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Effects of Chronic Cocaine Abuse on Verbal Learning and Memory

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EFFECTS OF CHRONIC COCAINE ABUSE ON VERBAL LEARNING AND MEMORY

by

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ABSTRACT

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The purpose of this study was to determine the existence and nature of verbal memory deficits in a group of chronic cocaine abusers. Functional impairment in a control group of recovering alcoholics was also examined. Participants were selected from groups of volunteers enrolled in various drug treatment programs at the Veterans' Hospital in Hampton, Virginia. All participants were at least 30 days abstinent at the time of testing. Twelve cocaine abusers with a mean use time of 10 years served as the experimental group. Ten alcoholics with a mean use time of 25 years served as the control group. Participants were given a battery of neuropsychological tests to assess attentional abilities, immediate and delayed free recall for word lists and stories, recognition ability, ability to profit from memory cues, and learning strategies. Both groups of substance abusers showed normal performance on tests of attentional abilities. Cocaine abusers showed decreased learning efficiency of word lists and had difficulty with immediate and delayed free recall of word lists and stories. In addition, cocaine abusers did not use the expected amount of semantic (meaning-based) processing in their learning of word lists. Both groups displayed normal performance on recognition testing and profited from memory cues. Overall, cocaine abusers showed verbal learning impairment that can be attributed to reduced learning efficiency and poor learning

strategies. Contrary to previous research, attentional impairments were not found in this study, possibly due to the relatively long abstinence times of participants in this study. It is possible that the types of impairments shown by cocaine abusers could be mitigated through training in the use of more efficient learning strategies, such as semantic processing and the use of memory aids. Future studies in this area might address the implementation and effectiveness of this type of training for cocaine abusers.

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INTRODUCTION

Cocaine has become one of the most sought-after and widely abused psychoactive drugs in North America. Recent studies reveal that as many as five million Americans are regular users of cocaine and 33 million report having experimented with the drug (McKim 1991). Cocaine has very powerful reinforcing effects that often lead to dependence after short periods of time. Individuals suffering from cocaine dependence often become involved in illegal activities, are unable to maintain social and occupational functioning, and experience many physical and psychiatric symptoms. Much research has been conducted concerning the physical and psychological consequences of chronic cocaine use, which include cardiac arrest, seizures, malnutrition, depression, and paranoid ideation (American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders 4th ed. 1994). However, less attention has been paid to cocaine's long-term effects on neuropsychological variables such as concentration abilities, visuomotor skills, and verbal learning and memory.

Mechanisms of Action of Cocaine

Cocaine is a psychomotor stimulant that facilitates the activity of monoamine neurotransmitters in the peripheral and central nervous systems (Sanchez-Ramos 1993). Peripherally, cocaine increases levels of epinephrine, which activates the sympathetic nervous system and leads to increased heart rate, blood pressure, and dilation of blood vessels. Overdose or chronic use of this drug causes an exaggeration of these effects

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The model journal article used for this thesis was: Cocaine-Induced Increases in EEG Alpha and Beta Activity: Evidence for Reduced Cortical Processing by R. Herning, B. Glover, B. Koeppl, R. Phillips, E. London (1994): Neuropsychopharmacology 11:1-9.

which can lead to hypertension, cardiac arrest, and brain hemorrhages. Disruption of cerebral blood flow consequent to hemorrhages associated with hypertension result in inadequate oxygen and nutrient delivery to brain tissues and can cause widespread tissue infarction, especially in subcortical structures (Lezak 1995). This type of damage may cause deficits in motor skills and memory.

In the central nervous system, cocaine blocks the reuptake of serotonin and the catecholamines- norepinephrine and dopamine. These neurotransmitters are prevented from being reabsorbed into the synaptic cell that released them and thus remain in the synapse longer than normal. This results in hyperstimulation of the post-synaptic cell. The pleasurable reinforcing effects of cocaine are believed to be caused by hyperstimulation of cells in the mesolimbic ("pleasure center") area of the forebrain by excess dopamine. After continued cocaine use, monoamines that remain in the synapses and are not properly reabsorbed will be metabolized as the body's homeostatic mechanisms attempt to normalize neural stimulation. The brain's supply of essential monoamines becomes depleted and many functions that depend on these neurotransmitters, such as mood and appetite regulation and memory functions can become impaired. Indeed, cocaine abusers often present with severe weight loss and depression. While these symptoms are fairly easy to detect, the more subtle cognitive and behavioral manifestations of cocaine-induced structural and chemical changes in the brain require careful investigation.

Neuropsychological Findings

Acute doses of cocaine are associated with subjective improvements in mood and

enhanced performance on tests of attention and reaction time (McKim 1991; Stillman et al. 1993). Performance effects are attributed to cocaine's stimulant properties which can counteract the effects of fatigue in repetitive tasks. However, chronic use of the drug can result in impairments in many cognitive and motor activities. Slowed reaction times in signal detection, visual tracking, and Sternberg memory tasks have been noted, indicating difficulties with attention, psychomotor coordination, and short-term memory (Bauer 1994; Herning et al. 1990). Ardila et al. (1991) tested chronic crack (freebase cocaine) abusers using a battery of standard neuropsychological tests and concluded that short-term verbal memory and attentional abilities were most sensitive to the detrimental effects of cocaine use. Abstracting abilities, naming, and short-term non-verbal memory were not significantly impaired. Other investigators have examined the relationship between patterns of cocaine use and degree of neuropsychological impairment. O'Malley et al. (1992) discovered a positive correlation between cocaine abusers' abnormal scores on the Story Memory Test of the Wechsler Memory Scale (WMS) and the number of grams of cocaine they had reported using in their lifetime. Deficient performance on arithmetic tests measuring concentration and mental tracking abilities were negatively related to days since last cocaine use. Ardilla et al. (1991) also found a negative relationship between lifetime cocaine intake and scores on the Digits subtest of the WMS and a calculated Memory Quotient reflecting overall memory abilities.

