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Verbal learning and memory in alcohol abusers and polysubstance abusers with concurrent alcohol abuse

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Abstract

To define the combined effects of drug and alcohol abuse on verbal learning and memory, 70 alcoholic and 80 polysubstance abuse (PSA) individuals with concurrent alcohol abuse were compared on a list learning task, the California Verbal Learning Test (CVLT). Despite demonstrating similar learning strategies, response styles, and error patterns, the PSA group nonetheless exhibited significantly greater recall deficits than the alcoholic group on the CVLT. These deficits were particularly evident in those who were heaviest abusers of cocaine. PSA participants did not, however, evidence greater recognition memory deficits. This pattern of greater deficits on recall than on recognition memory, as well as poor consolidation, is consistent with the initiation–retrieval difficulties of patient groups with subcortical dysfunction. It is concluded that the combined use of alcohol and drugs, cocaine in particular, may compound memory difficulties beyond what is typically observed in alcoholic individuals. (*JINS*, 1998, *4*, 319–328.)

Keywords: Alcohol abuse, Polydrug abuse, Learning, Memory, Neuropsychology

INTRODUCTION

Although neuropsychological impairments have been well documented in chronic alcoholics (Grant, 1987; Parsons & Farr, 1981; Ryan & Butters, 1986), more recent studies have also investigated the neurocognitive sequelae of substance abuse (Reed & Grant, 1990; Spencer & Boren, 1990). Deficits in learning and memory, attention, visuospatial abilities, and problem solving have all been observed (see Reed & Grant, 1990; Spencer & Boren, 1990). Investigators typically have examined the neuropsychological profiles of persons who abused a single substance, yet results across these studies have often been equivocal. For example, initial studies examining marijuana abuse found no evidence of associated cognitive impairment (Grant et al., 1978), but more recent investigations have demonstrated deficits in attention, information processing, and memory (Fletcher et al., 1996; Hooker & Jones, 1987; Varma et al., 1988; Wetzel et al., 1982). Many of these early studies of substance abuse suffered from methodological difficulties such as small or nonrepresentative samples or lack of adequate measures of drug consumption.

Further limiting the scope and generalizability of these earlier studies have been recent changes in the demographics of drug abuse, particularly increases in concurrent alcohol abuse among drug abusers (Mehrabian & Straubinger, 1989; Rainone et al., 1987). Although polysubstance abuse is prevalent, very few studies have investigated the combined effects of alcohol and polysubstance abuse on cognition. Those that did (cf. Grant et al., 1978, 1979) tended to have polydrug samples who were not necessarily alcoholic. Furthermore, many cognitive studies of polysubstance abuse have excluded individuals with concurrent alcohol abuse (e.g., Mittenberg & Motta, 1993; O'Malley et al., 1992). Given the potential for additive and/or interactive effects as well as the increased prevalence of combined drug and alcohol abuse, a critical examination of the cognitive status of such individuals appears warranted.

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In related research, recent neuroimaging studies have documented cerebral abnormalities associated with chronic drug abuse. Volkow et al. (1988b) reported deep white matter changes on magnetic resonance imaging (MRI) in patients abusing heroin and/or cocaine, and cerebral atrophy has been reported in chronic cocaine abusers (Pascual-Leone et al., 1991). Through the use of positron emission tomography (PET) and single photon emission computed tomography (SPECT), cerebral perfusion abnormalities have also been associated with cocaine abuse (Volkow et al., 1988a) and polysubstance abuse (Holman et al., 1991). Central stimulant drugs are associated with higher risk of cerebrovascular accident (Freilich & Byrne, 1992), and Volkow et al. (1988a) have suggested that the direct action of these drugs on cerebral vessels could induce ischemia, hemorrhage, and necrosis secondary to vasospasm. Volkow and colleagues further argue that stimulant drug abuse in combination with alcohol consumption may be particularly detrimental, inasmuch as alcohol is also associated with vasoconstriction (Altura & Altura, 1983) and a higher incidence of cerebrovascular accidents (Gill et al., 1986; Hillborn & Kaste, 1981). Consistent with these suppositions, Aasly et al. (1993) found white matter changes on MRI examinations in a group of young male drug abusers with concurrent alcohol abuse. These authors suggest that the structural brain changes are likely to be the result of high alcohol consumption in parallel with drug abuse.

