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Psychological and neuropsychological correlates of Postconcussional Disorder

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PSYCHOLOGICAL AND NEUROPSYCHOLOGICAL CORRELATES OF POSTCONCUSSIONAL DISORDER

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ABSTRACT

Chronic symptoms of Postconcussional Disorder (PCD) occur in a significant minority of mild brain injury patients. The latest research suggests an interactionistic perspective as the most logical and empirically supported pathogenesis for the development and maintenance of PCD. The interactionistic perspective implicates organic factors in the development of acute symptoms of PCD, and psychological factors in the maintenance of chronic symptoms. Possible psychological factors relevant in the maintenance of PCD symptoms include a grief response, a coping hypothesis, and the development of dysfunctional coping loops. Providing support for a psychological etiology of symptom maintenance is research indicating reduction of chronic symptoms using cognitive behavioral techniques. The present study examined the effects of mild traumatic brain injury (MTBI), postconcussion symptom status, stress, and psychological distress on Paced Auditory Serial Addition Test (PASAT) performance. There were no significant main effects or interactions affecting PASAT performance. Secondarily, a modified distress index was developed using the subscales of the Personality Assessment Inventory (PMDI), based the scoring of the Brief Symptom Inventory Positive Symptom Distress Index. Postconcussive symptoms were moderately correlated with PMDI scores and with specific scales on the PAI (anxiety, anxiety related disorders, depression, somatic complaints, borderline features, stress, and nonsupport). In addition, a 2 (MTBI status) X 2 (PCD symptom status) X 2 (stress) ANOVA with PMDI as the dependent variable yielded main effects of stress and symptom status, but not MTBI, on PMDI scores. Limitations of this study and implications for future research are discussed.

INTRODUCTION

Each year approximately 2 million people in the United States suffer closed head injuries (CHI); approximately 500,000 are severe enough to require hospitalization (Brown, Fann, & Grant, 1994). Other researchers have estimated the incidence of CHI in the United States at over 9 million (Miller & Berenguer-Gil, 1994). CHI account for about 10% of all emergency room visits (Sherer, Madison, & Hannay, 2000). This number has recently decreased, possibly due to decreased hospitalization of individuals with mild brain injuries. According to the National Head Injury and Spinal Injury Survey of 1980, the typical male is four times more likely to experience a head injury than a female (Kalsbeek, McLaughlin, Harris, & Miller, 1980). Men are believed to sustain more head injuries because they have been found to be greater risk-takers, more likely to be engaged in potentially dangerous work, more impulsive, and more likely to abuse alcohol or drugs. Alcohol is reported to be involved in 30% of head injuries suffered by males and 10% of those suffered by females (Bennett, 1987).

Historical Perspectives

Gennarelli (1986) described the modern definition of a concussion as trauma to the head delivering acceleration/deceleration forces to the brain with resultant mechanical strains and distortions causing the shearing or stretching of nerve fibers. The injury is often undetectable by common neuroimaging techniques (i.e., magnetic resonance imaging [MRI] and computerized tomography [CT]).

Symptoms following a concussion have been identified and named as early as the 1800's when Traumatic Neurasthenia, or “Railway Hysteria”, was the term used to describe symptoms of railway trauma that does not involve spinal cord lesions. Dana (1884) concluded that railway traumas could produce so severe a shock to the nervous system that the individual could become

neurasthenic, or hysterical. The symptoms that followed were identified as: sleeplessness, irritability, depression, memory disturbance, inability to do mental or physical work, headache, tinnitus, nervousness, vasomotor disturbance, excessive sweating, eye strain, enlarged pupils, spinal pain and twitches, and pulse irregularities. Lateralized sensory deficits were also thought to be indicative of this disorder (Dana). In 1889, the term "Vasomotorischen Symptomencomplex" was used to describe similar symptoms that were not due to an obvious physical impairment. Symptoms included headaches, dizziness, vasomotor instability, and intolerance of alcohol and were thought to be the result of disordered intracranial blood flow (as cited in Gasquoine, 1998).

In 1920, Dana again attempted to define this cluster of symptoms with reports of individuals with "nonfatal, nondestructive wounds of the head" who reported symptoms of headache, vertigo, insomnia, irritability, anxiety, depression, memory deficits, fatigueability, tinnitus, partial deafness, and loss of weight. Contrary to earlier definitions, there was no acknowledgment of unconscious mechanisms involved in this disorder. In 1928, Sir Charles Symonds suggested that a "temporary vascular embarrassment" was the cause of symptoms following a concussion. This cluster of symptoms, including headache, giddiness, inability to concentrate, defective memory, indecision, loss of emotional control, and fatigue, was later named "postcontusional syndrome". Shortly following Symonds' description of symptoms, Russell (1932) and Strauss and Savitsky (1934) developed the term "postconcussional syndrome" to describe individuals with persistent headache, dizziness, loss of memory, nervousness, or sleeplessness approximately six months post head injury. Russell was the first to suggest that posttraumatic amnesia (PTA) might be helpful in classifying the severity of the concussive injury. In 1961, Russell and Smith found a positive correlation between injury

severity and PTA and developed a widely adopted severity classification scheme based on retrospectively graded PTA duration. Russell and Smith further suggested that acceleration/deceleration forces during injury were responsible for structural brain injury in concussion.

The consistency among the reported symptoms leading up to the currently used term "postconcussional disorder (PCD)" is obvious. In all but one or two of the historical syndromes, headache, dizziness, cognitive disturbance, affective changes (anxiety, depression, and/or irritability), and changes in sleep or increased fatigueability were identified.

Defining a Head Injury

Closed head injuries, and therefore PCD, have varying definitions and descriptions. Definitions of a mild head injury (MHI) can range from loss of consciousness requiring hospitalization to simple cuts about the face or head leaving the brain unaffected. Most common definitions of MHI suggest impact to the brain involving at least some transitory alteration in consciousness (Kay et al., 1993) and are more appropriately termed mild traumatic brain injuries (MTBI). MTBI's have been shown to result in brain lesions typically in the frontal and temporal lobes (Alves, Macciocchi, and Barth, 1993).

Russell and Smith (1961) classified brain injuries as mild, moderate, or severe based on duration of PTA. Mild brain injuries are characterized by PTA less than 60 minutes, moderate brain injuries include those with PTA lasting from 60 minutes to 24 hours, and PTA in severe brain injuries lasts over 24 hours. This definition was expanded by Russell (1971) to include very severe injuries in which PTA extends beyond 7 days. In addition to PTA, duration of loss of consciousness can be used to grade injury severity. For example, Rimel, Giordani, Barth, Ball, & Jane (1981) defined MTBI as a cranial trauma characterized by (1) an initial loss of

consciousness of 20 minutes or less, (2) a Glasgow Coma Score (GCS) of 13-15 on emergency room presentation, and (3) hospitalization not exceeding 48 hours with associated cognitive impairments (e.g., attention, memory, and information processing speed deficits). A GCS score of 13-15 is indicative of a mild injury, while scores ranging from 9-12 suggest a moderate injury, and scores 3-8 suggest a severe injury (Sherer et al., 2000). For the vast majority of MTBI patients, the aforementioned cognitive difficulties do not persist for more than three months (Dikmen, McLean, & Temkin, 1986; Levin et al., 1987; Mittenberg & Strauman, 2000).

In 1993, the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine developed a definition of MTBI. The individual must have had a traumatically induced physiological disruption of brain function with at least one of: (1) any period of loss of consciousness; (2) any loss of memory for events immediately before or after the accident; (3) any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused); or (4) focal neurological deficit(s) that may or may not be transient. The severity of the disruption may not exceed the following: loss of consciousness of approximately 30 minutes or less, a GCS score of 13-15 after 30 minutes, and PTA not greater than 24 hours. Types of accidents that might lead to this type of injury include (1) head being hit by an object, (2) head striking an object, and (3) brain undergoing strong acceleration/deceleration movement, as in the case of whiplash injuries. Excluded in this definition are stroke, anoxia, tumor, and encephalitis. This definition allows for normal MRI, CT, electroencephalogram (EEG), and neurological evaluations (Kay et al., 1993).

A commonly observed characteristic of a MTBI is a concussion. A concussion typical follows an acceleration/deceleration injury to the head, and is usually accompanied by a short

loss of consciousness and amnesia followed by headache, dizziness, intellectual deficit (which is temporary in the majority of patients) and occasionally nausea (Becker, 1975). Generally, a concussion is considered to have occurred if, after a head injury, a person loses consciousness and suffers reduced alertness after regaining consciousness (Brown et al., 1994). Russell and Smith's (1961) classification of mild, moderate, and severe head injuries based on PTA duration is applicable for grading the severity of the head injury causing the concussion.

Postconcussional Disorder

If an individual has sustained a MTBI with concussion, he or she is at increased risk for developing symptoms of PCD. After a brain injury, PCD is a common diagnosis. PCD is normally associated with a MTBI, as its manifestations cannot be attributed to the myriad of other more severe symptoms that accompany moderate and severe head injuries. The diagnosis of PCD is somewhat controversial, with a lack of consensus on any one definition and a rather high degree of professional skepticism of the disorder and its legitimacy. Discrepant evidence exists about the frequency and duration of PCD, as well. Reports estimate the frequency of PCD development following MTBI between 20-80% and support the claim that individuals may experience postconcussive symptoms for up to and beyond 12 months post-injury (Bohnen & Jolles, 1992). Alves et al. (1993) found only female sex in combination with GCS score to be predictive of postconcussive symptoms at 12 months post-injury. Others have reported that high versus low self-report of postconcussional symptomatology better predicts performance on neuropsychological testing than history of head injury (Hanna-Pladdy, Gouvier, & Berry, 1997; Pinkston, Gouvier, & Santa Maria, 2000). Santa Maria, Pinkston, Miller, and Gouvier (2001) found stability of postconcussive symptoms to be more a function of reported symptom level and female sex than brain injury.

As PCD is diagnosed based predominantly on self-report, lack of objective data further this skepticism. Some researchers have suggested that PCD should only be diagnosed if symptoms persist beyond normal recovery period (commonly 3 months after injury) (Bohnen & Jolles, 1992). Others suggest that there are stages to recovery from MTBI. During the first stage, the individual may experience the short-lived symptoms of nausea, vomiting, drowsiness, and blurred vision, while an individual in the late stage is more likely to experience irritability and intolerance to noise (Bohnen & Jolles). Factor analysis revealed three types of MTBI patients: those with predominantly cognitive and affective symptoms, those with somatic symptoms, and those with relatively mild or no postconcussive symptoms (Bohnen & Jolles).

PCD can be a debilitating disorder lacking clear structural abnormalities as evidenced by typical neuroimaging techniques. As it may lack obvious organic correlates, PCD is thought by some to be an overreaction to an inconsequential brain injury. Although individuals with PCD often lack tangible evidence of an organic etiology, researchers have identified some commonalities among injured patients. Dana (1920) once stated that "patients from the compensation commission who come with bruised heads tell stories so startlingly similar that it seemed to me finally that there must be an underground school for the education of those who have been hit on the head and desire permanent total disability compensation." Dana's voice was prophetic; dozens of such schools have been opened in nearly every city under the supervisory auspices of the local and regional trial lawyers' organizations.

The International Statistical Classification of Diseases and Related Health Problems, 10th edition (ICD-10; World Health Organization, 1992) provides diagnostic criteria for Postconcussion Syndrome, coded 310.2. The ICD-10 criteria requires a history of head trauma with loss of consciousness preceding symptom onset by a maximum of four weeks and three or

more of the following symptom categories: (1) headache, dizziness, malaise, fatigue, noise intolerance; (2) irritability, depression, anxiety, emotional lability; (3) subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment; (4) insomnia; (5) reduced alcohol tolerance; or (6) preoccupation with above symptoms and fear of brain damage with hypochondriacal concern and adoption of sick role. The ICD-10 does not require neurological structural abnormalities for diagnosis, as these have been difficult to identify using typical neuroimaging techniques.

