The effect of depression on memory performance following traumatic brain injury.

Michelle Anne. Keiski

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The Effect of Depression on Memory Performance
Following Traumatic Brain Injury

by

Michelle A. Keiski

A Thesis
Submitted to the Faculty of Graduate Studies and Research
through the Department of Psychology
in Partial Fulfillment of the Requirements for
the degree of Master of Arts at the
University of Windsor

Windsor, Ontario, Canada

2002

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Abstract

The purpose of the current study was to compare the memory performance of depressed TBI subjects and non-depressed TBI subjects. The hypothesis was that differences between the groups would emerge on the recall and/or recognition components of the California Verbal Learning Test -II (CVLT-II) and the Rey Complex Figure Test (RCFT), after covarying a composite score reflecting neuropsychological impairment. The neuropsychological test results of 76 subjects who had sustained mild to severe traumatic brain injury (TBI) were obtained retrospectively. The subjects were classified as depressed or non-depressed on the basis of depression scores on the Personality Assessment Inventory (PAI). With regard to the CVLT-II, ANCOVA analyses indicated that the Long Delay Free Recall scores and the Recognition Discriminability scores of the depressed subjects (n = 24) were reduced relative to the non-depressed subjects (n = 19), after covarying for the neuropsychological composite score. However, recall was no more impaired than was recognition. Follow-up analyses indicated that the learning slope and semantic clustering of the depressed subjects were reduced compared to the non-depressed subjects. The findings were interpreted to indicate deficient encoding in the depressed subjects, related to the failure to benefit from the semantic organization of the CVLT-II list. With regard to the RCFT, ANCOVA analyses indicated that depressed subjects (n = 29) achieved lower scores on the Delayed Recall trial than did the non-depressed subjects (n = 30), whereas Recognition performance was similar across the groups. Hence, the RCFT performance of the depressed TBI subjects indicated retrieval deficits or difficulty with some other aspect of the recall task. The encoding deficits observed on the CVLT-II, but not on the RCFT,
may be due to the failure to initiate or maintain deliberate learning strategies on the
CVLT-II, whereas the incidental learning nature of the RCFT should preclude an
advantage of non-depressed subjects arising from the use of deliberate learning strategies.
The utility of the derived neuropsychological composite score and the relationship of the
PAI total depression score to depression subscale scores were also discussed. Finally,
limitations of the current study and future directions were reviewed.
Acknowledgements

First, I must gratefully acknowledge my advisor, Dr. Doug Shore, as this project would not have been possible without his perpetual and incessant availability and support. The numerous discussions regarding this project and the subsumed topics in general provided valuable guidance in the planning phase and in the interpretation of results. The feedback and counsel of my committee members, Drs. J. Casey and K. Munroe, were essential in refining the methodology, discussion and format of this project, as well as in defining the scope of this project from the outset. I would also like to thank Drs. L. Buchanan and J. Casey for their assistance in the preparation of a poster based on preliminary data.

My dearest thanks is extended to Dr. J. Hamilton, who graciously permitted me access to data she has amassed over the years as a result of her exemplary dedication to her clients. Her unwavering encouragement and support have been touching and inspiring.

I must also thank numerous friends and relatives for their encouragement and support. To my parents Danielle, Reijo and Ginny for instilling in me the sense that I could achieve whatever I set my mind to. To my sister for her intellectual discussion and reassuring words. To my grandparents Elsa and Kalevi for encouraging me in every possible way. To Darryl, Kayla and Brianna for their patience, understanding and unwavering support in my absence. To Geri for putting things back into perspective and attending the proposal and defense. To Nik for always being ready and willing to lend a helping hand.
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List of Abbreviations

BDI: Beck Depression Inventory
CANTAB: Cambridge Neuropsychological Test Automated Battery
CDS: Cognitive Difficulties Scale
CT: computerized axial tomography
CVLT: California Verbal Learning Test
DSM: Diagnostic and Statistical Manual of Mental Disorders
EEG: electroencephalogram
GCS: Glasgow Coma Scale
GOS: Glasgow Outcome Scale
GOAT: Galveston Orientation and Amnesia Test
MCMI: Millon Clinical Multiaxial Inventory
MMPI: Minnesota Multiphasic Personality Inventory
PAI: Personality Assessment Inventory
PASAT: Paced Auditory Serial Addition Test
PCI: Postconcussional Index
PCS: postconcussional syndrome
PIQ: Performance Intelligence Quotient
PTA: post traumatic amnesia
RAVLT: Rey Auditory Verbal Learning Test
RBMT: Rivermead Behavioral Memory Test
RCFT: Rey Complex Figure Test
SCL-90-R: Symptom Checklist 90 - Revised
TBI: traumatic brain injury

VIQ: Verbal Intelligence Quotient

WAIS: Wechsler Adult Intelligence Scale

WMS: Wechsler Memory Scale
Chapter I

Introduction

Approximately 35% to 45% of individuals who have sustained a traumatic brain injury (TBI) concomitantly experience depression (Busch & Alpern, 1998; Hoofien, Gilboa, Vakil, & Donovick, 2001; Kreutzer, Seel, & Gourley, 2001; Macniven & Finlayson, 1993). Both TBI and depression have been associated with memory deficits. As a result, the individual with comorbid TBI and depression presents a unique challenge to the assessing neuropsychologist. The question arises whether memory deficits should be attributed to the injury per se or to the concomitant depression. This determination is crucial for the accurate formulation of a diagnosis, prognosis and treatment plan. The differentiation also has important implications in the medicolegal assessment / treatment of individuals who have sustained a TBI. Unfortunately, current understanding of the effects of depression on memory in individuals who have sustained TBI is rather limited. Hence, the attribution of a memory deficit to either TBI or depression is difficult at best.

Memory in Depression

Seminal Studies of Memory Impairment in Depression

Seminal studies of memory performance in depressed patients documented memory deficits and identified factors that contributed to these memory deficits. In one such study, Weingartner, Cohen, Murphy, Martello, and Gerdt (1981) asked subjects to process a series of words either semantically or acoustically. The subjects were asked to recall these words following a 24-hour delay. Although the depressed subjects recalled as many acoustically processed words as controls, their recall of semantically processed words was impaired. The authors concluded that depressed patients were impaired in the
more elaborate processing of information. Specifically, their view was that the depressed patients failed to benefit from the “more elaborate or complete processing strategy of using semantic associations to establish more memorable trace events” (p.46).

In another part of the study, recall of word lists that varied in extent of organization was examined. The organization inherent in the lists was manipulated by the inclusion of semantic categories. The highly organized lists contained exemplars from four semantic categories and related exemplars were listed consecutively. In the moderately organized lists, the exemplars from the four semantic categories were presented in random order, whereas the non-organized list consisted of words that were not semantically related.

Depressed subjects adequately recalled words from the highly organized list. However, they recalled fewer words from the less organized lists than did the controls. Furthermore, the depressed subjects recalled items in a less clustered fashion during the recall of the moderately organized lists. In contrast, their clustering during recall of the highly organized list was similar to that of the controls. These findings indicate that recall deficits are more pronounced when the semantic structure of the list is less evident. Such an effect continues to be documented in recent literature.

Subsequently, Cohen, Weingartner, Smallberg, Pickar, and Murphy (1982) proposed that memory deficits observed in depression were related to a failure to initiate or maintain effort. Supporting this suggestion was the finding that memory performance was related to severity of depression and a measure of effort. Specifically, “effort” was measured by asking subjects to maintain grip strength, equivalent to half of their peak strength, for as long as possible. Significant positive correlations were obtained between
duration of motor persistence and memory performance on a task similar to the Consonant Trigrams Test. As a result, the authors concluded that "the most parsimonious explanation would be one based on a single deficit in the area of the central motivational state" (p.596). They further stated that "these findings raise considerable doubt as to the reasonableness of hypothesizing specific memory deficits in depression separable from the general deficits of motivation, drive and attention" (p.596).

Although the correlation between motor persistence and memory performance is noteworthy, an unequivocal relationship between motivation/drive and motor persistence has not been demonstrated. Moreover, the assumption of a relationship between motor performance and motivation may be untenable in a disorder in which subcortical dysfunction (Massman, Delis, Butters, Dupont, & Gillin, 1992), and basal ganglia dysfunction in particular (Kimbrell et al., 2002; Meyer et al., 2001), have been implicated. As a result, the conclusion drawn by these authors appears premature.

A study by Richards and Ruff (1989) is frequently cited as providing direct evidence that memory deficits in depression should not be attributed to reduced motivation. In this study, 30 depressed patients and 30 control subjects were randomly assigned to "motivation" and "non-motivation" conditions. In the motivation condition, subjects were informed that they were to be rewarded for surpassing a cutoff and positive feedback was provided during performance. A card-sorting task, purported to be sensitive to motivation, was utilized to ensure that the "motivation manipulation" had achieved the desired effect.

Data analyses demonstrated a potent effect of the motivation manipulation on card-sorting performance. Analyses of neuropsychological performance on a variety of
attention and memory measures indicated a significant main effect of depression, but not of motivation. Nor was there an interaction between depression and motivation. These results supported the notion that motivation deficits are unlikely to be the sole source of neuropsychological deficits in depressed patients.

In general, seminal studies confirmed memory deficits in individuals with depression and suggested that semantic processing and list organization moderated recall deficits. However, there was disagreement regarding the contribution of motivation and effort to these deficits. More recently, researchers have sought to characterize these memory deficits more precisely on a range of experimental and standardized measures. While the importance of motivation to memory deficits remains controversial, considerable agreement has been reached with regard to specific memory patterns in depression. A substantial body of evidence points to a dissociation between recall and recognition in depression.

*Dissociation of Recall and Recognition*

Ilsley, Moffoot and O’Carroll (1995) compared the memory performance of 15 inpatients who met DSM-III-R criteria for major depressive disorder to that of a control group with similar age, NART IQ, sex, and handedness. All subjects completed Digit Span from the Wechsler Memory Scale - Revised Edition (WMS-R), the Rivermead Behavioral Memory Test (RBMT) and an incidental memory task. The incidental memory task required subjects to rate how well each of a series of words (e.g., unlucky, lonely) described them or their current situation. Implicit memory was subsequently assessed by word-stem completion.
Ilsey and colleagues (1995) concluded that attention was intact in the depressed subjects since differences between the groups were not observed on Digit Span Forward or Digit Span Backward. Performance on the implicit memory task was unimpaired. On the RBMT, the depressed inpatients demonstrated reduced recall on the story and message subtests relative to controls. In contrast, their recognition of faces and pictures on the RBMT was equivalent to that of the controls. Overall, memory deficits were observed on memory tasks requiring recall, whereas deficits were not observed on memory tasks requiring recognition. Hence, the memory deficits of the depressed patients were attributed to retrieval difficulties.

Similarly, Fossati, Deweer, Raoux, and Allilaire (1995) reported that the memory deficits of their inpatient depressed subjects were best characterized as retrieval deficits. In their study, the performance of 10 depressed inpatients was compared with that of 10 controls matched for age and sociocultural level. The subjects were screened for alcoholism, Axis 2 diagnoses and neurological / medical conditions. Memory was assessed via performance on the Wechsler Memory Scale (WMS), the California Verbal Learning Test (CVLT) and the Grober and Buschke verbal learning task.

Consistent with the majority of prior evidence, Digit Span Forward and Digit Span Backward did not differ between the patients and controls. Furthermore, significant differences on the Logical Memory subtest of the WMS were not in evidence, suggesting intact attention and intact recall of organized narratives.

On the CVLT, the depressed subjects recalled fewer words across the five learning trials. Nonetheless, they benefited from repetition and the mean learning slope was similar to that of the controls. In learning the list, the depressed and control subjects
utilized semantic and serial organization equally across the trials, as demonstrated by similar semantic and serial clustering ratios. However, the depressed subjects were less consistent in their recall of words across the learning trials. Moreover, the depressed subjects performed as well as the control group on the recognition trial.

On the Grober and Buschke verbal learning task, depressed patients demonstrated deficits in free recall, although these disappeared when cues were provided. The recall of depressed patients was less consistent across trials. Furthermore, recognition performance was perfect for both patients and controls. In sum, reduced performance on the recall trial, accompanied by intact recognition, was observed in the depressed patients on both list learning tasks.

Fossati and colleagues (1995) described the memory deficits of their depressed patients as best characterized by retrieval deficits, which is consistent with the findings of Ilsley and colleagues (1995). The authors speculated about difficulty in initiating or maintaining processes necessary for active retrieval from memory. They noted that their findings directly contradict Weingartner’s theory that reduced motivation and attention interfere with organization and mnemonic strategies during initial information processing. First, deficits on measures traditionally believed to reflect attention (i.e., Digit Span Forward) are not typically found in depressed subjects. Second, the semantic clustering ratio on the CVLT did not differ between controls and depressed individuals in Fossati’s sample. Thus, it was suggested that depressed subjects utilize semantic organization to the same degree as controls. However, Fossati and colleagues cautioned that their sample size was quite small. In fact, reduced semantic clustering (Calev &
Erwin, 1985) and reduced performance on more sensitive attention tasks (Zakzanis, Leach, & Kaplan, 1998) have been reported in depressed individuals.

In sum, Ilsley and colleagues (1995) and Fossati and colleagues (1995) observed impaired recall performance, accompanied by intact recognition performance, in their depressed patients. The authors of both studies concluded that depressed subjects exhibited retrieval deficits. However, alternate explanations could account for discrepancies between recall and recognition performance.

For example, Calev and Erwin (1985) argued that recall is more difficult than recognition. Given the generalized impairment of psychiatric patients, this pattern of performance could simply be an artifact due to discrepant discriminating powers. Hence, Calev and Erwin chose to investigate the memory performance of depressed subjects with verbal recall and recognition tasks matched for difficulty.

Analyses of test performance indicated that depressed individuals performed better on the recognition trial than on the free recall trial, whereas no such discrepancy was observed in the controls, suggesting that the discrepancy between recall and recognition performance in depressed subjects is not due to differential difficulty of the tasks. Furthermore, the control subjects demonstrated greater clustering during free recall than the depressed individuals. The authors concluded that the controls demonstrated a "better organized retrieval system" (p. 128). On the other hand, it could be argued these findings are indicative of "better organized encoding".

In another study, depressed subjects completed a modified version of the Rey Auditory Verbal Learning Test (RAVLT) in which a list of 15 monosyllabic words was presented visually on a computer screen (Brand, Jolles, & Gispen-de Wied, 1992). Recall
was assessed following each of five learning trials and following a delay. The subjects were also asked to recognize the words amongst a set of target and distractor words.

The depressed individuals recalled fewer words on several of the learning trials. However, differences in delayed recall were not in evidence. Recognition performance did not differ between the patients and the controls, although response latencies were longer for the depressed individuals. The authors suggested that a ceiling effect might have caused the equivalence of recognition scores. As a result, they conducted a second experiment in which subjects were not allowed to “overlearn” the items.

The second experiment conducted by Brand and colleagues (1992) involved the same task, although recall and recognition were assessed following either one learning trial or three learning trials, as opposed to five. In the one trial condition, the depressed individuals were impaired on the immediate recall and delayed recall trials. The patients were also impaired on the recognition trial. Furthermore, their response latencies during the recognition trial were longer than those of the controls. In the three trial condition, the depressed individuals were impaired on immediate recall, although their delayed recall was equivalent to that of the controls. Recognition scores did not differ between the groups, although the depressed individuals demonstrated longer response latencies during the recognition trial.

The authors concluded that encoding of information is impaired at the beginning of a novel task when a substantial amount of effort is required. Thus, when depressed individuals are asked to recognize words following a single exposure, they may demonstrate deficits in recall and recognition. However, following repeated exposure to the words, deficits in recognition are no longer apparent and deficits in recall
predominate. The observation of impaired immediate recall, accompanied by intact delayed recall, is difficult to explain. Overall, the authors summarized their findings by stating that “when the depressives have greater familiarity with the learning material (i.e., when demands on effort have become smaller), the material is encoded into memory better, which results in better retrieval (delayed recall) and recognition memory” (p. 84).

A review of the relevant literature indicates that depressed individuals have frequently been found to demonstrate deficits in free recall. In several instances, this deficit in free recall is accompanied by intact recognition. This has led several investigators to conclude that memory deficits in depression are related to difficulties with the retrieval process. Some authors have recognized that inefficient encoding may hinder retrieval. Furthermore, recognition deficits have been observed when limited opportunity has been provided to learn the material. Whether deficits are apparent in recall, recognition, or both also appears to depend upon the organization inherent in the material.

Channon, Baker, and Robertson (1993) specifically studied the effect of list structure and organization on subsequent recall and recognition in a group of 24 depressed outpatients. The study was prompted by comments made by Hertel and Hardin (1990) which suggested that the memory impairments of depressed individuals were due to difficulty initiating mnemonic strategies which were not mandatory for task completion. On the other hand, if depressed subjects were provided with sufficient environmental cues to support these strategies, they would allocate sufficient cognitive resources. Hertel and Hardin’s comments imply that depressed subjects should be most
impaired on tasks “that permit but do not necessitate the spontaneous use of strategies” (p. 323).

Hence, Channon and colleagues (1993) devised three word lists that varied in degree of organization. The least structured list did not contain superordinate categories; all words were semantically unrelated. The moderately structured list contained exemplars from four superordinate categories presented in randomized order. The most structured list contained four categories, with exemplars from each category listed consecutively. If depressed individuals are solely impaired in initiating strategies or imposing organization, they should not demonstrate deficits in the recall of the least structured list since this list does not permit implementation of a semantic strategy. Furthermore, impairment should not be observed in the recall of the most structured list since the organization is clearly provided and initiation of a strategy is unnecessary. In contrast, failure to initiate a strategy and impose organization should lead to deficits in recalling the moderately structured list.

Recall and recognition performance were assessed for all three lists. The patients recalled fewer words from the moderately structured list than did controls, whereas they recalled as many words from the least structured and most structured lists as did controls. This finding supports the suggestion that depressed individuals are maximally impaired in the recall of moderately “structurable” information. With regard to recognition performance, repeated measures ANOVA failed to find differences between the groups. However, analyses of recognition sensitivity (i.e., d’) indicated reduced sensitivity for the depressed subjects, although an interaction with list organization was not obtained. Thus, recognition sensitivity may not differ systematically with learning material organization.
Channon and colleagues (1993) concluded that memory deficits in depression were most apparent with moderately “structurable” information. Furthermore, free recall conditions were more sensitive to organization related deficits than were recognition conditions. The authors stated that semantic organization allows the use of beneficial strategies during encoding and retrieval, whereas the benefit of such organization in forced choice recognition is less clear.

