The Prevalence Of Post-Traumatic Stress Disorder In Head-Injured Adults

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ABSTRACT

The present study investigated the prevalence of PTSD, as measured by the Penn Inventory, in a sample of 50 head-injured adults. Fifty-eight percent of the total sample scored within the clinical range on the Penn Inventory, and 17% of this subgroup experienced neurogenic amnesia for the traumatic event. The presence of neurogenic amnesia in the PTSD subgroup was unrelated to the severity of PTSD symptomatology as rated by the Penn Inventory. Scores on the Penn Inventory were highly correlated with the Beck Depression Inventory, the PK and PS scales of the MMPI-2, and eight of the ten clinical scales of the MMPI-2. Contrary to expectations, there were no significant relationships between scores on the Penn Inventory and premorbid and demographic variables, indices of head injury severity, or neuropsychological test performance. These results suggest that PTSD may occur in presence of head injury and concomitant neurogenic amnesia for the traumatic event.

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TABLE OF CONTENTS

ABSTRACT ii
TABLE OF CONTENTS iii
LIST OF TABLES iv
ACKNOWLEDGMENTS v

CHAPTER 1: INTRODUCTION 1
1.1 Clinical Features of Post Traumatic Stress Disorder (PTSD) 2
1.2 Assessment of PTSD 4
1.3 Neuropsychological Test Performance in PTSD 6
1.4 PTSD and Motor Vehicle Accidents 9
1.5 Traumatic Brain Injury 10
1.6 PTSD and Traumatic Brain Injury 11
a. Nondeclarative Memory Theory 14
b. Secondary Traumatization 17
1.7 Summary of the Literature and Purpose of the Present Study 19
1.8 Hypotheses 19

CHAPTER 2: METHOD 21
2.1 Participants
a. Criteria for Inclusion in Study 21
b. Descriptive Statistics of Sample 21

2.2 Procedure
a. PTSD Measures 25
b. Cognitive and Neuropsychological Measures 26

CHAPTER 3: RESULTS 29
3.1 Statistical Considerations and Alpha Levels 29
3.2 Tests of Hypotheses 29
3.3 Exploratory Analyses 34

CHAPTER 4: DISCUSSION 36
4.1 Findings Relevant to Main Hypotheses 36
4.2 Caveats 39
4.3 Conclusions and Directions for Future Research 40

REFERENCES 42
APPENDICES 50

LIST OF TABLES

Table 1: Head Injury Severity 23

Table 2: Summary of Physical Injuries 24

Table 3: Length of Hospitalization 24

Table 4: Demographic Data for the PTSD Subgroup 30

Table 5: Distribution of LOC and PTA for PTSD and Non-PTSD Subgroups 30

Table 6: Distribution of Retrograde Amnesia (RA) in PTSD Subgroup 31

Table 7: Correlations Between the Penn Inventory and Demographic, Premorbid, and Injury Severity Variables 33

Table 8: Correlations Between Penn Inventory and Scores on the Cognitive and Neuropsychological Tests 34

Table 9: Correlations Between Penn Inventory and Clinical Scales of the MMPI-2 35

Table 10: Correlations Between Penn Inventory and Content Scales of the MMPI-2 35

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CHAPTER 1
INTRODUCTION

The complex interaction between organic and psychological factors in the symptom presentation and prognosis of individuals with traumatic brain injuries has only recently been acknowledged. Previously, there was a tendency to attribute all of the cognitive and behavioral sequelae of traumatic brain injury to the neurological insult or to virtually ignore the implications of the brain injury and focus solely on emotional reactions to the event. Increased efforts to assess and treat both organic and psychological symptoms following traumatic brain injury have fueled controversy regarding the diagnostic compatibility of brain injury and post-traumatic stress disorder.

It has been a commonly held belief that traumatic brain injury (TBI) and post-traumatic stress disorder (PTSD) are mutually incompatible (Sbordone, 1992). However, recent clinical reports describe brain-injured individuals, some of whom display neurogenic amnesia[1] for the traumatic event, who meet full diagnostic criteria for PTSD (Layton & Wardi-Zonna, 1995; Horton, 1993; McMillan, 1991). Despite accumulating case studies which verify the co-occurrence of PTSD and TBI, there have been no published reports concerning the prevalence of PTSD in a brain-injured sample. Nor have there been any investigations concerning the clinical presentation of PTSD in brain-injured individuals who have neurogenic amnesia for the traumatic event.

The relationship between TBI and PTSD is particularly relevant for clinicians who treat the victims of motor vehicle accidents, as this population, more than any other, frequently present symptoms of both disorders (Norris, 1992; Hickling & Blanchard, 1992; Guberman, 1994). Understanding the relative contribution of neurological and psychological factors in brain-injured motor vehicle accident victims is a prerequisite for the implementation of appropriate rehabilitation or treatment, as well as the provision of realistic estimates of recovery.

The following sections critically review the contemporary knowledge of PTSD, and in particular, its occurrence in the survivors of motor vehicle accidents and brain-

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[1] The term "neurogenic amnesia" will be used throughout this paper in reference to neurologically-mediated amnesia due to head injury , including retrograde amnesia and posttraumatic amnesia, as opposed to "functional amnesia" which refers to memory loss with a psychogenic etiology.

injured individuals. This review concludes with a summary of the existing literature, followed by a discussion of the specific research questions and hypotheses which are investigated in this study.

1.1 Clinical Features of PTSD

Post-traumatic stress disorder (PTSD) is characterized by the development of a multi-faceted set of psychological and biological symptoms, for a duration of at least one month, following exposure to an extreme traumatic stressor. This syndrome first received official recognition in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980). According to its current definition in the DSM-IV (American Psychiatric Association, 1994), PTSD is characterized by three categories of symptoms which develop following exposure to a "psychologically traumatic event that is generally outside the range of usual human experience". The symptom clusters include: a) persistent re-experiencing of the traumatic event, b) persistent avoidance of cues reminiscent of the event and numbing of general responsiveness, and c) persistent symptoms of increased physiological arousal (see Appendix A for DSM-IV diagnostic criteria).

The emergence of symptoms in PTSD appears to have no specific time constraint. Symptoms may develop immediately following exposure to the traumatic event in some individuals, whereas other individuals only experience symptoms following a prolonged incubation period. Numerous surveys have indicated that PTSD symptoms may persist for decades after the initial traumatic event (Goldstein, van Kammen, Shelley, Miller & van Kammen, 1987; van Kammen, Christiansen, van Kammen & Reynolds, 1990) and that the severity of the trauma is more predictive of the duration of stress-related symptoms than is the recency of the event (Norris, 1992).

The prevalence of PTSD in community samples at any one point in time is estimated to be between 1% and 2% (Helzer, Robins & McEvoy, 1987), while lifetime prevalence rates within the general population are close to 10% (Breslau, Davis, Andreski & Peterson, 1991). Studies of at-risk individuals (e.g., combat veterans, victims of natural disasters or criminal violence) have yielded prevalence rates ranging from 3% to 58% (American Psychological Association, 1994). Further, it is assumed that these figures

under-estimate the true prevalence of PTSD, as this disorder is often under-reported. This is because sufferers often develop avoidance behavior and do not seek treatment, or they receive treatment for a co-morbid psychiatric disorder.

Levels of comorbidity in PTSD are high regardless of whether the sample is drawn from the community or treatment seekers (Kulka et al., 1988; Helzer et al., 1987). Sierles, Chen, McFarland, and Taylor (1983) evaluated a sample of inpatient combat-related PTSD patients for the presence of other psychiatric disorders. PTSD was the sole diagnosis in only 16% of their sample. Fifty-six percent met the criteria for one additional diagnosis, 20% for two additional diagnoses and almost 10% for three or more additional diagnoses. The most common comorbid disorders are: somatization disorder, schizophreniform disorder, panic disorder, social phobia, simple phobia, obsessive-compulsive disorder, generalized anxiety disorder, dysthymia, and depression (Kroll, Habenicht & MacKenzie, 1989; Gurvits et al., 1993; Sierles, Chen, Messing, Besyner & Taylor, 1986). Alcoholism, drug dependence, antisocial personality disorder and organic mental syndrome are commonly co-existing syndromes in combat-related PTSD patients (Sierles et al., 1983).

Specific characteristics of the traumatic event may predict the severity of post-traumatic symptoms. A dose-response relationship between the intensity of exposure to the traumatic event and the development of PTSD has been consistently documented (March, 1990; Shore, Tatum & Vollmer, 1986). Exposure to death or disfigurement has been cited as the strongest predictor of PTSD (Green, Lindy, Grace & Gleser, 1989). Further, individuals who experience physical injury are much more likely to develop PTSD symptomatology than their counterparts who experienced the same traumatic event but were unharmed. In a study of Vietnam veterans, 20% of the veterans who were physically injured during combat later met criteria for PTSD, whereas, only 4% of the uninjured veterans were diagnosed with PTSD (Pitman, Altman, & Macklin, 1989). Similar findings were reported in civilian populations following a traumatic event (Martini, Ryan, Nakayama & Ramenofsky, 1990; Schreiber & Galai-Gat, 1993). Generic components of the stressor such as sudden onset, lack of preparation, loss of controllability, trauma duration, threat to life and traumatic loss are also related to the

severity of the post-traumatic stress reaction (Lindy, Green & Grace, 1987; Foa, Steketee & Rothbaum, 1989).

The role of individual pre-morbid factors in the development of PTSD has also been investigated. McFarlane's (1988a, 1988b) research on firefighters who were exposed to a severe bush fire indicates that a personal or family history of psychiatric disorder is significantly related to the development of PTSD. Alcohol abuse and chronically depressed mood are also important predisposing factors (Green, Grace, Lindy, Gleser & Leonard, 1990). The relationship between post-traumatic reactions and personal characteristics such as age, sex and marital status have been addressed as well (Norris, 1992), with young, single females the most likely group to develop symptoms following exposure to a traumatic event. Finally, it has been shown that experiences of previous trauma may sensitize, or predispose, an individual to develop PTSD when confronted with a new traumatic event (Breslau et al., 1991).

To summarize, PTSD is a condition which carries the risks of chronicity, increased physical and psychiatric disturbances, and impairment in interpersonal and professional functions. Many factors, in addition to the traumatic experience itself, are related to the presence and severity of PTSD symptoms. Indicators of a good prognosis are healthy pre-morbid functioning, trauma of a lesser magnitude and brief duration, adequate post-traumatic social support, absence of an individual or family history of psychiatric disorder, and absence of medical and psychiatric comorbidity (Ursano, Fullerton, Kao & Bhartiya, 1995; McFarlane, 1988a, 1988b).

1.2 Assessment of PTSD

PTSD offers a diagnostic challenge to clinicians due to the diversity and complexity of its symptoms. Thus, multiple assessment strategies have been recommended for identifying this disorder. A variety of structured interviews have been recently developed which specifically focus on PTSD symptomatology. However, diagnostic interviews are time-consuming and often require specialized training to administer and, therefore, are generally impractical for general screening or research purposes. In contrast, self-report measures of PTSD have enjoyed widespread usage because they are better suited for diagnostic screening and research purposes.

Self-report questionnaires, which are commonly used in the assessment of PTSD, have emerged from two main sources. First, existing psychological tests were evaluated for their utility in assessing PTSD. Subscribers to this approach have focused on the MMPI and MMPI-2 (Butcher, Dahlstrom, Graham, Tellegen & Kaemmer, 1989), which possess a variety of well-established clinical and validity scales. In 1984, Keane, Malloy and Fairbank identified a group of MMPI items which effectively distinguished PTSD cases from non-PTSD cases. This 49-item scale, known as the PK scale, was found to correctly classify 82% of the original validation sample which was made up of 100 veterans with combat-related PTSD and 100 veterans without PTSD. Other investigators (Cannon, Bell, Andrews & Finkelstein, 1987; Hyer et al., 1986; Koretzky & Peck, 1990) using a variety of comparison groups, methodologies and cut-off scores, have reported that the overall efficiency or hit-rate for the PK scale ranges from 56% to 88%. In the revised version of the MMPI, the MMPI-2, the PK scale contains 46 of the original items. New norms for this scale are presented which are based on the standardization sample of 1138 males and 1462 females from the general population.

