psychology students. Testing generally took place over a 7-hour time span with one hour allowed for lunch. Additional breaks were given at any point requested by the participant, or when it appeared that fatigue was becoming a factor. All data obtained from the participants (history, medical records, premorbid standing, demographics and assessment results) was recorded on a neuropsychological evaluation face sheet, which was constructed and used by the neuropsychologist (Appendix C). For purposes of confidentiality identifying patient information was blacked out during archival data collection.

Following the interview and administration of the neuropsychological battery, each of the 137 participants were diagnosed as either meeting the diagnostic criteria for MTBI or PTSD.

Research Questions and Null Hypotheses

The overall question addressed by this study is to what extent there is a significant difference in neuropsychological assessment of attention/concentration and memory between individuals diagnosed with MTBI versus those diagnosed with PTSD. The null hypothesis for this question is that there is no difference in neuropsychological test scores for attention/concentration and memory between individuals diagnosed with MTBI versus those diagnosed with PTSD.

Should a significant differences arise, a secondary issue is to what extent a predictive equation can be formed using the results of the eleven neuropsychological measures that would discriminate between these two groups. In essence, the null hypothesis for this question is that no predictive equation can be formed using the results of the neuropsychological assessment measures that would maximize the class distinctions between the two groups.

A secondary question is the extent to time post-injury differentially effects performance on neuropsychological measures. The null hypothesis for this question is that there is no difference in neuropsychological test scores for attention/concentration and memory between individuals less that 6 ½ months, between 6 ½ and 12 ½ months, between

12 ½ and 24 ½ months, and more than 24 ½ months post injury, irrespective of group status.

Statistical Analysis

The purpose of the study is to ascertain to what extent there is a significant difference in neuropsychological assessment of attention/concentration and memory between individuals with MTBI versus those diagnosed with PTSD and the extent to which time post-injury plays a role in performance on selected neuropsychological tests. A one-way multivariate analysis of variance was used to determine any discernable difference between the MTBI and PTSD groups on each of the 11 dependent neuropsychological testing variables (Logical Memory I & II, Visual Reproduction I & II, Paired Associates, Stroop Color Word Interference Test, Digit Span Forwards & Backwards, Digit Symbol, Trails B, and Knox Cube), as well as the demographic variables of age and education. In addition, several one-way analyses of variance tests, as well as a chi-square test were utilized to test for potential covariates and presence of gender effects.

A 2 X 4 multivariate analysis was performed to determine the effect of length of time post injury on

neuropsychological performance across the dependent variables.

Chapter IV

Results

Research Questions and Null Hypotheses

The overall question addressed by this study is to what extent there is a significant difference in neuropsychological variables of attention/concentration, processing speed, and memory between individuals diagnosed with MTBI vs. those diagnosed with PTSD. The null hypothesis for this question is that there is no difference in neuropsychological test scores proven to be sensitive to attention/concentration, processing speed, and memory between individuals diagnosed with MTBI vs. those diagnosed with PTSD.

Should a significant differences arise a secondary issue is to what extent a predictive equation can be formed using the results of the eleven neuropsychological measures that would discriminate between these two groups. In essence, the null hypothesis for this question is that no predictive equation can be formed using the results of the neuropsychological assessment measures that would maximize the class distinctions between the two groups.

A secondary question is the extent time post-injury groups differ on neuropsychological measures. The null hypothesis for this question is that there is no difference in neuropsychological test scores on measures of attention/concentration, processing speed, and memory between individuals less that 6 ½ months, between 6 ½ and 12 ½ months, between 12 ½ and 24 ½ months, and more than 24 ½ months post injury.

Statistical Analysis

Several multivariate analyses of variance procedures were performed to test these hypotheses to avoid inflating the overall type I error rate by the use of fragmented univariate tests (Stevens, 1996). First, a one-way multivariate analysis of variance was performed, in which diagnostic group membership (two levels - MTBI & PTSD) was the independent variable, with Logical Memory I & II (percentile), Visual Reproduction I & II (percentile), Paired Associates (percentile), Stroop Color/Word Interference (percentile), Knox-Cube Test (age- equivalent scores), Trail Making Test- Part B (percentile), Digit Span Forward (percentile), Digit Span Backward (percentile), and

Digit Symbol Coding (percentile) serving as the 11 dependent variables.

The means, standard error, and confidence interval of the eleven neuropsychological dependent variables across both MTBI and PTSD groups are reported in Table 1 (Appendix D).

