

**TRAUMATIC BRAIN INJURY AND ATTENTION:
POSTCONCUSSION SYMPTOMS AND
INDICES OF REACTION TIME**

by

JOACHIM F.L. MURERIWA

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ABSTRACT

One of the consequences of traumatic brain injury is the postconcussion syndrome. The symptoms in this syndrome include headache, dizziness, poor memory, poor concentration, easy fatigue, drowsiness, irritability, sensitivity to light, sensitivity to noise, low alcohol tolerance, visual problems, auditory problems, nausea, vomiting, anxiety, and depression. Several factor analytic studies have shown that these symptoms load onto *cognitive* and *noncognitive* factors (Bohnen, Twijnstra, & Jolles, 1992). The aim of this study was to determine whether patients who report different symptoms also evidence differences in cognitive deficits, as indexed by reaction time.

For this purpose 106 subjects (mean age 25.92 years; SD=6.05) of both sexes were tested on 8 reaction time tasks adapted from Shum, McFarland, Bain, and Humphreys (1990). There were 54 traumatic brain injury patients (mean age 26.40 years; SD=6.23) drawn from three Pretoria hospitals. They were heterogeneous with respect to diagnosis and severity of injury. For the controls (N=52), the mean age was 25.43 years (SD=5.88). The eight reaction time tasks constituted 4 task variables, each with 2 levels. From these tasks, 36 reaction time indexes were derived. The indexes were classified into 4 groups, viz., reaction time (RT), movement time (MT), total reaction time (TT), and subtraction scores (SB, the difference between the 2 levels for each task variable).

RT reflects the *decision component* and MT reflects the *response execution component* of reaction time. Partial correlation coefficients for all symptoms ($p \leq 0,01$) showed that some symptoms were most frequently associated with RT whilst others were most frequently associated with MT. On factor analysis with varimax rotation, symptoms loaded predominantly with SB scores. Symptoms also loaded with different task variables, suggesting that they correlated with deficits on different stages of information processing. Taking into account possible methodological constraints that were discussed, these results confirm that different symptoms within the postconcussion syndrome correlate with different cognitive deficits. The correlations between symptoms and indices of reaction time are

moderated by the characteristics of the symptoms (frequency & intensity), and the duration since injury. These findings have significance for understanding the aetiology of the postconcussion symptoms and for planning treatment.

CHAPTER ONE

INTRODUCTION

Traumatic brain injury is a major source of disability both in South Africa and other parts of the world. Amongst the complications from these injuries are physical disability (Miller, 1990), personality change (DiCesare, Parente, & Anderson-Parente, 1990), the postconcussion syndrome (Brown, Fann, & Grant, 1994) and cognitive deficits (Burton & Volpe, 1994). All of these complications are important and legitimate subjects for investigation. In the course of one investigation, it is however not possible to study all the complications of traumatic brain injury in depth.

In the present investigation, I focus on the postconcussion syndrome. Factor analytic studies have shown that some of the symptoms load onto a *cognitive* factor whilst others do not. In the study by Levin, Gary, et al. (1987) for example, the symptoms poor concentration and poor memory loaded onto the *cognitive-depression* factor, whilst headache and dizziness loaded on the *somatic* factor. Bohnen, Twijnstra, and Jolles (1992) recently reported that the symptoms loaded onto two factors, namely, *post-concussive-cognitive complaints* and *emotional-vegetative* complaints. The aim of this study is to determine whether patients who report different symptoms also evidence differences in cognitive deficits, as indexed by reaction time.

The Postconcussion syndrome

Adams and Victor (1989) give the classic definition of concussion as an immediate traumatic event characterized by a loss of consciousness for a variable time, with transient physiologic changes, a period of retrograde amnesia, an absence of anatomic changes, and eventual recovery without deficit. This definition is accepted by those who subscribe to the view that the postconcussion

syndrome is largely psychological in origin (Miller, 1961; Mittenberg, DiGiulio, Perrin, & Bass, 1992). It will be made clear however, from Chapters 2 and 3, that concussion is associated neither with an absence of anatomic changes nor recovery without deficit.

After sustaining a concussion, some patients go on to develop what Strauss and Savitsky (1934) labeled as the postconcussion syndrome. Benton (1989) defined this syndrome as follows:

It is generally understood to refer to a condition in which a person who has sustained a concussion complains of a variety of somatic, cognitive, motor, or sensory disabilities which he attributes to the concussion. At the same time, convincing historical and clinical evidence of significant brain damage cannot be elicited. The typical history indicates that at the time of the accident and shortly thereafter, the person was comatose for a very brief period, if at all, and showed practically no retrograde and very little post-traumatic amnesia (Benton, 1989, p.3).

Benton further observed that there is often a striking discrepancy between the supposedly objective assessment of the physician and the subjective complaints of the patient. The symptoms that make up the syndrome include the following: Headache, dizziness, poor memory, poor concentration, easy fatigue, drowsiness, irritability, sensitivity to light, sensitivity to noise, low alcohol tolerance, visual problems, auditory problems, nausea, vomiting, anxiety, and depression (Cartlidge & Shaw, 1981; Jennett & Teasedale, 1981; Strauss & Savitsky, 1934; World Health Organization, 1992).

Estimates of the incidence of postconcussion symptoms after traumatic brain injury vary greatly. Studies have reported incidences of between 20% and 80% (Richardson, 1990; Rimel, Giordani, Barth, Boll & Jane, 1981; Rutherford, 1989). Rutherford, Merritt, and McDonald, (1977) reported that within three months of injury, an estimated 51% to 84% of patients reported postconcussion symptoms. The number of patients reporting these symptoms drops to 15% to

33% after one year (Rutherford, Merritt, & McDonald, 1978). The wide variation of estimates of the incidence of postconcussion symptoms has been attributed to methodological differences such as differences in the definition of mild head injury, the measures used to assess the symptoms, and the duration since injury (Dikmen, McLean & Temkin, 1986; Rutherford, 1989). Further confounding factors were recently enumerated by Jacobson (1995). These include the fact that studies differ widely in their use of controls, the inclusion of patients with past head injury, psychiatric history or substance abuse, timing of assessment, method of eliciting symptoms, presence of compensation claims, and attrition on follow-up.

The individual postconcussion symptoms themselves differ in their frequency of occurrence. For example, Oddy, Humphrey, and Uttley (1978) reported on the frequencies of the different postconcussion symptoms complained of by a group of 50 young adults who had been injured six weeks previously and had a PTA exceeding 24 hours. They found that 38% of the patients complained of having trouble remembering things, 35% complained of often losing temper, 35% reported becoming tired very easily, and 33% had difficulties concentrating when reading. Similarly, Mureriwa (1990) reported on a heterogeneous group of head trauma patients with a mean of 10 months since injury. Of the symptoms spontaneously reported by these patients, headache, dizziness, and anxiety were found to be the most frequent. Brown, Fann and Grant (1994) recently reviewed the literature and found that headache, dizziness, concentration, and memory problems were the most frequent symptoms following closed head injury. Of these symptoms, headache was the most common (Alves, Macciocchi, & Barth, 1993; Packard & Ham, 1994).

The incidence of postconcussion symptoms is related to the duration since injury. Rutherford (1989) identified what he labeled as early and late postconcussion symptoms. The early symptoms include headaches, dizziness, vomiting, nausea, drowsiness, and blurred vision. The late symptoms consist of headaches, dizziness, irritability, anxiety, depression, poor memory, poor concentration, insomnia, fatigue, and poor vision. In order to place the incidence of postconcussion symptoms in perspective, it is necessary to note the presence of these symptoms in non head-injured populations. These symptoms have been

reported to be common in the general population (Bohnen, Twijnstra, & Jolles, 1992; Wong, Regennitter, & Barrios, 1994) and also in non-head injured patients (Fullerton, Harvey, Klein, & Howell, 1981; Gouvier, Uddo-Crane, & Brown, 1988). Fox, Lees-Haley, Earnest, and Dolezal-Wood (1995) reported that similar symptoms are also common amongst non head-injured psychiatric patients.

Researchers in South Africa have not paid much attention to the postconcussion syndrome. This is surprising when one considers the fact that research elsewhere in the world has shown that postconcussion symptoms are the most frequent reason for medical referral after traumatic brain injury. Richardson (1990) for example, reported that 50 to 80% of patients who are admitted to hospitals after traumatic brain injury will complain of one or more of these symptoms. Furthermore, the postconcussion syndrome deserves much research attention because it is persistent (Alves, Colahon, O'Leary, Rimel, & Jane, 1986; Merskey & Woodforde, 1972; Rutherford, 1989), and is resistant to current treatments (Duckro, Greenberg, Schultz, & Burton, 1992; McMordie, 1988).

Cognitive Deficits

The Importance of Attention

Memory and attention are the most frequently cited cognitive deficits after traumatic brain injury (Evans, 1992). These two deficits are equally important, but attention was chosen as the focus of the present study because it is probably more ubiquitous. Sherrington (1940) described normal attention as possibly the highest form of mental integration. It plays an important role in many other psychological processes. Treisman and Gelade (1980), for example, pointed out that attention is crucial in perception, while Baddeley (1986) and Nissen (1986) pointed out that attention is important in learning and memory. Whyte (1992a) noted that attention improves the speed and accuracy of many tasks. Recently, LaBerge (1995a) summarized the major benefits of attention to the individual as follows: Attention enables individuals to make accurate perceptual judgements and actions; it enables individuals to make quick perceptual judgements and to act

quickly. Finally, attention enables individuals to engage in sustained processing of a mental activity.

Because attention plays such a crucial role in other psychological processes, when deficient, it can compromise attempts at rehabilitation in other spheres (Gray, Robertson, Pentland, & Anderson, 1992). A final reason for giving precedence to the study of attention is that as much as 10-15% of the population suffer from problems of attention (Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991). Deficits of attention are also found in many other psychological disorders (American Psychiatric Association, 1994; Van Zomeren & Brouwer, 1994). Among these are attention-deficit and disruptive disorders, mood disorders, and anxiety disorders. In recent years, researchers have shown that problems of attention are prevalent in individuals with the acquired immunodeficiency syndrome (Ingram, Bridge, Janssen, Stover, & Mirsky, 1990; Dunlop, Bjorklund, Abdelnoor & Myrvang, 1993). Any insights gained from investigating attention after head injury may therefore have wide application in other medical and psychological disorders.

The Assessment of Attention

There are many ways of assessing attention deficits, as will be shown in Chapter 4. The two broad approaches to the assessment of attention are the psychometric testing approach and the information processing approach (Shum, McFarland, & Bain, 1994a). In the former approach, psychologists use individual psychological tests or batteries of tests to confirm the presence of attention deficits. These tests are used to identify deficits in components of attention such as concentration, focused attention and divided attention (Ponsford & Kinsella, 1992). Some writers have criticized the use of such global tests of attention because they were empirically, rather than theoretically derived (Reitan & Wolfson, 1985; Shum, McFarland & Bain, 1994a). It has been suggested that the information processing approach is more theoretically tied to known cognitive concepts (Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991). In the information processing approach, the researchers measure the speed with which an individual performs specific and elementary cognitive operations. The speed of performance

on these operations is assessed through choice reaction time tasks. An important assumption in this approach is that information is processed in distinct, serial stages.

Deficits on stages of information processing have recently been investigated by several researchers. These include studies by Shum, McFarland, Bain, and Humphreys (1990), Murray, Shum, and McFarland (1992), Schmitter-Edgecombe, Marks, Fahy, and Long (1992), and Shum, McFarland, & Bain (1994b). The methodology used in these studies will be described in more detail in Chapter 4. The major criticism of the information processing approach is that by using highly specific laboratory tasks on comparatively small numbers of subjects, it lacks generality and clinical applicability (Lezak, 1985). In the present study, attention will be assessed using tasks adapted from the study by Shum, McFarland, Bain, and Humphreys (1990).

Aims and Rationale of Present Study

Because of the subjective nature of the postconcussion symptoms, discriminating the malingerer or the hypochondriac from a genuinely disabled patient is often difficult for clinicians (Gronwall, 1977; Youngjohn, Burrows, & Erdal, 1995). One way around this problem is to attempt to correlate the subjective symptoms with experimentally determined cognitive deficits. Only a few studies have examined the association between the presence of postconcussion symptoms and neuropsychological performance. These include the studies by Jakobsen, Baadsgaard, Thomsen, and Henriksen (1987), Leininger, Gramling, Farrel, Kreutzer, & Peck, (1990), and Gfeller, Chibnall & Duckro (1994). Jakobsen et al. successfully predicted the persistence of postconcussion symptoms by using reaction time tasks. These researchers gave simple reaction time tests to 55 patients one to five days after concussion. They found that the reaction time measured during the first few days of injury were significantly prolonged in patients complaining of concussion one month after injury, than in patients who reported no symptoms. After three months, patients with symptoms had longer reaction times than patients without symptoms, but the difference did not reach significance. The

study by Gfeller, Chibnall, & Duckro (1994) found that patients who reported postconcussion symptoms exhibited greater impairment on almost half of all the neuropsychological measures administered. Leininger, Gramling, Farrel, Kreutzer, & Peck (1990) found that symptomatic minor head injury patients, as compared with uninjured controls had deficits on several neuropsychological tests. These included the Category Test, the Paced Auditory Serial Addition Test -Revised (PASAT-R), Auditory Verbal Learning Test, and both the copy and memory trials of the Complex Figure Test.

In the present study, the intention is to contribute to the ongoing attempts to relate postconcussion symptoms to experimentally determined cognitive deficits. Further to this, the aim is to determine whether patients with different symptoms will show different cognitive deficits. Previous studies focussed on comparing patients and controls. In the present study on the other hand, the aim is to show that there are significant positive correlations between the presence of specific postconcussion symptoms and specific cognitive deficits. It is also the aim of this study to determine whether aspects of the postconcussion symptoms, viz., their presence, frequency, intensity, and severity correlate with indices of reaction time. The reasons for expecting these correlations will become clear from the literature review.

Outline of the study

The first three chapters deal with the review of the literature on traumatic brain injury, the postconcussion syndrome, and attention. The review of traumatic brain injury is done in Chapter 2 and begins with an overview of the ways in which the severity of traumatic brain injury is assessed. This is followed by a discussion of the mechanisms of traumatic brain injury, and the associated neuropathology and pathophysiology. One of the sequelae of traumatic brain injury is the postconcussion syndrome and this is discussed at length in Chapter 3. The bulk of this chapter is devoted to discussing the aetiology of this syndrome. Problems of attention feature prominently in the postconcussion syndrome. In Chapter 4, the characteristics and mechanisms of normal attention are discussed. In this

discussion, special emphasis is given to the information processing approach to the conceptualization of attention. This is followed by an overview of the neural bases of attention deficits after traumatic brain injury. The chapter ends with a description of the methods for assessing attention deficits. A brief summary of the review of the literature is given in Chapter 5, followed by the statement of the hypotheses. The pilot study is described in Chapter 6. Finally, in Chapters 7, 8, and 9, the methods, results and discussion are respectively described.

CHAPTER TWO

TRAUMATIC BRAIN INJURY

The phrase *traumatic brain injury* (TBI) refers to neural damage resulting from closed or open-head injuries following an accident involving the head (Stratton & Gregory, 1994). In open head injury, traumatic damage results from missiles such as bullets, or other sharp, pointed objects that forcibly penetrate the skull. The tissue damage is usually concentrated in the path of the intruding object (Strub & Black, 1988). Such injuries result in the exposure of the intradural contents to the atmosphere (Allen, 1986). Closed head injuries on the other hand, are non missile injuries (Hannay, 1982). These injuries are most frequently found after acceleration-deceleration processes, such as those from motor vehicle accidents. Closed head injuries are more frequently encountered in peacetime, whereas open head injuries are more common during war (McClelland, 1988). The present study focuses on closed head injury. In this chapter, I will discuss four aspects of traumatic brain injury. These are the severity of traumatic brain injury, epidemiology, the mechanisms of traumatic brain injury, and the neuropathology and pathophysiology of traumatic brain injury.

Severity of Injury

The three main measures of severity of brain injury are the duration of coma, the depth of coma, and the duration of post-traumatic amnesia (PTA) (Alexander, 1995). The advantages and disadvantages of these different ways of assessing severity will be discussed below.

Duration of Coma

Symonds (1928) first suggested that the duration of loss of consciousness (LOC) or coma is related to the severity of brain damage. He further suggested that the duration of unconsciousness depends on the amount, rather than on the location of brain lesions. Following on this, Russell (1971) proposed a four-category classification of patients based on the duration of loss of consciousness: Subjects who were unconscious for less than one hour were classified as mildly injured; those who were unconscious from 1 hour to 24 hours were moderately injured; those unconscious for one to seven days were severely injured; and those unconscious for more than seven days were classified as very severe. Bond (1983) criticized Russell's (1971) schema, stating that it over classified severe cases. He proposed instead, that mild brain injury should refer to loss of consciousness for less than one day; moderate injury as loss of consciousness for 1 to 7 days; severe injury as loss of consciousness for 7 to 28 days; and very severe injury as loss of consciousness for 28 days or more. Bond's classification can in turn be criticized for overclassifying mild cases. Current consensus is that mild traumatic brain injury involves loss of consciousness for 30 minutes or less (Kay et al., 1993).

The duration of loss of consciousness as a measure of severity of injury has been criticized for several reasons. Firstly, loss of consciousness is more closely associated with diffuse injury than with focal injury. For this reason, individuals with severe focal injuries may not be correctly classified (Sarno, 1988). Secondly, according to the centripetal model of cerebral concussion, loss of consciousness occurs only in injuries severe enough to affect the mesencephalic core of the brain (Ommaya & Gennarelli, 1974). Consequently, mildly injured persons would not be properly classified because these injuries may not necessarily affect the mesencephalic core of the brain. Thirdly, as Brooks, Bond, Jones and Rizvi (1980) pointed out, coma is usually brief and is seldom accurately measured. In such cases, one might incorrectly assume that an individual had not sustained brain injury. Finally, many studies have shown that the correlation of duration of coma

and outcome is usually low. Damage that is insufficient to cause coma may nevertheless lead to impaired cortical functioning associated with decreased cognitive abilities (Brooks, Campsie, Symington, Beattie, & McKinlay, 1986; Dikmen, Reitan, & Temkin, 1983; Rimel, Giordani, Barth, Boll, & Jane, 1981; Esselman & Uomoto, 1995; Rimel, Giordani, Barth, & Jane, 1982).

Depth of Coma.

A more widely used measure of severity of brain injury based on impaired consciousness is the Glasgow Coma Scale (GCS) (Jennett & Teasdale, 1977; Teasdale & Jennett, 1974). To determine a patient's Glasgow coma score, the examiner assesses the patient's eye opening response, the verbal output, and the motor responses. A fully conscious person shows spontaneous eye-opening, is oriented, and obeys commands. The patient in a deep coma on the other hand, will show no eye opening, no verbal response, and no motor response. Scores on the GCS range from 3 to 15.

Researchers have proposed different cut-off points on the GCS to classify patients into those with mild, moderate, and severe injuries. Teasdale and Jennett (1974) suggested that a score of eight or less reflected severe injury. Rimel et al. (1981) classified patients with initial GCS 9 through 12 as having moderate injury and those with 13 through 15 as having minor head injury. Born (1988) argued that the GCS is of limited scope because in severe cases, the score is determined solely by the patient's motor response. He proposed that clinicians should use the Glasgow-Liege Scale, which combines the Glasgow Coma Scale with the quantitative analysis of five brainstem reflexes. Stambrook, Moore, Lubusko, Peters, and Blumenschein (1993) recently also pointed out other limitations of the GCS. These are that GCS scores are frequently contaminated by alcohol or drug ingestion and other, non-neurological organ system trauma. The GCS is also criticized for having a restricted range in survivors. The GCS was not designed to make sharp distinctions in patients with less severe brain injuries. Matheson (1994) also pointed out that the duration of coma is frequently artificially prolonged

by medical treatment.

Post-traumatic Amnesia (PTA).

Russell (1932) described post traumatic amnesia as an early stage of recovery from TBI during which the patient is not sufficiently aware of the environment to be able to commit events to memory. Associated clinical features during PTA may include disorientation, defective attention, agitation, lethargy, and inappropriate and disinhibited behaviour and speech (Levin, 1989). Russell and Nathan (1946) measured PTA retrospectively to include the period from the time of injury to the time of full awareness, and the ability to retain a stable record of events. For the purpose of classifying the severity of head injury using PTA, Russell (1971) suggested that concussion with a PTA of less than one hour was mild. He proposed that concussion was moderate if the PTA was 1 to 24 hours and was severe if the PTA exceeded 24 hours. In most studies, the cut-off point for severe head injury is a PTA of 24 hours or more (McClelland, 1988b). This probably overclassifies severe cases. To overcome this, Matheson (1994) suggested the following classification:

Less than 5 minutes	very mild
5 to 60 minutes	mild
1 to 24 hours	moderate
1 to 7 days	severe
1 to 4 weeks	very severe
more than 4 weeks	extremely severe

More recent assessment procedures for PTA are prospective and exclude the period of coma. The first such prospective measure of PTA was the Galveston Orientation and Amnesia Test (Levin, O'Donnell, & Grossman, 1979). In prospective assessments of PTA, the measurements begin while the patients is in and emerging from PTA (Forrester, Encel, & Geffen, 1994). This is in contrast to retrospective assessment which is carried out in an interview after the state of PTA

has resolved. Retrospective assessments of PTA depend on the patient's subjective recollections, which makes them unreliable.

PTA has been shown to be a better predictor of outcome than duration of loss of consciousness (Brooks, 1984; Mandelberg & Brooks, 1975; Schacter & Crovitz, 1977). Long and Williams (1988) suggested that PTA might be a better predictor of outcome than duration of coma because it is of longer duration, and is closer in time to final outcome. Although PTA is a better predictor of outcome than duration of coma, it is nevertheless also a weak predictor (Brooks, 1976; Gronwall & Wrightson, 1981; Lishman, 1968; Mandelberg, 1976). Long and Williams (1988) suggested that the reason for this is that PTA is caused by diencephalic damage, a focal injury. According to the centripetal model of cerebral concussion (Ommaya & Gennarelli, 1974), diencephalic damage occurs only with moderate to severe injury. For this reason, PTA measures would not be a suitable measure for mild injuries. Despite these criticisms, PTA is still considered the best yardstick for predicting the severity of traumatic brain injury (Wilson, Teasdale, Hardle, Wiedman, & Lang, 1993).

Combined Indices of Severity

From the foregoing descriptions, it is clear that all the indices of severity of injury are inadequate for one reason or another. Nevertheless, as Alexander (1995) recently observed, "There is at present no biologically objective measure that quantifies the severity of neuropathology more accurately than the clinical measures of GCS, loss of consciousness (LOC), and PTA" (p. 1254). In order to increase the accuracy of outcome predictions, researchers and clinicians frequently use a combination of these indicators of severity. For example, Brown, Fann, and Grant (1994) defined mild traumatic brain injury as follows: "when the period of unconsciousness does not exceed 30 minutes, the GCS on admission is 13-15 and never falls below 13 on continued observation, and both a clinical neurologic examination and cranial CT scan (one assumes MRI could be substituted) are normal" (p. 16).

Wilson, Teasdale, Hadley, Wiedmann, and Lang (1994) justify the use of a

combination of indices by pointing out that these indices relate to different aspects of brain damage. They showed, for example, that although both PTA and coma were associated with lesions in central brain structures, only the PTA was related to hemispheric lesions as seen on magnetic resonance imagery (MRI). The definition of mild brain injury recommended by the Mild Traumatic Brain Injury Committee (1993) uses a combination of indices. They recommended that mild head injury is manifested by at least one of the following:

1. any period of loss of consciousness;
2. any loss of memory for events immediately before or after an accident;
3. any alteration in mental state at the time of the accident (eg, feeling dazed, disoriented, or confused); and
4. focal neurological deficit (s) that may or may not be transient;
but where the severity does not exceed the following:
 - . loss of consciousness of approximately 30 minutes or less;
 - . after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and
 - . posttraumatic amnesia (PTA) not greater than 24 hours (p. 86).

For moderate head injury, Williams, Levin, and Eisenberg (1990) proposed an initial (and lowest) GCS of 9 to 12 with or without positive radiological findings and loss of consciousness equal to or less than 20 minutes. Higher scores than moderate injury reflect severe injury. In other words, severe injury includes loss of consciousness for at least 30 minutes, a GCS equal to or less than 8, and PTA equals to or more than 24 hours.

Epidemiology of Traumatic Brain Injury

South Africa

South Africa has one of the highest accident rates in the world. Recently, Butchart, Nell, Yach, Brown, Anderson, Radebe, and Johnson (1991) reported that the incidence of non-fatal trauma in Johannesburg was 2,886 per 100,000 population. Of this number, 22,6% were injuries to the head and face. Injuries to the head and face are of special interest to neuropsychologists because such injuries may be associated with brain injury. The first major studies of TBI in South Africa date back to the Anglo-Boer war of 1899-1902 (De Villiers, 1984). According to De Villiers, the high rate of road accidents since then has been attributed to personal and extraneous factors. Amongst the personal factors are the high levels of alcohol consumption in South Africa; fatigue, which results from the fact that the country has long stretches of roads which connect sparsely scattered towns; and the low educational level of a large percentage of the population. The extraneous factors include the high traffic density in the cities and poor road design.

De Villiers (1984) also observed that assault was on the increase in South Africa. He attributed this to the population explosion in the lower socio-economic groups which leads to overcrowding, unemployment; and deculturalization. De Villiers (1975, 1984) observed that head injury caused by a knife is a common occurrence in South Africa. Butchart et al. (1991) confirmed this in their recent study which showed that in Johannesburg, sharp instruments are predominantly used during interpersonal violence.

Nell and Brown (1990) reported a major study on the epidemiology of traumatic brain injury in Johannesburg. Their study focused on morbidity, mortality and aetiology. The subjects of the study, who were aged between 15 and 65 years were classified into mild, moderate and severe on the basis of length of amnesia-unconsciousness and the GCS. The mild cases had a GCS of 13-15, and LOC of for 60 minutes or less. Patients with a GCS of 7-12 were classified as moderately injured. On the basis of GCS scores, this sample had 87.5% mild cases, and on

the basis of amnesia-unconsciousness 80.7% were mildly injured. The severity of injury was found to be unevenly distributed by race, with a significantly higher proportion of severe cases amongst whites. The severity of injury was also found to increase significantly with age for all three race groups. Nell and Brown used ICD-9 (World Health Organization, 1977) criteria for determining head injury diagnoses, and found that 89.16% had concussion, 1.86% had cerebral laceration or contusion, 7.96% had intracerebral Haemorrhage and 1.02% had unspecified intracranial injury. The Nell and Brown (1990) study also found significant differences across age, sex, and race.

With respect to morbidity, Nell and Brown (1990) found an overall TBI incidence of 316.42 per 100,000 population, with males outnumbering females by 4.80. Whites had the highest male to female ratio (40.15), followed by coloureds (7.81), and Africans (4.37). For all races combined, TBI increased with age from 15 to 45 years, after which it declined. For African males, the highest risk age groups were the 15 to 24 year olds and 25 to 44 year olds. Amongst the whites on the other hand, it was the younger age group (15 to 24 years) that was most at risk. The overall mortality was 80.73 per 100,000 population. The age range with the highest fatalities was the 25 to 44 year group, followed by those aged 45 to 64, and 15 to 24 years. For blacks, whites, coloureds, and Asians, the fatal TBI incidence was 88.47; 53.68; 45.01, and 46.23 respectively. There was thus a clear difference across race groups with respect to fatalities. Furthermore, for all race groups, there were more male than female fatalities.

Nell and Brown (1990) found that interpersonal violence was the largest single source of traumatic brain injury amongst South African blacks. It accounted for 50.95% of African nonfatal TBI, 39.53% amongst coloureds, 25% amongst Asians, and 10.14% amongst whites. Blunt instruments were found to account for most assaults. Traffic accidents on the other hand were more frequent amongst whites (69.57%). Amongst the Asians, coloureds, and blacks, traffic accidents accounted for 55.67%, 48.84%, and 25.56% respectively. The majority of white

motor vehicle accident victims were vehicle occupants, whilst the majority of black victims were pedestrians.

Cross-national Comparisons

The South African studies reviewed above clearly indicate that traumatic brain injury is a major health problem in this country and that it is distributed unevenly with respect to age, sex, and race. In order to place the South African context into proper perspective a limited review of the epidemiology of traumatic brain injury from one Western country, the USA, will be carried out. Several methodological constraints have been identified when attempting to make cross-national comparisons of the epidemiology of traumatic brain injury (Kraus, 1980). Hauser (1986) identified three reasons for this difficulty. Firstly, brain injury frequently forms part of an overall traumatic condition. It may therefore be classified as another type of trauma. Secondly, many brain-injuries that recover quickly and have a good outcome are frequently not recorded. Thirdly, the quality of medical care and record-keeping for trauma varies from country to country. Kraus, Rock, and Hemyari (1990) also highlighted further difficulties in comparing epidemiological studies both nationally and cross nationally. These difficulties include inconsistencies in the scope and definition of brain injury, differences in procedures for case finding, and the varying ways in which severity is measured.

In the USA, a major study, the National Head and Spinal Cord Injury Survey (HSCI) was carried out in 1974 to determine the extent of the problem nationally (Anderson, Kalsbeek, & Hartwell, 1980; Kalsbeek, McLaurin, Harris, & Miller, 1980). The HSCI survey determined that the incidence of TBI in the USA was 200 per 100,000 population. The incidence was twice as frequent in males than in females and occurred mostly in the 15-24 year age group. The most frequent cause of head injury was found to be motor vehicle accidents, which accounted for 49% of all cases. Motor vehicle accidents were also associated with the more severe injuries. The second most frequent source of injuries was falls (28.43%). Other causes, including assault, made up the remaining 22.57%. The most frequent diagnosis after head injury was concussion, which made up 74.5%,

followed by contusion and /or laceration, and haematoma which constituted 6.3% and 2.2% respectively.

In other subsequent USA studies, traumatic brain injury has been found to be the third commonest cause of death in the general population after vascular disease and malignant neoplasms (Askenasy & Rahmani, 1988). In adults under the age of 35, TBI is the major cause of death (Adamovich, Henderson & Auerbach, 1984). In the USA, as in South Africa, there is a much higher incidence of minor head injury than severe head injury. Population based studies of mild head injury in the USA have reported an incidence range of 120 to 280 injuries per 100,000 (Annegers, Grabow, Kurland, & Laws 1980; Kraus et al., 1984; Whitman, Coonley-Hoganson, & Desai, 1984). Recent studies have estimated that almost 90% of new traumatic brain injury patients sustain minor brain injury (Goldstein, 1990; Miller, 1993). South Africa is similar to the USA in that the victims of traumatic brain injury tend to be young males. The other similarity is that the majority of the victims sustain mild rather than severe injury. Finally, concussion constitutes the major TBI diagnosis.

South Africa differs from the USA in that the incidence of trauma is much higher. Nell and Brown (1991) found that overall, TBI occurrence is 58.2% higher in Johannesburg than in the USA. Amongst the South African blacks, coloureds, and Asians, the incidence is 97.88% higher. In South Africa, interpersonal violence contributes more to the incidence of trauma than motor vehicle accidents. Finally, in South Africa, the number of males who sustain injuries from motor vehicle accidents and interpersonal violence outnumbers that of females by a much larger margin than is the case in the USA.

The Mechanisms of Traumatic Brain injury

The mechanisms of traumatic brain injury are divided into two main categories, primary brain injury and secondary brain injury. Primary brain injury is the injury that occurs at the moment of impact, whereas secondary injury occurs after the impact. Secondary brain damage is a complication of primary brain injury. The duration during which secondary damage can occur varies from seconds to days.

Primary Brain Injury

History of the Study of Coup- Contrecoup Injuries

Many of the mechanisms of primary traumatic brain injury were known by the 1950's. Courville (1950) summarized what was known at the time. The ancient Greek scholars and physicians were aware of the *coup-contrecoup* phenomenon, whereby a blow to one part of the skull produces a fracture under the site of the blow. Damage to the brain occurs directly below the impact site as well as on the opposite side of the head. Interest in contrecoup injuries of the brain, as opposed to those of the skull, was initiated in the French Royal Academy of surgeons around 1776. Six main theories were since put forward to explain contrecoup injuries of the brain. These are the vibration or "echo" theory, the theory of transmitted force, the brain displacement theory, the pressure gradient theory, and the rotation theory.

The vibration theory. According to the vibration theory, the traumatic impact sets up vibrations which are reflected in damage to the opposite pole. The vibrations occur as an echo across space.

Theory of transmitted force. The proponents of this theory suggested that the impact resulted in a transmission of the applied force through the tissue of the brain. This force causes the contralateral structures to be thrust against the wall of

the skull.

Cranial deformation. The cranial deformation theory was also known as the *structure hoop* theory. According to this theory contusions result because the skull is like a resting hoop. When a resting hoop is struck on one side, the opposing arc approximates it. Because of this, the skull contralateral to the point of the blow is suddenly forced against the brain at this point, resulting in a bruise.

Brain displacement. According to this theory, contrecoup injuries result from the avulsion of the cerebral cortex from the overlying meninges.

The pressure gradient theory. This theory was based on the observation that there is a sudden fall in intracranial pressure opposite to the point of impact. This fall in pressure causes blood vessels at that point to rupture.

The rotation theory. Proponents of the rotation theory suggested that after a blow the brain is set in centrifugal motion in the direction of the line of force. In so doing, the brain is thrust against the irregularities on the interior of the skull. Of all the theories enumerated by Courville (1950), the rotation theory has since become the most influential. Because of the importance of this theory, it is appropriate to single it out for a fuller description.

Denny-Brown and Russell (1941) demonstrated the importance of rotation as a mechanism of injury by subjecting the heads of dogs and monkeys, which they used as experimental subjects, to a high rate of change. This procedure produced concussion, which they referred to as acceleration concussion. They further demonstrated that concussion produced by a crushing injury, in which the head is struck whilst it is supported on a hard surface (compression concussion), required a much greater force to produce the same effect. Following up on the work of Denny-Brown and Russell (1941), Holbourn (1943, 1945) applied rotational forces to gelatin models of the brain. He observed that contusions were produced, and that these contusions were maximal at the tips of the frontal and temporal lobes. Holbourn (1943) explained his results by suggesting that when the head receives a blow, the behaviour of the skull during and immediately after the blow, is determined by the physical properties of the skull and the brain, and Newton's laws of motion. Thus, following the blow, the brain rotates and forcibly comes into contact with the bony prominences of the skull.

Holbourn suggested that because of the brain's incompressibility and its feeble rigidity, it is injured by shear strains (the pulling apart of axons and the disruption of cell bodies) that develops as the brain is squeezed between the bony prominences. Although rotation was the main source of injury to the brain, a significant contribution was also made by other factors such as deformation of the skull, with or without skull fracture. Holbourne suggested that rotation and skull deformation were responsible for some intracranial haemorrhages and probably for concussion. Early support for Holbourn's hypothesis was provided by Pudenz and Shelden (1946). These researchers removed part of the skull of their experimental animals and replaced it with a lucite calvarium, so that they could observe the movements of the brain within the skull. From this procedure, they observed that the movements of the brain lagged behind the movements of the skull. Furthermore, the brain was least able to move at the tips of the temporal and frontal lobes because of the presence of the bony prominences. In this way, the tips of the temporal and frontal lobes were subjected to greater sheer strains than the other parts of the brain. Subsequent experimental studies such as those of Adams, Graham, Scott, Parker, and Doyle (1980) and Chu, Lin, Huang and Lee (1994) have confirmed that contusions occur mostly in the fronto-temporal regions, irrespective of where the head is struck.

Contemporary Theories

Pang (1985) proposed that the mechanisms of traumatic brain injury can be classified into acceleration-dependent and non-acceleration dependent factors. The rotation theory for example is a description of accelerational forces. Pang's classification incorporates most of the factors previously described by Courville (1950). In the next few paragraphs, Pang's description of the acceleration-dependent and non-acceleration dependent factors will be paraphrased.

Acceleration-dependent factors. According to Pang, acceleration is of two types, translation or linear acceleration, and angular acceleration. In translation, the resultant vector of a force applied to a rigid body passes through the centre of gravity, so that all the particles within the body will travel at the same acceleration

and in the same direction. In this way, the particles will not sustain intermolecular stress. Although the particles do not sustain intermolecular stress, translational acceleration nevertheless causes brain damage. This comes about because the acceleration causes both the skull and the brain to move, but with the brain moving more slowly than the skull. The differential speed has the effect of causing the brain to crowd towards the impact pole. When this happens, a pressure gradient is created at the impact pole and negative pressure is created at the antipole.

As the negative pressure at the anti-pole continues to a point below the vapour pressure, the liquid boils and forms bubbles as it turns into the gaseous state. Deceleration leads to condensation of gases and the bubbles will disappear. The rapid succession of formation and collapse of bubbles is called cavitation. Cavitation is responsible for damage to the anti-pole where it ruptures blood vessels and tears neural tissues diffusely. According to Pang, cavitation theory can explain traumatic damage to the cortical and subcortical areas, but it does not explain lesions to the deep brain structures where there is zero pressure.

In angular acceleration, the resultant vector of a force does not pass through the centre of gravity. Consequently, the body will rotate around its own centre of gravity. Because the head is attached to the cervical spine, some degree of rotation always occurs around the foramen magnum regardless of the direction of the impact vector. Pang (1985) cautions that both pure translation and pure angular acceleration are rare. What is more commonly found is a mixture of angular and linear acceleration. From this, the body will spin around its own centre of gravity whilst travelling linearly along the path of the translational vector.

Rotation is a form of angular acceleration and it is responsible for most brain damage after trauma. Pang proposed that the mechanisms of injury are as follows: When a blow inducing rotation is applied to the head, the skull rotates, whilst the brain initially remains stationary. In time the brain is also made to rotate by the frictional forces of the bony prominences, namely the falx and the sphenoid ridges, which produce shearing strain on the cingulate gyrus, the temporal poles, and the frontal poles. The bony prominences create shear strain by distorting the shape of the brain. Because the brain has a feeble rigidity, the shear strains lead

to the tearing of neural tissues in the deep portions of the brain. Although the shearing forces cause damage diffusely, they are more likely in certain areas, notably along the interfaces between substances of different densities such as between grey matter and white matter, the brain and blood vessels, and the brain and cerebrospinal fluid.

Non-acceleration dependent factors. Pang considered these factors to be rare and therefore to be of theoretical interest only. He divided the non-acceleration dependent factors into deforming and non-deforming mechanisms. The deforming mechanisms include impression trauma, ellipsoidal deformation, and skull vibration. Impression trauma results when the impacting surface is small. Initially the impacting object causes the skull to bend inwards. Next, the skull reverses, but instead of resuming its original configuration, it overshoots as a result of the inertia of the skull. By so doing, negative pressure is created at the point below the initial impact. This will have the effect of sucking up neural tissues and blood vessels, causing damage in the process. The mechanism involved in impression trauma is cavitation.

Ellipsoidal deformation occurs when there is a fronto-occipital blow to the supported head. The blow deforms the skull from its usual ellipsoidal shape to a spherical one. The spherically shaped skull will therefore have a larger volume. Because of this, destructive shearing strains will occur in and around the centre of the brain where a negative pressure will develop. This negative pressure develops because the portions of the brain at the anterior and the posterior will be forced to move towards the centre whilst the lateral sides of the brain will be forced to move towards the periphery of the sphere. The anatomical sites that are injured by this process are the walls of the ventricles and parts of the corpus callosum, and the hypothalamus. The mechanism of skull vibration is largely an extension of the theory of "echo" across space previously described and will therefore not be dwelt upon further.

Finally, Pang (1985) described one possible non-deforming mechanism, the mechanism of stress waves. The impact of the blow supposedly sets up shock waves which travel from the point of impact to the anti-pole. As these shock waves travel, they create zones of differential pressure where shearing strains may be

generated. Pang sees this mechanism, which is similar to the theory of transmitted force previously described, as being of little relevance because physical laws dictate that a shock wave may be expected only if the collision time is less than the travelling time of the acoustic wave in the head. These conditions are not met in blows to the head, and therefore stress waves are not generated.

Secondary Brain Damage

Secondary brain damage occurs for varying lengths of time after head trauma. Salazaar (1992) suggests that because the pathologic picture continues to evolve for hours and days, it is important to recognize that traumatic brain injury is a dynamic process. Much of the ultimate damage from head trauma appears to occur in a delayed or secondary fashion (Smith, Casey & McIntosh, 1995). Hypoxia and ischaemia are central mechanisms of cell injury within the nervous system (Bennett, O'Brien, Phillips, & Farrell, 1995). The other mechanisms are haemorrhage, haematoma, brain oedema, brain swelling, hydrocephalus.

Hypoxia and Ischemia

Under normal circumstances, the brain takes up about 25% of the body's oxygen supply (Snell, 1989). The term ischemia means an inadequate delivery of oxygen to the brain (Sahuquillo et al., 1993). After traumatic brain injury, deprivation of oxygen can result from cardio-respiratory arrest, reduced blood flow, and increased intracranial pressure related to the mass lesions such as haemorrhage and haematoma (Adams, Graham & Harriman, 1989). Hypoxic damage is usually seen in the arterial border zones between major cerebral arterial territories as well as diffusely throughout the cortex (Graham, et al., 1983).

According to Brierley (1976) the hippocampal regions have a selective vulnerability to hypoxia. Hall and Braughter (1993) showed that the free radicals and excitotoxic neurotransmitters released during hypoxia result in damage to the neurons.

Haemorrhage and Haematomas

By haemorrhage is meant intracranial bleeding. This occurs as a result of the rupture of blood vessels in different locations in the cranium. The three main locations affected during trauma are the space between the dura and the skull, that between the dura and the arachnoid matter, and within the brain substance itself (Snell, 1989). Haematomas are the blood clots resulting from haemorrhage. Like haemorrhages, haematomas are divided into extradural, subdural, and intracerebral.

Extradural. In extradural haemorrhage, the meningeal arteries and veins, particularly the anterior division of the meningeal are injured. According to Snell (1989), bleeding occurs under high pressure and tears up the meningeal layer of the dura from the internal surface of the skull. The haemorrhage frequently results from a fracture of the parietal bone. Walsh (1987) pointed out that the extradural haematoma has little relevance to neuropsychology because it is a neurosurgical emergency.