The current study was designed to examine whether polysubstance abusers with concurrent alcohol abuse would display greater impairments than alcoholics on a test of verbal learning and memory, the California Verbal Learning Test (CVLT; Delis et al., 1987). The CVLT was developed to enhance diagnosticians' accuracy in identifying and characterizing different memory disorders by not only evaluating the magnitude of learning and memory impairments, but also by evaluating the cognitive processes leading to impaired performance. The qualitative aspects of CVLT performance include identification of learning strategies and an analysis of error types. Thus, the CVLT is well suited to measure subtle changes in verbal learning and memory ability (Delis et al., 1991; Kramer et al., 1988).

METHODS

Research Participants

One hundred and fifty men participated in the study. All were selected from admissions to a 28-day inpatient alcohol and drug treatment program at the San Diego Department of Veterans Affairs Medical Center. Candidates with a previous history of neurologic or psychiatric disorders, head trauma, or metabolic disease were excluded from participation. The 150 participants were diagnosed as alcohol abusing and/or dependent according to DSM–III–R criteria (American Psychiatric Association, 1987), and 80 were further classified as polysubstance abusers (PSA; DSM–III–R criteria for abuse and/or dependence of other substances). Table 1 presents the two groups' demographic, affective and alcohol consumption data. The alcoholic participants were significantly older than the polysubstance abuse (PSA) participants [$t(148) = 4.4$, $p < .001$], but they did not differ in the number of years of formal education [$t(148) = 1.5$, $p = .14$], age-corrected scaled scores of the WAIS–R Vocabulary subtest [$t(148) = 1.8$, $p = .07$], or race/ethnicity [$\chi^2(3, N = 150) = 3.7$, $p = .29$]. The PSA participants demonstrated significantly higher scores on the Hamilton Rating Scale for Depression [$t(148) = 2.1$, $p = .04$], although neither group's mean score was indicative of a clinically significant degree of depression. The alcoholic participants did not differ from PSA participants in the average or maximum number of drinks per day during the 3-month interval prior to admission [average: $t(148) = 1.2$, $p = .21$; maxi-

Table 1. Demographic and group characteristics

Variable	Alcoholics ($N = 70$)	Polysubstance abusers ($N = 80$)
Age [*M*, (*SD*)]	44.0 (10.9)	37.6** (6.7)
Education [*M*, (*SD*)]	13.5 (1.9)	13.1 (1.4)
WAIS–R Vocabulary (age-corrected scaled score) [*M*, (*SD*)]	10.2 (2.5)	9.5† (2.3)
Hamilton Rating Scale for Depression (score at discharge) [*M*, (*SD*)]	6.5 (4.9)	8.3* (5.5)
Ethnicity (*N*)		
Mexican-American–Latino	4	2
African-American–Black	14	21
Caucasian–White	51	56
Malayan–Filipino–Other Asian	1	1
Mean number of days since last drink [*M*, (*SD*)]	15.8 (23.0)	20.5 (45.4)
Mean number of drinks per day [3 months prior to admission; *M*, (*SD*)]	13.9 (7.7)	12.2 (8.5)
Years of alcohol abuse [*M*, (*SD*)]	20.7 (11.5)	15.4** (7.8)
Years of drug abuse [*M*, (*SD*)]	n/a	15.4 (8.3)

*$p < .05$, **$p < .001$, †$p = .07$.

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mum: $t < 1$], or the number of days since their last drink ($t < 1$). Because the alcoholic participants were older than the PSA subjects, it was not unexpected that the alcoholic group demonstrated a significantly greater total number of years of alcohol abuse [$t(148) = 3.3$, $p = .001$].

PSA participants' drug consumption profiles are presented in Table 2. Mean lifetime estimates of the number of occasions of drug use and the median number of days since last use of the drug are presented. Quantifying the number of occasions of drug use was defined as any use of that substance per day. For example, an estimate of 500 occasions of cocaine use would indicate a minimum of 500 uses of cocaine, irrespective of the amount ingested per occasion. The PSAs reported significantly greater amounts and more recent use of substances than did the alcoholic group. Three drug types—marijuana, cocaine, and amphetamines (e.g., "crystal" methamphetamine)—were associated with the most recent and greatest amount of use by this sample of PSAs.

