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# **UMI**

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**The Relationship of Personality Disorders and Persistent Post Concussive Syndrome  
in Mild Head Injury**

by

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**A Dissertation Submitted in Partial Fulfillment of the  
Requirements for the Degree of**

**DOCTOR OF PHILOSOPHY**

**in the Department of Psychology**

**We accept this dissertation as conforming  
to the required standard**

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### ABSTRACT

The relationship of personality disorders and persistent post concussive syndrome (PPCS) in mild head injury was investigated. Personality disorders were measured with the Millon Clinical Multiaxial Inventory-II (MCM-II). Mild head injury referrals were compared to a moderate head injury group, (n=46), and to a non-head injured neurological control group, (n=93). There was little evidence to suggest that the mild traumatic brain injury (TBI) group had more personality disorders than either of the two comparison groups. The mild TBI group did endorse more passive-aggressive, aggressive-sadistic, self-defeating and borderline personality traits; however, the overall scores were below ranges which indicate a personality disorder. The relationship between personality disorders (the MCM-II) and emotional status, as measured by the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) was also examined. Neither maladaptive personality characteristics or psychological distress were related to performance on neuropsychological tests. The results are discussed within the context of physiological and psychological determinants of the PPCS.

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## LIST OF ACRONYMS

<b>AIRS</b>	<b>=</b>	<b>Average Impairment Rating Scale</b>
<b>BR</b>	<b>=</b>	<b>Base Rate score</b>
<b>C.C.</b>	<b>=</b>	<b>Canonical Correlation</b>
<b>CanVar</b>	<b>=</b>	<b>Canonical Variate</b>
<b>DSM (3-R, IV)</b>	<b>=</b>	<b>Diagnostic and Statistical Manual of Mental Disorders</b>
<b>HRNB or HRB</b>	<b>=</b>	<b>Halstead-Reitan Neuropsychological Battery</b>
<b>HII</b>	<b>=</b>	<b>Halstead Impairment Index</b>
<b>MCMII (II)</b>	<b>=</b>	<b>Millon Clinical Multiaxial Inventory</b>
<b>MHBI</b>	<b>=</b>	<b>Millon Behavioral Health Inventory</b>
<b>MHI</b>	<b>=</b>	<b>Mild Head Injury</b>
<b>MMPI (2)</b>	<b>=</b>	<b>Minnesota Multiphasic Personality Inventory</b>
<b>MVA</b>	<b>=</b>	<b>Motor Vehicle Accident</b>
<b>NRI</b>	<b>=</b>	<b>Neurologically Related Item (re: MMPI)</b>
<b>PCS</b>	<b>=</b>	<b>Post Concussive Symptoms/Syndrome</b>
<b>PPCS</b>	<b>=</b>	<b>Persistent Post Concussive Symptoms/Syndrome</b>
<b>SIP</b>	<b>=</b>	<b>Sickness Impact Profile</b>
<b>TBI</b>	<b>=</b>	<b>Traumatic Brain Injury</b>
<b>TPT</b>	<b>=</b>	<b>Tactual Performance Test</b>
<b>WAIS-R</b>	<b>=</b>	<b>Wechsler Adult Intelligence Scale-Revised</b>
<b>WMS-R</b>	<b>=</b>	<b>Wechsler Memory Scale-Revised</b>

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# **THE RELATIONSHIP OF PERSONALITY DISORDERS AND PERSISTENT POST CONCUSSIVE SYNDROME IN MILD HEAD INJURY**

## ***Introduction***

Mild head injury is one of the most common causes of neurological impairment and accounts for approximately 80% of all head trauma hospital admissions (Alexander, 1995; Kraus & Nourjah, 1988). Mild head injury is typically defined as an injury to the head resulting in a brief loss of consciousness or a period of being dazed with no loss of consciousness, post-traumatic amnesia of less than 1 hour, Glasgow Coma Scale greater than 13, and a negative neuroimaging scan (Alexander, 1995; Williams, Harvey, Levin & Eisenberg, 1990). Symptoms following a mild head injury include headaches, dizziness, fatigue, attention and concentration difficulties, memory problems, and increased sensitivity to light and sound (e.g., Binder, 1986). This constellation of symptoms is commonly referred to as post concussive symptoms and/or the Post Concussive Syndrome (PCS).

Although the majority of both neurological and neuropsychological symptoms resolve within 3 months (Levin et al., 1987; Levin, Williams, Eisenberg, High & Guinto, 1992), approximately 10 to 15% of people with mild head injuries do not recover within 3 months and complain of *persistent* post-concussive symptoms (Alves, Macciocci & Barth, 1993; Brown, Fann & Grant, 1994; Evans, 1992; McAllistar, 1992). Individuals who complain of persistent sequelae such as chronic headaches, attention-concentration difficulties, and memory problems will often report an inability to complete premorbid workloads and/or

premorbid social recreational activities despite few, if any, neurophysiological and neuropsychological indicators consistent with the individuals' complaints (Dikmen, McLean & Temkin, 1986; Fenton, McClelland, Montgomery, MacFlynn & Rutherford, 1993; Slater, 1989). It has therefore been important to investigate this small yet consistent subgroup of patients with mild head injury who acquire persistent post-concussive symptoms (PPCS).

The PPCS poses several difficulties for the neurologist and neuropsychologist alike. Neuroimaging scans do not always detect the full extent of reported deficits. Neuropsychological tests also do not always detect the deficits that would corroborate the client's complaints. The discrepancy between the magnitude of reported symptoms and the seemingly mild insult to the brain led clinicians and researchers to look at possible motivational or psychological contributions to the PPCS. For example, it was a common belief in the past that individuals seeking monetary compensation through insurance companies or employee compensation agencies were more likely to maintain their symptoms until compensation was obtained (e.g., Miller, 1961). The term was coined "accident neurosis" or "compensation neurosis".

Both physical and psychological factors have been used to explain why the majority of people recover from MHI within 3 months, and why a consistent minority report persistent sequelae. The most common explanation states that the post concussive symptoms experienced during the first three months post-injury are a result of neurophysiological influences (e.g., shearing forces in acceleration-deceleration injuries, edema, etc.). However, if post-concussive symptoms persist past 3 or 4 months then psychological factors have been presumed to contribute to the maintenance and possible exaggeration of the symptoms

poor coping mechanisms and individual vulnerability are *thought* to contribute to the psychogenesis of the PPCS (Barth, 1996; Cicerone, 1991; Fenton et al., 1993), although concrete, objective evidence is lacking.

Personality traits are "enduring patterns of perceiving, relating to, and thinking about the environment and oneself, and are exhibited in a wide range of important social and personal contexts" (Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, Revised (DSM-3-R), p. 335). It is when personality traits become inflexible, maladaptive, and cause significant distress or functional impairment do they become personality *disorders* (DSM-3-R). Longstanding pervasive personality traits or disorders are in contrast to mood states, which are more transient, situation-dependent and often short-lived emotional reactions.

Individuals with personality disorders or maladaptive personality traits are known to have difficulty adjusting to significant life changes. It is therefore logical to question the extent to which maladaptive personality styles and/or personality disorders per se, affect the outcome from mild head trauma. Given that the mild nature of head trauma is unlikely to affect brain-related personality structures, such as that seen in more severe damage (Parker, 1991), one aim of the present study therefore, is to investigate how premorbid personality disorders and maladaptive personality characteristics affect the adaption to, and, outcome from mild head trauma and persistent post concussive symptoms.

## REVIEW OF THE LITERATURE

It is a recently held belief that post concussive symptoms and the persistent post concussive syndrome are a corollary of both physiological and psychological factors (Lishman, 1988; Rutherford et al., 1978). In general, this view purports that cerebral sequelae caused by head injury commonly result in a nuclear group of symptoms. Initially these symptoms are firmly organic in origin. As the weeks pass, the severity of symptoms will recede by a natural process of healing. If the environment is left undisturbed, recuperation will, in most cases, be complete.

However, in some milder forms of head injury, obstacles interfere with the healing. Some obstacles may include being more acutely aware of symptoms and how they affect daily functioning, as well as anxiety, stress, and poor coping mechanisms. There may be a tendency to worry unduly, a family who over-focuses on the injury, pre-existing domestic problems, financial pressure, or resentment toward the accident itself. Later there may be the stress and anxiety around pursuing litigation, adding significantly to stress levels. This scenario provides the ideal ground for elaboration of symptoms. Within this framework, the evidence contributing to both the physiological and psychological aspects of PPCS will be reviewed below.

## *The Physiogenesis of Post Concussive Symptoms*

### *Definition of Mild Head Injury*

As described earlier, mild head injury is typically defined as an injury to the head resulting in a loss of consciousness of less than 20 minutes, or, a period of being dazed with no loss of consciousness, post-traumatic amnesia of less than 1 hour, Glasgow Coma Scale greater than 13, and a negative CT or MRI scan (Alexander, 1995; Williams, Harvey, Levin & Eisenberg, 1990). Trauma to the brain that causes some degree of disorientation or a loss of consciousness typically results in a constellation of symptoms, including one or more of the following: headaches, dizziness, vertigo, tinnitus, blurred vision, diplopia, light and noise sensitivity, diminished taste and smell, irritability, anxiety, depression, personality change, fatigue, sleep disturbance, decreased libido, decreased appetite, memory dysfunction, impaired concentration and attention, slowing of reaction time, and slowing of information processing speed (e.g., Evans, 1992). The most common complaints following mild TBI are headaches, dizziness, nausea, memory problems, fatigue, irritability, anxiety, insomnia, loss of concentration, and noise sensitivity (Binder, 1986; Dikmen, McLean & Temkin, 1986; Alves et al., 1993). Moreover, headaches, dizziness, nausea and memory problems appear to be symptoms that are unique to head injury patients compared to non-head injured hospitalized controls (Barth, Alves & Ryan, 1989; Bohnen, Twijnstra & Jolles, 1992).

In practice however, the lack of medical urgency of these cases rarely warrants an order for a CT scan when individuals present themselves at the Emergency Room of the hospital. The so called 'gold standard' for defining a mild head injury has become a brief loss of consciousness and GCS of 13 or higher. This definition is predominantly medically

driven and is typically given shortly after the patient is sent home from the ER.

Unfortunately, this definition, taken literally, could result in a considerable number of false negative diagnoses. For example, 22% of gun shot wounds to the head do not result in a loss of consciousness (Varney, 1997). A famous neurological case, a patient named Phineas Gage, suffered a serious injury in which a steel bar entered his brain by the orbital area, piercing the frontal lobes and exiting out the posterior end of the brain. This man did not lose consciousness, drove himself to the hospital, had a transitory PTA and was speaking coherently at the hospital. By definition, this man received a mild head injury!

Common practice in hospital emergency rooms is to use only the GCS. Yet, of those cases classified as mild by this criteria, more than 20% of cases will result in definite neurological complications (Kraus, & Nourjah, 1989). The lack of sensitivity of the GCS and LOC in predicting outcome in milder cases of head injury is now being realized (Hugenholtz, Stuss, Stethem, & Richard, 1988; Kraus, & Nourjah, 1989; Stein, Soettell, Young, & Ross, 1993; Vilkki, Ahola, Holst, Ohman, Servo, & Heiskanen, 1994). A more comprehensive definition is needed for mild brain injury, and in the absence of a sensitive neuro-imaging scans shortly following the actual injury, the terms "minor" and "mild" should be used cautiously. In particular, the definition should include some reference to the fact that the individual has to have received a hit, blow, whiplash or acceleration-deceleration mechanism, sufficiently hard enough to possibly inflict some level of disruption to brain tissue. The distinction of severity level in TBI should also include reference to the degree of neuro-cognitive impairment. The lasting effects on an injury to the brain, as it affects cognitive abilities, may be different from the status of the patient in the Emergency Room at the

hospital. Although "guesstimates" of severity of injury are made at hospital admission based on LOC, the application of a true severity level should be delayed until a measure of neuropsychological abilities can be obtained. The only difficulty with this suggestion is that the level of severity could not be made until at least three months following the assumed mild TBI.

### Neuro-Imaging Studies

With the advent of neuroimaging studies, clinicians were able to investigate and understand more clearly the neuropathology of MHI. In general practice, individuals with MHI typically do not warrant a referral for a computer tomography (CT) or Magnetic Resonance Imaging (MRI) scans, due to the "mild" nature of the trauma and lack of neurological signs on initial examination. It is with the advent of research projects funding the cost of neuroimaging that we are beginning to discover that even a brief loss of consciousness can result in visible damage to brain tissue. For example, in a sample of 690 mild head traumas, with suspected LOC and GCS  $\geq$  13, over 23% of patients had a intracerebral lesion identified on their CT scan (Stein, Spettell, Young & Ross, 1993).

In a series of studies where both CT and MRI scans were taken in hospital, over 80 percent of the scans depicted at least one visible lesion (Levin et al., 1987; Levin et al., 1992). Lesion sites were still detectable at 1-month post injury, although significantly smaller, and were non-detectable by the 3-month follow-up. These neurophysiological studies demonstrate that actual physical damage can occur, even in seemingly mild injuries, and give unequivocal support for the physiogenesis of post concussive symptoms. That the

lesions are no longer visible by 3 month follow-up is consistent with the typical recovery reported by the majority of MHI individuals.

In contrast, not all mild head injuries have normal imaging scans after three months (Ruff, Crouch, Troster, Marshall, Buchsbaum, Lottenberg, & Somers, 1994). Ruff et al., (1994), investigated 9 MHI patients, 4 of whom did not experience a loss of consciousness. The subjects had negative CT and MRI but demonstrated deficits on neuropsychological testing and were reporting significant post-concussive symptoms. Follow-up Positron Emission Tomography (PET) at one year post injury revealed several areas of reduced functioning in all 9 MHI patients. The PET findings were consistent with deficits found on the neuropsychological tests for those with and without a loss of consciousness. The authors conclude that both PET and neuropsychological testing appear to be sensitive to focal lesions and contusions, as well as to superimposed diffuse damage.

There is also preliminary evidence to suggest the PET scanning can detect seizure foci, missed by EEG, years following a medically defined mild head trauma (Varney, 1997). Symptoms of complex partial seizures have considerable overlap with post-concussive symptoms. Small lesions incurred at the time of the initial injury may produce scar tissue, which may then be vulnerable to develop into seizure activity. This is an important line of investigation in that, post-traumatic seizure syndrome could provide an alternative explanation to the so called persistent post concussive syndrome. It could explain the maintenance of PCS-like symptoms, as well as accounting for depressive presentations on interview and on psychological testing. Many patients treated with anti-convulsants instead of anti-depressants following a MHI are relieved of their symptoms (Varney, 1997).

While the Levin et al. studies provide physiological support for the more typical recovery process following MHI, the Ruff et al. and Varney studies provide physiological support for the minority of MHI individuals who do not follow the typical 3-month recovery. Thus, the symptom complex following a mild head injury may not necessarily be a functional disorder, but the result of an organically based neurological trauma. If organic injury is not found, perhaps it is a function of the sensitivity of the neuro-imaging techniques, with PET more sensitive than MRI (Ruff et al., 1994) and MRI more sensitive than CT (Levin et al., 1987; 1992; Wilson, Wiedman, Haddeley, Condon, Teasdale, & Brooks, 1988).

### Neuropsychological Studies

One of the most difficult problems in neuropsychological follow-up studies of mild head injury is attrition rates of the original subject sample. This is particularly problematic when studying concussion and/or mild head injury, in that from the onset, the examiner knows that 80 to 85 percent of the sample will no longer suffer sequelae from the brain trauma, and thus carry a low probability of returning 1 month, 3 months, or 1 year later for the sake of "research". Employment, family responsibilities, travel and incidental costs are realities that the subject samples need to consider first. Thus, many follow-up studies of mild head injury incur relatively high attrition rates. Overall, neuropsychological studies using MHI samples tend to be inconclusive, in that, for every study that finds neuropsychological deficits, there is one that fails to find differences. Part of this apparent inconsistency in results may be a function of the rate of attrition and the nature of the sample used. In reviewing the literature, it was noted that studies of neuropsychological functioning of mild

head injury tend to fall into roughly three categories: those that somehow minimize the attrition and maintain a majority of the original MHI sample; those consisting only of patients that complain of persistent post concussive symptoms; and those that partial their MHI sample based on neurological or subjective differences. Not surprisingly, the three styles of studies tend to result in 3 styles of outcomes.

Studies that are able to track and follow patients from hospital discharge to follow up time tend not to find differences on neuropsychological tests compared to controls. Levin et al. (1987) from an original sample of 155 one week following mild head trauma, continued with 57 subjects at the one month follow-up, and 32 at the 3 month follow-up. The MHI sample at one week performed significantly worse than non-concussed controls on 7 neuropsychological measures of attention, memory and information processing speed. By one month, differences were found on tests of short term attention and speed of information processing (e.g., digit span, PASAT, and finger tapping). By the 3-month mark, the MHI group were performing similarly to the controls on all measures. Although attrition rates seem high, they are fairly good compared to other studies. Therefore, the sample that was studied can be considered representative of a general MHI group (e.g., sample includes individuals with favourable and unfavourable recoveries).

Another study with good follow-up examined 436 head injured individuals with a full neuropsychological test battery at 1- and 12-months post-injury (Dikman, Machamer, Winn, & Temkin, 1995). Dikman et al., studied all head injury severities prospectively, however, the proportion of the sample with mild head injuries showed no significant neuropsychological differences when compared to a non-concussed, trauma control group.

An earlier study, (Dikman, McLean, & Temkin, 1986), also revealed no neuropsychological differences in a sample of 19 MHIs tested at 1- and 12-months post injury. As well, no differences were found between a small sample of MHI with and without PCS at 22 months post-injury on 4 neuropsychological tests (Bohnen, Jolles, Twijnstra, & Marshall, 1995).

Hugenholtz et al. (1988) examined simple and choice reaction time, 5 times over a three month period. The MHI group performed significantly slower and made more errors than controls on the complex choice reaction time paradigm on the first 3 visits. By the three month test session, group differences were no longer noted. Practice effects were likely to play some role in this reaction time study, however, the authors noted that the MHI group showed greater improvements in performance than did the control group. Evaluating attention, memory and intelligence in a sample of 50 MHI patients 1-month post injury revealed no significant differences when compared to controls (Gentilini, Nichelli, Schoenhuber, Bortolotti, Tonelli, Falasca, & Merli, 1985). However, when this same group of researchers evaluated attention and information processing speed using complex reaction time tests, differences were found. Gentilini, Nichelli, and Schoenhuber (1989) noted significant differences between a sample of 48 MHI and matched control groups on a variety of computer generated reaction time tests measuring selective attention, sustained attention and divided attention at 1 and 3 month follow-ups. This study demonstrates the sensitivity of reaction time tests in the measuring of information processing speed and attention.

This review would suggest that, in those studies utilizing MHI samples with and without post concussive complaints, neuropsychological deficits are found initially, but recovery is typically observed within a 3 month follow-up period. Thus, as described at the

beginning, full recovery is the rule by 3 months post-injury. A notable exception to this trend is a seminal study by Barth et al., (1983), who followed 70 MHI patients from hospital discharge to 3 months post-injury. This study is often heralded as one of the more important studies demonstrating true organic deficits in mild head injury. Using the Halstead Impairment Rating, 22 subjects received a rating of .7 (moderate to severe brain injury), 22 evidenced impairment between .3 and .6 (mild brain injury) and 26 with impairment ratings below .3 (no brain injury). Sixty three percent of this sample demonstrated neuropsychological deficits. This is a rather high proportion of the MHI sample compared to the typical "ten percent". Examining the subject sample and demographics, it would appear that this particular sample of MHIs may have been somewhat unrepresentative. For example, 14 of the subjects had a previously documented head injury, and 97% of the sample had a *documented* loss of consciousness, with mean length of LOC at 11.5 minutes. Most mild head injury samples have minimal to no loss of consciousness, even though the criteria indicate a maximum of 20 minutes. It is also not clear from the paper what proportion of patients were in motor vehicle accidents compared to falls and sport related injuries, as MVA accidents may incur more whiplash effect and therefore more diffuse damage. Finally, this study has been criticized on the fact that the MHI group was compared to test cut-off scores, rather than to a comparison or control group.

The second style of neuropsychological studies examine only MHI patients who continue to have PPCS, in essence the "10%". Typically, the results find marked differences in neuropsychological functioning compared to controls. An interesting study divided the MHI group into those that suffered a brief loss of consciousness and those that only received

a dazing experience following the trauma. Although there were no difference between the MHI-concussed and MHI-dazed groups, the MHI total group performed significantly worse on 5 of 8 neuropsychological tests (e.g., Category test, PASAT, RAVLT, Rey Osteirith Complex Figure copy and recall tests) (Leininger, Kreutz, & Hill, 1991). Average time since injury was 8 months and these were patients who continued to report PCS.

