PERSISTENT SYMPTOMS AFTER A TRAUMATIC BRAIN INJURY AND THEIR RELATIONSHIP TO THE PSYCHOLOGICAL IMPACT OF THE TRAUMA AND TO PTSD.

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ABSTRACT

The post-concussive syndrome (PCS) is the term used to describe the persisting and troublesome symptoms and complaints after a traumatic brain injury. Uncertainty exists about the cause of these symptoms, but usually the early symptoms are held to have their origin in the physical brain damage while the late-onset and persisting ones are thought to be caused by psychological factors. Previous studies indicate that head injured persons complain of more symptoms when they retained consciousness during the injury and when the injury was caused by someone else, especially if it was an assault. Such findings suggest that not only are psychological factors pertinent to the outcome of a head injury but that factors relating to the subjective impact of the traumatic event causing the injury may play a role. Because post-traumatic stress disorder (PTSD) is also a possible outcome of such an injury, it seems possible, if not likely, to confuse the two disorders, especially since they also share many symptoms in common. This study was intended to clarify the prevalence of symptoms of PCS and PTSD in persons who had suffered a brain injury more than three months previously, and to examine how these symptoms interact and whether different aspects of the subjective impact predicted either PCS or PTSD. The study was in two sections, Part 1 examining the records of 195 subjects, and results supported previous research showing that mild head injured subjects and those who had been assaulted, experienced more symptoms than those with injuries of greater severity or head injuries from other causes. Part 2 was developed to investigate these findings more fully and used 18 subjects who were administered questionnaires on PCS and PTSD symptoms and on the psychological (subjective) impact of the head injury trauma. Results showed that there was a trend for the subjective impact of the trauma to be associated with PCS and there was a significant association between the subjective impact and PTSD. It appeared that PTSD symptoms were acting as a confounding factor for the diagnosis of PCS after a traumatic brain injury with a high subjective impact. Another finding of this study was that, although subjects with memory of the trauma generally experienced more PTSD symptoms than those with no memory of it, there were several subjects who could not remember the trauma but who had a high number of intrusive PTSD symptoms. This contradicts the usual view that PTSD and amnesia for the trauma are incompatible.

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INTRODUCTION.

Mild head injury (MHI) is defined in terms of- minimal neurological complications and minimal amnesia following the injury. Typically these quickly resolve, but over 10% of those with MHI experience prolonged problems. Often occurring for several years after a head injury, these troublesome symptoms referred to collectively as the *post-concussive syndrome* (PCS), include a number of emotional and physical problems as well as the commonly occurring memory and concentration problems. In persons with MHI, PCS is consistently associated with poor functional outcomes such as failure to return to work (Binder, 1986) and represents a considerable challenge to the health services (McMordie, 1988; Karol, 1989).

The origin of PCS is not well understood. While older age and previous head injury are clearly associated with it (Dikmen, Temkin & Armsdale, 1989), there is controversy about whether it is primarily due to brain damage, issues surrounding compensation, or psychological factors operating before, during and after the injury (Lishman, 1973). Recent research acknowledges that PCS has different presentations and is best understood by considering different causal and mediating factors (Jacobson,1995; Kay, Newman, Cavelli, Ezrachi & Resnick, 1992; Lishman, 1988). For instance, PCS has been shown to be more severe when consciousness was maintained during the injury (Cicerone & Kalmar, 1995) and when the person blames someone else for what happened (Rutherford, 1989). However, the extent to which the circumstances of the injury and the emotional reaction to it affect PCS remain unclear (Bryant & Harvey, 1995).

While PCS may be associated with an unresolved emotional reaction to the traumatic incident causing the head injury, such a reaction is more likely to be diagnosed as *Post-traumatic Stress Disorder* (PTSD) which is described as an emotional and cognitive reaction to an extreme stressor resulting in characteristic intrusive, avoidance and arousal symptoms (American Psychiatric Association: Diagnostic and statistical manual of mental disorders, 4th.Edition, 1994). Estimates of the prevalence of clinical PTSD after a mild head injury range from 0% to 10% and appear to depend on the methodology of the study such as how PTSD is defined and the type of population used (Mayou & Radanov, 1996; Middelboe, Anderson, Birket-Smith & Friis, 1992; Sbordone & Litter, 1995). Trauma characteristics that increase the risk of PTSD developing after a mild head injury include severity of injury, exposure to death, personal assault and the absence of amnesia for the incident (Bryant & Harvey,1996; Kay et al, 1992). However, head injury victims are rarely asked about their fear and horror during the incident, or their grief, anger and

altered perceptions of themselves and the world after the incident. Consequently, very little is known about whether these thoughts and feelings are associated with either PTSD or PCS. Considerable uncertainty remains about the emergence and phenomenology of PTSD that occurs after a head injury (Epstein & Ursano, 1994).

Although emotional reactions are associated with both PTSD and PCS, the relationship between the two is unclear. Most research refers to either PTSD or PCS and not to both, and it is often unclear whether PTSD symptoms are to be regarded as a separate entity or whether they are part of an overall post-concussive syndrome in the same way that anxiety and depression are. This diagnostic dilemma is added to by the commonality of symptoms between PTSD and PCS (McAllister, 1994). Uncertainty remains then about differentiating both the etiological factors for, and the presentation of, PTSD and PCS. One notable attempt to clarify this situation is the study of Middelboe et. al. (1992) which found that 50% of mild head injury subjects had post-concussive symptoms and 20% had PTSD symptoms after one year and that all symptoms were predicted by psychiatric morbidity shortly after the injury and by minimal amnesia.

It is assumed that MHI is more likely to be associated with PTSD than severe head injury because the length of coma and duration of amnesia following the injury is minimal (Kay et al., 1992; McAllister,1994). It is also possible that the brief disruption in consciousness associated with a MHI increases the likelihood of PCS because of a more negative immediate impact of the accident and a better memory of it.

The present study sought to examine the relationships between the memory and impact of the traumatic event, core PTSD and PCS symptoms. It was hypothesised that both PTSD and PCS symptoms would occur with greater frequency when the traumatic event was remembered more clearly, and when the psychological reaction to it was more extreme. While this implies that they would occur more frequently with a MHI, it does not disregard the fact that PTSD and PCS may occur with more severe head injuries and other types of cerebral insult where the circumstances of the trauma make an extreme reaction more likely, such as with assault cases.

As a background to this study, an overview of traumatic brain injury with an emphasis on MHI is provided in Chapter 1. Chapter 2 focuses specifically on factors that previous studies have associated with PCS and develops the hypothesis that emotional and cognitive factors associated with the traumatic event causing the head injury may partly underlie PCS. Chapter 3 discusses PTSD in the context of a head injury. Chapter 4 brings PTSD and PCS together and considers how they might be differentiated at both

the etiological level and in terms of how they present. The remaining four Chapters focus on the hypotheses, methodology, results and summary of this research.

CHAPTER 1:
AN OVERVIEW OF HEAD INJURY.

1.1..CHAPTER OVERVIEW

The empirical and theoretical base which this research utilises is defined more clearly in this chapter. The reality of the problem of persisting symptoms after MHI is given a clinical and social perspective for discussing hypotheses about the causes of such symptoms.

1.2 EPIDEMIOLOGY

1.2.1 Incidence

In the USA traumatic brain injury of all grades of severity has an incidence ranging from 152 per 100,000 to 300 per 100,000 (Katz & Alexander,1994; Morse & Montgomery, 1992). New Zealand figures are similar. The Accident Compensation Commission had 7182 head injuries registered in the year ending March 1991, this representing an annual incidence of about 205 per 100,000. The proportion of head injured that are classified as mild is generally agreed to fall between 72% and 82% (Kay et al., 1992; Kraus & Nourjah, 1989). This corresponds well with the reported hospital admission rate for MHI of 130 per 100,000 (Morse & Montgomery, 1992). Given that only an estimated 18% of the mild head injured are hospitalised, it seems that these figures underestimate the true incidence of MHI (Kay et al, 1992). Extrapolating from this and the figures above, a more correct estimate of MHI annual incidence would appear to be 720 per 100,000.

This high incidence is supported by self report studies of head injury prevalence in adolescents. Binder and Rattock (1989) and Segalowitz and Lawson (1995) observed that 20% to 35% of adolescents will report a head injury incident with up to a half having some degree of unconsciousness. Body (1995) observed that 41% of a New Zealand sample of 14 to 15 year olds reported a head injury in the previous three years. Even though the reliability of these figures cannot be taken for granted, they clearly suggest that a considerable proportion of the population will have experienced a mild

head injury and could therefore have experienced the various sequelae of MHI.

1.2.2 Age and gender factors

Goldstein and Levin (1995) indicate that head injury incidence is bimodal with peak occurrence between the ages of 15 and 24 and over the age of 70. They report an incidence of 550 per 100,000 for 15 to 19 year olds dropping to much lower levels in mid-life and rising again in the older age group with an incidence of 200 per 100,000 in 65 year olds. Traumatic brain injury is a leading cause of mortality and morbidity in young people and is the leading cause of death in those under the age 35 (Morse and Montgomery, 1992). These researchers report that

- 62% of head injuries occur in 10 to 29 year old age group
- the overall incidence of TBI is two to three times greater in males than in females but with MHI the gender ratio becomes more equal reflecting possibly the ubiquitous nature of the causes of MHI, many of which occur in domestic settings
- motor vehicle accidents (MVA's) account for most head injuries in the young and falls account for most in the elderly

1.2.3 Alcohol intoxication and head injury

Although studies relating blood alcohol concentration (BAC) to brain injury incidence and outcome are equivocal, it appears that about half those with a brain injury diagnosis have a BAC greater than the legal driving limit of 0.1% with a greater prevalence of a positive BAC in those with mild head injuries (Kraus & Sorenson, 1994).

1.2.4 Causes of mild head injury

A recent New Zealand study has reported that falls and MVA's are usually associated with severe head injuries whereas assaults, collisions and "other factors" usually result in mild head injuries (Yeates, 1997). When all grades of head injury severity were considered, this study showed a similar ratio of causes as is given in Table 1.1 for just MHI. Table 1.1 summarises the causes of MHI from data derived from two large population studies of Alves, Macciocchi and Barth (1993) and Kraus and Nourjah (1989). These studies show a similar trend and indicate that about a third to a half of mild head injuries are caused by motor vehicle accidents.

Table 1.1

Percentage distribution of the causes of mild head injury

	N	MVA's	Bicycle accidents	Assaults	Falls	Sport	Other causes
Alves, Macciocchi and Barth (1993)	587	53	2.7	13.3	17.5	8.0	5.5
Kraus and Nourjah (1989)	2435	36	6.0	14.0	23.0	6.0	14.0

1.3 CLASSIFICATION OF HEAD INJURY

MHI is regarded as being on a continuum of severity of disruption of brain function that runs from very mild to extremely severe (McAllister 1994). The categories of mild, moderate and severe are distinguished by the neurological status observed soon after the injury. The American Congress of Rehabilitation Medicine (1993) has developed an operational definition for MHI which has clear upper and lower limits.

Using this system MHI is defined as a traumatically induced disruption of brain function manifested by at least one of the following :

- any period of loss of consciousness (LOC)
- any loss of memory for events immediately before or after the accident
- any alteration in mental state at the time of the accident
- focal neurological deficits that may or may not persist

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

- an LOC of about 30 minutes or less
- after 30 minutes, an initial Glasgow Coma Scale (GCS) score of 13-15
- post-traumatic amnesia (PTA) not greater than 24 hours

Variations of these criteria are used by other researchers. PTA is more often set at an upper limit of one hour (Kraus & Nourjah, 1989; Teasdale & Jennett, 1974) and a LOC

of 20 minutes was used by Rimel, Giordani, Barth, Boll and Jane (1981). The reliability of these measures has been questioned and Binder and Rattock (1989) specifically advise against using single criteria for MHI. This is supported by several findings. Williams, Levin and Eisenberg (1990) found that the outcome of patients with a GCS of 13 to 15 plus focal brain lesions or depressed skull fracture was similar to the outcome for patients with a moderate brain injury. Also, PTA which is the time between the injury and the recovery of memory of day today events, is difficult to measure accurately when less than a day and one acceptable method is to use standardised quantitative methods with a criterion score that indicates the absence of the amnesia (Gronwall, 1989a). Using a continuous score of 75 on the Galveston Orientation Assessment Scale as a marker is one such procedure. Because PTA relates to lesions in the cerebral hemispheres and LOC relates to lesions in more axial structures, both are needed for reliable severity estimates (Katz & Alexander, 1994).

Other criteria of mild head injury used by researchers such as Morse and Montgomery (1992) are:

- absence of skull fracture
- hospitalisation less than two days (designed to eliminate subjects with injuries other than head injury)
- no neurological emergencies such as haematoma evacuation or dealing with cerebral hypoxia

These criteria imply that there will be no persisting difficulties (Katz & Alexander, 1994). Because this is not always the case, Gronwall (1989a) suggests using later outcomes as criteria so that a MHI is defined retrospectively by the absence of symptoms after several weeks and a severe head injury by the presence of persisting impairments after many months. This perspective is not the usual one however as it ignores the distinction between subjective and objective symptoms and the need to make clinical decisions about severity early after the injury.

MHI then is classified according to a set of observations made proximal to the injury. The American Congress of Rehabilitation Medicine (1993) also allows that more distal cognitive, physical and behavioural symptoms be taken as evidence of MHI when they cannot be accounted for by other physical causes or emotional states. With a MHI these

symptoms are expected to abate after several months. With moderate and severe head injuries by contrast, more significant degrees of disability are expected to persist which are more clearly associated with the physical brain damage.

The term *post-concussive syndrome* (PCS) is applied in the literature to all cases where the head injured person makes persistent complaints about any of a constellation of symptoms that continue beyond a point in time when they are expected to have resolved, and the term is not synonymous with MHI (McAllister, 1994). However the term PCS does imply some doubt about the objective basis and validity of the complaints and for this reason it is more commonly used in the context of MHI (McAllister,1994).

Using GCS and PTA criteria, Morse and Montgomery (1992) classify those with a GCS scale score of 9-12 and a PTA of 1-24 hours as having a moderate head injury and those with a GCS score of 8 or less and a PTA of more than a day as having a severe head injury.

1.4 OUTCOMES OF HEAD INJURY

1.4.1 Severe head injury

Severe head injury outcomes are well researched and there is little contention that they have an organic basis compounded by psychological reactions to the injury and the social conditions it sets in motion. There are many ways of categorising these outcomes but an examination of the research of Morse and Montgomery (1992), Prigatano (1987) and Slagle (1990), reveals common categories:

1. Cognitive deficits occur of the sort that would appear as deficits on neuropsychological tests and which fall into five classes (1) attention and concentration problems (2) memory and learning problems (3) executive function problems such as occur with planning and self-regulation (4) general intellectual deficits (5) other focal deficits such as with language and reasoning.

2. Affective disorders are common with depression arising more often from damage to the left frontal lobe and mania more from damage to the limbic area or left parietal lobe (Silver, Yudovsky & Hales, 1991).

3. Psychotic reactions occur in 2% to 3% of cases

4. Problems of lack of awareness of the injury and defective self-perception are common.

5. Personality change occurs which tends to have three presentations (1) catastrophic reaction (2) symptoms of apathy, indifference and inflexibility (3) aggressive, disinhibited and antisocial behavior.

Not all research supports the presence of these problems however. Godfrey and Knight (1992) for example, examining the self-report and the report of significant others, found no significant evidence of psychiatric or emotional problems in their 26 subjects and concluded that the main problems were psychosocial ones based largely on skill deficits such as instigating behavioural exchanges.

1.4.2 Mild head injury

Mild head injury is on a continuum with severe head injury and may be associated with the same problems except that they can occur with less frequency and intensity (McAllister, 1994). For example, in their review of depression after TBI, Silver et al (1991) report a range of 19% to 39% for depression after mild head injury and a range of 35% to 60% for depression after severe head injury. What seems salient about MHI is that there is a somewhat different profile of **chronic symptoms** (PCS) and that there is no correspondence between symptom complaints and indices of brain damage. Cicerone and Kalmar (1995) suggest that PCS is not just more common after MHI but that the degree of symptom reporting is inversely proportional to the LOC. What seems distinctly possible is that there is a greater clarity of experiencing and remembering the traumatic event associated with a minimal LOC. Some of these experiences and memories may underly the chronic symptoms or provide circumstances where their natural abatement is hindered. Indeed this is the point of this research and subsequent chapters will develop this theme.

MHI outcomes can be divided into structural changes and clinical features, and will be discussed accordingly below.

Structural changes in the brain after a mild head injury.

The mechanical trauma of a MHI is usually accompanied by diffuse axonal injury and its associated membrane depolarisation. These can, in principle, account for the memory and concentration problems as well as some behavioral problems after MHI. One proposal is that psychosocial stresses encountered when the head injured person attempts to resume normal activities aggravate an already compromised neurotransmitter system (Dixon, Taft & Hayes, 1993).

Focal damage due to primary damage (e.g. hematomas) and secondary damage (e.g.anoxia) are rarer after a MHI than after a severe TBI although 3% to 14% of persons are reported to have either neurological crises or gross intra-cranial lesions confirmed by magnetic resonance imaging (Kraus & Nourjah,1989; McAllister, 1994). There is evidence then that brain injury considered trivial on the basis of the degree of altered consciousness has demonstrable neuropathological effects similar to those occurring with more severe head injuries.

Clinical outcome of mild head injury.

McAllister (1994) distinguishes between cognitive and behavioral sequelae of MHI with cognitive sequelae defined as those defects indicated by neuropsychological tests. These are commonly in the information processing, attention and memory domains and are extremely prevalent in the first few months. Behavioral sequelae are described as those problems that the patient complains of and he reports that 80% to 100% of patients will complain of cognitive, somatic and affective symptoms for several months after a MHI but these commonly disappear.

While neuropsychological tests may correlate with structural damage, they do not correlate with symptom complaints after three months or so (Cicerone & Kalmar,1995). It is possible that psychological or motivational factors account for why the individual complains or this may be because the context of a neuropsychological assessment does not resemble the context of daily living. Nemeth (1996) has suggested that some

structural or functional damage to the brain can be best inferred from a less "variable-centred "approach to assessment. For instance, he found that by asking his subjects to tell and show him what their limitations were, two key cognitive deficits emerged: (1) impaired ability for simultaneous attention to, and processing of, two discrete concepts, and (2) a reduced capacity and speed for processing new information and committing it to working memory.

Table 1.2 summarises some research on MHI outcomes. Results confirm that abnormal neuropsychological test results and symptom complaints are very common early after a MHI but will persist in only a small proportion of persons.

There are a number of possible reasons for different research results. These concern the reliability and validity of the measures used and the internal validity of the research design and these need to be kept in mind when interpreting the results illustrated in Table 1.2. as well as when designing further research with MHI subjects.

(1) There is inconsistency as to whether subjects with a prior head injury, pre-injury psychological disturbance, or those proceeding with compensation issues are excluded. Failure to exclude these subjects can lead to spuriously high symptom estimates (Dikmen, McLean and Temkin, 1986).

(2) Kay et al. (1992) suggest symptom measures should include symptom severity as well as frequency dimensions since it seems to be the severity of the symptoms that distinguishes PCS complainants from normal subjects. Fox, Lees-Haley, Earnest and Dolezal-Wood (1995) demonstrated that certain PCS symptoms have a high prevalence in individuals seeking psychotherapy, which puts the specificity of post-concussive symptoms to mild head injury in doubt.

(3) A selection bias for subjects with ongoing problems and the attrition of those without problems is often not accounted for in the research design.

Table 1.2

Summary of outcome studies of Mild Head Injury

Author	Type of study	Neuropsychological test results	Subjective symptoms
Rimel, Barth, Giordani, Boll and Jane, (1981).	*	*	79% had symptoms at 3 months
Watson, Fenton, McClelland, Lumsden, Headley et al.,(1995).	Prospective study of 26 MHI subjects	*	50% had symptoms at 1 year and this correlated with neurophysiological criteria
Gerber and Schraa (1995).	Comparisons between 32 MHI subjects, 44 orthopedic and 22 uninjured controls	*	17% of MHI patients have multiple symptoms at 6 months but a significant number of both other groups do also
Alves Macciochi and Barth (1993).	Looks at prevalence of PCS in MHI subjects	*	10% have symptoms at 1 year with 2-6% having multiple symptoms
Dikmen, Temkin and Armsdale (1989).	Series of studies comparing MHI subjects and uninjured controls	MHI subjects have defects in attention and delayed recall at 3 months. No differences at 1 year	5-10% of MHI subjects have symptoms at 1 year.
Levin, Mattis, Ruff, Eisenberg, Marshall et al.,(1987).	57 MHI subjects compared with 56 uninjured controls	Memory, attention and information processing speed defects in MHI subjects normalise at 3 months	Symptoms.common in MHI subjects up to 3 months.
Leininger, Kreutzer and Hill (1991).	Compares mild and severe head injured for MMPI profiles	*	MHI subjects indicate greater distress levels
Leininger, Gramling, Farrell, Kreutzer and Peck (1990).	Compares PCS subjects and uninjured controls	PCS subjects perform significantly worse on 4 of the 8 tests at 22 months after the injury	*
Batchelor, Harvey and Bryant (1995).	Compares MHI and uninjured controls	MHI subjects worse on all conditions of the Stroop test confirming there is a focused attention deficit after a MHI.	*
Dikmen, Machamer, Winn and Temkin (1995).	Compares 436 head injured subjects of all grades of severity with 121 orthopedic controls	Subjects whose PTA exceeded 1 hour had significantly worse results notably in performance I.Q. tests.	*
Goldstein & Levin (1995).	Compares MHI and uninjured controls over age 50	MHI significantly worse in fluency, similarities and memory tests	*

Note * designates no results in this area

(4).The type of symptom measure used is of paramount importance. Gerber and Schraa (1995) distinguished between two types of symptom checklist, those where subjects volunteer what symptoms they have and those where they endorse items. In their study, very different symptom profiles were produced from these two methods, with endorsement methods indicating significantly more symptoms in head injured subjects, orthopedic controls and uninjured controls. All other studies reviewed used endorsement methods and so the validity of the results must be questioned.

