Electrophysiological Investigation of Attentional Deficits in Traumatic Brain Injury

by

Louise C. Penkman
B.A. (Hons.), University of Ottawa, 1993

A Thesis Submitted in Partial Fulfillment
of the Requirements for the Degree of

MASTER OF SCIENCE

in the Department of Psychology

We accept this thesis as conforming
to the required standard

[redacted]

Dr. C. Mateer, Supervisor (Department of Psychology)

[redacted]

Dr. M. Corcoran, Departmental Member (Department of Psychology)

[redacted]

Dr. I. Dawson, Outside Member (Department of Nursing)

[redacted]

Dr. M. Languis, External Examiner (Emeritus Professor, Graduate Neuroscience Program
Ohio State University)

University of Victoria

Supervisor: Dr. Catherine Mateer

ABSTRACT

Visual event-related potentials (ERPs) were recorded in two target detection reaction time tasks in order to assess the role of automatic processes in the attentional deficits exhibited by individuals with traumatic brain injury (TBI). A second objective was to examine the issue of distractibility in this population. A group of 10 TBI subjects were compared to a normal control (NC) group. Subjects were also administered a brief neuropsychological test battery. The TBI group was found to be significantly slower than the NC group in both experimental task conditions. Latency and amplitude of the P3b and the P3a components showed no group or condition effects. The data showed a trend for NC subjects to process automatic and controlled tasks differentially. This trend was not evident for the TBI subjects. The results suggest that further research with larger sample sizes is needed.

Examiners:

[redacted]

Dr. C. Mateer, Supervisor (Department of Psychology)

[redacted]

Dr. M. Corcoran, Departmental Member (Department of Psychology)

[redacted]

Dr. I. Dawson, Outside Member (Department of Nursing)

[redacted]

Dr. M. Languis, External Examiner (Emeritus Professor, Graduate Neuroscience Program Ohio State University)

TABLE OF CONTENTS

List of Tables

List of Figures

Acknowledgments

I would like to express my thanks to my supervisor, Dr. Catherine Mateer and to my committee members. I would also like to thank Dr. Debra Allison and the Neuropsychology staff at the Gorge Road Hospital for their assistance with subject recruitment. I would like to extend a special thank you to Dr. Kimberly Kerns and Dr. Marlin Languis who both provided invaluable statistical and technical help and encouragement.

INTRODUCTION

The major cause of death in the young adult is traumatic brain injury (Campbell, Deacon-Eliot & Proulx, 1986). These injuries are most commonly the result of motor vehicle accidents (MVA), physical aggression, industrial, or sporting and leisure accidents. Due to greater safety standards in automobiles, and advances in emergency medical care and neurosurgical techniques, more and more of the victims of accidents resulting in traumatic brain injury (TBI) are surviving. In the United States between 200 and 300 people per 100 000 of the population are admitted to hospital each year for head injuries (Cohen, 1993). Injury can occur due to blunt impact, rotation of the head and brain, or to acceleration-deceleration forces which result in the brain being thrown back and forth against the interior surface of the skull. The latter is often the case in MVAs, in which the victim may not actually hit any external object with his or her head.

The neuropathology of non-penetrating traumatic brain injury can involve both focal lesions such as cortical contusions, and diffuse axonal injuries (DAI). Contusions are the result of the brain impacting on the rigid internal surface of the skull. The most frequent sites of contusions following TBI are the orbital regions of the frontal lobes and the poles of the temporal lobes (Auerbach, 1988). In fact, magnetic resonance imaging (MRI) has confirmed that the frontal region is the most common location of focal lesions after mild to moderate TBI (Levin, Amparo, & Eisenberg, 1987). DAI is a more widespread injury involving axonal shearing due to stretching of the axons when the brain is impacted. This may result in tearing and breaking of axons and ultimately in neuronal degeneration. Often, victims will lose consciousness which, if they survive, is followed by a period of post-

traumatic amnesia (PTA). PTA involves an inability to retain information about ongoing events and a wide constellation of behavioral disturbance (Levin, 1989). Following resolution of the acute stages of the injury, patients with TBI frequently experience a wide range of cognitive deficits. There is an extensive literature regarding these deficits, which include impairments in attention and concentration, problems with learning and memory, difficulties in planning, inability to make judgements, problems initiating activities, and decreased speed of information processing (Levin, 1989; Van Zomeren, Brouwer & Deelman, 1984; Ponsford & Kinsella, 1992; Bennett, 1988). In everyday life these deficits result in problems with activities of daily independent living, difficulties in maintaining employment, problems in managing finances, and difficulty in interpersonal relationships.

Relationship of Memory and Attention

Various experimental and theoretical paradigms and a wide range of clinical measures have been employed to investigate the nature of the cognitive deficits in patients with TBI. This research is essential, as knowledge of the specific cognitive deficits informs the planning and implementation of rehabilitation strategies designed to improve the quality of life for survivors of these injuries. Studies have examined speed of processing (Ponsford & Kinsella, 1992, Van Zomeren et al., 1984), memory deficits (Levin, 1989), and attentional dysfunction (Ponsford & Kinsella, 1992; Sohlberg & Mateer, 1987). Memory deficits are frequently documented in this population in both clinical and experimental reports (Levin, 1989; Auerbach, 1986). As a greater understanding of human memory is being achieved, it has become evident that memory is strongly related to attention. Perhaps

the simplest way to conceptualize this relationship is to say that information that is not attended to is poorly remembered (Nissen, 1986). Classic studies by Craik and Lockheart (1972) demonstrated that manipulating the level of processing that a stimulus receives affects the strength of the memory trace, as measured by the rate of forgetting. A study by Russell and D'Hollosy (1992) investigated the effects of attention on memory. Their results led them to conclude that both short and long term memory are directly dependent upon attention with regard to both what is remembered and the strength of the memory store. They went on to say that the strength of the memory store is directly equivalent to the amount of attention paid to the material that enters memory. Intuitively, this is not surprising. Nearly everyone can recall an experience when one was not able to remember information due to a low level of attention, most likely related to boredom or distraction. With regard to the TBI population, it appears that memory is likely to be secondarily impaired due to a disorder of attention (Van Zomeren et al., 1984). The exception to this statement may be those patients who sustain direct damage to the hippocampus, as this structure has been implicated in the consolidation of memory (Bauer, Tobias & Valenstein, 1993).

Models of Attention

As the focus of the discussion turns to attention and its relation to the cognitive deficits experienced by individuals with TBI, it is necessary to define what the term "attention" has come to represent in the field of neuropsychology. It is not a term that is easily conceptualized as its boundaries overlap with other constructs such as memory and

arousal. In addition, it is not a construct that can be discreetly localized within the brain, as for example, language has been localized to the left temporal area. Attention is considered by most to be a multidimensional cognitive capacity which directly impacts new learning, memory, communication, problem solving, perception, and all other domains of cognition (Sohlberg & Mateer, 1987). It is not a unitary construct and it is helpful to briefly review how researchers have partitioned and conceptualized attention. Empirical, factor analytic models of attention have been advanced to support the current componential understanding of attention within the field of neuropsychology (see Mateer & Mapou, 1996 for a review).

At its most basic level, or in Luria's terminology, the first functional unit (Luria, 1973), attention is manifested in consciousness, the level of arousal of the brain. The tectal and mesopontine regions of the recticular formation of the brainstem have been well established as essential to the maintenance of consciousness and the regulation of arousal. These structures comprise the basic, fundamental, and phylogenentically most primitive component of the attention system in the brain (Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991). This level of attention is severely compromised when a person sustains a TBI and loses consciousness. Some researchers maintain that the deficits seen in TBI are due to generalized lowered arousal and not impairment of a specific, discrete information processing component (Gronwall & Sampson, 1974).

Closely related anatomically to the maintenance of arousal is the concept of sustained attention. Midline thalamic structures appear to play a role in sustained attention, through the modulation of reticular activity (Yingling & Skinner, 1975). Sustained attention refers to the ability to maintain a consistent behavioral response for a period of time during

continuous and repetitive activity (Kerns & Mateer, 1996). The Continuous Performance Test (CPT) (Rosvold et al., 1956) is used to assess sustained attention, also referred to as vigilance.

The next aspect of attention to be discussed deals with perceptual selection. This is best demonstrated by the example of the 'cocktail party phenomenon'. Selective attention is analogous to attending to only one conversation amongst a background noise of many other conversations, such as occurs at a cocktail party or in many environmental settings. Therefore, selective attention refers to the selection of a particular stimulus as the focus of attention when distracting or competing stimuli are present. The earliest debates surrounding attention involved this component. D.E. Broadbent (1958) was the first to suggest the concept of an attentional filter necessary for a limited capacity information processing system to control which environmental stimuli were to be processed. Treisman (1964) advanced the concept of attenuation. She found that salient stimuli such as the subject's name (Moray, 1959) were detected when presented to the unattended channel. This led her to suggest that unattended channels were being monitored at some attenuated level. Deficits in selective attention present as excess distractibility.

Closely related to selective attention and used interchangeably by some authors (Weber, 1990), is the concept of focused attention. This term refers to the ability to focus one's attentional resources on a single stimuli in the context of others. In Sohlberg and Mateer's (1987) componential theory of attention, focused attention is defined as the ability to respond to specific sensory information. Like selective attention, focused attention tasks involve elements of capacity and control. Control is necessary to direct attention away from

one task and toward the task of interest. The task of interest can only be accomplished if it does not exceed the capacity of the attentional system.

Many behaviors carried out on a daily basis involve performing two or more tasks at once. To do this, divided attention is required. Divided attention can be thought of as being somewhat diametric to selective attention in that it involves the division of attentional resources between two or more tasks simultaneously, as opposed to the focusing of attentional resources on one task to the exclusion of all other available stimuli or responses. Divided attention is utilized by all individuals on a daily basis to carry out daily activities such as talking on the phone and making a cup of coffee at the same time. Divided attention becomes difficult as the limited capacity of the attentional system of the individual is reached. As multiple tasks become more demanding performance of these tasks decreases. It appears that the more automatic or overlearned the tasks, the greater the number of tasks that can be performed at once.

Sohlberg and Mateer (1987) introduce the concept of alternating attention. They define it as the capacity for mental flexibility which allows individuals to shift their focus of attention. It involves switching between tasks with different cognitive demands requiring different behavioral responses. In addition to divided attention, alternating attention is part of our daily repertoire of behavioral strategies used to cope with the numerous demands of daily life. Alternating attention involves control as the individual is required to assess the stimuli presented and to decide which should be attended to and when to shift to another stimulus.

Automatic and Controlled Processing

One of the most productive paradigms employed in the study of attention, particularly with relation to the study of attention deficits in TBI, involves the concept of automatic and controlled or effortful processing. An important ability of the human information processing system is its ability to learn. Certain behaviors when emitted under certain predictable conditions can become automatic. That is, conscious attention is no longer necessary for them to be carried out. This leaves our limited capacity attentional processor free to carry out other important tasks which are complex and which require controlled processing. A frequently cited example of an automatic process is the act of driving a car. When an individual is comfortable driving a car and when extenuating circumstances such as bad weather or heavy traffic do not interfere with the process, other behaviors can be carried out at the same time such as having a conversation on a cellular phone, reading street signs or even eating lunch. In 1977 Schneider and Shiffrin published two landmark studies which established the existence of these two distinct processing systems, automatic and controlled. They define controlled processing as being serial in nature, highly demanding of attentional capacity, and being under the conscious control of the individual. An example of controlled processing would be rehearsal of items which one wishes to store in long term memory. Automatic processing is thought to be parallel in nature; it is not capacity limited and it usually occurs without the conscious awareness of the individual. It is difficult to repress, ignore or modify. Automatic processing is established when an invariant relationship exists between a stimulus and a response. Therefore a task which is at first carried out in a controlled manner can, under these

conditions, become automatic. The hallmarks of automaticity are said to be an absence of interference between the automatic process and other concurrent activities, together with an apparently unstoppable tendency for the automatic stimulus to evoke its response (Baddeley, 1990). An example of this phenomenon is the Stroop effect in which subjects are required to name the colour in which words are printed. Colour naming is slowed when the word indicates a colour that is different from that in which it is printed, for example, saying "green" in response to the word "blue" written in green letters.

The automatization of certain behaviors is necessary for multiple tasks to be carried out successfully simultaneously. The automatic and controlled processing paradigm is closely related to the issue of attentional control, as certain attentional tasks requiring focused, alternating, or divided attention require that the allocation of the attentional resources be directed or controlled. Control is also required to efficiently allocate the attentional resources so that overlearned tasks are carried out automatically. In addition, during controlled processing automatic monitoring of unattended stimuli needs to be occurring, in the event that a salient stimulus should arise. It is important to note that automatic and controlled processes probably exist on a continuum. Certain tasks are probably more automatized than others and certain behaviors require controlled processing initially and, with practise, can become automatic.

Models of Attentional Control

Two interesting models of the executive control of attentional resources have been advanced (Norman & Shallice, 1986; Reason, 1984). These models are particularly

interesting as they both propose the existence of an executive attentional system which is responsible for the allocation of attentional resources in an automatic or controlled manner. The Norman and Shallice model has been termed the Supervisory Attentional System or SAS model. They looked at everyday cognitive errors committed by normal individuals as well as the breakdown of behavior in clinical samples, particularly frontal lobe patients. An example of a cognitive error is going into a familiar room to pick up an item and then leaving the room with a different item unintentionally. Inherent to their model is the assumption that actions can be performed in two ways: as well learned skills or via the supervisory attentional system. The concept of well learned skills is akin to automatic processing in Schneider and Shiffrin's terminology. These well learned skills are carried out relatively automatically and more than one behavior can occur simultaneously. When ongoing activities come into conflict one behavior must gain priority over the other. They suggest a mechanism termed contention scheduling to solve this problem. It is a relatively automatic process consisting of simple rules as to relative importance which are built into the system to establish which behavior should gain precedence.

The supervisory attentional system can be likened to the operation of will. It is capable of interrupting and modifying ongoing behavior by systematically biasing existing probabilities so as to make one line of action more likely to occur than another line of action. Shallice suggests that deficits in this supervisory attentional system will result in perserverative errors. In such circumstances, a strategy once adopted continues to run uninterrupted as the capacity to interrupt or change ongoing activity has been lost.

Additionally, behavior may be passively captured by whatever stimulus the environment presents resulting in behavior that is highly distractible and stimulus bound.

Reason (1984) approached the study of attentional control from a similar vein, also utilizing observations of the slips of action and cognitive errors of normal individuals. His model is an attempt to explain absent mindedness and other cognitive failures in every day life. Attention is an integral part of his model. It is composed of three levels. The first is termed schemata which consist of relatively permanent knowledge structures which provide detailed guidance for largely automatic sequences of speech, thought, action or perception. These are overlearned behavioral programs, again akin to the concept of automatic processing first advanced by Schneider and Shiffrin (1977). According to Reason, schemata can be activated by two factors. The first he terms domain specific, such as action and word schemata which receive their primary activation from intentions to say or to do certain things. The second factor comprises universal activating factors which exert their influence across all cognitive domains independently of the intention system. They involve the environmental context, the current need state or prevailing emotion, and influences from other schemata, particularly those groups of schemata which share common operations such as different cooking behaviors which all involve the boiling of water. Universal factors can also be influenced by the recency of schemata operation.

The intention system is the second level in Reason's model. It consists of a central processor and storage units. It assembles plans for future actions, monitors and guides ongoing actions, copes with changes in circumstances and detects and recovers from errors. The storage unit serves as a time extension to the central processor. It can be likened to

working memory in that it functions serially, has limited contents, is short lived, and is prone to interference.

The attentional control system is the third and highest level of Reason's model. Its inherent features are that attention is selective and limited; it is mobile and connected with conscious awareness; it is under intermittent voluntary control and is important in learning new behaviors. As the conscious components of a behavior decrease, it is likely that the behavior will become a relatively invariant sequence and therefore become schemata. In essence, as behaviors become overlearned, and when the need for controlled processing decreases, then automaticity develops.

Reason has developed an interesting model for the interaction of these three levels. He envisions a cognitive board which is a matrix of separate squares each representing individual schema. At any point in time the schemata have different activation levels. They are all affected by domain specific and universal activating factors and are potentially linked to one another by a complex nexus running throughout the board. If this board was to operate without the third level, the attentional control component, control of action would be dominated by the schemata with the highest activation levels. As activation is dominated by frequency and recency factors, these schemata would dominate the system resulting in repetitive behaviors. As mentioned, universal factors also play a role in activating schemata but are not sufficient to elicit higher order cognitive functions such as problem solving and forming new schemata.

Attentional Control of Automatic Processing in the TBI population

Researchers have explored the nature of the cognitive deficits manifested by patients who have sustained a TBI within each of the attentional paradigms previously mentioned: sustained attention, selective attention (Gronwell, 1987), speed of processing and arousal (Van Zomeren & Van Den Berg, 1985). The automatic and controlled processing paradigm has also been utilized as a framework within which to examine these deficits (Levin, 1989; Rugg, Cowan, Nagy, Milner, Jacobson, & Brooks, 1993; Heinze, Münte, Gobiet, Niemann, & Ruff, 1992). As illustrated by the models of Reason (1984) and Norman and Shallice (1986), there is an intimate relationship between the concept of automatic and controlled processing and the notion of attentional control. Attentional control appears to be a vital component necessary for the allocation of these different types of attentional resources. Hence the concept of attentional control provides an interesting framework within which to look at the cognitive deficits of individuals who have sustained a TBI. Weber (1990) defines control as an individual's ability to guide a selective process by directing and organizing whatever attentional capacity he or she has available. This involves a resistance to distractibility combined with monitoring of unattended stimuli in the case that something relevant should occur which demands attention. To refer again to the models of Reason, and Norman and Shallice, attentional control is conceptualized by them as directing and organizing attentional capacity.

The locus of injury associated with TBI is often focused in the orbitofrontal and prefrontal areas due to the susceptibility of theses regions to acceleration-deceleration and

blunt impact injuries. As discussed, neural systems composing these areas play a critical role in executive functions and in the control of attention (Cohen, 1993). Damage to these regions could therefore result in deficits of attentional control. This control deficit could be manifested in several different presentations. The frontal lobes are thought to be involved in mediating inhibitory processes which are necessary to selective and focused attention (Cohen, 1993). With a deficit of this inhibitory mechanism, distractibility and deficits on tasks of focused attention would be observed. Deficits on these types of measures have been reported in patients with TBI (Stuss et al., 1989, Levin, 1989; Baribeau, Ethier, & Braun, 1989).

It has also been suggested that the frontal cortex is important in responding to irrelevant environmental stimuli, with damage in this region diminishing the neural reaction to novel stimuli (Foster, Eskes, & Stuss, 1994). A deficit in the frontal control of attention could therefore be manifested as insufficient monitoring of environmental stimuli as suggested by the work of Rugg and colleagues (1993). They report evidence that this automatic attentional shifting appears to be impaired in the TBI population. Their research is electrophysiological in nature and will be examined more thoroughly in the electrophysiology section of this paper. This finding is interesting in light of the fact that the majority of research into the cognitive deficits experienced by this group has been directed towards investigating specific types of controlled or intentional information processing. The findings of Rugg and colleagues are suggestive of a different type of deficit in this population. These patients could be impaired in the automatic process responsible for

monitoring external stimuli and attending to relevant signals, or perhaps the attentional control system responsible for driving this automatic behavior is deficient.