Yarnell and Rossie (1988) conducted a "follow-back" study in which they examined 27 cases that had received a whiplash injury. Referrals followed a neurological evaluation and average time since injury was approximately 9 months. Of this file review, only 16 of the 27 had received a neuropsychological assessment. Half of this sub-sample had not yet returned to work and the other half were working at a reduced capacity. Neuropsychological testing revealed an average Halstead Impairment Rating of .4 (e.g., Mild Brain Injury Category). There was no comparison group, however the authors report that 88% of the sample of 16 were impaired on a complex motor flexibility test (e.g., fist-edge-palm), 86% were impaired on a vigilance task, 85% on working memory test (e.g., Serial 7's), 68% were impaired on the Halstead Category Test and 70% of the group were impaired on a auditory verbal list learning task.

Marsh and Smith (1995) also found clear neuropsychological deficits in a small sample of MHI who were complaining of PCS after 3 months. These studies suggest that when MHI samples are confined to only those subjects who are presenting with PPCS, neuropsychological deficits are found. It might be that when MHI samples include both the "typical" recovery individual as well as the PPCS individual, group differences are not found as the subset of the MHI with PPCS are masked by the larger group effect.

The idea that group effects can mask neuropsychological deficits in a subsample of MHI subjects is supported by studies that divide MHI subjects into subgroups. Perhaps one of the more comprehensive studies on attentional processes in mild head injury was reported by Gronwall (1989) using the Paced Auditory Serial Addition Test (PASAT). The PASAT is a complex task of information processing capacity in which the individual is given a series of numbers from 1 to 9 and is required to add the first number to the second, then the second to the third, then the third to the fourth, etc. Gronwall studied 237 subjects with mild head injury and has generated recovery curves for this sample, based on PASAT scores. Fifty-three percent of the sample had moderately impaired scores on the PASAT, taking approximately 4 to 6 weeks to obtain normal scores. Another 25% scored better than this later group and obtained normal scores within 2 weeks of the initial injury. The remaining 20% showed more severe impairment initially and impairment persisted for a longer period (Gronwall, 1986). The Gronwall studies excel in that there is a wide sampling of mild head injuries, and all followed from initial injury to at least 2 months post injury. As well, the range of recovery curves would appear to model the statistics given earlier, with 20% of a MHI sample being more substantially impaired and not reaching a full recovery within 3 months.

Williams, Levin, and Eisenberg, (1990), compared MHI patients with complications (e.g., focal lesion on imaging scans, skull fracture, etc.), to MHI patients without complications and to moderate TBI patients. The total sample size was relatively large (n=215) allowing for a fairly representative sample. The results indicated that the MHI sample with complications performed more similarly to the moderate TBI group than to the

MHI sample without complications. Deficits were found in the MHI with complications and the moderate TBI groups on the PASAT, recognition memory, word fluency and the Glasgow Outcome Scale.

Dividing a mild head injury sample (n=53) into those with and without soft neurological signs revealed that those with neurological soft signs showed deficits on psychomotor and spatial organization tests at 9 months post-injury compared to the MHIs without neurological signs (Cattelani, Gugliotta, Maravita, & Mazzucchi, 1996).

Interestingly, there were no differences found on a variety of tests of attention and memory.

Another example of where MHI samples tend to demonstrate neuropsychological deficits is in subject samples when over 90% of the head injuries are caused by MVAs (Leininger et al., 1990; Parker & Rosenblum, 1996; Yarnell & Rossie, 1988). In a typical sample, the cause of the head injuries varies, such as sporting injuries, MVAs, falls, bicycle accidents, assault, etc. It would appear that MVA brain injuries are slightly more debilitating, presumably due to the acceleration-deceleration forces compared to other causes, such as impact wounds, sport injuries and falls. Acceleration-deceleration forces causes stretching and shearing injuries in many areas of the brain; whereas, light impact wounds or falls are more likely to result in more focal damage.

Taken altogether, the neuropsychological data provides clear evidence that neuropsychological deficits can be documented immediately following a mild head injury. Improvements are typically seen over time, with the majority of the sample performing as well as non-TBI controls. The most consistent finding is, deficiencies in speed of processing, as well as attention and working memory abilities. When studies investigate only those

individuals complaining of persistent PCS, neuropsychological deficits are usually found when compared to non-TBI controls. As well, studies that examine their MHI sample based on neurological complications before testing, by rate of recovery during testing, or style of injury, tend to find deficits in a 15-20% subsample of the MHI group. When group differences are not found in testing, then the majority of good recoverers may be masking the problem areas of the subsample of MHIs that have persistent neuropsychological difficulties.

Both the neuroimaging and neuropsychological literature seem to indicate that recovery of function occurs within the first 3 months after injury, for the majority of individuals who sustain a MHI. In addition, these studies have also identified a subsample within large MHI groups who have clear neurological and/or neuropsychological deficits that persist past the 3 month mark.

It is curious that many literature reviews in this area support the prevailing belief that there is minimal physiological evidence to support the persistence of post concussive symptoms after 3 months. For example, a review paper by Binder (1986) concluded that MHI can cause persisting brain damage in a small percentage of individuals. This paper was probably one of the primary efforts in supporting the legitimacy of PPCS in ten percent of MHI sufferers. Yet, in his most recent articles (Binder, 1997; Binder, Rohling, & Larrabee, 1997), based on a meta-analytic review of 8 research studies, the authors concluded that the average effect of MHI on neuropsychological performance is undetectable. When one looks more closely at the studies included in the meta-analysis, it is not surprising that persisting deficits on neuropsychological tests were not found. For example, the definition of MHI used across the 8 studies varied considerably, and, studies of whiplash and/or cervical strain

were purposely not included. In addition, two of the studies examined MHI secondarily in samples of subjects who were alcoholics or HIV positive, and the sample sizes across the 8 studies ranged from 6 to 161. Most importantly however, Binder et al., (1997) only included studies that followed MHI groups prospectively. Clinical studies that investigated PPCS retrospectively (e.g., people complaining of post concussive symptoms following a MHI) were excluded. As was demonstrated in the present review, studies that follow MHI individuals from emergency room to whenever the follow-up time is, tend not to find group deficits on neuropsychological performance, because only a small percentage of this group would actually have persistent deficits. The ten percent of MHI samples that have persistent deficits are masked in the process of group means. Thus, it is not surprising that the Binder et al., (1997) article failed to find neuropsychological deficits in his analysis.

Binder dismisses the use of clinical studies that use samples who report persistence of symptoms following a MHI. He argues that symptomatic patients may differ in many ways from asymptomatic patients, and that although clinical studies are interesting, "their methodology cannot be employed to determine the *extent and frequency of neuropsychological deficits* in MHT" (pg. 422). This is an unusual argument, given that research typically demonstrates that only 10 percent of MHI that will report ongoing neuropsychological sequelae. The debate in the literature is whether the persistence of symptoms is organic or psychogenic in nature. By only using studies that include a representative sample of MHI, (e.g., with 90% who recover fully and 10% who do not), it would appear that this paper has simply re-established what has been understood: MHI, in general, exacts minimal neurological sequelae. The Binder et al., (1997) article did not

address the ten percent subgroup of MHI in any of the analyses. Again it must be stated that the area of debate surrounds the 10 percent who report ongoing post-concussive symptoms--not the 90% who recover. Certainly, when studies use large samples and use group averages, often the MHI group perform similarly to non-TBI controls, supporting the idea of "no evidence".

It has been demonstrated in the present review however, that when studies examine their MHI samples more closely, typically a small percentage are shown to have neurological complications and/or neuropsychological deficits consistent with their subjective complaints. As well, in studies that only investigate PPCS, or, the "ten percent", neuropsychological deficits are noted. Although a head trauma can be classified as "mild" based on LOC and GCS, mild head trauma with PPCS may fall outside the definition and expectations of mild TBI. Perhaps this "10 percent" are *physically* moderate brain injuries.

### ***The Psychogenesis of Persistent Post Concussive Syndrome***

Perhaps one of the most puzzling aspects of mild head injury is that the reported symptom complaints of the individual do not seem to "fit" the nature of the trauma. Despite the physical evidence just given to support the complaints made by individuals with PPCS, there has been considerable investigation regarding the psychological determinants of persistent sequelae following MHI.

Mild head injuries can be caused by a variety of events, such as a fall of a roof, being rear-ended while parked at a stoplight, or being concussed on the football field. The apparent trauma to the brain in such instances, would appear minor and unlikely to be

debilitating. The discrepancy between a seemingly minor head injury and the patient's complaints often lead clinicians to attribute the discrepancy to the patient, and his or her need for secondary gain and to explore psychosocial and emotional variables, in the attempt to find some justification for the client's complaints. The resulting body of literature is generally scattered, contradictory and atheoretical. A variety of possibilities have been examined, but few have been systematically and methodologically investigated and replication of findings is scant. In essence, the riddle of the PPCS is reflected in the research: inconsistency of results and uncertainty regarding relevant factors. Notwithstanding this state of affairs, some myths have been broken, some trends have been uncovered, and other lines of investigation show promise but need more systematic study.

### Compensation Neurosis

A commonly held notion to account for PPCS is what has been termed "compensation neurosis" or "accident neurosis". Compensation neurosis, in short, implies that the primary mechanism for the persistence of post-concussive symptoms is the need for individuals to seek monetary compensation for damages incurred (Levy, 1992). This view assumes that symptoms are consciously maintained by the injured party exclusively for the duration of the litigation process. Once a monetary settlement has been granted, it is believed that the symptoms will disappear (see, Levy, 1992, for review). This longstanding and still prevalent belief is in large part, a result of a seminal paper by Miller in 1961, based on his personal experience with medico-legal assessments as a neurologist. His clinical experience and the results of his paper are based on a sample comprised solely of head injuries pursuing litigation. Based on this extremely biased sample, he made several sweeping negative

generalizations about this population and concluded that the outcome was favorable only after compensation was granted.

Miller's methodology and unfounded conclusions have been duly criticized (see, Levy, 1992; Tarsh & Royston, 1985). Current research with mild head injuries appears to have debunked Miller's opinions. The presence of post concussive symptoms are no more existent in people with MHI pursuing or receiving compensation than those who have never pursued litigation (Fenton et al., 1992; Gfeller, Chibnall, & Duckro, 1994; Hugenholtz, Stuss, Stethem & Richard, 1988; Leininger, Gramling, Farrell, Kreutzer & Peck, 1990; Mendelson, 1982; Rutherford, Merrett & McDonald, 1978; Tarsh & Royston, 1985). The process of seeking compensation may substantially increase stress levels by increasing anxiety and frustration with the entourage of medical appointments and court proceedings; however, it seems that this has no bearing on whether the individual maintains his symptoms for court or whether settlement induces recovery.

### Psychosocial Variables

Other variables of interest have been age and gender. Early studies reported that women were more likely than men to develop PPCS, although men were more likely to sustain a head injury (i.e., Dikmen, Temkin & Armsden, 1985; Lishman, 1988; Rutherford, 1978). More recent studies have not confirmed this finding (Fenton et al., 1993; Mittenberg, Digiulio, Perrin & Bass, 1992). The relationship with age is less clear. It is legitimate to consider that the older brain (i.e., >50 years) would be more susceptible to injury following subtle trauma and this relationship has been confirmed in many studies (Dikmen, Temkin & Armsden, 1989; Fenton et al., 1993; Radanov, Stefano, Schnidrig & Ballinari, 1991;

Williams, Levin & Eisenberg. 1990). However, several other studies have not found this relationship to be true for their MHI samples (Alves, Macciocchi & Barth, 1993; Hugenholtz et al., 1988; Mittenberg et al., 1992; Rutherford, Merrett & McDonald, 1978). It is uncertain why the relationship of PPCS with age is so inconsistent, although it is likely a function of the particular subject samples in individual studies (e.g., predominantly young or old subjects, types of injury included in sample, etc).

Investigations involving other psychosocial variables have found that persons with PPCS were more likely to report higher symptom rates, report more sick leave from work, and have higher rates of health concerns compared to MHI persons without post concussive symptoms (Middlebow, Anderson, Birket-Smith & Friis, 1992). This statement however, seems somewhat circular, in that, if persons with PPCS have received more neurological damage, then naturally they would have more complaints and be less likely to have returned to work than those who had a full recovery.

Another study examined premorbid histories and found that persons with a mild head injury obtained in a MVA were twice as likely to have had significant life events, such as death, divorce, marriage, illness, in the year before the accident (Fenton et al., 1993). Premorbid burdens and pressure may make the ability to cope with even a mild head injury very difficult, possibly leading to slower recovery. Investigations into coping style or persons recovering from a MHI revealed that a poorer recovery was associated with avoidance, emotion focused thinking and wishful thinking (Malia, Powell, & Torode, 1995). In contrast, studies exploring other factors such as, dysfunctional families, child abuse, rape, or social interaction history failed to reveal any significant relationships to differentiate the

MHIs from the controls (Fenton et al., 1993; Radanov et al., 1991). Another study also revealed no differences between concussed and non-concussed controls in reference to drug use, premorbid psychological complaints, premorbid activity levels or premorbid symptom discomfort (Robertson, Rath, Fournet, Zehart, & Estes, 1994).

Explanations regarding who will develop PPCS are certainly inconclusive. Age and gender trends are inconsistent with as many studies reporting a relationship as there are studies reporting no relationship. Again, the exact nature of the MHI sample may delineate some of the inconsistencies. Mild head injury is too broad a category to be lumped under "less than 20 minutes loss of consciousness". Information regarding extent of whiplash, neurological complications, proportion of ages rather than average age need to be part of the sample description. There also does not appear to be any predominant premorbid psychosocial variables which characterize MHI groups with post concussive symptoms from those without. The one thing that is relatively certain, is that PPCS is not an artifact of the litigation process.

#### *Base Rates of Post Concussive Symptoms*

Another line of investigation has looked at the degree to which post concussive symptoms are truly unique to head injury. Persons with uncomplicated MHI are more likely than non-concussed controls to complain of post concussive symptoms, compared to emotional vegetative symptoms when given a checklist questionnaire that measures both areas (Bohnen, Twijnstra & Jolles, 1992). It has also been demonstrated that post concussive symptoms are found in the general population with some frequency, but are not as high as those suffering from a traumatic brain injury (Alves, Macciocchi & Barth, 1993).

An interesting study compared MHI and normal controls on a symptom checklist, in which subjects were required to complete a post concussive symptom checklist, twice. Subjects in the MHI group completed the questionnaire as it reflected their current symptoms and as they thought they were *before* the head injury. Control subjects answered the questionnaire as it reflected their current status and then how they think they *would be* if they had a head injury (Alves et al., 1992). The results showed that the general population has a rather accurate understanding of the effects of head injury without having had a head injury (i.e., they identified the Post Concussive Syndrome). Moreover, head injured persons were apt to significantly underestimate the degree to which many of their symptoms occurred before the accident. For example, MHI subjects rated several symptoms (i.e., the number of headaches, the number of times they forgot where they parked their car, etc.) as occurring much less frequently before the accident compared to after the accident, and much less often than normal controls reported these "symptoms". This study suggests that some degree of reporting of post concussive symptoms in mild head injured samples may not represent true "changes" from premorbid functioning. This data suggests that individuals with a MHI tend to attribute many instances of physical, cognitive and emotional symptoms to the head injury without appreciating that some symptoms are common occurrences and tend to occur anyway, regardless of whether they sustained a head injury or not.

#### *Psychological Factors: General Emotional Functioning*

Another area of research has attempted to investigate emotional and/or psychological functioning in groups of individuals with PPCS. Except for the literature using the

Minnesota Multiphasic Personality Inventory (MMPI), the majority of studies are not easily comparable because they use different tests. A study in Switzerland investigated whether the "neuroticism" scale of a German emotional functioning inventory could differentiate groups of people with MHI, with and without post concussive symptoms at a 6-month follow-up (Radanov et al., 1991). Both MHI groups revealed normal profiles on this German personality test. A study in Belfast used a series of questionnaires to measure various psychosocial variables that may affect outcome from MHI and reported that no significant differences between MHI and control groups on premorbid social difficulties, premorbid psychopathology, or current social interactions (Fenton et al., 1993). An Italian study compared a mild head injury sample with a non-head injured control group on the State-Trait Anxiety Inventory (Spielberger, Gorsuch & Lushene, 1970) and the Self Rating Depression Scale (Zung, 1965) at 9 months post-injury (Schoenhuber and Gentilini, 1988). Schoenhuber and Gentilini (1988), reported that the groups differed only on the self rating depression scale, with the MHI group reporting significantly more depressive symptoms compared to controls. No differences were found between groups on anxiety indices, whether state or trait.

These foreign studies do not allow for firm conclusions regarding the relationship between a variety of psychological variables and MHI, although support for higher depression rates in MHI groups has been found in many North American studies. Differences in culture and socialization may mask and/or enhance some areas of emotional adjustment to the head injury, depending on where the study is conducted. In addition, the measurement tools used in these studies makes it difficult to generalize to North American settings. One

psychological questionnaire that has been routinely studied in head injured populations is the Minnesota Multiphasic Personality Inventory (MMPI).

*Psychological Factors: The MMPI*

The MMPI and its revision, the MMPI-2 are the most widely used personality inventories for neuropsychological populations (Lees-Haley, Smith, Williams & Dunn, 1996; Wooten, 1983). This test consists of 10 main clinical scales and 3 validity and/or test-taking style scales, as well as a mirage of supplementary and content scales that are too numerous and detailed to review here. The MMPI-2 can be considered a measure of personality functioning, as well as an indicator for situational stress and psychological/emotional turmoil. It is not necessarily a test for personality *disorders*. Table 1 outlines the 10 clinical scales and the main interpretive descriptors of each.

Within neuropsychological populations, and traumatically brain injured populations in particular, five of the MMPI clinical scales are consistently elevated, Scales 1 (Hypochondriasis), 2 (Depression), 3 (Hysteria), 7 (Psychasthenia), and 8 (Schizophrenia) (Alfano, Finlayson, Stearns & Neilson, 1990; Bornstein, Miller & VanSchoor, 1988; Leininger, Kreutzer & Hill, 1991; Wooten, 1983). Simply interpreted, this typical profile describes individuals who endorse a mirage of somatic complaints, report perceived changes in thinking, as well as elevated levels of anxiety and mild depression.

There does not appear to be a positive relationship between severity of brain injury and severity of emotional functioning (e.g., Wooten, 1983), even through one might think that the more severe the brain damage, the more emotional adjustment problems the TBI

**Table 1: Scale Descriptors of MMPI-2**

Scale	Label	Interpretive Description
L	Lie (Defensiveness)	fake good, defensive, denying, little insight into their own motivations, over evaluate own worth, conventional and socially conforming, rigid and moralistic
F	F (Frequency)	fake bad, exaggerating symptoms and problems as a plea for help, may be resistant to test taking procedure, may manifest true neurotic or psychotic problems, random or all true responses
K	K (Subtle Defensiveness)	fake good, all false responding, making appearance of adequacy, control & effectiveness, shy and inhibited, lack self-insight and understanding, in absence of psychopathology then a measure of ego-strength
1 (Hs)	Hypochondriasis	excessive bodily concern, vague somatic complaints, fatigue, weakness, selfish, narcissistic, whiny, demanding, critical, unhappy & dissatisfied.
2 (D)	Depression	dysphoric, pessimistic of future, vegetative signs, feel useless, lack self-confidence, aloof, introverted, shy
3 (Hy)	Hysteria	react to stress via physical symptoms, headaches, chest pain, do not report severe emotional turmoil, lack in-sight to problems, need attention & affection from others
4 (Pd)	Psychopathic Deviate	difficulty with societal norms, rebellious toward authority, stormy family relationships, impulsive and strive for immediate gratification, insensitive to others
5 (Mf)	Masculinity/ Femininity	may be experiencing sexual identity problems, if male then stereotyped masculine behaviours and interests, if female then rejecting traditional female role, assertive
6 (Pa)	Paranoia	paranoid predisposition, excessively sensitive and overly responsive to needs of others, suspicious, guarded, hostility, resentment, argumentative, opinionated, moralistic
7 (Pt)	Psychasthenia	experiencing psychological turmoil, feel anxious, tense, agitated, worried, apprehensive, problems concentrating, often anxiety disorders, introspective, obsessive thinking
8 (Sc)	Schizophrenia	may have psychotic disorder, confused, disorganized, disoriented, poor judgement, may be in acute psychological turmoil, tend to lead schizoid lifestyle, alienated, aloof
9 (Ma)	Hypomania	unrealistic self-appraisal, overactive, energetic, talkative, bored easily, restless, low frustration tolerance, if T > 80, may be frank manic episode
10 (Si)	Social Introversion	socially introverted, insecure and uncomfortable in social situations, shy, reserved, timid, few social activities, prefers company of few close friends, described as distant

Note. Summarized in table form from MMPI-2: Assessing Personality and Psychopathology (pp.53-85) by J. R. Graham, 1990, New York:Oxford University Press.

person will experience. In fact, one study found that people with mild head injuries have more emotional distress compared to a group of severe head injuries on the MMPI (Leininger, Kreutzer & Hill, 1991). In particular, Leininger et al., (1991) found that the scores of the mild head injury group were statistically higher than the severe TBIs on MMPI

Scales 1, 3 and 7, indicating that milder injuries endorse more somatic concern, preoccupation with physical symptoms, substantial anxiety and difficulties with concentration. Some might conclude that the MMPI profiles of MHIs are, like post concussive complaints, in that the impact of the head injury on functioning far exceeds what would be expected given the mild trauma. Alternatively, persons with mild head injuries are very aware of the changes they have experienced due to the brain injury and can become quite overwhelmed with the struggle to return to the person they were. Severe TBI patients often suffer from a lack of insight and awareness, therefore their injuries are less noticeable to them, nor as subjectively debilitating.

