THE ROLE OF DEPRESSION IN VERBAL MEMORY FOLLOWING TRAUMATIC BRAIN INJURY

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The purpose of this study was to characterize the relationship between verbal memory and depression scores on the Personality Assessment Inventory following traumatic brain injury. Depression was associated with diminished delayed recall and recognition on the California Verbal Learning Test-II (CVLT-II), even after controlling for a neuropsychological composite score and/or a measure of motivation (i.e., the TOMM). There was no relationship between depression and recall on Verbal Paired Associates or Logical Memory when controlling for the same covariates. The findings were most consistent with depressed subjects failing to utilize the semantic organization of the CVLT-II list to enhance their learning.

Keywords: Depression; Memory; Traumatic brain injury

INTRODUCTION

A substantial number of individuals who have sustained traumatic brain injury (TBI) suffer from depression following their injury (Dikmen, Bombardier, Machamer, Fann, & Temkin, 2004; Kreutzer, Seel, & Gourley, 2001). Both TBI and depression have been associated with cognitive compromise including memory deficits, with some authors specifically noting the similarity between neuropsychological profiles characteristic of depression and moderately severe TBI (Veiel, 1997). As a result, the individual with comorbid TBI and depression presents a unique challenge to the assessing neuropsychologist who must distinguish the relative contributions of each influence on memory test performance. This determination is crucial for the accurate formulation of a diagnosis, prognosis, and treatment plan. The differentiation also has important implications in the medical-legal assessment of individuals who have sustained TBI. Several authors have stressed the importance of delineating relationships, if any, between affective disorders and cognition in TBI (Prigatano, 1987; Sweet, Newman, & Bell, 1992). Despite the potential clinical significance...
and the frequency with which the issue arises, there is a paucity of empirical reports in the literature specifically addressing this topic.

Newman and Sweet (1986) illustrated that depression impairs neuropsychological performance to a sufficient degree that the potential for misdiagnosis on an individual basis is unacceptably high. Others have emphasized that the impact of depression on memory is minimal relative to the impact of neurological dysfunction (Coughlan & Hollows, 1984; Gass & Russell, 1986). Nonetheless, the respective contributions of TBI and depression on observed cognitive deficits should be parsed in cases of comorbidity. In such cases, there may be additive or synergistic effects of depression and TBI.

One approach to studying the effects of comorbid depression in neurological patients involves longitudinally studying the cognitive changes that temporally coincide with remission of depression. In a case report of a depressed individual who had sustained moderate TBI, Payne (2000) noted difficulty organizing verbal information on a list-learning task, accompanied by adequate recall of more structured information (i.e., story recall, verbal paired associates). Following remission of depression, learning of the list was improved. In 15 TBI subjects enrolled in a sertraline trial, improvements on Logical Memory and the Buschke Selective Reminding Test were noted over the treatment period, although there was no control group to account for practice effects (Fann, Uomoto, & Katon, 2001). While these findings suggest a relationship between depression and list-learning performance, replication with larger samples and more rigorous designs 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 mixed, possibly due to divergent measures of depression and verbal memory. In a mild TBI sample, Ruttan (1998) found that increased levels of depression on the Millon Clinical Multiaxial Inventory-II predicted reduced passage recall, but that cognitive performance was largely independent of depression scores on the Minnesota Multiphasic Personality Inventory-II (MMPI-II). Also in mild TBI, higher scores on the Beck Depression Inventory predicted reduced long-term recall on the Buschke Selective Reminding Test (Atteberry-Bennett, Barth, Loyd, & Lawrence, 1986). Satz and colleagues (1998) found that performance on the Rey Auditory Verbal Learning Test was inversely related to examiner ratings of depression, but not self-reported ratings of depression on the Symptom Checklist-90 Revised, 6 months following moderate to severe TBI. Others have reported no relationship between depression on the MMPI and verbal memory in a mild TBI sample (Raskin, Mateer, & Tweeten, 1998). As of yet, a consistent picture depicting the prevalence, severity, and nature of memory deficits related to depression in comorbid TBI has not emerged.

Depression-related memory deficits in individuals free of frank neurological insults have been characterized to a greater degree. Impaired free recall, with intact or better-preserved recognition, has often been reported (Fossati, Deweer, Raoux, & Allilaire, 1995; Ilsley, Moffoot, & O’Carroll, 1995). The discrepancy between recall and recognition does not appear to be an artifact related to the greater difficulty of recall, since the discrepancy remains even when recall and recognition tasks are matched for difficulty (Calev & Erwin, 1985). A selective impairment in recall accompanied by intact recognition suggests that retrieval deficits are contributing...
to the memory deficits in depression, as does the alleviation of recall deficits with provision of cues (Fossati et al., 1995). It should be noted, however, that memory deficits have not been consistently demonstrated in young, depressed outpatients (Grant, Thase, & Sweeney, 2001; Purcell, Maruff, Kyrios, & Pantelis, 1997).