Additional evidence for the dysfunction of attentional control in the TBI group comes from a study investigating the ability to shift and focus attention following TBI. A cued reaction time task was used which measured the reaction time benefit of valid directional cueing and the reaction time cost of miscuing. Reaction time measures were similar for the TBI and the control group. The cost due to invalid cueing was also the same for both groups; however, the TBI group demonstrated reduced benefit with valid cueing as compared to controls. The authors suggest that TBI affects the normal ability to direct attention and to maintain attention at a specific location (Cremona-Meteyard, Clark, Wright, & Geffen, 1992).

In addition to problems inhibiting irrelevant responding, monitoring environmental stimuli and shifting attention, a deficit in the control of attention could result in automatic and controlled processes being allocated in an inefficient manner. Stimuli normally attended to automatically, may receive capacity limited, conscious or controlled processing. This could result in deficits on measures of controlled processing as well as on measures of automatic processing. If behaviors normally carried out automatically receive controlled or intentional processing, abnormalities may be noted; for example, they may occur more slowly. If there are less resources available due to the utilization of capacity limited controlled processing to carry out normally automatic tasks, performance on controlled processing tasks will decrease. Heinze and colleagues (1992) did indeed find TBI subjects to be impaired on tasks tapping both automatic and controlled processing. They utilized a

feature detection and a feature conjunction task to address the question of whether the cognitive slowness exhibited by the head injured population reflected deficits in capacity-limited stages of stimulus analysis, or whether dysfunctions of automatic processes without capacity constraints also played a role. In addition, these researchers utilized electrophysiological measures which will be discussed at length at a later point in this paper. Performance of the TBI subjects on the feature detection task, which is believed to tap automatic processes, was slower than that of normal controls. They also exhibited slower reaction times in the feature conjunction condition which assesses controlled processing. The authors interpret their results as being indicative of the possibility of an impairment of automatic processing, in addition to an impairment of controlled processing. It is plausible that the impairments are due to an inefficient allocation of attentional resources by an impaired attentional control system.

A study carried out by Levin and colleagues (1988) examined the memory performance of TBI subjects. These authors predicted that free recall of a word list would be impaired indicating impaired controlled processing. They conversely predicted that frequency information would be maintained as this is believed to be processed automatically. However, the results were surprising in that TBI subjects were impaired on both tasks. This led the investigators to carry out a replicative study and again they found that the performance of the TBI subjects on frequency estimation tasks was greatly diminished when compared to control group performance. These findings suggest that features which are ordinarily processed in an automatic mode require greater effort in survivors of severe head injury. If the head injured patient is expending greater capacity to

process relatively automatic contextual features, then there is less capacity for encoding the actual material to be learned (Levin, 1989). More information with regard to this issue will be informative to the design of rehabilitative programs.

The Application of Event-Related Potentials to the Study of Cognitive Deficits in TBI

An obstacle in information processing research is the difficulty in decomposing the information processing sequence (Kramer & Donchin, 1987). The recording of event related potentials (ERP) is proving to be increasingly helpful in this respect. The ERP is a transient series of voltage oscillations in the brain that can be recorded from the scalp in response to the occurrence of a discrete event (Donchin, 1981). ERPs can be divided into exogenous and endogenous components. The early or exogenous ERPs, elicited during the first 100 milliseconds (msec) after stimulus onset, are used frequently as a standard technique for diagnosing sensory deficits and certain neurological disorders such as Multiple Sclerosis. Abnormalities in these components are indicative of the integrity of the sensory pathway in question. The endogenous evoked potentials are believed to reflect cognitive functioning. They provide information about brain electrical events coinciding with cognitive processes.

The P300 component of the ERP is a positive going component which occurs at approximately 300 msec post stimulus. The P300 (P3b) is typically elicited when a stimulus of relevance to the subject occurs. Paradigms involving the recognition of a target among a group of stimuli are commonly used in studies wishing to examine the P300. Its amplitude is related to probability and certainty whereas its latency is believed to be related to task difficulty (Picton, 1992). It is thought to index the updating, or revising of templates in

memory (Deacon-Eliot, Campbell, Suffield, & Proulx, 1987). It involves maintaining a memory for a target, stimulus evaluation, matching and comparison ability, decision making and the initiation of a response. It is therefore believed to reflect the end of a mental matching response set (Picton, 1992). There is an extensive literature documenting the longstanding debate regarding the psychological underpinning of the P300 component. However, as Knight states (1990), it cannot be generated without initial phasic attention to a discrete stimulus. Therefore, the P300 can be used as a neurophysiologic index of attention capacity in humans.

The P300 appears to involve two components which can be elicited under different circumstances. A frontocentrally maximal and automatically elicited P300 has been termed P3a. It is typically generated 20 to 50 msec earlier than the P3b. It is elicited by unexpected, deviant stimuli with no task relevance and reflects passive orienting. It may represent a central nervous system component of the orienting response (Squires, Squires & Hillyard, 1975). This passive attention responsible for stimulus selection at the preattentive stage corresponds to automatic processing (Tachibana, Toda & Sugita, 1992). The P3a can be used as an index of involuntary automatic attention to potentially significant environmental events (Knight, 1990).

The P3b component, as discussed above, is usually the measure of interest as it is reflective of capacity and is related to controlled processing. The P300 has been utilized as a dependent variable in many studies with clinical populations because it appears to be variable among these groups. Its latency appears to increase with age and with age-related disorders such as Alzheimer's Disease (Tachibana et al, 1992; Williams, Jones, Briscoe,

Thomas & Cronin, 1991). Abnormalities have also been found in children with Attention Deficit Disorder (Robaey, Breton, Dugas, & Renault, 1992). Several researchers have found the latency of the P3b component to be delayed in the TBI population (Heinze et al, 1992; Papincolaou, Levin, Eisenberg, Moore, Goethe & High, 1984). Reductions in amplitude of this component have also been reported in this group (Baribeau et al, 1989; Campbell et al., 1986; Rugg, et al., 1988).

Interesting results have been documented when P300 is looked at within the context of automatic and controlled processing in both TBI and in normal controls. Rugg and colleagues (1993) utilized an oddball[1] paradigm which consisted of target, nontarget and novel stimuli to assess automatic and controlled processes in TBI. The most robust finding was that P3a amplitude was reduced and its latency was delayed as compared to controls. P3a was elicited by the novel sounds. This is suggestive of an impaired mechanism in the monitoring of external environmental stimuli. In contrast to the findings of Rugg and colleagues, Baribeau and colleagues (1989) report large P3a amplitudes to nontarget standard tones which they interpret as being indicative of excess distractibility, perhaps due to impaired inhibitory mechanisms. However, methodological differences exist in the two studies.

Ford and colleagues (1994) present evidence which supports the dissociation of P3a and P3b and their respective relation to automatic and controlled processing. They elicited P3a through novel, startling sounds and utilized the standard oddball paradigm to elicit P3b. They concur with Rugg and colleagues by stating that the P300 elicited automatically is

[1] The target is an "oddball" tone in that it differs from the standard tones (i.e., louder, softer, etc).

maximal centrally, with the P300 elicited through the standard oddball paradigm being maximal parietally. This underscores the need to employ scalp topography as a dependent measure when utilizing P300 to investigate automatic and controlled processing in addition to amplitude and latency measures. The amplitude of the P3a correlated significantly with frontal grey matter volume obtained through the use of Magnetic Resonance Imaging (MRI). The amplitude of the P3b correlated significantly with parietal grey matter volume. They conclude by stating that their data support the hypothesis that P300s elicited automatically and effortfully may be generated by different neural networks involving different areas of the brain. This finding is very interesting in light of our current investigation. If automatic processes are localized frontally they could be selectively impaired following TBI.

As previously mentioned Heinze and colleagues (1992) utilized a feature detection and a feature conjunction task to examine the possibility that automatic processes without capacity played a role in the deficits observed in TBI, in addition to controlled processing deficits. Their ERP findings were particularly interesting. In the two conditions (feature detection and feature conjunction), presumed to represent automatic and controlled processing respectively, control subjects showed different P300 wave forms. TBI subjects displayed qualitatively similar wave forms in the two conditions. This is supportive of the possibility of inefficient allocation of attentional resources resulting in a deficit in automatic processing. Perhaps TBI subjects utilized controlled processing in the feature detection condition which was processed automatically by normal control subjects.

The P300 is a very exciting measure to be used with the head injured population. Due to its close relationship to cognitive processes, any improvement in these processes could be reflected in P300 changes such as decreased latency. With increased research in this area P300 could become a physiological marker of improvement due to rehabilitation. On a larger scale, this type of research could have implications for studies of brain plasticity, as the generators of the P300 component perhaps change or reorganize due to the cognitive exercises employed in rehabilitation. Evidence already exists that P3b latencies can be decreased following cognitive rehabilitation (Baribeau et al., 1989).

PURPOSE OF THE STUDY

The objective of this study was to further investigate the role of automatic processes with relation to the cognitive deficits experienced by individuals with TBI, and to correlate behavioral measures of automatic and controlled processing with event related potential measures. Of particular interest is the P300 component. I assessed the performance of individuals with TBI and normal controls on a visual ERP task designed to tap controlled processing and on a similar task designed to tap automatic processing. It was a goal of this study to assess the differences in the P300 wave form in these two conditions as well as to compare the P300 between the two groups to establish whether TBI subjects process the task information in a qualitatively different manner than controls. As reported by Heinze and colleagues (1992) using a similar task, TBI subjects did not show as large a difference in P300 morphology between an automatic and controlled processing condition as controls did. These authors interpret their results as being indicative of impaired automatic processing mechanisms.

A second objective was to investigate the nature of the response of individuals with TBI to novel stimuli while engaged in tasks of differing levels of difficulty. Rugg and colleagues (1993) found that the P3a elicited by novel sounds evidenced delayed latency and reduced amplitude, while Baribeau and colleagues (1989) found increased amplitude of the P3a component suggesting heightened distractibility in the TBI group. A novel distractor was presented during the two visual ERP task conditions (controlled and

automatic) to determine whether P3a was affected by the level of difficulty of the primary task.

I suspected that TBI subjects would perform more slowly on both the automatic and controlled processing task as general slowing is a consistent finding in this population. I expected that both groups would evidence longer reaction times during the controlled processing condition. In addition, it was hypothesized that in the TBI group the wave forms recorded in the two conditions would not show as great a difference in morphology as wave forms recorded from control subjects, suggesting inefficient allocation of attentional resources. It was also expected that the P3b component would be delayed in latency, diminished in amplitude, or both.

With regard to the second objective, I suspected that the P3a component of the P300 potential would be reduced in amplitude and delayed in latency in TBI subjects as compared to normal control subjects, indicating impaired ability to monitor environmental stimuli. I proposed that this deficit is reflective of an impairment in automatic monitoring of the environment by TBI subjects, which could be indicative of a deficit in attentional control. I expected that the P3a response would not vary as a function of the level of difficulty of the primary task.

METHOD

Subjects

Ten TBI subjects were recruited from the wait list for admission to the Head Injury Rehabilitation Outpatient Program at the Gorge Road Hospital in Victoria, B.C., and from the Neuropsychology Clinic at the University of Victoria. Demographic and clinical information are provided in Tables 1and 3. All subjects except one evidenced post traumatic amnesia. Subjects were tested at least eight months post injury. Subjects ranged in age from 18 to 42 with a mean age of 33. This age range was chosen to exclude the possibility that developmental or ageing effects could be responsible for any ERP differences observed. Potential subjects who had sustained a previous head injury or experienced seizures were excluded from the study. Three potential subjects with TBI did not meet inclusion criteria and were therefore rejected. No subjects reported currently abusing alcohol; however subjects were not excluded on the basis of a prior history of substance abuse. Alcohol abuse is common in individuals who sustain traumatic brain injuries (Grafman & Salazar, 1987). At the time of testing only two subjects were gainfully employed; all others were unable to work.

Ten control subjects were selected from amongst the friends and family of the TBI subjects to assure demographic similarity (see Table 2). This method of selection of normal control subjects for use with a TBI population is suggested in the literature (Dikmen & Temkin, 1987). Eight potential NC subjects were excluded from the study. Subjects were matched for age, years of education, and gender. Matching criteria for age were plus or

minus five years; for education subjects were matched within two years of education. Statistical analysis revealed that groups did not differ significantly with respect to age (TBI group mean age = 33, SD = 7.27; NC group mean age = 33.7, SD = 8.45), or education (TBI group mean years of education = 13.2, SD = 1.93; NC group mean years of education = 13.6, SD = 1.71). The gender breakdown was equivalent between groups (four males, six females). IQ was estimated using the Kaufman Brief Test of Intelligence (K-Bit) (Kaufman & Kaufman, 1990). Statistical analysis revealed that groups did not differ significantly with respect to general cognitive ability (IQ) (TBI group mean IQ = 102.2, SD=11.16; NC group mean IQ = 108.5, SD = 9.86). Controls were screened for lifetime history of any psychiatric or neurological disorders and were in good physical health. All subjects had normal or corrected to normal vision. Several ERP files for certain subjects had to be discarded due to excessive eye artifact or muscle tension resulting in an unequal number of subjects for certain statistical comparisons. All subjects did produce some useable ERP data.

Table 1.

Subject Characteristics: Traumatic Brain Injured Group

Subject	Gender	Age	Education (yrs)	Pre-injury Occupation	IQ (K-Bit)
1	F	18	12	Prep. cook	100
2	M	34	12	Steward	75
3	F	29	12	Nanny (present)	98
4	F	35	16	Dental Assistant	106
5	M	34	11	Handyman (present)	102
6	F	41	15	Journalist	118
7	F	37	14	Office work	110
8	M	42	16	Teacher	102
9	F	35	13	Manager	108
10	M	25	11	Student	103

Table 2.

Subject Characteristics: Normal Control Group

Subject	Gender	Age	Education (yrs)	Occupation	IQ (K-Bit)
11	F	21	13	Receptionist	95
12	M	36	14	Catering worker	105
13	F	35	11	Homemaker	91
14	F	33	16	Retail	109
15	M	31	12	Promotions	108
16	F	34	13	Self-employed	109
17	F	46	15	Bookkeeper	111
18	M	48	16	Search and Rescue Controller	121
19	F	32	14	Office Work	122
20	M	22	12	Prosthetic Technician	114

Table 3.

TBI Group: Injury Profile

Subject	Time Since Injury (months)	Accident	PTA	CT	LOC	GCS
1	11	MVA (bleeding l. ear)	2 hrs	Normal	1 hr	15
2	15	Direct blow to l. temp area	Patchy	N\A	None	N\A
3	24	MVA (Motorcycle)	4 days	N\A	Unknown	15
4	17	MVA	3-4 wks	Swelling r. frontal lobe	1 wk	5
5	206	MVA	2-4 wks	Skull fracture l. frontal lobe	5 days	N\A
6	45	MVA	None	N\A	None	N\A
7	14	MVA	20 mins	N\A	5 mins	N\A
8	8	MVA	1 day	N\A	Unclear	N\A
9	16	MVA	4 hrs	L. temporal lobe contusion	10 mins	N\A
10	33	MVA	Patchy	N\A	5 mins	N\A

There are various methods used to assess the severity of TBI. Typically used measures include: Glasgow Coma Scale (GCS), duration of Post Traumatic Amnesia (PTA), and duration of loss of consciousness (LOC). GCS scores were available for only three subjects. This measure has limited utility as subjects who are being compared in the same study have frequently had GCS ratings carried out at differing intervals following their injury. Score on the GCS changes over time as the injured person begins to regain consciousness and orientation. The GCS has also been criticized on the grounds that it sacrifices information for reliability. There is some controversy over exactly how long after impact the GCS is a valid indicator of injury severity (Eisenberg & Weiner, 1987). Four subjects reported a loss of consciousness of greater then thirty minutes which would classify their injury as moderate to severe; however six subjects' TBI would be classified as mild by

the LOC criteria. Duration of PTA is also used to classify TBI, however different authors have utilized different durations to provide classifications (see Jones, 1992 for a review). Five subjects experienced PTA of more than seven hours in duration which can be considered severe. CT evidence was available for four subjects and damage to the frontal lobes was documented for two subjects. This information suggests that 60 percent of the subjects may have had a categorically different level of injury, placing them in the mild TBI category, depending upon which measure of severity is considered.

Procedures

Prior to participation in the study the experimental procedures and the purpose of the study were explained to subjects. All subjects completed a written informed consent form and were informed that they were free to withdraw from the study at any time, and that all of the data provided by them would remain strictly confidential.

ERP Tasks

Subjects were given a visual ERP task. This task was designed to assess subjects' ability to detect target stimuli as well as to assess their response to novel distractors while engaged in tasks with differential attentional demands. Two task conditions were administered.

Task One

The animal word condition was implemented to engage subjects in an automatic processing task. In this condition subjects sat in front of a computer monitor and were instructed to respond to target stimuli as quickly and accurately as possible by clicking on a mouse. Target stimuli were animal words consisting of four, five, six and seven letters, and were presented 15% of the time (p=0.15). Word reading is a task that has been established to be overlearned and processed automatically (Stroop, 1935). Reaction time was not expected to increase as a function of the number of letters in the word, indicating that subjects were processing the words in a parallel, automatic manner. The non-target stimuli (p=0.70) in this condition were non-animal words consisting of four, five, six and seven letters. Target and non-target stimuli were matched for imageability and for frequency of occurrence in the English language. Stimuli were presented as black letters against a pale grey background. Stimuli remained on the screen for one second with an interstimulus interval of two seconds. Novel distractor stimuli were also presented, appearing on the screen (p=0.15) in place of the target or non-target stimuli. Probability of the target and novel stimuli were identical to ensure that any differences observed in the P3a and P3b components would not be due to probability of occurrence. The novel distractor stimuli consisted of a face presented in the center of the screen. This stimulus was selected as a novel distractor as it is definitely distinct from lexical stimuli. Accuracy and latency measures were recorded as well as ERP data.

Task Two

A condition designed to assess controlled or effortful processing was also employed. In this condition target stimuli were words of four, five, six or seven letters which contained two vowels. Subjects processed the stimuli in a serial manner, searching each word letter by letter to detect two vowels. Non-target stimuli consisted of words of four, five, six or seven letters in length that did not possess two vowels (i.e. containing one, three or four vowels). Animal words were not used due to the possibility of a carry over effect for those subjects exposed to the animal word condition before the two vowel condition. The probabilities of the presentation of the targets and non-targets, interstimulus interval, and stimulus exposure time were the same in the two tasks. Distractor stimuli were the same as in the animal word condition. The order of the presentation of the two conditions was counterbalanced across subjects. Subjects were again encouraged to respond as quickly and accurately as possible with their dominant hand by clicking on a mouse. Subjects were practised on both tasks to ensure understanding of task requirements.