In contrast to the findings of Channon and colleagues (1993), reduced recognition on a list without inherent semantic organization has been reported in depressed patients (Wolfe, Granholm, Butters, Saunders, & Janowsky, 1987). The authors utilized a modified version of the Rey Auditory Verbal Learning Test (RAVLT) which included a recall version and a recognition version. Unipolar depressed patients recalled fewer words than controls on the five learning trials and the delayed recall trial. Furthermore, the slope from Trial 1 to Trial 5 was steeper in controls than in unipolar patients. On the recognition version, the depressed subjects recognized a lower percentage of previously recalled words than the controls. Overall, impaired recall and recognition performance on the RAVLT was demonstrated in this sample of unipolar inpatients. Furthermore, the reduced learning slope suggests difficulty learning the items of the RAVLT.

In addition, scores on the Beck Depression Inventory (BDI) were significantly correlated with delayed recall and delayed recognition performance in the unipolar patients. This provides evidence that recognition deficits may become manifest on particular verbal learning tasks. Although not explicitly addressed by these authors, it is possible that recognition deficits become apparent specifically when the information is not readily amenable to semantic organization, increasing the difficulty of the task.
Difficulty encoding poorly structured information may contribute to reduced performance on recall and recognition. This contrasts, however, with the intact recall of words from the least structured list demonstrated by Channon and colleagues (1993).

Zakzanis and colleagues (1998) stated that research on the neuropsychology of depression “often yields an ambiguous mix of results: decidedly significant, suggestive, convincingly null, and hopelessly inconclusive” (p. 111). In discussing their meta-analysis of previous findings, the authors addressed a number of important variables that may explain discrepancies between findings. As a result, several of their observations shall be reviewed in detail herein.

Selected studies, published between 1980 and 1997, included a healthy control group and were restricted to unipolar patients diagnosed according to DSM-III-R criteria. Effect sizes were calculated for the various neuropsychological tasks included in the 22 studies selected.

Tests of declarative, episodic memory were typically calculated to have effect sizes above the median effect size. Specifically, the RAVLT, the Rey Complex Figure Test (RCFT), and the Visual Reproduction and Logical Memory subtests from the WMS-R demonstrated effect sizes above the median. Measures found to have effect sizes below the median were: WAIS-R Information and Vocabulary, Wide Range Achievement Test Reading, Wisconsin Card Sorting Test categories, WAIS-R Similarities and Digit Span Forward, Auditory Consonant Trigrams, Paced Auditory Serial Addition Test (PASAT) and WAIS-R Picture Completion.

Zakzanis and colleagues (1998) first noted that it is unlikely that observed memory deficits are entirely attributable to attention deficits since effect sizes for
episodic memory tasks were greater than effect sizes for attention measures. They also noted that the pattern of effect sizes does not support the suggestion that memory deficits are attributable to inability to engage in effort-demanding cognitive tasks since the effect sizes for the PASAT and the Brown-Peterson Consonant Trigrams Test were quite small. However, effort-demanding memory measures were more sensitive to the effects of depression than automatic or superficial memory measures. As a result, the sensitivity of memory measures to depression appears to depend upon the effort required for completion (or task difficulty). The authors stressed that the effect of “effort” appears specific to memory processes rather than a broad range of cognitive processes. This finding suggests that depression related memory deficits are more likely to be observed on difficult tasks which require substantial effort.

The authors suggested that retrieval deficits could explain the observed deficits in episodic recall. That having been said, the authors pointed to the range of sensitivities calculated for various measures of episodic recall. As an example, they pointed to the greater sensitivity of the RAVLT over the CVLT. The CVLT supports encoding via its amenability to semantic organization. The authors stated that this finding supports an effect of depression on encoding processes in episodic memory. The authors summarized their findings by stating that “major depressive disorder is accompanied by dysfunction of effortful encoding of information along with an accompanying inefficiency of retrieving poorly encoded information from declarative memory” (p. 118).

In general, episodic recall measures appear most sensitive to the effects of depression, although these effects are accentuated when the material does not inherently support organization which facilitates encoding and/or retrieval processes. Recall
measures involving clearly organized material are less sensitive to the effects of depression. The authors comment that “if the aim is to differentiate memory deficits caused by depression from an underlying neurological dysfunction, ..., then the use of the RAVLT may result in a false-positive result... As such, the clinician would be in a better position to differentiate depression from [neurological dysfunction] on the basis of CVLT scores” (p. 117).

Massman, Delis, Butters, Dupont, and Gillin (1992) compared the performance of individuals with unipolar depression (n = 40), bipolar depression (n = 9), Huntington’s Disease (n = 20), Alzheimer’s Disease (n = 20) and age-matched controls (n = 40) on the CVLT. On the basis of a discriminant function, the depressed subjects were classified as resembling the normal controls (n = 32) or the Huntington’s patients (n = 17), whereas none were classified as resembling the Alzheimer’s patients. Thus, depressed individuals with memory impairment exhibit a memory pattern similar to that exhibited by patients with subcortical dysfunction.

The depressed subjects classified as similar to the Huntington’s group demonstrated reduced slope across the learning trials, reduced semantic clustering and impaired free recall performance. Their performance on recognition was considerably superior to their performance on free recall. The observation of reduced free recall performance, accompanied by improved recognition, in depressed individuals who demonstrate memory impairment is consistent with the majority of evidence in the literature on depression.

Following a summary of evidence pointing to depression related impairments on the CVLT, Delis, Kramer, Kaplan, and Ober (2000) concluded that a subgroup of
depressed patients exhibit impairment on the CVLT. When observed, the memory
deficits are characterized by reduced performance on free recall and improved
recognition. The authors concluded that the memory pattern of depressed individuals was
"indicative of retrieval deficits as the major contributor to poor performance" (p. 118).
Nonetheless, the authors acknowledged the possibility that a discrepancy between recall
and recognition may be the result of fragmented memory traces due to a "partial encoding
deficit" (p. 49).

_Dissociation of Implicit and Explicit Memory_

Much like the dissociation between recall and recognition, a dissociation between
explicit and implicit memory functions in depression has been reported (Denny & Hunt,
1992). Unipolar depressed individuals (n = 16) and normal controls (n = 16) were asked
to rate how well each of 24 words described them or their current life circumstances.
Individuals were not forewarned that they would be asked to recall the words. In the
explicit memory task, the subjects were required to list the words they could recall. In the
implicit memory task, the subjects were required to complete word fragments with
interspersed letters omitted from each word.

Depressed individuals explicitly recalled fewer words than the controls. In
contrast, there were no group differences on the word completion task. Hence, a
significant dissociation between explicit and implicit memory was observed. These
findings essentially mirrored those of Danion, Willard-Schroeder, Zimmerman, Grange,
Schlienger, and Singer (1991). The authors tentatively proposed that the relative
decrement on the free recall task might be due to impairment in a strategic, consciously
controlled aspect of memory retrieval. However, the authors called for caution in this
interpretation because the implicit memory task included letters of the word which may have served as cues, whereas the explicit memory task lacked these cues.

Illey and colleagues (1995) conducted a similar study, although the cues provided were equivalent in the explicit and implicit memory tasks. The authors examined incidental recall and priming of words presented in a self-rating task similar to that used by Denny and Hunt (1992). It was found that depressed individuals were not impaired on the implicit or the explicit word stem completion tasks. Hence, there is evidence to support the suggestion that deficits in explicit recall are reduced or rendered negligible when sufficient cues are provided.

Bazin, Perruchet, de Bonis, and Feline (1994) also ensured that their explicit and implicit tasks provided equivalent cues. Subjects were asked to read a list of 40 common nouns aloud and were informed that memory would subsequently be assessed. Relative to sex, age and education matched controls, the depressed subjects were impaired on the explicit task, despite intact implicit performance.

In sum, there have been discrepant reports about a potential dissociation between explicit and implicit memory performance when cues are equivalent across the tasks. Significant differences in methodology may account for the discrepant findings of Illey and colleagues (1995) and Bazin and colleagues (1994). First, Illey and colleagues utilized an incidental memory task. If depressed individuals are impaired in the deployment of purposeful learning strategies, it is likely that impairment will be most pronounced in deliberate learning situations. Secondly, Bazin and colleagues utilized a longer list, making recall more difficult and increasing the likelihood that explicit recall deficits would be observed. Third, Illey and colleagues utilized a self-rating task, which
generally imposes greater elaboration and self-referencing. It would appear reasonable to suppose that requiring recall of nouns, processed with less depth, would increase the likelihood that explicit memory deficits would be observed. In sum, incidental memory conditions, reduced task difficulty and greater depth of processing may reduce or abolish the explicit recall deficits of depressed individuals.

It should be noted that the failure to obtain a dissociation between explicit and implicit memory in depression has been reported elsewhere in the literature. For example, Elliott and Greene (1992) found both explicit and implicit memory to be impaired in their sample. Bazin and colleagues (1994) proposed that subjects in the Elliott and Greene study were found to be impaired on the implicit task because they noticed that all of the words on the implicit task had been previously presented and referred to explicit memory to complete the task. Including distractor items in implicit memory tasks was recommended, to reduce the likelihood that subjects ascertain the nature of the task.

In sum, several investigators have reported a dissociation between explicit and implicit memory performance in depressed patients. This dissociation has been interpreted as reflecting disruption of retrieval processes. However, several authors have recognized that this dissociation may be due to discrepant cue provision in explicit and implicit memory tasks. It is less clear whether the dissociation between explicit and implicit memory performance remains, when explicit and implicit conditions are matched for cue provision. Discrepant task requirements may be responsible for equivocal findings in studies which match cue provision in explicit and implicit memory conditions. Regardless, the finding of improved explicit recall with cue provision is consistent with
the observation that free recall is more likely to be impaired in depressed patients than is recognition.

Asymmetry of Verbal and Nonverbal Memory Deficits

Although the majority of studies on depression have focused on verbal memory, several studies have identified visual memory deficits in depressed subjects. For example, Richards and Ruff (1989) found depression related deficits in delayed recall on a trail learning task, in addition to Block Span. In contrast, there were no deficits on the Selective Reminding Test or Digit Span. The authors suggested that their patients demonstrated greater visuospatial deficits than verbal deficits, although they noted that deficits on the RCFT were not observed.

The failure to find depression related deficits on the RCFT contrasts with the findings of Rossi, Stratta, Nistico, Sabatini, Di Michele, and Casacchia (1990). These findings were derived from the RCFT performance of 20 depressed individuals prior to initiation of electroconvulsive therapy. The depressed subjects demonstrated dramatic impairments on the copy and immediate recall trials of the RCFT compared to matched controls. Delayed recall and recognition trials were unfortunately not administered.

Several points should be borne in mind when comparing the findings of Rossi et al. (1990) and Richards and Ruff (1989). First, depressed subjects undergoing electroconvulsive therapy are likely to be more severely depressed than other samples of depressed patients. Second, the depressed individuals assessed by Rossi and colleagues exhibited very poor copy scores and a recognition measure was not administered. In this instance, it is difficult to determine whether memory impairment was the major contributor to poor performance on the RCFT.
Immediate and delayed recall of the RCFT were found to be sensitive to depression in the meta-analysis conducted by Zakzanis and colleagues (1998). Given that the commercialization of a recognition trial for the RCFT (i.e., Meyers & Meyers, 1995) is relatively recent, the majority of studies utilizing the RCFT have not examined recognition performance in depressed patients. Investigations with the RCFT which include both recall and recognition components will hopefully help to elucidate the effects of depression on performance of this task.

Another meta-analysis revealed that memory deficits were more prevalent in verbal recall tasks than in visual recall tasks (Burt, Zembar, & Niederehe, 1995). However, studies that were not restricted to unipolar depression were included in the meta-analysis. As a result, the applicability of this finding to unipolar depression in particular is unclear.

Other evidence suggests that visual recall is affected to a greater degree than verbal recall. For example, Kalska, Punamaki, Makinen-Pelli, and Saarinen (1999) found that depressed individuals exhibited visual recall deficits relative to matched controls, in the absence of verbal recall deficits. In their study, the performance of 30 acute-care patients diagnosed with major depression was compared to that of controls matched for age, sex, education and level of intelligence (i.e., Information and Similarities of the WAIS-R). Memory assessment included Warrington’s Recognition Memory Test and the WMS-R. The depressed individuals performed more poorly on the Visual Memory Index, Figural Memory, Visual Paired Associates (immediate and delayed), and Visual Reproduction (immediate and delayed). Deficits in face recognition were not observed. Whereas visual memory deficits were apparent, verbal memory deficits were not in
evidence. The authors noted that it was unfortunate they were unable to examine visuospatial functions, independent of memory.

Consistent with these findings, Deptula, Manevitz, and Yozawitz (1991) reported greater impairment in visual memory than in verbal memory in depressed inpatients (n = 28) compared to matched controls (n = 14). The verbal and visual learning tasks consisted of 20 stimuli (animal names and geometric figures, respectively) presented over four learning trials. Impaired performance in the depressed group was observed for visual recall, but not for verbal recall. For each subject, difference scores between visual and verbal standard scores were then calculated. It was found that 89% of the depressed subjects exhibited an asymmetry of memory performance favouring verbal recall. In contrast, the control subjects failed to demonstrate an asymmetry.

These findings were inconsistent with those of Calev, Korin, Shapira, Kugelmass, and Lerer (1986). An asymmetry between visual and verbal recall was not detected when Calev’s verbal and visual matched-difficulty tasks were utilized. Deptula and colleagues (1991) suggested the inconsistent findings were due to their use of a multi-trial task, rather than a single trial task such as that used by Calev and colleagues (1986).

Taken together, the literature indicates that depression is associated with verbal and nonverbal memory deficits. A substantial proportion of the literature on depression has documented the presence of verbal memory deficits. However, a small number of studies, addressing both verbal and visual memory, have reported greater impairment in visual memory than in verbal memory. It is possible that the novel nature or increased difficulty of visual memory tasks is responsible for the asymmetrical memory deficit. Consistent with this line of reasoning, Calev and colleagues (1986) failed to find an
asymmetry when utilizing single-trial tasks matched for difficulty. As a result, it is unclear whether visual or verbal memory deficits predominate in depression.

Relevance of Patient Status and Age

A meta-analysis of findings pertinent to memory impairment in depression revealed that depression was more closely associated with memory performance in inpatients than in outpatients (Burt et al., 1995). With support from this finding, the authors stated that “since the memory performance of depressed inpatients was usually compared with that of controls who were neither depressed nor patients, multiple dimensions of group variation were encompasses that might explain these patient status effects” (p. 296).

One study directly addressed this possibility by comparing the memory performance of depressed inpatients (n = 18) with that of personality disordered inpatients (n = 18) and healthy controls (Golinkoff & Sweeney, 1989). Both patient groups were impaired on recall and recognition of a verbal paired associate learning task, relative to the control group. Hence, the observed memory impairment may not be specific to depression. Several confounding factors that may account for the patients’ deficient performance relative to controls were listed. These included lifestyle changes involving sleep cycle, diet, exercise, social support and patterns of socialization.

It should also be noted that memory deficits have not been consistently observed in younger depressed subjects. For example, Purcell, Maruff, Kyrios, and Pantelis (1997) failed to detect memory deficits in their young unipolar patients, only one of whom was an inpatient. Specifically, deficits were not in evidence on the delayed matching to
sample, spatial recognition and pattern recognition tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB).

Similarly, Grant, Thase, and Sweeney (2001) failed to observe memory deficits on the Visual Reproduction subtest of the Wechsler Memory Scale or the Hopkins Verbal Learning Test in 123 outpatients with unipolar, nonchronic depression (i.e., less than 24 months). Deficits on the memory tests from the CANTAB were not apparent, which is consistent with the findings of Purcell and colleagues (1997). Thus, memory deficits may not be in evidence in younger outpatients with unipolar depression.

On the other hand, Sweeney, Kmiec, and Kupfer (2000) reported a selective impairment of episodic memory on the CANTAB in young to midlife unipolar patients. On the delayed matching to sample task, unipolar patients performed more poorly than controls and the deficit became more pronounced as the delay increased. In contrast, there were no significant group differences on spatial recognition or pattern recognition scores. Nonetheless, severity of depression was correlated with performance on the spatial recognition task. The authors concluded that their young unipolar patients demonstrated selective mesiotemporal impairment. Although the subjects in this study were relatively young (mean age = 32.29 years; SD = 9.10), it should be noted that they were inpatients.

In sum, the presence of memory deficits in depressed outpatients has not been adequately demonstrated in the literature. It is possible that depression is less severe (or alternately, qualitatively distinct) in outpatients. Furthermore, memory deficits have not been consistently reported in younger depressed subjects. However, the measures used in most of these studies, namely visual recognition and delayed matching to sample tasks,
may be less sensitive to the effects of depression than other measures. Performance on recall tasks which require retention over a longer delay are perhaps more likely to be impaired in younger depressed outpatients. The finding of impaired recall of a moderately structured list in young depressed outpatients is consistent with this possibility (Channon et al., 1993). In general, tasks utilized to study the effects of depression in younger subjects should be relatively difficult, include a sufficient number of items and require retention over a delay to achieve appropriate sensitivity.

*Traumatic Brain Injury*

Memory deficits following moderate to severe TBI have been well documented. Whereas some researchers have attempted to characterize the memory deficits of TBI subjects on a variety of experimental and standardized tests of memory, others have documented the heterogeneity of memory patterns following moderate to severe TBI. The presence of neuropsychological deficits following mild TBI is more controversial.

*Memory Deficits Following Moderate to Severe TBI*

Brooks (1974a) examined the performance of 34 rather severely injured individuals on a continuous recognition measure. The stimuli consisted of 160 cards with either geometrical designs or nonsense figures. The trials were divided into seven blocks. For each subject, a corrected score was calculated by subtracting the number of false positives from the number of correct hits. The control group consisted of 34 orthopedic patients. The groups were adequately similar with regard to age and education.