Semantic processing and list organization appear to be important mediators of memory deficits in depression. Impaired recall of semantically processed words, despite adequate recall of acoustically processed words, suggests that depressed subjects fail to benefit from deeper, more elaborate processing in establishing memory traces (Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981). Based on their series of experiments, Weingartner and co-authors (1981) commented that depressed subjects failed to reorganize input to assist subsequent recall, although recall was improved when the organization or structure of the information was evident. Consistent with this, Channon, Baker, and Robertson (1993) found that depressed subjects were impaired in recalling words from a list if semantically related words were presented in randomized order, but not if they were presented in consecutive order. This study demonstrated that depressed individuals were most impaired in the recall of moderately structured or organized information, which was deemed consistent with the hypothesis of Hertel and Hardin (1990). This hypothesis asserts that the memory impairments of depressed individuals are due to difficulty spontaneously initiating strategies that are beneficial, but not mandatory, for task completion. On the other hand, if organization or mnemonic strategy is inherent to the task or is explicitly provided, memory deficits should not be observed.

Therefore, it appears that the California Verbal Learning Test (CVLT) would be well suited to evaluate the deleterious effects of depression on memory by virtue of its semantic structure with related exemplars presented in randomized order and its inclusion of both recall and recognition formats. Indeed, a subset of depressed individuals exhibit the pattern of improved recognition relative to recall on the CVLT (Massman, Delis, Butters, Dupont, & Gillin, 1992; see Delis, Kramer, Kaplan, & Ober, 2000, for a review). This pattern is generally believed to reflect retrieval deficits, although it should be noted that the accuracy of inferring retrieval deficits from recall–recognition discrepancies on the CVLT has been questioned (Wilde, Boake, & Sherer, 1995).

**RATIONALE FOR THE CURRENT STUDY**

The CVLT requires delayed recall of a moderately structured list (i.e., one in which semantically related exemplars are presented in randomized order) that others have found to be sensitive to depression (Channon et al., 1993; Fossati et al., 1995; Massman et al., 1992; Zakzanis, Leach, & Kaplan, 1998), albeit not in TBI. Therefore, it was hypothesized that depression would be associated with reduced delayed recall on the CVLT-II. It was further hypothesized that depression-related deficits in recall would be attenuated for Logical Memory, since passage recall is more structured and organized than most list-learning tasks. Others have reported decrements in recall of paired associates in depression (Golinkoff & Sweeney, 1989), and we expected to replicate this finding in TBI. The Verbal Paired Associates task serves as a useful comparison to the CVLT-II in that it similarly requires multi-trial
learning of a series of words, although semantic organization plays a lesser role. Given the discrepancy between delayed recall and recognition described in psychiatric samples (Calev & Erwin, 1985; Fossati et al., 1995; Ilsley et al., 1995), our initial analyses of memory performance focused on delayed free recall and recognition. It was anticipated that recognition would be less impaired than free recall, if deficits were observed on recall, consistent with the possibility of retrieval deficits.

The head-injured population is relatively heterogeneous with regard to the nature and severity of neuropsychological deficits, as well as course of recovery (Dikmen, Machamer, Temkin, & McLean, 1990; Millis & Ricker, 1994; Millis et al., 2001; Novack, Alderson, Bush, Meythaler, & Canupp, 2000; Wiegner & Donders, 1999). The traditional division of injury severity, according to GCS scores, is limited in its ability to capture the extent of cognitive impairment in any particular individual at any particular time. To quantify the relative severity of neuropsychological impairment at the time of assessment, a neuropsychological composite score, herein labeled the Average Performance Rating (APR), was devised. The measures that composed the APR were selected to maximize sensitivity to the sequela of TBI, while excluding memory measures. The composite score was used as a covariate in several statistical analyses to control for degree of neuropsychological impairment. The APR is utilized to examine the most salient question addressed by this research. If one controls for degree of impairment, is there a superimposed effect of depression on memory performance? That is, for any given level of impairment, is the memory performance of a depressed individual more compromised than the memory performance of a non-depressed individual?

It is assumed by many clinicians and researchers that any effect of depression on memory is secondary to reduced motivation. This view was iterated early on and supported by analyses of grip strength and persistence that were presumed to estimate effort (Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982), but was contradicted by experimental manipulation of motivation by Richards and Ruff (1989). In personal injury contexts with latent or manifest financial incentives for neurocognitive disability, the possibility of malingering (or the feigning/exaggeration of neurocognitive deficits) also warrants consideration. The Test of Memory Malingering (TOMM; Tombaugh, 1996) is often used to evaluate motivation or response bias with regard to memory performance in such forensic evaluations. One of the goals of the current work was to determine whether memory deficits accompanying depression remained significant after controlling for motivation, as quantified by scores achieved on the TOMM.

It has been noted that presence of a depressive disorder and severity of depressive symptoms are not always consistently associated with neuropsychological performance (Boone et al., 1995; Newman & Sweet, 1992). The presence of a depressive disorder (regardless of severity) could be associated with a given neuropsychological deficit, even if the magnitude of this deficit does not vary directly with changes in the severity of depressive symptoms. On the other hand, other neuropsychological deficits may vary directly in magnitude as severity of depressive symptoms waxes and wanes (e.g., Boone et al., 1995). Hence, depression was evaluated both as a categorical variable and as a continuous variable in the current study.

In sum, the purpose of the current work was to elucidate the relationship between depression and memory, specifically delayed recall and recognition, on three measures
of verbal memory in individuals who had sustained TBI. Additionally, we wished to
determine whether observed relationships between self-reported levels of depression
and objective memory performance were evident when controlling for a composite
score reflecting neuropsychological impairment and/or a measure reflecting motivation.
In general, it was hypothesized that depression would predict diminished delayed recall
on the CVLT-II, even after controlling for a neuropsychological composite score and/or
a measure of motivation. It was anticipated there might be evidence for depression-
related retrieval deficits, particularly on the CVLT-II.