PCD was researched for inclusion in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition and Text Revision (DSM-IV and DSM-IV-TR; American Psychiatric Association [APA], 1994, 2000) and it was concluded that there was a lack of evidence necessary to include PCD as an official category. Alternatively, PCD was included in Appendix B of the DSM-IV: Criteria Sets and Axes Provided for Further Study, and remains there in the DSM-IV-TR. The criteria proposed for further study and possible future inclusion include: (1) history of head trauma that has caused significant cerebral concussion as evidenced by loss of consciousness, PTA, or less commonly posttraumatic onset of seizures; (2) neuropsychological evidence of difficulty in attention or memory; and (3) three or more symptoms that last at least three months and have an onset shortly after head trauma or represent substantial worsening of previous symptoms (fatigue, disordered sleep, headache, dizziness, irritability, anxiety/depression/affective lability, changes in personality, or apathy/lack of spontaneity). The disturbance must cause significant impairment in social or occupational functioning and represent a significant decline from a previous level of functioning. In school-age children, the impairment may be manifested by a significant worsening in school or academic performance dating from the trauma. The symptoms may not meet criteria for Dementia Due to Head Trauma

and are not better accounted for by another mental disorder (e.g., Amnestic Disorder Due to Head Trauma or Personality Change Due to Head Trauma). Currently, a cluster of symptoms that meets criteria for PCD should be coded as Cognitive Disorder, Not Otherwise Specified (294.9) (APA, 2000).

Of the people who experience MTBI's there is a subgroup that performs within normal limits on neuropsychological tests, a subgroup for whom testing identifies cognitive impairment and a decline in IQ, and another subgroup that maintains a normal IQ, but exhibits deficits in attention, concentration, memory, information processing speed, perceptual ability, perceptual-motor skills, reasoning ability, or logical analysis. Independent of the results of testing, many MTBI patients report that "things are not the same" (Bennett, 1987). Bennett suggests many patients, for whom testing lacks sensitivity, may have been functioning within the high average range premorbidly. This suggests that test results within the average range for this subgroup still reflect a decline from premorbid functioning. Controlling for pending insurance claims and litigation supports these reports. Paul Green and associates' data show the frequently observed and reported lack of correlation between injury severity and reported deficit (Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990) is due to the noise introduced by the high variance of the low effort/malingering participants. When the participants who fail effort measures, such as the Word Memory Test or Computerized Assessment of Response Bias, are removed from the analysis, clear and strong injury severity–deficit correlations emerge (Green, Rohling, Lees-Haley, & Allen, 2001).

Clinical Features

Individuals with symptoms of PCD typically fall into three subgroups: those with predominantly physical symptoms, those with cognitive problems, and those with emotional or

behavioral problems. Physical symptoms may include nausea, vomiting, dizziness, headache, blurred vision, diplopia, convergence insufficiency, light and noise sensitivity, altered sense of taste and smell, unsteadiness or poor coordination, tinnitus, hearing loss, sleep disturbance, fatigueability, lethargy, or other sensory loss. Cognitive deficits may include problems with attention, concentration, perception, memory, speed of information processing, speech/language, and an increased sensitivity to lack of sleep, fatigue, stress, drugs, and alcohol. Emotional or behavior changes associated with PCD include emotional lability, irritability and aggression, personality change, fatigue and decreased energy, anxiety, depression, apathy, disordered sleep, loss of libido, and poor appetite (Alves et al., 1993; Anderson, 1996; Brown et al., 1994; Kay et al., 1993; Varney & Roberts, 1999). Most individuals with PCD will present with only a few of the aforementioned symptoms.

Gronwall (1989) described a unifying theme of the cognitive symptoms of PCD as "reduced information processing capacity". As MTBI patients have increased difficulty analyzing complex or abstract information, their processing speed, attention, and memory appear deficient. Neuropsychological testing of MTBI patients commonly yields results suggesting deficits on measures of reaction time, speeded information processing, and divided and selective attention (Ewing, McCarthy, Gronwall, & Wrightson, 1980; Gronwall & Wrightson, 1981; MacFlynn & Montgomery, 1987). Nayak (as cited in Mariadas, Rao, Gangadhar, & Hegde, 1989) and Gangadhar, Rao, and Hegde (1985) found patients with postconcussive symptoms to be significantly impaired in serial and parallel information processing and to have decreased capacity to withstand distraction. In a study of individuals with postconcussive symptoms, Mariadas et al. found only memory functions to be consistently deficient. They suggested that

since memory deficits are reliably demonstrated in individuals with PCD, they are among the "core" deficits of PCD.

Individuals with MTBI may incur tissue damage in any area of the brain; however, the areas of the brain most commonly damaged during mild brain trauma are the anterior parts of the temporal lobes and the orbital frontal lobes. The area and type of lesion may determine what postconcussive symptoms are evident. Frontal lobe injuries would result in difficulties in sustained concentration and attention, behavior flexibility, planning, and goal-oriented behavior. Temporal lobe damage would result in impairments of verbal memory (dominant hemisphere) or nonverbal memory (nondominant hemisphere). Some authors (Reitan & Wolfson, 1986) suggest that impairment of abstraction, reasoning, and logical analysis skills, sometimes referred to as "conation", ultimately prevent the MTBI individual from returning to work or other normal activities of daily functioning. Others (Bennett, 1987) suggest that the psychosocial and personality changes are far more disabling than the neuropsychological changes.

Course

Typically, individuals experiencing MTBI report postconcussive symptoms immediately after the injury and symptoms tend to decrease over the course of several months until the majority of individuals appear relatively symptom-free. For a significant minority, postconcussive symptoms may persist for years following the accident (Alves et al., 1993; Anderson, 1996; Bennett, 1987; Bohnen & Jolles, 1992; Brown et al., 1994). Patients who experience a second concussion typically experience more severely impaired functioning and longer recovery time, suggesting that the effects of concussion may be cumulative (Ewing et al., 1980). This evidence lends support for the brain reserve model, which suggests that there is a threshold that must be reached before functional impairment occurs and includes the concepts of

cognitive reserve and compensation (i.e., the ability to use alternate methods to approach a problems once the standard approach is no longer available) (Stern, 2002). Alves et al. (1993) found 2/3 of patients with uncomplicated MTBI to experience postconcussive symptoms at hospital discharge, 40-60% reported symptoms three months later, 25-45% six months later, and 10-40% 12 months later. Headache was the most commonly reported symptom with more than 50% of MTBI patients reporting headaches at discharge. The percent of headaches decreased over time, with 9-28% of MTBI patients still reporting headaches at a 12-month follow-up. Dizziness was also reported by 15% of MTBI patients at discharge and did not begin to decrease until six months post-MTBI (Alves et al.). Most patients in the Alves et al. study experienced one or two postconcussive symptoms, but it was rare for patients to experience the full symptom constellations consistent with PCD. Other studies have found a majority of MTBI patients to experience postconcussive symptoms within the first month after the accident, but a significant reduction in symptoms by three to six months post-injury (Levin et al., 1987). A review of literature by Bohnen, Twijnstra, and Jolles (1992) found 16-49% of MTBI patients to experience postconcussive symptoms at a six-month follow-up and 1-50% of patients at a 12-month follow-up. Brown et al. found that once symptoms are evident, they tend to persist. Symptoms at three and six months correlated as well as symptoms at six and 12 months (Alves, Colohan, O'Leary, Rimel, & Jane, 1986). This trend continues up to five years after injury. Mittenberg and Strauman (2000) reported that 38% of patients with MTBI were diagnosable with Postconcussion Syndrome according to ICD-10 criteria six weeks after injury, and 28% of untreated patients met criteria six months after injury. Leininger et al. (1990) found patients with postconcussive symptoms to evidence deficits in tests of reasoning, information processing, and verbal learning when compared with control participants up to 22 months post-injury. However,

while brain injured participants report more concentration difficulty and restlessness than controls, controls report similar difficulties with memory, apathy, frequent loss of temper, irritability, fatigue, and impatience (Gouvier, Uddo-Crane, & Brown, 1988). These results underscore the importance of considering base rates of postconcussive symptoms during assessment and diagnosis.

Ewing et al. (1980) examined the course of recovery in a sample of nonsymptomatic post-MTBI students. College students who had recovered from MTBI's experienced one to three years previously and controls were administered vigilance and memory tests under normal and hypoxic stress conditions. Both groups' performance was comparable in the normal testing condition. However, at a simulated altitude of 3,800 meters, where the students experienced additional stress in the form of mild hypoxia, the MTBI students reverted to a level of performance more typically seen immediately after concussion on some of the measures. The MTBI subjects in the hypoxia condition performed more poorly than the controls, and more poorly than their own performance at sea level. The hypobaric stressor had no effect on the performance of the controls. This level of performance is also seen in the elderly with no history of head injury. Previous comparisons of the effects of concussion, aging, and hypoxia are supported by these results (Gronwall & Sampson, 1974; McFarland, 1963). While patients may appear to return to baseline after recovering from MTBI and even perform within normal limits on neuropsychological tests, the addition of elevated stress to the condition may be a more sensitive measure of residual postconcussive symptoms, again lending support for the brain reserve model of dysfunction and recovery.

Even months after the injury, the MTBI patient often feels different. The individual "realizes things are different, but does not understand why. He or she feels indifferent, cannot

concentrate, has difficulty reasoning or thinking, is forgetful, loses his or her temper easily, misinterprets the actions of others or what they say, feels alienated from family, friends, and self, and makes inappropriate or 'dumb' social comments" (Bennett, 1987, p.13). The patient's family may think the patient looks good and they may place unrealistic expectations on the patient such as returning to work or other premorbid activities. The patient often returns to work or school and tries to interact with the world as he or she has always done in the past, except previous schemas may not be as effective as they once were (Bennett). If this is the case, the patient begins to feel generally unsure of him or herself. This phenomenon is termed "perplexity" and may cause patients to view their mental or emotional changes as "evidence of insanity" (Bennett; Lezak, 1978). In more mild cases the patient may suffer lowered self-esteem and lack of confidence. These patients may become socially withdrawn, depressed, angry, and/or paranoid. They may need confirmation from family and friends that they are "normal" and may become inappropriately attached to family and friends. These problems, if experienced, may lead to impairment in coping ability. An individual experiencing these new struggles, especially if the individual was premorbidly of high functioning, may lose trust in him or herself. One attempt to deal with these thoughts is for the individual to allow others to believe he or she is "normal", while the individual knows that it is an act. Often the individual does not realize his or her thoughts and emotions are typical of others with PCD (Bennett).

Kay (1993) described similar experiences of MTBI patients. The individual's sense of predictability and stability is lessened, especially without symptom validation. Kay suggests the individual may experience failure, fear, avoidance, anxiety, depression, loss of self-esteem, isolation, and alienation; depression usually does not occur until at least six months after the injury (Varney, Martzke, & Roberts, 1987). For patients who return to work or school,

Wrightson & Gronwall (1981) found 68% still report at least one symptom of PCD. At three months postinjury, only 34% of MTBI patients were able to return to work (Rimel et al., 1981), and at 12 months post-injury, 10% were still unemployed (Kay, Cavallo, & Ezrachi, 1992).

Associated Psychopathology

When diagnosing PCD, associated symptoms and psychopathology need to be assessed. Both the ICD-10 and DSM-IV proposed criteria for PCD include loss of consciousness (LOC) as a symptom. If LOC is not apparent, the clinician may consider posttraumatic stress disorder as an alternative diagnosis (Bohnen & Jolles, 1992). Alternatively, Varney & Varney (1995) suggested that significant brain injury can and often does occur without impact to the head and without resultant LOC or posttraumatic amnesia. Lack of evidence for LOC and PTA does not rule people out for possibly suffering the organic, neuropsychological, and psychological effects of a head injury, hence, the Kay et al. (1993) study group adopted "alteration of consciousness" as a more liberal definition of MTBI.

Another dimension for change in the head injured individual is personality. Personality change is independent of injury severity and changes may become more pronounced with time (Hibbard et al., 2000). These changes can often be more upsetting for the individual and family and more disabling than mild cognitive losses. Researchers have not identified a specific personality profile unique to individuals with MTBI, although symptoms from a cluster of known changes repeatedly occur (Varney, 1989; Varney & Roberts, 1999). MTBI individuals have been described as irritable, restless, unpredictable, impulsive, moody, childish, unmotivated, and insensitive to the needs of others (Hibbard et al.). Hibbard et al. suggest that personality changes following traumatic brain injury (TBI) are the result of an idiosyncratic mix of premorbid personality traits and emotional/behavioral postconcussive symptoms. Some

research suggests that premorbid personality traits become magnified after TBI, while others suggest that the emotional/ behavioral symptoms change the individual's personality in a way that decreases his or her ability to manipulate and manage environmental demands. There is limited research on how premorbid personality traits affect postmorbid adjustment, but four personality traits appear to stand out as predictors of poor post-TBI adjustment: narcissistic grandiosity, perfectionism, dependency, and borderline personality (Hibbard et al.).