Analyses indicated that the TBI group was consistently impaired on the corrected score relative to the controls. Furthermore, the learning slope across the blocks was steeper in the controls than in the TBI group. These findings imply that the TBI group
recognized fewer visual stimuli due to reduced initial acquisition of the information. Time since injury appeared unrelated to the memory performance of the individuals who had sustained TBI. However, length of posttraumatic amnesia (PTA) was inversely correlated with overall memory performance (i.e., sum of corrected scores on all 7 blocks). Given the relationship between PTA and recognition performance, the observed memory deficits were attributed to diffuse injury rather than to focal injury.

Although it appeared that reduced acquisition led to impaired recognition in the above study, an alternate explanation could account for the findings. It is possible that the TBI individuals encoded the stimuli but failed to identify to them as “hits” because they utilized a stringent decision-making criterion. As a result, a re-analysis of the data was conducted, utilizing signal detection indices (Brooks, 1974b). Sensitivity of recognition (d’) was reduced in the individuals with TBI. In contrast, the observer criterion (B) did not differ between the groups, suggesting that observed deficits in recognition were attributable to memory deficits per se rather than decision criteria.

Hannay, Levin and Grossman (1979) criticized Brooks’ choice of signal detection indices. Hannay and colleagues utilized “c” as a measure of response criterion rather than “B”. They conducted a study with 47 patients who sustained injuries that ranged from Grade I to Grade III. Subjects categorized as Grade I were conscious upon admission, whereas subjects categorized as Grade III were unconscious for more than 24 hours. In this study, stimuli were line drawings of familiar objects. Repeated stimuli belonged to one of several categories, namely flowers, birds, snails, moths, mushrooms, insects, fish and rodents. Six blocks of 20 stimuli were included in this visual continuous recognition paradigm.
The results clearly demonstrated that subjects classified as Grade II or Grade III had recognition sensitivities (d') which were reduced compared to controls and Grade I patients. Memory efficiency was related to duration of coma and not to focal injury, suggesting a greater role of diffuse injury in producing memory deficits. With regard to response criterion, Grade II subjects had significantly lower criterions, or c values, than did Grade I subjects and controls. Comparison of c values for the Grade III subjects, to Grade I subjects and healthy controls, approached but did not meet statistical significance. In general, these findings raised the possibility of less stringent decision criteria following TBI.

Brooks' (1974) visual recognition data was subsequently re-examined, utilizing "c" as a measure of observer criterion (Richardson, 1978; as cited in Hannay, Levin, & Grossman, 1979). Differences in observer criterion level (c) were not found.

In sum, visual recognition deficits have been documented in individuals who have sustained moderate to severe TBI. The reduced learning slope demonstrated by Brooks (1974a) suggests reduced rate of acquisition of geometric and nonsense designs. Whether response criteria are modified following TBI is less certain. The less stringent criterion reported by Hannay and colleagues (1979) would inflate the raw number of correct hits, as well as the raw number of false positives. Indices which account for both hits and false positives, such as d' or Brooks' (1974a) corrected score (i.e., hits - false positives), appear sensitive to the effects of TBI. Finally, visual recognition performance appears to depend upon duration of unconsciousness or PTA, suggesting an important role of diffuse injury.
More recently, deficits on standardized tests of memory have been documented following TBI. The WMS-R, RAVLT and RCFT performance of 13 individuals who had sustained "serious" TBI from 1 to 12 years prior (mean = 3.7 years) has been described (Crossen & Wiens, 1988). Several composite scores of the WMS-R were well below the normative values for individuals with comparable education levels. Specifically, General Memory, Verbal Memory, Visual Memory and Delayed Recall indices were significantly below the normative levels. The authors noted that the Delayed Recall composite score was particularly impaired, whereas the Attention/Concentration composite score fell within average limits. In addition, the head-injured subjects were significantly impaired on delayed recall of the RCFT. For the RCFT, delayed recall was the only score reported. Furthermore, all calculated measures of the RAVLT (i.e., Trials 1 to 5, Trial 5, Recall, Recognition and Intrusion Errors) were significantly impaired in the head-injured subjects.

In general, Crossen and Wiens (1988) demonstrated substantially impaired performance in their sample. However, it is difficult to characterize these memory deficits with any specificity or assert their representativeness to various populations, given the small number of subjects and the wide variability in time since injury. The authors noted that distinctions between storage and retrieval deficits could not be made on the basis of WMS-R performance. This is because verbal recognition is not assessed with this version of the WMS and visual recognition is only assessed on Figural Memory. With regard to the RAVLT, the authors did not speculate about deficits in theoretical memory processes, nor did they report statistical tests that might have elucidated these processes (e.g., learning slope).
Reid and Kelly (1993) examined the WMS-R performance of 20 subjects who had sustained acceleration deceleration closed head injuries. Subjects were screened for history of learning disability and psychiatric disorder. Controls consisted of friends and relatives of patients matched for age and education. Subjects were tested following emergence from PTA, as defined by two consecutive scores on the Galveston Orientation and Amnesia Test (GOAT) of at least 75.

The results indicated that head-injured subjects performed significantly worse than controls on all five indices of the WMS-R. Evidence of forgetting over a delay was obtained. Specifically, percentage of information retained on the delayed recall trial was reduced on the Logical Memory, Visual Reproduction, Visual Paired Associates and Verbal Paired Associates subtests. Reduced percent retention was most pronounced on the Logical Memory and Visual Reproduction subtests. In calculations with a larger sample, PTA was inversely correlated with the Visual Memory index, General Memory index and Delayed Memory index, as was Glasgow Coma Scale (GCS).

The findings in this study allow some elucidation of theoretical memory processes. Forgetting over a delay suggests impaired consolidation or storage. However, the majority of subjects in this study were in the acute stages of recovery. Specifically, 13 of the 20 subjects were assessed less than 6 weeks post-injury. Several subjects had emerged from PTA only a few days prior to assessment. As a result, the finding of impaired consolidation or storage may not be applicable to adequately oriented patients in the more chronic phases of recovery.

There is also difficulty interpreting the significance of reduced percent retention since any calculation of percentage depends upon the initial level of performance. A
simple example can best demonstrate this point. If a head-injured patient initially learns 10 words, whereas a control subject initially learns 20 words, and both subjects forget 2 words, the subjects demonstrate dissimilar percentage retention. The head-injured subject has retained 80% of acquired information, whereas the control subject has retained 90% of acquired information. Should one conclude that the head-injured subject demonstrates increased forgetting?

DeLuca, Schultheis, Madigan, Christodoulou, and Averill (2000) criticized studies of memory in TBI for their failure to control for initial levels of acquisition. That is, given the relationship of recall measures to initial acquisition, initial learning is a confound which should be controlled for when determining the cause of reduced recall performance.

DeLuca and colleagues (2000) equated their groups on initial level of learning by training their subjects to criterion on a modified version of the Selective Reminding Test. The subjects \( n = 28 \) had sustained moderate to severe TBI and were matched to controls \( n = 21 \) on age, sex and level of education. The TBI subjects completed the assessment between 13 and 74 months post injury (mean = 32.2 months; SEM = 2.7). Of the TBI subjects, 8 failed to meet the criterion and were not included in the analyses reviewed herein.

The results indicated that the head-injured subjects required more trials to reach the criterion than the controls. In contrast, their levels of recall and recognition were comparable to those of the controls. This finding suggests that once information has been encoded, it is adequately maintained and retrieved. It was also found that both groups recalled fewer words at the 90-minute delay than at the 30-minute delay. However, an
interaction between group and delay was not obtained. The authors concluded that the verbal learning deficits in their TBI sample were due to reduced capacity for initial learning, as opposed to retrieval deficits or rapid forgetting.

Shum, Harris, and O’Gorman (2000) reported findings similar to those of DeLuca and colleagues (2000) on a visual recognition task. The subjects were 28 individuals who had sustained severe TBI, divided into early-recovery (i.e., less than 1 year post-injury) and late-recovery (i.e., more than 1 year post-injury) groups. Visual recognition was assessed with the Shum Visual Learning Test which includes Chinese characters. This visual learning test proceeds with five learning trials, an interference trial, a post-interference trial and a 20-minute delayed retention trial. A discrimination task involving Chinese characters was administered to rule out visual-perceptual difficulties in the participants.

Significant differences between the early-recovery and late-recovery groups were not identified on the Shum Visual Learning Test. Differences between the head-injured subjects and the controls, however, were evident. The TBI individuals recognized fewer of the Chinese characters and their learning slope across the five learning trials was relatively flat. In contrast, a retention after interference index and a delayed retention index were similar across the groups. These findings suggest that capacity for acquisition of information is reduced following severe TBI. Nonetheless, it would appear that information that is acquired is adequately maintained.

Other researchers have presented evidence to support the suggestion of impaired consolidation following TBI (Vanderploeg, Crowell, & Curtiss, 2001). Their participants included a TBI sample with moderate to severe injuries (n = 55), a control group matched
on demographic factors (n = 55) and a control group matched on initial learning (i.e., Trial 5 and sum of Trials 1 to 5) of the CVLT (n = 55).

The slope across the five learning trials of the CVLT did not differ between the three groups. The equivalent learning slopes were cited as evidence that encoding was not impaired in the TBI group. In contrast, the TBI subjects demonstrated reduced short delay free recall compared to demographic-matched and acquisition-matched control groups. The authors argued that the rapid decrease of recall in the TBI subjects was due to impaired consolidation. The rate of forgetting between the short delay recall and the long delay recall trials did not differ between groups. The authors cited comparability between retrieval processes across groups by showing that all groups benefited equally from semantic and recognition retrieval cues.

The conclusion of impaired consolidation in the TBI subjects relies heavily on the finding of reduced short delay recall relative to the controls matched on initial levels of learning. This control group was drawn from a sample of Army veterans that demonstrated memory impairment. The specific nature of the memory impairment(s) of individuals comprising the acquisition-matched control group was undetermined. It may be unwise to conclude that TBI subjects are deficient in consolidating information because their delayed recall is worse than individuals with uncharacterized memory deficits.

In sum, empirical evidence has demonstrated reduced level of performance on various memory tasks following moderate to severe TBI. Whether these deficits are due to impaired encoding, consolidation or retrieval is subject to debate. There have been discrepant reports from studies that have controlled for initial levels of learning., DeLuca
and colleagues (2000) found similar levels of performance on recall and recognition in TBI patients who were trained to criterion, whereas Vanderploeg and colleagues (2001) found rapid forgetting in TBI patients matched to controls on Trial 5 and Trials 1 to 5 of the CVLT. It seems reasonable to suppose that the particular aspect of memory that is most compromised could vary with injury characteristics or time since injury. Inconsistent operationalization of encoding, consolidation and retrieval throughout the literature may also contribute to contradictory conclusions.

**Heterogeneity of Memory Patterns: Emergence of Memory Subtypes**

Several researchers have evaluated the performance of TBI subjects on the CVLT, although they have not sought to identify a particular pattern characteristic of all their patients. Rather, they have documented considerable heterogeneity in the pattern of performance of various individuals following TBI. The learning subtypes derived from CVLT analyses shall be briefly reviewed herein.

Millis & Ricker (1994) proposed that memory deficits following TBI would be characterized by a variety of subtypes, given the heterogeneity of injury parameters within a TBI sample. The CVLT was utilized to examine the heterogeneity of performance deficits in their sample (n = 65), 71% of whom were classified as having sustained a severe TBI. These investigators identified four distinct patterns, which they labelled Active, Passive, Disorganized and Deficient. The Active group utilized semantic processing to guide their learning of the list and demonstrated the steepest learning slope. The patients in this group were less severely injured than those in the other groups. The three remaining subtypes were characterized as having moderate to severe deficits in verbal learning. The Passive group utilized the less efficient strategy of serial clustering.
The Disorganized type failed to reliably apply either a semantic or a serial strategy and was inconsistent in their recall. The individuals in this group recalled more words during the cued recall trials than the free recall trials. The Deficient group was impaired in all respects and demonstrated slowed rate of learning, inconsistent recall and a high rate of false positive errors during recognition.

Curtiss, Vanderploeg, Spencer, & Salazar (2001) utilized a larger sample of individuals with TBI (n = 301) to characterize verbal learning subtypes on the CVLT. The derived subtypes were subsequently validated by external correlates. There was a considerable range of chronicity amongst the subjects. Two clusters fell within normal limits, with one cluster (n = 65) demonstrating use of a semantic encoding strategy and the other cluster (n = 103) demonstrating use of a serial encoding strategy. Another cluster (n = 70) was characterized by poor retention because recall scores were low relative to the 5th trial and recognition hits were reduced. The last cluster (n = 61) to be reviewed here was characterized by poor retrieval, primarily because cued recall was superior to free recall. The individuals that comprised the latter cluster also exhibited greater difficulty with control, as indicated by a higher number of intrusions and false positives. These individuals had the lowest scores on Percent Retained of the Logical Memory and Visual Reproduction subtests of the WMS-R. They were also more impaired on Trails B and Stroop Colour-Word.

Wiegner and Donders (1999) identified a four-factor solution via confirmatory factor analysis applied to their mixed severity sample of 150 non-litigating TBI subjects. The first cluster (n = 45) demonstrated performance within normal limits on all measures, whereas the second cluster (n = 26) demonstrated severely impaired scores on all
measures. The individuals in the second cluster were found to be younger and to have sustained more severe injuries. The remaining clusters exhibited moderate impairment. Individuals that comprised Cluster 3 (n = 37) committed more cued recall intrusions and recognized fewer words. The individuals that comprised Cluster 2 (n = 31) demonstrated superior performance on the recognition trial relative to the long delay free recall trial. The latter cluster is perhaps best characterized as demonstrating retrieval deficits.

In general, considerable evidence suggests heterogeneity in the memory deficits of TBI patients, at least on the CVLT. The subtypes derived in various studies are similar but not entirely consistent, perhaps due to samples which differ on several dimensions and discrepant statistical techniques. The heterogeneity of learning subtypes within TBI samples has been attributed to heterogeneity of injury parameters. Affective status may also contribute to heterogeneity in memory subtypes following TBI.

Heterogeneity of memory patterns in neurological samples has also been described on the RCFT. Meyers and Meyers (1995) describe five distinct memory patterns on the RCFT. The first subtype is a normal pattern characterized by T scores above 40 on Immediate Recall and Delayed Recall. The second is an attention pattern characterized by T scores below 25 on Immediate Recall, Delayed Recall and Recognition. The third pattern suggests impaired encoding. This difficulty is inferred when Immediate Recall and Delayed Recall are below a T score of 23, are relatively equivalent and Recognition is no more than 10 T score points higher than either recall score. The fourth is a disrupted storage pattern. This subtype is characterized by T scores that decrease with increasing delay. Put otherwise, Immediate Recall is superior to Delayed Recall, which in turn is superior to Recognition. The final subtype is a retrieval
pattern, which is characterized by improved Recognition compared to the recall trials. The subtypes are organized hierarchically in that later subtypes can only be inferred when the pattern has not met the criteria for previous subtypes. For example, an encoding pattern can only be inferred if the pattern of performance did not meet the criteria for the attention pattern.

Meyers and Meyers (1995) examined the prevalence of these RCFT profiles in a sample of 98 neurologically compromised subjects with a wide range of chronicity. The prevalence of the subtypes were as follows: 22.6% attention pattern, 9.3% encoding pattern, 10.3% storage pattern, 37.8% retrieval pattern and 20% normal or undifferentiated pattern. Acute patients were more likely to demonstrate attention or encoding patterns, whereas chronic patients were more likely to demonstrate storage or retrieval patterns. Thus, heterogeneity of memory patterns on the RCFT has been demonstrated in a neurologically compromised sample. Furthermore, the prevalence of the subtypes varied with chronicity.

In contrast, a relationship between chronicity and memory subtype on the CVLT was not observed in the larger sample examined by Curtiss and colleagues (2001). Although one might speculate that the contradictory findings are due to the use of the CVLT or the RCFT, the injury characteristics (including etiology) of the Meyers and Meyers (1995) sample are not described in sufficient detail to permit comparison of the neurological samples. The equivocal findings could thus be due to vastly dissimilar populations in the two studies.

One may wish to consider whether the subtype characterized by retrieval deficits occurs uniquely in mild, moderate or severe TBI. In the Wiegner and Donders (1999)
study, the individuals that comprised the cluster in which recognition exceeded free recall performance had sustained mild (48%), moderate (23%) and severe (29%) TBI. In the study by Curtiss and colleagues (2001), the individuals comprising the subtypes labelled “poor consolidation/good retention”, “poor retention” and “poor retrieval/poor control” did not differ with regard to an overall cognitive ability index. Potential associations between injury severity and RCFT subtype were not discussed by Meyers and Meyers (1995).

In sum, Meyers and Meyers (1995) found that a memory pattern suggesting retrieval deficits is more likely to be observed as chronicity increases. It seems plausible that this memory pattern emerges as attention and encoding deficits subside. In contrast, Curtiss and colleagues (2001) did not find differences in chronicity across their subtypes. As a result, it is difficult to characterize the relationship between chronicity and memory subtype with any certainty. With regard to injury severity, a retrieval pattern has been observed following mild, moderate and severe TBI (Wiegner & Donders, 1999). Furthermore, individuals with retrieval deficits demonstrate overall cognitive impairment similar to those with poor consolidation or poor retention (Curtiss et al., 2001).

Taken together, the aforementioned studies have provided considerable evidence of memory pattern heterogeneity following moderate to severe TBI. Retrieval deficits have been inferred in one of these subtypes. However, there is not sufficient evidence to suggest a clear relationship between retrieval deficits and various factors including chronicity, injury severity and overall cognitive impairment. Furthermore, the presence or type of psychopathology in individuals that comprise the distinct subtypes has not been adequately investigated.
Neuropsychological Sequelae of Mild Traumatic Brain Injury

Substantial evidence has confirmed neuropsychological impairment in individuals who have sustained moderate to severe TBI. The literature on deficits following mild TBI is more equivocal. Several authors have suggested that impairments following mild TBI are related to factors other than brain injury _per se._

Levin et al. (1987) conducted a multi-center study to investigate the effects of mild TBI on longitudinal neuropsychological performance. Levin and colleagues examined the performance of individuals who had sustained mild TBI at various points in time post-injury. Subjects with various neurological complications, such as focal neurological deficit, intracranial surgery (e.g., for skull fractures) and meningitis, were excluded. Subjects were also screened for various confounding factors including prior neurological disorder (including TBI), alcohol/drug abuse and psychiatric hospitalization. None of the subjects was hospitalized for more than 48 hours or had general anesthesia due to extracranial injuries. Neuropsychological measures administered included Digit Span, list learning of animal names, geometric design recall, Digit Symbol and PASAT.