No statistically significant differences were found between MTBI and PTSD group membership. Wilks' Lambda = .121, F = 1.068, p=.394. Thus, there is a failure to reject the Null Hypothesis, which stated that there is no difference in neuropsychological test scores for attention/concentration, processing speed, and memory between individuals diagnosed with MTBI vs. those diagnosed with PTSD. Multivariate findings, as well as follow-up ANOVA results are reported in Table 2 (Appendix D).

Three one-way analyses of variance tests were conducted to determine if education, age, or length of time post injury were acting as potential covariates. During analysis of length of time post injury, five outliers were identified (2 PTSD: 120 and 300 months post injury, as well as, 3 MTBI: 172 and two 324 months post injury). Analyses were performed both with and without these outliers, with no significant change in statistical significance (5 Ss included F = .174, p=.677; 5 Ss excluded F = .265, p=.608).

The following findings are with the five subjects excluded. Highest education achieved was very similar across both MTBI and PTSD groups (MTBI = 12.42 & PTSD = 12.66; F = .343, p= .559). Likewise, chronological age across MTBI and PTSD groups was generally the same (MTBI = 37.21 & PTSD = 36.22; F = .171, p= .680). Length of time post injury across MTBI and PTSD was quite similar (MTBI = 12.95 months & PTSD = 14.25 months; F = .265, p= .608). MTBI and PTSD diagnostic groups did not statistically differ on age, education, and length of time post injury. The sample size, means, standard error, and confidence interval of age, education, and length of time post injury across both MTBI and PTSD groups are reported in Table 3 (Appendix D).

Given that gender was unequally distributed in the MTBI group (Chi-Square = X^2 (1) = 4.99, p=.026), follow-up analysis was conducted to determine the presence of gender effects on the data. A 2 x 2 multivariate analysis of variance was performed. Gender (two levels) and diagnostic group membership (two levels) were the independent variables with eleven neuropsychological measures serving as the dependent variables. As before, no statistically significant differences were found between MTBI and PTSD group membership across the 11 dependent variables. A statistically significant gender effect was present, Wilks'

Lambda = .735, F = 3.133, p<.001; Hotelling's Trace = .361, F=3.133, p<.001. However, no Group X Gender interaction effect was found (Wilks' Lamba = .908, F = .880, p=.569. These findings suggest that while the two genders perform differentially across neuropsychological measures, the gender effect does not account for the failure to reject the null hypothesis between the MTBI and PTSD.

Due to the significance of gender at the multivariate level, subsequent one-way analyses of variance test were performed. Statistically significant gender effects were found on three neuropsychological variables: Logical Memory I (F = 7.38, p<.008), Logical Memory II (F = 7.98, p<.006), and Digit Symbol Coding (F = 13.25, p<.000). Females performed significantly better than males on all three tests. Means, standard error, and confidence interval for gender by group membership, across the eleven dependent variables are reported in Table 4 (Appendix D).

A 2 x 4 multivariate analysis was performed to determine the effect of length of time post injury on neuropsychological performance. Diagnostic group membership (2 levels: MTBI and PTSD) and length of time post injury (4 levels: < 6 ½ months, 6 ½ - 12 ½ months, 12 ½ - 24 ½ months, and > 24 ½ months post injury) were the independent variables, with the eleven neuropsychological measures

acting as the dependent variables. Once again, no statistically significant differences were found between MTBI and PTSD group membership across the 11 dependent variables. Likewise, no statistically significant differences were found between the four length of time groups across the 11 dependent variables (Wilks' Lambda = .703, F = .937, p=.577; Hotellings' Trace = .377, F = .928, p=.591). There was no diagnostic group membership X length of time post injury group interaction effect (Wilks' Lambda = .637, F = 1.22, p=.189; Hotelling's Trace = .506, F = 1.24, p=.168). Therefore, there is a failure to reject the Null Hypothesis, which stated that there is no difference in neuropsychological test scores of attention/concentration, processing speed, and memory between individuals less that 6 ½ months, between 6 ½ and 12 ½ months, between 12 ½ and 24 ½ months, and more than 24 ½ months post injury.

In sum, no statistically significant differences were found between the MTBI and PTSD group performance across the 11 neuropsychological measures. Subsequently, no follow-up discriminant analysis was indicated. Likewise, no statistically significant differences were found between the length of time post injury groups. Therefore, there was a failure to reject all three null hypotheses.

