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Neuropsychological Functioning in Posttraumatic Stress Disorder and Alcohol Abuse

Kristin W. Samuelson,
San Francisco Veteran's Affairs Medical Center and California School of Professional Psychology at Alliant International University

Thomas J. Metzler,
San Francisco Veteran's Affairs Medical Center and University of California, San Francisco

Johannes Rothlind,
San Francisco Veteran's Affairs Medical Center

Gerard Choucroun,
San Francisco Veteran's Affairs Medical Center

Thomas C. Neylan,
San Francisco Veteran's Affairs Medical Center and University of California, San Francisco

Maryanne Lenoci, and
San Francisco Veteran's Affairs Medical Center

Clare Henn-Haase
San Francisco Veteran's Affairs Medical Center and University of California, San Francisco

Michael W. Weiner and **Charles R. Marmar**
San Francisco Veteran's Affairs Medical Center and University of California, San Francisco

Abstract

Studies have shown differences in neuropsychological functioning between groups with posttraumatic stress disorder (PTSD) and control participants. Because individuals with PTSD often have a history of comorbid alcohol abuse, the extent to which an alcohol confound is responsible for these differences remains a concern. The current study compares neuropsychological testing scores in 4 groups of veterans with and without PTSD (PTSD+] and PTSD–, respectively) and with and without a history of alcohol abuse (ETOH+ and ETOH–, respectively): *n* for PTSD+/ETOH+ = 30, *n* for PTSD+/ETOH– = 37, *n* for PTSD–/ETOH+= 30, and *n* for PTSD–/ETOH– = 31. Results showed that PTSD, when alcohol, educational level, vocabulary, and depression are controlled for, was associated with decreased verbal memory, attention, and processing speed performance. Alcohol abuse history was associated with decreased visual memory performance. By controlling for alcohol and depression, the authors can more conclusively demonstrate that verbal memory and attention differences are associated with PTSD.

Keywords

PTSD; veterans; memory; attention; alcohol abuse

Correspondence concerning this article should be addressed to Kristin W. Samuelson, who is at the California School of Professional Psychology at Alliant International University, One Beach Street, Suite 100, San Francisco, CA 94133. E-mail: ksamuelson@alliant.edu.

Interest in neuropsychological functioning in posttraumatic stress disorder (PTSD) arose in response to patients' complaints of problems with memory, attention, and concentration. A number of studies have identified decreased performance in verbal memory and learning in participants with PTSD relative to control participants (Elzinga & Bremner, 2002; Gil, Calev, Greenberg, Kugelmass, & Lerer, 1990; Golier & Yehuda, 2002; Horner & Hamner, 2002; Koenen et al., 2001; Uddo, Vasterling, Brailey, & Sutker, 1993; Yehuda et al., 1995). Other studies have documented differences between groups with and without PTSD in the areas of working memory and attention (Brandes et al., 2002; Gilbertson, Gurvits, Lasko, Orr, & Pitman, 2001; Vasterling, Brailey, Constans, & Sutker, 1998; Vasterling et al., 2002) and processing speed (Brandes et al., 2002). In addition, many but not all studies have reported decreases in hippocampal volume (Bremner et al., 1995, 1997; Gilbertson et al., 2002; Gurvits et al., 1996; Stein, Koverola, Hanna, Torchia, & McClarty, 1997; Villarreal et al., 2002) and hippocampal N-acetylaspartate (Freeman, Cardwell, Karson, & Komoroski, 1998; Schuff et al., 1997, 2001) as well as associations between hippocampal atrophy and poorer verbal memory in PTSD (Bremner et al., 1995). Although Brandes et al. (2002) and Uddo et al. (1993) found impaired visual memory performance in individuals with PTSD, the majority of studies have failed to find PTSD-related deficits in visual memory (Bremner et al., 1995; Stein, Kennedy, & Twamley, 2002; Vasterling et al., 1998, 2002; Zalewski, Thompson, & Gottesman, 1994).

Findings of neuropsychological deficits in PTSD are not consistent, however, and several studies have not shown differences between samples with and without PTSD in memory performance (Crowell, Kieffer, Siders, & Vanderploeg, 2002; Neylan et al., 2004; Stein, Hanna, Vaerum, & Koverola, 1999; Stein et al., 2002; Zalewski et al., 1994) or attention (Golier et al., 1997; Yehuda et al., 1995). Such inconsistencies in findings may be attributed in part to sample limitations inherent in populations diagnosed with PTSD. Most neuropsychological studies of PTSD involve veterans with chronic PTSD who frequently exhibit comorbid psychiatric diagnoses. In particular, high lifetime rates of alcohol abuse frequently accompany PTSD, thereby confounding the attribution of neuropsychological deficits specifically to PTSD. In their review, Keane and Kaloupek (1997) found lifetime alcohol abuse and dependence rates varying from 24% to 84% in individuals with PTSD. Because alcoholism itself may impact neuropsychological functioning, we cannot be sure that neuropsychological deficits seen in patients with PTSD are in fact due to PTSD without adequately controlling for comorbid alcohol abuse and dependence.

Research studies on neuropsychological deficits in alcoholics tend to show more severe deficits in visual–spatial functions than in verbal functions (e.g., Butters, Cermak, Montgomery, & Adinofli, 1977; Donat, 1986; Fabian, Parsons, & Sheldon, 1994; Leber, Jenkins, & Parsons, 1981; Ryan & Butters, 1980), leading many alcohol researchers to suggest that the right hemisphere might be more vulnerable to the effects of alcohol abuse than the left hemisphere of the brain (see Ellis & Oscar-Berman, 1989, for review). Although the majority of studies show deficits in chronic or recently detoxified alcoholics, some studies have found neuropsychological problems to persist after the acute withdrawal period. Cross-sectional studies of recovering alcoholics who have been abstinent for several years have shown impairment particularly in visual–spatial tasks (Brandt, Butters, Ryan, & Bayog, 1983; Fama, Pfefferbaum, & Sullivan, 2004). A review of longitudinal studies (Parsons, 1996) showed that although neuropsychological performance improves over time in abstinent alcoholics, recovering alcoholics still show significant differences from control groups at retesting for several years. These studies illustrate the continued debate surrounding the long-term effects of alcoholism on neuropsychological functioning.

In their large study of neurocognition in veterans with and without PTSD, Barrett, Green, Morris, Giles, and Croft (1996) examined the impact of comorbid psychiatric disorders.

Veterans with PTSD alone did not show lower scores on measures of neuropsychological functioning compared with normal control participants. However, veterans with both PTSD and a concurrent diagnosis of depression, anxiety, or substance abuse performed significantly less well. These results suggested that a diagnosis of PTSD alone may not be strongly associated with neuropsychological impairment.