Data analyses of the initial evaluation indicated that the mild TBI subjects performed more poorly than matched controls, suggesting that the tests administered were sensitive to the early cognitive sequelae of mild TBI. The performance of the TBI subjects was improved from initial levels at one month and three months post-injury. Moreover, the performance of the TBI subjects at three months post-injury did not differ statistically from that of the controls. The authors concluded that “a single uncomplicated minor head injury produces no permanent disabling neurobehavioral impairment in the great majority of patients who are free of preexisting neuropsychiatric disorder and
substance abuse” (p. 234). Constraints in generalizing these findings to subjects with the confounding factors that were excluded in the study were acknowledged. The possibility that the observed improvement with time may have been due to practice effects was also acknowledged.

In his review of the neuropsychological sequelae of mild TBI, Binder (1986) stated that “the argument that persisting, measurable cognitive deficits typically occur after mild head injury is unsupportable, but one must consider the possibility of selective vulnerability (p. 328).” Previous head injury, age and socioeconomic status were cited as potential sources of vulnerability.

More recently, a meta-analysis was conducted to amalgamate findings pertinent to neuropsychological sequelae of mild TBI at least 3 months post-injury (Binder, Rohling, & Larrabee, 1997). Only prospective studies with an attrition rate of less than 50% were included. Effect sizes of mild TBI on neuropsychological performance were quite small. For example, the effect size on measures of memory acquisition was 0.19 standard deviations, whereas the effect size on measures of delayed recall was 0.13 standard deviations. The authors noted that these effect sizes are smaller than the measurement error of many standardized tests. It was concluded that persistence of neuropsychological sequelae in prospective samples was relatively uncommon.

In reviewing clinical implications of these data, Binder (1997) reported that only 7-8% of individuals who sustain mild TBI have persistent symptoms at 6 months post-injury. He noted that neuropsychologists frequently see patients who remain symptomatic following mild TBI, whereas the asymptomatic individuals are rarely seen by neuropsychologists. Multiple risk factors for poor prognosis following mild TBI were
reviewed. These risk factors included: increased age, premorbid psychological factors, limited educational status and prior head injury. Persistence of subjective complaints may also be attributable to malingering, somatoform disorder and/or psychiatric illness. In general, Binder stated that the injury may not be causally related to cognitive deficits, subjective complaints or extent of disability. A differential diagnosis of psychiatric factors was recommended.

Impaired performance on objective tests has been documented in the minority of individuals who present with chronic, subjective complaints following mild TBI. For example, impaired WMS-III performance was reported in individuals who had sustained mild TBI (Fisher, Ledbetter, Cohen, Marmor, & Tulsky, 2000). Subjects classified as having sustained mild TBI (n = 23) reported either a loss of consciousness or a diagnosis of concussion. The majority had sustained their injury as a result of an automobile accident. None had cerebral hemorrhage or skull fracture. Subjects with a Full Scale IQ less than 74 were excluded. Assessments took place a mean of 431 days post-injury.

None of the subjects were considered to be malingering, on the basis of scores on the Rey 15 Item procedure or F-K scores on the Minnesota Multiphasic Personality Inventory - 2nd Edition (MMPI-2). A control group obtained from The Psychological Corporation standardization sample was selected on the basis of individual matching for age, education, sex and ethnicity.

Significant intellectual differences (i.e., WAIS-III scores) between the mild TBI group and the controls were not obtained. However, the mild TBI group demonstrated reduced Auditory Immediate, Auditory Delayed, Visual Delayed and General Memory indices on the WMS-III compared to the controls. In discussing their findings, Fisher and
colleagues (2000) mentioned the controversy surrounding memory deficits in mild TBI. The authors cited evidence that neuropsychological sequelae are not demonstrated in the majority of individuals who sustain mild TBI. However, the authors added that memory impairments can be observed in individuals with mild TBI who are subjectively symptomatic. They pointed to speculation that depression may be responsible for the memory deficits in mild TBI. However, they noted that only two of their subjects were depressed.

Postconcussional Syndrome

Several patients report persistent postconcussional symptoms following mild TBI. Common symptoms of postconcussional syndrome (PCS) include headache, dizziness, irritability, anxiety, insomnia, easy fatiguability, concentration difficulties and memory difficulties (Binder, 1986).

Ponsford et al. (2000) examined postconcussional symptoms in a sample of individuals with mild TBI. These individuals reported symptoms of headaches, dizziness, fatigue and memory difficulties at one week post-injury. These subjective complaints were accompanied by neuropsychological deficits. Neuropsychological impairments were no longer in evidence by three months post-injury. Subjective difficulties had also resolved for the majority of individuals. Nonetheless, 24% of the subjects continued to report symptoms and distress. It was concluded that few individuals presented with persistent complaints at 3 months following mild TBI.

There has been significant debate as to the etiology of PCS symptoms in individuals who have sustained mild head injuries. The persistence of symptoms could be
related to litigation issues, emotional stress, depression and/or subtle neurological symptoms which are not yet understood (Trahan, Ross, & Trahan, 2001).

In reviewing evidence of postconcussive symptoms, Gasquoine (1997) stated that the persistence of subjective symptoms is unlikely accounted for by the memory and attention deficits experienced by individuals in the early phases following mild TBI. Emotional distress, as well as preoccupation with symptoms, is likely to accompany persistence of the symptoms. The author noted that such emotional distress is not specific to mild TBI; it may be observed following other types of injuries.

Gordon et al. (1998) examined a group of 143 individuals who had sustained a "blow to the head with altered mental status" but did not identify themselves as having been disabled by a head injury. They complained of headaches and memory problems, although they did not relate these symptoms to the head injury. It was found that these individuals presented with complaints similar in quality and quantity to those of individuals with known mild TBI. This finding suggests that individuals who have sustained a mild TBI may report subjective complaints, independent of malingering or litigation.

To further complicate the issue, PCS symptoms are reported by the general population, albeit to differing degrees in different samples. For example, individuals engaged in litigation unrelated to head injury endorse a higher number of PCS symptoms (Lees-Haley & Brown, 1993). Furthermore, a significant correlation between scores on the BDI and PCS symptoms has been identified in college students (Beck & Steer, 1987).

Trahan and colleagues (2001) noted the overlap in reported symptomatology in mild TBI and depression and discussed the importance of determining the etiology of
symptoms reported by individuals who have sustained a head injury. They sought to quantify PCS symptoms reported by depressed subjects, mild TBI subjects and healthy controls. The control group included 496 subjects screened for neurological and psychiatric illnesses. A clinically depressed group consisted of 56 participants with BDI scores greater than 20 who were screened for neurological illnesses. The mild TBI group consisted of 40 individuals free of depression who sustained their injuries more than 12 months prior to assessment. All individuals in the mild TBI group had returned to work or school at the time of the assessment. The authors devised a self-report measure, the Postconcussional Index (PCI), which quantifies headache, dizziness, insomnia, irritability, anxiety, depression, frustration, forgetfulness, poor concentration and fatigue. The authors then analyzed the PCI profiles of the three groups.

The depressed subjects reported a higher proportion of PCS symptoms than did the mild TBI or control groups. With regard to forgetfulness, 25% of the depressed group endorsed memory lapses at a frequency of at least twice per week. Similarly, 25% of the mild TBI group reported forgetfulness at this frequency. Furthermore, collapsing all groups, there was a substantial correlation between scores on the PCI and the BDI (r = 0.68, p < 0.001).

In general, these data indicate that depressed individuals report a greater number of PCS symptoms than the general population. As a result, the authors suggested that depression is an important factor to consider when PCS symptoms are more severe or persistent than would be predicted by the severity of the head injury. The endorsement of forgetfulness at similar frequencies in depressed and TBI subjects is also noteworthy.
These findings, however, did not directly address depression concomitant to head injury. Cattelani, Gugliotta, Maravita, and Mazzucchi (1996) studied 53 consecutive patients who had sustained mild TBI and presented with persistent PCS symptoms. The authors divided the subjects into two severity groups, based on the presence of CT or EEG abnormalities on admission. The individuals without detected abnormalities on admission had higher mean elevations on the neurotic triad (i.e., Scales 1 through 3) of the MMPI than did individuals with detected abnormalities. The authors concluded that "organic and psychogenic factors probably coexist in most cases, the mutual proportion and relevance being different in each case" (p. 194).

In their review of depression following mild TBI, Busch and Alpern (1998) commented that "the overlap between the criteria for a major depressive disorder and what constitutes PCS is enigmatic" (p. 95). They stated that "the reporting of memory, attention, concentration and thinking difficulties, sleep disturbance, fatigue, irritability, and anxiety as symptoms of MTBI may obscure diagnosis of a major depressive episode" (pp. 95-96). Conversely, the attribution of these symptoms to depression may obscure a diagnosis of mild TBI. In sum, it would appear that determining the etiology of PCS symptoms following mild TBI, in an individual with concomitant depression, presents many challenges. A similar problem presents itself in the evaluation of objective test performance.

*Neuropsychological Findings: Psychological vs. Organic?*

*Comparison of Neurological and Psychiatric Patients*

Given the deleterious effects of depression on neuropsychological performance, it would appear warranted to question whether depressed individuals are misdiagnosed as
neurologically impaired on the basis of neuropsychological findings. Newman and Sweet (1986) determined the number of depressed subjects that would be misdiagnosed with the Luria-Nebraska Neuropsychological Battery, utilizing various heuristic diagnostic rules. Their subjects were 20 depressed inpatients screened for neurological impairment.

It was found that 35% of the patients exceeded the recommended cutoff for the Intellectual Processes scale, 25% exceeded the cutoff for the Memory scale, and 20% exceeded the cutoff score for the Arithmetic scale. The authors then applied the rule that cerebral impairment should only be diagnosed if two or more elevations are above the recommended cutoffs, as recommended by Golden, Moses, Graber, and Berg (1981). When this criterion was applied, 40% of the depressed subjects were misdiagnosed as neurologically impaired. Utilizing a criterion of three or more elevations above the cutoff, 25% of the depressed subjects continued to be classified as neurologically impaired. Similarly, use of the Impairment scale incorrectly classified 25% of the patients as neurologically impaired. These findings emphasize the importance of understanding neuropsychological deficits which accompany depression in order to substantially account for their effects in the evaluation and diagnosis of individual patients.

Psychologists are frequently asked to differentiate between organic and psychiatric contributions to presentation, although there is a lack of appropriate research dealing with this issue (Coughlan & Hollows, 1984). To contribute to necessary research, the performance of neurological, depressed and control groups was evaluated on a variety of visual and verbal memory tests. The neurological group (n = 53) consisted of individuals with various neurological conditions, most of whom had sustained "very severe" TBI (i.e., PTA greater than 7 days). The depressed group (n = 48) consisted of
inpatients and day patients diagnosed by their psychiatrists. The control group (n = 139) consisted of healthy volunteers. Estimated premorbid IQ scores of the patient groups and estimated IQ scores of the control group were quite similar. Memory tests included recall of a story, learning of a word list over four learning trials, forced-choice recognition of words presented during a rating task, recall of a complex figure, recall of visual designs and facial recognition.

Performance on a memory test was considered to be impaired if it fell 2.0 standard deviations below the mean of the control group. The proportion of neurological patients classified as impaired was greater than the proportion of depressed patients classified as impaired. Within the depressed group, depression scores on the McNair and Lorr scales were not significantly correlated with memory performance, with the exception of Warrington’s forced-choice face recognition (r = -0.29). The authors noted that this correlation was low and did not result in impaired facial recognition performance by a significant number of depressed individuals. The possibility of a restricted range of depression scores, reducing correlations with memory performance, was not addressed by the authors.

Implications of these findings were summarized by the statement that "depression exerts little effect on the various memory tests we have investigated, and that such tests have a useful part to play in differentiating organic conditions from depression" (Coughlan & Hollows, 1984, p.166). Nonetheless, the possibility that inclusion of severely depressed subjects would have increased the neuropsychological deficits observed was acknowledged.
Moreover, the neurological group in this study consisted largely of individuals with severe injuries. The finding that severely injured individuals performed more poorly than moderately depressed subjects is perhaps not surprising. However, it is less clear how mildly or moderately injured subjects would compare to severely depressed subjects. This is likely a more common clinical problem. Furthermore, it should be noted that several of the depressed subjects were classified as impaired on the memory tests. Specifically, 23.4% of the depressed subjects were impaired on at least one of the verbal memory tests, whereas 17.0% were impaired on at least one of the visual memory tests.

In sum, the proportion of depressed individuals classified as neurologically impaired appears lower than would be the case in a group demonstrating hard neurological findings. Nonetheless, a number of depressed individuals perform neuropsychological tasks at levels typically held to indicate neurological impairment. This overlap creates the possibility of misdiagnosis on an individual basis.

The RCFT has also been utilized to characterize the memory performance of psychiatric (n = 30), neurological (n = 30) and control (n = 30) groups (Meyers & Lange, 1994). The three groups differed significantly on Immediate Recall and Delayed Recall, with the neurological patients performing most poorly and the psychiatric patients performing at an intermediate level. The neurological patients performed more poorly than the psychiatric and control groups on the recognition trial. In contrast, the psychiatric group demonstrated recognition scores similar to those of the control group. These findings indicate greater impairment in the neurological patients than in the psychiatric patients. The findings further suggest the possibility of a retrieval deficit in the psychiatric sample.
A discriminant function analysis utilizing recall and recognition measures was also performed. The discriminant function yielded an overall correct classification rate of 77.8%. In general, the combination of recall and recognition measures of the RCFT appears to hold some promise for the discrimination of neurological and psychiatric patients. Unfortunately, the psychiatric group in these studies was a mixed diagnosis sample which was not restricted to depression. The neurological group was also poorly described.

*Depression Concomitant to Neurological Dysfunction*

The aforementioned studies examined psychiatric status independent of neurological impairment, which does not specifically address the issue of comorbidity. Other studies have directly addressed the effect of depression in individuals with clear or suspected neurological insults.

For example, Gass and Russell (1986) examined the effect of depression on memory processes in individuals with suspected neurological impairment who had been referred for assessment. Classification as organic or non-organic was determined following examination by neurology staff. The organic group had various diagnoses including cerebral vascular accident, head injury, alcoholism, tumour, neurodegenerative diseases and multiple sclerosis. A total of 135 organic and 135 non-organic subjects were recruited for participation. All subjects completed the MMPI, the WAIS and the WMS-R. Individuals were considered to be depressed if their score on the Depression scale of the MMPI was at or above a T score of 70.

Performance on Digit Span and Logical Memory were not correlated with the Depression score. In contrast, performance on these tests was significantly correlated
with an index of overall cerebral impairment. Similarly, main effects were found for organicity, but not for depression. Furthermore, interactions were not in evidence. The authors stated that the memory decrements attributable to organicity were much larger than the decrements attributable to depression. They concluded that clinicians should “be conservative in attributing poor verbal memory performance to depression, particularly when there is evidence of organicity” (Gass & Russell, 1986, p. 262). They further commented that clinical lore about the association between depression and memory deficits may be due to the exaggerated complaints of depressed individuals. It should be noted, however, that Digit Span and Logical Memory are not amongst the tests most sensitive to depression related memory deficits in non-neurological samples. Furthermore, the depression scale of the MMPI is sensitive to factors other than depression (e.g., somatic complaints).

The relationship between cognitive complaints, objective test performance and psychological status was subsequently analyzed in 63 males who had sustained TBI of various severities (Gass & Apple, 1997). The subjects were assessed an average of 4.8 years post-injury. None were involved in litigation at the time of assessment. Cognitive complaints were evaluated via self-report on the Cognitive Difficulties Scale (CDS). Factor analysis of the CDS, utilizing a larger sample, produced a seven factor solution. Factor scores on these seven factors were calculated for each of the subjects and were retained for statistical analyses.

Elevations on the Depression scale of the MMPI-2 were found to be correlated with factor scores on attention/concentration, orientation/memory, and errand/name recall, as well as the total CDS score. In particular, the depressed individuals referred for
neurological examination presented with a higher frequency of cognitive complaints than their non-depressed counterparts. The complaints associated with depression demonstrated some specificity; they pertained to attention, memory and everyday recall. Depression elevations were unrelated to factor scores on domestic activities, facial recognition or task efficiency.

To evaluate the relationship between cognitive complaints and objective test performance, the subjects were categorized as “less complaints” or “more complaints” based on their total CDS score. Patients with “more complaints” were found to perform more poorly on Digit Span Forward, Digit Span Backward, Logical Memory I and II and Visual Reproduction I.

The group with more cognitive complaints had a higher mean Depression elevation on the MMPI-2. In addition, the “more complaints” group had higher mean elevations on the Psychasthenia, Anxiety and Obsessional scales. Amongst the MMPI-2 scales, the Psychasthenia scale demonstrated the strongest relationship to cognitive complaints. The Psychasthenia scale of the MMPI-2 overlaps with the Depression, Anxiety and Obsessions content scales. Greene (2000) states that the Psychasthenia scale “appears to be a composite of a number of different items reflecting generalized distress and discomfort that is common to most MMPI-2 scales” (p. 161). Gass and Apple (1997) concluded that cognitive complaints can predict objective test performance, although the complaints score predicts emotional distress to a greater degree. The authors commented that “to an extent, memory complaints reflect level of distress which, in turn can adversely affect memory functioning” (p.297).
In general, this study demonstrated that males with an elevated number of
cognitive complaints following TBI performed more poorly on several objective memory
tests. Furthermore, these TBI patients demonstrated higher elevations on the Depression
scale of the MMPI-2. Whether a direct relationship existed between memory performance
and depression elevations *per se* is difficult to determine. Regardless, the authors
interpreted their data as indicating a relationship between generalized emotional distress
and objective memory performance.