Subdural. Subdural haemorrhage results from the tearing of the superior cerebral veins where they enter the superior sagittal sinus (Snell, 1989). This happens when a blow to the head causes excessive anteroposterior displacement of the brain within the skull. Bleeding takes place under high pressure but the pressure is less than is the case with extradural haemorrhage. The blood accumulates in the potential space between the dura and the arachnoid. Subdural haemorrhage, unlike extradural haemorrhage spreads diffusely over the affected hemisphere. According to Jennett (1994) acute intracranial haematoma is the most common vascular complication after traumatic brain injury. Tsuchida, Harms,

Woodward, & Bullock (1996) described the importance of subdural hematoma as follows:

Acute subdural hematoma kills or disables more severely head injured patients than any other complication of intracranial trauma. The main pathological factor involved is ischemic neuronal damage, which is caused by raised intracranial pressure and local effect. (p. 104).

Haematomas lead to brain damage because they are mass lesions (Grubb & Coxe, 1978; Pang, 1985). They take up space in the cranium and thus lead to increased intracranial pressure. Adams, Graham and Harriman (1989) state that as a result of this pressure, extradural haematomas lead to a shift of the midline structures such as the ventricles, and compression of the midbrain. Subdural haematomas on the other hand lead to flattening of the convolutions of the brain and a narrowing of the sulci.

Intracerebral. According to Snell (1989) intracerebral hemorrhages occur as a result of the rupture of the thin-walled lenticulostriate artery, a branch of the middle cerebral artery. The haemorrhage occurs into the substance of the brain. It is found in more severe injuries. Like subdural hematomas, intracerebral hematomas can lead to increased intracranial pressure (Robertson, Gopinath, & Chance, 1995).

Oedema

Cerebral oedema refers to an increase in the volume of the water content of the brain. It is one of the normal physiological responses to tissue damage (Lezak, 1995). Adams and Victor (1989) distinguished between cellular and vasogenic oedema. Cellular oedema results from hypoxic injury which causes the cellular elements, viz the neurons, glia, and endothelial cells to imbibe fluid. Vasogenic oedema on the other hand results from the alteration of the blood-brain barrier caused by the trauma-induced physical disruption of the nerve tissue by the trauma. Cerebral oedema leads to brain damage by contributing to brain swelling.

Oedema, like haematoma, also causes brain damage by increasing the intracranial pressure (Lezak, 1995).

Brain Swelling

Brain swelling refers to the increase in the total volume of the brain. As was indicated in the previous section, such swelling can occur in some cases as a result of oedema. Diffuse brain swelling can also occur in areas adjacent to haemorrhages, haematomas, and contusions (Kaye, 1991). Kaye suggests that cerebral swelling involves a disturbance of the vasomotor tone. Bullock et al. (1992) state that there is evidence of reduced cerebral blood flow in the brain areas surrounding the swelling, which explains why there is always a surrounding zone of ischemic tissue and pyknotic dead neurones. The term *pyknotic* refers to a degenerative process in the cell, which involves the condensation and shrinkage of the cell nucleus. This occurs because reduced cerebral flow is associated with vasospasm (Kordestani, Martin, McBride, 1995). In addition to reducing blood flow, brain swelling also leads to raised intracranial pressure which, as previously pointed out, results in brain shift. It has also been suggested that axonal injury causes localized transport failures in the axon, which leads to swelling, as well as lysis of the axon, and Wallerian degeneration (Crooks, 1991; Povlishock, 1993).

Hydrocephalus

Hydrocephalus is an abnormal increase in the volume of the cerebrospinal fluid (CSF) within the skull (Snell, 1989). After traumatic brain injury, hydrocephalus results from atrophy in other parts of the brain. Bigler, Kurth, Blatter, and Abildskov (1992) found that because of the disintegration of severely damaged neuronal tissue, the brain shrinks. This allows the ventricles to increase their volume of CSF, leading to an increase in the ventricle-to-brain ratio. An increase in ventricle-to-brain ratio has been observed in association with diffuse axonal injury on the

cortex (Macnamara et al., 1992), and atrophy of the fornix and corpus callosum (Gale, Johnson, Bigler, & Blatter, 1995).

Neuropathology and Pathophysiology of Traumatic Brain Injury

Abnormalities In Brain Structure

Investigations of the abnormal brain structures after brain injury rely mostly on radiological techniques. The two most commonly used radiological techniques are the computerized tomography (CT) scans and magnetic resonance imaging (MRI). Both of these techniques produce three dimensional images of the brain. The CT scan and the MRI are equally sensitive in the detection of superficial soft-tissue injury (Orrison et al., 1994). There are however some differences between the two techniques. For example, Kurth, Bigler, and Blatter (1994) noted the following advantages that the CT scan has over MRI: "It requires less time to acquire the images, is superior to MR for visualizing bone, is less sensitive to motion effects (a common problem with patients, who are frequently agitated), is less expensive, and generally sufficient to make early diagnostic and treatment decisions" (p. 489). Focal and multifocal contusions as revealed by CT scans, are associated with an increase in the frequency in abnormalities of memory, concentration, speech, weaknesses of limbs, and consciousness (Eide & Tysnes, 1992). The CT scan is also more useful than MRI in identifying acute brain damage (Matheson, 1994).

Despite these advantages, the CT scan fails to identify many cases of brain injury. Astrup (1989) suggested that the CT may fail to identify lesions because it is only sensitive to focal injuries. It will therefore fail to identify a significant number of cases because, as was stated before, most closed head injuries lead to diffuse brain damage. According to Astrup, the CT scan fails to identify damage because diffuse injury occurs mainly at the cellular level, leaving gross anatomy unchanged. As a result, the brain looks surprisingly normal on CT scan,

considering the often serious clinical condition of coma.

The MRI has been found to be more sensitive than CT scan in identifying subtle neurological damage, such as that found in minor head injury (Eisenberg & Levin, 1989; Gandy, Snow, Zimmerman, & Deck, 1984; Jenkins, Teasdale, Hadley, McPherson, & Rowan, 1986; Levin, Williams, Eisenberg, High, & Guinto, 1992). Jacobs (1995) reports that within 1 week of head injury, MRI scans identify twice as many lesions as CT scans. Furthermore, the MRI has been shown to be superior to the CT in identifying cerebral contusions, shearing injury, subdural and epidural hematoma, and sinus involvement (Orrison et al., 1994). Both CT scans and MRI have been used to identify abnormalities arising from secondary brain damage. These abnormalities have already been described in detail. They include haematoma, oedema, brain swelling, and hydrocephalus. The other abnormalities that these imaging techniques can detect are contusions and diffuse axonal injury (DAI) which will be described below.

Contusions

Contusions are traumatic bruises on the surface of the brain, with resultant escape of the blood contents into the tissues of the brain (Matheson, 1994). Auerbach (1986) states that contusions have long been considered as the hallmark of traumatic brain injury and that they are usually correlated to some extent with the presence of skull fractures. Clifton, Grossman, Makala, Handel, & Sadhu (1980) observed that contusions are commonly bilateral, but asymmetrical. Contusions are focal injuries, found most frequently at the tips of the frontal and temporal lobes (Holbourn, 1943; Adams, Graham, Scott, Parker, & Doyle, 1980). The predilection for contusions on these two anatomical sites has been confirmed by both CT scans and MRI (Levin, Amparo, et al., 1987). The MRI reveals lesions in more detail than the CT scan (Adams, Graham, Murray, & Scott, 1982; Snow, Zimmerman, Gandy, Heir, & Deck, 1986). In minor head injury, small contusions are visible on MRI but not on CT scan (Auerbach, 1986; Jacobs, 1995).

Diffuse Axonal Injury

Strich (1956, 1970) was the first to describe diffuse axonal injury (DAI) after postmortems on people who had died after severe head injury. Although the axonal injury was diffuse, it was particularly evident in the white matter of the cerebral hemispheres, notably the corpus callosum and the brainstem. These initial findings were confirmed by subsequent studies (Adams, Graham, Murray, & Scott, 1982; Blumbergs, Jones, & North, 1989). Strich proposed that the DAI was caused by mechanical shearing forces at the moment of impact. Oppenheimer (1968) reported DAI in five patients who had sustained minor head injuries but had died of other causes. The pathology in minor head injury that Oppenheimer found differed from that of severe injury only in extent. Some recent studies have shown that DAI in the brainstem is much more widespread than had previously been thought (Hashimoto, Nakamura, Richard, & Frowein, 1993). Hilton (1995) explains that disruption of the white matter "in essence disconnects the cerebral hemispheres from any form of communication with other brain centres, in particular the reticular activating system" (p. 8).

Gennarelli et al. (1982) found that the shear strains and tensile forces that are associated with closed head injury lead to the formation of retraction balls. These retraction balls are, in essence, reactive swelling (Hilton, 1995). As the neurons are torn during impact, they retract and expel a ball of axoplasm. Furthermore, Gennarelli et al. found that in animal studies, the mechanism of mild head injury is the same as that for severe head injury. The only difference was that there was more damage in the later. Pilz (1983) showed that the frank separation from the cell soma occurs over a period of hours and that the large caliber, long-tract decussating axons are preferentially vulnerable to injury. Similarly, Lyeth, Jenkins, Hamm, & Dixon (1990) observed that cellular disruption may induce persistent metabolic/physiologic abnormalities but not cellular death or degeneration. In mild brain injury, the axons are not torn at impact. Instead, there is a focal compression or stretching of axons rather than a frank separation (Povlishock & Coburn 1989, Gennarelli, 1993).

Diffuse axonal injury is the most consistent pathology after moderate to severe head injury (Povlishok, Becker, & Cheng, 1986). This has been confirmed by more recent studies. For example, an increase in ventricle-to-brain ratio after traumatic brain injury is an indication of degenerative changes in the brain (Bigler, Kurth, Blatter, & Abildskov, 1992). Macnamara et al. (1992) reported a case, probably the first of its kind, of an individual who had CT scans taken 3 months prior to a severe traumatic brain injury, and after injury. The posttraumatic scan showed significant dilation of the ventricles, which reflected DAI. Neuropsychological tests which were performed on this patient showed global deficits, a finding which is consistent with diffuse brain injury. More recently, Bigler et al. (1994) compared the day of injury CT scans of 15 adult TBI patients with moderate to severe injury ($GCS \leq 10$) with scans taken two months after injury. These scans were contrasted with those of 21 medical controls who had normal scans. In 80% of the patients, there was demonstrable ventricle-to-brain ratio increases. According to Elson and Ward (1994), mild head injury, like moderate and severe injury, can also lead to diffuse axonal injury.

In spite of the successes reported in the use of CT and MRI scans, these radiological assessments still fail to identify many cases with brain damage (Newton et al., 1992). The reason for this is that CT and MRI procedures assess the structural integrity of the brain. In this way, they can identify contusions, and DAI but they cannot actively tap the current mental activity (Horton & Miller, 1985). They can identify some of the abnormalities of brain structure but cannot identify abnormalities of brain function (Gordon, 1994).

Abnormalities in Brain Function

Some radiological procedures were designed to assess brain function, rather than brain structure. There are two broad sets of measures. The first are the electrical measures electroencephalography (EEG), evoked potentials, and magneto-encephalography (MEG). The second set are the radio-isotope measures which include regional cerebral blood flow (rCBF), single photon emission computed tomography (SPECT), and positron emission tomography

(PET). In addition to radiological abnormalities, there are also abnormalities in neurotransmission and in neuropsychological function.

Electro-encephalography

Electroencephalography involves measurements of voltage changes from the brain. Bickford and Klass (1966) demonstrated that there is a slowing of EEG after minor head injury. Later, McFlynn, Montgomery, Fenton, and Rutherford (1984) reported acute theta abnormalities in individuals who had sustained minor head injury. Similarly, Montgomery, Fenton, McLelland, MacFlynn, & Rutherford (1991) demonstrated that there is a reduction in EEG theta power after minor head injury. Recently, McLelland, Fenton, and Rutherford (1994) reported that after head injury, there was a large amount of diffusely distributed abnormal slow wave activity. Furthermore, they also found that all the EEG channels showed significant correlations with symptom counts. A major difficulty with the use of EEGs in the past was that normal recordings have been reported in people with demonstrated brain damage (Horton & Miller, 1985). Nevertheless, Gevins et al. (1995) have shown that the more recent high-resolution EEGs can be quite sensitive and are likely to be put to greater use in future.

Evoked Potentials

The evoked potential is a change in the EEG record elicited by the momentary presentation of a sensory stimulus. These potentials can be classified by modality into auditory, visual and somatosensory. In terms of polarity, the waves can be either positive, designated by the letter P, or negative, designated by the letter, N. The waves are further labeled according to the number of milliseconds that elapse between the presentation of a stimulus and the appearance of the wave. So, the wave which occurs 100 milliseconds after a stimulus will be P100 or N100 depending on whether it is positive or negative. Finally, the evoked potential waves can be labeled in terms of their latency into early, middle, and late potentials. The term *event-related potentials* (ERP) is

nowadays preferred to the term evoked potentials, because it has now been realized that the electrical recordings do not just reflect the response *evoked* by a stimulus, but also a variety of situations *invoked* by the psychological demands of the situation (Donchin, Ritter, & McCallum, 1978). Coles and Rugg (1995) reviewed the ways in which ERPs are traditionally classified. Their description will be paraphrased below. Most reviews of ERPs classify the components into those that precede and those that follow events.

Event-preceding components. Two components have been identified. These are the readiness potential, and the contingent negative variation (CNV). The readiness potential was first identified by Kornhuber and Deeke 1965 (cited in Coles & Rugg, 1995). This is a slow, ramp-like negative shift that precedes the actual production of a voluntary hand movement by as much as 1000 ms. It is maximal at precentral sites. As the time of the impending hand movement approaches, the negativity becomes larger over the right scalp site, contralateral to the left hand movement. This has been referred to as the *Lateralized Readiness Potential*.

Walter, Cooper, Aldridge, McCallum and Winler (1964) showed that when a person is given a warning signal and is instructed to respond to or to anticipate a second stimulus, his or her EEG will show negative shifts in widespread cortical regions. Walter et al. described this phenomenon as contingent negative variation (CNV) or expectancy wave. This wave was maximal over the fronto-central regions of the brain. Loveless and Sanford (1974) showed that the CNV was actually composed of two components. The two components only become apparent when the interval between the two stimuli extended beyond the 1 sec originally used by Walter et al. At intervals of 6 sec and 15 secs, there are 2 negative waves, one following the first stimulus, and the other preceding the second stimulus. Loveless and Sanford labeled the first wave as "O" because they believed it reflected *orientation* to the stimulus. The second was the "E" wave, which they believed reflected *expectancy*. The researchers believed that perhaps the E wave was

actually the readiness potential.

Event-following Components. Event-following components have been broadly classified into exogenous and endogenous waves.

The exogenous are a set of components whose characteristics (amplitude, latency, and distribution) seem to depend on the physical properties of sensory stimuli, such as their modality and intensity...It has been claimed that their characteristics are immune to variations in the subject's state and to the nature of the interaction between the subject and the stimulus - that is, that they are not influenced by 'cognitive' manipulations (p. 15).

The endogenous components, on the other hand, depend on the nature of the interactions between the subject and the stimuli. They vary as a function of factors such as attention, task relevance, and so on. Coles and Rugg (1995) suggest that this dichotomy is a simplification of the real state of affairs. They point out that almost all of the sensory components have been shown to be affected by cognitive manipulations such as attention. According to these authors, one should speak of the exogenous-endogenous dimension that is roughly coextensive with time. "Thus, those ERP components that occur within the first 100ms of stimulus presentation tend to be exogenous, while those occurring later tend to be more endogenous" (Coles & Rugg, p. 16). The exogenous components include sensory components, the Nd (negative difference), which is also known as processing negativity, and the mismatch negativity (MMN).

The *sensory components* are the ERP deflections related to transmission of sensory information from the peripheral sensory system to the cortex and/ or the arrival of that information to the cortex. One of the most commonly researched sensory components is the brainstem auditory evoked potential (BAEP). The BAEP is related to auditory stimuli, and is evident in the auditory nerve as early as 10 ms. It can be detected in the cortex after 100 ms (Hillyard, 1993). A number of studies in the early 1980s reported that these waves were slowed in up to 27% of the victims of chronic head injury (Montgomery, Fenton, & McLelland, 1984; Narayan, Greenberg & Miller, 1981; Noseworthy, Miller, Murray, & Regan, 1981).

Rowe and Carlsson (1980) noted that these abnormalities are particularly associated with dizziness. After these initial publications, it was felt by many that brainstem auditory evoked potentials would be useful in the assessment of minor head injury. More recent investigations have, however, failed to confirm these initial optimistic reports (Schoenhuber, Gentilini, & Orlando, 1988; Rappaport, Hammele, & Rappaport, 1991). Campbell, Suffield, & Deacon (1990) suggested that this is because exogenous components such as the BAEP cannot detect structural damage of the higher centres or the functional nature of cognitive deficits. Although the brainstem auditory evoked potentials are frequently present long after injury, they correlate poorly with other measures of severity (Alexander, 1995). For the somatosensory processes, early evoked potentials have also been observed (Naatanen, 1992). In the visual sense however, only the later deflections seem to be evident. In other words, the deflections only become evident at the early cortical levels.

The *negative difference* (Nd) (Hillyard & Hansen, 1986) and the *processing negativity* (Naatanen, Gaillard, & Mantysalo, 1978) are descriptors of the same component. However, claims about the functional significance of each component are somewhat different. The Nd emphasizes the polarity and the operation used to identify the component. It is isolated by taking the difference between two ERP waveforms that are elicited in response to the same physical stimulus. "The critical comparison is between ERPs for the same stimulus when it is being attended versus when it is unattended. 'Processing negativity' emphasizes the fact that the component is related to some form of extra processing accorded to attended events on the basis of a preceding selection process" (Coles & Rugg, 1995, p. 17).

Hillyard and Hansen (1986) showed that the attended stimulus was associated with more negative deflections than the unattended, and the difference was maximal (auditory task) at 100 ms after the presentation of the stimulus. They concluded that the effect of attention was to modulate the amplitude of the N1 component. On the other hand, Naatanen, Gaillard, and Mantysalo (1978) argued that the effect of attention was to add a negative shift to the unattended ERP, a shift that is not specifically time-locked to the N1 component. They claimed the negative shift was associated with the processing of the target stimulus dimension.

The *mismatch negativity* (MMN) is a negative wave that was first described in relation to the physiological basis of the orienting response. Sokolov (1963) suggested that the following mechanisms might explain how the orientation reaction comes about: Repeated presentations of a stimulus results in an internal representation of the stimulus, a neuronal model. All incoming information is compared against such neuronal models. If the incoming stimulus is identical to the neuronal model of that stimulus there will be no reaction. In other words there will be habituation. If, on the other hand, the incoming stimulus is different from the internal representation, an orienting reaction will follow. The orientation reaction occurs because of the mismatch between the incoming stimulus and the neuronal model of that stimulus. The negative wave that is generated when there is such a mismatch is the mismatch negativity wave (MMN). It is an N200 wave.

Naatanen, Gaillard, and Mantysalo (1978) found that the MMN occurs even when the stimuli are unattended. They suggested that this wave reflected the automatic detection of physical deviance. Woldorff, Hackley, and Hillyard (1991) argued that this deviation does not reflect a purely automatic process, because its amplitude may be influenced by attention. NB: The mismatch negativity is N200a; N200's presence depends on the events being task relevant. In many instances, this later component co-varies with P300.

The most widely researched *endogenous wave* is the P300 (abbreviated as P3). This wave was first described by Sutton, Braren, Zubin, and John (1965). It is characterized by a parietally maximal scalp distribution and has a latency between 300-800 ms. Coles et al. suggest that the P3 is most widely researched because of its size (5-20 mv), and the ease with which it is elicited. The P3 has been separated into two components, P3a and P3b (Squires, Squires, & Hillyard, 1975). The P3a is frontal in origin whilst the P3b is parietal in origin. Several studies have shown that the P300 component is slowed after TBI (Levin, Gary, et al., 1987; Papanicolaou et al., 1984).

The latency of the P300 is a measure of the speed of cognition and has thus been used as an electrophysiological marker for the disorders of cognition (Pratap-Chand, Sinniah, & Salem, 1988). "The latency of the P300 component (measured in milliseconds) reflects the time taken by the individual to conclude

that a task-relevant stimulus has been presented" (Shores et al., 1990). This wave thus reflects, amongst other things, decision making. Recently, it has been demonstrated that the P300 latency decreases with patient improvement as assessed by neuropsychological tests (Onofr et al., 1991).

Cerebral Blood Flow

Taylor and Bell (1966) were probably the first to report that there is a significant slowing of cerebral circulation after traumatic brain injury. Reduced blood flow is detected using positron emission tomography (PET) scans and single photon emission computerized tomography (SPECT) (Gordon, 1994). The reduced blood flow renders the brain at risk for secondary ischaemia (Bouma & Muizelaar, 1992). Pogacnik (1989) reported on the differences in blood flow between regions of the brain after traumatic brain injury as follows:

Because of increased metabolism in the frontal regions, blood flow in the anterior temporal and central regions decreases (steal syndrome?). The interhemispheric differences can perhaps be accounted for by increased blood supply to the dominant hemisphere in the "deranged state" (interhemispheric steal?). Impaired adaptability of rCBF to metabolic needs is thus one of the demonstrable functional disorders in patients with the postconcussion syndrome (p. 314).

After traumatic brain injury, patients become hypermetabolic, hypercatabolic, and hyperglycemic (Roberts, 1995). This general increase in metabolism leads to a greater need for a supply in oxygen, which is carried by the blood. Injury to the brain is associated with reduced blood flow around cerebral contusions and intracerebral haematoma (Bullock et al., 1992). The cerebral blood flow is decreased by intracranial hypertension; cerebral vasospasm; occlusion, compression, or injury of intracranial nerves, hypocapnia, and hypotension (Robertson, Contant, Gokaslan, Narayan, & Grossman, 1992). Cerebral blood flow measures can identify more abnormalities than structural measures. For example,

Newton et al. (1992) recently assessed 19 patients, comparing the structural measures of CT scan and MRI with the functional measure SPECT. They found that SPECT identified 43 abnormalities, MRI identified 19, and CT scan identified only 13. Reduced cerebral blood flow has been shown to have significant correlations with neuropsychological deficits, notably problems of attention and memory (Ruff et al., 1994).

Neurotransmitter Abnormalities

Carlsson (1987) showed that after traumatic brain injury, there are alterations in arousal, attention and information processing, which are mediated in part by dopaminergic and noradrenergic neurons. Similarly, Meltzer and Lowy (1987) suggested that post-traumatic symptoms such as memory, anxiety, and aggression are mediated by abnormalities in serotonergic neurons. Hayes, Lyeth and Jenkins (1989) and Lyeth and Hayes (1992) reviewed the literature on neurotransmission abnormalities and noted that mechanical brain injury seems to lead to a large nonspecific release of both excitatory and inhibitory neurotransmitters.

The acute, net effect of TBI-induced agonist receptor interactions is excessive neuronal excitation. The resulting excitotoxicity significantly contributes to the pathophysiology of TBI. The excitation at the time of injury can result in functional deficits persisting long after the initial insult (Lyeth & Hayes, 1992, p. S469).

Many studies have noted an increase in the neurotransmitters glutamate and acetylcholine (Myseros & Bullock, 1995). The activation of the muscarinic cholinergic system located in the rostral pons contributes to components of behavioural suppression accompanying transient unconsciousness. A recent review by Hovda et al. (1995) found that the release of neurotransmitters results in a massive ionic flux, which, consequently, produces an increase in glycolysis. "This increase in glycolysis is followed by a metabolic diaschisis, which is related

to the degree and extent of behavioural deficits." (p. 903). Gualtieri (1995) suggests that these neurotransmitter abnormalities suggest a link with postconcussion symptoms:

The pathologic anatomy of CHI is reflected by the derangements in neurotransmission that come one step closer to elucidating the origin of the symptoms of PCS (*postconcussion syndrome*). The axonal tracts that are most likely to be disrupted in CHI are monoaminergic projections from the brainstem to cortical and subcortical structures. The majority of the monoaminergic neurons are located in the lower brainstem and from there project to all parts of the central nervous system (CNS). They make up a very expansive chemical network, part of the reticular formation, organized in tightly clustered groups with extensive homotypic interconnections and long projections to interacting target areas (Gualtieri, 1995, p. 130).

The neurochemical disturbances resulting from abnormal excitation may not be associated with structural alternations detectable at the light microscopic level. Bigler and Snyder (1995) propose that in some cases, there may be structural damage in mild traumatic brain injury that is below the threshold for detection by current anatomic imaging techniques. This is further reason for using assessment techniques that measure abnormalities in brain function, in addition to assessments of the structural integrity of the brain.

Neuropsychological Abnormalities

Neuropsychological assessments aim to identify abnormalities in a wide variety of psychological processes associated with brain injury. All psychological processes is potential targets for these assessments. The psychological processes include consciousness, sensation, perception, attention, memory, intellectual functions, emotional & motivational behaviour, and personality change. Numerous tests are currently in use to assess neuropsychological deficits after TBI. These are well reviewed in neuropsychological texts such as Lezak (1995).

Research using such tests has demonstrated neuropsychological deficits in patients. For example, Arcia and Gualtieri (1993) demonstrated an association between the presence of postconcussion symptoms and deficits on neuropsychological tests. The tests included finger tapping, continuous performance test, switching attention test, pattern comparison test, symbol digit substitution, and pattern memory test. Memory problems were most consistently associated with the neuropsychological deficits. The assessment of neuropsychological deficits can also be carried out using the information processing paradigms, the aims and rationale of which were described in Chapter 1. This is the approach to be used in the present study.

Summary

This chapter focussed on four aspects of traumatic brain injury, namely assessments of severity, epidemiology, mechanisms of injury, and pathology. The conventional indices of severity of injury are the duration of coma, the depth of coma, and the duration of post-traumatic amnesia. None of these measures is completely satisfactory as a measure of severity, when used on its own. Alexander (1995) points out however, that despite the inherent difficulties of these measures, they are still the best available. The utility of these measures is strengthened when they are used in combination, rather than singly. Wilson, Teasdale, Hadley, Wiedmann, and Lang (1994) recommend this use of multiple indices of severity, because these indices are sensitive to different forms of brain injury.

The epidemiological studies that were reviewed showed that South Africa has one of the highest accident rates in the world. As is the case in Western countries, the majority of victims of traumatic are young males and concussion is the most frequent diagnosis. The mechanisms of traumatic brain injury were divided into primary and secondary brain injuries. Ischaemia has been argued by many to be one of the most critical processes in secondary brain damage (Bennett, O'Brien, Phillips, & Farrell, 1995). Other secondary brain injury processes include hypoxia, haemorrhage, haematoma, oedema, brain swelling,

and hydrocephalus. Many of these processes are associated with raised intracranial pressure.

Traumatic brain injury is associated with several neuropathological and neurophysiological features. These were classified into abnormalities of brain structure and abnormalities of brain function. The abnormalities of brain structure include contusions and diffuse axonal injuries. Identification of these structural abnormalities has relied mostly on CT and MRI scans. These technologies have been very useful, but they have the short-coming that in some cases, the brain may look structurally normal, and yet be functioning abnormally. The abnormalities of brain function include slowing in aspects of the brain's electrical activities, slowing of cerebral blood flow, abnormalities of neurotransmission, and disturbed neuropsychological processes. All these abnormalities can contribute to the postconcussion syndrome, which is the subject of the following chapter.

CHAPTER THREE

THE POSTCONCUSSION SYNDROME: SYMPTOM CLUSTERS AND AETIOLOGY

Symptom Correlations and Symptom Clusters

In the past, there was some controversy as to whether or not the various postconcussion symptoms could be said to constitute a syndrome. Several writers argued that these symptoms did not form clear-cut clusters, therefore they did not really constitute a syndrome (Lidvall, Linderöth, & Norlin, 1974; Rutherford, Merritt, & McDonald, 1977). Contrary to these assertions, later researchers have since demonstrated strong correlations between pairs of postconcussion symptoms. For example, Mureriwa (1990) reported strong correlations between headache and dizziness, and between poor concentration and memory.

In addition to associations between pairs of symptoms, factor analytic studies have demonstrated symptom clusters within the postconcussion syndrome. Van Zomeren and Van Den Burg (1985) used principal components analysis of interview findings on patients who had sustained severe head injuries two years previously. From these interviews, they identified two clusters of complaints. The first group of complaints, which they labelled as impairment complaints, consisted of forgetfulness, slowness, poor concentration, and inability to divide attention between two simultaneous activities. These complaints were found to be positively related to severity of injury as measured by post-traumatic amnesia. The second cluster of complaints, which consisted of all other postconcussion symptoms such as headache and anxiety, were labelled as intolerances because they were not related to the severity of injury.

Levin, Gary, et al., (1987) used factor analysis on the symptoms reported

by 155 patients one week after injury. This analysis revealed five clusters of symptoms:

1. Cognitive/depression: thinking difficulties, poor concentration, recent memory problems, remote memory problems, and depression.
2. Somatic: dizziness, headache, vertigo, blurred vision, diplopia, and hearing difficulties.
3. Sensory/sleep: noise sensitivity, poor sleep, and hallucinations.
4. Gustatory/olfactory: loss of sense of taste, smell, and appetite.
5. Irritability/anxiety: anxiety, impatience, and temper.

Bohnen, Twijnstra, and Jolles (1992) identified two subgroups of patients with mild head injury. They found these two subgroups after administering a 26 item questionnaire which included postconcussion symptoms, cognitive-energetic complaints, and a series of emotional and vegetative complaints. Principal components analyses with varimax rotation were carried out on the responses of 71 patients ten days after trauma. The results of the analysis showed that the symptoms of headache, dizziness, and intolerance to environmental stimuli loaded together with items of decreased cognitive and work performance. Bohnen et al. referred to this group of symptoms as the *post-concussive/cognitive complaints*. The second subgroup was labelled as *emotional/vegetative*. It consisted of the items of emotional lability and depression, as well as some aspecific vegetative symptoms such as heart palpitations, wet hands, dyspnoea, and others. Jacobson (1995) recently classified the symptoms into somatic, and psychological symptoms. The somatic symptoms include headache, dizziness, and fatiguability. The psychological symptoms are subclassified into cognitive (poor memory and concentration), and affective (irritability, emotional lability, depression and anxiety). Because of the variation in the concussional symptoms across patients, it has been suggested that the postconcussion syndrome is a group of illnesses rather than one illness (Szymanski & Linn, 1992).

Aetiology of the Postconcussion Syndrome

For over 100 years, there has been major controversy about the aetiology of the postconcussion syndrome (Evans, 1994; Gualtieri, 1995). The two major points of view in the past were the view that the syndrome is the result of psychological factors, and the view that the syndrome has an organic basis. According to Trimble (1981), the idea that postconcussion symptoms might have a psychological basis was first suggested by Rigler in 1879. Rigler had observed that after a system of compensation for injuries sustained on the Prussian railways was instituted in 1871, there was an increased incidence of post-traumatic invalidism. From this, Rigler concluded that the patients' complaints were largely neurotic, arising from a desire for compensation. Erichsen (1882) held a contrary view to that of Rigler. According to Erichsen, even minor injuries could have resulted in genuine pathology of the spinal cord. Recently, Bohnen and Jolles (1992) suggested that the controversy about the aetiology of the postconcussion syndrome is fuelled by inconsistencies in the definition of minor head injury, the difficulty of objectively assessing postconcussion symptoms, and issues related to the methodology and design of studies on the outcome of mild head injury. The arguments for and against the two opposing views are discussed next.

Psychological Factors

In the past, the major psychological explanation for the postconcussion syndrome was that such patients had a compensation neurosis. Hyler, Williams, and Spitzer (1988) observed that although compensation neurosis is not listed in any official nomenclature, it is commonly used in forensic psychiatry in cases that involve litigation: "Traditionally, compensation neuroses involve the presence of physical or psychological symptoms following an injury, that seem out of proportion to the injury and are reinforced by claims of disability or the possibility of financial compensation" (p. 4).

Arguments in Favour of the Psychogenic Perspective

Perhaps the most well-known proponent of the compensation neurosis viewpoint of the postconcussion syndrome in the past was Henry Miller (Miller, 1961, 1966; Miller & Stern 1965). Miller (1961) based his thesis on an analysis of 200 cases of head injury referred for medico-legal evaluations and a follow-up study of 50 of those patients. The patients that Miller followed up had been found to have gross neurotic symptoms after an accident more than three years previously. From this study, Miller reported the following:

1. There was significant negative correlation between accident neurosis and the severity of injury as measured by the duration of unconsciousness or post-traumatic amnesia. Miller and Stern (1965) reported that the postconcussion syndrome was *conspicuously absent* in severe injury.
2. The neurosis was more common in people of lower socio-economic status than in the professional or managerial patients.
3. The most consistent clinical feature was the subject's conviction of unfitness for work.
4. The symptoms disappeared after the issue of compensation was settled, whether or not they won the application.

Further support for the psychogenic perspective comes from the following factors.

Support for the compensation hypothesis. Support for the compensation hypothesis has been suggested by studies on reattribution of symptoms, and studies on patients' desires for compensation. With respect to reattribution, Mittenberg, DiGiulio, Perrin, and Bass (1992) showed that patients are more likely than normal controls to underestimate the occurrence of symptoms identical to the postconcussion symptoms before trauma. Mittenberg et al. conclude from this that patients may reattribute benign emotional, physiological, and memory symptoms to their head injury. The reattribution comes about because of expectations of the

occurrence of postconcussion symptoms. Mittenberg, DiGiulio, and Bass pointed out that the generalizability of their study is limited by several methodological considerations. One of these considerations is that their head injury group was drawn from consecutive outpatient referrals for neuropsychological examination rather than from consecutive hospital admissions for mild head injury.

A recent study by Youngjohn, Burrows and Erdal (1995) used the Portland Digit Recognition Test with which they found that as much as 15% of patients undergoing neuropsychological evaluations for medico-legal purposes may be unconsciously or consciously motivated to do poorly. They suggested that this was because "Patients involved in workers' compensation or personal injury cases have a very unfavourable motivational context for neuropsychological assessment, in that they are rewarded with financial compensation for poor performance." (p.113). Finally, Binder, Martin and Rohling (1996) carried out a meta-analytic study of the literature on the issue of compensation. This study was based on 17 reports, covering 18 study groups, and a total of 2,353 subjects. They found that there was more abnormality and disability in patients with financial incentives despite having less severe injuries. The authors suggest that in assessing brain injured patients, the issue of secondary gain must always be considered.

Nonspecificity of symptoms to the postconcussion syndrome. Symptoms similar to those of concussion have been recorded in individuals who have not sustained a head injury. For example, as was mentioned earlier, these symptoms are prevalent in the general population (Wong, Regennitter, & Barrios, 1994), in non-head injured medical patients (Gouvier, Uddo-Crane, & Brown, 1988), and non head-injured psychiatric patients (Fox, Lees-Haley, Earnest, & Dozel-Wood, 1995).

One possible reason for the similarity of postconcussion symptoms to symptoms in other psychological disorders and controls is that stress may be implicated for all these groups (Mittenberg, Zielinski, & Fichera (1993). For individuals without brain injury, stress has been implicated in the following symptoms: headache (Ficek & Wittrock, 1995), dizziness (Schneider, Friedman, &

Fisher, 1995), memory problems (Diamond, Fleshner, Ingersoll, & Rose, 1996), fatigue (Libbus, Baker, Osgood, Phillips, & Valentine, 1995), irritability (Baldoni, Ercolani, Baldaro, & Trombini, 1995), poor concentration (Spurgeon, Gompertz, & Harrington, 1996), and anxiety and depression (Leonard & Song, 1996).

For the brain-injured individuals, the emergence and persistence of postconcussion symptoms is associated with social adversity before the accident (Fenton, McClelland, Montgomery, MacFlynn & Rutherford, 1993; McLelland, Fenton and Rutherford, 1994). It has been established that TBI victims have decreased social contact, decreased leisure activity, unemployment, marital and family disruptions, and problems with independent living (Morton & Wehman, 1995). These difficulties are compounded by the fact that most victims are young people in the early stages of establishing their independence. Finally, a brain-injured patient may have survived an accident in which other people died, and thus becomes liable to the experience of bereavement. Haynes (1994) recently showed that such bereaved patients had significantly greater decreased memory and attention abilities.

If the postconcussion symptoms persist, individuals may decide, or may be advised to litigate. Lees-Haley (1988, 1992) drew attention to the *litigation response syndrome*, whose symptoms are qualitatively similar to the postconcussion syndrome. Lees-Haley (1988) stated that the litigation response syndrome arises because for many people, litigation is a deeply frustrating and disturbing experience. The symptoms of the litigation response syndrome disappear after the settlement of compensation claims. Recently, Lees-Haley and Brown (1993) surveyed the complaints of personal injury claimants and confirmed the presence of many concussion-like symptoms. In similar vein, postconcussion symptoms can also be exacerbated by the issue of blameworthiness for the injury (Rutherford, 1989, Jacobson, 1995). An individual who can, and does blame someone else for the accident faces a different kind of emotional stress from an individual who blames himself or herself.

Another condition that may produce symptoms similar to the

postconcussion syndrome is post-traumatic stress disorder (PTSD). In the fourth edition of the diagnostic and statistical manual of mental disorders (DSM-V) (American Psychiatric Association, 1994), PTSD is classified as an anxiety disorder. The anxiety comes about because of the circumstances of the accident:

Closed head injury often occurs in terrifying circumstances-car accidents, attacks, industrial accidents-and-, if the injury is recalled, the experience of injury (something that the severe TBI patient never has) could be a potent generator of anxiety or even post-traumatic stress disorder or panic disorder. Anxiety in its own right, powerfully disrupts concentration and complex mental operations (Binder, 1986; Krapnick & Horowitz, 1981)(Alexander, 1995, p. 1257).

Criticisms of the Psychogenic Perspective

The weaknesses of the psychogenic view of the postconcussion syndrome have been highlighted by several authors. Judd and Burrows (1986) pointed out several methodological flaws in Miller's (1961) study. Firstly, the 50 patients that Miller followed up already had a predisposition to neurosis. Secondly, the injuries studied were heterogeneous, and the patients were not reassessed until two years after the finalization of claims. Furthermore, Miller inferred that patients were free from symptoms because they returned to work, rather than from a clinical examination. The study by Mittenberg et al. (1992) requires replication before its findings can be accepted. The authors themselves point out that their findings are weakened by the fact that they did not include a control group of patients with symptoms who were not litigating.

Methodological problems apart, a number of direct tests of Miller's (1961) claims have produced negative results. Several studies have shown that postconcussion symptoms persist even after settlement of compensation (Gfeller, Chibnall, & Duckro, 1994; Merskey and Woodforde, 1972; Tarsh & Royston, 1985; Weighill, 1983). Balla and Moraitis (1970) reported that the patients who do return

to work do so before the settlement of compensation claims. It has also been shown that large numbers of patients do not return to work even after the finalization of the litigation (Mendelson, 1995). Other studies have shown that there is no correlation between the postconcussion syndrome and compensation in either minor injuries (Rimel, Giordani, Barth, Boll, & Jane, 1981) or severe head injury (McKinlay, Brooks, & Bond, 1983).

Miller (1961) had claimed that postconcussion symptoms were confined to minor injuries. Kelly (1975) disconfirmed this as he found a high proportion of postconcussion symptoms in severely head injured patients. More important still, Kelly found postconcussion symptoms in severely head-injured people who were not claiming compensation. McKinlay, Brooks and Bond (1983) warn that failure to volunteer symptoms by the severely head injured does not necessarily imply the absence of symptoms. From their own study, McKinlay, Brooks and Bond observed that symptoms other than postconcussion symptoms were mentioned by patients because they were more dramatic. However, direct questioning uncovered postconcussion symptoms.

Perhaps the strongest argument against the psychogenic perspective are the findings of neurological abnormalities in association with symptoms, especially in mild head injury. Traditionally, concussion was equated with mild brain injury, and either an absence of, or minimal brain damage (Adams & Victor, 1989). There have been some dissenting views to this generally accepted definition of concussion. Symonds (1962) for example, argued that concussion was characterized neither by brief loss of consciousness nor by absence of damage to the nervous system. He pointed out that differences in duration of loss of consciousness were only quantitative, not qualitative. Irrespective of the duration of loss consciousness, concussed patients all pass through the stages of stupor and confusion on the way to recovery of clear consciousness. This view has been echoed by contemporary writers, such as Sweeny (1992), Parker (1994), and Esselman & Uomoto, (1995) who point out that brain injury can occur, not only in the absence of impact to the head, but also in the absence of loss of consciousness. In the present study, in line with these contemporary views,

cerebral concussion will be defined as a traumatic paralysis of the nervous system which is associated with impaired consciousness and/or amnesia, of whatever duration.

Organic Factors

Arguments in Favour of the Organic Perspective

A frequently cited reason for expecting postconcussion symptoms to have an organic basis is the high frequency and universality of the syndrome (McMordie, 1988). Pogacnik (1989) argued for this viewpoint as follows.

The occurrence of the symptoms in exactly the same form in patients of different ages, occupations, educational backgrounds and social levels, along with the fact that it is encountered only after craniocerebral trauma and not after other injuries (eg to the extremities, abdomen or chest), strongly suggests an organic cerebral basis for this syndrome (Karlin, 1976) (p. 313).

In similar vein, Brown, Fann, and Grant (1994) pointed out that these symptoms have increasingly been shown to have a predictable configuration. If the symptoms were the result of a desire for compensation, one would expect that the symptoms reported would vary widely between patients. This should be even more so for patients with less education, who are unlikely to be aware of the constellation of symptoms making up this syndrome (Aubrey, Dobbs, & Rule, 1989). The organic perspective is also supported by the presence of abnormal brain structure and abnormal brain function after traumatic brain injury. These include radiological abnormalities, cerebral blood flow abnormalities, and neuropsychological deficits, which were considered at some length in the previous chapter. The organic abnormalities associated with individual postconcussion symptoms have also been identified in some cases. These individual symptoms

are discussed below.

Headaches. McKenzie and Williams (1971) and Jackson (1977) suggested that concussion headaches are the result of vascular changes; muscle trauma, or radiculitis. The vascular changes in migraine headache are thought to take the form of alternating vasospasm and dilatation of blood vessels (Altchek & Vitori, 1990; Greenblatt, 1973). Allen (1986) pointed out that in almost all head injuries, there is an element of whiplash injury to the cervical spine, which is probably responsible for the headaches.