Researchers of some studies have attempted to address the comorbidity issue by matching participants with PTSD and control participants on alcohol abuse history (Bremner et al., 1993, 1995; Jenkins, Langlais, Delis, & Cohen, 1998) or by statistically controlling for lifetime history of alcohol use (Gilbertson et al., 2001), and they have continued to find PTSD-related neuropsychological deficits. Other studies have excluded participants with recent alcohol abuse histories altogether (Neylan et al., 2004; Stein et al., 2002). Although it is necessary to ensure that the effects of alcohol are controlled for, it is also important to study participants with PTSD and recent alcohol abuse histories, otherwise findings will not be representative of the larger population of veterans with PTSD. This concern is underscored by initial clinical impressions of high levels of alcohol abuse in returning Afghanistan and Iraq veterans with PTSD. By comparing four groups of individuals with and without PTSD and alcohol abuse history, it is possible to experimentally determine the independent contribution of PTSD to neuropsychological functioning, the independent contribution of alcohol abuse history, and the interactive effects of both conditions. Including participants with PTSD with and without a history of recent alcohol abuse offers an opportunity to better understand the factors that may contribute to lower neuropsychological performance in PTSD, and it may yield results that are generalizable to a real world clinical population of combat veterans with PTSD.

Comorbid depression diagnoses present an additional challenge in PTSD research. The review by Keane and Kaloupek (1997) reported rates of depression among individuals with PTSD to range from 28% to 84%. The effects of depressed mood on neuropsychological functioning have been well documented (Snyder & Nussbaum, 1998). The high rate of comorbidity and overlap of symptoms between these two disorders makes it extremely difficult to exclude individuals with current depression from PTSD studies. However, it is important to address the presence of comorbid depression and include measures of depression as covariates in PTSD studies.

The previously documented attention difficulties in PTSD (Uddo et al., 1993; Vasterling et al., 1998, 2002) also complicate the interpretation of lower scores on memory measures in this disorder. One possible explanation for memory difficulties in PTSD is that explicit memory performance may be impacted by impaired attention resources during processing. Siegel (1995) and others have theorized that the heightened emotional reactivity in those with PTSD disrupts attentional resources. Because attention and concentration difficulties are symptoms of PTSD, and attentional deficits have been documented in veterans with PTSD, it is possible that observed differences in memory actually reflect impaired attention. Impaired attention prevents sufficient registration of information, which in turn prevents consolidation and retrieval of memory. Gilbertson et al. (2001) found that after controlling for attentional differences, most neuropsychological tasks (executive functioning, perceptual motor speed, visual–spatial skills) were not altered in PTSD, with the exception of explicit memory functioning, as measured by the Wechsler Memory Scale—Third Edition (WMS–III; Wechsler, 1997b).

In the present study, we attempted to control for several potentially confounding factors that often complicate interpretations of PTSD neurocognitive studies. By using a 2 × 2 factorial design, examining groups with and without diagnoses of PTSD and with and without histories of alcohol abuse, we examined the independent and interactive effects of PTSD and alcohol. We defined history of alcohol abuse as a diagnosis of abuse or dependence within the last 5

years, on the basis of previous research suggesting neuropsychological deficits in alcoholics for several years postsobriety but not 5 or more years postsobriety (see Parsons, 1996, for a review). In addition, we statistically controlled for depression, vocabulary, and education level. To control for the role of attention, we then analyzed differences in neuropsychological performance after covarying for a measure of attention. Our primary hypothesis was that PTSD is associated with decreased performance in domains of verbal learning and memory, and working memory, attention, and processing speed when the effects of alcohol, education, vocabulary, and depression symptoms are controlled for. We also hypothesized that alcohol abuse history would be associated with decreased performance in visual memory and visual–spatial domains. We did not predict a PTSD × Alcohol interaction effect on any measure.

Method

Participants

Two hundred and forty-four male and female veterans, ranging in age from 22 to 60 years, were recruited from the San Francisco Veteran's Affairs Medical Center and from the community by using advertisements in the media for a study on magnetic resonance imaging and neuropsychological functioning in veterans. None of the participants were inpatients at the time of the study. An initial screening interview was performed, and written informed consent was obtained after participants were provided with a complete description of the study. The study protocol and consent form was approved by the Committee on Human Research at the University of California, San Francisco. Eleven participants with a history of head trauma, prolonged loss of consciousness (> 10 min), neurological disorder, or systemic illness affecting central nervous system function (including all neurologic disorders and diabetes) were excluded. Twenty-eight participants who were diagnosed during the screening interview with past or current alcohol abuse and dependence, lifetime history of psychotic disorder or bipolar disorder, and drug abuse or dependence within the previous 6 months were also excluded. In addition, 54 veterans were excluded because they did not meet appropriate PTSD criteria for the study, 15 veterans dropped out of the study after the initial interview, and 8 veterans were excluded because of positive toxicology screens.

The final sample consisted of 128 veterans in four groups. Groups were divided into participants with and without PTSD (PTSD+ and PTSD–, respectively) and with and without a history of alcohol abuse or dependence in the past 5 years (ETOH+ and ETOH–, respectively; ETOH = ethanol, or alcohol). Thirty veterans were identified as PTSD+/ETOH+, 37 veterans were identified as PTSD+/ETOH–, 30 veterans were identified as PTSD–/ETOH+, and 31 veterans were identified as PTSD–/ETOH–.

Demographic characteristics (age, gender, ethnicity, and education), trauma histories, and clinical and background variables [Clinician Administered PTSD Scale (CAPS, Blake et al., 1995); Lifetime Drinking History (LDH, Skinner & Sheu, 1982); Wechsler Adult Intelligence Scale—Third Edition (WAIS–III), Vocabulary (Wechsler, 1997a); and Symptom Checklist-90-R (SCL-90-R, Derogatis, 1994) Depression scale] are summarized in Table 1. The groups did not differ by age or gender, but the groups that were ETOH+ had fewer years of education than the other groups. As expected, groups that were PTSD+ had higher CAPS scores than groups that were PTSD–, but CAPS score differences between groups PTSD+ with and without alcohol abuse were not significant. The groups differed substantially by lifetime drinking, with both groups PTSD+ and ETOH+ having significantly higher scores. The groups differed significantly by current drinking totals in the past 3 months, with groups ETOH+ having significantly higher scores than groups ETOH–. Post hoc analyses revealed that the group identified as PTSD+/ETOH+ did not differ from the group identified as PTSD–/ETOH + on number of drinks in the past 3 months. Groups that were PTSD+ had significantly higher SCL-90-R Depression subscale scores, as well as lower WAIS–III Vocabulary scores, than

groups that were PTSD–. The participants were ethnically diverse although predominantly White (73%). The groups were comparable in ethnic composition. English was the dominant language for all participants.