Other researchers have suggested that the relationship between depression and
memory performance is due to poor functional outcome. Satz et al. (1998) investigated
relationships between depression, functional outcome and neuropsychological
performance in 100 individuals who had sustained moderate to severe TBI and 30
controls who had sustained injuries other than to the head. Individuals were assessed
approximately 6 months post-injury. Neuropsychological assessments included Grooved
Pegboard, Colour Trails, Word List Memory (total correct) and RAVLT (Trials 1-5 and
8).

Depression was found to be associated with poor functional outcome, as assessed
by the Glasgow Outcome Scale (GOS). Hence, the head-injured patients were divided
into good outcome and poor outcome groups based on GOS score. The poor outcome
group was further divided into depressed (n = 17) and non-depressed (n = 47) groups.
Classification of depressed and non-depressed individuals was based on Symptom
Checklist 90 - Revised (SCL-90-R) scores. Neuropsychological differences between the
depressed and non-depressed individuals with poor functional outcomes were not in
evidence.
Hierarchical regression analyses were also conducted to preserve the continuity of SCL-90-R scores. Age, education and GOS scores were entered prior to SCL-90-R scores. The results indicated that depression scores were not significant predictors of neuropsychological performance after variance due to age, education and GOS was accounted for. The authors concluded that neuropsychological differences between their groups were due to functional outcome rather than depression status.

In these analyses, the variance associated with functional outcome was removed prior to examining the relationship between depression and neuropsychological performance. Given the association between functional outcome and depression, one might assume that an unknown proportion of shared variance was removed by this statistical operation. Poor functional outcome may be integrally related to depression, rendering covariance of functional outcome rather questionable. Correlational analyses, independent of functional outcome, indicated an inverse relationship between RAVLT performance and an examiner-rated measure of depression (i.e., the Neurobehavior Rating Scale), but not a self-rated measure of depression (i.e., the SCL-90-R).

Bornstein, Miller, and van Schoor (1989) presented evidence which suggested that degree of neuropsychological impairment was related to depression in 125 patients who had sustained head injuries as a result of work-related accidents. Higher depression scores on the MMPI were related to greater impairment on a neuropsychological composite index, despite similar time since injury across groups. The relationship between depression and neuropsychological impairment remained significant after age and education were utilized as covariates. Analyses of performance on individual tests that comprised the impairment index were unfortunately not reported.
The relationship between depression and performance on individual neuropsychological tests was examined by Atteberry-Bennett, Barth, Loyd, and Lawrence (1986). Their goal was to identify demographic and neuropsychological variables that would predict depression scores on the BDI in a sample of individuals who had sustained mild TBI (n = 37). Regression analyses indicated that several neuropsychological variables and demographic data such as age and time since injury were significant predictors of depression scores. The combination of all variables accounted for 34% of the variance in BDI scores. The combination of Vocabulary, Sensory Perceptual and Selective Reminding Test recall scores accounted for 25% of the variance in depression scores.

Higher scores on Vocabulary were related to decreased levels of depression. The authors suggested that individuals with higher Vocabulary scores may have better access to coping strategies. A greater number of errors on the sensory exam was associated with decreased depression. The authors suggested that individuals with significant sensory deficits may be less capable of reporting depression. Impairment of long-term recall on the Selective Reminding Test was most strongly associated with increased depression. Although the intent of Atteberry-Bennett and colleagues (1986) was to identify variables that would predict the occurrence of depression following mild TBI, a causal relationship between variables cannot be determined. As a result, it is not clear whether recall deficits cause depression or depression causes recall deficits.

In a similar study, Ruttan (1998) examined potential relationships between neuropsychological performance and depression scores on the Millon Clinical Multiaxial Inventory - 2nd Edition (MCMI-II) (n = 72) and the MMPI-2 (n = 50) in individuals who
had sustained a mild TBI. Neuropsychological assessments included the Category Test, Logical Memory, Visual Reproduction, Trails B and Consonant Trigrams. Depression scores generally did not predict performance on the neuropsychological measures. The single exception was a significant relationship between Logical Memory performance and MCMI-II depression, indicating reduced narrative recall with increased depression elevations.

In sum, a few nomothetic studies have demonstrated an association between depression and verbal recall in individuals who have sustained a TBI. Some authors have suggested that the association between depression and neuropsychological performance is related to generalized emotional distress or functional outcome. Rather than examining cross-sectional group data, other researchers have described reversibility of certain neuropsychological deficits following remission of depression. This longitudinal approach characterizes the following case studies and a study on the effects of sertraline. 

*Longitudinal Studies: Remission of Depression in Neurological Patients*

Payne (2000) presented a case study of an individual with comorbid TBI and depression. The 26 year-old male had sustained a moderate TBI two years prior. His estimated premorbid abilities were in the High Average to Superior range. At the initial assessment, he demonstrated a significant discrepancy between verbal IQ (125) and performance IQ (95). He also demonstrated reduced information processing speed on the Adult Memory and Information Processing Battery and the WAIS-R. Attention was considered intact since Digit Span and Trails B performance exceeded the 75th percentile. With regard to memory performance, the authors stated that the patient demonstrated difficulty organizing verbal information for later recall, as reflected by his performance
on the List Learning task (Trials 1 to 5) of the Adult Memory and Information Processing Battery. In contrast, his recall of more structured information (e.g., story recall and RCFT) was less impaired. Thus, adequate maintenance of encoded information, as well as resistance to interference, was inferred.

Following four months of cognitive behavioural therapy, this individual’s BDI score had returned to the normal range at which point neuropsychological testing was repeated. Comparison of the initial and subsequent assessment results indicated that processing speed was improved and that the discrepancy between VIQ and PIQ was significantly reduced. A dramatic improvement on the learning trials of the List Learning task (i.e., from the 10th percentile to the 90th percentile) was also observed. The improvement in delayed recall of the list was less dramatic (i.e., from the 75th percentile to above the 90th percentile). The author suggested that list learning performance may be a marker of the effect of depression on memory.

Fogel and Sparadeo (1985) presented the case study of a 28 year-old lawyer who was assessed nine months following resection of a right parieto-occipital arteriovenous malformation. At the first assessment, the individual met criteria for DSM-III major depressive disorder. His Verbal IQ was 125, whereas his Performance IQ was 89. Qualitative analysis of his performance suggested significant impairment of right hemisphere functioning. For example, his Block Design performance included reversals and broken configurations. The numbers on his clock drawing were outside of the circle. His Visual Reproduction drawings were distorted, rotated and lacked overall configuration.
Following eight months of treatment for depression including desipramine and psychotherapy, the individual was reassessed. His Performance IQ remained essentially unchanged. However, his Block Design performance was qualitatively normal. The authors attributed his unchanged score to slowness. His WMS Memory Quotient increased from 110 to 128. The increase was primarily due to improved performance on the Visual Reproduction subtest. His reproductions of the designs were no longer distorted or rotated. Similarly, his clock drawing was normal.

Based on these observations, Fogel and Sparadeo (1985) stated that depression can accentuate the effects of a focal lesion by introducing de novo deficits, reducing sustained effort necessary for compensation or inducing neurochemical changes in the right hemisphere. They concluded that “when brain injury and depression occur in combination, a definitive cognitive prognosis must await resolution of the depression” (p. 123). The conclusion drawn by these authors relies on their attribution of the dramatic improvement to remission of depression rather than recovery from neurological insult. While it is true that the rate of improvement from neurological insult slows toward the end of the first year, the possibility that this individual’s improvement was the result of neurological recovery rather than psychiatric remission cannot be ruled out.

Although not specifically addressed by Fogel and Sparadeo (1985), their patient exhibited significant improvement in list learning performance. At the first assessment, the individual recalled 9/10 words by the fifth trial. He recalled 7 of the 10 items following a delay. At the second assessment, the individual recalled 10/10 words by the second trial of an alternate form. Furthermore, he recalled all of the items following a delay. This contrasted with his performance on Logical Memory, which remained stable
across testing sessions. Although recovery from surgery may have contributed to improved perceptual functioning and visual memory, it seems less likely that improvement in list learning performance was the direct result of neurological recovery. The improvement of list learning performance following remission of depression is consistent with the case presented by Payne (2000).

Neuropsychological changes in a group of patients, following reduction or remission of depression, have also been described (Fann, Uomoto, & Katon, 2001). Depressed subjects with mild TBI (n = 15) participated in a trial of sertraline. The patients completed neuropsychological evaluations prior to drug administration and eight weeks following initiation of sertraline. Depression scores decreased significantly and 10 patients demonstrated complete remission over the treatment period.

Vocabulary and Digit Span scores remained stable across the 8 week treatment period. In contrast, there was dramatic improvement of memory performance. In particular, improvements were noted on Logical Memory, Consistent Long Term Recall of the Buschke Selective Reminding Test and overall score on the Benton Visual Retention Test. Improvement appeared most robust for retrieval of recent verbal information. However, the possibility that practice effects were responsible for observed improvements cannot be ruled out, given the lack of an appropriate control group.

In sum, dramatic memory improvement following remission of depression was reported in two case studies. Memory improvement was similarly observed in a group of patients following sertraline treatment. It is noteworthy that several reports have suggested that list learning performance may be sensitive to depression in neurologically compromised patients.
Sweet, Newman, and Bell (1992) provided a review of the effects of depression in various neurological conditions. Their review of TBI is quite brief. They begin by quoting Prigatano (1987) who asserted that the interplay between affective disorders and cognitive deficits in TBI is important yet neglected. Although several reports have surfaced in the literature since 1992, these have been sporadic and their findings have been inconsistent. The quantity of research in this area is clearly not commensurate with the importance of the issue in clinical practice.

Sweet and colleagues (1992) criticized studies for failing to indicate the representativeness of their subjects to particular clinical populations. The proposed study is specifically relevant to referrals for neuropsychological assessment as a result of known trauma and potential brain injury. The assessment of memory functions, followed by formulations of etiology, prognosis and treatment plans, typically occurs at this level. Referral questions frequently involve the relative contributions of injury and psychological status to observed impairment.

Improved understanding of the interaction between depression and cognitive deficits following TBI will enhance the accuracy of neuropsychologists in assessing the needs of their clients and promote effective treatment (Sweet et al., 1992). It is with this goal in mind that the current study was undertaken.

**Summary of Evidence and Rationale for Study**

**Review of Relevant Findings**

The heterogeneity of memory patterns following moderate to severe TBI has been well documented (Curtiss et al., 2002; Meyers & Meyers, 1995; Millis & Ricker, 1994; Wiegner & Donders, 1999). One of the subtypes that emerges from these studies is
characterized by recognition performance which exceeds free recall performance. The affective status of the individuals who demonstrate this pattern of performance has not been sufficiently investigated.

The presence of memory deficits following mild TBI is relatively controversial. There is some consensus that memory deficits are not observed in the *majority* of individuals who sustain mild TBI. Memory deficits observed in the acute period tend not to persist in individuals free of premorbid neurological or psychiatric disorder who sustain “uncomplicated” mild TBI (Levin et al., 1987). However, memory deficits have been objectively documented in a sample of individuals who continue to report subjective symptoms of mild TBI (Fisher et al., 2000). Regardless, memory deficits generally decrease in magnitude as severity of injury decreases from severe to mild.

With regard to depression in non-neurological samples, greater impairment in recall tasks than in recognition tasks has been reported by several authors (Calev & Erwin, 1985; Fossati et al., 1995; Ilsley et al., 1995). Deficits in explicit recall, accompanied by intact implicit recall, have also been documented (Denny & Hunt, 1992; Danion et al., 1991), even when explicit and implicit conditions are matched for cue provision (Bazin et al., 1994).

With regard to neurological patients who experience concomitant depression, published case studies suggest that list learning performance improves following remission of depression (Fogel & Sparadeo, 1985; Payne, 2000). Improvements on Logical Memory, the Selective Reminding Test and the Benton Visual Retention Test were noted in TBI subjects enrolled in a sertraline trial (Fann, Uomoto, & Katon, 2001). Although these longitudinal studies have consistently indicated a relationship between
depression and list learning performance, replication with larger samples would be required prior to entertaining the reliability or generalizability of these findings.

Results from cross-sectional studies with larger sample sizes have been relatively inconsistent. Ruttan (1998) reported an inverse relationship between Logical Memory performance and MCMI-II depression, but not with MMPI-II depression. In another study, RAVLT performance was found to be inversely related to the depression rating from the Neurobehavior Rating Scale, but not the SCL-90-R (Satz et al., 1998). An inverse relationship between BDI scores and long term recall on the Selective Reminding Test has also been reported (Atteberry-Bennett et al., 1986). Furthermore, the combination of recall and recognition scores on the RCFT has demonstrated some utility in the discrimination of neurological and psychiatric patients (Meyers & Lange, 1994).

On the other hand, some authors have reported little or no effect of depression on various memory measures (Coughlan & Hollows, 1984; Gass & Russell, 1986). Some researchers have suggested that the relationship between depression and memory impairment is due to poor functional outcome (Satz et al., 1998) or generalized psychological distress (Gass & Apple, 1997). Overall, several studies have demonstrated deleterious effects of depression on memory performance in neurological subjects, particularly on verbal recall tasks, although the absence of such a relationship has also been reported.

**Appropriateness of the Comparison Group**

One criticism that has been levelled at studies which have detected differences between depressed and non-depressed groups, in non-neurological samples, is that depressed subjects are typically inpatients, whereas controls are typically outpatients
(e.g., Burt et al., 1995). Clearly, this scenario introduces several confounding factors which could account for group differences. In the current study, all subjects were outpatients referred for neuropsychological evaluation due to head injury.

Sensitivity of the Memory Measures Selected for Study

On occasion, depression related memory deficits have not been observed in younger depressed patients (e.g., Purcell et al., 1997). However, several of these studies have assessed memory with tasks such as recognition and delayed matching to sample, which may not be optimally sensitive to the effects of depression. As a result, the failure to detect group differences in young depressed patients should not necessarily be construed as indicating the absence of an effect of depression on memory. If an effect of depression is sought in younger depressed patients, one should attempt to maximize the sensitivity of the measures. In particular, recall of moderately “structurable” information should be evaluated. Furthermore, the memory tasks selected should be relatively difficult and include a sufficient number of items to be recalled to achieve appropriate sensitivity. Tasks believed to meet these requirements, namely the CVLT-II and the RCFT, were selected for use in the current study.

Similarly, assuming that the effect of depression is similar in psychiatric samples and neurological samples, the sensitivity of the memory measures included in several studies of depression following TBI may be less than optimal. Examples of such memory measures include: story recall (Coughlan & Hollows, 1984; Gass & Russell, 1986), forced-choice recognition of words (Coughlan & Hollows, 1984), facial recognition (Coughlan & Hollows, 1984), Digit Span (Gass & Apple, 1997; Gass & Russell, 1986) and Consonant Trigrams (Ruttan, 1998). Furthermore, given the differential effect of
depression on recall and recognition, both aspects of memory performance should be concurrently evaluated. Previous studies examining the effect of depression in neurological subjects have not consistently compared recall and recognition performance on the same measure.

The assumption made in the previous argument regarding measure selection was that the effect of depression would be similar in psychiatric and head-injured subjects. That this assumption holds has not been adequately demonstrated in the literature. Nonetheless, it was tentatively proposed that memory measures sensitive to depression in psychiatric samples may also be sensitive to depression in TBI samples. Thus, the current study proceeded with the notion that findings from the psychiatric literature may be relevant to depression in neurological studies. As a result, the memory measures selected as dependent variables were the CVLT-II and the RCFT, which are moderately “structurable”, relatively difficult and include both recall and recognition components.

Self-Report Measures of Depression

The measures of depression severity utilized in studies of depression following TBI have varied tremendously. The depression scales of several self-report measures include symptoms that may be attributable to head injury rather than to depression. Symptoms of depression which overlap with the cognitive and psychosocial consequences of TBI include “decreased energy, decreased initiation, irritability, difficulty with decision making, concentration and memory problems, lack of concern regarding physical appearance, decreased libido, sleep disturbance, self-criticism, egocentrism, increased emotionality and flat affect” (Corey, 1987, p. 31). Given the overlap in symptoms of depression and TBI, it is perhaps not surprising that a variety of
depression scales include items that tap sequelae common to TBI. In particular, several items that comprise the depression scale of the MMPI-2 are sensitive to the sequelae of TBI (Gass, 1991). Factor analysis of the BDI scores of TBI patients resulted in a five-factor solution, one of which included symptoms of TBI, rather than the three-factor solution proposed by Beck (Christensen, Ross, Kotasek, Rosenthal, & Henry, 1995; as cited in Rosenthal, Christensen, & Ross, 1998). The depression scale of the SCL-90-R is also weighted with common symptoms of mild to moderate TBI (Woessner & Caplan, 1995).

The PAI was the self-report measure included in the current study. To the knowledge of the author, investigation of the use of the PAI with TBI subjects has not been described in the literature. However, the PAI may be an ideal measure to investigate depression in TBI, given that depression scores are calculated on three non-overlapping subscales reflecting affective, cognitive and physiological aspects of depression. From a review of the items included in each subscale, the cognitive subscale would appear sensitive to low self-esteem and self-depreciation, whereas the physiological subscale would appear sensitive to physiological symptoms such as sleep difficulties and anergia. Both the cognitive and physiological aspects tapped by these subscales could be elevated due to symptoms associated with TBI, independent of the presence of depression. The affective subscale includes items sensitive to depressed mood and loss of interest/pleasure. It should be noted that the items that comprise the affective subscale are remarkably similar to the mandatory criteria for the diagnosis of a major depressive episode, according to recent DSM criteria. Hence, the comparison of the affective subscale score and the total depression scale is particularly relevant in a TBI population.
In general, the fact that the PAI includes three non-overlapping subscales yields some desirable properties. The scores on the three subscales can be utilized to determine whether a substantial proportion of the subjects have total depression scores in the clinical range due to endorsement of cognitive and physiological symptoms in the absence of significant affective symptoms.

In addition, the questions in the PAI are quite simple and straightforward, facilitating interpretation in cognitively impaired individuals. Furthermore, the items that comprise the affective scale demonstrate a high degree of face validity, suggesting greater consistency between item endorsement and information about mood elicited during a clinical interview than would be the case with other self-report measures of depression.