Chapter V

Discussion, Conclusions, & Recommendations

Introduction

This chapter provides an overview of the study and an interpretation of the results. The implications of the statistical findings are discussed, as well as limitations of the study and recommendations for future research.

Summary of Study

The purpose of this study was to explore the differences between MTBI and PTSD populations on eleven neuropsychological test measures sensitive to attention/concentration, processing speed, and memory, while determining the extent to which length of time post injury plays a role in the performance of the two diagnostic groups.

This study is comprised of archival data from 137 individuals who were referred to a board certified neuropsychologist for evaluation of mild traumatic brain injury from the years 1993 to 1999. The patient cases consist of 65 individuals who received a diagnosis of PTSD

and 72 individuals who received a diagnosis of MTBI. These individuals were selected from approximately 1200 archival files. Patients with a past history of alcohol/substance abuse, neurodegenerative disorder, learning disability, sequential PTSD following MTBI, previous head injury, and inpatient psychiatric treatment, as well as, those who experienced secondary hydrocephalus or hematoma following MTBI were excluded from the study.

There were three main null hypotheses in this study. The first stated that there would be no difference in neuropsychological test scores for attention/concentration and memory between individuals diagnosed with MTBI vs. those diagnosed with PTSD. The second was that no predictive equation could be formed using the results of the neuropsychological assessment measures that would maximize the class distinctions between the two groups. The third stated that there would be no difference in neuropsychological test scores for attention/concentration, processing speed, and memory between length of time post injury groups comprised on individuals less than 6 ½ months, between 6 ½ and 12 ½ months, between 12 ½ and 24 ½ months, and more than 24 ½ months post injury.

Statistical Findings

The research questions were tested using a one-way multivariate analysis of variance (Diagnostic Group x 11 neuropsychological dependent variables), a 2x2 factorial multivariate analysis of variance Diagnostic Group x Gender), and 2x4 factorial multivariate analysis of variance (Diagnostic Group x Post Injury Group). In addition, one-way analysis of variance, chi-square, correlation, and descriptive statistical treatments were applied as indicated by the test data. No statistically significant differences were found between the MTBI and PTSD group performance across the 11 neuropsychological measures. Subsequently, no follow-up discriminant analysis was indicated. Likewise, no statistically significant differences were found between the length of time post injury groups. Therefore, there was a failure to reject all three null hypotheses.

Discussion of Results

Given that PTSD is viewed as a stress reaction to a traumatic event, with a primary psychological etiology, and MTBI is seen as a mild/minor trauma to the brain, evidenced

by a brief loss of consciousness and post traumatic amnesia, with a primary neurological etiology (Shaw, 1998); one would expect to find some group differences on neuropsychological measures. However, when put into proper context, considering that both PTSD and MTBI typically have negative neurological findings (e.g. MRI, CT, neurological examination), both are associated with mild cognitive deficits, and both have been shown to lead to numerous emotional/behavioral sequelae, it becomes less surprising that no statistically significant group effect occurred. "To interpret neuropsychological data properly, each bit of data must be evaluated within context, or it may be unsuitably interpreted" (Lezak, 1995 p. 113).

Many researchers contend that the proper context in evaluating MTBI is length of time post injury. Although the literature varies considerably, most researchers agree that even a brain injury of mild severity may produce minimal brain damage, which results in cognitive weakness (Kibby & Long, 1995). Recovery from these mild cognitive weaknesses is typically estimated to occur anywhere between one and 22 months depending on the study, with the vast majority of the MTBI literature citing full cognitive recovery 3 to 6 months. PTSD has a different symptom course, with symptoms usually manifesting themselves within a month following the

traumatic event (must be present for at least a month to meet DSM-IV diagnostic criteria), and unlike normal adjustment to a mild neurological injury, stress reactions are likely to worsen with time (Shaw, 1998).

MTBI and PTSD groups were similar in time post injury (MTBI = 12.95 months & PTSD = 14.25 months), with 44% of the 137 cases reviewed less than 6 ½ months post injury. Due to their differing course and the data demographics, one might expect there to be significant differences between the time post injury groups and a strong interaction effect between the diagnostic group membership; however, no such findings were documented.