Another important subject of investigation is the degree to which neuropsychological deficits persist after the cessation of cocaine use. Berry et al. (1993) administered a battery of neuropsychological tests to cocaine dependent inpatients within

72 hours of last cocaine use and again two weeks later. The cocaine abusers showed deficiencies on visuospatial construction, verbal memory, and concentration at the first testing. These deficits persisted at the two week retesting. In addition, although both cocaine users and normal controls showed improvement on most measures at re-testing, the cocaine users exhibited less improvement than the controls on measures of verbal memory, psychomotor speed, concentration, and divided attention. The authors concluded that cognitive deficits can persist at least two weeks after the cessation of cocaine use and that any recovery from these deficits will be slowed, perhaps due to neurological damage resulting from long-term cocaine use.

Investigations into the neurological consequences of cocaine use have measured brain structure and metabolism in chronic cocaine users. MacKay et al. (1993) used magnetic resonance imaging techniques to examine brain phospholipid concentration in cocaine dependent patients. These patients had decreased amounts of lipid matter in the basal ganglia and thalamus, indicating a degeneration of lipid containing myelin and axonal membranes in these areas. This type of damage is thought to be a consequence of blood and oxygen loss resulting from cocaine-induced spasms of cerebral blood vessels. Although the experimenters could not determine whether these pathological changes were permanent and due solely to cocaine use, some intriguing evidence is provided that long-term cocaine use may damage brain structures related to memory functions. Strickland et al. (1993) explored possible relations between regions of abnormal cerebral blood flow and neuropsychological impairment in cocaine abusers. Measures of brain activity showed reduced cerebral blood flow in all patients which did not follow any

consistent pattern but ranged from moderate to severe and affected the frontal, temporal, and parietal lobes differently in each patient. Similarly, a global pattern of deficits was seen on the standard neuropsychological tests, with measures of attention, concentration, and verbal learning most consistently impaired. Although no specific conclusions relating physical to cognitive compromise could be drawn from this study, it provides compelling evidence for widespread physical and cognitive abnormalities consequent to chronic cocaine use. It is also interesting to note that the subjects in this study had been abstinent for at least six months, suggesting that these deficits persist after acute withdrawal and even through the early stages of recovery. However, studies such as these must be interpreted with caution due to the possible confounding effects of other drugs the patients may have used and the physical consequences of a drug abuse lifestyle, such as malnutrition and lack of sleep, which may impair cognitive functioning.

Gorelick (1992) addressed the issue of separating the effects of cocaine from confounding factors that can influence cognitive performance. He proposed that including comparison groups of non-cocaine using drug abusers would aid researchers in distinguishing the specific effects of cocaine from the more generalized consequences of chronic drug use. Findings of cognitive impairments which are more pronounced in the cocaine using group could then be more confidently attributed to the effects of this particular drug. When using this method, a comparison group should be selected whose members are users of substances that have physiological effects that are distinct from those of cocaine. Alcohol is one such substance.

Physical and Cognitive Effects of Alcoholism

Alcohol is a CNS depressant that dissolves in lipid membranes and inhibits the activity of nervous tissue. It is believed to exert most of its effects on receptors of the inhibitory neurotransmitter GABA. The reinforcing properties of alcohol are primarily attributed to the actions of its metabolite, acetaldehyde. Acetaldehyde causes the release of catecholamines such as norepinephrine that can result in pleasurable sensations and mood elevation (Maisto et al. 1995). Chronic alcohol consumption can lead to malnutrition, cirrhosis of the liver, and various types of neurological damage. The most common neurological syndrome associated with alcoholism is Korsakoff's syndrome, which is characterized by severe thiamine deficiency and associated brain "shrinkage", myelin degeneration, and limbic and thalamic lesions. Cognitively, Korsakoff's patients show loss of memory for past events, severe deficits in the learning of new material, and disorientation and confusion. Alcoholism without associated Korsakoff's syndrome has also been linked to reduced brain weight, diffuse demyelination, thinning of the corpus callosum, and shrinkage of the cerebral cortex and cerebellum. Animal models of alcoholism have shown damage to the hippocampus, although there are not yet many studies of this effect on humans (Harper and Kril 1993). Many investigators have found evidence of neuropsychological impairment in alcoholics. Glenn and Parsons (1992) tested adult male and female alcoholics on problem-solving, perceptual-motor skills, abstracting abilities, and memory for verbal and visual stimuli. Their participants showed decreased response accuracy on all four measures. Kane (cited in Delis et al. 1987) administered the California Verbal Learning Test to a group of male alcoholics

who had been abstinent for 20 to 120 days. Deficits were shown in immediate and delayed free recall measures, and subjects did not benefit from retrieval cues. The majority of research concerning verbal memory deficits in alcoholics has yielded findings that alcoholics have impaired encoding processes and are unable to utilize semantic strategies for encoding verbal information. They are thus unable to profit from retrieval cues that are semantically related to target words and display a high number of false positives and misses on recognition tests. Free recall is also impaired (Cermak 1977; Parker 1984; O'brien and Chafetz 1991). DeFranco et al. (1985) related duration of problem drinking to the presence of cognitive deficits. Participants who had abused alcohol for five or more years scored below normal ranges on measures of attention, psychomotor speed, and verbal and visual memory. Carlen and McAndrews (1991) defined the consumption rate of 30 or more drinks per week as the criteria at which 50 to 70% of problem drinkers will display memory deficits on standard neuropsychological tests.