More recently, an experimental measure of PTSD, the MMPI-2 PS scale, was developed by Schlenger and associates (Schlenger & Kulka, 1987; Schlenger et al., 1989). This 60-item scale includes the 45 items of the MMPI-2 PK scale in addition to 15 items from the experimental form of the MMPI (AX) that were shown to best discriminate a community sample of Vietnam veterans with PTSD from Vietnam veterans without PTSD. According to Graham (1990), the internal consistency coefficients, reliability and diagnostic accuracy of this scale are acceptable, however, additional empirical studies exploring the clinical utility of this scale are needed.

The second main approach to the assessment of PTSD focused on the development of specialized questionnaires to identify the symptoms of this disorder. "First-generation" tests within this category, such as the Mississippi Scale for Combat-Related PTSD (Keane, Caddell & Taylor, 1988), focused exclusively on the assessment of combat-related PTSD in males. Furthermore, the reliability and validity of these early measures remain largely unknown. In contrast, "second generation" specialized PTSD measures, such as the Penn Inventory (Hammarberg, 1992), are applicable to males and

females within both civilian and veteran populations. The 26-item Penn Inventory was designed to specifically address PTSD symptoms as defined by the DSM criteria. The psychometric properties of the Penn Inventory have been well-investigated and this measure's internal consistency, construct validity, test-retest reliability, and diagnostic accuracy are excellent.

Great strides have been made in the assessment of this complex and dehabilitating disorder. For pragmatic reasons, self-report inventories of PTSD are widely used by both clinicians and researchers. The PTSD scales of the MMPI-2 and the Penn Inventory are exemplary measures in that they are clinically useful in veteran and civilian PTSD samples, correspond with DSM-IV (American Psychiatric Association, 1994) criteria of PTSD, and possess well-documented reliability and validity.

1.3 Neuropsychological Test Performance in PTSD

Cognitive difficulties, including memory disturbances, learning difficulties and attention/concentration problems, are commonly reported by individuals with PTSD (Sutker, Winstead, Galina & Allain, 1990; Sutker, Winstead, Galina & Allain, 1991; Burstein, 1989; Wolfe & Charney, 1991; Everly & Horton, 1989). However, empirical investigations of the neuropsychological abilities of individuals with PTSD have failed to unequivocally confirm such deficits.

In a well-controlled study, Bremner and colleagues (1993) administered selected psychological and neuropsychological tests to 26 individuals with combat-related PTSD and 15 normal controls. The diagnosis of PTSD was based on the DSM-III-R criteria for PTSD as determined by the Structured Clinical Interview for DSM-III-R (SCID-R; Spitzer, Williams & Gibbon, 1987), consensus diagnosis by three research psychiatrists, and a score of greater than 107 on the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder (Keane, Caddell & Taylor, 1988). The groups were matched for age, sex, race, years of education, handedness, socioeconomic status and alcohol use. The authors compared the performance of the two groups on the Arithmetic, Vocabulary, Picture Arrangement, and Block Design subtests of the Wechsler Adult Intelligence Scale - Revised (WAIS-R); immediate and delayed Logical Memory and Figural Memory subtests of the Wechsler Memory Scale (WMS) and the Selective

Reminding Test. There were no significant differences between the groups on the WAIS-R subtests. The PTSD group did, however, perform significantly more poorly than the controls on the immediate and delayed Logical Memory subtests of the WMS. Further, the PTSD group obtained significantly lower scores than controls on the Selective Reminding Test.

Further evidence of deficits in attention, learning and memory in PTSD patients is provided by Uddo and colleagues (1993). These researchers examined the neuropsychological functioning of 16 combat veterans with chronic PTSD and a military sample of 15 individuals free of PTSD and other significant psychopathology. The groups did not differ in years of formal schooling, race or gender; however, there was a significant age difference, with the PTSD group participants an average of nine years older than individuals in the control group. The results indicated that the PTSD group performed more poorly than the comparison group on several measures of attention, memory and new learning. More specifically, the PTSD group performed more poorly on measures of verbal and visual immediate recall and learning, exhibited less facile acquisition of verbal materials with repeated exposures, were more sensitive to proactive interference, and showed greater perseveration on an auditory verbal learning test. Additionally, the PTSD group were relatively impaired on visual attention and tracking tasks and generated significantly fewer words on a verbal fluency test than did the comparison group.

There exists some evidence which suggests that the cognitive difficulties experienced by patients with PTSD are similar, both in nature and severity, to those experienced by individuals who display other psychiatric disorders. Gil and colleagues (1990) compared the performance of 12 patients with PTSD with that of age- and sex-matched psychiatric and normal controls on measures of intelligence and other neuropsychological abilities. Individuals who had undergone ECT, psychosurgery, or had a history of significant head trauma, alcoholism or drug abuse were excluded from the study. The psychiatric control group was matched with the PTSD group in terms of severity of symptomatology, however, they had no history of psychological trauma. The results of their investigations indicate that patients with PTSD have generalized

impairments in their cognitive functioning relative to normal controls, but their performance was not significantly different from that of the psychiatric controls. The authors concluded that PTSD patients display generalized cognitive impairments similar to those encountered in other psychiatric disorders such as depression and schizophrenia.

However, other researchers have failed to find clear neuropsychological deficits in individuals with PTSD when compared with normative expectations as well as matched psychiatric and normal controls. Dalton, Pederson, and Ryan (1989) examined the neuropsychological performance of over 100 male inpatients admitted to a specialized PTSD treatment program. Participants were administered a routine battery of tests, including the WAIS-R, Stroop Test, Rey Auditory Verbal Learning Test, Benton Visual Retention Test, Temporal Orientation, Serial Digit Learning, Shipley-Verbal, and the Trail Making Test. When the performance of the PTSD group was compared to normative expectations, very few and only minor deviations were evident. These authors concluded that PTSD symptomatology does not have a pronounced effect on neuropsychological performance and these patients performed in a manner expected of individuals with mild to moderate anxiety. However, the generalizability of these results is limited by this study's lack of a control group.

Zalewski, Thompson & Gottesman (1994) evaluated 241 Vietnam war veterans who met DSM-III criteria for PTSD but did not meet the criteria for a lifetime diagnosis of generalized anxiety disorder (GAD). Control groups were 241 veterans who did meet the criteria for a lifetime diagnosis of GAD (but not PTSD) as well as 241 nonpsychiatric veteran controls. The groups were matched for age, race, years of education, and handedness. All participants were administered the Block Design subtest of the WAIS-R, the California Verbal Learning Test, the Rey-Osterrieth Complex Figure Drawing Test and the Paced Auditory Serial Attention Test (PASAT). All three groups performed within normal limits and no neuropsychological deficits were evident in the PTSD group.

In summary, although there is some evidence that individuals with PTSD demonstrate cognitive dysfunction of a similar nature to that demonstrated by other psychiatric groups (i.e., anxious and depressed individuals), these findings remain equivocal. Many of the existing studies have utilized small sample sizes and failed to

incorporate well-matched control groups into their research methodology. Further, given the high rates of alcohol abuse, substance abuse, physical injury and head injury in PTSD samples, particularly in individuals with combat-related PTSD, it is surprising that very few of the previously-mentioned investigations screened for these conditions. At present, there have been no attempts to relate the presence of particular symptoms, such as re-experiencing phenomenon, to memory and attentional capacities in PTSD groups, nor have there been attempts to relate the severity of PTSD symptoms to neuropsychological abilities. Thus, further exploration of the memory, learning, attention and concentration abilities of civilians with PTSD is required.

1.4 PTSD and Motor Vehicle Accidents

Motor vehicle accidents (MVAs) are very common occurrences and may result in serious physical and psychological disabilities in individuals injured in such accidents. The devastating effects of involvement in an MVA is described by Norris (1992), who studied the frequency and impact of ten potentially traumatic events, including robbery, physical assault, sexual assault, tragic death, MVA, combat, fire, other disasters and other hazards. Her results indicate that when the frequency of the event and severity of post-traumatic symptoms are considered, involvement in an MVA emerged as the single most significant traumatic event affecting the general population among the events studied. Within Norris' sample, the lifetime frequency of involvement in MVA serious enough to cause injury to one or more passenger was 23%, with 12% of that group meeting diagnostic criteria for current PTSD. Other estimates of the prevalence of PTSD following serious MVAs range from 10% to 65% (Brom, Kleber & Hofman, 1993; Hickling & Blanchard, 1992). Within samples of MVA victims who were hospitalized for their injuries, the prevalence rates of PTSD are considerably higher (Kuch, Swinson & Kirby, 1985).

The symptom presentation of MVA-related PTSD appears to be consistent with the symptoms observed following other forms of trauma, with two notable exceptions. Survivors of MVAs often report a long-standing phobia of being a driver or passenger in a motor vehicle (Kuch et al., 1985). Driving-phobia may significantly impair daily functioning, and is often one of the first symptoms addressed in the treatment of

individuals with MVA-related PTSD (Burstein, 1989). Secondly, there is an unexpectedly high frequency of somatic complaints within this group as compared to other PTSD groups, with MVA-related PTSD patients reporting particularly high rates of headache and chronic pain (Hickling & Blanchard, 1992).

To summarize, involvement in a MVA is a relatively common traumatic occurrence which may result in physical and emotional disability. There is a high prevalence of PTSD in MVA survivors, and the classic symptoms of this disorder are often accompanied by driving phobia and chronic pain. It should be noted as well that the clinical presentation of PTSD in the survivors of MVAs may be influenced by their involvement in litigation for financial compensation for their physical and emotional injuries.

1.5 Traumatic Brain Injury (TBI)

Involvement in a MVA is the cause of approximately half of all traumatic brain injuries in adults (Guberman, 1994). Although the severity of brain injury following involvement in a MVA is highly variable, ranging from mild concussion to severe coma, a number of damaging mechanical forces are common to all closed head injuries. The most obvious of these is the coup-contrecoup effect. The blow at the point of impact is called the coup. Because the brain rests on its flexible stem in a liquid medium, the force of a blow to one side of the head may literally cause the brain to impact with the other side of the skull (contrecoup), resulting in damage to both the point of impact and the opposite area (Gurdijan, 1975). Additionally, due to the translatory force and rotational acceleration of the brain during a sudden impact, delicate nerve fibers and blood vessels may be stretched to the point of shearing. The bruising, shearing and strains caused by traumatic impact appear to play a significant role in producing loss of consciousness, or concussion (Ommaya & Gennarelli, 1974). Subsequent damage to brain tissue may be produced by hemorrhage and edema.

The severity of head injury following a MVA is highly variable, ranging from mild to severe. Initially it was thought that mild head injuries were not associated with any neurological damage. However, there is increasing evidence to suggest that head trauma not associated with alterations in consciousness can result in neuropathological

changes (Levin et al., 1987). Consequently, physical, cognitive and emotional sequelae have been reported following all degrees of severity of head injury. Symptoms of spasticity, fatigue, dizziness, headaches, and insomnia are commonly reported following TBI, and may last from days to months (Binder, 1986). The most common cognitive complaints following TBI are problems with attention, concentration and memory (see Binder, 1986 for review). Cognitive deficits following minor head injury often resolve themselves within three months of the trauma, while deficits associated with more severe head injuries tend to persist over time (Levin et al., 1987; Dikmen, McLean & Temkin, 1986).

Loss of consciousness (LOC) and post-traumatic amnesia (PTA) are commonly considered the two most useful clinical measures of head injury severity (McClelland, 1988). Although there are some variations in the definitions employed by various researchers, the severity of head injury has typically been classified as follows: a) Mild: LOC and/or PTA less than 1 hour, b) Moderate: LOC and/or PTA 1-24 hours, and c) Severe: LOC and/or PTA greater than 24 hours (Russell & Smith, 1961). Post-traumatic amnesia is most frequently defined retrospectively as the period from the time of the injury to full awareness and the ability to retain a stable record of occurring events (McClelland, 1988). Retrograde amnesia, on the other hand, refers to an inability to recall events that occurred prior to the onset of amnesia (Kolb & Wishaw, 1990). Retrograde amnesia may extend from a period of a few seconds to years. Although retrograde amnesia may be patchy and shrink towards the time of the injury, the injury itself and the events surrounding it may never be recalled (Crovitz, 1987).

In summary, MVAs are very common occurrences and account for approximately half of all adult head injuries. The high velocity of impact in a moving vehicle serves to intensify the shearing, stress and shock wave effects on brain tissue and multiplies the likelihood and extent of neurological damage. Brain injury severity, which is commonly defined by the length of unconsciousness and post-traumatic amnesia, is related to the severity and duration of physical, cognitive and behavioral symptoms.