METHOD

Participants

The data were collected by retrospective review of consecutive referrals to a
private neuropsychological practice for evaluation of potential sequelae arising from
closed head injury. The majority of subjects had been referred by attorneys,
insurance adjudicators, case managers, family physicians, and other medical practi-
tioners for the purpose of establishing disability, determining compensation, and/or
planning vocational rehabilitation. The sample is predominantly comprised of eval-
uations of a forensic nature; litigation or compensation seeking was ongoing or
plausible in most of the evaluations. Based on a priori inclusion criteria, all subjects
were between the ages of 18 and 49 years and none had sustained their injury prior to
age 15 years. All subjects sustained at least a mild brain injury, according to the cri-
teria of the Mild Traumatic Brain Injury Committee of the American Congress of
Rehabilitation Medicine (1993). Specific criteria included at least one of the follow-
ing: any period of loss of consciousness, any loss of memory (whether retrograde or
anterograde), any alteration in mental state, or focal neurological deficits.

Inclusion criteria were relatively broad to maintain the representativeness of the
sample to populations routinely evaluated in clinical practice, particularly in the con-
text of third-party evaluations. Subjects were not excluded due to prior history of learn-
ing difficulties, chronic pain, substance abuse, or litigation status. On the other hand,
subjects whose performance was considered grossly affected by a condition unrelated
to the brain injury (e.g., schizophrenia, developmental disability) were excluded, as
were subjects whose deficits prevented them from completing a substantial portion
of a comprehensive neuropsychological evaluation (e.g., aphasia, sensory impairment).

This sample is comprised primarily of young adults ($M = 33.7$ years;
$SD = 10.1$) in the post-acute phase of recovery (time since injury: $M = 17.8$ months;
$SD = 16.8$), most of whom had completed at least secondary education ($M = 13$
years; $SD = 2.2$). Of the 53 subjects, 37 were deemed to have sustained mild TBI,
whereas 16 were deemed to have sustained moderate to severe TBI. Most were
injured in a motor vehicle collision.

For the purpose of forming categorical groups based on depressive symptoms,
individuals with depression scores of at least 70 on the Personality Assessment
Inventory (PAI) were included in the depressed group ($n = 24$), whereas individuals
with depression scores of at most 60 were included in the non-depressed group
($n = 19$). There were no significant group differences with regard to age, education,
time since injury, sex, and injury severity.
Measures

Severity of depressive symptoms was quantified via the depression scale of the PAI (Morey, 1991). Tests of verbal learning and memory included the Verbal Paired Associates and Logical Memory subtests of the Wechsler Memory Scale-III (WMS-III; The Psychological Corporation, 1997), and the CVLT-II (Delis et al., 2000).

To quantify the severity of neuropsychological impairment at the time of assessment, a neuropsychological composite score labeled the APR was calculated for each subject. This was accomplished by computing the average of T scores achieved on each of verbal fluency (FAS), Trails B, Coding from the WAIS-III, and Grooved Pegboard (average of right and left hand performance). Raw scores for verbal fluency were converted to demographically corrected T scores using norms provided by Tombaugh, Kozak, and Rees (as cited in Spreen & Strauss, 1998), while T scores for Trails B and Grooved Pegboard were derived by reference to the Heaton, Grant, and Mathews (1991) norms. The measures that composed the APR were selected to maximize sensitivity to the sequelae of TBI, while excluding memory measures. The APR was used as a covariate in select statistical analyses to control for degree of neuropsychological integrity.

Performance on the TOMM was utilized to control for motivation in certain analyses. Scores on the first learning trial of the TOMM were selected in an attempt to maximize the range of scores within the sample and to enhance sensitivity to suboptimal effort for statistical purposes. Preliminary inspection of the data revealed that scores ranged from 27 to 50, but were negatively skewed. As recommended by Tabachnick and Fidell (2001), TOMM scores were reflected and subjected to logarithmic transformation. For ease of interpretation, scores were again reflected to ensure directionality consistent with the original measure (i.e., lower scores on the transformed TOMM variable are analogous to lower scores on the first trial of the TOMM). Substantial improvement in normality (i.e., skewness approaching 0) was confirmed following transformation. Subsequent references to TOMM scores refer to transformed TOMM scores.

Analyses

To minimize Type I errors, one delayed recall and one recognition variable for each of the three verbal learning tests were selected a priori for analyses. Selected scores for the CVLT-II were long delay free recall and recognition discriminability. For Logical Memory and Verbal Paired Associates, primary subtest scores were selected for analyses of delayed recall, whereas raw scores were selected for analyses of recognition. Given the disparate nature of the two verbal learning tasks from the WMS-III, use of the norm-referenced composite score reflecting auditory recognition was deemed inappropriate. Age was not correlated with raw scores on the recognition trial for Logical Memory, $r^2 (53) = -.238, p = .086$, or Verbal Paired Associates, $r (50) = -.116, p = .424$. Therefore, age was not included as a covariate in analyses involving these measures. Norm-referenced scores are reported herein, except for Logical Memory and Verbal Paired Associates recognition scores.