The current study could have proceeded with a design involving continuous depression scores as an independent variable. However, the subjects were divided into depressed and non-depressed groups, based on their total depression score, to compare the recall and recognition performance of the groups. Although several studies have documented differences between depressed and non-depressed groups, many studies have failed to find correlations between measures of depression severity and memory performance. Newman and Sweet (1992) stated that “mere presence of a depressive disorder has been found to be related more consistently to neuropsychological impairment than has severity of depression” (p. 23). Given the exploratory nature of the current study, a group design was considered appropriate.

To maximize the likelihood that subjects categorized as “depressed” were in fact depressed, as opposed to exhibiting slight elevations due to cognitive or physiological
symptoms which could be due to head injury sequelae, only subjects with total depression scores equal to or greater than a T score of 70 were included in the depressed group. Conversely, to minimize "false alarms", only subjects with depression elevations of 60 or less were included in the non-depressed group.

_Heterogeneity of Neuropsychological Performance in TBI Populations_

With regard to previous studies that have investigated the effect of depression in TBI patients, one further issue needs to be considered. The head-injured population is vastly heterogeneous with respect to injury characteristics and neuropsychological performance. Clearly, a substantial proportion of the variance in memory performance would be attributable to differences in injury parameters. Seeking an effect of depression in a sample with such heterogeneity may be analogous to "seeking a needle in a haystack", particularly if the effect size for depression is smaller than the effect size for head injury.

The traditional division of injury severity into mild, moderate, and severe, based on GCS scores, is limited in its ability to capture the extent of cognitive impairment in any particular individual at any particular time. For example, consider the neuropsychological performance of an individual who is free of risk factors for poor prognosis and who sustained a moderate brain injury two years prior to assessment. The performance of this individual may be more similar to the performance of an individual who sustained a mild brain injury one month prior to assessment than to the performance of an individual with risk factors who sustained a moderate brain injury three months prior to assessment.
In order to quantify the severity of cognitive sequelae associated with the injury, at the time of assessment, an Average Performance Rating was calculated for each subject. The following scores were included in the Average Performance Rating: verbal fluency (FAS), Trails B, Coding from the WAIS-III and Grooved Pegboard (average of right and left hand performance). Brief descriptions of these measures are provided in Appendix A. The average of the T scores on these four measures was utilized as a measure of the severity of cognitive sequelae following TBI. This rating was included as a covariate in statistical analyses to control for degree of cognitive dysfunction.

The four measures that comprised the Average Performance Rating were chosen to ensure sensitivity to the sequelae of TBI, while deliberately failing to include memory measures. Verbal fluency has been shown sensitive to injury severity in TBI (Peck et al., 1992; as cited in Spreen & Strauss, 1998). The Trailmaking Test is considered particularly useful in the assessment of cerebral dysfunction arising from mild TBI (Mitrushina, Boone, & D'Elija, 1999). The Processing Speed Index, which includes performance on the Coding subtest, has consistently demonstrated sensitivity to the sequelae of TBI (e.g., Fisher et al., 2000; Martin, Donders, & Thompson, 2000). Finally, the Grooved Pegboard task is a complex, multifaceted motor test that requires several cognitive skills (Francis, Fletcher, Rourke, & York, 1992). The task is known to be highly sensitive to lateralized damage (Mitrushina et al., 1999).

**Purpose and Hypothesis of the Current Study**

The current study was guided by findings of depression related memory impairments in psychiatric samples, despite limited empirical evidence that effects of depression would be similar in a neurological sample. As a result, memory measures
sensitive to depression in psychiatric samples were selected for study. Specifically, relatively difficult tasks which require retention of moderately "structurable" information over a delay, namely the CVLT-II and the RCFT, were selected for study. Furthermore, given the differential effect of depression on recall and recognition in psychiatric samples (i.e., recall is more compromised than recognition), performance on the recall and recognition trials of the CVLT-II and the RCFT were evaluated. The purpose of the current study was to compare the memory performance of depressed TBI subjects on recall and recognition components of these tasks to the performance of non-depressed TBI subjects.

The current study was designed to address the possibility that there would be an effect of depression on memory performance after controlling for degree of cognitive impairment at the time of assessment. The hypothesis was that depressed TBI subjects and non-depressed TBI subjects would differ on recall and/or recognition measures of the CVLT-II and the RCFT after covarying for a measure of cognitive impairment likely to be sensitive to the sequelae of TBI (i.e., the Average Performance Rating).

On the basis of the previously documented asymmetry between recall and recognition performance, two predictions were generated for both the CVLT-II and the RCFT.

1) The recall performance of the depressed group should be reduced relative to the non-depressed group after controlling for the Average Performance Rating.

2) Recognition performance should be less impaired than recall performance for the depressed group. Furthermore, the discrepancy between recall and recognition performance should be greater in the depressed group than in the non-depressed group.
Chapter II

Method

Participants

Participants in the Entire Sample (N = 76)

In total, the data from 76 subjects were obtained. The sex, injury severity, age, education and time since injury of the 76 subjects are presented in Table 1. Mechanisms of injury for the entire sample are described in Table 2.

Participants Included in the Depressed and Non-Depressed Groups (N = 59)

Individuals with depression elevations of at least 70 were included in the depressed group, whereas individuals with a T score of 60 or less were included in the non-depressed group. Of the 76 subjects, 30 had depression scores greater than or equal to a T score of 70, whereas 29 had depression scores less than or equal to a T score of 60. The remaining 17 subjects had intermediate scores on the depression scale. The sex and injury severity of individuals that comprised the depressed and non-depressed groups are presented in Tables 3 and 4 along with relevant Pearson Chi-Square values. Other demographic data for the groups, as well as group comparisons, are presented in Table 5. All 59 subjects who were classified as depressed or non-depressed had completed the RCFT. Hence, analyses involving the RCFT were based on data from these groups.

Participants Included in the CVLT-II Analyses (N = 43)

Of the 59 subjects who were classified as depressed or non-depressed, 43 had completed the CVLT-II. As a result, the CVLT-II analyses in the current study were based on the performance of 19 non-depressed subjects and 24 depressed subjects. The sex and injury severity of the individuals comprising these groups are presented in Tables
6 and 7 along with relevant Pearson Chi-Square values. Other demographic data for the depressed and non-depressed groups, as well as group comparisons, are presented in Table 8.
Table 1
Demographic Data for Entire Sample (N = 76)

<table>
<thead>
<tr>
<th>Sex</th>
<th>Male (N)</th>
<th>33</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female (N)</td>
<td>43</td>
</tr>
<tr>
<td>Injury Severity</td>
<td>Mild (N)</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>Moderate to Severe (N)</td>
<td>26</td>
</tr>
<tr>
<td>Other Demographics</td>
<td>Mean Age in Years (SD)</td>
<td>33.74(9.99)</td>
</tr>
<tr>
<td></td>
<td>Mean Education in Years (SD)</td>
<td>13.13(2.18)</td>
</tr>
<tr>
<td></td>
<td>Mean Time Since Injury in Months (SD)</td>
<td>20.41(20.50)</td>
</tr>
</tbody>
</table>

Table 2
Mechanisms of Injury for Entire Sample (N = 76)

<table>
<thead>
<tr>
<th>Mechanism of Injury</th>
<th>N</th>
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</thead>
<tbody>
<tr>
<td>Automobile Accident</td>
<td>55</td>
</tr>
<tr>
<td>Pedestrian Struck by Automobile</td>
<td>8</td>
</tr>
<tr>
<td>Assault</td>
<td>3</td>
</tr>
<tr>
<td>Snowmobile Accident</td>
<td>3</td>
</tr>
<tr>
<td>Sporting Accident</td>
<td>2</td>
</tr>
<tr>
<td>Cyclist Struck by Automobile</td>
<td>2</td>
</tr>
<tr>
<td>Motorcycle Accident</td>
<td>2</td>
</tr>
<tr>
<td>Falling Object</td>
<td>1</td>
</tr>
</tbody>
</table>
Tables 3 - 5: Depressed and Non-Depressed Groups for RCFT Analyses

Table 3
Sex by Depression Group

<table>
<thead>
<tr>
<th></th>
<th>Male N</th>
<th>Female N</th>
<th>Total N (by Dep Group)</th>
<th>Chi-Square</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed Group</td>
<td>12</td>
<td>17</td>
<td>29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Depressed Group</td>
<td>14</td>
<td>16</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total N (by Sex)</td>
<td>26</td>
<td>33</td>
<td></td>
<td>0.167</td>
<td>0.683</td>
</tr>
</tbody>
</table>

Table 4
Injury Severity by Depression Group

<table>
<thead>
<tr>
<th></th>
<th>Mod. to Severe N</th>
<th>Mild N</th>
<th>Total N (by Dep. Group)</th>
<th>Chi-Square</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed Group</td>
<td>8</td>
<td>21</td>
<td>29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Depressed Group</td>
<td>13</td>
<td>17</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total N (by Injury Severity)</td>
<td>21</td>
<td>38</td>
<td></td>
<td>1.595</td>
<td>0.207</td>
</tr>
</tbody>
</table>

Table 5
Comparison of Demographic Factors in Depressed and Non-Depressed Groups

<table>
<thead>
<tr>
<th></th>
<th>Depressed Group M(SD)</th>
<th>Non-Depressed Group M(SD)</th>
<th>t</th>
<th>U</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>35.66(9.21)</td>
<td>30.60(10.84)</td>
<td>---</td>
<td>310.50</td>
<td>0.059</td>
</tr>
<tr>
<td>Education</td>
<td>12.69(2.11)</td>
<td>13.47(2.10)</td>
<td>1.420</td>
<td>---</td>
<td>0.161</td>
</tr>
<tr>
<td>Months post-injury</td>
<td>25.62(24.86)</td>
<td>18.97(19.11)</td>
<td>---</td>
<td>368.50</td>
<td>0.313</td>
</tr>
</tbody>
</table>
Tables 6-8: Depressed and Non-Depressed Groups for CVLT-II Analyses

Table 6

Sex by Depression Group

<table>
<thead>
<tr>
<th></th>
<th>Male N</th>
<th>Female N</th>
<th>Total N (by Dep Group)</th>
<th>Chi-Square</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed Group</td>
<td>10</td>
<td>14</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Depressed Group</td>
<td>11</td>
<td>8</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total N (by Sex)</td>
<td>21</td>
<td>22</td>
<td></td>
<td>1.118</td>
<td>0.290</td>
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</tbody>
</table>

Table 7

Injury Severity by Depression Group

<table>
<thead>
<tr>
<th></th>
<th>Mod. to Severe N</th>
<th>Mild N</th>
<th>Total N (by Dep. Group)</th>
<th>Chi-Square</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed Group</td>
<td>6</td>
<td>18</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Depressed Group</td>
<td>9</td>
<td>10</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total N (by Injury Severity)</td>
<td>15</td>
<td>28</td>
<td></td>
<td>2.336</td>
<td>0.126</td>
</tr>
</tbody>
</table>

Table 8

Comparison of Demographic Factors in Depressed and Non-Depressed Groups

<table>
<thead>
<tr>
<th></th>
<th>Depressed Group M(SD)</th>
<th>Non-Depressed Group M(SD)</th>
<th>t</th>
<th>U</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>35.17(8.99)</td>
<td>30.37(11.39)</td>
<td>------</td>
<td>163.500</td>
<td>0.114</td>
</tr>
<tr>
<td>Education</td>
<td>12.46(2.08)</td>
<td>13.21(2.23)</td>
<td>1.141</td>
<td>-------</td>
<td>0.261</td>
</tr>
<tr>
<td>Months post-injury</td>
<td>20.71(18.32)</td>
<td>17.47(17.82)</td>
<td>------</td>
<td>196.000</td>
<td>0.433</td>
</tr>
</tbody>
</table>
Measures

Personality Assessment Inventory (Morey, 1991)

The PAI is a self-report personality questionnaire containing 344 items. All items are rated on a Likert scale that ranges from 1 to 4, or from false to very true. There are four validity scales and 18 clinical scales, with raw scores converted to T scores. Each scale consists of unique test items that do not overlap with other scales. The clinical scale relevant to the current study is the DEP, or depression, scale. The DEP scale is comprised of three non-overlapping subscales, which represent affective, cognitive, and physiological aspects of depression.

California Verbal Learning Test - II (Delis, Kramer, Kaplan, & Ober, 2000)

The CVLT-II is a multi-trial categorized list learning task. Each of the two lists of 16 words consists of exemplars from four semantic categories. The examiner begins by reading List A and the examinee is asked to recall as many words as possible, without regard to order. A total of five learning trials are conducted in this fashion. List B is then read once to the examinee who is asked to recall it immediately. This is known as the interference trial, or simply as List B. Immediately following the interference trial, the examinee is instructed to recall words from List A. This is the Short Delay Free Recall trial. This is followed by the Short Delay Cued Recall trial, in which the examinee is asked to recall items from particular semantic categories, one category at a time. For example, the examinee is asked to recall all tools listed in the first list. The examinee is then instructed to recall all fruits in the first list. Subjects are not informed that subsequent trials will take place. Following a 20 minute delay, free recall is assessed and cued recall is subsequently assessed. These trials are known as Long Delay Free Recall
and Long Delay Cued Recall, respectively. The examinee is then presented with 48 words and is asked to respond “yes” if the word was on List A and “no” if the word was not on List A. Distractors on the recognition trial include words presented on Trial B and words that are semantically related to the target words. This recognition trial is referred to as the Yes-No Recognition trial.

In the current study, the Long Delay Free Recall score was the recall measure utilized in all analyses. The score for this trial is calculated by converting the sum of correct responses to a z score. Recognition Discriminability (d’) for the Yes-No Recognition Trial is calculated by subtracting the z score of the False Positive Rate from the z score of the Hit Rate.

Rey Complex Figure Test (Meyers & Meyers, 1995)

The examinee is asked to reproduce an abstract complex figure on a blank sheet of paper. There is no time limit for the Copy trial. Examinees are not forewarned that they will later be asked to recall the figure. Following a three minute delay, the examinee is asked to reproduce the figure to the best of their ability. This is the Immediate Recall trial. Following a further 20 minute delay, the examinee is again asked to reproduce the figure from memory. This constitutes the Delayed Recall trial. Following another brief delay, the examinee is provided with 24 figures. Of the 24 figures, 12 figures are elements of the complex figure, whereas the other 12 figures are distractor items. The examinee is instructed to identify the figures that were elements of the original complex figure. This constitutes the Recognition trial.

The Delayed Recall score was utilized in the current study. Scoring of the RCFT figure involves awarding points for accurate reproduction of the elements of the figure, as
well as the correct placement of these elements. The maximum possible raw score is 36. Detailed scoring instructions are provided in the Professional Manual (Meyers & Meyers, 1995). The Recognition trial is scored by determining the correct number of responses (i.e., true positives + true negatives). The Delayed Recall score and the Recognition score are converted to a T score.

Brief descriptions of the four measures that comprise the Average Performance Rating can be found in Appendix A.

Procedure

The neuropsychological test results of individuals referred due to closed head trauma were obtained retrospectively. Consecutive referrals since 1999 were considered for inclusion if the assessment had been completed and scored at the time of data collection. Subjects were included if data were available for a substantial portion of the WAIS-III, the WMS-III and at least one of the RCFT or the CVLT-II. Based on a priori criteria, only subjects between the ages of 18 and 49 years at the time of assessment were included. Furthermore, individuals who sustained their injury prior to age 15 were excluded.

With regard to premorbid or concomitant conditions, inclusion criteria were relatively broad in order to maintain the representativeness of the sample. Only subjects whose performance was considered grossly affected by a well documented condition unrelated to the brain injury were excluded. For example, subjects with psychosis, schizophrenia (or schizoaffective disorder), mental retardation or severe developmental disability were excluded. Furthermore, subjects who could not complete a substantial portion of the subtests (e.g., due to gross motor disability or aphasia) were excluded. All
subjects demonstrated sufficient facility with the English language. Subjects were not excluded due to conditions such as premorbid depression, prior brain injury, substance abuse and/or learning disability.

All subjects presented as meeting criteria for having sustained at least a mild brain injury, according to the criteria of the Mild Traumatic Brain Injury Committee of the American Congress of Rehabilitation Medicine (1993). Specific criteria included at least one of the following: any period of loss of consciousness, any loss of memory (whether retrograde or anterograde), any alteration in mental state, or focal neurological deficits. Consistent with the aforementioned criteria, individuals meeting one or more of the following criteria were considered to have sustained a moderate to severe brain injury: loss of consciousness greater than 30 minutes, Glasgow Coma Scale less than 13 and post-traumatic amnesia greater than 24 hours.

Quantification of the severity of cognitive deficits was achieved by calculating an average T score on verbal fluency (FAS), Trails B, Coding from the WAIS-III and Grooved Pegboard (average of right and left hand performance) for each subject. The derived score was termed the Average Performance Rating. Brief descriptions of the measures included in the Average Performance Rating are provided in Appendix A.

Individuals with depression elevations of at least 70 were included in the depressed group, whereas individuals with a T score of 60 or less were included in the non-depressed group. The memory scores of the groups were compared via ANCOVA and repeated measures analyses, utilizing the Average Performance Rating as a covariate. In particular, the dependent memory measures consisted of Long Delay Free Recall and
Recognition Discriminability for the CVLT-II and Delayed Recall and Recognition for the RCFT.
Chapter III

Results

Given that the current work focuses upon the comparison of the depressed group to the non-depressed group, only the data from the subjects that comprised these groups were included in the analyses subsequently described. As a result, the data from the "borderline" depression group (i.e., depression T scores between 60 and 70) were not included in subsequent analyses, unless inclusion of the entire sample is explicitly stated.

Data Screening

Prior to commencing data analyses, the data were examined via several exploratory methods (e.g., scatterplots, boxplots). Inspection of the RCFT data indicated that several subjects had the lowest T score possible (i.e., a score of 20) on the recall and recognition components. Specifically, of the 59 subjects, 13 had T scores of 20 on the Delayed Recall trial and 8 had T scores of 20 on the Recognition trial. In order to preserve the variability of scores occurring below a T score of 20, raw scores for the Delayed Recall trial and the Recognition trial were utilized in all analyses. Age was negatively correlated with the recall score, $r(59) = -0.361, p = 0.005$, but not the recognition score, $r(59) = -0.049, p = 0.715$. As a result, age was utilized as a covariate in subsequent analyses involving the Delayed Recall scores.