1.6 PTSD and Traumatic Brain Injury

There are many similarities in the clinical presentation of patients who have

experienced a severe traumatic event and those who have suffered mild to moderate head injury. Both groups commonly report concentration difficulties, forgetfulness, sleep difficulties, fatigue, headache, emotional lability, irritability, poor frustration tolerance, depression and anxiety (Sbordone, 1992). In fact, four of the diagnostic criteria for PTSD are also common symptoms of TBI (e.g., inability to recall an important aspect of the event, restricted range of affect, increased irritability and outbursts of anger, and problems concentrating). According to a review of the literature, in the past, when this constellation of symptoms was present in an individual who had suffered a TBI, cognitive and behavioral changes were attributed either to the effects of the brain injury or to the emotional reaction to the event. Thus, the relationship between neurological injury and psychological sequelae following a traumatic event is not well understood.

The DSM-IV (American Psychiatric Association, 1994) definition of PTSD implies that a memory for the traumatic event is at least potentially available to consciousness. This has led some researchers, such as Sbordone (1992), to argue that PTSD and TBI associated with neurogenic amnesia are mutually incompatible and may not be diagnosed concurrently. Associated with this hypothesis is the assumption that memory (or psychologically repressed memory) for the event is a necessary requirement for the existence of re-experiencing phenomenon (i.e., flashbacks, nightmares about the event).

However, other researchers have challenged Sbordone's theory and present case studies of individuals with TBIs who also display PTSD (Layton & Wardi-Zonna, 1995; McMillan, 1991). In all of the case studies, individuals were screened for previous neurological and psychiatric difficulties and met the diagnostic criteria for PTSD according to DSM-III or DSM-III-R guidelines. In more than one of these cases, retrograde amnesia rendered the individual unable to recall the traumatic event.

For example, McMillan (1991) presented the case of an 18 year-old female who was involved as a passenger in a collision between two cars. A close friend of the patient's died in the accident and another friend sustained very severe and persisting injuries. The patient herself suffered a severe head injury and a CT scan conducted shortly after the accident revealed generalized brain swelling. She was unconscious for

three to four days following the injury and was an inpatient in a neuro-rehabilitation unit for three months. Post-traumatic amnesia was estimated to be about six weeks. Upon evaluation fourteen months following her injury, the patient reported symptoms commonly associated with severe head injury, including extreme fatigue, poor concentration, difficulty coping at work, occasional dizziness and severe headaches. She was reported to be irritable, verbally aggressive, moody and childish at times. Along with symptoms of depression, the patient met diagnostic criteria for PTSD. She reported frequent intrusive thoughts about her dead friend and was unable to prevent these thoughts from entering her mind. She suffered from continual and irrational survivor guilt, believing that she had somehow caused the accident or failed to prevent it. She showed cognitive and physical avoidance of reminders of the accident, refused to drive and did not talk about the accident to anyone. Additionally, her psychological distress increased on the anniversary of the accident. According to the material presented, this appears to be a case of PTSD in the presence of neurogenic amnesia.

Horton (1993) presented the case of a 29 year-old male who was involved in a multiple car crash in which the driver of another vehicle was killed. The patient suffered a mild concussion as a result of the accident, however, he did not lose consciousness. Classical symptoms of PTSD were reported by the patient, including repetitive, intrusive thoughts about the driver who was killed, difficulty sleeping, an inability to drive by the site of the accident, increased risk-taking behavior, and difficulty performing routine tasks at work. The patient reported memory difficulties, which were confirmed by neuropsychological testing. In addition to short-term verbal memory impairments, visuospatial deficits and impaired tactile-perceptual abilities were also identified. These test results were consistent with what would be expected following a mild closed-head injury and related PTSD.

Layton and Wardi-Zonna (1995) present two cases of brain-injured individuals who displayed concurrent PTSD. The first case involved a 40-year-old male who sustained a closed head injury with questionable loss of consciousness during an automobile accident. Retrograde amnesia was approximately five minutes, while post-traumatic amnesia was approximately six hours. The patient reported intense

psychological distress at exposure to events that symbolized or resembled the accident, an inability to recall important aspects of the trauma, markedly diminished interest in significant activities, sense of foreshortened future, feelings of detachment and estrangement from others, difficulty concentrating, and physiological reactivity while driving. Neuropsychological testing revealed mild cognitive difficulties consistent with focal right hemispheric dysfunction.

The second case presented by Layton and Wardi-Zonna (1995) concerned a 21 year-old female who was involved in a motorcycle-motorcycle accident. She sustained multiple orthopedic injuries and a closed head injury. The duration of coma was four days, with retrograde amnesia estimated to be one to four minutes, while post traumatic amnesia was two to three weeks. Along with symptoms of depression, this patient presented with PTSD. She experienced recurrent and intrusive images of motorcycles colliding, although she denied memory of the accident. She also complained of difficulties falling and staying asleep, and had recurrent and distressing dreams about the traumatic event. She unsuccessfully attempted to block the recurrent images of the accident. She experienced significant agitation and irritability, difficulties concentrating, feelings of isolation, and detachment from others. Attention and concentration difficulties, as well as deficits in planning and sequencing, were evident from neuropsychological testing.

Taken together, the foregoing case studies support the co-existence of TBI with neurogenic amnesia and PTSD. Various theoretical models might be utilized to explain how PTSD and TBI may co-occur, even in the presence of neurogenic amnesia for the traumatic event. The most compelling hypotheses, one involving the nondeclarative memory system and another focused on secondary traumatization phenomenon, are presented below.

a. Nondeclarative Memory Theory

Layton and Wardi-Zonna (1995) developed a framework to explain the co-occurrence of PTSD and brain injury associated with neurogenic amnesia. These authors point to contemporary memory theory which posits the existence of multiple and independent memory systems in the brain. These memory systems process and integrate

information at a number of levels with varying degrees of verbal and perceptual elaboration (Squire, 1987). A distinction is made between declarative and nondeclarative memory processes (Squire, 1992). **Declarative** memory refers to stored experience that is accessible to conscious recollection. It includes memory for facts, words, faces, scenes, stories, and events, and it is assessed by conventional tests of recall and recognition. Further, declarative memory depends on the integrity of brain structures and connections in the medial temporal lobe and the diencephalon. This is the memory system that is disrupted in neurogenic amnesia.

In contrast, **nondeclarative** memory, which mediates a variety of phenomena, is not accessible to consciousness. Information that is acquired during skill learning (motor skills, perceptual skills, and cognitive skills), habit formation, simple classical conditioning including some kinds of emotional learning, the phenomenon of priming, and other knowledge that is expressed through performance rather than recollection are all examples of nondeclarative memory (Squire, 1992). This unintentional, unconscious form of memory depends on multiple brain systems, including the basal ganglia, and is not disrupted by medial temporal lobe amnesia (Schacter, 1992). The functions of the nondeclarative memory system cannot be directly measured by subjective recollection or recognition. Rather, its existence is only inferred by the presence of objective changes in behavior as a function of prior experience (Schacter, 1987).

The dissociation between declarative and nondeclarative memory can be seen most dramatically in amnesic patients whose memory is found to be significantly impaired when tested with traditional, declarative memory tests of recall and recognition, yet whose nondeclarative memory functions are normal (Diamond & Rozin, 1984; Graf & Schacter, 1985). Numerous studies have shown that amnesic patients can acquire a variety of skills at a normal rate, including motor skills such as puzzle-solving (Brooks & Baddeley, 1976), perceptual skills such as serial pattern learning (Nissen & Bullemer, 1987), and cognitive skills such as reading mirror-inverted script (Squire & Frambach, 1990). Milner and her colleagues at the Montreal Neurological Institute reported that the profoundly amnesic patient, known as H.M., was capable of acquiring motor skills such as pursuit rotor and mirror tracing, even though he did not remember explicitly that he

had previously performed the task (Milner, Corkin & Teuber, 1968). Research on priming, which refers to the improved facility for detecting or processing a perceptual object based on recent experience, has also played an important role in dissociating declarative and nondeclarative memory. The results of numerous investigations suggest that amnesic patients exhibit fully intact repetition priming effects, whether the test materials are words, familiar objects, or entirely novel material such as nonwords, novel objects, or line patterns (Gabrieli, Milberg, Keane & Corkin, 1990; Haist, Musen & Squire, 1991; Schacter, Cooper, Tharan & Rubens, 1991; Musen & Squire, 1992a). Warrington and Weiskrantz (1968, 1970, 1974, 1978) found that amnesic patients could show normal retention of a list of familiar words when tested with word-stem or fragment cues, but were profoundly impaired on free-recall and recognition tests. Taken together, these studies support the existence of independently-functioning declarative and nondeclarative memory systems.

According to current memory theory, a single stimulus or event may have both declarative and nondeclarative properties, and thus, may be stored independently in both systems (Mishkin & Appenzeller, 1987). In this manner, an event may be registered consciously in the declarative system and later be consciously recalled. Concurrently, this same event may also change the behavior of the organism, independent of conscious memory (nondeclaratively). This occurs when autonomic reactivity is increased in response to an event, even though this event did not previously affect arousal.

Under normal circumstances, experiences are stored simultaneously in both declarative and nondeclarative systems and the effect of an experience on behavior would involve the combined effect of registration in both systems. However, in the event of neurogenic amnesia, the declarative system becomes dysfunctional while the nondeclarative system continues to operate. Thus, an individual who suffers brain injury during a traumatic experience may have no conscious recall of the event, but due to the intact functioning of nondeclarative memory, may be behaviorally changed as a function of the experience (see Appendix B). In particular, the nondeclarative system is thought to mediate autonomic responses, which in turn, could be associated with the PTSD symptoms of increased physiological arousal, hyperalterness and avoidance of stimuli

reminiscent of the traumatic event. However, this model does not address the mechanisms by which re-experiencing phenomenon (i.e., flashbacks, nightmares), which are required for the diagnosis of PTSD, may exist in individuals with neurogenic amnesia for a traumatic event.

Preliminary research in this area conducted by Zeitlin and McNally (1991) support the aforementioned theoretical model. These authors investigated declarative (cued recall) and nondeclarative (word completion) memory in 24 Vietnam veterans with PTSD and 24 Vietnam veterans without PTSD. All individuals included in the PTSD group met DSM-III-R criteria for PTSD as determined by the Structured Clinical Interview for DSM-III-R (SCID-R; Spitzer, Williams and Gibbon, 1987). Participants in both groups were presented with a word list, equally comprised of words related to combat, social threat, positive experiences and neutral experiences. Half of the participants in each group were randomly assigned to either an elaborative or nonelaborative encoding condition. Elaborative encoding required the participants to rate their liking of each word on a seven-point scale, whereas nonelaborative encoding required participants to simply count the number of letters in each word. Participants were tested with either a word-completion or cued-recall task. The test order and word sets used in each task were determined according to a balanced design. The results of this study indicated that only the PTSD patients exhibited an implicit memory bias for combat words that was not attributable to response bias. The implicit memory bias for disorder-specific threat information was apparent in both the primed and unprimed conditions. The results also indicated that the implicit memory bias was significantly related to the severity of PTSD even when the effects of length and severity of combat exposure were partialled out.

b. Secondary Traumatization

Secondary traumatization refers to the phenomenon by which an individual may develop PTSD symptoms subsequent to being described the details of a traumatic event, without having direct exposure to the traumatic event. Secondary traumatization has been explored in therapists who work intensely with clients with PTSD (James, 1994; McCann & Pearlman, 1990). Danieli (1994) describes the results of a study that

systematically examined the nature of the emotional responses and other problems experienced by psychotherapists in working with Nazi Holocaust survivors and their families. Reportedly, a proportion of these therapists were traumatized simply by listening to the traumatic experiences of their clients, and "a few found themselves sharing the nightmares" of the survivors they were treating. Similarly, Lindy (1988) found that therapists who worked with Vietnam veterans often experienced PTSD-like symptoms, such as nightmares, intrusive images, reenactments, amnesia, estrangement, alienation, irritability, psychophysiological reactions, and survivor guilt. Additional evidence comes from McCann and Pearlman (1990), who also observed that therapists may experience painful images and emotions associated with their clients' traumatic memories. Some of the clients' traumatic memories may become incorporated into the therapists' own memory system. In this manner, therapists may find themselves experiencing PTSD symptoms, including intrusive thoughts or images and painful emotional reactions, without having directly experienced the original traumatic event.