Theory and Measurement of Memory

Although there are many theories concerning the exact nature of human memory, there is general agreement on the existence of two distinct memory storage systems: short-term memory and long-term memory (Ashcraft 1994). Short-term storage is a limited capacity system that is thought to be part of a "working" memory system that is responsible for holding new information for transfer to long-term storage through the use of rehearsal. This system is thought to be highly attention-dependent. Impaired functioning in this system is manifested by reduced learning efficiency and loss of newly

acquired information after a few minutes. Learning, the process of acquiring information in long-term storage, is believed to involve three basic processes: encoding, storage, and retrieval (Reeves and Wedding 1994). Encoding involves processing and internally representing stimuli in a form for later storage. Encoding deficits are often characterized by failure to process stimuli at a semantic level and failure to profit from recall and recognition cues. The actual storage process involves consolidation- the transfer of information from short-term memory to long-term memory after encoding has occurred. Storage difficulties are characterized by intact short-term recall with abnormally high rates of forgetting on delayed-recall testing. Retrieval is the recall of information that is stored in long-term memory. Individuals with retrieval difficulties demonstrate poor free recall but normal performance with the aid of cues. Assessment techniques that address these issues are essential for the study of distinct memory deficits in substance abusers.

Neuropsychological assessment is an invaluable tool for detecting symptoms of brain damage such as attentional and memory impairments that may go undetected during a standard neurological exam (Kolb and Whishaw 1990). Verbal memory tests utilize stimulus lists of categorically related words or short stories to measure short and long-term recall. These measures are positively correlated with verbal intelligence. A thorough assessment of memory must include three distinct procedures (Lezak 1995): 1) A delay trial must supplement the immediate recall trial to determine if learning has occurred and if material has been stored in more than temporary short-term memory. 2) During the delay period, interference stimuli should be presented to clarify that any retention of the original material was due to learning and not just continuous rehearsal in

short-term memory. 3) Cued recall and recognition testing should be performed if the subjects' memory for stimuli is below normal limits on free recall testing. Cued recall techniques involve the presentation of stimuli that are related to the target in some aspect such as appearance or meaning. Recognition tests present the target stimulus itself among a group of distractors. Free recall is the spontaneous retrieval of information without the aid of cues. These testing methods can be used to determine if the problem is one of information storage or retrieval. Normal performance with the aid of cues indicates a retrieval rather than a storage problem.

Overall, neuropsychological studies of chronic cocaine users have shown a relatively consistent pattern of deficits in attention and concentration abilities, and new learning and memory involving verbal stimuli. It has been suggested by Miller (1985) that these deficits reflect global rather than localized brain damage as a consequence of drug use. This type of damage will become most evident when abusers are given tests that require the integration of several skills, such as the attention, concentration, and organization of material that is required in a verbal learning processes. Mittenberg and Motta (1993) have suggested that cocaine abusers' failure to recall new verbal material is due to a storage deficit and not an attentional impairment. Their participants showed impaired learning of new words on the California Verbal Learning Test, and retrieval cues did not significantly improve their subjects' memory scores. However, their subjects did display a normal ability to benefit from retrieval cues based on the limited number of words they remembered on delayed free recall. Their subjects also did not have an accelerated rate of forgetting. Taken together, these results appear to suggest an

attentional impairment that interfered with new learning and short-term memory processes, rather than a storage deficit. Indeed, several investigators (Ardila et al. 1991; Berry et al. 1993; O'Malley and Gawin 1990; O'Malley et al. 1992) have found evidence of attentional impairment in cocaine abusers. Further studies of verbal memory impairment in this population should include tests aimed at identifying attentional difficulties which could contribute to learning problems. Story memory tests would also be useful in testing attentional capacity and would provide a measure of the ability of short-term memory processes to handle large amounts of information. In addition, the learning strategies of cocaine abusers (use of semantic clustering, serial-order recall, etc) should be evaluated to determine if inefficient learning strategies are also contributing to memory impairment.

It appears that this issue deserves further exploration, as the specific nature of the memory deficit could provide clues concerning the physiological nature of cocaine-induced neurological damage and the prognosis for recovery. The short-term stages of memory processing such as attention to stimuli, sensory registration, and rehearsal of information are primarily dependent on neurotransmitter mediated synaptic activity (Lezak 1995). These processes will most likely be disturbed by monoamine depletion which could reverse itself with continued abstinence. Long-term memory processes such as consolidation and storage of information and retrieval abilities are the result of changes in brain cell structure and chemistry. These processes would probably be sensitive to cerebral tissue infarction secondary to chronic cocaine use and would be less likely to show recovery of function.

Research Purpose and Hypotheses

The purpose of the present study was to further assess the nature of verbal memory and learning impairment in cocaine abusers. The following hypotheses were proposed for this study:

Hypothesis 1: Both cocaine abusers and alcoholics will score in the impaired range on measures of attention.

Hypothesis 2: Both cocaine abusers and alcoholics will score in the impaired range on measures of free recall .

Hypothesis 3: Cocaine abusers will show normal ability to benefit from retrieval cues, as measured by recognition test performance. Alcoholics will not benefit from retrieval cues.

Hypothesis 4: Cocaine abusers will display a normal amount of semantic information processing in their recall of verbal information. Alcoholics will show lower than normal levels of semantic processing.