An argument could be made that psychological variables (e.g. depression, anxiety, irritability, etc.) associated with PCS or MTBI with poor adjustment might have confounded the data analyses. PCS is typically linked to deficits related MTBI that persist past the period of time which neurological recovery is thought to occur (3 to 6 months). In addition, PCS, like PTSD, is likely to worsen with time. The fact that there was no difference between individuals 6 ½ months versus 24 ½ months post injury makes psychological variables associated with PCS or poor adjustment unlikely confounds for this sample.

Likewise, a case could be made, based on the literature, that PTSD and MTBI existed co-morbidly in a percentage of the sample population. However, of the 137 individual cases, there was no instance in which a patient met the full criteria for both PTSD and MTBI. One case of sequential PTSD was excluded from the study to protect the integrity of the diagnostic group membership. In this particular instance, the individual suffered sequential PTSD. She was involved in a MTBI, with a brief loss of consciousness, only to be later exposed to an imminent and life- threatening situation (for which there was complete and vivid recall) soon after regaining consciousness.

"A test validated on clearly defined groups (e.g. brain damaged versus normal) may have low predictive validity. If it can identify "only those subjects whose brain damage is obvious, then the test serves no useful purpose, since it confirms what needs no confirmation" (Yates, 1966; as cited in Walsh, 1999 pg. 387). While many aspects of MTBI and PTSD individually, as well as their interaction, remain unclear, the findings from the current study appear to confirm that visually and verbally mediated neuropsychological measures of attention/concentration, processing speed, and memory are not sufficient to differentiate MTBI from PTSD in a clinical group. Likewise,

length of time post injury does not appear to impact either diagnostic group membership or neuropsychological test performance. These findings were confirmed in the absence of a significant interaction between diagnostic group membership and variables of age, education, and gender.

In fact, based on age-corrected normal population mean percentiles, general test performances for both MTBI and PTSD were in the average to above average range, demonstrated by the confidence intervals seen in Table One. This suggest that while a small majority may have performed significantly below average (approximately 5%), the overwhelming majority of both MTBI and PTSD individuals (95%) showed no discernable deficits on neuropsychological measures of attention/concentration, processing speed, and memory when compared to age-adjusted norm populations. While this certainly does not take into account potential individual declines from high average or superior range functioning, it is assumed, based on mean sample education of the two groups, that the number of individuals experiencing pre-morbid decline as measured by the selected battery of neuropsychological measures are relatively few.

Based on this study, the most important information in deriving differential hypotheses between MTBI and PTSD comes from a carefully elicited history during clinical

interview. That is not to say that neuropsychological measures are unnecessary, rather just the opposite is indicated, As cited above, one of the major implications from this study is the absence of significant performance deficits on Logical Memory I & II, Visual Reproduction I & II, Paired Associates-retention, Stroop Color/Word Interference Test, Knox-Cube Test, Trail Making Test- Part B, Digit Span Forward, Digit Span Backwards, and Digit Symbol Coding in individuals who meet the full diagnostic criteria of MTBI or PTSD. Therefore, for purposes of assessing effects of mild traumatic brain injury or an individual's overall functioning across functional domains (i.e. cognitive, emotional/behavioral) a comprehensive neuropsychological examination remains the best procedure currently available (cited in Hartlage, 1997). One can not diagnose MTBI or PTSD on neuropsychological measures alone. There remains too much unaccounted for variability to allow for identification of "individuals" based simply on their performance, observed in a vacuum.

Implications for Practice

There are three major implications for clinical neuropsychological practice. First, individuals who present

with a history of MTBI or PTSD with a benign premorbid neurological, psychological, and substance use history should be able to generally perform within normal limits on Logical Memory I & II, Visual Reproduction I & II, Paired Associates-retention, Stroop Color/Word Interference Test, Knox-Cube Test, Trail Making Test- Part B, Digit Span Forward, Digit Span Backwards, and Digit Symbol Coding. Secondly, with the exception of sequential injury, there was no evidence to support MTBI and PTSD presenting co-morbidly from the same incident. Lastly, clinical neuropsychological evaluations should take an idiosyncratic approach to each patient, taking into account the individuals pre-morbid history, such as native intellectual potential, education, occupation, psychological adjustment, psychosocial stressors, pertinent medical findings, etc. The nature of injury itself, as well as the patient's history since the injury is likewise crucial. Loss of consciousness, retrograde and anterograde amnesia, vivid recollection of event, recovery pattern, chronicity and severity of symptoms, participation in rehabilitation services, brain imaging findings, and current psychological profile are some of the variables which should be considered.