In a study by Hibbard et al. (2000), it was found that a large minority of post-TBI patients (24%) had preinjury personality disorders (PD). Antisocial and obsessive-compulsive PD's made up a large number of the preinjury PD's. This is consistent with previous research suggesting that individuals with certain personality traits (e.g., risk taking, impulsivity, and aggressiveness) may be at greater risk for experiencing a TBI. Although Axis II Personality Disorders cannot be diagnosed when symptoms are the result of a head injury, Hibbard et al. found that 2/3 of individuals would have met criteria for at least one personality disorder post-TBI. The most frequently occurring post-TBI personality patterns were borderline, obsessive-compulsive, avoidant, paranoid, antisocial, and narcissistic. Less common were passive-aggressive, histrionic, dependent, and schizoid characteristics. Borderline characteristics were most common post-TBI. Having a premorbid PD increased the frequency of developing post-TBI antisocial characteristics. Certain personality traits negatively affected over 30% of post-TBI individuals; these included loss of self-confidence, difficulty coping with cognitive and interpersonal failures, and difficulties related to negative affect (Hibbard et al.). These changes in personality following MTBI would be diagnosed as Personality Change Due to Head Trauma, with type specified.

Differential Diagnoses

Individual symptoms of PCD are not specific to PCD and may be attributed to numerous other syndromes. Symptoms of PCD occur in the general population and among patients with medical or psychiatric problems (Gouvier et al., 1988; Iverson & McCracken, 1997). Ruling out differential diagnoses entails neurological and neuropsychological examination of the patient from an organic as well as a psychological perspective (Mittenberg & Strauman, 2000). Using current DSM-IV criteria for PCD, a patient may not exhibit memory problems. However, if significant memory difficulties are identified as well as aphasia, apraxia, agnosia, or decreased executive functioning, the patient should be diagnosed with dementia in lieu of PCD.

The head injured patient may experience depression or emotional stress due to postconcussive symptoms (Bennett, 1987; Ruff, Wylie, & Tennant, 1993). Often the patient and his or her family are unaware of the symptoms accompanying a head injury and are confused about why the individual is not the same. Symptoms of depression are commonly the result of this confusion (Bennett). A depressive disorder and PCD are both characterized by sadness, irritability, sleep disturbance, fatigue, and difficulty with concentration or thinking; PCD symptoms do not include changes in appetite or weight, psychomotor agitation or retardation, suicidal ideation, or a history of depressive disorder. PCD and somatoform disorders share the symptoms of headache, fatigue, dizziness, blurred vision, memory difficulty, and hypochondriacal concern, but PCD is not associated with gastrointestinal, sexual, or urogenital symptoms, marked motor or sensory deficits, or psychogenic seizures. If the patient experiences physical problems as a result of the brain injury, it is important to keep in mind that personality inventories are likely to indicate elevated somatization (Mittenberg & Strauman, 2000; Ruff et al.).

Sbordone and Liter (1995) stress the importance of differentiating PCD from posttraumatic stress disorder (PTSD). While some authors argue that individuals sustaining MTBI may develop resultant PTSD, Sbordone and Liter found PCD and PTSD to be mutually exclusive. PTSD patients are able to provide detailed and emotional recollections of their traumas, while PCD patients are typically amnesic for the actual injury and are, therefore, unable to provide detailed recollections and do not become emotional upon recall. The characteristic symptoms of PTSD (intrusive thoughts, anxiety with exposure to the traumatic event, nightmares, reluctance to discuss the trauma, and hypervigilance) are not seen in individuals with PCD. However, memory problems, word-finding problems, problem solving deficits, fatigue, social/interpersonal problems, decreased libido, and photophobia are symptoms that are common to both PCD and PTSD. Sbordone and Liter recommend that PTSD be ruled out before making a diagnosis of MTBI or PCD. Bohnen and Jolles (1992) and Ruff et al. (1993) suggest assessing for an adjustment disorder with depressed and anxious mood if symptoms appear to be due to a neurotic reaction to the head injury.

Headaches, dizziness, and subjective intellectual impairment, as well as the patient's history differentiate PCD from generalized anxiety disorder (Mittenberg & Strauman, 2000). Postwhiplash syndrome should also be considered in lieu of PCD (Yarnell & Rossie, 1988). Symptoms of postwhiplash syndrome are very similar to those of PCD, such as headache and fatigability, but postwhiplash syndrome is also characterized by vestibular symptoms, neck pain, and cervical paresthesias. Although postwhiplash syndrome does not usually involve a brain injury, cervical whiplash and concussion may damage common brain structures (Varney & Varney, 1995).

Chronic pain should also be ruled out while assessing a MTBI patient for PCD. Chronic pain patients have often had accidents and complain of physical, cognitive and psychological symptoms similar to that of the PCD patient. A study by Iverson and McCracken (1997) found that 39% of chronic pain patients would meet full criteria for PCD, with the exception of a head injury. When assessing a patient with a CHI, gathering information from the patient's history will assist in differentiating PCD from chronic pain.

Finally, in assessing for PCD, the differential diagnosis of malingering must be ruled out. There are differing views on the rate of malingering among MTBI patients. While PCD was historically referred to as "accident neurosis" and thought to be an attempt for secondary gain (Miller, 1961), some clinicians agree that past rates of malingering have been overestimated (Bornstein, R. A., Miller, H. B., & van Schoor, 1988; Rimel et al., 1981). Other researchers have found that 33-47% of compensation-seeking patients are identified as exaggerating cognitive impairment by objective diagnostic instruments (Mittenberg & Strauman, 2000). Thus, it is very important that a patient be assessed for malingering in the presence of postconcussive symptoms, as well as with other presentations. The DSM-IV suggests that malingering should be strongly suspected if any combination of the following is noted: (1) medicolegal context of presentation; (2) marked discrepancy between the person's claimed stress or disability and the objective findings; (3) lack of cooperation during the diagnostic evaluation and in complying with the prescribed treatment regimen; and (4) presence of Antisocial Personality Disorder. The DSM-IV defines malingering as an intentional attempt to produce "false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs" (APA, 1994, p. 683). Those with *mildly* exaggerated symptoms, or the low effort group,

are probably the most numerous. Although this group does not fit the DSM-IV definition of malingering, it is essential that they be identified and the role of their low effort be considered in evaluation their level of actual disability.

A careful assessment of malingering should be conducted before a diagnosis is made, especially in a head injured population, as symptoms can be easily misattributed. Although the DSM-IV is a helpful aid in the diagnosis of malingering or low effort, it has some shortcomings. The DSM-IV warns that medicolegal involvement may suggest an increased risk of malingering, but many MTBI patients have been involved in car accidents or other types of accidents, and litigation involvement is relatively common. A marked discrepancy between the patient's complaints and objective assessment is also noted as an indicator of malingering. As research has shown that MRI and CT scans easily miss small lesions common following MTBI and are unable to provide reliable information regarding the functional status of brain tissue, a discrepancy between these test results and patient complaints should not be entirely unexpected. The DSM-IV also stresses lack of cooperation as an indicator for malingering. Patients with MTBI commonly exhibit limited cooperation; however, this may be due to frontal lobe disinhibition, distractibility, or other attentional problems, which are among the most common neuropsychological sequelae of brain injury. Lastly, the DSM-IV suggests that malingering should be suspected if the patient has an antisocial PD, but the premorbid presence of antisocial PD is a known risk factor for increased likelihood of CHI.

Ruff et al. (1993) suggest that suspected malingering can be ruled out by examining as much information about the patient as possible. They suggest: (1) reevaluate and psychometrically retest the patient, (2) review comorbid symptoms and establish their interactions, (3) review premorbid medical and academic records, (4) interview the patient,

family, and friends regarding pre- and postmorbid functioning, (5) determine the nature of any secondary gain, and (6) determine the independence of data measures. Ruff et al. identified indicators based on clinical impression to consider when assessing for malingering. These include premorbid factors, test performance, current activities of daily living, behavioral observations during the evaluation, postmorbid complaints, and indicators specific to personal injury litigants.

Pathogenesis

The pathogenesis of postconcussive symptoms is divided into two main perspectives: psychological and organic. Proponents of psychological pathogenesis cite lack of correspondence between severity of the injury and persistence of postconcussive symptoms as their main source of evidence (Bohnen & Jolles, 1992; Gasquoine, 1998). The psychological perspective is likely to consider postconcussive symptoms as the result of a posttraumatic neurosis, including hysteria, malingering, and anxiety reactions (Bohnen & Jolles; Gasquoine). Certainly, the emotional response and expectations following MTBI are apt to have an effect on the individual. Threats of a change in lifestyle or a lost career may also increase a patient's fear and anxiety (Bohnen & Jolles).

As one example of a theory originating from the psychological perspective, Mittenberg and Strauman (2000) propose the neuropsychological theory to explain why an individual may have postconcussive symptoms without associated organic damage. They suggest that when an individual experiences MTBI expectations are formed about what symptoms may develop. Normally occurring premorbid symptoms are subsequently attributed to the MTBI, and symptom expectations are thus confirmed. This theory is supported by the many non-litigant MTBI patients who underestimate premorbid postconcussive symptoms, and, therefore, overestimate

the change since injury (Hilsabeck, Gouvier, & Bolter, 1998). Mittenberg, Tremont, Zielinski, Fichera, and Rayls (1996) found an early cognitive behavioral intervention to prevent the development of PCD, further supporting the neuropsychological theory.

Evidence weakening the support for a psychological pathogenesis of PCD includes studies that assess for malingering or accident neurosis. Guthkelch (1980) reported a sample of 398 patients with compensation claims; after evaluation, only 6.8% were suspected to be malingering or suffering from accident neurosis. Other estimates of malingering approach 1/3 of litigants (Allen, Conder, Green, & Cox, 1997; Binder, 1997; Binder, Rohling, & Larrabee, 1997; Frederick, Sarfary, Johnston, & Powell, 1994; Greiffenstein, Baker, & Gola, 1994; Millis, 1992; Trueblood & Schmidt, 1993).

The organic pathogenesis of PCD suggests that symptoms are caused by "rotational sheer strains and corresponding diffuse axonal injury throughout the brain" (Anderson, 1996, p. 493-494; Bohnen and Jolles, 1992). This perspective has been supported by animal research on primates confirming that acceleration of the head without impact results in severe diffuse destruction of brain substance consisting of diffuse axonal injury (Alves et al., 1993; Anderson; Ommaya & Gennarelli, 1974). After a head injury, focal injuries may occur as contusions develop on the undersurface of the temporal and frontal lobes and the anterior poles of the temporal lobes due to contact with rough bony surfaces. The orbital frontal cortex is particularly sensitive to damage as it is in close proximity to the skull (Anderson). Patients with these types of injuries may never lose consciousness, but show clear patterns of psychological, behavioral, and neuropsychological changes (Varney & Varney, 1995).

Support for the organic perspective includes evidence that preexisting neurological disorders increase in symptom severity following MTBI (Naugle, 1987) and MTBI has been

implicated in the development of atypical neurological syndromes (Kitanaka, Sugaya, & Yamada, 1992). Additionally, headaches, anosmia, and diplopia identified 24 hours after injury have been correlated with higher symptom frequency six weeks after the injury (Bohnen & Jolles, 1992). There is evidence that immediately after MTBI physiological alterations occur. These physiological changes include: neuronal damage, reduced cerebral blood flow, disturbances in water metabolism, altered brainstem-evoked potentials, neurotransmitter changes, and brainstem dysfunction (Bohnen & Jolles). Gasquoine (1998) suggests that the organic perspective of PCD was strengthened subsequent to the use of PTA as an index of injury severity, identification of acceleration/deceleration forces as the mechanism of injury, development of methodology to separate organic from psychogenic sequelae (correlation with duration of PTA), and delineation of the neuropsychological sequelae of concussion via the experimental approach. Other researchers indicate that PTA is difficult to reliably assess if it is less than one hour and, therefore, a consistent relationship between PTA duration and persistence of postconcussive symptoms following MTBI is often difficult to show (Gronwall & Wrightson, 1980).