METHOD

Participants

The experimental group for this study consisted of 12 chronic cocaine abusers participating in the Substance Abuse Treatment Program (SATP) or Rehabitat Program for Homeless Veterans at the Veterans' Affairs Medical Center in Hampton, Virginia. They met criteria for cocaine dependence according to the Diagnostic and Statistical Manual of Mental Disorders and were 30-180 days abstinent at the time of testing. Abstinence was verified by periodic urine drug screens which are required for continued participation in the program. Individuals were excluded who met any of the following criteria: 1) history of diagnosed dependence on substances other than cocaine; 2) bipolar disorder, major depression, schizophrenia or Attention-Deficit/ Hyperactivity Disorder; 3) neurological disorders or learning disability; 4) current use of narcotics and barbiturates; 5) history of consuming 30 or more alcoholic beverages per week for five or more years; 6) IQ score below 89 (average) on the Shipley Institute of Living Scale. Two participants reported a history of head injury with loss of consciousness. One individual was injured in a high school football game, the other in a fight. Neither participant reported persistent memory deficits or seizures following these incidents, or received neuropsychological testing. The experimental group was composed of 11 men and 1 woman, ranging from 29 to 52 years of age, who had completed 12 to 16 years of education. Participants had a mean cocaine use time of 10 years and all reported past use of marijuana and/or heroin. The comparison group consisted of 10 alcoholic SATP or Rehabitat patients who meet DSM IV criteria for dependence on alcohol and were

30-150 days abstinent at the time of testing. Individuals were excluded if they reported: 1) more than two instances of cocaine use in the previous five years; 2) bipolar disorder, major depression, schizophrenia or ADHD 3) history of neurological disorders or learning disability; 4) current use of narcotics or barbituates; 5) IQ score below 89 (average) on the Shipley Institute of Living Scale. Four individuals in this group reported a history of head injury with loss of consciousness. Two individuals were in car accidents, one was involved in a fight, and one sustained a fall in childhood. None of the participants reported seizures or severe memory loss following these incidents, and none received neuropsychological testing. The comparison group was composed of 8 men and 2 women, ranging from 27 to 56 years of age, who had completed 12 to 16 years of education. These participants had a mean alcohol use time of 25 years and two individuals reported past use of marijuana and amphetamines. No participants in either group received a score greater than 17 (moderate depression) on the Beck Depression Inventory they were given prior to beginning the SATP or Rehabitat program. All participants provided informed consent and were treated in accordance with the "Ethical Principles of Psychologists and Code of Conduct" (American Psychological Association, 1992). A copy of the consent form is provided in Appendix A.

Materials

Demographic questionnaires eliciting information about gender, age, education, history of substance use, psychiatric disorders, medications, and memory problems were given. A sample questionnaire is provided in Appendix B.

Symbol Digit Modalities Test. The Symbol Digit Modalities Test (SDMT; Smith, 1982) was used to assess attention and concentration abilities and was administered in written form. This test contains 110 small blank squares, each paired with a specific symbol. The individual's task is to match each symbol with its correct number from the key at the top of the page. The test is scored by counting the number of correct matches the individual has made in 90 seconds. These raw scores are then converted to z scores. The test-retest reliability coefficient for the oral form has been reported at .76 (Lezak, 1995) and the SDMT has been shown to be sensitive to generalized brain defects.

California Verbal Learning Test . The California Verbal Learning Test (CVLT; Delis, Kramer, Kaplan & Ober, 1987) was used to assess free recall (List A total trials), recognition ability (discriminability index), and ability to profit from memory cues (long-delay cued recall). A semantic cluster ratio was also calculated to evaluate learning strategy by measuring category clustering of words in free recall. This first part of this test contains sixteen "shopping list" items (List A) that belong to either of 4 categories: tools, clothing, herbs and spices, or fruits. For the first 5 trials, the examiner reads the list items and the individual repeats back as many items as he or she can remember. The total number of words recalled over the trials is recorded as List A total trials. The examinee is then orally presented with a second shopping list (List B) of sixteen words that are either fruits, appliances, fish, or spices. The individual again repeats back as many items as he or she can remember. The individual is then asked to recall as many items as possible from List A and these responses are recorded as List A short-delay free recall. The individual is then orally given the List A category names as

cues and asked to name the items in each category (short-delay cued recall). After a 20 minute delay, the List A free and cued recall tests are repeated. Finally, the examinee is read a list of 44 words which contains List A and List B items, words that are semantically and phonetically related to List A or List B items, and non-related distracter items. The individual is asked to verbally indicate whether or not the word presented appears on List A. The total number of correct responses, false positives, and misses are used to calculate a discriminability index. (For purposes of this study, the List B recall, List A short-delay free and cued recall, and List A long-delay free recall scores were not analyzed.) The List A total raw scores were converted to t scores and all other raw scores were converted to z scores. Split-half reliability for this test has been reported at .77 (Lezak, 1995) and the CVLT has been shown to differentiate between normal subjects and those affected by neurological damage.

Wechsler Memory Scale-Revised. The Logical Memory subtest of the Wechsler Memory Scale-Revised (LM-R; Wechsler, 1987) was used to assess immediate and delayed free recall in a format where ideas are meaningfully related. This test consists of two short stories that contain individual ideas concerning names of people and places and various activities. Story A contains 24 ideas and Story B has 22 ideas. The stories are read to the examinee and he or she is asked to repeat back as many of the story ideas as possible immediately after each story is read. Thirty minutes later, the examinee is again asked to recall as many ideas from each story as possible in a delayed recall test. The examinee receives one point for each idea correctly remembered. These raw scores are then converted to percentile scores. This test has been shown to distinguish between

brain damaged patients and normals and also between those with left and right hemisphere lesions. Correlations between scores on the two stories range from .68 to .85 (Lezak, 1995) and test-retest reliability ranges from .61 to .74.