Evans (1992) also described different post-traumatic headache types. These include muscle contraction headaches, occipital neuralgia, supraorbital and infraorbital neuralgia, muscle contraction headaches, migraine, and cluster headaches. Packard and Ham (1994) recently suggested that nerve fibre damage, abnormal cerebral circulation, and neurochemical abnormalities may also be involved. Headache, is thus like the other postconcussion symptoms, notably memory and attention deficits, in that it has several types. Duckro, Chibnall, & Greenberg (1995) found that myofascial tenderness was positively related to measures of headache, anger, depression, and perceived disability and suggested that attention to muscle irritation is an important aspect of the workup of chronic posttraumatic headache patients.

Dizziness. The aetiology of dizziness, like that of many other postconcussion symptoms is not yet fully understood. Healy (1982) pointed out that minor head injury is often not an isolated injury. The minor head injury frequently occurs with further injury to the head, skull and neck. The result of this might be vestibular concussion which in turn causes post-traumatic vertigo and persistent dizziness, as well as tinnitus or hearing loss. Researchers have generally supported the view that dizziness is associated with brainstem dysfunction. Evidence for this comes from studies which show that brainstem conduction delay has been associated with persistence of symptoms (Montgomery, Fenton, McClelland, MacFlynn, & Rutherford, 1991).

According to Keane and Baloh (1992), the mechanism of post-traumatic positional vertigo is probably the dislodgement of calcium carbonate crystals from

the macula of the utricle. The dislodgement causes the crystals to become attached to the capula of the posterior semicircular canals. The ear's semi-circular canals are important in the sense of balance. Abnormalities such as the ones just described may thus result in the experience of dizziness and vertigo. Recently, Rubin, Woolley, Dailey, & Goebel (1995) demonstrated experimentally that individuals who had sustained minor head injury or whiplash had significantly more balance and postural problems than uninjured controls. Another recent finding is that persistent dizziness may be the result of unsuspected perilymphatic fistulas (Fitzgerald, 1995). These are ruptures that allow perilymph to leak from the inner ear to the middle ear space.

Anderson, Yolton, Reinke, Kohl, & Lundy-Ekman (1995) observed that dizziness unfortunately does not have a precise medical definition. "When dizziness is a presenting complaint, distinctions must be made between vertigo (a sense of false movement), near-syncope (a feeling of impending faint), disequilibrium (loss of balance), and ill-defined lightheadedness (an inability to concentrate or focus the mind)." (p. 545). These authors summarize the possible causes of dizziness as including conflicts between visual and vestibular information, vascular problems, adverse reactions to medication, psychological difficulties, systemic disease, and the effects of aging.

Fatigue. Fatigue may be the effect of damage to what Luria (1973) called the first functional unit of the brain. This unit, which is responsible for arousal, is made up of the reticular activating system and the medial aspects of the forebrain. Luria (1973) stated that when this unit is damaged one of the symptoms is that patients fatigue more easily. Gronwall and Wrightson (1974) suggested that fatigue after traumatic brain injury results from the patient's increased effort to perform as well as he or she used to do before the injury. This is the coping hypothesis explanation of the postconcussion syndrome.

Depression, anxiety, and irritability. Depression, anxiety, and irritability are due to damage to parts of the limbic system such as the septal region and the anteromedial thalamus (McClelland, 1988). It has been proposed that depression after trauma may also be due to the physical stresses which are applied to the

hypothalamus area of the brainstem during hyperflexion of the neck (Jackson, 1977; Martin, 1970; McKenzie & William, 1971). Depression is known to disrupt cognitive operations, particularly concentration, memory, and executive functions (Weingartner, Cohen, Martello, & Murphy, 1981). Such patients show PET abnormalities, with frontal hypometabolism (Dolan, Bench, & Brown, 1992). Gainotti (1993) found that emotional disorders result from the disruption of neural mechanisms subserving the modulation and control of emotional and social behaviour. With specific reference to depression, Jorge, Robinson, Arndt, Starkstein, Forrester, & Geisler (1993) found that the location of the brain lesion was associated with the development of major depression only in the acute stage. Transient depressive syndromes were associated with left dorsolateral frontal and/or left basal ganglia lesions. Persons with both anxiety and depression on the other hand had right hemisphere lesions. Jorge, Robinson, Arndt, & Forrester, (1993) further found that whereas acute onset depression occurring soon after injury is related to the brain injuries, late onset depression was mediated by psychosocial factors.

Memory. Memory deficit is like headache and attention deficit in that it takes several forms after traumatic brain injury. In the past 30 years or more, several dissociations of memory deficits have been identified. Milner (1965) for example, reported the case of an adult male patient (HM), who had developed severe memory disorder after bilateral hippocampectomy. HM was unable to commit new facts to memory. However, he could remember skills he had learned such as mirror drawing and pursuit rotor tests. The deficits observed on HM have since been confirmed in other patients with hippocampal damage (Victor & Agamanolis, 1990; Mattioli, Grassi, Perani, Cappa, Miozzo, & Fazio (1996).

Memory deficits after traumatic brain injury, like headache and dizziness, present in various forms. In a recent review of the literature, Nyberg and Tulving (1996) concluded that there are multiple long-term memory systems. The most widely researched are episodic, semantic, perceptual representation, and procedural memory: Episodic memory is a memory for biographical events. Semantic memory is memory for knowledge of the world, such as the meaning of

words. Perceptual representation refers to the images associated with a memory. Finally, procedural memory is the memory for actions, such as how to drive a car. Traumatic brain injury leads to impairment on some forms of memory, but not others. For example, it has been shown that traumatic brain injury leads to difficulties with episodic, but not semantic memory (Kinsbourne, 1987; Calabrese, Markowitsch, Harders, & Scholz, & Gehlen, 1995).

The hippocampus is not the only brain structure associated with memory deficits. A significant neurological correlate of memory is temporal horn volume (THV). THV is an indirect measure of hippocampal atrophy. Gale, Johnson, Bigler, and Blatter (1994) demonstrated that ventricular dilation, in particular THV, correlated with memory impairment in TBI. In addition to the increase in THV, Gale, Johnson, Bigler, & Blatter (1995) found that there is a significant reduction in most brain structures, including the fornix and the corpus callosum. The authors argued that the findings of an increase in THV and the reduction in brain structures is representative of diffuse white matter atrophy. The fornix has been shown to be important in the construction of episodic memories (Calabrese, Markowitsch, Harders, Scholz, & Gehlen, 1995).

Axonal injury has been shown to lead to deficits in central cholinergic transmission (Dixon, Taft, & Hayes, 1993; Myseros & Bullock, 1995). Cholinergic pathways are a component of memory systems (Squire & Davis, 1981). The memory problems found after traumatic brain injury are therefore in some instances also due to acetylcholine deficiencies. Dixon et al. (1995) suggested that memory deficits may be particularly associated with hippocampal cholinergic circuits. Apart from the above neuroanatomic sites and systems, which are damaged during trauma, it has been proposed that memory deficits may result from changes in information processing (eg attention problems) rather than damage to the neuroanatomical systems critical to memory (Reeder & Logue, 1994). Furthermore, being involved in an accident is a stressful event. Bremner, Krystal, Southwick, and Charney (1995) suggested that traumatic stress brings about physiological changes in the same regions as the regions responsible for memory. The brain structures that are affected both by stress and head injury are

the limbic structures, notably the hippocampus. They propose that therefore, traumatic stress may contribute to the amnesia seen after traumatic brain injury.

Poor concentration. This is one of the deficits of attention. Journals and books on traumatic brain injury rarely refer to poor concentration by itself. It is mentioned in the context of attention deficits, of which it is a component. The discussion of this symptom is thus well covered in chapter 4, which deals with attention.

Sensory deficits. Some of the visual and auditory impairments that arise after traumatic brain injury can be attributed to cranial nerve damage. Keane & Baloh (1992) point out that this is evidenced by the fact that the olfactory, facial, and the audio-vestibular nerves are damaged most often by blunt head injury. With respect to vision, there are variety of difficulties that have been noted after mild head injury. These include binocular, oculomotor, accommodative and visual field loss (Hellerstein, Freed, & Maples, 1995).

Low noise and light tolerance. Low noise and low light tolerance have been shown to be related to lowered thresholds in the auditory and visual senses (Bohnen, Twijnstra, Wijnen, & Jolles, 1991; Waddell & Gronwall, 1984). Cohen and Rein (1991) recently suggested that optometrists should routinely be involved in head trauma evaluations because accidents resulting in closed head trauma commonly affect the cranial nerves and can cause visual field defects, most commonly homonymous hemianopsia.

Nausea and Vomiting. The vomiting center is believed to be the nucleus tractus solitarius in the brainstem (Johns, 1995). Johns observed that the chemoreceptor trigger zone (the area postrema) in the floor of the fourth ventricle is a major afferent to the nucleus tractus solitarius. This trigger zone can be stimulated by pressure from hydrocephalus after closed head injuries.

Criticisms of the Organic Perspective

The factors that were enumerated earlier as supporting the psychogenic perspective can also be taken to be criticisms of the organic perspective. Additional criticisms of the perspective were recently summarized by Jacobson (1995, p. 686) as follows:

1. The persistence of postconcussion symptoms after organic deficits have largely resolved.
2. The variable prognosis of post-concussional symptoms after equivalent severity of head injury.
3. Symptom exacerbation in those without organic deficits
4. The impact of psychosocial factors (Fenton, McClelland, Montgomery, MacFlynn, & Rutherford, 1994).
5. The generally low correlations between organic (neuropsychological, EEG, BAEP, MRI scan) indices and postconcussion symptoms evident after 3 months (Lishman, 1988).

Youngjohn, Burrows, & Erdal (1995) pointed out that one of the most commonly cited evidence in favour of the organic perspective is neuropsychological test results. He further goes on to make the following important observation. "Unfortunately, neuropsychological test results may not be as objective as we might wish, because valid test results are dependent upon the best efforts for success on the part of the patient." (p. 113). Youngjohn et al., found that patients undergoing assessment for litigation purposes were motivated to do poorly, rather than well, on the tests. Another criticism regarding the use of neuropsychological tests was recently expressed by Alexander (1995) when he said, "Avoid the logical fallacy; *because everyone with a TBI from closed head injury has impaired concentration, it does not mean that in everyone with impaired concentration after closed head injury the cause is neurologic*" (p. 1257). Alexander goes on further to say:

There is not yet any reliable biologic measure of 'physiologicalness' - evoked responses, EEG, SPECT, or even statistical manipulation of neuropsychological tests. Studies that apply external measures to selected groups of the persistently symptomatic are doomed to find interesting correlations that shed absolutely no light on causation and thus no light on treatment." (p. 1258).

The Interactive Perspective

It is clear from the review of the psychological and organic explanations of the postconcussion syndrome that neither approach can fully account for the syndrome. There are valid points as well as valid criticisms of both sides of the controversy. It appears that the best approach is one which takes into account the interaction between the two sets of factors. Long and Novack (1986) suggested that there should be no place for dualistic thinking in the paradigm of minor head injury. According to Benton (1989), it was only by the second half of the twentieth century that the awareness of the multifactorial nature of the postconcussion syndrome came to be appreciated. Recently Bohnen and Jolles (1992) observed that in the view of many contemporary workers in the field, psychogenic and organic factors are so closely intertwined that it would be unnatural to try to separate them.

A good example of the interaction between organic and psychological factors is the *coping hypothesis* for the emergence of postconcussion symptoms (Gronwall & Wrightson, 1974; Hinkely & Corrigan, 1990 ; Van Zomeren, Brouwer, & Deelman, 1984). According to the coping hypothesis, traumatic brain injury leads to several cognitive abnormalities. The most important of these is a reduction in information processing capacity. To make up for this deficit, the patient exerts more effort.

This effort is an answer to the demands made by the social environment and the patient's own standards. Such demands are made specifically to

those patients who are not visibly handicapped, and whose injuries are not considered to be so severe as to prevent a complete resumption of previous activities. When the cognitive functions are not yet completely recovered, the resulting stress may lead to intolerances as secondary symptoms, especially in the less severely injured patients (Marsh and Smith, 1993 p. 554).

The intolerances which Marsh and Smith (1993) refer to include symptoms such as headaches and irritability.

Summary

In this chapter, the symptoms constituting the postconcussion syndrome and their aetiologies were discussed. Factor analytic studies such as those by Van Zomeren and Van Den Burg (1985), and Levin, Gary et al. (1987) have shown that these symptoms load into clear-cut clusters. For more than 100 years, there has been controversy in medico-legal circles about the aetiology of the postconcussion syndrome (Evans, 1994). Arguments have been put forward in favour of both psychological (Miller, 1961) and organic (McMordie, 1988) factors. Many contemporary writers now argue in favour of viewing this syndrome as the product of an interaction between psychological and organic factors (Bohnen & Jolles, 1992). The coping hypothesis (Gronwall & Wrightson, 1974; Hinkely & Corrigan, 1990 ; Van Zomeren, Brouwer, & Deelman, 1984) is a good illustration of how organic and psychological factors interact to bring about the postconcussion symptoms.

CHAPTER FOUR

ATTENTION: COMPONENTS, MECHANISMS, PHYSIOLOGY AND PATHOLOGY

When psychology was instituted as a scientific discipline at the turn of the nineteenth century, attention was one of the key areas of psychological investigation. At that time, attention was studied using introspective methods by researchers such as James (1890), and Titchener (1908). With the rise of behaviourism (Watson, 1913), however, the study of attention fell into disfavour because the concept of attention was considered as being too mentalistic. The proponents of gestalt psychology such as Rubin (cited in Luria, 1973) also rejected the concept of attention. The gestaltists argued that the selectivity and direction of attention were purely the result of the structural organization of the perceived field, and that the laws governing attention were thus nothing more than the structural laws of visual perception.

Hebb (1949) argued for the reinstatement of the study of attention in the mainstream of psychology. He pointed out that the existence of attention was undeniable, and that in fact, psychologists had all along been investigating it under the guise of concepts such as "attitude", "set", and "expectancy" (Gibson, 1941). In the wake of Hebb's publication, a number of experimental investigations such as those of Cherry (1953) were carried out in the 1950s. On the basis of investigations such as these, Broadbent (1957, 1958) formulated the filter theory of attention, which was to become very influential in the field of the experimental investigation of attention and set the stage for much of the work carried out in the past three decades.

By the early 1960s, attention had become fully accepted as a legitimate field of psychological investigation. Moray (1969) identified three reasons for this favourable change: The first was that psychologists had managed to redefine the concept of attention in stimulus-response terms. This redefinition resulted in the

concept of attention becoming more acceptable to behaviourally oriented psychologists. Secondly, after the second world war, and the technological advances that followed, it became necessary to find solutions to critical problems of communication because the advanced technologies generated rapid and extensive information. Lastly, the availability of new techniques and apparatus such as tape recorders eased the problems for the experimental studies of attention.

The Varieties and Components of Attention

William James (1890), one of the earliest and most influential psychologists of the time, defined attention as follows:

Everyone knows what attention is. It is the taking possession of the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration of consciousness, are of its essence. It implies withdrawal from some things in order to deal effectively with others (James, 1890, pp. 403-4).

Recently, Wells and Matthews (1994) defined attention more simply as “the selection or prioritization for processing of certain categories of information” (p. 19). This is the definition that will be adopted for the rest of this chapter. As attention became more widely experimented upon and theorized about, from the 1950s onwards, it soon became clear that there was no generally agreed definition of attention as James (1890) had claimed. This state of affairs was clearly demonstrated by Moray (1969), who reviewed the literature and identified six broad categories of the meaning of attention. These meanings of attention still

guide contemporary research and theory. Because of their ongoing influence, I will paraphrase Moray's list below:

1. **Mental concentration:** The person concentrates on some specific task, such as mental arithmetic, and tries to exclude all incoming stimuli which might interfere with the specific task.
2. **Vigilance:** This is a situation where nothing much is happening, but the observer is paying attention in the hope of detecting some event, whenever it does occur (watch keeping).
3. **Selective attention:** This is exemplified by the *cocktail party problem* described by Cherry (1953). An individual receiving several messages at once tries to select only one of them.
4. **Search:** A set of signals is presented and the observer hunts among them for some subset or single signal.
5. **Activation:** This is the everyday version of the *orientation reflex*. During activation the individual is getting ready to deal with whatever is going to happen next.
6. **Set:** Preparation to respond in a certain way, either cognitively or by an overt motor response.

Since Moray's (1969) paper, several attempts have been made to break down the concept of attention into a small set of components. As Stablum, Leanardi, Mazzoldi, Umilta, and Morra (1994) put it, "attention is not a unitary aspect of cognition, but rather comprises a variety of interacting processes and functions" (p. 603). After reviewing the literature I identified the following to be some of the ways in which theorists and researchers have tried to classify the components of attention.

1. Posner (1975): alertness, selectivity, and processing capacity.
2. Zubin (1975): selectivity, focus, and shift.
3. Pribram and McGuinness (1975): arousal, activation, and effort.

4. Shiffrin and Schneider (1977): automatic and controlled processing.
5. Posner, Walker, Friedrich, and Rafal (1984): disengage, move, engage.
6. Posner and Petersen (1990): orienting to sensory events, detecting signals for focal (conscious) attention, and maintaining a vigilant and alert state.
7. Mirsky, Anthony, Duncan, Ahearn, and Kellam (1991): focus-execute, sustain, encode, and shift.
8. Plude, Enns & Brodeur (1994): components of selective attention: orienting, filtering, searching & expecting.

Posner (1975) suggested that the various components of attention are not mutually exclusive. Furthermore, one can see from the above list of components that the schemes of the different theorists overlap a great deal. In the remaining part of this section, some of the above components will be described. For each of the components of attention, an attempt will be made to describe the corresponding neural bases. Because of its complexity and the large variety of activities with which it is associated, one can expect attention to have a complex neural base. Mirsky, Anthony, Duncan, Ahearn, and Kellam (1991) pointed out that the complexities of some of the models of attention proposed by cognitive psychologists are beyond our current knowledge of the correspondence between brain structure and brain function. In similar vein, Van Zomeren and Brouwer (1994) stated: " 'Attention' refers to a collection of states, processes, and abilities. It is impossible to relate all of its various aspects to separate cerebral structures" (p. 39). For these reasons, it has generally been found more useful to speak in terms of brain systems than of individual neuroanatomical structures when discussing the neural basis of attention.

Arousal

According to Duffy (1962), arousal refers to a variety of physiological states between the extremes of coma and excitation. Arousal is also a fragmented concept, taking as it does, three forms, viz cortical, autonomic and behavioural

(Lacey, 1967). An important component of arousal is the orientation reaction, first described by Sokolov (1963). Sokolov defined the orientation reaction as a nonspecific response to novelty. He further observed that the orientation reaction was accompanied by distinct physiological changes such as EEG desynchronization and momentary changes in heart rate and blood flow. For this reason, electrophysiologists often refer to the orienting reaction as *activation* (Van Zomeren & Brouwer, 1994). These changes are brought about by the sympathetic division of the autonomic nervous system. Some writers have argued that novelty *per se* is not enough for the elicitation of the orientation reaction. Additional concepts, such as the significance of the stimulus, were postulated as necessary conditions for the orientation reaction to be elicited by a change in stimulation (Bernstein, 1979; Maltzman, 1979). Gati and Ben-Shakhar (1990) observed that a mechanism that produces an orientation reaction to the slightest change in stimulation would not be functional.

The theory of neuronal models in the elicitation of the orienting response was discussed in chapter 3 (mismatch negativity, Sokolov, 1963). Pribram and McGuinness (1975) suggested that the hippocampus, which plays an important part in memory, compares incoming information with old information. If the incoming information is similar to the information in memory, the hippocampus inhibits arousal by the ascending reticular formation. On the other hand, if the information is novel, the hippocampus releases the reticular formation, thus allowing arousal to take place.

Montcastle (1978), Wurtz, Goldberg, and Robinson (1980), and Petersen, Robinson, and Morris, (1987), suggested that orienting to visual stimuli is controlled by the posterior attention system. This system is made up of the posterior parietal lobe, the lateral pulvinar nucleus of the posterolateral thalamus and the superior colliculus. Posner and Petersen (1990) showed that these three anatomical areas perform different functions in visual orienting: The parietal lobe first disengages attention from its present focus. The superior colliculus acts to move attention to the areas of the target. Finally, the pulvinar, which is involved in the filtering of sensory inputs, reads out data from the indexed locations.

Sharpless and Jasper (1956) distinguished between a phasic arousal pattern which is of short latency, brief duration, and is resistant to habituation; and tonic arousal, which is of longer latency, greater persistence, and is susceptible to habituation. The tonic arousal is mediated by the ascending reticular activating system (ARAS) below the mesencephalon, and phasic arousal is mediated by the ARAS above the mesencephalon. Moruzzi and Magoun (1949) demonstrated that arousal arises from the activities of the reticular activating system. Posner and Petersen (1990) expanded on this notion suggesting that one of the subsystems of attention is the *reticular component* which consists of the intralaminar thalamus, the midbrain raphe nucleus, and the locus coeruleus. The reticular activating system and related structures arouse the rest of the brain, probably because of the action of the noradrenergic, dopaminergic, and serotonergic fibers which they contain (Robins, 1986). Kinomura, Larsson, Gulyas & Roland (1996) found that when subjects shifted from a relaxed awake state to do an attention demanding reaction time task, PET recordings showed significant activation of the midbrain reticular formation and of the thalamic intralaminar nuclei. Trexler and Zappala (1988) following closely on the previous work of Magoun and Moruzzi (1949) and Luria (1966) proposed that there are three hierarchically integrated and interdependent anatomical systems which functionally regulate attentional processes. These are the brainstem, the diffuse thalamic system, and the thalamofrontal gating system. According to Trexler and Zappala, the brainstem is responsible for the tonic arousal of the telencephalon. The thalamic projection system is responsible for the phasic activation of the cerebral cortex, especially the associative cortex. The third system, the thalamofrontal gating system, which is physiologically and functionally connected with certain limbic structures (Nauta, 1971), is responsible for controlled or selective attentional processes. Some studies have suggested that although the cortex is aroused diffusely by the reticular activating system, the association areas are more densely innervated than the primary sensory areas (Clark, Geffen, & Geffen, 1989; Robins, 1986). The right hemisphere has also been thought to be more closely associated with arousal, particularly tonic arousal, than the left hemisphere (Jutai, 1984; Posner,

Inhoff, Friedrich, & Cohen, 1987).

Recently, Robins and Everitt (1995) reviewed studies on arousal, and concluded that the cortex is aroused from the brainstem by a number of distinct neurotransmitter systems. These systems perform different functions in arousal:

The ceruleocortical NA system seems to have a protective function of maintaining discriminability in stressful or arousing circumstances; the mesolimbic and mesostriatal DA systems play a role in the activation of output, whether cognitive or motor in nature; the cholinergic systems appear to enhance stimulus processing at the cortical level; and the 5-HT systems may serve to dampen the actions of the others-for example, by promoting behavioural inhibition and cortical de-arousal. (p. 715).

According to the schema proposed by Robins and Everitt (1995), the prefrontal cortex regulates its own inputs by sending feedback to the origins of the ascending arousal systems. The salience of the sensory inputs to the ascending systems is determined by descending inputs including novelty and conditioning.

Alertness

One can define alertness as a state of arousal characterized by an increased selective awareness of an aspect of the environment and a readiness to detect and respond to specified stimuli (Posner, 1975). As it stands, this definition incorporates some of the meanings of attention identified by Moray (1969). These are mental concentration, vigilance, selectivity, and activation. As previously stated, arousal can be divided into two forms, tonic and phasic. In like manner, alertness has been divided into tonic and phasic forms. Van Zomeren, Brouwer, and Deelman (1984) described the difference between tonic and phasic arousal as follows: Tonic alertness is continuing responsiveness to stimulation covering minutes or hours. The changes in tonic alertness occur slowly and involuntarily. They are mostly explained as resulting from physiological changes in the

organism, such as diurnal rhythms. Phasic alertness, on the other hand, occurs in anticipation of an event. The changes in phasic alertness occur rapidly and depend on the subject's interests and intentions.

In reaction time tasks, the contingent negative variation (CNV) has been taken to reflect vigilance or sustained attention during the 1.0 to 1.5 second period that typically separates the warning signal from the second, or target stimulus (Papanicolaou, 1987). Segalowitz, Unsal & Dywan (1992) showed that the CNV is more closely associated with frontal lobe activity than with activities in the posterior regions of the brain. The frontal lobe is the origin of the N200 which is associated with orientation, a precursor of alertness (Squires, Squires, & Hillyard, 1975). Drake (1990) suggested that the N200 reflects stimulus classification. Recent studies by Naatanen and Alho (1995) suggest that the MMN is generated in at least three brain areas including the frontal lobe: The main contribution to the MMN is the auditory complex, in the supra temporal plane. The other sources are the right lateral temporal cortex and the right frontal cortex. Naatanen and Alho suggest that the temporal sites reflect a preperceptual detection of a mismatch. The frontal sites, on the other hand "might be associated with conscious perception of, or attention switch to, stimulus change." (p. 315).

In addition to the N200, alertness is also associated with the P300. Karlin (1970) suggested that the P300 reflected a cessation of a state of alertness, a change of state from high to low arousal. Hillyard and Kutas (1983) proposed that this wave is elicited by task relevant stimuli that occur unexpectedly and require a motor response or a cognitive decision. Like the N200, the P300 is believed to be, at least partially, the result of a mismatch between the mnemonic templates of the frequently occurring stimuli and the rare stimulus (Halgren et al., 1980). The P300 is elicited by unattended events that produce orienting (Pritchard, 1981). A related idea is that the P300 reflects the resolution of uncertainty or a cessation of vigilance once the sought-after rare stimulus occurs (Papanicolaou, Loring, & Eisenberg, 1985).

The P3a, which predominates in the frontal areas, is related to alertness. It is linked to processes involved in the involuntary capture of attention by salient

events (Knight, 1991). Posner and Petersen (1990) proposed that there are three interrelated subsystems for visual attention. These are, orienting, target detection, and alerting. The ability to sustain the alert state depends heavily on the integrity of the posterior aspects of the right hemisphere (Heilman, Watson, & Valenstein, 1983). Posner and Petersen further found that the alerting system requires norepinephrine pathways which are lateralized more to the right hemisphere. During tasks that demand sustained vigilance, normal subjects show right frontal and parietal activation as measured by cerebral blood flow studies (Pardo, Pardo, & Raichle, 1990). This finding was confirmed by Pardo, Fox, & Raichle (1991):

These data identify and localize in healthy humans an anatomically distinct and asymmetric neural system mediating sustained attention to sensory stimuli. Vigilance then, is one cognitive component of human attention that is right-lateralized. The vigilance system encompasses both right prefrontal and right superior parietal cortices (p. 63).

Recently, Arguin, Cavanagh, and Joanette (1993) demonstrated that damage to the left hemisphere leads to a delay in the alerting effect of a warning signal for the processing of contralesional targets. An analysis of the lesions did not establish a clear correlation between attention deficit and a specific brain area. However, analyses of the volumes of the lesions suggested that "a mass effect, rather than lesion localization may be the most plausible anatomical account for the alerting deficit observed" (p. 320).

Selectivity

As Moray (1969) indicated, many early writers viewed selectivity as being attention *par excellence*. For example, James's (1890) definition of attention emphasized its selective aspects. Hebb (1949) defined attention as the selectivity of response. Later, Treisman (1969) defined attention as the selective aspects of perception and response. These definitions of attention imply that humans

perceive and respond to only a limited portion of the sensory inputs from the environment. Cherry (1953) suggested that such selectivity is necessary because humans have a limited processing capacity. To demonstrate this, Cherry used an experimental procedure called dichotic listening. She had subjects listen to two separate tape-recorded messages simultaneously delivered to the two ears. The subjects were instructed to shadow one ear, that is, to repeat the message as it arrived. The results of the experiments showed that the subjects could tell very little about the message delivered to the non-shadowed ear.

On the basis of selectivity, three types of attention can be distinguished. These are focused attention, divided attention, and sustained attention. Focused attention, as James (1890) stated, implies the withdrawal from some things in order to deal more effectively with others. As such, focused attention requires the ability to ignore distraction. The second type of selective attention, divided attention, refers to the ability to do two or more things simultaneously. The third type of selective attention is vigilance, also known as sustained attention. Vigilance is studied when interest is in long-term performance decrement, mainly in detecting infrequent and unpredictable weak signals, as a function of the time on task (Naatanen, 1992).

The selection of a channel for further processing in terms of the filter theory (Broadbent, 1958) is reflected by the emergence of the N1 component of the evoked potential (Hillyard, Hink, Schwent, Picton, 1973). Naatanen, Gaillard, and Mantysalo (1978) found that the P300 is related to more complex stimulus evaluations, such as selectivity. According to a review of the literature by Coles, Smid, Scheffers, and Otten (1995), the critical variables affecting the amplitude of the P300 are the subjective probability of the eliciting event, "and the relevance or utility of the event in the context of the task as appraised by the subject" (p. 95). The latency on the other hand is affected by events occurring before the elicitation of the P300. These have been found to be stimulus evaluation and categorization.

Clark, Geffen, and Geffen (1987) and Carlsson (1988) carried out some studies which showed that the thalamus acts as the filter for sensory inputs. They suggested that the filter is controlled by feedback systems modulated by

dopaminergic mesencephalo-striatal pathways. Trexler and Zappala (1988), whose model was discussed above, suggested that the thalamofrontal gating system was responsible for selectivity in humans. A review of the literature by Robinson and Petersen (1992) suggested that in animal models at least, the pulvinar of the thalamus is involved in the act of choosing relevant from irrelevant visual information.

For auditory and somatic information, the lateral and the medial pulvinar of the thalamus are involved. Cohen et al. (1988) investigated sustained attention in subjects carrying out an auditory discrimination task. The control tasks in this experiment were somatosensory stimulation and resting. Brain activity during the tasks was monitored through positron emission tomography. The investigators established that higher glucose metabolic rates in the mid-prefrontal cortex and lower metabolic rates in the anterior and the superior posterior cortices were associated with the processing of stimuli that are important to the organism in determining goal-directed behaviour. On the other hand, lower metabolism in the midprefrontal and hippocampal cortices with possible metabolism in the orbitofrontal and temporal cortices were observed in subjects receiving, and possibly processing stimuli that do not contain information important for behaviour. Cohen et al. suggested that some of these regional metabolic rates were linked with the processes of response extinction, habituation, or inhibition.

The role of the thalamus, particularly the pulvinar in selective attention has been confirmed in a recent reviews (Kinomura, Larsson, Gulyas, & Roland, 1996; Newman, 1995). Laberge (1995b) for example stated, "The pulvinar mechanism apparently is directly controlled by axon fibres from the posterior parietal cortex, whose cells are driven , in turn, both by bottom-up inputs originating in the eye and top-down inputs arising from the prefrontal cortex" (p. 649).

Focus, sustain, encode, and shift

These four elements of attention were proposed by Mirsky, Anthony, Duncan, Ahearn, and Kellam (1991). Mirsky et al. based their classification of the

components of attention in part on Zubin's (1975) list. Their intention was to identify components that can be correlated with brain functional systems. According to Mirsky et al. (1991) the *focus* element represents the ability to select target information from an array for enhanced processing. The *sustain* element means vigilance in the sense of the ability to maintain focus and alertness over time. *Shift* is the "ability to change attentive focus in a flexible and adaptive manner" (p. 112). Mirsky et al. (1991) attempted to provide evidence for the validity of this scheme by carrying out factor analyses of the performance of 203 adult neuropsychiatric patients and normals as well as an epidemiologically-based sample of 435 elementary school children on a wide range of neuropsychological tests.

Mirsky et al. found that the ability to focus depends on the activities of the superior temporal and inferior parietal cortices as well as the structures of the corpus callosum. These researchers adopted Mesulam's (1985) suggestion that the execution of responses relies on the integrity of the inferior parietal and the corpus striatal regions. With respect to the sustain element, Mirsky et al. stated: "Sustaining a focus on some aspect of the environment is the major responsibility of rostral brain structures, including the mesopontine reticular formation and midline and reticular thalamic nuclei"(p. 133). Finally, Mirsky et al. suggested that the encoding of stimuli depends upon the hippocampus and amygdala, whilst the capacity to shift from one salient aspect of the environment to another is supported by the prefrontal cortex. A recent review by Rafal and Robertson (1995) confirmed the findings that the parietal lobe, particularly the temporo-parietal junction, is involved in the disengagement of visual attention. Rafal and Robertson further found that the right parietal lobe may be critical for shifting attention between locations, whilst the left parietal lobe is critical for shifting attention between objects. In monkeys, the specific area responsible for the shift component of attention is believed to be Brodmann's area 7. Steinmetz and Constantinidis (1995) found that cells in this area respond to covert shifts of attention away from the point of fixation. This same area was found by Corbetta, Shulman, Miezin, and

Petersen (1995) to be activated by detection of conjunction features. In this study, subjects were required to search for signals whose position was indicated by colour, motion, or a conjunction of colour and motion.

Automatic and Controlled Processing

Schneider and Shiffrin (1977) and Shiffrin and Schneider (1977) distinguished between automatic and controlled information processing. As the term *automatic* implies, this form of processing does not require attention. Shiffrin and Schneider proposed that automatic processing does not require attention because it is well-learned in long-term memory. When inputs are being processed automatically they will be processed in parallel rather than serially. For this reason, such tasks do not interfere with each other. *Controlled* processing on the other hand involves search, rehearsal, and other processes which are demanding of attention. In controlled processing therefore, the division of attention is more difficult. With practice, tasks that previously required controlled processing can also become automatic (Anderson, 1987). According to Wells and Matthews (1994), "The key criteria for automaticity are thus *independence from resources*, and its *insensitivity to voluntary control*" (p. 28).

Effort and the Strategic Control of Attention

Kahneman (1973) wrote a very influential essay on effort as a component of attention. He viewed attention as having two major dimensions, the selective and the intensive. The selective aspect of attention has already been discussed in previous sections. By the intensive aspects of attention, Kahnemann meant that attention varies in amount. Increased attention is the result of increased effort and is correlated with physiological arousal. Pribram and McGuiness (1975) viewed effort as the activity which controls attention by coordinating arousal and activation. The manner in which this control takes place is as follows: Two reciprocal neural systems converge on the amygdala, which probably controls the emotional tone of arousal. The first system arises from the dorsolateral aspect of

the frontal lobe. Pribram and McGuiness propose that this input facilitates arousal. The other input arises from the orbitofrontal cortex and is supposed to have an inhibitory effect on arousal. Commenting on Pribram and McGuiness's model, Van Zomeren and Brouwer (1994) said that "The frontoamygdala influence may be conceived as a finely tuned determinant, controlling visceromotoric arousal initiated by the hypothalamic mechanism during orienting" (p. 43).

An important aspect of the Pribram and McGuiness (1975) formulation is that it makes reference to control. The idea that attention exercises a controlling function over other psychological processes has been suggested by several writers. Eysenck (1982) for example, suggested that effort is a nonspecific resource of limited capacity which is importantly involved in the performance of an enormous variety of tasks. Similarly, Posner (1975) suggested that effort is an overseer which is tied to concentration and the ability to respond to information rapidly. Shallice (1982) proposed that there is a supervisory attentional control system during information processing. The following description of the strategic control of attention given by Whyte (1992a) provides a clear picture of the nature of this supervisory function:

Effective performance involves a delicate balance between two opposing needs: The need to allocate attention in a goal directed fashion related to current performance priorities, and the need to attend to important unexpected environmental events that are not part of the current task. An excess of the former tendency results in lack of awareness of critical, but unforeseen information. An excess of the latter results in distractibility (p. 946).

According to Van Zomeren and Brouwer (1987), the term control refers to what Luria (1966) referred to as the executive functions of the brain. These functions comprise planning, programming, regulation, and verification of goal-directed behaviours. Because of the controlling function of effort, one can justifiably regard it as a higher order component of attention than the other

components such as alertness and selectivity. Auerbach (1986) suggested that in terms of the neurological substrates of attention, one can identify a group of components which he labeled as the *attentional control matrix*. This matrix includes arousal and alertness, vigilance, ability to initiate set shifting, ability to inhibit inappropriate set shifting, and speed of processing. The mental control components are believed to arise from the activities of the frontal lobes. This is consistent with Luria's (1966) proposal that the frontal lobes act as a kind of superstructure over the other two functional units of the brain.

Brain Systems and Attention

For all the components of attention discussed above, attempts were made to identify the neural basis of the components. It is clear from the various descriptions that multiple brain areas are involved in attention. This is consistent with the influential theory put forward by Luria (1966, 1973) regarding the role of the brain in psychological processes, including attention. It is therefore appropriate to discuss Luria's proposals about attention. This description will be followed by a more recent model of neural networks in attention.

Luria's Model of the Neural Basis of Attention

Luria (1966, 1973) cautioned that higher mental processes, including attention, cannot be strictly localized to specific brain structures. He proposed that the brain can be divided into three functional units which act concertedly to bring about higher mental processes. The first functional unit is made up of the ascending and descending reticular formation, some limbic structures such as the hippocampus, and the medial aspects of the forebrain. This unit, according to Luria, is responsible for regulating cortical tone, and for waking. The reticular activating system of the first functional unit sends projections to widespread areas of the brain to arouse them. Attention, like all other mental states, requires this background level of waking and arousal. The second functional unit of the brain is

located in the posterior regions of the brain. This includes the parietal, occipital, and temporal lobes. The unit is responsible for receiving, analyzing, and storing information. In discussing attention, Luria (1973) did not make specific reference to the role of the second functional unit. However, one may assume that he viewed the parietal, occipital, and temporal lobes to be involved as one pays attention to spatio-temporal, visual, and auditory information.

Luria's third functional unit is situated in the frontal lobe. This unit is responsible for the programming, regulation, and verification of mental activity. The frontal lobe is responsible for the higher voluntary forms of attention. In this, its essential role is to inhibit responses to irrelevant stimuli and to preserve goal-directed, programmed behaviour and higher forms of verification of activity (Konorsky, Lawicka, & Brutowsky, cited in Luria, 1973).

Neural Networks of Attention

Mesulam (1981, 1990) proposed an integrated cortical network consisting of four components which modulate directed attention within extrapersonal space. The first of these is the posterior parietal cortex which is responsible for the sensory representation of extrapersonal space. Inputs from the association cortices bordering on the parietal, occipital, and temporal lobes converge in this area. The second component consists of the limbic structures including the cingulate gyrus, the substantia inominata and the basal forebrain. These structures play a fundamental role in assigning motivational valence to complex events occurring in extrapersonal space. The third component, comprising the frontal eye-fields (part of Brodmann's area 8) and surrounding regions, are responsible for motor representation. These regions provide a stage of efferent integration for the initiation or inhibition of motor mechanisms involved in exploratory or attentive behaviour. The frontal eye-fields, together with the superior colliculus of the midbrain, modulate head and eye movements. Finally, there is the reticular component, which consists of the intralaminar thalamus, the midbrain raphe nucleus, and the locus coeruleus. The reticular structures are responsible

for the widespread arousal of cortical structures.

The posterior parietal cortex, the cortex around the cingulate gyrus, and the frontal cortex have reciprocal relations with each other. They all receive afferents from the reticular structures. The input and output structures that are relevant to directed attention are organized as follows: The inputs from the reticular structures, limbic structures, and the sensory association cortices all converge into an area of the dorsolateral area of the inferior parietal lobule. This dorsolateral area then sends outputs to the frontal eye-fields and to the superior colliculus. These outputs are believed to coordinate the sequences necessary for fixing the visual stimulus on the fovea of the eye, for scanning, exploring, fixating, and manipulating motivationally relevant events within extrapersonal space. The neural networks as described by Mesulam (1981, 1990) were recently supported in a review by LaBerge (1995a).

The Mechanisms of Attention

The theories that have been put forward to explain the mechanisms of attention can be divided into two groups, the bottleneck theories and capacity models (Kellogg, 1995). These two approaches will be discussed next.

Bottleneck Theories

The first major bottleneck theory was put forward by Broadbent (1957, 1958). Broadbent stated that the nervous system acts to some extent as a single communication channel. Information flows through the nervous system in stages. At some stage in the information flow, there is a filter which allows only a portion of the information to pass on for more complex processing. The selection can take place either in the earlier stages or in the latter stages. Bottleneck theories have also been referred to as *structural theories*, because they suggest that there is a

fixed location in the system for the bottleneck beyond which the capacity is limited (Naatanen, 1992). The limitation is evidenced in particular, by the difficulty or inability to carry out more than one task at a time, the problem of divided attention. Bottleneck theorists can be categorized into two types on the basis of whether they favour early or late selection. Early and late selection were recently defined by Mangun and Hillyard (1995) as follows:

From the psychological standpoint, early selection is taken to mean that the processing of a stimulus need not be completed before the event can either be selected for further processing or rejected as irrelevant. This view implies a bottleneck in information processing in that the brain protects higher, limited capacity processing systems from being overloaded by irrelevant inputs. In contrast, late selection theorists have argued that both attended and irrelevant stimuli are fully analyzed before any selection between them takes place (p. 41).

Broadbent's Early Selection Theory

Broadbent (1958) suggested that information coming from various channels arrives in parallel at the sense organs. A *channel* in this context means sensory inputs that have distinctive physical properties such as location in space, intensity, frequency, etc. The next stage in the information flow is the short-term buffer store where information is stored only for a few seconds. The short-term buffer store is a preattentive level in the processing of information. From this store, one class of events is selected to pass through a filter for further perceptual analysis by a limited processing capacity channel. Broadbent suggested that information is not selected at random to pass on from the short-term sensory buffer. The probability of a particular class of events being selected is increased by certain properties of the events such as physical intensity and frequency. The selection of channels is also influenced by certain states of the organism such as the drive states like hunger. Information that is not selected to pass through the filter decays in the short-term buffer store and is thus lost. On the other hand, the selected

information passes either into a long-term store where the conditional probabilities of past events are stored, or it is selected for response. If the information is not stored in long-term memory or selected for response, it goes back into the short-term store.