Procedure

All participants were tested during the 3rd or 4th week of their inpatient treatment stay. The CVLT (Delis et al., 1987) was administered according to the standard procedure by trained psychometrists as part of a larger set of neuropsychological tests. The administration of the CVLT begins with an oral presentation of a "shopping" list of 16 items (List A) over five learning trials. The list consists of four items from each of four semantically distinct categories (*fruits*, *herbs and spices*, *clothing*, and *tools*). Adjacent words on the list are from different categories, which allows assessment of the participant's learning strategy (i.e., whether they recall words clustered in semantic categories or attempt to recall the list in the order presented). Following the five learning trials, a second, interference list (List B) is presented for one trial. Immediate recall (both free and category-cued) of List A is then tested. After a 20-min interval during which nonverbal testing occurs, delayed recall (both free and category-cued) and yes–no recognition of List A are assessed. During immediate and delayed recall, responses not on the list are counted as intrusion errors. In all, three broad areas of mnemonic function are evaluated with the CVLT: acquisition, retention, and error responding.

The paper-and-pencil protocols were scored using the CVLT scoring software (Fridlund & Delis, 1987). Descriptions of the CVLT variables analyzed in this study are provided in a number of sources (e.g., Delis et al., 1987, 1991). Fortunately, extensive normative data on the various CVLT indices made it possible to convert raw scores to standard scores within different age ranges and by sex, controlling for the effects of these two important variables. For example, a mean standard score of −1.0 would indicate that subjects scored on the average of 1 standard deviation below persons of their age and gender group in the CVLT normative sample. This standard score conversion was vital for the current investigation because of the age difference between the alcoholics and polysubstance abusers and because all of the participants were men.

RESULTS

Preliminary Group Comparisons

Univariate analyses

Because both groups were comprised of participants diagnosed as alcohol abusing and/or dependent, we expected any group differences, if present, to be small. Thus, al-

Table 2. Drug use characteristics (lifetime indices)

Substance	Alcoholics ($N = 70$)	Polysubstance abusers ($N = 80$)
Mean lifetime estimate of the number of occasions of drug use [*M*, (*SD*)]		
Marijuana	276 (1425)	3529 (4645)
Cocaine	17 (58)	1245 (2082)
Amphetamines	12 (52)	789 (1515)
Opiates	0 (1)	501 (1562)
Barbiturates	5 (28)	175 (547)
Hallucinogens	9 (41)	103 (237)
Median number of days since last drug use		
Marijuana	1825 [$N = 37$]	30 [$N = 75$]
Cocaine	1095 [$N = 22$]	33 [$N = 72$]
Amphetamines	730 [$N = 9$]	66 [$N = 58$]
Opiates	5110 [$N = 3$]	1460 [$N = 31$]
Barbiturates	3650 [$N = 9$]	2000 [$N = 37$]
Hallucinogens	3650 [$N = 11$]	1912 [$N = 54$]

Note. Brackets indicate the number of subjects reporting any lifetime use of that substance.

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though a relatively large number of statistical tests were conducted, a testwise alpha level of .01 (two-tailed) was adopted as a compromise between lowering the potential for capitalization on chance and efforts to maintain sufficient power in this exploratory study. Subsequent to these initial comparisons, however, a second tier of analyses, with data reduction procedures, was also performed to further address the possibility of capitalization on chance because of the large number of statistical comparisons.

Table 3 shows the standard scores for both groups on each of the CVLT variables. PSA participants' performances were significantly poorer than those of the alcoholic participants on five measures of item learning and recall, and significantly greater on a summary measure of improvement on recognition memory relative to free recall (Recognition Discriminability *vs.* List A Trial 5 Recall). A borderline nonsignificant trend ($.01 < p < .05$) was also observed on the long-delay free recall trial ($p = .012$), the number of free recall intrusions ($p = .015$), and on a measure reflecting the use of an efficient organizational strategy during learning (i.e., semantic cluster ratio, $p = .027$).