Logical Memory Paragraphs Multiple Choice Questionnaire. The Logical Memory Paragraphs Multiple Choice Questionnaire (LM-MC; L.Warner, personal communication, January 21, 1996) was given as a supplement to the LM-R to test memory for the complex ideas and names of people and places that are presented in the stories in a recognition format. It consists of 32 multiple-choice questions; 17 questions for Story A and 15 questions for Story B. A copy of the test is provided in Apppendix C. This test was administered in written form. It is an experimental measure that has been utilized with clinical patients by its developer. Raw scores from this test were compared directly to raw scores from the normative sample without further conversion. The reference group upon which its scoring system is based consists of 48 patients having a measured Wechsler Adult Intelligence Scale verbal IQ between 85 and 115 and scoring between the 16th and 84th percentile on the LM-R. Twenty-four patients were female and 24 were male, ranging in age from 18 to 76 years. Thirty-two of the patients had sustained closed head injuries, 13 had suffered strokes, one had ADHD, one had a seizure disorder and closed head injury, and one was diagnosed as normal and unimpaired.

Procedure

Prospective participants were recruited through SATP and Rehabitat group meetings. Individuals expressing a desire to participate in the study were given an appointment with the experimenter, at which time they completed a consent form and the

demographic questionnaire. Any participant reporting a condition listed in the exclusionary criteria was excused from the study. The testing session began with the administration of the CVLT. Following this the LM-R material was presented and immediate free recall testing was done. Participants then completed the SDMT . The session concluded with the 30 minute LM-R delayed recall given at the appropriate time and the LM-MC following.

All participants were tested individually and received the tests in the above order. Approximate testing time was 60 minutes. Participants were given the option of receiving feedback concerning their performance. Those wishing to receive feedback were given a scheduled appointment to review their test results with the experimenter and a licensed clinical psychologist.

For those individual participants who desired feedback concerning their performance, standard clinical interpretative procedures were followed. This involves the conversion of raw scores to either z scores, t scores, or percentile scores based on norms for the person's age group. These standard scores were then compared to a table of clinical performance ranges (L. Warner, personal communication, December 6, 1996) to determine if the individual demonstrated impairment. A copy of this table is provided in Appendix D.

Impairment is defined as a deviation of behavior from an ideal standard (Lezak, 1995). This ideal standard is the established performance level of a normative sample of individuals who have taken the test and were chosen to be representative of individuals who will take the test in clinical settings. This sample is generally chosen to be

representative of the general population in demographic characteristics such as age, sex, race, and education and does not contain individuals with severe intellectual deficits or brain damage.

RESULTS

Statistical Analysis

T tests were used to compare the means of scores in the experimental and control groups to each other. Z tests were used to compare the mean of scores in either group to the mean (50) of a set of t scores. This was done for purposes of determining clinical impairment in the group, given that the abilities that were measured are normally distributed in the population. When z tests were used, standard scores that were reported as deviations from the mean were converted to t scores. For test scores that were reported as percentiles, the data were interpreted according to the table in Appendix D and the number of impaired participants in each group was noted. A list of all test score means and standard deviations for the experimental and control groups is provided in Appendix E.

Due to the fact that multiple t tests and z tests were performed on the data, a Bonferroni inequality was used to minimize the probability of a Type I error. The overall alpha level was set at .10, with each individual alpha being .005.

Measures of Attention

The hypothesis that both alcoholics and cocaine users would score in the impaired range on measures of attention was not supported by the data. To assess attention, scores from the SDMT were subjected to a t test to compare the performance of experimental

and control groups. A significant difference was not found between the groups. A z test was performed to determine if either group showed impairment compared to norms. Neither alcoholics ($z = 1.27$, $p>.005$) nor cocaine users ($z = 1.30$, $p>.005$) showed clinical impairment.

Measures of Free Recall

The hypothesis that both groups of substance abusers would show impairment in free recall tests was not supported, but cocaine users did show evidence of impairment on some of the tests used. Free recall measures consisted of the LM-R immediate and delayed recall tests and the CVLT List A totals. On the LM-R immediate and delayed recall measures, a significant difference was not found between the cocaine users and alcoholic controls when the Bonferroni inequality was used. However, it is noteworthy that a difference existed between the group means for both tests that was significant at an alpha level of .05 before the Bonferroni correction. For the LM-R immediate recall, the mean of percentile scores for cocaine abusers was 50.75 and the mean for alcoholics was 77.70. For the LM-R delayed recall, cocaine users had a mean of 41.00 and alcoholics had a mean of 69.70. Overall, on the LM-R immediate recall, no subjects scored in the clinically impaired range (at or below the 10th percentile), but 1 alcoholic and 4 cocaine abusers scored below average (at or below the 11th percentile). On the LM-R delayed recall test, 2 cocaine abusers scored in the clinically impaired range and 2 cocaine abusers scored below average. The CVLT List A total trials was subjected to a t-test for experimental and control group comparison. No difference was found between the groups on this measure. A z test was then used to compare each group to norms.

Alcoholics as a group did not show clinical impairment ($\underline{z} = 2.34$, $p>.005$), but cocaine users did ($\underline{z} = 4.73$, $p< .005$).