Group differences on selected delayed recall and recognition measures were first evaluated by independent samples $t$-tests and Mann–Whitney $U$ tests for variables
with normal and non-normal distributions, respectively. For each measure of verbal memory, an ANCOVA analysis was conducted to determine whether the depressed group performed more poorly than did the non-depressed group, after covarying for a composite score of neuropsychological integrity (i.e., the APR). Subsequent analyses utilized TOMM scores as a covariate to determine whether group differences were evident after accounting for motivation.

The relationship between severity of depressive symptomatology and memory performance was examined via correlational analyses. Non-parametric correlations were used for variables with non-normal distributions. For variables with normal distributions, partial correlation coefficients were then computed to evaluate the extent of relationship between severity of depressive symptoms and memory performance when controlling for APR and TOMM scores.

**RESULTS**

**Comparison of the Depressed and Non-Depressed Groups**

The delayed recall and recognition performance of the depressed \((n = 24)\) and non-depressed \((n = 19)\) groups on all verbal memory measures are listed in Table 1, as are group comparisons. Post-hoc analyses on supplementary CVLT-II variables

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**Table 1** Verbal memory performance for the depressed \((n = 24)\) and non-depressed \((n = 19)\) groups

<table>
<thead>
<tr>
<th>Memory measure</th>
<th>Depressed (M (SD))</th>
<th>Non-depressed (M (SD))</th>
<th>Group comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>California Verbal Learning Test-II (z-scores)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial 1</td>
<td>-0.48 (1.06)</td>
<td>-0.47 (0.96)</td>
<td>0.018</td>
</tr>
<tr>
<td>Trial 5</td>
<td>-0.81 (1.04)</td>
<td>0.13 (1.09)</td>
<td>2.892**</td>
</tr>
<tr>
<td>Learning Slope</td>
<td>-0.52 (0.99)</td>
<td>0.39 (1.09)</td>
<td>2.877**</td>
</tr>
<tr>
<td>Short Delay Cued Recall</td>
<td>-0.96 (1.07)</td>
<td>0.13 (1.08)</td>
<td>3.302**</td>
</tr>
<tr>
<td>Short Delay Free Recall</td>
<td>-1.00 (1.07)</td>
<td>0.05 (1.12)</td>
<td>3.137**</td>
</tr>
<tr>
<td>Long Delay Cued Recall</td>
<td>-0.90 (1.05)</td>
<td>0.11 (1.14)</td>
<td>2.989**</td>
</tr>
<tr>
<td>Long Delay Free Recall</td>
<td>-1.25 (1.33)</td>
<td>0.21 (1.03)</td>
<td>3.943**</td>
</tr>
<tr>
<td>Recall d’</td>
<td>-0.94 (1.00)</td>
<td>0.29 (1.05)</td>
<td>3.910**</td>
</tr>
<tr>
<td>Recognition d’</td>
<td>-0.83 (1.12)</td>
<td>0.42 (0.77)</td>
<td>4.164**</td>
</tr>
<tr>
<td>Response Bias</td>
<td>0.16 (0.94)</td>
<td>0.16 (0.94)</td>
<td>0.080</td>
</tr>
<tr>
<td>Consistency</td>
<td>-0.06 (0.98)</td>
<td>0.63 (0.70)</td>
<td>2.596*</td>
</tr>
<tr>
<td>Primacy</td>
<td>-0.13 (0.97)</td>
<td>0.16 (1.08)</td>
<td>0.903</td>
</tr>
<tr>
<td>Middle</td>
<td>-0.88 (1.55)</td>
<td>-0.58 (1.40)</td>
<td>0.648</td>
</tr>
<tr>
<td>Recency</td>
<td>0.69 (1.65)</td>
<td>0.21 (0.99)</td>
<td>210.500</td>
</tr>
<tr>
<td>Serial Clustering</td>
<td>0.05 (0.76)</td>
<td>-0.08 (1.06)</td>
<td>-0.434</td>
</tr>
<tr>
<td>Semantic Clustering</td>
<td>-0.38 (0.66)</td>
<td>0.79 (1.61)</td>
<td>119.000**</td>
</tr>
<tr>
<td>Verbal Paired Associates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delayed Recall (scaled score)</td>
<td>8.75 (2.54)</td>
<td>10.37 (2.36)</td>
<td>2.139*</td>
</tr>
<tr>
<td>Recognition (raw score)</td>
<td>23.25 (1.70)</td>
<td>23.74 (0.93)</td>
<td>194.500</td>
</tr>
<tr>
<td>Logical Memory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delayed Recall (scaled score)</td>
<td>8.00 (3.34)</td>
<td>8.95 (3.01)</td>
<td>0.965</td>
</tr>
<tr>
<td>Recognition (raw score)</td>
<td>23.96 (3.29)</td>
<td>25.42 (2.78)</td>
<td>1.549</td>
</tr>
</tbody>
</table>

\(d’ = \text{discriminability.}\)

\(*p < .05, \quad **p < .01.\)
are also included in Table 1. Table 2 summarizes the results of ANCOVA analyses comparing the memory performance of the depression groups, after controlling for either the neuropsychological composite score (i.e., the APR) or the measure of motivation (i.e., TOMM score).

**California Verbal Learning Test–II.** The depressed group showed reduced recall on the long delay free recall trial and reduced recognition discriminability relative to the non-depressed group (see Table 1). These group differences remained significant in ANCOVA analyses, whether controlling for neuropsychological composite scores or motivation scores, as shown in Table 2.