(5) The type of injury may produce a different outcome. Leininger, et al., (1990) suggest that MVA subjects are more prone to acceleration and deceleration injuries which may lead to different outcomes from other injury types.

(6) The nature of the group which is being compared with the MHI or PCS group is of critical importance. The study by Rimel et al.(1981) used normative data thus making comparisons with studies using other types of control groups difficult. Levin et al., (1987) state that the most valid comparison criterion is the subject's pre-injury self but that this is rarely applied experimentally.

CHAPTER 2

THE POST-CONCUSSIVE SYNDROME (PCS)

2.1 STRUCTURE OF PCS

While there is some consistency across studies in the sorts of symptoms complained of, there is great variability in the frequency with which these symptoms are reported as illustrated in Table 2.1. The three studies cited show a wide variation in individual symptom prevalence with the study done at 6 months showing a larger proportion of subjects reporting each symptom than in the study done at 3 months. This calls into question the reliability of symptom endorsement procedures.

Table 2.1

Frequency in percentages of post-concussive symptoms in three studies

	Keshavan, Channabasavanna and Reddy (1981), 3 months after injury	Mittenberg, Tremont, Zielinsky, Fichera and Rayles (1996), 6 months after injury	Youngjohn, Burrows and Erdal (1995), 6 months or more after injury
Headache	47	86	75
Dizziness	30	50	28
Fatigue	37	82	13
Anxiety	28	58	20
Insomnia	27		57
Sensitivity to noise	30	67	
Concentration problems	8	80	35
Memory problems	8	80	83
Depression		56	56
Sensitivity to light		83	30
Blurred vision		75	19
Irritability	17	82	30
Tinnitus			83

Cicerone and Kalmar (1995) and Jacobson (1995) indicate that the concept of a uniform post-concussive *syndrome* is outmoded. There is strong evidence that symptoms appear in clusters which Cicerone and Kalmar (1995) propose have different etiologies and they recommend exploring the correlates of these clusters. They already note a relationship between clusters and both functional outcomes and treatment needs.

While the differentiation of clusters appears to be in its infancy, some work has already been done. Hinkeldy and Corrigan (1990) found two symptom clusters, (1) those related to the severity of the head injury and this includes slowness, poor concentration and attention problems and (2) those unrelated to severity but related to anxiety and this includes dizziness, irritability, headaches, fatigue and depression. Rutherford (1989) noted the consistent appearance of drowsiness, nausea, vomiting, blurred vision, dizziness and fatigue early after a MHI and he attributes this along with attention deficits directly to the injury. Lishman (1988) differentiates this early symptom cluster from later onset symptoms which he attributes to psychological factors. (Cicerone and Kalmar 1995) assessed 50 subjects at least 3 months after the injury and using a cluster analysis found five clusters which are shown in Table 2.2.

Table 2.2
Clusters of Post-concussive Symptoms

Cognitive cluster	*Affective cluster*
concentration problems	irritability
memory problems	low frustration tolerance
difficulty with decisions	anxiety
mental fatigue	depression
slowed thinking	feeling overwhelmed
Sensory cluster	*Somatic cluster*
sensitivity to noise and light	dizziness
	balance problems
Solitary symptoms	nausea
headache	visual problems
sleep disturbances	appetite changes

It is possible that some of these individual symptoms or symptom clusters are related to specific causes. For instance, the somatic and sensory cluster could be associated with otological damage or damage to the cervical nerves or muscles. Cicerone and Kalmar (1995) suggest that if specific etiological correlates of these symptom clusters can be found, then this may facilitate prediction of outcome and appropriate treatments.

One cluster of symptoms that several researchers have found following a head injury are those associated with post-traumatic stress disorder. Bryant and Harvey (1995) found these to occur early after a head injury, moreso in subjects with clearer memories of the accident. Gerber and Schraa (1995) found that a group of symptoms normally associated with post-traumatic stress disorder occurred with a similar frequency in both MHI and orthopedic subjects with symptoms of general fearfulness, nightmares, being easily startled and recurrent thoughts about the accident occurring in both groups up to 6 months after the injury.

2.2 CLARIFYING THE DIAGNOSIS OF PCS

PCS is diagnosed according to the symptoms which are presumed to be caused by the head injury in some way Both the structure of the symptom profile and the etiology are relevant to a valid diagnosis of PCS. A number of persons diagnosed as having PCS have only a few of, or a circumscribed cluster of, the typical PCS symptoms. One of the issues that previous research has generally failed to address has been whether such persons have a more singular cause of these symptoms. For instance, it might be that someone with headache, dizziness and tinnitus had only an otological disorder subsequent to the head injury. Or it may be that someone with a cluster of cognitive problems has only an underlying attentional defect. Along these lines, Nemeth (1996) has suggested that a qualitative approach to assessment is more likely to detect how one single symptom may give rise to subsequent problems. He suggests for instance, that in some cases post-trauma depression may give rise to another set of symptoms, or that frustration with memory and concentration problems may be the basis for other PCS problems. This issue raises the question of how PCS can be more clearly classified.

It is possible to conceptualise PCS as an umbrella term embracing all persisting problems after a head injury. This would encompass cerebral damage, otological and whiplash

problems, as well as symptoms of anxiety, depression, post-traumatic stress, and somatoform disorder. In contrast, it is possible to conceptualise PCS as a tentative classification awaiting clarification of its various component elements or concurrent disorders. Such a view would encourage the use of more valid assessment techniques with clear diagnostic parameters that measure not just organic damage but also the patient's belief system and symptom patterns. Integrative models take a multifactorial approach to PCS etiology and demand that PCS patients be assessed for a wide range of possible etiological factors which seems a formidable task. With integrative models, the distinction between etiological factors and presenting symptoms becomes blurred since the two form a cyclical process. For instance, the distress with which the patient may view the troublesome symptoms and the horror of the accident is both the result of prior events and the cause of later events.

Nevertheless, the need to distinguish between different contributing factors and different outcome patterns remains since this will affect treatment. For each person with PCS, questions need to be asked about how *their* symptoms originated, how they are maintained, and what they mean for that person (Dikmen & Levin, 1993; McAllister, 1994).

2.3 THE ETIOLOGY OF PCS

Previous research has often utilised three alternative perspectives for looking at PCS. This section summarises some findings from the perspectives of organic damage, compensation seeking, and contributing psychological factors and recent proposals that a multifactorial approach to PCS is more valid are discussed.

2.3.1.Evidence for an organic basis to PCS

There is considerable evidence that PCS may sometimes be based on organic damage either in the brain itself or in other structures in the head.

2.3.1 (i) Evidence that brain damage itself contributes PCS

There is general agreement on the nature and extent of brain damage in the weeks following a MHI and it is possible that although this damage normally subsides, it may persist in some persons and account for persisting symptoms. Studies using

neurophysiological measures suggest there is a relationship between PCS and altered brain function and structure:

- while EEG measures typically do not correlate with PCS symptoms (Binder, 1986) there is an association between left temporal recovery and psychological morbidity after one year (Watson et al., 1995).
- some studies show a minimal association between brainstem altered evoked potentials (BAEP's) and PCS (e.g. Montgomery, Fenton, McClelland, MacFlynn & Rutherford, 1991; Zasler 1993). But the "Belfast studies" cited in Jacobson (1995) showed that all those MHI subjects with symptoms continuing beyond 6 weeks also had abnormal BAEP's. However those subjects whose symptoms emerged later on usually had normal BAEP's. This suggests that a subgroup with brainstem damage and continuing symptoms can be differentiated from a subgroup with functional symptoms.
- the clearest evidence of a relationship between neurophysiological abnormality and PCS comes from studies of cerebral blood flow (Barnes, 1991). Neurological Single Photon Emission Computerised Tomography (NeuroSPECT) studies, which measure regional cerebral perfusion, have located abnormalities in the mesio-antero temporal and the orbital-frontal areas of MHI subjects with catastrophic emotional reactions to their injury and this ocurred alongside normal CT and MRI scan results (Varney, Bushnell, Nathan, Kahn, Roberts et.al., 1995).
- Goldstein and Levin (1995) discuss the possibility that the accumulation of beta A4 amyloid protein in the traumatised brains of older persons is associated with the intellectual decline sometimes seen in them.

Neuropsychological measures can supplement neurophysiological measures as evidence of brain damage, although the validity of these measures as evidence of a specific brain injury needs to be accounted for in each individual case. Caplan (1993) comments that neuropsychological test deficits can be just as easily accounted for by depression medication and medical illness as by brain injury. Table 1.2 illustrates that there is a small but significant proportion of MHI persons who demonstrate persisting deficits on neuropsychological tests. Although there is no correlation between neuropsychological test performance and PCS, there are some grounds for believing that problems with

information processing and attention may contribute to PCS:

- measures of information processing and attention are more valid measures of diffuse axonal injury than are other neuropsychological tests which tend to measure focal injury (Gentilini, Nichelli & Shoenhuber, 1989)
- the Stroop test can differentiate between PCS and non-PCS subjects (Bohnen, Twinstra & Jolles, 1992)
- information processing measures such as the Paced Auditory Serial Addition Test correlate with prevalence and rates of improvement of PCS (Gronwall, 1989b; Leininger et al., 1990)
- Gronwall (1989b) suggests that information processing deficits persist indefinitely after a MHI and this may manifest itself only when the individual endures biolgical stressors (e.g.alcohol ingestion or episodes of hypoxia) or psychosocial stressors.

2.3.1 (ii) Non cerebral organic causes of PCS

Whiplash and other forms of cervical injury can produce the same set of symptoms as those following a MHI. (Mayou & Radanov, 1996). The whiplash may occur in conjunction with the impact head injury or in association with rotational forces on the brain where there is no impact injury (Sweeney, 1992). Both these researchers claim that differential diagnosis of MHI and whiplash is essential since they can have the same origin, presentation and outcome but a different treatment is required.

Parker (1990) discusses the potential for trauma to the temporo-mandibular joint concomitant to the head injury , and this can cause a range of symptoms stemming from autonomic dysfunction and trigger point activity. Included here are tinnitus, headache and blurred vision, all of which are part of the PCS presentation. Sataloff and Speigel (1993) show how dizziness, tinnitus and hearing disturbances can be accounted for by otological damage caused by trauma to cranial nerves 7 and 8, middle ear injury and labyrinthine concussion that occured concomitant to the head injury.

There appears then to be a number of potential physical causes of the symptoms associated with PCS that may originate from non-cerebral injuries associated with the brain injury. Zasler (1993) provides an overview of these and he stresses an

interdisciplinary approach to their assessment and treatment.

2.3 2 PCS and seeking compensation

The view that PCS has its basis in conscious or unconscious symptom exaggeration with financial compensation as the main reinforcer was put forth most emphatically by Miller (1961) and termed by him as the "compensation neurosis". He believed that since MHI by definition did not lead to organically based problems, then those with symptoms must be exaggerating them. There is some evidence supporting this view. Youngjohn, Burrows and Erdal (1995) observed that symptoms similar to PCS are commonly complained of by personal injury litigants without a head injury and they reported that 72% of PCS subjects who were proceeding with litigation cases feigned a poor performance on neuropsychological testing. This position is challenged though by reports of persistent disability in the absence of litigation and compensation claims (Dikmen et al., 1995; Rimel et al., 1981) and in spite of claims settlement (Leininger et al.,1990).

Binder (1990) suggests looking for reinforcers of symptom expression other than financial compensation. He says that a diagnosis of PCS should be accompanied by an assessment of non-monetary reinforcers for that person such as unusual dependency needs, the need to play the sick role, and the need to nurture their suffering. Jacobson (1995) noted that the head injured person's family often influences compensation seeking and this may influence the expression of symptom complaints by the person.

The finding of Rimel et al. (1981) that mildly head-injured individuals of higher socioeconomic status are much more likely to resume employment over a three month period than were those of lower socioeconomic status is open to a number of interpretations. It is possible that motivational factors are present. It is also possible that higher socioeconomic individuals have greater cognitive potential to overcome the effects of the head injury.

Nevertheless, it seems reasonable to expect that some persons with a head injury will feign dysfunction and discomfort in order to obtain some reward and it is suggested that every head-injured person with a financial incentive to fake bad must be assessed for the possibility of malingering (Binder, 1990).

2.3.3 Psychological factors contributing to PCS

The proposal that psychological factors may underly PCS is supported by the fact that symptoms sometimes emerge several months after the accident and by the absence of a correlation between symptom complaints and objective indices of severity. In fact, complaints are more likely when the head injury is mild and is associated with minimal disruption of consciousness (Cicerone & Kalmar, 1995). Lishman (1988) provides a model of PCS where psychological factors are conceptualised as having an input at three levels, pre-trauma, peri-trauma and post-trauma. This framework will be used to discuss other research showing the effect of psychological factors on the occurrence of PCS.

2.3.3 (i) Pre-traumatic factors

These factors focus on the premorbid characteristics of the head injured person. It has been shown that being female, high estimates of neuroticism, having low social competence, low self-esteem, and poor social support, and having had a previous psychological disorder, will increase the likelihood of having PCS (Fenton, McClelland, Montgomery, McFlynn & Rutherford, 1993; Keshavan, Channabassavanna & Reddy, 1981) .

2.3.3 (ii) Peri-traumatic factors

These are the factors relevant to the time of the head injury event and they revolve around the circumstances and significance of the accident and the short term reactions to it. Although a number of investigators have proposed that these factors are related to later problems, there has been little systematic investigation of this area (Bryant & Harvey, 1995). Some research has been done though and this suggests that grieving for what was lost because of the trauma and the triggering of unresolved emotional issues from the past are significant responses after a head injury (Dikmen, Temkin & Armsdale, 1989; Haynes,1994; Kay et al., 1992).

The circumstances in which the accident happened, such as coming close to death or being victimised can also lead to fear, anger, and changes in self-concept (Lishman, 1988). For example, more frequent and more severe PCS has been associated with having received a head injury in an assault compared to other causes and because the PCS emerged later, this was more likely to be due to psychological factors although injuries to the left parietal lobe appear to predict later emotional difficulties also (Dunlop, Udvarhely, Stedem, O'Connor, Issacs et.al., 1991)

2.3.3 (iii) Post-traumatic factors

Research in this area has addressed firstly, the processes of coping with the injury and its resulting symptoms, and secondly, retrospective appraisals of the significance of the accident and the associated emotional reactions.

Studies on coping examine the situations, cognitive processes and behaviors underlying symptom expression and experience, such as how PCS complaints vary with daily stress levels (Gouvier, Cubic, Jones, Brantley & Cutlip, 1992). Other studies have found that there is a subset of persons who may be predisposed to misattributing random symptoms to the head injury and this includes those with an expectation of problems and those with chronic adversity before the accident (Binder, 1986; Jacobson, 1995). The fact that some psychological interventions can prevent and ameliorate PCS and the effectivenes of stress inoculation and cognitive restructuring of negative self-statements about the injury also suggests a role for psychological factors in mediating PCS (Mittenberg et al., 1996).

Other studies have shown an association between PCS and how the person appraises the injury event. For example, PCS has been found to vary with early fears, concerns and anxiety dreams about the accident, and with attributing blame for what happened to someone else (Adler, 1945; Lidvall, (1974, cited in Lishman, 1988); Rutherford, Merrett & McDonald, 1977).

While it appears that how the head injured person deals with the problematic symptoms is reasonably well researched, there is a need for more information on how the person

deals with the trauma of the accident and how this affects later symptoms. There seems to be a need for more research on how the person's reactions to the accident both at the time it occurred (peri-traumatic factors) and later on (post-traumatic factors) affect the development of later symptoms.

2.4 AN INTEGRATIVE MODEL OF PCS

The previous discussion has drawn a distinction between the organic, compensation based, and psychogenic views of PCS. Distinctions have also been drawn between pre-traumatic, peri-traumatic, and post-traumatic factors, and between appraisals of the person's symptoms and appraisals of the accident. Jacobson (1995) stresses the arbitrariness of such distinctions and proposes a model which integrates all of these factors.

> *The variability of patient responses to a mild head injury is more understandable and more predictable, if the multiple influences on post-concussional symptoms and their changing combinations over time are jointly onsidered. Organic forces contend with a rubric of psychological factors, including attention, anxiety (stress responses and personality trait), prior learning history, the meaning of the situation, blame, and environmental responses.* (Jacobson, 1995, p 685)

This is a cognitive-behavioral perspective emphasising that the person's beliefs and appraisals at any point in time will be a major controlling influence on symptom experience and expression. This is a broad view that recognises the influence of social factors, motivational and operant factors, stress response factors and cognitive factors and draws a parallel between PCS and other disorders with a recognised psychological basis such as somatoform disorders and chronic pain states.

One criticism of the Jacobson (1995) model might be that it seems to downplay the potential effect of objective organic causes on patient disability and discomfort. Some persons may have circumscribed symptoms with an entirely organic origin. In contrast, the model proposed by Kay et al. (1992) stresses the relevance of objective neurological and other organic factors to many cases of PCS, but these inputs are incorporated into feedback loops where it is the *subjective experience* of these disabilities and discomforts

that is crucial to later outcomes. For instance, pain causes inability to function, inability to function causes anxiety and depression, this maintains the pain, and so forth.

2.5 CONCLUSION

This Chapter has examined the structure and etiology of PCS. Recent research suggests that PCS has a variable cluster-based presentation with a multifactorial etiology. This integrative view demands an exacting assessment procedure to determine the context of early symptoms, organically based deficits such as in memory and attention, and psychosocial factors from which later symptoms develop. The circumstances of the trauma and the attitudes and emotional responses to it have been proposed as psychosocial factors associated with PCS by previous investigators but there has been little work done in this area.

One of the problems of investigating this area is the confounding role of Post-traumatic Stress Disorder (PTSD) which is the diagnostic category usually associated with severe emotional reactions to trauma. It seems to be of vital importance to know whether a person's complaints are due to injured brain tissue, symptom exaggeration, and feeling frustrated, angry or sad about all that has happened, or whether they are due to the physiological, cognitive and dissociative processes associated with PTSD. Chapter 3 discusses PTSD and its association with head injury and PCS.

CHAPTER 3:

POST-TRAUMATIC STRESS DISORDER

3.1 INTRODUCTION

The essential feature of PTSD is the development of characteristic symptoms following exposure to an extreme stressor. This involves both a *traumatic event* such as a threat of death and a *response* of fear, helplessness or horror (DSM-IV, 1994). Opinion is divided as to whether PTSD can occur after a concussion and much of the debate centres around the head injured person's perception and awareness of the traumatic event. The issue of how the conditions of Criterion A of PTSD can be met by a head injured person are discussed in this chapter. The relationship between head injury and PTSD is of interest in two ways. Firstly, persons who are not coping with a head injury after a traumatic event may need to be assessed for PTSD as a possible contaminant. Secondly, persons with PTSD after an accident involving physical injury may need to be assessed for an undisclosed head injury which could be affecting their coping efforts.

3.2 DIAGNOSIS OF PTSD

There are considerable problems with the differential diagnosis of PTSD from other disorders which it may co-occur with or be confused with. Green (1994) notes that in about 80% of cases, PTSD occurs alongside other psychiatric and psychosocial disturbances complicating its diagnosis and treatment such as depression, somatoform disorder, suicide attempts and relationship problems. PTSD is diagnosed according to six criteria which includes 17 core physical and psychological symptoms (DSM-IV, 1994) and which are described in Table 3.1.

Table 3.1

Outline of the Diagnostic Criteria for PTSD as given in DSM-I V, (1994)

Criterion A: The witnessing or experiencing of an extremely traumatic event and a response of fear, helplessness or horror.

Criterion B: The traumatic event is persistently experienced in one or more of the following ways:

(1) recurrent and intrusive distressing recollections of the event

(2) recurrent distressing dreams of the event

(3) acting or feeling as if the traumatic event were reoccuring

(4) intense psychological distress when exposed to aspects of the trauma

(5) intense physiological distress when exposed to aspects of the trauma

Criterion C: Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness as indicated by three or more of the following:

(1) efforts to avoid thoughts and feelings associated with the trauma

(2) efforts to avoid places, people and activities associated with the trauma

(3 inability to recall important aspects of the trauma

(4) markedly diminished interest in significant activities

(5) feeling detached or estranged from others

(6) restricted range of affect (e.g., unable to have loving feelings)

(7) sense of foreshortened future

Criterion D: Arousal symptoms: two or more of these: difficulty falling or staying asleep, irritability and anger outburts, difficulty concentrating, hypervigilance, exaggerated startle.

Criterion E: Duration of symptoms longer than one month.

Criterion F: Disturbed social and occupational functioning.

Other aspects of PTSD include the following.