Measures of Performance With Retrieval Cues

The hypothesis that cocaine users would benefit from retrieval cues was supported by the data from the CVLT. The hypothesis that alcoholics would not benefit from retrieval cues was not supported. To assess performance with the aid of retrieval cues, the CVLT discriminability index and long-delay cued recall measures, and the LM-MC were analyzed. No difference was found between the groups on the CVLT measures using a $\underline{t}$ test. Both groups showed adequate performance on the discriminability index, ($\underline{z} = .95$, $p>.005$ for alcoholics and $\underline{z} = .58$, $p>.005$ for cocaine users). Neither group showed a deficiency on long-delay cued recall ($\underline{z} = 3.16$, $p>.005$ for alcoholics, and $\underline{z} = 0.29$, $p>.005$ for cocaine abusers). However, it can also be noted here that the long-delay cued recall results for alcoholics were significantly lower than the norms at an alpha of .05 before the Bonferroni correction. Impaired performance on the experimental LM-MC was shown by 2 alcoholics and 5 cocaine abusers. For this test, impairment was defined as fewer than 23 of the 32 multiple-choice questions answered correctly. This criterion was based on the lowest number of questions answered correctly by persons in the clinical reference group scoring in the unimpaired range on the LM-R.

Measures of Semantic Processing Ability

The hypotheses that cocaine abusers would show normal levels of semantic processing and alcoholics would show decreased semantic processing were not supported by the present findings. Semantic processing ability was assessed using scores on the

CVLT semantic clustering ratio. A difference was not found between the two groups using a t test. However, cocaine abusers were deficient on this measure ($z = 3.46$, $p<.005$), while alcoholics were not ($z = 1.27$, $p>.005$).

DISCUSSION

This study investigated attentional and verbal memory impairment in recovering cocaine abusers. Functional deficits in a control group of recovering alcoholics were also examined. Impaired performance was not found on tests of attentional abilities for cocaine abusers or alcoholics. Cocaine abusers showed reduced learning efficiency and had difficulty with free recall of words and stories. Both groups performed normally on recognition testing, and benefited from retrieval cues on delayed recall. Cocaine abusers did not demonstrate expected levels of semantic information processing in their learning of verbal material. The following sections provide a more detailed description of the test results as they relate to the hypotheses of this study.

It should first be noted that when interpreting the findings from this study, several limiting factors must be considered: 1) The sample size was relatively small and a significant amount of variability was noted in test performance within the groups. 2) Several participants in each group reported a history of head trauma which can negatively affect performance on neuropsychological tests. 3) There were no means of determining the level of premorbid functioning for any of the participants before they began using cocaine or alcohol. Therefore, the exact degree of impairment present that was due to substance abuse cannot be ascertained. 4) Although exclusionary criteria were followed, most participants also reported a history of polysubstance abuse that can confound efforts to isolate the detrimental effects of cocaine or alcohol. 5) The participants were mainly male high school or college graduates in their late 30s to early 50s with IQs in the average range. This sample is not representative of the entire

population of substance abusers. However, many factors such as polysubstance abuse and head injury are frequently found in the population of substance abusers seeking treatment (Gorelick 1992). In addition, individuals participating in recovery groups often have higher intelligence levels and socio-economic status than the general population of substance abusers. While these variables may confound the specific efforts of this study, they provide external validity with regard to the nature and degree of impairments that will be found in a clinical setting

The results of the SDMT test used to measure attentional abilities did not support the hypothesis that both groups of substance abusers would show attentional impairment. There was no difference between the groups on this measure and neither group showed clinical impairment. Previous studies with these groups have found attentional impairments in both cocaine users and alcoholics (DeFranco et al. 1985; Ardila et al. 1991). The relatively long abstinence times for both groups (from 30 to 180 days) in this study compared to previous studies could be a factor accounting for the difference. In accordance with Lezak's (1995) model of memory functions, attention to stimuli is regulated primarily by neurotransmitter-mediated activity that can be disturbed by acute drug use but may return to normal when drug use is discontinued. These findings provide positive support for the prospect of recovery of function with continued abstinence.

With regard to free recall abilities, cocaine abusers showed a decreased number of words learned on List A totals. In addition, cocaine abusers performed worse than alcoholics on the LM-R story memory immediate and delayed recall measures, with a

few subjects in the cocaine group showing clinical impairment on the delayed recall portion. These findings provide some support for the hypothesis that both groups would show impaired free recall, although it was not predicted that cocaine abusers would perform worse than alcoholics on any measure. Some sources of difficulty in learning can include poor encoding and failure to process information for meaning (Reeves and Wedding 1994). Poor delayed recall can be caused by storage or retrieval problems. These concepts will be explored further in the following sections.

Both groups of substance abusers scored within normal limits on the CVLT recognition tests of discriminability and long-delay category cued recall. On the experimental multiple-choice LM-MC for LM-R story ideas, although exact statistical analysis could not be performed, overall more cocaine abusers (5) showed impairment than did alcoholics (2). The hypothesis that cocaine abusers would profit from retrieval cues is supported by the CVLT findings, but not the LM-MC findings. However, based on the fact that cocaine abusers learned fewer LM-R story ideas initially than alcoholics, they still could have benefited from retrieval cues for the few ideas they did learn, as was the case for the CVLT. The hypothesis that alcoholics would not benefit from retrieval cues was not supported by the data. This group showed normal performance with the aid of visual and semantic memory cues. For cocaine abusers, the data point to the conclusion that they are more likely to suffer from retrieval rather than storage difficulties, but will benefit from the use of visual or semantic retrieval aids. This is indicated by the fact that normal recognition performance was seen on the CVLT in the presence of impaired free recall. These findings on neuropsychological tests indicate that

the material was actually stored in long-term memory, but the individual requires prompting with memory cues to retrieve it (Lezak 1995).