These findings suggest that depression exerts a deleterious effect on recall and recognition, rather than a selective effect on recall. Nonetheless, it was possible that recall was more impaired than was recognition in the depressed subjects, consistent with a retrieval deficit. Repeated-measures ANCOVA analyses, with depression status as a between-subject variable and CVLT-II trial (i.e., recall vs recognition) as a within-subject variable, were conducted to determine whether a decrement in recall relative to recognition was associated with depression. When covarying for the APR, there was no interaction between depression group and CVLT-II trial, \( F(1, 40) = 0.318, p = .576 \), although there was a significant main effect for depression status, \( F(1, 40) = 16.897, p = .000 \). Similarly, there was no interaction between depression group and CVLT-II trial, after controlling for TOMM scores, \( F(1, 40) = 0.369, p = .547 \), although the main effect for depression continued to be apparent, \( F(1, 40) = 11.184, p = .002 \).

Sequentially controlling for APR and TOMM scores would be redundant if both covariates had analogous relationships with the dependent variable, namely memory performance. Therefore, multiple regression analyses utilizing the entire sample \((N = 53)\) were conducted to ensure that each of the covariates independently contributed to the prediction of memory performance on the CVLT-II. The combination of the neuropsychological composite score and the measure of motivation significantly predicted delayed recall of the list, \( F(2, 50) = 16.830, p = .000, R^2 = 0.402 \), with significant independent contributions from APR scores, \( \beta = .494, t = 4.342, p = .000 \), and TOMM scores, \( \beta = .284, t = 2.499, p = .016 \). Similarly,
the combination of predictors accounted for a significant proportion of the variance in recognition of the words, $F(2, 50) = 21.068, p = .000, R^2 = 0.457$, with significant independent contributions from the APR, $\beta = .502, t = 4.635, p = .000$, and the TOMM, $\beta = .335, t = 3.089, p = .003$.

Depression was associated with diminished recall and recognition on the CVLT-II. However, the findings thus far did not support the hypothesis that depression-related memory decrements were best characterized as retrieval deficits. Therefore, a wider range of CVLT-II variables were included in post-hoc analyses to further characterize the nature of memory deficits in the depressed group. As can be seen in Table 1, the depressed group recalled fewer words than the non-depressed group on all delayed recall trials, whether or not trials were cued by semantic categories. The learning slope was less steep for the depressed subjects; they recalled fewer words on the fifth learning trial, despite similar recall on the first learning trial. The depressed group showed less evidence of semantic clustering and their recall was less consistent. The groups did not differ with regard to scores reflecting primacy and recency effects.

**Wechsler Memory Scale-III.** As shown in Table 1, the depressed group ($n = 24$) performed as well as the non-depressed group ($n = 19$) on delayed recall and recognition of short stories. The depressed subjects recalled fewer word pairs than non-depressed subjects, although this group difference was no longer significant when controlling for either neuropsychological composite scores or TOMM scores. Non-parametric analyses failed to show group differences for recognition of word pairs. However, it should be noted that these scores were markedly skewed and likely reflected a ceiling effect, casting doubt on the utility of this finding. Given the limited range of scores on Verbal Paired Associates recognition and the apparent ceiling effect, only limited analyses on this measure are reported herein.

**Analyses Utilizing Depression as a Continuous Variable**

**California Verbal Learning Test-II.** Correlation coefficients for continuous depression scores and CVLT-II variables in the entire sample ($N = 53$) are presented in Table 3. Increasing severity of depressive symptoms was associated with worsening delayed recall and recognition discriminability. Higher levels of depression were associated with reduced recall on the fifth learning trial, but not on the first learning trial. This was also reflected in a negative correlation between depression scores and learning slope. Depressive symptoms were related to inconsistent recall and diminished semantic clustering.

Partial correlation coefficients, controlling for the APR and TOMM scores, are also listed in Table 3. Controlling for these scores attenuated the magnitude of correlations between depression and memory variables, although most of the relationships obtained in the first analysis remained significant (with the exceptions of short delay free recall and long delay cued recall). Specifically, higher levels of depression continued to predict reduced learning slope, delayed free recall, recognition, and consistency of recall, after controlling for the neuropsychological composite score and the measure of motivation.
Computed correlation coefficients for depression and memory measures are listed in Table 4, as are partial correlations controlling for APR and TOMM scores. When analyzing continuous depression scores from the entire sample ($N = 53$), depression was not significantly correlated with delayed passage recall or recognition. Higher levels of self-reported depressive symptoms predicted recall of fewer word pairs. However, this relationship was no longer significant when controlling for APR and TOMM scores. Recognition of the word pairs appeared unrelated to depression scores, although a ceiling effect was apparent in the recognition data.