Bornstein et al., (1988) investigated whether there were particular subgroups of "MMPI profiles" within a head injured population. The TBI group was comprised of various severities of head injury, with over half of the sample classified as mild. Using a cluster analysis, they uncovered 4 subgroups of MMPI profiles: 1) normal profiles (20% of sample); 2) Scales 1, 2, 3 elevated only (35% of sample); 3) the typical TBI profile with scales 1, 2, 3, 7, and 8, elevated (40% of the sample); and 4) profiles in which almost all scales were significantly elevated (i.e., a "cry for help" profile; (7% of sample). Clinicians would likely agree that the Bornstein et al., (1988) cluster analysis is a fair representation of the most common emotional responses that head injured clients present with at a clinic: the ones who *are* coping well with their head injuries, those that are coping fairly well with some focus on physical problems and somatization, those that are having some difficulties coping with their head injuries with considerable focus on physical somatic complaints, anxiety, depression and thinking problems and lastly, those that are not coping at all.

One must keep in mind that the items of the MMPI were included to discriminate various neurotic and psychiatric disorders from "normals". A common mechanism found in many psychological disorders is to convert stress and emotional pain into physical maladies. It follows then that several questions on the MMPI measure stress-related physical and cognitive symptoms as part of the descriptive picture of various emotional and psychological disorders. Several of the *physical* symptoms covered by the MMPI are very similar to neurologically based post concussive symptoms. This has led some researchers to correct for the overlap of neurological content in MMPI questions.

One study uncovered 44 MMPI items that, when literally interpreted, represented true neurological symptoms (Alfano, Finlayson, Stearns & Neilson, 1990). Alfano et al. (1990), rescored the MMPIs of a general neuropsychology sample by simply deleting the 44 neurologically related items (NRIs). Due to item overlap among Clinical Scales, this resulted in 89 scoreable points being removed from the overall profile configuration, which in turn calls into question the clinical validity of these neurologically corrected profiles (i.e., a profile is considered unscorable if between 10 and 30 items are left unanswered (Butcher, Dahlstrom, Graham, Tellegen & Kaemmer, 1989; Graham, 1990). Despite the methodological problems, the neurological correction led to interesting results. Specifically, almost a third of the sample revealed a normal profile and another third had considerably different profiles from the originally scored profile. The authors concluded that elevated MMPI profiles within neuropsychological populations may be artificially inflated due to the endorsement of items reflecting actual neurological symptoms as opposed to symptom profile of a psychological disorder.

Gass and Russell (1991) identified 42 neurologically related items on the MMPI and rescored the subjects original MMPIs by prorating the NRIs based on the number of non-NRI items endorsed for a particular scale. This procedure allows for consideration of a proportion of NRI item endorsement to reflect some emotional concerns. A simple frequency analysis of NRIs per scale revealed that almost 60% of the questions that load on Scale 1 (Hypochondriasis) were ranked as NRIs. The other "neuropsychology scales" (i.e., 2, 3, 7, 8) contained between 20 to 30 percent NRIs. The remaining scales contained less than 10% NRIs. Comparing the original profiles to the corrected profiles uncovered significant differences with average T-score point decreases of 19 on Scale 1, 11 on Scale 8, 7 on Scale 2 and 6 T-score points on scales 3 and 7. Conventional scoring and interpretation would have found significant neurotic and psychotic pathology in over 83% of the sample; whereas the corrected profiles revealed psychopathology in 64% of the sample--a notable difference. Appreciation of the neurological content of MMPI items must be made when interpreting profiles within a neurological/neuropsychological population.

Gass (1991b) applied a seemingly more clinically and statistically valid correction factor using the revised MMPI-2. He compared item endorsement of a large sample of head injured patients with those of the MMPI-2 normative sample. Test items qualified as an NRI if it had both discriminative power in separating the TBI from the normal controls, and a high frequency endorsement by TBI responders. The items identified would therefore have maximum discriminative power statistically, as well as being highly relevant in a clinical sense. The analysis identified 14 critical neurologically related items, accounting for more than 24% of the variance between the 2 groups. These items reflect neurological symptoms

such as headache phenomenon, concentration and memory changes, fatigue, sleep related difficulties, weakness and numbness. These items are very characteristic of post concussive symptoms. In fact, if all 14 items were endorsed, which is fairly probable in many cases, the average associated incremental impact on T-scores would be as high as 13 points on Hs, 12 points on D, 10 points on Hy, 12 points on Pt, and 20 points on Sc (Gass, 1991b).

Given the extent of MMPI items that can be interpreted as post-concussive symptomatology, both researchers and clinicians must be cautious when interpreting the profiles in neuropsychological populations. Optimally, profiles should be scored twice, once in the traditional sense and secondly by incorporating a neurological corrections factor (Gass, 1991b).

It can be seen that individuals with traumatic brain injuries report a considerable amount of stress and emotional problems in their responses on the MMPI, even when neurological factors are controlled. The literature and clinical experience with the MMPI and traumatic brain injury, highlights the emotional turmoil many patients have in adjusting to their head injury.

#### *Psychological Factors: Functional Outcome with the Sickness Impact Profile*

Another popular measurement tool in evaluating a person's psychological adjustment to a head injury is the Sickness Impact Profile (SIP: Bergner, Bobbitt, Carter & Gibson, 1981). The SIP is a 136-item questionnaire that assesses various aspects of how the patient perceives that health related issues have impacted on their life. This questionnaire is more

functionally oriented, compared to the MMPI, tapping both physical and psychosocial areas, and is recommended as an ecologically valid tool (Judd & Fordyce, 1996; Lezak, 1995).

Studies using the SIP with brain injured populations have reported that the Psychosocial Scale, and its subscales, are sensitive to patients' quality of life judgements and are more frequently elevated as problematic areas compared to scales measuring physical maladies (Dikman et al., 1995; Klonoff, Costa & Snow, 1986; Klonoff, Snow & Costa, 1986). This may appear to be in contrast to the MMPI, where endorsement of physical complaints elevates and exaggerates psychological functioning scales. However, the Physical Scale on the SIP appears to tap more broad-based physical areas, such as ambulation, mobility, and body care and movement, as opposed to the more post-concussive-like symptoms found on the MMPI.

Although the SIP was created for health issues in general, there is evidence to suggest its clinical utility within neuropsychological populations. The SIP represents the functional assessment of daily life activities and some scales have been shown to correlate well with neuropsychological functioning (Klonoff, Costa & Snow, 1986; McSweeney, Grant, Heaton, Prigatano & Adams, 1985).

McSweeney et al. (1985) applied canonical correlations between neuropsychological measures of the Halstead-Reitan Neuropsychological Battery (HRNB; Reitan & Wolfson, 1993) and the SIP and identified 2 significant correlations: 1) between poorer performance on psychomotor and motor tests (e.g., Trails B, grip strength, grooved pegboard) and endorsed problems in the areas of Body care, Home Management, Mobility, and Socialization scales, and 2) higher number of errors on the Aphasia Screening test with an elevated

Communications scale. Klonoff, Costa & Snow (1986) also used canonical correlation and provide preliminary evidence suggesting that neuropsychological difficulties in memory and constructional abilities were related to a high degree of reported problems in Psychosocial Functioning (overall scale).

These studies clearly provide support for the functional, or ecologically valid aspect of some neuropsychological tests. In addition, the SIP appears to be particularly relevant to neuropsychological populations and provides the researcher and clinician with a more functional assessment of how the head injury has impacted on the examinee's lives.

Dikman et al, (1995) separated their sample of TBI subjects into mild, moderate, and severe subgroups. Head injury subjects as a group reported significantly higher psychosocial dysfunction compared to normal control and trauma control groups (e.g., significant medical trauma without head injury). The mild head injury group, specifically, were less likely to be employed and more likely to have lower income and greater limitations of psychosocial functioning compared to the normal control group. However, the extent of the psychosocial limitations with the MHI group did not differ from the 'trauma' control group. The authors suggest that non-TBI related factors were contributing to the subjects' post-concussive symptoms, such as a general difficulty coping and adapting to the traumatic event, as opposed to the TBI sequelae per se.

Data from MMPI and SIP studies provide clear evidence of the emotional impact of a traumatic brain injury. The few studies that analyze MHI groups separately, also suggest that these measurement tools are sensitive to persons with a milder TBI, although more research is needed in this area. Not all persons who suffer a head injury, however, are overwhelmed

and distraught, many cope very well with their cognitive deficits and do not allow the traumatic event to "take over" daily functioning. One differentiating factor between those who do and do not cope well following a TBI could be personality style or the presence of a personality disorder .

### Personality Disorders

There are very few studies that have investigated the possibility of "true" personality disorders within head injured populations. Middlebow, Anderson et al. (1992) investigated particular personality traits in post concussive patients using the Millon Behavioral Health Inventory (MHBI) (Millon, Green & Meagher, 1982), with a special emphasis on coping styles. The MHBI is a 150-item self report inventory intended to assess the patient's style of relating to health care personnel and treatment plans, as well as major psychosocial stressors. They found that two personality styles were predominant in their patients with significant post concussive symptoms. The first was a Forceful Personality Style, describing domineering, aggressive, impatient and easily angered individuals who may not follow treatment regimens and who are distrustful. The Sensitive Personality Style was also identified, describing patients as unpredictable, negativistic with passive-aggressive traits including guilt-ridden, moodiness, and as complaining and anticipating disappointments. In contrast, the Cooperative Personality Style was a protective factor in developing PPCS, characterizing patients as compliant, dependent and eager to take and follow advice, although they may lack initiative and tend to deny their problems. Middlebow, Anderson et al. (1992) conclude that certain personality factors predispose these individuals for developing PPCS and that the immediate organic and emotional aspects of the MHI may serve as only a precipitator for the

development of a more comprehensive morbidity than would be expected when considering the relative low impact forces of the head injury.

One study examined both Axis I and Axis II disorders in a small TBI sample of all severities, using the structured psychiatric interview of the DSM-3-R (VanReekum, Bolago, Finlayson, Garner, & Links, 1996). Seven of the eighteen TBI subjects received at least one DSM-3-R personality disorder. In fact, these 7 subjects received a sum total of 22 personality disorders. The most commonly identified personality disorders were Borderline Personality Disorder (4 out of 7 subjects), and Avoidant Personality Disorder (5 out of 7). They also report that no subject received a diagnosis of paranoid, schizotypal, or self-defeating personality disorder. Although only a small sample of 18, as much as 39% were diagnosed with at least one personality disorder by DSM-3-R criteria.

Streeter, VanReekum, Shorr, & Bachman (1995), investigated the relationship between TBI and Borderline Personality Disorder (BPD) in a sample of male veterans. Forty-two percent of the BPD sample had a prior TBI, whereas only 4% of the psychiatric controls had a history of TBI. Using a questionnaire that identifies BPD retrospectively, the chart reviews of the Borderline group revealed that the diagnosis of the personality disorder occurred *after* the traumatic brain injury. The authors conclude that the TBI contributes to the development and/or exacerbation of borderline personality characteristics, rather than persons with BPD being at higher risk for acquiring a TBI.

The Millon Clinical Multiaxial Inventory is a commonly used test for measuring personality disorders in many clinical settings, although not commonly used within neuropsychological populations. Only one study using the MCMI in a head injured

population would appear to have been published (see Table 2 for a description of the scales of the MCMI-II). Snibbe, Peterson and Sosner (1980) examined the MMPI and MCMI profiles of Workers' Compensation claimants among 4 subgroups: head injury, psychiatric stress and strain, low back pain, and miscellaneous. In the head injury group, the most frequently elevated personality disorder was Submissive-Dependent, more recently labelled Dependent Personality on the MCMI-II. As the main emphasis of this study was to compare and contrast the 4 claimant groups, there was minimal information pertaining to specific post concussive symptoms, severity of head injury or a more indepth analysis of the personality styles within the TBI group. Nonetheless, the authors concluded that the MCMI is useful in identifying premorbid long-standing personality styles as opposed to more transient symptom states such as what is measured on the MMPI.

In summary, apart from the lack of relationship between post concussive symptoms and litigation, few demographic or developmental variables have been uncovered to describe the "typical" PPCS patient. Studies examining psychological/emotional functioning describe these individuals as overly concerned with somatic symptoms, having attention-concentration difficulties, reporting unusual sensory experiences, along with mild depression and anxiety. This description of emotional functioning could just as easily describe a person with a seizure disorder. Investigation of defined personality disorders are too few to warrant any general statements, although symptoms consistent with Borderline personality disorder appear to have some relationship with traumatic brain injury.

In contrast, there *is* considerable consistency across these studies in identifying, in general, the emotional and/or psychological consequences following a mild brain injury.

Table 2: Scale Descriptors of the MCMI-II Personality Scales

CLINICAL PERSONALITY PATTERNS		
Scale	Label	Interpretive Description
1	Schizoid	lack of close relationships, lack of interest or ability in expressing emotions, private and prefer to be alone, detachment is comfortable for them, not insightful, lead unemotional lives, etc.
2	Avoidant	predominant avoidant & dependant traits, hypersensitive to the possibility of rejection, concerned about risk of interpersonal relation, ill at ease socially, nervous, uncomfortable, can be compassionate, etc.
3	Dependent	believe they are unable to take care of selves, need others to protect and support them, feel inadequate or insecure, see themselves as less effective or capable than everyone else, followers, submissive, etc.
4	Histrionic	dramatic, colourful, emotional, seek stimulation, quickly "react" in the situations around them--involvement does not last though, make good 1st impressions, demanding and controlling, etc.
5	Narcissistic	believe they are special & superior to others, exaggerate their ability, rationalize to inflate own worth, depreciate others, make good 1st impressions, proud & dignified, must feel recognized and do not like compromise, etc.
6A	Antisocial	competitive & feel they must fend for themselves, mistrustful & suspicious, see selves as strong, assertive & self-reliant, contemptuous of the weak, impulsive, if crossed--may respond with anger and vindictiveness, etc.
6B	Aggressive/ Sadistic	a measure of antagonism, a more pathological variant of Antisocial style, abusive, forceful, commanding, militant, intimidating, touchy, excitable, irritable, react with anger when confronted or when autonomy threatened, etc.
7	Compulsive	disciplined, believe in hard work, orderly, big planners, conscientious, efficient, dependable, overly ingratiating and respectful to authority figures, perfectionistic, inflexible, overcontrolled emotions, etc.
8A	Passive- Aggressive	conflictual, they believe they need people to help them--yet they also feel they cannot afford to depend on others, thus compliant but not giving full support, moody, irritable & pessimistic, angry & stubborn to guilty & contrite, sulk, etc.
8B	Self-Defeating	self-sacrificing, martyr-like, allow others to take advantage of them, demigrate selves into believing they deserve their fate, pattern repeated in relationships--thus prone to being abused, somewhat masochistic, etc.
SEVERE PERSONALITY PATHOLOGY		
S	Schizotypal	more severe variant of schizoid or avoidant, behavioural peculiarities & eccentricities, detached from world, tend to lead meaningless, drifting lives, emotionally bland, socially detached, pervasive discomfort with others, cognitive confusion and perceptual distortions very common, self-absorbed & ruminative, etc.
C	Borderline	conflicting & ambivalent feelings, from inconsolable need to deep resentment, attachment disorders, labile & intense emotions, frequent mood swings, strong fears of insecurity and abandonment, lack a sense of own identity, often have punishing conscience and prone to acts of self-mutilation, etc.
P	Paranoid	vigilantly distrust others, have an abrasive, hostile, irritable & irascible demeanour, readily attack & humiliate anyone who is perceived as controlling them, distort events to support suspicions, rigid & argumentative, exasperate others, etc.

Note: Summarized in table format from Interpretive Guide to the MCMI (pp. 74-114) by J.P. Choca, L.A. Shanley & E. VanDenburg, 1992. Washington, DC: American Psychological Association

Regardless of whether the origin and maintenance is organic in nature or not, there is a real emotional phenomenon occurring following a MHI that needs to be addressed.

### ***A Paradigm Shift***

So far, the emphasis in the investigation of PPCS is to uncover or discover why this 10 to 15% do not maintain the typical recovery from a mild traumatic brain injury. Given the fragile nature of brain tissue, could we not restate the question into, why is it that 85% actually do recover favourably? Dr. Nils Varney and his colleagues are strong advocates for the neurological validity of persistent post-concussive symptoms. Instead of viewing MHI as a condition that has a low probability of lasting sequelae, consider that the 10% with lasting sequelae translates into approximately 160 000 people a year who seek medical and neuropsychological care (U.S. estimates).

Varney argues strongly for the legitimacy of the symptoms reported in the 10% of mild TBI sufferers who fail to recover as completely as those who suffered a mild TBI and *do not* report post concussive symptoms. Varney's discussions highlight two areas in describing "the 10%"; first, the misrepresentation of post concussive symptomatology and depression following a MHI as part of a seizure-spectrum disorder, and second, the lack of investigation into prefrontal amotivational symptoms. The former area is most relevant to the current proposal.

Post concussive symptoms overlap considerably with symptoms of partial-complex seizures, for example: anesthetics, headache, photophobia, nausea, abrupt mood shifts, memory gaps, irritability, confusion, occasional speech problems, abdominal sensations,

anxiety, intrusive thoughts and depression (cf. Varney & Shepard, 1991). Perhaps the persistence of symptoms, and/or the patients' report of deterioration of symptoms over time, may in fact be some form of seizure phenomenon. A large percentage of persons with depression and a history of traumatic brain injury, who had been treatment resistant to typical anti-depressant medications, were no longer reporting depressive symptoms and post-concussive phenomenon when placed on a trial of anti-convulsant medication (Roberts, Paulsen, Marchman, & Varney, 1988).

It is well known that a considerable proportion of persons who suffer a closed head injury become depressed at some point following the injury. Many individuals do not even report onset of depressive symptoms until at least 6 months following the head injury (Varney, Marzke, & Roberts, 1987). Although one could argue that the adjustment to the changes experienced following the TBI is what brings on the depression, one might also consider the possibility of a seizure disorder. A whiplash injury causes stretching and shearing of white matter, tiny multi-focal haemorrhages and air bubbles. It is reasonable to infer that these small lesions create scar tissue which may make the area vulnerable to the development of multi-focal seizure activity. Thus, a seizure-spectrum disorder may be masked as a psychological disorder such as depression, or as a persistent post-concussive syndrome.

Varney and colleagues call for a paradigm shift in the conceptualization of mild head injury and post concussive syndrome. He agrees that stress and poor adjustment may exacerbate the symptomatology, however, the sequelae acquired following MHI is predominantly neurological and organic, not functional and psychological. Although it is

doubtful that neurologists and neuropsychologists will totally abandon psychological theories for PPCS, Varney brings to surface several important variables that need to be kept in mind when conducting a thorough assessment.

### ***A Neuropsychological Model of Functional Disability***

A very comprehensive model of functional recovery, merging both the physiological and psychological sequelae and the interaction of the two was developed by Thomas Kay and his colleagues (Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992). The premise of the model by Kay et al., states that the responses of the individual to the presence of primary neurological and physical injuries will largely determine how functionally disabled the person will become and whether a mild head injury will develop into a complicated, or persistent, post concussive syndrome. When the primary brain injury resolves, it is possible for symptom reactions to perpetuate themselves and take on a life of their own. Although the process of recovery is similar for most people with a mild TBI, including changes in cognitive efficiency and processing capacity; the experience of failure and frustration when returning to premorbid activities, along with development of anxiety and depression resulting in a shaken sense of self, and a possible downward spiral of worsening psychological issues causing more cognitive deficits, lowering the depression, etc., can occur for some.