Alcoholics showed normal levels of semantic information processing (category clustering of words in free recall) on the CVLT, but cocaine abusers did not. These results are contradictory to the hypothesis of this study that alcoholics would have difficulty with semantic processing, but not cocaine abusers. They also appear to contradict cocaine abusers' normal performance on the CVLT long-delay cued recall, which uses semantic category cues. A possible explanation for the cocaine abusers' results is that their poor learning strategies or low motivational levels prevent them from using semantic learning strategies during initial learning, but they will recognize and respond to semantic memory aids when prompted. The results found for alcoholics are inconsistent with previous research indicating that alcoholics have difficulty with semantic information processing and do not generally encode information in this manner on verbal learning tasks (Cermak 1977; Parker 1984).

Conclusion

The results of this study show definite patterns of verbal learning impairment in cocaine abusers. The most consistent difficulties displayed by this group were reduced learning efficiency and poor learning strategies. Cocaine abusers' performance on the tests given indicate that they do have the ability to retain the reduced amount of information they do learn and are also able to profit from retrieval cues. Their impaired learning efficiency could thus be improved with education on the proper use of tools to aid in memory, such as an emphasis on processing new information for meaning, rather

than rote memorization. The degree and type of cognitive impairments found in substance abusers is an important issue affecting substance abuse education, treatment, and prospects for recovery, and cannot be ignored (Meek et al. 1989). Many otherwise intelligent and motivated individuals with specific cognitive impairments can become frustrated with their inability to learn and concentrate in a recovery program. This may be lead to unnecessary relapse. Identification of individuals with these difficulties and further studies which implement learning aids for these individuals are essential in a clinical recovery setting.

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APPENDIX A

CONSENT FORM

Department of Veterans Affairs

VA RESEARCH CONSENT FORM

Subject Name: _____ **Date** _____

Title of Study: Effects of Chronic Cocaine Abuse on Verbal Memory and Learning

Principal Investigator: JAMES ROBINSON, Psy.D. **VAMC:** HAMPTON

DESCRIPTION OF RESEARCH BY INVESTIGATOR

1. **Purpose of study and how long it will last:**
2. **Description of the study including procedures to be used:**
3. **Description of any procedures that may result in discomfort or inconvenience:**
4. **Expected risks of study:**
5. **Expected benefits of study:**
6. **Other treatment available:**
7. **Use of research results:**
8. **Special circumstances:**

I understand that I am being asked to give my voluntary consent to participate in a research study which will require me to complete three tests measuring attention span and memory for word lists and short stories. The purpose of this study is to test verbal learning and memory skills in recovering substance abusers. The testing will take about 60 minutes of my time. In addition to completing the tests, I will be asked to fill out a brief questionnaire.

This study will involve approximately 60 persons who have been diagnosed with dependency on either cocaine or alcohol. Individuals who have a history of diagnosed dependency on other substances or who have any of the psychiatric or medical conditions listed on the questionnaire are excluded from the study.

SUBJECT'S IDENTIFICATION (I.D. plate or give name-last, first, middle)

Department of Veterans Affairs

VA RESEARCH CONSENT FORM
(Continuation Page 2 of 2)

Subject Name: _____ Date _____

Title of Study: Effects of Chronic Cocaine Abuse on Verbal Learning and Memory

Principal Investigator: JAMES ROBINSON, PsyD VAMC: HAMPTON

The results of my tests will be evaluated by the test administrator and scored according to standardized procedures. I understand that any information obtained about me from the research, including answers to the questionnaire, will be kept strictly confidential. I also understand that data derived from this study could be used in reports, presentations, and publications, but that I will not be individually identified. I understand that I have the right to refuse to participate or withdraw from this project at any time without penalty or loss of benefits to which I am otherwise entitled. If I desire feedback concerning my performance on the tests I will take I may leave my name and a number where I may be reached. I will be contacted later in the week and informed of the time and place when individual feedback sessions will be given.

The benefit of my participation in this study is that it may be helpful in identifying learning and memory problems among recovering substance abusers. This information could be used to modify drug treatment and education programs to address these issues. I realize that I will not be compensated for my participation. There are no risks associated with involvement in this project. If I have any further questions concerning the study, I am free to contact Dr. James Robinson at 722-9961 ex. 2140 to discuss them.

I certify that I have read the preceding or it has been read to me, that I understand its contents, and that any questions I have pertaining to the research or my rights as a research subject have been answered by Dr. James Robinson. A copy of this consent form will be given to me. My signature below means that I have freely agreed to participate in this project.

Department of Veterans Affairs

VA RESEARCH CONSENT FORM

(Continuation Page 2 of 3)

Subject Name: _____ **Date** _____

Title of Study: _____

Principal Investigator: JAMES ROBINSON, Psyd **VAMC:** HAMPTON

RESEARCH SUBJECTS' RIGHTS: I have read or have had read to me all of the above. Dr. Robinson has explained the study to me and answered all of my questions. I have been told of the risks or discomforts and possible benefits of the study. I have been told of other choices of treatment available to me.

I understand that I do not have to take part in this study, and my refusal to participate will involve no penalty or loss of rights to which I am entitled. I may withdraw from this study at any time without penalty or loss of VA or other benefits to which I am entitled.

The results of this study may be published, but my records will not be revealed unless required by law.

In case there are medical problems or questions, I have been told I can call Dr. Robinson at 722-9961 during the day and Dr. _____ at _____ after hours. If any medical problems occur in connection with this study the VA will provide emergency care.

I understand my rights as a research subject, and I voluntarily consent to participate in this study. I understand what the study is about and how and why it is being done. I will receive a signed copy of this consent form.

_____ _____

Subject's Signature Date

_____ _____

Signature of Subject's Representative* Subject's Representatives

_____ LISA KAMINSKI

Signature of Witness Witness (print)

_____

Signature of Investigator

*Only required if subject not competent.

