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THE EFFECT OF TRAUMATIC BRAIN INJURY ON ATTENTION: STAGES OF INFORMATION PROCESSING AND AUTOMATIC AND CONTROLLED INFORMATION PROCESSING

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THE EFFECT OF TRAUMATIC BRAIN INJURY UPON ATTENTION: STAGES OF INFORMATION PROCESSING AND AUTOMATIC AND CONTROLLED INFORMATION PROCESSING

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ABSTRACT

THE EFFECT OF TRAUMATIC BRAIN INJURY UPON ATTENTION: STAGES OF INFORMATION PROCESSING AND AUTOMATIC AND CONTROLLED INFORMATION PROCESSING

By

James David Thomas

The effects of traumatic brain injury (TBI) on two aspects of information processing were investigated in a test of Van Zomeren's (1981) model of attentional deficits. In Experiment 1, the first three stages of the Sternberg Paradigm were administered to severely injured TBI patients and matched controls. Patients were enrolled in a post-acute day-treatment program. The patients were slower than controls and all task manipulations reached significance. Contrary to the expected results, no group by task-factor interactions were found and this failed to support Van Zomeren's model. These results are interpreted in light of a new model of posttraumatic cognitive reorganization. In Experiment 2, a second sample of TBI patients and matched controls were evaluated for their ability to establish automatic attentional responses after extended practice. A modified Sternberg Paradigm was used. As was expected, control subjects reduced their response times with practice and demonstrated a longer response time when the targets and nontargets were

reversed. In contrast, TBI patients did not come to perform the task more rapidly, suggesting that they failed to develop automatic responses. However, the TBI patients were as slowed as the controls by the target reversal and this suggested that an automatic response had created a response conflict. These results provide only equivocal support for the hypothesis of the study and suggestions for further research are presented.

Dedicated, with all of my love, to Laura and Ethan.

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This research would not have been possible without the gracious assistance of the patients and staff at the Center for Neuropsychological Rehabilitation and their families who volunteered to be subjects. They gave of their time for the promise of better rehabilitative treatments for persons with traumatic brain injury.

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LIST OF ABBREVIATIONS

CHI ..... closed head injury

CM ..... consistent mapping of responses on stimuli
In this task paradigm associated with the research of Schneider and Shiffrin (1977), specific responses are always associated with specific imperative (i.e., target) stimuli

CPT ..... Continuous Performance Test

CRT ..... choice reaction time

CT ..... computerized axial tomography

DAD ..... divided attention deficit

DAI ..... diffuse axonal injury

FAD ..... focused attention deficit

IPT ..... information processing theory

ISI ..... inter-stimulus interval

LCCP ..... limited capacity central processor

MRI ..... magnetic resonance imaging

PTA ..... post-traumatic amnesia

RT ..... reaction time

TBI ..... traumatic brain injury

VM ..... varied mapping of stimuli onto responses
In this task paradigm associated with the research of Schneider and Shiffrin (1977), specific responses are inconsistently associated with the imperative stimuli

INTRODUCTION

In recent years neuropsychologists have found great utility in using attentional constructs to explain the cognitive and emotional problems that can follow traumatic brain injury (TBI). Cognitive deficits that are common in TBI, such as confusion, perplexity, slowed responding, distractibility, disorientation and anterograde amnesia have been described in terms of attentional dysfunction (Lezak, 1978; Mack, 1986; Nissen, 1986; Van Zomeren, Brouwer, & Deelman, 1984). The neurobehavioral syndromes of left hemi-spatial neglect and acute disturbances of mental status have been hypothesized to reflect damage to attentional systems (Heilman, 1979; Karnath, 1988; Mesulam, 1985). Attentional deficits have been related to the poor impulse control, affective dysregulation, and irritability found both in post-traumatic personality changes (Kwentus, Hart, Peck, & Kornstein, 1985; Prigatano, 1987) and the post-concussional syndrome (Klove, 1987). Further, a few studies have proposed that the failure to return to work after TBI is often caused by attentional deficits (Brooks, McKinlay, Symington, Beattie, & Campsie, 1987; Rimel, Giordani, Barth, Boll, & Jane, 1981).

Diffuse damage to cerebral white matter and more focal damage in the frontal and temporal lobes have been identified as common in TBI. This pattern has been demonstrated in experimental studies of simulated human brain injury (Holbourn, 1943), in a primate model of TBI (Ommaya et al., 1971; Ommaya & Gennerelli, 1974), and in human autopsy studies (Adams, et al., 1977; Courville, 1937; Stritch, 1961). Several researchers have suggested that damage to the executive systems and disconnections between processing areas are responsible for the high incidence of attentional deficits following TBI (e.g., Auerbach, 1986; Lezak, 1978; Van Zomeren, 1981, ch. 2). This may explain why subjective complaints such as mental slowness, poor concentration, difficulty with tasks performed simultaneously, and fatigue are among the most commonly reported by TBI survivors (Hinkeldey & Corrigan, 1990; Van Zomeren & Van den Burg, 1985).

Although this suggests that attentional deficits have an important role in producing post-traumatic symptoms, the research supporting this relationship is problematic. The majority of the reports linking attentional dysfunction to symptom formation have been observational and post hoc, not empirical. More importantly, the available reports of clinical experimentation into post-traumatic attentional deficits have produced ambiguous findings.

The present research has been designed to investigate attentional processes in TBI. To this end, several aspects

of attention and attentional dysfunction following TBI will be reviewed. First, an overview of attention definitions will be presented that will consider the selective aspect of attention and its relationship to information processing theory. The effects of TBI on attention and information processing will be reviewed and then two tests of the prevailing theory of post-traumatic attentional deficits will be presented.

An Overview of Attention

Definitions of Attention

Attention can be defined as the set of mental processes that determines the significance of stimuli and limits the prediction of behavior on the basis of stimulus considerations alone (Kahnaman, 1973, p. 2). This broad definition subsumes a large number of more specific processes that have been hypothesized to mediate perception and cognition (Kinchla, 1980). Several of these mechanisms are identified below.

Moray (1969, p. 6) identified seven broad categories of attention. He defined these as (a) mental concentration, described as the ability to focus on one task while ignoring other incoming sensory stimuli; (b) vigilance, described as the detection of events that occur between intervals of no signals; (c) selective attention, described as the receiving of a single source of information when several signals are present; (d) search, described as the situation that occurs when a set of

signals are present and the observer must hunt for some specified target stimulus; (e) activation, described as an individual's preparation to deal with a signal or event; (f) set, described as the preparation to respond in a certain way to a signal; and (g) Neisser's (1967) conception of Analysis by Synthesis, a hypothetical mechanism defined as the active translation of sensory events into neural codes.

Posner and Boies (1971) described three senses of attention that they found in the psychological literature. These were (a) the alertness or the maintenance of optimal sensitivity to the environment, (b) the selection of specific stimuli from the environment, and (c) the limited capacity of cognitive systems.

Lezak (1983, pp. 34-35) identified three types of attention. The first of these, pure attention, she described in terms of two mechanisms, concentration and tracking. The former of these was defined as the ability to select or exclude stimuli from consciousness and the latter as the ability to direct the "train of thought" sequentially. She also described the mechanisms of consciousness, defined as the psychological awareness of the self ranging from full alertness to stupor and activity rate defined as the speed at which mental operations and motor responses could be performed.

Posner and Rafal (1987) discussed attention in terms of (a) a general state of activation or arousal, (b) a

process of selection from competing sources of stimulationfor conscious processing, and (c) a capability to sustain concentration or vigilance over time.

Wood (1986) described three component mechanisms of attention from the perspective of the kinds of deficits that follow brain injury. He described these as (a) capacity defined both as the amount of information that can be processed at a given time and the immediate memory span, (b) visual scanning defined as the ability to attend to the entirety of the visual field, and (c) the focusing and sustaining of attention which were defined as the abilities to attend to important environmental stimuli in response to task set and concentration, respectively.

Given the large number of hypothetical cognitive mechanisms described as attentional, it is difficult to know which are redundant and which are logically necessary for a working definition of attention (Gronwall, 1987). However, two basic classes of attentional processes have been considered to be fundamental and independent (e.g., Kahneman, 1973; Posner & Rafal, 1987). One type has been defined by the constructs of arousal, concentration, and vigilance. Typically these factors have been related to the intensity of response to environmental stimuli and the effort mobilized during task performance. The other class has been termed selective attention and is the focus of the present research.

Varieties of Selective Attention

The selective aspect of attention has a long history and has been used in diverse theoretical approaches. James (1890/1950) stated that attention implied the "withdrawal from some things in order to deal effectively with others" (p. 404). Estes (1950) suggested that learning (defined as the increased probability of a particular response) was dependent upon the sampling of subsets of all available stimuli during acquisition. Discussing the relation of psychological phenomena to electro-encephalographic data, Thatcher and John (1977) described attention as "...the focusing of perceptual mechanisms upon inputs in particular sensory modalities or upon the specific configuration of stimuli which correspond to a unique event in the environment" (p. 91).

Two kinds of selective mechanisms have been proposed (Davis, Jones, & Taylor, 1984). The first of these has been termed focused attention and has been defined as the extent to which a task can be performed in the presence of distracting stimuli (Kahneman & Treisman, 1984). Filtering or gating processes have been hypothesized to limit the interference and the related performance decrement often caused by the presence of irrelevant (i.e., distracting) stimuli (Posner, 1964). The second form of selective attention has been termed divided attention and has been defined as the extent to which two sources of information can be followed or two tasks can be combined without a loss

of performance (Kahneman & Treisman, 1984). Research paradigms in this area typically have required a subject to perform two tasks simultaneously and thus divide cognitive mechanisms between competing activities.

Using any of these definitions for clinical diagnoses requires the links between the definition, the measurement paradigm, and the underlying psychological reality to be formally evaluated through the process of constuct validation (Sanders, 1984). This has not yet been done. An alternative approach involves describing attention in context of the functioning of a larger information processing system. In this manner various forms of attention may be described as epiphenomena of the larger system and not as entities.

Information Processing Theory

Historical Overview

Information processing theory (IPT) may be defined as the set of measurement techniques, paradigms, and theory addressed to the goal of determining and describing the cognitive mechanisms that are active during the performance of various psychological tasks (Townsend & Ashby, 1983, p. 6). Although IPT is a recent invention, it's origins extend back to the middle of the 19th century and the earliest empirical work into attention. These include Bessel's study of individual differences in reaction time (RT) beginning about 1819 (Boring, 1950, pp. 134-153); Helmholtz's report of the relative slowness of neural

transmission using RT methodology in 1850 (Boring, 1950, p. 48; Posner, 1978, pp. 13-14); and Donders' (1868/1969) use of RT to determine the duration of mental processes.

Attention, as measured with RT methodology, continued to be popular until about 1920 when the logical problems of Donders' method and the rise of Behaviorism reduced its acceptance (Brebner & Welford, 1980; Posner, 1978). However, during and soon after World War II there was a revival in attentional research because of the development of human factors engineering and of advancements in decision theory and cybernetics (Townsend & Ashby, 1983, p. 6).

IPT is based in Shannon and Weaver's (1949) discussion of signal integrity in communications systems. Their contribution was two-fold. First, they introduced a method for the quantification of the information in a signal by borrowing concepts from statistical physics and thermodynamics. Second, they described the structural organization of a system or channel that explained the movement of information. These innovations were then incorporated into the study of human cognition (Miller, 1953).

IPT was initially applied within psychology to explain Merkel's 1885 report that choice reaction time (CRT) increased as the number of signal alternatives (stimuli) increased from one to ten (cited in Hick, 1952). At the time that Shannon and Weaver published, it was known that

CRT increased as task difficulty increased; however, there was no theory explaining this phenomena (Laming, 1968, p. 1). Hick (1952) discovered that CRT was positively and monotonically related to the amount of information present in a stimulus array. He proposed that a quantitative relationship existed between the addition of an alternative to a stimulus set and the resulting increase in response time. Crossman (1953) extended this to the situation where the stimuli were not all equally probable and Hyman (1953) did so with the situation where the stimuli were presented under conditions of temporal uncertainty.

Later research demonstrated the limitations of this approach (e.g., Fitts, 1964; Laming, 1968); however, it was this work that caused attention to be conceptualized as the processes that control the processing of information (Posner, 1978). Three classes of IPT models of attention can be found in the literature, each based on a metaphor for the human information processing system (Wickens, 1984).

Information Processing Models of Selective Attention

The oldest of the three models was proposed by Broadbent (1958) and based on the metaphor of a pipeline. He suggested that multiple signals initially enter a large capacity sensory buffer; however, the remainder of the system was hypothesized to have a limited capacity. In order for information to move through the system, it was hypothesized that a large proportion of the incoming

information would be rejected by an active filter located immediately after the sensory buffer. The channel was thus protected from overload. The filter mechanism has been referred to as a bottleneck because it restricts the flow of information through the system (Kahneman, 1973).

Broadbent suggested that the filter could be "tuned" to allow only information having specific attributes to pass and that it functioned in an "all-or-none" fashion. He located the filter at an early stage of the system because research using shadowing determined that people could detect changes in the physical properties of auditory stimuli (e.g., gender of speaker) but not the semantic content (Cherry 1953). The task paradigm called shadowing presents two auditory stimuli dichotically and requires the subject to repeat the information from one ear while ignoring the other ear. If information presented to the unattended ear has not been filtered efficiently, then it may be detected and recalled (Sheer & Schrock, 1986, p. 99).

Broadbent's model stimulated considerable research that was designed to clarify the location and nature of the filter. Broadbent (1958) and Treisman (1964,1969) argued that the filter acted at the level of perceptual processing or pattern analysis: however other research contradicted this conclusion. For example, subjects detected their own name about 50 percent of the time (Moray, 1959) and demonstrated a galvanic skin response to affectively

charged words presented to an unattended ear (Corteen & Wood, 1972). Thus the processing system could extract semantic meaning from an unattended source and the filter would have to be located after the perceptual mechanisms, relatively late in the system (Egeth & Bevan, 1973).

Several researchers demonstrated that the filtering was not wholly explained by Broadbent's conceptualization (e.g., Deutsch & Deutsch, 1963; Treisman, 1960). Further, selection mechanisms not using filtering have been proposed. Norman (1968) proposed that incoming stimuli were analyzed in long term memory and then independently assigned a level of importance that selected stimuli for further processing. Neisser argued that stimuli are neither filtered or ignored, but instead may not be processed by the cognitive system (1967, p. 213). Broadbent's model was abandoned in the early 1970's because these issues could not be resolved (Lambert, 1985).

The metaphor of the single, limited communication channel was replaced by a computer metaphor termed the limited capacity central processor (LCCP). It was hypothesized that many mental operations at all stages of processing would require the LCCP; however, the LCCP could only contribute to one task at a time. The well documented performance decrements seen in tasks performed both simultaneously and in close temporal proximity were hypothesized to be caused by a competition for the LCCP (e.g., Kerr, 1973; Posner & Boise, 1971; Welford, 1967).

The limitation in task performance continued to be conceptualized as a bottleneck; however, it was not tied to a single processing stage.

Several key studies demonstrated the limitations of the LCCP model. Allport, Antonis, and Reynolds (1972) demonstrated that music students could sight-read music while performing a shadowing task, without a decrement in either performance. Likewise, Shaffer (1975) demonstrated that an experienced transcription typist could perform a transcription typing task while simultaneously reciting nursery rhymes or shadowing prose, with only a nominal level of error. This result has not been found with all task pairings (Salthouse, 1986); however, that it could occur at all argues against a unitary processing mechanism.