Logistic regressions

A series of logistic regression (LR) analyses were then performed in an effort to assess the relation between group membership and each of the CVLT variables independent of the potentially confounding effects of education level, verbal intellectual ability (as indexed by the age-corrected WAIS–R Vocabulary score), and severity of depressive symptoms (as indexed by the Hamilton Rating Scale for Depression score). Although educational attainment strongly predicts performance on many neuropsychological tasks, it may not necessarily be redundant with intellectual ability; thus, WAIS–R Vocabulary was also used to assess verbal intellectual ability. LR analyses confirmed the stability of group differences on all five of the CVLT variables demonstrating significant group differences after education, age-corrected WAIS–R Vocabulary and Hamilton Rating Scale for Depression scores were taken into account (see Table 3). In addition, none of the variables demonstrating borderline nonsignificant trends attained significance following the LR analyses.

Principal components analysis

Nineteen age-residualized CVLT measures identical to the variables used by Delis et al. (1988) were then submitted to a principal components analysis (PCA), which included two variables that were combinations of other variables (e.g., *List B* vs. *List A Trial 1 Recall*, and *Short-Delay Free Recall* vs. *Trial 5 Recall*). These additional analyses were undertaken (1) to minimize capitalization on chance by reducing the large number of CVLT variables into a smaller set of conceptually meaningful groups (i.e., learning and memory, error responding, learning strategy, etc.), and (2) to de-

Table 3. Standardized (*Z*) scores of alcoholic and polysubstance abuse (PSA) participants on variables derived from the California Verbal Learning Test (CVLT)

	Alcoholic ($N = 70$)	PSA ($N = 80$)	*t* test	Logistic regression
Score	*M* (*SD*)	*M* (*SD*)	*p* value	*p* value*
List A Total Recall (*T* score)	48.9 (12.1)	42.7 (13.9)	.005	.007
List A Trial 5 Recall	−0.10 (1.32)	−0.93 (1.82)	.002	.003
List B Total Recall	−0.50 (1.26)	−0.68 (1.26)	.398	.519
Semantic Clustering	−0.29 (1.10)	−0.68 (1.03)	.027	.059
Serial Cluster Ratio	0.21 (1.26)	0.38 (1.12)	.409	.507
Percent Primacy Recall	0.07 (1.07)	0.19 (0.96)	.483	.369
Percent Recency Recall	−0.39 (0.84)	−0.34 (0.93)	.740	.860
Recall Consistency	−0.40 (1.07)	−0.58 (1.00)	.300	.154
Learning Slope	0.03 (1.09)	−0.09 (1.25)	.547	.416
Short Delay Free Recall	0.27 (1.14)	−0.60 (1.38)	<.001	<.001
Short Delay Cued Recall	0.10 (1.13)	−0.64 (1.41)	.001	.001
Long Delay Free Recall	0.00 (1.18)	−0.54 (1.37)	.012	.028
Long Delay Cued Recall	0.14 (1.22)	−0.70 (1.61)	<.001	<.001
Recognition Discriminability	−0.24 (0.84)	−0.39 (0.72)	.259	.248
Discriminability *versus* Trial 5 Recall	−0.14 (1.16)	0.54 (1.58)	.003	.006
False Positives	0.30 (0.95)	0.45 (0.71)	.273	.141
Free Recall Intrusions	−0.31 (0.97)	0.11 (1.13)	.015	.036
Cued Recall Intrusions	−0.31 (0.81)	−0.08 (1.34)	.195	.142
Perseverations	0.44 (1.46)	0.35 (1.46)	.698	.556

*Logistic regressions were performed in which education level, WAIS–R Vocabulary score, Hamilton Rating Scale for Depression score, and a single CVLT variable were simultaneously entered as predictors of group membership. Two-tailed *p*-values associated with the independent contributions of each CVLT variable are reported.

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termine if the current sample of participants would yield a comparable factor structure to the normative sample factor structure of the CVLT obtained by Delis et al. (1988). Thus, age-residualized standard scores from 19 CVLT indices were factor analyzed using a principal components procedure. Factors whose eigenvalues were greater than 1.0 were retained and loadings greater than .50 were considered significant.