This paradigm provides a measure of the amplitude and latency of the P3b component while subjects are engaged in a task requiring automatic processing and one requiring controlled processing. The P3a component is also elicited by presenting novel distractors. This measure is valuable for establishing whether TBI subjects are more distractible than normal control subjects as evidenced by larger P3a amplitude, or if TBI subjects do not monitor environmental stimuli while engaged in a primary task which would be suggested by delayed P3a latency or smaller amplitude. Testing was carried out in

the morning to guard against the possibility of time of day effects as ERPs have been reported to vary as a function of arousal level throughout the day (Geisler & Polich, 1990).

ERP Recording

Electrodes were placed on the scalp for 20 response sites using an electrode cap according to the 10-20 International system. Electrodes placed on the mandibles served as references. Electrode impedance was kept below five KΩ. Electroencephalographic (EEG) data were recorded using a Bio-Logic Brain Atlas (Bio-Logic Systems). Seven minutes of EEG data were recorded for each subject in each task condition. Data were recorded in four 3.5 minute blocks with subjects taking short rest breaks in between. EEG was amplified 20 000 times and filtered with a 60 Hz notch filter with a band pass of 0.01-30 Hz. Sampling rate was 200 Hz from 250 msec prior to stimulus onset to 1030 msec post-stimulus. An EEG marking method was used whereby stimulus onset was registered by a 'TTL' pulse signal. This signal was delivered to an inactive channel (Oz) from the Macintosh computer which served to present the stimuli. 'TTL' pulses identified whether a target, non target or novel stimuli had been presented. ERPs were then derived off-line. Microvolt values of artifact were identified by visual inspection individually for each subject and then an automatic artifact rejection system was used to reject trials contaminated with eye blinks, eye movements or excessive muscular activity.

ERP Data Analysis

EEG was averaged using an editing program which rejected trials with microvolt values above a pre-set value as mentioned above. The microvolt values used for the rejection of artifact for each subject were determined by measuring the amplitude of electrical activity due to blinks and other artifactual sources (i.e., eye movements, muscular activity). This value was set at 50 μV for 17 of 20 subjects. Two subjects had artifact rejection levels set at 55 μV and one subject had artifact rejection levels set at 60 μV. A maximum of 30 trials was used to produce average wave forms elicited by target stimuli for each subject. A maximum of 30 trials was also used to produce average wave forms to novel stimuli. A maximum of 140 trials were averaged to produce non target wave forms for each subject. This averaging procedure was carried out for both task conditions. Although 30 trials were recorded to be used for creating averaged wave forms to both target and novel stimuli, strict artifact rejection criteria resulted in some wave forms being created from fewer than 30 trials. This resulted in the loss of data for some subjects who did not produce enough artifact-free trials to create reliable wave forms (see Table 4). P3a and P3b wave forms were determined through visual inspection. Peak amplitude was measured with respect to the mean of the 250 msec pre-stimulus baseline. Latency was measured with respect to stimulus onset. In addition, peak amplitude and latency values were selected using a Brain Mapping Utilities Peak Seeking program (Roberts, Miller & Languis, 1987). This was carried out as a check against the visual inspection.

Table 4.

Subjects with Missing Data for ERP Analysis
X = missing data

Subject	Animal Word Condition		Two Vowel Condition	
	Target	Novel	Target	Novel
3 (TBI)	X	X		
4 (TBI)				X
12 (NC)				X
14 (NC)	X	X		X
17 (NC)		X		
18 (NC)			X	X

Neuropsychological Test Battery

A neuropsychological test battery lasting approximately 60 minutes was administered in order to obtain additional information regarding subjects' current level of functioning in different cognitive domains, particularly attention and memory. These tests are non-invasive, paper and pencil standard psychometric measures.

The Kaufman Brief Test of Intelligence (K-BIT) (Kaufman & Kaufman, 1990) was administered lasting approximately 30 minutes. This measure was carried out in order to obtain an estimation of IQ for the purpose of matching the two groups. The California Verbal Learning Test (CVLT) (Delis, Kramer, Kaplan & Ober, 1987) was also administered. It is a frequently used measure of memory function which involves having subjects learn a list of 15 words which are presented in the auditory modality. Scores utilized for the purposes of this study included the total number of words recalled over five

trials, number of words recalled for the first trial and for the fifth trial, number of words recalled following the presentation of a distractor list of another 15 words, and the number of words recalled following a twenty minute delay (delay trial). The Brown-Peterson Consonant Trigrams task (CCC) (Brown, 1958) was administered as it has been shown in the literature to be particularly sensitive to the deficits of individuals with TBI (Stuss, Ely, Hugenholtz, Richard, LaRochelle, Poirier, & Bell, 1985). It is a measure of short term working memory with interference. It involves presenting subjects with three letters and then having them perform the distracting task of counting backwards by threes. Subjects are told to stop after either nine, eighteen, or thirty-six seconds and are then asked to recall the letters. Three scores are provided: total number of letters correctly recalled for the nine, eighteen, and thirty-six second trials. A standard Stroop Colour Word Test was administered as a measure of focused attention (Stroop, 1935). This test requires subjects to name coloured dots (red, blue, green) as quickly as possible. They are then presented with words and asked to read them as quickly as possible. The interference trial of the Stroop task requires subjects to read colour words (red, blue, green) printed in different colours (i.e., the word "red" printed in blue ink). The Brief Test of Attention (BTA) (Schretlen, Bobholz & Brandt, 1989) was administered as a measure of executive attentional control. Subjects were presented with a series of letters and numbers on a tape recorder. They were asked to count the letters and ignore the numbers, and were then asked to count the numbers and ignore the letters. The total score is composed of the number of correct responses. Neuropsychological assessment followed the ERP test session.

STATISTICAL ANALYSES AND RESULTS

The following section will investigate the hypotheses proposed previously. This will entail a reiteration of the hypotheses, a description of the statistical procedures utilized, and the subsequent findings. An alpha level of 0.05 was used for all statistical tests. Correlations of $p<.05$ were interpreted only if the correlation was of 0.30 magnitude or larger. Correlation magnitude was defined according to Cohen's (1988) criteria for correlation effect size (small: r = .1; medium: r = .3; large r = .5).

Analysis of Neuropsychological Data

A multivariate analysis of variance (MANOVA) was carried out to investigate differences between the two groups on the neuropsychological measures administered. Due to the changes in performance with age all neuropsychological raw scores were converted into Z scores to account for age and gender differences. Analyses were carried out for total recall score, recall on trials one and five, and the short delay with interference and long delay trial of the CVLT. Consonant Trigrams was analyzed by looking at performance on each of the three time conditions (9, 18, and 36 seconds). Total score on the BTA was utilized. For the Stroop task, time to read the colour trial, time to read the colour word trial (interference trial) and, the difference in reading times between the colour word and the colour trial were the measures of interest.

Groups did not differ on any of the CVLT measures. Performance differences between groups on the BTA approached statistical significance, $F(1, 17) = 4.11529$, $p =$

.058. Although group differences on the 18 and 36 second Consonant Trigrams conditions were statistically significant, the 9 second condition only approached significance, F(1,18) = 4.02571, p = .06. The Stroop difference score (time to read interference trial - time to read colour trial) also approached significance, F(1,18) = 4.15763, p = .056. Several measures revealed statistically significant group differences exceeding 0.05; these are presented in Table 5.

Table 5.

Neuropsychological Measures Which Revealed Significant Group Differences[a]

Test	TBI Group		Control Group		F	P
	Mean	SD	Mean	SD		
Stroop (colour trial)	-1.5870	1.8115	-.1090	1.0769	4.9184	.040
Stroop (interference trial)	-2.6010	2.4415	-.319	1.0132	6.49318	.021
CCC (18)	-1.05	.86	-.147	.885	5.35881	.033
CCC (36)	-1.189	1.138	.214	1.461	5.74146	.028

[a] Scores are presented in the form of Z scores.

Analyses of Behavioural Data

HYPOTHESIS: *It was predicted that TBI subjects would perform more slowly on both the automatic and controlled processing tasks as compared to controls. Additionally, it was*

predicted that both groups would exhibit longer reaction times to the controlled processing (two vowel) condition as compared to the automatic processing (animal word) condition.

A repeated measures MANOVA was carried out to look at mean number of correct responses and reaction time data. There were significant main effects for group, $F(1,18) = 8.56$, $p = .009$, and condition, $F(1,18) = 156.2$, $p = .000$. Follow up with a one-way ANOVA indicated that there was no difference in the accuracy of target detection between the TBI and NC groups for either condition, $F(1,18) = 1.3595$, $p = .2588$ (animal words); $F(1,18) = 1.0636$, $p = .3161$ (two vowel words). TBI subjects correctly detected 85.9% of the targets in the automatic (animal word) condition and 78.4% of the targets in the controlled (two vowel) condition. NC subjects correctly detected 97.8% and 94.6% of the targets in the automatic and controlled conditions respectively. Paired t-tests revealed that both groups made significantly more errors in the controlled condition than in the automatic condition, $t(20) = 4.31$, $p = .047$, (see Figure 1). Commission errors were examined using paired t-tests; both groups made commission errors (false positives) but between group differences were not significant. However, differences between conditions for commission errors did reach statistical significance, $t(20) = -4.38$, $p = .000$, with both groups making more errors in the two vowel condition. Means (with standard deviations in parentheses) for false positives for the TBI group were 1.3 (1.252) for the automatic condition, and 18.9 (15.975) for the controlled condition. For the NC group means were .9 (.738) and 10.9 (11.827) for the automatic and controlled conditions respectively. These differences in error rates between

the two conditions confirms the added capacity load of the two vowel (controlled processing) condition.

Figure 1.

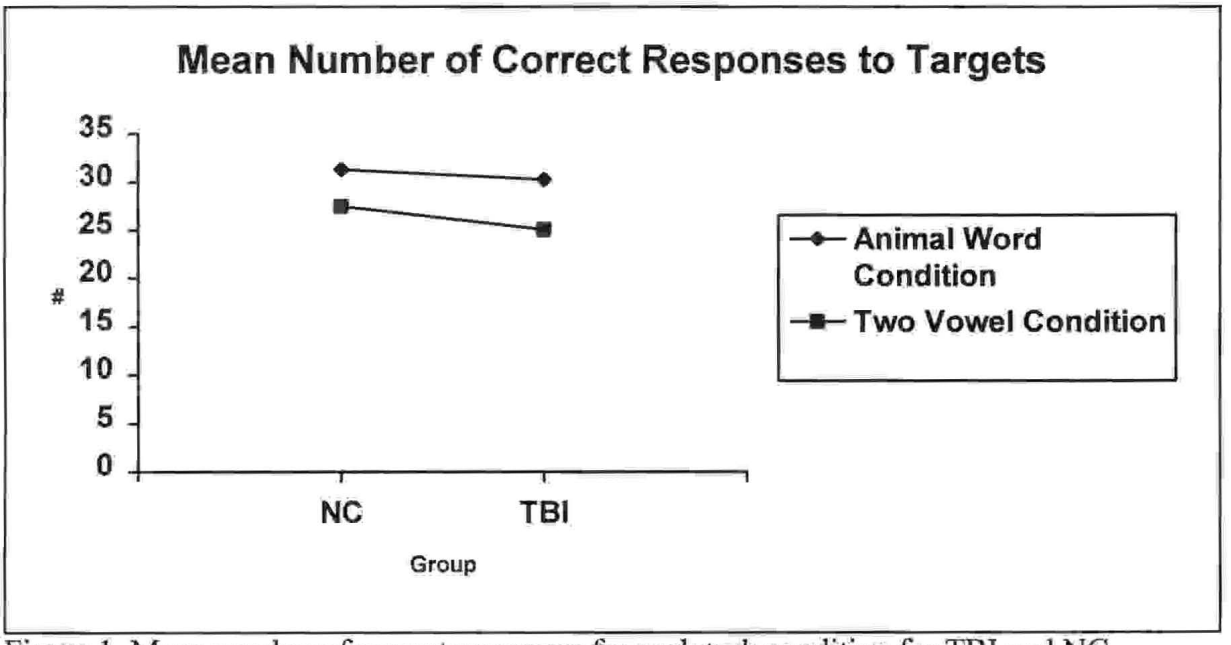

Figure 1. Mean number of correct responses for each task condition for TBI and NC subjects.

Both groups were also significantly slower in the two vowel condition than in the animal word condition, F (1,18) = 156.20, p = .000. Analysis revealed a significant interaction effect for group by measure, F (1,18) = 8.25, p =.01. Follow up with a one-way ANOVA indicated that the TBI group responded more slowly than the NC group in both conditions: F (1,18) = 9.72, p = .0059 and F (1,18) = 4.4637, p = .0489, respectively (See Figure 2).

Figure 2.

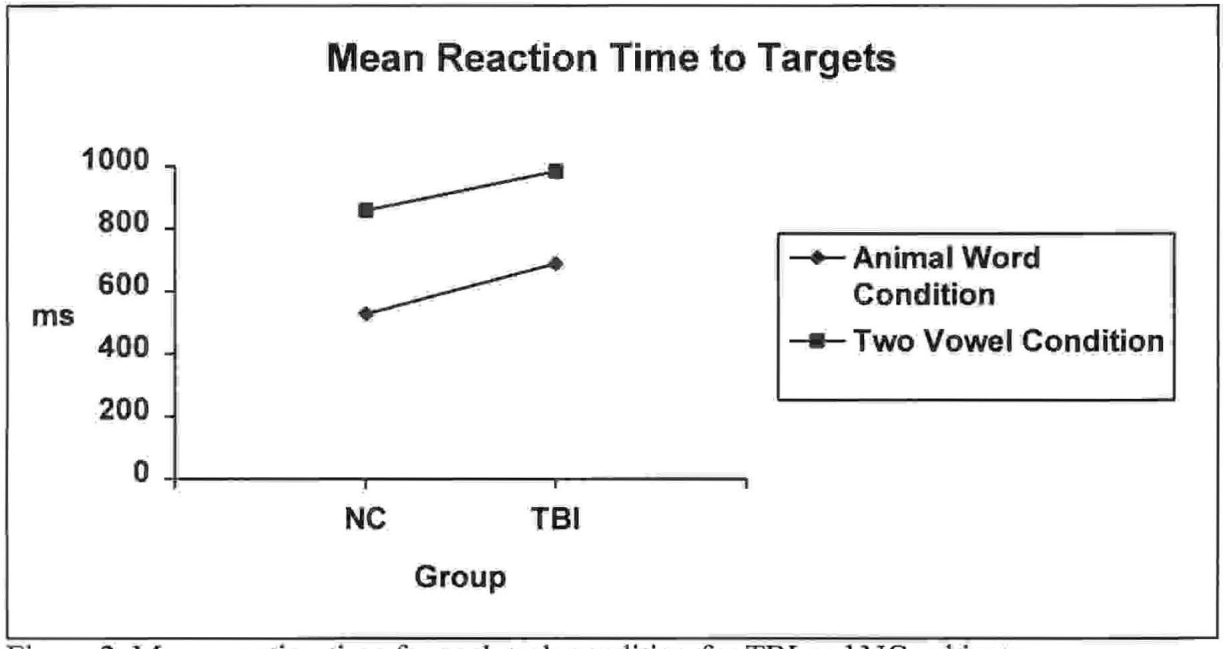

Figure 2. Mean reaction time for each task condition for TBI and NC subjects.

Analysis of ERP Data

HYPOTHESIS A: *It was predicted that the TBI subjects would not demonstrate a large difference in the amplitude and/or latency of the P3b wave form between conditions as compared to control subjects. It was also predicted that the P3b wave forms recorded in the TBI group would be delayed in latency and diminished in amplitude, as compared to controls.*

Grand mean ERPs recorded at midline frontal, central, and parietal sites (Fz, Cz, Pz) were used in the analysis. In each group, a large, parietally-distributed positivity was observed 450 - 500 msec after target presentation in both conditions. This is the P3b component as it is maximal parietally, is sensitive to low probability trials, and occurs

within an appropriate time window for the P300 component. The presentation of irrelevant novel stimuli elicited a positivity which occurred 375-425 msec after stimulus onset. This is the P3a component as it occurs earlier than the P3b and is elicited passively, without the subject being required to make any type of response.

P3b Component

Latency measures and baseline to peak measures of amplitude were submitted to a three factor (wave measure X condition X group) multivariate repeated measures analysis of variance. As previously mentioned, due to poor wave form resolution, trials for some subjects were rejected resulting in unequal N's for some analyses. Data were transformed using a square root transformation to account for outliers. MANOVA revealed no significant effects for group, $F(1,15) = .44$, $p = .515$ or condition, $F(1,15) = .8$, $p = .386$. No interaction effects were significant. However, there were obvious trends in the data which supported my hypothesis that TBI subjects would not show a large difference in P3b amplitude and/or latency between conditions, as compared to NC subjects. Large standard deviations may have obscured the finding of significant effects. Means and standard deviations for both groups in each condition are presented in Table 6.

Table 6.

Mean Amplitudes of the P3b for TBI and NC Subjects[a]

Condition	Mean	SD
	TBI Group	
Animal words (Condition 1)	7.489	5.3197
Two Vowel words (Condition 2)	6.496	4.4931
	NC Group	
Animal words (Condition 1)	10.7933	7.2872
Two Vowel words (Condition 2)	3.9989	2.0149

[a]Values are Microvolts.

The NC group evidenced larger P3b amplitudes to the presentation of animal words than the TBI group. For the two vowel word condition the situation was reversed with the TBI group exhibiting the larger amplitude wave form. What is interesting to note is that the TBI group demonstrated very little difference in peak amplitude between the two conditions whereas the NC group showed a large difference in amplitude across conditions, although neither of these differences reached statistical significance (see Figure 3). The same trend is evident in P3a amplitude to novel stimuli and will be discussed. Mean averaged wave forms for responses to target stimuli for NC subjects in the animal word and two vowel condition are presented in Figures 5 and 7, respectively. Mean averaged wave forms for TBI subjects

for responses to target stimuli in the animal word condition are presented in Figure 4. Mean averaged wave forms for responses to target stimuli in the two vowel condition are presented in Figure 6.

Figure 3 .

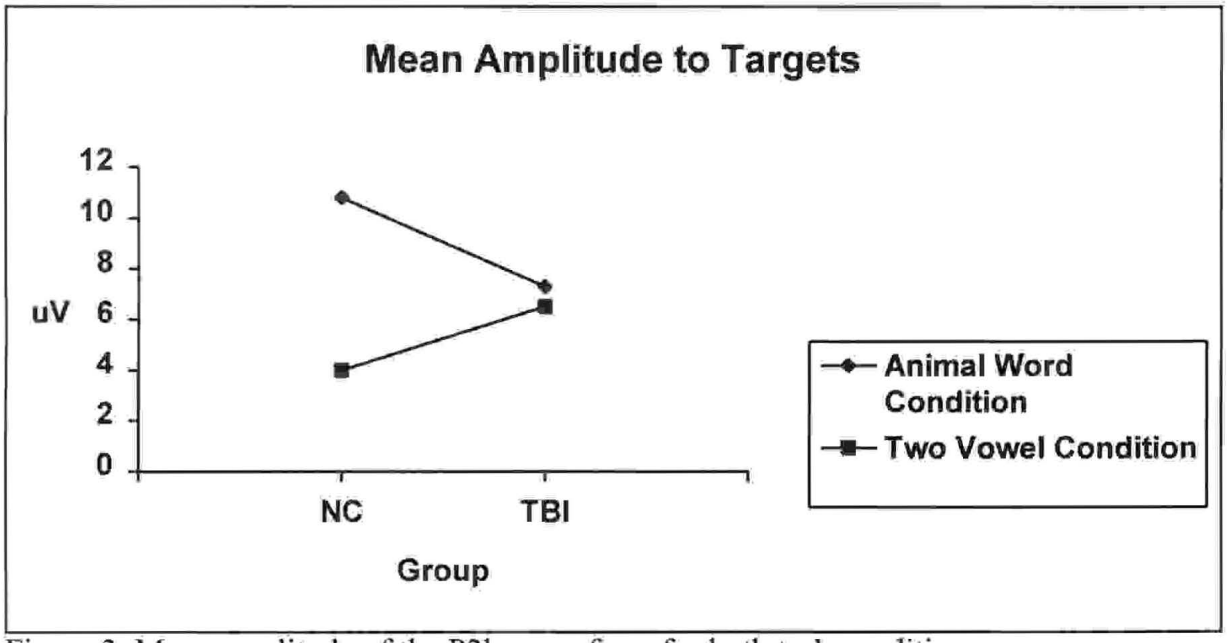

Figure 3. Mean amplitude of the P3b wave form for both task conditions.