The most recent model in IPT is the resource allocation model that takes its metaphor from microeconomics. This model proposed that the phenomena typically called attentional result from the investment of processing resources to various tasks in the way that machinery and raw materials are assigned to manufacturing (Kahnaman, 1973; Navon & Gopher 1979; Norman & Bobrow, 1975). As Wickens (1984) has noted, attention can be viewed as a finite commodity that is conceptually equivalent to the sum of the information processing mechanisms that are available at a given time. The nature of the resources and the mechanisms that regulate their

allocation vary between theorists; however, the resource allocation model assumes that resources can be allocated in a graded fashion to any hypothetical stage or cognitive operation. Thus, during the performance of a task some proportion of the available resources are in use while others remain available.

Phenomena attributed to Broadbent's selective filter can be explained by this mechanism (Norman & Bobrow, 1975). For example, during the performance of a shadowing task, a subject could allocate the majority of the available resources to a primary task and the remaining resources could then be used to follow the other message. If the other message did not require substantial resources, perhaps because of a large physical differences in the stimuli, then it would be easily detected. However, if the second message required greater resources then it might never be adequately processed. Thus the bottleneck would be more apparent than real and a filter mechanism would not be required to explain selective attention.

The performance decrements seen in the dual-task paradigm and attributed to divided attention can also be explained by the resource allocation model (Wickens, 1984). Some tasks require more resources and thus either interfere with or prevent the performance of a second task. If all resources were not allocated to the primary task, then a second task could be performed as was

described above (i.e., Allport, Antonis, & Reynolds, 1972; Shaffer, 1975).

The resource allocation model provides a powerful tool in the analysis of attention; however, it cannot explain all attentional phenomena. Two other approaches in IPT include the distinction between automatic and controlled modes of processing and the evidence supporting independent information processing stages. These will be considered below.

Automatic and Controlled Processing

Schneider and Shiffrin (1977) defined controlled processing as the temporary activation and allocation of cognitive resources for the completion of a given task. When two tasks compete for resources, then a performance decrement occurs that has been called a divided attention deficit (DAD). The use of the word "deficit" in this context does not indicate a pathological state and instead refers to the expected and normal performance decrement.

Automatic processing has been defined as the situation where processing resources are allocated to a task or stimulus because of the configuration of the input stimuli and not because of the expenditure of effort or active attentional control (Schneider & Shiffrin, 1977). Unlike controlled processes, automatic processes do not require active processing and are unavoidable once the stimulus configuration has been perceived (Laberge, 1981). Also, automatic processing has been reported to control the

registration in memory of event frequency. Several papers (Hasher & Zacks, 1979, 1984; Zacks, Hasher & Sanft, 1982) have reported that the temporal and spatial locations of stimuli and the frequencies of categories of words can be learned without intention during the performance of other tasks. Schneider and Shiffrin have identified the focused attention deficit (FAD) as arising when an individual knows that a particular stimulus is irrelevant for a given task but cannot ignore that stimulus and the presence of the stimulus impairs task performance. Reason (1984) has indicated that this can occur with errors of both commission and omission.

In a series of experiments employing a multiple frame, visual search paradigm (Schneider & Shiffrin, 1977; Shiffrin & Schneider, 1977), it was found that automatic processes are acquired by high levels of practice when a particular response is consistently paired with a specific stimulus configuration. This has been termed the consistent mapping (CM) of responses onto stimuli. Conversely, varied mapping (VM) reflects the situation where stimuli and responses are changed from trial to trial. VM does not produce automatic processing.

The constructs of controlled and automatic processing enhance the explanatory power of resource allocation theory and help to explain older, related phenomena. These include: (a) the reduction of the attentional demands of a task through habit (James, 1890/1950), (b) the decrease in

response latencies with overlearning (Laberge, 1975), and (c) the equivalence of CRT at different levels of task difficulty after practice (Mowbray & Rhoades, 1959).

Processing Stages and the Sternberg Paradigm

A second aspect of IPT not specifically addressed by resource allocation theory involves the structural organization of the resources that underlie information processing. The most widely accepted model proposes that the hypothetical processing mechanisms are sequentially arranged into a series of stages between stimulus and response (Shallice, 1991; Smith, 1968; Sternberg, 1969b). In theory, the processing of a given stimulus in a CRT task proceeds through an initial perceptual analysis (i.e., an encoding stage), followed by a categorization process, then response selection (i.e., a decision stage) and finally the execution of the appropriate response.

The experimental study of these cognitive mechanisms began with Donders' (1868/1969) characterization of CRT as a composite of the sum of the durations of the individual processing stages. He believed that these stages functioned independently with each stage contributing a unique, constant time to the response latency. He assumed that the stages functioned sequentially, with each stage completing its operation before the following stage was invoked. From these axioms, Donders proposed that the durations of processing stages could yield information about their basic properties, and that these could be

studied by subtractively decomposing CRT. For this purpose he devised the Subtractive Method. A central proposition of this method is that tasks can be devised in order to include or exclude entire processing stages while not altering those stages common to all tasks. This has been called the assumption of pure insertion.

Although the subtractive method guided much of the RT research prior to 1970, it was criticized on both logical and empirical grounds (Laming, 1968; Pachella, 1974; Sternberg, 1969a) and largely rejected. Sternberg (1969a, 1969b) proposed an alternative way of studying information processing stages that he termed the Additive Factors Method. He accepted the concept of independent processing stages; however, he rejected Donders' assumption of pure insertion. Using the logic of analysis of variance, he proposed that the cognitive processes attributed to various stages could be operationalized as separate factors in a multi-factor experiment. For example, the discriminability and number of stimuli could be independently manipulated to influence both stimulus encoding and categorization stages respectively. Sternberg proposed that the lack of a significant statistical interaction between two factors would indicate that the processing mechanisms were additive and thus independent. Conversely, if two factors were found to interact statistically, then they would be expected to influence a common mechanism or stage.

Sternberg (1966, 1969a, 1969b) conducted a number of experiments testing the additive factors method using a visual recognition-memory, binary-choice task, typically manipulating four factors. Subjects were required to hold various numbers of target stimuli in memory (e.g., 1,2 or 4 digits, letters, or designs) and then respond to the presentation of a test stimulus that was either one of the stimuli held in memory or a novel stimulus. The subject was to respond positively if the test stimulus matched a target held in memory. Sternberg (1966) hypothesized that this task involved a serial and exhaustive comparison of the test stimulus to those held in memory, thus the description of the task as memory scanning. Sternberg's four factor model and his operationalizations are presented in Figure 1.

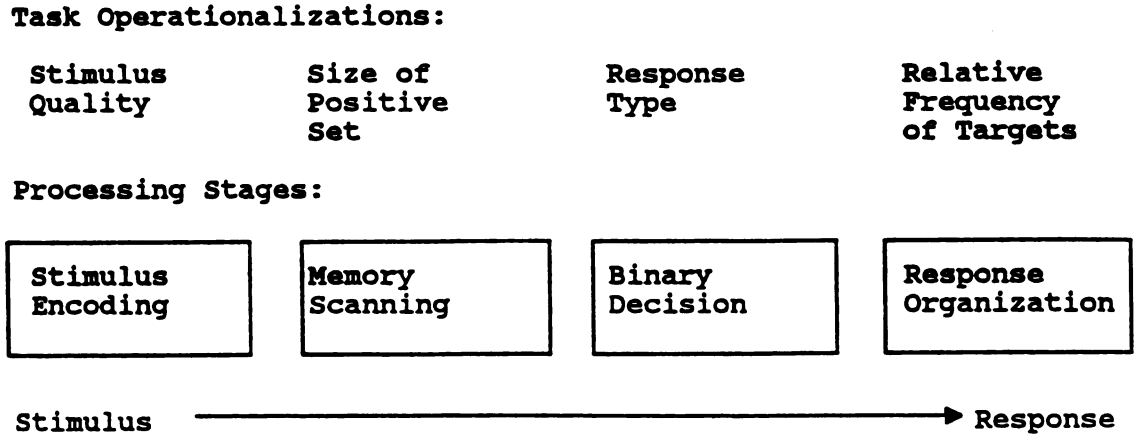


Figure 1. Sternberg's Four Factors and Operationalizations

In addition to manipulating the memory comparison stage by controlling the size of the target set, Sternberg controlled the stimulus encoding stage by presenting the test stimuli in either a highly discriminable or a degraded form. He influenced the binary decision stage by including both target and nontarget stimuli, thus requiring both positive and negative responses. The response organization stage was controlled by varying the relative frequencies of targets and non-targets in the ensembles of test stimuli. Comparing five of the six possible pairs of these factors, Sternberg concluded that each of the factors was independent and thus additive in the context of the 4 stage model.

Sternberg's (1969a, 1969b) primary method of analysis involved comparing pairs of factors by plotting the mean RT's of one factor against the mean RT's of each level of a second factor. If the factors were independent then the slopes would be expected to be parallel. This technique was found to be of special value when the set size was regressed against the other factors. In this situation, the slope of the function represents the speed of the memory scanning process and the y-intercept reflects the speed of the combined stimulus encoding and response processes. Using digits as stimuli, Sternberg found that the slope of the function was linear with a constant increase of about 38 msec. Thus the addition of each new item to the memory set increased response latency by a

fixed amount. The slope did not change when set size was regressed against the three other factors. In contrast, the y-intercept varied as a function of task parameter, with a larger y-intercept produced by the degraded stimuli, the absence of a target (i.e., a negative response), and a lower frequency of targets relative to distractors. The use of these operational definitions in conjunction with the Additive Factors Method has been referred to as the Sternberg Paradigm.

Sternberg's findings have been replicated in a number of studies (e.g., Blackman, 1975; Kristofferson, 1972a; Nickerson, 1966) suggesting a stability for the stages suggested by Sternberg and a generalizability of the Additive Factors Method. Further, memory scanning has been demonstrated in non-human primates (Sands & Wright, 1982) suggesting a biological basis for this cognitive process.

Some research has failed to replicate memory scanning and the additive factors method has been criticized on theoretical grounds (e.g., Pachella, 1974; Pieters, 1983; Townsend & Ashby, 1983, pp. 227-243); however, as Sternberg (1975) noted the additivity of those factors studied has been supported when there has been a speed-accuracy trade-off that favors accuracy and when error rates have been kept below about 10 percent.

Automaticity and Memory Scanning

Although the controlled-automatic processing distinction and the additive factor-memory scanning

paradigms come from separate literatures, there are commonalities. Shiffrin and Schneider (1977) suggested that serial processing and the independence of the stages proposed by Sternberg were examples of controlled processing. They also hypothesized that Sternberg's encoding stage equated to their automatic encoding process.

More importantly they noted that Sternberg's procedures worked against the establishment of automatic processing because of the small number of trials allowed for training and the lack of CM conditions. Sternberg (1975) reported that extended practice on a memory scanning task using a VM condition caused a reduction of the y-intercept but did not change slopes. Other research (Briggs & Blaha, 1969; Kristofferson, 1972b; Ross, 1970) demonstrated that when the target set was held constant over trials (i.e., consistent mapping), then the slope of the regression of memory set size against other factors became negatively accelerated (i.e., curvilinear). Each new stimulus included in the target ensemble added a progressively smaller contribution to the RT, indicating that the stages no longer were independent. These results suggested that practice and the use of a CM procedure produced automatic processing and a reduction in the resources that were required for stimulus processing.

Synthesis

The above discussion describes two major traditions in attentional research. The older sought to identify the types of attention related to various task paradigms; however, these constructs largely represent post-hoc labels that have not been psychometrically validated. The other approach has been derived from information processing theory and describes attention in terms of the allocation of cognitive resources to the performance of tasks. Two constructs postulated to underlie selectivity (i.e., focused and divided attention) can be explained within the resource allocation model; however, other attentional mechanisms cannot be. Among these are the automatic-controlled processing dimension and the independence of processing stages as determined by the additive factors method. Taken together, these hypothetical mechanisms and task paradigms provide a background for an understanding of the current theory of the effects of TBI on attention.

Traumatic Brain Injury and Attention

Historical Overview

The history of research into attentional deficits following TBI can be divided into three periods that roughly correspond to the development of method and theory in neuropsychology. From earliest to most modern these include (a) clinical case reports and anecdotal descriptions of behavior, (b) the use of objective tests in

clinical assessment, and (c) the use of IPT in neuropsychological research.

The best example of the case report method can be found in Meyer's (1904) review of records from the Worchester Insane Hospital. He presented data on 31 patients admitted between 1896 and 1902 who had experienced head trauma. He attempted to relate cerebral damage found at autopsy with various behavioral disturbances and to classify the patient's symptoms (i.e., delirium, post-ictal confusion and post-traumatic psychosis). Although he did not study attentional dysfunction per se, he reported that slowness of thought, distractibility, and fatigability were common. Levin (1991) has reviewed other reports where similar descriptions have been offered.

Much of the early research employing psychometric assessment was conducted on heterogeneous patient samples and thus is not useful in determining the nature of attentional deficit following TBI. The conception of brain damage as a unitary factor was not rejected until the mid-1950's (e.g., Yates, 1954). Thus, the current review will consider the findings of only those studies limited to TBI patients.

The initial study in this period was conducted by Conkey (1938). She used tests supplied by Babcock and Halstead to study the cognitive deficits of 20 TBI patients and their recovery in the first year post injury. She concluded that TBI patients had a limited ability to

sustain effort, fatigued easily, and were less able to shift between tasks. Later, Ruesch (1944a; 1944b) drew similar conclusions using tests of visual RT, visual threshold, dark adaptation, and the tachistoscopic perception of simultaneity. He reported that TBI patients were impaired in sustaining their performance over time, had impaired "visual judgement", and demonstrated a reduced mental speed.

Dencker and Lofving (1958) tested 31 pairs of monozygotic twins, where one twin had suffered TBI and the other was uninjured. Among the 25 dependent variables they collected were several that they related to four attentional factors. These factors were labeled as concentration (e.g., digit span, serial learning); response speed (e.g., verbal fluency, finger tapping, visual and auditory RT); "shifting set" (e.g., mirror reversal drawing) and fatigability (e.g., ten choice, visual RT measured over an extended interval). Using a paired t-test procedure, only four dependent measures were found to discriminate the two groups and only two of these were among the attentional factors. These measures included mirror drawing and the extended CRT. Thus the TBI patients could be seen as being impaired in terms of shifting set and fatigability; however, the construct validity of their factors was not formally evaluated.

These empirical studies are superior to case reports; however, they also have limitations. As Gronwall (1987)

noted, for the most part they are descriptive and thus designed only to show that some tests can measure group differences. Such studies do not provide an interpretative analysis of the essential nature of attentional deficits. The lack of construct validation for hypothetical attentional factors compounds this problem, as does the potential problem with test difficulty. Incorrect conclusions regarding the differential impairment of two cognitive abilities can be caused by administering tests that have different mean item difficulties (Chapman & Chapman, 1973). Thus Dencker and Lofving's (1958) finding of an attentional deficit involving fatigue and not response speed could have resulted from the use of CRT to operationalize the former factor and simple RT for the latter.

Information Processing Studies

Miller (1970) conducted the first study that evaluated TBI related attentional deficits from the standpoint of IPT. He evaluated five severely injured TBI patients and five normal controls matched for age, gender, and occupation with a visual RT task that had one, two, four, or eight stimulus-response pairs. Using Hick's (1952) definition of information, Miller demonstrated that RT increased linearly in both groups as the amount of information (i.e., number of choices) increased and that the TBI patients were slower than controls at all levels of information. He suggested that TBI produced a general

slowing of information processing. He also demonstrated that the TBI group was differentially affected by the amount of information contained in the stimulus array and thus produced a greater slope compared to the controls when RT was plotted against information. Because both regression lines had the same y-intercept, Miller concluded that TBI caused a selective impairment of a central decision mechanism but spared other mechanisms, including sensory registration and response execution.