As shown in Table 4, the varimax-rotated (orthogonal) factor matrix revealed a seven-factor solution. The eigenvalues associated with the solution were 6.4, 1.8, 1.7, 1.5, 1.2, 1.1, and 1.0, each accounting for 34%, 10%, 9%, 8%, 6%, 6%, and 5% of the variance, respectively. The *first* factor closely approximated the *General Verbal Learning and Memory* factor of Delis et al. (1988) and contained significant loadings from the following variables: List A Total Recall, Semantic Clustering, Consistency of Item Recall, Short-Delay Free Recall, Short-Delay Cued Recall, Long-Delay Free Recall, Long-Delay Cued Recall, and Recognition Hits. List B Recall and Short Delay *versus* Trial 5 Recall were the only two variables that were contained in the first factor described by Delis et al. that did not significantly load on this first factor in the current investigation. It should be noted, however, that Delis et al. considered factor loadings to be significant if they were greater than .4, and List B Recall displayed the smallest loading (.42) on this first factor in the Delis et al. study. The two indices loading on the *second* factor (List B Recall; List B *vs.* List A Trial 1 Recall) were identical to those of the *Proactive Effect* factor of Delis et al. (1988).

The *third* factor in the present study revealed a significant loading on the Learning Slope variable, which corresponds exactly to the *Acquisition Rate* factor in the Delis et al. study. The *fourth* factor, which showed a significant loading on False Positive Errors, did not demonstrate any obvious similarity with those obtained by Delis et al. (1988). Indices loading on the *fifth* factor (Semantic Clustering; Serial Clustering) were identical to those of the *Learning Strategy* factor of Delis et al. (1988). The *sixth* factor in the present study revealed significant loadings on the Percent Primacy Recall and Percent Recency Recall variables, which corresponds exactly to the *Serial Position Effect* factor in the Delis et al. study. The *seventh* and final factor showed significant loadings on Cued Recall Intrusions and Perseverations, which corresponds approximately to the error responding or *Response Discrimination* factor described by Delis et al. In all, six of the seven factors derived from the present PCA very closely approximated those obtained by Delis et al. (1988).

Univariate analyses with factor scores

Following the PCA, independent samples *t* tests, with component scores as dependent variables, were performed for each component or factor (see Table 4). Results revealed that the PSA participants obtained significantly lower component scores than alcoholic participants only on the first *General Verbal Learning and Memory* component [$t(148) = 3.3, p = .001$]. All other comparisons did not attain statistical significance ($ts < 1$).

Follow-Up Comparisons

Regression analyses: Demographic and consumption influences

In a follow-up analysis, a number of demographic, drug and alcohol consumption variables were entered into two separate regression analyses in order to evaluate their relative contributions to PSA participants' performance on the *General Verbal Learning and Memory* factor. These variables included (1) education level and WAIS–R Vocabulary scores, (2) mean and maximum number of drinks per day during the 3 months prior to admission, (3) days since last drink prior to admission, (4) total number of years of alcohol abuse, and (5) lifetime estimates of the number of occasions of marijuana, hallucinogen, opiate, amphetamine, and cocaine use. Because the drug consumption variables had such wide ranges (i.e., 0–10,000 or more reported occasions of drug use), all individual data points for each of these five drug use variables were natural-log (*ln*) transformed in order to reduce the impact of any outliers in the analysis.

Influence of alcohol consumption. The first regression analysis was undertaken to determine the effects of recent use of alcohol, since previous research has suggested that recent use is more predictive of neuropsychological performance than estimates of cumulative use (see Grant, 1987, for review). The regression (with backward elimination) was run in a stepwise manner, with education and WAIS–R Vocabulary forced in initially as a set. With this procedure it was pos-

Table 4. Results of principal components analysis of California Verbal Learning Test variables

Principal component	Eigenvalue	Proportion of variance	$t(148)$	p value
Verbal Learning and Memory	6.38	33.6	3.34	<.001
Proactive Interference Effect	1.78	9.4	<1.00	n.s.
Acquisition Rate	1.72	9.0	<1.00	n.s.
False Positive Errors	1.48	7.8	<1.00	n.s.
Learning Strategy	1.15	6.1	<1.00	n.s.
Serial Position Effect	1.08	5.7	<1.00	n.s.
Response Discrimination	1.00	5.3	<1.00	n.s.