Figure 4.

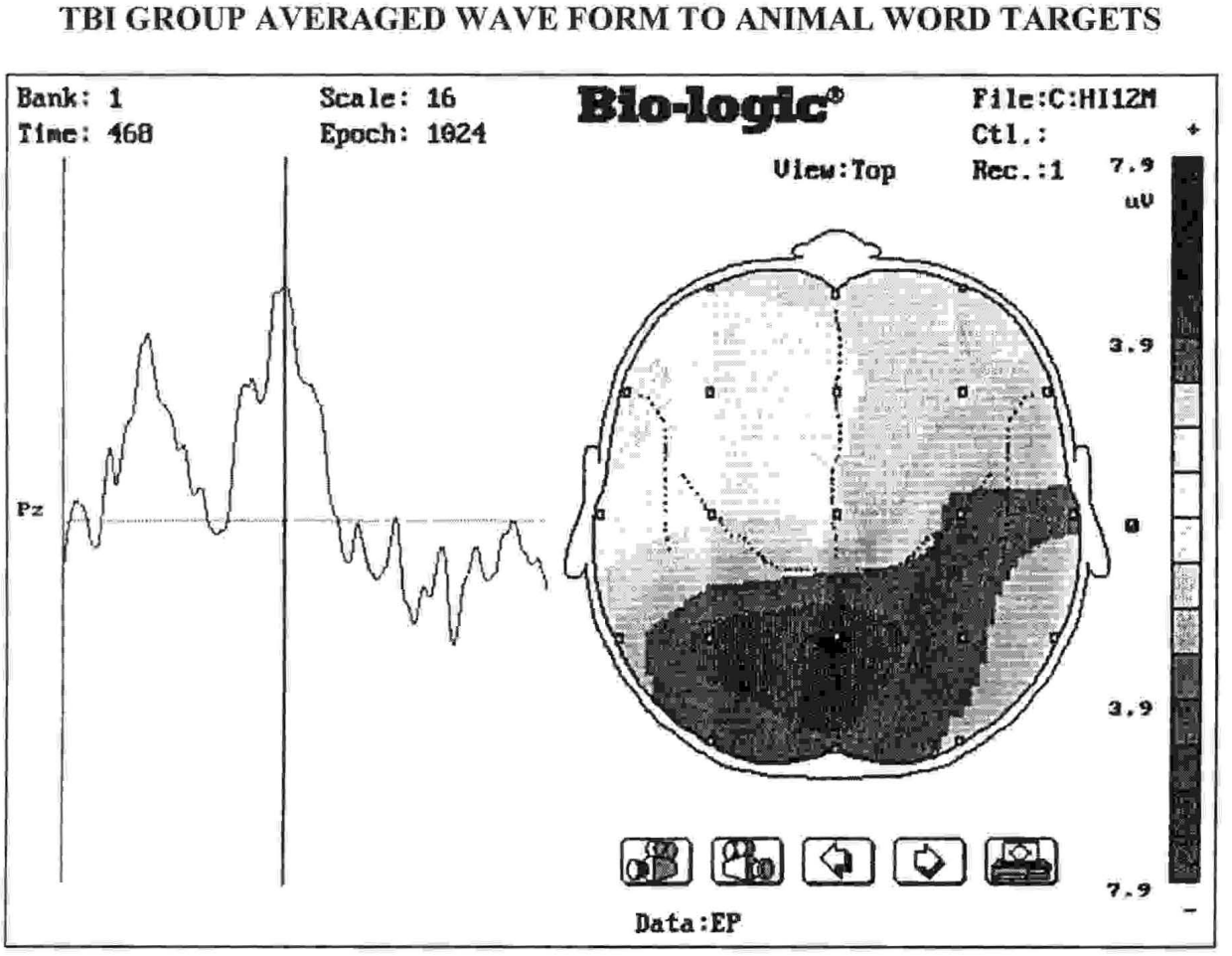

Figure 4. Averaged P3b wave form to animal word targets for the TBI group. The darkest scalp area represents the area of the highest amplitude electrical activity; this corresponds with the P3b wave form.

Figure 5.

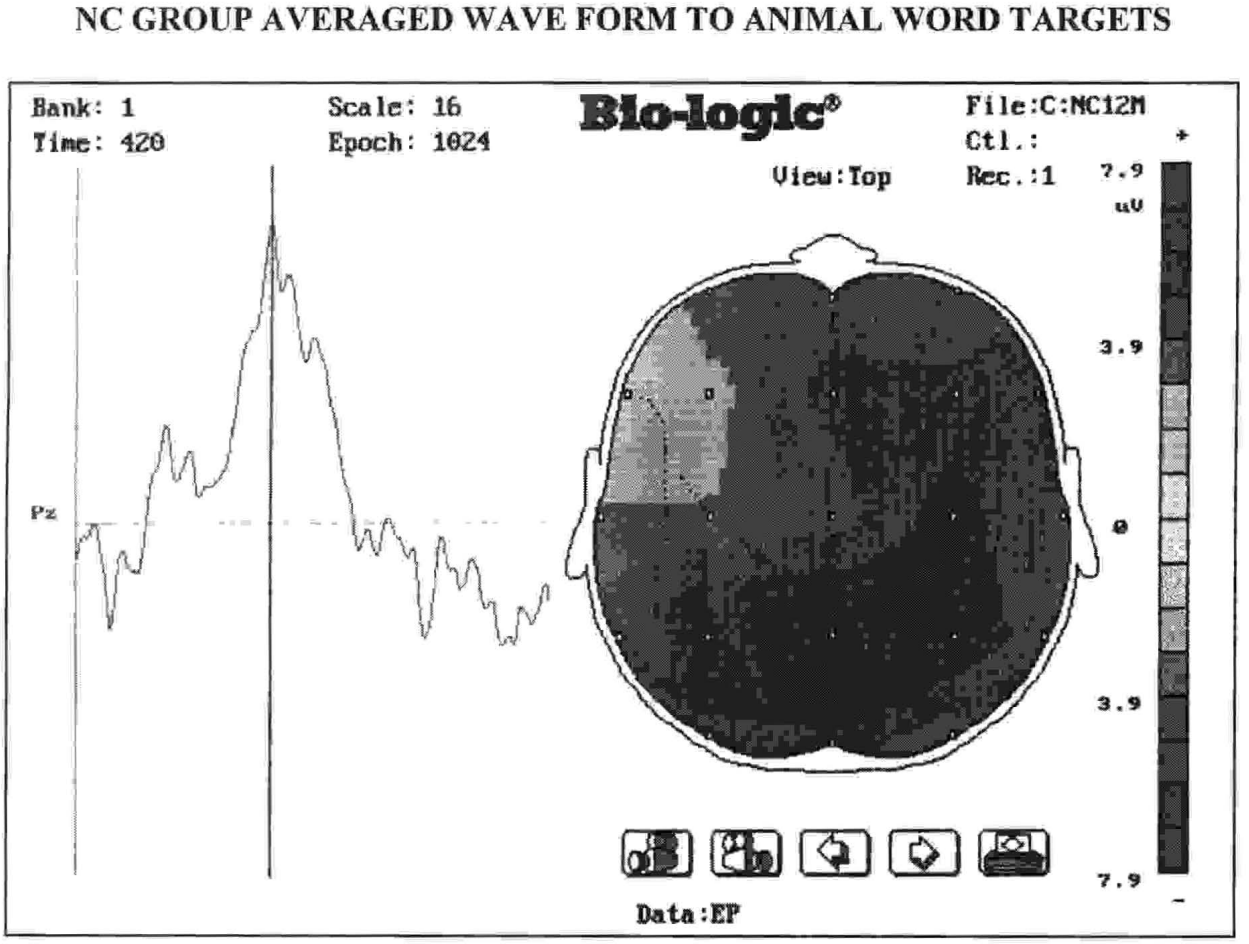

Figure 5. Averaged P3b wave form to animal word targets for the NC group. The darkest scalp area represents the area of the highest amplitude electrical activity.

Figure 6.

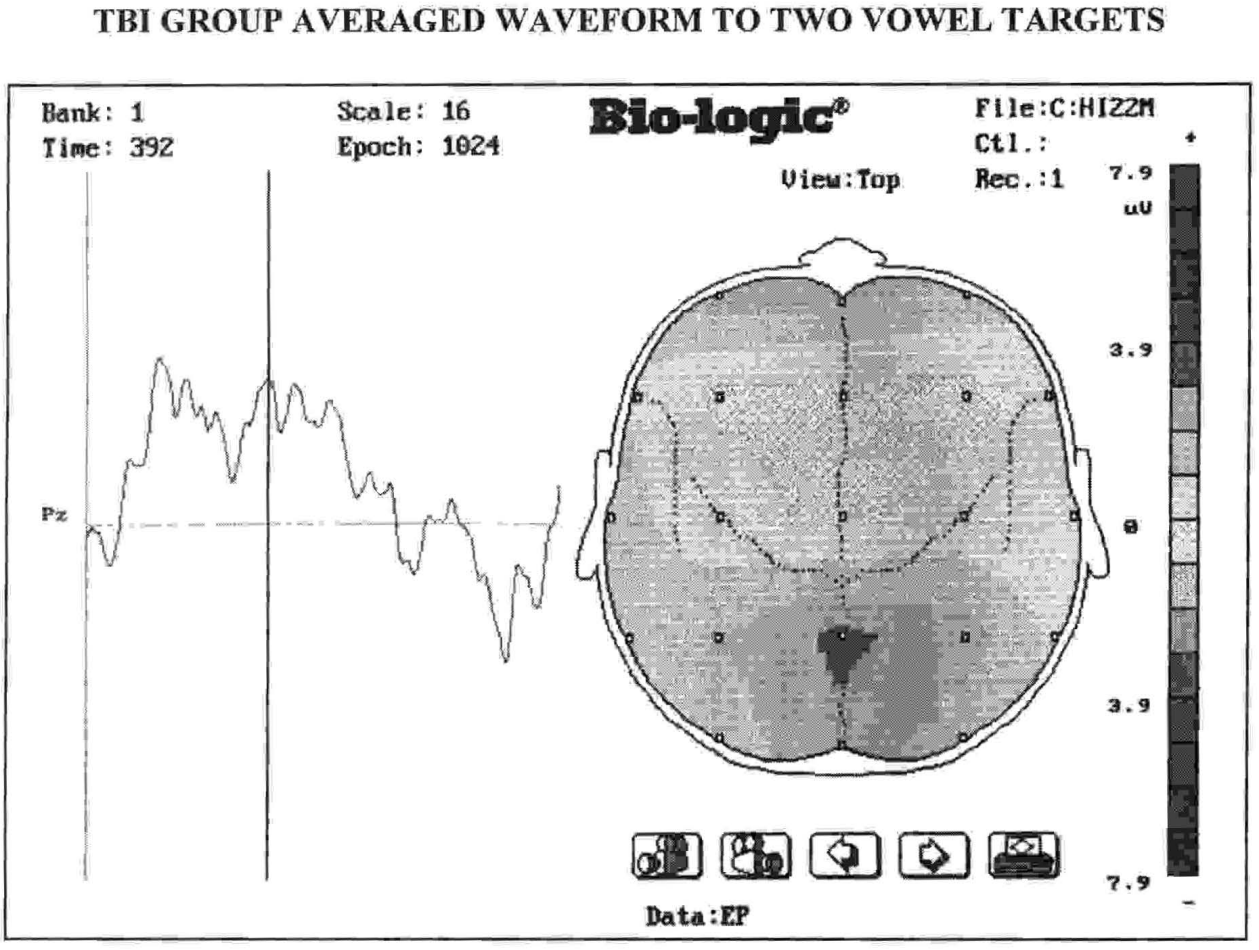

Figure 6. Averaged P3b wave form to targets in the two vowel condition for the TBI group. The darkest scalp area represents the area of the highest amplitude electrical activity; this corresponds with the P3b wave form.

Figure 7.

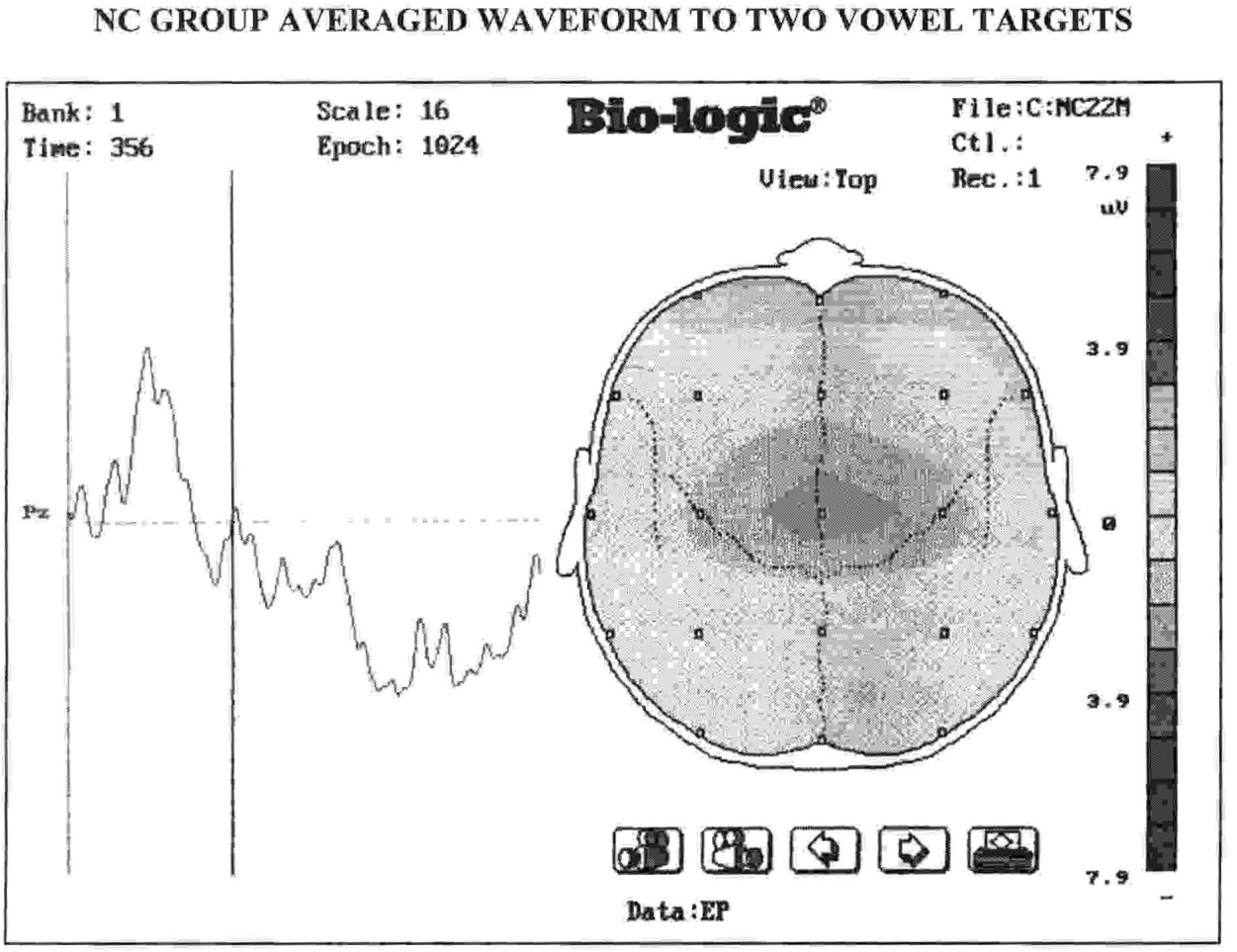

Figure 7. Averaged P3b wave form to targets in the two vowel condition for the NC group. The darkest scalp area represents the area of the highest amplitude electrical activity.

There were no trends in the data observed for P3b latency. In fact, mean latencies for the two groups were very similar. Means for the animal word condition (with standard deviations in parentheses) were as follows: TBI group 451.5556 msec (48.5157), NC group 439.1111 msec (55.4537). For the two vowel word condition means and standard deviations were: for the TBI group 472.8 msec (124.5051) and for the NC group 465.7778 msec (171.6638).

HYPOTHESIS B: *It was predicted that the P3a component would be reduced in amplitude and delayed in latency in TBI subjects as compared to NC subjects.*

P3a Component

Latency measures and baseline to peak measures of amplitude were submitted to a separate 3 factor (wave measure X condition X group) multivariate repeated measures analysis of variance. Data were transformed using a square root transformation to account for outliers. MANOVA revealed no significant effects for group, F (1,12) = 1.06, p = .324 or condition, F (1,12) = .59, p = .458. No interaction effects were significant. This data does not support the hypothesis that the P3a component would be reduced in amplitude and delayed in latency in TBI subjects as compared to NC subjects. As mentioned briefly above, the same trend was evident in the response to novel stimuli data as was observed in the response to target stimuli (see Figure 8). Again, small sample size and large standard deviations may have obscured the finding of significant effects. Means and standard deviations for both groups in each condition are presented in Table 7.

Table 7.

Mean Amplitude of the P3a Component for TBI and NC Subjects[a]

Condition	Mean	SD
	TBI Group	
Animal words (Condition 1)	5.70778	3.6729
Two vowel words (Condition 2)	4.89	2.9899
	NC Group	
Animal words (Condition 1)	6.59875	4.72931
Two Vowel words (Condition 2)	2.2271	2.5284

[a]Values are Microvolts.

Figure 8.

