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THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

EFFECTS OF ALCOHOL ON PAIRED ASSOCIATE LEARNING AND MEMORY

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PHILOSOPHY

BY

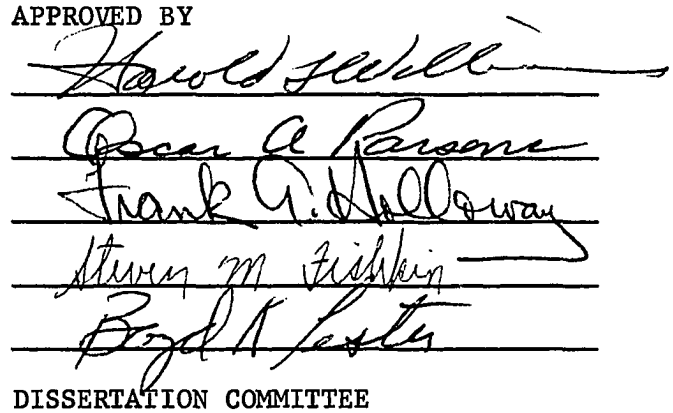
CYNTHIA LEE JANES

Oklahoma City, Oklahoma

1970

EFFECTS OF ALCOHOL ON PAIRED ASSOCIATE LEARNING AND MEMORY

APPROVED BY



DISSERTATION COMMITTEE

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EFFECTS OF ALCOHOL ON LEARNING AND RECALL OF PAIRED ASSOCIATES

CHAPTER I

INTRODUCTION

Alcohol is the most commonly used drug in America, yet we seldom consider its effects on human behavior. Anecdotally, memory loss has been associated with excessive use of alcohol, but there have been relatively few systematic studies of this effect. Three avenues of investigation have been employed to assess the relationship between alcohol consumption and cognitive functioning. The first is the study of normal subjects who are temporarily intoxicated, the second involves research on chronic alcoholics, and the third approach is clinical and experimental investigation of Korsakoff patients. For these patients, excessive use of alcohol has resulted in a unique set of symptoms, called the Korsakoff syndrome, the chief characteristic of which is memory impairment. The fact that Korsakoff patients frequently are persons who have chronically overindulged in alcohol suggests that alcohol plays a significant role in the memory problem. Therefore, the nature of memory impairment in the Korsakoff patient, as well as in the temporarily inebriated normal subject and chronic alcoholic, is of interest.

Verbal learning theories assert that forgetting is a function of one of two mechanisms, interference or decay. It will also be of

interest, therefore, to examine the implications of previous alcohol studies for these two theoretical positions.

Alcohol and Intellectual Functions

Jellinek and McFarland (1940) and Carpenter (1962) have reviewed all previously published investigations of the effects of alcohol on memory and other intellectual processes. All of these studies showed that cognitive functions are impaired by alcohol. The Jellinek and McFarland (1940) review reported that the performance of inebriated subjects was always impaired on memory tests. The results of later studies (Carpenter, 1962) which used better controls and more refined techniques, supported the earlier work (Jellinek & McFarland, 1940) showing that intellectual processes were disrupted by alcohol. Studies completed since the publication of the Carpenter (1962) review agree that alcohol impairs both learning and memory (Kalin, 1964; Storm, Caird, & Korbin, 1965; Storm & Caird, 1967; Tartar, 1968; and Goodwin, et al., 1969).

Several investigations have examined the ability of subjects to retain material that was learned under the influence of alcohol, and all of them showed that when tested while sober, subjects who had learned while sober remembered better than subjects who had learned while intoxicated.