However, Kay et al. propose that the extent of disintegration of the self will depend on both personality characteristics and the adequacy of the environmental response. First, individual differences and environments will determine whether symptom focus will be minimal or exaggerated. Some individuals will become overly focused on every instance of

the changes they experience and others will acknowledge them and work through the changes. Second, the model identifies five vulnerable personality styles, based on clinical experience that may also differentially affect the individual's ability to adapt, and they are: the overachiever, the dependent person, the insecure person, the grandiose person and the person with borderline personality characteristics (not disorder). Third, emotional issues that occurred prior to the head injury may be triggered again, increasing their sense of vulnerability and helplessness.

Thus, the model identifies an interplay of reciprocal relations between the following areas: neurological factors, physical factors, psychological factors, objective cognitive factors and subjective cognitive factors, all of which contribute to functional outcome. What Kay et al. (1992) make clear in their model, is that recovery from a mild head injury is not a simple thing. Predictability of outcome from MHI may be more a function of premorbid personality characteristics and level of adaptability rather than just a function of neurological healing or neuroticism as implied in the past. This model is gaining some support (Parker, 1996; Ruff, Camenzulis, & Mueller, 1996; Ruff, Crouch, Troster, Marshall, Buchsbaum, Lottenberg, & Somers, 1994), however an objective study of actual personality disorders has yet to be published.

### *Purpose of the Study*

The main focus of this study is to investigate the presence of personality disorders in a mild head injury population with PPCS, and to examine if personality characteristics contribute to the outcome of mild TBI. Whether personality disorders are more prevalent in

the subgroup of mild head injuries who develop persistent symptoms following head injury has not been systematically studied to date. On what basis then, might personality disorders even be considered a factor in the psychogenesis of PPCS? First, research conducted with the MMPI and head injured populations has given some insight into common psychological disorders and personality styles within the head injured population (e.g., anxiety, depression, somatization of psychological pain). It is therefore a reasonable step to query whether personality disorders are also prominent. Second, when an individual does not meet the expectations of what is known in various medical and mental health disciplines, there is sometimes a tendency to question the client's verity and presume some form of secondary gain. These assumptions make for adversarial relationships between the head injured person and the people they rely on for medical advice and financial assistance (i.e., neurologists, neuropsychologists and third party payers). A personality disorder may account for some of the negative reactions mild head injured people receive from professionals and may also explain the individual's need to depend on medical formulations to help them cope, if they lack the internal resources to do the same.

Accordingly, the present study will be one of the first attempts to establish the presence or absence of personality disorders in a mild head injured population with PPCS, using a personality test that is consistent with the DSM-IV and a heuristic understanding of personality disorders. This may in turn, help further delineate a predictive variable of poor recovery from mild head injury, which could be subsumed under the constructs of "psychogenesis" and "individual vulnerability". The presence of a personality disorder, or even strong indications of maladaptive personality style, would have ramifications on

predicting outcome and treatment recommendations for head-injured individuals.

Specifically, recovery in 3 months may not be as likely in the subgroup of people with MHI and accompanying personality disorders compared to those without a personality disorder.

It is understood that the MCMI-II is only a clinical tool that can be used for the investigation of a personality disorder, and that it is not diagnostic of a personality disorder on its own. If an individual receives a clinically relevant score on the Borderline Personality Disorder Scale of the MCMI-II, this is not sufficient evidence to diagnose the individual with that particular personality disorder. A proper diagnosis should also include a comprehensive psychiatric interview, background history and information from significant others. The use of the MCMI-II in the present study is an attempt to measure personality disorders more formally than what has been done in the past, but the test is not assumed to be a sufficient source for diagnosis.

The present study will also attempt to establish whether or not the MCMI-II is a useful diagnostic instrument to use with the head injured population--will it add to the understanding of the psychological status over and above what is obtained with the widely used MMPI-2? Pragmatically, the MCMI-II would be an easy addition to a neuropsychological test battery in that it is a True/False questionnaire format, similar to the MMPI-2, but it contains only 175 questions. To the author's knowledge, only one published study has utilized the MCMI with a head injured population (Snibbe, Peterson & Sosner, 1980). These authors concluded that the MCMI offers useful information on longstanding personality styles, as opposed to more acute, transient symptom states, such as is found with the MMPI.

It is unclear why such a paucity of published information is available on the MCMI/MCMI-II with head injured and/or neuropsychological populations. One reason may be that the Millon manual advises that the MCMI/MCMI-II should not be used on "normal" people as the normative data is based on a "psychiatric" population. Unfortunately, the manual does not clearly define what constitutes "psychiatric". For example, does "psychiatric" imply only individuals seeking inpatient psychiatric treatment or does it include all possible diagnoses within the DSM IV, including adjustment disorders and V-Codes?

However, some authors claim that given the difficulty in establishing inclusion criteria to exist under the term "psychiatric", the use of the MCMI-II with "normal" populations is a valid practice, albeit with some interpretive cautions (Choca, Shaney & VanDenburg, 1992). In particular, Choca et al. (1992) advise that the examiner using the MCMI should be aware that the instrument was designed and standardized on a psychiatric population and that the scale descriptors must be altered and/or scaled down when interpreting profiles of non-psychiatric clients. Moreover, Millon (1992) himself states that the MCMI-II is appropriate for individuals who are referred to a psychological or psychiatric agency. Thus, a head injured population who experience psychological distress and cognitive sequelae as a result of a neurological insult is well within the boundaries of appropriate use of the MCMI-II.

***The Present Study:***

The purpose of the present study is to investigate personality disorders within a head injury population and to explore how these disorders relate to emotional functioning and the

persistence of post concussive symptoms. In addition, this study will be one of the first attempts to establish the clinical utility of the MCMI-II in neuropsychological populations. As detailed previously, 85 to 90 percent of individuals who sustain a mild traumatic brain injury recover from the post concussive sequelae within three months of the original injury. Fifteen to ten percent of individuals who sustain a mild TBI continue to report persistent post concussive symptoms for months to years past the 3-month recovery window. It is this latter group that is of interest in the present study; namely, individuals with a documented mild TBI who have not made a full recovery, and who are reporting ongoing symptoms and difficulties returning to premorbid functional levels. Thus, within the present study, the mild TBI group is also a PPCS group. This study does not include a group with mild TBI who have recovered from their brain injury.

The clinical group of interest consists of individuals who have sustained a mild head injury and who were reporting post concussive sequelae for more than 6 months following the initial traumatic brain injury. Comparison control groups will include a moderately-severe head injured sample, as well as a non-head injured, general neurological control group.

***Hypotheses:***

- 1) The first hypothesis addresses the issue of whether or not personality disorders are differentially represented among the three groups. The psychogenesis assumption would predict that the mild TBI group with persistence of post concussive symptoms would have more personality disorders compared to the other groups, and that personality factors are the reason this group has not recovered. The physiogenesis

assumption would claim that neurological damage is the cause of the persistence of post concussive symptoms, and that personality disorders have a nonsignificant role in explaining group differences. Given that the null hypothesis cannot be directly tested (i.e., the physiogenesis argument), this hypothesis will address the psychogenesis argument. Thus, it is hypothesized that the mild head injury group will evidence more personality problems in the way of higher scores on the MCMI-II scales compared to a group with moderately severe head injuries and a general neurological group.

- 2) It will be assumed that a proportion of MMPI-2 profiles will be clinically elevated, regardless of neurological condition. The physiogenesis assumption would contend that the psychological distress experienced by the mild TBI, the moderate TBI, and general neurological groups would be a function of the changes experienced since the acquired neurological condition. The psychogenesis argument, however, would suggest that the factors contributing to increased emotional distress will differ across groups. The psychogenesis notion would maintain that MMPI-2 profiles will be elevated in the moderate TBI and neurological control groups as a function of the difficulties adapting to an acquired neurological condition. In contrast, the mild head injury group will experience psychological stress for different reasons, such as a longstanding personality disorder. Thus, it is hypothesized that emotional/psychological stress (i.e., MMPI-2 scores) will be associated with the presence of personality disorders (i.e., MCMI-II scores) within the mild head injury

group, but will not be related to personality disorders in the moderate head injury group or the neurological group.

- 3) The third aspect of this investigation will examine whether neuropsychological test performance is associated and/or affected by the presence of personality disorders. This aspect of the study is exploratory in nature.

## METHOD

### *Subjects*

#### Selection Criteria

Subjects consisted of consecutive adult referrals to a hospital based neuropsychology out-patient service, in a Southern Ontario city. The hospital funded service provided neuropsychological assessments only, no rehabilitation services were offered. Major referral sources to the neuropsychologist were from neurologists and family physicians affiliated with the hospital. Another referral source were insurance companies requesting neuropsychological assessments for motor vehicle accident victims. Clients receiving services from this clinic, between 1992 through to 1996, were given the opportunity to sign a consent form (i.e., completely voluntary), allowing their raw test data to be used, at some future time, for research purposes. Psychology files were screened for endorsed consent forms and subjects were placed into one of three groups according to the following criteria.

Mild Head Injury Group with PPCS (PPCS): Inclusion criteria included 1) a documented or suspected injury to the head, 2) estimated loss of consciousness less than 20 minutes or a dazing experience with no loss of consciousness, 3) no evidence of cerebral or brainstem contusion or mass lesion, 4) neuropsychological testing performed between 6 months and 4 years post-injury 5) no history of prior head injury, alcoholism, cerebral disease, or significant psychiatric disorder, 6) premorbid intelligence estimates greater than 85, 7) age range between 19 and 55, and 8) English as a first language.

Moderate Head Injury Group (Mod TBI): Inclusion criteria included 1) a documented injury to the head, 2) estimated loss of consciousness greater than 20 minutes and less than 48 hours, 3) neuropsychological testing administered within 6 months to 4 years post-injury, 4) no history of prior head injury, alcoholism, cerebral disease, or significant psychiatric disorder, 5) premorbid intelligence estimates greater than 85, 6) age of injury between 19 and 55, and 7) English as a first language.

General Neurological Group (Neuro): Inclusion criteria included 1) neurological patients who did not have a history of traumatic brain injury (e.g., stroke, early Huntington's Disease, Multiple Sclerosis, seizure disorder, etc.), 2) no history of prior head injury or significant psychiatric disease, 3) premorbid intelligence estimates greater than 85, 4) age of onset between 19 and 55, and 5) English as a first language.

If more than one neuropsychological assessment had been administered on a given client, then the assessment closest to the injury onset date and/or the assessment closest to the onset date which included an MCMI-II was used in the present analysis. For the general neurological group, the neuropsychological assessment performed closest to the original referral which also included an MCMI-II was used for the present study (i.e., an attempt to keep the time interval between date of assessment and injury onset comparable to the TBI groups).

### Actual Sample

Approximately 749 patient files were reviewed for the purposes of the study. A total of 228 files were suitable for inclusion based on the above criteria. The

remaining 521 files were excluded for the following reasons: the MCMI-II was not available for data entry because an earlier version (e.g., MCMI) was administered, or, the MCMI-II was invalid ( $n = 136$ ), age requirements were not met ( $n = 89$ ), English was a second language ( $n = 45$ ), consent for research was refused ( $n = 20$ ), neuropsychological testing was not completed ( $n = 93$ ), patient sustained more than one documented traumatic brain injury ( $n = 21$ ), patient sustained 3 or more motor vehicle accidents ( $n = 17$ ), patient sustained a severe head injury ( $n = 19$ ), premorbid estimates and actual IQs were below 85 ( $n = 17$ ), referral question regarded adult assessment of possible developmental issues (i.e., learning disability, Attention Deficit Hyperactivity Disorder, etc.) ( $n = 23$ ), co-morbid psychiatric disorder ( $n = 16$ ), and 25 further files were excluded for various "other" reasons.

The final sample was based on 228 client files, 89 in the Mild Head Injury Group, 46 in the Moderate Head Injury Group and 93 in the Neurological Control Group. Table 3 outlines the mean age, education, IQ, loss of consciousness and length of time between onset of injury and testing across the three groups<sup>1</sup>.

There was a significant difference in ages among the 3 groups ( $F(2,225) = 5.99, p < .003$ ), with the Neuro group being older than the PPCS and Mod TBI, who did not differ from each other. There were no significant differences among the three groups on any of the other variables in Table 3. The average education level

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<sup>1</sup>Subject involvement with lawyers and third party payers (e.g., motor vehicle insurance agents) was not consistently documented in the patient files. Although litigation status was an important variable to consider, particularly when discussing stress and emotional adjustment issues, this variable was unable to be reliably assessed, and therefore not included in the statistical analyses.

**Table 3: Means and Standard Deviations of Age, Education, I.Q., and Time Between Injury and Assessment Across the Three Groups.**

SUBJECTS	Age (in years)		Education (in years)		Intelligence		Time Since (in months)	
	M	s.d.	M	s.d.	M	s.d.	M	s.d.
PPCS <sup>a</sup>	36.6	10.5	12.4	2.5	98.3	10.3	23.7	27.0
Moderate TBI <sup>b</sup>	34.7	11.5	12.3	2.6	98.1	13.0	22.8	35.5
Neurological Controls <sup>c</sup>	40.5	9.3	12.5	2.7	96.2	9.9	24.6	25.3

<sup>a</sup>  $n = 89$     <sup>b</sup>  $n = 46$     <sup>c</sup>  $n = 93$

across groups was approximately 12 years and the mean time between illness onset and testing was nearly 2 years. Mean Full Scale IQs across the three groups were 98, 98, and 96, respectively. Sex was equally distributed within the mild TBI and Neuro groups (males = 45 and 46 and females = 44 and 47, respectively). There were more than twice as many males than females in the Mod TBI group (males = 32, females = 14). This finding was not unexpected, as men are more likely to sustain a moderate to severe head injury compared to women.

Table 4 identifies the causes of traumatic head injuries for the mild and moderate TBI groups, and Table 5 identifies the neurological illness or primary investigative complaint of the Neurological control group. From Table 4, motor vehicle accidents (MVA) accounted for 73 % of the PPCS group, and 65 % of the Mod TBI group. The Neuro group consisted of a broad range of neurological diagnoses and/or complaints, including stroke, seizure disorder, people complaining of "general memory problems", tumor, Multiple Sclerosis, Huntington's, toxic exposure, etc (see Table 5). The "Other" category includes diagnoses such as "query" Multiple Sclerosis, query anoxia, various neurological soft signs, acquired memory problems following non-neurological surgery (e.g., breast cancer), migraine, Tourette's Syndrome, Raynaud's Disease, Bells Palsy, etc. The sample appears relatively representative of typical referrals to a Neuropsychological service.

**Table 4: Frequency and Percentages of Causes of Brain Injury Within the Mild and Moderate Brain Injury Groups**

CAUSE OF INJURY	PPCS Group		Moderate TBI Group	
	<u>n</u>	percent	<u>n</u>	%age of group
Motor Vehicle Accident	65	73	30	65
Falls	7	8	5	11
Hit by object	4	4.5	0	0
Pedestrian*	4	4.5	2	4.3
Bicycle	1	1.1	2	4.3
Motorcycle	3	3.4	4	9
Other	5	5.6	3	7

\* = Pedestrian hit by moving vehicle

**Table 5:            *Frequencies and Percentages of Referral Diagnosis or Referral Question Within the Neurological Control Group***

REFERRAL DIAGNOSIS	Neurological Control Group	
	<u>n</u>	Percentage
Stroke	15	11
General Memory Problem	12	13
Seizure Disorder	9	10
Tumor	8	8.6
Multiple Sclerosis	6	6.5
Toxic Exposure	5	5.4
Medical Problems	5	5.4
Meningitis/Encephalitis	4	4.4
Huntington's Disease	3	3.2
"Other"	25	27

## ***Measures***

***Intelligence:*** IQ was measured by the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981), the most widely used test of psychometric intelligence (Jarvis & Barth, 1994; Lees-Haley, Smith, Williams & Dunn, 1995; Lezak, 1995).

## ***Neuropsychological Tests***

- 1) **The Halstead-Reitan Neuropsychological Battery (HRB)** (Reitan & Wolfson, 1993). The HRB is the most researched and validated neuropsychological test battery (Jarvis & Barth, 1994) providing for the assessment of several important neuropsychological domains. Although various tests have been added over time, for the purposes of this study, the "HRB" will consist of the 8 tests (12 scores) that are included in the Average Impairment Rating (AIR).

***The Halstead Category Test (HCT):*** The category test is a test of problem solving, judgment, abstract reasoning, concept formation and the ability to modify behaviour from corrective feedback. Subjects are shown slides of various geometrical designs on a projector screen. These designs are intended to remind the person of a number between 1 and 4. Subjects are instructed to pull down on the numbered keys on the keyboard that corresponds to the number suggested by the pattern on the screen. There are 7 subtests, each with one underlying principle or idea that runs throughout the subtest, and the individual's task is to figure out the underlying principle. Some examples of underlying principles include ordinal position of the odd shape, proportion of

shape that is solid vs. perforated, etc. When subjects pull down on the correct key, a pleasant bell is sounded and a buzzer is heard when the response is incorrect. The last subtest does not have an underlying principle; it consists of slides from the previous 6 subtest and is considered a memory trial. A total of 208 slides are presented and the number of errors made represent the subject's score.

*Tactual Perception Test (TPT):* The TPT is a test that measures tactile-kinesthetic problem solving abilities and spatial memory. On the TPT, subjects are blindfolded and situated in front of a rectangular board that is positioned upright, on which there are 10 differently shaped spaces. Wooden blocks that correspond to the spaces on the board are placed on the table between the subject and the board. Subjects are asked to place each block into its corresponding space on the board, as quickly as s/he can. There are three trials, one with the dominant hand, one with the nondominant hand and on the last trial subjects are allowed to use both hands. Following the third trial, the blocks and form board are put away before the blindfold is removed, then the subjects are asked to draw a picture of the board. Subjects are required to detail the correct shape as well as the general location of the shapes on the board. This is an incidental memory task which has been shown to be particularly sensitive to cerebral trauma (Jarvis & Barth, 1994; Reitan & Wolfson, 1993).

*Speech-Sounds Perception Test:* This is a test of auditory verbal sustained attention, phonemic hearing, sound-symbol association, and concentration, in which the subject has a response sheet with 60 sets of 4 "nonsense" words which all contain the "ee" sound. A recorded voice states one of the 4 nonsense words and the patient is to underline the corresponding word on the answer sheet. The test set is divided into 6 sets of 10 nonsense words and the voice on the recording cues the examinee as to which set or column and to the item number (e.g., Column B, the first word is 'theets'). The number of errors constitutes the subject's score.

*Seashore Rhythm Test:* This is a test of auditory, nonverbal attention and concentration in which the subject listens to 30 pairs of rhythmic patterns and is required to discriminate whether the two patterns are the same or different. The test is divided into 3 sets of 10 pattern pairs and the subject is cued as to when each column begins, however, there are no cues to know what item number they should be on. This test moves fairly rapidly and the task demands good concentration. The number of correct discriminations constitutes the subject's score.

*Finger Oscillation Test (Finger Tapping):* This is a test of simple motor speed and coordination in which the examinee taps a lever as rapidly as possible, which is attached to a mechanical counter. The subject is asked to tap as fast as s/he can for 10 seconds with the index finger. Five trials within 5 taps of each other are given for the dominant hand and then for the non-dominant

hand. The average of the five trials, per hand, constitutes the score. Average score for the dominant hand is used in the Halstead Impairment Index, and the lowest score, regardless of dominance, is used in the Average Impairment Rating.

*Halstead Impairment Rating (HII):* The HII is a composite measure of 7 HRB tests. Each of the scores is compared to a cut-off score indicating brain-damage or no brain-damage. The index score indicates the proportion of those test scores that are in the range characteristic of brain-impaired patients. The seven test scores include the Category Test (cut-off score is 51 errors and above), the TPT total score (cut-off score is 15.7 minutes and above), the TPT memory score (cut-off score is 5 shapes recalled or less), the TPT localization score (cut-off is 4 or less correctly placed blocks), the Seashore Rhythm Test (cut-off is 6 errors or more), the Speech-Sounds Perception Test (cut-off is 8 errors or more), and dominant hand finger tapping (cut-off is 50 taps or fewer). The formula for calculating the HII is to divide the number of tests in the impaired range by the number of total HII tests administered, with scores ranging from 0 to 1.0. A HII between 0.0 and 0.2 is considered to be in the normal range, a HII score between .3 and .4 is considered to indicate mild brain damage, between .5 and .78 as indicating moderate brain damage and HII scores between .8 and 1.0 to imply severe brain damage.