In a series of studies, Gronwall and Sampson (1974) examined the nature of attentional deficit in TBI from the perspective of Broadbent's (1958) model. They studied 10 normal controls and 20 concussed TBI patients. The patients were divided into severely and mildly injured groups on the basis of the length of their hospital stay. Subjects were given a test of paced mental arithmetic that required them to add pairs of adjacent digits in an aurally presented series of 61 digits. Subjects were given five trials with the inter-stimulus interval (ISI) varied in 0.4 increments between 2.4 to 0.8 seconds. They found that the severely concussed patients performed more poorly than persons in the other groups and that the mildly concussed patients did not differ from the controls. Overall, test performance was a function of ISI where for all groups shorter ISI's produced poorer performances. However, a significant group by ISI interaction demonstrated that the the severely concussed patients performed more poorly at

the longer ISI's than either of the other two groups but gave a similar performance to the other groups at the shorter ISI's.

Gronwall and Sampson (1974) concluded that information processing speed is slowed following severe concussion but also acknowledged two alternative interpretations. They noted that their findings could have resulted from the continuous-paced nature of the task, or could have been caused by a specific impairment of stimulus encoding or of response production mechanisms. These hypotheses were tested with a CRT task that manipulated the amount of information to be processed (i.e., 1, 2, 4, 6, 8, and 10 stimulus-response pairings) and the difficulty of the subjects' decision (i.e., stimulus-response compatibility). Normal controls and mildly concussed patients were tested with both spatially adjacent (i.e., high compatibility) and non-adjacent (low compatibility) stimuli and response buttons. In the high compatibility condition, increasing the number of choices significantly increased CRT but did so equally for the two groups. In the low-compatibility task an interaction between the factors of group and amount of information was found, indicating that the TBI patients were more affected than controls by the increase in the quantity of information when response production was made more difficult. This indicated that pacing per se did not explain the finding of slowed information processing after TBI because the CRT

trials were discreet. Gronwall and Sampson analyzed the regression slopes and intercepts and concluded that the slowing was not related to response production or movement. They hypothesized that the slowing could result from selective impairments in either stimulus classification or decision processes.

The hypothesized perceptual impairment was rejected by the results of two studies. First, Gronwall and Sampson (1974) demonstrated that mildly concussed TBI patients were not significantly impaired relative to controls on a task involving the auditory perception of English sentences masked by white noise (Experiment 3). Second, they demonstrated that TBI patients were not more likely to experience intrusion errors on a two message, shadowing task relative to controls (Experiment 6).

They concluded that the absence of perceptual errors demonstrated that attention deficits after TBI did not arise from an impairment of selective attention, defined both in terms of the filtering of interference and the ability to resist distraction. As noted above, in current usage these two functions would be subsumed under the construct of focused attention, which in turn would be seen as an aspect of selective attention.

The authors hypothesized that the information to be processed and the instructions controlling the manipulation of this information compete for capacity available in the information processing system. They suggested that

concussion in TBI causes a central deficit that results in a reduction in this capacity. They suggested that the reduced capacity resulted from reduced arousal caused by damage to the brain stem reticular formation; however, they presented no supporting data.

Van Zomeren (1981) and his colleagues repeated the research (i.e., Gronwall & Sampson, 1974; Miller, 1970) that demonstrated that TBI patients are more sensitive to task complexity, as defined by the number of choices in a CRT task, than are uninjured controls (Experiment 4.1). They also showed that this effect was greater in persons who sustained more severe injuries (Experiment 4.2 and 5.1) and that it diminished as time elapsed between injury and test (Experiment 4.3 and 5.1).

Van Zomeren presented three hypotheses to explain the effect of task complexity and thus be the cause of attentional dysfunction in TBI. He proposed that TBI could reduce arousal and thus slow the execution of responses. Secondly, he proposed that a single stage of processing could be prolonged by TBI and suggested that stimulus identification and response selection were likely candidates. Finally he suggested that TBI patients could require an extra stage of processing involving a verification of the accuracy of a response that would serve to prolong RT.

He tested the first hypothesis in a study that compared a group of eight severely injured TBI patients

with a group of controls matched on age and gender. TBI patients were tested between 6 weeks and 6 months after injury. Subjects were given both simple RT and 4 choice CRT with an apparatus that allowed response time to be divided into decision and movement components. Decision time was defined as the interval between the onset of target stimulus and the release of a "start" button. Movement time was defined as the interval between the release and the pressing of a response button. The TBI group was found to be slower than the control group on the decision time as task complexity increased; however, this was not found for movement time. In a separate study Van Zomeren (1981, pp. 100-102) demonstrated that EEG slowing (a correlate of reduced arousal) did not differentiate controls and TBI patients during an auditory signal detection task administered over a 30 minute period. With these results Van Zomeren rejected the arousal-response execution hypothesis.

In a test of his second hypothesis, Van Zomeren tested 20 TBI patients and a matched group of uninjured controls (Experiment 6.2a). The patient group had injuries ranging from mild to very severe and were tested between three and 12 months after injury. Movement and decision time were measured with a 4 choice CRT task that presented eight stimulus lights and response buttons to the subject. Subjects were told which four of the eight light-button pairs would be used in each test. After CRT had been

measured, subjects were tested under a distraction condition where one of the four unused stimuli was illuminated simultaneously with the true target. The distraction significantly slowed the decision times for both subject groups; however, a significant interaction between group and distraction was found, indicating that the TBI patients were more affected by the irrelevant stimulus relative to the controls. Analysis of the movement time data indicated that the TBI patients moved more slowly than controls but that this factor was not affected by distraction (i.e., no group by task interaction was found). Van Zomeren concluded that the prolongation of decision time was caused by a response conflict. He reasoned that the subjects' initial task experience without distraction caused them to attempt a response to both the target and irrelevant stimulus when both were presented.

In order to demonstrate that the distraction effect resulted from a response conflict, he repeated the distraction experiment with 12 healthy controls but also introduced a fixed duration warning stimulus on half of the trial blocks. He hypothesized that the warning stimulus would eliminate time uncertainty and reduce RT by facilitating response selection. As before, distraction slowed decision time; however, the warning stimulus also speeded decision time. Neither condition affected movement time. Additionally, a significant interaction was found between the distraction and warning factors, suggesting

that distraction affected decision time by acting on the response selection stage.

Van Zomeren's Interpretation

On the basis of these studies, Van Zomeren and his colleagues (Van Zomeren, 1981; Van Zomeren, Brouwer, and Deelman, 1984) attempted to describe the nature of the attentional deficit that follows TBI in two ways. First, they proposed that TBI caused a generalized slowing of information processing not limited to any particular stage or process. This was characterized as an impairment of divided attention where tasks requiring controlled, capacity-demanding processing would be impaired in TBI patients relative to controls. They noted that real world tasks typically require several simultaneous cognitive processes and that TBI patients would be less able to divide attention (i.e., allocate the necessary resources) because of the generalized slowing. The primary support for this involved the disproportionate effect that information load (i.e., amount of information to be processed) had in slowing the RT of TBI patients. Further, they hypothesized that diffuse axonal damage throughout the white matter was the cause of the impairment. Secondly, they hypothesized that tasks not requiring controlled processing would not be affected by the slowing because they would not be affected by capacity limitations. Thus they stated that focused attention was not impaired after TBI. In support of this conclusion, they reported two

unpublished studies using the Stroop paradigm where distraction failed to impair the performance of TBI patients more than controls. These hypotheses have found wide currency in neuropsychology and rehabilitation and have been widely reported in reviews of TBI (e.g., Cohen, Sparling-Cohen, & O'Donnell, 1993; ch. 11).

Challenges to Van Zomeren's Conclusions

Although the effect of task complexity on RT has been well supported by research, it is difficult to find support for the conclusion that this reflects only deficits of divided attention and excludes focused attention deficits. Challenges can be mounted both on the basis of Van Zomeren's interpretation of his data and from other related TBI research.

First, Van Zomeren's (1981) report that distraction primarily acted on the response selection stage argues against his conclusion that TBI causes a generalized slowing of information processing. His data support his second hypothesis, implicating a specific processing impairment after TBI, not a generalized impairment affecting all stages.

Second, the significant interaction between the factors of distraction and group membership showed that distraction disproportionately slowed the TBI patients. He downplayed the importance of this finding, reporting high correlations between the RT's produced in the distraction and non-distraction conditions. He concluded that

"...distraction obviously introduces no specific attention problems for the patients" (p.123). However, this is a weak argument based on a post-hoc data analysis that contradicts his primary findings.

Third, Van Zomeren's rejection of focused attention deficits in TBI is problematic. He did not directly test either automatic processing or focused attention in TBI patients, basing his conclusion on the finding that the distraction factor interacted with the provision of a warning signal in TBI patients. He noted that his subjects had participated in an earlier RT study and reasoned that the previous learning would have elicited an automatic response that slowed the required controlled processing, if distraction acted on a response process. Unfortunately, he did not report details regarding the number of trials and the mapping of stimuli on responses, and so this interpretation cannot be evaluated. Further, his labeling this situation (if true) a deficit of controlled processing is problematic. Activating an automatic process in the context where it interferes with ongoing behavior, by definition is an FAD (Schneider, Dumais, & Shiffrin, 1984, p. 9). Gronwall (1987, p. 366) offered a similar criticism when she suggested that the response conflicts found in several studies (including Van Zomeren's own) can be interpreted as FAD's.

Finally, several studies have reported deficits in the automatic processing of event frequency in TBI patients

(Levin, Goldstein, High, & Williams, 1988; Tweedy & Vakil, 1988). Because freedom from capacity limitations is a feature of automatic processing and focused attention, this finding contradicts Van Zomeren's conclusion.

More recently, Van Zomeren and Brouwer (1994; chapter 4) selectively reviewed the literature that has accumulated since the publication of Van Zomeren's original monograph (1981). Although they allowed that subacute patients might be more sensitive to interference from dominant response tendencies (i.e., FAD's), they downplayed this possibility and essentially reiterated Van Zomeren's original conclusions. Further, they failed to consider the disconfirming evidence noted above and attributed the generalized processing impairment to neural noise caused by axonal shearing in cerebral white matter.

Attentional Dysfunction and Processing Stages

In response to Van Zomeren, a few studies have explored the impairment of specific information processing stages in TBI patients. Brouwer (1985) attempted to study automatic and controlled processing deficits with a modified memory scanning task with a VM paradigm. His TBI patients were moderately to very severely injured and tested both in the post-acute period of recovery (i.e., within 90 days post-injury) and more chronically (i.e., between 157 and 314 days post-injury). He required TBI and control subjects to hold 1, 2, or 3 digits in short term memory (i.e., target set size). He then presented them

with integer addition problems at three levels of difficulty (i.e., task complexity) and required them to respond positively if the sum matched a digit in the target set and negatively if it did not (i.e., response type). He argued that task complexity should be independent of target set size because simple addition is overlearned and free of capacity limitations (i.e., automatic). Conversely, he predicted a three-way interaction between the factors of group, target set size, and response type because the TBI patients were expected to have an impairment of controlled processing.

Brouwer (1985) found that TBI patients were slowed relative to controls on both test occasions. At the time of the post-acute testing, the TBI patients were more affected by the three task manipulations relative to the controls. During the second test sessions the only differential effect was that of memory load. He found that none of the three-way interactions he had specified were significant on either occasion and he concluded that neither automatic nor controlled processing was impaired by TBI. He concluded that TBI caused a generalized slowing of processing, but did not impair a specific processing stage.

Stokx and Gaillard (1986) conducted four experiments using the Additive Factors Method with TBI patients. The first three of these were designed to test the four stages of processing proposed by Sternberg (1969b). The last was designed to replicate the distraction effect found by Van

Zomeren (1981). Their subjects were sampled from a pool of 13 severely injured TBI patients (coma between 1 and 8 weeks) tested at more than 2 years after injury and normal controls matched on age, gender, and education.

Experiment 1 used a 3 choice CRT task to test the stages of response selection and motor preparation by manipulating stimulus-response compatibility and response uncertainty, respectively. In the former condition the response buttons were either adjacent to the stimulus lights (i.e., high compatibility) or associated with another of the lights in the apparatus (low compatibility). The latter condition offered either a fixed or variable interval between stimuli. They hypothesized that an impairment in either stage would be demonstrated in a group by factor interaction. Stokx and Gaillard found that the three main effects were significant; however, the interactions were not. The patients were thus slower than controls, and both a stimulus-response incompatibility and a variable prestimulus interval produced longer response times.

Stokx and Gaillard (1986) used a binary CRT task to investigate the stimulus encoding stage in their second experiment. They operationalized this stage by presenting pairs of digits in either an intact or degraded format and separated by either 6 or 45 degrees of visual angle. They employed two integers (4 and 5) and required subjects to indicate with a button press whether the pairs were the

same or different. They hypothesized that an impairment of stimulus encoding would be demonstrated through a group by factor interaction. Although all three main effects were found to be significant, no significant interactions were found. However, a trend toward a significant group by stimulus quality interaction was found.

In Experiment 3, they studied the memory scanning stage and response preparation (a factor outside of Sternberg's model) with a binary CRT task. They manipulated both the size of the memory set (i.e., 1, 2, or 4 characters) and the time interval between trials (i.e., ISI). The inclusion of both targets and nontargets allowed a test of the binary decision stage. As before, they expected a stage to demonstrate TBI related impairment when a significant interaction was found between the factor that operationalized the stage and the group membership factor. All main effects were significant; however, only the interaction between group and ISI was significant. Thus, although the TBI patients were slower than controls, they were not impaired either by task complexity or by the processing related to binary decision relative to controls. In contrast to this, the TBI patients required significantly more time to prepare for the next stimulus.

In their fourth experiment, Stokx and Galliard (1986) manipulated both the ISI and the presence of distracting stimuli presented simultaneously in a bimanual, binary choice CRT task. Target stimuli were distinguished from

nontargets and distractors in terms of their spatial location. All main effects were found to be significant but the interactions were not. Thus neither the effect of ISI nor Van Zomeren's distraction effect could be replicated. Stokx and Galliard concluded that none of the factors they studied was implicated in the attentional deficits found after TBI. They did however report a marked heterogeneity in their TBI sample with a few TBI patients demonstrating a clear impairment of stimulus encoding. More importantly, the TBI patients were generally slower than the control sample.

Shum, McFarland, Bain, and Humphreys (1990) criticized Stokx and Gaillard's (1986) research because of the latter's use of multiple two-factor studies. They argued that this approach assumed that processing stages were independent in both controls and TBI patients and that this had not been established. They advocated that all processing stages should be studied simultaneously in a multi-factor experiment. They defined the processing stages of feature extraction, stimulus identification, response selection, and motor adjustment. These were operationalized in terms of stimulus quality (i.e., standard or degraded test stimuli), stimulus discriminability (i.e., closely packed or spatially distinct test stimuli), stimulus-response compatibility (spatially adjacent or nonajacent stimulus-responses pairings), and stimulus uncertainty (i.e., a varied or

fixed interval between the warning and test stimuli). They used a four choice CRT task where an arrow generated on a video screen above a set of response buttons indicated the required response.

A pilot study using uninjured college undergraduates demonstrated that all main effects were significant; however, all of the interactions were nonsignificant. This demonstrated that in control subjects, the manipulations appropriately prolonged the duration of their respective stages and that these stages were independent in accordance with Sternberg's model of information processing.