Information about comorbid mood and anxiety disorder diagnoses and past drug abuse or dependence diagnoses are also included in Table 1. There were significant differences between groups that were PTSD+ and PTSD– in rates of major depressive disorder and anxiety disorders (including obsessive–compulsive disorder, panic disorder, generalized anxiety disorder, or social phobia). Mood and anxiety disorders are so common among individuals with PTSD that they are often conceptualized as part of the PTSD psychopathology (Sutker, Uddo-Crane, & Allain, 1991). Thus, participants were not excluded from the study on the basis of these comorbidities. Psychiatric medication use is also summarized in Table 1. Participants with PTSD had significantly higher rates of psychiatric medication (primarily antidepressant) use. Only eight participants out of the entire sample were taking benzodiazepines, evenly distributed among the four groups, $\chi^2(3, N = 128) = 1.11$, $p = .78$.

Measures

Structured diagnostic interviews—Participants were interviewed by a clinical psychologist with extensive PTSD experience in using the Structured Clinical Interview for *DSM–IV* Diagnosis (First, Spitzer, Williams, & Gibbon, 1996) and in using the CAPS, and they were classified into groups. A second rater listened to 20% of the taped CAPS interviews, and interrater reliability was obtained. The intraclass correlation coefficient for the two raters was .984. The Structured Clinical Interview for *DSM–IV* Diagnosis was used to diagnose comorbid psychiatric disorders.

Veterans with past but not current PTSD, or current subsyndromal PTSD, were not included in the study. Veterans with a history of alcohol abuse or dependence in the past 5 years were included in groups that were ETOH+, whereas veterans with no history of alcohol abuse or dependence anytime in the past 5 years were included in groups that were ETOH–. In the group identified as PTSD+/ETOH+, 25 of the 30 participants had alcohol dependence diagnoses and 5 had alcohol abuse diagnoses. In the group identified as PTSD–/ETOH+, 24 of the 30 participants had alcohol dependence diagnoses, compared with 6 with abuse diagnoses.

SCL-90-R—Participants completed the SCL-90-R, a 90-item self-report clinical rating scale of psychiatric symptomatology. Of interest in the current study was the Depression subscale score, which consists of 13 items of depressive symptomatology such as feelings of hopelessness, worthlessness, and suicidality.

Lifetime drinking history—We obtained lifetime alcohol use histories on all of the participants by using the LDH.

Neuropsychological variables—All participants were administered a battery of neuropsychological tests that assessed three domains of cognitive functioning: verbal memory; visual memory and visual–spatial skills; and attention, working memory, and processing speed.

Verbal memory—To assess verbal memory and learning, we used three variables from the California Verbal Learning Test (CVLT; Delis, Free-land, Kramer, & Kaplan, 1988). The CVLT measures both recall and recognition of word lists over a number of trials. The Trial 1 score provides a measure of initial acquisition of verbally presented material, whereas the Total Trials 1−5 score provides a measure of verbal learning. The Long-Delay Free Recall score reflects the test takers' ability to retain the word list over a period of 20 min, and is a measure of delayed verbal memory. Participants also completed the Logical Memory I and II subtests

of the WMS–III. For Logical Memory I, the participant listens to two stories read by the examiner and immediately after hearing each story, is asked to retell it from memory. Thirty min later, the participant is asked to retell the stories, which constitutes the Logical Memory II score. The Percent Retention score is a measure of how much of the stories the participant remembered relative to his or her immediate recall. Low scores on the Logical Memory subtests suggest weaknesses in learning of or memory for orally presented narratives, whereas low scores on Percent Retention suggest difficulties with retaining auditory narratives after a delay.

Visual memory and visual–spatial skills—To assess short and long-term visual memory, we used the Visual Immediate Index and Visual Delayed Index scores of the WMS–III. These indices are composed of the Faces and Family Pictures subtests, immediate and delayed recall. In the Faces subtest, the participant is asked to remember photographs of faces and then is shown a series of faces and must identify whether the face was one he or she was asked to remember. For the Family Pictures subtest, the participant is shown scenes with family members and asked to remember aspects of the scene. Whereas Faces is a test of recognition, Family Pictures is a test of recall, but low scores on both indicate memory deficits with visually presented material. For Faces II and Family Pictures II, which make up the Visual Delayed Index, participants are asked to identify or recall the faces or family pictures 30 min later. Visual–spatial skill was assessed by using the Block Design subtest of the WAIS–III, which requires the participant to replicate pictures of geometric patterns with two-color blocks.

Working memory, attention, and processing speed—Working memory and attention were assessed by using the Letter Number Sequencing, Spatial Span, and Digit Span subtests of the WMS–III. For the Letter Number Sequencing subtest, a measure of auditory working memory, the participant is read a series of numbers and letters and is asked to repeat them, saying the numbers first in ascending order and then the letters in alphabetical order. In the Spatial Span subtest, the examiner taps cubes on a board in a specified sequence, and the participant must tap the blocks in the same sequence. The Digit Span subtest consists of Digit Span Forward, in which the participant is asked to repeat back a series of digits in the same sequence as the examiner presented, and Digit Span Backward, in which the examinee must repeat back the digits but in reverse order. Digit Span Forward is a measure of focused attention, whereas Digit Span Backward demands more effort from working memory, requiring the participant to manipulate information stored in the short-term memory (Wechsler, 1997b). To assess processing speed, we administered the Digit Symbol subtest of the WAIS–III. The examiner is presented with a key of numbers corresponding with symbols and is required to use the key to copy the symbols under the numbers. The task measures psychomotor speed and set shifting.

Intellectual functioning—Participants were administered several subtests of the WAIS–III, and the Vocabulary subtest score was used as an estimate of intellectual functioning. The Vocabulary subtest is often used as a “hold” test from which estimates of premorbid functioning are derived in brain-injured populations (Lezak, 1995).

A clinical psychologist administered the neuropsychological battery.The neuropsychological tests were administered in the same order for all participants. The first 11 participants did not receive some tests (Digit Symbol, Logical Memory I and II, Letter Number Sequencing) that were added to the battery after their completion. Testing took approximately 2 hours including a midsession 15-min break. Participants were instructed to abstain from using alcoholic beverages and were breathalyzed before neuropsychological testing. Participants also had urinalysis for drug toxicology on the day of neuropsychological assessment. Data from 2 participants who had positive toxicology screens were excluded from the analysis.

Data Analysis

We conducted individual analyses of covariance (ANCOVAs) on measures from three domains of neuropsychological functioning—verbal memory; visual–spatial and visual memory; and attention, working memory, and processing speed. In each analysis, we covaried for background or clinical variables in which there were differences between groups, such as education, vocabulary performance, and depression. Post hoc analyses of group effects were made for each analysis, adjusting for multiple comparisons by using a modification of Bonferroni's method for correlated multiple outcomes, grouping together these domain variables (Sankoh, Huque, & Dubey, 1997). Our four-group model accounts for alcohol abuse history in the past 5 years but may not adequately account for current alcohol use. Therefore, we next conducted analyses on variables previously seen to be significantly different between groups that were PTSD+ and PTSD–, covarying for level of current alcohol use determined by total drinks in the past 3 months on the LDH. We also conducted ANCOVAs on all neuropsychological measures, comparing participants with lifetime histories of drug dependence or abuse with participants without drug dependence or abuse. If any differences were detected, we then entered drug history as a covariate to determine if any previously significant differences seen between groups that were PTSD+ and PTSD– remained after controlling for drug history. Finally, to control for attention in secondary analyses, we conducted individual ANCOVAs on variables with significant differences in the initially specified ANCOVAs, using the Digits Forward score as a covariate.