The raw RCFT scores (i.e., Delayed Recall and Recognition) and the CVLT scores (i.e., Long Delay Free Recall and Recognition Discriminability) were examined for extreme univariate outliers relative to their group, as was the Average Performance Rating. Extreme scores were detected for the CVLT-II data. In particular, one subject had $z$ scores of -3.0 for the free recall and the recognition trials. Another subject had a free
recall score of $z = 0$, whereas her score on recognition was $z = -3.0$. The extreme nature of these recognition scores is clearly demonstrated in the boxplot, scatterplot and residuals plot presented in Appendix B. Both subjects were young women who had sustained mild injuries. Hence, injury parameters are unlikely to account for the degree of impairment suggested by their recognition scores. Given the extreme nature of these scores and their potential influence on subsequent analyses, the CVLT-II data from both of these subjects were excluded from subsequent analyses.

*Examination of the PAI Depression Subscales*

The subjects were classified as depressed or non-depressed on the basis of their total depression score. The depression subscale scores were subsequently examined to determine the concordance between the subscale scores and the overall depression score in each of the depression groups. The total depression scale was strongly correlated with the various subscale scores. Specifically, the correlations of the total scale with the affective, cognitive and physiological subscales were: $0.942 \ (p = 0.000)$, $0.865 \ (p = 0.000)$ and $0.891 \ (p = 0.000)$, respectively.

Of the 30 subjects classified as non-depressed, none had elevations at or above a T score of 65 on the affective subscale or cognitive subscale. However, six subjects (20%) had elevations at or above a T score of 65 on the physiological subscale. Hence, individuals classified as non-depressed on the basis of a T score of 60 or lower on the total depression scale did not report affective symptoms to a clinically significant degree. However, several of the non-depressed subjects had elevations in the clinical range on the scale intended to assess physiological symptoms of depression.
Of the 29 subjects classified as depressed, two subjects (7%) had affective scores below a T score of 65, whereas 7 subjects (24%) had cognitive scores below this score. With regard to the physiological subscale, two subjects (7%) had scores in the normal range. In general, the majority of subjects classified as depressed, on the basis of a total depression score at or above a T score of 70, reported affective symptoms to a clinically significant degree. However, approximately one quarter of the depressed subjects did not report substantial cognitive symptoms.

Overall, the classification of subjects on the basis of the total depression scale score was relatively successful in identifying subjects who reported significant affective symptomatology and those who did not. Only two individuals (approximately 3%) had affective subscale elevations which were discordant with their total depression scale elevation. In sum, the PAI total depression scale adequately reflected endorsement of affective symptoms which resemble the first two diagnostic criteria for depression (i.e., the mandatory criterion of either depressed mood or loss of interest/pleasure).

It should be noted, however, that the subscale scores of subjects with borderline levels of depression, as determined by the total scale, were not examined. Based on criteria established a priori, subjects with borderline scores on the total depression scale were not classified as depressed or non-depressed, increasing the probability that subjects within the respective categories were either clearly depressed or clearly not depressed.

*Utility of the Average Performance Rating*

Ideally, use of the Average Performance Rating as a covariate should remove variance in memory performance associated with the cognitive sequelae of TBI, while
failing to remove variance associated with depression. Hence, analyses were conducted to
determine the extent to which these ideal conditions applied to the current data.

First, one must consider whether there is a probable relationship between the
Average Performance Rating and the cognitive sequelae of TBI. Although acute
indicators of severity have limited accuracy in predicting cognitive outcome, the
moderate to severe injury group should demonstrate more impaired performance than the
mild injury group. In fact, the mean Average Performance Ratings for the mild TBI and
moderate to severe TBI groups were compared and failed to reach significance, \(t(57) = 1.746, p = 0.086\). In this context, it should be noted that the individuals with moderate to
severe TBI were assessed at a mean time since injury of 29.57 months (\(SD = 28.59\)),
whereas the individuals with mild TBI were assessed at a mean time since injury of 18.18
months (\(SD = 16.80\)). The discrepant levels of chronicity across the groups might account
for the failure of the Average Performance Rating comparison to reach significance.
Regardless, given that the comparison approached significance, it was considered
worthwhile to examine whether a group difference would be significant in a slightly
larger sample. In fact, utilizing the data from all 76 subjects regardless of depression
group, the moderate to severe TBI group had significantly lower Average Performance
Ratings than did the mild TBI group, \(t(74) = 2.135, p = 0.036\). Overall, the derived
Average Performance Rating scores appeared sensitive to injury severity.

Second, one must ensure that the Average Performance Rating is not substantially
related to depression, such that variance due to depression is not inadvertently being
removed by the covariance operation. Data analyses indicated that the Average
Performance Rating was not correlated with depression score, \(r(59) = -0.181, p = 0.171\).
This continued to be the case when the entire sample was included in the correlational analysis, $r(76) = -0.155$, $p = 0.180$. Hence, the Average Performance Rating appeared sensitive to the sequelae of TBI, while failing to be substantially associated with depression.

Furthermore, the use of the Average Performance Rating as a covariate necessitates a correlation between it and the memory measures utilized as dependent variables. The correlations between the Average Performance Rating and the memory scores utilized in the ANCOVA analyses are presented in Table 9. As illustrated, all correlations were significant and in the direction that would be predicted. Specifically, individuals with greater cognitive impairment (i.e., lower Average Performance Rating scores) had significantly lower memory scores. The correlation between the Average Performance Rating and the RCFT Delayed Recall raw score, controlling for age, was also significant, $r(56) = 0.3866$, $p = 0.003$.

Analyses were also conducted to ensure that the assumptions of linearity, homogeneity of variance and homogeneity of regression were tenable. The assumptions were met for all relevant analyses.

*California Verbal Learning Test -II Analyses*

The Long Delay Free Recall scores of the depressed and non-depressed groups were compared in an ANCOVA analysis, utilizing the Average Performance Rating as a covariate. The Average Performance Rating was a significant covariate for the recall score, $F(1,40) = 17.455, p = 0.000$. After controlling for the Average Performance Rating, the effect of depression was also significant $F(1,40) = 11.902, p = 0.001$. The effect size for depression ($\eta^2 = 0.229$) was smaller than the effect size for the Average
Table 9

Correlations Between the Average Performance Rating and Dependent Memory Measures

<table>
<thead>
<tr>
<th>Dependent Memory Measure</th>
<th>Correlation</th>
<th>p</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVLT-II Long Delay Free Recall (z score)</td>
<td>0.587</td>
<td>0.000**</td>
<td>43</td>
</tr>
<tr>
<td>CVLT-II Recognition Discriminability (z score)</td>
<td>0.657</td>
<td>0.000**</td>
<td>43</td>
</tr>
<tr>
<td>RCFT Delayed Recall (raw score)</td>
<td>0.430</td>
<td>0.001**</td>
<td>59</td>
</tr>
<tr>
<td>RCFT Recognition (raw score)</td>
<td>0.547</td>
<td>0.000**</td>
<td>59</td>
</tr>
</tbody>
</table>

** indicates satisfaction of significance at an 0.01 alpha level
Performance Rating ($\eta^2 = 0.304$). Review of the estimated adjusted means (evaluated at an Average Performance Rating T score of 41.87) indicated that the recall score was significantly lower in the depressed group ($M = -1.098, SE = 0.211$) than in the non-depressed group ($M = 0.0184, SE = 0.238$). In Figure 1, a scatterplot is presented to illustrate the relationship between the CVLT-II Long Delay Free Recall score and the Average Performance Rating. The line of best fit for depressed and non-depressed subjects is superimposed to demonstrate the group difference.

ANCOVA was also utilized to analyze group differences on the Recognition Discriminability index of the CVLT-II. The Average Performance Rating was significantly related to the recognition score, $F(1,40) = 27.619, p = 0.000$. There was also a significant between-subjects effect of depression after covarying the Average Performance Rating, $F(1,40) = 14.690, p = 0.000$. The effect size of depression on the recognition score was considerable ($\eta^2 = 0.269$), although the effect size of the Average Performance Rating was larger ($\eta^2 = 0.408$). Review of the estimated adjusted means (evaluated at a T score of 41.87 for the Average Performance Rating) indicated that recognition was reduced in the depressed group ($M = -0.690, SE = 0.158$) relative to the non-depressed group ($M = 0.240, SE = 0.179$). A scatterplot is presented in Figure 2 to illustrate the relationships among the variables.

Overall, the ANCOVA analyses indicated that both recall and recognition measures were reduced in the depressed group relative to the non-depressed group after controlling for a measure of cognitive impairment. The possibility remained that recall was more impaired than recognition in the depressed group, whereas the discrepancy between recall and recognition was negligible in the non-depressed group. Hence, a
Figure 1. CVLT-II Long Delay Free Recall z scores as a function of the Average Performance Rating. The line of best fit for each of the depression groups is superimposed.
Figure 2. CVLT-II Recognition Discriminability z scores as a function of the Average Performance Rating. The line of best fit for each of the depression groups is superimposed.
repeated measures analysis, utilizing the Average Performance Rating as a covariate, was conducted to determine whether there was an interaction between depression and CVLT-II trial (i.e., recall vs. recognition). The interaction term was not significant, $F(1, 40) = 0.390, p = 0.536$, suggesting that the discrepancy between recall and recognition was no greater in depressed subjects than in non-depressed subjects. The estimated mean scores on the recall and recognition trials (evaluated at a T score of 41.87 on the Average Performance Rating), for each group, are illustrated in Figure 3.

The combination of reduced recall and recognition suggests the possibility of difficulty encoding the list. Furthermore, the literature on depression suggests the possibility of reduced semantic processing, at least in psychiatric samples. As a result, additional analyses were conducted to determine whether the groups differed on the semantic clustering ratio and the learning slope of the CVLT-II.

The Shapiro-Wilk statistic indicated violation of normality for semantic clustering scores in the non-depressed group ($0.860, df = 19, p = 0.010$) and the depressed group ($0.829, df = 24, p = 0.010$). As a result, Mann-Whitney U analyses were conducted to evaluate the possibility of group differences on the semantic clustering scores. A significant difference in the semantic clustering ratios of the groups was detected, $U = 119.00, p = 0.006$. The depressed group had a lower mean clustering score ($M = -0.3750, SD = 0.6635$) than the non-depressed group ($M = 0.7895, SD = 1.6100$). Consistent with this finding, depression elevations were negatively correlated with the clustering ratio when the depression score was evaluated as a continuous variable, $r(43) = -0.391, p = 0.01$, even after controlling for the Average Performance Rating, $r(40) = -0.365, p = 0.017$. In sum, these findings indicated that the depressed group utilized the semantic
Figure 3. CVLT-II adjusted mean scores on the recall and recognition trials by depression group. Although the depressed group achieved lower scores than the non-depressed group on both trials, the lines are parallel and there is no interaction.
organization of the list to a lesser degree than did the non-depressed group in learning the list.

In addition, the mean learning slope score was steeper in the non-depressed group ($M = 0.3947$, $SD = 1.0875$) than in the depressed group ($M = -0.5208$, $SD = 0.9943$) and this difference was statistically significant, $t(41) = 2.877$, $p = 0.006$. The learning slope score was also found to correlate negatively with depression scores, $r(43) = -0.380$, $p = 0.012$, even after controlling for the Average Performance Rating, $r(40) = -0.3500$, $p = 0.023$. In sum, depression scores of the PAI were inversely related to the semantic clustering ratio and the learning slope of the CVLT-II. Moreover, this relationship appears unlikely to be accounted for by level of cognitive impairment arising from the injury.

In sum, both the Long Delay Free Recall score and the Recognition Discriminability score of the CVLT-II were found to be reduced in the depressed group, relative to the non-depressed group, after covarying for the Average Performance Rating. Follow-up analyses indicated that the semantic clustering ratio and the learning slope of the CVLT-II were reduced in the depressed group. Moreover, it appeared unlikely that cognitive impairment arising from the injury accounted for group differences on the semantic clustering ratio and the learning slope.

*Rey Complex Figure Test Analyses*

For the RCFT analyses, the data of 30 non-depressed and 29 depressed subjects were available. To compare the raw Delayed Recall scores of the depressed and non-depressed groups, an ANCOVA was conducted, utilizing both age and the Average Performance Rating as covariates. The Average Performance Rating was a significant
covariate, $F(1,55) = 7.607, p = 0.008$, whereas age approached significance, $F(1,55) = 3.969, p = 0.054$. After covarying for age and the Average Performance Rating, the effect of depression on the RCFT recall score was significant, $F(1,55) = 4.477, p = 0.039$. The effect size ($\eta^2$) for depression was 0.075, which was smaller than the effect size for the Average Performance Rating ($\eta^2 = 0.121$). Review of the estimated adjusted means (evaluated at 33.09 years of age and a T score of 42.44 for the Average Performance Rating) indicated that the non-depressed group had higher raw RCFT recall scores ($M = 17.951, SE = 1.121$) than did the depressed group ($M = 14.482, SE = 1.141$).

The scatterplot of the relationship between RCFT Delayed Recall scores and the Average Performance Rating, for both groups, is presented in Figure 4. Note, however, that this plot is not an entirely accurate representation of the ANCOVA results since age is not a covariate in the scatterplot. Nonetheless, the scatterplot was included for illustration purposes.

Raw RCFT Recognition scores were also subjected to ANCOVA analysis, although the Average Performance Rating was the only covariate utilized. Again, a significant effect of the Average Performance Rating was observed, $F(1, 56) = 19.927, p = 0.000$. A between-subjects effect for depression, after controlling for the Average Performance Rating, approached but did not reach statistical significance, $F(1,56) = 3.039, p = 0.087$). The adjusted means (evaluated at an Average Performance Rating of 42.44) were relatively similar across the groups. Specifically, the depressed group had an adjusted mean of 18.906 ($SE = 0.371$), whereas the non-depressed group had an adjusted mean of 19.825 ($SE = 0.364$).
Figure 4. RCFT Delayed Recall raw scores as a function of the Average Performance Rating. The line of best fit for each of the depression groups is superimposed.
The data are illustrated in Figure 5. Note that the regression slopes are not entirely parallel. However, there was not a significant interaction between depression group and the Average Performance Rating, $F(1, 55) = 1.020, p = 0.317$.

In sum, the RCFT Delayed Recall score was significantly lower in the depressed group than in the non-depressed group after covarying the effects of the Average Performance Rating and age. In contrast, group differences on the RCFT Recognition score approached but did not reach statistical significance.

A repeated measures analysis was conducted to confirm whether the discrepancy between recall and recognition was greater in the depressed group than in the non-depressed group. In this analysis, the Average Performance Rating was utilized as a covariate, whereas age was not. As previously described, age was negatively correlated with the recall score but was not correlated with the recognition score. Age was not a significant covariate in the ANCOVA analysis of the RCFT Delayed Recall scores. As a result, age was not utilized as a covariate in the repeated measures analysis.

The interaction between depression group and RCFT trial (i.e., recall vs. recognition) approached significance after covarying for the Average Performance Rating, $F(1, 56) = 3.785, p = 0.057$. The adjusted mean scores (evaluated at a T score of 42.44 on the Average Performance Rating) on the Delayed Recall trial and the Recognition trial, for each group, are presented in Figure 6. Review of this figure suggests that recall is more impaired than recognition in the depressed subjects, and to a greater degree than in non-depressed subjects. However, the relationship depicted in Figure 6 fails to reach statistical significance.
Figure 5. RCFT Recognition raw scores as a function of the Average Performance Rating. The line of best fit for each of the depression groups is superimposed.
Figure 6. RCFT adjusted mean scores on the recall and recognition trials by depression group. The interaction between depression group and RCFT trial approached significance.
In summary, the depressed group achieved significantly reduced scores on the Delayed Recall trial of the RCFT relative to the non-depressed group, after covarying the Average Performance Rating. Differences on the Recognition scores of the RCFT failed to reach significance in the ANCOVA analyses. Finally, repeated measures analyses were conducted to determine whether recall was more impaired than recognition in the depressed group, and to a greater degree than was observed in the non-depressed group. The relevant interaction term approached but did not meet statistical significance.
Chapter IV

Discussion

The purpose of the current work was to compare the memory performance of depressed TBI subjects and non-depressed TBI subjects. In particular, the hypothesis was that differences between the groups would emerge on the recall and/or recognition components of the CVLT-II and the RCFT after covarying for a composite score reflecting neuropsychological impairment which was labelled the Average Performance Rating. On the basis of the previously documented asymmetry between recall and recognition performance, two predictions were generated for both the CVLT-II and the RCFT. 1) The recall performance of the depressed group should be reduced relative to the non-depressed group after controlling for the Average Performance Rating. 2) Recognition performance should be less impaired than recall performance for the depressed group. Furthermore, the discrepancy between recall and recognition performance should be greater in the depressed group than in the non-depressed group.

To compare the performance of the depressed and non-depressed subjects, scores achieved on the recall and recognition components of the CVLT-II and the RCFT were analyzed independently via ANCOVA, utilizing the Average Performance Rating as a covariate. Repeated measures analyses were subsequently conducted to determine whether recall was impaired to a greater degree than was recognition.

Main Findings Concerning the CVLT-II

With regard to the CVLT-II, the current findings indicated that the depressed group achieved lower recall and recognition scores than did the non-depressed group. However, recall was no more impaired than was recognition. In follow-up analyses, the
semantic clustering ratio and the learning slope were found to be reduced in the depressed group. Taken together, these findings strongly suggest deficient encoding in the depressed TBI subjects. Furthermore, the depressed group appears to have utilized the semantic organization of the list to a lesser degree, hindering their learning of the list.

In the literature documenting memory impairment in depressed subjects without frank neurological insult, the reduction of both recall and recognition scores has been described in a few studies. For example, impaired recall and recognition on a modified version of the RAVLT was noted in depressed subjects when limited opportunity was presented to learn the information (Brand et al., 1992). It was suggested that insufficient familiarity, leading to greater demands on effort, was responsible for less than adequate encoding in the depressed subjects. However, recognition deficits were only observed following a single learning trial. The CVLT-II includes five learning trials. It would be difficult to argue that exposure to the words on the CVLT-II had been inadequate. Rather, inferring that the depressed subjects failed to fully benefit from repetition, due to reduced semantic processing and/or encoding, seems a more adequate explanation.