3.2.1. Partial PTSD.

A large proportion of people exposed to trauma have been estimated to experience a partial form of PTSD where not all the DSM-IV (1994) criteria apply (Bryant & Harvey, 1995; Taylor & Koch, 1995). Often this takes the form of the person having intrusive symptoms without avoidance symptoms.

3.2.2 Acute stress Disorder.

DSM-IV (1994) uses a classification of Acute Stress Disorder for when a group of intrusive, avoidance, arousal and dissociative symptoms cause significant distress within one month of a traumatic event. Bryant and Harvey (1995) report that about a quarter of those exposed to a serious trauma will get symptoms of acute stress disorder but when symptoms start to occur more than one month after the trauma, a diagnosis of PTSD is made. Everly (1995) also makes a clear distinction between acute symptoms early after a trauma suggesting they are a normal occurrence, and persisting or delayed symptoms which are an abnormal occurrence and the diagnosis of PTSD should be reserved for these.

3.2.3 Secondary symptoms of PTSD

As well as having the core symptoms, PTSD may be accompanied by a number of secondary symptoms which are not specific to PTSD or a necessary part of the diagnosis (McFarlane, Atchison, Rafalowicz & Papay, 1994). These can occur after a head injury as part of the PCS thus providing a diagnostic dilemma in head injured persons. One possible approach to diagnosis may be to take the presence of the core symptoms as suggesting a diagnosis of PTSD at least, and the presence of secondary symptoms without core symptoms as suggesting a diagnosis of PCS. The implication from evidence cited in earlier chapters is that a diagnosis of concurrent PTSD and PCS requires both the presence of core PTSD symptoms and the demonstration that PCS symptoms had an independent basis. At the moment there does not appear to be any research which has utilised this methodology. Table 3.2 illustrates the secondary symptoms.

Table 3.2

Secondary Symptoms of PTSD

1. memory disturbances	10. need excessive sleep
2. concentration difficulties	11. difficulty staying asleep
3. word finding difficulties	12. blurred vision
4. problem solving difficulties	13. irritability and anger
5. fatigue	14. headaches
6. anxiety	15. dizziness
7. easily distractible	16. can't recall aspects of the event
8. depression	17. impulsiveness
9. reduced libido	

3.3 ETIOLOGICAL AND RISK FACTORS FOR PTSD

There are a number of risk factors for developing PTSD. These include the type and severity of the trauma, having a history of psychological disorder, the degree of perceived threat of death or injury during the trauma, and the type of social support and other community resources available (Frederikson, Chamberlain & Long, 1992). Frederikson et al., (1992) describe risk factors and etiological factors in terms of a time process model that differentiates pre-traumatic, peri-traumatic, and post-traumatic periods. Etiological factors are categorised as biological, behavioral, cognitive and psychosocial with recent research favouring an integration of all these factors as an explanation of PTSD (Frederikson et al., 1992).

An example of these integrative models is the two factor model of Everly (1995). The first factor involves neurological hypersensitivity due to chronic arousal of the limbic system and the hypothalamic-pituitary adrenal axis with fear memories being encoded in the amygdala. The second factor is psychological hypersensitivity based on declarative and semantic memories encoded in the hippocampus and focusing on a violation of the traumatised person's world-view of safety and security and sense of self. Approaches such as this embrace each of the biological, behavioral and cognitive theories described below.

3.3.1 Biological theories

Ver Allen and van Kammen (1990) review the biological models of PTSD and propose that PTSD is due to a process of change and damage at various brain sites such as the amygdala, the hypothalamic-pituitary-adrenal axis and the locus coeruleus (with the latter identified as a brain trauma center that integrates arousal, responsiveness and fear memories which may be ongoing as the victim remembers the traumatic event). Kolb (1987) presents a neuropsychological model which proposes that the locus coeruleus and medial hypothalamic nuclei are dissociated from cortical control so that the original perceptual, cognitive, affective and somatic clinical expressions of the trauma are re-activated.

3.3.2 Behavioral theories

Typical of the behavioral models is that of Kean, Fairbank, Caddell, Zimmering and Bender (1985) who suggest that PTSD is based on an increase in stimulus generalisation so that the individual cannot avoid noxious cues. Subsequent over-arousal from the wide array of sources is considered to elicit ongoing memories of the traumatic event.

3.3.3 Cognitive theories

Cognitive approaches focus on the perception and interpretation of the traumatic events and the meaning given to them. Creamer (1995) sees this as a two stage process. Firstly the trauma must be perceived as threatening and frightening at the time it is occuring. Secondly the later interpretation of it must result in a severe challenge to the existing beliefs, assumptions and self-structure (cognitive schemata) that the individual operates from. Cognitive models propose that information and images about the trauma continue to severely arouse the person in a cyclical intrusive/avoidance pattern until the person's cognitive schemata can comfortably assimilate them (e g. Horowitz, 1986). There are variants of this model and recent proposals are that a challenge to those schemas most related to the central needs of the individual, such as those concerning trust, security and a positive sense of oneself and the world, is most likely to cause PTSD (Janoff-Bulman, 1985; McCann & Pearlman, 1993)

While there is some support for biological models of PTSD through animal models and research in other areas of stress, the cognitive models of PTSD have little empirical

backing (Creamer, Pattison & Burgess, 1992). In response to this deficit, these investigators have provided some validity for cognitive models by showing that the cognitive processing of trauma information accounts for more variance in outcome than the objective aspects of the trauma. Their research enlarges on the work of Foa, Skeketee and Rothbaum (1989) who show that traditional behavioral models do not adequately account for all PTSD reactions and that concepts of *meaning* must be brought in to explain all outcome phenomena. Psychosocial models of PTSD enlarge on the cognitive models by emphasising that the meanings and appraisal given to the traumatic events can be modified by coping efforts and the social context in which the trauma occurs (Green, Wilson & Lindy, 1985).

3.4 CLARIFYING CRITERION A OF PTSD

DSM-IV (1994) defines this criterion as

> *the person has been exposed to a traumatic event in which both of the following were present: (1) the person witnessed or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the integrity of self or others, (2) the person's response involved intense fear, helplessness or horror* (DSM-IV, 1994, p 427)

Criterion A is etiologically based rather than symptom based. There are some difficulties with the operationalisation of this criterion and with its appropriateness for a diagnosis of PTSD. Kasle (1990) suggests that diagnostic criteria should not include etiological factors but should be based on clinical symptoms and the course of the disorder. This principle is applied in some cases such as where early child abuse is inferred from adult symptoms (Zlotnick, Zabrinsky, Shea, Costello, Begin, et al., 1996).

Another problem with Criterion A stems from the lack of clarity between objective and subjective aspects. Baum, O'Keefe and Davidson (1990) suggest differentiating the objective spatial and temporal traumatic event (stressor), from the subjective appraisal of this stressor and considering their respective impacts on the outcome independently. Green (1990) extends these distinctions and distinguishes between eight different aspects of the objective stressor saying that this is validated by these aspects each predicting different outcomes.

These eight types of objective stressor suggested by Green (1990) are:

- threat to one's life or bodily integrity
- severe physical harm or injury to self
- the injury or harm is intentional
- exposure to the grotesque
- violent or sudden loss of a loved one
- witnessing or learning of violence to a loved one
- learning of exposure to a noxious agent or condition
- causing death or severe harm to another

Ullman (1995) provides further validation of this approach by showing that stressors involving physical assault cause greater severity and frequency of all categories of PTSD symptoms than stressors that occur accidently.

3.5 CRITERION A AND BRAIN INJURY

The different perspectives on Criterion A are relevant to diagnosing stress responses and PTSD after a brain injury. Firstly, it is possible that different degrees and types of objective stressor and subjective response may predict different outcomes such as acute stress disorder, full PTSD, partial PTSD, or PCS. Secondly, while there is usually little doubt about the history of an objective stressor during a brain injury, there is generally doubt about the subjective experience of the stressor in head injuries that involve amnesia. Opinion seems to be divided into the following somewhat incompatible areas.

(1) Some consider that PTSD is not possible after a brain injury because the amnesic barrier surrounding a it prevents the registration of any trauma (e.g. Ruff, Wyllie and Tennant, 1993). This view sees that the proven subjective experience of a *trauma* is necessary for a diagnosis of PTSD regardless of the existence of later symptoms.

(2) Others see that where PTSD might occur, the stressor must be clearly in memory. Sbordone and Litter (1995) operationalise this as being able to remember to within 15 minutes of the event causing the head injury. Since a mild head injury is defined by its having a post-traumatic amnesia of less than one hour, this rules out in

principle most head injuries resulting in PTSD. Indeed, Sbordone and Litter (1995) found no cases of PTSD in their sample of mild head injured when they used this very stringent criterion of remembering a trauma.

(3) Others take the view that having a head injury with loss of consciousness or amnesia does not rule out getting PTSD. One explanation for this is that PTSD may be mediated by non-conscious as well as by conscious processes. Layton and Wardi-Zonna (1995) consider that retrograde and post-traumatic amnesia do not preclude PTSD occuring as they hypothesise it to be mediated by implicit memory processes rather than explicit or semantic processes. This view is supported by several case studies showing the co-occurrence of mild and severe head injury with PTSD (Horton, 1993; McMillan, 1991). Also in support of this view, Van der Volk and Fisler (1995) confirm that traumatic events are mediated by different neurological mechanisms than ordinary stressful events. They showed that representations of trauma have

- greater use of implicit memory processes
- less semantic representation
- greater sensori-motor, somatic and visceral representation
- less use of hippocampally based memory

(4) Even discounting the above argument, it can be argued that alterations in consciousness after a head injury do not preclude PTSD when the stressor falls *outside* the amnesic barrier which for a mild head injury is very short anyway. The events surrounding some types of head injury would make this quite possible. For example, a head injury may be preceded by horrifying events such as seeing an assailant approach or seeing a car accident unfold with sufficient time before the brain injury for consolidation of the memory to occur. Similarly, the scenario of "coming to" in the carnage of a road accident, an intensive care ward or in one's house after a mugging must be a reality for some people. Indeed, the conceptualisation of PTSD as derived from combat stress and disaster studies indicates that PTSD is as likely to occur from prolonged stressors as from acute ones (Breslau, 1990; Ver Allen & van Kammen, 1990). The thrust of cognitive theories of

PTSD is that the appraisal process is extended in time well beyond the moment of "trauma" (Green, 1994). An examination of the eight typologies of stressor suggested by Green (1990) shows that they are all potentially applicable to a mild head injury and that they all would seem to entail an appraisal process that ran beyond the moments just after the infliction of a head injury by days and weeks. In support of this position, Bryant (1996) discusses two cases of severe head injury which later developed PTSD. Two explanations were given to account for this. The first was that PTSD was due to pseudo-memories reconstructed from information about the traumatic event obtained vicariously after a return to consciousness and related to the person's emotional needs at the time (e.g. recognising the extent of their injuries or confronting the loss of their old identities). The second explanation is that the trauma may have been encoded via implicit memory processes at the time it occurred.

3.6 CONCLUSION

Head injuries often occur in a context where PTSD is quite likely to develop even disregarding the brain injury. Epstein and Ursano (1994) suggest for example, that PTSD is especially likely to develop because of the head injured person's reduced ability to deal with the changed self-perceptions and arousal states associated with a head injury. The circumstances of an accident involving a head injury provide a suitable setting then for both PTSD and PCS to develop.

CHAPTER 4

THE INTERACTION OF PTSD AND PCS

4.1 INTRODUCTION

Several studies indicate that PTSD symptoms and PCS symptoms sometimes co-occur after a head injury with typical PTSD symptoms being flashbacks and dreams about the accident, physiological reactivity on exposure to scenes of the accident, increased arousal and a disturbed sleep cycle (Rubenstein, 1993). Rates of PTSD symptoms in head injured patients have been found to be similar to those in orthopedic patients with up to 22% of head injury patients reporting avoidance symptoms after one year (Gerber & Schraa, 1995; Middelboe et al. 1992). As discussed in Section 2.2, it is possible in cases where PTSD is diagnosed in concurrence with PCS that the symptoms of "PCS" are really the secondary symptoms of PTSD, that is that all symptoms are due to PTSD. Davidoff, Labstien, Kessler and Mark (1988) reclassified all PCS subjects as having PTSD, this unusual perspective deriving from their definition of PTSD as the *accompanying emotional response to* a *trauma.* Their position ignores all other potential causes of PCS previously discussed.

Although the amnesic period does not appear to be a complete barrier to PTSD developing, it seems to be a partial barrier as the study of Bryant and Harvey (1995) indicated. With their sample of road accident victims, 27% of those who had a mild head injury (with some RA and PTA) exhibited PTSD symptoms and 42% of those without a head injury had PTSD symptoms in the first month. It was concluded that although intrusive and avoidance symptoms are less common in amnesic mild head injured subjects than in those without a head injury, they still occurred. A drawback in this study was that the exact content of intrusive symptoms and whether they were related to the accident itself or the period outside of the amnesia was not clarified.

4.2 DIFFERENTIATING PTSD FROM PCS

The *presentation* of the two disorders can be differentiated by the presence or absence of core PTSD symptoms. From the *etiological* perspective, the question remains as to whether there are any particular factors that are associated with the development of PTSD rather than PCS such as those concerning psychological impact.

4.2.1 Psychological impact

It can be argued on conceptual grounds that PTSD is more likely to occur with extreme traumas associated with horrifying and threatening events. PCS by itself may occur when the trauma is less severe but is accompanied by circumstances and thinking patterns that set chronic arousal and dysphoric states in motion. Everly (1995) suggests that full-blown PTSD may only occur after more extreme stressors and a more extreme reaction to them. He describes PTSD as a spectrum disorder where the characteristic neurologic and psychologic hypersensitivity starts to occur with more intense reactions. Therefore it is *more likely* that PTSD would occur when the victim's awareness was undisturbed although as previously argued, this is not always the case. Thoughts at the time of the trauma such as "I am going to get killed now" are likely to be more potent in evoking intense reactions than retrospective thoughts such as "I might have got killed".

With a mild head injury, it is likely that full-blown PTSD would be uncommon because most mild head injuries do not occur in terrifying or horrifying circumstances. Also, most involve some degree of amnesia which seems to reduce the likelihood of PTSD. What does seem possible is that some mild head injuries will occur in circumstances that engender ongoing anger, fear, feeling vulnerable, grievances, guilt, blame and self-doubt. That is, a sequence of cognitive and emotional processes may be set in motion which makes persisting symptoms (PCS) more likely. There is some support for this as PCS has been associated with styles of blame and early anxiety.

At this point however, there appears to be no empirical data to explain the connection between ongoing awareness after a mild head injury and increased PCS. It is possible that vivid memories of the incident mediate the chronic autonomic system hyperarousal that Jacobson (1995) believes drives the troublesome symptoms. One of the proposals of this research is that *there is* a connection between PCS and the circumstances of the

head injury, ongoing awareness, and reactions that involve guilt, blame anger and self-doubt and such-like. This research does not propose to *explain* the connection.

4.2.2 Assessing how the psychological impact of the trauma affects PTSD and PCS

The survey planned as part of this research required the use of an instrument for assessing those cognitive and emotional factors hypothesised to partly lead to PTSD and PCS. The development of this involved examining a number of factors that have been identified in previous research as conceptually or empirically associated with PTSD or PCS as either etiological agents or risk factors. Apart from categorising the *type* of traumatic event that occurred and a clear memory of events, all other factors considered focus on the meaning, interpretation and impact of the traumatic incident since that is the focus of this research. Most of the factors considered are related to PTSD and have been discussed in a general way earlier in the chapter.

In contrast, there are few cognitive factors found so far that associate PCS with the impact of the trauma causing the minor head injury. Kay, et al, (1992) have suggested that the trauma can have an effect in various ways. It may set off unresolved emotional issues from the past such as feeling vulnerable, unprotected or unresponded to when hurt, or unable to get retribution when wronged. Giving information about head injury and its effects can reduce the likelihood of PCS by altering expectations of problems to come (Dikmen & Levin, 1993). Rutherford (1989) has drawn a distinction between causal responsibility and culpability for the traumatic event. While both are related to who caused the injury, culpability implies a moral dimension and seems to be associated with a worse outcome. For instance, where an organisation or employer is held culpable, PCS has been shown to be more severe. Lishman (1988) discusses how the fear of future accidents, fear of early symptoms, and fear about longer term consequences can affect the severity of PCS. For Lishman (1988), the type of trauma, the meaning given to it, and the personal context in which it occurred are of great importance for later outcomes.

4.2.3 The centrality of meaning and context

The theme of *meaning* also runs constantly through the PTSD literature. Foa et al, (1989) suggest that meaning is a broad term that encompasses a range of "meaning words" such as threats, losses, and perceptions. Cognitive theories of PTSD state that the world no longer appears meaningful and comprehensible to victims of trauma and that the meaning attributed to an event is a powerful determinant of outcome (Janoff-Bulman, 1985; Peterson, Prout & Schwarz, 1995). Foa, et al., (1989) discuss meaning in terms of appraisal and attribution processes. Appraisal processes are those that attach meaning to a stressful event and seem to address the question "what is it?" Attribution processes are part of this appraisal and seem to ask causal questions like "why is it?"

Cognitive theories of PTSD emphasise that appraisal and attribution are done in a context of pre-held beliefs, assumptions, needs, previous experience, and ongoing agendas as well as the environmental context. Pilowski (1985) and Shalev, Schreiber and Galai (1993) have used vignettes to describe how this context determines the highly individualised reaction to a traumatic event. For instance, in the vignettes used by Pilowski (1985), the expectation of not being found when injured, or of being annhialated, and the imagery of having one's skull crushed, all lead to different idiosyncratic somatic reactions.

4.3 DEVELOPING A HEAD INJURY IMPACT SCALE

A number of appraisal and attribution factors to be explored in this study will be discussed alongside the objective characteristics of the stressor.

(i) Type and severity of stressor.

The objective stressor dimension is central to the diagnosis of PTSD and is the major criterion which distinguishes PTSD from other anxiety disorders. For objective indices of the trauma, other research has used measures such as the amount of time Vietnam veterans had engaged in certain types of combat and the amount of body surface burned (Roca, Spence & Munster, 1992). With head injured persons, questions can be asked about the number of previous head injuries and whether there are serious injuries other than the head injury. Categorisation of the type of incident into motor vehicle accidents,

falls, assaults, sporting accidents, and domestic or industrial accidents could also be regarded as a objective categorisation of context.

Having a previous head injury is regarded as a major predictor of PCS (Lishman, 1988). Having other injuries has predicted PTSD in some research (e.g. Blanchard, Hickling, Taylor Loos, Forneris & Jaccard, 1996; Shalev, Schneiber & Galai, 1993) but the finding is not unanimous (e.g. Bryant & Harvey, 1996). DSM-IV (1994) states that PTSD is worse when the trauma had a human cause and Kay et al. (1992) note that PTSD is more likely when the mild head injury occurred as part of a personal attack. PCS is also associated with incidents involving assault (Dunlop et al., 1991).

(ii) Experiencing the threat of severe injury or death to self or others.

This is a focal feature of the DSM-IV description of PTSD and has consistently been shown to be necessary for PTSD to occur after motor vehicle accidents and criminal assaults (Blanchard, et al., 1996; Davis & Friedman, 1985). Threat to one's life or safety involves other dimensions such as how much the persons sense of invulnerability was violated and how much control the person had over what was happening (Janoff-Bulman, 1985). Threat then is a multifaceted concept and the person should be assessed for the severity of threat experienced during the trauma as well as an ongoing sense of violated safety and helplessness. The incorporation of the helplessness paradigm into the victimisation paradigm still requires some validation (Foa et al., 1989) with the evidence to date suggesting that a sense of helplessness has only a minor role to play in PTSD development. Peterson and Seligman (1983) believe that helplessness is best operationalised as a general belief in the uncontrollability of future traumatic events and anxiety about how to cope with them.

(iii) Feeling out of control after the trauma

The anxiety implied by this has been associated with both PTSD (e.g. McCann & Pearlman, 1993) and PCS (McAllister, 1994).

(iv) The experience of fear during the traumatic incident.

This may be seen as the emotional counterpart of feeling threatened and would appear to be a necessary element for the classical conditioning processes which may underly some

PTSD symptoms. Fear of dying has been shown to be one of the four main predictors of PTSD after a motor vehicle accident (Blanchard, et al., 1996). Rossi and Cheek (1988) have presented an elaborate theory of state-dependent learning, behavior and memory where emotions, somatic states and cognitions experienced during a trauma can underly later psychosomatic responses when the appropriate states are re-encountered. Rossi and Cheek (1988) present their theory as an explanation for both PTSD and ongoing somatic and emotional reactions to trauma. Their theory implies that not only can external cues such as frightening sounds and sights become conditioned stimuli but also internal cues such as pain states and cognitions.

(v) Experience of horror during the traumatic incident.

This is also central to the DSM.IV concept of PTSD. Exposure to the grotesque has been shown to contribute to the severity of PTSD after air crashes, technological disasters and during Vietnam combat (Green, 1990).

(vi) Altered self-concept.

Cognitive theories of PTSD focus on the shattering of assumptions about oneself and the world. Janoff-Bulman (1985) says that severe trauma can induce a negative view of the self and only when a renewed positive sense of self emerges can healing be said to have occured. Head injured persons could be asked about whether they feel they have changed since the injury occurred and whether such change was good or bad.