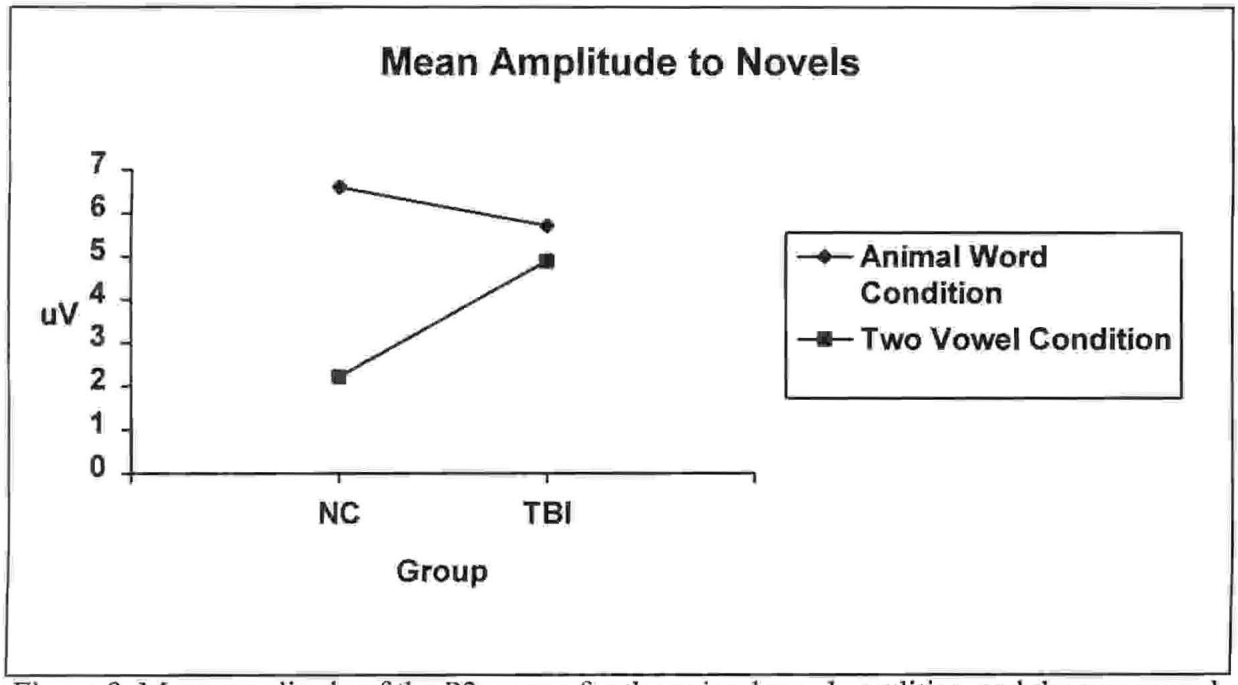

Figure 8. Mean amplitude of the P3a wave for the animal word condition and the two vowel condition for normal control and TBI subjects.

No trends were noted for latency of the P3a component between groups or conditions. Due to the large standard deviations data were analyzed for both the P3b and P3a wave forms with extreme outliers removed. This did not result in the finding of any significant group or condition differences.

Additional Analyses

In order to further investigate the trend observed in our data, a three factor (wave measure X condition X group) multivariate repeated measures analysis of variance was carried out to look at the amplitude and latency of the P200 (P2) component. The P2 is a positive going mesogenous component occurring approximately 200 msec post-stimulus

and is thought to reflect stimulus discrimination. There is evidence that the P2 component is reduced in amplitude after TBI (Clark, O'Hanlon, Wright, & Geffen, 1992). MANOVA revealed no significant effects for the P2 wave form in the target detection condition for group, F (1,16) = .14, p = .711 or condition, F (1,16) = 2.44, p = .138. No interaction effects were significant. Similarly, there were no significant interaction effects or main effects for group, F (1,12) = .1.34, p = .269 or condition, F (1,12) = .96, p = .346, for the P2 recorded to the presentation of novels. The trend observed in the P3b and P3a data was not evident for the P2 wave form.

A condition (2) X wave measure (2) X site (3) by levels of group (2) multivariate repeated measures analysis of variance was also carried out to examine the distribution of the P300 component. There were no significant effects or interactions found. Both groups demonstrated a posterior-anterior gradient for the P3b component to target stimuli for the animal word condition. This gradient was also evident for the two vowel condition in the TBI group. The NC group, however, showed larger amplitude frontally (Fz) than centrally (Cz) with the largest amplitude still occurring at Pz. Although non-significant, this finding supports the trend observed that NC subjects demonstrated differential processing of the two conditions as indicated by amplitude and scalp topography discrepancies, whereas the TBI subjects showed little difference in amplitude or scalp topography between conditions .

P3a responses to novel stimuli produced the largest amplitudes at Cz for the NC group. This is typical of the P3a component which is usually maximal frontocentrally. The TBI group again maintained the same scalp topography as seen in their response to target stimuli (a posterior-anterior gradient).

Two separate MANOVAs (wave measure (2) X target type (novel or target) (2) X group (2)) were carried out for each of the two conditions (animal words and two vowel words) to examine any difference in latency or amplitude between the P3a and P3b component between groups. Results revealed no significant effects.

Due to the small sample size and the large variability evident in the data, I carried out a Mann-Whitney U- Wilcoxon Rank Sum W test which is a nonparametric measure. No measures reached significance; however group differences in P3a amplitude in the two vowel condition approached statistical significance $\underline{U} = 15.5$, $p = .0901$.

Due to the previously noted variability in the TBI sample, five subjects with more severe injury characteristics (i.e., LOC greater than 30 minutes and a period of PTA of two hours or more) were subjected to a repeated measures analysis of variance for P3b to targets and the P3a to novels, to determine if the variability in severity of initial injury in the sample may have precluded the finding of significance. The five NC subjects who were recruited as matches for these TBI subjects were used as the comparison group. Results of these analyses yielded no significant group or condition effects.

Due to the large variability in the data and to the greater difference between standard deviations for each condition observed in the NC group as compared to the TBI group, statistical comparisons were performed on variance differences (Keppel, 1982). P3b amplitude measures for each condition were transformed to absolute deviation scores according to the following formula: Deviation score = group mean - individual score. These deviation scores were analyzed by MANOVA for repeated measures. Results of this analysis did not yield any significant group differences.

Qualitative Investigation

As two subjects with TBI in the sample had documented evidence of damage to the frontal lobes, I conducted a qualitative examination of the amplitude and latency of their P300 data. The subject with a right sided swelling of the frontal lobe documented by CT scan experienced a period of unconsciousness of one week in duration. The interval of PTA was estimated at three to four weeks. This subject evidenced a mean P3b amplitude in the animal word condition of 17.64 μV. This is much larger than the mean of the TBI group in this condition (M=7.4 μV). Similarly, it is much larger than the NC mean for this condition (M=10.7 μV). Latency of the P3b for this subject in the animal word condition was within the range of the TBI group (subject mean = 456 msec; TBI group mean = 451 msec). For the two vowel condition, this subject demonstrated a reduction in amplitude as compared to the animal word condition (5.39 μV). This amplitude is larger than the mean for the NC group in the two vowel condition (M=3.99 μV), but it is smaller than the TBI group mean (M=6.49 μV). Latency of the P3b in the two vowel condition was prolonged in this subject (780 msec). Mean latency for the TBI group was 472 msec and for the NC group mean latency was 465 msec. Data for this subject was only available for the animal word condition for the P3a response to novel stimuli. Again this subject's mean amplitude was larger than the mean amplitude for either of the experimental groups but the difference was not as marked as for the P3b (subject mean amplitude = 7.59 μV; TBI group mean = 5.7 μV; NC group mean = 6.5 μV). Latency of the P3a response was also slowed in comparison to both experimental groups (subject mean latency = 532 msec; TBI group

mean = 425 msec; NC group mean = 369 msec). Also notable is that this subject made a large number of commission errors in the two vowel condition (42).

The other subject with documented damage to the frontal lobes sustained a skull fracture and a contusion of the left frontal lobe was noted on the CT scan. This subject experienced a comparable period of loss of consciousness to the subject discussed above (five days), and a comparable estimated duration of PTA (two - four weeks). However, this subject demonstrated a different pattern of results. Amplitude of the P3b in both the animal word and the two vowel condition was reduced compared to both experimental groups. In the animal word condition this subject demonstrated a mean P3b amplitude of 0.85 μV (see above paragraph for means for the experimental groups). Latency of the P3b in this condition was 432 msec. In the two vowel condition mean amplitude of the P3b was 1.59 μV and mean latency was 344 msec. Data for the P3a response to novel stimuli was available for this subject for both conditions. In the animal word condition amplitude of the P3a was 1.83 μV which is again reduced in comparison to both group means. Latency of the P3a in this condition was 480 msec which is slightly longer than the TBI group mean latency. For the two vowel condition, this subject evidenced a P3a amplitude which was virtually identical to the TBI group mean and larger than the NC group mean (subject mean amplitude = 5.02 μV; TBI group mean = 4.8 μV; NC group mean = 2.2 μV).

Relationship Between Measures

An alpha level of 0.05 was used for all correlations and all correlations presented have p values of 0.05 or less. The relationship between reaction time (RT) and ERP

measures was assessed separately for each group. For the control group these correlations were not significant. For the TBI group mean RT was positively correlated with P3b latency for the animal word condition, $r(9) = 0.8105$. Conversely, RT was negatively correlated with P3b latency for the two vowel condition, $r(10) = -0.6294$. RT was also correlated with mean number of correct responses. Interestingly, both groups demonstrated an inverse relationship between RT and mean correct responses for the two vowel condition: TBI group, $r(10) = -0.7796$; NC group, $r(10) = -0.6977$. Correlations between RT and mean correct responses for the animal word condition were not significant.

Exploratory Analyses

Exploratory correlations were carried out between P3b amplitude and latency for each condition and neuropsychological test scores for both groups separately. No correlations reached significance for the control group. For the TBI group P3b amplitude for the two vowel condition was negatively correlated with three of the CVLT measures and with IQ as measured by the KBit (see Table 8 for correlations).

Table 8.

Correlations Between CVLT and IQ Scores and P3 Amplitude for the TBI Group (Two Vowel Condition)
(*if P<.05, two-tailed)

	CVLT (total)	CVLT (trial 5)	CVLT (Delay)	CVLT (Short Delay)	IQ
P3b amplitude	-.7035*	-.7614*	-.7460*	-.7020*	-.5889*

Correlations were carried out between neuropsychological measures and P3a amplitude and latency for each condition for both groups separately. Two significant correlations were found for the NC group. Latency of the P3a in the animal word condition correlated positively with performance on the BTA, $r(10) = 0.8696$. In addition, amplitude of the P3a for the animal word condition was inversely related to performance on the short delay trial of the CVLT, $r(10) = -0.7113$. No other correlations were significant. For the TBI group, P3a amplitude for the two vowel condition was positively correlated with performance on the fifth trial of the CVLT, $r(10) = 0.6795$, and with performance on the long delay trial of the CVLT, $r(10) = 0.8133$.

Correlations were also carried out between neuropsychological measures and behavioural measures for the experimental tasks (mean reaction time (RT), mean number of correct responses (Correct), and mean number of commission errors (Commissions)) separately for both groups. Correlations for the NC group are provided in Table 9.

Table 9.

Correlations Between Behavioural and Neuropsychological Measures (NC Group)
(*if P<.05, two-tailed)
AW = animal word condition
TV = two vowel condition

	RT (AW)	RT (TV)	Correct (AW)	Correct (TV)	Commissions (AW)	Commissions (TV)
BTA	-.0065	.23	.0307	.2566	-.6243	-.1620[a]
CCC 9secs	-.6360*	-.4363	-.0254	.8112*	.0337	-.4114
CCC 18secs	-.1413	-.1793	.2173	.6729*	-.2702	-.6179[b]
CCC 36secs	-.6483*	-.2891	-.1966	.6917*	-.0078	-.2064
IQ	-.4779	-.3313	.3026	.6229[a]	-.2060	-.7415*

[a]p=.054
[b]p=.057

No correlations were significant between the behavioural measures for the experimental tasks and the CVLT or Stroop measures for the NC group. For the TBI group several correlations reached or approached significance; they are presented in Table 10.

Table 10.

Correlations Between Behavioural and Neuropsychological Measures (TBI Group)
(*if P<.05, two-tailed)
AW = animal word condition
TV = two vowel condition

	RT (AW)	RT (TV)	Correct (AW)	Correct (TV)	Commissions (AW)	Commissions (TV)
BTA	-.3771	-.2742	.8550*	.5206	-.3497	-.5581
CCC 18secs	.3347	.6234[a]	-.0972	-.3914	-.0052	-.0981
CVLT trial 5	-.2457	.1457	.6427*	.3420	-.3127	-.7113*
Stroop (colour)	-.4234	-.5012	.7291*	.4067	-.2396	-.3175
Stroop (cw)	-.6231[a]	-.5748	.7118*	.8512*	-.0628	-.5812
Stroop (diff.)	.5028	.4848	-.4869	-.7964*	-.0249	.4572
IQ	-.3919	.0619	.7106*	.2879	-.3069	-.5662

[a]p=.054

For the TBI group no correlations were significant between experimental behavioural measures and the 9 and 36 second trials of the Consonant Trigrams task. The total score, recall on trial one, and recall on the short and long delay of the CVLT were also not significantly correlated with experimental behavioural measures.

Although the groups were matched for age and education I carried out correlations between these demographic variables and neuropsychological measures, behavioural responses to the experimental tasks, and ERP measures. For the NC group age was related to: number of correct responses on the animal word condition, $r(10) = 0.7185$, performance on trial 5 of the CVLT, $r(10) = 0.6559$, performance on the delay trial of the CVLT, $r(10) = 0.6908$, and latency of the P3b in the two vowel condition, $r(9) = -0.6669$. Correlations for

the TBI group revealed that age was related only to performance on the 18 second trial of the Consonant Trigrams task, $r(10) = 0.6732$. Education was not related to any measures for the TBI group. For the NC group education correlated with performance on the 18 second trial of the Consonant Trigrams task, $r(10) = 0.6353$, and the 9 second trial, $r(10) = 0.6888$, with performance on the 36 second trial approaching significance, $r(10) = 0.6168$, $p = 0.058$. No correlations carried out for P3a measures and these demographic variables were significant for either group.

Due to the possible effects of recovery on performance, time since injury (in months) was correlated with ERP measures, behavioural measures on the experimental task, and neuropsychological measures for the TBI group. Time since injury was found to be inversely related to performance on the first trial of the CVLT, $r(10) = -0.6646$.

DISCUSSION

Several findings in this study differed from the stated hypotheses. The analyses of subjects' performance on neuropsychological measures, performance on the experimental tasks, event-related potentials, and the correlational analyses will be discussed separately.

Neuropsychological Results

No specific hypotheses were advanced with regard to the performance of subjects on neuropsychological tests. Testing was carried out in a descriptive manner in order to provide information about the types of cognitive problems that the subjects with TBI were experiencing. The two groups were matched on the basis of gender, age, and education, and did not differ with respect to IQ; therefore it is unlikely that group differences in performance on neuropsychological measures are due to demographic variables, educational level, or discrepancies in overall cognitive ability between groups.

Similar performance of the two groups on the CVLT measures suggests that the TBI subjects were able to encode, consolidate and retrieve a list of words presented orally as well as the NC subjects when there were no distractions present or extraneous demands made for cognitive resources. When the demands on attention and working memory were increased, TBI subjects performed less well than the NC subjects as evidenced by group differences on the Consonant Trigrams test, the Stroop test, and by group differences which approached significance for performance on the Brief Test of Attention. Intact ability to learn new material has been documented in patients with frontal lobe lesions (Shimamura,

Janowsky, & Squire, 1991), including patients with frontal lobe lesions due to TBI (Levin, Goldstein, Williams, & Eisenberg, 1991), although recall after a delay was impaired in this latter group.

The Consonant Trigrams test provides a measure of working memory under interference. The present findings concur with previous findings that this test is sensitive to the effects of TBI (Stuss et. al, 1985). Deficits on this test have been interpreted in the literature as an increased susceptibility to the effects of interference (Stuss, Kaplan, Benson, Weir, Chiulli, & Sarazin, 1982). This interpretation fits with the data as the TBI subjects in this study performed as well as the NC subjects on a measure of memory with no distractions (CVLT). There are many reports of TBI patients doing well on formal memory testing while having trouble remembering in the "real world" which is often noisy and distracting.

The Stroop test also proved to be sensitive to cognitive deficits in this sample. This test was of particular interest as it requires the active inhibition of an overlearned automatic response. Attentional control is believed necessary to exercise this inhibition. Lezak (1995) reported that patients who do poorly on this task often have difficulty concentrating, and difficulty in warding off distractions. The frontal lobes are believed to exert an inhibitory influence over the rest of the brain (Knight, 1984); therefore, damage to this region could result in decreased performance on the Stroop test. I examined speed on the colour naming trial as well as speed on the interference (colour word) trial. In addition, I looked at a difference score between the two trials to see if TBI subjects are disproportionately slower on the interference trial than normal controls. The TBI subjects were significantly slower on

both timed trials and the cost in speed on the interference trial approached statistical significance, suggesting that there is a trend for TBI subjects in this study to have more difficulty inhibiting the automatic word reading response. The finding of decreased performance of TBI subjects on the interference trial of the Stroop test is supported in the literature (Bohnen, Twinjstra & Jolles, 1992), although studies have also found no significant group differences apart from general slowing (Stuss et. al, 1985).

Group differences on a third measure, the Brief Test of Attention, closely approached statistical significance and deserve mention. The BTA is a test which requires that subjects listen to a series of letters and numbers. They are first required to count only the letters, ignoring the numbers and then to count the numbers and ignore the letters. This is viewed as a task of executive attentional ability (Schretlen, Bobholz, & Brandt, 1989). Counting numbers and ignoring letters requires attentional control to actively ignore one stimuli and attend to the other. It is therefore a task of focused attention with the irrelevant stimuli serving as distractors. Differences on this task may indicate that the TBI group in this study was more distracted by the irrelevant stimuli and therefore performed less well than the normal control group.

The constellation of tests on which TBI subjects performed more poorly than controls have been hypothesized to require top-down, controlled processing to attend to the relevant material. When the material presented to TBI subjects was provided without distractions and requirements of the task were non complex, as in the case of the CVLT, they did as well as the normal group. TBI subjects appear to be impaired in their ability to ignore distracting information and to read quickly while inhibiting an irrelevant response.

These processes fall under the rubric of attentional control, and impairment in this area is suggested. Slowed speed of processing and/or response is also evident by slow performance on the Stroop test (colour and colour word trials) and poor performance on a paced measure such as the BTA.

The neuropsychological battery administered in this study was not comprehensive but was included for the purpose of describing the sample, and therefore firm conclusions cannot be drawn. The findings noted above do serve to confirm the presence of cognitive deficits in this sample. Due to the clear group differences on some of the measures utilized and to the questions raised by the findings as they relate to other variables in the study, hindsight suggests that a more comprehensive battery would have provided valuable information. Measures of initiation such as the Controlled Oral Word Fluency Test and/or Design Fluency may have provided additional information about cognitive deficits in this sample.