The early selection theories have received support from electrophysiological studies. Zani and Proverbio (1995) gave subjects a task to decide on the relative sizes of visual stimuli. They found early, enhanced latero-occipital P90 positivity as well as an occipital N115 negativity to relevant patterns. There was also a stronger, left-sided selectivity. A review by Hillyard, Mangun, Woldorff, & Luck (1995) also supported early selection theories for both auditory and visual attention. They found that the earliest evoked potential changes (20-50 ms for auditory and 70-90 ms for visual) were noticeable over the primary auditory and visual sensory areas respectively. They are thus modality specific, suggesting that "attention acts to modify processing in cortical areas that encode elementary stimulus features rather than fully analyzed pattern or object representations." (P. 679). The longer latency ERPs were found in association with brain areas that process multiple stimulus features and feature combinations, the association cortices. Mangun and Hillyard (1995) found that although there is early selection for both auditory and visual senses, it is still unresolved whether the two modalities differ in the cortical level at which the earliest selection takes place:

For visual attention, there has been no convincing evidence from ERP recordings (or from other neuroimaging techniques) for any stimulus selection at, or prior to, the level of the primary visual cortex. Rather, the initial effects of visual-spatial attention appear to modulate information transmission between the primary cortex and visual association areas of the occipital lobe. For auditory attention, however, the short latency of the earliest attention effect (20-50ms) and its localization in the superior temporal lobe are consistent with sensory modulation in either primary or

secondary auditory cortex (p. 77).

Late Selection Theories

Late selection theories arose because a number of research reports contradicted some aspects of Broadbent's (1958) filter theory. For example, Moray (1959) demonstrated that the filter seemed to have a selective block. He showed that in shadowing experiments, subjects are able to perceive important information, such as the subject's own name, coming through the to-be-ignored channels. This finding was replicated by Oswald, Taylor, and Treisman (1960). Similarly, Gray and Wederburn (1960) showed that in dichotic listening tasks, parts of the rejected message may slip in when they fit well into the relevant message. Findings such as these necessitated changes to Broadbent's original formulation of the filter model. Treisman (1960, 1961, 1969) proposed that incoming sensory information is analyzed, prior to the filter, for physical characteristics such as pitch, intensity, location, and so on. The filter then selects one channel for further processing. However, the non-attended channels are not completely blocked. Instead, they are attenuated, that is, they continue to be processed but are given less priority than the selected channel. Further on in the sequence of information flow, the attended and the attenuated channels arrive at a second stage of selection. Treisman (1960) described this second stage as follows:

Shadowing experiments suggest that there is a single channel system for analyzing meaning, presumably comprising the matching of signals with some kind of "dictionary" and its store of statistical probabilities and transition probabilities gradually learned through the continual use of language. If this is so, one should be able to avoid the "identification paradox" pointed out by Moray (1959, p. 246).

Treisman proposed that in the dictionary or store of known words, some

units or groups of words have permanently lower thresholds for activation, or are permanently more available than others (eg one's own name). The incoming attended message activates the relevant dictionary unit which in turn leads to a response. The attenuated channels also arrive at the second selection stage but they will fire only if the unit which it activates has a lowered threshold. Treisman's (1960) approach differs from that proposed by Broadbent (1958) not only in that the latter involved early selection, but also in that Treisman's theory involved two stages of selection, an early stage and a late stage. Both Treisman's approach and that of Broadbent differed from those who proposed a purely late selection model.

Deutsch and Deutsch (1963) were probably the earliest proponents of late selection. They proposed that each central structure which is excited by the presentation of a specific quality or attribute of the senses is given a preset weighting of importance. An individual will be alerted to the stimulus depending on two factors, namely, the level of general arousal and the importance of the message. Deutsch and Deutsch likened arousal to a fluctuating standard interacting with the preset weighting of importance. When the individual is highly aroused, he or she will attend to every incoming message. On the other hand, when asleep, the individual will respond only to the most important messages, such as one's own name. According to Deutsch and Deutsch (1963) and Deutsch, Deutsch, and Lindsay (1967), all sensory inputs are recognized at the highest level, that is, they are analyzed for meaning. The filter selects the sensory message for response, not for further analysis.

As a result of the contributions of the two-stage selection models and the late selection models, Broadbent (1970, 1971, 1982) modified his filter theory, so that it could incorporate these latter contributions. In his revisions, Broadbent (1970) introduced the notion of *stimulus set* and *response set* (pigeon-holing). By stimulus set Broadbent meant that sensory inputs which satisfy certain anticipated physical parameters are selectively processed whilst those that do not are attenuated or blocked. Broadbent defined response set as the selection of certain classes of responses as having higher priority of occurrence even if the evidence for them is not especially high. To clarify this further, Mulder (1983) explained that

if in the selection of a channel, controlled processing is applied to early aspects of information processing such as pitch and colour, stimulus set is said to occur. Response set on the other hand is said to occur when controlled processing is applied to late aspects of information processing.

In a simplified psychological model attentional mechanisms can act at either the input or output levels of information processing. At the input or sensory level they enable discriminations between relevant and irrelevant material and set priorities for the processing of input information, processes corresponding to selection or directed attention. At the output level they efficiently orchestrate the responses appropriate to the task, processes corresponding to motor intention" (Bench et al., 1993, p. 907).

Criticisms of the Bottleneck Theories

The first possible criticism of the bottleneck theories was already noted in previous sections. It is the fact that in shadowing experiments, meaningful information from the to-be-ignored channel often breaks through into awareness (Treisman, 1960). This means that, contrary to filter theory (Wells & Matthews, 1994) this information will have been analyzed for semantic content. The second criticism is related to the first. As Meyer et al. (1995) put it, the bottleneck theories lack computational flexibility. According to the theory, information is processed on a first-come-first served basis. This leaves no room for the processing of high priority information first. If this was the nature of our processing of information, we would have great difficulties adapting to changing circumstances and changing priorities. Finally, Meyer et al. (1995) noted that the assumptions of bottleneck theories seem to be neurophysiologically implausible:

Contrary to them, information processing in the brain is 'massively parallel' and 'distributed' throughout components of many interconnected neural

networks (Anderson & Hilton, 1981; Rumelhart & McClelland, 1986). There are no obvious brain sites that constitute immutable response-selection bottlenecks of the sort to which PRP (*psychological refractory period*) effects and other multi-task performance decrements have been attributed (Allport, 1980, 1987; Newman, 1987). (p. 165).

Capacity Models of Attention

The Effort Component of Attention

Capacity models of attention are also called resource models. In Best's (1986) view, the second phase in theory building with regard to the mechanisms of attention began with the reconceptualization of the problem which occurred with the publication of Kahneman's (1973) book on effort. Kahneman stated that the performance of any task requires some effort. Even when task demands are at zero, some effort, known as spare capacity, is expended in the continuous monitoring of the environment. This spare capacity is reduced as the individual pays more attention to the primary task. The reduction of capacity when an individual performs more than one task at a time implies a limitation of resources. Kahneman (1973) did not specify what these limited resources were. In Best's view, research suggested that the limited resources might be some basic and elementary processes of the nervous system. Whyte (1992a) suggested that the limited resource might be arousal.

As was previously mentioned, Kahneman (1973) emphasized the fact that attention has both selective and intensive aspects, and that the latter was related to effort. Van Zomeren and Brouwer (1994) acknowledged this concept by suggesting that attention may be said to be qualified by selection and quantified by intensity. To explain the factors that influence the intensity of attention Kahneman cited the work of Berlyne (1960, 1970). Berlyne, whose work focused only on involuntary attention, had suggested that the intensity of attention was related to the level of arousal, alertness, and activation. Berlyne further suggested that the

level of arousal of an organism is largely controlled by the properties of the stimuli to which it is exposed. The properties of the stimuli include collative variables such as novelty, complexity, and incongruity.

According to Kahneman (1973), the study of attention at the time dealt mostly with involuntary attention and thus made little reference to arousal and the intensive aspects of attention. He suggested that the intensive aspects of attention applied to voluntary attention as they did to involuntary attention. He proposed that voluntary control over effort is limited. The effort invested in a task is determined mainly by the intrinsic demands of the task.

Multiple Resources and Resource-free Processing

The idea that there is a single, limited processing capacity has been questioned by some. For example, Allport, Antonis, and Reynolds (1972) proposed that there are several processing mechanisms rather than just one. Because of this, dissimilar tasks which rely on different resources do not interfere with each other. This idea was echoed by Wickens (1984) who suggested that one should view simultaneous task performance in terms of *time sharing*:

Tasks are assumed to demand resources for their performance, and these resources are limited in their availability. Therefore, when the joint resource demand of two tasks exceeds the available supply, time-sharing efficiency drops and will be more likely to do so as the difficulty of either component task increases (p. 63).

Navon (1984) argued that all information processing, not just automatic processing, is resource free. He proposed that there is no central processing capacity. The deterioration of performance in dual tasks is due to outcome conflict between two processes rather than to a competition for a limited resource. Recently, Meyer et al. (1995) proposed a new theoretical framework for understanding processing resources, the executive computational model. They

propose that there is "no immutable decision or response-selection bottleneck for executing task procedures at a central cognitive level" (p. 173). Instead multiple procedures can be executed simultaneously with distinct sets of production rules. For example, a person can drive and talk on the cellular phone at the same time because the two activities rely on different production rules, making it possible for them to occur in parallel. The reason why people may not be able to do more than two tasks at once is that there is a limitation of the peripheral sensory and motor mechanisms, and not limited cognitive capacity. For example it is impossible to keep both hands on the steering wheel and to hold the telephone at the same time. The executive processes may, in this instance, engage in flexible task scheduling (ie when approaching an intersection, the individual stops using the phone and uses both hands for driving. He will resume use of the phone after crossing the intersection).

Attention and the Concepts of Information Processing

Mulder (1983) maintains that the main assumption of the information processing approach is that there are a few symbolic, computational operations such as encoding, comparing, locating, storing, retrieving, deciding, etc, which ultimately account for intelligence. As Tromp and Mulder (1991) put it, "cognition refers to the basic ability of the brain to analyze, store, retrieve, and manipulate information in order to solve problems" (p. 821). Because of the dominant position of the information processing approach, it is not surprising that the theories of attention, starting with Broadbent (1958), discuss attention within the framework of information processing. For example, the bottleneck theories discussed in a previous section are attempts to explain how sensory information coming from multiple channels gets selected for further processing. The capacity theories are also concerned with the issue of information. They grapple with the question of whether or not the resources required for processing information are limited, or whether information processing requires any resources at all. Zubin (1975)

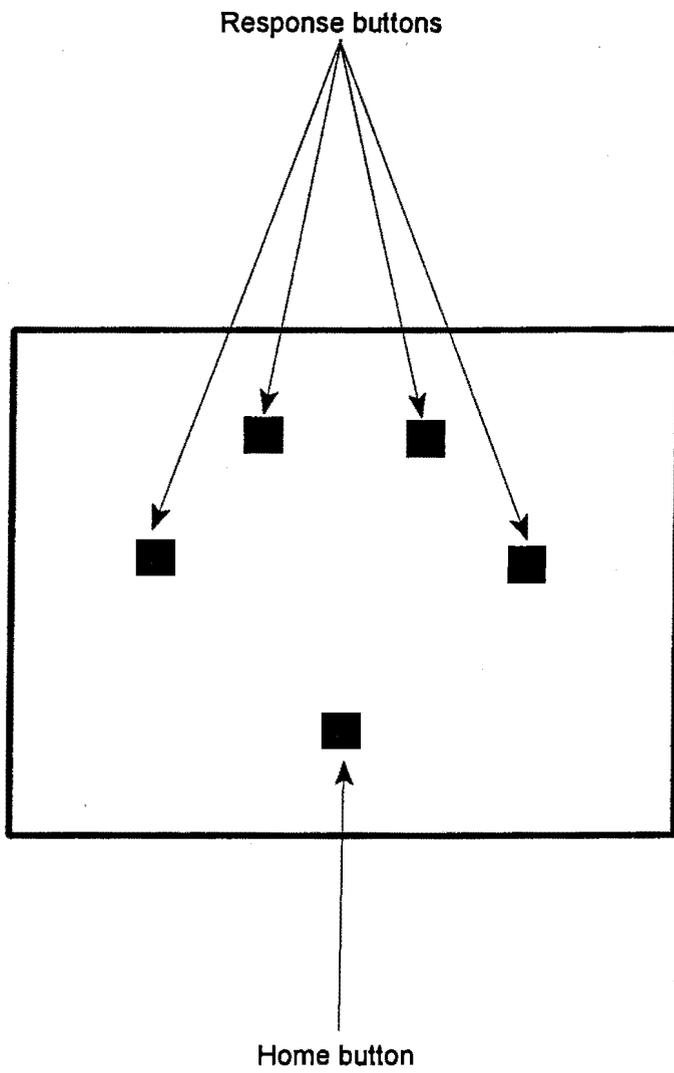
suggested that the attentional elements of focus, sustain and shift are important aspects of the regulation of information processing. Likewise, it can be argued that components of attention, in addition to those cited by Zubin, also play a crucial role in the control of information processing

Attention, acting as the supervisory control previously described, exerts some control over some of the stages of information processing. Mulder (1983) for example, suggested that stages of information processing such as serial comparison, binary decision, and choice all require controlled attentional processes. Because of its controlling function, attention can be expected to affect the speed and accuracy of information processing (Whyte, 1992a). Mulder drew attention to the distinction between fast process research, which relies on automatic information processing, and slow process research, which relies on controlled processing. According to Mulder, the use of response time in fast process research requires the use of reaction time paradigms. Reaction time tasks have been found to be so important in information processing research that Van Zomeren and Brouwer (1994) said of them, "Given the proper stimuli and instructions, almost any neuropsychological impairment can be expressed in a RT task as slow information processing" (p. 39). Because of the importance of reaction time studies in investigating information processing, they will be described at some length in the following section.

Reaction Time

The reaction time experiment was first introduced by Helmholtz in 1850 (Leahy, 1980). Helmholtz carried out reaction time experiments in order to measure the speed of nerve conduction. His method entailed stimulating the nerve of a frog's leg at different points, near and far from the target muscle. The time it took for the animal to respond to the stimulation was labeled as the reaction time. Contemporary reaction time experiments are designed in a variety of ways and use a variety equipment. One of the most commonly used equipments is response box with a home button, and varying numbers of response buttons arranged in a

Figure 1: Reaction time box with four response buttons



semi-circle (Jensen & Munro, 1979). An example of a four-response button reaction time box is shown in Figure 1.

The home button is so called because it is the button from which the subject begins to make a response, and the one to which the subject must return after making a response. At the beginning of the reaction time experiment the subject sits with the index finger depressing the home button. A stimulus will then be given for the subject to respond to. This stimulus may be a sound, or some visual stimulus flashed on the computer screen (as is the case in the present study). The subject is required to respond by lifting his or her finger as quickly as possible from the home button and depressing one of the response buttons depending on the instruction. In a simple reaction time experiment, for example, only one response button and the home button would be visible and accessible to the subject. The other three response buttons would be covered by a mask. In the simple reaction time experiment for example, the subject may be required to depress the response button as soon as he or she sees a luminous arrow flashed on a computer screen. In a two, three, or four choice reaction time experiment, there would be two, three, or four response buttons respectively. The subject will be required to press one of the four buttons, depending on the test stimulus given. For example, as is the case in the present study, the stimulus may be a luminous arrow on the computer screen pointing in the direction of one of the response buttons. The subject is required to depresses the button to which the arrow points.

Indices of Reaction Time

Two main measurements are taken during reaction time experiments. These are *reaction time (RT)* and *movement time (MT)* (Jensen & Munro, 1979). RT is the duration, in milliseconds, from the time the test stimulus is presented, to the time the subject lifts his or her finger from the home button. MT is the duration in milliseconds from the time the subject lifts the finger from the home button to the

time he or she depresses the appropriate response button.

Some researchers have suggested that RT reflects decision time, the length of time required for stimulus evaluation and response programming, whilst MT is a measure of the time it takes to complete a response (Carroll, 1981; Houlihan, Campbell, & Stelmach, 1994). So, whilst RT reflects cognitive processes, MT is supposed to reflect the motor component of the reaction time. Other researchers have defined reaction time as the sum of RT and MT, which is then referred to as total reaction time (Dunlop, Bjorklund, Adelnoor, & Myrvang, 1993). Smith and Carew (1987) stressed the importance of using both RT and MT rather than just total reaction time. The reason for this is that different subjects may use different strategies when responding. For example in the *detection strategy*, the subjects may lift the finger from the home button simply after noticing the presence of a stimulus, rather than after evaluating the stimulus. The result would thus be a lengthening of movement time. Other subjects on the other hand, may fully evaluate the stimulus and make a decision about how to respond before they make any movement. This is the *decision strategy*. The result would be a longer RT and shorter MT. The experimenter is thus never sure which strategy each subject uses. Smith and Carew (1987) further pointed out that there may be a range of possibilities between the extremes of the detection strategy and the decision strategy. Another reason for separating reaction time from movement time is that correlations between reaction time and movement time are either nonsignificant or significant and small (Whitley & Montano, 1992).

Task Complexity and Warning Signals

Researchers have identified two parameters of reaction time that are related to traumatic brain injury. These are task complexity and the presence of a warning signal. Task complexity refers to the numbers of choices that the subject has during a reaction time task. If, for example, there is only one response button on the reaction time box (with the other buttons masked), the subject makes all his or her responses by depressing this one button. The task requirement might be to depress the button when a certain stimulus is flashed on the computer screen. In

this case, the reaction time task is referred to as *simple reaction time*, because there is only one, simple choice. On the other hand, when there is a choice of two or more response buttons, the task is more complex, and it is called *choice reaction time*. With the choice of responding by pressing one of two response buttons, the task is referred to as *two-choice reaction time*.

Blackburn and Benton (1955) showed that a two-choice reaction time task did not produce a more severe retardation of response than simple reaction time. This led to the belief that increasing task complexity does not lead to better discrimination between patients and controls. Contrary findings were however reported by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956), Norman and Svahn (1961), Miller (1970), Van Zomeren and Deelman (1976, 1978), and Van Zomeren and Brouwer (1987). These researchers found that in order to demonstrate the complexity effect, one must use more than two choices. Van Zomeren (1981) established that there is an interaction between task complexity and severity of injury. The trend towards a complexity effect is clearest in the more severe cases.

The other reaction time parameter that has been investigated is the presence or absence of a warning signal in the test. Woodrow (1914) first reported that the most rapid reaction times were obtained when subjects were given a warning signal 2 seconds before the test signal. He suggested that a 2 second foreperiod gave the "maximal adaptation of attention" (p 64). Blackburn & Benton (1955) showed that reaction time in patients with cerebral disease is longer than that of normals when the preparatory interval of 2 seconds is used. Costa (1962) reported that in people with brain damage, a preparatory interval of 2 seconds has no relation to reaction time, whereas in normals, there are more rapid responses over constant preparatory intervals of 0.5 seconds or 2 seconds. Costa concluded from this that normals controls develop an expectancy or set based on the characteristics of the preparatory interval. People with cerebral damage on the other hand do not develop such an expectancy.

Explanations for the Slowing in Reaction Time after TBI

The central issue in the information processing approach with respect to traumatic brain injury is that patients are significantly slower than uninjured individuals. Gronwall and Sampson (1974) and Van Zomeren and Deelman (1978) proposed that the prolonged reaction time indicated a state of underarousal. Van Zomeren (1981) concluded that the slowness was not caused by underarousal because in his series of experiments, both EEG recordings and heart rate activity unequivocally pointed in the direction of increased rather than decreased levels of arousal.

According to Brouwer (1985), the main cause of slowness is a delay in access to stored knowledge. In other words, the slowness is really an aspect of memory deficits. Tromp and Mulder (1991) suggested that mental slowness after traumatic brain injury is due to reduced redundancy of memory representations, causing a delay in the retrieval of information stored in memory. "Redundancy implies that, in the normal brain, knowledge is stored in multiple ways, and that multiple access routes can be taken to reach an item of knowledge. It makes the system less vulnerable to damage (Powell, 1981)." (p. 822). Tromp and Mulder propose that declarative knowledge can have either high or low redundancy according to its familiarity, whilst procedural knowledge, which is formed through experience by solving the same type of problem in many slightly different contexts (Anderson, 1987), is necessarily represented in a highly redundant way. After traumatic brain injury, the retrieval of both declarative and procedural knowledge will be delayed. Procedural knowledge however, because of its greater redundancy, will be relatively less affected.

The issue of redundancy has also been used to explain the differences between left and right hemisphere brain damage. Goldberg and Costa (1991) reviewed the literature and came to the conclusion that the left hemisphere is superior in the use of well routinized, familiar codes (ie procedural knowledge), whereas the right hemisphere is superior in handling novel information (ie declarative knowledge). As was seen earlier, the hypothesis that was put forward

by Tromp and Mulder (1991) is that declarative knowledge, which is less redundant than procedural knowledge, is more vulnerable to brain injury. The differences between the right and left hemispheres has anatomical correlates:

The left hemisphere contains relatively more cells than fibres, and is organized in a modular way. The right hemisphere, on the other hand, contains more fibres than cells, and has more interconnected association areas. Hence, shearing of axon tissue, as happens in head injury, will affect the right hemisphere more than the left, and therefore have more impact on the processing of novel information" (Tromp & Mulder, 1991, p. 827).

Analysis of Stages of Information Processing

One of the most frequently debated approaches to information processing is the stage theory. Sternberg (1969) defined stage theory as follows:

This *stage* theory implies that reaction-time (RT) is a *sum*, composed of the durations of the stages in the series, and suggests that if one could determine the component times that add together to make up the RT, one might then be able to answer interesting questions about mental operations to which they correspond. The study of RT should therefore prove helpful to an understanding of the structure of mental activity. (p. 421).

Two main methods have been designed for identifying and measuring the duration of the different stages of information processing. These are the *subtractive method*, and the *additive factors method* (AFM). Sternberg sees these two methods as ways of decomposing RT.

The Subtractive Method

The subtractive method was introduced by Donders in 1868 (Donders, 1969). Donders was interested in quantifying mental processes, in particular, the length of time required for mental processes. He assumed that information processing takes place in stages. In order to establish the duration of a stage, Donders asked his subjects to carry out two tasks. The second task required all the stages of the first task and an additional stage. The difference between the mean reaction times of the two tasks over a number of trials was taken to be an estimate of the duration of the additional stage. In other words, the reaction time in simple versions of a task is subtracted from the reaction time in a more complex version (Mulder et al., 1995).

After Donders's (1969) publication, a lot of experimental work was carried out utilizing the subtractive method. For example, In 1880, Wundt (cited in Sternberg, 1969) investigated the information processing stages of stimulus detection, identification, and response organization as follows: The reaction time achieved by the subject when instructed to respond after merely identifying the presence of a stimulus was taken to be an estimate of stimulus detection. The reaction time after recognizing the stimulus was taken to be an estimate of identification. Response time was the time between recognition of the stimulus and the making of a response.

Towards the end of the nineteenth century, the subtractive method began to fall into disfavour (Jastrow, 1890). According to Jastrow, there were two main reasons for this. Firstly, introspective data suggested that it would be difficult to devise experimental tasks that could add or delete one of the stages between stimulus and response without also altering other stages. In other words, there were doubts whether pure insertion was possible. Secondly, the differences in mean reaction times varied excessively from one subject to another, and from one laboratory to another. The revival of interest in the subtractive method came with the emergence of better styles of experimentation, and better tests of validity than the method of introspection (Sternberg, 1966).

The Additive Factors Method (AFM)

Sternberg (1969) introduced the additive factors method for identifying stages of information processing. He proposed that the duration of each stage of information processing is influenced by variables that do not influence any other stages. These variables are referred to as *task variables*. The task variables are stimuli that are presented in a factorial reaction time experiment, to which the subject is instructed to respond in a specified way. Each task variable has two levels, an easier one and a more difficult one. The levels of the task variables are called *task conditions*. The presentation of different levels of the task variable is referred to as *manipulation of the task variable*.

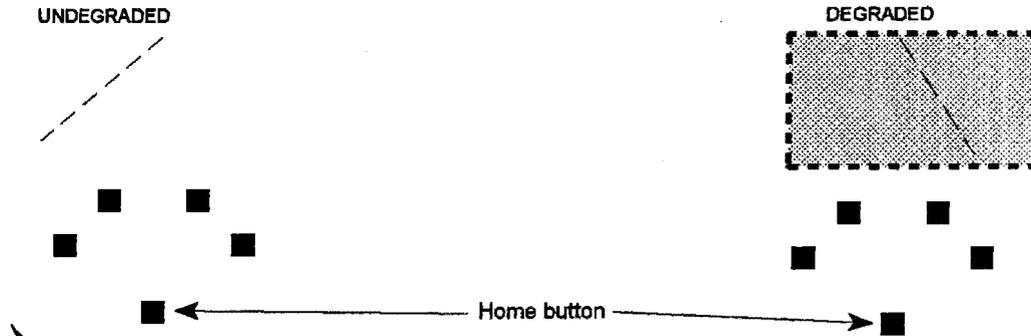
To illustrate these ideas, I will briefly describe an experiment by Shum, McFarland, Bain and Humphreys (1990), which was carried out to identify four stages of information processing. The four stages of information processing were *feature extraction*, *identification*, *response selection*, and *motor adjustment*. The corresponding task variables (with the task conditions in brackets) were as follows: *signal quality* (degraded vs undegraded), *signal discriminability* (similar vs dissimilar), *signal-response compatibility* (compatible vs incompatible), and *foreperiod uncertainty* (fixed foreperiod vs variable foreperiod).

In the experiment, a reaction time box similar to the one illustrated in Figure 1 was used. The stimuli consisted of luminous lines on a computer screen pointing in the direction of one of the four response buttons. At the beginning of the experiment the subject sat with his or her index finger depressing on the home button. An auditory warning signal in the form of a beep generated by the computer acted as a warning signal that the luminous arrow was about to appear on the screen. When the luminous arrow appeared, the subject was required to lift the finger from the home button and depress one of the four response buttons, depending on the task condition. The task conditions are described below.

In the *degraded condition*, there was a random dot pattern (pixels) which were superimposed on the luminous line. This made this signal more difficult to extract from the background than in the *undegraded* condition where the

Fig. 2: The experimental conditions for three task conditions

(A). The two signal quality conditions



(B). The two stimulus discriminability conditions



(C). The two S-R compatibility conditions



luminous line appeared against a clear screen background. The luminous lines for the *similar* condition pointed at positions much closer together than in the *dissimilar* condition (See Fig. 2 for illustration. For the *similar* condition, it was thus more difficult to identify the response button to which the line pointed, than in the *dissimilar* condition.

In the *compatible* condition, the subject was required to depress the button corresponding to the direction of the arrow. For the *incompatible* condition on the other hand, the subject was required to depress the response button opposite to the direction of the arrow (See Fig. 2). The *incompatible* condition was more difficult than the *compatible* condition. Finally, for the motor adjustment stage, the duration between the warning signal and the presentation of the luminous arrow was either short and fixed at 1.5 ms for the *fixed foreperiod*, or was longer and variable (3.5, 4.4, or 5.5 milliseconds) for the *variable foreperiod*. The *variable foreperiod* condition is more difficult than the *fixed foreperiod* condition. The task conditions were presented in a factorial design. This means that the eight task conditions were combined two at a time. The resulting 24 factorial task conditions are shown in Table 1.

Sternberg (1969) defined a stage as one of a series of successive processes that operates on an input to produce an output, and contributes an additive component to reaction time. By additivity Sternberg meant a property of independence for mean reaction time. The mean score at any stage is not related in any systematic way to the mean score for any other stage. In other words, at each stage of information processing, the duration of the stage is influenced only by the manipulation of the relevant task variable. For example, the stage of feature extraction is affected by signal quality alone. The duration of the stage will be longer for the degraded stimulus and shorter for the undegraded stimulus. If there is true independence of stages, the duration of feature extraction should not be affected by task variables from another stage.

Table 1. Factorial design task conditions

Factorial tasks ^a	Deg	Und	Comp	Incom	Simil	Disim	Fixed	Var
Deg	X ^b	X						
Und	X	X						
Comp	✓ ^c	✓	X	X				
Incom	✓	✓	X	X				
Simil	✓	✓	✓	✓	X	X		
Disim	✓	✓	✓	✓	X	X		
Fixed	✓	✓	✓	✓	✓	✓	X	X
Var	✓	✓	✓	✓	✓	✓	X	X

Note

^a Deg = degraded; Und = Undegraded; Comp = compatible; Incom = incompatible; Sim = similar; Disim = dissimilar; fixed = fixed foreperiod; Var = variable foreperiod.

^b X = Nonconditions. This is either because the task is being combined with itself, or, because it is combined with a task condition in the same task variable. The latter would result in a contradiction in terms. For example, a stimulus cannot both be degraded and undegraded.

^c ✓ A factorial task condition, for example *degraded compatible*.

The general idea is that when factors influence no stages in common, their effects on mean reaction time will be independent and additive. That is, the effect of one factor will not depend on the level of the others....On the other hand, when two factors...influence at least one stage in common...there is no reason to expect their effects on reaction time to add; the most likely relation is some sort of interaction (p. 282).

\ Sternberg (1969) further suggested that whilst this is the general rule, exceptions are possible. The additive factors method differs from the subtractive method in that the experimental factors are designed to selectively influence, rather than to insert, a stage. The additive factors method is based on the following assumptions:

1. Information processing consists of a sequence of independent stages.
2. Each stage receives input from a preceding stage.
3. The transformation produced in each stage is independent of any prior stage, and
4. The total reaction time is the sum of the processing times produced in each stage.

Subsequent to the introduction of the additive factors method by Sternberg (1969), interest in the investigation of processing stages increased amongst researchers (Shum, McFarland, Bain & Humphreys, 1990). The additive factors method has some shortcomings, however. Sternberg was aware of this and pointed out that exceptions are possible to his general scheme of how stages can be identified. He also pointed out that his method revealed the stages that existed but did not reveal stage order and stage duration. He agreed with the views of some earlier writers, such as Jastrów (1890), who argued that the overall duration of a stage is more difficult to study and is of less interest than whether such a stage exists, what influences it, what it accomplishes, and what its relationship to other stages is. Other criticisms of the additive factors method were summarized

by Sanders (1980):

1. There is a problem with the basic logic of the additive factors method. Sternberg (1969) had stated that if variables affect different processing stages, then their effects on reaction time add. This statement cannot be logically reversed to the statement that if two variables have additive effects they affect different processing stages. There is always a possibility that a third variable affected both stages, or that there was additivity by coincidence.
2. The additive factors method is built on the assumption that the reaction process is a single serial dimension in the sense that the output of one stage cannot serve as input for more than one stage. It is quite feasible that an experimental variable may affect not only the duration of a stage, but also its output.
3. When deciding that two variables have additive effects, the null hypothesis is accepted. Sanders (1980) points out, however, that the traditional prejudices against the null hypothesis have been subject to much criticism (Rouanet, 1978).
4. The speed-accuracy phenomenon is a well recognized factor in reaction time experiments. The additive factors method does not make provision for this factor.
5. The principle of seriality and independence has been shown to be non-universal. Taylor (1976) for example, described successive stages that overlap in time, so that an increase in time required for one stage may have the effect of decreasing the time required by a subsequent stage.

Further criticisms of the additive factors method (AFM) were recently summarized by Coles, Smid, Scheffers, and Otten (1995).

Most notable of these are the assumptions of *selective influence* and *constant output*. Just as a change of condition may involve more than an insertion of a stage, so it may also involve more than a change in the

duration of a particular stage. It might affect the functioning of other stages and it might also influence the quality of the output of the stage. In addition, appropriate use of the AFM is critically dependent on the presence of a single, all-or-none, discrete instance of information transmission between stages of processing. The idea of discrete transmission is also associated with an additional assumption that each stage of processing is active in series and that no two stages are active in parallel. Deviations from these conditions of discrete transmission and serial ordering of processing can render application of the AFM invalid (p.89).

Sanders (1980, 1990) suggested that despite these limitations of the additive factors method, it has proved very useful as a first step in discovering processing stages. He suggested further that a moderate optimism is justified that at least for limited sets of problems, the method will work, and that in any case, it is advantageous to use this method in order to extend the data base.

Task Variables and Physiological Changes

Electrophysiological studies have been used in an attempt to identify the neural mechanisms associated with different stages of information processing. For example, the decision about the identity of an object is reflected in ERP latency changes. Smulders, Kok, Kenemans, & Bashore (1995) showed that the latency of the evoked potential P300 is lengthened as the stimulus is degraded. In their experiment, the latency of the P300 was not affected by an increase task complexity. Decary & Richer (1995) compared the performance of patients with frontal lesions with those of patients with temporal lobe lesions in an S-R compatibility task amongst other tasks. They found that both groups of patients were impaired compared to normals, but those with frontal lesions were more greatly affected. The motor preparation stage was shown to delay the latencies of N200 and P300 components of event related potentials (Fiori, Ragot, & Renault, 1992). Coull et al. (1995) recently investigated the effects of catecholamines on attentional processes. They found that a low tryptophan drink had differential

effects on stimulus-response compatibility during attentional search.

Although stimulus-response incompatibility has been found to affect reaction time, it does not affect P300 latency (McCarthy & Donchin, 1981; Ragot, 1984). Similarly, Smid, Mulder, Mulder, and Brands (1992) found that although an increase in response selection difficulty resulted in an increase of about 70 ms in reaction time, it did not influence the latency of the P300. Coles, Smid, Scheffers, and Otten (1995) suggest that these findings can be interpreted to mean that "the P300 is emitted after the completion of those processes that are needed for the evaluation of the stimulus, but that the timing of the P300 is independent of the processes associated with response selection and execution." (P. 97).

Neurological Substrates of Attention Deficits after TBI

Normal attention has a complex neurological substrate, as was shown in discussing the components of attention. Likewise, the neurological substrates of attention deficits after traumatic brain injury should be expected to be complex. From the review of the literature in chapter 2, it was pointed out that closed head injury tends to be associated with diffuse injuries. This means that there is a wide range of deficits possible because of the involvement of widespread areas of the brain. The review of that literature also suggested that whilst the brain is injured diffusely in closed head injury, the tips of the frontal and temporal lobes are particularly vulnerable. In the next few paragraphs, the brain lesions that are associated with some of the commonly reported attention deficits will be discussed.

Frontal Lesions

Luria (1973) observed that damage to the prefrontal lobe results in the loss of the ability to form an intention to pay attention. Damage to the dorsolateral portions of the frontal lobe leads to what Hecaen and Albert (1975) later called the "pseudo-depressed" state. This state is characterized by slowness, perseveration, and lack of initiative. Damage to the orbito-frontal cortex leads to disinhibition which manifests as distractibility, impulsivity, and hyperactivity. This distinction between dorsolateral and lateral aspects of the frontal lobe was recently illustrated using brain injury in monkeys. Dias, Robbins, and Roberts (1996) showed that damage to the lateral prefrontal cortex (Brodmann's area 9) was associated with loss of inhibitional control in attentional processes. On the other hand, damage to the orbito-frontal cortex is associated with loss of inhibitory control in affective processing. The frontal lobes have also been shown to be crucial in sustained attention (Wilkins, Shallice, & McCarthy, 1987). The right hemisphere is better than the left hemisphere in sustaining attention on a voluntary basis (Tartaglione, Oneta, Manzione & Favale, 1987).

Fronto-limbic and Reticular Lesions

The frontal lobes have very close connections with the limbic system and the reticular activating system (Nauta, 1973). Weintraub and Mesulam (1985), and Mesulam (1985) suggested that there is an *attentional control matrix* consisting of concentration, vigilance, perseverance, and response inhibition, which correlate with the functions of the ascending reticular pathways and the frontal lobes. Auerbach (1986) suggested that this control matrix should be expanded to include arousal and alertness, vigilance, ability to initiate set shifting, ability to inhibit inappropriate set shifting, and speed of processing. The disorders of attention arising from damage to this part of the brain include perseveration, impulsiveness, distractibility, impersistence, slowness in processing, and confusion.

Trexler and Zappala (1988) suggested that there is a thalamofrontal gating system which is responsible for the controlled or selective attentional processes.

Damage to the thalamofrontal gating system leads to increased susceptibility to interference. It has been suggested that the thalamus acts as a filter for sensory inputs (Carlsson, 1988). The pulvinar of the thalamus for example, has been shown to be involved in the act of choosing relevant from irrelevant visual information in animal studies (Robinson & Petersen, 1992).

Posterior Lesions

\ Posterior lesions, as has already been noted, are less frequently encountered than the anterior lesions discussed above. The posterior lesions fall within Luria's (1973) proposed second functional unit of the brain which is responsible for receiving, analyzing, and storing information. The primary zones of the posterior lobes are more modality specific than the secondary and the tertiary zones. In these primary zones, one can expect auditory, visual, and spatial attention deficits with damage to the temporal, occipital, and parietal lobes respectively. The posterior regions, especially in the right hemisphere appear to be involved in subconscious or automatic processing (Posner, 1975). This suggests that damage in this region may lead to reduced capacity for automatic processing. The end result of this is a regression from automatic to conscious and effortful processing of information (Van Zomeren, 1981).

Memory deficits are very common after traumatic brain injury (Brooks, 1975). These deficits can occur with lesions to any of the lobes, but are particularly associated with temporal lobe damage and damage to diencephalic structures, notably the hippocampus (Milner, 1965). It has been suggested that memory disorders may be, at least in part, secondary to attention deficits (Kinsbourne, 1987; Nissen, 1986; Russell & D'Hollosy, 1992). However, it is likely that the source of the underlying attention difficulty itself may lie elsewhere in the brain, notably the frontal lobes. #

A well-known attention deficit associated with posterior deficits is unilateral spatial neglect. This condition was first described by Geschwind (Heilman, Watson, & Valenstein, 1983). Patients with unilateral spatial neglect behave as if they are not aware of one side of their body and one side of extrapersonal space.

This is evidenced for example by failure to wash one side of the body. Whyte (1992a), suggests that spatial neglect is a disorder of selective spatial attention. This disorder is found in association with right parietal lobe lesions (Heilman, Watson, & Valenstein, 1983). Posner, Walker, Friedrich, and Rafal (1984) suggested that attention can be divided into the components of disengage, move, and engage. Posner, Inhoff, Friedrich, and Cohen (1987) showed that damage to the parietal lobe can lead to a severe deficit of the disengage component whilst the move and the engage components remain intact.

\ A recent study by Mosidze, Mkheidzw & Makashvilli (1994) confirmed that the right parietal and occipital regions are crucial for visuo-spatial attention, whilst the left hemisphere is not. Similarly, Egly, Rafal, Driver, and Starrveveld (1994) tested the ability of neurologically injured and intact controls to detect luminance changes in one of four locations. They found that damage to the right hemisphere was associated with a spatial detection deficit, whilst left hemisphere lesions were associated with object deficits.

Assessment of Attention Deficits after TBI

In a recent review of the methods used in assessing attention, Whyte[#] (1992b) observed that there are no pure tests of attention. Attention, rather, may be seen as the substrate in the performance of all conscious tasks. Whyte (1992b) further observed that one can view the assessment of attention in terms of levels. These levels vary from the firing of individual neurons, to the generation of event related potentials (ERPs), to changes in accuracy and speed, and overt movements of the organs, through to clinical symptomatology. Whyte noted that there are trade offs related to these levels. The lower molecular levels are associated with greater precision but less immediate relevance to behaviour. The higher levels are more clearly related to behaviour but are less precise. The level to be chosen depends on the clinical question.

Whyte (1992b) suggested that in order to assess attention, one has to rely

on measures of motor, perceptual, and cognitive activity. Just as with the theoretical study of attention itself earlier in the century, little attention has been given to the development of psychological tests of attention. Stuss and Benson (1986) observed that the most frequently used tests are not derived from theories of attention. Furthermore, as Van Zomeren and Brouwer (1994) recently commented, only a few tests which meet basic psychometric standards have been developed. With these caveats in mind, the methods used in the assessment will now be described. These methods will be grouped under the various components of attention, viz. arousal and alertness, selective attention, strategic control of attention, speed of information processing and deficits on stages of information processing. Before describing the assessment of these components, a brief description will be given of the initial observation and interview, and the tests of mental control, as suggested by Van Zomeren and Brouwer (1994).

Observation and Interview

The observation of attentional deficits is made from the clinician's own contact with the patient, as well as reports from the patient's relatives or other acquaintances, and from other health personnel. Reported behaviour may include descriptions such as "fidgetiness", "easily distracted", "cannot concentrate" and so on. In the interview, rating scales are frequently used. Examples of rating scale are the Neurobehavioural Rating Scale (NRS) (Levin, High, & Goethe, 1987) and the Attentional Rating Scale (Ponsford and Kinsella (1991). The NRS contains items like, "Rapidly fatigues on challenging cognitive tasks or complex activities, lethargic." One of the items on the Attentional Rating scale is "was unable to pay attention to more than one thing at once." In addition to rating scales, interview questionnaires are also frequently used. Van Zomeren and Van Den Burgh (1985) developed the Trauma Complaints List. Sample items from this questionnaire are "Do you think that you respond more slowly in a conversation?" and "Has your tolerance for bustle decreased?"

The tests of mental control are easy-to-use procedures that can be used at the bedside, and require no specific equipment. They are frequently used by

neurologists as screening tests. The tests include counting back from 10. More frequently used is the Serial Sevens test (Smith, 1967; Luria, 1966), and the Digit Span Forward and Backward (Wechsler, 1981). It has been shown that after head injury, some patients who pass the Digit Span Forward may fail the Digit Span Backward (Brooks, 1984). Weber (1988) recently developed the Attentional ~~+~~ Capacity test, a mental control test consisting of eight subtasks of increasing difficulty. Tests specific to components of attention will now be enumerated. Detailed descriptions of the individual tests will not be given because these tests are well described in neuropsychology texts such as Lezak (1995). The purpose here is simply to give an overview.

Arousal and Alertness

As previously described, arousal and other components of attention including alertness and effort are associated with autonomic nervous system activity. Arousal can be ascertained from EEG spectral analysis. For example, hippocampal theta rhythm is associated with heightened attention (Adey, 1969). The indices of autonomic activity following phasic arousal include changes in pupil size, heart rate, and electrodermal response (Beatty, 1982). Apart from measures of autonomic activity, other measures of arousal include the critical flicker fusion frequency (Smith & Misiak, 1976), and reaction time measures (Van Zomeren, 1981). Tonic arousal may be assessed through behavioural observation, but probably only at its extremes. Whyte (1992b) suggests that these measures may be confounded by neuropsychological underarousal which may sometimes lead to behavioural overarousal, agitation, and restlessness.

Reaction time measures have been used to assess phasic arousal by comparing an individual's performance with and without a warning signal. Costa (1962) showed that in normal controls, the presence of a warning signal led to a reduction in reaction time but this was not the case with brain damaged individuals. The difference between patients and normal controls on warned

versus unwarned tasks is more evident in choice reaction time tasks (Craig, Davies, & Matthews, 1987; Lansman, Farr, & Hunt, 1984).