(vii) Distress and care early after the accident

Lishman (1988) describes fear and anxieties about early symptoms as predictive of PCS and Dikmen and Levin (1993) found that reducing such anxieties with appropriate information reduced the likelihood of PCS occurring. The head injured person could be asked about early fears and whether early professional help was found to be adequate.

(viii) Losses occurring as a result of the trauma.

Haynes (1994) discusses grief after a head injury and his research indicates that head injured persons have a grief response similar to a bereaved person. The head injured person stands to lose life as it was known to them and could reasonably be asked about significant losses and the impact of these.

(ix) Blame for the trauma.

The style of attributing blame for what has happened has been shown to predict both PCS (Rutherford, 1989) and PTSD (Foa, et al.,1989). Those who blame themselves and assume some personal responsibility for future events adjust better after a trauma (Baum, O'Keefe & Davidson, 1990). Blame can be directed at another person or agency or at oneself. When directed at oneself, it can be attributed to either fixed characterological features (characterological self-blame) or to erratic and changeable behaviors (behavioral self-blame). Janoff-Bulman (1985) says that behavioral self-blame attributions are associated with the best outcome after a trauma and Jacobson (1995) says mild head injuries have a better outcome when behavioral self-blame is used.

(x) Anger and resentment about what has happened.

There is no documented association between these factors and either PTSD or PCS. It is reasonable to expect though that the ongoing autonomic hyperactivity associated with strong anger and resentment could facilitate symptom development. It is likely that anger and resentment would be associated with blaming another person or with claims for compensation.

(xi) Finding meaning in what happened.

Shalev et al. (1993) captured some interesting and pertinent information from victims of a terrorist attack by asking how the trauma affected the person most and whether they were able to rationalise the event. Although most of the assessment process so far asks questions related to *meaning*, head injured subjects could be asked a general question about how the head injury has affected them most and whether it has any personal significance for them. This would allow the subjects to give a response in their own words and not in terms dictated by the researcher.

(xii) Questions about remembering the trauma.

Previous Chapters have discussed evidence that both PTSD and PCS are more likely to occur when the amnesia and loss of consciousness during the trauma are minimal.

4.4 CONCLUSION

This chapter has considered some of the problems surrounding the questions "Has this person got PCS, PTSD or both?" and "What are the circumstances of the trauma causing the head injury and initial responses to it that may influence the development of either PTSD or PCS?" Previous research findings emphasise two factors as consistently associated with PCS and PTSD. Firstly, being aware of the traumatic event as it occurred and subsequently being able to remember it seems important although clinical lore allows that aspects of the trauma can be held as dissociated memories in the case of PTSD. Secondly, what the trauma *means* to the person seems to be critical.

CHAPTER 5

RESEARCH HYPOTHESES

5.1 SUMMARY OF PREVIOUS CHAPTERS

Previous Chapters have put PCS and PTSD into perspective as two possible outcomes of traumatic brain injury. Prior research supports the view that PCS has numerous underlying factors operating as an integrated set. Clinical PTSD appears to be an uncommon outcome of TBI although some core PTSD symptoms can occur in about 20% of head injured persons. PTSD seems likely to occur only as part of an extreme subjective response to a severe objective traumatic event and being aware of the trauma as it occurred, and later, seems an important part of this.

Both PCS and PTSD have been shown to be more frequent and more severe when the head injury is mild, when there was minimal amnesia and LOC at the time of the head injury, and when the injury occurred in an assault or because of someone else's actions. The common thread emerging from this is that the memory and psychological impact of the head injury incident/accident sometimes plays a role in the development of PCS and PTSD.

PTSD and PCS are accepted as occurring independently of each other after a head injury in that a person can have one without the other. Previous research suggests though that both become more likely when the psychological impact of the incident/accident becomes greater and there is uncertainty in the research literature about how to differentiate the two disorders conceptually and diagnostically in such cases. This is because persistent emotionally based symptoms after an acute trauma are usually regarded as some form of PTSD, and because PTSD and PCS share many common symptoms.

5.2 THE AIMS OF THIS RESEARCH

The main part of this study (Part 2) is an extension of previous research and of an earlier part of the study done by the researcher (Part 1) showing that MHI and having the head injury caused by an assault are related to a higher prevalence of PCS. These preliminary results suggest that minimal amnesia during the injury, better memory of the accident, and the psychological impact of the accident are related to PCS and PTSD symptoms. This research aims to investigate further the proposal that persistent complaints after a brain injury may be related to having been psychologically traumatised (i.e. reporting a high psychological impact) as much as to the physical brain injury. In Part 2 of this study, psychological impact is operationalised in terms of 12 dimensions which are measured separately and which have all been associated with PCS and/or PTSD previously. This study aims to clarify any relationships between these 12 dimensions and the total psychological impact score with PCS and PTSD measures.

It seems to be particularly relevant to know also which aspects of the psychological impact of a trauma are most likely to lead to PTSD and not PCS. This research aims to clarify this by examining which psychological impact dimensions are more strongly associated with each of the PCS and PTSD measures.

It is possible also that there are objective aspects of the injury or trauma that increase the likelihood of a more severe psychological impact One factor which may serve as an objective measure of trauma severity is having multiple injuries. It is of interest then to compare symptom prevalence in those with multiple injuries and those with only a head injury.

In summary then, the proposal underlying this research is that the high psychological impact of a head injury can mediate both PCS and PTSD. These two disorders are expected to both co-occur and interact under conditions of high psychological impact. The previously noted problem of classifying a head injured person as PTSD, PCS or both is an individual and clinical issue beyond the scope of this study. For the purpose of operationalising these classifications in this research however, separate measures of (1) PCS symptoms, (2) PTSD symptoms, and (3) full classification of PTSD, are used.

Below is a model illustrating a number of postulated variables which may underly PCS and/or PTSD and which is derived from the time-process model for PTSD of Frederikson et al., (1995) and the PCS model of Lishman (1988). It is the framework underlying the hypotheses for this current study although not all the factors investigated are illustrated.

Pre-trauma factors	Peri-trauma factors BRAIN INJURY EVENT	Post-trauma factors	Possible outcomes
Personality Prior brain damage Drug use Age Gender Psychological problems	Psychological impact factors: fear, threat to life, horror, awareness, loss of control Brain damage Other injuries	Psychological impact factors: anger, losses, blame, meaning, early anxiety, social support, coping skills	PCS PTSD symptoms Full PTSD Other disorders Adjustment

Figure 1

Model representing factors operating before, during, and after a brain injury.

5.3 RESEARCH HYPOTHESES

Two subject groups are utilised in order to examine these questions. The two parts of the study and their associated hypotheses are as follows.

5.3.1. PART 1

In this part, an existing data base of head injured subjects from the Massey University Psychology Clinic is used to provide information on (1) cause of the head injury (2) head injury severity and (3) the frequency of nine symptoms (anxiety, irritability, headaches, dizziness, eye, smell, taste and ear problems, and flashbacks). Eight of these symptoms are those of PCS and the other, flashbacks, is a core PTSD symptom.

Hypotheses used for Part 1 of this research are as follows:

Hypothesis 1

More subjects with a mild head injury will report PCS symptoms and flashbacks than subjects with a moderate/severe head injury.

Hypothesis 2

Subjects reporting flashbacks will report more PCS symptoms than subjects not reporting flashbacks.

Flashbacks are a core PTSD symptom (Criterion B) and this hypothesis is based on the proposal that core symptoms are likely to be accompanied by arousal symptoms (Criterion D) and secondary PTSD symptoms (McFarlane et al., 1994). Subjects with even a partial PTSD are likely to have more problem symptoms than subjects without PTSD symptoms because they are more likely to have associated anxiety, depression, irritability, dizziness and headaches.

Hypothesis 3

Head injuries resulting from a personal assault will be associated with more PCS symptoms than will head injuries received in other ways.

5.3.2. PART 2

Part 2 of the study involved administration of 4 questionnaires obtaining information about (1) the severity of the head injury, (2) the extent of other injuries, (3) the psychological impact and memory of the head injury accident which are seen to have an etiological or mediational role, and (4) outcome variables of PCS, the incidence of full PTSD, and the incidence of PTSD symptoms (partial PTSD). Part 2 of the study was an extension of Part 1 in that it sought to examine further the prevalence of PCS and PTSD symptoms after a brain injury and the relationships between the psychological impact of the injury and these symptoms.

Hypothesis 4

Brain injured subjects will frequently report symptoms of PTSD.

Previous Chapters have highlighted the uncertainty about the extent of PTSD symptoms after a TBI and recent findings appear to contradict prior viewpoints that PTSD symptoms were rare (e.g. Bryant, 1996; Bryant & Harvey, 1995,1996; Middelboe et al., 1992)

Hypothesis 5

Subjects with other injuries as well as a brain injury will report more PTSD and less PCS symptoms than subjects with a brain injury only.

While some previous research indicates that the extent of physical injury is a predictor of PTSD, there is no evidence that it predicts PCS. Psychological theories of PCS can in fact provide a rationale for minimal injuries predicting later symptom complaints because of such factors as (1) minimal public acknowledgement of the impact of the head injury and its early effects, and (2) the consequent unrealistic expectation of an early return to pre-injury competence and the straining of the head injured person's coping capacity (McAllister, 1994).

Hypothesis 6

Subjects who can remember the head injury event will report a higher total psychological impact.

This hypothesis examines whether subjects who remember the event as it was happening (and who therefore have negligible PTA or RA), will report a higher psychological impact than those who cannot remember it.

Hypothesis 7

Subjects with MHI will report more PCS and more PTSD symptoms, a higher psychological impact, and a clearer memory of the trauma than subjects with moderate/severe head injuries.

Previous research has shown that both PTSD symptoms and PCS are more common with MHI and the possibility that this is being mediated by a high psychological impact and by remembering the trauma is being examined in this hypothesis.

Hypothesis 8

Subjects who remember the head injury trauma, and subjects who rate the trauma as having a higher psychological impact on HIIQ dimensions, will report more PCS and PTSD symptoms.

Hypothesis 9

Subjects, when considered as a group, will endorse some psychological impact dimensions more strongly than others.

This proposes that subjects will perceive some impact dimensions as more salient to the head injury and their current psychological functioning than others. Because of their referral to the Massey Clinic, all 18 subjects can be regarded as having PCS at some level and an examination of how the group tended to endorse the HIIQ Likert scale impact dimensions may provide some insight into the functioning of persons with PCS.

Hypothesis 10

Subjects with a DSM-IV classification of PTSD will have higher psychological impact ratings than those not classified as having PTSD.

Since PTSD is conceptualised as originating from the psychological impact of a trauma, this hypothesis examines the construct validity of the DSM-IV measure as well as of PTSD itself. It is expected that subjects with PTSD would report higher impacts especially of those dimensions conceptually linked with PTSD such as fear, threat to life, and experience of horror.

CHAPTER 6
METHODOLOGY

6.1 SUBJECTS

Part 1.

From the original data base of 232 subjects at the Massey University Psychology Clinic, 195 had been assessed later than three months after the injury. Of these, 42 had a mild head injury, 37 had a moderate head injury, and 116 had a severe head injury. Since these subjects were patients at the Clinic, they did not represent a random sample of head injured persons but instead represented those head injured persons with persistent problems.

Part 2.

Eighteen brain injured patients seen more recently at the Clinic were asked to participate in an interview and to complete 4 questionnaires. In most cases, this was done in conjunction with other neuropsychological testing. While this research focuses essentially on persons whose brain injury was as a result of an impact injury, (i e. TBI), several subjects were included who had experienced a brain injury in a non-impact incident but in a way that was potentially traumatic. Characteristics of the subjects are given in Table 6.1.

Table 6.1

Characteristics of the subjects in Part 2

Subject	Severity	Age	Gender	Months since injury	Prior brain injury	Pre-injury psych, problem	Other injuries as well	Cause
1	severe	36	M	4	has had four	0	no	fall off horse
2	severe	28	F	13	0	0	no	MVA
3	severe	27	M	27	0	0	yes	MVA
4	mild	28	M	72	0	0	yes	MVA
5	mild	29	M	12	0	0	yes	MVA
6	mild	33	F	4	0	0	yes	sport
7	severe	49	F	12	0	0	yes	fall
8	severe	14	F	2	0	0	yes	neck injury
9	severe	65	M	2	0	0	yes	MVA
10	severe	35	M	36	0	0	yes	MVA
11	severe	41	F	120	0	0	yes	MVA
12	*	37	F	26	0	0	yes	electric shock
13	severe	17	F	9	0	0	yes	MVA
14	mod	17	M	3	0	0	no	MVA
15	*	23	F	26	0	0	no	asthma attack
16	*	41	M	*	0	0	no	solvent poison
17	severe	37	M	36	0	0	yes	MVA
18	severe	26	F	17	0	0	yes	MVA

* = no information available

6.2. MEASURES

6.2.1 Measures used in Part 1

Data from the subjects in Part 1 was obtained from the standard patient information forms and clinical interview used for Massey University Psychology Clinic clients. This provided information on head injury severity, cause of the head injury, the prevalence of nine PCS symptoms, and the prevalence of flashbacks.

6.2.2 Measures used in Part 2

Scales were used to assess (1) the psychological impact of the head injury (2) PTSD symptoms and full PTSD (3) PCS.

(1) The Head Injury Impact Questionnaire (HIIQ)

A questionnaire called the Head Injury Impact Questionnaire (HIIQ) was developed specifically for this study. The full questionnaire is included in Appendix A and includes qualitative questions and a quantitative part which will be outlined in Table 6.2. Eleven questions sought qualitative information about prior psychological treatment, drug use, injuries received other than a head injury, other peoples' injuries in the accident, what the head injury meant to them and how the accident has affected them most. This qualitative information was intended to provide insight into the subject's psychosocial status as well as the objective aspects of the trauma.

A further 21 items, each to be scored on a 5 point Likert scale, ask about the impact (e.g. fear, threat, losses, anger) that the head injury trauma had on the subject at the time it occurred and now. These 21 items provide measures of 12 *impact* dimensions and one measure of *memory for the accident.* Where there are several items that make up a measure, the scores from each item will be averaged. On the Likert scale, a score of 5 designates a high psychological impact and 1 designates a low impact except for items 16, 17, 18, and 21 where the scoring is reversed.

The HIIQ will be examined quantitatively in three different ways depending on which hypothesis is being tested.

(1) scores for each of the 12 dimensions can be added to provide a total psychological impact score (maximum score of 60)

(2) scores from the Likert scale for each dimension can be used as they are (e g. *feeling angry about it* on a 1-5 scale). Scores were used this way when examining Hypotheses 9 and 10.

(3) individual scores for each psychological dimension can be categorised as high impact or low impact so that the subject sample can be divided into high and low psychological impact groups for each dimension. Scores of 0, 1, and 2 will define a low impact and scores of 3, 4, and 5 will define a high impact. This allows comparison of high and low

impact groups on PCS and PTSD scores and the scoring is done in this way when examining Hypothesis 8.

For the *memory* dimension, a score of 1 represents inability to remember the head injury incident and scores of 2, 3, 4, and 5 represent the ability to remember it.

Table 6.2

Abbreviated item content corresponding to 12 psychological impact dimensions and memory measure on the HIIQ

Dimension	Item nos.	Abbreviated items
1) feeling angry about it	22	• anger at what happened
	23	• resentment at what happened
(2) blame someone else	19	• how much was someone else to blame
(3) was accident preventable	18	• could accident be prevented
	21	• can you prevent it happening again
(4) experience of loss	29	• how much have you lost from accident
(5) feeling life threatened	10	• was life in danger
(6) feeling out of control	11	• out of control during accident
	24	• how safe from similar accidents
	25	• how much in control of life now
(7) feeling scared at time	12	• how scared during the accident
(8) horror at the time	13	• anything gruesome at the accident
(9) unable to make sense of it	14	• was it hard to believe what was happening
	28	• do you feel the victim of a senseless act
(10) negative early effects	15	• how upset by effects of injury early on
	16	• how well cared for by medical people
	17	• how well cared for by family
(11) blaming self	20	• how much were you to blame for what happened
(12) self-change after accident	30	• how much change since accident
memory of head injury incident	26	• memory for what happened
	27	• memory for feelings/thoughts at time

(2) PTSD Measures

In order to measure the relationship between PTSD and the head injury impact, measures of PTSD symptom frequency and full PTSD are required. The Impact of Events Scale and the DSM-IV criteria for PTSD were used respectively for these purposes.

The Impact of Events Scale (IES)

This was developed by Horowitz, Wilner and Alvarez (1979) and is a 15 item questionnaire designed to assess the frequency of symptoms clinically associated with post traumatic stress syndromes. The IES permits assessment over time and has been used clinically and in research in cases of personal injury, combat stress, and bereavement (Schwarzwald, Solomon, Weisenberg & Mukilincer, 1987). The scale items were divided into intrusive and avoidance subgroups by Horowitz et al (1979) originally because of clinical experience and this division is supported by later research (e.g. Schwarzwald et al., 1987). These two subgroups have a correlation of 4.2 which Zilberg, Weiss and Horowitz (1982) say is small enough to infer some independence of the subgroups. This correlation reduces to 0.15 when the scale is administered early after a trauma, and Zilberg et al. (1982) say that this supports the idea that the person's fixation in either the intrusive or avoidance phases is the basis of pathological post-traumatic states.

High indices of sensitivity to change after therapy and the matching of high scores to other diagnostic markers of post-trauma stress syndromes support the validity of the IES (Horowitz et.al., 1979). The scale has a split half reliability of 0.86, a good test-retest reliability, and a high internal consistency of both the subscales and the entire scale (Horowitz et.al., 1979).

The IES has 15 questions, each scored on a 4 point scale of *not at all, rarely, sometimes* and *often*, which Horowitz et al. (1979) suggest should be scored 0, 1, 3, and 5 respectively. While this would generate a total score, it is not certain what the diagnostic and treatment implications of the scores might be (Schwarzwald et al., 1987). It may therefore be more appropriate to use the scale as an indicator of symptom frequency for dimensional comparisons with other variables and this is how this research will use it. Previous researchers investigating PTSD and PCS have used the IES in this way with

scores greater than 9 and 19 representing medium and high scores respectively on the intrusive section (Bryant & Harvey, 1995; Middelboe et al.,1992). The IES is included in Appendix C.

DSM-IV (1994) criteria for PTSD

Many of the currently available assessment and diagnostic instruments for PTSD derive from research on veterans (Vreven, Gudanowski, King & King 1995). However, the psychometric properties of diagnostic measures for civilian populations is being established and Kean, Wolf and Taylor (1987) state that there is a trend towards a structured battery of tests to enhance diagnostic accuracy through the collection of potentially convergent information. There are problems with reliability with the diagnosis of PTSD (Watson, Juba, Manifold, Kucala & Anderson, 1991) One study, (Malloy, Fairbanks & Kean, 1983), used behavioral, physiological and self-report measures to correctly identify 100% of PTSD cases. Watson et al., (1991) suggest that an acceptable instrument for diagnosing PTSD should have these characteristics: (1) contents reflecting contemporary DSM criteria (2) well documented and substantial reliability and concurrent validity against current DSM criteria (3) the presence of dichotomous statements on the presence/absence of the disorder and each of its DSM criteria (4) continuous measures of the severity and/or frequency of the disorder and each of its symptoms (5) simple enough to be administered by a trained subprofessional.

Watson et al., (1991) consider that no current instrument satisfies all of these criteria. One method, applicable to this research, that goes some way towards satisfying these criteria is to simply use the DSM criteria themselves, reworded in a way that head injured persons can easily understand. The cognitive deficits and emotional lability of some head injured persons means it is important to use tests that are simple to understand and score. The DSM-IV diagnostic protocol provides a clear statement of whether full PTSD is present or not, and in this research, it is used for this purpose. The amended scale is included in Appendix C. (It is acknowledged that in clinical practice the DSM-IV, (1994) protocol would not be used just as a series of items to be endorsed but rather as a format for the interview and for seeking clarification of the whole symptom profile on which the final diagnosis would be made. This issue of the validity of the diagnostic measure of PTSD used in this study will be addressed in the concluding chapter).

(3) PCS Measure

Researchers and clinicians describe PCS in terms of up to 23 typical symptoms, any number of which may be present after a head injury. However there is no instrument, checklist or assessment scale specifically for PCS that has established psychometric properties and is widely used. For this reason, a scale was developed for this study based largely on using those items on the Symptom Checklist 90 R (SCL-90-R) that represent PCS symptoms.

The SCL-90-R is a widely used instrument of self-reported general psychopathology. It is composed of 91 items describing symptoms most commonly identified by psychiatric and medical patients. Past research documents a variable factor structure of the SCL90 with different populations. One study (Rauter, Leonard & Swett, (1996) indicates that it has one large factor accounting for 42% to 70% of the variance. Rauter et.al., (1996) interpret recent research to indicate that the SCL-90 is particularly subject to extraneous influences and response bias. This is supported by the study of Woessner and Caplan (1995) on the use of the SCL90-R for mild and moderately head injured persons. They found a response bias where almost all symptoms were endorsed more than they would be for a normal population. In particular, those symptoms commonly associated with a head injury were endorsed more frequently and as being of greater severity. Woessler and Caplan (1995) conclude that care is needed when interpreting the SCL90-R in neurological patients.

Not only does the SCL90-R have different validity considerations in a head injured population but it also needs to be interpreted differently when used with PTSD patients. Weathers, Litz, Keane, Herman, Steinberg et.al., (1996), have successfully used SCL90 endorsement patterns for diagnosing combat-related PTSD, even though the scale does not include the typical clinical symptoms of PTSD. Care is needed then when interpreting research that matches the SCL90 with other PTSD instruments.