**Table 3** Correlations between depression scores and California Verbal Learning Test-II

<table>
<thead>
<tr>
<th>Memory measure</th>
<th>Correlation</th>
<th>Partial correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>.001</td>
<td>.090</td>
</tr>
<tr>
<td>Trial 5</td>
<td>-.377**</td>
<td>-.280*</td>
</tr>
<tr>
<td>Learning Slope</td>
<td>-.350**</td>
<td>-.283*</td>
</tr>
<tr>
<td>Short Delay Free Recall</td>
<td>-.373**</td>
<td>-.267</td>
</tr>
<tr>
<td>Short Delay Cued Recall</td>
<td>-.405**</td>
<td>-.312*</td>
</tr>
<tr>
<td>Long Delay Free Recall</td>
<td>-.511**</td>
<td>-.447**</td>
</tr>
<tr>
<td>Long Delay Cued Recall</td>
<td>-.360**</td>
<td>-.250</td>
</tr>
<tr>
<td>Recall Discriminability</td>
<td>-.410**</td>
<td>-.302**</td>
</tr>
<tr>
<td>Recognition Discriminability</td>
<td>-.486**</td>
<td>-.413**</td>
</tr>
<tr>
<td>Response Bias</td>
<td>.081</td>
<td>.042</td>
</tr>
<tr>
<td>Consistency</td>
<td>-.369**</td>
<td>-.302*</td>
</tr>
<tr>
<td>Primacy</td>
<td>.043</td>
<td>.062</td>
</tr>
<tr>
<td>Middle</td>
<td>-.100</td>
<td>-.088</td>
</tr>
<tr>
<td>Recency$^a$</td>
<td>-.022</td>
<td>–</td>
</tr>
<tr>
<td>Serial Clustering</td>
<td>.114</td>
<td>.067</td>
</tr>
<tr>
<td>Semantic Clustering$^a$</td>
<td>-.362**</td>
<td>–</td>
</tr>
</tbody>
</table>

*Note.* Partial correlation coefficients control for both the Average Performance Rating and the Test of Memory Malingering scores. $N = 53$.

$^a$Non-parametric Spearman rank correlation coefficient, $r_s$.

$^*$p < .05, $^{**}$p < .01.

**Wechsler Memory Scale-III.** Computed correlation coefficients for depression and memory measures are listed in Table 4, as are partial correlations controlling for APR and TOMM scores. When analyzing continuous depression scores from the entire sample ($N = 53$), depression was not significantly correlated with delayed passage recall or recognition. Higher levels of self-reported depressive symptoms predicted recall of fewer word pairs. However, this relationship was no longer significant when controlling for APR and TOMM scores. Recognition of the word pairs appeared unrelated to depression scores, although a ceiling effect was apparent in the recognition data.

**Table 4** Correlations between depression scores and Wechsler Memory Scale-III measures

<table>
<thead>
<tr>
<th>Memory measure</th>
<th>Correlation</th>
<th>Partial correlation</th>
</tr>
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<tbody>
<tr>
<td>LM Delayed Recall</td>
<td>-.231</td>
<td>-.125</td>
</tr>
<tr>
<td>LM Recognition</td>
<td>-.257</td>
<td>-.162</td>
</tr>
<tr>
<td>VPA Delayed Recall</td>
<td>-.303*</td>
<td>-.173</td>
</tr>
<tr>
<td>VPA Recognition$^a$</td>
<td>-.116</td>
<td>–</td>
</tr>
</tbody>
</table>

Partial correlation coefficients control for both the Average Performance Rating and the Test of Memory Malingering scores. $N = 53$. LM = Logical Memory; VPA = Verbal Paired Associates.

$^a$Non-parametric Spearman rank correlation coefficient, $r_s$.

$^*$p < .05, $^{**}$p < .01.
DISCUSSION

The findings failed to indicate any relationship between depression and passage recall, whether depression was analyzed as a categorical or as a continuous variable. Depression was associated with decrements in word pair recall, but this association was abolished by controlling for a neuropsychological composite score and/or a measure of motivation. Depression did not predict recognition of the word pairs, although recognition scores for the Verbal Paired Associates task were markedly skewed. This likely reflected a ceiling effect, casting doubt on the utility of this finding. The level of difficulty and sensitivity are presumably too low to address the questions raised in this study.

Depression was associated with impoverished performance on the CVLT-II, consistent with prior reports identifying a relationship between depression and list learning performance in TBI samples (Atteberry-Bennett et al., 1986; Payne, 2000; Satz et al., 1998). Furthermore, for any given level of performance on a composite score comprised of neuropsychological measures sensitive to the sequelae of TBI, there was a superimposed effect of depression on delayed recall and recognition of the list. Likewise, depression was associated with reduced recall and recognition of the list when controlling for a measure of motivation. Hence, the observed memory decrement does not appear entirely attributable to reduced motivation or greater TBI-related impairment in the depressed subjects. Rather, the findings imply that depression exerts a relatively specific effect on memory processes measured by the CVLT-II. The relationship between depression and memory was generally consistent, whether depression was analyzed as a categorical variable or a continuous variable, suggesting that increasing severity of depressive symptoms is associated with increased magnitude of verbal memory decrements on this measure.

The findings herein provide support for the position that depression-related decrements on particular measures of verbal memory (specifically the CVLT-II) may be observed in young, depressed outpatients who have sustained at least mild TBI. This contrasts with the absence of a relationship between depression and CVLT recall and recognition reported by Raskin et al. (1998). Methodological differences that might account for the discrepant results include use of the MMPI to quantify severity of depressive symptoms and subjects scoring below a T score of 70 having been categorized as non-depressed.