One of the arguments against the organic perspective is lack of correspondence between brain damage and severity of postconcussive symptoms. While MRI's and CT's are useful in diagnosing acute hematomas and parenchymal and extra-parenchymal lesions, many lesions are too small to be detected using these neuroimaging methods. Methods such as positron emission tomography (PET) and single-photon emission computerized tomography (SPECT) are able to identify smaller lesions and yield information regarding functional status of brain tissue (Kant, Smith-Seemiller, Isaac, & Duffy, 1997), but, as of yet, are not readily available and economically feasible for neurological assessment of MTBI (American Academy of Neurology, 1996; Davalos

& Bennett, 2002). However, it appears that brain damage (i.e., lesions) may be more related to postconcussional symptoms than previously thought, but the imaging equipment predominantly used to identify organic damage may lack sensitivity for identifying even widespread microscopic damage. As previously mentioned, expected relationships between injury severity and deficit emerge when effort variables are considered (Green et al., 2001).

Alternatively, an interaction perspective, the very essence of the study of neuropsychology, may be more accurate than either a purely psychological or organic perspective and appears to have the most support in the literature. An interaction perspective of the development and maintenance of postconcussive symptoms suggests that acute symptoms are likely related to organic pathogenesis, while chronic symptoms may be more related to psychological factors (Bohnen & Jolles, 1992; Lishman, 1988; Mittenberg and Strauman, 2000). An estimated 25-38% of MTBI patients develop emotional sequelae (Klonoff & Thompson, 1969), many during the chronic stage. Stanczak, Gouvier, & Long (1983) suggest that nonorganic sequelae might develop as a grief response in reaction to "actual or perceived loss of functional integrity or autonomy and/or a loss of social or financial status" (p. 17). Alternatively, Stanczak et al. suggest nonorganic sequelae might be a result of the exacerbation of premorbid personality factors following MTBI.

When assessing MTBI patients it is important to consider the impact of the environment on the patient's postconcussive symptoms. According to the coping hypothesis, developed in 1985 by Van Zomeren and Van den Burg to explain the development and maintenance of postconcussive symptoms, environmental stressors may exacerbate these symptoms. The coping hypothesis suggests that symptoms worsen as the patient makes a chronic effort to compensate for organic deficits. When the patient experiences stress and strain, postconcussive symptoms

develop. As the patient continues to deal with environmental demands, the symptoms are exacerbated. The quicker the patient resumes premorbid activities before recommended, the more likely he or she is to be unable to cope with the stress of daily life in addition to his or her cognitive deficits. For example, litigation and compensation claims may be stressful enough to cause the development of postconcussive symptoms according the coping hypothesis (Bohnen & Jolles, 1992). The coping hypothesis is supported by research identifying stress as a factor contributing to poorer performance on cognitive tests (Ewing et al., 1980; Hanna-Pladdy, Berry, Bennett, Phillips, & Gouvier, 2001).

During the assessment phase of rehabilitation, it is important to develop a conceptual framework in order to identify and assess the multiple factors involved in symptom presentation after MTBI and to assist in the development of an individualized treatment plan (Kay, 1993). Kay presents seven assumptions guiding the development of a neuropsychological framework. The first assumption points out that minor head injury may result in MTBI and, therefore, notes the difference between the two terms with minor head injuries requiring only cuts about the face, head, or neck, without necessarily injury to the brain. Second, symptoms may be transient or permanent. Third, an individual's personality and social/environmental factors determine his or her response to the initial symptoms. These factors may result in symptom lessening over time or in the maintenance or magnification of symptoms. The fourth assumption states that primary deficits after MTBI may lead to a shaken sense of self. Fifth, if primary deficits remain undiagnosed, psychological overlay accumulates, which, in time, may become more disabling than the underlying primary deficits. Sixth, personality and environmental factors interact with primary deficits to determine the level of functional disability. Lastly, an individual's outcome after MTBI is a product of at least the following: extent of damage to the brain, persistent

symptoms of injury to the head, personality style of the individual, family and social support systems, job and home requirements, age and medical factors, legal status, and adequacy of medical response to injury.

A neuropsychological framework is helpful in understanding functional disability following MTBI (Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992). This model is comprised of three main components affecting outcome: physical, psychological, and neurological. The physical factors include physical effects of the injury that are a detriment to functioning, such as pain, fatigue, sleep problems, sensory deficits, balance problems, and effects of medication. Psychological factors include premorbid or acquired internal structures or responses that affect functioning. These may include personality style, affective status, sense of self, degree of psychological overlay, psychosocial situation, and response to or motivation for being in litigation. The neurological factors are a combination of premorbid factors, such as age or previous injuries to the brain, and injury-related factors. These neurological factors determine the extent of damage to the brain and whether damage is temporary or permanent.

The model differentiates between objective cognitive deficits (determined directly by damage to the brain or neurological factors) and subjective cognitive deficits (those breakdowns experienced by the individual and identified on neuropsychological testing that may be caused by psychological and physical factors, as well as objective cognitive deficits due to brain injury). Therefore, while actual neurological damage may be causing objective cognitive deficits affecting functioning, the severity of deficits may actually be minimal. Certain personality factors or other psychological factors may be causing or exacerbating these minor deficits to significantly affect functioning. Appropriate treatment will be based on which of the above assessments is accurate. If the functional deficits are predominantly due to objective cognitive

deficits, cognitive remediation might be the best treatment. If subjective cognitive deficits are causing functional impairment, psychological factors should be the targets of change for improved functioning. Physical factors may also influence subjective cognitive deficits either directly or indirectly through the psychological factors. For example, the expectation of pain causes anxiety, which reduces concentration.

The connections between neurological and objective cognitive factors and the other factors may weaken in time and eventually have no influence within the system. Although initial symptoms have resolved, this "dysfunctional loop" remains strong based on the other connections among physical, psychological, and subjective cognitive factors, which continue to influence the functional outcome (Kay, Newman, et al., 1992). Even without objective symptoms the functional disability may still be quite severe for the individual. An example of a dysfunctional loop remaining after symptom resolution is the cognitive dysfunction loop. In this situation, the individual has initially experienced some confusion or memory loss. These objective symptoms could activate psychological factors (anxiety), which in turn initiate subjective cognitive factors (concentration problems), which affects functional outcome (problems at work), which negatively affects the individual's psychological status (increased anxiety). Even with the dissipation of the original objective symptoms (i.e., confusion and memory loss), the cognitive dysfunctional loop will likely persist. Kay, Newman, et al. (1992) proposed a second dysfunctional loop within this model, the dysfunctional pain loop. In this instance the flow begins with physical factors, such as the perception of pain, which influence the individual's functional outcome, which influences psychological factors, such as elevated anxiety or depression, which may further support and intensify the perception of pain. Even with objective pain reduction, the loop is self-perpetuating. The strength of the connections between

the factors and relative weights of the factors is ideographic. To determine how to proceed with treatment, it is essential to develop a framework to direct the selection of interventions. Without identifying the source of the functional deficits, the therapist might easily waste the client's time and money by using an ineffective treatment.

Since a treatment plan is developed based on results of the assessment, it is critical to accurately assess the client and factors leading to the functional outcome. The organic, as well as the nonorganic sequelae, should be evaluated. Nonsymptomatic clients with a history of MTBI have been shown to perform at levels seen immediately after injury when subjected to hypobaric stress and compared to non-injured controls (Ewing et al., 1980). Although there is no self-report of persisting symptoms, symptoms are apparent on measures of complex attention and memory when stress is introduced. These results support the theory that mild cognitive deficits may persist for years, even with mild head trauma. However, only a minority of these head injured individuals actually report postconcussive symptoms, and, in fact, at a rate similar to the base rate of these symptoms in the general population (Gouvier et al., 1988). If a sample of mildly head injured individuals performs more poorly on a task of complex attention while under stress than a non-head injured sample, then what factors lead to the report of postconcussive symptoms in only a minority of people with mild head injuries? Hanna-Pladdy et al. (2001) found postconcussive symptoms to vary more as a function of level of subjective stress than head injury status. However, the role of psychopathology was not addressed in either symptom reporting or cognition functioning.

Limitations of Research Criteria for PCD

The problem of base rates must be revisited to comprehensively cover PCD. The hallmark features of PCD are headaches, memory problems, dizziness, tinnitus, sensitivity to

noise, concentration problems, visual disturbances, fatigue, irritability, and impatience (Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995a). While Gouvier et al. (1988) found PCD patients to have more concentration problems and restlessness than a control group of college students, all of the other symptoms of PCD were similar across groups. Other researchers (Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995b; Fox et al., 1995a; Lees-Haley & Brown, 1993) have found similar results. From workers' compensation to other personal injury litigants to psychiatric populations to normal populations, PCD symptoms are reported at high rates. On the other hand, during validation of the Postconcussion Syndrome Checklist (PCSC; Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992), the PCSC was found to reliably differentiate between a group of head injured participants and a control group.

Although discrepant findings exist regarding the discriminant validity of the PCD symptoms, researchers as long ago as Dana (1920) reported a similar pattern of symptoms following head injury in litigation as well as nonlitigating populations. These differences might be explained by qualitative differences in these symptoms that are unique to PCD.

PURPOSE OF STUDY

Summary

Looking over the history of PCD, essential diagnostic features and associated symptoms have been studied for at least 200 years. PCD is usually the result of a mild head injury; however, head injuries have varied in definition culminating in the now widely used definition of MTBI provided by the Kay et al. (1993) group. While the majority of individuals with a history of MTBI are relatively symptom-free by three months post-injury, a significant minority continues to report postconcussive symptoms and may be diagnosed with PCD. PCD is defined by physical, cognitive, and emotional/behavioral symptoms; however, the pathogenesis of these symptoms and, more importantly, the mechanisms involved in their maintenance is unclear. Purely organic factors appear to play a role, especially initially, but cannot explain the maintenance of symptoms months or years post-injury. Similarly, purely psychological hypotheses, while partially explaining the maintenance of symptoms, cannot account for the commonly seen objective neuroimaging evidence of brain lesions associated with MTBI.

As most researchers and clinicians now agree, an interactionistic perspective, with acute symptoms primarily related to organic factors and chronic symptoms to psychological factors, appears to have gained the most empirical support. Recent research using neuroimaging technology supports the presence of organic changes during the first couple of weeks post-MTBI, which likely results in the development of early postconcussive symptoms. However, the maintenance of symptoms beyond expected recovery times has several etiological hypotheses including a grief response hypothesis (Stanczak et al., 1983), the coping hypothesis (Van Zomeren & Van den Burg, 1985), and the development of dysfunctional loops (Kay, Newman, et al., 1992). Lending support to maintenance of symptoms having a psychological etiology is

research supporting reduction of chronic symptoms using cognitive behavioral strategies (Mittenberg et al., 1996). In addition, Mittenberg's research on "expectation as etiology" promotes psychological factors in the development of PCD (Mittenberg, DiGiolio, Perrin, & Bass, 1992; Mittenberg & Strauman, 2000). Still, the interaction is not entirely clear.

Ewing and associates (1980) did one of the first studies looking at the effects of relatively mild head injuries (at least one year post-injury) on cognitive functioning. Their participants experienced PTA ranging from less than one hour to two days, but all were considered to have sustained mild head injuries. In a stressful condition involving mild hypoxia simulating an altitude of 3,800 meters, the nonsymptomatic mild head injury group performed significantly worse on memory and vigilance tasks than a nonsymptomatic non-head injured group, and significantly worse than their own performance at sea level. There were no significant differences between groups on the Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977), a test of complex attention. The authors hypothesized that this was due to practice effects (the test was given twice, once at the start of the experiment and a second time during the stress condition), and habituation to the stress condition, as the participants spent at least 30 minutes in the stress condition before taking the PASAT. These are two limitations of this study, rendering the results associated with the PASAT virtually uninterpretable. In addition, the Ewing et al. study is limited in that only nonsymptomatic participants with and without MTBI were evaluated. A group of symptomatic MTBI and non-MTBI participants would add an entire dimension to this study. Lastly, Ewing et al. did not investigate the current level of psychological distress in the participants. It is possible that the MTBI participants were experiencing a greater level of psychological distress than the non-MTBI participants, which led

to a decrement in memory and vigilance performance due to an inability to cope with the stress condition. These limitations will be addressed in the current study.