Secondary traumatization is not limited to the therapists of traumatized individuals; this phenomenon has also been observed in the family members of individuals with PTSD. Maloney (1988) studied the psychological status of the wives of a sample of Vietnam veterans. He observed that the wives of men with PTSD may "identify so strongly with their men that they have authentically internalized their partners' stressor imagery." Many of the wives who participated in this study reported experiencing PTSD-like symptoms similar to those of their husbands.

According to the secondary traumatization hypothesis, an individual who personally experienced a traumatic event, but has no memory of it due to neurogenic amnesia, may develop PTSD symptoms simply by imagining or being described the details of the event. Thus, secondary traumatization may occur if an individual is exposed to pictures of the traumatic event in the media or through others' descriptions of the event. These newly introduced pictures or descriptions of the event, rather than images of the actual traumatic event, may be the source of re-experiencing phenomenon (i.e., flashbacks or nightmares) in brain-injured individuals with PTSD who have neurogenic amnesia for the traumatic event.

1.7 Summary of the Literature and Purpose of the Present Study

The study of PTSD, which was introduced as a psychiatric diagnosis only 15 years ago, remains largely descriptive in nature. Within the current literature, emphasis has been placed on the clinical phenomenology of PTSD as well as disorder identification and differentiation. Considerable debate has recently focused on the feasibility of diagnosing PTSD in the presence of traumatic brain injury, and how to distinguish the symptoms of the two disorders, should they co-exist. Given the high incidence of head and brain injuries in MVA victims, and the frequency of MVA-related PTSD, questions concerning the co-occurrence of these disorders are most appropriately addressed in the survivors of MVAs.

This study was designed to address the following questions:

1. What proportion of head-injured MVA accident survivors display PTSD?
2. Is there a unique presentation of PTSD in head-injured individuals with neurogenic amnesia for the traumatic event?
3. What is the relationship between the Penn Inventory and the PK and PS scales of the MMPI-2 in assessing PTSD in a head-injured population?
4. Which premorbid, demographic and injury variables are related to PTSD in a head-injured group?
5. What is the relationship between performance on neuropsychological measures and endorsement of PTSD symptoms in a head-injured group?

1.8 Hypotheses

In this study's attempts to further the understanding of the relationship between head injury and PTSD, the following hypotheses will be tested:

1. Given accumulating reports of traumatically brain injured adults who meet diagnostic criteria for PTSD, it was predicted that a proportion of this sample of head-injured MVA survivors would display PTSD, regardless of neurogenic amnesia for the traumatic event.

2. The nondeclarative memory theory forwarded by Layton and Wardi-Zonna (1995)

may explicate the occurrence of increased physiological arousal and avoidance symptoms of PTSD in individuals with neurogenic amnesia for the traumatic event, while the phenomenon of secondary traumatization could account for the presence of re-experiencing symptoms of PTSD in this same group. Thus, it was expected that there would be no difference in the clinical presentation of PTSD in head-injured individuals with neurogenic amnesia for the traumatic event and those without neurogenic amnesia for the traumatic event.

3. It was predicted that scores on the Penn Inventory would be highly correlated with scores on the MMPI-2 PK and PS scales, as these measures have been shown to accurately assess the presence and severity of PTSD symptomatology. A high correlation between the MMPI-2 PK and PS scale was also predicted, given the significant item overlap of these two scales.

4. Based on the existing literature, it was predicted that there would be a positive and significant relationship between the severity of PTSD symptomatology and a) exposure to death or disfigurement, b) personal history of previous traumas, c) history of previous psychological or psychiatric treatment, d) increased severity of physical injuries, and e) increased age.

5. A negative relationship between scores on measures of attention, memory and learning and the severity of PTSD symptoms was hypothesized based on the findings of existing studies of the neuropsychological abilities of individuals with PTSD.

CHAPTER 2
METHOD

2.1 Participants

The data used in the present study was obtained from the clinical files of 53 adults who were consecutively seen for neuropsychological assessment following a head injury and suspected brain injury[2]. Participants were assessed between 1992 and 1996. All of the participants were involved in motor-vehicle accidents (including vehicle-pedestrian, vehicle-bicycle, vehicle-motorcycle and motorcycle-motorcycle accidents), and all of these individuals were involved in litigation regarding their injuries. English was the first language for all participants.

In the analyses that follow, participants who scored at or above the diagnostic cut-off score of 35 on the Penn Inventory were regarded as the PTSD subgroup, while those individuals who obtained scores below 35 on the Penn Inventory were regarded as the non-PTSD subgroup.

a. Criteria for Inclusion in Study

Participants were excluded on the basis of: 1) the presence of pre-existing, non-traumatic neurological impairment (e.g., tumor or stroke), 2) performance below the recommended cut-off score on the Victoria Symptom Validity Test (VSVT; Slick, Hopp & Strauss, 1995), a forced-choice recognition memory test used to assess symptom validity, 3) questionable validity of responses on the MMPI-2[3]. These criteria led to the exclusion of three subjects. One individual was excluded from the sample due to prior neurosurgical removal of a brain tumor. A second participant was excluded due to a significantly below-chance score on the VSVT, which is suggestive of malingering. A third individual was excluded from the sample due to a Fb score of 120 on the MMPI-2, which is indicative of an invalid response style.

b. Descriptive Statistics of Sample

Due to the exclusion of the three individuals as mentioned above, 50 participants

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[2] It is important to note that this sample is comprised of individuals with suspected brain injuries. Verification of brain injury through neurological assessment or neuroimaging was unavailable for a substantial proportion of the participants.

[3] As suggested by Munley et al (1995), only participants with MMPI-2 Fb scale elevation < T = 115; VRIN score < T = 100; and F scale < T = 110 were included.

were included in the final analyses. The sample was comprised of 23 males (46%) and 27 females (54%). The participants ranged in age from 15 to 75 years (M = 31 years, SD = 10.8 years). The majority of the participants (60%) completed at least 12 years of education at the time of the assessment, with years of education ranging from eight to 17 years. Forty-two percent of the participants were unemployed at the time of the assessment, 24% were students, and 4% were homemakers. Socioeconomic status was classified according to the Blishen Socioeconomic Index (Blishen, Carroll & Moore, 1987). Of the portion of the sample who were gainfully employed, the mean SES Index was 38.73 (SD = 10.9), which falls in the second lowest of the six class intervals outlined by Blishen et al. (1987) and can be categorized as "lower-middle class".

Twenty percent of the participants in this sample reported receiving previous psychological or psychiatric treatment. A small proportion of those individuals who had received previous treatment may have been treated specifically for symptoms of PTSD, however, information concerning the type of treatment received and the efficacy of the interventions in alleviating PTSD symptoms was unavailable.

On average, participants were neuropsychologically assessed two and a half years following their involvement in the MVA (M = 32.0 months; SD = 25.5 months). Loss of consciousness (LOC) and post-traumatic amnesia (PTA) were used as measures of the severity of head injury. Information regarding LOC and PTA was obtained from medical records when available, and from retrospective patient reports when documentation was lacking. Table 1 presents the distribution of LOC and PTA in the sample.

Table 1
Head Injury Severity

	N	Percent
Loss of Consciousness		
No LOC	14	28
less than 1 hour	26	52
1 to 24 hours	1	2
1 to 5 days	1	2
6 to 10 days	6	12
11 to 14 days	1	2
more than 2 weeks	1	2
Posttraumatic Amnesia		
No PTA	19	38
less than 1 hour	12	24
1 to 24 hours	4	8
1 to 3 days	3	6
4 to 7 days	4	8
1 to 6 weeks	6	12
more than 6 weeks	2	4

As can be seen from the above table, 80% of the sample experienced no LOC or LOC for less than one hour. Sixty-two percent of participants experienced no PTA or PTA for a duration of less than one hour. Thus, the majority of the participants in the sample experienced mild head injuries (according to the classification of head injury severity previously outlined). However, it should be noted that a substantial proportion (between 18% and 30%) of the sample suffered severe head injuries.

Forty percent of participants were taking prescription medication at the time of the assessment, most commonly analgesics, anti-depressants, and anxiolytics. The physical injuries reported in participant's medical records are presented in Table 2. As can be seen below, the severity of physical injuries ranged from mild to severe. The length of hospitalization may provide some indication of the severity of physical injuries experienced by the participants. Table 3 presents the distribution of the participant's length of hospitalization.

Table 2
Summary of Physical Injuries

	N	Percent
Lacerations and/or bruises	29	58
Bone and/or skull fracture	26	52
Muscle strain and/or whiplash	21	42
Concussion	13	26
Collapsed lung and/or lung contusions	5	10
Internal organ injuries	5	10
Dental injuries	5	10
Cerebral hemorrhage	2	4
Cerebral edema	2	4
Hemiparesis	2	4
Visual loss	2	4
Hearing loss	2	4
Facial nerve palsy	1	2
Bone dislocation	1	2
Seizures	1	2
Ruptured artery	1	2
Limb amputation	1	2

Table 3
Length of Hospitalization

	N	Percent
Not hospitalized	18	36
1 to 7 days	11	22
1 to 4 weeks	13	26
1 to 2 months	5	10
2 to 3 months	2	4
more than 3 months	1	2

2.2 Procedure

Following a clinical interview by a registered psychologist specialized in clinical neuropsychology, participants were administered a routine battery of psychological and neuropsychological tests. Based upon clinical judgment, various tests were added to or deleted from the routine battery for each participant.

a. PTSD Measures

i) The Penn Inventory of PTSD

This 26-item self-report measure of the severity of PTSD was designed for use in both civilian and veteran populations (Hammarberg, 1992). The 26 items which comprise the Penn Inventory were written to correspond closely to the DSM-III-R criteria of PTSD, and thus this measure may be used for diagnostic purposes. Each item is rated from 0-3 on a Likert-type scale and thus a continuous total score ranging from 0-78 may be obtained by summing the scores on each item.

The Penn Inventory may also be fitted with a cut-off score for the purposes of dichotomous decision making. Its clinical utility is evident in its excellent sensitivity and specificity. Sensitivity refers to a test's ability to identify "true-positives", while specificity refers to a test's ability to identify "true-negatives". Through three independent studies, Hammarberg (1992) determined the psychometric properties of the Penn Inventory. Using the maximal cut-off score of 35, the overall sensitivity of the Penn Inventory was .95 and the overall specificity was .89. The overall efficiency or "hit-rate" of this measure, which refers to the proportion of both positive and negative diagnoses that were correctly identified by utilizing the cut-off score of 35, was 93%. The internal consistency (Cronbach's alpha of .94) and test-retest reliability ($r = .96$) of this measure have also been established. The construct validity of the Penn Inventory was established by correlating scores on this measure with scores on other tests thought to measure similar and dissimilar constructs. Within the standardization sample, the Penn Inventory was found to be highly correlated with the Beck Depression Inventory ($r = .84$) and the Beck Anxiety Inventory ($r = .56$). There was also a high correlation between the Penn Inventory and two "first-generation" measures of combat-related PTSD, the Impact of Events Scale and the Mississippi Scale ($r = .72$ and $r = .85$, respectively). The correlation between the Penn Inventory and the Combat Exposure Scale, a measure of the duration and type of combat experiences to which war veterans are frequently exposed, was relatively low ($r = .37$), suggesting that the Penn Inventory measures more than simply the length and severity of a traumatic exposure.

ii) MMPI-2 PK Scale

This 46-item scale has an internal consistency of .86 in normal samples and a test-retest reliability of .88. The suggested maximal cut-off score for the MMPI-2 PK scale is a T-score of 83 in males and a T-score of 79 in females (Lyons & Keane, 1992). This scale has been shown across a number of studies to have very good diagnostic specificity and sensitivity as well as clinical utility. The items which comprise this scale reflect great emotional turmoil. Some items deal with anxiety, worry, sleep disturbance, guilt and depression. Other items reflect the presence of unwanted and disturbing thoughts, lack of emotional control and feelings of being misunderstood and mistreated (Graham, 1990).

iii) MMPI-2 PS Scale

Items on this 60-item scale significantly overlap with those of the PK scale, and this scale has only been used experimentally thus far. Internal consistency coefficients for males and females in the MMPI-2 standardization sample were .89 and .91, respectively. Although the 60 items listed in the MMPI-2 manual for the PS scale comprise an experimental grouping only, there continues to be ongoing research on these items by Schlenger and his associates, as well as other researchers in the field of PTSD.