It should be noted that most of the depressed subjects did not demonstrate grossly impaired memory. Rather, there appeared to be a performance decrement of approximately one standard deviation for delayed recall and recognition, which is roughly consistent with the magnitude of memory decrements on the CVLT reported for depressed subjects without TBI (Otto, Bruder, Fava, Delis, Quitkin, & Rosenbaum, 1994). Inspection of the group means and estimated marginal means reveals a small degree of improvement in standardized scores from the delayed free recall format to the recognition format in the depressed subjects. However, a similar degree of improvement was seen in the non-depressed subjects. There was not a significant interaction between depression group and memory measure trial (i.e., recognition vs recall), inconsistent with a priori expectations of a recall–recognition discrepancy or a retrieval deficit for the depressed subjects.
Post-hoc analyses were conducted to clarify the nature of the depression-related memory deficits on the CVLT-II. Depression was associated with reduced delayed recall and recognition, which suggests reduced learning or encoding of the list. Indeed, the depressed subjects recalled fewer words at the fifth learning trial despite similar recall at the first learning trial, which converged with the finding of reduced learning slope. Depression was not associated with the score reflecting the primacy effect. On the other hand, depression was associated with reduced semantic clustering. In general, the depressed subjects appear to have utilized the semantic organization of the list to a lesser degree, hindering their learning of the list, which subsequently resulted in reduced recall and recognition of the words.

The nature of the observed memory deficits accompanying depression could be viewed as being consistent with several explanations posited by those involved with research on depression, albeit not in TBI. First, the findings could be construed as reflecting inefficiency in effortful encoding (cf. Zakzanis et al., 1998). Second, depressed individuals may fail to benefit from semantic processing to an optimal degree when encoding information (Weingartner et al., 1981). Third, depressed subjects may be impaired in initiating spontaneous mnemonic strategies that promote optimal task performance but are not mandatory for task completion (Hertel & Hardin, 1990). In particular, depressed subjects may fail to initiate organizational strategies in learning a list of semantically related words presented in staggered order, as suggested by the findings of Channon and colleagues (1993). These explanations are not inherently mutually exclusive. It could be that depressed subjects’ failure to implement the mnemonic strategy of semantic clustering leads to decreased ability to benefit from the semantic organization of the list, subsequently resulting in impaired recall and recognition of the list, which constitutes a failure in effortful encoding.

Prior research suggests that frontal lobe volume predicts semantic clustering on the CVLT (Kramer et al., 2005) and that semantic organizational processes at encoding are associated with increased left prefrontal cerebral blood flow in normal controls (Fletcher, Shallice, & Dolan, 1998). Decreased prefrontal cerebral blood flow has been linked to memory deficits in depression (Dolan, Bench, Brown, Scott, & Frackowiak, 1994). Furthermore, depression-related differences in prefrontal EEG activity during a memory task have been attributed to the failure of depressed subjects to initiate a learning strategy, consistent with Hertel’s (2000) cognitive-initiative framework (Nitschke, Heller, Etienne, & Miller, 2004). It is tempting to speculate that prefrontal dysfunction contributes to the depressed subjects’ failure to employ organizational strategies that benefit learning and memory, and that the depression-related memory deficits observed on the CVLT-II reflect impairment in executive functions more so than impairment in core memory processes.

Overall, the findings were not consistent with the retrieval deficits specific to depression, inferred from recall–recognition discrepancies, noted in several psychiatric studies (Calev & Erwin, 1985; Fossati et al., 1995; Ilsley et al., 1995; Massman et al., 1992). It is possible that cognitive profiles associated with depression differ in TBI and strictly psychiatric populations, a possibility that is at least partially supported by the discriminant function analyses of Aloia, Long, and Allen (1995). That said, recognition deficits have been observed in depressed patients without neurological insult, with several researchers concluding that depression-related deficits may also extend to encoding (Brand, Jolles, & Gispen-de Wied, 1992; Golinkoff &
Sweeney, 1989; Wolfe, Granholm, Butters, Saunders, & Janowsky, 1987; Zakzanis et al., 1998). Furthermore, the association between depression and consistency on the CVLT-II observed in the current study suggests the possibility of retrieval inefficiencies, superimposed upon the aforementioned learning difficulties.

It should additionally be specified that depression-related deficits in recognition were not due to a conservative response bias. Response bias on the CVLT-II recognition trial appeared unrelated to depression, whether depression was analyzed as a continuous or categorical variable. It could be speculated that depression is less consistently related to a conservative response bias in neurological samples than in non-neurological samples, since response bias in depressed TBI subjects may be more determined by injury parameters than by affective status. For example, frontal lobe dysfunction secondary to TBI may be associated with a liberal response bias (cf. Kramer et al., 2005), which counteracts or precludes a conservative response bias due to depression.

The hypothesis that the CVLT-II would be more sensitive to depression-related decrements in verbal memory than Logical Memory or Verbal Paired Associates was supported by reduced delayed recall on the CVLT-II, even after controlling for a neuropsychological composite score and motivation, whereas passage recall and word pair recall were not reduced after controlling for the same covariates. While it is tempting to attribute the different findings across the three verbal memory measures to the nature and task demands of those measures, some caution is warranted in this conclusion since the measures are not equated for difficulty or normative populations.