Brain injuries and subsequent sequelae are often multi-modal in impact and do not occur in a vacuum, neither should the interpretation of neuropsychological data. The patient's premorbid and injury history is the proper context in which to interpret neuropsychological findings.

Limitations

1. Obtaining "objective" measures of psychopathology would have been helpful to better speak to differences in emotional/behavioral constructs between the two diagnostic groups.
2. Obtaining a symptom validity measure on all patients instead of just a few would have been helpful in better eliminating any potential response bias.
3. Size of sample: although the N=137, a respectable number, surpassed proposal estimates, a larger sample size could have provided better generalizability of results.
4. Inter-rated reliability from an outside source. Although diagnostic membership was decided upon by an assessment team comprised of a board certified neuropsychologist and two advanced doctoral students, reliability of PTSD and MTBI group membership would have potentially been improved with

a third party evaluator from an outside source. Logistically, this was not feasible.

5. Since neither racial identity nor SES is part of the neuropsychological face sheet that was utilized, this data was addressed or co-varied for.
6. Perhaps one reason why cognitive deficits were not found was the absence of an estimated pre-morbid level of overall cognitive functioning. This would make more clear the discrepancy between pre-injury and post-injury more salient. Several the patient's cognitive functioning as measured by the selected measures may have declined from "Superior" or "High Average" ranges to "Average."

Directions for Further Research

Future studies should address the limitations of the current study. For example, further researchers should obtain an "objective" measure of psychopathology, as well as a symptom validity test. Pooling resources from several neuropsychology clinics (using the same protocol) would increase sample size, while providing increased inter-rated reliability. In addition, future studies might look at similar type designs as the current study, but employing different tests to tease out group membership, as well as

the inclusion of measures that would measure pre-morbid cognitive functioning. In addition, demographic data of ethnicity and social economic status would be useful in providing a more complete understanding of the test results.

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Appendix A

Common Symptoms of MTBI

(Bennett & Raymond, 1997)

Physical

- •Headache
- •Vertigo
- •Diplopia
- •Blurred Vision
- •Photophobia
- •Sonophobia
- •Fatigue
- •Nausea/ Vomiting

Cognitive

- •Disorientation
- •Memory Problems
- •Fluctuating Attention/Concentration
- •Reduced Insight and Judgement
- •Poor Abstract Reasoning
- •Language Difficulties

Emotional

- •Depression
- •Anxiety
- •Irritability and Frustration
- •Phobic Reaction
- •Disinhibition
- •Somatic Preoccupation
- •Reduced Self-Esteem and Self-Concept

Common Symptoms of PTSD

(Davidoff et al. 1988; Dikmen & Levin, 1993; Sbordone & Liter, 1995)

Physical

- •Sleep Disturbance
- •Headaches
- •Vertigo
- •Reduced libido
- •Appetite Problems
- •Fatigue

Cognitive

- •Memory Problems
- •Attention/Concentration Difficulties
- •Chronognosy (loss of temporal sequencing)
- •Word Finding Difficulties

Emotional

- •Avoidance of thoughts, feeling, activities or situation associated with the injury
- •Persistent reliving of the event
- •Depression
- •Reduced self-esteem
- •Apathy
- •Loss of Affect
- •Anxiety
- •Disinhibition
- •Abulia
- •Irritability
- •Anger Outbursts
- •Emotional Lability

Appendix B

CONTINUUM OF INJURY

DEFINING SYMPTOMS

Malingering Continuum	Somatoform Disorders	Stress Reactions	Mild Traumatic Brain Injury with poor Adjustment	Mild Traumatic Brain Injury with good Adjustment	Moderate Traumatic Brain Injury	Severe Traumatic Brain Injury
No LOC/PTA Inconsistent, findings minimization of effort, exaggeration of deficits	No LOC/PTA Pain sx, GI sx psuedoneurologic sx not fully explained by or in excess of medical condition (ex: Conversion)	No LOC/PTA Anxiety and reexperiencing phenomema avoidant tendencies and signs of arousal (ex: PTSD)	Brief LOC/PTA Negative neurologic findings but shaken sense of self with subjective cognitive dysfunction loop (ex: MTBI)	Brief LOC/PTA Negative neurologic findings, minimal cognitive deficits except for attention, concentration, IPS, memory, efficiency.	Moderate LOC/PTA Positive neurologic findings, noticeable physical and/or cognitive deficits. May need long term treatment.	Lengthy LOC/PTA Positive neurologic findings, obvious physical and cognitive deficits. May need long term placement or supervision

POST CONCUSSIVE SYNDROME

1. Symptoms likely to worsen with time.
2. Severity of symptoms in excess of what would be expected .
3. Depression and anxiety antecedent to cognitive or physical deficits.
4. ? Premorbid make-up ?