Results

Primary Analyses

Verbal memory—We conducted individual ANCOVAs on six measures of verbal memory —CVLT total score, Trial 1 score, and Long-Delay Free Recall score, and the WMS–III Logical Memory I, Logical Memory II, and Logical Memory Percent Retention scores. We controlled for education, Vocabulary, and depression. Because depression and PTSD share several symptoms, we constructed a residualized variable of the SCL-90-R Depression subscale that partials out shared variance with the CAPS, and we entered this residualized variable into the model as a covariate. Post hoc analyses of group effects adjusting for multiple comparisons were then conducted with a modification of Bonferroni's method for correlated multiple outcomes. Results are shown in Table 2. There was a main effect for PTSD on both CVLT total score and Trial 1 score, and we did not find a main effect for alcohol or an interaction effect. There were no significant differences at the Bonferroni-adjusted alpha level between groups on the three Logical Memory measures, although there was a trend for lower Logical Memory Percent Retention in groups that were PTSD+.

Working memory, attention, and processing speed—We conducted three separate ANCOVAs for measures of working memory (WMS–III Letter Number Sequencing and Spatial Span), attention (WAIS–III Digit Span), and processing speed (WAIS–III Digit Symbol). Results are shown in Table 3. We again adjusted for multiple comparisons of these variables with Bonferroni's method. After covarying for education, Vocabulary, and the residualized SCL-90-R Depression score, there was a main effect for PTSD on Letter Number Sequencing, Digit Span, and Digit Symbol. There were no main effects for alcohol or interaction effects on any of these measures.

Visual memory—We conducted three ANCOVAs for measures of visual–spatial reasoning (Block Design) and visual memory (WMS–III Visual Immediate Memory Index and WMS–III Visual Delayed Memory Index). Results are shown in Table 4. Adjusted multiple comparison alphas were again used for these analyses. After covarying for education, Vocabulary, and the residualized SCL-90-R Depression score, we did not find either main

effects for PTSD and alcohol or an interaction effect for Block Design. There was a main effect for alcohol on the WMS–III Visual Immediate Index. There were no main effects for PTSD on any measures, and there were no interaction effects.

Secondary Analyses

Having found lower scores in groups that were PTSD+ compared with groups that were PTSD– on measures of verbal memory, attention, and processing speed, we then conducted univariate analyses covarying for current alcohol use by using a measure of total number of drinks in the past 3 months obtained from the LDH. After covarying for current level of alcohol use, in addition to education, Vocabulary, and SCL-90-R Depression residualized score, the main effects for PTSD persisted on all measures: the CVLT Total Score, $F(1, 120) = 7.10$, $p = .009$; Digits Forward, $F(1, 118) = 14.66$, $p = .000$; and Digit Symbol, $F(1, 108) = 11.33$, $p = .001$.

Given the findings of differences in short-term visual memory performance between groups that were ETOH+ and ETOH–, we conducted bivariate correlations of WMS–III Visual Immediate Index and current alcohol use amount by group. None of the correlations were significant, suggesting that frequency of recent alcohol use is not related to visual memory impairment.

We also conducted ANCOVAs to determine if participants with lifetime drug abuse or dependence diagnoses performed differently on the neuropsychological tests. After covarying for education, Vocabulary, and SCL-90-R Depression residualized score, there was a significant difference between groups on Digit Symbol performance, $F(1, 108) = 5.90$, $p = .017$, with the drug history group ($M = 61.62$, $SD = 13.25$) showing decreased performance compared with the group without lifetime substance abuse diagnoses ($M = 69.36$, $SD = 15.89$). We then conducted ANCOVAs, entering drug history as an additional covariate, comparing groups that were PTSD+ and PTSD– on the Digit Symbol measure, and there remained a significant difference between groups, $F(1, 109) = 8.91$, $p = .003$.

Univariate analyses were next conducted to determine if the verbal memory, working memory, and processing speed differences seen in the groups that were PTSD+ were a function of attentional problems. After covarying for attention by using the Digits Forward score on the Digit Span subtest—in addition to covarying for education, SCL-90-R Depression residualized score, and Vocabulary—we still found a significant difference on CVLT total score, $F(1, 120) = 4.96$, $p = .028$; CVLT Trial 1, $F(1, 120) = 8.30$, $p = .005$; Letter Number Sequencing, $F(1, 112) = 16.23$, $p = .030$; and Digit Symbol, $F(1, 110) = 8.67$, $p = .004$.

Discussion

The results confirmed our hypotheses that PTSD would be associated with decreased performance on measures of verbal learning, working memory, attention, and processing speed. Alcohol abuse history was associated with decreased performance on a measure of immediate visual memory. There were no interaction effects of PTSD and alcohol. After covarying for several potential confounds—including education, an estimate of intellectual functioning, depression, current level of alcohol use, and drug history—differences between individuals with and without PTSD remained significant. Given these results, we can more conclusively determine that lower scores on tests of verbal learning, working memory, attention, and processing speed among the participants who were PTSD+ in this sample are not due to the confounding effects of alcohol or depression.

Our findings suggest that memory deficits are not global but restricted to specific tasks. On memory measures, veterans with PTSD showed deficiencies relative to veterans without PTSD in the initial registration and learning of word lists but not on retention. Although we found

differences on CVLT performance, we did not see differences between participants who were PTSD+ or PTSD– on paragraph recall, suggesting that verbal memory may be more compromised when the task does not involve a narrative context. In the group with PTSD, working memory was impaired when the task involved auditory material (i.e., Digit Span, Letter Number Sequencing) but not visual material (Spatial Span). Similarly, participants with PTSD did not exhibit deficits on immediate or delayed visual memory. Consistent with previous research (Vasterling et al., 1998, 2002; Yehuda et al., 1995), these findings of weaknesses in tasks involving attention, working memory, and new learning are suggestive of frontal-limbic abnormalities.