While we have come to believe that psychological factors play a role in the maintenance of postconcussive symptoms, it is unclear how symptomatic MTBI patients differ from nonsymptomatic MTBI patients in terms of psychological distress. Why many MTBI patients have lasting residual postconcussive symptoms is an unanswered question. If symptomatic and nonsymptomatic groups differ in level of psychological distress, the specific differences have not been identified. In other words, does psychological distress play a role in symptom maintenance, and, if so, what are the specific characteristics of psychological distress common to symptomatic MTBI patients?

In addition, it is unclear how these patients respond to stressful situations and whether there is a difference between symptomatic and nonsymptomatic MTBI patients in their response to stress. While prior research has indicated that stress plays a greater role in report of postconcussive symptoms than MTBI status (Hanna-Pladdy et al., 2001), the interaction of psychological factors has not been studied. Even patients denying postconcussive symptoms have been shown to exhibit cognitive deficits when stress was induced (Ewing et al., 1980). Thus we must wonder if an underlying, reduced ability to cope with stress is affecting the expression of postconcussive symptoms.

The difference between symptomatic head injured and symptomatic non-head injured people will likely be found in the combination of and connections between organic and psychological factors. Exacerbation of premorbid personality factors or post-morbid development of dysfunctional loops could account for these differences. Although there is evidence that stress increases the relative number, frequency, and intensity of postconcussive

symptoms irrespective of head injury status (Gouvier et al., 1992), comparison of the psychological profiles of symptomatic MTBI and nonsymptomatic MTBI has not been thoroughly investigated. This study proposes to examine the relationship between stress, report of postconcussive symptoms, MTBI status, and psychological distress. While many studies compare head injured to non-head injured participants, these groups are confounded by differences in their symptom status. Therefore, the additional factor of symptom status was added to this study to create a basic four-group design (i.e., MTBI symptomatic, MTBI nonsymptomatic, non-MTBI symptomatic, and non-MTBI nonsymptomatic). Each of these four groups was further divided with half of each group participating in control/reading condition and half participating in a stress condition, thereby creating eight groups of subjects.

Research Questions and Hypotheses

Question 1

Do people with mild brain trauma perform worse on cognitive and attentional tasks than those without a history of mild brain trauma?

Hypothesis 1. It is hypothesized that MTBI participants will perform significantly more poorly on a test of complex attention, as measured by the PASAT, than non-MTBI participants.

Question 2

Do stress and history of a mild head injury interact to affect performance on cognitive and attentional tasks?

Hypothesis 2. Based on previous research (Ewing et al., 1980), stressed, MTBI participants performed significantly more poorly on tests of vigilance and memory, but not on a test of complex attention, than stressed, non-MTBI participants. Ewing et al.

hypothesized that this pattern was due to test practice effects and habituation to the stress condition. Controlling for this study's limitations, it is hypothesized that stressed, MTBI participants will perform significantly more poorly on a test of complex attention, as measured by the PASAT, than the other three groups: stressed non-MTBI participants, non-stressed, MTBI participants, and non-stressed, non-MTBI participants.

Question 3

Does self-report of postconcussional symptomatology affect performance beyond the main effects and interaction effects of stress and history of MTBI?

Hypothesis 3. It is hypothesized that symptomatic, stressed MTBI participants will perform significantly more poorly on a test of complex attention, as measured by the PASAT, than nonsymptomatic, stressed, MTBI participants (see Table 1 for hypothesized differences between groups).

Table 1. Hypothesized Differences in PASAT Scores Among Groups

	Symptomatic		Nonsymptomatic	
	Stress	No stress	Stress	No stress
MTBI	*			
No MTBI				

* Group that will be significantly different from all others.

Question 4

Is the development and maintenance of postconcussive symptoms related to level of psychological distress?

Hypothesis 4. It is hypothesized that level of psychological distress will be significantly positively correlated with level of PCD symptomatology after three months.

Question 5

Which components of psychological distress will be most related to maintenance of postconcussional symptomatology ?

Hypothesis 5. It is hypothesized that levels of depression, anxiety, anxiety-related disorders, trait stress, somatic complaints, level of perceived non-support, and borderline features will each be significantly positively correlated with level of PCD symptomatology after three months.

METHOD

Participants

A power analysis was done to determine the number of participants needed for power = 0.80, alpha = .05. Eighty total participants were estimated to yield enough power to find a true difference. To obtain this estimate, effect sizes from current research measuring the effects of postconcussional symptoms (Hanna-Pladdy et al., 2001) and history of MTBI (Maddocks & Saling, 1996; Ewing et al., 1980) on complex attention was used. The average effect size for main effects was considered large ($f = 0.52$) and indicated a need for approximately 10 participants per group. Estimating interaction effect sizes as medium to high medium (i.e., $f \approx 0.30$) indicated that 10 participants per group would allow enough power to detect interactions.

Two hundred and five undergraduate psychology students at Louisiana State University in Baton Rouge volunteered to participate in this study for class extra credit. Participants responded to notification on the LSU research website asking for volunteers to participate in an experiment on postconcussional disorder. The notification asked for individuals with mild or no head injuries to complete tests of attention and personality questionnaires. Initially, consecutive participants were accepted for participation until the first of the four groups was filled. Then, participants were screened using only the first 20 minutes of the procedure (i.e., consent form, interview, and postconcussive symptom checklist) to ensure that they met the criteria necessary to be included in one of the remaining groups.

All participants were screened for inclusion and exclusion criteria. Criteria were kept at a minimum to keep the sample as heterogeneous as possible, and, therefore, increase external validity. Inclusion criteria was age 18 years or older. Exclusion criteria was history of moderate

or severe brain trauma, mild brain trauma within the past three months, illiteracy, neurologic disease, and seizure disorder. A total of 80 participants completed the entire experiment.

Participants were divided into four groups based on self-reported history of MTBI, as indicated by any alteration in mental state at the time of the accident (Kay et al., 1993), and symptomatic presentation, as measured by the PCSC (Gouvier et al., 1992). To obtain a high symptom group (i.e., 68th percentile or above) and a low symptom group (i.e., 32nd percentile or lower) participants with PCSC total scores greater than 0.5 standard deviations from the mean were included in data analysis based on previous research using similar cutoffs (Hanna-Pladdy et al., 2001). Participants with low PCSC scores (scores $\leq$57) were assigned to the nonsymptomatic group and participants with high PCSC scores (scores $\geq$ 72) were assigned to the symptomatic group. Groups were symptomatic MTBI (n = 20), nonsymptomatic MTBI (n = 20), symptomatic non-MTBI (n = 20), and nonsymptomatic non-MTBI (n = 20). Participants in each of these four groups were further alternately divided into two groups based on their exposure to an experimentally induced stress condition, thereby creating eight groups with 10 subjects in each group.

Materials

Structured Clinical Interview

A structured clinical interview (see Appendix A) was developed and given to participants in order to collect the following information: sex, age, race, education, parent's socioeconomic status (SES) as determined by annual salary, mental health history, neurological history, head injury information, if applicable, reading level, and, for females, information regarding menstrual cycle. Reading level at or above the 4th grade was required and was measured by the

participants' ability to read and comprehend a sentence determined to be above the 4th grade reading level using the Flesch-Kincaid readability statistic provided by Microsoft Word.

Postconcussion Syndrome Checklist

The PCSC (Gouvier et al., 1992) is a self-report questionnaire that has been shown to be a valid measure of the symptoms associated with PCD. Participants rate the frequency, intensity, and duration of 10 symptoms commonly occurring following a mild brain injury. The total score on the PCSC is able to reliably make the distinction between a head injured, nonsymptomatic control group from a head injured patient population with PCD. Therefore, the total score from this measure was used in this study to determine group membership based on symptomatology (see Appendix B).

Paced Auditory Serial Addition Test

The PASAT (Gronwall, 1977; Gronwall & Sampson, 1974) is a neuropsychological measure of complex attention that has been shown to be effective at detecting subtle attentional deficits in symptomatic mild brain injured patients (Leininger et al., 1990; Lezak, 1995). To ease interpretation, and because it is the most difficult of the four trials on the PASAT, total number correct on trial four was used in analysis for this study (see Appendix C).

Personality Assessment Inventory (PAI)

The PAI (Morey, 1991) is a self-report measure consisting of 344 questions with a four-point Likert response format. The PAI is designed to provide information on DSM clinical syndromes, treatment considerations, interpersonal style, and validity indicators. Some of the PAI's benefits include development based on rational and empirical item selection, heterogeneous normative group, and non-overlapping scales. The PAI requires a 4th grade reading level. The PAI contains critical items regarding suicidality, each of which was inspected

for each participant before he or she left the experiment. Participants endorsing any of these critical items were assessed for suicidal intent before leaving the experiment.

For the purposes of this study, a PAI Modified Distress Index (PMDI), a general descriptive of clinical elevations, was calculated based on a composite of the various scaled scores. The PMDI was calculated using the Somatic Complaints, Anxiety, Anxiety-Related Disorders, Depression, Mania, Paranoia, Schizophrenia, Borderline Features, Antisocial Features, Alcohol Problems, Drug Problems, Aggression, Suicidal Ideation, Stress, and Nonsupport scales from the PAI.

This descriptive index score was based on the scoring procedures used for the Brief Symptom Inventory (Derogatis & Spencer, 1982). The BSI is a measure of self-reported psychological problems that asks the reporter to identify how distressed he or she was by specific symptoms during the past week. The response format for the BSI is a 5-point rating scale with zero being "not at all" distressed and 5 being "extremely" distressed. The BSI sums all responses to yield a Positive Symptom Total (PST) score. Since a score of zero indicates no distress, the BSI divides the PST by the number of non-zero responses to obtain a Positive Symptom Distress Index (PSDI), that is, an average severity rating of endorsed responses.

For the purposes of the PAI, t-scores were converted to z-scores. Since the scales used to calculate the PMDI are unipolar scales with low scores indicating normality, or lack of psychopathology, and participant distress/psychopathology was the construct of interest, scales with $z<0$ were recoded to $z = 0$ based on the same premise used in calculating BSI scores, that is, scores of zero or below indicate a lack of distress in that clinical area. By recoding scores below $z = 0$ to zero, high levels of psychopathology on specific scales were not lost by very low scores on other scales. Z-scores were then summed yielding a score similar to the Positive Symptom

Total on the BSI (Derogatis & Spencer, 1982), which was divided by the total number of non-zero responses yielding the PMDI (similar to the BSI's Positive Symptom Distress Index).

Design and Procedure

The following procedures were completed at the LSU Psychological Services Center at the Baton Rouge campus. Undergraduate Chancellor's Aid students and undergraduate students registered for PSYC 4999 and PSYC 2999 were trained as research assistants (RA). All involved researchers were certified in the Human Participant Protections Education for Research Teams course as suggested by the National Institutes of Health. RA's were trained by the author, a Master's level clinical psychology graduate student, in clinical interviewing skills and followed an interview protocol developed by the author ensuring that all necessary information was obtained. In addition, RA's were trained in administration of the PASAT, PAI, and instructed in the mental arithmetic procedure (Shostak & Peterson, 1990) for the stress condition. All administration techniques were conducted according to standardized directions as per the test manual or research procedures descriptions. The author observed the RA's practicing on non-participants before they worked with study participants. Instructions were to be read verbatim by RA's in order to minimize experimenter error. RA's were randomly observed by the author to ensure that directions were being read verbatim and followed as specified. There were no observations of noncompliance with instructions.

The consent form was presented to each participant orally and in writing with each participant retaining a copy of his or her signed consent form (see Appendix D). The investigators' names and contact numbers were on each consent form. Each participant was given between one and four extra credit points for completion of this study to use in a participating psychology class. To ensure that participants provided optimal effort on all

measures and responded to questions in an honest manner, a motivation procedure was used. Participants were told that measures in the study contained validity information and participants' honesty and effort would be assessed. If participants were determined to be answering questions candidly and providing optimal effort on all measures, they would be entered in a drawing at the end of the semester for a $50 gift certificate to a local restaurant. In actuality, all participating students were entered in the drawing regardless of their response style.