Subjects used in this research were given the SCL-90 as part of their overall head injury assessment programme *outside of this research*, but it was decided to use an altered scoring procedure of the scale for this study. Of the 91 items of the SCL-90-R, there are 37 that correspond to 17 of the typical post-concussive symptoms. In some cases

therefore, several SCL90-R items correspond to one post- concussive symptom. But there are six typical post-concussive symptoms that do not have corresponding items on the SCL-90. In the altered SCL-90 used in this study, only those items that correspond to post-concussive symptoms were scored and where several items correspond to one symptom, the mean of these item scores was used as the PCS symptom score. The decision on which SCL-90-R items correspond to PCS symptoms was made on a rational basis. For instance, the symptom of *fatigue* was considered to correspond to three items about feeling low in energy, feeling weak in parts of the body, and feeling that everything is an effort. It is acknowledged that some information may be lost in this process of combining items.

As well, six extra items were added to make up for those symptoms not accounted for on the original SCL90. There is then a total of 42 checklist items corresponding to 23 PCS symptoms. Subjects were asked how much discomfort each symptom had caused in the last month and had to answer on a scale of 0 to 4. Total scores for each subject could therefore range between 0 and 92. These scores could then be used as a dimensional measure of PCS severity for establishing relationships with other variables. The altered scale used in this research is called the PCS Measure and is illustrated in Appendix B along with Table B showing the items on the PCS Measure that correspond to each symptom. Table 6.3 below lists the 23 symptoms that comprise PCS and this has been developed from the work of other researchers cited in earlier chapters (e.g. Gerber & Schraa, 1995; McAllister, 1994).

Table 6.3 *PCS Symptoms*

headache	frustration	vision problems
dizziness	depression	hearing problems
eating problems	anxiety	tinnitus
nausea	difficulty with decisions	alcohol intolerance
sleeping problems	control of aggression	hypersensitivity to light
memory problems	sexual problems	hypersensitivity to noise
concentration problems	doing things slowly to ensure correctness	thinks something is wrong with mind
irritability		

6.3 PROCEDURE

6.3.1 Part 1

Part 1 of this study involved retrospective examination of the records of 232 head injured clients at the Massey Psychology Clinic. Using the computer database, the following factors were recorded for each subject: (1) head injury severity using PTA length as a criterion with one hour and 24 hours being the demarcation points for moderate and severe head injury respectively (2) presence or absence of each of 8 PCS symptoms and of flashbacks at least three months after the injury (3) cause of the accident. This provided information to test Hypotheses 1, 2 and 3. Data was recorded as percentages and testing the statistical significance of results was done using the large sample test for comparing binomial proportions.

6.3.2. Part 2

Part 2 of the study utilised 18 clients who were more recently referred to the Massey Clinic for investigation of the outcomes of a head injury. Each client agreed to complete the four questionnaires used in this part of the study in addition to their overall neuropsychological assessment at the clinic. The use of subjects who had mild head injuries, severe head injuries and in three cases brain injuries received from an asthma attack, solvent poisoning and asphyxiation, allowed comparisons to be made between these groups. While the major part of this research was quantitative, it was also intended to use some qualitative information from the interviews from Part 2. The intention of this was twofold, firstly to examine whether factors such as having prior head injuries and pre-injury psychological problems had any bearing on symptom patterns, and secondly to understand how PTSD, where it occurred, was influenced by situational and personality factors. Information gathered was unstructured and consisted of comments made and impressions gained in the interviews.

Hypotheses were tested by comparing groups of either MHI or moderate/severe TBI subjects, high/low psychological impact subjects, or other injury/no other injury subjects on PCS or PTSD status. Statistical significance was examined using the Mann-Whitney Rank Sum test..

6.4 ETHICAL CONSIDERATIONS

The researcher made every effort to ensure that research procedures met scientific standards of competency and were sensitive to the welfare and dignity of the participants.

For Part 1 of this study, the information used was some of that previously obtained in the routine clinical interview at the Massey Psychology Clinic. This information was stored under coded identities and was frequently used for research purposes by Massey staff and students. Clients had been made aware of, and agreed to, such use of data when enrolling at the Clinic.

With Part 2 of this study, 14 of the 18 participants had questionnaire material included as part of their routine neuropsychological programme
at the Massey Clinic. All subjects were verbally informed of the inclusion of extra assessment material and how it was to be used. For 4 of the 18 subjects, questionnaire material was posted to them after a personal request and explanation was made to them by the research Supervisor.

The New Zealand Psychological Society's Code of Ethics expresses several concerns about use of research subjects and these are considered here with respect to the subjects in Part 2.

(1) Informed consent covering what sort of questions were going to be asked, what was going to be done with the information, and the right to withdraw at any time, was discussed with each participant.

(2) Confidentiality was assured in that the information was to be held only by the researcher and Supervisor and was not accessible through computer or office files.

(3) Potential harm to the participant was minimised as each of them was concurrently seeing the Supervisor for other assessment and/or therapy.

CHAPTER 7

RESULTS

7.1 PART 1

The databased records of 195 subjects with traumatic brain injury were reviewed for prevalence of the symptoms of post-concussive syndrome and post-traumatic stress disorder. Information was limited to what had been already entered, and not all symptoms of these disorders had been recorded. With particular reference to PTSD, the absence of data on the period of retrograde amnesia (RA), made it impossible to determine whether or not the subjects recalled the accident. Nonetheless, it was expected that preliminary examination of what data was available would highlight areas for further investigation in Part 2 of this study.

Based on previous findings that MHI was more likely to be associated with symptoms of PCS, comparisons were made between groups of different TBI severity. Further, a comparison was made between groups with and without flashbacks for prevalence of PCS symptoms. This would clarify the degree to which the PCS and PTSD symptoms overlap. Finally, since assault has been associated with more PCS and PTSD symptoms previously, comparisons were made between a group with TBI due to assaults and a group with TBI due to other causes. Almost all subjects had experienced difficulty with memory in some way after their injury. This is a symptom of both PCS and PTSD.

Hypothesis 1

More subjects with MHI will report PCS symptoms and flashbacks than subjects with moderate/severe brain injury

Comparison of the significance of the difference between percentages of each group reporting symptoms was examined using z-scores derived from the large sample test for comparison of binomial proportions. Results as shown on Table 7.1 revealed a significant difference for *anxiety* ($z = 1.83$, $p < .05$) with the MHI group reporting higher levels. Further, there was a trend for the MHI group to more frequently report other symptoms, with the exception of smell and eye problems.

Table 7.1

Percentage of subjects with MHI and moderate/severe brain injury who have symptoms

	Mild *N=42*	Moderate/severe *N=153*
Irritability	64	55
Headaches	35	36
Anxiety	26	15*
Flashbacks	15	9
Dizziness	14	10
Ear problems	10	6
Eye problems	7	18
Smell problems	2	6
Taste problems	2	2

* = $p < 05$

Clearly, irritability (57%), headaches (35%), and anxiety (17%) stand out as the most commonly reported symptoms for all TBI severity groupings as shown in Figure 2. Ear, eye, taste and smell problems are less frequently reported symptoms associated with PCS. Flashbacks are also reported more (but not significantly) by the MHI group.

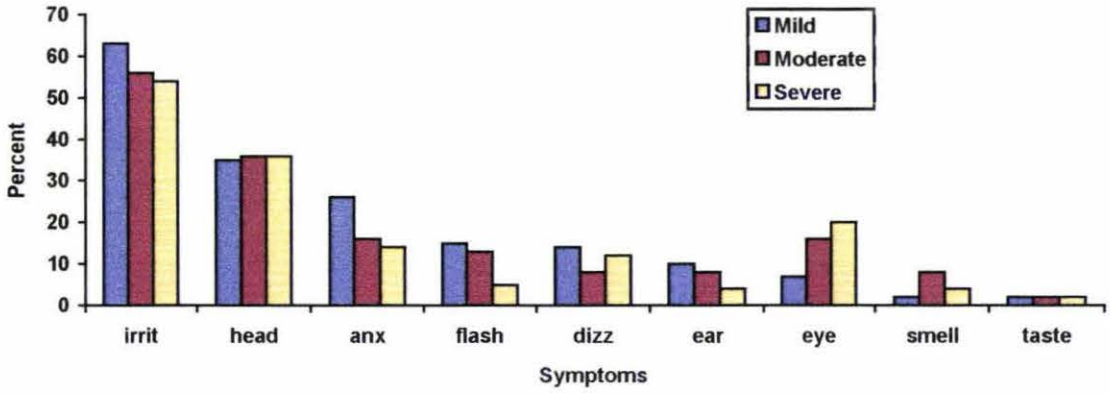

Figure 2

Percentage of mild, moderate and severely head injured subjects with symptoms.

Hypothesis 2

Subjects reporting flashbacks will report fewer PCS symptoms than subjects not reporting flashbacks.

The basis for this hypothesis was that "flashbacks", a core PTSD symptom usually only associated with PTSD, would be therefore less likely to be associated with PCS. If this was revealed to be the case, (ie. comparison between a group reporting flashbacks and another without flashbacks revealed more PCS symptomology in the latter), weight would be added to the argument that the two syndromes were separate. If the reverse occurred, (ie. PCS symptomology occurred more in a group with flashbacks compared to a group without), then weight would be added to the argument that PCS is really PTSD. No difference between the groups would suggest merely that there is a considerable overlap between the groups in terms of PCS symptomology.

The only core PTSD symptom about which information was available was "flashbacks" although anxiety, irritability, dizziness, depression and headaches are secondary PTSD symptoms. Again, comparison of the significance of difference between percentages of each group reporting symptoms was examined using z-scores derived from the large sample test for comparison of binomial proportions. As shown in Table 7.2 below, the group reporting flashbacks also reported more irritability and significantly more anxiety ($z = 7.2$, $p < .05$) and depression ($z = 3.8$, $p < .05$) than the group not reporting flashbacks suggesting that there is at least a considerable overlap between PCS and PTSD symptoms. Eye, ear, smell and taste problems however are much more associated with the PCS group.

Table 7.2

Percentage of subjects reporting and not reporting flashbacks who also reported other symptoms

	Flashbacks N = 18	No flashbacks N = 168
Depression	83	38*
Irritability	77	61
Anxiety	77	12*
Headaches	33	37
Dizziness	0	14
Ear problems	5	8
Eye problems	5	19
Smell problems	0	5
Taste problems	0	3

* = $p.<.05$

Hypothesis 3

Head injuries resulting from a personal assault will be associated with more PCS symptoms than will head injuries received in other ways.

As shown in Table 7.3, assault was associated with more symptoms than head injury from other causes and the difference between the groups was significant for dizziness *(z=2.3, p <.05)*, headache *(z=2.17, p <.05)*,and depression *(z=1.76, p <.05)*.

Table 7.3

Causes of head injury and percentage of subjects who complain of problems

	Assault *N=15*	Combined non-assault causes *N = 220*	Falls *N=41*	Collisions *N=27*	MVA's *N=110*
Irritability	78	62	60	68	62
Depression	65	42*	50	38	37
Headache	65	37*	43	41	31
Dizziness	34	13*	18	16	9
Anxiety	32	23	28	29	11

* = p < .0

Table 7.3 also shows that other than for irritability, there was a tendency for falls and collisions to be associated with more symptoms than MVA's.

In summary, Part 1 suggests that there is a considerable overlap between the PCS and PTSD syndromes in the co-existence of reports of flashbacks, irritability and headaches. PTSD is more associated with depression and anxiety, while PCS is more associated with sensory problems.

7.2 PART 2

In this part of the study, 18 subjects with TBI who had not been part of the database sample were asked specific questions arising partly from Part 1. They were given four questionnaires focusing on PCS (derived from the SCL90-R), psychological impact of the head injury event (the HIIQ), and PTSD (the Impact of Events Scale and the DSM IV criteria). These four measures are included in Appendices A-C.

Raw scores for these 18 subjects are shown in Table 7.4 below and scores on specific HIIQ dimensions are listed in Appendix D. Testing of hypotheses 4-9 was conducted using Mann-Whitney Rank Sum tests for comparison of non-parametric group means, and Spearman Rank correlation coefficients for examining relationships of continuous measures. Group N's differed for some measures as several subjects failed to complete parts of the questionnaires correctly.

The HIIQ *memory of trauma* scale used in Table 7.4 gives subjects' scores on a 1-5 scale and this was interpreted to mean that a score of 1 indicates no memory of the trauma and scores greater than 1 indicate some memory of it.

Table 7.4

Subjects' raw scores for PCS, IES, HIIQ total impact, and memory measures and PTSD classification

Subject and head injury severity	PCS measure	Memory of trauma (from HIIQ)	Total impact (from HIIQ)	IES score (PTSD symptoms)	PTSD class. (DSM.IV criteria)
1 severe	64	1	16	13	no
2 severe	55	2	20	30	no
3 severe	45	5	42	*	yes
4 mild	42	5	43	45	no
5 mild	38	5	29	23	no
6 mild	34	1	32	30	yes
7 severe	34	1	*	2	no
8 severe	34	3	41	15	yes
9 severe	32	1	24	21	yes
10 severe	30	1	25	0	no
11 severe	28	1	29	32	yes
12 hypoxia	28	5	29	6	no
13 severe	28	1	28	*	no
14 mod	26	1	31	16	no
15 hypoxia	26	1	40	16	no
16 hypoxia	23	3	30	2	no
17 severe	7	1	12	1	no
18 severe	2	1	26	7	no
Mean	32		29	16	
SD	14		9	13	

*= no score available

PCS SYMPTOMOLOGY

Specific comparison of PCS prevalence between Part 2, Part 1 and other studies is shown on Table 7.5. For the purpose of the table, Part 2 subjects who endorsed PCS items as either "not at all" or :a little bit", were considered to be relatively untroubled by the symptom, and those rating items "moderately", "quite a bit", or "extremely", were classified as experiencing the symptom. Subjects in all studies were at least three months post head injury. More information about symptomology and generally a higher prevalence of complaints, (notably concentration, fatigue, anxiety and dizziness), is reported in Part 2 than has been reported for other studies. The Part 2 subjects however, reported considerably less sensitivity to noise and tinnitus than other studies.

The three subjects with MHI obtained high PCS scores (ranked 4, 5 and 6 on Table 7.4). However, little else can be said of the MHI and PCS interrelationship due to the low numbers.

Table 7.5

Percentage of Part 2 subjects who report PCS symptoms and comparisons with results from other studies

	Part 2 subjects N=18	Part 1 subjects N=195	Middelboe et.al.,(1991) MHI subjects N=51	Youngjohn et al.,(1995) MHI subjects N=55	Keshavan et al., (1981) Mainly severe TBI N=60
Memory	89	100	25	83	8
Concentration	83	*	25	33	8
Having to do things slowly	72	*	*	*	*
Fatigue	66	*	21	13	36
Headache	61	35	32	75	46
Anxiety	61	17	18	20	28
Sleep problems	55	*	18	57	36
Irritability	55	57	21	30	16
Frustration	55	*	*	*	*
Difficulty with decisions	55	*	*	*	*
Feelings associated with depression	55	*	*	56	*
Dizziness	38	11	25	28	30
Feeling something is wrong with mind	33	*	*	*	*
Visual problems	22	15	18	19	3
Tinnitus	22	*	*	83	*
Light sensitivity	22	*	*	30	*
Alcohol intolerance	16	*	11	*	1
Problems with aggression	16	*	*	*	1
Noise sensitivity	11	*	*	*	30
Ear problems	5	7	7	*	1
Appetite problems	5	*	*	*	*
Nausea	5	*	*	*	*
Sexual problems	0	*	*	*	3

* = no results available

PTSD SYMPTOMOLOGY

Hypothesis 4

Part 2 brain injured subjects will frequently report symptoms of PTSD.

Prevalence of PTSD symptoms reported on the IES and using the DSM-IV criteria are outlined in Table 7.6. The IES items were developed so as to have an intrusive section (items 1,4,5,6,10,11,and 14) and an avoidance section (items 2,3,7,8,9,12 and 13). The DSM-IV criteria were divided into an intrusive symptom section (items 1-6), an avoidance symptom section (items 7-13) and an arousal symptom section (items 14-18).

Table 7.6

Number of symptoms of DSM-IV PTSD and scores on the IES for each subject.

		DSM-IV PTSD measure				IES		
Subject	Memory of trauma	Intrusive symptoms	Avoidance symptoms	Arousal symptoms	PTSD classif.	Intrusive symptoms	Avoidance symptoms	Total IES score
1	no	2	2	3	no	7	6	13
2	yes	2	2	4	no	12	18	30
3	yes	3	3	4	yes	-	-	-
4	yes	3*	2	0	no	21	24	45
5	yes	2	1	1	no	18	5	23
6	no	4*	3	4	yes	19	11	30
7	no	0	1	4	no	1	1	2
8	yes	2*	3	5	yes	9	6	15
9	no	1	4	3	yes	7	14	21
10	no	1	2	2	no	0	0	0
11	no	3	4	4	yes	18	14	32
12	yes	0	4	3	no	6	0	6
13	no	0	2	3	no	-	-	-
14	no	2*	2	2	no	9	7	16
15	no	2	1	5	no	10	6	16
16	yes	2	2	4	no	1	1	2
17	no	0	3	1	no	1	0	1
18	no	1	1	1	no	2	5	7

* = has flashbacks

Table 7.6 illustrates that PTSD symptoms were frequently reported by Part 2 subjects, although interpretation of these scores is made difficult by the absence of norms or standardised comparison groups. However, 14 of the 18 subjects recorded at least one intrusive symptom on the DSM-IV measure which satisfies the Criterion B threshold for a diagnosis of PTSD. Those subjects who had lowest symptom rates (subjects 7, 10, 17 and 18) all had severe head injuries and the three MHI subjects (subjects 4, 5 and 6) were amongst the highest scorers, and this is re-emphaised again in Hypothesis 7. Other issues illustrated by Table 7.6 are as follows.

The relationship between the DSM-IV and IES measures.

Since these measure the same factors in the same subjects, scores from the intrusive and avoidance sections of each instrument should correlate significantly. The Spearman Rank correlation coefficient is 0.67, and although this is significant ($z = 2.59$, $p < .05$), it is not as high as might be expected given that both measures are derived from the same theoretical constructs.

Memory of the trauma and intrusive symptoms.

Also seen on Table 7.6, several subjects who had no memory of the trauma had either flashbacks (subject 14) or a classification of PTSD (subjects 6, 9 and 11). This contradicts the usual view that the RA and PTA associated with a head injury precludes the formation of intrusive symptoms. There is a tendency however (not significant) for subjects with memory of the event to have more intrusive symptoms (N=6; mean=11) than those with no memory of the event (N=10; mean = 6.4).

High prevalence of PTSD symptoms compared to other studies.

Comparison of results from Parts 1 and 2 and those from three other similar studies, as shown in Table 7.7, reveals that subjects in Part 2 of this study report a higher prevalence of PTSD symptomology and intrusive symptoms than subjects in other studies.

Table 7.7

Prevalence of PTSD symptoms in Part 2 subjects compared to other studies.

Symptom or measure	Comparison	
IES Intrusive (Score of 9 or >)	Middelboe et al., (1992) 20%	Part 2 of this study 37%
IES and DSM-III or DSM-IV PTSD symptoms	Bryant and Harvey (1995) 1 month after the injury 27%	Part 2 of this study At least 3 months after the injury 27%
Flashbacks	Part 1 of this study 10%	Part 2 of this study 22%

EFFECT OF OTHER INJURIES

Hypothesis 5

Subjects with other injuries as well as a brain injury will report more PTSD and less PCS symptoms than subjects with a brain injury only.

As shown in Table 7.8, there was no significant difference between the groups on prevalence of PTSD symptoms (as measured by the IES) or PCS symptoms, although there was a tendency for brain injury alone to be associated with more PCS symptoms as proposed by the hypothesis.

Table 7.8

Mean PCS and IES scores for subjects with and without other injuries

	Other injuries *N = 13*		No other injuries *N = 5*		
	Mean	SD	Mean	*SD.*	
PCS	29	(12)	39	(23)	NS
IES (PTSD symptoms)	15	(14)	15	(10)	NS

MEMORY OF TRAUMA AND PSYCHOLOGICAL IMPACT

Hypothesis 6

Subjects who can remember the head injury event will report a higher total psychological impact.

Using data from the *memory of trauma* and *total HIIQ impact* columns of Table 7.4, the mean HIIQ total impact score for the seven subjects who remember the trauma is 33 and the mean for the ten subjects who do not remember the trauma is 26. While this is not a significant result, it does indicate a trend for memory of the head injury event to be associated with a greater impact.

MHI AND ITS ASSOCIATION WITH OTHER MEASURES

Hypothesis 7

Subjects with MHI will report more PCS and more PTSD symptoms, a higher psychological impact, and a clearer memory of the trauma, than subjects with moderate/severe head injuries.

The three subjects in this study who were not involved in a TBI but who received a brain injury in other ways were excluded from this investigation. The results reported in Table 7.9 show the MHI group obtained significantly higher scores than the moderate/severe group on the IES ($U(3,10)=3$, $p < .05$) and for total HIIQ impact ($U(3,11)=4$, $p<.05$).