Selective Spatial Attention

Whyte (1992a) described various measures of selective spatial attention. These include pencil and paper tests of hemi-spatial inattention such as Line Bisection (Schenkenberg, Bradford, & Ajax, 1980), Letter Cancellation (Diller & Weinberg, 1977), and the drawing of symmetrical figures such as clocks (Battersby, Bender, Pollack, & Khan, 1956). For patients who are not able to read and write, tests such as the Motor Free Visual Perception tests have been used (Colarusso & Hamill, 1972).

Focused Attention

The Stroop Word Colour Test (Stroop, 1935) is one of the oldest measure of focussed attention. Focussed attention is usually assessed in the auditory and visual areas (Van Zomeren & Brouwer, 1994). Auditory attention can be tested using the dichotic listening tasks (Cherry, 1953) which were previously described. Visual tests include the Letter Cancellation Task (Diller et al., 1974), the Trail-making tests (Reitan, 1958), reaction time tests with distraction (Van Zomeren, 1981). The visual focused attention tasks take the form of visual search of a target stimuli in a field of distractor stimuli (Van Zomeren & Brouwer, 1994).

Divided Attention

Deficits in divided attention are frequently evidenced by reduced speed in the performance of tasks (Ponsford & Kinsella, 1992). This reduced performance can thus be demonstrated through reaction time tests (Van Zomeren, Brouwer, & Deelman, 1984). A commonly cited measure of divided attention is the Paced Auditory Serial Addition Test (PASAT) (Gronwall & Sampson, 1974). Gronwall and Sampson reported that recently concussed patients performed significantly more

poorly on the PASAT compared to normal controls, and nonconcussed accident victims. Furthermore, the degree of impairment on the PASAT correlated positively with the severity of injury. Patients with mild concussion produced performances which were three times as slowed as those of normals. The severely injured were found to be five times as slow as normal people.

Many subsequent studies have confirmed the slowing of mental processes as measured by the PASAT (Levin, High, Goldstein, & Williams, 1988; Stuss, Stethem, Hugenholtz, & Richard, 1989). Gronwall and Wrightson (1981) showed that the return of PASAT to normal correlated with the recovery from concussion symptoms and improvement of cognitive functions except memory. Dyche & Johnson (1991) used the children's version of the PASAT, the CHIPASAT, and found that its scores yielded low but significant correlations with Arithmetic and Digit Span subtests of the WISC-R. These WISC-R scores are known to be diagnostic of attention problems.

Sustained Attention

One of the earliest tests developed to assess sustained attention is the Continuous Performance Test (CPT) developed by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956). These researchers found that patients were impaired in sustained attention as compared to normal controls. In the past, the visual modality was used in these continuous performance tasks. Recently, Baker, Taylor, & Leyva (1995) showed that auditory presentation of stimuli made the task more difficult, and thus increased its sensitivity.

Van Zomeren and Brouwer (1994) propose that sustained attention deficits present as time-on-task effects, lapses of attention, and intraindividual variability. Time-on-task effects refer to a decrement in performance over time. For example, in a reaction time task, the patient takes a longer to respond, the longer he or she has been involved in the task. Intraindividual variability means that in a continuous task, the individual shows fluctuations in performance. Lapses of attention are a special form of variability of performance. Van Zomeren and Brouwer define such lapses as "sudden dips in level of performance lasting a few seconds at the most"

(p. 38). They suggest that on continuous reaction time tasks, a lapse is any response that exceeds two standard deviations.

Other tests for sustained attention are the cancellation tasks such as the Letter Cancellation tests (Talland, 1965; Talland & Schwaub, 1964;), Digit Vigilance tests (Lewis & Kupke, 1977) and perceptual Speed (Moran & Mefferd, 1959).

Strategic Control of Attention

As stated by Kahnemann (1973), the effort component of attention controls the other components so that attention is allocated in accordance with one's ongoing intentions and plans. Shallice (1982) referred to this as the supervisory attentional control. The Wisconsin Card Sorting Test (Drewe, 1974; Heaton, 1981) is a widely used measure of this supervisory control. The other measures of supervisory control are Digits Backwards (Wechsler, 1981), Serial Sevens (Smith, 1967), PASAT (Gronwall & Sampson, 1974), the Tower of London task (Shallice, 1982) and the Trail-making test (Reitan & Wolfson, 1985). Van Zomeren and Brouwer (1994) point out that the strategic control of attention is not routinely assessed in current clinical practice.

Assessment of Deficits on Stages of Information Processing

Most of the neuropsychological tests used in assessing attention have only a tenuous link with cognitive psychology. This idea was discussed in Chapter 1. According to Mirsky, Anthony, Duncan, Ahearn, and Kellam (1991), the concepts of attention that these tests supposedly measure, such as concentration, are global and less concrete than measures of the elements or components of attention. Assessing the impairments on stages of information processing is one way of using more clearly defined cognitive concepts. In the past few years, the additive factors method for assessing stages of information has been used on clinical populations, as the following examples show. The major studies that have been reported using the additive factors method on patients with brain damage are

those of Stokx and Gaillard (1986), and Shum, McFarland, Bain, and Humphreys (1990).

Stokx and Gaillard (1986) carried out their study with the aim of attributing the slowness displayed by chronic patients to a particular stage in information processing. They carried out three experiments to investigate four stages of information processing, viz, stimulus encoding, memory comparison, response selection, and motor preparation. These four stages were respectively identified through the manipulation of the task variables of stimulus degradation, set size, stimulus-response compatibility, and time uncertainty. The main effects of the four task variables were found to be significant, suggesting the existence of four independent processing stages. However, there was no significant difference between the experimental groups of closed head injury and normal controls on any of the task variables. Contrary to expectation then, this study failed to provide evidence that a specific stage in information processing is impaired in victims of closed head injury.

Shum, McFarland, Bain and Humphreys (1990) also set out to attribute the mental slowing to a particular stage of information processing. They criticized the study by Stokx and Gaillard (1986) for two reasons. Firstly, the study had used a small sample. Because of this, the study lacked sufficient experimental power to identify differences between the groups and the experimental variables. Secondly, the four task variables were manipulated in three separate experiments. The results therefore, could not show conclusively that the effects of the four variables when considered together were additive. The method that these researchers used was described earlier in this chapter. Shum et al. confirmed the existence of four information processing stages. Furthermore, contrary to the findings reported by Stokx and Gaillard (1986), Shum et al. found that different groups of patients were impaired on different stages of information processing. The severe short-term group (GCS at admission < 8; and duration since injury less than 12 months) were impaired on identification and response execution. The severe long-term group (GCS at admission < 8; and duration since injury at least 12 months and not more than 54 months) was impaired only on the response execution stage. There was no evidence of impairment on any of the information processing stages for the mild

short-term group (GCS on admission > 8).

In addition to the above two studies, follow-up studies include that by Murray, Shum, and McFarland (1992), which confirmed the existence of independent stages of information processing in head-injured children and normal controls. The severely injured children were found to be significantly more impaired in response selection and motor execution than the normal controls.

Another study utilizing the additive factors method on head-injured subjects was reported by Schmitter-Edgecombe, Marks, Fahy and Long (1992). This study showed that not all stages of information processing are impaired after severe head injury. The severely head-injured were significantly more impaired than normals on the stage of decision making/response selection. There was no significant difference on the memory comparison stage. Schmitter-Edgecombe et al. conclude from this that some processes are more resilient than others to the effects of severe head injury. In concluding the discussion of the different tests of attention, note is taken of the warning by Van Zomeren and Brouwer (1994) that each test will inevitably tap several aspects of attention:

Thus if we describe a test that seems appropriate for studying the capacity to divide attention, it should be born in mind that the subject in this particular situation must be alert, perceiving selectively, and able to sustain attention for at least several minutes to finish the test. Also, the subject's supervisory attentional control must be active during testing. And let us hope that he will show no lapses of attention as a result of lack of sleep the preceding night (p. 159).

The proposal that most tests tap several aspects of attention is supported by the fact that some tests are used in assessing more than one component of attention. For example, as is clear from previous descriptions, Letter Cancellation is used in assessing selective spatial attention, focused attention, and sustained attention. Furthermore, It has been suggested (e.g. Cronbach, 1957; Sternberg, 1977) that the information processing and psychological testing approaches should be used in a complementary fashion. Shum et al. (1994a) recently

attempted this. They used Posner and McLeod's (1982) *Cognitive Correlates Method*. The cognitive correlates method is based on the assumption that "if elementary cognitive processes (as inferred from specific experimental tasks) are involved in the performance of more complex cognitive operations (as measured by psychological tests), then the subjects' performances on the tasks and tests should correlate with each other" (p. 532). Shum et al. showed that three components of attention, visual-motor scanning, sustained selective visual scanning, and sustained auditory scanning, could be predicted by six indices of information processing. The indices were mean reaction time, mean movement time, feature extraction, identification, response selection, and motor adjustment.

Summary

As a research topic, attention was popular with the earliest experimental psychologists such as James (1890) and Titchener (1908). Attention fell into disfavour in the 1920s due to the rise of behaviourism and gestaltism. There was a resurgence of interest in attention after the influential publications of Hebb (1948) and Broadbent (1958) and the rise of cognitive psychology in general.

From the early days of the revival of attention a research topic, it has become clear that attention is a heterogeneous rather than a homogeneous concept. Moray (1969) identified six meanings of attention used by researchers. These were mental concentration, vigilance, selective attention, search, activation, and set. After Moray, various attempts have been made to break down the concept of attention into its components rather than to continue using ill-defined concepts such as mental concentration.

One of the earliest such attempts was the breaking down of the concept of attention into the overlapping components of alertness, selectivity, and processing capacity (Posner, 1975; Posner & Boies, 1971). Zubin (1975) suggested the components of selectivity, focus, and shift. Pribram and McGuinness (1975) proposed that attention was related to the processes of arousal, activation, and

effort. The distinction between automatic and controlled attentional processes was introduced by Shiffrin and Schneider (1977). More recently, Posner, Walker, Friedrich, and Rafal (1984) suggested that attention can be split into the components of disengage, move, and engage. Posner and Petersen (1990) dealing specifically with visual attention identified the components of orienting to sensory events, detecting signals for focal attention, and vigilance. Finally, Mirsky, Anthony, Duncan, Ahearn and Kellam (1991) used factor analysis on the results of performance on neuropsychological tests to confirm the existence of the components of focus-execute, sustain, encode, and shift. Recently, Plude, Enns & Brodeur (1994) identified orienting, filtering, searching & expecting, as components of selective attention. Of the above sets of components, the following were discussed at some length: arousal; alertness; selectivity; focus, sustain, encode, and shift; automatic and controlled processing; and effort and the strategic control of attention.

Overall, components of attention are associated with multiple brain areas. This is consistent with Luria's writings regarding the involvement of the three functional systems of the brain in all psychological processes. Luria's approach to attention was described. This was followed by a description of Mesulam's (1981, 1990) suggestion of neural networks as the neurological basis of attention.

In an attempt to describe the mechanism of attention, two sets of theories have been proposed. There are the bottleneck theories, and the capacity models of attention. The bottleneck theories state that individuals are continuously exposed to multiple sources of information and it is always necessary to select some channel(s) and ignore others. The bottleneck theories are further subdivided into those which favour early selection (Broadbent, 1958), and those which favour a two-stage (Treisman, 1961) selection or late selection (Deutsch & Deutsch, 1963; Deutsch, Deutsch, & Lindsay, 1976). The idea that there is a bottleneck in information processing has recently been criticized because it is not consistent with the physiological properties of the brain (Meyer et al., 1995). With respect to the early versus late selection, early selection has received support from the ERP studies.

The capacity models began with Kahneman (1973) who theorized about

effort as a component of attention. These models state that the resources for processing information are limited. For this reason, when an individual attempts to carry out two or more tasks simultaneously, there will be mutual interference in performance. Some capacity theorists have suggested that there are several processing mechanisms, and not just one. For this reason, tasks will only interfere with each other if they compete for the same resources (Allport, Antonis, & Reynolds, 1972; Wickens, 1984). Finally, suggestions have been made by theorists like Navon (1984), that all information processing is resource free. The deterioration of performance in dual tasks is due to outcome conflict between two processes. Meyer et al. (1995) suggested that the reason why people may not be able to do more than two tasks at once is that there is a limitation of the peripheral sensory and motor mechanisms, and not limited cognitive capacity.

The relationships between information processing concepts and attention were dealt with next. It was shown that both the bottleneck and capacity theories of attention deal with information processing. Reaction time tasks have been used extensively as an experimental paradigm for measuring speed and accuracy in information processing. The two main methods in assessing information processing stages are the subtractive method and the additive factors method. Both methods rely on the use of reaction time tasks. The subtractive method was the first to be developed (Donders, 1969). Several pertinent criticisms were levelled against this method and it was eventually dropped and replaced by the additive factors method (Sternberg, 1969). This method is also liable to several pertinent criticisms. It has however continued to be used because of its practical value (Sanders, 1980, 1990).

Consistent with the fact that normal attention has a complex neural base, attention deficit was also found to have a complex neural base. All three functional systems of the brain (Luria, 1966, 1976) are implicated in one or other of the components of attention. Special note was made of the contributions of the frontal lesions, fronto-limbic and reticular lesions, and the parietal lobe lesions.

The final major section of the chapter dealt with the assessment of attention. Whyte (1992) has argued that there are no pure tests of attention. Rather, indirect measures of individuals' performance on a wide variety of

conscious tasks. The assessment of attention is also conceptualized as taking place on a continuum of levels, from the firing of individual neurons through to symptomatology. The levels vary in their degree of accuracy and clinical utility. In clinical practice, many tests, some of which were empirically rather than theoretically derived, are frequently used. These tests are described in many textbooks of clinical neuropsychology, for example Lezak (1995).

The assessment tests used in the attention components of arousal, focussed attention, selective attention, and the strategic control of attention were enumerated. The chapter ended with a review of the assessment of attention through the analysis of deficits on stages of information processing.

CHAPTER FIVE

SUMMARY OF LITERATURE REVIEW AND HYPOTHESES

Summary and Aims

In the preceding four chapters, a review of the literature on traumatic brain injury, the postconcussion syndrome, and attention, was carried out. From the review, it was shown that traumatic brain injury is a significant health problem in South Africa (Nell & Brown, 1991). It was also shown that even so-called minor head injury can lead to significant structural and/ or functional abnormalities of the brain (Gordon, 1994).

One of the most frequently reported clinical conditions after traumatic brain injury is the postconcussion syndrome, which was reviewed in Chapter 3. Factor analytic studies, such as those by Levin, Gary et al. (1987) have shown that the symptoms of the postconcussion syndrome load onto meaningful and clear-cut clusters. The aetiology of the postconcussion syndrome has been a controversial topic for more than a century, with the two major positions being the organic and the psychological perspectives (Evans, 1994). Many contemporary writers argue that the postconcussion syndrome is the product of an interaction between organic and psychological factors (Bohnen & Jolles, 1992).

One of the symptoms in the postconcussion syndrome is attention deficit, which patients spontaneously report as concentration difficulties or slowness of thought processes (Evans, 1992; Whyte, 1992a). Deficits of attention are also suspected to be involved in other postconcussion symptoms, notably memory problems (Nissen, 1986). Because of the prominence of this deficit in the postconcussion syndrome, the nature and mechanisms of attention were discussed at some length in Chapter 4. It was shown that attention is not a unitary aspect of cognition, but rather, comprises of a variety of interacting processes and

functions (Stablum, Leanardi, Mazzoldi, Umilta, & Morra, 1994). It is comprised of many components (Posner, 1975; Plude, Enns, & Brodeur, 1994).

Also discussed in Chapter 4 were the various procedures for the assessment of attention deficits after traumatic brain injury. These procedures differ in the degree of precision and immediate relevance to behaviour (Whyte, 1992b). Assessment of impairments on stages of information processing offers a way for operationally defining attention deficits more precisely than the commonly used neuropsychological tests and test batteries (Shum et al., 1994a). Although the assessment of deficits on stages of information processing are a promising approach, serious criticisms have been levelled against it (Sanders, 1990).

The aim of the present study is to determine whether postconcussion symptoms differ in the extent to which they correlate with experimentally determined cognitive deficits. Previous neuropsychological studies have mostly attempted to demonstrate that patients with the postconcussion syndrome have more cognitive deficits as compared to controls (Gfeller, Chibnall, & Duckro, 1994). In the present study, the focus is not on the difference between patients and controls. The comparison of interest is that between patients who report different symptoms on cognitive tasks as indexed by reaction time.

The reaction time tasks were adapted from those previously used by Shum et al. (1990), in their investigation of deficits on stages of information processing. These tasks were described and illustrated in Chapter 4 (Figure 2). To recapitulate, these tasks involve the manipulation of four task variables, signal quality (degraded vs undegraded stimulus), stimulus-response compatibility (compatible vs incompatible), stimulus discriminability (similar vs dissimilar), and foreperiod uncertainty (fixed foreperiod vs variable foreperiod). The difference between the Shum et al. study and the present study is that in the former, the experimental design was factorial, whilst in the later the design is nonfactorial. In other words, the task conditions were not combined two at a time as is the case in factorial designs (see Table 1). Instead, only the original eight task conditions were used. These are shown in Table 2.

Table 2. Nonfactorial task conditions

Task variable	Nonfactorial task conditions
Signal quality	Degraded vs Undegraded
Signal discriminability	Similar vs Dissimilar
signal-response compatibility	Compatible vs Incompatible
Foreperiod uncertainty	Fixed foreperiod vs Variable foreperiod

The advantage of factorial designs is that statistical procedures are available for identifying stages of information processing from the reaction time data. The disadvantage of this design is that there are more task conditions, and these conditions are more complex. For example on the task variable *signal quality* there are only two task conditions, viz. *degraded* and *undegraded*. In the factorial design on the other hand, there are twelve possible task conditions as shown in Row 2 and Row 3 of Table 1. The tasks are more complex because they involve two task conditions, and not one, as in the nonfactorial design. For example, for the task nonfactorial design a stimulus is simply degraded or undegraded. In the factorial design on the other hand the task may be *degraded and incompatible*.

Because of the complexity and the number of tasks in the factorial design, it takes relatively longer to test each subject than in the nonfactorial design. In the study by Shum et al. (1990), testing each participant took approximately one and a half hours. This excluded the time for the interview and the filling out of questionnaires. Spending more than one and a half hours on each participant was considered impractical in the outpatient settings in which this study was going to be carried out. After consulting with the doctors, patients are normally anxious to leave the hospital to catch their transport home. They would not be willing to wait for hours at the outpatient department to be tested for research purposes.

The advantage of using the nonfactorial task design is that a much larger sample can be used. Furthermore, if any differences are found between groups, there would be a strong case for suspecting that differences would also be found on the more complex tasks of the factorial designs. There would then be a justification for planning more expensive and time consuming factorial studies investigating deficits on stages of information processing. At the same time, this simpler experiment would help to highlight errors to be avoided, and new strategies to be used, in future factorial designs. To achieve the above aims, the following hypotheses were formulated.

Hypotheses

Hypothesis 1

Patients who have sustained a concussion will show greater frequency and intensity of postconcussion symptoms than controls; and patients will be slower in performing reaction time tasks.

Previous studies such as those by Bohnen et al., (1992) and Wong, (1994) have shown that symptoms similar to those found after a concussion are also common in the general population. Furthermore, these symptoms are also found in non-head injured patients (Fullerton et al., 1981; Gouvier, Uddo-Crane, & Brown, 1988) and in the psychiatric population (Fox, Lees-Haley, Earnest, & Dolezal-Wood, 1995). The first prediction of Hypothesis 1 is that patients will report greater frequency and intensity of symptoms. There are at least three reasons why patients may have greater symptomatology. First, because of the concussion, patients may have organic pathologies that increase the probability of symptoms. For example, as was indicated in the literature review (Chapter 3), abnormal cerebral circulation may contribute to postconcussion headaches (Evans, 1992; Packard & Ham, 1994). Secondly, the stresses associated with traumatic brain

injury may lead to more symptoms (Mittenberg, Zielinski, & Fichera, 1993; McLelland, Fenton & Rutherford, 1994). Finally, as Mittenberg, DiGiulio, Perrin, & Bass (1992) recently found, the expectation that one will experience symptoms after head injury may contribute to the emergence of those symptoms.

The second prediction of Hypothesis 1 is that patients will be slower on reaction time tasks. The slowness of patients compared to controls is due to the fact that traumatic brain injury can lead to damage to brain structures and systems known to be crucial for some cognitive processes. For example, in the literature review, it was indicated that the frontal and temporal lobes are particularly vulnerable to injury (Adams, Graham, Scott, Parker, & Doyle, 1980; Chu, Lin, Huang & Lee, 1994). Damage to the temporal lobes may result in memory problems (Cooke & Kausler, 1995). Because of this deficit, the patient may have difficulties remembering instructions, and this will result in errors or slowness of response (Tromp & Mulder, 1991). Traumatic brain injury also leads to several kinds of attention deficit. For example, damage to the first functional unit of the brain (Luria, 1973), may lead to reduced arousal and alertness; damage to posterior brain regions leads to deficits in spatial attention (Mesulam, 1990); and damage to the frontal lobe leads to loss of ability to inhibit responses to irrelevant stimuli (Luria, 1973). Furthermore, traumatic brain injury may lead to reduced processing capacity, which gives rise to the need to exert more effort (Gronwall & Wrightson, 1974). Increased effort in turn leads to fatigue, which further compromises performance on reaction time tasks (Van Zomeren, 1981).

Hypothesis 2

For both patients and controls, the symptoms tend to form clusters. However, the clusters identified for the patients differ from those identified for the controls.

Previous factor analytic studies have already shown that postconcussion symptoms tend to form clusters (Van Zomeren & Van Den Burgh, 1985; Levin, Gary et al., 1987). There is no reason to believe that the symptoms of patients in

South Africa do not also form clear-cut clusters. However, as Levin, Mattis et al. (1987) showed, the aggregation of symptoms reported differ from centre to centre, even in the same country. The postconcussion symptom patterns also change over time (Rutherford, 1989). It is therefore difficult to determine *a priori* the number and the constituent symptoms of the clusters of symptoms in the present sample. The present hypothesis was included in order to establish the actual symptoms reported and the manner in which they cluster in this particular sample.

None of the reviewed studies compared the symptom clusters between patients and controls. It is proposed here that the symptom clusters will not be identical. There may be similarities between some of the clusters, namely, the constellations of symptoms arising from stress (McLelland, Fenton & Rutherford, 1994). However, because the patients have an additional traumatic insult to the brain, it is to be expected that there will be additional symptoms that are not found in the control group.

Hypothesis 3

For both patients and controls, level 1 tasks will be associated with significantly longer response times than level 2 tasks.

As was described in the literature review, there are four task variables, each with two levels or tasks conditions (Shum et al., 1990). For each task variable, one of the task conditions is meant to be more difficult than the other. The more difficult task will lead to longer reaction time than the easier task. In this study, the more difficult task condition will be referred to as level 1, and the less difficult will be referred to as level 2. The level 1 and level 2 task conditions for the four task variables are shown in table 3.

Table 3: Level 1 and level 2 task conditions

Level 1	Level 2
Degraded	Undegraded
Incompatible	Compatible
Similar	Dissimilar
Variable foreperiod	Fixed foreperiod

The hypothesis states the obvious, in that the tasks were designed with the express aim of making the level 1 tasks more difficult than the level 2 tasks. Previous studies have used similar tasks, with the same aim of having significant differences in performance on the two levels (Shum et al., 1990; Stokx & Gaillard, 1986; Murray, Shum, Bain, & Humphreys, 1992; Shum, McFarland & Bain, 1994b). It is nevertheless important to confirm that this is the case for the present sample, before testing other hypotheses which rely on this assumption.

Hypothesis 4

The magnitude of the difference between level 1 and level 2 tasks will be significantly greater for patients than it is for controls .

In hypothesis 3, it was suggested that the level 1 task is more difficult than the level 2 task. This means that subjects are expected to be faster in their reaction times on level 2 than on level 1. It is predicted here that this difference will be significantly greater for patients than for controls. This expectation is based on the fact that previous research has suggested that brain damage is associated with greater sensitivity to increased complexity on reaction time tasks (Van Zomeren & Brouwer, 1987). Because of the limited processing capacity after traumatic brain injury, patients often have to exert more effort than controls in order to cope with even simple tasks (Gronwall & Sampson, 1974; Hinkely & Corrigan, 1990 ; Van Zomeren, Brouwer, & Deelman, 1984). With the more difficult tasks, it is reasonable to expect that even more effort will be required. As more

effort is exerted, there will be more fatigue, which in turn will lead to significantly slower responses.

Hypothesis 5

The number of correlations between postconcussion symptoms and indices of reaction time varies as a function of the subjective ratings of symptom frequency, intensity, and duration.

Jakobsen, Baadsgaard, Thomsen, and Henriksen (1987) showed that it is possible to predict the persistence of postconcussion symptoms from the subjects' performance on reaction time tasks a few days after injury. Lishman (1988) also found that there is a positive correlation between postconcussion symptoms and slowing on reaction time tasks, but this correlation disappeared after three months. More recently, Arcia and Gualtieri (1993) also showed that symptom presence is associated with deficits on a number of neuropsychological tests. For this study it is proposed that more cognitive deficits would be identified if the symptoms are quantified beyond just endorsing the fact that they are present. The way to quantify the symptoms is to record the patient's subjective rating of each symptom's frequency and intensity. Gouvier, Cubic, Jones, Brantley, and Cutlip (1992) have suggested that recording symptom frequency, intensity and duration increases the conciseness of assessing postconcussion symptoms.

The indices of reaction time are classified into four groups: Reaction time (RT, the duration from the presentation of a stimulus to the lifting of the subject's finger from the home button), movement time (MT, the duration from the lifting of the finger from the home button to the depressing of the response button), total reaction time (TT, the sum of RT and MT), and subtraction score (SB, the respective differences between the RTs, MTs, and TTs of level 1 and level 2 task conditions). There will thus be eight RT scores, eight MT scores, eight TT scores, and 12 SB scores. The total number of reaction time indices for 8 task conditions will be 36. In this study then, both the symptoms and the reaction times are broken down into specific aspects. This large number of indices was used because, as

Houlihan, Campbell, and Stelmach (1994) suggested, predictive power is increased when several reaction time measures are used.

Hypothesis 6

The various postconcussion symptoms will differ in the extent to which they correlate with reaction time, movement time, total reaction time, and subtraction scores.

In the literature review, it was indicated that different components of reaction time, viz., reaction time (RT) and movement time (MT), involve different capabilities, namely decision time and response execution (Jensen & Munro, 1979). Decision time was referred to as the cognitive component of reaction time. The postconcussion symptoms have also been classified into *cognitive* and *noncognitive* ones (Bohnen, Twijnstra & Jolles, 1992; Levin, Gary, et al., 1987). In this hypothesis, the prediction is that symptoms will correlate either with RT or with MT depending on whether they are cognitive or not. Total reaction time and SB scores are both derived from RT and MT. It is predicted that likewise, symptoms will differ in the extent to which they correlate with total reaction time and SB. The specifics of the relationships between postconcussion symptoms and brain lesions is not yet fully understood (Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992). Some of the proposed neural bases of the postconcussion symptoms were discussed in Chapter 3. For example, some forms of depression after traumatic brain injury may be attributable to frontal lobe damage (Jorge et al., 1993a). The cognitive component of reaction time, notably *response selection*, has also been attributed to frontal lobe damage (Decary & Richer, 1995). An overlap in the neurological aetiology of depression and deficits on the cognitive component of reaction time may lead to a strong correlation between the two. Establishing

correlations between the various postconcussion symptoms and indices of reaction time may help to suggest the underlying neurological bases of both.

Hypothesis 7

The various postconcussion symptoms will differ in the extent to which they correlate with the task variables signal quality, signal discriminability, S-R compatibility, and foreperiod uncertainty

According to the stage theories of information processing (Sternberg, 1969), the different task variables require different cognitive operations. In factorial experiments (Table 2), signal quality involves *feature extraction*, signal discriminability involves *identification*, S-R compatibility involves *response selection*, and foreperiod uncertainty involves *motor adjustment* (Shum et al., 1990). In the postconcussion syndrome, some symptoms are cognitive, whilst others are not (Bohnen, Twijnstra & Jolles, 1992). It is expected that symptoms will differ in the extent to which they relate to the various cognitive demands inherent in these tasks. For example one can expect that patients with visual problems will have more difficulties with *signal quality* than other patients. Because a nonfactorial design was used in the present study, any significant relationships found can only suggest, rather than confirm, relationships between specific symptoms and stages of information processing.

Hypothesis 8

The various symptom clusters will differ in the extent to which they correlate with reaction time, movement time, total reaction time, and subtraction scores.

This hypothesis is an extension Hypothesis 6. The only difference here is that one is considering collections of symptoms, rather than individual symptoms. It is important to investigate clusters of symptoms because postconcussion

symptoms do not typically occur in isolation (Levin, Gary et al., 1987; Mureriwa, 1990). It is anticipated that symptoms with a common aetiology may show similar cognitive deficits. For example, low noise and low light tolerance are both associated with cranial nerve abnormalities (Cohen & Rein, 1992; Bohnen, Twijnstra, Wijnen, & Jolles, 1991; Waddell & Gronwall, 1984).

CHAPTER SIX

PILOT STUDY

Aims

The aims of the pilot study were as follows:

1. To provide the researcher with experience in the administration of the reaction time tasks as well as the research questionnaires.
2. To test the computerized reaction time programme which was custom-made for this research.
3. To determine what difficulties, if any, patients may have in taking part in the computerized reaction time tasks.
4. To obtain an indication of the distribution of the reaction time data on a South African hospital sample. In previous research on stages of information processing, most reaction time data is reported to be positively skewed (Shum, McFarland, Bain, & Humphreys, 1990). In other words, in a normal distribution curve, the scores tend to pile up to the left of the curve. It was considered necessary to determine if this was the case on the present sample, so that the research tasks could be redesigned as necessary.

Method

Subjects

To obtain patients to take part in the pilot study I requested referrals from neurosurgeons at the Ga-Rankuwa and HF Verwoerd hospitals in Pretoria. The patients were to have the following characteristics:

1. History of traumatic head injury
2. Age: 18-40 years
3. Sex: both
4. Symptoms: All patients complaining of one or more postconcussion symptoms (Appendix E).
5. No history of neurological conditions such as epilepsy or previous head trauma, or a history of psychiatric disorders.
6. Ability to communicate in English.

A total of 22 patients were referred but only 16 were included in the study. The six patients who were excluded had been referred from the adult male neurosurgical ward at Ga-Rankuwa hospital. They were excluded because, although they were considered well enough to be discharged from hospital, when I tested them, they were unable to follow the instructions. In other words they did not appear to understand what was required and continued to make incorrect responses in spite of these mistakes being pointed out to them. Of the subjects that remained, nine were from Ga-Rankuwa hospital (eight from the neurosurgical outpatient department and one from the male neurosurgical ward), and seven were from the neurosurgical outpatient department at HF Verwoerd hospital.

The control subjects took part in response to advertisements placed on notice boards at the Medical University of Southern Africa (Medunsa). No payment was offered for taking part.

Instruments

1. A 16 MHZ 386 desktop Astro computer with colour monitor.
2. A wooden reaction time box (Hick Box)(Fig. 3). This consists of 1 square home button and 4 square target (or response) buttons. The five buttons measured one square centimetre each. Each of the target buttons was located 16cm from the home button.
3. Questionnaires
 - 3.1 Demographic Data Form (Appendix A) . This form was specifically designed for the present study. It elicited demographic information such as sex and age. Information about the dates of admission and discharge was obtained from the patient's files. The last four questions were for establishing hand dominance. These were borrowed from Nell (1990).
 - 3.2 Trauma Details Form (Appendix B) . This form was also specifically designed for the present study. The aim of the form was to obtain information from the patient about the source and severity of injury. The answers to questions 10 to 15 were obtained from the patients' hospital files.
 - 3.3 Screening Questionnaire (Appendix C). This questionnaire was adapted from the World Health Organization's (1986) *Neurobehavioural Core Test Battery* by Nell and Taylor (1992). The information it obtains relates to previous head injuries, and the presence of other medical conditions.
 - 3.4 Pretest Questionnaire (Appendix D). (World Health Organization, 1986). The pretest questionnaire asks about the amount of sleep in the past 24 hours, as well as the consumption of fluids and drugs.
 - 3.5 Postconcussion Symptom Checklist 1 (Appendix E) . This is a list of postconcussion symptoms compiled by myself from the literature reviewed in Chapter 3.

Figure 3. Computer and Hick Box

Computer screen
with 8 stimulus lines →

Hick Box with 4 response
buttons and a home button



Procedure

At the time that this project began, I was employed as a Senior clinical psychologist and Senior lecturer at Ga-Rankuwa hospital and Medunsa. I also had an honorary appointment as a clinical psychologist at the HF Verwoerd hospital. Apart from carrying out the research project, I provided service to these patients as part of my employment. The service included doing clinical assessments, filling in medico-legal forms, counselling, and referring patients on to other specialists.

Questionnaire Administration

The first questionnaire to be administered was the Demographic Data form (Appendix A). Information on dates of admission and discharge was obtained from the hospital files. For the controls, the questions relating to hospitalization were not asked. The next form was the Trauma Details (Appendix B). This was administered to patients only. The first nine questions were asked from the patients directly, and the answers to the remaining questions were obtained from the hospital files. All information provided by the patients was double-checked against the information in the hospital files. The Screening Questionnaire (Appendix C), and the Pretest Questionnaire (Appendix D) were administered to both patients and controls.

The administration of the Postconcussion symptom checklist 1 was more involved than the first four questionnaires. I sat next to the patients and placed the questionnaire in front of them. I would then say:

On this form I have a list of symptoms that you may or may not have been experiencing since your injury." As I said this I would indicate the column on the form labelled symptom. "I would like you to indicate to me how frequently this symptom bothers you" (pointing to the column frequency). If this symptom never bothers you we will give it a score of 0. If it bothers you only sometimes we will give it a score of 1. If the symptom bothers you most of the time we will give it a score of 2.

Finally, I would like you to indicate in this column (pointing to the column intensity) how intense this symptom is for you. If the symptom is not a problem at all to you we will give it a score of 0. If it is a mild problem we will give it a score of 1. If it is moderate we will give it a score of 2; and if it is severe we will give it a score of 3.

After these instructions, I filled in the questionnaire according to the responses indicated by the patients. Further explanations of the scoring rules were given as required by the patients. Some patients responded that the symptoms "used to be present" but were not currently present. Only current symptoms were endorsed on the form.

Reaction Time Tasks: Task Variables and Task Conditions

The reaction time experimental procedure closely followed that used by Shum, McFarland, Bain, and Humphreys (1990). This experiment was described in chapter 4. A diagrammatic illustration of the four task variables was given in Figure 2, chapter 4. For each of the 8 task conditions there were two signals. The first one was a warning signal. It was a 256 Hz beep from the computer, lasting .07 seconds. The second signal was the imperative signal, a 10cm luminous line on a dark screen background. This line appeared in the centre of the screen pointing in the direction of one of the four response buttons. The next pair of signals appeared 2 seconds after the response button had been depressed. With the exception of the *variable foreperiod condition*, the warning signal was presented 1.5 seconds before the appearance of the imperative signal.

The following instruction was given at the beginning of the experiment:

I am going to use this computer and this Hick Box to measure how fast you are in responding. You will make all your responses on this Hick Box. The pictures that you must respond to will appear on this computer screen. As you make your responses, the computer will calculate how fast you are responding. You must use only the index finger of one hand for doing this

(This would be the preferred hand as established by the hand dominance examination). Try to respond as quickly as you can without making mistakes. Now we can begin....

After the subject was seated comfortably in front of the experimental equipment, the researcher typed in the appropriate commands on the computer keyboard. This led to the appearance of eight luminous lines on the computer screen with a degraded background (See Fig. 4). Four of the lines were thick and clearly visible. The other 4 were faint and difficult to discriminate against the background. The subject was then asked "Do you see these eight lines on the screen?" If the answer was yes, the subject was told: "Please point to each of those lines in turn."

If the subject was unable to point to all eight lines correctly, he or she would be excluded from the study for visual reasons. If, on the other hand the subject pointed to all the lines correctly, the experimenter said: "These lines are going to appear on the screen one at a time. I would like you to show me which of these lines has appeared by pressing down on one of the four buttons on this Hick Box. Let me show you how we are going to do this..."

The experimenter then pressed the ENTER key on the keyboard. This brought on another screen page, with the prompt for providing the patient's name. After the subject's name had been logged on, the familiarization practice for the subject would begin. This was meant to familiarize the subject with the position of the home button and the target buttons. It was also meant to give the subject practice in responding to the warning and the imperative signals. The familiarization practice consisted of nine trials. The instructions for doing this practice were shown on the computer screen and were as follows:

On the wooden box in front of you there are five black buttons. The one at the centre, situated below the other four is the "start" button. The four buttons arranged in a semi-circle near the top of the box are the "target buttons." These are numbered 1, 2, 3, & 4.

In order to start the experiment, you must place your forefinger on the start button and keep it pressed down. After a few seconds a beep will sound from the computer to warn you that the experiment is about to begin. The beep will be followed by an arrow appearing on the computer screen. This arrow will be pointing in the direction of one of the four target buttons. As soon as possible after the appearance of the arrow, lift your finger from the start button and depress the button corresponding to the direction of the arrow. Try to respond as quickly as you can without making a mistake. Tell me when you are ready to begin...

After the familiarization practice, the formal tests began. The eight task conditions were presented in random sequence. The instructions for carrying out each reaction time task appeared on the computer screen and were read out and explained to the subject. The instructions were for each of the eight task conditions were as follows:

1. Undegraded

- * Keep your forefinger pressing down on the start button.*
- * When the arrow appears on the screen lift your forefinger and quickly depress the target button corresponding to the direction of the arrow.*

2. Degraded

- Keep your forefinger pressing down on the start button.*
- * When the arrow appears on the screen lift your forefinger and quickly depress the target button corresponding to the direction of the arrow.*

3. Dissimilar

- * Keep your forefinger pressing down on the start button.*
- * When the arrow appears on the screen lift your forefinger and quickly depress the target button corresponding to the direction of the arrow.*

4. Similar

- * Keep your forefinger pressing down on the start button.*
- * When the arrow appears on the screen lift your forefinger and quickly depress the target button corresponding to the direction of the arrow.*

5. Compatible

- * Keep your forefinger pressing down on the start button.*
- * When the arrow appears on the screen lift your forefinger and quickly depress the target button corresponding to the direction of the arrow.*

6. Incompatible

- * Keep your forefinger pressing down on the start button.*
- * When the arrow appears on the screen lift your forefinger and quickly depress the target button AT THE POSITION OPPOSITE TO THE DIRECTION OF THE ARROW.*

In other words, when the arrow points:-

- In the direction of button 1

** You should depress button 4.*

- In the direction of button 2

** You should depress button 3.*

- In the direction of button 3
- * You should depress button 2.

- In the direction of button 4
- * You should depress button 1

8. Foreperiod Uncertainty

In this part of the experiment, the length of the waiting period between the beep from the computer and the appearance of the arrow on the screen will vary. Sometimes the waiting period will be short and sometimes it will be long.

- * *Keep your forefinger pressing down on the start button.*
- * *When the arrow appears on the screen lift your forefinger and quickly depress the target button corresponding to the direction of the arrow.*

The testing was discontinued if a subject made five consecutive errors. To discontinue, the experimenter said, "That will be enough for today. Press this button" (Indicating either ESC, in order to abort the test without saving results, or 'Q' for quitting and saving results up to this point). These subjects would be excluded from the study.

Results

Questionnaires

There were no problems with the forms eliciting personal details, and the Screening Questionnaire. Patients were able to understand the requirements of the Postconcussion Symptom Checklist. An impression was gained however, that a fuller description of the symptoms would be obtained if patients were asked to rank their symptoms in some way. Patients found it difficult to estimate duration of

loss of consciousness or post-traumatic amnesia. They tended to repeat what someone else had told them. Most were unable to tell the time of the accident or the assault.

Reaction Time

The testing of normal controls was abandoned after four respondents had been tested because, the computer, a large desktop computer, broke down. The breakdown occurred due to the fact that the computer equipment had to be frequently connected and disconnected and transported in the car between the office and the hospital clinics. Failure to test more normal controls was not considered as a serious setback because no significant problems had been noted when testing normal subjects during the practice phase.

Several observations were made about the way in which the reaction time tasks were performed: Judging by its face value, it had been expected that the *incompatible* task condition would be the most difficult for the subjects. Subjects were observed to make many errors on this task. However, the subjects who were able to perform the task seemed to find it more interesting than all the other task conditions. When asked for comment, subjects indicated that they enjoyed this task because it involved more challenge than the other tasks.

It was observed that for most subjects, the response required for the *incompatible* task condition tended to be carried over to whichever task condition followed. In other words, the subjects tended to respond by depressing the button opposite to the direction of the arrow, thus resulting in error scores. Many subject also made errors on the *similar* task condition. Those who had difficulties in this task seemed to find it unpleasant and frustrating.

Some subjects who had quick reaction time (RT) nevertheless recorded slow movement time (MT). The delay in depressing the response buttons was due to one of two factors: On some occasions, subjects clearly aimed for the correct target button but missed it. They would hit the Hick Box in the vicinity of the button first then move over to depress the correct button. On other occasions, subjects touched the correct button but did not depress it firmly enough. They would then

either realize the problem and depress the button for the second time, or would not realize the problem and go back to the home button. With practice however, this problem was eliminated.

Error Counts. Whenever the subject failed to depress the required response button on each trial, this was recorded as an error. The number of errors made on each condition were counted. These are shown in Table 4.

There were high error rates on three task conditions, compatible, incompatible, and similar. In experiments on stage analysis, of which this study is a modification, the aim is to have as low an error rate as possible (Shum, McFarland, Bain, & Humphreys, 1990). This is because identification of stages is based on calculations on correct responses, which should be at least 90%. Errors

Table 4 :Percentages of errors on task conditions

Task condition	Errors %
Degraded	6.64
Undegraded	7.03
Compatible	10.55
Incompatible	22.27
Similar	19.14
Dissimilar	7.03
Fixed foreperiod	7.03
Variable foreperiod	6.64

are always eliminated. The high error rates on some of the task conditions are therefore not acceptable. It is proposed that the errors could have been less if the subjects had been given more practice trials.

Distribution of Reaction Time Measurements. For the pilot study, only RT scores were used in the following analyses.