These results also add to the alcoholism literature, suggesting effects of alcohol abuse history on short-term visual memory performance. Because visual memory but not verbal memory was compromised in our participants with significant alcohol abuse histories, our findings offer partial support for the “right hemisphere hypothesis” of alcoholism, suggesting that the right hemisphere may be particularly vulnerable to the effects of alcoholism. These results are consistent with other studies showing impaired visual memory in recently detoxified alcoholics (Donat, 1986; Leber et al., 1981). However, in our sample there was not a relationship between frequency of recent alcohol use and visual memory performance, suggesting that impairments seen are due to a longer term history of alcohol abuse or dependence or to genetic or early developmental risk factors for alcohol abuse. These findings highlight the continued need for alcohol abuse researchers to examine visual–spatial and visual memory performance in alcoholics well past the acute withdrawal period. They also suggest the need for familial studies of gene markers and neurocognitive testing to determine if visual–spatial and visual memory deficits are heritable risk factors for alcohol abuse.

Because the groups that were PTSD+ had significantly lower scores on a measure of attention, it was possible that observed differences in verbal memory actually reflected impaired attention. After covarying for the effects of passive auditory attention, the differences on CVLT measures, Letter Number Sequencing, and Digit Symbol remained significant. These results suggest that difficulties in both verbal learning and memory as well as set shifting and mental flexibility are not merely an artifact of reduced passive auditory attention. However, it may also be that the measure of attention used in this study is not sufficiently sensitive to detect alterations in attention that might account for effects on memory. Future studies might continue to study this hypothesis, using a measure of attention such as the Continuous Performance Test (Conners, 1994).

Several limitations should be noted when interpreting the results of this study. First, it is difficult to adequately identify individuals with significant alcohol abuse histories. By using the categorical *DSM–IV* diagnosis to identify our participants with a history of alcohol abuse, we may be including individuals in our groups with ETOH– who are binge drinkers yet do not meet the diagnostic criteria of abuse or dependence. In addition, many of our participants with a history of alcohol abuse were presently in remission, with some having been sober for nearly 5 years. As alcohol effects have been most consistently identified in current or newly abstinent drinkers (e.g., Beatty, Tivis, Stott, Nixon, & Parsons, 2000; Grant, 1987), it is certainly possible that the variation in recency of alcohol abuse or dependence limited our ability to detect a significant effect for alcohol. We attempted to account for this limitation by covarying for current alcohol use in secondary analyses of measures that detected differences in individuals who are PTSD+ and PTSD–; all differences previously seen remained significant. However, our study had limited power to detect effects of recent drinking given the small number of recent drinkers. It is important to note that post hoc analyses revealed the group PTSD+/ETOH + did not differ from the group PTSD–/ETOH+ on number of drinks in the past 3 months, underscoring our success in controlling for recent heavy alcohol use, arguably the most important PTSD/alcohol confound in neurocognitive testing.

Ideally, neuropsychological studies comparing group differences should match participants in each group on key demographic variables, such as age and education, which will likely have an impact on the dependent variable. When groups are not matched, as was the case in the current study with education level, a less satisfactory approach applies ANCOVA, with differences in demographic variables controlled for (Adams, Brown, & Grant, 1985). It can be particularly difficult to match participants in PTSD and alcoholism research on education level, but researchers should continue to strive to do so.

In addition, our neuropsychological battery did not include systematic measures of executive function. Past studies have included measures of executive functioning (e.g., Stein et al., 2002), and future researchers might continue to test these skills by using a design similar to ours. Finally, it is possible that exposure to toxins such as Agent Orange may worsen veterans' neuropsychological functioning, a possibility not explored in the present study. Future studies might explore in more detail the deployment and toxic exposure histories of enlisted military personnel. Recognizing these limitations, the present findings add to the literature documenting neuropsychological differences in individuals with and without PTSD. Verbal and working memory difficulties were more prominent than visual impairments in veterans with PTSD.

Most important to note is that these differences cannot be accounted for by alcohol abuse history, depression, or attentional disturbances.

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References

Adams K, Brown G, Grant I. Analysis of covariance as a remedy for demographic mismatch of research subject groups: Some sobering simulations. Journal of Clinical and Experimental Neuropsychology 1985;7:445–462. [PubMed: 4031027]

Barrett DH, Green ML, Morris R, Giles WH, Croft JB. Cognitive functioning and posttraumatic stress disorder. American Journal of Psychiatry 1996;153:1492–1494. [PubMed: 8890689]

Beatty WW, Tivis R, Stott HD, Nixon SJ, Parsons OA. Neuropsychological deficits in sober alcoholics: Influences of chronicity and recent alcohol consumption. Alcoholism: Clinical and Experimental Research 2000;24:149–154.

Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, et al. The development of a clinician-administered PTSD scale. Journal of Traumatic Stress 1995;8:75–90. [PubMed: 7712061]

Brandes D, Ben-Schachar G, Gilboa A, Bonne O, Freedman S, Shalev A. PTSD symptoms and cognitive performance in recent trauma survivors. Psychiatry Research 2002;110:231–238. [PubMed: 12127473]

Brandt J, Butters N, Ryan C, Bayog R. Cognitive loss and recovery in long-term alcohol abusers. Archives of General Psychiatry 1983;40:435–442. [PubMed: 6838323]

Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM, et al. MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. American Journal of Psychiatry 1995;152:973–981. [PubMed: 7793467]

Bremner JD, Randall P, Vermetten E, Staib L, Bronen RA, Mazure C, et al. Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse—a preliminary report. Biological Psychiatry 1997;41:23–32. [PubMed: 8988792]

Bremner JD, Scott T, Delaney R, Southwick S, Mason J, Johnson D, et al. Deficits in short-term memory in post-traumatic stress disorder. American Journal of Psychiatry 1993;150:1015–1019. [PubMed: 8317569]

Butters N, Cermak L, Montgomery K, Adinofli A. Some comparisons of the memory and visuoperceptive deficits of chronic alcoholics and patients with Korsakoff's disease. Alcoholism: Clinical and Experimental Research 1977;1:73–80.

Conners, K. The Conners Continuous Performance Test. Multi-Health Systems; Toronto, Ontario, Canada: 1994.

Crowell T, Kieffer K, Siders C, Vanderploeg R. Neuropsychological findings in combat-related posttraumatic stress disorder. Clinical Neuropsychologist 2002;16:310–321. [PubMed: 12607144]

Delis DC, Freeland J, Kramer JH, Kaplan E. Integrating clinical assessment with cognitive neuroscience: Construct validation of the California Verbal Learning Test. Journal of Consulting & Clinical Psychology 1988;56:123–130. [PubMed: 3346437]

Derogatis, LR. SCL-90-R: Symptom Checklist-90-R administration, scoring, and procedures manual. Pearson Assessments; Minneapolis, MN: 1994.