For example, a study by Kalin (1964) showed that subjects had difficulty remembering stories they had composed while drinking. His subjects wrote stories to three TAT pictures during a cocktail party. The first story was written before they began to drink, the second after twenty-five minutes of drinking, and the third following an additional twenty-five-minute period. Consumption of alcohol was ad lib., and no

comparison of amount consumed to performance on story recall was reported. The following day, while sober, subjects were asked to rewrite the three stories. Controls, who had consumed non-alcoholic beverages at another party, recalled the third story best. Experimental subjects, however, remembered the first story, the one written prior to alcohol ingestion, better than either of the other two. Their recall of the last story was very poor. A rather surprising finding in this study was that recall by alcohol subjects of the first TAT story was superior to that of controls, a fact which suggests that alcohol may counteract the interfering effects of interpolated material, i.e., information learned between the acquisition and recall of other information. Under normal, non-alcoholic conditions, the learning of interpolated material impairs memory for the original information (i.e., retroactive interference). Kalin's results show that for alcohol subjects, learning interpolated material did not have the expected effect. This study also demonstrated that the more a subject had drunk when he wrote a story, the less he could remember when sober.

An explanation for the memory deficit of subjects learning under alcohol and tested while sober is offered by Storm, Caird, and Korbin (1965), Storm and Caird (1967), and Goodwin, et al., (1969). The Storm group required both alcoholic patients and normals to learn serial lists either while sober or inebriated. After a forty-eight-hour period, while sober, all subjects were tested for retention. Those who learned while sober relearned the serial lists significantly faster than those who had been intoxicated during the learning period. Goodwin, et al. (1969) compared normal subjects who had learned while intoxicated

and while sober for twenty-four hour recall of rote-learned sentences and word associations. They found that on these tasks, performance of subjects who had learned while inebriated was grossly inferior to that of subjects who had been sober during learning.

The results of the above studies are interpreted by their authors in terms of a dissociation hypothesis. Subjects performed better when trained and tested in the same state (drunk or sober), than when in different states. Thus, it may be that the memory deficit of subjects who learn while intoxicated and are tested while sober is attributable to their changed state, rather than to the effect of alcohol per se. The findings of Vogt (1930), for example, that lines of the Odyssey learned under the influence of alcohol were much more difficult to relearn than lines memorized while sober, may be due to the change of state between learning and relearning. In both the Storm and Caird (1967) and Goodwin, et al. (1969) studies, however, statistical analysis showed that the effect on test performance of having been intoxicated during training, as well as the effect of changed state, was significant. Thus, memory impairment in these studies may result both from dissociation and the direct effect of alcohol.

Although investigators agree that alcohol affects memory, no study includes an analysis of the stage(s) of memory affected, with the exception of that by Goodwin, et al. (1969). Most current models of memory recognize at least three stages: registration, retention, and retrieval (Melton, 1963; and Weiner, 1966). Goodwin, et al., reported that after one relearning trial (while sober) the memory deficit of those subjects who had learned while drunk disappeared. They concluded that

this transient deficit was due to impairment of retrieval functions, but they attributed this effect to the altered state rather than to specific effects of the drug.

Alcohol impairs cognitive performance and learning, as well as memory. For example, in problem solving tasks, small amounts (0.33 ml/kg) of alcohol facilitated performance, while large doses (1.0 ml/kg) reduced the efficiency of performance (Carpenter, et al., 1961). Both time to solution and number of problems solved were found to be curvilinear functions of alcohol dose. A later study (Carpenter & Ross, 1965) showed that alcohol differentially affected persons with high and low task proficiency. The impairment in performance of subjects with high pre-alcohol task proficiency scores was an increasing function of alcohol dose. Performance of subjects whose proficiency was poor prior to alcohol intake improved with small doses of alcohol, but declined with stronger concentrations.

Alcohol's disruption of rote learning has been repeatedly demonstrated (Korman, Knopf, & Austin, 1960; Storm, Caird, & Korbin, 1965; Storm & Caird, 1967; Tartar, 1968; and Goodwin, et al., 1969). Both chronic alcoholics (Storm & Caird, 1967) and normals (Storm, Caird, & Korbin, 1965) required significantly more trials to master serial lists when under the influence of alcohol, than when sober. The same effect has been found with alcoholized normal subjects for paired associate learning (Tartar, 1968), and for the rote learning of sentences (Goodwin, et al., 1969).