Shum, McFarland, Bain, and Humphreys (1990) went on to study the equivalence of processing stages in TBI patients and normal controls. They tested three groups of TBI patients selected on the basis of severity of and time since injury. From a pool of 17 normal controls they culled control groups for each patient group, matching on the basis of age, gender, and education. Each pairing of groups was analyzed separately. For the acute, severely injured group, all five main effects were significant as were the interactions of group and both stimulus identification and response selection. For the post-acute (i.e., chronic), severely injured patients the main effects representing the four processing stage factors were significant; however, no significant difference between groups was found. Only the interaction between group membership and response selection was significant. In the

acute, mildly injured group, only the four processing stage factors (i.e., task main effects) attained significance. The factor of group membership was nonsignificant and no interaction was significant.

The authors concluded that their acute, severely injured TBI patients demonstrated a generalized slowing of information processing relative to controls. They also noted that these patients were selectively impaired in the cognitive processes underlying stimulus identification and response selection. However, they noted that this situation changed as patients recovered. In the post-acute phase (greater than one year after injury) the TBI patients were not generally slower than controls, but may have had a greater difficulty selecting appropriate responses. The mildly injured patients did not differ from controls in terms of these processing stages.

Haut, Petros, Frank, and Lamberty (1990) compared 12 TBI patients at least one year post injury and 16 controls matched for age, education, and vocabulary scores on a variant of the Sternberg Paradigm. In this study, severity of injury was not well described; however, unconsciousness was reported as being greater than five days. They manipulated the amount of information to be maintained in short-term memory with three levels of setsize (i.e., 2, 4, and 6 digit stimuli) and the type of response required (i.e., target present versus target absent). The target rate was maintained at 50 percent. All three main effects

were highly significant; however, the more interesting finding was the significant interaction found between the factors of group and setsize. No other interactions were found to be significant. Additionally, the groups were not distinguishable by simple RT. These results corroborated Van Zomeren's work (Experiment 4.1) and the bulk of the earlier RT research in that TBI patients were disproportionately affected by information load.

Schmitter-Edgecombe, Marks, Fahy, and Long (1992) suggested that two patterns of information processing deficits were possible after TBI. They suggested that the deficits could take either the form of a impairment in the latter stages of processing or a generalized information processing deficit affecting all stages. In a test of this hypothesis, they predicted that TBI patients would demonstrate impairments across the task factors of stimulus encoding, memory comparison, and response selection (i.e., from early to late processing). Two studies were conducted comparing a sample of chronic, severely injured TBI patients to an uninjured control group matched for sex, age, education, and household income. The same subjects participated in both studies.

The first experiment was designed to operationalize the encoding and response-selection stages of information processing by manipulating the discriminability of target stimuli and the compatibility of stimuli and responses, respectively. On each trial, subjects were first cued to

either respond in accord with the target stimulus or to give the opposite response. Then they were presented the target stimuli (the words "left" and "rite") in either a standard or degraded form. Subjects were to then respond by pressing a button to the left or the right of midline. As in the earlier studies using RT, the TBI patients were significantly slower than controls and both task manipulations significantly increased RT. Additionally, the two group by task factor interactions were also significant. Thus the TBI patients were more affected by stimulus quality and stimulus-response compatibility than the controls, suggesting that TBI caused deficits in both an early stage of information processing (i.e., stimulus encoding) and a late stage (i.e., decision making).

The second experiment used a modified memory scanning procedure with 3 levels of memory set size (i.e., 1, 2, and 4 digits) and both positive and negative responses (i.e., target presence and absence) with a VM paradigm. Again, the main effects were all significant. More importantly, the only significant interactions were found for group and response type as well as for response type and memory set size.

Schmitter-Edgecombe, Marks, Fahy, and Long (1992) concluded that their hypothesized, generalized information processing deficit could not be supported because of the failure to find a group by memory set size interaction. This meant that TBI patients were no more affected by the

amount of information held in short-term memory than were controls. They speculated that memory-scanning may be relatively unaffected by TBI. They also rejected the hypothesis of a specific deficit in the latter stages of information processing, noting that in their sample both stimulus encoding (i.e., early) and response-execution (i.e., late) processes were impaired.

Synthesis

Information processing studies of attentional dysfunction after TBI provide some insights into the nature of the underlying cognitive impairment. Several studies have suggested that early in recovery, the information processing deficit may be generalized with impairment across cognitive mechanisms (Brouwer, 1985; Gronwall & Sampson, 1974; Van Zomeren, 1981). This is not the most attractive conclusion for several reasons.

First, accepting the existence of a generalized impairment suggests that any test of information processing speed should be diagnostic for TBI; however, it has been shown that simple RT does not always differentiate TBI patients from controls (Van Zomeren, 1981). Second, several studies have shown that TBI patients demonstrate impairments on specific stages of processing which would argue against a generalized dysfunction (Schmitter-Edgecombe, Marks, Fahy, & Long, 1992; Shum, McFarland, Bain, & Humphreys, 1990). Third, some of the reasoning

supporting a generalized information processing disorder is difficult to accept.

As noted above, Gronwall and Sampson (1974) found a specific impairment of stimulus encoding; however, they rejected this conclusion because of negative findings in two studies that used shadowing and filtering paradigms. These tests may not have addressed the same processing mechanisms or may have had different degrees of difficulty (i.e., Chapman & Chapman, 1973). Thus, the negative findings may have been artifactual. In spite of their conclusion of a generalized disorder, Van Zomeren (1981) reported finding a specific impairment in decision mechanisms while Brouwer (1985) reported a specific impairment in the memory comparison process on both testing occasions.

Another major problem involves Van Zomeren, Brouwer, and Deelman's (1984) conclusion regarding the sparing of automatic processing with impairment of controlled processing. The most common finding in this literature is the disproportionate effect of complexity or processing load in TBI patients relative to controls which supports the conclusion that controlled processing is impaired by TBI. However there is little support yet for Van Zomeren's contention that automatic processes are not impaired by TBI. As noted above, the logic supporting this hypothesis is problematic and a direct test of automatic processing

after TBI has not been conducted. The combined influence of these problems makes further research a necessity.

Predictions

The present research was designed to test two aspects of attention that may be impaired after TBI. Experiment 1 examined the hypothesis that TBI causes a generalized information processing disorder but does not impair any specific stage of information processing.

TBI patients and normal controls were compared on the Sternberg Paradigm using a VM procedure to study stimulus encoding, memory comparison, and response selection. In contrast to previous studies, these factors were operationalized in the same manner described by Sternberg (1969a, 1969b).

It was expected that in the normal control group the variables operationalizing the processing stages would produce significant main effects and that all two-way task interactions would be nonsignificant, consistent with Sternberg's original findings. This represents a methods check on the testing paradigm with the task manipulations prolonging RT and these effects being independent.

In the tests of the effects of TBI, it was expected that the TBI patients would be slower than controls on an overall test of group differences and that all three task related factors would produce significant main effects. It was expected that the generalized information processing deficit would be demonstrated by significant group by

task-factor interactions indicating that the TBI patients were more affected at all stages of information processing relative to controls. The other two-way and higher-order interactions were expected to be nonsignificant.

Experiment 2 was designed to investigate the existence of automatic processing deficits following TBI. Using a CM procedure with the Sternberg Paradigm and extended periods of practice, it was expected that normal controls would develop automatic processing to the target stimuli but that this would not occur with TBI patients. Brouwer's (1985) hypothesis regarding the ease of access to memory after TBI suggests that TBI patients would have difficulty establishing automatic responses even after extended practice with consistent mapping of stimuli to responses.

It was anticipated that after the CM training, the TBI patients would fail to show a prolongation of RT when presented with a novel set of targets (i.e., when there was a change in the imperative stimulus and response). It was expected that the uninjured control subjects would demonstrate a prolongation of RT relative to the TBI patients who had not established the automatic response. The prolongation of RT in this context is termed a negative transfer of training. Because RT may also be affected by subject fatigue after extended practice, the control and TBI patient groups were compared in terms of their RT to the CM condition on both the first trial block after two day's training (Block 19) and the last trial block of the

training (Block 30) and also their RT in the memory set reversal condition (Block 31). It was expected that the main effects of group and target block (i.e., standard target set versus reversed target set) would be significant and the interaction would also be significant. Further it was expected that a test of vigilance would not differentiate TBI patients from controls.

In both experiments several variables related to subject characteristics were considered. First, it was expected that age and depression would be positively correlated with RT while education would be unrelated to RT. Second, it was expected that in the TBI groups RT would be positively correlated with several measures of severity of injury (i.e., length of coma and post traumatic amnesia). Recovery (i.e., time since injury) was expected to be inversely correlated with RT.

METHOD

Experiment 1

Subjects

TBI patients. Six TBI patient volunteers were selected from consecutive admissions to the Day Treatment Program of the Center for Neuropsychological Rehabilitation (CNR), Indianapolis, IN. CNR is an independent, out-patient clinic that provides rehabilitation therapies to a variety of post-acute neurologic etiological groups.

Family interviews and a review of hospital records indicated that patients had been rendered unconscious by non-penetrating head trauma for at least one hour (range = 0.3 - 84 days, *M* = 17.7, *SD* = 32.6); had a minimum Glascow Coma Scale (GCS; Teasdale & Jennette, 1974) rating of 8 or less (range = 3 - 4, *M* = 3.17, *SD* = 0.4); and had a post-traumatic amnesia (PTA) duration exceeding one hour (range = 7 - 97 days, *M* = 33.8, *SD* = 35.2). Thus they had experienced moderate to very severe concussion (Russell, 1971). TBI patients were tested between 51 and 286 days after injury (*M* = 176.0, *SD* = 89.3) and thus were in the post-acute phase of recovery. They had been hospitalized because of their trauma and had received traditional inpatient rehabilitation for between 7 and 155 days (*M* =

48.5, SD =54.0). At the time of testing, they had received CNR treatment for between 22 and 198 days (M = 68.0, SD = 67.9).

Exclusion criteria included the following: previous head trauma or other neurological disease, alcohol dependency, premorbid psychiatric treatment, seizures within the prior month and treatment with depressant or stimulant medications. Subjects receiving anti-seizure medication were not excluded from the study; however, because the two most common anti-seizure medications (i.e., carbamazepine and phenytoin) have been shown to increase RT (Gallassi, Morreale, Lorusso, Procaccianti, Lugaresi, & Baruzzi, 1988), the medications and dosage were recorded for later analysis.

The TBI patients had a memory-span of 5 or more digits on the Digit-Span Forwards subtest of the Wechsler Memory Scale-Revised (Wechsler, 1987) because this level of short term memory for digits was necessary for the study (range = 5 - 8, M = 6.2, SD = 1.2). Patients were free from PTA (Russell, 1971) as defined by a T-score on the Galveston Orientation and Amnesia Test (Levin, O'Donnell, & Grossman, 1979) of more than 70. TBI patients were able to comprehend the task instructions, were able to generate a vocal response that would trigger a voice activated switch, and had vision that was correctable to 20 / 20. They were blind to the hypotheses of the study. Written informed consent was obtained from all TBI patients.

Information regarding the injuries sustained by the TBI patients is presented in Table 1. The location of cerebral lesions was identified from computerized axial tomography (CT) reports and from reports of neuropsychological testing conducted both in the hospital and at CNR. These data are presented in Table 2.

Control subjects. Six healthy volunteer control subjects were recruited from the friends and family members of CNR patients, CNR employees, and from the community. They were selected on the basis of their matching the TBI patients on the variables of age (within two years) and gender. Only individuals meeting the above inclusion and exclusion criteria were included in the study. All control subjects were without a history of central nervous system disease and gave written informed consent for their participation in the study. The demographics of the TBI patients and their matched controls are presented in Table 3. An analysis of these data demonstrated that the TBI patients were not different from the controls in terms of age ($t(5) = 0.0$, $p=1$); however, the TBI patients were less educated ($t(5) = 2.9$, $p < .05$).

Apparatus

Environment. Subjects were tested at CNR in an enclosed cubicle. They were seated in an upholstered chair with a computer monitor visible at eye level through a 5 cm by 12.5 cm window in the cubicle wall. Air flow was maintained with a fan vented to the outside room. Subjects

Table 1. Experiment 1: Characteristics of the TBI Patient Sample

Subect	Type of Injury	GCS	Length of Coma	Length of PTA	Time Since Injury	Length of Hosp Rehab	Length of CNR Rehab	Anti-Convlst Dose	GOAT Score	Digit Span
001	MVA	4	3.0	9.0	178	27	35	0	100	8
002	MVA	3	7.0	14.0	261	17	198	0	100	6
003	MVA	3	4.0	23.0	106	45	22	0	100	6
004	Beating	3	0.3	7.0	51	7	29	0	100	5
005	MVA	3	84.0	97.0	286	155	36	0	100	6
006	MVA	3	8.0	53.0	174	40	88	400	100	7

All time intervals are reported in days.
MVA = Motor Vehicle Accident
GCC = lowest Glascow Coma Scale - scores range from 3 (obtunded) to 15 (alert and responsive)
Anti-Convlst Dose = Carbamazepine dose - dose in miligrams per day
GOAT = Galveston Orientation and Amnesia Test - scores range from 0 to 100 (i.e., disoriented to oriented)
Digit Span scores represent raw forward memory span.

Table 2. Experiment 1: Localization of Brain Injury

Subject	CT Narrative	Neuropsychological Testing
001	Multiple medial frontal/ parietal punctuate white matter hemorrhages, multiple frontal contusions, medial right frontal gliding hemorrhage, diffuse white matter edema	Mild to moderate deficits of memory and attention
002	Left frontal hemorrhagic contusion, basilar skull fracture, right orbital and mandibular fractures	Left hemiparesis, visual-spatial deficits
003	Bilateral subdural effusions in the frontal/ temporal regions - worse on the left	Right upper extremity dyscoordination, dysarthria, auditory comprehension deficits, dysgraphia, verbal and visual spatial deficits
004	Left temporal skull fracture, left temporal punctate hyperdensities with a multi-focal distribution suggesting left temporal contusions	Verbal and visual-spatial deficits, verbal and visual-spatial memory moderately to severely impaired
005	Right frontal subdural hygroma, large right hematoma extending from the parietal area to the temporal fossa	Left hemiparesis, diploplia, dysphagia, memory deficits, diffuse deficits across all cognitive domains
006	Bilateral frontal lobe contusions, superimposed on diffuse cerebral injury, small subarachnoid deficits hemorrhage, small amount of blood in the right occipital horn of the ventricle, increased intracranial pressure	Verbal and visual-spatial deficits, Memory and attentional deficits, cerebellar gait disturbance

Table 3. Experiment 1: Demographics of TBI and Control Subjects

	TBI	Control
Gender		
Male	5	5
Female	1	1
Age in Years		
Mean	34.5	34.5
SD	10.1	9.7
Range	20-45	22-47
Years of Education		
Mean	12.0	15.8
SD	2.1	3.0
Range	8-14	12-20

were aurally isolated from environmental noises by white noise (Coulbourn Instruments No. S81-02) presented over headphones.

Computer hardware. The Sternberg Paradigm was administered by an International Business Machines compatible microcomputer operating at 8.0 MHz. This machine presented the stimuli, measured RT, and stored data to magnetic disk. Stimuli were displayed on an RGB monitor

(640 by 200 pixel resolution). This equipment minimized the problems attributed to glare and to after-image persistence as occurs with monochrome displays (Lincoln & Lane, 1980). Subject responses were acquired with a voice activated switch (Coulbourn Instruments No. S28-24) connected to the computer's joyport. As configured, the computer system duplicated the functioning of an electromechanical tachistoscope and reaction time apparatus (e.g., McKeever, 1986).