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sible to determine what effect the recent alcohol use variables might have on CVLT performance while controlling for the effects of factors that influence performance on neuropsychological tasks; that is, verbal intellectual ability (Schafer et al., 1991). Only two predictor variables, WAIS–R Vocabulary ($p < .002$) and a borderline significant effect for the number of drinks per day in the 3 months prior to admission ($p = .07$), were major contributors to performance on the *General Verbal Learning and Memory* component ($R^2 = .167$, $p < .001$). Neither maximum number of drinks per day (in the previous 3 months) nor days since last drink accounted for a significant portion of the variance on the *General Verbal Learning and Memory* component.

Influence of drug consumption. The second regression analysis utilized the estimates of cumulative drug and alcohol use. Again, the regression (with backward elimination) was run in a stepwise manner, with education and WAIS–R Vocabulary forced in initially as a set. Again, only two predictor variables, WAIS–R Vocabulary ($p = .001$) and years of alcohol abuse ($p = .007$), contributed to performance on the General Verbal Learning and Memory component ($R^2 = .224$, $p < .001$). None of the remaining cumulative drinking or drug use variables accounted for a significant portion of the variance on the *General Verbal Learning and Memory* component.

Effects of Cocaine Abuse

Because of evidence suggesting that cocaine abusers may be differentially vulnerable to learning and memory deficits (Ardila et al., 1990; Manschreck et al., 1990; Mittenberg & Motta, 1993; O'Malley et al., 1992), a final analysis of the PSA participants was performed. The PSA participants were divided into two groups based on the amount of cocaine use they reported; those who had reported less than 500 occasions were separated from those who reported 500 or more occasions of cocaine use (*PSA/cocaine*) and were directly compared on the CVLT. Both groups reported comparable levels of alcohol use (i.e., years of alcohol use; mean and maximum number of drinks per day in the 3 months prior to admission; and days since last drink), marijuana, opiate and hallucinogen use (all ts < 1.1, based on *ln* transformations of drug use information because of the large ranges of values). PSA participants ($N = 35$) did not differ from PSA/cocaine participants ($N = 45$) on years of education ($t < 1$), race ($\chi^2 < 1$), WAIS–R Vocabulary [$t(78) = 1.6$, $p = .11$], or Hamilton Rating Scale for Depression scores ($t < 1$). PSA participants ($M = 35.8$ years, $SD = 7.1$) were significantly younger than the PSA/cocaine group [$M = 39.0$ years, $SD = 6.2$; $t(78) = 2.1$, $p = .04$]. Thus, age-residualized CVLT scores were again utilized for these analyses.

Table 5 shows the CVLT scores of these two subgroups of PSA participants. PSA/cocaine participants' performances were significantly poorer than PSA participants on five measures: (1) List A Total Recall [$t(78) = 2.2$, $p = .03$], (2) List B Recall [$t(78) = 2.5$, $p = .01$], (3) Short-Delay Free Recall [$t(78) = 2.2$, $p < .03$], (4) Perseverations [$t(78) = 2.1$, $p = .04$], and (5) the measure of an efficient organizational strategy (Semantic Clustering) [$t(78) = 2.9$, $p = .005$]. A signif-

Table 5. Standardized scores of polysubstance abusers who reported more (PSA–Cocaine) or less than (PSA) 500 occasions of cocaine use on the California Verbal Learning Test (CVLT)

	PSA ($N = 35$)	PSA–Cocaine ($N = 45$)
Score	*M* (*SD*)	*M* (*SD*)
List A Total Recall (*T* score)	46.2 (14.2)	39.8 (13.1)*
List A Trial 5 Recall	−0.74 (1.80)	−1.07 (1.84)
List B Total Recall	−0.29 (1.10)	−0.98 (1.31)*
Semantic Clustering	−0.31 (1.13)	−0.96 (0.85)**
Serial Cluster Ratio	0.31 (1.25)	0.42 (1.01)
Percent Primacy Recall	0.14 (0.77)	0.22 (1.08)
Percent Recency Recall	−0.54 (0.61)	−0.17 (1.09)
Recall Consistency	−0.37 (0.97)	−0.73 (0.98)
Learning Slope	−0.14 (1.11)	−0.04 (1.35)
Short Delay Free Recall	−0.23 (1.33)	−0.89 (1.37)*
Short Delay Cued Recall	−0.31 (1.37)	−0.89 (1.40)
Long Delay Free Recall	−0.34 (1.24)	−0.69 (1.46)
Long Delay Cued Recall	−0.46 (1.52)	−0.89 (1.67)
Recognition Discriminability	−0.29 (0.71)	−0.47 (0.73)
Discriminability *versus* List A Trial 5 Recall	0.46 (1.52)	0.60 (1.63)
False Positives	0.40 (0.60)	0.49 (0.79)
Free Recall Intrusions	−0.11 (0.76)	0.29 (1.32)
Cued Recall Intrusions	−0.06 (1.24)	−0.09 (1.43)
Perseverations	−0.03 (1.27)	0.64 (1.54)*

*$p = .05$, **$p = .01$.