*Trail Making Test:* The first part of this test, Trails A, is a measure of visual scanning ability and psycho-motor speed. The subject is given a piece of paper

with 25 numbered circles distributed in a random pattern, and is required to connect the circles with lines in numerical order as quickly as possible. The second part, Trails B, is a sheet of paper with both numbers and letters, encased in circles, distributed in a random pattern. The subject is asked to connect the circles, alternating between numbers and letters (e.g., from 1 to A, then A to 2, then 2 to B, etc). This is a test of divided attention, visual scanning ability and psychomotor speed. Completion time constitutes the subject's score.

*Aphasia Screening Test:* This measure was designed to screen for a variety of areas of dysfunction including dysnomia, dyslexia, spelling dyspraxia, dyscalculia, dysgraphia, constructional dyspraxia and right-left confusion. The test consists of 33 simple questions in which subjects are asked to draw-name-spell simple geometrical shapes, name pictured objects, copy or write simple words or statements, read simple phrases, repeat words and phrases, simple arithmetic and follow simple commands. For the purposes of the Average Impairment Rating (see below), this test was quantified and scored according to the criteria outlined in the manual with errors costing from 1 to 4 points. The total error score of all items except for copying items constitutes the subject's score. In addition, the Average Impairment Rating included a ranked "spatial relations" score, which was based on level of performance of copying a Greek cross, two times. The subject's drawing is compared to sample drawings and given a rank between 1 and 4.

***Sensory-Perceptual Exam:*** This is a test to screen for sensory perceptual impairment in tactile, auditory and visual modalities in both a unilateral and double simultaneous stimulation paradigm. Tactile perception is assessed by a light touch to either hand or both simultaneously, then either hand and contralateral side of face either singly or simultaneously. The subject's eyes are closed during each administration. Auditory perception is assessed by a light rubbing of fingers beside either or both ears while the subjects eyes are closed. Visual perception is measured by the examiner sitting in front of the patient and making a slight movement of the fingers at the periphery of the patient's vision while s/he focuses on the bridge of the examiner's nose. Also included is a measure of finger agnosia, in which the fingers are numbered 1 through 5 and the subject identifies which finger the examiner touched, by number, while the eyes are closed. Finally, finger tip number writing is administered in which the examinee is asked to identify numbers written with a stylus on the fingertips of his/her hand by the examiner. A comparison of the number of errors on the two sides of the body is the important information obtained from this portion of the battery.

***Av - I Score:*** This is also a score included in the Average Impairment Rating and is based on the ratio of the Digit Symbol subtest of the WAIS-R to three other Performance subtests of the WAIS-R, namely, Block Design, Object Assembly and Picture Arrangement. The scaled score of the digit symbol is

considered to be suggestive of brain injury, if it is relatively lower than the average of the other 3 Performance subtests, minus one.

*The Average Impairment Rating (AIR):* The AIR was developed by Russell, Neuringer and Goldstein (1970) and is a modification of the HII. The AIR includes more tests from the Halstead-Reitan battery, and is based on an expanded normative base, relative to the HII. Whereas a single cut-off score is used, per test, in the HII, the AIR places each score along a continuum of scores crossing 6 categories. The ranking of each score from 0 (above average) to 5 (severe impairment) were considered relative to standard deviations from the mean of the normative group, and/or, representative of known brain injury impairment (e.g., the spatial relations score where the subject's drawing is compared to 4 different levels of impaired drawings). The tests included in the AIR are as follows: Category Test, TPT total score, TPT memory score, TPT localization score, Seashore Rhythm Test, Speech-Sounds Perception Test, Finger Tapping (worst hand), Trails B, Aphasia Screening errors, Spatial Relations Score, Sensory-Perceptual errors, and the Av - 1 ranking. To obtain the AIR, a ranking number of 0 to 4 is given to each of the 12 tests, these numbers are added and divided by 12. The average rating of the 12 tests is the Average Impairment Rating. An AIR above 1.55 indicates the mild end of the brain injury spectrum.

## 2. Other Neuropsychological Tests

*Wechsler Memory Scale-Revised* (WMS-R) (Wechsler, 1987): The WMS-R is a relatively widely used battery of memory tests. It consists of 12 subtests that measure various aspects of auditory-verbal memory, visual memory and attention and concentration. The overall scores consist of 5 Index scores created from 2 or more of the subtests (i.e., Verbal Memory Index, Visual Memory Index, General Memory Index, Attention/Concentration Index and the Delayed Recall Index). Research has indicated that the WMS-R is sensitive to memory problems in a wide variety of neuropsychological populations (Spren & Strauss, 1991; Wechsler, 1987).

*Grooved Pegboard* (Klove, 1963): The grooved pegboard requires both motor speed and fine motor coordination for successful completion. The test requires subjects to place 25 notched pegs in slotted holes, as rapidly as they can; the hole being oriented in random directions on a board. The test simulates placing a key in a grooved hole. One trial is given with each hand, and the time, in seconds, to place all 25 pegs constitutes the score.

*Grip Strength*: A measure of strength of grip (Lezak, 1983) is routinely used in neuropsychological test batteries. This is a measure of grip strength and laterality as the patient is asked to squeeze the hand dynamometer as hard as possible. Two alternating trials are given for both hands, and the average of the 2 measurements is recorded.

*Consonant Trigrams Test:* This is a test of short-term retention with distraction (Lezak, 1995). The subject is given 3 consonants followed immediately by a number. The distracter task is to count backwards, by three's, until the examiner knocks on the table. At this point the subject is asked to recall the original 3 consonants. The distracter tasks (i.e., counting backwards) occurs for 3, 9 or 18 seconds, with 5 trials at each delay time. The number of correctly recalled consonants constitutes the overall score for this test.

### Achievement Test

*Wide Range Achievement Test, Third Edition (WRAT-3):* The WRAT-3 is the most recent edition of a widely used screening tool for reading recognition, spelling and computational arithmetic. Two versions of each subtest are available; however, the Tan Version was the most consistently used. Standard scores and Grade Levels are obtained.

### Tests of Motivation

*Rey 15-Item Test:* This task is presented as a test requiring the memorization of 15 different items in 10 seconds. That *15 items* need be remembered is stressed to make the task seem difficult. In fact, subjects only have to remember 3 general ideas (i.e., capitol and lower case letters a,b,c; Roman and Arabic numbers 1,2,3; and 3 simple shapes). The principle underlying this task is that the subject who consciously or unconsciously wishes to exaggerate their memory deficits, will perform poorly on a test that all but the

most severely brain damaged patients perform easily (Lezak, 1995). A score below 9 is considered suspect.

*Frederick Test* (Frederick & Foster, 1991): This is a forced-choice test of cognitive ability, based on the Test of Nonverbal Intelligence (TONI; Brown, Sherbenou, & Johnsen, 1982), in which subjects are given a variety of puzzle or matrix-like designs that are incomplete and they are to choose which of two possible answers would best complete the pattern. The subject is told that the 100 questions get progressively more difficult, when in fact the difficulty levels are randomly presented. Guessing is discouraged. The rationale underlying this task is that individuals who were severely impaired or using random responding would perform at least at chance levels. Individuals who, either consciously or unconsciously, were attempting to exaggerate their disability levels would likely perform below chance levels in their ability to "look bad". There are four scores that are used in the decision making process: 1) overall score, with subject scores less than 43% correct being considered suspect; 2) the slope--when test items are plotted from easiest to hardest, valid responders would be expected to perform at 100% on the easier items and at chance level for the difficult items, or, when they reached their ceiling. This visual plot would demonstrate a negative slope. So called "malingerers" are assumed to perform below chance overall, thus requiring to perform well below chance on the easy items and at chance on the difficult, or, ceiling items. This would produce a flat, or, small positive slope; 3) A consistency ratio (CR) in which

the ratio of the number of equivalent item pairs in which both items were answered correctly to the maximum possible number of item pairs in which both items could be answered correctly: The assumption here is that malingerers are assumed to perform inconsistently, and 4) slope  $\times$  CR measure, a score found to be extremely sensitive to biased responding if below the cutoff score of  $-.0041$  and the subject's overall score is below average. Discriminant validity data demonstrates specificity and sensitivity relative to other measures of response bias (Frederick & Foster, 1991; Frederick, Sarfaty, Johnston & Powel, 1994).

#### Tests of Emotional Functioning and Personality

**MMPI-2** (Hathaway & McKinley, 1951): This is primarily a test of current emotional status, psychopathology and personality characteristics. It consists of 567 statements in which the subject responds True or False as it applies to them in the last few weeks. This is a widely used and well researched test of psychological functioning. Although several types of scores can and have been derived on computer scoring programs, the present study will include the original 10 clinical scales and the three validity scales (see Table 1 for a review of the scales and their interpretive descriptors). Computer generated T-scores with a normative reference group are generated. A T-Score  $> 65$  is considered significant and within the clinical range.

**MCMI-II**: The MCMI-II is a 175-item true/false questionnaire, similar in format to the MMPI-2. It is based on Millon's personality theory and is

heuristically congruent with the DSM-IV. The test has 22 scales, although of interest in the present study are the 13 personality disorder scales. Scoring is complicated and is performed by a computer scoring program. The reference group is psychiatric patients, and Base Rate (BR) scores are generated for each scale. These scores reflect how the patient's responses compare with the prevalence of characteristics in a psychiatric population. A BR score of 35 is the "normal" median, a BR > 75 indicates the presence of a disorder and a BR score > 85 on a particular scale indicates that this disorder is most prominent or salient for the individual (see Table 2 for scale descriptors).

### *Data Analysis*

#### Hypothesis One:

This hypothesis investigated whether the personality disorders, as measured by the MCMI-II differed across groups. In particular, that the PPCS group would have higher scores on the MCMI-II personality disorder scales, compared to the Mod TBI and Neuro groups. A One-way, between subjects MANOVA was executed to determine which MCMI-II scales were elevated across the 3 groups. Univariate  $F$  tests were examined only if the overall, multivariate  $F$  test was significant.

Comparison of group means utilized the Tukey post hoc analyses, and a Bonferroni

familywise correction factor was used to adjust significance levels for the post hoc tests<sup>2</sup>.

*Hypothesis Two:*

This hypothesis investigated the relationship of psychological /emotional stress to the presence of personality disorder within each group. Canonical correlations were used to examine the relationship between MCMI-II clinical scales and MMPI-2 clinical scales, within the three separate groups. The canonical correlation method examines the relationship between two data sets, along a series of underlying dimensions. A canonical correlation, unlike a simple correlation, places no limits on the number of variables to be placed in the correlational analysis. Whereas simple  $r$  examines the relationship between two variables,  $x$  and  $y$ , canonical correlation examines the relationship between two sets of variables,  $X = (b_1x_1 + b_2x_2 + \dots + b_px_p)$  and  $Y = (c_1y_1 + c_2y_2 + \dots + c_qy_q)$ . The canonical correlation procedure defines a weighted linear combination of MCMI-II personality disorder scales (i.e., the "X set") and a linear combination of MMPI-2 clinical scales (i.e., the "Y set") to uncover the maximum variance overlap between the two data sets (e.g., joint variance). This becomes the first canonical correlation, or canonical variate. Then the next canonical variate is selected, such that it is orthogonal to the first canonical variate, and so on. The maximum number of possible canonical correlations equals the smaller number of

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<sup>2</sup>A profile analysis is another line of investigation when examining multiple dependent variables across groups. It was not felt to be appropriate in the present MANOVA because the hypothesis was investigating differences among the groups on individual test scales. A profile analysis would investigate whether the test *profiles* differed among the three groups.

variables in either the X or Y set. For the present analysis, there were 13 MCMI-II personality disorder scales and 13 MMPI-2 clinical scales, thus, 13 possible canonical correlations could be produced.

*Neuropsychological Variables:*

Do personality disorders affect performance on neuropsychological tests? The third line of investigation in this study is exploratory in nature and will examine whether there is a relationship between number of personality disorders and neuropsychological test results. Initially, a MANOVA will be conducted to examine overall group differences on the neuropsychological tests. Theoretically, the neurological control group and the moderate head injury group would be expected to perform more poorly compared to the mild head injury group. According to the "psychogenesis" theory of PPCS, the relationship between personality factors and neuropsychological performance would predict that poorer scores on neuropsychological tests would be related to personality problems (e.g., poor motivation, exaggeration of impairment for attention, etc.), particularly within the mild TBI group. According to the "physiogenesis" theory of PPCS, there would be no relationship between neuropsychological test performance and characterological problems. A simultaneous multiple regression analyses will investigate this question, within each group. The dependent variable, represents the number of personality disorders, and the predictor variables, represents those neuropsychological test scores believed to be particularly sensitive to brain injury.

## RESULTS

### **MCMI-II**

Hypothesis One states that the PPCS group would demonstrate higher scores on the MCMI-II compared to the Mod TBI and Neuro groups. There was an overall significant difference of MCMI-II scores across groups (multivariate  $F(26, 426) = 2.079, p < .01$ ). Examining the univariate tests, Table 6 details which of the 13 personality disorder scales differed across the three groups. Significant group differences were found specifically on the following personality disorder scales: Dependency ( $F(2, 225) = 4.855, p < .01$ ), Aggressive-Sadistic ( $F(2, 225) = 6.542, p < .01$ ), Passive-Aggressive ( $F(2, 225) = 5.127, p < .01$ ), Self Defeating ( $F(2, 225) = 3.739, p < .05$ ), and Borderline ( $F(2, 225) = 6.893, p < .001$ ).

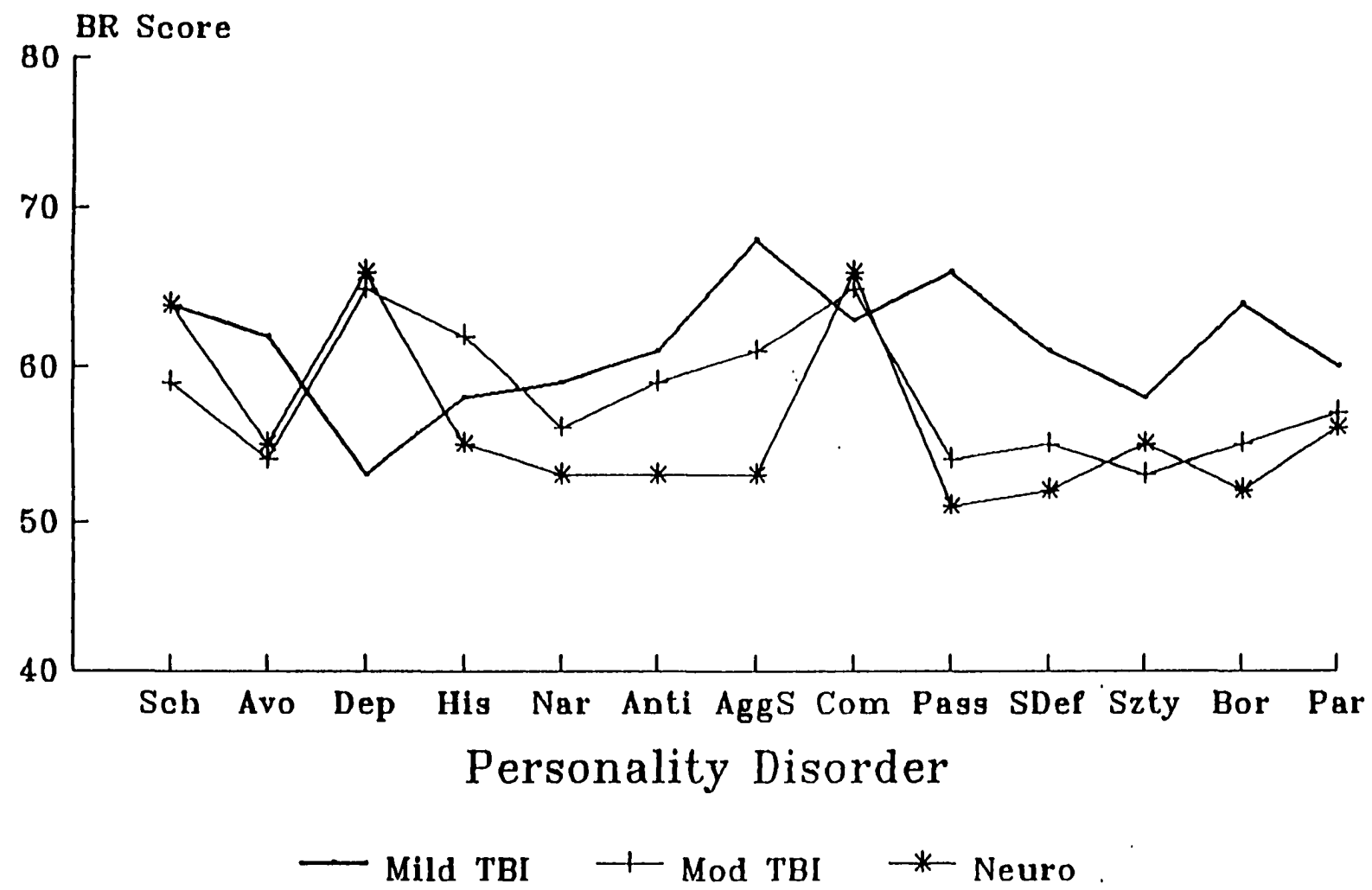
If the assumption that PPCS is a function of maladaptive personality characteristics is true, then the analysis of group means on the MCMI-II would ultimately reflect that the Mild TBI group with PPCS have higher scores compared to the Mod TBIs and Neuro groups. Keep in mind that this particular group of Mild TBI represents the 10 percent who report the persistence of post concussive symptoms, and there is no a priori reason to predict that the Mod TBIs and Neuro group means should differ from each other. Figure 1 plots the group means for each personality disorder scale. Post hoc tests indicate that the PPCS group scored significantly higher on the Passive-Aggressive, Borderline, Aggressive-Sadistic, and Self-Defeating scales, compared to the Neuro group. Using the Tukey HSD test with correction for unequal n's and alpha maintained at .05, no statistically significant

**Table 6: MANOVA of MCMI-II profiles across PPCS, Moderate TBI and Neurological Control Groups**

<i>Multivariate Test of Significance with (26, 426) D.F.</i>		
	F	p
Wilkes Lambda	2.077	<b>.002*</b>
<i>Univariate F-tests with (2, 225) D.F.</i>		
Schizoid	0.942	.391
Avoidant	2.478	.086
Dependant	4.855	<b>.009</b>
Histrionic	1.415	.245
Narcissistic	0.932	.395
Antisocial	2.269	.106
Aggressive-Sadistic	6.542	<b>.002</b>
Compulsive	0.746	.476
Passive-Aggressive	5.127	<b>.007</b>
Self Defeating	3.739	<b>.025</b>
Schizotypal	1.029	.359
Borderline	6.893	<b>.001</b>
Paranoid	0.874	.419

\* Bold print indicates  $p < .05$

### FIGURE 1: MCMI-II Mean Scale Scores For All Groups



differences were found between the moderate TBI group and the mild TBI group on any of the five scales. However, there was a trend for the Moderate TBI group to score more similarly to the neuro controls than to the PPCS group. Thus, the mild TBI group scored significantly higher on Passive-Aggressive, Aggressive-Sadistic, Self-Defeating and Borderline personality traits compared to the neurological control group, with moderate TBI's falling in between.

On the Dependent Personality Disorder scale, the PPCS group was found to have significantly *lower* scores compared to the Mod TBIs and Neuro groups, who did not differ from each other. This result would appear to indicate that having a more severe neurological condition, like the moderate TBI and neurological group, may place noticeable constraints on adaptive coping, diminishing the individuals's self-efficacy, and making him or her more dependent on others for decision making, emotional support and quality of life.

Interpretive evaluations for the MCMI-II suggests that a base rate (BR) score greater than or equal to 75 indicates that the individual has endorsed characteristics very similar to the normative psychiatric population who had the particular personality disorder. A base rate score exceeding 85 would indicate the most predominant personality style. Figure 1 demonstrates that group means across all 13 personality disorder scales did not reach a clinically significant range. However, how many personality disorders (e.g., base rate score  $\geq 75$ ) were in each group? Table 7 lists what percent of each group reached a BR  $\geq 75$  for each personality scale. A greater

**Table 7: The Number and Percentage of MCMI-II Base Rate Scores Over 75 within Each Personality Disorder, by Group**

MCMI-II SCALE	PPCS Group (Total = 89)		Moderate TBI Group (Total = 46)		Neuro Group (Total = 46)	
	<u>n</u>	%age of group	<u>n</u>	%age of group	<u>n</u>	%age of group
Schizoid	20	22	6	13	17	18
Avoidant	23	26	9	20	20	22
Dependant	25	28	17	37	20	45
Histrionic	29	33	11	24	27	29
Narcissistic	31	35	8	17	21	23
Antisocial	25	28	7	15	15	16
Aggressive-Sadistic	36	40	16	35	20	22
Compulsive	24	27	16	35	22	24
Passive-Aggressive	34	38	7	15	25	27
Self-Defeating	20	22	2	4	11	12
Schizotypal	8	9	2	4	5	5
Borderline	21	24	3	7	12	13
Paranoid	12	13	4	9	6	6

percentage of the PPCS sample obtained clinically elevated personality disorder scores compared to the other two groups across all scales except the Dependent personality scale, and the Compulsive scale. The average number of scales on the personality inventory that were elevated, per subject, was 3.5 for the PPCS group, 2.3 for the Moderate TBI group, and, 2.4 for the Neurological group.