One study showed that under stressful conditions, alcohol may facilitate the learning of serially presented nonsense syllables (Korman,

Knopf, & Austin, 1960). However, an analysis of this investigation reveals some methodological peculiarities that might account for this apparent facilitatory effect. These investigators used a single dose of alcohol (30 ml) with no adjustment for body weight, and did not report blood alcohol levels. Furthermore, they specify that time since last food ingestion was not controlled. It is therefore impossible to estimate with any accuracy, the blood alcohol levels of these subjects. However, assuming subjects had fasted at least four hours, a typical fasting period for alcohol studies, the dose of 30 ml would raise the blood alcohol concentration only .02 to .03 percent in the average male. This dose would approximate half that consumed by the low dose group of Carpenter, et al. (1961). In the Carpenter study, low doses actually improved problem solving. Such amounts of alcohol are generally not high enough to cause intellectual impairment, but might serve to relax the subject so that he is able to perform more effectively.

Despite methodological shortcomings of a number of studies, the conclusion that alcohol generally impairs learning, retention, and cognitive performance, seems inescapable.

Korsakoff's Syndrome and Memory

In a series of papers published in the late 1880's, the Russian physician, S. S. Korsakoff, described what he termed "a special form of psychic disorder which occurs in conjunction with multiple neuritis" (Victor & Yakovlev, 1955, p. 396), which is now called the Korsakoff syndrome. Korsakoff felt that psychic disturbances accompany a number of diseases, and that they are especially evident in cases of chronic alcoholism. The following description of the Korsakoff syndrome is taken

from a translation of one of Korsakoff's papers (Victor & Yakovlev, 1955, pp. 396-399).

> The story usually begins with vomiting which may sometimes be very persistent and then extreme weakness develops. If previously the patient was able to walk, he begins to stagger, the gait becomes insecure, and finally he cannot get up at all. . . . Then paralyses of the lower extremities become noticeable, affecting particularly frequently the extensors of the thighs and movements of the toes and ankle. Quite frequently following paralysis of the legs, the paralysis occurs also in the arms in which movements of wrists and fingers are first affected. . . . These symptoms appear either in the form of an intolerable irritability and an extreme restlessness, or in the form of an extreme depression of psychic activity with a profound impairment of memory. . . . At times the disorder or memory manifests itself in an extraordinarily peculiar amnesia, in which the memory of recent events, those which just happened, is chiefly disturbed, whereas the remote past is remembered fairly well. . . . One may note that at times he utterly confuses events and that he remembers absolutely nothing of what goes on around him; he does not remember whether he has had dinner, whether he was out of bed. Depending on the conditions under which the disease develops, the onset and the course vary. Thus, in alcoholism, the disease frequently begins with symptoms resembling delirium tremens and then follow paralysis and characteristic disturbances of memory.

Since Korsakoff published his observations, numerous subsequent studies of these patients have verified his findings. Talland (1960) performed a comprehensive study of memory disorder of Korsakoff patients. Extensive interviews with twenty-four persons with chronic Korsakoff's disease revealed a significant degree of confusion in memory for past events. Generally, the patients were able to recall experiences that preceded their hospitalization more accurately than those that had taken place quite recently, but there were exceptions to this overall finding. Typically, the Korsakoff patient was unable to give the correct date, to estimate accurately the time since his hospitalization, or to recall, even within wide margins, the number of interviews he'd had in a certain room.

If the memory of the Korsakoff patient is better for older events than for more recent ones, as is usually found in clinical interviews, then this differential memory should be clearly delineated in an experimental setting. Talland (1959) predicted that for normal subjects engaged in serial-list learning, interpolation of a second list would make the first more difficult to remember. However, he expected the second list to have little effect on the Korsakoff patients' memory of the original list. Using both nonsense syllables and words, Talland required both Korsakoff patients and normal subjects to learn a single serial list. After being tested for retention of this list, subjects learned a second list. After testing for retention of the second list, the original list was relearned and retention was measured again. Talland's prediction was confirmed by the finding that learning of the second list had negligible interference effects for the patients. Thus, Korsakoff patients did not exhibit retroactive inhibition.