Performance on the Experimental Tasks

As predicted, both groups responded more slowly and made significantly more errors in the two vowel condition than in the animal word condition. This confirms the increased difficulty of the two vowel task condition. Also as predicted, TBI subjects performed more slowly in both task conditions. This is consistent with the robust finding of slowed response time in this population (see Van Zomeren & Brouwer, 1994 for a review). Some interpretations which have been proffered for the slowed response time include: a lack of preparation to respond, fatigue, and general cognitive slowing (Deacon-Eliott et. al,

1987). Slowed response time is more evident when TBI subjects perform choice reaction time tasks as opposed to simple reaction time tasks (Van Zomeren, 1981). It has been suggested that this is due to a complexity "effect" (Van Zomeren & Deelman, 1978); an increasing number of stimulus alternatives slows the decision making process down. In this study, the relative increase in reaction time with the increasing task difficulty was not different between TBI subjects and controls. Brouwer obtained similar results in subjects with TBI using a binary choice paradigm (Brouwer in Van Zomeren & Brouwer, 1994). This finding concurs with the model of general cognitive slowing. One would expect a differential increase in reaction time between the easy and difficult conditions between groups if there was something above and beyond general slowing driving the performance difference.

It is evident from the findings that some part of the cognitive activity involved in making a target-nontarget decision and responding is slowed in the TBI group. I am assuming that the slowed response time was not related to any motoric difficulties on the part of the subjects; none were noted or reported. It has been shown that TBI subjects tend to exhibit reaction times similar to controls when engaged in a simple reaction time as opposed to a choice reaction time task, suggesting that their slowness of responding is related to cognitive and not motoric factors (Van Zomeren, 1981). I am assuming that the TBI subjects are similar in this respect to other samples that have been studied. The findings suggest that responding to a task is slowed whether the stimuli are processed in an automatic, parallel fashion, or subjects perform a more controlled, serial search of the stimuli. This statement, however, is presupposing that TBI subjects did process the stimuli

in the animal word condition in an automatic, parallel manner, and used serial search to examine the two vowel stimuli. If automatic processing is deficient in the TBI subjects they may have utilized controlled, serial processing to carry out the task, resulting in slowed responding to an easy task which should have placed minimal demands on the attention system. Stuss and colleagues (1989) argued that TBI subjects did process redundant, unnecessary information. The TBI subjects in this study may not have used the fastest and most efficient strategy to process stimuli. Such utilization of controlled, capacity limited processing for information which could be processed in an automatic manner, could be due to inefficient allocation of attentional resources or to inflexibility in cognitive strategy. Presently however, there is not enough evidence to argue in favour of this hypothesis over the hypothesis of general cognitive slowing.

The data may have yielded more conclusive results regarding deficits of attentional control if I had utilized a task which involved response inhibition, such as the Stroop task. However, constraints were placed upon the task because of the recording of ERPs. ERPs are obscured by any extraneous artifact such as muscular activity, tension or vocalization. Therefore I was limited in my choice of mode of response.

Although the behavioural data, when looked at in isolation, appear to provide additional converging evidence for the finding of general cognitive slowing in the TBI population, when looked at in the context of the ERP data another alternative is suggested. This will be discussed in the next section.

ERP Results

Contrary to my hypotheses there were no differences between the two groups in P3b nor P3a amplitude or latency. I predicted that the NC group would show a difference in the amplitude and latency of the P3b wave form between the two conditions that would not be evident in the TBI group. Although statistical significance was not achieved, a trend in this direction was observed with NC subjects showing a decrease in amplitude of the P3b wave form for the two vowel condition that TBI subjects did not demonstrate. Interestingly, this trend was also evident for the P3a wave; NC subjects again demonstrated reduced amplitude for the two vowel condition that was not evident for TBI subjects.

There are differences in the scalp topograpy of the P300 wave in different task conditions (Courchesne, Hillyard & Galambos, 1975). In this study, both groups demonstrated a posterior-anterior amplitude gradient, with amplitude being largest for the more posterior recording sites, for the animal word condition. This is the typical scalp topography observed when the P3b is measured in a target detection task. Additional support for differential processing of automatic and controlled task demands in the NC group comes from the nonsignificant trend for a change in P3b topography in the two vowel condition. In this condition amplitude was greatest centrally in the NC subject group, while TBI subjects maintained the same posterior-anterior gradient as in the animal word condition. Given the previously described trend toward amplitude differences, the topography data further support potential differences in cognitive processing on these tasks between the groups.

No significant differences were found between groups for the P200 (P2) component. This component was examined due to prior reports that earlier components are sometimes more sensitive to the effects of TBI (Heinze et. al, 1992; Clark et. al, 1992). The groups did not differ in latency and amplitude measures of this component suggesting that both groups were able to discriminate the stimuli and were attending to the task to a similar degree. However, I only looked at P2 as recorded at site Pz. Clark and colleagues (1992) found that P2 was significantly smaller for their TBI sample at site Cz, suggesting that underlying damage reflected in a reduced P2 response may be located frontocentrally and not in parietal areas.

The findings of no significant differences between TBI and NC groups for the P3b component are consistent with those of Clark and colleagues (1992) who found no significant differences in P3b amplitude or latency between TBI and NC subjects. Other studies have reported delayed latency (Heinze et. al, 1992; Papincolaou, et. al, 1984; Cremona-Meteyard & Geffen, 1994), and reduced amplitude of the P3b in subjects with TBI (Baribeau et. al, 1989; Campbell et al., 1986; Rugg, et. al, 1988). However, the qualitative difference in wave forms observed in the study by Heinze and colleagues (1992) was observed in this study. These researchers carried out a complex analysis of several components of the ERP wave form. Interestingly, amplitude differences of the P3 component did not reach statistical significance in their study, although they did find differences in P3 latency.

The P3 is generally regarded as a sensitive indicator of cognitive deficits in TBI (Pratap-Chand, Sinniah & Salem, 1988). In this study the neuropsychological measures

utilized appeared to be more sensitive to the subjects' deficits than the ERP measures. In light of large standard deviations however, it is possible that differences in the ERP measures were obscured. The trend in the data and the qualitative difference in wave forms between groups does warrant discussion. The following is purely speculative and is based on a trend in the data for the amplitude of the NC wave forms to decrease between conditions as compared to TBI subjects, and is not based on a statistically significant finding.

The attenuated P3b which has been found in previous studies has been interpreted by many as a reduction in the cognitive capacity of TBI subjects. In this study, the NC group shows a reduced amplitude of the P3b in the two vowel condition as compared to the animal word condition. The TBI group does not show this; amplitude remains similar in both conditions. Could this mean that the NC group demonstrated a reduced cognitive capacity when engaged in a difficult task? Due to the fact that NC subjects performed near ceiling on both tasks, responded more quickly, and did better than TBI subjects on most neuropsychological measures administered, it seems an unlikely explanation. The answer may lie in examining the factors which affect the amplitude of the P3b. Picton (1992) states that "introspection suggests that the amplitude of the P300 wave varies with the amount of conscious attention paid to a stimulus." Empirically, amplitude demonstrates an inverse relationship with probability of stimulus occurrence. Amplitude also decreases with task difficulty. When adequate attention is paid to a task, amplitude and certainty (confidence) are believed to covary (Picton, 1992). Both groups of subjects may have exhibited larger amplitudes for the animal word condition because it was a relatively easy task and both

groups felt certain that they were responding correctly. For the two vowel condition, it is likely that NC subjects were more confident of their responses than TBI subjects. Therefore, the amplitude of their P3b wave decreased as a function of their increased effort (due to task difficulty) and their continued confidence that they were making the correct response. TBI subjects were less certain, as evidenced by their higher error rates and slower reaction time, and therefore the amplitude of the P3b component reflected this by remaining fairly large.

Why might it be that TBI subjects were less certain of their responses? Consistent with the notion of general cognitive slowing, TBI subjects may not have had time to perceive and encode the stimulus, make a decision, and consider a response in the two second interstimulus interval. They therefore may have been uncertain of the response that they chose. This uncertainty could be due to reduced cognitive capacity leaving TBI subjects unable to attend to as much information at a time as the NC group. This reduced cognitive capacity would result in more time needed to process a given amount of information (Weber, 1990). The nonsignificant amplitude difference could be related to reduced confidence and certainty on the part of the TBI subjects as a result of increased task demands which exceeded their cognitive capacity and/or processing efficiency.

Although general cognitive slowing likely plays a role in the TBI subjects' delayed response time, it is likely not the only factor affecting performance. Consider the very similar latencies of the P3b component in the two groups. Latency of the P3b is believed to be related to task difficulty (Picton, 1992) and stimulus evaluation (McCarthy & Donchin, 1981). According to Roth, Ford and Kopell (1978), latency of the P3b depends on the speed of identification of the stimulus and decision making. If latency does indeed reflect time

taken to evaluate a stimulus, then the TBI subjects in this study are no slower than the NC group in stimulus evaluation. Whatever is slowing down their responding follows the process of stimulus evaluation. If P3b latency does reflect stimulus evaluation I can assume that TBI subjects are able to perceive and encode a stimulus, and decide if it is a member of a particular category as quickly as the NC subjects. What follows these processes is the selection and initiation of a response. Selection and control of a response involves: the intention to respond, the initiation of the response and inhibition of other competing responses, active switching from one response mode to another, and executive regulation of responding (Cohen, 1993). As the task involved only one decision, to respond or not to respond, there was no inhibition of other competing responses or active switching. It is therefore possible that TBI subjects may have been impaired in their intention to respond or in their initiation of a response.

Prior to a response, a response intention forms; this intention reflects the response preparation. As suggested by Deacon-Eliott and colleagues (1987) TBI subjects may be inadequately prepared to respond. This has been investigated electrophysiologically using the contingent negative variation (CNV) with mixed results (see Deacon-Eliott et. al, 1987 for a review). Deacon-Eliott and colleagues (1987) report results which are very intriguing in light of the present findings. Briefly, these authors found that TBI subjects could be classified into subgroups based on their CNV wave form. One group termed "overprocessors" appeared unable to inhibit irrelevant processing. The other group termed "underprocessors" appeared to be ill-prepared to respond. The overprocessing hypothesis is similar to the idea I am advancing that TBI subjects may overprocess information, utilizing

capacity limited resources when they do not need to do so. The underprocessors may have an impairment of response intention and/or initiation.

The frontal lobes may play a critical role in intentional activity (Heilman and Watson, 1991; Stuss & Benson, 1986). The premotor areas of the frontal lobes perform the functions of sequencing, organizing and integrating actions. The supplementary motor area appears to mediate preparatory arousal to action at a preconscious stage in the generation of movement (J.W. Brown in Lezak, 1995). The reciprocal projections of the frontal lobes provide further evidence of their role in intention and initiation of actions. They receive projections from the limbic system, which is believed to play a role in motivation. The frontal lobes project to the striatum which through the thalamic nuclei project back to the frontal areas. Hypokinesia (delay in initiating a response) is only one of a variety of motor problems which may arise following frontal lobe damage. Motor impersistence, defective response inhibition, and motor perseveration have also been reported (Heilman & Watson, 1991). Impairment in response initiation has been reported in patients with frontal, limbic, reticular, and hypothalamic lesion sites (Cohen, 1993). Clinical examples of problems in initiation of behaviour have also been reported in TBI samples (Lezak, 1995; Levin, et al., 1991). A deficit in the ability to form response intentions and/or to initiate behaviour may manifest as a decreased ability to "get going", lowered motivation, decreased initiative and productivity, as well as a reduced rate of emitted behaviours presenting as reduced spontaneity.

The data do not provide strong evidence that an impairment of attentional control resulting in inefficient allocation of resources exists. Unexpectedly, what they do suggest is

that the damage sustained due to TBI in the sample may have affected some aspect of the frontal network of response control. This is manifested as slowed formation of a response intention or slowed initiation. One potential problem for this hypothesis lies in the previously mentioned finding that TBI subjects are no slower than controls in simple reaction time tasks (Van Zomeren, 1981). However, this is likely due to the ease of these tasks. Once the response pattern has been established, the pattern can be left to run as an automatic, low level process. In the experimental tasks, responding could not be automatized as every individual stimulus had to be evaluated and response intentions formed or inhibited. This finding of delayed reaction time compared to controls in the presence of no differences in P3b latency is not new. Other authors have reported similar findings (Clark et. al, 1992); however they did not interpret this finding.

The conclusion that TBI subjects may be slowed in their ability to form a response intention and/or initiate a response is based upon the assumption that P3 latency is truly a measure of stimulus evaluation and decision making. Knight (1990) presents evidence that target detection can occur in the absence of a P300 wave form. Patients with unilateral lesions of the temporoparietal junction were able to correctly perform a target detection task at control levels but produced virtually no P3b. He suggests that P300 generation may not be critical to performance of this task and might be linked to later stages of orientation to and memory registration of the target stimuli. Discussion of these issues is beyond the scope of this paper but the issues are worth mentioning as an alternative explanation for the significant reaction time findings despite nonsignificant differences in P3 latency.

Currently, there are reports in the literature of the P3a component being both enhanced in TBI subjects suggesting distractibility (Baribeau et. al, 1989), and reduced suggesting insufficient monitoring of environmental stimuli (Rugg et. al, 1993). The findings do not support or contradict either of these positions. When comparing my findings with those of Baribeau and colleagues (1989) and Rugg and colleagues (1993) it is important to note that in both of these papers stimuli were presented in the auditory modality. Auditory distractibility and visual distractibility may be two very different constructs. It appeared that TBI subjects in this study were no more distracted by irrelevant visual stimuli than NC subjects. Other studies have found similar results (see Van Zomeren & Brouwer, 1994 for a review). However, the trend of a greater reduction in amplitude of the P3a for NC subjects in the two vowel condition could indicate that they had increased the level of attention paid to the primary task and were no longer attending to the same degree to the novel distractor. The TBI group showed little change in amplitude of the P3a between conditions. This suggests that they did not inhibit attending to the novel distractor, perhaps contributing to their performance decrement in the more difficult condition. There is some evidence that patients with prefrontal lesions do not habituate to irrelevant environmental events (Knight, 1984). If this is the case with the TBI group, then they may not have been able to ignore the novel distractor when task demands increased. Its presentation was still able to evoke a large amplitude P3a. The more demanding nature of this task condition may have required that the NC subjects habituate more quickly to maintain performance levels. The TBI subjects, on the other hand, may not have habituated at all in either task condition. The larger amplitude P3a observed to novels in the animal

word condition for both groups could be reflective of "extra" attentional capacity available due to the automaticity of the task; therefore subjects could "afford" to attend to the novel stimuli without cost to performance.

The prefrontal cortex exerts a net inhibitory influence through thalamic relay nuclei. This inhibitory pathway serves to gate sensory input and allow an individual to focus on a relevant task to the exclusion of irrelevant stimuli. A novel stimulus may be noted initially but then the orienting response habituates. Damage to this area can result in a break down of this inhibitory system which may produce excess distractibility (Knight, 1991).

There are several limitations in this study which may have precluded the finding of significant results. There has been some criticism of the use of peak amplitude as the measure of interest in ERP studies. Unsal and Segalwitz (1995) suggest that peak amplitude of single trials is reduced through averaging and that mean amplitude is actually a better, more sensitive measure. Additionally, trial-to-trial latency, referred to as latency jitter, may also reduce amplitude of the averaged wave form. TBI may lead to variable latency over trials and this may affect the validity of the amplitude of the averaged wave form. It is possible that the measures of amplitude may have been affected by latency jitter. These authors also point out weaknesses in using the baseline to peak amplitude differential as a measure of peak amplitude. They suggest that this measure can be questionable if baseline activity fluctuates from trial to trial randomly. They suggest using a peak-to-trough-to-peak measure. However the majority of the averaged wave forms were based on at least 15 samples and therefore baseline is likely to be relatively stable.

Gevins and Cutillo (1971), suggest that when ERP peak amplitudes are measured with respect to a pre-stimulus baseline, the effects of expectancy need to be considered. This is especially important when interstimulus intervals are constant, as they were in this study. The contingent negative variation (CNV) is a slowly increasing negative potential which occurs between two stimuli typically in tasks involving a contingency or association of paired stimuli (i.e., if ....then...). The CNV is believed to reflect expectancy, preparatory processes, and the build up of cerebral potentiality (Gevins & Cutillo, 1971). Although there was no contingency in this study, subjects may have begun to expect the next stimulus due to the constant interstimulus interval. This may have resulted in increased negativity of the prestimulus baseline, which in turn may have resulted in an artifactual reduction of measures of P3b amplitude.

The study of ERPs is a very complicated endeavour and there are many different components which have been implicated as markers of abnormality in clinical populations. I chose the P300 component due to its long history of research and its apparent relation to attention. Recently the Nd or processing negativity has been receiving attention. The Nd is observed in a dichotic listening task. A subject attends to stimuli delivered in one ear and ignores stimuli delivered to the other. The effect of attending increases the amplitude of the N100 component and the P200 component. The unattended wave form is then subtracted from the attended wave form to produce the Nd. This difference measure has been shown to be significantly different in subjects with TBI (Baribeau et. al, 1989). The TBI group in this study may exhibit differences in brain electrical activity related to attention but I may not have targeted the crucial component. The CNV, as mentioned above, is related to response

preparation. If the subjects do indeed demonstrate an impairment in response initiation or intention, the CNV may have revealed significant differences between groups.

The distractor used in this study (a human face) may have been a relatively weak distracting stimulus as there were no conflicting response tendencies associated with it. TBI subjects may actually be more distractible than NC subjects if the distracting stimulus is more salient. Additionally, I did not record ERPs associated with incorrect responses. This may have shed some light on the issue of response certainty by allowing comparison of the wave forms associated with correct and incorrect responses.

An additional limitation regarding the stimuli used in this study is that the subjects were required to scan the word in the two vowel condition to determine if the word was a target. By requiring subjects to serially search each word, I may have inadvertently introduced additional artifact into the recording of the P3b due to saccadic eye movements. This may have contributed to the loss of data due to artifact.

Aside from technical and design limitations, the sample size in this study although typical of most ERP studies in this area was small. There was a considerable degree of variability in the data as evidenced by large standard deviations which made the finding of significance difficult. Selection of subjects resulted in a fairly heterogeneous group. This was due to a limited availability of willing subjects who satisfied the inclusion/exclusion criteria. The TBI group varied considerably in both the time since injury and in the initial severity of injury. However, other authors did not find statistically significant differences in type or severity of attentional disorders between TBI patients with differing levels of

chronicity and severity (Trexler & Zappala, 1988). The heterogeneity of the TBI group combined with the small sample size is likely the most serious limitation of this study.

Trexler & Zappala (1988) did find that classification of patients on the basis of lesion location produced significant differences on measures of attention. This suggests that the sequelae of TBI may be better investigated if patients are not looked at as a group but are subdivided on the basis of presence or absence of lesions and the location of those lesions. Qualitative analysis of the data of two subjects with documented damage to the frontal lobes concurs with this assertion. As noted in a previous section, the two subjects in this study with CT evidence of frontal damage evidenced lesions with differing lateralization. Examination of ERP data for these subjects revealed that they each presented a very different picture. Lesions to the left frontal lobe are associated with a decrease in verbal fluency (output) (see Lezak, 1995; Heilman & Valenstein, 1993 for a review), and behavioural inhibition (C. Mateer, personal communication, July 18, 1996). Right frontal lobe lesions have been reported to be related to problems in perceptual organization and planning, motor impersistence, constructional deficits, disinhibition, and behavioural excess (Lezak, 1995; C. Mateer, personal communication, July 18, 1996). Qualitiative examination of these two subjects' data were surprisingly consistent with the current understanding of lateralization within the frontal lobes. The subject with damage to the right frontal lobe evidenced generally larger P3 amplitude than either the NC or TBI group mean P3 amplitude. Additionally, this subject was very impulsive as evidenced by a large number of commission errors. These observations of "excess" are consistent with the theory that right frontal damage leads to an enhancement of behaviours. Conversely, the subject with

damage to the left frontal lobe produced P3 wave forms of very small amplitude suggesting a suppression of responding. At the present time it is unclear how the current understanding of behavioural and cognitive differences due to lateralization of frontal lesions relates to diminished or excessive P300 response. This observation is mentioned as further evidence that research into the deficits of TBI would benefit from classification of subjects on the basis of lesion location as opposed to classification on the basis of severity or chronicity of injury. In addition to differences in the localization of functions to the left and right area of the frontal lobes, behavioural distinctions are made based upon lesions of the dorsolateral versus the orbitofrontal regions of the frontal lobes (Heilman & Valenstein, 1993). This underscores the point that lesion location is important in enabling both researchers and clinicians to define the nature of a patient's deficits.