### **MMPI-2**

MMPI-2 profiles were re-scored using a neurological correction factor (Gass, 1991), to control for the probability of true neurological symptoms that could be masked as psychiatric symptoms on the MMPI-2. Comparison of the original MMPI-2 and the neurologically corrected MMPI-2 (MMPI-2nc) used a 1-way, between subject MANOVA on the difference scores. This analysis revealed no multivariate group differences ( $F(26, 406) = 1.25, p = .189$ ). As there was no significant change in MMPI-2 scores using the neurological correction factor, the original MMPI-2 scores were used in the remaining analyses.

MMPI-2 profiles were examined for validity. Criteria used to denote an invalid profile were based on guidelines recommended by Graham (1990), and were as follows: Lie and K scales with T-scores greater than 70 and F scales with T-scores greater than 99. Although the F scale cutoff is relatively high, this scale also measures degree of pathology and can remain interpretable when scored in the higher ranges. F scale scores over 99 clearly denote invalidity or deviant test-taking practise. Thirteen percent of the total sample ( $n = 30$ ) met these three criteria and were

excluded from all further analyses. The percentage of invalid profiles was relatively consistent across the PPCS, Mod TBI and Neuro groups (12%, 9%, and 16%, respectively). Twenty-six percent of the total sample obtained "normal" profiles (e.g., all subscales with T-score below 70). However, only 17% of the PPCS group obtained "normal" profiles, compared to 31% and 33% of the Mod TBI and Neuro group, respectively.

The MANOVA investigating an overall group effect of MMPI-2 subscales did not reveal significant differences ( $F(26, 366) = 1.30, p = .160$ ), and therefore, univariate tests were not examined. Collapsing the three groups together, clinically elevated MMPI-2 scales (e.g.,  $T > 65$ ) were found on scales Hs, D, Hy, Pa and Pt. Figures 2, 3, 4 and 5 display the mean T-score profiles for the Total group, the PPCS group, the Mod TBIs and the Neuro Groups, respectively. On visual inspection of the four graphs, it appears that the profile pattern is very similar across the three groups. The PPCS group has generally higher group mean scores and has clinically elevated scores on scales Hs, D, Hy, Pt, and Sc, very similar to the Total group profile. In contrast, the Mod TBI and Neuro groups had clinically elevated scales on Hs, D, and Hy only. The Pt and Sc scales were elevated, but they did not fall above the  $T=65$  cut-off.

In general, the total sample's MMPI-2 results are similar to what has been previously found in neurological populations, with scales Hs, D, Hy, Pt and Sc constituting the "typical" neurological profile. The PPCS group had clinically elevated groups scores on all five scales. The Mod TBI and Neuro groups did not

FIGURE 2 MMPI-2 Mean Scale Scores  
Total Group

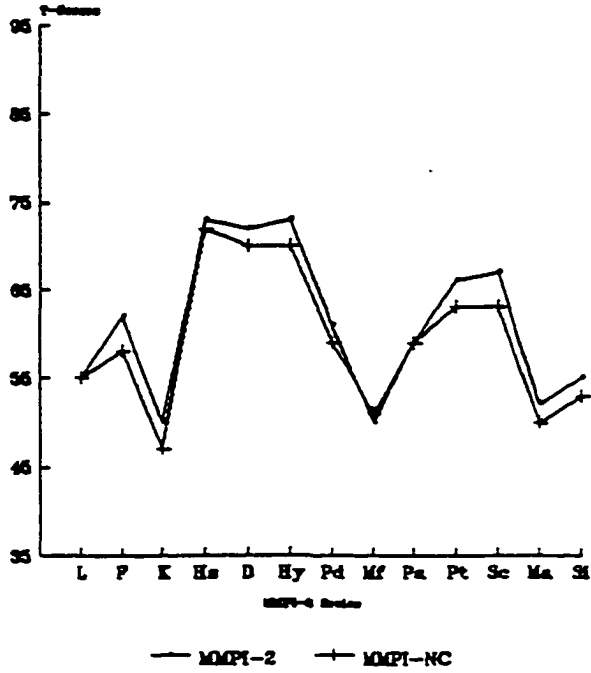


FIGURE 3: MMPI-2 Mean Scale Scores  
for Mild TBI Group

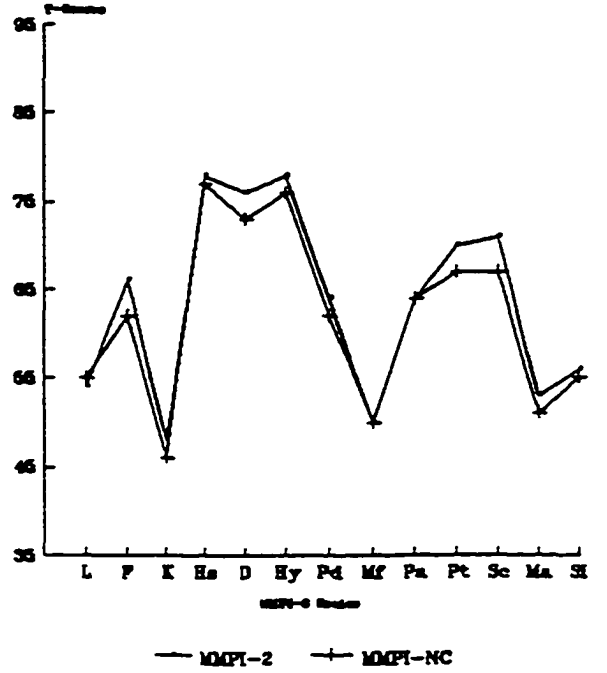


FIGURE 4: MMPI-2 Mean Scale Scores  
for Moderate TBI Group

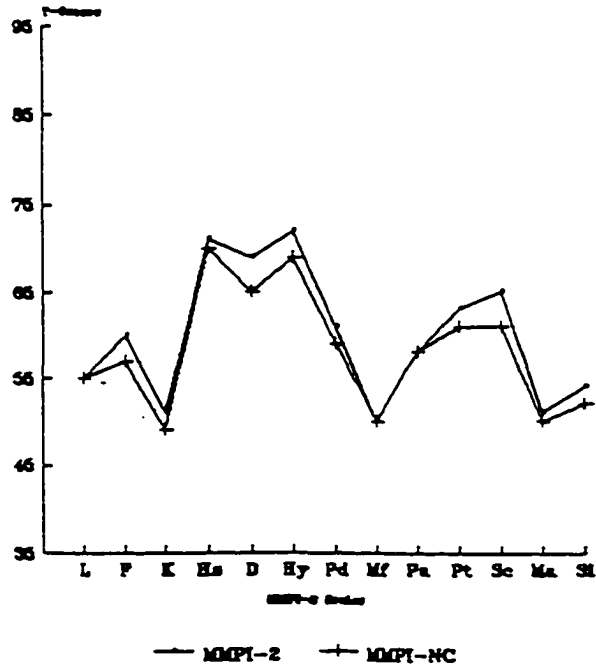
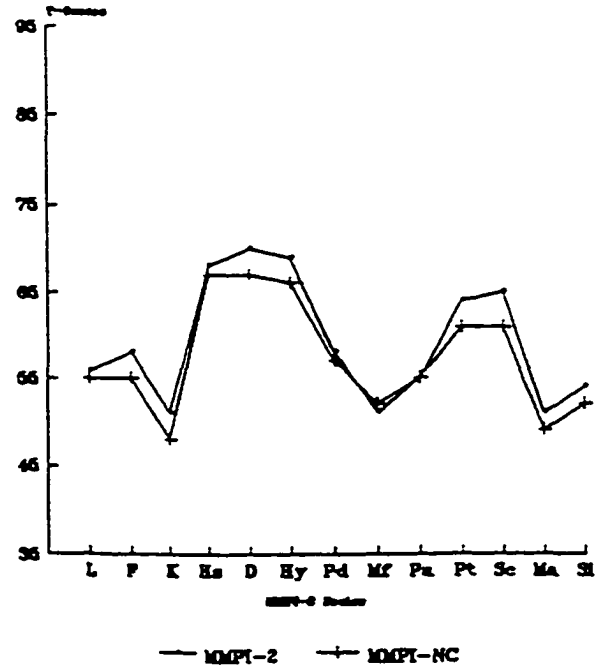


FIGURE 5: MMPI-2 Mean Scale Scores  
for Neurological Group



reveal clinically elevated T-scores on the Pt and Sc subscales. Interestingly, when the current sample is collapsed into one complete group, Figure 1 shows that the means of the Pt and Sc subscales become elevated. In essence, the PPCS group appears to have brought the Total group mean scores up to clinically elevated levels for scales Pt and Sc.

Table 8 lists the percentage of each group that had a T score greater than 65 within the three groups. In the PPCS group, the frequency of subscale T-scores was highest for Hs (85%), Hy (78%), D (76%) and Sc (62%). In the Mod TBI group, the frequency of subscale scores was highest for Hy (64%), Hs (62%), and D (55%). In the Neuro group, subscales D (60%), Hy (59%), and Hs (56%) had the highest frequency of clinically elevated scores.

**Canonical Correlations:**      *What is the Relationship Between the MCMI-II and the MMPI-2?*

Canonical loadings can be thought to be similar to factor loadings in factor analysis, in that the loadings are the simple correlation,  $r$ , between each subtest from the MCMI-II and the MMPI-2, with the canonical variate. The loadings that are represented in bold type-face in Table 9 share at least 50% of its variance with the canonical variate and are considered to have "loaded" on that variate. A cut-off loading of .7 may seem high, considering that canonical loadings greater than .3 has been recommended for inclusion as being a relevant finding (Tabachnik & Fidell, 1996). A higher cut-off was used in the present analysis to aid in the clinical

**Table 8: The Number and Percentage of MMPI-2 T-Scores Over 65 within Each Psychological Scale, by Group<sup>a</sup>**

MMPI-2 SCALES	PPCS Group (Total = 78)		Mod TBI Group (Total = 42)		Neuro Group (Total = 78)	
	<u>n</u>	%age of group	<u>n</u>	%age of group	<u>n</u>	%age of group
L (lie)	12	15	4	9	12	15
F (frequency)	32	41	11	26	21	27
K (defensiveness)	2	3	4	9	5	6
Hs (hypochondriasis)	66	85	26	62	44	56
D (depression)	59	76	23	55	46	60
Hy (hysteria)	61	78	27	64	46	59
Pd (rebelliousness)	34	44	12	29	18	23
Mf (masculine-feminine)	2	3	4	10	10	13
Pa (paranoia)	33	42	9	21	16	21
Pt (anxiety)	45	58	16	36	29	37
Sc (bizarre thinking)	48	62	19	45	33	42
Ma (hypomania)	16	21	4	10	14	18
Si (social introversion)	20	26	7	17	17	22

<sup>a</sup> Invalid MMPI-2 profiles have been removed from the group data (invalidity based on:  $F \leq 99$ ,  $L \leq 70$ , and  $K \leq 70$ )

relevance of the overall canonical correlations matrix. Canonical correlations are often mathematically correct, but criticized for the lack of meaningfulness in interpretation. Thus, for ease of interpretation and to provide clinically relevant findings, a canonical loading was included in interpretation if it is shared at least 50% of its variance with the canonical variate.

The value of the canonical correlation represents the multivariate correlation of the set of MCMI-II tests with the set of MMPI-2 tests that load on the overall variate. The two "sets" of subtests, make up the two parts of the canonical variate pair. Thus, the value of the canonical correlation is the correlation between the canonical variate pair. The following is a contrived result for illustrative purposes. Presume that the Schizoid and Schizotypal scales of the MCMI-II loaded on the first canonical variate, and we will label this "Set#1 of CanVar A". Also presume that Sc (Bizarre Thinking) and Pa (Paranoia) scales of the MMPI-2 loaded on the first canonical variate, and we will label this "Set#2 of CanVar A". Set#1 and Set#2 are considered to be the canonical variate pair for CanVar A. If the Canonical Correlation equals .75, then this is the multivariate correlation of Set#1 with Set#2 on CanVar A. Thus, the correlation of Set#1 (Schizotypal and Schizoid) with Set#2 (Sc and Pa) is .75.

#### Mild Head Injury/PPCS Group

There were two significant canonical correlations within the PPCS sample. The first canonical correlation equalled .900 ( $p < .000$ ), accounting for 81% of the variance of the first canonical variate pair. The second canonical correlation equalled .884 ( $p < .000$ ), accounting for 78% of the variance of the second canonical variate

pair. Table 9 shows the canonical loadings of the MCMI-II scales and MMPI-2 scales to this canonical variate pair for each group.

From Table 9, the first canonical variate pair included Histrionic Personality Disorder, Narcissistic Personality Disorder, Antisocial Personality Disorder from the MCMI-II (i.e., Set#1 of CanVar1), and, Ma (Hypomania) from the MMPI-2 (i.e., Set#2 of CanVar1). The personality characteristics measured by the Ma scale of the MMPI-2 is highly correlated to the characteristics measured in the Histrionic, Narcissistic and Antisocial personality scales of the MCMI-II (C.C. #1 = .900). The descriptors of the Ma scale include the following: over activity, impulsiveness, restlessness, unrealistic self-appraisal, unqualified optimism, social gregariousness, deep seated resentment, agitation and proneness to aggressive outbursts. One can easily see that these descriptors include a combination of histrionics, narcissism and antisocialism.

Interpreting all four scales in combination, the first canonical correlation describes characteristics of a person who is outgoing, friendly, self-confident and charming to others, but harbours a strong need for attention, affection and a need to feel special. They are constantly seeking stimulation, are conspicuous, have a tendency toward over activity, and have an unrealistic appraisal of themselves. They express their feelings easily with intense, short-lived emotions with a tendency toward hostility. They have a tough, matter-of-fact view of the world and may have feelings of dissatisfaction concerning what they are getting out of life.

On the second canonical variate pair, Avoidant Personality Disorder, Self-Defeating Personality Disorder, and Schizotypal Personality Disorder from the MCMI-II (i.e., Set#1 of CanVar2), was highly correlated with the K Scale (e.g., a validity scale tapping defensiveness) and Si (Social Introversion) scale from the MMPI-2 (i.e., Set#2 of CanVar2). Thus, in this second canonical correlation for the PPCS group, Set#1 (Avoidant, Self-Defeating and Schizotypal scales of the MCMI-II) was correlated .884 with Set#2 (K and Si of the MMPI-2).

Disregarding the direction of the loadings for the moment, all five scales are measuring a similar set of attributes. The commonality of the five scales revolves around social awkwardness, introversion, detachment from close relationships, being distant, cautious and rigid in their beliefs (i.e., overly conventional). The five scales differ in what dynamics are maintaining the individuals isolation and detachment. For example, the Schizotypal prefers solitude and is left alone by others due to their eccentricities and unusual thinking, whereas, the Avoidant Personality actually desires closeness and social relatedness. However, due to the high level of resentment caused by past hurts, this individual evades relationships to avoid further experiences of rejection. The Self-Defeating personality style find themselves alone and introverted because they relate in a self-sacrificing and martyr-like manner, placing themselves in situation where others take advantage of them and may abuse them. The Si and K scales of the MMPI-2 also include submissiveness to authority and inflexible, moralistic beliefs.

**Table 9: Canonical Correlation Loadings by Group**

MCMI-II/MMPI-2 Scales	PPCS Group		Moderate TBI Group		Neurological Controls	
	CanVar 1	CanVar 2	CanVar 1	CanVar 2	CanVar 1	CanVar 2
Schizoid	<b>-.265</b>	<b>-.588</b>	<b>-.202</b>	<b>-.633</b>	<b>-.410</b>	<b>.396</b>
Avoidant	<b>-.109</b>	<b>-.797</b>	<b>.142</b>	<b>-.860</b>	<b>-.770</b>	<b>.400</b>
Dependant	<b>-.160</b>	<b>-.307</b>	<b>.234</b>	<b>-.122</b>	<b>-.063</b>	<b>.207</b>
Histrionic	<b>.789</b>	<b>.259</b>	<b>.160</b>	<b>.239</b>	<b>-.142</b>	<b>-.829</b>
Narcissistic	<b>.689</b>	<b>.192</b>	<b>.455</b>	<b>.139</b>	<b>-.169</b>	<b>-.875</b>
Antisocial	<b>.732</b>	<b>-.110</b>	<b>.312</b>	<b>-.380</b>	<b>-.567</b>	<b>-.580</b>
Aggressive-Sadistic	<b>.416</b>	<b>-.165</b>	<b>.408</b>	<b>-.425</b>	<b>-.468</b>	<b>-.589</b>
Compulsive	<b>-.413</b>	<b>-.262</b>	<b>-.080</b>	<b>.076</b>	<b>.193</b>	<b>-.057</b>
Passive-Aggressive	<b>.628</b>	<b>-.562</b>	<b>.450</b>	<b>-.664</b>	<b>-.887</b>	<b>-.289</b>
Self Defeating	<b>.225</b>	<b>-.711</b>	<b>.083</b>	<b>-.702</b>	<b>-.781</b>	<b>.161</b>
Schizotypal	<b>.115</b>	<b>-.715</b>	<b>.237</b>	<b>-.657</b>	<b>-.687</b>	<b>.199</b>
Borderline	<b>.586</b>	<b>-.581</b>	<b>.286</b>	<b>-.645</b>	<b>-.914</b>	<b>-.151</b>
Paranoid	<b>.343</b>	<b>-.449</b>	<b>.556</b>	<b>-.403</b>	<b>-.480</b>	<b>-.687</b>
L (Lie)	<b>-.444</b>	<b>.087</b>	<b>-.306</b>	<b>-.052</b>	<b>.515</b>	<b>.029</b>
F (Frequency)	<b>.217</b>	<b>-.600</b>	<b>-.015</b>	<b>-.564</b>	<b>-.656</b>	<b>.028</b>
K (Defensiveness)	<b>-.437</b>	<b>.694</b>	<b>-.492</b>	<b>.743</b>	<b>.905</b>	<b>.183</b>
Hs (Hypochondriasis)	<b>-.213</b>	<b>-.068</b>	<b>-.612</b>	<b>-.491</b>	<b>-.329</b>	<b>-.027</b>
D (Depression)	<b>-.332</b>	<b>-.635</b>	<b>-.436</b>	<b>-.651</b>	<b>-.511</b>	<b>.469</b>
Hy (Hysteria)	<b>-.199</b>	<b>.100</b>	<b>-.771</b>	<b>-.416</b>	<b>-.211</b>	<b>.096</b>
Pd (Rebelliousness)	<b>.505</b>	<b>-.283</b>	<b>-.381</b>	<b>-.092</b>	<b>-.418</b>	<b>.115</b>
Mf (masculine/feminine)	<b>-.104</b>	<b>-.037</b>	<b>-.410</b>	<b>-.197</b>	<b>-.109</b>	<b>-.166</b>
Pa (Paranoia)	<b>.215</b>	<b>-.442</b>	<b>-.408</b>	<b>-.484</b>	<b>-.422</b>	<b>.140</b>
Pt (Anxiety)	<b>-.048</b>	<b>-.535</b>	<b>-.493</b>	<b>-.489</b>	<b>-.540</b>	<b>.396</b>
Sc (Bizarre Thinking)	<b>.209</b>	<b>-.491</b>	<b>-.454</b>	<b>-.517</b>	<b>-.599</b>	<b>.104</b>
Ma (Hypomania)	<b>.712</b>	<b>-.067</b>	<b>-.155</b>	<b>.125</b>	<b>-.271</b>	<b>-.792</b>
Si (Social Introversion)	<b>-.453</b>	<b>-.823</b>	<b>.149</b>	<b>-.862</b>	<b>-.581</b>	<b>.662</b>

CanVar = Canonical Variate; C.C. = Canonical Correlation

PPCS: C.C.1 = .900 ( $p < .000$ ); C.C.2 = .884 ( $p < .000$ ).