b. Cognitive and Neuropsychological Measures

i) Wechsler Adult Intelligence Scale - Revised (WAIS-R)

The WAIS-R (Wechsler, 1981) is one of the most frequently administered measures of overall intellectual functioning. It is suitable for individuals aged 16 to 74 years. The WAIS-R is composed of 11 subtests; six subtests are related to verbal abilities (Verbal Intelligence Quotient; VIQ) and the remaining five subtests are related to non-verbal, or performance skills (Performance Intelligence Quotient; PIQ). Scores on the VIQ and PIQ are summed to provide the Full Scale Intelligence Quotient (FSIQ). The WAIS-R FSIQ, VIQ, and PIQ scores were used in the analyses.

ii) Wisconsin Card Sorting Test (WCST)

The WCST (Heaton, 1981) assesses the ability to form abstract concepts, and shift and maintain the mental set. This measure requires participants to match the cards in two decks to four "key cards". Although they were not informed how to match the cards (i.e. by shape, number or color), they were told whether their responses were correct or

incorrect. After ten consecutive correct matches are achieved (i.e. completion of a "category"), the examiner changes the "correct" matching principle. Included in the analyses were the participants' scores for the total number of correct categories achieved, the number of perseverative errors made, and the number of "failures to maintain set".

iii) Rey Auditory Verbal Learning Test (RAVLT)

The RAVLT (Rey, 1964) assesses immediate verbal memory, new learning, susceptibility to interference, and recognition memory. A list of 15 nouns were read aloud to participants across five consecutive trials. Each trial was followed by a test of free-recall. A second list of 15 different words was then presented, followed by a free-recall test of that list. Immediately following this, recall of the original word list was assessed, and recall was measured again following a 30-minute delay. Finally, a test of visual recognition for the words on the original list was given. Analyses were performed on the total number of words recalled across the five administration trials.

iv) Rey Visual Design Learning Test (RVDLT)

The RVDLT (Rey, 1964) assesses immediate visual memory, new learning, and recognition memory. The test consists of 15 geometrical designs, which were presented to participants at a rate of two seconds per design. Participants were then asked to draw as many of the designs as they could recall. This procedure was repeated five times, followed by a test of visual recognition memory. The total score across the learning trials was utilized in the analyses.

v) Rey-Osterrieth Complex Figure Test

The Rey-Osterrieth Complex Figure Test (Rey, 1941) assesses long-term memory for visual material, as well as graphomotor functioning and visual-perceptual skills. Participants were asked to copy a complex geometrical drawing and then to reproduce the drawing from memory following a 30-minute delay. Only the long-delay recall score was analyzed for the purposes of this study.

vi) California Verbal Learning Test (CVLT)

The CVLT (Delis, Kramer, Kaplan & Ober, 1987) is another verbal list-learning task in which a list of words from four semantic categories were presented across five learning trials, followed by the presentation of an interference list, and then tests of free-

recall and cued-recall of the original after both a short and long delay. The summary score, or the total number of words learned across the five learning trials, was used in the analyses.

vii) Paced Auditory Serial Attention Test (PASAT)

The PASAT (Gronwall, 1977) is a very sensitive measure of mental speed, mental control, computational ability and attention (Spreen & Strauss, 1991). Participants were requested to listen to a random series of numbers ranging from 1 to 9, presented at varying speeds, and add each number to the number immediately preceding it. The total number of correct responses given in the first trial (2.4 second interval) was used in the analyses.

CHAPTER 3
RESULTS

The following chapter is divided into two main sections. The first section provides information concerning the statistical considerations and alpha levels used in the subsequent analyses. The second section is comprised of the results of the statistical analyses relevant to the central hypotheses. This entails a reiteration of each hypothesis followed by the relevant findings. The results of the exploratory analyses are contained in the final section of this chapter.

3.1 Statistical Considerations and Alpha Levels

All analyses were conducted using the SPSS for Windows 6.12 statistical software package. To control for experiment-wise error, an alpha level of .05 was adopted for predicted comparisons. Bonferroni-corrected alpha levels were adopted for unpredicted comparisons. Correlational effect size was determined using Cohen's (1988) criteria for behavioral science research, where correlations of .50, .30, and less than .30 are considered large, medium, and small correlations, respectively.

3.2 Tests of Hypotheses

Hypothesis 1 - *A proportion of this sample of head-injured MVA survivors will display PTSD, regardless of neurogenic amnesia for the traumatic event.*

The results of a frequency analysis indicate that 58% percent (n=29) of this sample of brain-injured adults scored at or above the diagnostic cut-off score of 35 on the Penn Inventory (PTSD subgroup). Males and females were equally represented in the PTSD subgroup (48% and 52%, respectively). A Student's t-test analysis revealed no significant differences in the mean age, educational level, duration of time since injury, or head injury severity of the individuals who comprised the PTSD subgroup and the remainder of the sample. Demographic data for the PTSD subgroup, including age, education level and duration of time since the injury, are presented in Table 4.

Table 4
Demographic Data for the PTSD Subgroup

	Range	Mean	Standard Deviation
Age at Testing	15-75 years	31 years	12.4 years
Months since MVA	2-168 months	34.8 months	30.0 months
Years of Education	8-16 years	11.8 years	2.3 years

The distribution of head injury severity variables for the PTSD subgroup are presented in Table 5.

Table 5
Distribution of LOC and PTA for PTSD and Non-PTSD Subgroups

	PTSD Subgroup (n = 28) n (%)	Non-PTSD Subgroup (n = 21) n (%)
Loss of Consciousness		
No LOC	8 (27.6)	6 (28.6)
less than 1 hour	14 (48.3)	12 (57.1)
1 to 24 hours	0 (0.0)	0 (0.0)
1 to 5 days	0 (0.0)	1 (4.8)
6 to 10 days	5 (17.2)	1 (4.8)
11 to 14 days	1 (3.4)	1 (4.8)
Posttraumatic Amnesia		
No PTA	10 (34.5)	9 (42.9)
less than 1 hour	9 (31.0)	3 (14.3)
1 to 24 hours	1 (3.4)	3 (14.3)
1 to 3 days	1 (3.4)	2 (9.5)
4 to 7 days	3 (10.3)	1 (4.8)
1 to 6 weeks	4 (13.8)	2 (9.5)
more than 6 weeks	1 (3.4)	1 (4.8)

It is clear from the above table that the head injury severity of the PTSD subgroup ranged from mild to severe. The distribution of retrograde amnesia (RA) in this subgroup is presented in Table 6. The extent of RA in the individuals in the PTSD subgroup is particularly important as this indicates neurogenic amnesia for the traumatic event. As can be seen in the following table, five participants or 17% of the PTSD subgroup experienced retrograde amnesia greater than 1 hour (i.e., neurogenic amnesia for the traumatic event).

Table 6
Distribution of Retrograde Amnesia (RA) in PTSD Subgroup

	N	Percent
No RA	17	58.6
less than 1 hour	7	24.1
1 to 24 hours	1	3.4
1 to 7 days	2	6.9
1 to 6 weeks	2	6.9

Hypothesis 2 - *There will be no difference in the symptoms of PTSD in head-injured individuals with neurogenic amnesia for the traumatic event and those without neurogenic amnesia for the traumatic event.*

No significant differences were found in the mean Penn Inventory score of the PTSD subgroup with neurogenic amnesia for the traumatic event (PTSD+NA) and the PTSD subgroup without neurogenic amnesia for the traumatic event (PTSD-NA), according to a Student's t-test analysis (PTSD+NA Subgroup $\underline{M} = 42.8$, $\underline{SD} = 4.1$; PTSD-NA Subgroup $\underline{M} = 41.3$, $\underline{SD} = 5.8$).

The 26 items of the Penn Inventory were then classified on a logical basis into: a) re-experiencing symptoms[4], b) avoidance and numbing symptoms[5], and c) increased arousal symptoms[6]. Inter-item reliability coefficients[7] were derived for the three subscales: re-experiencing symptoms $\underline{r} = .53$, avoidance and numbing symptoms $\underline{r} = .74$, and increased arousal symptoms $\underline{r} = .40$. A Student's t-test analysis was used to test for differences in the above symptom clusters between the PTSD+NA subgroup and the PTSD-NA subgroup. Although the results were not significant, there was a trend for the

[4] Corresponds with symptom cluster B in the DSM-IV diagnostic criteria for PTSD. Includes Penn Inventory items 7,8,13,17,21,24.

[5] Corresponds with symptom cluster C in the DSM-IV diagnostic criteria for PTSD. Includes Penn Inventory items 1,2,3,5,10,12,14,15,18,20,22,23,25,26.

[6] Corresponds with symptom cluster D in the DSM-IV diagnostic criteria for PTSD. Includes Penn Inventory items 4,6,9,11,16,19.

[7] Given that the items were classified on a logical basis according to DSM-IV criteria of PTSD, only moderate inter-item correlations were expected; if an individual experiences one of the symptoms of Cluster D (i.e., difficulty concentrating), there is no reason to expect that he or she would demonstrate all of the other Cluster D symptoms (i.e., sleep difficulties, anger outbursts, hypervigilance, or exaggerated startle response), at a similar level of severity.

PTSD-NA subgroup to endorse more re-experiencing symptoms (t (27) = 1.84, p = .076) than the PTSD+NA subgroup. Another trend emerged in which participants in the PTSD+NA subgroup rated themselves as experiencing more avoidance and numbing symptoms (t (27) = -1.83, p = .078) than participants in the PTSD-NA subgroup. Given the small number of participants in the PTSD+NA and PTSD-NA subgroups, the above trends may be very meaningful. There were no significant differences between the groups in their endorsement of symptoms of increased physiological arousal.

Hypothesis 3 - *The Penn Inventory will be highly correlated with the MMPI-2 PK and PS scales. The MMPI-2 PK and PS scales will be highly correlated with each other.*

Pearson Product Moment correlations revealed a strong relationship between the Penn Inventory and the MMPI-2 PK and PS Scales (r = .63 and r = .67, respectively). Not surprisingly, a high correlation exists between scores on the PK and PS scales (r = .94).

Hypothesis 4 - *There will be a positive and significant relationship between the severity of PTSD symptomatology and a) exposure to death or disfigurement, b) personal history of previous trauma, c) history of previous psychological or psychiatric treatment, d) increased severity of physical injuries*[8]*, and e) increased age.*

A standard multiple regression was performed between total score on the Penn Inventory as the dependent variable and exposure to death or disfigurement, history of previous trauma or psychological treatment, severity of physical injuries and age as independent variables. The results of this analysis indicate that this combination of variables did not contribute significantly to prediction of the total score on the Penn Inventory. When the relationship between scores on the Penn Inventory and each of these

[8] Due to the moderate correlation between the number of physical injuries reported in participant's medical files and the duration of their hospitalization (r = .45), length of hospitalization was used as a measure of severity of physical injuries.

variables was analyzed independently, none of the variables were significantly related to the endorsement of PTSD symptoms.

Table 7

Correlations Between the Penn Inventory and Demographic, Premorbid, and Injury Severity Variables

Variables	Penn Inventory Total Score	
	r	p
Exposure to Death	- .12	.20
Exposure to Disfigurement	- .04	.39
Previous Treatment	- .09	.27
Previous Trauma	- .12	.20
Physical Injury Severity	.06	.34
Age	.04	.40

Hypothesis 5 - *Scores on measures of attention, memory, and learning will be negatively related to the severity of PTSD symptoms.*

No significant relationships were found between total scores on the Penn Inventory and the FSIQ, VIQ or PIQ of the WAIS-R, total categories, perseverative errors or failure to maintain set on the WCST, total scores on the RAVLT or RVDLT, 30-minute recall score of the Rey-Osterrieth Complex Figure Test, total score on the CVLT, or score on the first administration trial of the PASAT. The results of the correlational analyses between the Penn Inventory and scores on the aforementioned measures of attention, memory and learning are presented in Table 8.