DEFINITIVE NEUROLOGIC INJURY

1. Symptoms usually improve with time.
2. Severity of symptoms consistent with or less than expected.
3. Depression and anxiety consequent to cognitive or physical deficits .

Appendix C

Identifying Information

Name: | Referral:

DOE: / / | Age | DOB: / / | Sex

CC:

Admit (ER) Sx:

HPI

Date: / / | Time Post:

Description:

LOC | CT/MRI

RA | PTA

Sz | EEG

Hosp:

Premorbid Hx

Med/Dev:

OCC/Educ:

Est IQ (FOI) | Voc

Soc:

1. Mental Status Examination

Arous OR P P T

Dom: H E F

App:

Emot:

Mot Sx:

Thougt Proc:

Content:

Speech:

IPS:

Attitude:

2. Attention SX

DS F B V:F B

Age:(A) (V)

ACQ: 592 D

3. Sensation

A Visual Flds:

DSS: L R

Pupils:

C/U Acty:OD 20/ OS 20/

B Audition: CNVIII

DSS: L R

Rhine: A B A B Weber

C Somatheasis CNV

Dermatones

DSS: L R

D Neglect/Inattention

4. Perception

A Visual:Trck PC

VFD/JOL: %

MVPT: %

FAR: %

Faces: %

Hooper %

Sim/GC %

Stroop (C) =t

B Auditory

SRT =Rank

Wepman

C Somatheasis

Stereo L R

Graph L R

Fing Nam L R

5. Motor

A CN V VII

III,IV, VI

IX, X

XI

XII

B Strength: L R

C Dexterity: L R

D Speed: L R

E Ext:

F Coord/Tremor:

G AMR:L R FNF L R

H Gait/Station

I Reflexes: DTR:

Release:

6. Perceptual Motor

A Visual: DS:

Trails A: %

BD/3D:

Beery/Ben:

Draw/AS:

B Auditory:

R/L Disc:

Limb

Buccofacial

Ideational

C Somasthetic

TPT(t) =t

Dom =t

NonDom =t

Both =t

Mem Loc

7. Memory/Learning

Rey Port

A Vis/Spat: VRI: %

VRII: %

Recognit:: =SS

VPA

VIQ BVRT/CVMT

Rey Copy =SS

Complex Imm =SS

Del =SS

B Aud/Verb: LMI: %

LMII: %

Recognit: /10 /11

=SS

PA: E:

H:

VEQ SDL %

Bushke

C Delay Q:

8. Language: AS

Recept PPVT %

SIL %

A Aud:: Y/N SSPT

ALPS-L

MAE (AC) %

MAE (Token) %

B Vis: WRAT-R %

Stroop W =t

ALPS-R GG

MAE (RC) %

GORT %

C MAE (SenR) %

Expressive Dysarth

A Voc: MAE (VisN) %

MAE (CFL) %

GORT-P&P Odd

B Graph AS

WRAT-S %

9 Executive MZ:

A Sequencing PA

Trails B %

B Response Inhib

Stroop CW =t

C Regulating

Interleaved

Error Use

Rampart PSV

10 Abstraction

A Verbal:Comp Sim

B Math: AS WAIS-R

WRAT-A %

C Sorting/Problem Solving

WCST

Category

Ravens %

Proverbs

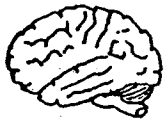
SIL: Voc

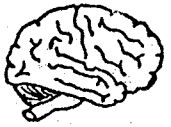
AQ CQ

11 Global Indices WAIS-R: VIQ PIQ FSIQ

DS DRS WMS(I) (D)

MMSQ Folstein Imp Ind AIR





Conclusions:

Recommendations:

Appendix D

Table One

Dependent Variable	Group Membership	Mean	Std. Error	95% Confidence Interval Lower	Upper
Logical Memory I	MTBI	62.14	3.37	55.46	68.82
	PTSD	56.52	3.37	49.85	63.20
Logical Memory II	MTBI	61.77	3.32	55.18	68.35
	PTSD	56.93	3.32	50.34	63.52
Visual Reproduction I	MTBI	70.72	3.48	63.81	77.63
	PTSD	65.78	3.48	58.87	72.70
Visual Reproduction II	MTBI	64.39	4.05	56.36	72.42
	PTSD	67.10	4.05	59.07	75.13
Paired Associates Reten.	MTBI	93.88	1.93	90.05	97.72
	PTSD	96.48	1.93	92.65	100.32
Stroop Color/Word Test	MTBI	50.95	1.11	48.74	53.16
	PTSD	48.99	1.11	46.78	51.19
Knox Cube Test	MTBI	13.66	.46	12.75	14.57
	PTSD	13.24	.46	12.78	14.16
Trail Making Test- Part B	MTBI	52.22	3.58	45.11	59.33
	PTSD	57.32	3.58	50.21	64.43
Digit Span Forward	MTBI	49.39	3.92	41.63	57.16
	PTSD	41.80	3.92	34.03	49.57
Digit Span Backward	MTBI	54.02	3.57	46.94	61.11
	PTSD	53.58	3.57	46.50	60.67
Digit Symbol Coding	MTBI	40.85	3.63	33.65	48.04
	PTSD	43.28	3.63	36.09	50.48

Table Two

Multivariate Tests

Design: Diagnostic Group + Time Post-Injury Group + Interaction

Diagnostic Group: PTSD vs. MTBI

	Value	F	df	Error df	Significance
Wilks' Lambda	.877	1.056	12	90	.407
Hotelling's Trace	.141	1.056	12	90	.407

Time Post-Injury Group: ≤ 6.5, 6.5 – 12.5, 12.5 – 24.5, and >24.5 (months)

	Value	F	df	Error df	Significance
Wilks' Lambda	.703	.937	36	266	.577
Hotelling's Trace	.377	.928	36	266	.591

Diagnostic Group * Time Post-Injury Group: Interaction

	Value	F	df	Error df	Significance
Wilks' Lambda	.637	1.222	36	266.6	.189
Hotelling's Trace	.506	1.246	36	266	.168

Table Three

Oneway for potential covariatess (all Ss included)

ANOVA

		Sum of Squares	df	Mean Square	F	Sig.
Chronological Age	Between Groups	36.205	1	36.205	.193	.661
	Within Groups	25343.562	135	187.730		
	Total	25379.766	136			
Highest Education Achieved	Between Groups	.351	1	.351	.070	.792
	Within Groups	620.881	123	5.048		
	Total	621.232	124			
Months Post Injury	Between Groups	423.669	1	423.669	.174	.677
	Within Groups	328313.3	135	2431.951		
	Total	328737.0	136			

Descriptives

		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean Lower Bound	95% Confidence Interval for Mean Upper Bound	Minimum	Maximum
Chronological Age	MTBI	72	37.5833	14.5687	1.7169	34.1599	41.0068	15.00	71.00
	PTSD	65	36.5538	12.6701	1.5715	33.4143	39.6934	13.00	78.00
	Total	137	37.0949	13.6607	1.1671	34.7868	39.4029	13.00	78.00
Highest Education Achieved	MTBI	67	12.4627	2.3376	.2856	11.8925	13.0329	8.00	20.00
	PTSD	58	12.5690	2.1367	.2806	12.0072	13.1308	7.00	19.00
	Total	125	12.5120	2.2383	.2002	12.1158	12.9082	7.00	20.00
Months Post Injury	MTBI	72	23.7986	55.6946	6.5637	10.7110	36.8862	1.00	324.00
	PTSD	65	20.2769	41.0942	5.0971	10.0943	30.4596	1.00	300.00
	Total	137	22.1277	49.1649	4.2004	13.8211	30.4344	1.00	324.00