Computer software. Programs directing the computer's operation were written in a compiled dialect of the BASIC programming language, Turbo Basic (Borland, 1987). Compiling the software allowed it to execute at speeds necessary for RT research (Grice, 1981; Price, 1979) and provided microsecond resolution for the timing routines, allowing an accurate method for measurement and controlling the length of stimulus presentations and delay intervals. The compiler passed tests of accuracy for floating point operations and dynamic range for numeric variables, following the procedures advocated by Miller (1982).

Covariate measures. The Beck Depression Inventory (Beck, 1978) was completed by each subject because past research has disclosed that depressive states can prolong reaction time (Bruder, Yozawitz, Berenhaus, & Sutton, 1980; Martin & Rees, 1966). This measure consisted of 21 sets of four statements describing various aspects of depressive symptomatology. Within each set, the first statement

reflected an absence of depression while the following statements described depressive symptoms of increasing severity. These statements were assigned numeric values on a 4-point ordinal scale (i.e., from 0 to 3). The subject's task was to indicate which of the four statements in each item set most accurately described his or her feelings over the previous week. The score on this measure was the sum of 18 of the items. Three items (14, 15 and 20) were omitted because they reflected inability to work, diminished physical attractiveness, and health concerns.

Because some research has shown that TBI may prolong response time, independent of other cognitive functions (Van Zomeren, 1981, pp. 15-22), simple RT was assessed in all subjects. This was done with the same computer apparatus that was used with the Sternberg Paradigm. Following a warning stimulus (a "plus" sign) and a variable inter-stimulus interval (ISI) between 1 and 3 seconds, an imperative stimulus (a random digit) was displayed in the standard form. On any single trial block, subjects were to respond as quickly as possible, either yes or no to the onset of all imperative stimului.

Sternberg Paradigm. The Sternberg memory scanning task was presented with the computer apparatus using a varied set procedure where responses are inconsistently associated with the digit stimuli. Stimuli consisted of the digits 0 to 9 created by a 7 by 12 matrix of white block graphic characters displayed against a black

background. Stimuli measured 4.8 cm high by 3.8 cm wide and were centrally presented. Subjects sat 84 cm from the monitor which resulted in an extra-foveal stimulus subtending 2 degrees and 35 minutes of visual angle. Digit stimuli could be presented in two possible formats. Standard stimuli were easily read and discriminated. Degraded stimuli consisted of the digits described above masked with an overlay of fourteen block graphic characters. The mask was generated from a random number procedure with the constraint that no more than two masking blocks occur in any one row. Masking characters were white if the underlying square making up the digit was black and was black if the underlying area was white. The digit stimuli are illustrated in Figure 2.

Procedure

Subjects were tested over two days. Before each day's testing, simple RT was measured in two blocks of 20 trials each. The order of the responses required for each trial block was counterbalanced over the two days. Following this the standard and degraded forms of each digit were presented in pairs to familiarize subjects with the degraded forms. Then each of the 10 degraded stimuli was displayed and the subjects were asked to name them. Verbal feedback was offered correcting any errors. This was done until a block of 10 correct responses was made. The sequence of events for a trial of the Sternberg Paradigm was demonstrated and subjects were told that they would be

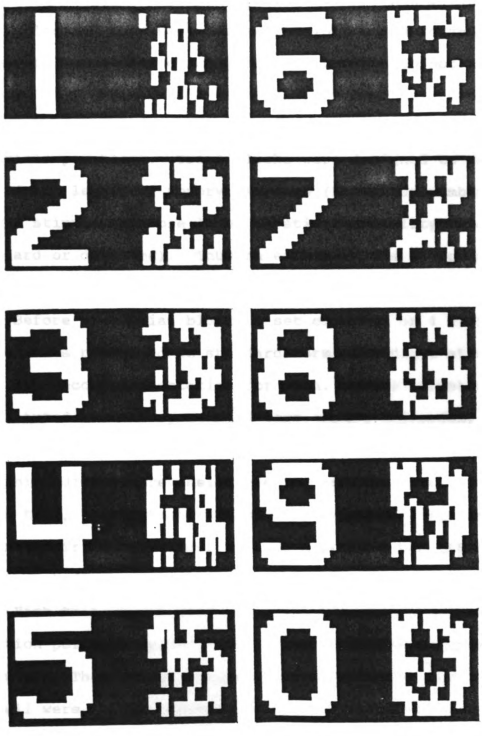


Figure 2. Standard and Degraded Digit Stimuli

tested with both standard and degraded stimuli and that the number of digits in the memory set would change between trial blocks. They were instructed in a standardized manner to respond as quickly as possible but to not make errors.

On each day the subjects received 12 trial blocks with 20 trials per block. Trial blocks were designed to present one of the levels of memory set size (i.e., the number of target stimuli) and one level of stimulus quality (i.e., standard or degraded). Thus on each day, the six possible pairings were presented twice.

Before each trial block, a set of 1, 2, or 4 digit stimuli was presented in standard form, one digit at a time with a 3 second presentation for each. These target digits constituted the memory set and were chosen at random, without replacement from the 10 available digits. The remaining digits were designated the negative set. After this, the subject was asked to name these digits to assure their memorization. If the subject could not name the target digits, then those digits were presented again.

Each trial began with a 1 second presentation of a fixation point (a "plus" sign) which served as a warning stimulus. Then a test stimulus was presented. Test stimuli were either items from the memory set for that trial block or chosen from the digits that comprised the negative set. Half of the test stimuli within a trial block were digits from the memory set and the other half

were digits from the negative set. Thus the rate of positive responses was maintained at a fifty percent level. Digits from the memory set were randomly distributed within a test block. The subject was to say the word yes if the test stimulus was a member of the memory set or say the word no if it was not. The subject's response terminated the trial and the display of the test stimulus. Reaction time was operationalized as the interval between the onset of the test stimulus and subject response. The interval between test trials was standardized at two seconds for all trails. If the subject inadvertently responded prior to the presentation of the test stimulus, the trial was restarted.

The experimenter recorded the subject's response and the occurrence of any situation that could invalidate the data of a particular trial, such as a cough, yawn, or a failure to respond with sufficient loudness to activate the response apparatus. Only incorrect responses were counted as errors. If a subject was unable to consistently correctly respond to a degraded stimulus (suggesting a specific problem with the degraded form of a stimulus) or failed to respond correctly in a consistent manner to a member of the positive set (suggesting a failure of recall of the memory set) then the trial block was aborted. Depending on the difficulty, the stimulus pairs were displayed or the memory set was shown again and then the trial block was repeated. At the end of each trial block,

subjects received feedback on their accuracy, a repetition of the instruction to respond both quickly and without error, as well as reinforcement for their effort.

Data Analysis

The RT data for the twelve trial blocks conducted on the second day's testing were first corrected for invalid trials. Values of less than 100 msec and those that represented either incorrect responses or spoiled trials were excluded from further analysis.

The patient and control groups were compared on the demographic variables, the modified Beck Depression Inventory, simple RT and the error rates found in the Sternberg Paradigm. Median RT's for the Sternberg Paradigm trials were computed for each of the twelve combinations of the three independent variables of memory set size (i.e., 1, 2, and 4), stimulus type (i.e., standard vs degraded), and response type (i.e., positive vs negative). These data were subjected to a 2 x 3 x 2 x 2 repeated measures multivariate analysis of variance (MANOVA) using the Statistical Package for the Social Sciences (SPSS/PC+; Noruiss, 1988). In this analysis group membership (TBI versus control) was the between-subjects factor and the three task factors were the within-group factors. The main effect of setsize and the related interactions were tested with Bartlett's test of sphericity. When this indicated a violation of the assumption of homogeneity of variance-covariance matricies required for the use of the averaged

F test of significance, then the more conservative multivariate test of significance was used (Norusis, 1988). The effect sizes of these tests were computed from the method suggested by Friedman (1968) and converted into Cohen's *f* (1988). Cohen has suggested that a small effect size be defined as *f* equal to 0.10, a medium effect size defined as *f* equal to 0.25, and a large effect size defined as *f* equal to or greater than 0.40.

Error rates were computed for each subject to assess the equivalence of the groups. Finally, correlations between the RT data and the variables related to the injury in the patient group (i.e., PTA, coma length, anti-convulsant medication dosage and time since injury) were computed to evaluate the effect of TBI on the RT data.

Experiment 2

Subjects

TBI patients. A new sample of six volunteer TBI patients was collected from consecutive admissions of CNR patients that met the inclusion and exclusion criteria for Experiment 1. From family interviews and hospital records it was determined that they had been rendered unconscious by non-penetrating head trauma and had minimum Glascow Coma Scale scores below 8 (range = 3 - 4, *M* = 3.2, *SD* = 0.4). They were comatose for between 3 and 63 days (*M* = 25.8, *SD* = 24.5) and had a PTA ranging from 14 to 104 days (*M* = 54, *SD* = 37.9). These data indicate that the patients suffered severe to very severe concussion.

They had been hospitalized for between 21 and 127 days (M = 81.7, SD = 42.9) and had received treatment at CNR for between 8 and 210 days (M = 94.8, SD = 78.9). The average time between injury and testing was 218.5 days (SD = 123.8). All subjects were tested within one year after onset and were thus in the post-acute period of recovery. Written informed consent was obtained from the patients or their parents. A description of these subjects and their injuries is presented in Table 4. Information regarding the localization of their brain injuries is presented in Table 5.

Control subjects. Six healthy volunteer subjects were recruited from CNR employees and the friends and family members of CNR patients and employees. They were selected on the same basis as Experiment 1, met the same inclusion criteria, and gave written informed consent for their participation. The demographics of the TBI patients and their matched controls are presented in Table 6. An analysis of this data disclosed that the groups did not differ in terms of age ($t(5) = 0.83$, $p > .05$) or education ($t(5) = 0.49$, $p > .05$).

Apparatus

The testing environment, computer hardware, and software were maintained from the first to the second study; however, a few modifications to the testing procedure were made.

Table 4. Experiment 2: Characteristics of the TBI Patient Sample

Subect	Type of Injury	GCS	Length of Coma	Length of PTA	Time Since Injury	Length of Hosp Rehab	Length of CNR Rehab	Anti-Convlst Dose	GOAT Score	Digit Span
001	MVA	3	63.0	104.0	302	127	150	0	100	6
002	MVA	3	49.0	14.0	307	117	100	0	100	5
003	MVA	4	3.0	25.0	55	21	10	0	100	7
004	MVA	3	10.0	28.0	190	61	91	400	100	5
005	MVA	3	21.0	93.0	359	112	210	0	98	7
006	MVA	3	9.0	60.0	98	52	8	400	97	7

All time intervals are reported in days.
MVA = Motor Vehicle Accident
GCC = lowest Glascow Coma Scale - scores range from 3 (obtunded) to 15 (alert and responsive)
Anti-Convlst Dose = Carbamazepine dose - dose in miligrams per day
GOAT = Galveston Orientation and Amnesia Test - scores range from 0 to 100 (i.e., disoriented to oriented)
Digit Span scores represent raw forward memory span.

Table 5. Experiment 2: Localization of Brain Injury

Subject	CT Narrative	Neuropsychological Testing
001	Acute brain stem injury, otherwise within normal limits	Left hemiparesis, left neglect, short term memory and attentional deficits, dyspraxia, dysarthria, perceptual deficits
002	Bilateral mandibular fractures, otherwise within normal limits	Left hemiparesis, left neglect, visuo-spatial deficits, mild generalized cognitive deficits, disorganized hypervose language, concrete, dysarthria
003	Depressed nasal fractures, nondepressed left orbital roof fracture, enlarged ventricles bilaterally in the frontal area-greater on the right	Moderate to severe diffuse cognitive deficits, short term memory and problem solving deficits, impaired attention, verbose and disinhibited speech
004	Right temporal lobe contusion with intraparenchymal hemorrhage	Left hemiplegia, verbal memory deficits, dysnomia, attention and arousal deficits, verbal fluency diminished
005	Within normal limits	Dysarthria, dystaxia, visuospatial deficits, verbal and visual spatial memory deficits, diffuse cognitive deficits
006	Bilateral scattered punctuate hemorrhages, intraventricular hemorrhages in the right right lateral and fourth ventricles, hydrocephalus	Short term memory deficits for both verbal and visuo-spatial stimuli, problem solving deficits, impaired abstraction, concrete

Table 6. Experiment 2: Demographics of TBI and Control Subjects

	TBI	Control
Gender		
Male	2	2
Female	4	4
Age in Years		
Mean	28.3	29.0
SD	13.0	12.1
Range	16-44	17-42
Years of Education		
Mean	12.3	12.8
SD	2.9	2.2
Range	10-16	10-16

Covariate measures. Subjects were administered a computerized form of the Continuous Performance Test (CPT) independently of the RT testing. The CPT has been identified as a test of vigilance or sustained attention because subjects are required to respond to a simple repetitive task over a relatively long time period. Software was written to replicate the original task

parameters devised by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956) implementing their two task conditions.

In both of these, 645 letter stimuli were presented sequentially with an 800 msec display duration and a 600 msec interval between stimuli. Stimuli were displayed in the center of a computer screen and responses were measured with a push-button switch wired to the computer game port. Subjects were to respond with their preferred hand; however, when hemiparesis prevented this the unaffected hand was used.

The first task condition (the X condition) required subjects to press the response button when the letter X was displayed and make no response to non-target stimuli. In the second condition (the AX condition), subjects were to respond only when the letter X had been preceded by the letter A. A 20 percent target rate was maintained for both parts with targets randomly distributed through each of the 5 minute blocks comprising the 15 minute test. Nontarget stimuli were randomly selected from the remainder of the alphabet. Before testing began, subjects practiced the paced responding. All subjects were able to perform the tasks. The frequency of correct stimulus detections and false alarms (i.e., responses made in the presence of a distractor) for each part of the test were recorded.

Subjects also completed the Beck Depression Inventory with the same item omissions as used in the first study.

Sternberg Paradigm. The Sternberg Paradigm was administered in the second study with the same task parameters; however, a consistent mapping of responses to stimuli was used. Sternberg (1969a, 1969b) referred to this as the fixed set procedure where the same target stimuli (i.e., the positive set) are used across all trial blocks. The non-target stimuli (i.e., the negative set) are also constant across trials. In the present study, a procedure designed by Kristofferson (1972b) was used. The digits used to initially create the positive set were randomly selected from the available ten digits; however, when less than four target stimuli were to be presented in a given trial block, the target stimuli were counterbalanced so that the four digits would be represented equally throughout the study.

Procedure

In the second experiment subjects were tested with the Sternberg Paradigm over three consecutive days. They were introduced to the stimuli and the task as in the first experiment. Accuracy and speed were equally emphasized. On the first two days of testing, the subjects received nine trial blocks each day, with 40 trials per block. The relative frequency of targets and non-targets was maintained at 50 percent and these were randomly distributed throughout the trials.

On each of the first two days, the subjects were tested at one level of stimulus type (i.e., standard or

degraded) and this was counterbalanced within each subject group. Subjects received three trial blocks of each of the three levels of the memory set size factor (i.e., 1, 2, or 4 items in the target set) on each of these days. The covariate measures were administered after RT data had been collected on the first or second day of testing.

On the third day, subjects received 13 trial blocks of 40 trials with the same four target stimuli that had been assigned to the positive set. In these trials all stimuli were presented in a standard format. Thus over the three days, subjects received 1200 trials using the same set of target stimuli. Then a 31st, and final trial block was administered. This trial block had the same organization as the preceding ones; however, the identity of the targets and non-targets was reversed. The new target set consisted of four digits that had been part of the negative set in all of the preceding trial blocks. All other procedural aspects of data collection were the same as in the first experiment.