Table 7.9

Differences in mean scores between MHI and moderate/severely head injured for PCS, PTSD symptoms, total psychological impact, and memory of trauma

	Mild			Moderate/severe		
	N	Mean	SD	N	Mean	SD
PCS	3	38	(9)	12	32	(16)
IES (PTSD symptoms)	3	32	(18)	10	14	(10) *
HIIQ total impact	3	35	(7)	11	25	(10) *
Memory	3	3.6	(2.3)	12	1.2	(1.4)

$p < .05$

PSYCHOLOGICAL IMPACT AND ITS ASSOCIATION WITH PCS/PTSD

Hypothesis 8

Subjects who remember the head injury trauma, and subjects who rate the trauma as having a higher psychological impact on HIIQ dimensions, will report more PCS and PTSD symptoms.

Testing this Hypothesis involved using the HIIQ Likert scales to form high and low psychological impact groups for analysis of each dimension. For the purpose of this study, a low impact was defined as a Likert score of 1 or 2 and a high impact as a Likert score of 3, 4, or 5. Results in Table 7.10 suggest that subjects who *can remember the trauma* (Likert score of 2 or more) and subjects who rate the trauma as having a higher psychological impact (Likert score of 3 or more) in six dimensions tend to have higher PCS and IES scores but results were significant in only two cases. Subjects with high impact ratings had significantly higher IES scores than subjects with low impact ratings for *negative early effects* ($U(4,12)=1$) and for *inability to make sense of the trauma* ($U(5,11)=10$).

Table 7.10 shows that there is a stronger association between psychological impact and PTSD than there is between psychological impact and PCS. This can be interpreted from an examination of the effect sizes for differences in IES (PTSD) scores which shows effect sizes close to, or above, the standard deviation of 13 for five of the dimensions. By comparison, the effect sizes for differences in PCS scores between high and low impact groups only approaches the standard deviation of 14 for one dimension (*blaming self).*

Table 7.10

Mean PCS and PTSD scores for high and low psychological impact groups and for the two groups representing ability to remember the trauma.

HIIQ Dimension	Impact group	N	Mean PCS score	effect size	Mean IES score (PTSD symptoms)	effect size
Remembering the trauma of the accident	Can	7	38	10	17	3
	Can't	11	28		14	
Blaming self	High	4	40	11	26	12
	Low	13	29		14	
Negative early effects	High	5	37	8	33	23*
	Low	13	29		10	
Unable to make sense of trauma	High	6	36	6	26	16*
	Low	12	30		10	
Horror at the time	High	5	35	5	27	14
	Low	12	30		13	
Feeling angry about it	High	12	32	5	18	4
	Low	6	27		14	
Feeling scared at time	High	5	36	4	26	13
	Low	12	32		13	
Feeling life threatened	High	5	34	3	14	-1
	Low	13	31		15	
Feeling out of control	High	11	32	0	17	3
	Low	7	32		14	
Was accident preventable	High	11	31	-3	14	-7
	Low	6	34		21	
Blame someone else	High	10	29	-7	18	2
	Low	7	36		16	
Experiencing of loss	High	14	27	-8	14	-8
	Low	3	35		22	
Self change after trauma	High	15	31	-10	16	-7
	Low	2	41		23	

* $p < .05$

Using the same data as in Table 7.10, Figures 3 and 4 illustrate the differences in mean scores of high and low psychological impact groups for PCS and PTSD respectively.

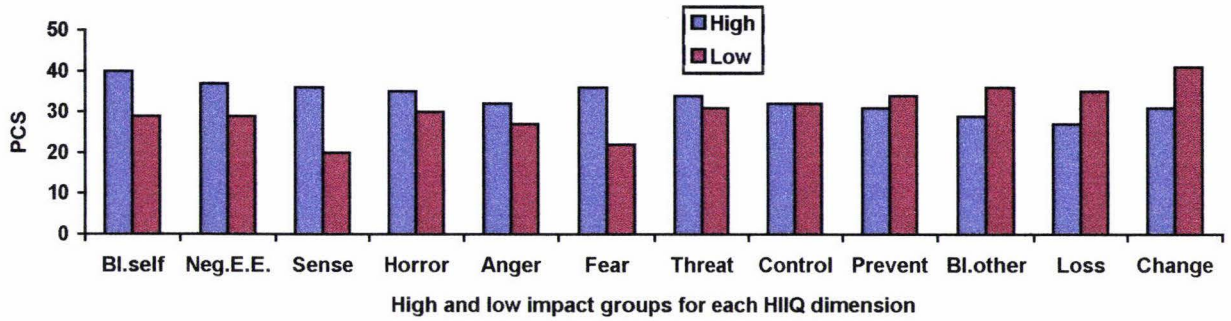

Figure 3

Differences in mean PCS scores for high and low impact groups on HIIQ dimensions.

In Figure 3, the dimensions on the left of the graph show a stronger association between high impact and PCS whereas those on the right show a stronger association between low impact and PCS. This inconsistency, and the failure to find any significance with these results, indicate a lack of support for the hypothesis that PCS is related to high impact, although there is a trend for this to occur.

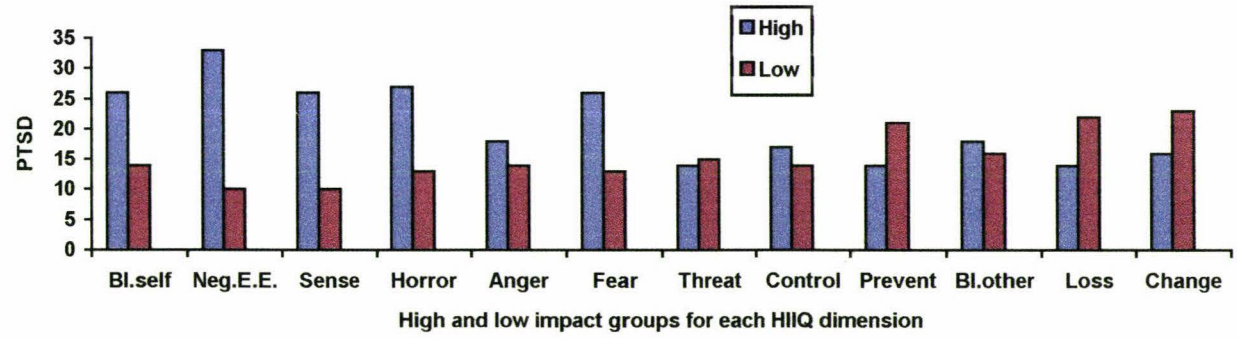

Figure 4

Differences in mean IES (PTSD) scores for high and low impact groups.

Figure 4 illustrates the differences in mean IES (PTSD) scores for high and low impact groups on each impact dimension. High impact is associated with higher mean scores for those dimensions on the left of the graph but as reported earlier, this is significant for only *negative early effects* and *inability to make sense of it.*

Hypothesis 9

Subjects, when considered as a group, will endorse some psychological impact dimensions more strongly than others.

While the previous hypothesis used scores from the HIIQ Likert scales to form high and low impact groups, examination of this hypothesis used the overall group scores on the scales. Figure 5 uses data from Table D in the Appendix to illustrate the salience of some impact dimensions compared to others. While no comparisons were being made either within the subject group or to an external group, the results suggest that many brain injured subjects experience anger, and have a sense of loss, blaming another, and self change due to the events, and this may have some therapeutic relevance.

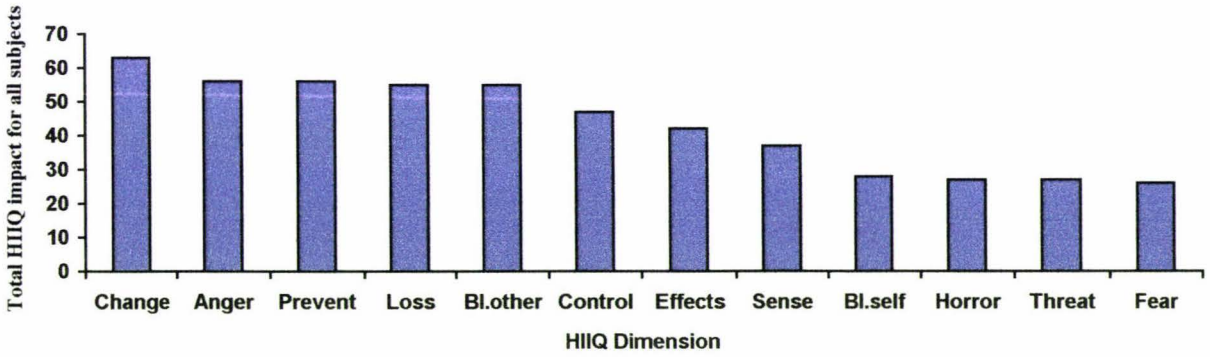

Figure 5

Total scores for all subjects on each HIIQ psychological impact dimension.

Hypothesis 10

Subjects with a DSM-IV classification of PTSD will have higher psychological impact ratings than those not classified as having PTSD.

Although this hypothesis is similar to Hypothesis 8 in that both examine the relationship between psychological impact and PTSD, here a *classification* of PTSD/ non-PTSD is used instead of *mean scores* on the IES scale. The rationale for using this hypothesis is to use the HIIQ and Likert scales differently from the way they were used in Hypothesis 8. Instead of using these scales to develop high and low psychological impact groups, subjects in the PTSD and non-PTSD groups (derived from the DSM-IV criteria) had their mean scores for each of the impact dimensions compared. This allows examination of the association of each separate impact dimension with DSM-IV classified PTSD.

None of the results were significant but subjects with a classification of PTSD had higher mean scores than the non-PTSD group for *remembering the trauma* and for nine psychological impact dimensions as shown in Table 7.11 and Figure 6.

Examining Hypotheses 8 and 10, those impact dimensions where **high psychological impact** was associated with both measures of PTSD used in this study (ie. IES scores and having a DSM-IV classification of PTSD) are *feeling life was threatened, feeling scared at the time, experiencing horror at the time, unable to make sense of the trauma, negative early effects, feeling angry about what happened, blaming someone else, and blaming oneself.*

Table 7 .11

Mean scores on the psychological impact dimensions of the HIIQ for PTSD and non-PTSD groups

Dimension	PTSD group N=5 Mean score	Non-PTSD group N=13 Mean score	Effect size
Remembering the trauma	2.2	2.1	0.1
Blaming self	2.0	1.5	0.5
Negative early effects	3.0	2.0	1.0
Unable to make sense of it	2.4	1.9	0.5
Horror at time	2.6	1.2	1.4
Feeling angry about it	3.4	3.0	0.4
Feeling scared at the time	2.2	1.5	0.7
Feeling life threatened	2.2	1.3	0.9
Feeling out of control	2.4	2.8	-0.4
Was accident preventable	3.0	3.0	0
Blame someone else	4.2	2.9	1.3
Experience of loss	3.0	3.2	-0.2
Self change after trauma	3.8	3.6	-0.4

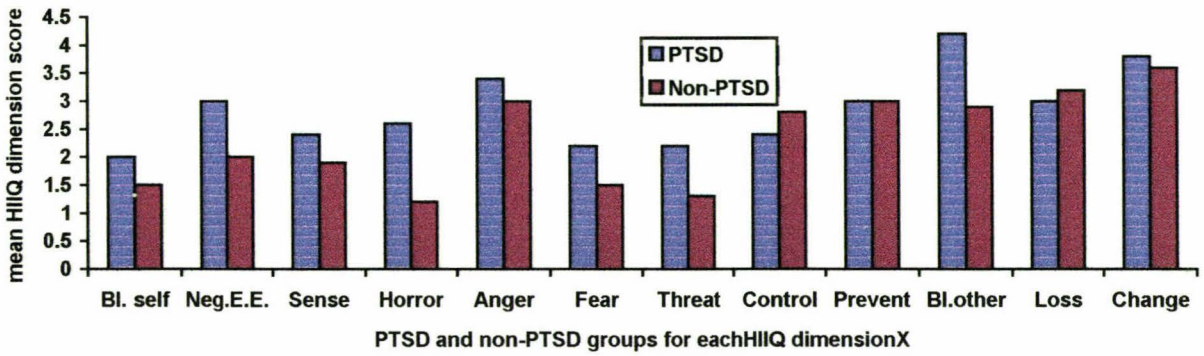

Figure 6

Comparison of mean scores on HIIQ dimensions for PTSD and non-PTSD groups.

CHAPTER 8

DISCUSSION

The proposal central to this research was that symptoms and illness behavior experienced some time after a brain injury may be related to the psychological effects of being in a traumatic incident as much as to the physical injury. The following discussion presents evidence to support this and interprets this in the context of discussion in previous chapters.

8.1 PCS AND FACTORS ASSOCIATED WITH IT.

Early chapters discussed the absence of a uniform definition and description of PCS. This in turn lead to uncertainties about which subjects to include, or exclude from PCS studies, and also to differences in symptom patterns and frequencies found in these studies. This current study took the approach typified by McAllister (1994) and Keshavan et al., (1981), that PCS was the term for the group of symptoms existing sometime after a head injury that could not obviously be explained by brain damage or other neurological or psychiatric disorders. This approach was appropriate for this study because it allowed that, while the persisting symptoms may indeed have their basis in brain damage or some psychiatric disorder, this was not altogether clear. The referral of the subjects to the Massey Clinic had in fact been to clarify the origin of the problem symptoms as well as for appropriate treatment of them. Early chapters identified PTSD as a psychiatric disorder that could possibly be misdiagnosed as PCS mainly because these disorders share many symptoms, but also because persons with a traumatic brain injury are not often asked about the presence of core PTSD symptoms.

The results of this investigation are discussed in terms of the structure of PCS, the hypotheses, and the findings of other studies on PCS.

8.1.1 Structure of PCS

Of the five studies that report symptom rates as was illustrated in Tables 2.1 and 7.5, the study of Mittenberg et al., (1996) shows the highest overall symptom frequencies followed by Part 2 of the current study. The only symptom that showed a consistent frequency across the five studies was depression (55%-56%) with other symptoms having a widely variable prevalence.

The absence of uniform prevalence rates makes it difficult to establish the common components of PCS. For instance, Keshavan et al., (1981) operationalised PCS as having three of the following symptoms: headache, dizziness, anxiety, sleeplessness and noise intolerance. However, this is not consistent with the findings of this study or others which, as Table 7.5 shows, suggest that the structure of PCS involves primarily cognitive problems (memory, concentration, fatigue and slowness), secondly emotional problems (anxiety, irritability and depression), and lastly somatic and sensory problems. Part 2 also identified that symptoms that other studies have not asked about such as *having to do things slowly* and *difficulty with decisions* may be commonly endorsed.

8.1.2 Factors associated with PCS..

Mild head injury

The results of this study suggest that there is a trend for MHI to be associated with PCS although the association is significant only for anxiety. There are a number of possible reasons why more mild head injured should report more symptoms. Previous research has suggested that psychosocial circumstances such as returning to a stressful work environment may tax incompletely recovered cognitive faculties, and it is possible that because persons with a MHI have less tangible injuries, they are expected to return to normal functioning earlier than those with more severe head injuries (McAllister, 1994). As well, a severe head injury is often associated with awareness of deficits. It is therefore quite likely that the clearer awareness of deficits following mild compared to severe head injuries, may mediate both the experience and expression of symptoms.

Another possible reason for MHI subjects to report more symptoms that emerged from this study was the significant association between MHI and the reporting of high

psychological impact. It is possible that the short amnesia and associated greater awareness and impact of what was happening during the accident may provide a substrate for later complaints to emerge.

Clearer memory of the trauma

Subjects who remembered the trauma more clearly had more symptoms and this is in accord with previous research (e.g. Cicerone & Kalmar, 1995). It remains uncertain though whether more symptoms occur because of the better memory of the trauma *per se* or because the injury is probably mild which is itself associated with PCS complaints for reasons just discussed.

Assaults

A finding in Part 1 of this study that supported other studies (e g., Dunlop et al., 1991) was that more persons who were assaulted, complained of headaches, dizziness and depression than did persons injured in MVA's or in other ways, and MVA's generally accounted for less symptoms than other causes. Dunlop et al., (1991) found that symptoms arising after an assault were usually delayed suggesting that they are caused by a psychological reaction rather than a biological one. It is possible that the cognitive and emotional changes associated with victimisation and the possible legal aftermath have an effect on developing PCS complaints. This area appears under-researched and one of the aims of this Part 2 of this research had been to investigate whether psychological impact factors associated with being victimised were related to PCS symptoms.

Psychological impact

High scores on seven impact dimensions were found to be associated with reporting high PCS scores. Although none were significantly associated, the most notable of these, in terms of effect size, were (i) negative early effects (ii) self blame (iii) inability to make sense of the trauma and (iv) horror.

The influence of *negative early effects* on PCS scores reflects the findings of other studies showing that indices of distress and anxiety soon after the injury can predict later symptoms (Lidvall, et al., (1974, cited in Lishman, 1988); Middelboe, et al., 1992). Why self-blame should be related to higher PSC scores is uncertain. The HIIQ

questionnaire item for self-blame asked "how much do you think you were to blame for what happened?" and this does not give any information as to whether characterological or behavioral self-blame was underlying the attribution. Janoff-Bulman (1985) reports that behavioral self-blame (attributing the event to one's changeable behavior) is more likely to cause a poor outcome than characterological self-blame (attributing the event to one's fixed character). As well, the association of self-blame and PCS found in this study is contrary to the findings of Rutherford (1989) that blaming someone else has a greater impact on PCS.

Being unable to make sense of the trauma, and feeling threatened, scared, horrified and out of control during the accident are early responses which might be expected to cause problematic conditioned responses and chronic arousal which in turn may mediate PCS. They are also likely to occur as responses to incidents that involve ongoing legal and medical problems which may themselves drive the PCS. That is, immediate impacts of the trauma may mediate PCS or may merely be correlates of other factors which mediate the PCS.

8.2 PTSD SYMPTOMS AND ASSOCIATED FACTORS

In this study, 10% (Part 1) to 37% (Part 2) of subjects presenting with problems after a brain injury reported core symptoms of PTSD and a greater number reported secondary PTSD symptoms. For Part 2, this is a greater prevalence than was expected from previous studies. The results suggest that PTSD may be related to the following factors.

Memory of the trauma.

Subjects in the present study who remembered the event had higher IES scores with more intrusive symptoms than those with no memory of the event. Although this was not a significant association, it tends to support other studies associating minimal RA and PTA with more PTSD symptoms.

Contrary to this trend however, were four subjects with no memory of the traumatic event who had a high number of intrusive symptoms. Although an explanation for this is outside the area of this study, Bryant (1996) has suggested that pseudomemories (derived from information gained after the event) and implicit memory processes account

for intrusive symptoms occurring after a head injury involving RA and PTA. Implicit memory theories propose that head injured persons encode aspects of the trauma in perceptual non-declarative memory systems (Horton, 1993). For the amnesic subjects in this study who have intrusive symptoms however, more thorough assessment of the phenomenology and topography of the symptoms would have been needed before any further comments could be made.

Mild head injury

Significantly more subjects with MHI than with moderate/severe head injury reported PTSD symptoms. This can be interpreted as supporting previous research showing that minimal amnesia during TBI facilitates consolidation of memories of the trauma (e g., Bryant & Harvey, 1995).

Psychological impact

Eight dimensions were found to be related to PTSD. The most notable of these, in terms of effect size, were (i) negative early effects (ii) inability to make sense of the trauma (iii) feeling scared (iv) experiencing horror (v) feeling out of control (vi) self-blame (vii) blaming someone else.

Subjects with higher scores on these eight dimensions reported more PTSD symptoms than those with lower scores. This supports conceptualisations of PTSD suggesting that it is based on reactions involving fear, horror, and threat to life. However, in this study only the *inability to make sense of the trauma* and *negative early effects* significantly influenced PTSD scores. The inability to make sense of events could be viewed as challenges to the person's world view and so the finding here is in accord with cognitive conceptalisations of PTSD that symptoms are related to the failure to adjust long-held cognitive schemas about self and the world.

The high salience of the dimension *negative early effects* in this study supports the findings of Middelboe et al., (1992) that distress and anxiety early after the head injury is associated with high IES scores one year later.

Other injuries

This study found no association between having other injuries and PTSD symptoms, and while this contradicts some findings that extent of injury is a powerful predictor of PTSD (e g., Blanchard et al., 1996), it supports other findings of no association (e g., Bryant & Harvey, 1995; Green, 1994).

8.3 THE MEDIATING ROLE OF PSYCHOLOGICAL IMPACT

One of the aims of this study was to examine the relationship between PTSD, PCS and psychological impact. High standard deviations relative to the mean scores were obtained for the three measures of PCS, PTSD and psychological impact, and this, together with the small number of subjects, made it difficult to find many significant relationships. However, there were several tendencies.

Firstly, reporting a high psychological impact is likely to be associated with more PTSD than PCS symptoms. This is what would be expected as PTSD is conceptualised as a reaction to more severe traumas whereas PCS can have numerous other factors underlying it as well as the emotional impact of the trauma. The issue of which particular impact dimensions underly PTSD as compared to PCS is still unclear though as high scores for six impact dimensions coincided with higher scores for both PTSD and PCS.

Secondly, subjects who report both a high psychological impact and higher PCS scores also tend to report higher PTSD symptom scores. This highlights the ongoing problem discussed in earlier chapters of differentiating between PTSD and PCS when there are more severe reactions to the head injury and its circumstances. It is uncertain whether subjects reporting high psychological impact have PTSD symptoms and separate PCS or whether the PCS is really just a part of the PTSD. This is a clinical assessment issue that involves investigations of etiological factors (e g. does organic brain damage or motivational factors underly the symptoms?) as well as the veracity and pattern of the symptoms.