Table 8: Correlations Between Penn Inventory and Scores on the Cognitive and Neuropsychological Tests

Variables	Penn Inventory (Total Score)	
	r	p
WAIS-R FSIQ	- .14	.37
WAIS-R VIQ	- .08	.64
WAIS-R PIQ	- .24	.15
WCST Categories	- .05	.74
WCST Perseverative Errors	- .09	.56
WCST Failure to Maintain Set	- .08	.66
RAVLT Total Score	- .07	.74
RVDLT Total Score	- .22	. 25
R-O Complex Figure - Recall	- .01	. 94
CVLT - Total Score	- .13	. 49
PASAT 2.4 Second Trial	- .06	. 70

A correlation matrix including the Penn Inventory, the MMPI-2 PK and PS scales, and the cognitive and neuropsychological variables of interest is presented in Appendix C.

3.3 Exploratory Analyses

Exploratory analyses of the construct validity of the Penn Inventory were conducted. A very high correlation was found between scores on the Penn Inventory and scores on the Beck Depression Inventory ($r = .80$). Moderate to high correlations were found between scores on the Penn Inventory and scores on numerous Clinical and Content Scales of the MMPI-2 (see Table 9 and Table 10).

Table 9
Correlations Between Penn Inventory and Clinical Scales of the MMPI-2

Scale	Correlation	p
1 (Hypochondriasis)	.45	.002
2 (Depression)	.58	.000
3 (Hysteria)	.33	.032
4 (Psychopathic Deviate)	.45	.002
5 (Masculinity-Femininity)	.30	.051
6 (Paranoia)	.48	.001
7 (Psychasthenia)	.60	.000
8 (Schizophrenia)	.59	.000
9 (Hypomania)	.06	.713
0 (Social Introversion)	.53	.000

Table 10
Correlations Between Penn Inventory and Content Scales of the MMPI-2

Scale	Correlation	p
Content Scales		
ANX (Anxiety)	.70	.000
FRS (Fears)	.24	.129
OBS (Obsessiveness)	.41	.007
DEP (Depression)	.67	.000
HEA (Health Concerns)	.44	.003
BIZ (Bizarre Mentation)	.36	.016
ANG (Anger)	.34	.028
CYN (Cynicism)	.27	.076
ASP (Antisocial Practices)	.11	.474
TPA (Type A)	.36	.019
LSE (Low Self-Esteem)	.42	.005
SOD (Social Discomfort)	.40	.008
FAM (Family Problems)	.41	.006
WRK (Work Interference)	.59	.000
TRT (Negative Treatment Indicators)	.58	.000

CHAPTER 4
DISCUSSION

There exists ongoing debate as to the appropriateness of the diagnosis of PTSD when it occurs in the presence of a traumatic head injury. In particular, Sbordone (1992) asserts that PTSD is theoretically incompatible with neurogenic amnesia for a traumatic event. However, no research to date has investigated the presence of PTSD in a sample of head-injured adults. To that end, the present study evaluated the endorsement of PTSD symptoms on the Penn Inventory in a sample of 50 adult, head-injured survivors of motor vehicle accidents who were referred for neuropsychological evaluation of suspected brain injury. Further, the relationship between the Penn Inventory and the MMPI-2 scales of PTSD were assessed, as was the extent to which premorbid, demographic, head injury severity, and neuropsychological variables were related to scores on the Penn Inventory. Exploratory analyses of the construct validity of the Penn Inventory were also conducted.

4.1 Findings Relevant to the Main Hypotheses

It is significant that 58% of this sample of adult MVA survivors referred for neuropsychological assessment of suspected brain injury scored within the clinical range on the Penn Inventory of PTSD. Although recent case studies (Layton & Wardi-Zonna, 1995; Horton, 1993, and McMillan, 1991) have provided evidence of the co-occurrence of PTSD and TBI, the current finding of scores above the Penn Inventory cut-off for PTSD in over half of the head-injured participants evaluated in this study was unexpected. Further, 10% of the total sample (five participants) obtained scores within the clinically significant range on the Penn Inventory despite experiencing neurogenic amnesia for the traumatic event. This finding directly contradicts Sbordone's (1992) theory that PTSD and head injury associated with neurogenic amnesia are mutually incompatible. Rather, it appears as though an individual may develop both disorders following a single traumatic event, such as involvement in an MVA.

The second hypothesis, in which it was predicted that the pattern of symptom endorsement on the Penn Inventory would be similar in head-injured individuals with neurogenic amnesia for the traumatic event (PTSD+NA) and head-injured individuals who were able to recall the traumatic event (PTSD-NA), was largely supported. The

findings indicate that both groups evidenced a similar level of PTSD symptom severity as measured by the Penn Inventory. However, it is notable that in spite of a relatively small sample size, there was a trend for the PTSD-NA subgroup to acknowledge more re-experiencing symptoms in comparison to the PTSD+NA subgroup, and a trend for the PTSD+NA subgroup to endorse more symptoms of increased arousal and emotional numbing than the PTSD-NA subgroup. There were no group differences in the ratings of increased physiological arousal symptoms.

Layton and Wardi-Zonna (1995) proposed that in the event of a head-injury associated with neurogenic amnesia, the declarative memory system becomes dysfunctional while the nondeclarative system continues to operate. Thus, due to the assumed intact functioning of the nondeclarative memory, an individual may be behaviorally changed as a function of an event for which there is no conscious recollection. In particular, the nondeclarative memory system is thought to mediate autonomic responses, which in turn, could be associated with the PTSD symptoms of avoidance of stimuli associated with the trauma, increased physiological arousal, and hyperalertness. In contrast, secondary traumatization phenomenon might explain the presence of PTSD re-experiencing symptoms in individuals who, due to neurogenic amnesia, have no conscious recollection of the traumatic experience. The recently expanding literature on secondary traumatization suggests that an individual may develop PTSD symptoms subsequent to being described the details of a traumatic event, despite never having personally experienced the traumatic event. Thus, an individual rendered unconscious during a traumatic event may subsequently report re-experiencing symptoms of PTSD, such as nightmares of the event or flashbacks, if they are exposed to pictures or descriptions of the event or perhaps if they imagine themselves in that situation. Nonsignificant trends were evident in the patterns of symptom endorsement on the Penn Inventory by the PTSD+NA and PTSD-NA subgroups. However, given the small number of individuals in the PTSD+NA and PTSD-NA subgroups, these nonsignificant trends may be practically meaningful. These results would appear to support Layton and Wardi-Zonna's (1995) theory regarding the role of nondeclarative memory in the development of the avoidance symptoms of PTSD in individuals with neurogenic

amnesia for the traumatic event. However, further research is required to confirm this finding.

The observed high correlations between scores on the Penn Inventory and the MMPI-2 PK and PS scales were expected, and support the construct validity of the Penn Inventory in this sample. Correspondingly, given the considerable overlap in the items which comprise the MMPI-2 PK and PS scales, the observed highly significant relationship between these two measures was anticipated. This finding suggests that the use of the experimental MMPI-2 PS as well as the MMPI-2 PK scale, which has already been shown to be psychometrically sound and clinically useful, would be redundant.

The absence of a significant relationship between the severity of PTSD symptoms and premorbid, demographic and injury-related variables was unexpected. Exposure to death or disfigurement, personal history of previous trauma, history of psychological or psychiatric treatment, increased severity of physical symptoms and increased age, which have been cited in the literature as important predisposing factors (Green et al., 1989; Martini et al., 1990; McFarlane, 1988a; McFarlane, 1988b; Breslau, et al., 1991) were not related to the endorsement of PTSD symptoms on the Penn Inventory in this sample. It is possible that different factors are related to the expression of PTSD symptomatology in head-injured individuals. However, it is equally likely that a significant relationship between the preceding predisposing factors and PTSD symptom endorsement on the Penn Inventory was not evident given the low occurrence of these events and the study's moderate sample size.

The results of this study also suggest that the endorsement of PTSD symptoms as measured by the Penn Inventory was not related to performance on measures of attention, memory and learning. This finding is consistent with the notion that PTSD and TBI are different phenomenon and that the Penn Inventory is relatively insensitive to the presence and severity of neuropsychological symptoms.

Exploratory analyses confirmed the significant relationship between the Penn Inventory and the Beck Depression Inventory, as well as many of the clinical scales of the MMPI-2. Taken together, these results support the construct validity of the Penn Inventory in this sample.

A major implication of these findings is that all individuals involved in MVAs who have suffered head injuries should be assessed for PTSD, regardless of whether they recall the accident. Accurate assessment of relevant neurological and psychological contributions to behavioral, cognitive and emotional changes in individuals head-injured in a MVA is crucial. The results of this study support the use of the Penn Inventory in assessing PTSD symptoms in head-injured samples.

The aforementioned findings also have important implications for therapeutic interventions with head-injured individuals with PTSD. As discussed by Layton and Wardi-Zonna (1995), therapies directed at re-activating memory for the traumatic event, which is a common approach with patients with PTSD who display functional amnesia for the event, is contraindicated in head-injured populations. Within groups of individuals with PTSD who have been head-injured, it is not possible to equate a failure to recall important aspects of the trauma with emotional defenses such as avoidance or denial. Instead, treatments aimed at de-sensitizing patients to the anxiety-provoking cues and memories of the traumatic incident are more likely to be beneficial according to the multiple memory system view of PTSD and TBI. A better understanding of the relationship between PTSD and cognitive abilities may inform clinicians about the formation of particular symptoms and the patient's capacities to discuss and modify them through both insight-oriented and more behaviorally-based exposure therapies.

4.2 Caveats

Although the number of participants in the present study is reasonable compared to the sample sizes of other studies of PTSD, it is still relatively small by general research standards. Thus, the generalizability of these results may be limited. Replication studies may be useful in clarifying whether the obtained results accurately portray the relationship between PTSD symptoms and head injury.

Secondly, the retrospective nature of the present study is problematic in that some important information obtained was difficult to verify. Further, due to this retrospective design, it was impossible to obtain some important information which could have impacted the results of the study. In particular, there was limited information regarding the intensity of exposure to the traumatic event, pre-morbid functioning, or the adequacy

of social supports, all of which have been shown to be related to the development of PTSD (Ursano, Fullerton, Kao & Bhartiya, 1995). A prospective research design would have allowed for an evaluation of these variables. Similarly, the findings of the present study are limited by the lack of neuroimaging results which might have confirmed the type and severity of neurological injury.

Thirdly, despite the practicality of a self-report measure of PTSD, such as the Penn Inventory, for research purposes, the lack of a semi-structured interview to further explore the nature of the reported symptoms or corroborative reports from family members to validate the participants' self-report, limit the generalizability of these results. The possibility of an artificial inflation of scores on the Penn Inventory due to participants responding in regards to previous traumas, in addition to symptoms associated with the MVA, must also be acknowledged.

Finally, it is possible that there is a unique pattern of PTSD symptom endorsement on the Penn Inventory in this head-injured sample due to the fact that all of the participants were involved in litigation for their injuries. In a review of the literature, Evans (1992) reported that litigation does not typically influence test performance or resolution of difficulties in head-injured individuals. Even upon settlement of their cases, these persons continue to experience difficulties for extended periods of time. However, it is conceivable that involvement in litigation, which commonly involves the repetition of the details of the traumatic experience on many different occasions, may serve to intensify and prolong the symptoms of psychological distress. Thus, it is possible that the present results are not applicable to non-litigating populations.

4.3 Conclusions and Directions for Future Research

The present study demonstrated that PTSD symptoms as measured by the Penn Inventory may occur in head-injured individuals, even in the presence of neurogenic amnesia for the traumatic event. Future research may proceed in several directions. First, evaluation of nondeclarative memory functioning in head-injured individuals with and without PTSD would serve to validate Layton and Wardi-Zonna's (1995) theory. Similarly, further exploration of the secondary traumatization hypothesis is necessary. For example, individuals with neurogenic amnesia could be interviewed regarding the

means by which they were exposed to the details of their traumatic experience. Well-controlled investigations of the relationship between PTSD and neuropsychological abilities are also required. Finally, explorations regarding how neurological damage affects an individual's ability to deal with psychological trauma are warranted.

Note: To obtain further information concerning the raw data and coding scheme utilized in this study, please contact Ms. Laura Shepard or Dr. Esther Strauss, Department of Psychology, University of Victoria.

REFERENCES

American Psychiatric Association. (1980). Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

Binder, L. (1986). Persisting symptoms after mild head injury: A review of the postconcussive syndrome. Journal of Clinical and Experimental Neuropsychology, 8, 323-346.

Blishen, B.R., Carroll, W.K. & Moore, C. (1987). The 1981 socioeconomic index for occupations in Canada. Canadian Review of Sociology and Anthropology, 24(4), 465-488.