Donat DC. Semantic and visual memory after alcohol abuse. Journal of Clinical Psychology 1986;42:537–539. [PubMed: 3711356]

Ellis RJ, Oscar-Berman M. Alcoholism, aging, and functional cerebral asymmetries. Psychological Bulletin 1989;106:128–147. [PubMed: 2667007]

Elzinga BM, Bremner JD. Are the neural substrates of memory the final common pathway in posttraumatic stress disorder (PTSD)? Journal of Affective Disorders 2002;70:1–17. [PubMed: 12113915]

Fabian MS, Parsons OA, Sheldon MD. Effects of gender and alcoholism on verbal and visual–spatial learning. Journal of Nervous and Mental Disease 1994;172:16–20. [PubMed: 6690649]

Fama R, Pfefferbaum A, Sullivan E. Perceptual learning in detoxified alcoholic men: Contributions from explicit memory, executive function, and age. Alcoholism: Clinical and Experimental Research 2004;28:1657–1665.

First, MB.; Spitzer, RL.; Williams, JBW.; Gibbon, M. Structured clinical interview for DSM-IV (SCID-I; Patient Version). Biometrics Research, New York State Psychiatric Institute; New York: 1996.

Freeman T, Cardwell D, Karson C, Komoroski R. In vivo proton magnetic resonance spectroscopy of the medial temporal lobes of subjects with combat-related posttraumatic stress disorder. Magnetic Resonance in Medicine 1998;40:66–71. [PubMed: 9660555]

Gil T, Calev A, Greenberg D, Kugelmass S, Lerer B. Cognitive functioning in post-traumatic stress disorder. Journal of Traumatic Stress 1990;3:29–45.

Gilbertson MW, Gurvits TV, Lasko NB, Orr SP, Pitman RK. Multivariate assessment of explicit memory function in combat veterans with posttraumatic stress disorder. Journal of Traumatic Stress 2001;14:413–432. [PubMed: 11469166]

Gilbertson MW, Shenton ME, Ciszewski A, Kasai K, Lasko NB, Orr SP, et al. Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. Nature Neuroscience 2002;5:1242–1247.

Golier J, Yehuda R. Neuropsychological processes in post-traumatic stress disorder. Psychiatric Clinics of North America 2002;25:295–315. [PubMed: 12136502]

Golier J, Yehuda R, Cornblatt B, Harvey P, Gerber D, Levengood R. Sustained attention in combat-related posttraumatic stress disorder. Integrative Physiological Behavioral Sciences 1997;32:52–61.

Grant I. Alcohol and the brain: Neuropsychological correlates. Journal of Consulting & Clinical Psychology 1987;55:310–324. [PubMed: 3597943]

Gurvits TV, Shenton ME, Hokama H, Ohta H, Orr SP, Lasko NB, et al. Magnetic resonance imaging study of hippocampal volume in chronic, combat-related post-traumatic stress disorder. Biological Psychiatry 1996;40:1091–1099. [PubMed: 8931911]

Horner MD, Hamner MB. Neurocognitive functioning in posttraumatic stress disorder. Neuropsychology Review 2002;12:15–30. [PubMed: 12090717]

Jenkins MA, Langlais PJ, Delis D, Cohen R. Learning and memory in rape victims with posttraumatic stress disorder. American Journal of Psychiatry 1998;155:278–279. [PubMed: 9464211]

Keane TM, Kaloupek DG. Co-morbid psychiatric disorders in PTSD: Implications for research. Annals of New York Academy of Sciences 1997;821:24–34.

Koenen KC, Driver KL, Oscar-Berman M, Wolfe J, Folsom S, Huang MT, et al. Measures of prefrontal system dysfunction in posttraumatic stress disorder. Brain and Cognition 2001;45:64–78. [PubMed: 11161363]

Leber WR, Jenkins RL, Parsons OA. Recovery of visual–spatial learning and memory in chronic alcoholics. Journal of Clinical Psychology 1981;37:192–197. [PubMed: 7204599]

Lezak, M. Neuropsychological assessment. 3rd ed.. Oxford University Press; New York: 1995.

Neylan TC, Lenoci M, Rothlind J, Metzler TJ, Schuff N, Du AT, et al. Attention, learning, and memory in posttraumatic stress disorder. Journal of Traumatic Stress 2004;17:41–46. [PubMed: 15027792]

Parsons, OA. Alcohol abuse and alcoholism.. In: Adams, RL.; Parsons, OA., editors. Neuropsychology for clinical practice: Etiology, assessment, and treatment of common neurological disorders. American Psychological Association; Washington, DC: 1996. p. 175-201.

Ryan C, Butters N. Further evidence for a continuum of impairment encompassing male alcoholic Korsakoff patients and chronic alcoholic men. Alcoholism: Clinical and Experimental Research 1980;4:190–198.

Sankoh AJ, Huque MF, Dubey SD. Some comments on frequently used multiple endpoint adjustment methods in clinical trials. Statistics in Medicine 1997;16:2529–2542. [PubMed: 9403954]

Schuff N, Marmar C, Weiss D, Neylan T, Schoenfeld F, Fein G, Weiner M. Reduced hippocampal volume and n-acetyl aspartate in posstraumatic stress disorder. Annals of the New York Academy of Sciences 1997;821:516–520. [PubMed: 9238242]

Schuff N, Neylan T, Lenoci M, Du A, Weiss D, Marmar C, Weiner M. Decreased hippocampal N-acetylaspartate in the absence of atrophy in posttraumatic stress disorder. Biological Psychiatry 2001;50:952–959. [PubMed: 11750891]

Siegel DJ. Memory, trauma, and psychotherapy: A cognitive science view. Journal of Psychotherapy Practice and Research 1995;4:93–122.

Skinner HA, Sheu WJ. Reliability of alcohol use indices: The Lifetime Drinking History and the MAST. Journal of Studies on Alcohol 1982;43:1157–1170. [PubMed: 7182675]

Snyder, P.; Nussbaum, P. Clinical neuropsychology: A pocket handbook for assessment. American Psychological Association; Washington, DC: 1998.

Stein MB, Hanna C, Vaerum V, Koverola C. Memory functioning in adult women traumatized by childhood sexual abuse. Journal of Traumatic Stress 1999;12:527–534. [PubMed: 10467560]

Stein MB, Kennedy CM, Twamley EW. Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. Biological Psychiatry 2002;52:1079–1088. [PubMed: 12460691]

Stein MB, Koverola C, Hanna C, Torchia MG, McClarty B. Hippocampal volume in women victimized by childhood sexual abuse. Psychological Medicine 1997;27:951–959. [PubMed: 9234472]

Sutker PB, Uddo-Crane M, Allain A. Clinical and research assessment of posttraumatic stress disorder. Psychological Assessment: A Journal of Consulting and Clinical Psychology 1991;3:520–530.

Uddo M, Vasterling JJ, Brailey K, Sutker PB. Memory and attention in combat-related post-traumatic stress disorder (PTSD). Journal of Psychopathology & Behavioral Assessment 1993;15:43–52.