This issue could be clarified by comparing groups with high psychological impact and high PCS scores that were differentiated in terms of high and low IES (PTSD) scores. This would identify the frequency with which psychological reactions to trauma (and events surrounding it) could occur in the absence of PTSD. Since this was not possible due to low numbers in the current study, the most that can be concluded is that PTSD symptoms appear to confound the diagnosis of PCS for those subjects who report a more severe psychological impact of the accident.

8.4 LIMITATIONS OF THIS STUDY

Part 1 The main limitation was the necessity to use data on symptom prevalence obtained by other investigators in conditions of unknown reliability and validity. As well, only a few of the symptoms that comprise PCS and only one PTSD symptom (flashbacks) had been recorded.

Part 2 Limitations here can be addressed in the following categories.

Measures used. One of the major issues limiting the internal and external validity of this research is the reliability and validity of the four assessment instruments used.

As there was no existing instrument for assessing the psychological impact of a head injury, the Head Injury Impact Questionnaire (HIIQ) was devised by the researcher. However, this had no established psychometric properties for use either as a measure of separate impact dimensions or total impact and this may have reflected on the appropriateness of the scores obtained and used. For instance, using one or two questions to tap broad dimensions such as *the significance of losses* or *the degree of self-change since the accident* has the potential to produce misleading data. As well, the procedure of splitting Likert scores to provide high and low impact groups was not part of the original research design and was decided on after the measures were administered as the most suitable way of obtaining groups for comparison.

The IES was chosen as the measure of PTSD symptoms because its ease of administration and stated psychometric properties appeared to best suit the research needs. However, at the outset it was found to have items that were too difficult or

ambiguous for some of the subjects to understand (notably item two), and for this reason the modified DSM-IV criteria were added as a separate measure. The difficulty that subjects in this study had with the IES is noteworthy since two studies cited frequently in the background to the study (Bryant & Harvey, 1995, and Middelboe et al., 1992) had also used it.

In retrospect, the DSM-IV protocol for classifying PTSD cases, as used in this study, did not go into sufficient detail about symptoms. The objective for using it was because it was thought to be easy for head injured persons to understand but this was probably at the expense of the specificity and sensitivity required of a properly validated measure. The fact that four subjects with severe head injury and only one with MHI endorsed sufficient items to be classified as having PTSD cases is contrary to previous researchers findings on the prevalence of PTSD after a head injury.

Research design. The main limitation here is the use of a retrospective rather than prospective research design. Asking subjects about their immediate reactions to an event that may have occurred several years ago is not going to provide information of the same reliability and validity as asking them soon after the event.

Subjects. The use of only 18 subjects limited not just the external validity of this study but posed limits on achieving statistical significance. For example, in many instances when using Mann-Whitney calculations, a low value of U had to be matched against a critical value that was especially low because of the small N.

8.5 SUGGESTIONS FOR FURTHER RESEARCH

This research was conducted taking a broad approach to a number of contentious issues such as defining PCS and clarifying its origins, and differentiating PTSD from PCS. From previous comments in this chapter, further research could take several directions.

(1) Using measures that are psychometrically evaluated for use with head injured subjects both clinically and in research.

The PCS measure. There appears to be no standardised instrument for assessing PCS and this may be partly responsible for the anomalies in other research on PCS structure and TBI outcomes. A suitable instrument would need measures of both frequency and severity of symptoms as well as norms for non-clinical and psychiatric populations.

The PTSD measure. Assessment of emotionally and behaviorally based symptoms such as flashbacks, disturbing dreams and avoidance behaviors may be more reliable and valid when done in the context of a clinical interview rather than by the endorsement of single items on a questionnaire.

The psychological impact measure (HIIQ). Previous studies, as well as this one, have indicated that distress early after a head injury is one of the factors most associated with later outcomes. Therefore the development of a reliable and valid measure of the psychological impact of a head injury with established predictive validity for later outcomes, may have potential clinical use.

(2). Research design.

In this study, it was assumed that the dimensions of the HIIQ represented thoughts and feelings which acted after the head injury to mediate later outcomes. Integrative models of PCS and PTSD use feedback cycles rather than linear processes as a paradigm and such models may regard assumptions of linearity (such as that alterations in a single variable such as *self-blame* may perceptibly alter a complex structure such as PCS) as overly simplistic. On this basis, it is possible that the associations found in this study are spurious and reflect nothing more than subjects' bias and attitudes towards answering questionnaire items that rely on styles of self-perception.

Further research on the origin of PCS symptoms might reduce the likelihood of making such Type 1 errors in two ways: firstly by using multivariate research designs which presume that variables act in an inter-related rather than singular fashion, and secondly by using a more fine grained analysis of associations between variables. For instance, the possibility that attention or memory defects underly irritability and contribute to depressive behaviors could be examined by looking at the fine grained cognitive processes surrounding those defects and this could necessitate the case study approach.

Thirdly, a qualitative research design could be used to examine the influence of personality and personal history factors on head injury outcomes. For instance, Keshavan et al ., (1981) using reports of relatives of the head injured person, concluded that subjective symptoms correlated primarily with pre-trauma neuroticism. With reference to PTSD, Shalev et al., (1993) found that the content of his subjects' intrusive symptoms was determined partly by the highly idiosyncratic set of personal agendas and activities operating at the time the accident happened. It is acknowledged that the quantitative data seen on Table 7.4 of this study disregards qualitative factors. For instance, subject one had an extremely high PCS score indicating somatic and psychological distress which may have been due partly to his history of multiple head injuries and to having had cancer previously. Similarly, subjects 17 and 18 recorded particularly low PCS and IES scores which appeared from the clinical interview to be due to both psychological denial and the lack of awareness of self-limitation that often accompanies severe brain injury. A qualitative approach then could be an appropriate vehicle for understanding how cognitive styles, personality traits and personal meanings influence PCS and PTSD.

Fourthly, a prospective research design could be used so that, for example, an assessment of the psychological impact early after a head injury could be matched with both early and later symptoms.

(3). Research that utilises the findings from this study.

Because of limited subject numbers and the finding of few significant associations, the conclusions made from this study can only be tentative. Several directions that this study has taken though appear to be fruitful areas for further research.

(i) The development of a consensual and operational definition of PCS may facilitate comparison of results in future studies. Such a definition would need to be clear about symptom frequency norms and whether the term PCS included other diagnostic categories such as brain damage, depressive and anxiety disorders, and PTSD.

(ii) Clarification of the significance of PTSD symptoms after a head injury may have therapeutic use. A third of the subjects in Part 2 of this study reported moderate to severe intrusive symptoms and the clinical significance of this is unclear. Clarification of the mechanisms underlying intrusive symptoms in subjects who were unconscious during the head injury may facilitate further understanding of memory processes after trauma.

8.6 SUMMARY

This research has aimed to clarify the relationship between the psychological impact of a brain injury and its circumstances, PTSD and PCS. The underlying proposal was that persons who report a higher impact will have more PTSD and PCS symptoms. Most of the results of this research support this proposal although only a few of these results are significant. MHI subjects reported not only more PCS and PTSD symptoms but also a higher psychological impact and clearer memory of the brain injury incident and it is possible that this impact and memory mediate the development of the symptoms.

A number of conclusions can be drawn from these research findings. Just as other research has demonstrated the importance of the physical effects of a brain injury, this research has demonstrated the importance of the effects of the psychological impact of the brain injury incident on later outcomes. Although the issue of whether the person has PTSD, PCS or both, is a clinical issue, this research has shown that both sets of symptoms are likely to occur after a brain injury with a high psychological impact.

PTSD is conceptualised as stemming from traumatic experiences involving high levels of fear, horror, life threat, and challenges to the person's cognitive schemas of self and the world and this is supported by there results of this study. PCS has been empirically associated with a range of factors and current models of PCS attempt to integrate pre-morbid, organic damage, motivational, cognitive and emotional factors into a unified process. This study has shown that cognitive and emotional factors centering on the incident causing the head injury may sometimes significantly contribute to this process.

REFERENCES

Accident compensation Corporation (1991). *ACC Injury Statistics, Vol. 3 and 4.* Wellington: Accident Compensation Corporation.

Adler, A. (1945). Mental symptoms following head injury. *Archives of Neurology and Psychiatry, 53*, 33-43.

Alves, W., Macciocchi, S.N. & Barth, J.T. (1993). Postconcussive symptoms after uncomplicated mild head injury. *Journal of Head Trauma Rehabilitation, 8*, 48-59.

American Congress of Rehabilitation Medicine. (1993). Definition of mild head injury. *Journal of Head Trauma Rehabilitation, 8*, 86-87.

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

Barnes, M.P. (1991). Outcome of head injury. *Current Opinion in Neurology and Neurosurgery, 4*, 12-16.

Batchelor, J., Harvey, A.G. & Bryant, R.A. (1995). Stroop colour-word test as a measure of attention deficit following mild head injury. *The Clinical Neuropsychologist, 9*, 180-186.

Baum, A., & O'Keefe, M.K. & Davidson, L.M. (1990). Acute stressors and chronic response. *Journal of Applied Social Psychology, 20*, 1643-1654.

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

Binder, L.M. (1990). Malingering following head trauma. *The Clinical Neuropsychologist, 4*, 25-36.

Binder, L.M. & Rattock, J. (1989). Assessment of the post-concussive syndrome after mild head injury. In M. Lezak (Ed.) *Assessment of the Behavioral consequences of Head Trauma (pp 37-48)*. New York: Alan R. Lisk Inc..

Blanchard, E.B., Hickling, E, L., Taylor, A.E., Loos, W.R, Forneris, C.A. & Jaccard J. (1996). Who develops PTSD from motor vehicle accidents? *Behavioral research and therapy, 34,* 1-10.

Body, C. M. (1995). *Incidence and Neuropsychological Sequelae of Head Injury in a New Zealand Adolescent Sample.* Massey University Thesis.

Bohnen, N., Twinjstra, A. & Jolles, J. (1992). Post traumatic and emotional symptoms in different subgroups of patients with mild head injury. *Brain Injury, 6,* 481-487.

Breslau, N. (1990). Stressors: continuous and discontinuous. *Journal of Applied Social Psychology, 20,* 1686-1673.

Bryant, R.A. (1996). Post-traumatic stress disorder, flashbacks, and pseudomemories in closed head injury. *Journal of Traumatic Stress 9,* 621-629.

Bryant, R.A. & Harvey, A.G. (1995). Acute stress responses: a comparison of head injured and non-head injured patients. *Psychological Medicine, 25,* 866-873.

Bryant, R.A. & Harvey, A.G. (1996). Initial post-traumatic responses following motor vehicle accidents. *Journal of Traumatic Stress, 9,* 223-233.

Caplan, B. (1993). Rehabilitation of post-concussive disorder. *Journal of Head Trauma Rehabilitation, 8,* 114-116.

Cicerone, K.D. & Kalmar, K. (1995). Persistent postconcussion syndrome: The structure of subjective complaints after mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 10*, 1-17.

Creamer, M. (1995). A cognitive processing formulation of post trauma reactions. In R.J. Kleber, C.R.Figley, & B.P Gersons (Eds.). *Beyond trauma.* (pp 55-73). New York: Plenum Press.

Creamer, M., Pattison, P., & Burgess, P. (1992). Reaction to trauma: a cognitive processing model. *Journal of abnormal psychology, 101*, 452-459.

Davidoff, D.A., Labstein, D.F., Kessler, H.R. & Mark, V.H. (1988). Neurobehavioral sequelae of mild head injury: A consideration of the Post-concussive Syndrome versus Post traumatic Stress Disorder. *Cognitive Rehabilitation, March/April*, 8-13.

Davis, R.C., & Friedman, L.N. (1985). In C.R.Figley (Ed.). *Trauma and its Wake* (pp 90-111). New York: Brunner Mazel.

Dikmen, S.S., & Levin H.S. (1993). Methodological issues in the study of mild head injury. *Journal of head trauma rehabilitation, 8*, 30-37.

Dikmen, S.S., Machamer, J.E., Winn, H.R. & Temkin, N.R. (1995). Neuropsychological outcome at 1-year post head injury. *Neuropsychology, 9*, 80-90.

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

Dikmen, S.S., Temkin, N. & Armsdale, G. (1989). Neuropsychological recovery: Relationship to psychosocial functioning and post concussive complaints. In H.S. Levin, H.M. Eisenberg & A.C. Benton (Eds). *Mild Head Injury.* (pp.229-244). New-York: Oxford University Press.

Dixon, C.E., Taft, W.C. & Hayes. (1993). Mechanisms of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8,* 1-12.

Dunlop, T.W., Udvarhelyi, G.B., Stedem, A.F., O'Connor, J.M., Issacs, M.L., Puig, J.G. & Mather, J.H. (1991). Comparison of patients with and without emotional /behavioral deterioration during the first year after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 3,* 150-156.

Epstein, R.S. & Ursano, R.J. (1994). Anxiety disorders. In J.M. Silver, S.C. Yudofsky & R.E. Hales (Eds.). *Neuropsychiatry of traumatic brain injury,* (pp.285-312). Washington: American Psychiatric Press.

Everly, G.S. (1995). An integrative two factor model of post traumatic stress. In G.S.Everly & J.M.Lating (Eds.). *Psychotraumatology.* (pp. 28-45). New-York: Plenum Press.

Fenton, G., McClelland, R., Montgomery, A., MacFlynn, G. & Rutherford, W. (1993). The post-concussive syndrome: social antecedents and psychological sequelae. *British Journal of Psychiatry, 162,* 493-497.

Foa, E.B., Skeketee, G., & Rothbaum, B.O. (1989). Behavioral/Cognitive conceptualisations of post-traumatic stress disorder. *Behavior Therapy, 20,* 155-176.

Fox, D.D., Lees-Haley, P.R., Earnest, K. & Dolezal-Wood, S. (1995). Post-concussive symptoms: Base rates and etiology in psychiatric patients. *The Clinical Neuropsychologist, 9,* 89-92.

Frederikson, L.G., Chamberlain, K. & Long, N. (1995). PTSD: A time-process view of contributing Factors. Occasional Papers in Psychology, Massey University.

Gentillini, M., Nichelli, P. & Shoenhuber, R. (1989). Assessment of attention in mild head injury. In H.S Levin, H.M.Eisenberg & A.C.Benton (Eds.). *Mild Head Injury*. (pp.163-175). New York: Oxford University Press.

Gerber, D.J. & Schraa, J.C. (1995). Mild Traumatic Brain Injury: Searching for the syndrome. *Journal of Head Trauma Rehabilitation, 10*, 28-40.

Godfrey, H. & Knight, R. (1992). Psychological consequences of head injury: Afinal repport for the Accident Compensation Corporation of New Zealand. Department of Psychology, University of Otago.

Goldstein, F.C. & Levin, H.S. (1995). Neurobehavioral outcome of traumatic brain injury in older adults: Initial findings. *Journal of Head Trauma Rehabilitation, 10*, 57-73.

Gouvier, W.D., Cubic, B., Jones, G., Brantley, P. & Cutlip, Q. (1992). Post-concussive symptoms in a normal and a head injured college population. *Archives of Clinical Neuropsychology, 7*, 193-212.

Green, B.L. (1990). Defining trauma: Terminology and generic stressor dimensions. *Journal of Applied Social Psychology, 20*, 1632-1642.

Green, B.L. (1994). Psychosocial research in traumatic stress: An update. *Journal of Traumatic Stress, 7*, 341-358.

Green, B.L., Wilson, J.P. & Lindy, J.D. (1985). Conceptualising post-traumatic stress disorder: A psychosocial framework. In C.R.Figley (Ed.). *Trauma and its Wake*. (pp 53-69). New York: Brunner Mazel.

Gronwall, D. (1989a). Behavioral assessment during the acute stages of traumatic brain injury. In M. Lezak (Ed.). *Assessment of the Behavioral Consequences of Head Trauma.* (pp. 19-36). New York: Alan R Liss Inc.

Gronwall, D. (1989b). Cumulative and persisting effects of concussion on attention and cognition. In H.S Levin, H.M.Eisenberg & A.C.Benton (Eds.). *Mild Head Injury.* (pp 153-162*)*. New York: Oxford University Press.

Haynes, S.D..(1994). The experience of grief in the head injured adult. *Archives of Clinical Neuropsychology, 9*, 323-336.

Hinkeldey, N.S. & Corrigan, J.B. (1990). The structure of head injured patients neurobehavioral complaints: A preliminary study. *Brain Injury, 4*, 115-133.

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

Horowitz, M. (1986). *Stress response syndromes.* Northvale, New Jersey: Jason Aranson.

Horowitz, M.J., Wilner, N. & Alvarez, W. (1979). Impact of event scale: A measure of subjective stress. *Psychosomatic Medicine, 4*, 209-218.

Jacobson, R.R. (1995). The post-concussional syndrome: Physiogenesis, psychogenesis and malingering. An integrative model. *Journal of Psychosomatic Research, 39*, 675-695.

Janoff-Bulman, R. (1985). The aftermath of victimisation: Rebuilding shattered assumptions. In C.R.Figley (Ed.). *Trauma and its Wake*. (pp 15-35). New York: Brunner Mazel.

Kay, T., Newman, B., Cavallo, M., Ezrachi, O. & Resnick, M. (1992). Towards a neuropsychological model of functional disability after mild traumatic brain injury. *Neuropsychology, 6,* 371-384.

Karol, RL. (1989). The duration of seeking help following traumatic brain injury: The persistence of non-neurological symptoms. *The Clinical Neuropsychologist, 3,* 244-249.

Kasle, S.V. (1990). Some considerations in the study of traumatic stress.. *Journal of Applied Social Psychology, 20,* 1655-1665.

Katz, D.I. & Alexander, M.P. (1994). Traumatic brain injury: Predicting the course of recovery and outcome for patients admitted to rehabilitation. *Archives of Neurology, 51,* 661-670.

Keane, T.M., Fairbank, J.A., Caddell, J.M., Zimmering, R.T., & Bender, M.E. (1985). A behavioral approach to assessing and treating PTSD in Vietnam veterans. In C.R. F. Figley (Ed.). *Trauma and its Wake* (pp 257-294).New York: Brunner Mazel

Keane, T.M., Wolfe,J. & Taylor, K.L. (1987). PTSD : Evidence for diagnostic validity and methods of psychological assessment. *Journal of Clinical Psychology, 43,* 32-43.

Keshavan, M.S., Channabasavanna, S.M. & Reddy, G.N. (1981). Post-traumatic psychiatric disturbances: Patterns and predictors of outcome. *British Journal of Psychiatry 138,* 157-160.

Kolb, L.C. (1987). A neuropsychological hypothesis explaining post-traumatic stress disorders. *American Journal of Psychiatry, 144,* 989-995.

Kraus, J.F. & Nourjah, P. (1989). The epidemiology of mild head injury. In H.S. Levin, H.M. Eisenberg & A.C. Benton (Eds.). *Mild Head Injury*. (pp 8-22). New York: Oxford University Press.

Kraus, J.F.& Sorenson, S.B. (1994). Epidemiology. In J.M. Silver, S.C.Yudofsky & R.E Hales (Eds.) *Neuropsychiatry of traumatic brain injury*, (pp.3-42). Washington: American Psychiatric Press.

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

Lee, J., Giles, K. & Drummond, P.D. (1993). Psychological disturbance and an exaggerated response to pain in patients with whiplash injury. *Journal of Psychosomatic Research, 37*, 105-110.

Leininger, B.E., Gramling, S.E., Farrell, A.D., Kreutzer, J.S. & Peck, E.A. (1990). Neuropsychological deficits in symptomatic mild head injury patients after concussion and mild concussion. *Journal of Neurology, Neurosurgery and Psychiatry, 53*, 293-296.

Leininger, B.E. Kreutzer, J.S. & Hill, M.R. (1991). Comparison of mild and severe head injury emotional sequelae using the MMPI. *Brain Injury, 5*, 199-205.

Levin, H.S., Mattis, S., Ruff, R.M., Eisenberg, H.M., Marshall, L.F, Tabbador, K., High, W.M. & Frankowski, R.F. (1987). Neurobehavioral outcome following mild head injury: A three centre study. *Journal of Neurosurgery, 66*, 234-243.

Lishman, W.A.(1973). The psychiatric sequelae of head injury. *Psychological Medicine, 3*, 304-318.

Lishman, W.A. (1988). Physiogenesis and psychogenesis in the post-concussive syndrome. *British Journal of Psychiatry, 153*, 460-469.

Malloy, P., Fairbanks, J.& Keane, T. (1983). Validation of multimethod assessment of PTSD in Vietnam veterans. *Journal of Consulting and Clinical Psychology, 51,* 488-494.

Mayou, R. & Radanov, B.P. (1996). Whiplash neck injury. *Journal of Psychosomatic Research, 40,* 461-474.

McAllister, T.W. (1994). Mild traumatic brain injury and the post-concussive syndrome. In J.M.Silver, S.C.Yudofsky & R.E.Hales (Eds.). *Neuropsychiatry of Traumatic Brain Injury, (*pp.357-392). Washington: American Psychiatric Press.

McCann, I.L. & Pearlman, L.A. (1993). *Psychological trauma and the adult survivor: Theory, therapy and transformation.* New York: Brunner/Mazel.