Interpretations of Correlations

No specific hypotheses were advanced as to the direction or size of correlations expected in the behavioural and ERP data. Both groups demonstrated an inverse relationship between RT and correct responses for the two vowel condition. This is a counterintuitive finding as one might expect that longer time taken to evaluate a stimulus would result in increased correct responding. In this sample, faster responders were obtaining more correct responses. No relationship was demonstrated for NC subjects between reaction time and P3b latency, whereas TBI subjects demonstrated an interesting pattern of correlations. For the animal word condition, RT and P3 latency were positively correlated; for the two vowel condition the opposite relationship was observed. There is a

long history in the field of electrophysiology of a debate concerning the nature of the relationship between P3 latency and reaction time (Picton, 1992). It has generally been resolved that P3 latency and reaction time are both related to stimulus evaluation but that reaction time is also related to response selection whereas P3 latency is not (McCarthy & Donchin, 1981). When accuracy is emphasized reaction time tends to occur after P3. When speed is stressed, RT precedes the P300 by about 50 msec (Picton, 1992). As mentioned earlier, latency of the P300 component is believed to reflect the length of time needed to evaluate a stimulus (Donchin & Coles 1988). As faster responding is related to both number of correct responses and longer latency P3b in the two vowel condition, perhaps the longer stimulus evaluation time in the TBI subjects resulted in greater confidence regarding the correct response and therefore faster response time.

Exploratory correlations were carried out between P3b amplitude and latency for each task condition and neuropsychological test scores. As these correlations were carried out in an ad hoc fashion with no particular hypotheses being advanced, their interpretation should be limited. No neuropsychological test measures were related to P3b amplitude or latency for the control group. IQ and three of the CVLT measures examined showed an inverse relationship with P3b amplitude for the two vowel condition in the TBI group. This correlation is interesting as it provides support for the previously stated idea that a reduction in P3b amplitude is due to an increase in certainty. Subjects with poorer performance on neuropsychological measures might be expected to be less certain of their responses to the more difficult task condition and would demonstrate a larger amplitude P3b. There were no statistically significant differences between groups on CVLT measures; in fact this was the

only measure apart from IQ that did not show group differences. Covariation of CVLT scores with P3b amplitude suggests that subjects who remembered more words on the CVLT also demonstrated a smaller P3b amplitude in the two vowel condition, similar to the NC profile. This suggests that TBI subjects with less cognitive impairment were more confident of their responses for the difficult task condition.

Exploratory correlations between neuropsychological measures and P3a amplitude and latency were also carried out. For normal control subjects a positive correlation between performance on the BTA and latency of the P3a component in the animal word condition, and an inverse relationship between P3a amplitude and performance on the short delay trial of the CVLT suggests that the NC group was resistant to distraction. Lower amplitude and longer latency of the P3a component to novel distractors was related to good performance on these two measures of resistance to interference. This relationship was also present for the TBI subjects although it only approached statistical significance. For the TBI group, the CVLT measures were related to amplitude of the P3a component in the two vowel condition. However unlike the relationship observed for the P3b, this relationship was positive suggesting that the more words that subjects recalled on the fifth trial and long delay trial was related to a higher amplitude response to novel stimuli. Looking at this relationship together with the CVLT relationship with the target P3b it seems that better performance on the CVLT was related to smaller amplitude P3bs to targets and larger amplitude P3as to novels.

Neuropsychological measures were also correlated with behavioural measures on the experimental task. It appears that for the NC group those who performed well on the

Consonant Trigrams task made less errors in the two vowel condition. This might suggest that the experimental task is tapping some sort of working memory. Subjects were required to activate a memory of what constituted a vowel (a, e, i, o, u). They had to read a word, searching for matches to the vowels that they had activated in memory and after comparing the letters to the vowels they had to count them, decide if two vowels were present, and then make their response. IQ also correlated with correct responses in this condition and was inversely related to commission errors.

The absence of significant correlations between reaction time and the neuropsychological measures for both groups suggests that speed of responding is not related to performance on the neuropsychological tests. TBI subjects demonstrated a correlation between number of correct responses to the animal word condition and performance on the BTA, fifth trial of the CVLT, interference trial of the Stroop test, and IQ. Performance on the interference trial of the Stroop test was the only measure to correlate with number of correct responses for both task conditions. This suggests that subjects who did better on the experimental tasks were able to focus their attention as evidenced by better performance on the Stroop test. This pattern of correlations suggests, that like the NC group, TBI subjects who did better on neuropsychological measures also exhibited better performance on the experimental tasks.

There are many reports in the literature of changes in the performance of TBI subjects on cognitive measures over time following injury (Van Zomeren & Brouwer, 1994; Clark et. al, 1992; Dikmen & Timken, 1987). Subjects in this study were tested at least eight months post injury and would therefore be considered chronic according to the

classification suggested by Van Zomeren and Brouwer (1994). There were no significant correlations in the data between time since injury and ERP variables. Other authors have found significant correlations between ERPs and time since injury (Clark et. al, 1992). Performance on the first trial of the CVLT was negatively correlated with time since injury suggesting that as TBI subjects "recover" they are better able to perform a short term memory task.

General Implications, Limitations, and Future Directions

The task of the researcher who studies event related potentials is to "interpret the functional significance of a poorly understood wave form in terms of its underlying anatomical origins and its cognitive significance" (Deacon-Eliott et. al, 1987). Using event related potentials, a reaction time task, and neuropsychological measures, I sought to examine the integrity of automatic processes in individuals who have sustained a TBI. I suspected that an impairment of automatic processes may exist or may be the result of inefficient allocation of attentional resources resulting in automatic tasks being processed in a controlled, serial manner. I also investigated the nature of the response of TBI subjects to novel stimuli while engaged in tasks of differing levels of difficulty in order to shed some light on the issue of distractibility in this population. The data did not provide answers to my initial questions but offered some new possibilities to explore.

It appears that the TBI subjects in this study did perform more slowly on an automatic processing task than NC subjects. This could be an indicator of deficient automatic processing mechanisms, inefficient allocation of attentional resources, or general

cognitive slowing. The relative increase in reaction time between the automatic and controlled processing conditions was no different between TBI subjects and NC subjects. In addition, TBI subjects were slower on both timed trials of the Stroop task that were examined. This is consistent with the general cognitive slowing hypothesis of traumatic brain injury.

This hypothesis is entrenched in the TBI literature and has received much support from both empirical reaction time studies and neuropathological findings. When the head is impacted, or thrown back and forth against the skull as is the case in acceleration-deceleration injuries typical of motor vehicle accidents, the axons are stretched and torn. This diffuse and often widespread axonal damage is believed to result in slowed transmission of information within the brain. The cumulative noise hypothesis, which states that following this type of injury the signal-to-noise ratio in the brain decreases, has been advanced as one mechanism of cognitive slowing (Van Zomeren & Brouwer, 1994). Decreased signal strength has also been postulated (Brouwer, 1985 in Van Zomeren & Brouwer, 1994). This theory suggests that the strength of associations between nodes decreases due to loss of axonal tissue resulting in longer access time. It is highly likely that a large component of the difficulties experienced by individuals with TBI is due to cognitive slowing.

More recently, as TBI has been associated with damage to the frontal lobes, researchers have begun to look for deficits in higher order cognitive processes. It is important to discover if these deficits truly exist and to describe them in order to inform rehabilitation strategies. The challenge is to tease these deficits apart from general cognitive

slowing which is most likely present concurrently. This study provided evidence of cognitive deficits in addition to general slowing of processing speed. I examined the difference in cost due to naming colours in the presence of response competition (word reading) on the Stroop task in addition to time taken to name colours for the interference trial. I found group differences which approached statistical significance in cost due to the presence of a conflicting response tendency. This suggests that the TBI group may have been impaired in the attentional control mechanisms of focused attention and/or response inhibition in addition to being slower than normal controls. Group differences on a test which purports to measure executive attentional ability (the BTA) also approached significance. The TBI subjects were also deficient in performing a working memory task in the presence of interference. Findings of a stronger significance level may have been found if I had used subjects where radiological evidence was available to denote the presence or absence of focal lesions and to ascertain lesion location.

Although I was unable to demonstrate differential processing of automatic and controlled processing tasks by NC and TBI subjects using measures of P3b amplitude and latency, I was able to utilize the ERP measures to challenge and augment the theory of general cognitive slowing. TBI subjects in this study appear to be able to evaluate stimuli as *quickly as NC subjects as indexed by similar P3b latencies. Later stages of the response process seem to be deficient resulting in slowed reaction time. I propose that the TBI* subjects in this study were slowed in their ability to form an intention and/or initiate a response. As discussed at length previously, this function is believed to be subserved by the frontal regions. This deficit in the ability to form a response intention and initiate a response

likely presents challenges in the daily lives of survivors of TBI. On the experimental tasks this deficit may present as slowed response time but on a day-to-day basis it may involve problems in initiating activities, problems in carrying out activities quickly, decreased productivity and an apparent lack of motivation.

Future research should employ the CNV paradigm. It may be that the tasks employed to elicit a P300 do not adequately address the attentional control problems of subjects with TBI. The more complex contingencies set up to evoke the CNV may be more sensitive to these deficits. The CNV is also recorded frontally, and may be generated in the frontal lobes (see Stuss & Benson, 1986 for a review). Although I was not able to definitively state that TBI does impair some aspect of automatic processing of stimuli, I did not refute this hypothesis. TBI subjects were slower on an easy task which should not have taxed their capacity. This could be due to general cognitive slowing or to an impairment of automatic processing mechanisms. Further research is necessary to separate these two possibilities.

The present findings add to a growing body of research which documents the presence of deficits in cognitive functions which are believed to be subserved by the frontal lobes in individuals who have sustained a TBI. Subgroups of patients are being identified based on their clinical presentation and lesion site. What seems evident in the literature and from the results of this investigation is that research into the sequelae of TBI will continue to find inconsistent results unless subjects are divided into subgroups based upon their pathological presentation. The qualitative examination of two subjects' data suggest that left-right differences in lesion location should also be taken into account. Salient findings

will continue to be obscured if several subjects within a group differ in lesion location from the rest of the sample. Subjects need to be divided on the basis of clear radiological evidence to consider the confounding effects of diffuse axonal injury and extra-frontal lesions. Some authors suggest the use of metabolic imaging (such as the PET scanner) which may augment morphological lesion evidence (Levin et al., 1991). Studies which can more clearly elucidate the nature of deficits which follow TBI in the presence of no lesion site, lesions to the different regions of the frontal lobes, and/or other circumscribed areas, will more provide more clear information about the types of deficits these patients are experiencing. One way of doing this is to include several subgroups of TBI subjects which are divided on the basis of neuroimaging information and to compare them to each other and to a normal control group.

I utilized the framework of frontal lobe dysfunction in guiding my ideas about the deficits observed in subjects with TBI. I was not able to obtain radiological evidence of lesion location for all of the subjects and frontal damage was only discerned for two subjects. I was forced to work on the assumption that the frontal lobes were most likely injured as they are the most common site of injury (Levin et al., 1987). It is possible that I may have examined a sample of TBI subjects with intact and well functioning frontal lobes. This is unlikely as nine of the ten TBI subjects sustained head injuries in motor vehicle accidents which typically involve the brain being thrown back and forth against the skull due to the impact even if the head is not hit directly. What is most likely is that the sample was mixed with some subjects having lesions in frontal regions with or without concomitant lesions to other areas, whereas other subjects likely evidenced no clear lesion site at all. This

underscores the issue discussed above that carefully controlled studies which utilize neuroimaging techniques to separate this heterogeneous group into different subgroups will provide more clear and definitive results.

This sample consisted of Caucasian subjects from one geographical area. This, and the small sample size limits the generalizability of the findings. Although mentioned previously, it is important to stress the issue of small sample size in light of large variability. This is a significant weakness of this study. Future studies of this type should utilize larger sample sizes to account for the variability inherent in the TBI population and in ERP data.

As mentioned, the finding of possible slowed response intention and initiation in this study may present individuals with TBI with large challenges in daily life. However, this deficit may not be identified on a traditional neuropsychological test battery or may be attributed to general cognitive slowing. As noted by Mateer and Mapou (1996) measures of adaptive functioning in attentionally demanding and ecologically valid contexts should be used in the assessment of cognitive deficits in individuals with TBI. Development of questionnaires and checklists for families and individuals with TBI may provide additional information about the ways that damage to the frontal lobes results in losses of adaptive behaviour. This data would inform researchers and clinicians alike. I am aware of one study which utilized a measure of adaptive functioning in addition to traditional neuropsychological measures and electrophysiological measures (Dywan & Segalowitz, 1996).

On a clinical front, neuropsychologists need to develop tools which are sensitive to these deficits in switching, response inhibition, response intention, initiation, allocation of

resources, processing strategies, and deficits in self-awareness to name a few of the cognitive functions believed to be subserved by the frontal lobes. The frontal lobes have been more resistant than other brain areas to parting with their secrets, therefore neuropsychology has developed fewer measures for assessing the integrity of the frontal lobes. Due to the frequency with which people are injured in motor vehicle and other serious accidents, additional neuropsychological measures sensitive to functions of the frontal lobes are badly needed. Measures which do not rely heavily on speed of processing will best reveal patients' deficits in addition to general cognitive slowing. More accurate identification of the cognitive functions which are lost or limited following specific types of TBI will be useful in the development of specific rehabilitation programs.

Conclusions

The study of traumatic brain injury is both scientifically and clinically lucrative. It provides information about how the brain is organized and interconnected. It also provides information about the resiliency and plasticity of the brain, its capabilities to heal itself or to develop new connections in place of damaged ones. Research provides clinicians with new understandings of their patients' problems and informs rehabilitative strategies.

This study attempted to assess the integrity of automatic processes in TBI subjects. I suspected that automatic processing may be deficient due to inefficient allocation of attentional resources. A second objective was to determine if TBI subjects were more distractible than normal control subjects, or if they were less distractible and were insufficiently monitoring external stimuli. Neuropsychological, reaction time, and

electrophysiological measures were utilized to address these issues. This study provides support for the following conclusions:

1. Individuals who have sustained a traumatic brain injury of moderate severity show evidence of general slowing of cognitive processes;
2. These individuals also exhibit some impairment in attentional control as indicated by poor performance on neuropsychological measures requiring top down controlled processing;
3. They are no slower than normal controls in stimulus evaluation and decision making as evidenced by similar P300 latencies;
4. Neuropsychological measures were more sensitive to the deficits of TBI subjects in this study than the P300 event-related potential.

The second main hypothesis of this study could not be clearly supported or refuted. Analysis of the P3a wave form indicated that TBI subjects were not more or less distractible than NC subjects; however poorer performance on neuropsychological measures of performance under interference suggests that they are more susceptible to distraction.

This study, although unable to definitively answer the specific questions it set out to address, does suggest that the deficits that individuals with TBI experience are in addition to general cognitive slowing. This study also stressed the importance of utilizing subgroups of TBI subjects divided on the basis of lesion location, or on the basis of the presence or absence of a discrete area of damage in future studies. This will help to elucidate the

specific problems that patients with diffuse axonal injury alone, and patients with DAI in the presence of specific lesions experience. This will serve to better inform neuropsychological assessment of these patients as well as rehabilitation strategies.

REFERENCES

Auerbach, S. H. (1986). Neuroanatomical correlates of attention and memory disorders in traumatic brain injury: an application of neurobehavioral subtypes. Journal of Head Trauma Rehabilitation, 1(3), 1-12.

Baddeley, A. (1990). Human memory: Theory and practise. East Sussex, U.K.: Lawrence Erlbaum Associates Ltd.

Bennett, T. (1988). Use of the Halstead-Reitan neuropsychological test battery in the assessment of head injury. Cognitive Rehabilitation, 6(3), 18-24.

Baribeau, J., Ethier, M., & Braun, C. (1989). A neurophysiological assessment of selective attention before and after cognitive remediation in patients with severe closed head injury. Journal of Neurological Rehabilitation, 3(2), 71-92.

Bauer, R. M., Tobias, B., & Valenstein, E. (1993). Amnesic disorders. In K. E. Heilman & E. Valenstein (Eds.), Clinical Neuropsychology (3rd ed.) (pp. 523-602). New York: Oxford University Press.

Bohnen, N., Twijnstra, A. & Jolles, J. (1992). Performance in the Stroop colour word test in relationship to the persistence of symptoms following mild head injury. Acta Neurologica Scandinavica, 85, 116-121.

Broadbent, D. E. (1958). Perception and communication. London: Pergamon.

Brown, J. (1958). Some tests of the decay of immediate memory. Quarterly Journal of Experimental Psychology, 10, 12-21.

Campbell, K., Deacon-Elliot, D., & Proulx, G. (1986). Electrophysiological monitoring of closed head injury. I: Basic principles and techniques (the first of a three part series). Cognitive Rehabilitation, 4(5), 26-32.

Clark, C.R., O'Hanlon, A.P., Wright, M.J., & Geffen, G.M. (1992). Event-related potential measurement of deficits in information processing following moderate to severe closed head injury. Brain Injury, 6(6), 509-520.

Cohen, J. (1988). Statistical power analysis for the behavioural sciences. New York: Academic Press Inc.

Cohen, R. A. (1993). The neuropsychology of attention. New York: Plenum Press.

Courchesne, E., Hillyard, S.A. & Galambos, R. (1975). Stimulus novelty, task relevance and the visual evoked potential in man. Electroencephalography and Clinical Neurophysiology, 39, 131-143.

Craik, F. I. M., & Lockheart, R. S. (1972). Levels of processing: A framework for memory research. Journal of Verbal Learning and Verbal Behavior, 11, 671-684.

Cremona-Meteyard, S. L., Clark, C. R., Wright, M. J. & Geffen, G. M. (1992). Covert orientation of visual attention after closed head injury. Neuropsychologia, 30(2), 123-132.

Damasio, A.R. & Anderson, S.W. (1993). The frontal lobes. In K.M. Heilman & E. Valenstein (Eds.) Clinical neuropsychology, 3rd Ed. (pp. 409-460). Oxford: Oxford University Press.

Deacon-Eliott, D., Campbell, K. B., Braxton Suffield, J., & Proulx, G. B. (1987). Electrophysiological monitoring of closed head injury. III. Cognitive evoked potentials. Cognitive Rehabilitation, 5(3), 12-21.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (1987). The California verbal learning test (CVLT) adult version: Research edition. San Antonio: The Psychological Corporation Harcourt Brace Jovanovich, Inc.