The finding of simultaneously reduced recall and recognition was also reported by Wolfe and colleagues (1997) who administered a modified version of the RAVLT to unipolar patients. The learning slope was also found to be reduced. Overall, the findings of Wolfe and colleagues (1997) were consistent with an encoding deficit and were quite analogous to findings in the current study. The RAVLT, however, differs from the CVLT-II in that it includes words that are not semantically related.

Studies which have utilized moderately “structurable” lists (e.g., Channon et al., 1993) or the CVLT in particular (e.g., Fossati et al., 1995; Massman et al., 1992) have
yielded evidence more consistent with a retrieval deficit. Specifically, these studies have shown that free recall tends to be compromised in depressed subjects, whereas recognition is relatively intact. The results from the current study were inconsistent with those from the aforementioned studies which included depressed subjects without TBI. It appears plausible that the differences emerged due to the discrepant populations studied. In general, the memory profiles associated with depression may differ in psychiatric and neurological patients. In particular, the effect of depression on list learning performance may be different in TBI subjects than in subjects without TBI. The results from a discriminant function analysis at least partially support the suggestion that depression related patterns of performance on neuropsychological tests are different in neurological and psychiatric samples (Aloia, Long, & Allen, 1995).

With regard to studies investigating a potential influence of depression specifically in neurological patients, recall deficits on list learning tasks have been observed relatively consistently. One such finding pertained to an inverse relationship between BDI scores and long-term recall on the Selective Reminding Test (Atteberry-Bennett et al., 1986). Similarly, Satz and colleagues (1998) reported an inverse relationship between examiner-rated depression and RAVLT performance. In a longitudinal study, deficits in Consistent Long Term Recall of the Selective Reminding Test abated over the course of a sertraline trial in depressed subjects who had sustained mild TBI (Fann et al., 2001). Furthermore, reversibility of list learning deficits following remission of depression has also been noted in two case reports of neurologically compromised patients (Fogel & Sparadeo, 1985; Payne, 2000). The results of the current study are consistent with the aforementioned findings of reduced free recall on list
learning tasks in neurological patients with concomitant depression. The current study also identified reduced Recognition Discriminability on the CVLT-II in depressed TBI subjects. Unfortunately, prior studies of depression in TBI subjects have provided little information about the possibility of recognition deficits.

Overall, the results pertaining to CVLT-II performance were inconsistent with the hypothesis that recall would be more impaired than recognition, hence suggesting a retrieval deficit. Rather, the results quite consistently suggested that encoding deficits were responsible for impaired recall and recognition performance in the depressed TBI subjects. Furthermore, the reduced semantic clustering demonstrated by the depressed subjects suggests difficulty utilizing the semantic organization of the list to facilitate subsequent recall. Interestingly, Payne (2000) noted that their single depressed patient who had sustained a moderate TBI exhibited difficulty organizing verbal information for later recall and that this improved following remission of depression.

The failure to fully benefit from semantic processing in recall tasks has been noted in depressed patients without TBI (Weingartner et al., 1981). In addition, it has been suggested that impairment in effortful encoding may be responsible for the memory deficits of depressed subjects (Zakzanis et al., 1998). Overall, the findings in the current study could support either: 1) impaired effortful encoding or 2) a failure to benefit from semantic organization in the depressed TBI subjects. Alternately, a combination of both explanations may be appropriate. Specifically, the failure to engage in effortful encoding may lead to decreased ability to benefit from the semantic organization of the list, resulting in impaired recall and recognition on the CVLT-II.
Main Findings Concerning the RCFT

The findings involving the RCFT were more consistent with the original hypothesis. Specifically, recall was found to be reduced in the depressed group compared to the non-depressed group, whereas significant differences on recognition performance failed to emerge. When the data were subjected to repeated measures analysis, the interaction between depression and RCFT trial approached significance. Hence, the findings from the current study generally supported the notion that recall of the Rey Figure was impaired in depressed subjects, whereas recognition was relatively intact.

Intact recognition scores suggest that encoding has proceeded appropriately. Furthermore, intact recognition following a delay suggests adequate storage of information. Impaired recall scores, accompanied by intact recognition scores, may reflect difficulty retrieving adequately encoded and stored information from long-term memory. Thus, a discrepancy between recall and recognition, favouring recognition, is typically construed as indicating a retrieval deficit. An alternate possibility is that encoding is only partial, resulting in fragmented memory traces which are best accessed when sufficient cues are provided to match to the fragmented memory traces (see Delis et al., 2000).

Published studies examining the effect of depression on neuropsychological performance in TBI subjects have not examined recall and recognition on the RCFT. However, the findings obtained by Meyers and Lange (1994) are relevant to the current study. In their study, the RCFT performance of psychiatric patients (mixed diagnosis) was compared to that of healthy controls. The psychiatric group achieved lower scores on Delayed Recall of the RCFT than did the normal controls, although equivalent
recognition scores were achieved. The finding of impaired recall, accompanied by adequate recognition, in the psychiatric group is similar to that obtained in the current study. With regard to recall scores in isolation, a meta-analysis indicated that Delayed Recall was sensitive to depression in non-neurological samples (Zakzanis et al., 1998). The results of the current study suggest that this holds true in TBI subjects as well.

Impaired recall, in the face of intact recognition, generally indicates difficulty with some aspect of the recall task. While it is tempting to construe the discrepancy between recall and recognition as indicating a retrieval deficit, or a partial encoding deficit (see Delis et al., 2000), several alternate explanations could account for this finding. First, adequate performance of the RCFT recall task demands several skills not recruited in the recognition task. For example, reproduction of the figure in the recall trial requires substantial motor planning and organization, as well as graphomotor control and visuo-constructive skills. Hence, the asymmetrical performance of the depressed subjects on the recall and recognition tasks could be due to impairment of neuropsychological skills independent of memory. Second, completion of the recall trial could be characterized as more effortful than completion of the recognition trial. The notion that depressed individuals are impaired on neuropsychological tasks due to reduced motivation / effort was advanced by Cohen and colleagues (1982). As a result, the selective impairment on the Delayed Recall trial of the RCFT may be due to deficient deployment of effort rather than a memory deficit per se. Third, it may simply be the case that the recall task is more difficult, magnifying its sensitivity to depression related impairments (see Calev & Erwin, 1985). This possibility could not be addressed without the use of tasks matched for difficulty such as those utilized by Calev and colleagues.
The Average Performance Rating

A composite score on four measures, known to be sensitive to the neuropsychological sequelae of TBI, was utilized in the current study to remove variance attributable to variables extraneous to depression (i.e., cognitive impairment arising from the injury). In particular, the average T score on verbal fluency (FAS), Coding from the WAIS-III, Trails B and Grooved Pegboard was labelled the Average Performance Rating and was utilized as a covariate in statistical analyses. The composite score was a significant covariate in all the statistical analyses in which it was included. Furthermore, the Average Performance Rating was substantially associated with the dependent memory measures examined herein, while failing to be substantially associated with the total depression score on the PAI. Overall, the derived Average Performance Rating was quite successful in achieving the goal for which it was devised.

Although the Average Performance Rating was described as a composite score which reflected neuropsychological impairment attributable to head injury, it may have been concurrently sensitive to a number of variables, such as current and premorbid intellectual functioning or the presence of learning disability. It could be argued that it is equally desirable to remove such extraneous variance, even if it is unrelated to the presence and/or severity of TBI. Be that as it may, analyses described within the current work suggested that the derived Average Performance Rating was sensitive to injury severity. In general, it is likely that the Average Performance Rating was sensitive to injury parameters and brain integrity in general.

It should be noted, however, that the composite score was a more effective covariate in the CVLT-II analyses than in the RCFT analyses. That this is the case can be
roughly determined by comparing the effect sizes of the Average Performance Rating in the CVLT-II and the RCFT analyses. Note also that the scatter around the line of best fit tends to be larger in the RCFT scatterplots than in the CVLT-II scatterplots. Hence, the Average Performance Rating was less accurate in predicting RCFT scores than in predicting CVLT-II scores.

The more limited success at removing extraneous variance in the RCFT analyses may have had the undesirable effect of reducing the ability to detect differences across the depression groups. In particular, the failure to reduce the total error term could be responsible for the failure of certain comparisons to reach statistical significance. For example, the near significant interaction between depression and RCFT trial (i.e., recall is reduced relative to recognition in the depressed group, and to a greater degree than in the non-depressed group) could reflect this statistical phenomenon. Given that ANCOVA analyses indicated that recall was reduced in the depressed group compared to the non-depressed group and such group specific impairment was not observed on recognition, this would appear to be a plausible interpretation.

However, the ANCOVA comparison of RCFT recognition scores across the groups also approached significance. It is possible that statistically significant differences would have been detected with greater power (or a more effective covariate). Regardless, the difference in adjusted mean scores was relatively small and the effect size for depression would likely be quite small. Hence, it seems unlikely that any statistically significant difference in RCFT recognition performance between the depression groups would indicate a robust and clinically meaningful effect.
*Comparison of the CVLT-II and the RCFT*

In general, the CVLT-II appeared more sensitive to depression related impairments than did the RCFT. While this could have been a statistical artifact due to the discrepant predictive power of the Average Performance Rating, differences in task design might also lead to differential sensitivity to depression. In particular, the CVLT-II supports deliberate mnemonic strategies (i.e., by semantic clustering), whereas performance on the RCFT should not be influenced by deliberate mnemonic strategies due to the incidental recall nature of this task. As a result, the CVLT-II could be more sensitive to depression related memory impairments specifically because performance on this task reflects difficulty in the implementation of deliberate learning strategies.

Hertel and Hardin (1990) suggested that depressed individuals are impaired in the initiation of mnemonic strategies during the course of initial learning. The current findings regarding the CVLT-II could support this suggestion. Specifically, the combination of reduced semantic clustering and reduced learning slope in the depressed subjects could indicate a failure to implement a semantic mnemonic in learning the list. In general, the current findings regarding the CVLT-II imply that depressed subjects are less able to benefit from the semantic organization of the list, which hinders their learning of the list and subsequently leads to impaired recall and recognition. It is tempting to attribute these findings to deficient deployment of a mnemonic strategy consistent with the hypothesis of Hertel and Hardin (1990).

In contrast, the finding of impaired Delayed Recall on the RCFT is unlikely due to the failure to deploy *deliberate* learning strategies. The incidental learning paradigm utilized in the administration of the RCFT should preclude deliberate mnemonic
strategies in all subjects. Nonetheless, the extent to which the subjects apprehend the organization of the figure could influence the degree to which they subsequently recall the figure. It is possible that the recall of depressed subjects is reduced because they failed to organize the information during the initial copy of the figure. However, it is impossible to verify this possibility without quantifying the quality of organization in the copy of the figure. In this context, use of an organizational scoring system, such as that developed by Waber and Holmes (1985) for use with children, would shed light on this possibility. Unfortunately, such data were not available for evaluation in the current study.

Overall, the depressed subjects may fail to initiate deliberate mnemonic strategies during the learning phase of the CVLT-II, hindering their encoding of the list. Decreased performance on both recall and recognition components of the CVLT-II may reflect this difficulty. On the other hand, the depressed subjects are not disadvantaged by their failure to implement deliberate mnemonic strategies on the RCFT, since neither depressed nor non-depressed subjects are likely to initiate deliberate learning strategies during the copy phase. This may account for equivalent levels of learning on the RCFT and, subsequently, equivalent levels of recognition across the groups.

The Personality Assessment Inventory

The high concordance rate between the total depression scale and the affective subscale of the PAI was notable. As has been suspected and described by a variety of authors, several non-depressed TBI subjects reported an elevated number of physiological symptoms frequently associated with depression. However, none of the subjects with total depression elevations in the normal range (i.e., a T score of 60 or less)
had affective subscale T scores greater than 65. In contrast, two subjects with total depression scales of at least 70 had affective subscale elevations below 65. This pattern of responses on the PAI is presumably analogous to a previously described phenomenon, referred to as “masked depression”, which occurs infrequently in TBI patients (Jorge, Robinson, & Arndt, 1993, p. 98).

Although exclusion of the subjects with “masked depression” from the depressed group might have been warranted, the data from both subjects were retained in the current study. The memory scores of both individuals did not emerge as outliers relative to the depressed group, nor did their scores emerge as outliers in the prediction of memory scores from the Average Performance Rating. Hence, despite their denial of depressed mood and loss of interest on the PAI, their memory performance was consistent with that of other individuals classified as depressed. Comparison of the memory performance of individuals with high total depression scores who deny affective symptoms and those who endorse affective symptoms appears potentially worthwhile, although such a comparison would necessitate a greater number of subjects who denied affective symptoms than were available in the current sample.

*Limitations of the Current Study and Future Directions*

The representativeness of the current sample and the generalizability of the current findings warrant explicit mention. The data were obtained retrospectively from a private neuropsychology practice. The obtained sample consisted of individuals referred for neuropsychological evaluation. Typically, these referrals occurred a minimum of several months post-injury and assessments were funded through the private sector. Hence, the individuals within the obtained sample were more likely to: 1) have sustained
a moderate to severe brain injury; or 2) continue to report and/or demonstrate difficulties in the more chronic phase of recovery. As a result, the population would likely have differed on several dimensions had the study been prospective in nature and had the sample consisted of recruited participants who presented at the Emergency Department immediately following injury. As an example, the proportion of individuals demonstrating significant symptoms of depression was perhaps more elevated in the current sample than would be the case in a prospective sample. Furthermore, the proportion of individuals demonstrating memory deficits following mild TBI is likely to be more elevated in the current sample than would be manifest in a prospective sample. In general, the current sample may not be representative of individuals who sustain TBI and do not present for neuropsychological evaluation at a secondary or tertiary level of care.

Secondly, the sample in the current study was limited to subjects between the ages of 18 and 49 years of age. The findings of the current study may not generalize to older subjects. In a geriatric population, the symptoms of depression including memory impairment can be severe enough to resemble dementia and be labelled "pseudo-dementia" (American Psychiatric Association, 2000). Hence, the effect of depression on memory performance may be qualitatively distinct and/or more severe in a geriatric sample. Similarly, one would not assume that the current findings generalize to children who had sustained a brain injury. In general, the current findings do not mandatorily generalize to pediatric or geriatric populations.

With regard to injury severity, the current sample included fewer subjects who had sustained moderate to severe TBI than mild TBI. In fact, the range of injury severity
was likely restricted by the design and scope of the current study. Only subjects capable of completing the vast majority of a rigorous neuropsychological evaluation would have been considered for inclusion. As a result, extremely impaired individuals were not included in the current sample. Moreover, it was not possible to explicitly address the possibility that depression effects on memory performance were discrepant in mild TBI and moderate to severe TBI groups, given the limited sample size. In particular, the inclusion of only eight individuals with moderate to severe TBI in the depressed group rendered such an analysis unlikely to be fruitful. However, the comparison of effects of depression on memory performance by injury severity in a larger sample would be valuable.

In the current study, the subjects were divided into depressed and non-depressed groups on the basis of total depression scores on the PAI. However, the use of analyses which preserved the continuous nature of the depression scores, such as multiple regression, may have been warranted. Such analyses should be considered in future investigations. Furthermore, the relationship between memory performance and affective subscale scores on the PAI may warrant further investigation.

Finally, the potential influence of skills germane to memory task completion which do not reflect memory capacity per se, such as verbal, visual perceptual and motor skills, were not directly evaluated in the current study. Similarly, the potential influence of intellectual functioning was not directly addressed. Accounting for these factors, and directly evaluating their influence, may be worthwhile in future studies.

Overall, the findings from the current study indicate that depression exerts a deleterious effect on memory performance. In particular, depression was associated with
reduced memory performance even after controlling for a composite score reflecting overall neuropsychological impairment. This finding stresses the need to account for possible effects of depression when conducting neuropsychological assessments of depressed individuals who have sustained TBI. In addition, the findings in the current study highlight the importance and utility of controlling for extraneous variance in performance when conducting research with TBI samples, which are frequently vastly heterogeneous with respect to neuropsychological performance.
Appendix A: Measures Comprising the Average Performance Rating

1. *Verbal Fluency (FAS)*: The examinee is provided one minute to list as many words as possible beginning with a particular letter. The letters are presented in the following order: F, A and S. The score achieved is simply the number of correct responses; credit is not awarded for repetitions (including root words with different suffixes). The raw score is converted to a standardized score, according to age and education stratified norms (Tombaugh, Kozak, & Rees, 1996; as cited in Spreen & Strauss, 1998).

2. *Coding subtest from the WAIS-III*: In this subtest, the examinee is required to rapidly inscribe numbers which are matched to abstract symbols in the provided key. The score is calculated by summing the correct responses. Use of the WAIS-III (1997) normative data yields a scaled score.

3. *Trails B*: The examinee is instructed to connect circles containing consecutive letters and numbers interspersed across a sheet of paper, although the letters and numbers must be connected in an alternating fashion (i.e., 1, A, 2, B, 3, C, etc...). Errors are immediately corrected by the examiner. The time required to complete the task is utilized to determine the score achieved. Demographic stratified norms were utilized to calculate the standardized scores in the current study (Heaton, Grant, & Mathews, 1991).

4. *Grooved Pegboard*: The Grooved Pegboard task requires the speeded placement of grooved pegs into grooved slots organized in a series of rows. The time required to
complete the task is recorded. The time to completion was converted to a demographic corrected T score, according to the Heaton and colleagues' norms (1991). Completion with the dominant hand precedes completion with the non-dominant hand. In the current study, the average of the dominant and non-dominant hand T scores was utilized in the calculation of the Average Performance Rating.
Appendix B: Extreme Scores on the CVLT-II

Figure B1. Boxplot of CVLT-II Recognition z scores in the non-depressed group. The marker at $z = -3.0$ represents two extreme scores.
Figure B2. Scatterplot of the relationship between the Average Performance Rating and the CVLT-II Recognition Discriminability score in the non-depressed group. Note the two extreme outliers in the lower right quadrant.
Figure B3. Standardized residuals plot from a regression of CVLT-II Recognition Discriminability and the Average Performance Rating. Note the two extreme scores with standardized residuals of approximately -2.5.
References


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