Another issue that requires consideration is the heterogeneity of memory patterns following TBI, including the variety of subtypes identified on the CVLT (Curtiss, Vanderploeg, Spencer, & Salazar, 2001; Millis & Ricker, 1994; Vanderploeg, Crowell, & Curtiss, 2001; Wiegner & Donders, 1999). The current study did not specifically address the possibility that different memory patterns (e.g., encoding vs retrieval deficits) would be evident across the subjects with TBI, independent of depression. It is, however, an inherent assumption of the current research that affective status could contribute to heterogeneity in memory patterns following TBI. A comparison of the affective status of individuals comprising the various CVLT subtypes would be helpful in addressing this possibility.

In the current study, analyses using depression as a categorical variable proceeded by classifying individuals according to scores on the depression scale of the PAI. It should be recognized that this method is not necessarily concordant with establishing the presence of a depressive disorder meeting diagnostic criteria as in a structured clinical interview. Empirical studies specifically investigating the utility of the PAI in evaluating depression in TBI have been lacking. As a result, the sensitivity and specificity of the PAI depression scale in TBI populations are not known.

The sample in this study was comprised of individuals who had sustained a range of severity of TBI. It is possible that cognitive effects of depression differ by severity of injury or other injury parameters, but the small sample size precluded rigorous analyses of such possibilities. Nonetheless, the analyses herein showed a statistically significant effect of depression on the CVLT-II across the mixed severity sample after controlling for a neuropsychological composite score derived from performance on measures sensitive to brain injury sequelae. Supplementary repeated
measures ANCOVA analyses also failed to show any significant interaction between depression and APR scores in predicting CVLT-II recall and recognition. On the basis of these findings, it might be inferred that effects of depression may be similar across a range of severity of TBI, but further research is required to address this issue.

The representativeness of the current sample and the generalizability of the current study warrant explicit mention. Mean demographic statistics indicate the sample includes mostly young individuals, with some post-secondary education, who sustained mild head injuries more than 1 year prior to evaluation. Hence, the findings should not be generalized to subjects who are older, are experiencing severe depression requiring hospitalization, or are in the acute period of recovery (e.g., rehabilitation setting). However, the sample may be representative of individuals referred for neuropsychological evaluation by third parties in many independent practices, particularly those servicing the personal injury sector.

Considering the composition of the current sample, it is also important to note that prior literature indicates a small to negligible effect size of mild head injury on neuropsychological performance, including memory, more than 3 months following injury (Binder, Rohling, & Larrabee, 1997). On the other hand, research suggests that deemed malingering status and psychological status (e.g., depression) contribute significantly to the prediction of memory performance following head injury (Suhr, Tranel, Wefel, & Barrash, 1997). The date herein are consistent with this literature. The findings in the current study additionally suggest a mild memory decrement on the CVLT-II secondary to depression that is not entirely attributable to exaggeration or feigning of amnesic deficits, at least as measured by the first trial of the TOMM. Therefore, the possibility that depression accounts for mild memory deficits should be considered when evaluating individuals who are presenting for neuropsychological evaluation of TBI, following the acute period of recovery.

Furthermore, given the association between depressive symptoms and subjective over-estimation of memory deficits (Coleman, Sackeim, Prudic, & Devanand, 1996; Larrabee & Levin, 1986), depressed individuals with mild TBI may present with mild memory deficits in conjunction with memory complaints of far greater severity. In these cases, the impact of depression on the discrepancy between subjective reports of memory and objective memory performance should be taken into account. This is particularly relevant to the determination of malingering, as in the Slick et al. criteria (Slick, Sherman, & Iverson, 1999). Depression might be considered a differential diagnosis (i.e., Criteria D) that accounts for mild memory deficits and “over-stated” self-report of cognitive impairment in individuals who have sustained mild TBI. These forensic implications require further consideration and validation, based on research using larger samples.

Finally, the use of covariates in the current study warrants further consideration. Scores achieved on the first trial of the TOMM were used to control for motivation or response bias on the memory tasks. Although scores on the first trial of the TOMM are not used for the detection or diagnosis of malingering on an individual basis in clinical settings, it was believed that use of these scores as a covariate might enhance sensitivity to sub-optimal effort even below the level necessary for a clinical diagnosis of malingering, strictly for the purpose of statistical control across a group. This approach is consistent with a conceptualization of response bias as occurring
on a continuum of severity, rather than a more dichotomous classification of malingering, in forensic settings. Statistical analyses herein showed that scores on the first trial of the TOMM independently contributed to the prediction of memory performance in this sample. The utility of this approach might be considered for future investigations of a forensic nature. Likewise, use of neuropsychological composite scores (e.g., the APR used herein) to control for degree of neuropsychological impairment, as in the current study, may prove fruitful in research with cognitively heterogenous populations such as those with TBI.

In sum, we have extended prior findings by simultaneously considering recall and recognition on three measures of verbal learning, hypothesized to be differentially sensitive to depression. We demonstrated deleterious effects of depression on CVLT-II performance after controlling for a neuropsychological composite score and/or a measure tapping motivation. The impoverished delayed recall and recognition of the depressed subjects on the CVLT-II are most consistent with depressed subjects failing to utilize the semantic organization of the list to enhance their learning of the words. In contrast, depression-related decrements on passage or word pair learning were either not observed or were not significant after controlling for a neuropsychological composite score or a measure of motivation. It is tempting to speculate that prefrontal dysfunction in depression impacts memory function via its effect on semantic organizational strategies that benefit learning on the CVLT-II.

REFERENCES