Dikman, S. & Temkin, N. (1987). Determination of the effects of head injury and recovery in behavioral research. In H.S. Levin, J. Grafman, & H.H. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp. 73-88). New York: Oxford University Press.

Donchin, E. (1981). Surprise!....Surprise? Psychophysiology, 18, 498-513.

Donchin, E. & Coles, M.G.H. (1988). Is the P300 component a manifestation of context updating? Behavioral and Brain Sciences, 11, 357-374.

Dywan, J. & Segalowtiz, S.J. (1996) Self- and family ratings of adaptive behavior after traumatic brain injury: Psychometric scores and frontally generated ERPs. Journal of Head Trauma Rehabilitation, 11,(2), 79-95.

Eisenberg, H.M. & Weiner, R.L. (1987). Input variables: How information from acute injury can be used to characterize groups of patients for studies of outcome. In H.S. Levin, J. Grafman, & H. M. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp.13-29). New York: Oxford University Press.

Ford, J. M., Sullivan, E. V., Marsh, L., White, P. M., Lim, K. O., & Pfefferbaum, A. (1994). The relationship between P300 amplitude and regional cerebral grey matter

volumes depends upon the attentional system engaged. Electroencephalography and Clinical Neurophysiology, 90, 214-228.

Foster, J. K., Eskers, G. A., & Stuss, D. T. (1994). The cognitive neuropsychology of attention: A frontal lobe perspective. Cognitive Neuropsychology, 11(2), 133-147.

Geisler, M.W. & Polich, J. (1990). P300 and time of day: Circadian rhythms, food intake, and body temperature. Biological Psychology, 31(2), 117-136.

Gevins, A.S. & Cutillo, B.A. (1971). Signals of Cognition. In A. Remond (Ed.), Handbook of electroencephalography and clinical neurophysiology Vol. 2 (pp.335-379). Amsterdam: Elsevier.

Grafman, J. & Salazar, A. (1987). Methodological considerations relevant to the comparison of recovery from penetrating and closed head injuries. In H.S. Levin, J. Grafman, & H. M. Eisenberg (Eds.), Neurobehavioral recovery from head injury. New York: Oxford University Press.

Gronwall, D. (1984). Advances in the assessment of attention and information processing after head injury. In H. S. Levin, J. Grafman, & H. M. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp. 355-371). New York: Oxford University Press.

Gronwall, D. M. A. & Sampson, H. (1974). The psychological effects of concussion. Auckland, New Zealand: Auckland University Press.

Heilman, K.M. & Watson, R.T. (1991). Intentional motor disorders. In (H.S. Levin, Eisenberg, H.M. & Benton, A.L. (Eds.). Frontal lobe function and dysfunction. (pp. 199-213). New York: Oxford University Press.

Heinze, H. J., Münte, T. F., Gobiet, W., Niemann, H., & Ruff, R. M. (1992). Parallel and serial visual search after closed head injury: electrophysiological evidence for perceptual dysfunctions. Neuropsychologia, 30(6), 495-514.

Jones, C.L. (1992). Recovery from head trauma: A curvilinear process? In C.J. Long & L.K. Ross (Eds.) Handbook of head trauma: Acute care to recovery. (pp. 247-270). New York: Plenum Press.

Kaufman, A.S. & Kaufman, N.L. (1990) The Kaufman Brief Intelligence Test. USA: American Guidance Services, Inc.

Keppel, G. (1987). Design and analysis: A researcher's handbook 2nd Ed. New Jersey: Prentice-Hall, Inc.

Kerns, K. A. & Mateer, C. A. (1996). Walking and Chewing Gum: The Impact of Attentional Capacity on Everyday Activities. In: S.J. Sbordone & C.J. Long (Eds.) Ecological validity of neuropsychological testing (pp. 147-169). Delray Beach, Florida: GR Press/St. Lucie Press.

Knight, R.T. (1984). Decreased response to novel stimuli after prefrontal lesions in man. Electroencephalography and Clinical Neurophysiology, 59, 9-20.

Knight, R.T. (1990). Neural mechanisms of event-related potentials: evidence from human lesion studies. In J.W. Rohrbaugh (Ed.) Event-related brain potentials (pp. 3-18). New York: Oxford University Press.

Knight, R.T. (1991). Evoked potential studies of attention capacity in human frontal lobe lesions. In H.S. Levin, H.M. Eisenberg, & A.L. Benton (Eds.). Frontal lobe function and dysfunction (pp 139-153). New York: Oxford University Press.

Kramer, A. F., & Donchin, E. (1987). Brain potentials as indices of orthographic and phonological interaction during word matching. Journal of Experimental Psychology: Learning, Memory and Cognition, 13(1), 76-86.

Levin, H. S. (1989). Memory deficits after closed head injury. Journal of Clinical and Experimental Neuropsychology, 12(1), 129-153.

Levin, H.S., Amparo, E., Eisenberg, H.M., et. al. (1987). Magnetic resonance imaging and computerized tomography in relation to the neurobehavioral sequellae of mild and moderate head injuries. Journal of Neurosurgery, 66, 706-713.

Levin, H. S., Goldstein, F. C., High, W. M. Jr., & Williams, D. (1988). Automatic and effortful processing after severe closed head injury. Brain and Cognition, 7, 283-297.

Levin, H.S., Goldstein, F.C., Williams, D.H. & Eisenberg, H.M. (1991) The contribution of frontal lobe lesions to the neurobehavioral outcome of closed head injury. In (H.S. Levin, Eisenberg, H.M. & Benton, A.L. (Eds.). Frontal lobe function and dysfunction. (pp. 318-338). New York: Oxford University Press.

Lezak, M.D. (1995). Neuropsychological assessment, 3rd Ed. New York: Oxford University Press.

Luria, A. R. (1973). The working brain: An introduction to neuropsychology. New York: Basic Books.

Mateer, C.A. & Mapou, R.L. (1996). Understanding, evaluating, and managing attention disorders following traumatic brain injury. Journal of Head Trauma Rehabilitation, 11(2), 1-16.

McCarthy, G. & Donchin, E. (1980). A metric for thought: A comparison of P300 latency and reaction time. Science, 211, 77-79.

Mirsky, A.F., Anthony, B.J., Duncan, C.C., Ahearn, M.B., & Kellam, S.G. (1991). Analysis of the elements of attention: A neuropsychological approach. Neuropsychology Review,2(2), 109-145.

Moray, N. (1959). Attention in dichotic listening: Affective cues and the influence of instructions. Quarterly Journal of Experimental Psychology, 11, 56-60.

Nissen, M. J.(1986). Neuropsychology of attention and memory. Journal of Head Trauma Rehabilitation, 1(3), 13-21.

Norman, D. A., & Shallice, T. (1986). Attention to action: Willed and automatic control of behavior. In R. J. Davidson, G. E. Schwarts, & D. Shapiro (Eds.), Consciousness and Self Regulation: Advances in Research and Theory, Vol. 4 (pp. 1-18). New York: Plenum Press.

Papincolaou, A. C., Levin, H. S., Eisenberg, H. M., Moore, B. D., Goethe, K. E. & High, W. M., Jr. (1984). Evoked potential correlates of posttraumatic amnesia after closed head injury. Neurosurgery, 14(6), 676-678.

Picton, T. W. (1992). The P300 wave of the human event-related potential. Journal of Clinical Neurophysiology, 9(4), 456-479.

Ponsford, J. & Kinsella, G.(1992). Attentional deficits following closed head injury. Journal of Clinical and Experimental Neuropsychology, 14(5), 822-838.

Pratap-Chand, R., Sinniah, M. & Salem, F.A. (1988). Cognitive evoked potential (P300): a metric for cerebral concussion. Acta Neurologica Scandinavica, 78, 185-189.

Rallo, J.L. (1986). N2-P3 waves associated with unexpected attractive and easily recognizable visual stimuli. In W.C. McCallum, R. Zappoli & F. Denoth (Eds.), Cerebral Psychophysiology: Studies in Event-Related Potentials (EEG Suppl. 38) (pp. 135-137). Elsevier Science Publishers B.V. (Biomedical Division).

Reason, J. T. (1984). Absent-mindedness and cognitive control. In J. E. Harris, & P. E. Morris (Eds.), Everyday memory, actions and absent-mindedness (pp. 113-132). London: Academic Press.

Robaey, P., Breton, F., Dugas, M. & Renault, B. (1992). An event-related potential study of controlled and automatic processes in 6-8 year old boys with attention deficit hyperactivity disorder. Electroencephalography and Clinical Neurophysiology, 82, 330-340.

Robers, R.L., Miller, D.C., & Languis, M.L. (1987). Brain Mapping Utilities Program. Ohio: Biotech Interface Company.

Rosvold, H. E., Mirsky, A. F., Sarason, I., Bransome, E. D. Jr., & Beck, L. H. (1956). A continuous performance test of brain damage. Journal of Consulting Psychology, 20, 343-350.

Roth, W.T., Ford, J.M. & Kopell, B.S. (1978). Long-latency evoked potentials and reaction time. Psychophysiology, 15(1), 17-23.

Rugg, M. D., Cowan, C. P., Nagy, M. E., Milner, A. D., Jacobson, I. & Brooks, D. N. (1988). Event related potentials from closed head injury patients in an auditory "oddball" task: evidence of dysfunction in stimulus categorization. Journal of Neurology, Neurosurgery, and Psychiatry, 51, 691-698.

Rugg, M. D., Pickles, C. D., Potter, D. D., Doyle, M. C., Pentland, B., & Roberts, R. C. (1993). Cognitive brain potentials in a three-stimulus auditory "oddball" task after closed head injury. Neuropsychologia, 31(4), 373- 393.

Russell, E. W., & D'Hollosy, M. E. (1992) Memory and Attention. Journal of Clinical Psychology, 48(4), 530-538.

Schneider, W., & Shiffrin, R. M. (1977). Controlled and automatic human information processing: I. Detection, search and attention. Psychological Review, 84(1), 1-66.

Schretlen, D., Bobholz, J.H., & Brandt, J. (In Press). Development and psychometric properties of the Brief Test of Attention. The Clinical Neuropsychologist.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. Psychological Review, 84(1), 127-190.

Shimamura, A.P., Janowsky, J.S., & Squire, L.E. (1991). What is the role of frontal lobe damage in memory disorders? In (H.S. Levin, Eisenberg, H.M. & Benton, A.L. (Eds.). Frontal lobe function and dysfunction. (pp. 173-195). New York: Oxford University Press.

Sohlberg, M. M., & Mateer, C. A. (1987). Effectiveness of an attention training program. Journal of Clinical and Experimental Neuropsychology, 9, 117-130.

Stuss, D.T. & Benson, D.F. (1986). The frontal lobes. New York: Raven Press.

Stuss, D.T., Ely, P., Hugenholtz, H., Richard, M.T., LaRochelle, S., Poirier, C.A. & Bell, I. (1985). Subtle neuropsychological deficits in patients with good recovery after closed head injury. Neurosurgery, 17(1), 41-47.

Stuss, D.T., Kaplan, E.F., Benson, D.F., Weir, W.S., Chuilli, S., & Sarazin, F.F. (1982). Evidence for the involvement of orbitofrontal cortex in memory functions: An interference effect. Journal of Comparative Physiology and Psychology, 96, 913-925.

Stuss, D. T., Stethem, L. L., Hugenholtz, H., Picton, T., Pivik, J. & Richard, M. T. (1989). Reaction time after head injury: fatigue, divided and focused attention, and consistency of performance. Journal of Neurology, Neurosurgery, and Psychiatry, 52, 742-748.

Stroop, J.R. (1935). Studies of interference in serial verbal reaction. Journal of Experimental Psychology, 18, 643-662.

Squires, N.K., Squires, K.C., & Hillyard, S.A. (1975). Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. Electroencephalography and Clinical Neurophysiology, 42, 387-401.

Tachibana, H., Toda, K., & Sugita, M. (1992). Age-related changes in attended and unattended P3 latency in normal and control subjects. International Journal of Neuroscience, 66, 277-284.

Treisman, A. M. (1964). Selective attention in man. British Medical Bulletin, 20, 12-16.

Trexler, L.E. & Zappala, G. (1988). Neuropathological determinants of acquired attention disorders in traumatic brain injury. Brain and Cognition, 8, 291-302.

Unsal, A. & Segalowitz, S. J. (1995). Sources of P300 attenuation after head injury: Single-trial amplitude, latency jitter, and EEG power. Psychophysiology, 32, 249-256.

Van Zomeren, A.H. & Brouwer, W.H. (1994). Clinical neuropsychology of attention. New York: Oxford University Press.

Van Zomeren, A. H., Brouwer, W. H., & Deelman, B. G. (1984). Attentional deficits: the riddle of selectivity, speed and alertness. In N. Brooks (Ed.), Closed head injury: Psychological, social and family consequences (pp. 74-107). Oxford: Oxford Press.

Van Zomeren, A.H. & Deelman, B.G. (1978). Long-term recovery of visual reaction time after closed head injury. Clinical Neurology and Neurosurgery, 79, 81-90.

Van Zomeren, A. H., & Van Den Berg, W. (1985). Residual complaints of patients two years after severe head injury. Journal of Neurology, Neurosurgery and Psychiatry, 48, 21-28.

Weber, A. M. (1990). A practical clinical approach to understanding and treating attentional problems. Journal of Head Trauma Rehabilitation, 5(1), 73-85.

Williams, P. A., Jones, G. H., Briscoe, M., Thomas, R., & Cronin, P. (1991). P300 and reaction-time measures in senile dementia of the Alzheimer type. British Journal of Psychiatry, 159, 410-414.

Yingling, C. D., & Skinner, J. E. (1975). Regulation of unit activity in nucleus reticularis thalami by the mesencephalic reticular formation and the frontal granular cortex. Electroencephalography and Clinical Neurophysiology, 39, 635-642.

Appendix A

Consent Form Utilized in this Study

UNIVERSITY OF VICTORIA
DEPARTMENT OF PSYCHOLOGY
P.O. BOX 3050, VICTORIA, B.C.
V8W 3P5
TELEPHONE (604) 721-6356 / FACSIMILE: (604) 721-8929

INFORMED CONSENT TO PARTICIPATE IN A RESEARCH PROJECT

I understand that this research project, directed by Dr. Catherine Mateer, is studying the problems with attention that people who have sustained a traumatic brain injury experience. I am aware of the fact that the electrical activity in my brain will be recorded. I understand that participation in this study involves one testing session. During the session I will receive an electroencephalogram (EEG) and I will do some tasks on a computer. I realize that I may observe a very mild skin irritation where the electrodes contact my skin. The EEG will take approximately one hour of my time. Following the EEG I will be administered a series of neuropsychological paper and pencil tests which will provide information about my attention and my memory. This will also take approximately one hour of my time.

I understand that my participation is completely voluntary and that I can withdraw from the study at any time without explanation.

I understand that any data collected in the study will remain confidential; test results will be kept in a locked filing cabinet. Furthermore, I understand that my name will not be attached to any published results, and that my anonymity is guaranteed by using code numbers to identify the results obtained from individual subjects.

I understand that my neuropsychological test battery results will be recorded on paper. It is my choice as to whether they are destroyed after the information has been used or if I wish them to be retained. The data from my electroencephalographic recording will be kept on magnetic tape. This data will be coded and my name will not be attached to it.

I understand that whether I participate or chose not to participate will have no bearing on my medical treatment at the Gorge Road Hospital.

NAME: _____
ADDRESS: _____
TELEPHONE: _____
SIGNATURE: _____
EXPERIMENTER: _____
DATE: _____

FOR RESEARCH PARTICIPANTS UNDER THE AGE OF 19 YEARS ONLY

I am the parent or legal guardian of _____. I have read and understood this document, and provide permission for my son or daughter to participate in this study.

NAME: _____
SIGNATURE: _____

Appendix B

Screening Sheet for TBI Subjects

Name: _____

D.O.B.: _____ Gender: _____

Highest level of education attained: _____

Occupation: _____

Is your first language English? _____ Other languages spoken: _____

Date of Injury: _____

Type of Injury:

_____
_____
_____
_____

GCS: ___ LOC: _____

Previous psychiatric/neurologic condition?

_____
_____
_____

Alcohol consumption: Y/N # of drinks per week: _____

Drug use: Y/N Frequency: _____

Medications:

Appendix C

Screening Sheet for Normal Control Subjects

Name: _____
D.O.B.: _____ Gender: _____

Highest level of education attained: _____
Occupation: _____
Is English your first language? _____ Other languages spoken _____
Do any of the following apply to you?

1. Head trauma with loss of consciousness ___
2. Skull fracture ___
3. Neurologic surgery ___
4. Cerebral Vascular Disease (stroke, TIA, etc.) ___
5. Seizures (epilepsy) ___
6. Migraine headaches >1/month ___
7. Meningitis or encephalitis ___
8. Learning disabilities ___
9. Psychiatric disorders ___
10. Drug use (meds or otherwise) ___
11. Alcohol consumption ___ drinks per week
12. Diabetes ___
13. Other chronic diseases ___
14. Visual impairment (episodic visual probs. or uncorrected vision probs) _
15. Paralysis or sensory loss of any parts of the body ___
16. Other _____

_____

Medications:

Appendix D

Target Words

Animal Word Condition	Two Vowel Condition
horse	pool
wolf	nurse
tiger	pilot
bear	chapel
mouse	ankle
sheep	taxi
zebra	jade
lion	plumber
donkey	ketchup
lamb	nail
monkey	rose
turtle	cave
eagle	barge
buffalo	radish
kitten	spear
snake	garden
deer	cove
goat	glue
turkey	spade
rabbit	lake
skunk	grocer
mule	hail
raccoon	muffin
mole	loaf
falcon	napkin
gazelle	parcel
dove	pecan
dolphin	pearl
parrot	rhubarb
pony	shovel

CURRICULUM VITAE

Surname: Penkman Given Names: Louise Carol

Place of Birth: Billinge, England
Date of Birth: March 27, 1970
Status: Canadian Citizen

Educational Institutions Attended:

University of Victoria	1994 to present
University of Ottawa	1989 to 1993

Degrees Awarded:

B.A. (Honours)	University of Ottawa	1993

Honours and Awards:

Natural Sciences and Research Council PGS B Fellowship	1996 to 1998
Norma Wilson Graduate Bursary	1995
Annie Greskiw Graduate Award	1995
Vancouver Foundation Medical Services Summer Research Scholarship	1995
Royal Ottawa Hospital Ken Vardigan Bursary	1995
University of Ottawa Entrance Scholarship	1989 to 1990

Academic Experience:

Teaching Assistant University of Victoria	1995 to 1996

Partial Copyright License

I hereby the grant the right to lend my thesis to users of the University of Victoria Library, and to make single copies only for such users or in response to a request from the Library of any other university, or similar institution, on its behalf or for one of its users. I further agree that permission for extensive copying of the thesis for scholarly purposes may be granted by me or a member of the University designated by me. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Title of Thesis: Electrophysiological Investigation of Attentional Deficits in Traumatic Brain Injury

Author [redacted]
Louise Carol Penkman

Sept 26/96
Date