Table Four

Variable	Group	Gender	Mean	Std. Err	Low	High
Logical Memory I	MTBI	Male	54.09	3.76	46.62	61.55
		Female	70.20	5.59	59.12	81.27
	PTSD	Male	51.63	4.81	42.09	61.16
		Female	61.42	4.72	52.06	70.78
Logical Memory II	MTBI	Male	52.09	3.71	44.73	59.45
		Female	71.45	5.51	60.53	82.36
	PTSD	Male	53.33	4.74	43.93	62.73
		Female	60.53	4.65	51.30	69.76
Visual Reproduction I	MTBI	Male	72.04	3.90	64.31	79.77
		Female	69.40	5.78	57.93	80.86
	PTSD	Male	66.11	4.98	56.24	75.97
		Female	65.46	4.89	55.77	75.15
Visual Reproduction II	MTBI	Male	67.59	4.53	58.61	76.56
		Female	61.20	6.72	47.88	74.51
	PTSD	Male	67.92	5.78	56.46	79.38
		Female	66.28	5.68	55.03	77.53
Paired Associates Reten.	MTBI	Male	93.27	2.16	88.98	97.55
		Female	94.50	3.21	88.14	100.85
	PTSD	Male	96.33	2.76	90.86	101.80
		Female	96.64	2.71	91.26	102.01
Stroop Color/Word Test	MTBI	Male	50.40	1.24	47.94	52.87
		Female	51.50	1.84	47.84	55.15
	PTSD	Male	50.55	1.59	47.40	53.70
		Female	47.42	1.56	44.33	50.52
Knox Cube Test	MTBI	Male	13.55	.515	12.53	14.57
		Female	13.77	.764	12.26	15.28
	PTSD	Male	13.40	.658	12.10	14.71
		Female	13.08	.646	11.81	14.36
Trail Making Test- Part B	MTBI	Male	49.50	4.01	41.55	57.44
		Female	54.95	5.95	43.16	66.73
	PTSD	Male	57.51	5.12	47.37	67.66
		Female	57.12	5.02	47.16	67.08
Digit Span Forward	MTBI	Male	44.34	4.38	35.65	53.02
		Female	54.45	6.50	41.57	67.32
	PTSD	Male	44.00	5.59	32.91	55.08
		Female	39.60	5.49	28.72	50.49
Digit Span Backward	MTBI	Male	55.20	4.00	47.28	63.12
		Female	52.85	5.93	41.09	64.60
	PTSD	Male	52.96	5.10	42.84	63.07
		Female	54.21	5.01	44.28	64.14
Digit Symbol Coding	MTBI	Male	28.25	4.06	20.20	36.29
		Female	53.45	6.02	41.51	65.38
	PTSD	Male	37.18	5.18	26.91	47.45
		Female	49.39	5.09	39.30	59.47

Appendix E

OKLAHOMA STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD

Date: June 8, 1999 IRB #: ED-99-133

Proposal Title: "MILD TRAUMATIC BRAIN INJURY Vs POST TRAUMATIC STRESS DISORDER: A DIFFERENTIAL DIAGNOSIS "

Principal Investigator(s): John Romans
Daniel Johnson

Reviewed and Processed as: Exempt

Approval Status Recommended by Reviewer(s): Approved

Signature:

Carol Olson, Director of University Research Compliance

June 8, 1999
Date

Approvals are valid for one calendar year, after which time a request for continuation must be submitted. Any modification to the research project approved by the IRB must be submitted for approval. Approved projects are subject to monitoring by the IRB. Expedited and exempt projects may be reviewed by the full Institutional Review Board.

VITA

Daniel A. Johnson

Candidate for the Degree of

Doctor of Philosophy

Dissertation: MILD TRAUMATIC BRAIN INJURY Vs. POST TRAUMATIC STRESS DISORDER: A DIFFERENTIAL DIAGNOSIS

Major Field: Applied Behavioral Studies in Education

Biographical:

Personal Data: Born in Fresno, California, on January 3, 1972, the son of Walter and Lily Johnson

Education: Graduated from Nettleton High School, Jonesboro, Arkansas in May of 1990; received Bachelor of Arts degree in Psychology from Wheaton College, Wheaton, Illinois in May of 1994; received Master of Science degree in Psychology in August of 1996 from University of Memphis, Memphis, Tennessee. Completed the requirements for the Doctor of Philosophy degree in Applied Behavioral Studies at Oklahoma State University in July, 2000.

Experience: Completed clinical rotations at George W. Jackson Mental Health Center, Payne County Youth Services, Northeast Oklahoma Rehabilitation Hospital, Cushing Regional Hospital, St. Francis Hospital, Comprehensive Community Rehabilitation Services, and Stillwater Regional Medical Center. Completed an APA Neuropsychology Specialty internship at the New Orleans VA Medical Center with external rotations at Tulane University's Neuropsychology Laboratory in the Department of Psychiatry and Neurology.

Professional Memberships: National Academy of Neuropsychology, International Neuropsychological Society, New Orleans Neuropsychology Society, and American Psychological Association.