Data Analysis

Initially the RT data for the first and last two trial blocks of the third day's testing were corrected for invalid trials as described above and median RT's were computed. The patient and control groups were compared on the demographic variables, the modified Beck Depression Inventory, and the CPT. The RT data were subjected to a 2 x 3 repeated measures MANOVA from the SPSS/PC+ programs,

with the three trial blocks as the within-group factor and group membership (TBI vs control group) as the between-subjects factor. The assumption of homogeneity of the variance-covariance matrices for this analysis were assessed in the same manner as Experiment 1 and effect sizes were computed in the same way. Finally, correlations between the RT data and injury related variables were computed.

RESULTS

Experiment 1

An analysis of the covariate measures demonstrated that the TBI patients did not score differently (M = 5.8, SD = 4.8) than the controls (M = 2.7, SD = 2.9) on the Beck Depression Inventory, $t(5) = -1.5$, $p > .05$, and were not slower in terms of simple RT (M = 508.7, SD = 185.3) than the controls (M = 349.3, SD = 56.0), $t(5) = -1.72$, $p > .05$.

When the groups were compared on the error rates for the Sternberg Paradigm data it was found that the TBI patient group (M = 10.0, SD = 4.6) was not significantly different than the control group (M = 10.2, SD = 5.3), $t(5) = -0.06$, $p > .05$. Further the error rate was at 4% for the TBI patients and 5% for the controls and thus within the acceptable 10% limit for accuracy advocated by Sternberg (1975).

The adequacy of the present computer implementation of the Sternberg Paradigm was evaluated with a 3 x 2 x 2 repeated measures MANOVA conducted with the control group data. As can be seen in Table 7, the three main effects of setsize, stimulus quality, and response type, were all significant. In contrast, the interactions were all

Table 7. Experiment 1: MANOVA for the Control Group

Source	F	df	p<	f
Setsize	46.46	2,10	.000	3.04
Stimulus Quality	30.30	1,5	.003	2.50
Response Type	21.84	1,5	.005	2.07
Setsize x Stimulus Quality	1.03*	2,4	.44	0.68
Setsize x Response Type	1.39*	2,4	.35	0.83
Stimulus Quality x Response Type	0.02	1,5	.90	0.08
Setsize x Stimulus Quality x Response Type	0.13	2,10	.88	0.16

* Sphericity rejected, F estimated from Hotellings' T statistic.

nonsignificant. All of the main effects and two of the three two-way interactions produced large effect sizes according to Cohen's criteria (1988). Thus in uninjured subjects, the computer software provided a measurement paradigm replicating that described by Sternberg (1969a, 1969b). RT was prolonged by each of the three task factors and these factors were structurally independent.

The means of the median RT's for both groups are presented in Table 8. The main analysis of the RT data for Experiment 1 involved a 2 x 3 x 2 x 2 repeated measures MANOVA that is presented in Table 9. This disclosed that, overall the groups were significantly different, with the TBI patients having longer RT's than the controls. Each of the main effects attributable to task factors was highly significant.

Table 8. **Experiment 1: Means and Standard Deviations for the Median RT Data for the Three Task Conditions**

	Size of Target Set		
Stimulus-Response Condition	1	2	4
Control Group			
Standard Stimuli			
Targets Present	466.83 (117.97)	529.25 (97.41)	609.17 (92.90)
Targets Absent	515.33 (105.54)	580.67 (131.54)	716.17 (181.25)
Degraded Stimuli			
Targets Present	587.08 (116.10)	690.08 (169.07)	741.08 (134.19)
Targets Absent	624.92 (118.98)	727.25 (146.82)	859.83 (209.89)
TBI Patient Group			
Standard Stimuli			
Targets Present	698.83 (213.58)	764.67 (201.24)	899.83 (203.50)
Targets Absent	769.67 (230.73)	856.25 (244.13)	1003.50 (315.87)
Degraded Stimuli			
Targets Present	822.67 (213.42)	899.67 (217.31)	1072.33 (326.42)
Targets Absent	890.17 (223.11)	1036.33 (270.32)	1278.25 (371.85)

Standard deviations are in parentheses.

Table 9. Experiment 1: MANOVA Comparing TBI Patients and Control Subjects on the Three Task Conditions

Source	F	df	p<	f
Group	6.29	1,10	.03	0.79
Setsize	59.58	2,20	.000	2.44
Stimulus Quality	63.07	1,10	.000	2.51
Response Type	44.04	1,10	.000	2.10
Group x Setsize	2.48	2,20	.11	0.50
Group x Stimulus Quality	0.72	1,10	.42	0.27
Group x Response Type	2.89	1,10	.12	0.54
Setsize x Stimulus Quality	1.06*	2,9	.39	0.49
Setsize x Response Type	6.47	2,20	.007	0.80
Stimulus Quality x Response Type	1.29	1,10	.28	0.36
Group x Setsize x Stimulus Quality	0.74	2,20	.49	0.27
Group x Setsize x Response Type	0.05	2,20	.61	0.22
Group x Stimulus Quality x Response Type	1.86	1,10	.28	0.43
Setsize x Stimulus Quality x Response Type	1.17	2,20	.33	0.34
Group x Setsize x Stimulus Quality x Response Type	0.49	2,20	.62	0.22

* Sphericity rejected, F estimated from Hotelling's T statistic.

Contrary to the hypotheses of Experiment 1, the two-way interactions between the group and the task factors were all nonsignificant; however, the remaining two-way interactions involving the task factors produced a more complex pattern. As expected, the interactions of setsize and stimulus quality, and of stimulus quality and response

type were nonsignificant. However, in contrast to the original predictions, the interaction between setsize and response type was significant. None of the other higher-order interactions was statistically significant. All of the main effects and two-way interactions produced moderate to large effect sizes indicating the ability of the statistical tests to detect real differences (Cohen, 1988).

As can be seen in Figure 3, the TBI patients were slower than the controls and both groups produced a longer RT when stimuli were degraded, when no targets were present, and when holding a larger number of stimuli in short term memory. The interaction involving setsize and response type in the TBI group can also be seen.

Figure 3 suggests that at the highest level of setsize, the TBI patients were somewhat more slowed by the cognitive processes related to response formulation than they were at the lower levels of setsize. In theory this might occur when a relatively larger proportion of information processing resources from a common pool was directed to the maintenance of the target stimuli in short term memory and fewer resources would be available for decision making. This would indicate a lack of structural independence of the latter processing stages in both groups.

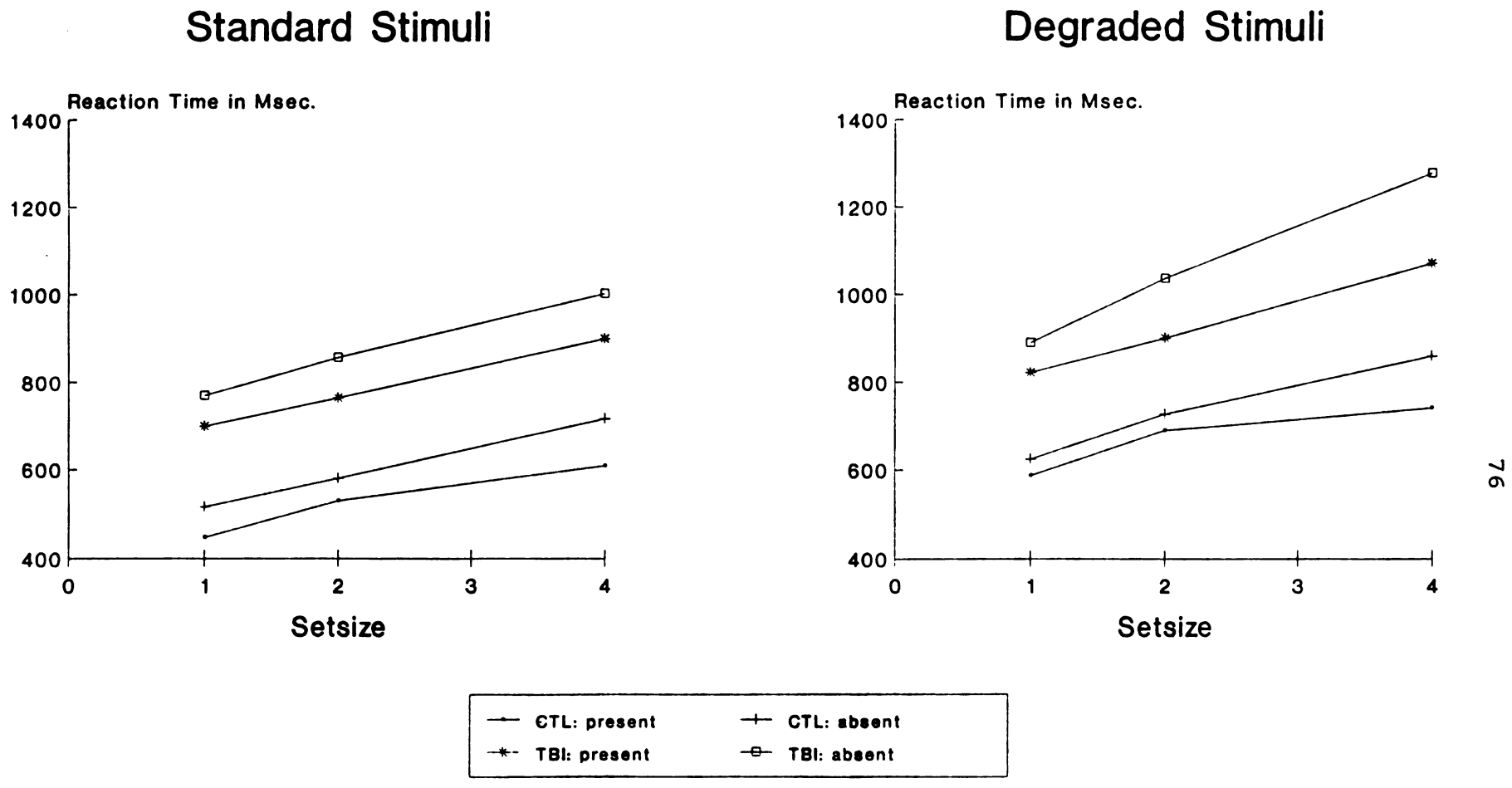


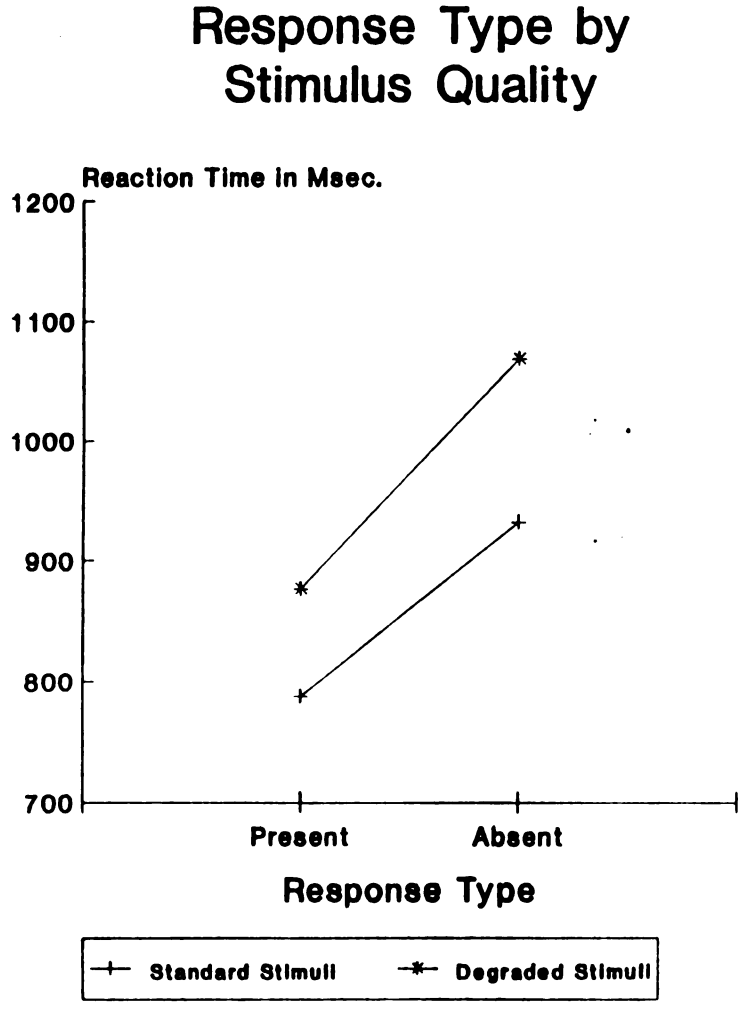
Figure 3. **Experiment 1: Means of the Median RT Performances as a Function of Setsize, Stimulus Quality, Response Type, and Group Membership**

This is an anomalous finding given the absence of a group by task factor interaction in the MANOVA of both groups (Table 9) and when no task factor interactions were found in the MANOVA of the control group (Table 8). To explore this further, a MANOVA of the task factors was performed on the data from the TBI patients. This analysis is presented in Table 10.

The main effects for the three task factors were found to be significant as were the interactions between set size and response type and between stimulus quality and response type. The interactions are graphically presented in Figure 4. These results suggest that stages of processing are not independent after TBI. The implication here is that TBI causes the decision process to require a larger share of the available resources, creating a kind of competition for

Table 10. Experiment 1: MANOVA for the TBI Group

Source	F	df	p	f
Setsize	26.97	2,10	.000	2.32
Stimulus Quality	33.03	1,5	.002	2.57
Response Type	24.13	1,5	.004	2.20
Setsize x Stimulus Quality	2.38	2,10	.14	0.69
Setsize x Response Type	4.11	2,10	.05	0.91
Stimulus Quality x Response Type	6.37	1,5	.05	1.13
Setsize x Stimulus Quality x Response Type	1.37	2,10	.30	0.52



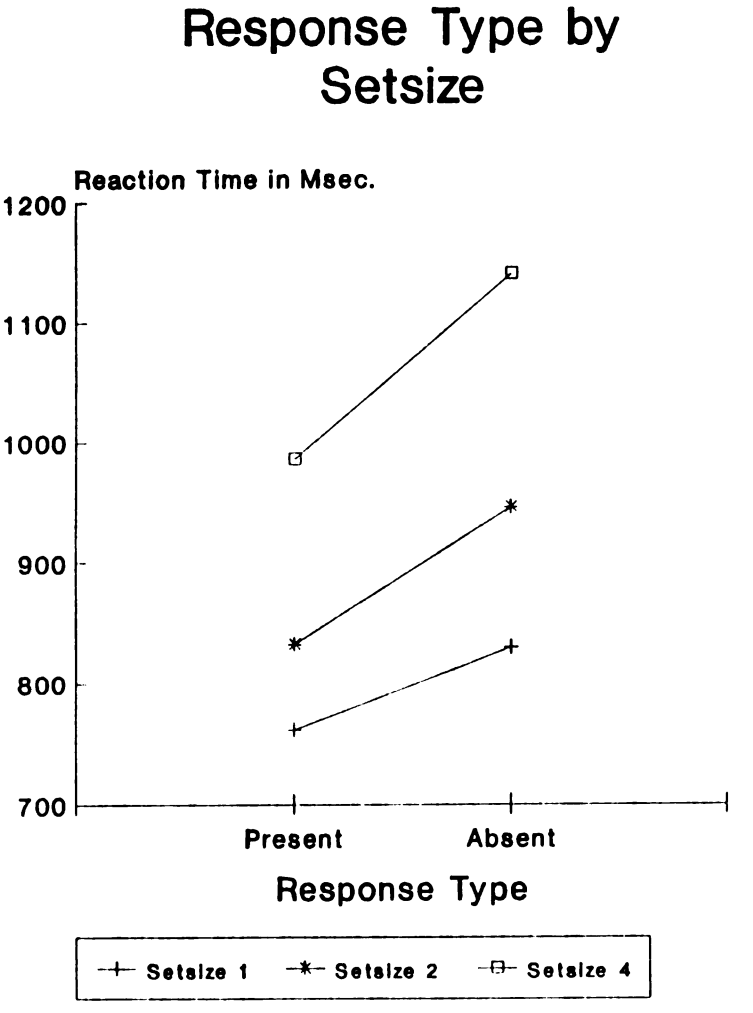


Figure 4. Experiment 1: Means of the Median RT Performances for Two Interactions Found in the MANOVA of the TBI Patients

information processing resources by either stimulus encoding or short term memory processes.