APPENDIX B

DEMOGRAPHIC QUESTIONNAIRE

Demographic Questionnaire pg. 1

Please take a few minutes to answer the following questions about yourself. Fill in the blanks or check the appropriate response.

Age-_____ Gender: Male___ Female____

Number of Years of Education: _____

Substance Use History: Drug of Choice_____

Approximate amount used per week_____

Length of time you have used_____

When did you last use?_____

(for cocaine users only):

Number of alcoholic beverages consumed weekly_____

Has drinking been a problem for you?____ If so, for how long?_____

(for alcohol users only):

Have you ever used cocaine?____ If so, for how long?_____

Amount used weekly_____ Date of last use_____

Other Substances:	Marijuana	Heroin	Amphetamine (Speed)
Amount used weekly	_____	_____	_____
Length of use	_____	_____	_____
Date of last use	_____	_____	_____

Present State:

Do you... Have memory problems_____ Forget meetings or appointments_____

Have trouble concentrating_____ Feel easily distracted_____

Are you taking any medication?(describe)_____

_____

Have you ever had: Head Injuries____ Seizures_____ Stroke_____

Have you ever been knocked unconscious in a fall or fight? _____

Have you been diagnosed with: Severe Depression_____ Schizophrenia_____

Bipolar Disorder_____ ADHD_____

APPENDIX C

LOGICAL MEMORY PARAGRAPHS

MULTIPLE CHOICE QUESTIONNAIRE

WECHSLER MEMORY SCALE-REVISED
Multiple Choice Questions

Story A:

1. What was the main character's first name?

   Angie Anna
   Allison Annabelle

2. What was her last name?

   Thompson Towers
   Smith Jones

3. Where was her home?

   South Carolina South Detroit
   South Boston South Dakota

4. What did the main character do? She was...

   employed unemployed
   studying on disability

5. What was she employed as?

   cashier therapist
   waitress cook

6. Where did she work?

   church office
   school hospital

7. Where did she work?

   classroom cafeteria
   office gym

8. Where did she report what happened?

at the newspaper at the administrator's office
at the principle's office at the City Hall Station

9. What did she report?

that she had been held up that she was sick
that she had been beaten up that she had car trouble

10. Where had she been held up?

at home on Elm Street
on State Street at the store

11. When had she been held up?

the night before four days earlier
two days earlier the morning before

12. What was stolen?

25 cents jewelry
clothes 56 dollars

13. How many children did she have?

one three
two four

14. What bill was due?

electric phone
rent water

15. For how many days had she and her family not eaten?

one three
two four

16. Who was touched by her story?

the doctor the school teacher
the lawyer the police

17. What did the police do for her?

booked her — took up a collection
gave her a ride home — wrote down the details of what happened

Story B:

1. What was the main character's first name?

Donald — Paul
Robert — David

2. What was his last name?

Smith — Miller
Jones — Watson

3. What was he doing?

driving — reading
sleeping — eating

4. What was he driving?

a dump truck — a fire truck
a pick-up truck — a ten-ton truck

5. What time of day was it?

morning — mid-day
night — afternoon

6. Where was he?

in Massachusetts — in Mississippi
in Mexico — in Minnesota

7. What was his truck carrying?

chickens — horses
eggs — gravel

8. Where was he going?

Nashville St. Louis
Memphis Atlanta

9. What happened?

his engine died he had a flat tire
he ran out of gas his axle broke

10. What happened to the truck?

it hit a bridge it skid into a ditch
it hit a tree it hit another truck

11. What happened to the main character?

he hit the windshield he was thrown out of the truck
he was thrown against the dashboard his head hit the door of the truck

12. What happened to him?

he was badly shaken he cut his forehead
he had a headache he broke his arm

13. What did he doubt?

that his truck could be fixed that he could walk home
that help would come that he could still drive the truck

14. What happened next?

his 2-way radio buzzed another truck drove up
a police car drove up his lights went out

15. What did he say on the 2-way radio?

this is Mr. Miller this is grasshopper
I need help this is elephant man

APPENDIX D

CLINICAL GUIDELINES FOR INTERPRETING TEST SCORES

Percentile Score	T score	Z score	Interpretation
98-99	> or = 70	+2	Very Superior
90-97	63-69	+1.3	Superior
75-89	57-62	+0.67	High Average
26-50-74	44-50-56	0	Average
11-25	38-43	-0.67	Low Average
3-10	31-37	-1.3	Mild-Moderate Impairment
< or = 2	< or = 30	-2	Severe-Profound Impairment

APPENDIX E

STATISTICAL SUMMARY OF TEST RESULTS

Test	Group	Mean	Standard Deviation
SDMT (z scores)	Cocaine	1.30	1.03
	Alcoholics	1.27	0.99
LM-R immediate recall (percentiles)	Cocaine	50.75	23.80
	Alcoholics	77.70	28.22
LM-R delayed recall (percentiles)	Cocaine	41.00	23.22
	Alcoholics	69.70	22.14
CVLT List A total (t scores)	Cocaine	36.33	13.32
	Alcoholics	42.60	20.29
CVLT discriminability (z scores)	Cocaine	2.83	0.58
	Alcoholics	2.70	0.95
CVLT sem. clust (z scores)	Cocaine	2.00	0.74
	Alcoholics	2.70	1.16
CVLT long delay cued (z scores)	Cocaine	2.92	0.51
	Alcoholics	2.30	1.64
LM-MC (raw scores)	Cocaine	22.58	4.77
	Alcoholics	26.20	4.78

VITA

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