McFarlane, A.C., Atchison, M., Rafalowicz, E. & Papay, P. (1994). Physical symptoms in post-traumatic stress disorder. *Journal of Psychosomatic Research, 38,* 715-727.

McMillan, T.M. (1991). PTSD and severe head injury. *British Journal of Psychiatry, 159,* 431-433.

McMordie, W.R. (1988). Twenty year follow-up on the prevailing opinion on the post-traumatic or post-concussive syndrome. *The Clinical Neuropsychologist, 2,* 198-212.

Middelboe, T., Anderson, H.S., Birket-Smith, M. & Friis, M.L. (1992). Mild head injury: Impact on general health after one year. A prospective study. *Acta Neurologica Scandinavica, 85,* 5-9.

Miller, H. (1961). Accident neurosis. *British Medical Journal, 1,* 991-998.

Mittenberg, W., Tremont, G., Zielinski, R.E., Fichera, S. & Rayles, K.R. (1996). Cognitive behavioral treatment of post-concussive syndrome. *Archives of Clinical Neuropsychology, 11*, 139-145.

Montgomery, C.E., Fenton, G.W., McClelland, R. J., MacFlynn, G. & Rutherford, W.H. (1991). The psychobiology of mild head injury. *Psychological Medicine, 21*, 375-384.

Morse, P.A. & Montgomery, C.E. (1992). Neuropsychological evaluation of traumatic brain injury. In R.F. White. (Ed.). *Clinical syndromes in adult neuropsychology. The practitioners handbook*. (pp. 85-175). New York: Elsevier science publishers.

Nemeth, A. J. (1996). Behavior descriptive data on cognitive, personality, and somatic residua after relatively mild brain trauma: Studying the syndrome as a whole. *Archives of Clinical Neuropsychology, 11*, 677-695.

Parker, R.S. (1990). *Traumatic brain injury and neuropsychological impairment*. New York: Springer-Verlag.

Peterson, K.C., Prout, M.F. & Schwartz, R.A. (1995). *Post-traumatic Stress Disorder: A Clinicians Guide*. New York: Plenum Press.

Peterson, C. & Seligman, M.E. (1983). Learned helplessness and victimisation. *Journal of Social Issues, 2*, 103-116.

Pilowski, I. (1985) Cryptotrauma and "Accident Neurosis". *British Journal of Psychiatry, 147*, 310-311.

Prigatano, G. (1987). Psychiatric aspects of head injury: Problem areas and suggested guidelines for research. In H.S. Levin, J Grafman & H. M Eisenberg (Eds.). *Neurobehavioral recovery from head injury.* (pp. 215-233). N.Y: Oxford University Press.

Rauter, U.K., Leonard, C.E. & Swett, C.P. (1996). SCL90-R factor structure in an acute involuntary adult psychiatric sample. *Journal of Clinical Psychology, 52,* 625-629.

Reviere, S.L. (1996). *Memory of childhood trauma.* New York: Guilford Press.

Rimel, R.W., Giordani, B., Barth, J.T., Boll & Jane. (1981). Disability caused by mild head injury. *Neurosurgery, 9,* 221-228.

Roca, R.P., Spence, R. J. & Munster, A.M. (1992). Post-traumatic adaptation and distress among adult burn survivors. *American Journal of Psychiatry, 149,* 1234-1238.

Rossi, E.L & Cheek, D.B. (1988). *Mind-body therapy.* New York: W.W. Norton and Co..

Rubenstein, D. (1993). Psychiatric aspects of mild brain injuries. In S. Mandel, R. Sataloff, & S. Shapiro (Eds.). *Mild head trauma: Asessment, management and rehabilitation.* (pp. 107-122). New York: Springer Verlag.

Ruff, R.M., Wylie, T. & Tennant, W. (1993). Malingering and malingering-like aspects of mild closed head injury. *Journal of Head Trauma Rehabilitation, 8,* 60-73.

Rutherford, W.H. (1989). Post-concussive symptoms: Relationship to acute neurological indices, individual differences and circumstances of injury. In H.S. Levin, H.M.Eisenberg & A.C Benton (Eds.). *Mild Head Injury.* (pp 217-228). New York: Oxford University Press.

Rutherford, W.H., Merrett, J.D. & McDonald, J.R. (1977). Sequelae of concussion caused by minor head injuries. *Lancet, 1*, 1-4.

Sataloff R.T. & Spiegel, J.R. (1993). Neurologic evaluation and treatment following minor head trauma. In S Mandel, R.T. Sataloff, & S.R. Shapiro. (Eds.). *Minor Head Trauma.* (pp. 159-202). New York: Springer Verlag.

Sbordone, R.J. & Litter, J.C. (1995). Mild traumatic brain injury does not produce PTSD. *Brain Injury, 9*, 405-412.

Schwarzwald, J., Solomon, Z., Weisenberg, M. & Mikuliner, M. (1987). Validation of impact of events scale for psychological sequelae of combat. *Journal of Consulting and Clinical Psychology, 55*, 251-256.

Segalowitz, J.C. & Lawson, S. (1995). Subtle symptoms associated with mild head injury. *Journal of Learning Disabilities, 28*, 309-319.

Shalev, A.Y., Schreiber, S. & Galai, G. (1993). Early psychological responses to traumatic injury. *Journal of Traumatic Stress 6*, 441-449.

Silver, J.M., Yudofsksy, S.C. & Hales, R.E. (1991). Depression in traumatic brain injury. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 4*, 12-23.

Slagle, D.A. (1990). Psychiatric disorders following closed head injury: An overview of biopsychosocial factors and their etiology and management. *International Journal of Psychiatry in Medicine, 20*, 1-35.

Sweeney,J.E. (1992). Non-impact brain injury: Grounds for clinical study of the neuropsychological effects of acceleration forces. *The Clinical Neuropsychologist, 6,* 443-459.

Taylor, S. & Koch, W.J. (1995). Anxiety disorders due to motor vehicle accidents: Nature and treatment. *Clinical Psychology Review, 15,* 721-738.

Teasdale, G. & Jennett, B. (1974). Assessment of coma and impaired consciousness. *Lancet, 11,* 81-84.

Ullman, S.E. (1995). Adult trauma survivors and post traumatic sequelae: An analysis of re-experiencing, avoidance and arousal criteria. *Journal of Traumatic Stress, 8,* 179-187.

Van der Volk, B.A. & Fisler, R. (1995). Dissociation and the fragmentary nature of traumatic memories: Overview and exploratory study. *Journal of Traumatic Stress, 8,* 505-523.

Ver Allen, P & van Kammen, D.P. (1990). The biological findings in post-traumatic stress disorder: A review. *Journal of Applied Social Psychology, 20,* 1789-1821.

Varney, N.R., Bushnell, D.L., Nathan, M., Kahn, D., Roberts, R., Rezai, K., Walker, W. & Kirchner, P. (1995). NeuroSPECT correlates of disabling mild head injury: Preliminary findings. *Journal of Head Trauma Rehabilitation, 10,* 18-28.

Vreven, D.L., Gudanowski, D.M., King, C.A. & King, D.W. (1995). The civilian version of the Mississippi PTSD scale: A psychometric evaluation. *Journal of Traumatic Stress, 8,* 91-98.

Watson, M.R., Fenton, G.W., McClelland, R.J., Lumsden, J., Headley, M. & Rutherford, W.H. (1995). The post-concussive state: Neurophysiological aspects. *British Journal of Psychiatry, 167,* 514-521.

Watson, C.G., Juba, M.P., Manifold, V., Kucala, T. & Anderson, P.E. (1991). The PTSD interview: Rationale, description, reliability, and concurrent validity of a DSM.111 based technique. *Journal of Clinical Psychology, 47,* 179-188.

Weathers, F.W., Litz, B.T., Keane, T.M., Herman, D.S., Steinberg, H.R., Huska, J.A. & Kraemer, H.C. (1996). The utility of the SCL90 -R for the diagnosis of war-zone related post-traumatic stress disorder. *Traumatic Stress, 9,* 111-127.

Williams, M., Levin, H.S. & Eisenberg, H.M. (1990). Mild head injury classification. *Neurosurgery, 27,* 422-428.

Woessner, R. & Caplan, B. (1995). Affective disorders following mild to moderate brain injury: Interpretive hazards of the SCL90. *Journal of Head Trauma Rehabilitation, 10, 78-89.*

Yeates, J.M. (1997). Epidemiology and Consequences of Head Injury: A comparison of Focal/Sharp Head Injuries and Generalised/Blunt Head Injuries. Massey University Thesis.

Youngjohn, S.R., Burrows, L & Erdal, K. (1995). Brain damage or compensation neurosis? The controversial post-concussive syndrome. *The Clinical Neuropsychologist, 9,* 112-123.

Zasler, N.D. (1993). Mild traumatic brain injury: Medical assessment and intervention. *Journal of Head Trauma Rehabilitation, 8, 13-29.*

Zilberg, N.J., Weiss, D.S. & Horowitz, M.J. (1982). Impact of event scale: A cross validation study and some empirical evidence supporting a conceptual model of stress response syndromes. *Journal of Consulting and Clinical Psychology, 50, 407-414.*

Zlotnick, C., Zabriski, A., Shea, T., Costello, E., Begin, A., Pealstein, T. & Simpson, E. (1996). The long term sequelae of sex abuse: Support for a complex post-traumatic stress disorder. *Journal of Traumatic Stress, 9*, 195-205.

APPENDIX A

THE HEAD INJURY IMPACT QUESTIONNAIRE
HIIQ

HEAD INJURY QUESTIONNAIRE

Subject Code.....

Date.....

This questionnaire asks you 32 questions about your head injury and the events that caused it. The first 9 questions ask you to write or tick in the box provided. The next 21 questions ask you to Circle the number on the scale that best describes how you remember the injury.

The first 5 questions ask you about parts of your medical background BEFORE the injury occurred.

1. Were you ever treated by a Doctor or Specialist for emotional or psychological problems.

No	
Yes	
Unsure	

2. Did you have an alcohol problem before the injury

No	
Yes	

3. How much alcohol did you use to drink in a week

4. Did you have a drug problem before the injury

No	
Yes	

5. If the answer to this question is "yes" what drugs did you regularly use

The next 12 questions ask you about the accident and your reaction to it ***at the time***

6. Did you get other injuries as well as head injury

No	
Yes	

7. If you had other injuries what were they

8. Was anyone else injured or killed at the same time you were injured

No	
Yes	

9. If the answer to this question was "yes", can you describe what happened

10.How much did you think your life was in danger

1.....2.....3.....4.....5

No Danger | Moderate Danger | Extreme Danger

Can't Remember	

11.How much did you feel out of control when the accident was happening

1.....2.....3.....4.....5

Fully in Control | Still had Control | Completely out of Control

Can't Remember	

12.How scared were you

1.....2.....3.....4.....5

Not scared at all | Moderately Scared | Extremely Scared, Terrified

Can't Remember	

13.Was there anything gruesome or really disturbing about the accident

1.....2.....3.....4.....5

No there was nothing | Moderately disturbing | Very disturbing and horrible

Can't Remember	

14.How hard was it to believe what was happening

1.....2.....3.....4.....5

Easy to believe Everything was familiar | Things were strange but not unreal | Hard to believe. Things seemed unreal

Can't Remember	

15.How upset were you by the effects of the head injury

1.....2.....3.....4.....5

Not upset at all | Moderately upset | Extremely upset

16.How well were you cared for by medical people after the injury

1.....2.....3.....4.....5

Very badly | Moderately well | Very well

17.How well were you cared for by your family after the injury

1.....2.....3.....4.....5

Very badly | Moderately well | Very well

The next questions ask you about how you think and feel about the accident ***NOW***

18.How much could the accident have been prevented

1.....2.....3.....4.....5

Could easily be prevented | Could not be prevented

19.How much was someone else to blame for what happened

1.....2.....3.....4.....5

No one else was to blame | Others partly to blame | Another person or organisation was Completely to blame

20.How much were you to blame for what happened

1.....2.....3.....4.....5

Not at all | I was partly to blame | It was my fault entirely

21.How much can you prevent a similar event happening again

1.....2.....3.....4.....5

Easily preventable | Impossible to prevent

22.How angry are you at what happened

1.....2.....3.....4.....5

Not at all | Quite angry | Extremely angry

23.How much do you resent the way things turned out

1.....2.....3.....4.....5

Not at all | Quite resentful | Extremely resentful

24.How safe do you feel now from similar accident happening again

1.....2.....3.....4.....5

Very safe | quite safe | Not safe at all

25.How much are you in control of your life now

1.....2.....3.....4.....5

Completely in Control | Some Control Only | Out of Control

26.How clearly can you remember what happened when you were injured

1.....2.....3.....4.....5

Can't Remember Anything | Some Memory | Very Clearly

27.How clearly can you remember your thoughts and feelings at the time of the accident

1.....2.....3.....4.....5

Can't Remember Anything | Some Memory | Very Clearly

28.How much do you feel the victim of a senseless act

1.....2.....3.....4.....5
Not at all Moderately Extremely

29.How much have you **Lost** from having this happen to you

1.....2.....3.....4.....5
Nothing Quite a lot Everything

30.How much have you changed since the accident

1.....2.....3.....4.....5
Not at all Some change Feel a different person now

Can you answer the last two questions briefly in your own words

31.If you have changed since the accident, in what ways are you different

32.How has the accident affected you most

APPENDIX B

PCS MEASURE

CHECKLIST OF PCS SYMPTOMS DERIVED FROM AMENDED SCL90-R

Below is a list of problems and complaints that people sometimes have. Read each one carefully and select one of the numbered descriptors that best describes HOW MUCH DISCOMFORT THAT PROBLEM HAS CAUSED YOU IN THE LAST MONTH INCLUDING TODAY. Place that number in the open block to the right of the problem. Do not skip items and print your number clearly. If you change your mind, erase your first number completely. Read the example below before beginning.

Example

HOW MUCH WERE YOU DISTRESSED BY	Answer	*Descriptors*
		0 Not at all
		1 A little bit
		2 Moderately
Ex. Trouble remembering things.....	☐	3 Quite a bit
		4 Extremely

1.	Head aches.....	☐
2.	Faintness or dizziness.....	☐
3.	Overeating.....	☐
4.	Nausea or upset stomach.....	☐
5.	Feeling low in energy or slowed down.....	☐
6.	Feeling weak in parts of your body.....	☐
7.	Feeling everything is an effort.....	☐
8.	Trouble falling asleep.....	☐
9.	Awakening early in the morning.....	☐
10.	Sleep that is restless or disturbed.....	☐
11.	Trouble remembering things.....	☐
12.	Trouble concentrating.....	☐
13.	Feeling easily annoyed or irritated.....	☐
14.	Temper outbursts that you could not control.....	☐
15.	Getting into frequent arguments.....	☐

16. Feeling blocked in getting things done..... ☐
17. Feeling blue..... ☐
18. Feeling no interest in things..... ☐
19. Feeling hopeless about the future..... ☐
20. Feeling lonely even when you are with people..... ☐
21. Feelings of worthlessness..... ☐
22. Nervousness or shakiness inside..... ☐
23. Feeling fearful..... ☐
24. Trembling..... ☐
25. Worrying too much about things..... ☐
26. Suddenly scared but for no reason..... ☐
27. Heart pounding or racing..... ☐
28. Feeling tense or keyed up..... ☐
29. Feeling so restless you could not sit still..... ☐
30. Spells of terror or panic..... ☐
31. Difficulty making decisions..... ☐
32. The idea that something is wrong with your mind..... ☐
33. Having thoughts about sex that bother you a lot..... ☐
34. Having to do things very slowly to ensure correctness..... ☐
35. Difficulty seeing..... ☐
36. Difficulty hearing..... ☐
37. Ringing in the ears..... ☐
38. Alcohol intolerance..... ☐
39. Unusual sensitivity to light..... ☐
40. Unusual sensitivity to noise..... ☐
41. Having urges to beat, injure, or harm someone..... ☐
42. Shouting or throwing things.....

The following table lists the PCS symptoms with the corresponding questions in the checklist. Where there are several questions relating to one symptom, the mean of the scores was used.

Table B

PCS symptoms and corresponding Checklist items

- headache 1
- dizziness 2
- eating problems 3
- nausea 4
- fatigue 5, 6, 7
- sleeping problems 8, 9, 10
- memory problems 11
- concentration problems 12
- irritability 13, 14, 15
- frustration 16
- depression 17, 18, 19, 20, 21
- anxiety 22, 23, 24, 25, 26, 27, 28, 29, 30
- difficulty with decisions 31
- thinks something is wrong with the mind 32
- sexual problems 33
- doing things slowly to ensure correctness 34
- problems with seeing 35
- problems with hearing 36
- tinnitus 37
- alcohol intolerance 38
- hypersensitivity to light 39
- hypersensitivity to noise 40
- control of aggression 41, 42

APPENDIX C

QUESTIONAIRE USED FOR CLASSIFICATION OF PTSD

DSM-IV CRITERIA FOR PTSD MODIFIED FOR THIS STUDY

HAVE YOU HAD ANY OF THE FOLLOWING SYMPTOMS FOR LONGER THAN A MONTH

Write yes or no after each question

1. Do you get frequent images and thoughts about the accident
2. Do you have dreams about the accident
3. Do you sometimes feel as if the accident, or parts of it, were happening again
4. Do you get flashbacks of the accident
5. Do you feel really upset with things that remind you of the accident
6. Does your body get tense or shaky with things that remind you of the accident
7. Do you have to make constant efforts to avoid thoughts and feelings about the accident
8. Do you constantly avoid things or places that remind you of the accident
9. Do you sometimes feel that you can't remember the accident or important parts of it
10. Do you still enjoy doing all the normal activities you did before the accident
11. Do you feel really separate or different from other people
12. Do you often feel numbed or emotionally dead
13. Do you often sense that your future may be different from other people's
14. Do you have difficulty falling asleep
15. Do you often get irritable or angry
16. Do you have difficulty concentrating
17. Are you constantly on your guard
18. Are you jumpy and easily startled
19. Do the symptoms we have just discussed greatly affect your work, family life or social life

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For the purpose of this research, a DSM-IV diagnosis of PTSD was given when a reply of yes was given to these questions:

- one or more of questions 1-6 (Criterion B)
- three or more of questions 7-13 (Criterion C)
- two or more of questions 14-18 (Criterion D)
- question 19 (Criterion F)

IMPACT OF EVENT SCALE

On _____(date) you experienced _____(life event)

Below is a list of comments made by people after stressful life events. Please check each item, indicating how frequently these comments were true for you DURING THE PAST SEVEN DAYS. If they did not occur during that time, please mark the "not at all" column.

	Not at all	Rarely	Sometimes	Often
1. I thought about it when I didn't mean to.				
2. I avoided letting myself get upset when I thought about it or was reminded of it.				
3. I tried to remove it from memory.				
4. I had trouble falling asleep or staying asleep because of pictures or thoughts about it that came into my mind.				
5. I had waves of strong feelings about it.				
6. I had dreams about it.				
7. I stayed away from reminders about it.				
8. I felt as if it hadn't happened or it wasn't real.				
9. I tried not to talk about it.				
10. Pictures about it popped into my mind.				
11. Other things kept making me think about it.				
12. I was aware that I still had a lot of feelings about it, but I didn't deal with them.				
13. I tried not to think about it.				
14. Any reminder brought back feelings about it.				
15. My feelings about it were kind of numb.				

APPENDIX D

PART 2 SUBJECTS' RAW SCORES ON THE HEAD INJURY IMPACT QUESTIONNAIRE

Table D

Likert scale scores on the dimensions of the Head Injury Impact Questionnaire

Subject	Dimension												
.	Thr.	Con	Fear	Hor.	Sen.	Eff.	Prev	B.O	B.S.	Ang	Los.	Ch.	Tot.
1	0	1	0	0	1	2	1	1	1	2	4	3	16
2	0	2	0	0	1	2	4	2	3	3	4	4	20
3	5	3	4	3	4	4	3	5	1	5	3	2	42
4	5	4	5	5	3	3	2	3	3	2	4	4	43
5	1	3	1	2	4	4	2	1	2	5	2	2	29
6	1	1	3	1	4	4	3	1	5	3	2	4	32
7	0	3	*	*	2	2	*	*	*	4	*	*	*
8	5	4	4	4	3	2	4	5	2	3	2	3	41
9	0	1	0	0	1	2	4	5	1	3	3	5	24
10	0	2	0	0	1	2	4	2	3	3	4	4	25
11	0	3	0	5	0	3	1	5	1	3	3	5	29
12	1	3	2	1	1	1	3	5	1`	2	4	5	29
13	0	3	0	0	2	2	5	5	1	3	3	4	28
14	1	3	1	1	4	2	4	5	1	2	4	3	31
15	5	4	5	5	2	2	2	1	1	5	3	5	40
16	3	4	1	0	2	2	5	5	1	2	3	3	30
17	0	1	0	0	0	1	1	1	1	1	3	3	12
18	0	2	0	0	2	2	3	4	1	5	4	3	26
Total	27	47	26	27	37	42	56	55	28	56	55	63	

Thr = threat to life
Con = feeling out of control
Hor = feelings of horror
Sens. = inability to make sense of it
Eff. = negative early effects
Prev. = preventability

B.O. = blame other
B.S. = blame self
Ang = anger
Los = loss
Ch. = change in self
Tot = total impact score
* = no score available