It is difficult to reconcile the difference in the findings between the analysis conducted on the combined groups and that done with the individual groups. However, it is possible that higher-order interactions in the combined groups analysis acted to obscure the important two-way interactions.

Correlations computed between the task factors and the injury related factors are presented in Table 11. Correlations between Glascow Coma Scale scores and the RT values were negative (as would be expected) and nonsignificant. The severe injury incurred by the majority of the TBI patients restricted the range of the Glascow Coma Scale and limited the expression of this relationship. Correlations computed between the 12 RT variables and both the duration of coma, PTA, and length of hospitalization were positive and moderately strong, with the majority attaining statistical significance. Because higher levels of each of these three variables indicated a greater severity of injury, these results suggested that RT increased as severity of injury increased. In contrast, time since injury was not found to be related to RT and this was likely due to the relatively homogeneity of the patient group. A similar pattern of correlations was found for simple RT. Because only one of the subjects was

Table 11. Experiment 1: Correlations Between Task Conditions and Injury Related Variables

Task Conditions	Glascow Coma Scale	Coma	Post-Traumatic Amnesia	Time Since Injury	Hosp Rehab
Setsize 1					
Standard Stimuli					
Targets Present	-.48	.76*	.77*	.48	.84*
Targets Absent	-.45	.79*	.81*	.59	.86*
Degraded Stimuli					
Targets Present	-.52	.43	.60	.23	.58
Targets Absent	-.39	.76*	.81*	.55	.86*
Setsize 2					
Standard Stimuli					
Targets Present	-.43	.73*	.67	.48	.80*
Targets Absent	-.31	.80*	.79*	.56	.89**
Degraded Stimuli					
Targets Present	-.46	.49	.59	.27	.63
Targets Absent	-.26	.43	.50	.13	.60
Setsize 3					
Standard Stimuli					
Targets Present	-.26	.68	.63	.44	.78*
Targets Absent	-.37	.78*	.71	.43	.85*
Degraded Stimuli					
Targets Present	-.35	.78*	.76*	.38	.87*
Targets Absent	-.28	.77*	.80*	.35	.88**
Simple RT	-.42	.86**	.86**	.62	.92**

* $p < .05$ ** $p < .01$ *** $p < .001$ (1 tailed tests)

prescribed an anticonvulsant, the effect of this on RT could not be evaluated.

Correlations between the RT of the task conditions and patient characteristic variables are reported in Table 12. In the control group a clear relationship between age and RT was demonstrated, with increasing age causing a relative prolongation of RT. This relationship was not found in the TBI patient group, presumably because the effects of injury overshadowed the effects of age. The effect of education on RT across tasks was ambiguous. Five of the twelve correlations were significant and six others were moderately strong though nonsignificant. These were negative relationships, indicating that as education increased, RT decreased. As before, this relationship was not found in the TBI patient group and this outcome may also reflect the potent effect of injury. In contrast a strong relationship was found between scores on the Beck Depression Inventory and RT in the patient group. TBI patients endorsing higher levels of depressive symptoms tended to have longer RT scores. This relationship was not found in the control group.

Experiment 2

An analysis of the covariate measures for the groups studied in Experiment 2 revealed that the TBI patient group (M = 6.3, SD = 4.3) did not differ from the control group (M = 7.2, SD = 6.2) on the Beck Depression Inventory, $t(5) = 0.23$, $p > .05$.

Table 12. Experiment 1: Correlations Between Task Conditions and Subject Characteristic Variables

	Controls			TBI		
Task Conditions	Age	Educ	Beck	Age	Educ	Beck
Setsize 1						
Standard Stimuli						
Targets Present	.91**	-.76*	.02	-.67	.10	.94**
Targets Absent	.92**	-.72*	-.07	-.60	.06	.94**
Degraded Stimuli						
Targets Present	.77*	-.55	.22	-.51	.13	.79*
Targets Absent	.80*	-.54	.17	-.59	.15	.95*
Setsize 2						
Standard Stimuli						
Targets Present	.90**	-.66	-.24	-.64	.07	.97***
Targets Absent	.80*	-.57	-.17	-.62	.19	.98***
Degraded Stimuli						
Targets Present	.76*	-.73*	-.19	-.55	.14	.88**
Targets Absent	.85*	-.86*	-.27	-.57	.39	.86*
Setsize 3						
Standard Stimuli						
Targets Present	.81*	-.78*	-.38	-.59	.21	.99***
Targets Absent	.50	-.07	.27	-.72*	.20	.97***
Degraded Stimuli						
Targets Present	.88**	-.69	-.22	-.75*	.30	.96***
Targets Absent	.94**	-.69	-.16	-.73*	.42	.93**
Simple RT	.76*	-.60	.27	-.64	.08	.93**

* $p < .05$ ** $p < .01$ *** $p < .001$ (1 tailed tests)

When the groups were compared in terms of their performance on the Continuous Performance Test (CPT) it was found that in the X condition, the frequency of correct detections did not differentiate the groups (TBI: M = 126.3, SD = 3.6; control: M = 128.5, SD = 0.6), t(5) = 1.55, p > .05. Also the frequency of false alarms in this condition did not differentiate the groups (TBI: M = 4.5, SD = 4.2; control: M = 1.7, SD = 1.2), t(5) = -1.93, p > .05. Similarly in the AX condition, both the frequency of correct detections (TBI: M = 122.3, SD = 6.8; control: M = 128.5, SD = 0.8; t(5) = 2.19, p > .05) and the frequency of false alarms (TBI: M = 4.3, SD = 4.9; control: M = 2.0, SD = 1.9; t(5) = -.95, p > .05) failed to differentiate the groups. Thus overall, both groups demonstrated an intact ability to sustain their attention during a simple, repetitive cognitive task.

The means of the median RT data for Experiment 2 are presented in Table 13. These data were analyzed with a 2 x 3, repeated measures MANOVA comparing group membership with RT over trial blocks. Trial block 19 was the first block given on day 3 while trial block 30 was the last block before the targets and nontargets were reversed. Trial block 31 was the reversal trial. This analysis is presented in Table 14. It disclosed that the effects of both group membership and performance over trial blocks were significant. Further the interaction of group and trial blocks was significant.

Table 13. Experiment 2: Means and Standard Deviations for the Median RT Data for Three Trial Blocks

Group	Block 19	Block 30	Block 31
Control Group			
Mean	577.8	550.1	631.3
SD	26.8	22.0	77.7
TBI Patient Group			
Mean	703.2	773.7	884.8
SD	53.3	78.2	130.2

Table 14. Experiment 2: MANOVA Comparing Groups and Trial Blocks

Source	F	df	p	f
Group	34.46	1,10	.000	1.86
Trial Block	7.85*	2,9	.01	1.32
Group x Trial Block	4.44	2,20	.03	0.67

* Sphericity rejected, F estimated from Hotelling's T statistic.

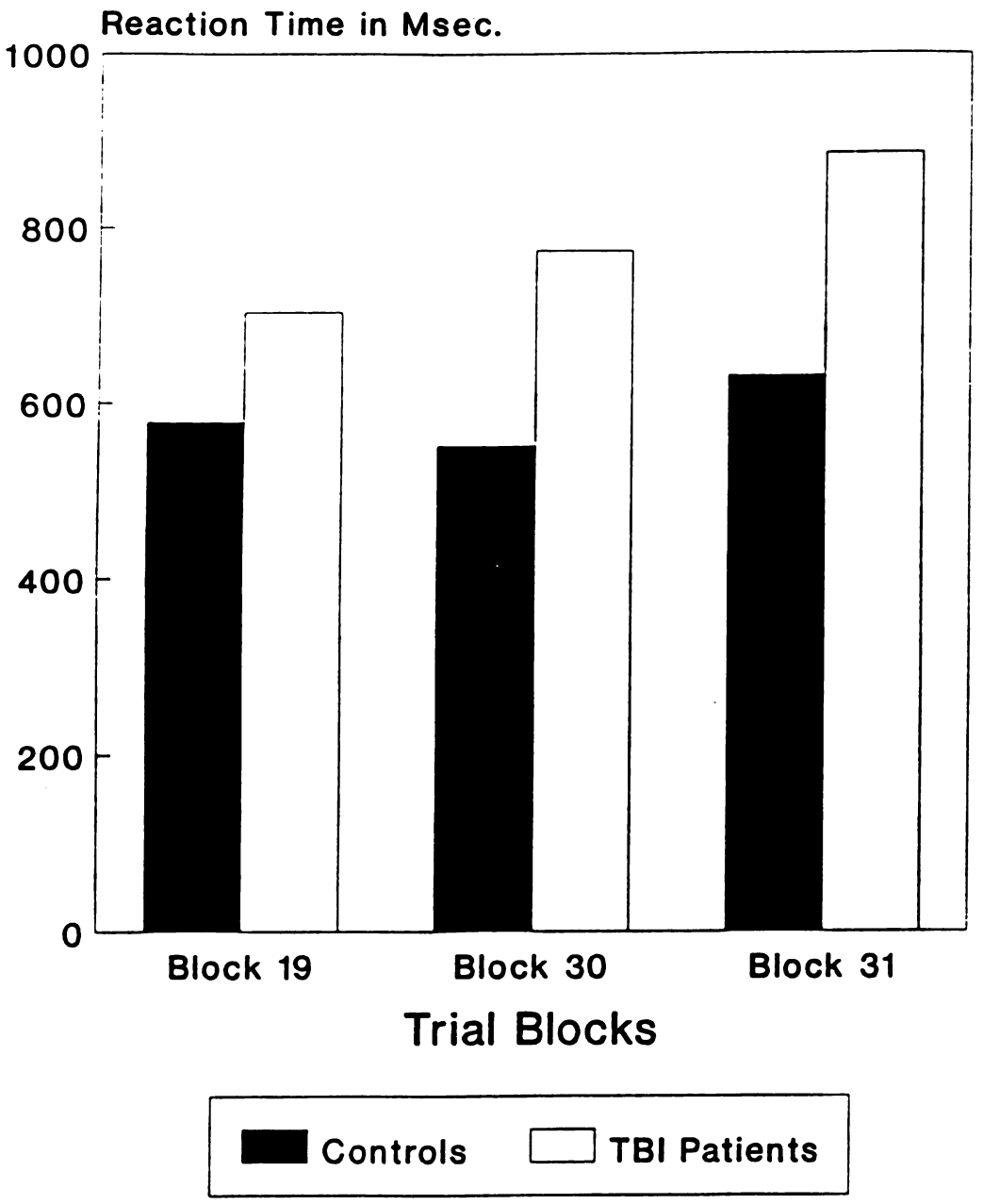


Figure 5. Experiment 2: Means of the Median RT Performances for TBI Patients and Controls Over Three Trial Blocks

As can be seen in Figure 5, the TBI patients consistently produced longer RT scores than did their matched controls. Further, the control subjects improved between trial block 19 and trial block 30 ($t(5) = 3.17$, $p < .05$) producing quicker RT's as would be predicted by Shiffrin and Schneider (1977). As the controls responded to the CM condition (i.e., the same set of targets) they developed automatic responses that facilitated their RT.

When trial blocks 30 and 31 were compared, the control group's performances were not found to differ significantly ($t(5) = -2.34$, $p = .07$); however, using Cohen's (1988) method, the power of this test was found to be less than 0.41, suggesting that a real effect may have been obscured. Thus it is still possible that the control subjects developed an automatic response and when the targets and non-targets were reversed for trial block 31, the RT of controls increased as the automatic response conflicted with the new task requirements. This is the Focused Attention Deficit (FAD) described by Shiffrin and Schneider.

As was hypothesized, TBI patients failed to demonstrate a reduction in RT between trial blocks 19 and 30 and thus failed to demonstrate the creation of an automatic response. When the targets and non-targets were reversed, the RT of the TBI patients was further slowed.

A post hoc analysis of the contrasts used to test these effects helped to clarify these findings. As would

be expected, trial blocks 19 and 30 ($t(20) = -4.06$, $p < .01$, and blocks 30 and 31 ($t(20) = -3.87$, $p < .01$) differed significantly. More importantly, the group by trial block interaction contrast was significant for trial blocks 19 and 30 ($t(20) = 3.30$, $p < .01$) while the contrast for trial blocks 30 and 31 was not ($t(20) = .60$, $p > .05$. Thus the difference in the decreasing RT in the controls and the increasing RT in the TBI patients was significant between trial blocks 19 and 30. However, between trial blocks 30 and 31 the groups differed only in terms of their absolute levels of RT.

This lack of interaction complicates the interpretation of these data. Both groups appeared to be equally affected by the target reversal which suggests that they both demonstrated an FAD (i.e., a performance decrement caused by a response conflict). However, for this to be true the group by trial interaction found between trial blocks 19 and 30 would need to be caused by a performance decrement in the TBI group that was independent from other psychological processes such as fatigue. The unimpaired performance of the TBI group on the CPT requires this. Indeed, Goldstein (1942) suggested that some brain-injured patients show a lengthening of RT that only occurs after at least 10 trials have been administered.

In order to assess this possibility, the data from trial blocks 19 and 30 were reanalyzed. For each group and each trial block, t-tests were computed to compare the

first 10 and last 10 trials. In the absence of significant differences between the first and last 10 trials, it can be concluded that RT was not affected by the number of trials in a trial block.

For the control group, this analysis produced nonsignificant results for trial block 19 ($t(5) = -0.83$, $p > .05$) and trial block 30 ($t(5) = -1.44$, $p > .05$). Similar nonsignificant results were obtained for trial block 19 for the TBI patients ($t(5) = -0.25$, $p > .05$); however, the results were less clear for trial block 30. This test did not achieve significance by standard criteria ($t(5) = -2.31$, $p = .07$); however, the computed probability value raised the question of the relative power of the test. Using Cohen's (1988) approach, the power of this test was computed at 0.90, suggesting that a real difference between the first and last 10 trials could have been found.

Taken together, these data provide support for the hypothesis that the TBI and control groups differed in terms of their ability to develop automatic processes following high levels of practice. It appeared that the control group produced faster RT's with high levels of practice and demonstrated a performance decrement when the target reversal occurred. However, the results from the TBI group were more ambiguous. They failed to demonstrate a practice effect and in the absence of this, it is difficult to label the performance decrement of the TBI

group between trials 30 and 31 as an FAD. Several possibilities will be considered below.