Two hundred and five participants were screened for inclusion and exclusion criteria (i.e., age >17 years, no history of seizures, no history of other neurological conditions, reading level above 4th grade, history of moderate or severe head injury, history of mild head injury within the past three months, and PCSC score between 58 and 71). All participants were screened using the clinical interview and PCSC. Based on information gained during the clinical interview and completion of the PCSC, RA's assigned participants to appropriate groups (symptomatic MTBI, nonsymptomatic MTBI, symptomatic non-MTBI, and nonsymptomatic non-MTBI) or excluded them from further participation. One hundred and twenty-five participants were excluded participants due to scoring between 58 and 71 on the PCSC or because they qualified for groups that were already filled. Participants who qualified for further participation were given an appointment time within the next two weeks to continue the study. Within each of the four groups, participants were alternately assigned to either a stress or control condition by RA's. Following group assignment and alternate placement of each participant in the stress or control condition, participants in the stress condition were instructed to engage in a mental arithmetic procedure for 3 minutes.

The mental arithmetic procedure was adapted from a study by Shostak & Peterson (1990) and required the participants to count backward from a four-digit number by decrements of a

two-digit number as rapidly and accurately as possible for three minutes, until they were told to stop. During the instructions for this task, participants were told that this task was correlated with important aspects of intellectual functioning and that their speed and accuracy would determine their score. The research assistant modeled the task at a rate of performance faster than most people's ability, thereby establishing an implicit standard for the participants to match. The examiner held a stopwatch in an obvious position and for each mistake said "wrong" and gave the last correct number. At the end of each 3-minute time period the participant was instructed to stop. For each implementation of the stress condition, different four-digit starting numbers and two-digit decrement numbers were chosen (see Appendix E). The mental arithmetic procedure has been often used as the experimental analogue for induction of physiological arousal. Researchers have shown a correlation between arousal induced by mental arithmetic procedures and subjective appraisal of stress (Manuck, Proietti, Rader, & Polefrone, 1985). Researchers have found that the mental arithmetic task reliably increased physiological reactions commonly associated with stress including heart rate (Anderson, 1981; Fahrenberg, Walschburger, Foerster, Myrtek, & Muller, 1983; Manuck & Garland, 1980), systolic blood pressure (Anderson; Manuck & Garland; Lawler, 1980), and frontalis muscle tension (Arena, Blanchard, Andrasik, Cotch, & Myers, 1983; Feuerstein, Bush, & Corbisiero, 1982).

Participants in the control condition engaged in silent passage reading during the same three minute time periods so that they would have the same interruptions, but not the added stress component. Control participants read from selected main articles in Time magazines, Spring 2003 issues.

A manipulation check was used to ensure participants in the stress condition felt elevated levels of stress. Each participant rated their stress level according to a seven-point scale with 7

being “the most stress they’ve ever experienced” and 1 being “the most relaxed they’ve ever felt.” Stress ratings occurred at the beginning of the study (immediately after the consent form was discussed and signed) and after the first presentation of the mental arithmetic or reading procedure.

Following this stress induction/control procedure, the PASAT was administered to all participants. After the PASAT, all participants engaged in their respective stress or control condition for three additional minutes, then were instructed on completion of the PAI. RA’s left the examination room during the completion of the PAI. To ensure that the participants in the stress condition continued to experience elevated stress while completing the PAI, RA’s returned to the examination room every 15 minutes and directed participants to engage in their respective stress induction or control procedure for 3 minutes; that is of every 18 minutes, three were spent in the stress/reading condition.

Before participants left, critical items assessing suicidal ideation and intent on the PAI were inspected. Participants endorsing “slightly true” or higher on any of these critical items were further assessed for suicidality by the author. When warranted, participants were given referrals for the Student Mental Health Center on LSU’s Baton Rouge campus. Eleven students were assessed for suicidality. No participants reported current passive or active suicide plans; 2 were provided the phone number to the Student Mental Health Center due to prior passive ideation.

At the end of the session, each participant was debriefed regarding the stress condition. Since future participants might have contact with past participants, the full purpose of the study was not revealed, but participants were told that some of the procedures were meant to induce stress and designed to be overly difficult. They were welcomed to return to the study site or

email or call the researcher at the end of the semester for a full disclosure of purpose, procedures, and results.

RESULTS

Preliminary Analysis

Demographic Information

Two hundred and five participants were screened to participate in the present study. Of these people, one qualifying subject withdrew from the study after 30 minutes stating that she did not want to complete the lengthy PAI. Eighty participants with the following demographics completed the study: mean age of 19.82 (*SD* = 3.23), 63 women (78.8%) and 17 men (21.2%), 69 Caucasian (86.3%), 9 African American (11.3%), 1 Hispanic (1.3%), and 1 other (1.3%) (see Table 2 for participant characteristics by group).

Table 2. Participant Characteristics by Group

	Nonsymptomatic	Symptomatic
Age		
Controls	19.95 (*SD* = 1.61)	19.55 (*SD* = 1.93)
MTBI	20.30 (*SD* = 5.90)	19.50 (*SD* = 1.36)
Sex		
Controls	f = 18, m = 2	f = 17, m = 3
MTBI	f = 14, m = 6	f = 14, m = 6
Education		
Controls	14.00 (*SD* = 1.43)	13.85 (*SD* = 1.93)
MTBI	13.25 (*SD* = 1.16)	13.68 (*SD* = 1.20)
GPA		
Controls	3.32 (*SD* = 0.45)	2.96 (*SD* = 0.47)
MTBI	3.11 (*SD* = 0.55)	3.12 (*SD* = 0.61)
Mental Health History		
Controls	no = 13, yes = 7	no = 14, yes = 6
MTBI	no = 15, yes = 5	no = 12, yes = 8

The participant groups did not differ significantly with regard to age, sex, race, socioeconomic status, education, GPA, or mental health history. However, there was a main

effect of symptom status on reported initial stress, $F(1,72) = 14.10$, $p < .001$. Participants in the symptomatic groups ($M = 4.08$, $SD = 1.05$) reported more initial stress than participants in the nonsymptomatic groups ($M = 3.13$, $SD = 1.24$). The length of time post injury for the MTBI participants at the time of testing ranged from six months to 17 ½ years ($M = 5.71$ years, $SD =$ .69 years). There was not a significant difference between symptomatic MTBI and nonsymptomatic MTBI participants in terms of length of time post injury, $t(1,39) = .63$, $p = .53$.

Group Assignment

The PCSC was used to classify participants as high symptomatic or low symptomatic (Gouvier et al., 1992). Participants with PCSC scores greater than 0.5 standard deviations from the mean were placed into either the symptomatic or nonsymptomatic group. This cutoff was chosen based on prior research with symptomatic participants (Hanna-Pladdy et al., 2001). However, to ensure that this cutoff resulted in two significantly different groups in terms of reported level of PCD symptoms, a two-way analysis of variance (ANOVA) was performed with PCSC score as the dependent variable and MTBI status and symptom status as the independent variables. There was a main effect of symptom status only, $F(1,76) = 439.39$, $p < .001$. Symptomatic MTBI ($M = 78.89$, $SD = 6.59$) and symptomatic non-MTBI ($M = 81.51$, $SD = 8.44$) groups endorsed significantly more symptoms than both nonsymptomatic MTBI ($M = 50.84$, $SD = 4.16$) and nonsymptomatic non-MTBI ($M = 51.69$, $SD = 4.52$) groups. Additionally, neither the symptomatic groups nor the nonsymptomatic groups significantly differed in their rates of symptom reporting. These results indicate that two distinct groups, based solely on PCD symptom report, were selected during the screening process.

To determine whether the stress condition (Shostak & Peterson, 1990) was effective in increasing the participants' reported stress level, a one-way ANOVA was conducted for the

difference score between first and second stress ratings. There was a main effect of stress, $F(1,78) = 37.24$, $p < .001$. Participants completing the mental arithmetic task reported a greater increase in stress than those in the control/reading condition. Participants in the stress condition reported significantly more stress following the stress induction procedure ($M_{difference} = 1.26$, $SD = 1.51$), $t(1,39) = -5.30$, $p < .001$. Participants in the reading condition reported significantly less stress after reading ($M_{difference} = -.48$, $SD = .99$), $t(1,39) = 3.04$, $p = .004$.

Neuropsychological Analyses

A 2 (MTBI status: injured vs. non-injured) x 2 (stress condition: stressed vs. non-stressed) x 2 (symptomatic status: symptomatic vs. nonsymptomatic) analysis of covariance (ANCOVA), with degree of psychological distress (as measured by the calculated PAI Distress Index score) entered as a covariate, was conducted for the PASAT fourth trial score, which was converted to a z-score. The ANCOVA was not significant for main effects or interactions.

To determine whether covarying psychological distress had masked effects of MTBI, stress, or symptom status on PASAT performance, a second 2 X 2 X 2 ANOVA was run without covarying psychological distress. This ANOVA was also not significant for main effects or interactions on PASAT performance.

Based on examination of group characteristics, beginning stress level was significantly higher for symptomatic participants than nonsymptomatic participants. Therefore, another 2 X 2 X 2 ANCOVA was run with initial stress level as a covariate. As with the original ANCOVA, there were no main effects or interactions, therefore, covarying the effects of initial stress level did not have a significant effect in the analysis.

The MTBI group was further divided into those who met MTBI criteria (Kay et al., 1993) by a history of LOC, PTA, or RA and those who met criteria by only history of alteration of

consciousness. An ANOVA comparing these two groups' performance on the PASAT indicated no significant differences.

Psychological Distress

To determine the relation between general psychological distress and postconcussive symptoms, a Pearson correlation was calculated. PMDI was significantly correlated with PCSC score ($r = .34$, $N = 80$, 2-tailed, $p = .001$). Higher general psychological distress was associated with higher PCSC scores.

A Pearson correlation was also calculated between PCSC score and subscales of the PAI. Results indicated that the PCSC is significantly correlated with the following PAI scales: Anxiety, Borderline Features, Depression, Somatic Complaints, Stress, Anxiety-Related Disorders, Nonsupport, Negative Impression Management, Positive Impression Management, Mania, Schizophrenia, Suicidal Ideation, Treatment Rejection, and Warmth (see Table 3 for correlations). Of the seven scales predicted to be correlated with the PCSC, all seven were correlated at the $p < .001$ level (Mean $r = .432$). To analyze divergent validity of this prediction, all other scales of the PAI were entered into a Pearson correlation. Of the 15 scales not predicted to be correlated with the PCSC, eight were correlated at least at the $p < .05$ level (Mean $r = .237$). PAI scales predicted to be significantly correlated with the PCSC were, in fact, correlated significantly more often than PAI scales not predicted to be correlated with the PCSC, $\chi^2(1, N = 22) = 4.8$, $p < .05$.

Table 3. PCSC Correlations with PAI Subscales

	Variable	Mean (in z-scores)	SD	Correlations 1. PCSC
Predicted Correlations	1. PCSC	0.06	1.14	1.00
	2. Somatic Complaints	-0.03	0.87	0.42**
	3. Anxiety	0.53	1.13	0.57**
	4. Anxiety Related Disorders	0.34	1.01	0.35**
	5. Depression	0.12	0.92	0.43**
	6. Borderline Features	0.55	1.01	0.49**
	7. Stress	0.08	0.98	0.41**
	8. Nonsupport	-0.15	1.03	0.35**
Non-Predicted Correlations	9. Inconsistency	-0.20	0.85	0.21
	10. Infrequency	-0.26	0.68	0.10
	11. Negative Impression Mgmt.	-0.02	0.70	0.40**
	12. Positive Impression Mgmt.	-0.39	0.92	-0.34**
	13. Mania	0.33	0.98	0.25*
	14. Paranoia	0.24	1.18	0.33**
	15. Schizophrenia	0.12	1.14	0.52**
	16. Antisocial Features	0.55	1.08	0.11
	17. Alcohol Problems	0.26	1.11	-0.04
	18. Drug Problems	-0.02	1.00	0.07
	19. Aggression	-0.16	0.96	0.10
	20. Suicidal Ideation	-0.11	0.97	0.22*
	21. Treatment Rejection	0.03	0.85	-0.41**
	22. Dominance	0.14	0.99	-0.19
	23. Warmth	0.23	1.02	-0.27*

* : Correlation is significant at the 0.05 level (2-tailed)
** : Correlation is significant at the 0.01 level (2-tailed)

To determine whether general psychological distress, as measured by the PMDI, was affected by MTBI status, PCD symptom status, and/or stress, a 2 X 2 X 2 ANOVA with PMDI as the dependent variable was conducted. There were main effects of stress, $F(1,72) = 4.52$, $p < .05$, and PCD symptom status, $F(1,72) = 7.22$, $p < .01$, on psychological distress. The participants who participated in the stress condition, regardless of PCD symptom status and MTBI status, reported a greater level of psychopathology than participants in the reading

condition. Additionally, symptomatic participants, regardless of MTBI status and stress condition, reported a higher level of psychopathology than nonsymptomatic participants.