In Korsakoff's discussion of the memory deficit of patients with multiple neuritis no reference is made to stages of the memorial process. Meissner (1968), however, has recently reviewed a number of theories concerning the nature of the Korsakoff patient's memory deficit, in terms of the three phases. His review shows that there is little consensus about which portion of the memory process may be most susceptible to the Korsakoff syndrome. Brodmann (1902 & 1904), for example, considered the memory problem to be a failure of the registration process, and Gregor (1909) asserted that the impairment was due to defective attention. According to a third investigator, Crahay (1957), both registration and retrieval were affected, but the retrieval function was more severely impaired.

More investigators have emphasized deficits in retrieval than in the other two memorial phases. For example, Lidz (1942) found that prompting aided his patients' recall, and theorized that the memory deficit in Korsakoff's syndrome resulted from inability to voluntarily recall information (Meissner, 1968), i.e., impairment of the retrieval function. Talland (1959) examined the ability of the Korsakoff patient to recall and recognize items on serial lists. Performance of Korsakoff patients was found to be inferior to that of controls in both measures. A later investigation (Talland, 1960) which used recall tests, such as digit and running memory span, information, single words, time span, counting, figures, and memory for the experimental tasks performed, showed the Korsakoff patients to be inferior to controls. However, performance of the patients on a series of recognition tests closely approximated that of controls. This suggested that for Korsakoff patients, the memory problem was limited primarily to retrieval processes, with retention affected to a lesser degree. Meissner (1968) found that Korsakoff patients were able to retain the solution to a simple problem-solving task, but as the task was made more difficult, their memory of solution became much poorer than that of normals. Since all subjects had learned each task to the same criterion, Meissner concluded that retention and retrieval, but not registration mechanisms, were impaired in Korsakoff patients.

Some studies of Korsakoff patients have suggested a deficit in learning, as well as one in memory. In his early work Talland (1959) noted that Korsakoff patients learned serial lists more slowly and forgot them more quickly than did normal subjects. Victor, et al. (1959),

tested cognitive functions of Korsakoff patients by administering the Wechsler-Bellevue Intelligence Scale and the Wechsler Memory Scale. One of the most reliable findings was that Korsakoff patients demonstrated marked impairment in learning new material involving word association and short stories.

Theories of Forgetting

Explanations of forgetting fall into two general categories, the trace decay theory and the interference theory. The trace decay explanation, in which memory loss is a simple function of time, has been considered by some as a valid theory. The longer the time since learning, the less material retained. For example, Brown (1958) has interpreted the results of his studies as supporting a decay rather than an interference interpretation of forgetting. However, Jenkins and Dallenbach (1923) provided early evidence that the passage of time alone could not adequately account for forgetting. They found that subjects remembered significantly more information when they slept during the retention interval than when they spent the time awake. They attributed the more rapid rate of forgetting observed in the awake state to interference from new experience.

Since the Jenkins and Dallenbach experiment, evidence favoring interference theory has mounted (Underwood, 1957; Slamecka & Ceraso, 1960). A significant portion of modern verbal memory literature is devoted to studies of the influence of various kinds of interfering material. Two basic paradigms have been developed for the determination of interfering effects. One examines effects of material learned prior to the material to be remembered, while the other looks at effects of

material learned subsequent to that to be remembered. If the interference theory is correct, then forgetting can be accounted for by the interfering effects of material learned either before or after the material to be remembered. In the retroactive inhibition (RI) paradigm, two groups of subjects learn material "A". One group subsequently learns material "B", while the other group engages in a relatively non-interfering task, such as cancelling digits or adding columns of numbers. Following a retention interval, both groups are tested for "A". Underwood (1948, 1949, & 1957) noted that another kind of interference, which he termed proactive inhibition (PI), often affected recall. Subjects who learned "A" prior to learning "B" retained less of "B" than subjects who learned only "B", after comparable retention periods. Although the significance of PI has been demonstrated (e.g., Koppenaal & O'Hara, 1962; Postman & Stark, 1964; and Houston, 1967), RI has received more attention in the experimental literature.

The above studies provide evidence that inebriated normals, chronic alcoholics, and Korsakoff patients, all experience at least some degree of memory deficit. The memory impairment of all these persons may be related to their use of alcohol. There is little agreement concerning which memorial functions may be most impaired by alcohol, or whether it impairs memory by potentiating interference or decay effects. The experiment reported here addressed these issues.