Correlations between the RT on the three trial blocks and the factors related to the injury are presented in Table 15. These correlations were largely nonsignificant though generally in the expected direction. Glascow Coma Scale was inversely related to RT and as the length of coma increased, RT on trial blocks 19 and 30 increased. Further, time since injury and length of hospitalization (both measures of severity of injury) were positively correlated with RT on trials 19 and 30. Thus, performance on a 4-choice binary task may be marginally related to

Table 15. Experiment 2: Correlations Between Task Conditions and Injury Related Variables

Conditions	Glascow Coma Scale	Coma	Post-Traumatic Amnesia	Time Since Injury	Hosp Rehab
Trial Blocks					
Block 19	-.50	.80*	-.05	.80*	.82*
Block 30	-.84*	.60	-.03	.71	.74*
Block 31	-.60	.08	-.34	.38	.27
Trial Block Differences					
Block 19-30	.88**	.08	-.01	-.29	-.31
Block 30-31	.14	.42	.49	.07	.26

* $p < .05$ ** $p < .01$ (1 tailed tests)

severity of injury; however, it is also the case that no relationship was found between the length of post-traumatic amnesia and RT. Difference scores for the RT scores on trial blocks 19 and 30, and trial blocks 30 and 31 were also correlated with the injury related variables in order to assess the effects of the CM training in the former and the target set reversal in the latter. With the exception of the significant relationship between Glascow Coma Scale scores and the change in RT between trial blocks 19 and 30, these were unremarkable. Only two subjects received anticonvulsant medication and so this factor could not be evaluated.

Table 16. Experiment 2: Correlations Between Task Conditions and Subject Characteristic Variables

	Controls			TBI		
Conditions	Age	Educ	Beck	Age	Educ	Beck
Trial Blocks						
Block 19	-.04	-.30	-.21	-.68	-.87*	-.05
Block 30	.34	-.20	.20	-.59	-.79*	-.40
Block 31	.40	.51	-.23	-.55	-.70	-.67
Trial Block Differences						
Block 19-30	-.41	-.17	-.47	.21	.34	.64
Block 30-31	-.28	-.52	.26	.29	.35	.63

* $p < .05$ (1 tailed tests)

Correlations between the RT scores both across the trial blocks and the two difference scores, and the subject variables are presented in Table 16. These were generally nonsignificant for both groups and no pattern emerged.

DISCUSSION

Experiment 1

In Experiment 1, contrary to the expected results, TBI patients did not demonstrate the kind of generalized slowing of information processing that was predicted by Van Zomeren's (1981) model. When the data from both groups were analyzed, the TBI group was slower than the matched control group in each of the task conditions; however, the expected two-way, group by task-factor interactions were not found. Thus the amount of information to be processed did not have a disproportionate effect on the TBI patients. This finding provides a strong challenge to Van Zomeren because he argued that the task complexity effect was caused by a greater sensitivity to information load in TBI patients relative to controls.

Van Zomeren's (1981) model was not supported in another way. He hypothesized that the information processing deficit of TBI would not be limited to any particular stage. In the present analysis, the pattern of task-factor interactions in the TBI data suggested that the processing stages had been selectively affected by the injury. More specifically, the data suggested that the structural independence of processing stages was lost

between the stimulus encoding and binary decision stages as well as between the memory scanning and decision stages. This organization is illustrated in Figure 6. Because the present control group and Sternberg's (1969b) own uninjured subjects produced no task-factor interactions, it may be concluded that the non-standard organization was caused by TBI.

It is reasonable to hypothesize that the slowing of RT that follows TBI is related to the non-standard structural organization, not a simple slowing of all three measured processes. However, it is noteworthy that the third, two-way interaction (i.e., between stimulus encoding and memory scanning) approached significance in the TBI data. The relatively large effect size (i.e., 0.69) argues against

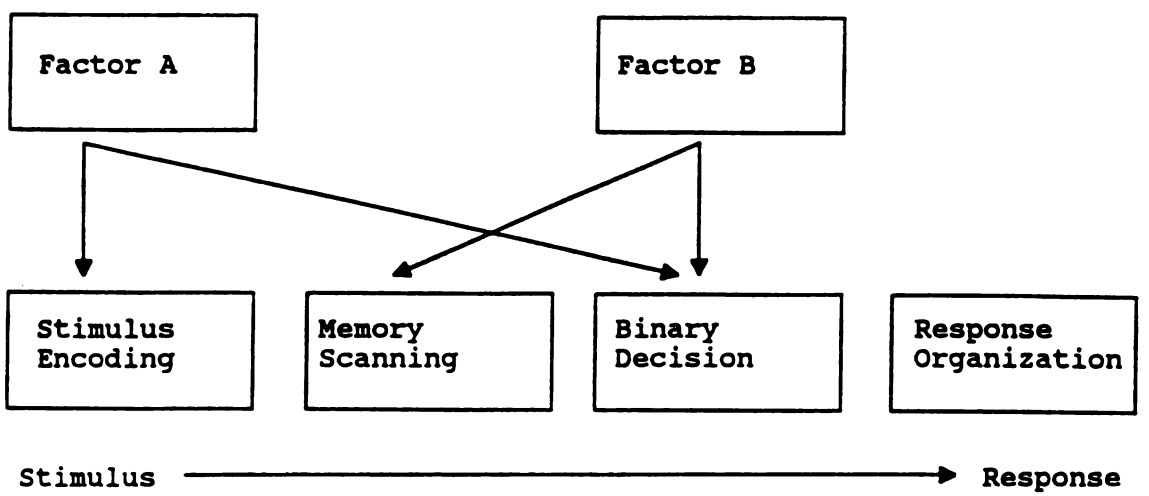


Figure 6. Sternberg's Four Factors Related to Higher Order Factors Caused by TBI

the possibility that the test of the interaction was not sensitive to a real effect and that in actuality, all three stages were affected by TBI. Thus, Van Zomeren's hypothesis to this effect requires modification.

An alternative hypothesis can be posited based on a consideration of the TBI data. Although the reason for the task-factor interactions cannot be directly determined, it is possible that TBI causes the later stages of processing to provide resources to the earlier stages. Response processes may have a special capability in assisting the impaired mechanisms or perhaps the earlier stages pass incompletely processed data to the later stages for completion.

Sternberg (1969a) addressed this general issue when he commented on situations where processing stages might not be structurally independent. He hypothesized that the duration of any particular stage of processing might be increased when a previous stage had to process a greater amount of information than was usual and would thus use more of the available resources (p. 288). In the present study, if the stimulus encoding and memory scanning stages had been impaired by TBI, then the decision stage may have provided the resources that allowed the CRT tasks to be performed, albeit more slowly.

This interpretation presumes that processing stages can be selectively impaired by brain damage and a few studies have reported this. Wolcott, Saul, Hellige, and

Kumar (1990) found that degraded letter stimuli prolonged the RT of right hemisphere stroke patients more than it did both normal controls and left hemisphere stroke patients. Similar results have been found with unpublished pilot data from this laboratory using the standard and degraded digit stimuli described in Experiment 1. Schweinberger, Buse, Freeman, Schonle and Sommer (1992) found that right hemisphere stroke patients demonstrated deficits in either the encoding or memory scanning of faces but not of digits, compared to both controls and left hemisphere stroke patients. Their methodology prevented them from determining which mechanism was impaired and so these results are somewhat ambiguous. Swinney and Taylor (1971) assessed aphasic subjects (i.e., presumably having focal left hemisphere damage) and matched controls using the Sternberg Paradigm and digit stimuli. They found a specific impairment in the memory scanning stage as well as an interaction between memory scanning (i.e., memory set size) and the decision stages (i.e., response type). Taken together, these studies suggest that focal lesions can impair specific processing stages and can create interactions between processing stages.

Some of the studies using the Sternberg Paradigm with TBI patients have also found impairments of specific processing stages, contradicting Van Zomeren. Although these studies used operationalizations different than Sternberg's and in some cases did not test more than two

factors simultaneously, several impairments of single stages were found. Stokx and Gallaird (1986) found an impairment in a response preparation process and a trend toward a significantly slowed CRT in a stimulus encoding stage. Shum, McFarland, Bain and Humphreys (1990) found specific impairments in both stimulus identification and response selection stages. Schmitter-Edgecomb, Marks, Fahy, and Long (1992) found impairments in stimulus encoding and response execution stages.

This leads to an important point. The kind of impairments found in Experiment 1 can be caused by focal damage, not only diffuse white matter damage as Van Zomeren suggested. Supporting this is the work of Rao, St. Aubin-Faubert, and Leo (1989). They compared multiple sclerosis patients and healthy subjects with a variant of the Sternberg Paradigm. If Van Zomeren's hypothesis was true then damage to white matter, regardless of etiology, should produce generalized slowing. Using nondegraded digit stimuli, they found a significant interaction involving group and set size but no significant main effect or interaction with response type. This indicated a slowing of serial comparisons without impairment of the later decision stage. Thus, isolated damage to the white matter, does not necessarily have a generalized effect on processing speed.

In the present study, post-acute TBI resulted in a pattern of injury that can be described as both diffuse and

multi-focal (see Table 2). Deficits included unilateral hemiparesis, dysgraphia, and visual spatial deficits that are consistent with lateralized damage as well as both attentional and memory deficits that may be related to diffuse injuries. CT scanning disclosed both localized pathology (contusions and hemorrhages) and diffuse white-matter damage. In spite of the white matter damage, no specific memory scanning impairment was found in the present data. Thus it is necessary to hypothesize that the primary information processing deficits after TBI involve damage to specific stages and the way in which the brain compensates for damage, not the disconnection of processing mechanisms caused by white matter damage.

This leads to the question of how best to characterize the attentional deficit in TBI. Many researchers have used the term slowing of information processing; however, this phrase has little specific meaning. This term has been used to refer to slowing caused by damage to a single stage of processing, to concurrent damage in multiple stages, to increased noise in the information processing system secondary to disconnection, and to the slowing that results from increases in task complexity. Experiment 1 may provide the basis for a different, more specific, characterization.

As has been suggesed here, RT may be prolonged after TBI when some mechanisms (but perhaps not all) are damaged and thus less efficient. The impaired stages then rely on

other cognitive processes (likely decision mechanisms in the later stages of processing) to perform their functions. This situation would reduce the overall capacity of the information processing system and produce a DAD. Depending on the tasks used in assessment, it would give the appearance of either slowing all cognitive mechanisms or only the common response stage. Interestingly, this nicely summarizes the majority of the research into attention deficits after TBI.

Obviously these findings require replication to assess the stability of the reorganized task-factor structure and several methodological points need to be considered. Although the present sample was small, it appeared adequate for the analysis and this likely had several causes. First, the careful use of computer technology likely reduced the RT measurement error. Many of the studies in the existing literature have used a computer keyboard to measure subjects' responses, have used slow liquid crystal and monochrome displays to present stimuli, and may have used unreliable timing measurements. The present study avoided these problems. Secondarily the use of a vocal response likely reduced the variability of RT attributable to lateralized cerebral damage and manual button presses. Lastly, the matching of groups and the use of repeated-measures MANOVA likely provided adequate design sensitivity.

Using these same techniques, any replication should include the fourth stage of processing studied by Sternberg (1969a) by varying the relative frequency of targets to nontargets across trial blocks. Unfortunately, this would increase the required number of trial blocks unless fewer trials are included in each block to make the testing more manageable (Hamsher & Benton, 1977).

Experiment 2

In Experiment 2, as was expected, TBI patients failed to develop an automatic response with extended practice on a Sternberg Paradigm task using an unchanging set of target digits (CM training), unlike their matched controls. Controls reduced their RT with practice and then showed a statistically significant decrement in RT when a novel set of targets was introduced. This was consistent with automatic processing, where a highly practiced response is inappropriately evoked by a stimulus and slows task performance. In contrast, the TBI patients demonstrated longer RT's with increasing levels of practice, suggesting that no automatic response was acquired.

However, the TBI patients also demonstrated the same degree of slowing as controls when the targets were reversed. This is problematic because the lack of an interaction between the effects of group and trial blocks for the target reversal suggested that both groups were equally affected by an FAD. Thus the TBI patients may have actually developed an automatic response; however, their RT

may have been slowed during training by a separate process unrelated to automaticity. A performance factor may have obscured the benefits of practice by inhibiting response speed.

This was explored by comparing the first and last 10 trials of the last CM trial block in the TBI group. It was expected that an inhibitory process would cause the RT of the last 10 trials to exceed that of the first 10 trials; however, no such difference was found. Thus a simple inhibitory process operating within a trial block seems unlikely; however, it is possible that this kind of process could have an influence over trial blocks.

Other evidence argues against an inhibitory process. First, in Experiment 2, the TBI patients did not perform differently than controls on the Continuous Performance Test and thus were unlikely to have a deficit of vigilance (i.e., sustained mental effort). Second, Benton and Blackburn (1957) compared an etiologically mixed sample of brain-injured patients and a hospital control group, looking for changes in RT as a function of practice. After 30 simple RT trials and another 30 CRT trials, no trend for a poorer RT performance was demonstrated. However, it is notable that 13 percent of the brain-injured group demonstrated a 20 percent slowing of CRT from their initial levels. Similarly, Ponsford and Kinsella (1992) found no deficits in vigilance when they studied severely injured

TBI patients and matched orthopedic controls using a binary CRT task over a 45 minute interval.

Taken together, these studies suggest that impaired vigilance cannot account for the performance decrement seen in Experiment 2. However, it is still possible that RT is inhibited in TBI patients when a complex CRT task is presented over a relatively large number of trials. Benton and Blackburn's (1957) tasks were simple and were presented over a small number of trials. Ponsford and Kinsella's (1992) procedure was similar to Experiment 2 in terms of the duration of testing; however, they employed a simple binary comparison task that probably used fewer processing resources than the memory scanning task.

A replication of Experiment 2 will be required to determine the cause of the observed performance decrement. The easiest way to do this would involve adding a fourth day of testing that would include an additional practice trial block with the target stimuli. If a group by trial block interaction occurs between a trial block early in the third day of practice and that on the fourth day that is similar in form to Experiment 2, then the performance or inhibitory process would be confirmed.

Another way to approach the issue of automaticity after TBI would involve administering the Sternberg Paradigm to TBI patients and controls over several days using a CM procedure. As Kristofferson (1972b) discovered, high levels of CM practice (i.e., daily

sessions over seven weeks) will produce a negatively accelerated slope for increasing levels of setsize in uninjured subjects. This indicates the effect of automaticity in information processing, presumably because fewer cognitive resources are required as task complexity increases. If TBI patients fail to demonstrate this kind of slope, then it could be concluded that they do not develop automatic processing.

Conclusions

The present research provides another demonstration of the slowing of RT that has been so consistently reported in the TBI literature. The data from Experiment 1 are not inconsistent with Van Zomeren's (1981) use of the concept of the DAD to explain posttraumatic attentional deficits; however, most other aspects of Van Zomeren's model were not supported. The greater benefit of Experiment 1 lies in the creation of a new model that views slowed RT in terms of an alteration in the allocation of processing resources post-injury. This model is more parsimonious than that of Van Zomeren and is consistent with other experimental data. Experiment 2 provided some support for the hypothesis that TBI patients fail to develop automatic processes as a result of practice; however, the results were were complicated by the lack of an expected interaction. Both studies will need to be replicated; however the second study will require a procedural modification to better

differentiate automaticity related to FAD's from performance effects.

Taken together, these studies also point the way to the possibility of better measurement technologies and clinical interventions in neuropsychology. A more complete understanding of the processing deficits caused by TBI may allow attentional deficits to be measured more specifically and not just as residual components of currently available tests. This decoupling of attention also should allow for a better assessment of severity and recovery if some form of attention or some reorganization of processing resources is unique to TBI. Further, if attentional constructs evolve beyond simple labels and factors defined by measurement paradigms, then it may be possible to design specific exercises and remediation strategies that hasten recovery. To date, this possibility has not been realized (e.g., Novack, Caldwell, Duke, Bergquist, & Gage, 1996), probably because of the lack of adequate theory.

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