Sex

A one-way ANOVA was conducted to determine whether PCSC score was affected by sex. There was not a main effect of sex on PCD symptoms, $F(1,78) = .12$, $p = .73$. The effect of phase of menstrual cycle on PCSC scores was also analyzed using a one-way ANOVA. There was not a main effect of phase on PCSC scores, $F(3,25) = .67$, $p = .58$. Females in the luteal phase were compared to females in non-luteal phases using a one-way ANOVA. There was not a main effect of luteal phase, $F(1,27) = 2.08$, $p = .16$.

DISCUSSION AND CONCLUSIONS

Researchers have found that nonsymptomatic participants with a history of MTBI placed in stressful conditions show more cognitive difficulties than controls (Ewing et al., 1980). MTBI history and stress have been found to be positively correlated with the development and maintenance of symptoms, with stress typically accounting for more of the variance in symptom presentation than MTBI status. Symptomatic MTBI participants exposed to high stress not only show slower information processing and subtle memory problems, but also increase in their report of postconcussive symptoms (Hanna-Pladdy et al., 2001). It has been hypothesized that while organic factors contribute to the development of PCD, psychological factors also play an important role in the development and maintenance of symptoms. It thus seems that report of psychopathology might contribute not only to the development and maintenance of PCD, but, with the addition of stress, create an additive effect on cognitive functioning.

Neuropsychological Analysis

This study examined the role of mild head injury, report of postconcussive symptoms, and stress, while controlling for the effects of psychological distress. This study hypothesized that *MTBI participants would perform significantly more poorly on the PASAT than non-MTBI participants*. This hypothesis was not confirmed. This study also hypothesized that *stressed, MTBI participants would perform significantly more poorly on the PASAT than the other three groups (stressed non-MTBI participants, non-stressed, MTBI participants, and non-stressed, non-MTBI participants)* and *symptomatic, stressed MTBI participants would perform significantly more poorly on the PASAT than nonsymptomatic, stressed, MTBI participants*. These hypotheses were also not confirmed.

The lack of significant results is surprising based on the findings reported in the literature. The initial ANCOVA yielded very small effect sizes (ranging from a high of $\eta^2 = .035$ for stress to a low of less than $\eta^2 = .001$ for the interaction between PCD symptoms and stress), significantly smaller than findings in the previous research on which the power analysis for this study was calculated (Ewing et al., 1980; Hanna-Pladdy et al., 2001; Maddocks & Saling, 1996). In fact, the Ewing et al. (2001) study ($N = 20$) had an effect size of 0.81. Similar published studies found effect sizes for main effects and interactions of MTBI, PCD symptom status, and stress on cognitive functioning to be medium ($f = .25$) to large ($f = .40$) (Hanna-Pladdy et al., 2001; Maddocks & Saling, 1996).

There are a number of reasons that significant results might not have been obtained. The method by which groups were determined must be considered as it may have assisted in lowering the effect sizes. Criteria used to identify MTBI participants were the widely used criteria developed by Kay and associates (1993). Under this definition, any traumatically induced physiological disruption of brain function resulting in the participant feeling dazed qualifies as a mild brain injury. At the other end of the continuum, the term mild brain injury can encompass someone with a blow to the head resulting in loss of consciousness for 30 minutes and PTA for 24 hours. In a college population, it is likely that students with a history of MTBI have recovered sufficiently to be accepted into and function within the academic setting. The majority of the current MTBI sample (83%) had no history of loss of consciousness, posttraumatic amnesia, or retrograde amnesia, but still met MTBI criteria (Kay et al., 1993) merely by report of transitory alteration of consciousness. None of the MTBI participants reported loss of consciousness exceeding 5 minutes. Alteration of consciousness encompassed dizziness, confusion, visual disturbances, and feeling dazed, disoriented, or confused. Headache

only, following a blow to the head, was not considered as indicative of MTBI for this study. A definition of "very" mild brain injury is rarely used as most researchers classify based on mild, moderate, and severe criteria, but it would assist in the classification of mild brain injured patients.

In 2002, a Task Force on Mild Traumatic Brain Injury was set up under the auspices of the European Federation of Neurological Societies (EFNS; Vos et al.). This Task Force, performing a search of journals in the MEDLINE database, published a more specific division of the mild brain injury group. They classified mild brain injury into four categories: (0) GCS = 15, no LOC, no PTA, head injury without traumatic brain injury, and no risk factors; (1) GCS = 15, LOC < 30 minutes, PTA < 1 hour, no risk factors; (2) GCS = 15, risk factors present; (3) GCS = 13-14, LOC < 30 minutes, PTA < 1 hour, with or without risk factors. The risk factors referenced in this classification include unclear or ambiguous accident history, continued PTA, retrograde amnesia longer than 30 minutes, trauma above the clavicles including skull fracture, vomiting, focal neurological deficit, seizure, age less than 2 years, age greater than 60 years, coagulation disorders, and high energy impact (Vos et al., 2002).

Examination of the MTBI and non-MTBI groups in this study indicates that it is likely that the MTBI criteria used (Kay et al., 1993) did not result in two distinctly different groups. The Kay et al. criteria encompass such a large range of injury severity that distinct differences likely exist between someone at the low end of the spectrum and someone at the high end. Based on Vos et al.'s (2002) criteria, approximately 83% of the current college sample fell into Category 0 and 17% fell into Category 1 with no known participants meeting criteria for Category 2 or 3. The results of this study then indicate that there are no significant effects or interactions of PCD symptom status, stress, and MTBI status (no MTBI versus Category 0-1

MTBI) on complex attention. This interpretation of results should not be confused with the overly general assumption that MTBI patients do not differ from non-MTBI patients, which is not accurate when considering the methodology of this study. Based on the current sample, no conclusions can be drawn regarding MTBI patients in Categories 2 or 3. When Category 0 (n = 83) participants were compared to Category 1 (n = 17) participants, there was not a significant difference. However, there was a large difference in number of subjects per group and the effect size ($\eta^2 < .01$) was extremely small.

These newly developed criteria (i.e., Vos et al.) will likely be beneficial in future research on mild brain injury and help to more specifically define groups. It is strongly recommended that this criteria, or similarly specific criteria, be used in future research on mild head injury. While the liberal Kay et al. criteria is needed to ensure that all possible brain injuries, and, therefore, any resultant symptoms, and identified, the further delineation provided by Vos et al. is needed to differentiate severity levels of MTBI.

Since the results were nonsignificant when psychological distress was covaried, it was possible that removing the effects of psychological distress might be removing one of the key variables. However, when psychopathology was not covaried, results remained nonsignificant. In fact, there were no main effects or interactions of MTBI, symptom status, or stress on complex attention regardless of whether psychological distress was covaried in the analysis.

Another consideration was participant effort and motivation. Observations of participants during the experiment, particularly during the administrations of the PASAT, suggested some variations in motivation. Participants appeared to be initially motivated, but as trials became more difficult, participants sometimes gave up or laughed at the level of difficulty and their performance. The PASAT is a difficult task and has been shown to induce negative

mood in normal mood participants (Holdwick & Wingenfeld, 1999). To ensure that using only PASAT trial 4 as the dependant variable had not obscured any real differences, four exploratory ANCOVA's were conducted using PASAT trials 1, 2, 3, and an average of all four trials as dependent variables. As with PASAT trial 4, there were no significant effects or interactions of the independent variables on any of the PASAT trials or composite score.

Based on the observations made during the testing of participants and results, as well as previous research revealing a strong severity–deficit correlation when poor-effort participants are excluded (Green et al., 2001), future studies of this nature should include an assessment of effort with removal of participants failing the effort test. Although few study volunteers receiving college credit would have any tangible reason to malinger, it is possible that they might not be motivated enough to provide full effort, especially on a test as difficult as the PASAT.

Lastly, the method of data collection for stress level must be considered. Current stress levels were obtained at the start of the experiment and immediately following the stress or reading condition. While participants in the stress condition reported a significant elevation in level of stress following the mental arithmetic procedure, self-report data must always be interpreted cautiously. Participants tend to want to respond in the way the researcher is biased (Rosenthal, 2002). Although previous researchers have recorded reliable physiologic increases in stress during the mental arithmetic task used in this study (Shostak & Peterson, 1990), without obtaining the same physiological measures, the participants' true internal states, such as blood pressure or muscle tension, are not known, only that they reported increases in stress.

Psychological Distress

With the interaction hypothesis of PCD pathogenesis gaining more support in the literature, it is important to determine what role psychopathology plays in the development and

maintenance in PCD. The hypothesis stating *level of psychological distress would be significantly positively correlated with level of PCD symptomatology after three months* was supported by a significant correlation between PMDI scores and PCSC scores. PMDI scores accounted for 12% of the variance in PCD symptom report. While correlated, there is still a large amount of variance that is unaccounted for by psychopathology. The hypothesis stating *levels of depression, anxiety, anxiety-related disorders, trait stress, somatic complaints, level of perceived non-support, and borderline features would each be significantly positively correlated with level of PCD symptomatology after three months* was also supported by significant correlations between PCSC scores and each of the above mentioned PAI clinical scales. Additionally, divergent validity was assessed by determining correlations between PCSC scores and PAI scales not hypothesized to be correlated. Of the scales hypothesized to be correlated with the PCSC, 7/7 were correlated at $p < .001$; of those not hypothesized to be correlated, 5/15 were correlated at $p < .001$ and 3/15 were correlated at $p < .05$. Discriminant validity was shown by the significant difference in number of significant correlations between predicted and non-predicted PAI and PCSC correlations.

Upon examining the effects of MTBI status, PCD symptom status, and stress on psychological distress, analysis revealed that participants with high levels of stress and high PCD symptom report reported greater levels of psychological distress. Previous research has not examined the role of psychopathology on PCD symptom status or its additive effect on cognitive functioning with MTBI and stress. These findings indicate that this is an area that needs further research if we are to more specifically understand the multifactorial pathogenesis of PCD.

Sex

Researchers have found female sex to be a contributing factor to PCD symptoms (Alves et al., 1993; Santa Maria et al., 2001). This finding was not supported in this study. Females were not significantly more likely than males to report PCD symptoms. However, since this finding had been observed in the past, phase of menstrual cycle was examined to determine whether phase was a contributing variable in female PCD scores. A significant relationship between phase and PCD symptom report was not found. These results might have been due to the small number of subjects in three of the four phases at the time of testing; 52% of subjects were in the luteal phase (n = 15), 21% were in the late follicular phase (n = 6), 17% were in the menses phase (n = 5), and 10% were in the peri-ovulatory phase (n = 3). Females are in the luteal phase for half of the cycle and therefore, without a large enough group of participants, the size of the other groups can easily be too small to detect a difference. The small effect size ($\eta^2 <$ .1) might be an indication that there were not enough subjects to detect a statistically significant difference. The luteal phase is marked by the highest level of progesterone; to determine whether level of progesterone had an effect on report of postconcussive scores, non-luteal phase subjects were collapsed into one group and compared to females in the luteal phase. There was not a main effect of luteal phase. This analysis was also marked by a small effect size ($\eta^2 < .1$). Future research should re-investigate the relationship between sex and postconcussive symptoms ensuring a large enough sample size in each group.

Conclusion

The study of PCD and its developing and maintaining factors is an important area needing further research. When 2 million Americans suffer head injuries every year, and the vast majority of those head injuries are considered mild, the pattern of postconcussional

symptoms following MTBI deserves continued investigation. Some limitations of this study were discussed and recommendations were made for future research. Limitations include more females than males in this sample, non-blinded examiners, low power, large range of MTBI criteria, possibly low effort on measures, and the inherent limitations of self-report data. Related recommendations include using more specific criteria for classifying mild head injury (Vos et al., 2002), controlling for effort, recording behavioral observations, and obtaining physiological measures of stress versus self-report.

Replication of Ewing et al.'s (1980) study with symptomatic groups is a necessary next step. Using the less common four group design allows the researcher to examine the effects of MTBI status (MTBI versus no MTBI) and symptom status (presence of PCD symptoms versus nonsignificant PCD symptoms) on the construct of interest. If only head injured subjects are compared to controls symptom status is a confound (see Figure 1). For example, a hypothetical finding showing that MTBI subjects perform more poorly on a measure than controls could, in fact, be due to the performance of the *symptomatic* MTBI subjects. However, by using only a two group design, this difference would be obscured. This more informative four-group design should be used in future research examining PCD and MTBI.