Statement of the Problem

The purpose of the present investigation was to determine effects of alcohol on learning and memory. Specifically, the following questions were asked:

(1) Does alcohol impair the acquisition of verbal paired associates?

(2) If so, does it do so by retarding acquisition of the response pool, or the stimulus-response (S-R) associations, or both?

(3) Does alcohol affect paired-associate memory?

(4) If so, is the effect due to impairment of retention or retrieval or both?

(5) Under alcohol, what are the effects of material learned both before and after the material to be remembered?

(6) Can interference theory or decay theory adequately explain forgetting under alcohol?

The study examined the effect of a single dose of alcohol on three phases of the memorial process, acquisition, retention, and retrieval. Acquisition, or registration, implies the laying down of a trace, retention implies storage for some period of time, and retrieval implies recall of the trace into awareness.

In order to study these three phases of memory, it was necessary that subjects learn some specified material. In the present case, the material to be learned was paired associate lists. The paired associate method was chosen for two reasons. First, with paired associates it is possible to maximize interference effects by pairing the same stimuli, "A", first with one set of responses, "B", and then with a new set of responses, "C". Under these conditions, subjects who receive the placebo dose (controls) should experience some interference effects. The degree of interference can be measured and compared to that for alcohol subjects. Second, because paired associates are lists of stimulus-response pairs,

one can tabulate subjects' success or failure on each item in each presentation of a given list, and in later recall trials.

Differences between alcohol and control subjects in acquisition, or learning, were assessed by comparing number of trials required by both groups to reach criterion. In order to elucidate the nature of the memory impairment under alcohol, two kinds of memory test, free recall and matching, were used. With these two measures, it was possible to ascertain whether alcohol impairment was due to loss of the associations or simply to unavailability of responses.

Free recall performance was scored in two ways, one called the easy scoring method and the other, the hard scoring method. In the free recall (hard) test, subjects were required both to recall the responses and correctly match them to the appropriate stimuli.

In free recall (easy) correct pairing of responses with stimuli was not required. The score was the number of responses recalled. In the matching test, stimuli and responses were provided, but had to be correctly associated. Several of the possible outcomes for these tests would permit inferences concerning which aspect of the memorial process was most affected by alcohol. For example, suppose that alcohol caused deficit in free recall (hard). This could result from loss of associations or of responses or from impairment of retrieval mechanisms, or from all three. Certain outcomes on the other two tests would reduce these alternatives to one or two possible interpretations. Other outcomes would not. Thus, if the inebriated subject showed marked improvement on free recall easy, but not on matching one could infer loss of associations but not of the response pool. Improved performance on

matching but not on free recall (easy) would be more difficult to interpret, because interpretation depends on assumptions about the relation between associations and responses. Clearly, in the latter case, the associations were retained. However, poor performance on free recall (easy) could have resulted either from loss of responses from memory or from defective retrieval. If, as is assumed here, the retention of associations implies retention of stimulus-response sets, then the latter case implies specific impairment of response retrieval mechanisms. This problem will be considered again in the discussion. Uniformly poor performance on all three tests would indicate loss of the associations, and impairment of retrieval and/or retention of responses as well.

In the present study, both PI and RI were examined. This made it possible to determine effects on memory of material learned both before and after the material to be remembered. In addition, the question of whether interference effects can account for all forgetting under alcohol was addressed. The PI and RI groups learned two lists, A-B and A-C. Control groups learned only one list, A-B, and performed a copying task instead of learning the A-C list. This task involved copying backwards a portion of a technical article on plants (Galston, 1969), and was chosen because it required a high degree of concentration, but was meaningless.

CHAPTER II

METHOD

Subjects

Subjects were 50 male students, mean age 24.3 years, ranging from 22 to 26 years. All characterized themselves as moderate social drinkers. When possible, each subject's report concerning his own drinking behavior was validated by consulting his friends. The friends were asked if they considered the subject's drinking behavior to be moderate. A person's drinking behavior was described as moderate if he sometimes drank enough to get high on weekends. Subjects were randomly assigned to experimental conditions. They were paid $2.40 per hour for their participation in the experiment.