Mod TBI: C.C.1 = .965 ( $p < .000$ ); C.C.2 = .930 ( $p < .000$ ).

Neuro: C.C.1 = .876 ( $p < .000$ ); C.C.2 = .862 ( $p < .000$ ).

bold print denotes the scales that have been considered to load on the variate.

Now examining the direction of the correlations, 4 out of 5 scales had a negative loading on the canonical variate. This would imply that *low* scores on these scales are related to the canonical variate, thus, a *lack of* social awkwardness, introversion and social isolation. The K scale had a positive loading, and, in the absence of psychological disturbance, can be a measure of ego-strength.

In summary, the first canonical correlation indicates that the Histrionic, Narcissistic and Antisocial scales from the MCMI-II are highly related to the Ma from the MMPI-2. Interpreting the canonical variate pair, the following characteristics are associated: outgoing, sociable and charming individual, with a strong need for attention, affection, along with a strong resentment toward what they feel they are getting from life. The second canonical correlation indicates that the Avoidant, Schizotypal and Self-Defeating scales of the MCMI-II are highly related to the K and Si scales of the MMPI-2. The scales in this canonical variate pair describe the following personality characteristics: a degree of ego-strength, the social relatedness, competitiveness, being self-indulgent, and harbouring resentment toward others. These mentioned qualities are found in the absence of social awkwardness, social avoidance and fear of rejection.

#### Moderate Head Injury Group

There were two significant canonical correlations within the Moderate TBI sample. The first canonical correlation equalled .965 ( $p < .000$ ), accounting for 93% of the variance. The second canonical correlation equalled .930 ( $p < .000$ ),

accounting for 87% of the variance of the second canonical variate pair. Examining Table 9 for CanVar 1, no scales from the MCMI-II, and, only one subscale, Hy, from the MMPI-2 loaded strongly on the first canonical variate. The Hs scale, also from the MMPI-2, was the next highest loading at .612 (e.g., 37% of the variance is shared with the canonical variate). Both subscales were negatively correlated with this variate. Low scores on Hy describe people who are not excessively worried about their physical symptoms, although they have a narrow range of interests, lack industriousness, are constricted and conforming and may appear content with an uneventful lifestyle. These descriptors have also been used to describe the behavioural characteristics of persons who have suffered some form of damage to the prefrontal area of the brain following an injury. This canonical correlation may be measuring non-personality disordered characteristics and revealing the amotivational aspects of the prefrontal injury in a subset of the moderate TBI group. There was no strong relationship between MCMI-II and MMPI-2 scales on this canonical correlation.

On the second canonical variate pair, Avoidant Personality Disorder and Self-Defeating Personality Disorder from the MCMI-II (i.e., Set#1 of CanVar 2), and the K and Si scales from the MMPI-2 (i.e., Set#2 of CanVar 2), were highly related. Thus, the canonical correlation of Set#1 (Avoidant and Self-Defeating from the MCMI-II) and Set#2 (K and Si) was .930. These four personality scales also loaded in the second canonical variate pair in the PPCS group. This finding provides further

support that the Si and K scales of the MMPI-2 are highly related to the Avoidant and Self-Defeating scales of the MCMI-II.

As described earlier, low scores on Avoidant, Self-Defeating and Si represent an absence of social isolation, introversion and social awkwardness. High scores on the K scale, in the absence of psychological disturbance, may indicate ego-strength. The second canonical variate pair, in both the PPCS and the Mod TBI group were exceptionally similar, indicating a relatedness of the particular scales on the MCMI-II and the MMPI-2, as well as a consistency of constructs across the two TBI groups.

In summary, for the moderate TBI group, the first canonical correlation did not represent a high degree of relatedness between the MCMI-II and the MMPI-2, in that only one scale, Hy, from the MMPI-2 loaded on the canonical variate. This canonical variate appears to have some features consistent with personality changes associated with damage to the prefrontal area of the brain. These characteristics would include the following: a narrow range of interests, lack of motivation and initiative, and contentment with an uneventful lifestyle. The second canonical correlation indicated that the Avoidant and Self-Defeating scales of the MCMI-II were highly related to the K and Si scales of the MMPI-2. The scales in this canonical variate pair describe the following personality characteristics: a degree of ego-strength, the social relatedness, competitiveness, being self-indulgent, and harbouring resentment toward others. These qualities are found in the absence of social awkwardness, social avoidance and fear of rejection. This second canonical variate pair was very similar to the second canonical variate pair within the PPCS group.

### General Neurological Group

There were two significant canonical correlations within the General Neurological Group. The first canonical correlation equalled .876 ( $p < .000$ ), accounting for 77% of the variance of the first canonical variate pair. The second canonical correlation equalled .862 ( $p < .000$ ), accounting for 74% of the variance of the second canonical variate pair. As can be seen in Table 9, the first canonical variate pair, the Borderline Personality Disorder, Passive-Aggressive Personality Disorder, Self-Defeating Personality Disorder, Avoidant Personality Disorder, Schizotypal Personality Disorder of the MCMI-II (i.e., Set#1 of CanVar 1), were highly correlated with the K scale (i.e., Set#2 of CanVar 1) of the MMPI-2. Thus, the canonical correlation of Set#1 from the MCMI-II (Borderline, Passive-Aggressive, Self-Defeating, and Avoidant) with Set#2 from the MMPI-2 (K) was .876.

All loadings from the MCMI-II were negative, thus indicating low scores on these subscales are associated together, and with high values of the abstract canonical variate. The K scale of the MMPI-2 had a positive loading, and as mentioned previously, is an indication of ego-strength. With this variate pair including low scores on 5 personality disorder scales, interpretation of this canonical variate pair characterizes the *absence* of any dysfunctional personality characteristics or disorders. Thus, a lack of personality disorders on the MCMI-II is related to psychological health and ego strength, as measured by the MMPI-2.

On the second canonical variate pair, Narcissistic Personality Disorder, Histrionic Personality Disorder, and Paranoid Personality Disorder from the MCMI-II

(i.e.g, Set#1 of CanVar 2) were highly correlated with Ma from the MMPI-2 (i.e., Set#2 of CanVar 2). Thus, the canonical correlation between Set#1 from the MCMI-II and Set#2 from the MMPI-2 was .862. All loadings were in the negative direction. This pattern of loadings again describes the absence of dysfunctional personality traits. The absence of flamboyant, gregarious, and extroverted personality traits found on the MCMI-II scales of Narcissism, Histrionics and Paranoid, is correlated with the reliable, dependable, lethargic, and humble personality traits, as measured by the MMPI-2.

In examining Table 9 for the overall relationship of the MCMI-II and the MMPI-2, there are a few scales that are consistently related, regardless of group membership. The Avoidant and Self-Defeating scales of the MCMI-II and the K scale of the MMPI-2 are represented in each of the three groups. As well, several more scales were represented in two of the three clinical groups. This included three scales from the MCMI-II (e.g., Histrionic, Narcissistic, and Schizotypal), and two scales from the MMPI-2 (e.g., Ma and Si). The MMPI-2 K, Ma, and Si scales appear to be highly related to some scales on the MCMI-II. The similarity of the canonical correlations across groups was unexpected. The first canonical correlation in the PPCS group is similar to the second canonical correlation of the Neuro Group, and, the second canonical correlation in the PPCS and the Mod TBI Group were strikingly similar. These findings provide preliminary support for the relationship between particular personality scales of the MMPI-2 and particular scales of the MCMI-II.

**Neuropsychological Variables:** What is the relationship between the MCMI-II and neuropsychological test performance?

Before the critical analyses were executed on the neuropsychological test data, the Fredrick's and 15-item Test were examined to eliminate those profiles that were suspect with respect to adequate motivation. Using the cut-off scores recommended by the Fredrick's authors, there were no test profiles that had a score below 43 and a CR X Slope ratio below  $-.0041$ . Only four subject in the entire sample obtained a score of less than 9 on the 15-Item test, 2 from the PPCS group, and one each from the Moderate TBI and Neurological group. Because these subjects did not have corresponding "suspect" scores on the Fredricks, they were not considered to be conclusive for malingering, and were therefore included in the analyses. Table 10 details the means and standard deviations on all of the tests used in the testing battery. A MANOVA was used to investigate overall group differences across some of the neuropsychological tests. Only those tests that were believed to be the most sensitive measures from the battery were included in the final analysis.

According to Reitan and Wolfson (1993), the most sensitive measures of brain injury from the HRB are the Halstead Impairment Index (HII), the Category Test, Trails B, the Sensory-Perceptual Exam and the Tactual Performance Test (TPT). In fact, Reitan and Wolfson claim that the Localization score claims to be the most sensitive aspect of the TPT. However, examining the group means on the Localization score, very little variability across groups was found. Therefore, the first (dominant hand) and second (non-dominant hand) trials were included in the

**Table 10: Means and Standard Deviations on Neuropsychological Tests in the PPCS, Moderate TBI and Neurological Groups**

Neuropsychological Tests	PPCS		Moderate TBI		Neurological Group	
	M	s.d.	M	s.d.	M	s.d.
<i>Category Test***</i>	47.88	27.73	43.89	26.30	54.85	27.96
<i>Tactual Performance Test-Dominant</i>	6.76	2.82	6.78	2.83	8.06	3.99
<i>Tactual Performance Test-NonDominant</i>	5.22	2.61	4.90	2.12	6.33	3.14
Tactual Performance Test-Both	3.35	2.33	3.30	1.78	4.16	2.65
Tactual Performance Test-Total***	15.37	6.79	15.02	5.93	18.33	7.79
Tactual Performance Test-Memory***^	7.45	1.46	7.86	1.30	7.41	1.66
Tactual Performance Test-Localization***^	4.27	2.31	4.75	2.04	4.01	2.30
Trails A	33.18	31.35	30.93	10.37	35.54	15.53
<i>Trails B**</i>	72.94	27.99	72.76	26.72	90.78	59.74
Speech-Sounds Perception***^	53.59	8.09	54.93	3.97	52.26	10.36
Seashore Rhythm Test***^	25.76	3.22	26.22	2.99	25.41	4.06
Finger Tapping-Dominant**^	47.19	8.91	47.65	8.30	44.95	9.16
Finger Tapping-NonDominant**^	42.64	8.82	42.57	9.19	41.07	7.53
Aphasia Screening Test**	.94	1.48	1.24	1.92	1.05	1.48
Spatial Relations Score**	.59	1.04	.33	.82	.76	1.56
<i>Sensory-Perceptual Errors**</i>	11.29	14.25	6.17	6.67	9.07	8.56
Average - 1 Rank**	1.46	1.05	1.67	1.26	1.80	1.19
<i>Halstead Impairment Rating</i>	.35	.27	.31	.19	.43	.28
<i>Average Impairment Rating</i>	1.09	.55	0.99	.46	1.24	.63

\*\*\* = Test used in the calculation of the HII and the AIRS

\*\* = Test used in the calculation of the AIR

\* = Test used in the calculation of the HII

^ = Higher scores indicate better performance, otherwise, lower scores indicate better performance

*Italic print indicates tests used in statistical tests*

**Table 10 Cont'd: Means and Standard Deviations on Neuropsychological Tests in the PPCS, Moderate TBI and Neurological Groups**

Neuropsychological Tests	PPCS		Moderate TBI		Neurological Group	
	M	s.d.	M	s.d.	M	s.d.
WMS-R <sup>a</sup> Verbal Memory Index <sup>^</sup>	92.27	14.76	95.54	13.56	90.58	14.42
WMS-R Visual Memory Index <sup>^</sup>	99.98	16.84	103.07	16.07	97.18	17.14
<i>WMS-R General Memory Index<sup>^</sup></i>	94.01	16.37	97.61	15.84	91.05	16.67
WMS-R Attention/Concentration Index <sup>^</sup>	96.35	16.95	99.65	15.17	96.75	14.60
WMS-R Delayed Index <sup>^</sup>	93.16	16.51	96.37	17.39	90.47	15.97
<i>Consonant Trigram Test, Total Score<sup>^</sup></i>	42.59	7.19	43.57	6.64	40.52	7.81
Grip Strength-Dominant Hand <sup>^</sup>	35.92	15.73	38.45	10.87	34.77	12.76
Grip Strength-NonDominant Hand <sup>^</sup>	32.71	14.95	34.49	11.42	32.45	12.53
Grooved Pegboard-Dominant Hand	69.25	15.21	70.53	13.28	78.28	22.94
Grooved Pegboard-Nondominant Hand	75.53	15.91	77.60	26.92	83.83	26.52
15 Items <sup>^</sup>	13.85	2.34	13.95	1.68	13.49	1.95
Fredricks-Total Score <sup>^</sup>	78.83	9.98	81.64	11.03	80.14	10.11
Fredricks-Slope	-.00560	.00233	-.00498	.00257	-.00578	.0025
Fredricks-Consistency Ratio (CR)	.8291	.0965	.8362	.1518	.8474	.0816
Fredricks-Slope X CR	-.00449	.00210	-.00412	.00207	-.00456	.00224

<sup>a</sup> = Wechsler Memory Scale-Revised

<sup>^</sup> = Higher scores indicate better performance, otherwise, lower scores indicate better performance

*Italic print indicates tests used in statistical tests*

analysis. As well, the Average Impairment Rating (AIR) was included because it is a summary score of 12 neuropsychological variables from the extended HRB. As a measure of information processing and working memory, the Trigram Test total score was included, and the General Memory Index from the Wechsler Memory Scale-Revised was used as the measure for memory. Finally, given that age has been identified as affecting neuropsychological performance, and that the neurological control group was significantly older than the two TBI groups, age was used as a covariate.

As can be seen from Table 11, any group differences in neuropsychological test performance can be accounted for by the effects of age. With age being partialled out of the equation as a covariate, there were no group differences on neuropsychological test performance  $F(18, 414) = 1.32, p = .169$ . Simultaneous Multiple Regression was used to examine if neuropsychological test performance, specifically the performance on the 9 identified neuropsychological tests (i.e., the X variables), was related to the number of personality disorders (i.e., the Y variable), within each group separately. The frequency or number of personality disorders was not associated with neuropsychological test performance, in any of the three groups: for the PPCS group, R Square = .129,  $p = .298$ ; for the Moderate TBI group, R Square = .259,  $p = .263$ ; and for the Neurological Group, R Square = .073,  $p = .695$ .

**Table 11: MANOVA of Neuropsychological Test Scores Across Groups, Age as Covariate**

<i>Multivariate Test of Significance with (9, 207) D.F. Effect: Covariate Age</i>		
	<b>F</b>	<b>p</b>
Wilkes Lambda	6.50	.000
<i>Univariate Tests of Significance with (1, 215) D.F.</i>		
AIRS	42.60	.000
Category	47.75	.000
HII	38.09	.000
Sensory Perceptual	5.61	.019
TPT Dominant Hand	6.30	.013
TPT Non-Dominant Hand	16.60	.000
Trails B	16.39	.000
Trigram Total	1.45	.229
WMS-R General Memory Index	6.67	.010
<i>Multivariate Test of Significance with (18, 414) D.F. Effect: Group</i>		
Wilkes Lambda	1.32	.169
<i>Univariate Tests of Significance with (2, 215) D.F.</i>		
AIRS	1.23	.295
Category	.65	.522
HII	1.11	.331
Sensory Perceptual	3.15	.045
TPT Dominant Hand	1.77	.174
TPT Non-Dominant Hand	2.70	.070
Trails B	2.39	.094
Trigram Total	2.75	.066
WMS-R General Memory Index	2.20	.113

Although the PPCS group had more personality disorder scales elevated than the moderate TBI and Neurological groups, the number of MCMI-II scales with a BR  $\geq 75$  did not predict poor neuropsychological performance. There is presumably some degree of relatedness among the 9 neuropsychological measures included in the regression equation. This would be particularly the case for the two summary scores, HII and AIRS, as many of the individual test scores are included in the summary scores. Therefore, a stepwise regression analysis was then attempted, however, none of the 9 neuropsychological tests were included as relevant predictors. This was the case in all three clinical groups.

## DISCUSSION

### *Personality Disorders in PPCS*

The present study was an attempt to investigate the relationship between personality disorders and mild head injury with persistent post concussive symptoms (PPCS). Those that support the view that PPCS is a corollary of premorbid psycho-emotional issues would agree that maladaptive personality characteristics would play a key role in the manifestation of PPCS. More specifically, that poor adaptation skills and coping in the face of a significant life stressor would be the reason that this subgroup of mild head injury sufferers fails to make the "3-month recovery period". However, the effect of personality disorders had not been systematically measured in complicated mild head injury populations.

To date, research studies will often mention that personality and/or personality disorders are probable contributory factors in the maintenance of post concussive symptoms. However, the area of personality disorders has not been measured systematically, either by psychometric testing, or, by clinical interview. The present study attempted a more methodical measurement of personality disorders, using a measurement tool that is congruent with the psychologist's heuristic understanding of personality disorders, namely, the MCMI-II. It should be noted that the measurement of personality disorders on the MCMI-II is not thought to be an exhaustive resource for diagnosis. Nonetheless, the use of the MCMI-II is one of the first attempts at incorporating some form of standardized

measurement of maladaptive personality traits.

The data revealed that, compared to the neurological group, the mild head injury group with PPCS had generally higher scores on all but two of the personality disorder scales of the MCMI-II. The mild head injury group evidenced *significantly* higher scores on passive-aggressive, aggressive-sadistic, self-defeating and borderline personality disorder scales. In contrast, they scored significantly lower in the area of dependent personality traits.

Compared to the Moderate TBI group, the PPCS group did not demonstrate statistically higher scores on the MCMI-II scales, even though it was predicted that the PPCS group would score significantly higher than both the moderate TBI and neurological group. The reasoning here was that the moderate TBI and neurological group complain of PCS symptoms because they sustained a *real* neurological impairment, as opposed to the more *questionable* neurological impairment sustained in a milder injury. There was only a trend for the moderate TBI group to score more similarly to the neurological group than to the PPCS group, but many group scores also fell very much "in between". One reason why the moderate TBI group were not significantly different than the PPCS group might be the much smaller sample size (e.g., almost half). If the moderate TBI group contained numbers congruent with the other two groups, the trend may have been more clear.

What does this say about this group of mild head injured people who continue to report post concussive symptoms almost 2 years past the original

injury? It must be kept in mind that although the PPCS group scored higher on passive-aggressive, aggressive-sadistic, self-defeating and borderline scales, the overall BR scores were in sub-clinical ranges and not indicative of full personality disorders. Interpretation of sub-clinical elevations on these four scales suggests that this group maintains an internal struggle of competing virtues. On the one hand, they tend to lean on others for attention and security and want others to "feed" them with emotional well-being and support. On the other hand, they present as martyr-like, with passive-reliance on others, resentment towards those people they need and a general sense of antagonism. Along with this internal struggle comes moodiness, irritability, contrariness, depression, anger and possible aggressive outbursts.

Within the context of traumatic brain injury, which is so often accompanied by insurance investigation and/or litigation proceedings, this struggle between needing and resenting could also represent the real-life drama of TBI. In other words, their struggle could be one of "being heard" for what they believe to be true symptoms and problems and at the same time resenting the process of having to prove themselves to multiple physicians, psychologists, psychiatrists, insurance agents and lawyers. Unfortunately, litigation status was not consistently documented in the clinical files of the present subject sample and therefore, the influence of this variable in the present sample could not be examined. It could be reasonably assumed, given the motor vehicle insurance laws in Ontario, that all subjects who received their TBI through a car accident were in some form of

litigation, at least with insurance adjusters, and perhaps also with legal counsel. To fully test whether litigation status affects scores on the MCMI-II, one could use a control group of personal injury claimants who do not have a head injury, and compare their MCMI-II profiles to that of the PPCS group. This may delineate whether elevations on those 4 personality disorders is associated with having PPCS, or, being involved in antagonistic negotiations with lawyers and insurance agents.