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icant group difference was also demonstrated on the *General Verbal Learning and Memory* [$t(78) = 2.2, p = .03$], and *Proactive Effect* components [$t(78) = 2.0, p = .05$]. It should be noted that in no instance was the PSA/cocaine group's mean performance better than that of the PSA group for any of the quantitative CVLT measures of acquisition, item recall, or recognition memory.

DISCUSSION

The present findings demonstrated that the PSA group performed significantly worse than the alcoholic group on a number of CVLT indices of learning and memory, even though the alcoholic group reported a greater number of years of alcohol abuse. Also, when variables were grouped through the use of principal components analysis, which provided a factor structure highly similar to that of the original CVLT factor structure reported by Delis et al. (1988), the *General Verbal Learning and Memory* factor was significantly lower in the PSA group compared to the alcoholic group. No differences were found on any of the other factor groupings associated with learning strategies, serial position effects, or response discrimination (error responding). These findings suggest that although alcoholic and PSA participants may have approached performing the CVLT through similar strategies, response styles and error patterns, PSA participants nonetheless showed reduced learning and recall relative to the alcoholic participant.

Comparisons of individual CVLT variables also revealed that PSA participants did not differ from alcoholic participants on recognition memory testing. This pattern of impaired performance on recall tasks relative to recognition memory tasks suggests difficulties with retrieval of information rather than actual forgetting of material. A number of studies have documented that patients with subcortical dysfunction and/or lesions demonstrate this pattern of performance (Bondi et al., 1993; Bondi & Kaszniak, 1991; Butters et al., 1985, 1986; Cummings, 1992; Delis et al., 1991; Martone et al., 1984). For example, although patients with Huntington's disease are often as severely impaired as amnesic or demented patients on tests of verbal recall, their recognition memory of comparable material is consistently superior to that of the amnesics or patients with dementia of the Alzheimer type (Butters et al., 1985, 1986; Delis et al., 1991; Folstein et al., 1990). This pattern of performance may be a function of poor initiation of retrieval strategies rather than actual forgetting; nondemented Parkinson's disease patients also display a pattern of superior recognition memory relative to free recall (Bondi et al., 1993; Massman et al., 1990; Taylor et al., 1986). Given that this pattern of performance has been associated with subcortical involvement, the current findings suggest that the memory deficits following polysubstance abuse may be associated with subcortical or corticostriatal dysfunction.

Recent findings of subcortical white matter abnormalities and microvascular changes on MRI in a small group of substance abusers also provide support for the presence of subcortical deficits associated with polysubstance abuse (Volkow et al., 1988a, 1988b). Aasly et al. (1993) found subcortical white matter MRI changes in young drug abusers who also had heavy alcohol consumption. Shear et al. (1994) have also documented white matter loss and significant volumetric white matter increase with abstinence in chronic alcoholic individuals using volumetric MRI analyses. Our results suggest that the combination of alcohol and drug use results in unique performance decrements among PSA participants. This hypothesis is supported both by the early observations of Grant et al. (1979) that polydrug users who also abused alcohol were more impaired neuropsychologically than were young alcoholics as well as by the more recent findings of Aasly et al. (1993) and Shear et al. (1994).

Perfusion studies using PET and SPECT scans in cocaine abusers have also found reductions in cortical perfusion (Holman et al., 1991), blood flow (Volkow et al., 1988a), and glucose utilization in the basal ganglia (Volkow et al., 1991). Given evidence of vascular changes in chronic cocaine abusers (Ardila et al., 1990), the white matter and the basal ganglia may be particularly vulnerable to the effects of vascular changes associated with combined alcohol and drug abuse. Such changes may contribute to the learning and retrieval deficits observed in the PSA participants, especially in those with a history of significant cocaine use.