Except for the *dissimilar* task condition, the reaction times were positively

Table 5: Measures of central tendency

Condition	Mean	Median	S.D.	Skewness
Degraded	89.73	61.00	86.44	4.10
Undegraded	56.37	49.00	53.65	12.47
Similar	61.90	60.00	26.44	0.71
Dissimilar	45.67	50.00	44.45	-2.16
Fixed foreperiod	61.57	55.00	21.28	1.43
Variable foreperiod	71.61	65.00	27.90	1.40
Compatible	60.31	50.00	43.04	4.52
Incompatible	66.33	55.00	42.64	5.18

skewed. This is consistent with previous studies, which have shown that reaction time data tends to be positively skewed (Shum, McFarland, Bain & Humphreys, 1990; Stokx & Gaillard, 1986; Murray, Shum, & McFarland, 1992; Shum, McFarland, & Bain, 1994). Positive skewness indicates that the tasks are not too difficult for the subjects (Shum, McFarland, Bain & Humphreys, 1990). The negative skew found for the *dissimilar* task condition is surprising, because one would have expected this task condition to be the easiest. Perhaps the task was too easy, and as such was not sufficiently arousing for subjects to exert maximal attention. It did not capture the subjects' attention (Berlyne, 1960).

Outliers. An outlier is any score which is 2 standard deviations higher or lower than the mean for each condition (Shum, McFarland, Bain & Humphreys, 1990). When analysing reaction time data for identifying stages of information processing, the accepted practice is to eliminate outliers (Tabachnick & Fidell, 1983). Sternberg (1969) noted that one way of handling the positive skewness in RT distributions was to use logarithmic transformations. He however did not recommend logarithmic transformation because such transformation would destroy meaningful additive or interactive relations between variables. Shum, McFarland, and Bain (1994) suggested that deletion of outliers allows a more direct interpretation of results. In the present study it was decided to eliminate outliers in order to be consistent with previous studies. The number of outliers as a

percentage of correct responses is shown in Table 6.

The percentage of outliers is comparable to that of previous studies (Shum, McFarland, Bain, & Humphreys, 1990; Stokx & Gaillard, 1989; Murray, Shum, & McFarland, 1992). For example, in Shum, McFarland, & Bain, (1994), the percentage of outliers ranged from 3.32% to 7.18%.

Mean differences between level 1 and level 2 tasks. All incorrect responses and outliers were eliminated for the reasons outlined above. From the remaining responses, the average scores for each subject on each condition were calculated. After this was done, all the cases which had less than 10 responses out of 16 (62,25%) on any of the four task conditions were excluded from further analyses. From the original 16 subjects, 11 remained. T-tests for repeated measures were used to compare the means for the level 1 and level 2 tasks. The level of each task condition is shown in brackets in Table 7.

There were two significant differences and two nonsignificant differences. The lack of significance is probably due to the smallness of the sample. Such small samples have, as was previously stated, been one of the criticisms of research in stage analysis (Lezak, 1985). Furthermore, the lack of significance may be attributable to the unacceptably high error rate in the task conditions compatible, incompatible and similar (Table 4). Because of the high error rate on a small sample of subjects, it means that only a few responses were used in the calculations. The validity of the result thus becomes questionable.

Procedural Modifications

On the basis of the results of the pilot study, the following modifications were adopted for the main study.

1. The postconcussion symptom checklist would be modified to include a ranking of symptoms
2. The subjects would be given more intensive practice on all task conditions. As was previously stated, it is desirable in this kind of experiment that the error rate be as low as possible (Shum, McFarland, Bain, & Humphreys,

1990). The focus is on speed and not on accuracy. Increased practice would help to eliminate errors and increase the number of trials that could finally be included in the calculations. In this way, there would be a greater chance of detecting significant differences if they exist. Giving sufficient practice in this way, is the strategy used by previous researchers such as Shum et al.

3. When subjects showed perseveration after completing the *incompatible* task condition, this was pointed out to them. They would further be allowed to practice the new condition until they obtained 5 successive correct responses.
4. If a subject knew the correct response but made the mistake of hitting the side of the response button or not depressing it hard enough, they would be allowed to restart the trial. If the problem was persistent however, the subject would be excluded from the study.

Table 6: Percentages of outliers on task conditions

Task condition	Outliers (%)
1 Degraded	2,73
2 Undegraded	3,9
3 Compatible	3,13
4 Incompatible	1,95
5 Similar	3,91
6 Dissimilar	4,30
7 Fixed foreperiod	5,86
8 Variable foreperiod	9,38

Table 7: Mean task condition differences (N = 11).

Task condition	Mean	Correlation	t-value	df	p
Degraded (1) Undegraded (2)	73.89 52.70	0.93	3.27	10	0.008
Incompatible (1) Compatible (2)	58.30 51.91	0.64	- 1.72	10	0.11
Similar (1) Dissimilar (2)	54.70 50.50	0.86	- 1.32	10	0.215
Variable foreperiod (1) Fixed foreperiod (2)	66.50 57.50	0.902	- 3.40	10	0.007

6. A portable computer would be used instead of a desktop computer. This was mainly for the ease of transportation of the equipment. It was also envisaged that there was less chance of a further breakdown when transporting a smaller computer.
7. In order to increase the subject pool, permission was sought to find patients at 1 Military Hospital in Pretoria.

CHAPTER SEVEN

METHOD

Subjects

A total of 144 subjects were involved in the study. Of these, 90 were patients and 54 were controls. The patients were consecutive outpatients from Ga-Rankuwa Hospital, HF Verwoerd Hospital and 1 Military Hospital. These patients were referred in response to a request made to neurosurgeons at the three hospitals as was done in the pilot study.

From the original 144 subjects, two control subjects were eliminated from the study because they could not follow instructions. None of the controls were excluded for reasons such as history of mental illness or previous head injury (Appendix C). Thirty six patients were excluded from the study for reasons stated in Table 8.

Table 8: Reasons for exclusion

Reason	N
Could not follow instruction and/ or poor mastery of English	18
Currently on medication	8
Poor vision (Failed to identify 8 lines in screen of Fig. 3).	3
Injury to right hand	4
Other neurological condition (2 epilepsy, 1 history of stroke).	3
TOTAL	36

I did not ask the doctors to keep records of the subjects that they did not refer to me because of the exclusion criteria that I had given them. The reason for this was that the doctors in these clinics work under a great deal of time pressure, and I needed to reduce the paper-work they did for me to a minimum. No records are therefore available on the number of subjects who the doctors did not refer for reasons such as previous head trauma.

The majority of the subjects were excluded because they could not follow instructions. This was in spite of the fact that these patients were judged fit enough for discharge by the medical personnel. From the Screening Questionnaire (Appendix C), none of the subjects indicated that they had a history of psychiatric illness. As a result of the exclusions, a total of 106 subjects remained. These comprised 54 patients and 52 controls.

The control subjects took part on a voluntary basis in response to advertisements. Written advertisements were placed on notice-boards at the University of South Africa (Unisa), the Medical University of Southern Africa (Medunsa), and Ga-Rankuwa Hospital. Subjects from these three locations were invited to encourage friends and relatives to take part. As a result of this, a number of subjects were seen from the suburbs of Mabopane and Mamelodi in Pretoria, and Fourways in Johannesburg. Subjects from Mabopane, Mamelodi, and Fourways were seen at houses belonging to my friends in those suburbs.

Research locations. The research locations for the patients and control subjects are shown in Tables 9 and 10. The majority of patients were seen at HF Verwoerd Hospital.

Table 9: Treatment hospitals and sex distribution for patients

Hospital	Males	Females	Total
Ga-Rankuwa	12	3	15
HF Verwoerd	22	3	25
1 Military	14	0	14
TOTAL	48	6	54

Table 10 Control subjects by research location

Location	Males	Females	Total
Ga Rankuwa Hospital	3	2	5
HF Verwoerd hospital	0	1	1
1 Military hospital	7	1	8
Mabopane	6	3	9
Mamelodi	6	6	12
Medunsa	7	0	7
Johannesburg	0	6	6
Unisa	3	1	4
TOTAL	32	20	52

Demographic Characteristics

Age

The age distributions for both patients and controls are shown in Table 11. No significant difference in mean age between patients and controls were found at the 5% level ($t = -0.82$; $df = 104$; $p = 0,41$).

Education

The mean number of years at school for patients was 9.98 years ($SD = 1.2$), and that for the control group was 8.6 years ($SD = 2.0$). The controls had a significantly higher mean number of years at school than patients ($t = 4.03$; $df = 104$; $p = 0.05$).

Table 11: Age distributions for patients and controls

Category	Mean	SD	N
Patients			
Males	26.35	6.05	48
Females	26.80	8.15	6
Total	26.40	6.23	54
Controls			
Males	24.32	4.47	32
Females	27.21	7.41	20
Total	25.43	5.88	52
Hospital^a			
GR	24.36	4.31	15
HF	27.54	7.01	25
1 Mil	26.16	5.70	14
Total	25.92	6.05	106

^aGR = Ga Rankuwa Hospital; HF = HF Verwoerd Hospital; 1 Mil = 1 Military Hospital.

Occupation

The occupational groupings of the subjects are shown in Table 12. Important differences between patients and controls in respect of their occupations are apparent from this data. There were more unskilled workers amongst the patients than amongst the controls. Only one patient was a professional but there were seven amongst the controls. There were twice as many soldiers amongst the patients than amongst the controls. Finally, considerably more controls had high school and tertiary level education than patients. Overall, controls were of a higher occupational status than patients

Table 12: Occupational groupings

Occupation	Controls	Patients
Unskilled	10	16
Semiskilled	4	5
Technician	3	2
Professional*	7	1
Security officer	1	3
Soldier	8	14
Primary school student	1	2
High school student	5	2
Tertiary student	12	6
Administrative	1	3
Totals	52	54

*The professionals were 3 nurses, 2 teachers, an economist, and a student advisor.

Hospital Data

Brain Injury Data

The Trauma Details Form (Appendix D) was used to obtain the brain injury data of patients. Where patients were unable to provide the required information, the data was obtained from the hospital files. For patients who could provide the information, their answers were double-checked against the information in the files.

Mechanisms of Injury

Data on the source of injury was available on 52 patients. The other 2 patients were not able to provide such information and there was no information available in the patients' files. The distribution of the mechanisms of injury is shown in Table 13.

Table 13: Sources of trauma (N=52)

Hosp ^a	MVA ^b	Sport	Assault	Falls	Total
GR	5	0	8	1	14
HF	13	1	9	1	24
1 Mil	8	3	3	0	14
Total	26	4	20	2	52

^aHosp = Hospital; GR = Ga-Rankuwa hospital; HF = HF Verwoerd Hospital; 1 Mil = 1 Military Hospital; ^bMVA = Motor vehicle accident.

Consistent with previous South African studies (Nell & Brown, 1991), the majority of injuries were sustained in motor vehicle accidents and assaults.

Duration of Loss of Consciousness

Ga-Rankuwa Hospital. The duration of loss of consciousness was estimated from the information provided by the patient about the time of the accident and the time of regaining consciousness (Trauma Details Form, Appendix B). The same information was also sought from the hospital file, especially the "Nurses' Notes", to confirm the patient's report. Some patients regained consciousness after they had been to theater, and this made it impossible to determine whether or not the duration was affected by the procedures and medications given there.

HF Verwoerd Hospital: The same strategy as at Ga-Rankuwa hospital was used. A notable problem at this hospital was that patients often had more than one hospital file, the original one and one or more temporary files. The relevant information was frequently not present in the file the patient was currently using. For such patients, there was no way of verifying the patient's report about loss of consciousness.

1 Military Hospital. The same problems as at Ga-Rankuwa hospital were encountered. Furthermore, once a patient had been discharged, it was no longer possible to access their files.

Estimates of the duration of loss of consciousness based on the patients' reports and perusal of the patients' files for all the hospitals are shown in Table 14. Because of the difficulties pointed out above, these results should be considered only as rough estimates.

Information on the duration of loss of consciousness (LOC) was not available for more than 50% of the sample. Data was available on only 25 patients. Using criteria described in chapter 2 (Russell, 1971; Bond, 1983; Kay et al, 1993), the number of patients with mild ($LOC \leq 30$ minutes), moderate ($LOC = 1-24$ hours) and severe (> 24 hours) brain injury was nine, seven, and nine

Table 14: Estimates of duration of loss of consciousness (N = 51)

Duration	N
Briefly dazed	3
Few minutes to 30 minutes	6
1 hour to 24 hours	7
> 24 hours to 14 days	9
Information not available from any source	26
TOTAL	51

respectively. On this subsample at least, the patients were evenly distributed between mild, moderate and severe. This result suggests that this sample was heterogeneous with respect to severity of injury.

Duration of Post-traumatic Amnesia

The presence of post-traumatic amnesia was assessed in terms of whether patients had a post-traumatic history of short-term memory deficits, inattentiveness, disinhibited behaviour, and restlessness (Appendix B). None of the patients could confidently state for how long these symptoms lasted. The nurses notes did indicate, in some instances, that a patient had these symptoms at some time. It was also not possible to reliably determine the dates when symptoms began and the dates on which the symptoms abated. Because of these difficulties, the attempt to get estimates of PTA was abandoned.

Diagnoses

The diagnoses were not recorded at the time of interviewing the patient. This information was sought after completing the experimental part of the study. The reason for this was so that the most up-to date opinion on the diagnosis, such as the discharge diagnosis, could be obtained.

Ga-Rankuwa Hospital. At this hospital, the International Classification of Diseases (ICD) (World Health Organization, 1992) diagnostic criteria are used for

classifying patients. Diagnostic and other information on all patients is kept on a computerized data base. Of the 15 Ga-Rankuwa Hospital patients who took part in the study, the diagnoses of only eight patients was available on the database. There were three patients diagnosed as cerebral oedema; three were diagnosed as fracture, vault of skull; and two were diagnosed as having had a skull defect.

H.F. Verwoerd Hospital. As was indicated when discussing estimates of duration of loss of consciousness, patients often did not bring a file with the original diagnostic information. Follow-ups were made at the hospital records office. Diagnoses for only nine patients were found. These are listed in Table 15. From this table it is clear that the diagnoses at HF Verwoerd Hospital were highly variable.

Table 15: Patient diagnoses at H.F. Verwoerd Hospital (N = 9)

Diagnosis	N
Temporal contusion	1
Subdural haematoma	2
Broca's aphasia	1
Anterior fossa defect	1
Skull fracture-occiput	1
Base of skull fracture	1
Bilateral frontal contusion	1
Frontal lobe injury	1

Military Hospital. No information can be provided for diagnoses at 1 Military Hospital. It was not possible to access these patients' records once they had been discharged.

On the basis of the limited number of records available from the three hospitals, the patients in this study were heterogeneous in terms of diagnoses.

Instruments

1. A 486 DX66 Sunrace colour notebook computer
2. Hick Box. (Fig. 3)
3. Questionnaires
 - 3.1 Demographic Data Form (Appendix A).
 - 3.2 Trauma Details Form (Appendix B).
 - 3.3 Screening Questionnaire (Appendix C).
 - 3.4 Pretest Questionnaire (Appendix D).
 - 3.5 Postconcussion Symptom Checklist 2 (Appendix F). Several changes were made to the checklist as used in the pilot study. An additional column was added for the patient to indicate a ranking of symptoms. Features of the concussion syndrome checklist used by Gouvier, Cubic, Jones, Brantley, and Cutlip (1992) were also incorporated into the new form: The frequency of symptoms column was increased from a three-point rating to a four-point rating; a column for rating the duration of symptoms was added.
 - 3.6 Symptom Ranking Matrix form (Appendix G): This form which I designed after the pilot study was only used for subjects who had difficulty in ranking symptoms. Its use is explained below.

Procedure

As was the case for the pilot study, subjects were seen after being referred by the neurosurgeons for neuropsychological assessment.

Questionnaires

Questionnaires 3.1 to 3.4 (Appendixes A, B, C, and D) were administered in the same way as for the pilot study.

The Postconcussion Symptom Checklist 2 (Appendix F) was administered in the same way as the Postconcussion Symptom Checklist 1 (Appendix E). The following aspects were added. After the form had been completed for ratings of frequency, duration, and intensity, subjects were asked to rank their symptoms. They were given the following instruction:

"We have just finished identifying the symptoms that bother you. You have reported that you are frequently bothered by....(*A list of all symptoms reported as present would be restated*). Look at the symptoms indicated on this form again and tell me which of these symptoms bothers you most or is the most serious problem for you"

After the subject had indicated a symptom, the subject was asked to identify the second most bothersome symptom. This process continued so that the subject could identify the third and subsequent most bothersome symptoms. For subjects who found it difficult to rank their symptoms, I used the ranking matrix form (Appendix G) as a basis for discussing symptom rankings. The symptoms that the subject would have endorsed were ticked on both axes of the form. The subject was then asked to compare 2 symptoms at a time:

I would like you to think of two of your symptoms at a time and tell me which of the two bothers you more or is a more serious problem for you than the other. Between the two symptoms headache and dizziness (for instance) which you mentioned, which would you say is the more serious?"

If the subject identified one of the symptoms as being more serious than the other, that symptom would be written down in the cell where the two symptoms intersect. If the subject responded that the symptoms were the same neither of the symptoms would be counted. After all the comparisons had been made the number of times that each symptom had been rated more severe than another was counted. I would then say to the subject: "From what we have just been doing, you mentioned headache (for instance) as a serious problem more

frequently than all the other symptoms. Is it your experience that it is the most serious problem?"

The same strategy was used to identify the second, and subsequent, most bothersome symptoms.

Reaction Times

As a result of the observations made during the pilot study, the following modifications were made to the reaction time testing procedure: In addition to the familiarization practice, the computer programme now had the facility for the subject to have more practice trials at the beginning of each task condition. If a subject made any mistake at the beginning of each trial the experimenter said: "That last response was incorrect, let me give you a chance to practice again before beginning the full test." The experimenter then pressed the letter R (for repeat) on the keyboard and the experiment would restart from trial number one. Previous responses on this condition would automatically be erased. If the subject was still unable to perform the task, after a further five trials, the experimenter said: "Good, that will be all for today. You can stop now." By pressing the escape button, the experiment would end for this subject.

After the subject had understood the instructions, he or she was told: "You can now start. The first few trials are only for practice." The subject would then begin. If he or she made mistakes these would be pointed out, and the subject told to retry. If a subject managed five correct responses successively, he would be told: "I think you can now do this well. Let us now start the full set of trials from the beginning." The experimenter then pressed the 'R' button on the keyboard in order to (R) repeat the trial. The full set of 16 trials would then follow. From this point onwards any further errors made would be pointed out to the subject, but the experiment would continue until all the 16 trials had been done.

CHAPTER EIGHT

RESULTS AND DISCUSSION

Questionnaires and Postconcussion Symptoms

Administration of the questionnaires presented no problems except for one item on the Concussion Symptoms Checklist 2 (Appendix F). After the pilot study, it had been decided to use an additional column for subjects to rate the duration of the symptom. It became apparent from the beginning of the attempts to ask for this rating that it was meaningless for several symptoms. For example, whilst it makes sense to ask someone for how long he or she has had memory problems, it does not mean much to ask how long the forgetfulness lasts. Similarly, for the symptoms light sensitivity and noise sensitivity, the problems naturally last for as long as the irritating stimulus is present. The item *duration* on this questionnaire was therefore omitted in assessing all subjects.

The postconcussion symptoms that were endorsed as present by patients as well as controls are shown in Table 16. It is clear from the table that some symptoms had very low counts. For the patients, the symptoms with very low counts included auditory problems, nausea, alcohol sensitivity, and vomiting. The controls had even more of these low count symptoms. The significance of these low symptom counts is that some of the findings reported in this study may be an artefact of small samples.

Another potential difficulty is the question of how accurately subjects reported symptoms, and the validity of the symptom constructs. As is the case with most of the available symptom checklists, the symptoms are subjectively, rather than objectively assessed. The accuracy and truthfulness of these reported symptoms is thus not

Table 16: Frequency of symptoms endorsed as present by patients and controls.

Symptoms	Patients (N=54)		Controls (N=52)	
	Frequency	%	Frequency	%
Headache	32	59.30	23	44.20
Fatigue	28	51.90	9	17.30
Dizziness	22	40.70	6	11.50
Memory	22	40.70	14	26.90
Irritability	20	15.40	8	15.40
Visual	18	33.30	9	17.30
Noise sensitivity	17	31.50	8	15.40
Concentration	16	29.60	18	34.60
Drowsiness	16	29.60	0	0.00
Depression	13	24.10	8	15.40
Light sensitivity	12	22.20	6	11.50
Anxiety	11	20.40	10	19.20
Auditory	6	11.10	0	0.00
Nausea	6	11.10	0	0.00
Alcohol sensitivity	2	3.70	2	3.80
Vomiting	2	3.70	0	0.00

known. Because the symptoms are not operationally defined, each patient may have a different understanding of the meanings of the symptoms (Bohnen & Jolles, 1992). It is feasible that some subjects may confuse the meaning of one symptom with the meaning of another symptom. For example, on the Postconcussion Symptom Checklist 2 (Appendix F) the following symptoms could be mistaken to mean the same thing: fatigue and drowsiness; visual problems and light sensitivity; noise sensitivity and auditory problems; anxiety and depression. Furthermore, as was pointed out in Chapter 4, symptoms such as dizziness do not have a precise medical definition. Dizziness for example, is often confused for vertigo, near-syncope, and ill-defined lightheadedness (Anderson, Yolton, Reinke, Kohl, & Lundy-Ekman, 1995).

The other problem relates to the validity of symptom constructs. The Postconcussion Symptom Checklist 2 (Appendix F) was designed for American subjects (Gouver, Cubic, Jones, Brantley, & Cutlip, 1992). The question is whether those symptoms mean the same thing to the South African sample used in this study. Nell (in press) argues that the problems of construct comparability multiply as one moves across cultures. Two of these difficulties were identified by Nell, Myers, Colvin, & Rees (1993) whilst attempting to administer the Profile on Mood States (POMS) on a sample of factory workers whose mother tongue was Zulu or Sotho. The first difficulty that the test administrators noted was that the number of synonyms in these African languages was inadequate for all the synonyms in the POMS on a given dimension. "For example, fatigue is described by the terms worn out, listless, sluggish, weary, bushed; anger is described by the terms peeved, grouchy, annoyed, ready to fight, and bad-tempered" (Nell et al., 1993, p. 310). The second difficulty was that the subjects had difficulties distinguishing between *traits* and *states*. They tended to respond in terms of their current state, and not the mood trait.

In the present study, because of the problem of small samples, and the potential problems in the comparability of constructs just described, the findings based on the symptom checklist (Appendix F) ought to be treated as tentative.

Preliminary Analyses of Reaction Time Data

Errors and Outliers

As was the case with previous studies using these tasks (Shum, McFarland, Bain, & Humphreys, 1990), the number of errors and outliers was calculated. The percentage of errors and outliers made by patients and controls is shown in Tables 17 and 18.

The percentage of errors was much less than that found in the pilot study. The highest error rate was for the *similar* condition, where the number of correct responses was just under 90%. On all the other task conditions, the correct response rate was more than 90%. The error rate was now comparable to that found in previous studies (Shum, McFarland, Bain, & Humphreys, 1990; Murray, Shum, & McFarland, 1992; Shum, McFarland, & Bain, 1994).

The percentages of outliers for the eight task conditions are shown in Table 18. It is clear from this table that the percentage of outliers for all eight tasks was very low. They are comparable to previous studies (Shum, McFarland, Bain & Humphreys, 1990). This means that very few responses would be eliminated during statistical analyses.

Data Skewness

The positively skewed nature of reaction time data was discussed in the pilot study. As a result of this positive skew, it has been suggested that outliers should be eliminated (Tabachnick & Fidell, 1983). In the present data set, after the errors and outliers had been eliminated, the skewness of the data for the eight different tasks was as shown in the Table 19. The task conditions are grouped in pairs, with the level 1 tasks recorded first.

Table 17: Percentages of errors on reaction time tasks

Task	Patients	Controls	Combined
Degraded	2.87	1.68	2.32
Undegraded	2.87	1.56	2.27
Compatible	2.25	1.08	1.71
Incompatible	4.41	4.09	4.26
Similar	10.25	8.89	2.43
Dissimilar	1.23	2.64	1.88
Fixed foreperiod	1.84	1.08	1.49
Variable foreperiod	1.23	1.32	1.27

Table 18: Counts of outliers as a percentage of correct responses.

TASK	PRT ^a	PMT ^b	CRT ^c	CMT ^d	CORT ^e	COMT ^f
Degraded	4.54	2.22	1.22	0.12	3.00	1.25
Undegraded	6.54	1.48	1.22	0.24	4.07	0.91
Compatible	5.14	4.19	1.94	1.09	3.66	2.76
Incompatible	6.86	5.57	1.75	2.63	4.51	4.22
Similar	6.39	4.79	0.92	1.98	3.57	3.23
Dissimilar	5.81	0.93	1.11	0.12	3.66	0.56
Fixed foreperiod	5.01	1.36	1.09	0.36	3.20	0.90
Variable foreperiod	7.78	1.35	1.22	0.00	4.76	0.73

Note

^a PRT = Patients' reaction time; ^b PMT = Patients movement time

^c CRT = Controls' reaction time; ^d CMT = Controls movement time

^e CORT = Combined reaction time (Patients & Controls).

^f COMT = Combined movement time (Patients and controls).

Table 19: Skewness: Reaction time, movement time, and total reaction time

Task condition	Reaction time		Movement time		Total reaction time	
	P ^a	C ^b	P	C	P	C
Degraded	-0.06	1.44	0.47	0.85	0.15	2.08
Undegraded	-0.09	0.58	1.01	0.71	0.35	0.58
Incompatible	0.26	0.14	0.52	0.64	0.38	0.54
Compatible	-0.17	0.41	0.57	0.29	0.28	-0.09
Similar	0.04	0.55	0.45	0.39	0.36	0.03
Dissimilar	0.05	0.36	0.84	0.19	0.46	0.40
Variable foreperiod	0.02	0.25	0.86	0.21	0.70	0.24
Fixed foreperiod	0.01	0.65	0.47	0.32	0.43	0.47

Note: ^aP = patients; C = controls

With the exception of the reaction time indices of the *degraded*, *undegraded*, and *compatible* task conditions, as well as the total reaction time for the task condition *compatible*, all scores were positively skewed. This suggests that the tasks were relatively easy for the subjects. Furthermore, with the exception of reaction time and total reaction time scores of the *degraded* task for controls, and the *undegraded* movement time for patients, all the scores were less than one. They thus approached normality, which makes them suitable for inferential statistics.

Tests of the Hypotheses

Hypothesis 1

Patients who have sustained a concussion will show greater frequency and intensity of postconcussion symptoms than controls; and patients will be slower in performing reaction time tasks.

There are two parts to this hypothesis. In the first part, it is predicted that there are significant differences between patients and controls with respect to the ratings of the frequency and intensity of postconcussion symptoms. The second part of the hypothesis states that patients will be slower than controls. The results for each portion of the hypothesis will be reported in turn.

Frequency and Intensity of Postconcussion Symptoms

Cross tabulations to establish the distribution of the subjects according to the ratings of symptom frequencies and ratings of symptom intensities were performed. These entailed 3 x 2 tables (3 rating categories for frequency/ intensity and 2 groups ie patients and controls). The cross tabulations resulted in 16 tables. Of these, 11 tables had some cells with frequencies less than 5. Chi-square tests were therefore not suitable for comparing patients and controls.

As an alternative to the chi-square, t -tests for independent measures were performed on the means of the frequency ratings and intensity ratings. From the 16 symptoms, a total of 32 t -tests were attempted (16 for symptom frequency ratings + 16 for symptom intensity ratings). It was not possible to compute t -tests on 8 ratings because of empty cells in one or other of the groups. These were the frequency ratings for drowsiness, hearing problems, nausea, and vomiting; and the intensity ratings for drowsiness, anxiety, nausea, and vomiting. There thus remained 24 comparisons. The results of the t -tests for independent measures comparing patients and controls are shown in Tables 20 and 21.

The first part of Hypothesis 1 was confirmed for symptom frequencies (Table 20). Of the nine symptoms that were used in analyses, seven showed significant differences between patients and controls. In four out of these symptoms, patients reported greater frequency, and in two of the symptoms, controls reported greater frequency. These results indicate that overall, patients report greater symptom frequencies than controls. This finding is contrary to some previous studies which found that patients and controls report these symptoms equally (Bohnen et al., 1992; Wong, 1994). There is at least one possible reason why differences may have been detected in the present study, contrary to some previous studies. In the present study, there was greater conciseness, in that symptom frequency ratings, rather than mere symptom presence were used for making the comparisons.

Symptom intensity ratings were analysed in the same way as symptom frequency ratings. The results are shown in Table 21. Hypothesis 1 was also confirmed for symptom intensities. Comparisons were made for 11 symptoms. There were significant differences for seven symptoms and no differences were found for four symptoms. The symptoms for which there were no differences are poor concentration, alcohol sensitivity, anxiety, and depression. On all the symptoms on which there was a significant difference, patients had greater mean intensities than

Table 20: t-tests for independent samples on symptom frequencies.

Symptom	Mean ^a	t	df	2-tail p
Headache	1.0	2.94	90.79	0.004
	0.5			
Dizziness	0.05	3.93	70.68	0.000
	0.1			
Memory	0.6	2.69	89.25	0.009
	0.2			
Fatigue	0.8	4.45	75.36	0.000
	0.1			
Noise sensitivity	0.05	1.99	91.15	0.05
	0.2			
Irritability	0.75	3.46	70.46	0.001
	0.19			
Concentration	0.5	0.7	90.19	0.4
	0.4			
Visual	0.6	2.2	90.22	0.02
	0.2			
Anxiety	0.29	0.75	98.32	0.45
	0.21			

Note

^aFor each symptom, the mean for the patients is reported in the first row and the mean for the controls are in the second row.

Table 21: t-tests for independent samples on symptom intensities.

Symptom	Mean ^a	t	df	2-tail p
Headache	1.2	3.59	88.39	0.001
	0.5			
Dizziness	0.8	4.4	65.04	0.000
	0.1			
Fatigue	0.9	4.72	69.24	0.000
	0.1			
Noise sensitive	0.5	2.6	76.61	0.01
	0.1			
Irritability	0.85	3.2	74.58	0.001
	0.23			
Light sensitive	0.3	2.5	73.50	0.01
	0.0			
Concentration	0.6	0.5	101.1	0.57
	0.5			
Alcohol sensitive	0.09	0.76	70.01	0.07
	0.03			
Visual	0.64	2.57	83.72	0.01
	0.21			
Anxiety	0.38	0.81	99.32	0.42
	0.26			
Depression	0.46	0.92	99.84	0.35
	0.30			

Note

^aFor each symptom, the mean for the patients is reported in the first row and the mean for the controls are in the second row.

controls. For symptom intensity ratings then, as for symptom frequency ratings, patients had higher scores than controls.

Differences on Reaction Time Indices

Patients and controls were compared on their performance on three reaction time indices. These are reaction time (RT), movement time (MT), and total reaction time (TT). To test for the differences, *t*-tests for repeated measures were used. The results are shown in Tables 22, 23, and 24.

There were significant differences for all eight task conditions for the three reaction time indices (RT, MT, and TT). The second part of Hypothesis 1 was therefore confirmed, so that, overall, Hypothesis 1 was confirmed. It can further be observed that patients performed slower than controls on all the conditions.

Hypothesis 2

For both patients and controls, the symptoms tend to form clusters. However, the clusters identified for the patients differ from those identified for the controls.

Hypothesis 2 was confirmed, as will be clear from the results presented below. For this analysis, the symptoms were coded into six levels of severity. Initially it was intended that the level of severity would be determined by symptom rankings, symptom frequency, and symptom intensity as shown on the Postconcussion Symptom Checklist 2 (Appendix F). On this basis, the most severe symptom would be one with a rank of 1, a frequency rating of 3, and an intensity rating of 3. When this criterion was applied to headache, which was reported most frequently by both patients and controls (Table 16), the symptom was coded as most severe by only 8 patients and 2 controls. Because of the small number of subjects involved, the use of

Table 22: t-tests for independent samples on reaction time (RT).

Task condition	Mean ^a	SD	t	df	2-tail p
Incompatible	56.70	13.05			
	47.62	11.18	3.76	104	0.000
Compatible	46.05	9.23			
	39.99	8.16	3.46	104	0.001
Undegraded	50.30	10.19			
	40.38	8.54	5.33	104	0.000
Degraded	62.03	16.25			
	46.15	13.08	5.53	100.5	0.000
Similar	51.52	12.19			
	44.26	8.49	3.25	101.5	0.002
Dissimilar	47.29	9.47			
	39.44	8.49	4.41	104	0.000
Variable foreperiod	62.11	12.05			
	51.48	10.08	4.80	104	0.000
Fixed foreperiod	56.56	11.16			
	46.11	9.51	5.07	104	0.000

Note

^aFor each task condition, the mean for the patients is reported in the first row and the means for the controls in the second row.

Table 23 t-tests for independent samples on movement time (MT).

Task condition	Mean ^a	SD	t	df	2-tail p
Incompatible	48.54	15.75			
	40.23	10.82	3.17	94.18	0.002
Compatible	46.14	13.69			
	36.30	8.25	4.41	88.32	0.000
Undegraded	48.89	16.54			
	36.95	9.73	4.55	86.35	0.000
Degraded	50.50	15.72			
	38.07	10.00	4.84	90.33	0.000
Similar	51.23	15.60			
	41.71	11.88	3.52	104	0.001
Dissimilar	47.69	14.89			
	36.58	8.52	4.82	84.96	0.000
Variable foreperiod	48.03	16.15			
	36.47	8.72	4.61	82.13	0.000
Fixed foreperiod	46.22	14.45			
	36.80	8.62	4.09	87.07	0.000

Note

^a For each task condition, the mean for the patients is reported in the first row and the means for the controls in the second row.

Table 24: t-tests for independent samples on total reaction time (TT).

Task condition	Mean ^a	SD	t	df	2-tail p
Incompatible	104.82	22.96			
	87.64	16.95	4.37	104	0.000
Compatible	91.97	18.48			
	76.67	11.24	5.17	88.06	0.000
Undegraded	99.22	22.04			
	77.19	12.83	6.32	85.84	0.000
Degraded	111.80	26.86			
	84.37	17.80	6.22	92.39	0.000
Similar	102.11	22.99			
	86.03	16.36	4.16	95.85	0.000
Dissimilar	94.88	20.34			
	76.03	13.39	5.65	92.04	0.000
Variable foreperiod	109.56	24.68			
	87.84	14.91	5.51	87.68	0.000
Fixed foreperiod	102.52	22.89			
	82.88	13.96	5.35	88.21	0.000

Note

^a For each task condition, the mean for the patients is reported in the first row and the means for the controls in the second row.

rankings was dropped as a severity rating criterion. Only symptom frequency and symptom intensity ratings were subsequently used. The categories of ratings using this system are shown in Table 25.

The symptom clusters were analysed at different durations since injury. The different durations since injury were assessed cross-sectionally, and not longitudinally. For each patient, the duration since injury was calculated by subtracting the date of trauma from the date of assessment (Trauma Details Form, Appendix B).

Taking into account the durations since injury was done in view of previous research reports, such as that by Rutherford (1989), which indicate that symptoms

Table 25: Classification of symptoms according to severity^a.

	Intensity			
Frequency	0	1	2	3
0	0	X	X	X
1	X	1	2	4
2	X	2	3	5
3	X	4	5	6

Note.

^a Symptom severity from 0 = no symptoms; to 6 most severe. In the no symptom category (0), the symptom has zero frequency and zero intensity; in the most severe category (6), the symptom has a frequency rating of 3 and an intensity rating of 3.

change over time. Initially, it was proposed to make cut-off points at two weeks (acute stage), three months, and more than three months. The cut-off at three months was made because it has been reported that there are generally low correlations between organic indices of brain injury and postconcussion symptoms after three months (Lishman, 1988). It was thus important to make this demarcation, with a view to comparing reported symptoms and performance on reaction time.

When two weeks since injury was used as the cut-off point for acute injury, only 12 patients met the criteria for being at that acute stage. This number was

considered too small for factor analyses. To increase the number of patients in the earliest duration since injury, the cut-off was moved to 21 days. This stage was referred to as acute/subacute. The next cut-off point was at three months (90 days). Only patients whose date of injury could be ascertained were included in the analyses. Because of low counts, the symptoms auditory problems, nausea, alcohol sensitivity, and vomiting were excluded from factor analyses. Symptoms clusters were identified by principal components analysis with varimax rotation. The results of factor analyses for patients and controls are described below.

Patients

Factor Analyses for 1 to 21 Days Since Injury

The results for the acute/subacute stage are shown in Table 26. Factor 1, which explains 36.7% of the variance was labelled as *somatic* because of the prominence of the symptoms headache and dizziness. The factor label *somatic* has been used for headache and dizziness by previous researchers such as Levin, Gary et al. (1987). Factor 1 was also called *cognitive* because of the symptom memory. This cognitive nature of this factor is strengthened by the fact that poor concentration, on Factor 3, also loaded highly on Factor 1. Furthermore, memory also loaded highly on Factor 3, which has a cognitive component. The association between cognitive symptoms and somatic symptoms can be explained in terms of the coping hypothesis (Gronwall & Wrightson, 1974; Hinkely & Corrigan, 1990 ; Van Zomeren, Brouwer, & Deelman, 1984) which was described in Chapter 3. To recapitulate, the coping hypothesis states that traumatic brain injury may be associated with cognitive problems, specifically slowed information processing. In order to cope with this

Table 26: Symptom clusters 1- 21 days since injury (N = 15).

Symptoms	Factor 1 Somatic/ cognitive	Factor 2 Visual/ arousal	Factor 3 Cognitive / depression
Dizziness	,85	,12	,10
Headache	,75	,10	,09
Fatigue	,69	,21	-,00
Memory	,64	,07	,56
Noise	,53	-,10	,39
Light	,18	,82	-,08
Drowsiness	,46	,72	,02
Visual	,12	,68	,19
Anxiety	,15	-,61	-,26
Irritability	,29	-,02	,78
Depression	-,13	,33	,74
Concentration	,46	,39	,58

Note.

Factor	Eigenvalue	% of Variance	Cum %
1	4,41	36,7	36,7
2	1,75	14,6	51,3
3	1,36	11,3	62,6

impairment, patients exert more effort when dealing with cognitive tasks. The increased effort is stressful, and leads to somatic symptoms such as headache and dizziness. The coping hypothesis does not imply that cognitive deficits are the only cause of the somatic symptoms. As the review of the literature in Chapter 4 showed, numerous factors, both organic and psychological, contribute to these symptoms. Increased coping effort may lead to somatic symptoms in some cases, and an exacerbation of somatic symptoms in other cases.

At first glance, it appears that fatigue and noise sensitivity do not fit in easily with the other symptoms on Factor 1. However, as the literature review in Chapter 3 suggested, fatigue and symptoms such as dizziness (Montgomery, Fenton, McClelland, MacFlynn, & Rutherford, 1991), and noise sensitivity (Bohnen, Twijnstra, Wijnen, & Jolles, 1991) may all have brainstem dysfunction in common.

Factor 2 has two visual symptoms, namely light sensitivity and visual problems, and an arousal symptom (drowsiness). For this reason, the factor was labelled as *visual/arousal*. The importance of arousal for Factor 2 is further suggested by the fact that fatigue, another arousal symptom, which loads on Factor 1, also loaded moderately on Factor 2. The symptom anxiety had a negative loading on Factor 2, suggesting that it does not belong with the other symptoms that load here.

Factor 3 was called *cognitive/depression* because of the symptoms poor concentration (cognitive) and depression. Levin, Gary et al. (1987), cited in Chapter 3, also found a similar factor. Irritability may be an emotional reaction to either or both of these two symptoms. Poor concentration also loads high on both Factor 1 and Factor 2. The reason for the loading of poor concentration, a cognitive symptom, with somatic symptoms in Factor 1 has already been discussed. It is also reasonable that poor concentration should load with arousal problems. The brain injuries that lead to arousal deficits affect the brain diffusely (Luria, 1973). Such injuries are also bound to affect cognitive processes, including concentration, as was discussed in Chapter 2.

Factor Analyses for 22-90 Days Since Injury

The symptoms loaded onto three factors as shown in Table 27. Three subsets of symptoms are discernible in Factor 1. They are *arousal* (drowsiness and fatigue), *cognitive* (memory and concentration), and *somatic* (headache and dizziness). Possible explanations for the associations between arousal, somatic, and cognitive symptoms were already given in the discussion for Table 26. Irritability may be an emotional reaction to any of the other symptoms, or a combination of symptom. Mittenberg, Zielinski, and Fichera (1993) have suggested that one of the most frequent causes of irritability and poor concentration is fatigue, which also loads on this factor. The symptom drowsiness has almost as high a loading on Factor 1 as it has on Factor 2.

Factor 2 has two visual sense symptoms (light sensitivity and visual problems), and two emotional symptoms (anxiety and depression). The presence of light sensitivity together with visual problems may be the result of the poor operational definitions of symptoms in the study. It is feasible that the sensitivity to light that some of the patients complained of were the visual problems that they also endorsed on the questionnaire. Anxiety and depression have also been found to load together after traumatic brain injury in some previous studies (Bohnen, Twijnstra, & Jolles, 1992). There is no readily apparent reason why the emotional symptoms in Factor 2 should load together with visual problems.

Noise sensitivity is the only symptom on Factor 3. It is notable that the somatic symptoms headache and dizziness (Factor 2) also load highly on Factor 3. A possible explanation for the association is that brainstem damage may be associated with both dizziness (Montgomery, Fenton, McClelland, MacFlynn, & Rutherford, 1991), and noise sensitivity (Bohnen, Twijnstra, Wijnen, & Jolles, 1991; Waddell & Gronwall, 1984).

Table 27. Symptom clusters 22 - 90 days after injury (N = 16)

Symptoms	Factor 1 Arousal/ Cognitive/Somatic	Factor 2 Visual/emotional	Factor 3 Noise sensitivity
Memory	,94	,02	-,18
Concentration	,92	,16	,22
Irritability	,92	-,03	-,01
Fatigue	,85	,14	,05
Headache	,76	,02	,57
Dizziness	,68	,00	,63
Drowsiness	,68	,67	,14
Light	,02	,96	,17
Anxiety	,11	-,83	,03
Depression	,16	,67	-,32
Visual	,63	,63	-,13
Noise	-,05	-,06	,85

Note.