Maybe the MCMI-II in a head injured population is not measuring the same thing as in other groups—because of their circumstances, they "read" the statements differently. The notion that neurological samples may respond differently to the content of a questionnaire compared to a psychiatric sample has been well researched with the MMPI-2. There have been some attempts at creating a neurological correction factor to control for the similarities between somatization symptoms and common neurological symptoms. The neurological correction factor utilized in the present study was modelled after Gass, (1991b), in which 14 items were clinically and statistically identified to discriminate between neurological samples and the normative group. By removing these "overlapping" items, it is assumed that artificial elevation of MMPI-2 T-scores is controlled. Gass (1991b) reported that endorsement of all 14 neurologically related items could increase clinical scales from 10 to 20 T-score points. Graphically, the neurologically corrected profiles are somewhat lower on the 5 MMPI-2 scales known to be high points in neurological profiles (see Figures 2, 3, 4, and 5). In

the present study however, the correction factor did not significantly reduce MMPI-2 profiles. One possible reason may be that the statistical procedure used to compare the two sets of scores was not sensitive enough to detect the changes. Alternatively, Gass's study did not apply the correction factor to his actual TBI sample. It is not known then how the correction factor affects the actual profiles--only the theoretical profiles. For example, there maybe the potential to lower the Hs scale by 12 T-score points, but how often this actually occurs on profiles given to neurological individuals has not been demonstrated. Nevertheless, proponents of the psychogenesis argument would suggest that the PPCS individuals would likely endorse as many post concussive symptoms as were available, as this is the reason these people have presumably failed to return to premorbid status. In the present study, there was no indication that the PPCS group over endorsed post concussive symptoms, because the neurological correction factor failed to reveal any meaningful differences in T-score points across the three groups.

Although the correction factor results presented by Gass were not replicated in the current project, the MMPI-2 profiles were very similar to that found in the majority of studies on neurological populations with the MMPI and MMPI-2. More specifically, Figure 1 shows that scales 1, 2, 3, 7, and 8 were clinically elevated when the total sample was examined. Interestingly, when the MMPI-2 profiles were looked at separately (see Figures 2, 3 and 4), the 5-scale high point profile was maintained for only the mild head injury group. Figures 3 and 4 demonstrate that the moderate head injury and neurological control group

had only scales 1, 2, and 3 elevated above a T-score of 65; scales 7 and 8 remained relative high points in the overall profile, however they were not elevated to the clinical range. The PPCS group would therefore appear to manifest more difficulties in the areas of anxiety, psychological turmoil and confusion than do groups with seemingly more serious neurological problems. Future research with general neurological samples should attend to the diagnoses making up the sample, in particular, the mild head injury subgroup with PPCS.

All three groups were approximately equal in the percentage of MMPI-2 profiles that were deemed invalid by reference to the guidelines made in the interpretive guide. An MMPI-2 profile can be deemed invalid if the subject has over-endorsed critical items, as seen in a T-score over 99 on the F scale. So the percentage of over-endorsers was equally represented in the three groups. However, the mild head injury group had half the number of normal profiles in their sample, as compared to the moderate TBI and the neurological control group. Thus, the mild head injury group were no more likely to invalidate the profile by over-reporting of symptoms, but, they were less likely to have a profile with none of the scales in the clinical range (e.g., T-score over 65). The observation that only the PPCS group had scales 7 and 8 clinically elevated may explain the lower percentage of normal profiles within this group compared to the moderate TBI and neurological groups.

In summary, the MCMI-II profiles indicate that the PPCS group endorse more passive-aggressive, aggressive-sadistic, self-defeating and borderline-like

traits compared to the Moderate TBI and Neurological groups. However, the overall scores on these four scales were not high enough to be categorized as "personality disordered". The MMPI-2 results indicate that the group with PPCS generally report more feelings of anxiety, rumination and psychological turmoil (e.g., scales 7 and 8) compared to the groups with seemingly more neurological damage. Those that believe PPCS is a function of psychogenesis could use this information to support the notion that it is emotional not physical damage causing their distress. Alternatively, the lack of evidence to support actual personality disorders suggests that the psychological differences found on MCMI-II and MMPI-2 profiles in the PPCS sample could just as easily be indicators of reasonable reactions to the TBI, rather than premorbid risk factors to PPCS.

### *Personality Disorders and Psychological Functioning*

Another goal in the present study was to examine the relationship between the presence of a personality disorder(s) and degree of disability in psychological well-being. The assumption was made that personality disorders found in the Mild TBI group with PPCS would be premorbid. It has been well documented that moderate and severe traumatic brain injury can alter personality functioning to various degrees. An individual's personality may change following a TBI due to lack of insight, lack of motivation, poor impulse control, intellectual and neuro-cognitive deficits, and caregivers often describe their loved ones as "he's not the same person any more". However, it is not often the case that a traumatic brain

injury can "cause" a Histrionic Personality Disorder, or that an injury to a certain part of the brain will cause Anti-Social Personality Disorder. Although maladaptive personality traits are often reported to be heightened following trauma to the brain, brain injury has not been documented as causing personality disorders, as described and diagnosed in the DSM-IV. Thus, any personality disorder that was identified on the MCMI-II were considered to be of a premorbid nature.

It was reasoned that individuals with a personality disorder would be likely to endorse a high frequency of problems on the MMPI-2. Thus, it was predicted that there would be a relationship between personality disorders on the MCMI-II and clinically elevated T-scores on the MMPI-2 within the mild head injury or PPCS group. If the notion of psychogenesis is correct, then the PPCS group are "not coping" due to their personality disorders, not due to actual brain injury problems. However, if the moderate head injury and the neurological group had some coping and stress-related problems as identified on the MMPI-2, then this would be a function of adjusting to a noted brain insult, as opposed to a personality disorder.

This hypothesis was examined using canonical correlations of the MCMI-II personality disorder scales and the MMPI-2 scales. Each group carried two significant canonical correlations. For the mild head injury group, the first canonical variate pair exhibited characteristics of being socially outgoing, gregarious, self-confident, with a tendency toward social manipulation, being

overly dramatic, and as harbouring resentment and some hostility to those who hold authority over them. In other words, a mix of histrionic, narcissistic and antisocial traits, coupled with some ruminations and anxiety. The second canonical variate pair related scales from the MCMI-II and the MMPI-2 that suggest an absence of social awkwardness, isolation and introversion.

These correlations of traits within the PPCS group could be consistent with the notion that PPCS individuals have difficulty coping with their sustained concussion due to maladaptive personality traits which makes change and adaption difficult for them. In addition, this mix of dysfunctional personality traits may explain why some of these individuals do not elicit the compassion and help they need from medical professionals and third party payers.

Across the two canonical correlations in the PPCS group, a total of six scales on the MCMI-II were differentially correlated with three scales on the MMPI-2. The correlations between the two different personality tests were strong, and, give support for overlapping content between the two tests. However, according to the psychogenesis argument, more scales on the MCMI-II would have been expected to be correlated with clinically elevated MMPI-2 scales.

The first canonical variate pair in the Moderate TBI group described this group as having a narrow range of interests, lacking industriousness, as well as being shy, inhibited, secluded by choice and overly conforming to persons who have authority over them. Interestingly, these descriptors are similar to those that can sometimes result from a pre-frontal injury.

Unexpectedly, the second canonical variate pair was almost identical to the second canonical variate pair found in the PPCS group. It was not predicted or expected that similar associations between the MCMI-II and MMPI-2 scales would occur across groups. This finding could also suggest similar underlying personality structures within the PPCS, the Moderate TBI, and the Neurological groups.

Two scales from the MCMI-II and 3 scales from the MMPI-2 were represented in the two canonical correlations for the Moderate TBI group. This is consistent with the hypothesis that the MCMI-II and MMPI-2 would not be closely associated in the Moderate TBI group, as compared to the PPCS group.

The neurological group had the most MCMI-II and MMPI-2 scales load on the two canonical variates, but the clinical scales had negative correlations, thereby noting the absence of these traits. In essence, the neurological group was the least emotionally and characterologically troubled group, who may present with good ego-strength if not somewhat overly conventional and compliant.

A total of eight scales from the MCMI-II and two scales from the MMPI-2 were represented in the two canonical correlations for the Neuro group. Contrary to the second hypothesis, there was a relatively high degree of association between the MCMI-II and the MMPI-2 in this group. The high degree of association was, in general, an indication of a lack of personality problems, indicated from both personality tests.

In general, there was some degree of overlap, or shared variance, between MCMI-II scales and MMPI-2 scales. There were two significant multivariate correlations found within each group, however, there were far fewer MMPI-2 scales than MCMI-II personality scales that loaded on each canonical variate. The K, Si, and Ma scales from the MMPI-2 were the most frequently related scales to the MCMI-II, and must somehow be tapping personality traits that are consistent with the characteristics measured on the personality disorder scales of the MCMI-II. Also of interest is the lack of concordance between the MCMI-II scales and the MMPI-2 scales, particularly in the case where scales from the two tests are supposedly measuring similar constructs. For example, the Hysteria scale from the MMPI-2 and the Histrionic scale of the MCMI-II were not correlated. Similarly, there was a lack of association between Pd and Antisocial Personality Disorder, or, Sc and Schizotypal Personality Disorder, or, Pa and Paranoid Personality Disorder. This lack of concordance also highlights that the MCMI-II is accounting for a considerable amount of variance across these three groups, over and above what is traditionally sought with the MMPI-2.

Qualitatively speaking then, the idea that the MCMI-II and MMPI-2 would be more correlated in the PPCS group compared to the other two clinical groups did not receive strong support. The MCMI-II results so far, do not support the hypotheses that there would be a higher degree of personality dysfunction in the PPCS group. Nor does the data support the idea that MCMI-II personality disorder scales would be associated with higher MMPI-2 profiles. Given that the

MCMI-II personality scales did not reach clinically significant levels, the hypothesis that personality disorder traits are more likely found in PPCS samples than other neuropsychological groups, was not found. The psychological profile of the PPCS group seems more out of reaction to a difficult, life-changing situation, rather than presence of premorbid psychiatric problems.

### *Personality Disorders and Neuropsychological Functioning*

The neuropsychological test data revealed some interesting results. With respect to personality disorders, regression analysis revealed that the number of personality disorders had little association with neuropsychological test scores. Thus, the number of characterological problems was not a factor in differentiating neuropsychological performance in these three groups. This finding must be interpreted with caution, because the inter-correlation among the neuropsychological tests included in the regression analysis may be masking any real effects. Alternatively, this finding is consistent with other studies that have found neuropsychological testing relatively resistant to the effects of emotional and personality dysfunctions (Bornstein, Miller & VanSchoor, 1988; Gass, 1991b; Novak, Daniel & Lowly, 1984; Reitan & Wolfson, 1997).

Examining the neuropsychological performance across the three groups, age was identified as a significant variable in accounting for group differences. Overall, the neurological group scored more poorly than the mild and moderate TBI groups, but these tests differences were a direct effect of age--the

neurological group was significantly older than the other two groups.

Interestingly, Reitan and Wolfson's (1996) take a strong position that although age (and education) can affect test scores within non-neurologically impaired subjects, it has no effect on neurologically impaired subjects. The present study included many tests from the Halstead-Reitan battery, and found age to be the most salient factor in accounting for variability in test scores across three neurological groups. Given that many neuropsychological tests are measured by the amount of time it takes to complete a task, and that response times decrease with age, it is not surprising that age can be a significant factor on test performance. The idea that age and education affect neuropsychological performance has been identified in other studies as well (Fenton et al., 1993; Heaton, Grant & Matthews, 1991; Gass, 1991b).

Perhaps the most important finding is that there was no difference in neuropsychological performance between the mild and moderate TBI groups. In fact, looking at the group means in Table 10, the PPCS group often scored somewhat worse than the moderate TBI group. There is the possibility that the neuropsychological tests used in the current battery was not sensitive enough to detect differences between the mild and moderate TBI groups. Inclusion of a supra-span list learning task and/or a complex information processing task such as the Paced Auditory Serial Addition Test (PASAT), may have been more relevant to the current TBI samples. Alternatively, the finding that the PPCS group scored more similar to the Moderate TBI group could suggest that this PPCS group has

suffered a more serious injury, resulting in neuro-cognitive deficits more similar to what is measured in a moderate TBI. Moreover, this lack of difference between PPCS and Moderate TBI demonstrates, at least in this sample, that differentiating between mild and moderate TBI is not a black and white issue. As it stands now, the classification of severity of injury is given before neuro-cognitive damage is assessed. As suggested earlier, severity of traumatic brain injury should include a neuro-cognitive outcome measure.

In examining the group means (Table 10), very few scores would be considered to be below normal or within a "brain-injury" range. The results also vary depending on what scoring method one chooses. Using those tests included in the Halstead Impairment Rating, only the TPT localization score, the finger tapping speed (dominant hand), and the HII itself just made the cut-offs for mild impairment ranges within the PPCS group. Within the moderate TBI group, only the finger tapping and HII mean scores imply brain injury and again only just within the limits of mild. This does not make for strong support of neuropsychological impairments for either the mild or moderate group. The lack of neuropsychological performance differences between the TBI groups will be discussed shortly.

The Average Impairment Rating (Russell, Neuringer, & Goldstein, 1970) is an extension of the HII employing more of the tests used in the HRB and has extended the normative base from which scores are ranked. Using AIR criteria, only the TPT (non-dominant hand, both hands and localization) were indicative of

mild brain injury within both the mild and moderate TBI groups. The overall AIR would place both groups within normal ranges, considering that an AIR score greater than 1.55 indicates mild damage and the group means for the mild and moderate TBI groups were 1.09 and .99, respectively.

The HRB has been well criticized for the small size of its original clinical sample, for the size of the normative sample and the questionable nature of the subjects that made up the normative group (i.e., Lezak, 1995). Better norms have since been published, correcting for age, gender and education levels (Heaton, Grant & Matthews, 1991). Using group means as an individual profile, the tests scores were compared to the Heaton et al. (1991) normative base. Test scores in this program are transposed into T-scores with a mean of 50 and a standard deviation of 10. For the present purposes, a score that lies one standard deviation below the mean will be considered an indication of impaired performance.

According to the Heaton et al., (1991) norms, the following tests would fall at or below a T-score of 40 for the PPCS group: Category Test, TPT (dominant hand), TPT (total score), Trails A, Finger Tapping (nondominant hand) and Sensory Perceptual errors. For the moderate TBI group, TPT (dominant, nondominant, and total), and finger tapping (nondominant) tests had T-scores at or below 40.

Although slightly different results occur, depending on which reference group one uses for comparison, the test scores are mostly within non-impaired, or normal, ranges, or *just* fall into the "mild damage" category. Some might expect

the scores to have been generally lower and more indicative of brain damage, given the clinical groups. However, group means are not clinically useful when neuropsychological test data is being considered. A neuropsychological profile can only be interpreted within the context of the pattern of test scores, in relation to the individual's premorbid status and current functional level. By averaging test scores, individual profiles are lost and fluctuations within a particular profile, which is the most informative information, is obscured by group averages. For example, if a subject scored well on the Category Test but was very impaired on the TPT dominant hand score, this would be very useful information clinically. Take another subject who did very poorly on the Category test and had minimal difficulty on the TPT. This is another clinically relevant finding, which represents a different neuropsychological profile from the first subject. Yet, when these scores are averaged as a group score, both end up falling within normal ranges. Therefore, generalizations regarding the averaged group scores on neuropsychological tests should not be weighed too heavily.

To summarize the neuropsychological findings, the most important variable in accounting for group differences was age, not head injury severity. In addition, the mild and moderate TBI groups performed very similar to each other. One could argue that this particular "mild" TBI group was more impaired than what is usually seen in PPCS victims, or, that this "moderate" group was less impaired than what is typically seen. This possibility is unlikely however, considering that both samples have been well screened to eliminate most confounding variables.

Alternatively, there exists another possibility to explain why the PPCS and moderate TBI group achieved similar neuropsychological levels. It may be that those individuals who sustain a mild traumatic brain injury and complain of post concussive symptoms at one year post injury, have not suffered a mild injury, but a more serious injury that places them within a moderate TBI group from a neuro-cognitive standpoint. Perhaps this ten percent of definably mild head injuries are neurologically moderate head injuries.

In conclusion, the present study attempted to investigate one aspect of the psychogenesis argument of PPCS, namely, the presence of personality disorders, as measured by the MCMI-II. The results did reveal that the mild TBI, or PPCS group demonstrated higher scores on passive-aggressive, aggressive-sadistic, self-defeating and borderline characterological traits. Although higher scores on these scales were obtained compared to the moderate TBI and the general neurological group, overall scores were not in the clinically significant range. In fact, the highest mean base-rate score was at least 15 points below the cut-off to be suggestive as a personality disorder. Nonetheless, given that these traits were found to be more predominant in the PPCS group, there is the suggestion that maladaptive personality traits could possibly account for the persistence of symptoms.

Alternatively, endorsement of these items on the MCMI-II could represent the conflict involved in the medical and litigational processes. For example, if there was something truly wrong with someone, and the medical community

continues to inform him or her that nothing is wrong, this would likely cause some degree of confusion and frustration. Over time, it is not surprising that an individual might become a bit passive-aggressive, somewhat anti-establishment, resentful, moody and despondent with medical professionals and third-party agents. The MCMI-II is purported to measure longstanding personality disordered traits. In the present study the four "elevated" scales reached only sub-clinical levels. Perhaps these group differences are indicative of situational response styles as opposed to premorbid pathology.

### *Summary*

In review of the literature, there is a fair degree of acceptance that individuals who receive a concussion and/or sustain a mild head injury should recover within three months of the occurring injury. Although it is also accepted that 10 to 15 percent of individual's with a mild head injury will continue to present with ongoing symptoms, few research papers are willing to consider the possibility of enduring physiological causes. Examination of specific psycho-social and emotional causes usually fail to differentiate complicated mild TBI's from uncomplicated mild TBI's. Kay et al. (1992) have advanced a very detailed and believable functional theory of how recovery can "go wrong" in this 10 percent. There is a shortage of supportive evidence so far, despite the theory's ecological validity. Many discussions recount nonsignificant psychological and emotional causes in their study, but will consider personality factors as contributory, although

they had not specifically measured personality disorders. The present study was an attempt to measure personality disorders somewhat more formally, using the MCMI-II. The results did not provide support that the PPCS group are more likely to have maladaptive personality traits and/or disorders.

It was not too long ago that researchers and clinicians believed that actual damage to brain tissue was not likely in milder head traumas. The existence of post concussive symptoms was noted, but not attributed to physiological injuries, because of the seemingly inconsequential nature of the injury. It was with the advent of neuro-imaging techniques and large research grants that uncovered statistics to suggest that over 20% of mild traumatic brain injuries admitted to hospital emergency rooms show visible evidence of brain damage on CT scans (Stein et al., 1993), and over 80% of mild TBI's admitted to emergency rooms show positive MRI scans (Levin et al., 1987; Levin et al., 1992). Although the injuries are no longer detectable by three months post injury, the neuro-cognitive sequelae may remain problematic. For example, the PET scans identified damage in PPCS sufferers as long as one year after the initial injury (Ruff et al., 1994).

Support for physiological damage can also be seen in neuropsychological studies. Neuropsychological studies that differentiate between complicated and non-complicated mild TBI, or between PPCS and recovered Mild TBI, generally find neuropsychological deficits in the complicated or PPCS subgroup of the sample (Cattelani et al., 1996; Gronwall, 1986; Marsh & Smith, 1995; Leininger, Kreutz & Hill, 1991; Williams, Levin & Eisenberg, 1990; Yarnell & Rossie, 1988).

When studies fail to find differences between mild TBI and controls, it is often the case that the mild TBI group includes subjects who have made full recovery, thereby masking the 10 percent in the way of group averages.

There has been a general lack of support to suggest that failure to recover in this 10 percent is due to litigation (Fenton et al., 1992; Gfeller, Chibnall, & Duckro, 1994; Hugenholtz, Stuss, Stethem & Richard, 1988; Leininger, Gramling, Farrell, Kreutzer & Peck, 1990; Mendelson, 1982; Rutherford, Merrett & McDonald, 1978; Tarsh & Royston, 1985). Moreover, the evidence is not convincing to implicate any of the following factors in accounting for PPCS: dysfunctional families, child abuse, rape, general neuroticism, premorbid social or psychiatric histories, drug use, premorbid psychological complaints, premorbid activity levels or premorbid symptom discomfort (Fenton et al., 1993; Radanov et al; 1991; Robertson et al., 1994).

A more comprehensive study therefore, would include a large sampling of mild traumatic brain injuries and concussions, compare those who have recovered within the allotted time to those who have not, and control or monitor whiplash injuries from impact injuries. This study would also require the assistance of the physics of the accident that caused the injury. Measurements would need to include MRI and PET imaging in conjunction with neuropsychological assessment at the 3 and 12-month post injury marks. Measurement of personality functioning would include a comprehensive diagnostic assessment with a detailed psychiatric interview, psychometric tests and collateral interviews. Information on litigation

status and rehabilitation history should also be important variables to be measured and considered.

It is always difficult to assess and determine the amount of influence various factors may have on an individual's outcome, when each possible factor is investigated in seclusion of other relevant issues. Psychological, neurological and psychiatric variables need to be investigated simultaneously, in order to adequately investigate the relative causative roles of physiogenesis and psychogenesis of the Persistent Post Concussive Syndrome.

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