It should be noted, however, that vascular damage may not be the only etiologic factor contributing to the observed pattern of CVLT performance. A host of studies have also implicated the role of glutamate and N-methyl-D-aspartate (NMDA) receptor disruptions in alcohol, cocaine, and amphetamine use, particularly in structures related to memory such as the hippocampus and ventral striatum (Hunt, 1993; Lovinger, 1993; Rossetti et al., 1992). Given the greater density of NMDA receptors in subcortical structures, neurochemical abnormalities may potentially contribute to the observed "subcortical" pattern of CVLT performance in our sample of PSA participants. Also, intravenous drug use introduces the possibility of additional neurotoxic factors, such as injection of particulate matter, infectious agents, or other toxins that may have an affinity for subcortical structures (see Ballard et al., 1985). Nevertheless, future studies of polysubstance abusers, particularly those investigating metabolic rates or cerebral blood flow, should pay careful attention to perfusion abnormalities within subcortical regions. Such efforts may reveal at least some of the underlying physiologic changes associated with the cognitive deficits following combined alcohol and drug abuse.

It is of interest to note that the regression analysis of PSA participants' cumulative drug use did not select cocaine use as a significant predictor of CVLT performance, yet a subsequent binomial analysis of cocaine use (i.e., little or no use *vs.* substantial use) did reveal significantly poorer CVLT performances in the group with substantial cocaine use. Perhaps the reliance on self-reports and the difficulty in accurately estimating one's number of occasions of drug use may have contributed to this apparent discrepancy. Furthermore, the drug use variables had such wide variances that

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logarithmic transformations were necessary, whereas the alcohol use variables did not demonstrate such wide variations. Based on the distribution of these values, it is also not surprising that years of alcoholism was selected as a significant predictor whereas the drug use variables were not. Taken together, these complementary findings suggest that the combination of alcohol and cocaine abuse contributes to the CVLT performance decrements, whereas alcoholism without concomitant cocaine abuse may not impair performance to the same degree. These findings are in accord with recent investigations of the cognitive sequelae associated with cocaine abuse (Manschreck et al., 1990; Mittenberg & Motta, 1993).

It is well documented that the cognitive deficits associated with alcoholism show recovery with abstinence (Grant, 1987). Recent findings have also documented increases in white matter volume in abstinent alcoholics (Drake et al., 1994; Shear et al., 1994). It is unclear, however, whether the deficits observed in the current group of PSA participants would demonstrate a similar pattern of improvement following a period of abstinence. Grant et al. (1978) observed that polydrug users who achieved abstinence or at least greatly reduced drug intake tended to demonstrate improvements in general neuropsychological functioning over a period of 3 months. Stuss and Cummings (1990), however, suggest that if the pattern of subcortical dysfunction is associated with small vessel infarctions, one might expect little or no improvement following a period of abstinence. Future longitudinal studies, therefore, should focus on patients with combined alcohol and drug abuse to determine if improvements in the cognitive deficits are observed and if the pattern of recovery of function is comparable to that of alcoholic groups. In addition, information on the structural and functional integrity of both cortical and subcortical structures should be combined with neuropsychological findings of patients with alcohol and drug abuse to determine more precisely the cognitive sequelae of combined alcohol and drug abuse.

Finally, caution is needed when interpreting these findings given some of the preexisting differences observed on potentially confounding variables such as age, verbal intelligence, and depression ratings between the two groups of participants. In addition, the present study relied on the normative reference group of the CVLT (Delis et al., 1987), which may not have been completely comparable to the alcoholic and PSA groups on demographic or other background characteristics, for the conversion of raw scores to age- and gender-corrected standard scores. However, logistic regression analyses, controlling for the effects of factors such as education, verbal intelligence and depression ratings, confirmed that they were not contributing to the CVLT differences between alcoholic and PSA groups. Furthermore, a study by Otto et al. (1994) demonstrated that severity of depression on the Hamilton Rating Scale in 156 outpatients with major depression was not associated with performance on the CVLT (all p values $> .29$). Nonetheless, samples for which demographic, affective, and other characteristics are better matched between alcoholic and PSA participants will improve on the preliminary findings noted in the present study.

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