Bremmer, J.D., Scott, T.M., Delaney, R.C., Southwick, S.M., Mason, J.W., Johnson, D.R., Innis, R.B., McCarthy, G., & Charney, D.S. (1993). Deficits in short-term memory in posttraumatic stress disorder. American Journal of Psychiatry, 150, 1015-1019.

Breslau, N., Davis, G.C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. Archives of General Psychiatry, 48, 216-222.

Brom, D., Kleber, R.J., & Hofman, M.C. (1993). Victims of traffic accidents: Incidence and prevention of post-traumatic stress disorder. Journal of Clinical Psychology, 49, 131-140.

Brooks, D.N. & Baddeley, A. (1976). What can amnesic patients learn? Neuropsychologia, 14, 111-122.

Burstein, A. (1989). Post-traumatic stress disorder in victims of motor-vehicle accidents. Hospital and Community Psychiatry, 40, 295-297.

Butcher, J.N., Dahlstrom, W.G., Graham, J.R., Tellegen, A.M., & Kaemmer, B. (1989). MMPI-2: Minnesota Multiphasic Personality Inventory - 2. Manual for Administration and Scoring. Minneapolis: University of Minnesota Press.

Cannon, D.S., Bell, W.E., Andrews, R.H., & Finkelstein, A.S. (1987). Correspondence between MMPI PTSD measures and clinical diagnosis. Journal of Personality Assessment, 51, 517-521.

Cohen, P. (1988). Statistical Power Analysis for the Behavioral Sciences (2nd Ed.). New York: Academic Press.

Crovitz, H.F. (1987). Techniques to investigate post-traumatic and retrograde amnesia after head injury. In H.S. Levin and H.M. Eisenberg (Eds.), Neurobehavioral Recovery from Head Injury. pp. 330-340. New York: Oxford University Press.

Dalton, J.E., Pederson, S.L., & Ryan, J.J. (1989). Effects of post-traumatic stress disorder on neuropsychological test performance. International Journal of Clinical Neuropsychology, 11, 121-124.

Danieli, Y. (1994). Countertransference and trauma: Self-healing and training issues. In M. Williams & J. Sommer (Eds.). Handbook of Post-Traumatic Therapy. Westport, CT: Greenwood Press.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1987). The California Verbal Learning Test. New York: Psychological Corporation.

Diamond, R. & Rozin, P. (1984). Activation of existing memories in anterograde amnesia. Journal of Abnormal Psychology, 93, 98-105.

Dikmen, S., McLean, A. & Temkin, N. (1986). Neuropsychological and psychosocial consequences of minor head injury. Journal of Neurology, Neurosurgery & Psychiatry, 49, 1227-1232.

Everly, G., & Horton, A. (1989). Neuropsychology of post-traumatic stress disorder: A pilot study. Perceptual and Motor Skills, 68, 807-810.

Foa, E.B., Steketee, G., & Rothbaum, B.O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. Behavior Therapy, 20, 155-176.

Gabrieli, J.D.E., Milberg, W., Keane, M.M. & Corkin, S. (1990). Intact priming of patterns despite impaired memory. Neuropsychologia, 28, 417-427.

Gil, T., Calev, A., Greenberg, D., Kugelmass, S., & Lerer, B. (1990). Cognitive functioning in post-traumatic stress disorder. Journal of Traumatic Stress, 3(1), 29-45.

Goldstein, G., van Kammen, W., Shelley, C., Miller, D.J., & van Kammen, D.P. (1987). Survivors of imprisonment in the Pacific theater during World War II. American Journal of Psychiatry, 144, 1210-1213.

Graf, P. & Schacter, D.L. (1985). Implicit and explicit memory for new associations in normal and amnesic subjects. Journal of Experimental Psychology: Learning, Memory & Cognition, 11, 511-518.

Graham, J.R. (1990). MMPI-2: Assessing Personality and Psychopathology. New York: Oxford University Press.

Green, B.L., Grace, M.C., Lindy, J.D., Gleser, G.C., & Leonard, A. (1990). Risk factors for PTSD and other diagnoses in a general sample of Vietnam veterans. American Journal of Psychiatry, 147, 729-733.

Green, B.L., Lindy, J.D., Grace, M.C., & Gleser, G.C. (1989). Multiple diagnoses in posttraumatic stress disorder: The role of war stressors. Journal of Nervous and Mental Disease, 177, 329-335.

Gronwall, D.M.A. (1977). Paced Auditory Serial Attention Task: A measure of recovery from concussion. Perceptual and Motor Skills, 44, 367-373.

Guberman, A. (1994). An Introduction to Clinical Neurology. Boston: Little, Brown and Company.

Gurdijan, E.S. (1975). Recent developments in biomechanics, management, and mitigation of head injuries. In D.B. Tower (Ed.). The Nervous System. Vol. 2: The Clinical Neurosciences. New York: Raven Press.

Gurvits, T., Lasko, N., Schachter, S., Kuhne, A., Orr, S., & Pitman, R. (1993). Neurological status of Vietnam veterans with chronic posttraumatic stress disorder. Journal of Neuropsychiatry, 5, 183-188.

Haist, F., Musen, G. & Squire, L.R. (1991). Intact priming of words and nonwords in amnesia. Psychobiology, 19, 275-285.

Hammarberg, M. (1992). Penn Inventory for posttraumatic stress disorder: Psychometric properties. Psychological Assessment, 4, 67-76.

Heaton, R.K. (1981). Wisconsin Card Sorting Test. Odessa, FL: Psychological Assessment Resources.

Helzer, J.E., Robins, L.N., & McEvoy, L. (1987). Post-traumatic stress disorder in the general population: Findings of the Epidemiologic Catchment Area Survey. New England Journal of Medicine, 317, 1630-1634.

Hickling, E.J., & Blanchard, E.B. (1992). Post-traumatic stress disorder and motor vehicle accidents. Journal of Anxiety Disorder, 6, 285-291.

Horton, A. M. (1993). Posttraumatic stress disorder and mild head trauma: Follow-up of a case study. Perceptual and Motor Skills, 76, 243-246.

Hyer, L., O'Leary, W.C., Saucer, R.T., Blount, J., Harrison, W.R., & Boudewyns, P.A. (1986). Inpatient diagnosis of post-traumatic stress disorder. Journal of Consulting and Clinical Psychology, 54, 698-702.

James, B. (1994). Long-term treatment for children with severe trauma history. In M. Williams & J. Sommer (Eds.). Handbook of Post-Traumatic Therapy. Westport, CT: Greenwood Press.

Keane, T. M., Caddell, J.M., & Taylor, K.L. (1988). The Mississippi scale for combat-related PTSD: Three studies in reliability and validity. Journal of Consulting and Clinical Psychology, 56, 85-90.

Keane, T.M., Malloy, P.F., & Fairbank, J.A. (1984). Empirical development of an MMPI subscale for the assessment of combat-related posttraumatic stress disorder. Journal of Consulting and Clinical Psychology, 52, 888-891.

Kolb, B & Wishaw, I. (1990). Fundamentals of Human Neuropsychology (3rd Ed.). New York: W.H. Freeman and Company.

Koretzky, M.B., & Peck, A.H. (1990). Validation of cross-validation of the PTSD subscale of the MMPI with civilian trauma victims. Journal of Clinical Psychology, 46, 296-300.

Kroll, J., Habenicht, M., & Mackenzie, T. (1989). Depression and post-traumatic stress disorder in Southeast Asia refugees. American Journal of Psychiatry, 146, 1592-1597.

Kuch, K., Swinson, R.P., & Kirby, M. (1985). Post-traumatic stress disorder after car accidents. Canadian Journal of Psychiatry, 30, 426-427.

Kulka, R.A., Schlenger, W.E., Fairbank, J.A., Hough, R.L., Jordan, B.K., Marmar, C.R., & Weiss, D.S. (1988). National Vietnam Veterans Readjustment Study. Research Triangle, NC: Research Triangle Institute.

Layton, B.S., & Wardi-Zonna, K. (1995). Post-traumatic stress disorder with neurogenic amnesia for the traumatic event. The Clinical Neuropsychologist, 9, 2-10.

Levin, H., Mattis, S., Ruff, R., Eisenberg, H., Marshall, L., Taboddor, K., High, W. & Frankowski, R. (1987). Neurobehavioral outcome following minor head injury: A three-center study. Journal of Neurosurgery, 66, 234-243.

Lindy, J.D. (1988). Vietnam: A Casebook. New York: Brunner/Mazel.

Lindy, J.D., Green, B.L., & Grace, M. (1987). The stressor criterion and post traumatic stress disorder. Journal of Nervous and Mental Disease, 175, 269-272.

Lyons, J.A., & Keane, T.M. (1992). Keane PTSD Scale: MMPI and MMPI-2 update. Journal of Traumatic Stress, 5, 111-117.

Maloney, L.J. (1988). Posttraumatic stresses of women partners of Vietnam veterans. Smith College Studies in Social Work, 58, 122-143.

March, J.S. (1990). The nosology of posttraumatic stress disorder. Journal of Anxiety Disorders, 4, 61-82.

Martini, D.R., Ryan, C., Nakayama, D., & Ramenofsky, M. (1990). Psychiatric sequelae after traumatic injury: The Pittsburgh Regatta incident. Journal of the American Academy of Child and Adolescent Psychiatry, 29, 70-75.

McCann, I.L. & Pearlman, L.A. (1990). Vicarious traumatization: A framework for understanding the psychological effects of working with victims. Journal of Traumatic Stress, 3(1), 131-149.

McFarlane, A.C. (1988a). The etiology of post-traumatic stress disorders following a natural disaster. British Journal of Psychiatry, 152, 110-121.

McFarlane, A.C. (1988b). The longitudinal course of post-traumatic morbidity: The range of outcomes and their predictors. Journal of Nervous and Mental Disorders, 176, 30-39.

McMillan, T.M. (1991). Post-traumatic stress disorder and severe head injury. British Journal of Psychiatry, 159, 431-433.

Milner, B., Corkin, S. & Teuber, H.L. (1968). Further analysis of the hippocampal amnesic syndrome: A 14 year follow-up study of H.M. Neuropsychologia, 6, 215-234.

Mishkin, M., & Appenzeller, T. (1987). The anatomy of memory. Scientific American, 256, 80-89.

Musen, G. & Squire, L.R. (1992a). Nonverbal priming in amnesia. Memory and Cognition, 43, 281-194.

Nissen, M.J. & Bullemer, P. (1987). Attentional requirements of learning: Evidence from performance measures. Cognitive Psychology, 19, 1-32.

Norris, F.H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. Journal of Consulting and Clinical Psychology, 60, 409-418.

Ommaya, A.K., & Gennarelli, T.A. (1974). Cerebral concussion and traumatic unconsciousness. Brain, 97, 633-654.

Pitman, R.K., Altman, B., & Macklin, M.L. (1989). Prevalence of post-traumatic

stress disorder in wounded Vietnam veteran. American Journal of Psychiatry, 146, 667-669.

Rey, A. (1941). L'examen psychologique dans les cas d'encephalopathie traumatique. Archives de Psychologie, 28, 286-340.

Rey, A. (1964). L'Examen Clinique en Psychologie. Paris: Press Universitaire de France.

Russell, W.R. & Smith, A. (1961). Post traumatic amnesia in closed head injury. Archives of Neurology, 5, 16-29.

Sbordone, R.J. (1992). Distinguishing traumatic brain injury from post-traumatic stress disorder. The Neurolaw Letter, 1, 3.

Schacter, D.L. (1987). Implicit memory: History and current status. Journal of Experimental Psychology: Learning, Memory and Cognition, 13, 501-518.

Schacter, D.L. (1992). Understanding implicit memory: A cognitive neuroscience approach. American Psychologist, 47, 559-569.

Schacter, D.L., Cooper, L.A., Tharan, M. & Rubens, A.B. (1991). Preserved priming of novel objects in patients with memory disorders. Journal of Cognitive Neuroscience, 3, 118-131.

Schlenger, W., & Kulka, R.A. (1987). PTSD Scale Development for the MMPI-2. Research Triangle Park, NC: Research Triangle Institute.

Schlenger, W., Kulka, R., Fairbank, J., Hough, R., Jordan, B., Marmar, C., & Weiss, D. (1989). The prevalence of post-traumatic stress disorder in the Vietnam generation: Findings from the National Vietnam Veterans Readjustment study. Report from the Research Triangle Institute. Research Triangle Park, NC.