Vasterling JJ, Brailey K, Constans JI, Sutker PB. Attention and memory dysfunction in posttraumatic stress disorder. Neuropsychology 1998;12:125–133. [PubMed: 9460740]

Vasterling JJ, Duke LM, Brailey K, Constans JI, Allain AN, Jr. Sutker PB. Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparisons. Neuropsychology 2002;16:5–14. [PubMed: 11853357]

Villarreal G, Hamilton DA, Petropoulos H, Driscoll I, Rowland LM, Griego JA, et al. Reduced hippocampal volume and total white matter volume in posttraumatic stress disorder. Biological Psychiatry 2002;52:119–125. [PubMed: 12114003]

Wechsler, D. Wechsler Adult Intelligence Scale—Third Edition (WAIS–III). The Psychological Corporation; San Antonio, TX: 1997a.

Wechsler, D. Wechsler Memory Scale—Third Edition (WMS–III). The Psychological Corporation; San Antonio, TX: 1997b.

Yehuda R, Keefe RS, Harvey PD, Levengood RA, Gerber DK, Geni J, et al. Learning and memory in combat veterans with posttraumatic stress disorder. American Journal of Psychiatry 1995;152:137–139. [PubMed: 7802106]

Zalewski C, Thompson W, Gottesman II. Comparison of neuropsychological test performance in PTSD, generalized anxiety disorder, and control Vietnam veterans. Assessment 1994;1:133–142. [PubMed: 9465143]

Table 1

Demographic, Clinical, and Background Variables

Variable	PTSD+/ETOH+ (*n* = 30)			PTSD+/ETOH− (*n* = 37)			PTSD−/ETOH+ (*n* = 30)			PTSD−/ETOH− (*n* = 31)			*F* or χ^2	*p*	Significant contrasts
	M	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%			
Age	48.57	7.7		47.89	9.4		43.60	12.0		46.42	9.6				None
Main effect for PTSD													*F*(3, 124) = 3.46	.065	
Main effect for ETOH													*F*(3, 124) = 0.384	.537	
Interactive effect													*F*(3, 124) = 1.02	.315	
Education	13.50	1.6		14.89	2.4		14.17	1.7		15.39	2.0				
Main effect for PTSD													*F*(3, 124) = 2.69	.104	
Main effect for ETOH													*F*(3, 124) = 13.58	.000	ETOH+ < ETOH−
Interactive effect													*F*(3, 124) = 0.059	.809	
Gender													χ^2(3, *N* = 128) = 0.09	.880	None
Men			93			84			96			84			
Women			7			16			4			16			
Ethnicity															None
Asian			3			3			0			3			
Black			21			22			10			26			
Hispanic			3			6			10			0			
White			73			68			80			71			
CAPS total score	65.16	16.93		65.13	19.66		3.07	4.81		0.87	2.08				
Main effect for PTSD													*F*(3, 124) = 704.32	.000	PTSD+ > PTSD−
Main effect for ETOH													*F*(3, 124) = 0.207	.650	
Interactive effect													*F*(3, 124) = 0.218	.641	
SCL-90-Depression subscale	1.77	0.72		1.85	0.96		0.70	0.47		0.46	0.72				
Main effect for PTSD													*F*(3, 124) = 85.14	.000	PTSD+ > PTSD−
Main effect for ETOH													*F*(3, 124) = 0.346	.557	

Variable	PTSD+/ETOH+ (*n* = 30)			PTSD+/ETOH− (*n* = 37)			PTSD−/ETOH+ (*n* = 30)			PTSD−/ETOH− (*n* = 31)			*F* or χ^2	*p*	Significant contrasts
	M	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%			
Interactive effect													*F*(3, 124) = 1.40	.240	
WAIS-III Vocabulary	46.40	10.63		50.08	9.58		51.47	7.54		53.87	8.17				
Main effect for PTSD													*F*(3, 124) = 7.55	.007	PTSD+ < PTSD−
Main effect for ETOH													*F*(3, 124) = 3.56	.061	
Interactive effect													*F*(3, 124) = 0.157	.693	
LDH total drinks	83,745	86,234		21,657	38,050		45,950	41,442		15,289	44,925				
Main effect for PTSD													*F*(3, 123) = 5.03	.027	PTSD+ > PTSD−
Main effect for ETOH													*F*(3, 123) = 22.17	.000	ETOH+ > ETOH−
Interactive effect													*F*(3, 123) = 2.55	.113	
LDH 3-month drink total	90.02	209.75		26.23	54.12		166.41	240.48		53.71	105.09				
Main effect for PTSD													*F*(1, 122) = 3.08	.082	
Main effect for ETOH													*F*(1, 122) = 8.90	.003	ETOH+ > ETOH−
Interactive effect													*F*(1, 122) = 0.683	.410	
Other diagnoses															
MDD			27			43			3			6	χ^2(3, *N* = 128) = 4.55	.033	PTSD+ > PTSD−
Anxiety disorders			40			30			10			3	χ^2(3, *N* = 128) = 3.90	.048	PTSD+ > PTSD−
Past drug abuse or dependence			53			32			50			6	χ^2(3, *N* = 128) = 18.57	.000	PTSD+ > PTSD−, ETOH+ > ETOH−
Current psychiatric medication use			56			30			43			26	χ^2(3, *N* = 128) = 7.80	.050	PTSD+ > PTSD−
Traumatic event															
Vietnam combat			63			57			10			10			

Variable	PTSD+/ETOH+ (*n* = 30)			PTSD+/ETOH− (*n* = 37)			PTSD−/ETOH+ (*n* = 30)			PTSD−/ETOH− (*n* = 31)			*F* or χ^2	*p*	Significant contrasts
	M	*SD*	%	*M*	*SD*	%	*M*	*SD*	%	*M*	*SD*	%			
Gulf War combat			3			14			0			0			
Military-related			24			21			17			16			
Nonmilitary			13			8			46			33			
None			0			0			17			42			

Note. All scores are raw scores. Military-related traumas include accidents or physical or sexual assaults. Nonmilitary traumas include child abuse and accidents. PTSD+ = current diagnosis of posttraumatic stress disorder (PTSD); PTSD− = no history of diagnosis of PTSD; ETOH = ethonol, or alcohol; ETOH+ = diagnosis of alcohol abuse or dependence in the past 5 years; ETOH− = no diagnosis of alcohol abuse or dependence in the past 5 years; CAPS = Clinician Administered PTSD Scale; SCL-90-R = Symptom Checklist—90-R; WAIS—III = Wechsler Adult Intelligence Scale—Third Edition; LDH = Lifetime Drinking History; MDD = major depressive disorder.