Factor	Eigenvalue	% of Variance	Cum %
1	5,86	48,8	48,8
2	2,62	21,8	70,6
3	1,43	11,9	82,5

Factor Analyses for 91 Days or more Since Injury

The results for the duration 91 days or more after injury are shown in Table 28. Five factors were identified. Factor 1 was labelled *arousal/ somatic* because of the arousal symptom fatigue, and the two somatic symptoms headache and dizziness. The two somatic symptoms did not load highly with any other factor. Fatigue had a moderately high loading on factor 3. Factor 2 was labelled *cognitive* because of two symptoms, memory, and concentration. Anxiety loaded negatively with this factor. Noise sensitivity had a low loading, and furthermore also loaded on both Factor 2 and Factor 5. Factor 3 was made up of two visual symptoms (light sensitivity and visual problems), and the one arousal symptom (drowsiness). The reason for the association between visual problems and arousal is not readily apparent. This association was also reported for the duration 1 to 21 days (Table 26). There was one symptom each for Factor 4 and Factor 5. It is notable that depression loads on the irritability factor (0.27), and irritability likewise loads on the depression factor (0.27). This suggests that these symptoms have something in common. It could be that depression gives rise to irritability. Another possibility is that the two symptoms have a common aetiology, notably damage to limbic structures (McLelland, 1988), as was discussed in Chapter 3.

Controls

The results for the controls are presented in Table 29. Three factors were identified. For Factor 1 there were three sets of symptoms. Headache and dizziness are classified as *somatic complaints* as was done for patients. Levin, Gary et al. (1987) classified noise sensitivity as a *sensory* symptom. The same label was adopted here. The other sensory symptom is light sensitivity. Irritability, which also loads moderately on Factor 2 is possibly partly an emotional reaction to the somatic

Table 28: Symptom clusters 91 days + after injury (N = 19).

Symptoms	Factor 1 Arousal/ Somatic	Factor 2 Cognitive	Factor 3 Visual/ arousal	Factor 4 Depression	Factor 5 Irritability
Fatigue	,85	-,05	-,34	,18	-,06
Dizziness	,79	-,02	,13	-,12	,06
Headache	,76	-,02	,17	,06	,13
Concentration	,01	,88	,06	,42	-,05
Anxiety	,28	-,70	,20	-,03	,01
Memory	,62	,67	,04	-,13	,21
Noise	,46	,59	,12	-,37	,36
Light	,09	-,09	,81	-,29	,14
Visual	-,06	-,07	,79	,21	,09
Drowsiness	,26	,37	,64	,39	-,36
Depression	,03	,14	,05	,91	,27
Irritability	,17	,07	,13	,27	,92

Note

Factor Eigenvalue % of Variance Cum Pct

1	3,35	27,9	27,9
2	2,09	17,4	45,3
3	1,87	15,6	60,8
4	1,33	11,1	72,0
5	1,09	9,1	81,0

Table 29: Symptom Clusters for Controls (N = 52)

	Factor 1 Somatic/sensory & irritability	Factor 2 Cognitive/ arousal & visual	Factor 3 Anxio/depressive
Noise sensitivity	,84	-,10	,01
Irritability	,81	,23	-,07
Light sensitivity	,65	,12	,14
Headache	,62	-,04	,04
Dizziness	,46	-,01	,26
Memory	-,11	,79	,19
Visual	,22	,76	-,12
Concentration	-,20	,63	,50
Fatigue	,07	,49	,03
Depression	,19	,19	,87
Anxiety	,11	-,01	,86

Note

Factor	Eigenvalue	% of Variance	Cum Pct
1	2,83	25,7	25,7
2	2,18	19,8	45,5
3	1,38	12,6	58,1

and sensory symptoms. Factor 3 has three sets of symptoms, cognitive, arousal and visual. These are memory problems and poor concentration (cognitive), fatigue (arousal), and visual problems. Factor 3 consists of two emotional symptoms, that were classified as *anxi/depressive*.

Hypothesis 2 was confirmed by the results presented in Tables 26, 27, 28, and 29. None of the symptom clusters identified for the patients were identical to the clusters for controls. It is notable however, that for both patients and controls, headache and dizziness always loaded on the same factor. This suggests that there is a strong relationship between these two symptoms, which have been classified as *somatic*.

Hypothesis 3

For both patients and controls, level 1 tasks will be associated with significantly longer response times than level 2 tasks.

The results for this hypothesis were analysed in terms of three categories of indices of reaction time. These were reaction time (RT), movement time (MT), and total reaction time (TT). To test for differences, *t*-tests for repeated measures were applied. The results are shown in Tables 30, 31, and 32. The RT differences are shown in Table 30. For both patients and controls, one tailed *t*-tests for independent samples revealed that Level 1 tasks had significantly longer response times than level 2 tasks. The hypothesis was thus confirmed for RT.

The hypothesis was not confirmed for movement time (Table 31). From the eight comparisons, there were five significant differences and three nonsignificant differences. For both patients and controls, no significant differences were found for the signal quality (degraded vs undegraded) conditions. Furthermore, for the controls, there were no significant differences for the foreperiod uncertainty (fixed foreperiod

Table 30: Mean reaction time (RT) differences between Level 1 and Level 2 tasks for patients (N = 54; df = 53) and controls (N = 52; df = 51)

TASKS ^a	Group ^b	MEAN	SD	r	t	p
Incompatible	P	56.7	13.05			
	C	47.62	11.18			
Compatible	P	46.05	9.23	0.74	-9.00	0.000
	C	39.99	8.16	0.65	-6.51	0.000
Similar	P	51.52	12.19			
	C	44.26	8.49			
Dissimilar	P	47.29	9.47	0.65	-3.36	0.001
	C	39.44	8.49	0.67	-4.53	0.000
Variable	P	62.11	12.05			
	C	51.48	10.08			
Fixed	P	56.56	11.16	0.81	-5.71	0.000
	C	46.11	9.51	0.87	-7.87	0.000
Degraded	P	62.03	16.25			
	C	46.15	13.08			
Undegraded	P	50.3	10.19	0.76	8.15	0.000
	C	40.38	8.54	0.76	4.85	0.000

Note

^a For each pair of reaction time tasks, the scores for the level 1 tasks are reported in the first row, and those for the level 2 tasks are reported in the second row.

^b P = patients; C = controls.

Table 31: Mean movement time (MT) differences between Level 1 and Level 2 tasks for patients (N = 54; df = 53) and controls (N = 52; df = 51)

TASKS ^a	Group ^a	MEAN	SD	r	t	p
Incompatible	P	48.54	15.75			
	C	40.23	10.82			
Compatible	P	46.14	13.39	0.85	-2.12	0.03
	C	36.60	8.25	0.83	-4.30	0.00
Similar	P	51.23	15.60			
	C	41.71	11.88			
Dissimilar	P	47.89	14.89	0.83	-2.79	0.00
	C	36.58	8.52	0.81	-5.26	0.00
Variable	P	48.03	16.15			
	C	36.47	8.72			
Fixed	P	46.22	14.45	0.94	-2.44	0.01
	C	36.80	8.62	0.84	0.49	0.62
Degraded	P	50.50	15.72			
	C	38.07	10.00			
Undegraded	P	48.89	16.54	0.83	1.21	0.23
	C	36.95	9.73	0.58	0.90	0.37

Note

^a For each pair of reaction time tasks, the scores for the level 1 tasks are reported in the first row, and those for the level 2 tasks are reported in the second row.

^b P = patients; C = controls

Table 32: Mean total reaction time differences between Level 1 and Level 2 tasks for patients (N = 54; df = 53) and controls (N = 52; df = 51)

TASKS ^a	Group ^a	MEAN	SD	r	t	p
Incompatible	P	104.82	22.96			
	C	87.64	16.95			
Compatible	P	91.97	18.48	0.81	-7.05	0.00
	C	76.67	11.24	0.82	-8.01	0.00
Similar	P	102.11	22.99			
	C	86.03	16.36			
Dissimilar	P	94.88	20.34	0.79	-3.78	0.00
	C	76.03	13.39	0.73	-6.48	0.00
Variable	P	109.56	24.68			
	C	87.84	14.91			
Fixed	P	102.52	22.89	0.92	-5.42	0.00
	C	82.88	13.96	0.86	-4.71	0.00
Degraded	P	111.80	26.86			
	C	84.37	17.80			
Undegraded	P	99.22	22.04	0.88	7.29	0.00
	C	77.19	12.83	0.70	4.10	0.00

Note.

^a For each pair of reaction time tasks, the scores for the level 1 tasks are reported in the first row, and those for the level 2 tasks are reported in the second row.

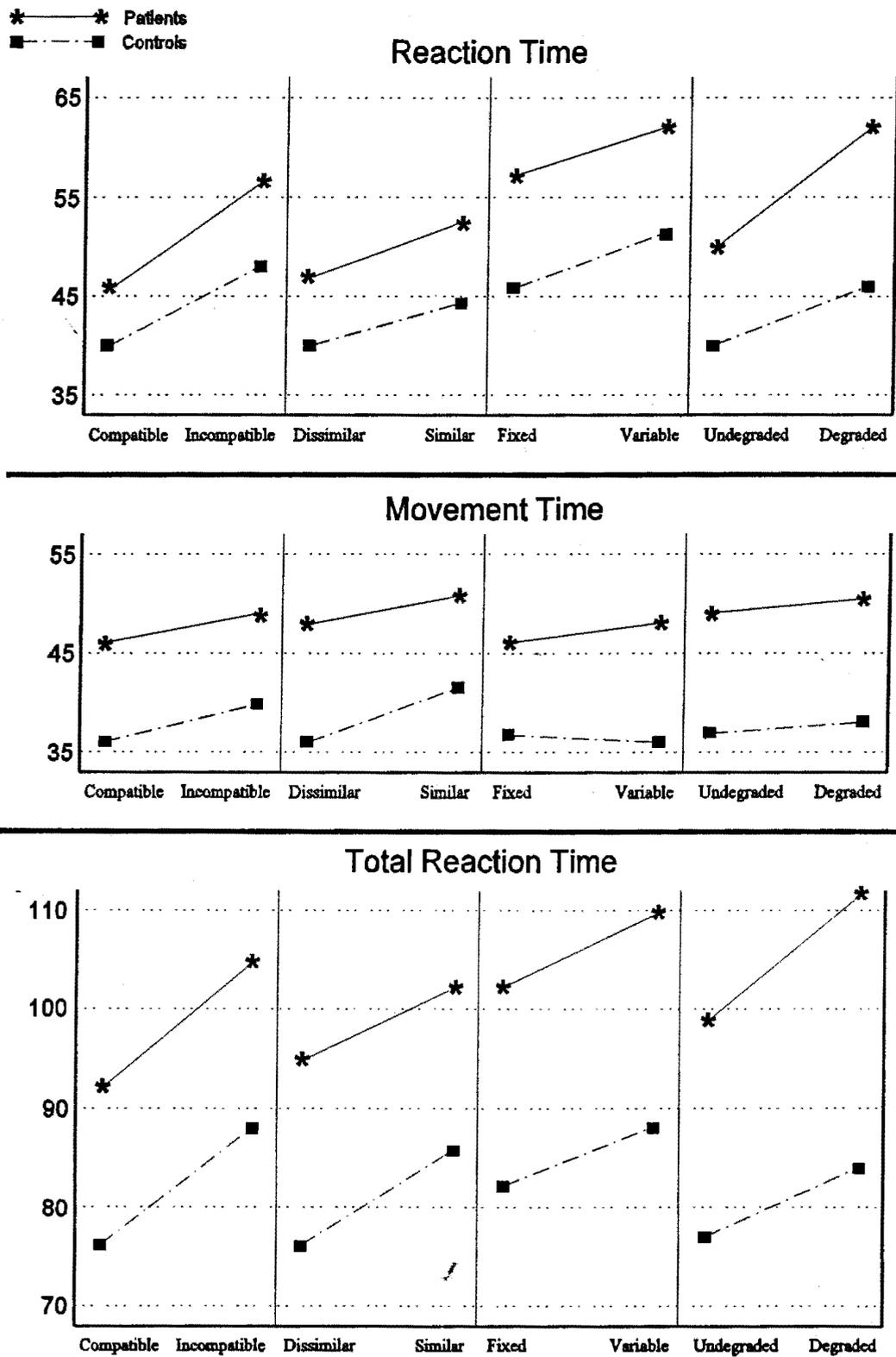
^b P = patients; C = controls.

and variable foreperiod) task conditions. Because MT does not contain decision time, (Jensen & Munro, 1979), the hypothesis was not confirmed for response execution only. On total reaction time, Hypothesis 3 was supported for both patients and controls.

The results from Tables 30, 31, and 32, are illustrated graphically in Figure 4. From these graphs, a number of observations can be made about the trends of the relationships between level 1 tasks and level 2 tasks in patients and controls.

1. All the plots, with the exception of movement time for foreperiod uncertainty (fixed - variable) of the patient group, sloped upwards from level 2 to level 1. This suggests that for both patients and controls, level 1 tasks were more difficult than level 2 tasks.
2. The line representing the patients lies higher than that for controls on all tasks. This suggests that the response times for patients are longer than those for controls on all tasks.
3. Wider gaps between the two plots are found for total reaction time than for either reaction time or movement time. This suggests that total reaction time discriminates between patients and controls better than movement time and reaction time.
4. The gaps between patient plots and control plots are wider in some tasks than others. In reaction time and in total reaction time, the gaps for signal quality (Undegraded - Degraded) and foreperiod uncertainty (Fixed - Variable) are wider than those for stimulus discriminability and S-R compatibility. These

Figure 4. Graphical representation of mean differences between task conditions.



features suggest that signal quality and foreperiod uncertainty discriminate between patients and controls better than the latter two task variables.

5. The slope of the plots appears greatest for total reaction time followed by reaction time. Movement time has the smallest slope of all the three categories of reaction time indices. The smallness of slopes in movement time are particularly evident on the foreperiod uncertainty and the signal quality tasks. From these graphs, it seems that total reaction time enhances the differences between level 1 and level 2 tasks more than either reaction time or movement time alone.

6. The slopes for the patients and controls are generally not parallel. Foreperiod uncertainty RT and S-R total reaction time appear to be the exceptions. This suggests that generally, the magnitude of the differences between level 1 and level 2 tasks is different for patients and controls: On reaction time, the magnitude of the difference for patients is greater than that for controls on all tasks. On movement time, the results appear to be mixed. For S-R compatibility and stimulus discriminability, the magnitude of the differences appears to be greater for the controls than for the patients. The magnitude of the difference for foreperiod uncertainty is greater for patients and controls. For signal quality the trend is unclear. Finally, on total reaction time the results are also mixed. The slopes are parallel for S-R compatibility. For stimulus discriminability, the magnitude of the difference is greater for controls than for patients. For foreperiod uncertainty and signal quality, the magnitude is greater for the patients. These shapes suggest that contrary to predictions, controls perform relatively worse than patients when the conditions are made more complex in some tasks.

For both patients and controls, level 1 task conditions were found to be relatively more difficult than level 2 tasks in terms of reaction time (RT) and total

reaction time (TT). This did not however hold true for the movement time (MT) scores of the task variables *foreperiod uncertainty* (controls), and *signal quality* (controls and patients).

Hypothesis 4

The magnitude of the difference between level 1 and level 2 tasks will be significantly greater for patients than it is for control subjects.

Difference scores between the level 1 tasks and the level 2 tasks were computed by subtracting the latter from the former. *t*-tests for independent measures were then performed on these new scores, comparing patients and controls on the reaction time, movement time, and total reaction time. The results appear in Tables 33, 34 and 35. As the results in Table 33 show, Hypothesis 4 was not confirmed for RT. There were significant differences for the *foreperiod uncertainty* tasks, and *signal quality*, but not for the *S-R compatibility* tasks or the *stimulus discriminability* tasks. Hypothesis 4 was also not confirmed for movement time (Table 34) and total reaction time (Table 35). The result shows that even though controls are faster than patients on all tasks (Tables 22, 23, and 24), the two groups respond similarly to a change in the difficulty levels on the latter two task variables.

Hypothesis 5

The number of correlations between postconcussion symptoms and indices of reaction time varies as a function of the subjective ratings of symptom presence, frequency, intensity, and severity.

Table 33: The magnitude of the difference between level 1 and level 2 tasks on reaction time for patients (N = 54) and controls (N = 52) (df = 104)

Task pairs	Group ^a	Mean	SD	t	p
Compatible/ Incompatible	1	10.70	8.84	1.83	0.70
	2	7.60	8.44		
Dissimilar/ Similar	1	4.14	9.41	0.04	0.68
	2	4.81	7.67		
Fixed/ Variable	1	1.81	5.46	2.13	0.03
	2	-0.33	4.85		
Degraded/ Undegraded	1	11.92	10.66	3.27	0.00
	2	5.77	8.58		

Note

^a1 = patients; 2 = controls.

Table 34: The magnitude of the difference between level 1 and level 2 tasks on movement time: Patients (N = 54) Vs controls (N = 52) (df = 104).

Task pairs	Group ^a	Mean	SD	t	p
Compatible	1	2.39	8.29	-0.87	0.38
Incompatible	2	3.62	6.08		
Dissimilar	1	3.33	8.77	-1.16	0.24
Similar	2	5.13	7.03		
Fixed	1	1.81	5.46	2.13	0.03
Variable	2	-0.33	4.85		
Degraded	1	1.51	9.19	0.22	0.82
Undegraded	2	1.12	8.98		

Note

^a 1 = patients; 2 = controls.

Table 35: The magnitude of the difference between level 1 and level 2 tasks on total reaction time: Patients (N = 54) Vs Controls (N = 52) (df = 104).

Task pairs	Group ^a	Mean	SD	t	p
Compatible	1	12.85	13.40	0.82	0.41
Incompatible	2	10.97	9.88		
Dissimilar	1	7.23	14.01	-1.12	0.26
Similar	2	9.99	11.12		
Fixed	1	7.03	9.55	1.24	0.21
Variable	2	4.95	7.58		
Degraded	1	12.57	12.67	2.19	0.03
Undegraded	2	7.17	12.62		

Note

^a1 = patients; 2 = controls.

Symptom Presence

Symptom presence means that the subject reported experiencing the symptom, irrespective of the symptom's frequency or intensity. One tailed partial correlations controlling for age and education were performed for controls. The same calculations were done for patients with age, education and duration since injury controlled.

Because of the need to control for age, education, and duration since injury, the number of cases used in calculations was reduced. For that reason, it was decided that calculations would not be carried out for cases with frequencies less than 10. For the patients, these were auditory problems, nausea, alcohol sensitivity, and vomiting. For the controls the symptoms were drowsiness, dizziness, and light sensitivity. Correlations between symptom presence and indices of reaction time for controls are shown in Table 36. Only three symptoms in the control group had significant correlations with reaction time. Two of the symptoms, poor memory and irritability had negative correlations. This finding is surprising because it suggests that control subjects with these two symptoms have less reaction time deficits than subjects without deficits. As was shown in Table 17, the error rate for the *similar* task condition was higher relative to the other task conditions. Calculations of the correlation co-efficients with poor memory were thus based on relatively fewer reaction time scores. This may explain the unexpected finding. The likely reason for the negative correlations between the task condition *compatible* and irritability is the small number of control subjects who endorsed this symptom. Overall, because of the small number of symptoms endorsed by the controls, relative to patients, the correlation co-efficients reported need to be interpreted with caution.

The results for the patients are shown in Tables 37 and 38. These results are summarized in Table 50, where they are discussed together with the results from symptom frequency, intensity, and severity.

Symptom Frequency Ratings

One tailed partial correlations between the subjects' frequency ratings for each symptom and the reaction time indices were performed. Age and education were controlled for in the control group. In the patient group, age, education and duration since injury were controlled for. For the patients, the symptom headache was found not to correlate with any of the reaction time indices. The symptoms for which significant correlations were found are listed in Tables 39, 40 and 41. The results from these three tables are included in the summary in Table 50, where they are discussed together with results from symptom presence, intensity, and severity.

For the control subjects, correlations between reaction times and headache, dizziness, memory, irritability, alcohol sensitivity, visual problems, and auditory problems were found to be nonsignificant. The symptoms with which there were significant correlations are shown in Table 42. Five symptoms were found to correlate with reaction time indices for the control group. Four of these symptoms were associated with the task variable *signal quality*, and one was associated with an index from the task variable *S-R compatibility*. All the indices were subtraction scores. This means that if only the conventional indices of RT, MT, and total reaction time had been used, no significant correlations would have been found.

Symptom Intensity

The symptom intensity results for controls are shown in Table 43. The symptoms headache, fatigue, irritability, noise sensitivity, alcohol sensitivity, concentration, and visual problems, were not found to correlate significantly with any of the reaction time indices. As was the case for symptom presence, memory had a negative correlation with the stimulus discriminability tasks. Anxiety, and depression were the only other symptoms that correlated with reaction time indices. All but one of

Table 36: Symptom presence and reaction time indices for controls

Symptom	Task condition	Index	r ^c
Memory	Similar/ Dissimilar	MT/SB	-0.4288**
	Fixed/ Variable	MT/SB	0.3229*
Irritability	Compatible	RT	-0.3184*
Depression	Degraded/ Undegraded	MT/SB	0.3057*

Note

^a MT/SB = movement time, subtraction score; ^b RT = reaction time; ^c * p < 0.01, ** p < 0.001

Table 37: Symptom presence and reaction time indices for patients: dizziness, memory, drowsiness, irritability, and noise sensitivity

Symptom	Task condition	Index ^a	r ^b	
Dizziness	Similar	RT	0.3692*	
	Incompatible	RT	0.3154*	
	Degraded/ Undegraded	MT/SB	0.3040*	
	Similar/ Dissimilar	MT/SB	-0.3192*	
	Degraded/ Undegraded	TT/SB	0.3395*	
	Memory	Similar/ Dissimilar	TT/SB	0.3167*
		Similar	RT	0.3669*
Drowsiness		Dissimilar	MT	0.3470*
	Incompatible	MT	0.3457*	
	Similar/ Dissimilar	RT/SB	0.3390*	
	Irritability	Similar	RT	0.3522*
Noise sensitivity		Degraded/ Undegraded	MT/SB	0.2486*

Note

^a RT = reaction time; MT/SB = movement time (subtraction); TT/SB = total reaction time (subtraction); MT = movement time; RT/SB = reaction time (subtraction).

^b * p < 0.01

Table 38: Symptom presence and reaction time indices for patients: light sensitivity, concentration, anxiety, and depression

Symptom	Task condition	Index ^a	r ^b
Light sensitivity	Compatible	MT	0.3053*
	Dissimilar	MT	0.3688*
	Similar/ Dissimilar	MT/SB	0.3385*
Concentration	Similar	RT	0.3378*
	Similar/ Dissimilar	RT/SB	0.5090**
Anxiety	Similar/ Dissimilar	RT/SB	0.3794*
Depression	Similar	RT	0.3417*
	Undegraded	RT	0.3252*

Note

^a RT = reaction time; MT/SB = movement time (subtraction); TT/SB = total reaction time (subtraction); MT = movement time; RT/SB = reaction time (subtraction).

^b * p < 0.01; ** p < 0.001

Table 39: Symptom frequency ratings and reaction time indices for patients: memory

Symptom	Task condition	Index ^a	r ^b
Memory	Similar	RT	0.3528*
	Compatible	MT	0.3138*
	Degraded	MT	0.3083*
	Fixed	MT	0.3568*
	Similar	MT	0.3364*
	Variable	MT	0.3340*
	Degraded	TT	0.3300*
	Undegraded	TT	0.3258*
	Compatible	TT	0.3156*
	Similar	TT	0.4392**
	Fixed	TT	0.3676*
	Similar/ Dissimilar	RT/SB	0.3680*
	Similar/ Dissimilar	TT/SB	0.3146*

^a RT= reaction time; MT = movement time; TT = total reaction time; RT/SB = reaction time (subtraction score); TT/SB = total reaction time (subtraction score).

^b * p < 0.01; ** p < 0.001

Table 40: Symptom frequency ratings and reaction time indices for patients: fatigue

Symptom	Task condition	Index ^a	r ^b
Fatigue	Compatible	MT	0.3503*
	Degraded	RT	0.3096*
	Degraded	MT	0.3321*
	Fixed	RT	0.3109*
	Fixed	MT	0.3247*
	Similar	MT	0.3575*
	Variable	MT	0.3184*
	Incompatible	MT	0.3327*
	Undegraded	MT	0.4254**
	Degraded	TT	0.3915*
	Undegraded	TT	0.4124*
	Compatible	TT	0.3558*
	Incompatible	TT	0.3024*
	Similar	TT	0.3186*
	Fixed	TT	0.3526*
Variable	TT	0.3240*	

Note

^aRT= reaction time; MT = movement time; TT = total reaction time.

^b * p < 0.01; ** p < 0.001

Table 41: Symptom (other than memory and fatigue) frequency ratings and reaction time indices for patients:

Symptom	Task condition	Index ^a	r ^b
Dizziness	Similar	RT	0.3151*
Drowsiness	Dissimilar	MT	0.3449*
	Similar	RT	0.3644*
	Similar	TT	0.3728*
	Similar/ Dissimilar	RT/SB	0.3863*
Irritability	Similar	RT	0.3354*
Noise sensitive	Degraded/ Undegraded	MT/SB	0.3511*
Light sensitive	Compatible	MT	0.3104*
	Degraded	MT	0.3432*
	Dissimilar	MT	0.3395*
	Similar/ Dissimilar	RT/SB	0.3058*
Concentration	Similar	RT	0.3004*
	Similar/ Dissimilar	RT/SB	0.4583**
Visual	Similar	RT	0.3240*
Anxiety	Undegraded	MT	0.3075*
Depression	Similar	RT	0.3631*
	Similar/ Dissimilar	RT/SB	0.3753*

Note

^a RT = reaction time; MT = movement time; TT = total reaction time, RT/SB = total reaction time (subtraction score); MT/SB = movement time (subtraction score).

^b * p < 0.01; ** p < 0.001

Table 42: Symptom frequency ratings and reaction time indices for controls

Symptom	Task condition	Index ^a	r ^b
Fatigue	Degraded/ Undegraded	TT/SB	0.3333*
Noise sensitive	Compatible/ Incompatible	MT/SB	- 0.3400*
Concentration	Degraded/ Undegraded	MT/SB	0.3360*
Anxiety	Degraded/ Undegraded	TT/SB	0.3119*
Depression	Degraded/ Undegraded	TT/SB	0.3161*

Note

^a TT/SB = total reaction time (subtraction score); MT/SB = movement time (subtraction score).

^b * p < 0.01; ** p < 0.001

the reaction time indices were subtraction scores. The results for patients are shown in Tables 44, and 45, and 46. These results are summarized in Table 50, where they are discussed together with results summarized from symptom presence, frequency, and severity.

Symptom Severity

For every subject, each symptom was coded for severity using the symptom rankings, symptom frequency, and symptom intensity ratings derived from the Postconcussion Symptom Checklist 2 (Appendix F). The symptoms were rated 1 to 4 in terms of severity as follows:

1. Symptoms subjectively ranked as 1 and rated 2 or 3 on both symptom frequency and symptom intensity.
2. Symptoms with a rating of 2 or 3 on both frequency and intensity, irrespective of the subjective ranking.
3. Symptoms with a rating of 2 or 3 on either frequency or intensity irrespective of subjective ranking.
4. Symptoms with a subjective ranking greater than 1, frequency less than 2, and intensity less than 2.

To test the hypothesis, 1-tailed partial correlations between tasks and symptoms were performed. For the patient group, age, education, and duration since injury were held constant, and for the controls, age and education were held constant.

In the control group, the symptoms headache, fatigue, noise sensitivity, alcohol sensitivity, concentration, and auditory problems were not found to correlate with any of the reaction time indices. The symptoms that correlated with reaction time indices are shown in Table 47. Some symptoms had negative correlations, whilst others had positive correlations. Only negative correlations were anticipated because the measures of severity decrease from 1 (highest) to 4 (lowest). As was argued for

symptom presence (Table 38), the positive correlations are probably due to the small number of control subjects who endorsed these symptoms. These results must therefore be interpreted with caution.

For the patients, no significant correlations were found for headache, and auditory problems. The symptoms that correlated with reaction time indices are indicated in Tables 48 and 49. These results are summarized in Table 50, together with correlations for symptom presence, frequency, and intensity.

Summary of Correlations Between Symptom Presence, Frequency, and Intensity, with Indices of Reaction Time.

The number of times that correlations were found between reaction time indices and symptom presence, symptom frequency, symptom intensity, and symptom severity were counted. These are shown in Table 50. Hypothesis 5 was confirmed by these results, which show that the number of correlations with reaction time indices varies as a function of the subjective ratings of symptom presence, frequency, intensity, and severity. When one looks at the totals, it is clear that symptom intensity ratings had the highest number of correlations with reaction time indices. Symptom frequency ratings had the second largest number of correlations. Symptom presence and symptom severity had an almost equal number of correlations. The totals for the symptoms indicate that symptoms varied widely in the number of correlations they had with reaction time indices. Inspection of this table suggests that the symptoms can be classified into two groups, those with multiple correlations (15-26 correlations) and those with few correlations (3-9 correlations). The two groups are shown in Table 51.

Table 43: Symptom intensity ratings and reaction time indices for controls

Symptom	Task condition	Index ^a	r ^b
Memory	Similar/ Dissimilar	MT/SB	-0.3944*
	Degraded/ Undegraded	TT/SB	0.3054*
Depression	Degraded/ Undegraded	MT/SB	0.4030*
	Degraded/ Undegraded	MT	0.3503*
	Degraded/ Undegraded	MT/SB	0.4030*
	Degraded/ Undegraded	TT/SB	0.3054*

Note

^a MT/SB = movement time (subtraction score); TT/SB = total reaction time (subtraction score); MT = movement time.

^b * p < 0.01

Table 44: Symptom intensity ratings and reaction time indices for patients: headache, dizziness, and memory

Symptom	Task condition	Index ^a	r ^b
Headache	Degraded/ Undegraded	MT/SB	0.3004*
	Compatible/ Incompatible	RT/SB	0.3049*
	Compatible/ Incompatible	TT/SB	0.3622*
Dizziness	Dissimilar	RT	0.3223*
	Similar	RT	0.3349*
	Dissimilar	TT	0.3220*
	Similar	TT	0.4322**
	Fixed	TT	0.4165*
	Variable	TT	0.2992*
	Similar/ Dissimilar	MT/SB	-0.3400*
Dissimilar	MT	0.3223*	
Memory	Compatible	MT	0.3211*
	Dissimilar	MT	0.3615*
	Fixed	RT	0.3551*
	Fixed	MT	0.3735*
	Similar	RT	0.3856*
	Similar	MT	0.3067*
	Variable	MT	0.3420*
	Incompatible	MT	0.3264*
	Degraded	TT	0.3836*
	Undegraded	TT	0.3329*

Note

^a MT/SB = movement time (subtraction score); RT/SB = RT(subtraction score)

TT/SB = total reaction time (subtraction score); RT = reaction time; MT = movement time.

^b * p < 0.01; ** p < 0.001

Table 45: Symptom intensity ratings and reaction time indices for patients: drowsiness, irritability, noise and light sensitivity, concentration, visual problems, and depression.

Symptom	Task condition	Index ^a	r ^b
Drowsiness	Dissimilar	MT	0.3257*
	Fixed	MT	0.3065*
	Similar	RT	0.2603*
	Incompatible	MT	0.3331*
	Similar	TT	0.2994*
	Fixed	TT	0.3238*
	Similar/ Dissimilar	RT/SB	0.3286*
Irritability	Similar	RT	0.3140*
	Undegraded	RT	0.3086*
	Similar/ Dissimilar	RT/SB	0.3163*
Concentration	Similar	RT	0.3707*
	Similar	TT	0.3319*
	Similar	RT	0.5399**
Visual	Similar	RT	0.3094*
Depression	Similar	RT	0.3140*
	Undegraded	RT	0.3086*
	Similar/ Dissimilar	RT/SB	0.3163*

Note

^a MT = movement time; RT = reaction time; RT/SB = reaction time (subtraction score).

^b * p < 0.01; ** p < 0.001

Table 46: Symptom intensity ratings and reaction time indices for patients: noise and light sensitivity

Symptom	Task condition	Index ^a	r ^b
Noise sensitivity	Degraded/ Undegraded	MT/SB	0.3064*
Light sensitivity	Compatible	MT	0.3130*
	Degraded	MT	0.3737*
	Dissimilar	MT	0.3863*
	Fixed	MT	0.3215*
	Variable	MT	0.3096*
	Incompatible	MT	0.3158*
	Undegraded	MT	0.3619*
	Undegraded	TT	0.3535*
	Dissimilar	TT	0.3108*
	Similar	TT	0.3205*
	Fixed	TT	0.3273*
Variable	TT	0.3179*	

Note

^a MT/SB = movement time (subtraction score); MT = total reaction time; TT = total reaction time.

^b * p < 0.01

Table 47: Symptom severity ratings and reaction time indices for controls

Symptom	Task condition	Index ^a	r ^b
Memory	Similar/ Dissimilar	RT/SB	0.3705*
	Compatible Compatible	RT TT	0.3184* 0.3016*
Visual	Similar	RT	-0.2961*
	Compatible/ Incompatible	MT/SB	-0.3096*
	Similar/ Dissimilar	RT/SB	0.2549*
	Degraded	TT/SB	0.3535*
Depression	Degraded	MT	-0.3397*
	Degraded/ Undegraded	MT/SB	-0.4410**
	Degraded/ Undegraded	TT/SB	-0.3603*

Note

* RT/SB = reaction time (subtraction score; RT = reaction time; TT = total reaction time; MT/SB = movement time (subtraction score); MT = movement time; TT/SB = total reaction time (subtraction score).

^b * p < 0.01; ** p < 0.001

Table 48: Symptom severity ratings and reaction time indices for patients: Dizziness, memory, fatigue, drowsiness, irritability, noise sensitivity.

Symptom	Task condition	Index ^a	r ^b
Dizziness	Similar/ Dissimilar	RT	-0.3267*
	Similar/ Dissimilar	RT/SB	-0.3044*
	Similar/ Dissimilar	MT/SB	-0.3256*
Memory	Similar/ Dissimilar	RT/SB	-0.3147*
Fatigue	Undegraded	MT	-0.3032*
	Undegraded	TT	-0.3219*
Drowsiness	Incompatible	MT	-0.3012*
Irritability	Similar	RT	-0.3400*
Noise sensitivity	Degraded/ Undegraded	MT/SB	-0.3219*

Note

^a RT = reaction time; RT/SB = reaction time (subtraction score); MT/SB = movement time (subtraction score); MT = movement time; TT = total reaction time.

^b * p < 0.01; ** p < 0.001

Table 49: Symptom severity ratings and reaction time indices for patients: light, concentration, visual, anxiety, depression

Symptom	Task condition	Index ^a	r ^b
Light sensitivity	Compatible	MT	-0.3341*
	Degraded	MT	-0.3508*
	Dissimilar	MT	-0.3496*
	Incompatible	MT	-0.3148*
	Undegraded	MT	-0.3725*
Concentration	Similar	RT	-0.3076*
	Similar/ Dissimilar	RT/SB	-0.4903**
Visual	Similar	RT	-0.3168*
Anxiety	Similar	RT	-0.3185*
	Similar/ Dissimilar	RT/SB	-0.4435**
Depression	Similar/ Dissimilar	RT/SB	-0.3719*
	Similar/ Dissimilar	TT/SB	-0.3575*

Note

^a MT = movement time; RT = reaction time; RT/SB = reaction time (subtraction score); TT/SB = total reaction time (subtraction).

^b * p < 0.01; ** p < 0.001

Table 50: Summary of associations between symptom presence, frequency, intensity and severity, with indices of reaction time.

Symptoms	Aspects of Symptoms				
	PRE ^a	FRQ ^b	INT ^c	SEV ^d	TOTAL
Headache	0	0	3	0	3
Dizziness	5	1	8	3	17
Memory	3	13	10	1	26
Drowsiness	3	4	7	1	15
Irritability	1	1	3	1	6
Noise	1	1	1	1	4
Light	3	4	12	5	24
Concentration	2	2	3	2	9
Anxiety	1	1	0	2	4
Depression	2	2	3	2	9
Fatigue	0	16	0	2	18
Visual	0	1	1	1	3
TOTAL	20	46	51	21	138

Note

^a symptom presence; ^b symptom frequency; ^c symptom intensity

^d symptom severity

Table 51: Classification of postconcussion symptoms based on the number of correlations with indices of reaction time

Group	Correlations	Symptoms
Group 1	Multiple correlations	Memory, light sensitivity, fatigue, drowsiness, and dizziness.
Group 2	Few correlations	Headache, irritability, low noise tolerance, poor concentration, anxiety, depression, visual problems.

The Group 1 symptoms consist of one cognitive symptom (memory), one stimulus intolerance symptom (light sensitivity), two arousal-related symptoms (fatigue and drowsiness), and a somatic symptom (dizziness). Group 2 symptoms include a somatic symptom (headache), emotional symptoms (irritability, anxiety, and depression), a cognitive symptom (poor concentration), stimulus intolerance (low noise tolerance), and a sensory symptom (visual problems).

Both groups have cognitive, somatic, and stimulus intolerance symptoms. The main difference is that additionally, Group 1 has the arousal-related symptoms of fatigue and drowsiness. Because of potential problem of inaccuracies in reporting symptoms that was discussed earlier, it is possible that for some patients, these two symptoms are one and the same. However, even if that is the case, the important issue is that the symptom relates to arousal. Arousal deficits reflect lesions of the first functional unit of the brain, which is responsible for the regulation of cortical tone and waking (Luria, 1973). Deficits of the first functional unit affect the brain diffusely. For this reason, it is not surprising that Group 1 symptoms are associated with more cognitive deficits as indexed by reaction time, than the Group 2 symptoms.

Hypothesis 6

The various postconcussion symptoms will differ in the extent to which they correlate with reaction time, movement time, total reaction time, and subtraction scores.

Hypothesis 6 was confirmed by the results. The number of times each symptom correlated with reaction time (RT), movement time (MT), total reaction time (TT), or subtraction scores (SB, the respective differences between the RTs, MTs, and TTs of level 1 and level 2 task conditions) were counted. These counts are listed in Table 52.

The results in Table 52 were analysed qualitatively, with the aim of identifying patterns of relationships between symptoms and reaction time indices. The results of the qualitative analyses are shown in Table 53. In this table, the reaction time index with the highest number of correlations for each symptom determines the pattern to which each symptom belongs.

The Pattern 1 symptoms would have shown no correlations if only the conventional reaction time indices (RT and MT) had been used. For this reason, subtraction scores can be considered as being more sensitive than the conventional indices in detecting correlations between reaction time indices and the symptoms headache and low noise tolerance.

As was indicated in the literature review, the RT scores are associated with the *decision component* of reaction time, and MT scores are associated with the *response execution* component of reaction time (Jensen & Munro, 1979). These results thus suggest that the symptoms of Pattern 2 are more closely associated with decision making, and those of Pattern 3 are more closely associated with response execution. In Pattern 2, the symptom *poor concentration* is clearly cognitive, and it is thus not surprising that it was most frequently associated with RT.

Table 52: Summary of correlations between symptoms and indices of reaction time

SYMPTOM	RT ^a	MT ^b	TT ^c	SB ^d	TOTAL
Dizziness	6	1	4	(3.9)6	17
Memory	4	11	7	(2.6)4	26
Drowsiness	2	7	3	(1.9)3	15
Irritability	5	0	0	(0.6)1	6
Noise	0	0	0	(2.6)4	4
Light	0	17	5	(1.3)2	24
Concentration	5	0	1	(1.9)3	9
Anxiety	1	1	0	(1.3)2	4
Depression	5	0	0	(2.6)4	9
Visual	3	0	0	(0.0)0	3
Headache	0	0	0	(1.9)3	3
Fatigue	2	8	8	(0.0)0	18
TOTAL	33	45	28	32	138

Note

^a reaction time; ^b movement time; ^c total reaction time

^d subtraction score. The score in brackets is the calculated score. The maximum possible score for SB is 12, whilst the maximum possible score for RT, MT, and TT is 8 scores each. To make the SB score comparable to the other scores, the calculated score was obtained by the following formula: Original SB score x 8/12.

Table 53: Patterns of correlations between symptoms and indices of reaction time.

Pattern	Reaction Time Index	Symptoms
Pattern 1	Subtraction scores only	headache, low noise tolerance.
Pattern 2	Most frequent scores RT	Dizziness, irritability, poor concentration, visual problems, depression.
Pattern 3	Most frequent scores MT	Memory, fatigue, drowsiness, low light tolerance.

The Pattern 3 symptoms correlated most frequently with the response execution component of reaction time (MT). The poor memory symptom, is clearly cognitive and seems more appropriately to belong to Pattern 1. Fatigue and drowsiness are arousal symptoms. These two symptoms are suggestive of damage to the first functional unit of the brain (Luria, 1973). Because damage to the first functional unit affects the brain diffusely, one would have expected these symptoms to be associated with both *decision time* and *response execution*. There is no easily apparent reason why low light tolerance should belong with Pattern 3.

The qualitative data in Tables 52 and 53 needs to be confirmed by quantitative analyses. This is done in Hypothesis 8 where symptoms are grouped into clusters and are examined at different durations since injury.

Hypothesis 7

The various postconcussion symptoms will differ in the extent to which they correlate with the task variables signal quality, signal discriminability, S-R compatibility, and foreperiod uncertainty

Hypothesis 7 was confirmed by the results. The scores for the task variables are the subtraction (SB) scores. As was explained in Chapter 5, these scores are obtained by subtracting the scores of the level 2 task conditions from the scores of the level 1 task conditions for each task variable. The number of times that each symptom correlated with each of the four task variables was counted. These counts are shown in Table 54. From the totals indicated in Table 54, it is clear that the task variable *signal discriminability* had the largest number of correlations with symptoms. *Signal quality* and *S-R compatibility* had smaller numbers, whilst foreperiod uncertainty had no correlations with symptoms. The symptoms *fatigue* and *visual problems* did not have correlations with any of the task variables. It is noteworthy that fatigue and drowsiness differ with respect to correlations with task variables. Earlier (Hypothesis 5, Table 51), it was speculated that for some patients, these two

symptoms might be identical in meaning. The present result suggests that the two symptoms are distinct.