Schreiber, S., & Galai-Gat, T. (1993). Uncontrolled pain following physical injury as the core-trauma in post-traumatic stress disorder. Pain, 54, 107-110.

Shore, J.H., Tatum, E.L., & Vollmer, W.M. (1986). Psychiatric reactions to disaster: The Mount St. Helen's experience. American Journal of Psychiatry, 143, 590-595.

Sierles, F.S., Chen, J.J., McFarland, R.E., & Taylor, M.A. (1983). Posttraumatic stress disorder and concurrent psychiatric illness: A preliminary report. American Journal of Psychiatry, 140, 1177-1179.

Sierles, F.S., Chen, J.J., Messing, M.L., Besyner, J.K., & Taylor, M.A. (1986).

Concurrent psychiatric illness in non-Hispanic outpatients diagnosed as having posttraumatic stress disorder. Journal of Nervous and Mental Disease, 174, 171-173.

Slick, D.J., Hopp, G., & Strauss, E. (1995). Victoria revision of the Hiscock and Hiscock Digit Memory Test: Version 2.2. Test manual. Independent: Victoria.

Spitzer, R.L., Williams, J.B.W. & Gibbon, M. (1987). Structured Clinical Interview for DSM-III-R (SCID). New York: New York State Psychiatric Institute, Biometrics Research.

Spreen, O. & Strauss, E. (1991). The Compendium of Neuropsychological Tests: Administration, Norms and Commentary. New York: Oxford University Press.

Squire, L.R. (1987). Memory and brain. New York: Oxford University Press.

Squire, L.R. (1992). Declarative and nondeclarative memory: Multiple brain systems supporting learning and memory. Journal of Cognitive Neuroscience, 4, 232-243.

Squire, L.R. & Frambach, M. (1990). Cognitive skill learning in amnesia. Psychobiology, 18, 109-117.

Sutker, P.B., Winstead, D.K., Galina, Z.H., & Allain, A.N. (1990). Assessment if long-term psychosocial sequelae among POW survivors of the Korean conflict. Journal of personality Assessment, 54, 170-180.

Sutker, P.B., Winstead, D.K., Galina, Z.H., & Allain, A.N. (1991). Cognitive deficits and psychopathology among former prisoners of war and combat veterans of the Korean Conflict. American Journal of Psychiatry, 148, 67-72.

Uddo, M., Vasterling, J.J., Brailey, K., & Sutker, P.B. (1993). Memory and attention in combat-related post-traumatic stress disorder (PTSD). Journal of Psychology and Behavioral Assessment, 15, 43-52. _

Ursano, R.J., Fullerton, C.S., Kao, T., & Bhartiya, V. (1995). Longitudinal assessment of posttraumatic stress disorder and depression after exposure to traumatic death. Journal of Nervous and Mental Disease, 183, 36-42.

van Kammen, W.B., Christiansen, C., van Kammen, D.P., & Reynolds, C.F. (1990). Sleep and the POW experience: 40 years later. In E. Giller (Ed.). Biological assessment and treatment of PTSD (pp.161-172). Washington, DC: APA Press Inc.

Warrington, E.K. & Weiskrantz, L. (1968). New method of testing long-term retention with special reference to amnesic patients. Nature, 217, 972-974.

Warrington, E.K. & Weiskrantz, L. (1970). Amnesia: Consolidation or retrieval? Nature, 228, 628-630.

Warrington, E.K. & Weiskrantz, L. (1974). The effect of prior learning on subsequent retention in amnesic patients. Neuropsychologia, 12, 419-428.

Warrington, E.K. & Weiskrantz, L. (1978). Further analysis of the prior learning effect in amnesic patients. Neuropsychologia, 16, 169-176.

Wechsler, D. (1981). Wechsler Adult Intelligence Scale - Revised. New York: Psychological Corporation.

Wolfe, J., & Charney, D.S. (1991). Use of neuropsychological assessment in posttraumatic stress disorder. Psychological Assessment, 3, 573-580.

Zalewski, C., Thompson, W., & Gottesman, I. (1994). Comparison of neuropsychological test performance in PTSD, generalized anxiety disorder, and control Vietnam veterans. Assessment, 1, 133-142.

Zeitlin, S.B. & McNally, R.J. (1991). Implicit and explicit memory bias for threat in post-traumatic stress disorder. Behavior Research and Therapy, 29, 451-457.

APPENDICES

List of Appendices

APPENDIX A: DSM-IV Diagnostic Criteria for PTSD

APPENDIX B: Hypothesized Position Of PTSD in Memory System Model

APPENDIX C: Correlation Matrix for Scores on PTSD Scales and Scores on Cognitive and Neuropsychological Tests.

APPENDIX A: DSM-IV DIAGNOSTIC CRITERIA FOR PTSD

A. The person has been exposed to a traumatic event in which both if the following were present:
(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
(2) the person's response involved intense fear, helplessness, or horror.

B. The traumatic event is persistently re-experienced in one (or more) of the following ways:
(1) recurrent and intrusive distressing recollections of the event, including images, thoughts and perceptions.
(2) recurrent distressing dreams of the event.
(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening and when intoxicated).
(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

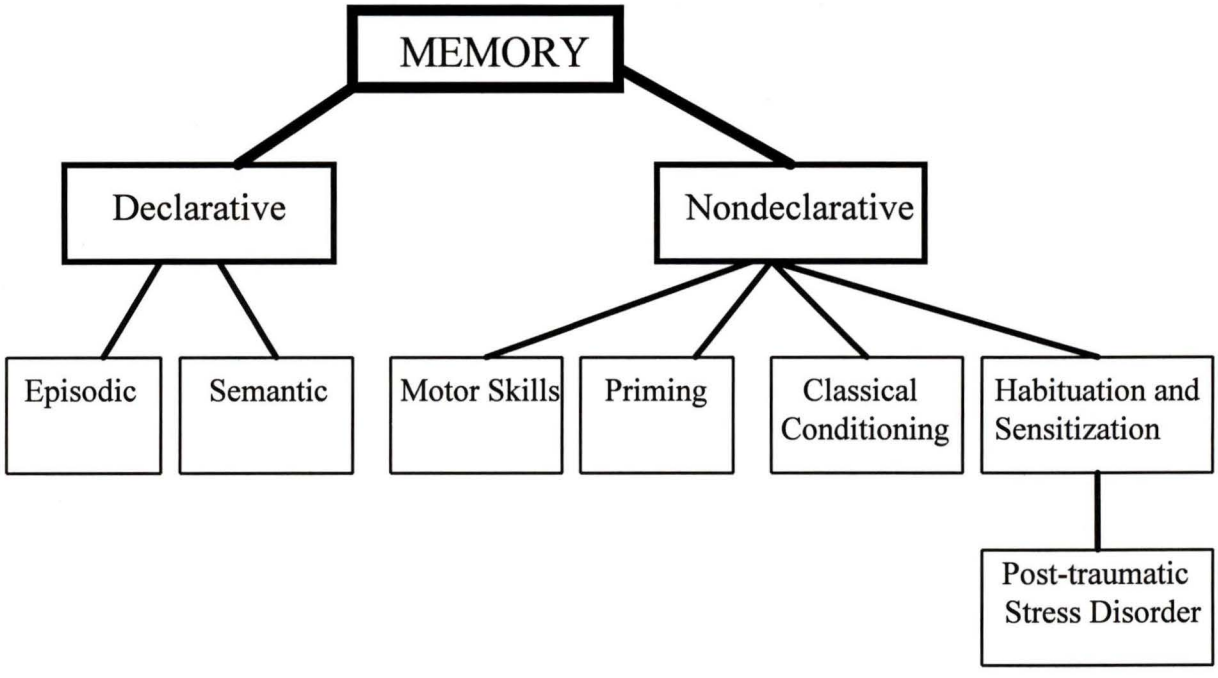
C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:
(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma.
(2) efforts to avoid activities, places or people that arouse recollections of the trauma.
(3) inability to recall an important aspect of the trauma.
(4) markedly diminished interest or participation in significant activities.
(5) feeling of detachment or estrangement from others.
(6) restricted range of affect (e.g., unable to have loving feelings).
(7) sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:
(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

APPENDIX B: HYPOTHESIZED POSITION OF PTSD IN MEMORY SYSTEM MODEL



APPENDIX C: CORRELATION MATRIX FOR SCORES ON PTSD SCALES AND SCORES ON COGNITIVE AND NEUROPSYCHOLOGICAL TESTS.

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Penn Inventory													
2. MMPI-2 PK	.63**												
3. MMPI-2 PS	.67**	.94**											
4. WAIS-R FSIQ	-.14	-.36*	-.39*										
5. WAIS-R VIQ	-.08	-.26	-.27	.92**									
6. WAIS-R PIQ	-.24	-.44**	-.46**	.82**	.56**								
7. WCST Categories	-.05	-.05	-.15	.14	.08	.12							
8. WCST Per. Errors	.09	.15	.17	-.03	.04	-.02	-.74**						
9. WCST FMS	.08	.10	.10	-.28	-.29	-.22	-.11	-.05					
10. PASAT 2.4	-.06	-.16	-.19	.37*	.34*	.36*	.22	-.08	.06				
11. RAVLT Total	-.07	-.21	-.08	.22	.20	.24	-.21	.07	.47	.25			
12. RVDLT Total	-.22	-.33	-.25	.41*	.36	.41*	-.00	-.11	-.27	.39	.53*		
13. R-O Recall	-.01	-.13	-.10	.30	.11	.47**	.03	-.08	-.15	.02	.25	.43	
14. CVLT Total	-.13	-.30	-.41	.61**	.59**	.49*	.43*	-.32	-.03	.26	.45	.41	-.13

NOTE: Penn Inventory = Total Score on the Penn Inventory; MMPI-2 PK = T-Score on MMPI-2 PK Scale; MMPI-2 PS = T-Score on MMPI-2 PS Scale; WAIS-R FSIQ = Wechsler Adult Intelligence Scale (Revised) Full Scale Intelligence Quotient; WAIS-R VIQ; Wechsler Adult Intelligence Scale (Revised) Verbal Intelligence Quotient; WAIS-R PIQ = Wechsler Adult Intelligence Scale (Revised) Peformance Intelligence Quotient; WCST Categories = Wisconsin Card Sorting Test Total Categories; WCST Per. Errors = Wisconsin Card Sorting Test Perseverative Errors; WCST FMS = Wisconsin Card Sorting Test Failure to Maintain Set; PASAT 2.4 = Paced Auditory Serial Addition Test 2.4 Second Delay Trial; RAVLT Total = Rey Auditory Verbal Learning Test Total Score (Trials 1-5); RVDLT Total = Rey Visual Design Learning Test Total Score (Trials 1-5); R-O Recall = Rey-Osterrieth Complex Figure Test 30-Minute Recall Score; CVLT Total = California Verbal Learning Test Total Score (Trials A1-5).

* $p < .05$.
** $p < .01$.

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The Robert and Douglas Vickery Graduate Award	1996

Publications and Presentations:

Janzen, L.A. & Nanson, J.L. (1993). Neuropsychological evaluation of pre-schoolers with fetal alcohol syndrome. Poster presentation at the Society for Research in Child Development, March 24-29, 1993, New Orleans, LA.

Hay, D., Janzen, L. & Soule, K. (1993). Children's affective responses and causal attributions in success and failure: Implications for self-esteem. Poster presentation at the Society for Research in Child Development, March 24-29, 1993, New Orleans, LA.

Janzen, L.A. & Nanson, J.L. (1994). A neuropsychological evaluation of pre-schoolers with fetal alcohol syndrome. Neurotoxicology and Teratology, 17(3), 273-279.

Publications and Presentations (continued):

Sherman, E., Janzen, L. & Joschko, M. (1995). Attention and Obsessive-compulsive behaviours in children with Tourette Syndrome. Paper presented at the International Neuropsychological Society, February 7-11, 1995, Seattle, WA.

Sherman, E., Shepard, L. & Joschko, M. (1996). Sustained attention and social functioning in children with Tourette Syndrome. Paper presented at the International Neuropsychological Society, February 14-17, 1996, Chicago, IL.

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Title of Thesis: The Prevalence of Post-Traumatic Stress Disorder In Head-Injured Adults

Author: [redacted]

Laura Ann Shepard
30 September 1996