Table 2

Verbal Memory Performance

Test	PTSD+/ETOH+ (*n* = 30)		PTSD+/ETOH− (*n* = 37)		PTSD−/ETOH+ (*n* = 30)		PTSD−/ETOH− (*n* = 31)		*F*	*p*	Significant contrasts (Bonferroni alpha = .019)
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*			
CVLT Trials 1−5	44.50	11.29	46.49	10.20	52.30	9.90	52.26	9.36			
Main effect for PTSD									*F*(3, 121) = 7.863	.006	PTSD + < PTSD−
Main effect for ETOH									*F*(3, 121) = 0.089	.766	
Interactive effect									*F*(3, 121) = 0.349	.556	
CVLT Trial 1	5.93	1.89	5.84	1.89	7.00	1.68	7.55	1.82			
Main effect for PTSD									*F*(3, 121) = 12.91	.000	PTSD + < PTSD−
Main effect for ETOH									*F*(3, 121) = 0.219	.641	
Interactive effect									*F*(3, 121) = 1.14	.288	
CVLT Long-Delay Free Recall	9.70	3.34	10.92	2.63	11.37	2.71	11.10	2.84			None
Main effect for PTSD									*F*(3, 121) = 0.710	.401	
Main effect for ETOH									*F*(3, 121) = 0.058	.810	
Interactive effect									*F*(3, 121) = 2.36	.127	
WMS—III Logical Memory I Recall	46.58	9.40	45.34	10.20	49.37	9.20	49.68	8.00			None
Main effect for PTSD									*F*(3, 112) = 0.416	.520	
Main effect for ETOH									*F*(3, 112) = 1.65	.201	
Interactive effect									*F*(3, 112) = 0.232	.631	
WMS—III Logical Memory II Recall	30.38	6.96	27.91	7.73	32.07	6.40	32.81	5.77			None
Main effect for PTSD									*F*(3, 112) = 1.397	.240	
Main effect for ETOH									*F*(3, 112) = 4.743	.032	
Interactive effect									*F*(3, 112) = 1.988	.161	
WMS—III Logical Memory % Retention	89.04	7.10	85.16	10.30	91.33	8.40	91.35	6.50			None
Main effect for PTSD									*F*(3, 112) = 4.954	.028	
Main effect for ETOH									*F*(3, 112) = 3.462	.065	
Interactive effect									*F*(3, 112) = 1.509	.222	

Note. All scores are raw scores. All analysis of covariance covaried for education, WAIS—III Vocabulary raw score, and SCL-90-R Depression residualized score. PTSD+ = current diagnosis of posttraumatic stress disorder (PTSD); PTSD− = no history of diagnosis of PTSD; ETOH = ethanol, or alcohol; ETOH+ = diagnosis of alcohol abuse or dependence in the past 5 years; ETOH− = no diagnosis of alcohol abuse or dependence in the past 5 years; CVLT = California Verbal Learning Test; WMS—III = Wechsler Memory Scale—Third Edition; WAIS—III = Wechsler Adult Intelligence Scale—Third Edition.

Table 3

Working Memory, Attention, and Processing Speed Performance

Test	PTSD+/ETOH+ (*n* = 30)		PTSD+/ETOH− (*n* = 37)		PTSD−/ETOH+ (*n* = 30)		PTSD−/ETOH− (*n* = 31)		*F*	*p*	Significant contrasts (Bonferroni alpha = .025)
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*			
WMS—III Spatial Span	15.17	3.22	15.19	3.50	16.10	3.32	15.90	3.02			
Main effect for PTSD									*F*(3, 120) = 3.125	.574	None
Main effect for ETOH									*F*(3, 120) = 7.978	.370	
Interactive effect									*F*(3, 120) = 0.029	.865	
WMS—III Letter Number Sequencing	10.00	1.96	9.84	2.47	11.93	2.45	11.58	2.19			
Main effect for PTSD									*F*(3, 112) = 12.481	.001	PTSD + < PTSD−
Main effect for ETOH									*F*(3, 112) = 1.010	.317	
Interactive effect									*F*(3, 112) = 0.140	.709	
WMS—III Digit Span	16.27	3.92	15.30	4.21	18.70	3.85	19.52	4.46			
Main effect for PTSD									*F*(3, 121) = 12.90	.000	PTSD + < PTSD−
Main effect for ETOH									*F*(3, 121) = 1.06	.306	
Interactive effect									*F*(3, 121) = 2.04	.156	
WAIS—III Digit Symbol	62.04	13.33	60.17	16.63	71.17	12.54	72.27	15.38			
Main effect for PTSD									*F*(3, 109) = 11.726	.001	PTSD + < PTSD−
Main effect for ETOH									*F*(3, 109) = 0.225	.636	
Interactive effect									*F*(3, 109) = 0.155	.695	

Note. All scores are raw scores. All analyses of covariance covaried for education, WAIS—III vocabulary raw score, and SCL-90-R Depression residualized score. PTSD+ = current diagnosis of posttraumatic stress disorder (PTSD); PTSD− = no history of diagnosis of PTSD; ETOH = ethanol, or alcohol; ETOH+ = diagnosis of alcohol abuse or dependence in the past 5 years; ETOH− = no diagnosis of alcohol abuse or dependence in the past 5 years; WMS—III = Wechsler Memory Scale—Third Edition; WAIS—III = Wechsler Adult Intelligence Scale—Third Edition.

Table 4

Visual—Spatial and Visual Memory Performance

Test	PTSD+/ETOH+ (*n* = 30)		PTSD+/ETOH− (*n* = 37)		PTSD−/ETOH+ (*n* = 30)		PTSD−/ETOH− (*n* = 31)		*F*	*p*	Significant contrasts (Bonferroni alpha = .028)
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*			
WMS—III Visual Immediate Index	17.33	4.80	20.59	3.70	18.73	4.10	20.55	14.90			
Main effect for PTSD									*F*(3, 121) = 0.005	.946	
Main effect for ETOH									*F*(3, 121) = 6.411	.013	ETOH + < ETOH−
Interactive effect									*F*(3, 121) = 0.576	.449	
WMS—III Visual Delayed Index	18.10	4.70	20.59	4.30	18.50	3.90	20.77	4.70			None
Main effect for PTSD									*F*(3, 121) = 1.031	.312	
Main effect for ETOH									*F*(3, 121) = 4.218	.042	
Interactive effect									*F*(3, 121) = 0.001	.979	
WAIS—III Block Design	38.73	11.52	38.11	11.22	41.33	12.08	41.61	12.08			
Main effect for PTSD									*F*(3, 121) = 0.304	.583	None
Main effect for ETOH									*F*(3, 121) = 0.476	.492	
Interactive effect									*F*(3, 121) = 0.059	.808	

Note. All scores are raw scores. All analyses of covariance covaried for education, WAIS—III vocabulary raw score, and SCL-90-R Depression residualized score. PTSD+ = current diagnosis of posttraumatic stress disorder (PTSD); PTSD− = no history of diagnosis of PTSD; ETOH = ethanol, or alcohol; ETOH+ = diagnosis of alcohol abuse or dependence in the past 5 years; ETOH− = no diagnosis of alcohol abuse or dependence in the past 5 years; WMS—III = Wechsler Memory Scale—Third Edition; WAIS—III = Wechsler Adult Intelligence Scale—Third Edition.