The above qualitative analyses of the relationships between symptoms and task variables will be confirmed by quantitative analyses for symptom clusters in Hypothesis 8.

Hypothesis 8

The various symptom clusters will differ in the extent to which they correlate with reaction time, movement time, total reaction time, and subtraction scores.

A rating scale of 0 to 6 for symptom severity was used was done in hypothesis 2. Principal components analysis with varimax rotation was used to identify clusters of postconcussion symptoms and reaction time indices (Appendixes H, I, J, and K). The data from these factor matrices are summarized in Tables 55, 56, 57, and 58.

Column 1 of each table contains the Factor number. This is followed by column 2 which has the percentage of variance explained by each factor. In Column 3 are given the reaction time indices that loaded on the respective factor. The maximum possible number of indices in the various categories of reaction time are as follows: RT (8), MT (8), total reaction time (TT) (8), subtraction scores (SB) (12). There are four task variables, viz., *signal quality*, *signal discriminability*, *S-R compatibility*, and *foreperiod uncertainty*. The scores for the task variables are represented by SB. This is because SB is the difference between the level 1 and level 2 scores in each task variable (see Table 1). SB scores are derived from RT, MT, and TT scores. The number of SB scores relating to each task variable will be shown in brackets. In factorial experiments, such as those reviewed in Chapter 4 (Table 1), deficits on task variables correspond to deficits on specific stages of

Table 54: Summary of correlations between symptoms and task variables

<u>Symptom</u>	<u>Task Variables</u>				<u>Total</u>
	<u>Quality^a</u>	<u>Discr^b</u>	<u>Com^c</u>	<u>Forep^d</u>	
Headache	1	0	2	0	3
Dizziness	2	4	0	0	6
Memory	0	4	0	0	4
Fatigue	0	0	0	0	0
Drowsiness	0	3	0	0	3
Irritability	0	1	0	0	1
Noise sensitive	4	0	0	0	4
Light sensitive	0	2	0	0	2
Concentration	0	3	0	0	3
Visual	0	0	0	0	0
Anxiety	0	2	0	0	2
Depression	0	4	0	0	4
TOTAL	7	23	2	0	32

Note

^a signal quality; ^b signal discriminability

^c S-R compatibility; ^d foreperiod uncertainty.

information processing. The results for the present study are based on a nonfactorial design (Chapter 5, Table 2). The deficits on specific task variables can therefore only suggest, rather than confirm, deficits on the corresponding stages of information processing.

The fourth column gives the components of reaction time. Two components are given, *decision time*, and *response execution time* (Jensen & Munro, 1979). According to Jensen and Munro, in reaction time experiments, the RT scores reflect decision time whilst the MT scores represent response execution. The decision as to which component of reaction time is associated with each factor depends on the reaction time indices reported in Column 2. Where both RT and MT scores are represented in Column 2, the component is described as *mixed*. In the fifth column are the symptoms that loaded on each of the factors.

Controls

Hypothesis 8 was confirmed for the controls (Table 55). The symptoms loaded on two of the four factors on Table 55. Factor 2 consisted of the symptom memory and all of the RT scores. This means that memory loaded with decision time, the cognitive component of reaction time. Further to reflecting the cognitive component of reaction time, Factor 2 also reflected slowing of information processing on the task variable *foreperiod uncertainty*. In factorial studies, slowed responses on the foreperiod uncertainty task variable suggests deficits on the *motor adjustment* stage of information processing.

The remaining symptoms loaded on Factor 4, which reflected both decision time and response execution. One would have expected poor concentration, which is a cognitive symptom, to load on Factor 2 together with memory. This unexpected finding may be related to the problem of the subjects' understanding of constructs such as concentration, which was discussed earlier. Finally, for Factor 4, the conventional reaction time indices (RT and MT) did not load with the symptoms. The

Table 55. Reaction time indices and symptom clusters in controls (N = 52)

Factor	% of Variance	Indices	component	Symptoms
1	31	8 MT scores 5 TT scores 2 SB scores (MT) - Signal discriminability (1) - S-R compatibility (1)	Response execution	No symptoms
2	13.4	8 RT scores 2 TT scores 1 SB (TT) - Foreperiod uncertainty (1)	Decision time	Memory
3	8.4	6 SB scores (RT & MT) - Signal discriminability (2) - S-R compatibility (2) - Foreperiod uncertainty (2)	Mixed	No symptoms
4	7.5	1 TT 3 SB scores (RT & MT) - Signal quality (3)	Mixed	Anxiety Dizziness Noise sensitivity Irritability Concentration Light sensitivity Headache Fatigue

presence of both RT and MT is detected only because of the SB scores, which loaded on this factor. These SB scores were derived from the task variable *signal quality*. The findings of the present study therefore suggest that for controls, with the exception of memory, the symptoms are associated with the *feature extraction* stage of information processing.

Patients

Factor analyses at 1-21 days since injury. Hypothesis 8 was not confirmed for this duration since injury because all the symptoms loaded on one factor (Factor 3, Table 56). All the symptoms loaded on Factor 3, which reflects decision time, the cognitive component of reaction time. This suggests that in the acute/subacute stage after injury, cognitive problems are more prominent than response execution. It is striking that the only reaction time indices that loaded with the symptoms were SB scores. If only the conventional reaction time indices of RT and MT had been used, the cognitive deficits would not have been detected. The task variables that the SB scores represent are *signal quality* and *S-R compatibility*, which, in factorial studies, would suggest deficits on the *feature extraction* and *response selection* stages of information processing respectively.

Factor analysis at 22 - 90 days since injury. Hypothesis 8 was confirmed for the duration 22 - 90 days after injury (Table 57). Symptoms loaded on three of the four factors. The majority of the symptoms loaded on Factor 2, which reflects the cognitive component of reaction time. Two of the symptoms, memory and concentration, are cognitive, and it is thus logical that they should load with the cognitive component of reaction time. These cognitive symptoms loaded with *somatic* (headache and dizziness), and *arousal* (drowsiness and fatigue) symptoms. As was pointed out earlier with respect to Table 27, the symptoms poor concentration and irritability are frequently the result of fatigue (Mittenberg, Zielinski, & Fichera, 1993), which also loads on Factor 2.

Table 56. Reaction time indices and symptom clusters in patients (N = 15): 1-21 days after injury.

Factor	% of variance	Indices	component	Symptoms
1	24.3	8 MT scores 6 TT scores	Response execution	No symptoms
2	20.1	8 RT scores 1 TT score 3 SB scores (MT) - Signal quality (2) - Signal discriminability (1)	Mixed	No symptoms
3	15.4	3 SB scores (RT) - S-R compatibility (2) - Signal quality (1)	Decision time	Noise Concentration Dizziness Memory Irritability Anxiety (-) headache Depression Light Visual problems Drowsiness Fatigue
4	9.5	1 TT score 6 SB scores (RT & MT) - Signal discriminability (2) - Foreperiod uncertainty (3) - S-R compatibility (1)	Mixed	No symptoms

Table 57. Reaction time indices and symptom clusters in patients (N = 16): 22-90 days after injury.

Factor	% of variance	Indices	component	Symptoms
1	48.3	8 TT scores 8 MT scores 8 RT scores	Mixed	No symptoms
2	11.4	2 SB scores (RT & TT) - Signal discriminability (1) - Foreperiod uncertainty (1)	Decision time	Concentration Irritability Headache Dizziness Memory Drowsiness Fatigue
3	8.6	3 SB scores (RT & MT) - S-R compatibility (1) - Signal quality (2)	Mixed	Light sensitivity Anxiety (-) Depression Visual
4	8	6 SB scores (MT & TT) - S-R compatibility (2) - Signal discriminability (2) - Foreperiod uncertainty (2)	Response execution	Noise sensitivity

The symptoms on Factor 2 are similar to the symptoms on Factor 1 (Table 26) and are identical to the symptoms found on Factor 1 (Table 27). The cognitive symptoms identified by factor analyses on symptoms alone (Tables 26 and 27) correlated with the cognitive component of reaction time (Table 57). This is an important finding, in that it suggests that the patients understood the constructs on the symptom checklist (Appendix F), and reported them accurately. The task variables with which these symptoms loaded are *signal discriminability* and *foreperiod uncertainty*. In factorial studies these would correspond to the *identification* and *motor adjustment* stages of information processing respectively.

The symptoms on Factor 3, which are similar to those of Factor 2 in Table 27, loaded with both RT and MT. In Table 27 these symptoms were labelled as *visual/emotional*. Only the SB scores loaded on this factor, and they were derived from the task variables *S-R compatibility* and *signal quality*. It is significant that the visual symptoms (light sensitivity and visual problems) loaded here, together with *signal quality*, a task variable that involves visual abilities (degraded vs undegraded task conditions). This finding suggests that the constructs visual problems and light sensitivity (Appendix F) were accurately reported by patients. The symptom anxiety loaded negatively on this factor, and it may be that the meaning of this symptom was not properly understood by patients.

Finally, Factor 4 had only one symptom, noise sensitivity, which was associated with response execution. The conventional reaction time indices (RT and MT) were absent. Noise sensitivity was associated with three of the four task variables, ie, *Signal discriminability*, *S-R compatibility*, and *foreperiod uncertainty*. Noise sensitivity is an auditory symptom, and it is significant that the only task variable with which it did not correlate was signal quality, which involves visual abilities. If this symptom was reported accurately, the present findings suggest that noise sensitivity is associated with a wider range of deficits (3 out of 4 task variables) than other symptoms.

Factor analysis at 91+ days after injury. Hypothesis 8 was confirmed for this duration. Symptoms loaded on all four factors (Table 58). Factor 1 has one symptom,

anxiety, which, on previous occasions was found to load negatively with other symptoms (Tables 26, 27, and 28) and with reaction time indices (Table 57). It also is notable that anxiety is the only symptom which loaded with practically all of the conventional reaction time indices (8 TT, 8 MT, and 7 RT). Anxiety is thus associated with both the decision time and the response execution components of reaction time. If anxiety was reported accurately, the present results suggest that this symptom becomes more significant in the chronic stages after traumatic brain injury. A similar finding for the symptom depression is reported below.

Factor 2 consisted of fatigue and two somatic symptoms. This factor is identical to Factor 1 on Table 28, which was labelled as *arousal/somatic*. In the literature review (Chapter 4), it was suggested that fatigue may be associated with injuries to the first functional unit of the brain (Luria, 1973). As Luria suggested, deficits of the first functional unit affect functions in widespread areas of the brain. For that reason, it makes sense that both the decision time and response execution components of reaction time also load on this factor. However, there were more RT scores than MT scores. As Appendix K shows, of the six SB scores that loaded on this factor, only one was an MT score. The symptoms on Factor 2 loaded with three of the four task variables, viz, *S-R compatibility*, *Signal quality*, and *signal discriminability*.

There is no easily apparent reason for the loading of visual problems with irritability on Factor 3. One possibility is that irritability is an emotional reaction to visual difficulties. The two symptoms are associated with both the decision time and the response execution components of reaction time. Because of the visual problems, one would have expected the task variable *signal quality* to load here too, but this was not the case. However, the *signal discriminability* task variable which also loaded on Factor 3, is visually demanding.

The six symptoms of Factor 4 loaded with only one reaction time index, *degraded (MT, SB)*. If this finding is not an artefact of small samples, it suggests that

Table 58. Reaction time indices and symptom clusters in patients (N = 19): 91+ days after injury.

Factor	% of variance	Indices	Component	Symptoms
1	43.4	8 TT scores 8 MT scores 7 RT scores	Mixed	Anxiety
2	11.1	1 RT score 6 SB scores (RT & MT) - S-R compatibility (3) - Signal quality (2) - Signal discriminability (1)	Mixed	Fatigue Dizziness Headache
3	8.8	5 SB scores - Foreperiod uncertainty (3) - Signal discriminability (2)	Mixed	Visual problems Irritability
4	7	1 SB score (MT) - Signal quality	Response execution	Concentration Drowsiness Memory Noise Depression Light

in the chronic stages, several of the symptoms become less associated with organic deficits, as indexed by reaction time. Furthermore because of the symptoms poor concentration and memory, one would have expected these symptoms to load with the cognitive component of reaction time, and not with response execution, as was the case here.

The possibility that the aetiology of symptoms is different at different durations since injury has been mentioned with respect to anxiety above. This possibility has also been investigated in the case of depression. Jorge, Arndt, and Forrester (1993) found that whereas acute onset depression is related to brain injuries, late onset depression is mediated by psychosocial factors. These findings are consistent with previous studies, which report that there are generally low correlations between organic indices of brain injury and postconcussion symptoms after three months (Lishman, 1988).

For all the three durations since injury, symptom clusters differed on the number of task variables with which they correlated. In terms of the stage theories of information processing (Sternberg, 1969; Shum, McFarland, Bain, & Humphreys, 1990), this suggests that some symptom clusters are associated with more stages of information processing than others. None of the clusters in Tables 56 -58 was associated with all four task variables. The maximum number of associations was three.

There is a striking difference between the associations of individual symptoms and task variables, and that between symptom clusters and task variables. The results for individual symptoms, which were reported in Table 54 suggested that most symptoms correlated with *stimulus discriminability*. The task variable *foreperiod uncertainty* was not associated with any symptom. Contrary to the findings for individual symptoms, all four task variables were associated with at least one symptom cluster (Tables 56-58).

Another interesting finding is that many symptom clusters correlated with clusters of subtraction scores more than they did with the conventional indices of reaction time (RT, MT, & TT). This finding attests to the usefulness of using a wide

variety of reaction time indices (Houlihan, Campbell, & Stelmach, 1994), and the need to include amongst these, the subtraction scores.

CHAPTER NINE

CONCLUSIONS AND RECOMMENDATIONS

Main Findings

Correlations Between Symptoms and Reaction Time Indices

The main finding of this study is as follows: There is a significant positive correlation between the kinds of postconcussion symptoms patients report, and the kinds of cognitive deficits that they have, as indexed by reaction time tests. The two areas in which symptoms differ are on the components of reaction time (Table 52 & 53), and on task variables (Table 54).

With respect to components of reaction time, some symptoms are more closely associated with the *decision time* component of reaction time, while others are more closely associated with the *response execution* component. This result confirms the findings from factor analytic studies (Bohnen, Twijnstra, & Jolles, 1992) that subjectively reported postconcussion symptoms load onto *cognitive* and *noncognitive* factors. The importance of the present investigation is that symptom factor loadings derived from subjective reports were confirmed by experimental assessments of cognitive deficits.

The task variables used in the present study were *signal quality*, *S-R compatibility*, *stimulus discriminability*, and *foreperiod uncertainty*. As is clear from Table 54, most symptoms were associated with *stimulus discriminability*. The symptom *headache* is the only one which correlated with *S-R compatibility*. In factorial experiments (Shum McFarland, Bain, & Humphreys, 1990), these task variables have been used to assess deficits on stages of information processing. The stages of information processing are *feature extraction*, *identification*, *response selection* and *motor adjustment* (Chapter 5, Table 2).

Because the present study was nonfactorial in design, the results obtained can only suggest, rather than confirm, deficits on specific stages of information processing. In this light, the present study suggests that most symptoms were associated with the *identification* stage of information processing. The results in Table 54 suggest that *feature extraction* was associated with only three symptoms, headache, dizziness, and noise sensitivity. Headache is the only symptom associated with *response execution*. No symptoms correlated with foreperiod uncertainty. In the study by Shum, McFarland, Bain, & Humphreys, (1990), deficits after brain injury were found in the *identification* and *response selection* stages of information processing.

Factors Moderating the Correlations Between Symptoms and Indices of Reaction Time

The correlations between symptoms and indices of reaction time are moderated by the following factors: characteristics of symptoms (Hypothesis 5, Tables 36 - 50), clusters of symptoms (Hypothesis 8, Tables 55 - 58), and duration since injury (Hypothesis 8, Tables 56 - 58).

The characteristics of symptoms that were investigated were the subjective ratings of symptom presence, symptom frequency, symptom intensity, and symptom severity. It was found that symptom intensity had the largest number of correlations with symptoms. This was followed by symptom frequency, symptom severity, and symptom presence respectively (Table 50). This finding confirms the suggestion by Gouvier, Uddo-Crane, & Brown (1988), that assessing different aspects of postconcussion symptoms (frequency, intensity, and duration) gives a more concise measure of those symptoms than noting the mere presence of a symptom.

The associations between clusters of symptoms and reaction time indices were assessed at different durations since injury. The component of reaction time with which a symptom correlated differed, depending on whether the symptom was considered on its own, or as part of a symptom cluster. For example considered singly, poor concentration and depression correlated with decision

time, because the most frequent score was RT (Pattern 2, Table 52). However, in clusters, these symptoms correlated with response execution three months or more after injury (Factor 4, Table 58). These findings suggest that interactions between symptoms changes the manner in which they correlate with reaction time indices. Symptom clusters vary with duration since injury (Table 26, 27, and 28). In some cases, the pattern of these clusters changes when indices of reaction time are added into the factor analyses (Table 26 vs Table 56; Table 28 vs Table 58).

Other Findings

Patients vs Controls

The focus of this study was not on the differences between patients and controls, as was pointed out in Chapter 1. However, the following significant findings are restated because they help to place the findings on patients into proper perspective, and are of interest in the diagnosis of brain injury.

1. *Patients reported higher frequencies and intensities of on most of the postconcussion symptoms than controls (Table 20 & 21).*

The symptoms for which there were no significant differences were: poor concentration and anxiety (frequency); and poor concentration, anxiety, and depression (intensity).

2. *On all reaction time tasks, patients were slower than controls. (Table 22, 23, & 24).*

Previous studies (Jakobsen, Baadsgaard, Thomsen, & Henriksen, 1987; Shum, McFarland & Bain, 1994b) have reported similar findings.

3. *Although patients and controls may report similar symptoms, the clusters formed by the symptoms in the two groups are different (Tables 26-28 vs Table 29).*

The patients group had a larger number of factors than the control group. Because of the imprecise way in which the symptoms are defined, it is probable that the symptoms described by patients and controls are only similar in name, and are not identical. It was pointed out in Chapter 3 that many of the names of many of the symptoms refer to a variety of conditions. For example, the symptom *dizziness* may refer to vertigo, near syncope, and ill-defined lightheadedness (Anderson, Yolton, Reinke, Kohl, & Lundy-Ekman, 1995).

4. *Overall, the magnitude of the difference between levels of task conditions is not significantly greater for patients than it is for controls.*

This finding is contrary to what had been predicted in Hypothesis 4. It suggests that even though controls are faster than patients on all tasks, the two groups are affected similarly by a change in the difficulty levels of task conditions.

Subtraction Scores

Subtraction (SB) scores are the product of the differences between Level 1 and Level 2 tasks in each task condition. They are a measure of the increase in complexity between the two levels. These scores are derived from the conventional indices of reaction time, namely reaction time (RT), movement time (MT), and total reaction time (TT). Each SB score thus always necessarily reflects one of these conventional indices.

In this study, many symptoms and symptom clusters showed correlations with SB scores and not with any of the conventional indices (Table 53, 55, 56, 57, & 58). The SB scores are thus more sensitive in detecting correlations between

symptoms and reaction time than the conventional indices from which they are derived. Many significant correlations would have been missed if only the conventional indices had been used.

Methodological Issues

Overall, the aims of the study, to show that cognitive deficits can be predicted from reported symptoms was achieved. However there are several weaknesses in the study, which could limit the generalizability of these findings. These are classified into problems relating to the sample (subjects), the instruments, and procedures.

Subjects

The interpretation of these results is bound to be affected by the fact that the patients were consecutive attenders at outpatient clinics, rather than a random sample of head injury patients. The control subjects were volunteers, and were thus also not a random sample. Another difficulty with these samples is that they were not well-matched demographically. The control group had more subjects with higher education and professional occupations (Table 12). However, the crucial comparisons in this study were not between patients and controls. The focus was on the differences between patients who report different symptoms.

A nonfactorial design was used with the view to increasing sample size. A total of 144 subjects were assessed initially, and it appeared that the sample was adequately large. The sample was however eventually reduced to 106 (54 patients and 52 controls) for reasons shown in Table 8. These two samples were adequate for comparing the reaction time performance of patients and controls. The samples were however small, when subjects were classified according to symptoms reported. For some symptoms, there was such a low count that they were excluded from several analyses. As a consequence of this, the results were

available for only 12 of the 16 original symptoms. The number of subjects endorsing the remaining symptoms was acceptable, but small, especially when subjects had to be matched for age, education, and duration since injury. It is important that future studies use much larger samples than were used here. The present study has highlighted the need for large initial samples, because many subjects eventually have to be dropped, for reasons such as failure to follow instructions.

Instruments

Symptom Checklists.

The hypotheses in this study were confirmed by significant but low correlations between symptoms and reaction time indices. It is possible that the low correlations were the result of the heterogeneity of patients classified under each symptom. The Postconcussion Symptom Checklist 2 (Appendix F) asked the subject to endorse whether or not he or she experienced a particular symptom. These symptoms however, may represent more than one condition. For example, Evans (1992) indicated that several types of headache are possible after traumatic brain injury. The same applies to memory (Cohen, 1984; Ewert, Levin, Watson, & Kalisky, 1989), dizziness (Anderson, Yolton, Reinke, Kohl, & Lundy-Ekman (1995), and possibly the other symptoms too. The patients classified under each symptom may therefore have been more heterogeneous than was suspected. In future, it is important to establish not only that the subject has a headache, but also to establish what kind of headache. Some symptoms, notably fatigue and drowsiness, may be confused for each other. Future studies need to define symptoms operationally, so that they can be understood in the same way by all subjects.

Reaction Time Tasks

The error rate for the task condition *similar* was considerably higher than that for all the other task conditions (Table 17). This may suggest that this task condition was relatively the most difficult. The higher the number of errors on a task condition, the less the number of trials used in the calculations of correlations. It is noteworthy that this task condition was most frequently associated with postconcussion symptoms (Table 54). To avoid this problem in future studies, preliminary statistical and psychometric studies ought to be carried out to ensure that the task variables are equal in level of difficulty. For example, how far apart should the lines be in the similar condition as opposed to the dissimilar? Similarly, in the task variable signal quality, the task condition *degraded*, involved the degrading of the image on the computer screen by 7%. This is an arbitrary level of degradation, which was used here in order to be consistent with previous studies (eg Shum, McFarland, Bain, & Humphreys, 1990). In addition to equating levels of difficulty, appropriate numbers of practice trials should be determined from trials on large numbers of subjects. In the present study, an arbitrary cut-off of 9 trials was used.

A final procedural issue is that more would have been achieved in this investigation if it had been a multidisciplinary and prospective study. As a multidisciplinary study, the clinical and diagnostic information would have been more readily available, and it would have been more accurate. It is desirable that neurosurgeons be formally involved in studies of this kind. One of their roles would be to use acceptable research and clinical instruments for assigning diagnoses to patients. The diagnoses that were available from Ga-Rankuwa hospital had been assigned mostly by registrars as part of their routine ward work. There was no way of checking on the accuracy of diagnoses. It is for this reason that no hypotheses relating to diagnosis and severity of injury were formulated.

In a prospective study, more reliable information about indices of severity of injury would be obtained. The researcher would be in a position to get information from patients and/or their relations about the time of the accident, the behaviours and deficits indicative of post-traumatic amnesia, and the evolution of the

postconcussion symptoms. With respect to post-traumatic amnesia, scales such as the Galveston Orientation and Amnesia Test (Levin, O'Donnell, & Grossman, 1979), and Westmead Post-traumatic Amnesia Scale (Shores, Marosszeky, Sandanam, & Batchelor, 1986) and the Glasgow Coma Scale-Extended (GCS-E) (Nell, 1997) would be used prospectively.

Implications of the Study

Notwithstanding the methodological constraints just cited, this study makes important theoretical and practical contributions to our understanding of the postconcussion syndrome. With regard to theory, the study confirmed the existence of three classes of symptoms in the postconcussion syndrome based on cognitive deficits. These are the symptoms associated with decision time, response execution, and those associated with both decision time and response execution. The study also suggests that symptoms, and clusters of symptoms can be classified in terms of deficits on specific stages of information processing. As far as I can tell, no previous study has investigated such associations. The fact that certain symptoms and indices of reaction time load together is important theoretically because it suggests clues about the aetiology of the postconcussion syndrome. Hypotheses for future studies can be formulated for each of the symptom/reaction time index clusters (Table 56-58), in the context of what is known about the mechanisms of traumatic brain injury (Chapter 2), the aetiologies of individual postconcussion symptoms (Chapter 3), and the physiology of attention and information processing (Chapter 4).

The most significant practical implication of this study is that it suggests a method for predicting cognitive deficits on the basis of symptoms reported by patients. Such predictions are useful because they will provide a framework for further assessments, and for the planning of treatment and rehabilitation. In assessing the postconcussion syndrome, this study has confirmed the importance of considering the duration since injury (Rutherford, 1989). The importance of

assessing the frequency and intensity of postconcussion symptoms, instead of just reporting on their presence (Gouvier, Uddo-Crane, & Brown, 1988) was also confirmed. Finally, this study highlighted the importance of subtraction scores in detecting cognitive deficits associated with symptoms.

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APPENDIX

APPENDIX ADemographic Data Form

- 1.
2. Surname and Initial:.....
3. Institution:.....
4. Hospital Number:.....
5. Research Number:.....
6. Group: Patient/ Normal Control P / C.
7. Sex: M / F.
8. Date of Birth: / /
9. Age:.....
10. Date of Admission: / /
11. Date of Discharge: / /
12. Marital status: Married / Single / Divorced / widowed
13. Highest education standard attained:.....Year 19...
14. Occupation/ profession:.....
15. Current employment:.....

Date: / /

Hand Dominance

Show me how:

1. You would hammer in a nail
2. You would throw a stone
3. You would write your name
4. Dominance: Right... Left... Mixed...

Left Right

--- ---
 --- ---
 --- ---

APPENDIX BTrauma Details Form

Date: / /

1. Research Number.....
2. Date of trauma: / /
3. Time of trauma:.....
4. Source of Trauma:..(Assault/ Mva/ Fall/ Falling object
other.....
5. MVA: Driver / Pedestrian / Passenger / NA
6. Assault: Fight / Unknown assailants / NA / Other.....
7. Assault Instrument.....
.....
8. Loss of consciousness:..T / F.
9. Duration loss of consciousness:.....
10. Glasgow coma score on admission: /15
11. PTA symptoms: (History of deficits in short-term memory, inattentiveness,
disinhibited, restlessness other.....
12. Duration of PTA.....
13. Duration of hospitalization:.....
14. Duration since trauma:.....
15. Diagnosis.....

APPENDIX C

Screening Questionnaire

Research Number.....

Date: / /

- 1. Have you ever been dazed or unconscious as a result of
 - A car accident in which you were the occupant _____
 - A car accident in which you were a pedestrian struck by a vehicle. _____
 - A blow to the head by a blunt object (stick, brick, iron bar, back of axe) _____
 - A penetrating wound on the head (bullet knife driven into head, sharp axe wound) _____
 - Accident while playing sport or falling _____
 - Nearly drowning in water _____
 - Suffocating in gas as a result of a gas stove or a fire inside the room. _____
 - Suffocating from exhaust fumes from a car or tractor. _____
 - Losing consciousness from poison or poison gases. _____
 - Losing consciousness while at work on the farm or for any reason. _____
 - What happened?.....
- 2. For how long were you dazed or unconscious?.....
- 3. Did you go to hospital as a result of this injury? _____
- If yes
- 4. How long were you in hospital?.....(Days/ weeks).
- 5. Have you ever had a diagnosis of any of the following conditions?

Epilepsy (falling sickness, seizures, convulsions)

Explain:
.....

—

A psychiatric diagnosis

Explain:.....
.....

—

Diabetes

—

Any disease affecting your nervous system

Explain:.....

—

Injury to you arm or hand (specify R, or L)
by breaking or crushing

Give details:.....
.....

6. Have you ever been admitted to hospital at any time
in the past?

—

(Examiner notes hospital stays of any duration
for any of the conditions in 5).

Year:.....

Hospital:

Conditions for which admitted:.....

.....

Source: Nell 1992.

APPENDIX DForm 5: Pretest Questionnaire

Date: / /

Research Number.....:.....

1. How much sleep did you get last night:
 About the same as usual
 Less than usual
 More than usual

2. Right now I am feeling:
 Energetic
 Fresh
 Average
 Tired
 Exhausted

3. In the last 24 hours, my consumption of coffee, tea or Coca-Cola has been:
 I do not drink coffee, tea or Coca-Cola
 About the usual amount
 Less than usual
 More than usual

4. In the last 24 hours, my smoking has been:
 I do not smoke
 About the usual amount
 More than usual

5. In the last 24 hours, my alcohol intake has been:
 I do not drink
 About the usual amount
 Less than usual
 More than usual

6. Medications:.....Source: NES-2 & WHO-NCTB

APPENDIX EPostconcussion Symptom Checklist 1

Research Number:.....

Date: / /

		<u>Ratings</u>						
<u>Symptom</u>		<u>Frequency</u>			<u>Intensity</u>			
1	Headache	0	1	2	0	1	2	3
2.	Dizziness	0	1	2	0	1	2	3
3	Memory	0	1	2	0	1	2	3
4.	Fatigue	0	1	2	0	1	2	3
5.	Drowsiness	0	1	2	0	1	2	3
6.	Irritability	0	1	2	0	1	2	3
7.	Noise	0	1	2	0	1	2	3
8.	Light	0	1	2	0	1	2	3
9.	Alcohol	0	1	2	0	1	2	3
10.	Concentration	0	1	2	0	1	2	3
11.	Visual	0	1	2	0	1	2	3
12.	Hearing problems	0	1	2	0	1	2	3
13.	Nausea	0	1	2	0	1	2	3
14.	Vomiting	0	1	2	0	1	2	3
15.	Anxiety	0	1	2	0	1	2	3
16.	Depression	0	1	2	0	1	2	3

APPENDIX F

Postconcussion Symptom Checklist 2

Research Number.....

Date / / .

Symptom	Rank	Ratings												
		Frequency			Duration				Intensity					
Headache	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Dizziness	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Memory	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Fatigue	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Drowsiness	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Irritability	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Noise sensitive	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Light sensitive	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Alcohol	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Concentration	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Visual	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Auditory	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Nausea	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Vomiting	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Anxiety	-	0	1	2	3	0	1	2	3	4	0	1	2	3
Depression	-	0	1	2	3	0	1	2	3	4	0	1	2	3

KEY

- i) frequency:
0 = Never; 1 = sometimes; 2 = most of the time; 3 = all the time.
- ii) Duration: 0 = not at all; 1 = A few secs; 2 = A few mins; 3 = A few hours;
4 = constant.
- iii) Intensity:
0 = None; 1 = mild; 2 = moderate; 3 = severe.

APPENDIX G

Symptom Ranking Matrix

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	X															
2		X														
3			X													
4				X												
5					X											
6						X										
7							X									
8								X								
9									X							
10										X						
11											X					
12												X				
13													X			
14														X		
15															X	
16																X

Key:

- | | |
|----------------------|------------------------|
| 1. Headache | 9. Alcohol sensitivity |
| 2. Dizziness | 10. Poor concentration |
| 3. Memory | 11. Visual problems |
| 4. Fatigue | 12. Auditory problems |
| 5. Drowsiness | 13. Nausea |
| 6. Irritability | 14. Vomiting |
| 7. Noise sensitivity | 15. Anxiety |
| 8. Light sensitivity | 16. Depression |

APPENDIX H

Factor analysis of reaction time indices and symptoms for controls (N = 52)

	Factor 1	Factor 2	Factor 3	Factor 4
Compatible (MT)	,94	-,01	,07	,04
Fixed foreperiod (MT)	,94	,03	,05	-,05
Similar (MT)	,94	,01	,17	,09
Incompatible (MT)	,92	,14	,02	,02
Variable foreperiod (MT)	,90	,05	-,11	,02
Dissimilar (MT)	,86	,22	-,11	-,07
Undegraded (MT)	,86	,03	-,02	,13
Degraded (MT)	,71	-,13	,18	-,54
Similar (TT)	,71	,52	,40	,08
Incompatible (TT)	,70	,62	-,18	,06
Fixed foreperiod (TT)	,69	,59	-,07	-,02
Variable foreperiod (TT)	,65	,62	-,09	,03
Undegraded (TT)	,62	,61	,06	,05
Similar (MT, SB)	,54	-,24	,43	,25
Compatible (MT, SB)	,37	,26	-,06	-,02
Compatible (RT)	-,05	,91	,24	,03
Undegraded (RT)	-,01	,88	,10	-,07
Variable foreperiod (RT)	,19	,87	-,05	,05
Dissimilar (RT)	,07	,87	-,24	,07
Fixed foreperiod (RT)	,18	,84	-,15	,02
Similar (RT)	,05	,83	,45	,02
Incompatible (RT)	,18	,83	-,30	,07
Degraded (RT)	,05	,80	-,19	-,35
Dissimilar (TT)	,59	,69	-,22	,00
Compatible (TT)	,64	,65	,22	,05
Memory	,17	-,19	-,01	,02
Fixed (TT, SB)	-,01	,12	-,05	,09
Similar (TT, SB)	,33	-,07	,86	,11
Similar (RT, SB)	-,01	,13	,85	-,06
Compatible (RT, SB)	,29	,23	-,63	,07
Compatible (TT, SB)	,47	,32	-,57	,04
Fixed foreperiod (MT, SB)	-,04	,03	-,29	,13
Fixed foreperiod (RT, SB)	,04	,15	,19	,07
Degraded (TT, SB)	-,00	,12	-,12	-,87
Degraded (MT, SB)	-,13	-,18	,22	-,74
Depression	-,09	-,09	-,18	,62
Degraded (TT)	,45	,52	-,05	-,58
Anxiety	-,17	,02	,11	,52
Degraded (RT, SB)	,09	,33	-,39	-,47
Dizziness	,05	,08	-,17	,41
Noise	,02	,28	,14	,35
Irritability	,14	,15	,18	,32
Concentration	,04	-,03	-,07	,28
Light	,12	,08	,15	,28
Headache	-,01	-,06	,21	,22
Fatigue	-,13	,07	-,03	-,15

APPENDIX I

Factor analyses of reaction time indices and symptoms 1 - 21 days after injury.

	Factor 1	Factor 2	Factor 3	Factor 4
Fixed foreperiod (MT)	,94	-,10	,04	,16
Undegraded (MT)	,94	-,24	-,09	-,03
Degraded (MT)	,93	,08	-,04	-,09
Dissimilar (MT)	,92	,04	-,04	-,15
Compatible (MT)	,90	-,23	-,19	,16
Undegraded (TT)	,88	,38	,10	,02
Incompatible (MT)	,85	-,32	,00	-,19
Similar (MT)	,83	-,31	-,20	,15
Fixed foreperiod (TT)	,82	,38	,13	,32
Variable foreperiod (MT)	,82	-,03	,18	-,06
Variable foreperiod (TT)	,76	,54	-,05	-,23
Compatible (TT)	,72	,37	-,20	,20
Dissimilar (TT)	,66	,59	-,04	-,28
Degraded (TT)	,64	,62	-,17	-,10
Degraded (RT)	-,06	,89	-,13	-,04
Undegraded (RT)	,05	,88	,27	,10
Variable foreperiod (RT)	,22	,85	-,04	-,17
Dissimilar (RT)	-,01	,83	-,06	-,34
Compatible (RT)	-,06	,82	-,11	,08
Incompatible (RT)	-,24	,80	,39	,01
Similar (RT)	-,14	,75	,16	,50
Fixed (RT)	,14	,73	,17	,31
Degraded (MT, SB)	,09	,65	,11	-,13
Incompatible (TT)	,47	,55	,42	-,12
Degraded (TT, SB)	-,16	,54	-,47	-,22
Similar (MT, SB)	,25	,45	-,23	,34
Noise	,13	,12	,86	,23
Concentration	,09	,20	,86	,17
Dizziness	-,13	,01	,75	-,05
Memory	,02	-,02	,74	,52
Irritability	-,02	,15	,65	,02
Compatible (RT, SB)	-,28	,39	,64	-,06
Anxiety	,09	,06	-,63	,14
Compatible (TT, SB)	-,19	,21	,60	-,30
Degraded (RT, SB)	-,17	,34	-,60	-,20
Headache	-,08	,22	,57	,00
Depression	,12	,54	,56	,10
Light	,37	-,27	,50	,23
Visual	-,13	-,04	,49	-,15
Drowsiness	,06	-,26	,43	,17
Fatigue	,03	-,28	,38	-,21
Similar (RT, SB)	-,15	,20	,23	,84
Similar (TT, SB)	-,03	-,19	,00	,82
Fixed foreperiod (TT, SB)	-,01	,27	-,26	-,79
Similar (TT)	,53	,29	-,03	,67
Compatible (MT, SB)	,03	-,19	,32	-,61
Fixed foreperiod (SB)	,16	,42	-,22	-,54
Fixed foreperiod (MT, SB)	-,08	,12	,32	-,44

APPENDIX J

Factor analyses of reaction time indices and symptoms: 22 - 90 days after injury.

	Factor 1	Factor 2	Factor 3	Factor 4
Variable foreperiod (TT)	,97	,02	,11	,04
Fixed foreperiod (TT)	,97	,16	,08	-,13
Fixed foreperiod (MT)	,95	,15	-,01	,01
Compatible (TT)	,95	,25	,05	,14
Incompatible (TT)	,94	,16	-,03	-,21
Degraded (TT)	,94	,16	,11	,15
Variable foreperiod (MT)	,93	,17	-,06	,14
Undegraded (TT)	,93	,14	-,03	,10
Dissimilar (TT)	,91	,30	,08	-,12
Similar (TT)	,91	,28	,18	,20
Similar (MT)	,89	,10	,07	,35
Incompatible (MT)	,89	,24	,13	-,16
Variable foreperiod (RT)	,88	-,14	,27	-,18
Fixed (RT)	,87	,20	,23	-,26
Degraded (MT)	,86	,18	,16	,18
Compatible (RT)	,85	,05	,17	-,20
Dissimilar (MT)	,85	,33	,09	-,04
Incompatible (RT)	,85	,05	-,30	-,25
Undegraded (TT, MT)	,83	,30	-,16	,24
Degraded (RT)	,81	,10	,05	,09
Compatible (MT)	,80	,34	-,06	,36
Undegraded (RT)	,80	-,20	,30	-,19
Similar (RT)	,78	,49	,31	-,05
Dissimilar (RT)	,72	,12	,12	-,23
Degraded (RT, SB)	,59	,31	-,17	,29
Concentration	,38	,86	,10	,01
Irritability	,30	,84	-,03	-,03
Headache	,11	,83	,05	-,03
Dizziness	,15	,78	-,03	,19
Memory	,38	,75	-,06	,09
Drowsiness	,33	,69	,58	-,02
Fatigue	,52	,66	,02	,27
Similar (RT, SB)	,38	,66	,35	,20
Fixed foreperiod (TT, SB)	,37	-,61	,18	,05
Light	,32	,04	,88	,04
Anxiety	-,39	,26	-,70	-,38
Depression	-,00	,15	,69	-,07
Compatible (RT, SB)	,10	,01	-,66	-,09
Visual	,17	,57	,59	-,18
Degraded (MT, SB)	-,31	-,28	,47	-,18
Degraded (TT, SB)	,27	,08	,31	,15
Compatible (MT, SB)	,32	-,10	,31	-,81
Compatible (TT, SB)	,32	-,13	-,18	-,78
Similar (TT, SB)	,15	,02	,23	,70
Similar (MT, SB)	-,05	-,41	-,04	,61
Fixed foreperiod (MT, SB)	,33	,15	-,20	,57
Fixed foreperiod (TT, SB)	,38	-,40	,11	,51
Noise	-,09	,15	,06	,34

APPENDIX K

Factor analyses of reaction time indices and symptoms 91 days+ after injury.

	Factor 1	Factor 2	Factor 3	Factor 4
Dissimilar (TT)	,98	,09	-,04	-,03
Variable foreperiod (TT)	,96	,16	-,08	,05
Compatible (TT)	,94	,22	-,02	,00
Undegraded (TT)	,94	,11	,07	-,08
Similar (MT)	,93	,13	,06	,14
Fixed foreperiod (TT)	,93	,16	,26	,02
Variable foreperiod (MT)	,92	,00	-,24	,07
Dissimilar (MT)	,90	,08	-,23	,13
Compatible (MT)	,89	,06	-,25	,05
Undegraded (MT)	,89	,04	-,12	-,04
Fixed foreperiod (MT)	,88	,07	,03	,18
Degraded (TT)	,87	,42	-,12	,11
Degraded (MT)	,87	,15	-,07	,25
Similar (TT)	,84	,29	,34	,19
Dissimilar (RT)	,83	,09	,21	-,23
Variable foreperiod (RT)	,81	,37	,13	-,01
Fixed foreperiod (RT)	,81	,22	,46	-,14
Incompatible (TT)	,78	,58	,08	-,05
Incompatible (MT)	,78	,40	-,09	,05
Undegraded (RT)	,77	,17	,28	-,11
Degraded (RT)	,68	,58	-,14	-,05
Compatible (RT)	,62	,35	,35	-,07
Similar (RT)	,56	,47	,53	,18
Anxiety	,37	,15	,13	-,30
Compatible (TT, SB)	,27	,84	,18	-,09
Compatible (RT, SB)	,31	,75	,18	-,25
Degraded (RT, SB)	,34	,72	-,46	,03
Fatigue	,22	,70	-,13	,11
Degraded (TT, SB)	,37	,68	-,35	,33
Compatible (MT, SB)	,21	,68	,19	,02
Incompatible (RT)	,57	,64	,33	-,19
Dizziness	,40	,56	,11	,37
Headache	-,09	,49	-,04	,24
Similar (RT, SB)	-,18	,49	,43	,47
Fixed foreperiod (TT, SB)	,10	,01	-,84	,07
Similar (TT, SB)	-,07	,39	,70	,41
Similar (MT, SB)	,05	,10	,68	,02
Fixed foreperiod (RT, SB)	-,20	,18	-,65	,24
Fixed foreperiod (MT, SB)	,34	-,15	-,64	-,19
Visual	,13	-,19	,39	,32
Irritability	,15	-,17	,32	,23
Concentration	-,29	-,08	-,09	,80
Drowsiness	-,06	,12	,02	,72
Memory	,25	,14	-,15	,70
Degraded (MT, SB)	,23	,25	,06	,58
Noise	,14	-,10	,06	,53
Depression	-,01	,07	,16	,40
Light	-,21	-,20	,13	,23