Symptomatic MTBI	Nonsymptomatic MTBI
Symptomatic Non-MTBI	Nonsymptomatic Non-MTBI

Symptomatic MTBI	
	Nonsymptomatic Non-MTBI

Figure 1. Four Group Versus Two Group Design

Secondly, determining the specific role of psychopathology in the development and maintenance of postconcussive symptoms and its interaction with stress might help to determine individuals at risk for developing PCD. With this information, early interventions (Hanna-

Pladdy et al., 2001; Mittenberg et al., 1996) tailored more specifically to the needs of these individuals may be implemented.

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APPENDIX A

STRUCTURED CLINICAL INTERVIEW

Participant #:_____ Date:_____

Gender: M F Age:_____

Race: Caucasian

African American

Hispanic

Asian

Other:_____

Years of education (years & college status):_____

Current GPA:_____

Estimated parents' income: $0 – 20,000

20, 001 – 40, 000

40, 001 – 60, 000

60, 001 – 80, 000

Greater than 80, 001

Have you ever had a head injury? Y N

(Have you ever been hit on the head hard enough to make you see stars [dizziness, trouble concentrating after, confused] or lose consciousness, even for a few minutes?)

If Yes, describe head injury(ies):

(1)_____

_____

When did this happen?_____

Knocked out? Y N If yes, for how long? _____ *(20 minutes or less? ___)*

Where on your head were you hit?_____

Was your brain exposed? Y N _____

Do you remember what you were doing immediately before the accident? Y N

If not, what is the last thing you remember and how long before the accident was it?_____

What is the first thing you remember after the accident? *(PTA? Y N)*

_____

(2)_____

_____

When did this happen?_____

Knocked out? Y N If yes, for how long? _____ *(20 minutes or less? ___)*

Where on your head were you hit?_____

Was your brain exposed? Y N _____

Do you remember what you were doing immediately before the accident? Y N

If not, what is the last thing you remember and how long before the accident was it?_____

What is the first thing you remember after the accident? *(PTA? Y N)*

_____

(3)_____

_____

When did this happen?_____

Knocked out? Y N If yes, for how long? _____ *(20 minutes or less? ___)*

Where on your head were you hit?_____

Was your brain exposed? Y N _____

Do you remember what you were doing immediately before the accident? Y N

If not, what is the last thing you remember and how long before the accident was it?_____

What is the first thing you remember after the accident? *(PTA? Y N)*

_____

Have you ever been in counseling/mental health treatment? Y N

If yes, please describe:_____

Have you ever been hospitalized for mental health reasons? Y N

Have you ever been diagnosed with a mental health disorder, like depression or anxiety? Y N _____

FOR FEMALES ONLY:

What was the first day of your last period?_____

What was the last day of your last period?_____

How long is your menstrual cycle (day 1 of period to day 1 of next period)?_____

Are you taking any form of birth control? Y N

If yes, what type/brand?_____

If an oral contraceptive, what pill are you on?_____

Rule-outs

Do you have any neurologic conditions: Y N

Do you have a seizure disorder? Y N

Have you ever had a head injury when you were knocked out for more than 20 minutes? Y N

Have you had any head injuries, been knocked out, or "seen stars" within the past three months? Y N

Please read this sentence and explain what it means.

I do not worry about other people's concerns and things that I cannot control.

APPENDIX B

POSTCONCUSSION SYNDROME CHECKLIST

Participant #:_____ Date:_____

Please rate the frequency, intensity, and duration of each of the following symptoms based on how they have affected you *during the past 2 months* according to the following scale:

FREQUENCY	INTENSITY	DURATION
1=Not at all	1=Not at all	1=Not at all
2=Seldom	2=Vaguely present	2=A few seconds
3=Often	3=Clearly present	3=A few minutes
4=Very often	4=Interfering	4=A few hours
5=All the time	5=Crippling	5=Constant

	FREQUENCY	INTENSITY	DURATION
Headache	_____	_____	_____
Dizziness	_____	_____	_____
Irritability	_____	_____	_____
Memory Problems	_____	_____	_____
Difficulty Concentrating	_____	_____	_____
Fatigue	_____	_____	_____
Visual Disturbances	_____	_____	_____
Aggravated by Noise	_____	_____	_____
Judgment Problems	_____	_____	_____
Anxiety	_____	_____	_____

Thank you for your time and effort in the completion of this form.

Raw total score = _____z-score = _____

APPENDIX C

PASAT

Participant Number:_____ Date:_____
Examiner:_____

Instructions: (On tape recording) If the participant is having difficulty, provide a written demonstration: **Let me show you what they mean.** Write down *'5,3,7,4,2'*. **You see, you add the '5' and the '3' together, and say '8'; then you have to forget the '8' and remember the '3'. When the '7' comes along, you add it to the '3', and say '10'; now you have to remember the '7'. All right. What do you say after '4'?** Keep going until the participant understands.

PASAT Score Sheet

2	2.4	2.0	1.6	1.2		2.4	2.0	1.6	1.2		2.4	2.0	1.6	1.2
7 (9)					8 (12)					5 (13)				
5 (12)					7 (15)					4 (9)				
1 (6)					1 (8)					8 (12)				
4 (5)					6 (7)					2 (10)				
9 (13)					3 (9)					1 (3)				
6 (15)					5 (8)					7 (8)				
5 (11)					9 (14)					5 (12)				
3 (8)					2 (11)					9 (14)				
8 (11)					7 (9)					1 (10)				
4 (12)					5 (12)					3 (4)				
3 (7)					3 (8)					6 (9)				
2 (5)					4 (7)					2 (8)				
6 (8)					7 (11)					9 (11)				
9 (15)					1 (8)					7 (16)				
3 (12)					5 (6)					8 (15)				
4 (7)					8 (13)					2 (10)				
5 (9)					3 (11)					4 (6)				
8 (13)					4 (7)					7 (11)				
6 (14)					6 (10)					6 (13)				
4 (10)					8 (14)					3 (9)				

Total Correct

2.4" pacing _____
2.0" pacing _____
1.6" pacing _____
1.2" pacing _____ z-score _____

APPENDIX D

INFORMED CONSENT FORM

Louisiana State University
236 Audubon Hall
Baton Rouge, LA 70803-5501
(225) 578-1494 Phone • (225) 578-4661 Fax

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1. Study Title:
   Psychological and Neuropsychological Correlates of Postconcussional Disorder

2. Performance Site:
   Louisiana State University

3. Investigators:
   The investigators listed below are available to answer questions about the research, M-F, 8:00 a.m. – 4:00 p.m.

   Wm. Drew Gouvier, Ph.D. & Joy Wymer, M.A.
   578-1494

4. Purpose of the Study:
   The purpose of this research project is to identify the psychological and neuropsychological correlates associated with postconcussive symptoms.

5. Participants
   A. Inclusion criteria: ≥18 years old
      Current undergraduate students at LSU
   B. Exclusion criteria: Individuals who have suffered a moderate or severe head injury
      Individuals who have suffered a mild head injury *within the past 3 months*.
      Neurological disease or seizure disorder
      Illiteracy
   C. Maximum number of participants: 200

6. Study Procedures:
   Each participant will be interviewed about their medical and psychological history, complete a questionnaire about physical, cognitive, and/or emotional symptoms take a test of cognition, and complete a personality questionnaire. Some of these procedures may be conducted under stressful conditions. Interview plus questionnaires should not exceed two 2 hours and will occur at one scheduled appointment.

7. Benefits:

Each participant will receive four (4) extra credit points for full participation in this two (2) hour study. Students who respond candidly to questionnaires, as measured by internal validity scales, on each measure, will be entered in a drawing for a chance to win a $50 gift certificate to a local restaurant. Information gained from this study may help us to determine why certain individuals recover more slowly from head injuries and how they can be better treated.

8. Risks/Discomforts:

There is no known risk associated with participation in this study above what might be experienced during an average day.

9. Injury/Illness:

To assure that participants' privacy is respected, this study will be anonymous.

10. Right to Refuse:

Participation in this study is completely voluntary and participants may change their minds and withdraw from the study at any time without penalty.

11. Privacy:

Participants' names on consent forms will not be able to be linked to interview and questionnaire responses. Additionally, consent forms will be stored separately from data.

The LSU Institutional Review Board (which oversees university research with human participants) and Wm. Drew Gouvier, Ph.D. may inspect and/or copy the study records.

Results of the study may be published, but no names or identifying information will be included in the publication.

12. Financial Information:

There is no cost to the participants. Participants will receive four (4) extra credit points for participation in this study.

13. Withdrawal:

You may withdraw from this study at any time. However, extra credit points will not be given for less than full participation. To withdraw, inform the principal investigator or research assistant of your decision.

14. Removal:

If it becomes apparent that the participant is not responding in a forthright manner or additional information suggesting that a participant meets exclusion criteria is disclosed later in the study, the participant will be removed from the study without his or her consent.

The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigator or research assistants. If I have questions about participants' rights or other concerns, I can contact Robert C. Mathews, Chairman, LSU Institutional Review Board, (225) 578-8692. I agree to participate in the study described above and acknowledge the investigator's obligation to provide me with a signed copy of the consent form.

Participant Signature_____

Participant Name (Print)_____

Date_____

Participant phone number_____ or email_____ (for gift certificate purposes only)

Witness Signature_____

Date_____

APPENDIX E

MENTAL ARITHMETIC PROCEDURE

Mental Arithmetic Procedure Participant #:_____ Date:_____

~ Before PASAT, before PAI, during PAI (every 15 minutes)

~ "This is an arithmetic test that is highly correlated with important aspects of intellectual functioning. I will give you a starting number and ask you to count backward by another number for 3 minutes. For example, I could say *Begin at 100 and count backward by 3's*. You are to count backward as quickly as you can without making mistakes. If you make a mistake, I will say *Wrong*, give you the last number you got correct, and you will continue counting backward from that point. You score will be based on your speed and number correct. *Model task. Start at 1100 and count backward by 12's.* Do you have any questions? Now begin at 1300 and count backward by 17's. Go." *Time 3 minutes. Mark stop point.*

~ 2nd and additional presentations: "I am going to give you a new number to start counting from and a new digit to count backward by. Again, your performance will be measured by your speed and accuracy. Begin at #### and count backward by ##. Go." *Time three (3) minutes.* Stop. *Mark stop point.*

1300 (17)	1780 (13)	2801 (14)	3395 (16)	2277 (17)
1283	1767	2787	3379	2260
1266	1754	2773	3363	2243
1249	1741	2759	3347	2226
1232	1728	2745	3331	2209
1215	1715	2731	3315	2192
1198	1702	2717	3299	2175
1181	1689	2703	3283	2158
1164	1676	2689	3267	2141
1147	1663	2675	3251	2124
1130	1650	2661	3235	2107
1113	1637	2647	3219	2090
1096	1624	2633	3203	2073
1079	1611	2619	3187	2056
1062	1598	2605	3171	2039
1045	1585	2591	3155	2022
1028	1572	2577	3139	2005
1011	1559	2563	3123	1988
994	1546	2549	3107	1971
977	1533	2535	3091	1954
960	1520	2521	3075	1937
943	1507	2507	3059	1920
926	1494	2493	3043	1903
909	1481	2479	3027	1886
892	1468	2465	3011	1869
875	1455	2451	2995	1852
858	1442	2437	2979	1835

VITA

Joy Helena Wymer earned her Bachelor of Science with a double major in psychology and Russian at James Madison University in Harrisonburg, Virginia, in May 1996. She attended The Citadel in Charleston, South Carolina, where she earned her Master of Arts in psychology in May 1999. Ms. Wymer is currently a Doctor of Philosophy candidate in the Department of Psychology at Louisiana State University and Agricultural and Mechanical College in Baton Rouge, Louisiana. She is in the Adult Clinical Psychology program and is specializing in neuropsychology under the supervision of Wm. Drew Gouvier, Ph.D. Her main research interests are postconcussional disorder, malingering, and dementia profiles. Ms. Wymer plans to complete her predoctoral internship at the Medical University of South Carolina in Charleston, South Carolina, in July 2004.