Lists

Each list consisted of eight consonant-vowel-consonant (CVC) nonsense syllables, each paired with a two-syllable adjective. All adjectives were stressed on the first syllable. No nonsense syllable-adjective pair began with the same letter. No consonant was repeated within a given list of nonsense syllables. Nonsense syllables were chosen from Archer (1960), and had association values of 40 to 60 percent. The association values of adjectives (Thorndike and Lorge, 1944) within a given list approximately equalled those of all other lists.

Design

The design was a 2x2x2x2 factorial, with repeated measures over the last factor. There were two alcohol doses, alcohol subjects (E) received .72 ml per pound body weight while controls (C) received 15 ml in gingerale; two positions of list A-B, first or second, one or two lists learned, and two recall tests, free recall and matching. The memory of each subject was tested by both free recall and matching. In addition to these eight groups, two other groups, one E and one C, performed the copying task, then learned the A-B list. The memory of these groups was then tested by the matching test only. These groups are referred to as C21m and E21m, and differ from C21 and E21 only because they were not given the free recall test. The design is diagrammed in Table 1. In the group designation, the first number following either E or C refers to the position of A-B learning with respect to the additional task. The second number refers to the number of lists learned. For example, E12 designates those subjects who got the high dose of alcohol, learned list A-B first, and learned two lists.

Procedure

Subjects were instructed to refrain from eating for a minimum of four hours before the experiment, and to abstain from alcoholic beverages for a period of twenty-four hours before the experiment.

Prior to ingestion of alcohol, the subject received paired associate instructions over a tape recorder. Typed copies of the instructions were provided, and subjects read along with the recording. The subject was then given five trials on a practice list, a trial being defined as the presentation of all eight stimulus-response pairs.

TABLE 1

EXPERIMENTAL DESIGN

HIGH DOSE ALCOHOL (E)

Designation	First List Learned	Second List Learned	Free Recall & Matching tests
E11	A-B	copying	A-B
E12	A-B	A-C	A-B
E21	copying	A-B	A-B
E21m	copying	A-B	A-B (Matching test only)
E22	A-C	A-B	A-B

LOW DOSE ALCOHOL (C)

Designation	First List Learned	Second List Learned	Free Recall & Matching tests
C11	A-B	copying	A-B
C12	A-B	A-C	A-B
C21	copying	A-B	A-B
C21m	copying	A-B	A-B (Matching test only)
C22	A-C	A-B	A-B

The paired associate lists were presented on a Lafayette memory drum. Each CVC stimulus was exposed for two seconds, followed immediately by a two-second exposure of the same CVC paired with an adjective. The intertrial interval was four seconds.

Following the paired associate instructions and practice the subject was given a Breathalyzer test to ascertain that he had not been drinking prior to the experimental session.

Twenty-five of the subjects (Group C) received 15 ml of 190 proof grain alcohol, 5 ml floated on each of three gingerale drinks. This dose was calculated to bring the blood alcohol concentration (BAC) to approximately .01 percent. The remaining subjects (Group E) received .72 ml alcohol per pound body weight, the amount necessary for a peak blood level of approximately .11 percent. For all subjects, the alcohol was evenly distributed over three drinks, the first drink being mixed with 250 ml gingerale, and the other two with 200 ml gingerale. Each subject was allowed one hour to consume his beverages, and was given a Breathalyzer test to determine his blood alcohol level fifteen minutes after the final drink had been consumed.

Following the Breathalyzer test, five additional practice trials were given on a second practice list. After an interval of one minute, the appropriate groups then learned list A-B or A-C to a criterion of at least six pairs correct,[1] or engaged in copying a technical article backwards. The amount of time a given subject spent copying was determined by the time an A-C, A-B subject with the same alcohol dose spent learning list A-C. Following list learning or the copying task, each subject learned either list A-C or A-B or performed the copying task, depending on the condition to which he had been assigned. Those subjects who performed the copying task second engaged in this task the same period of time the A-B, A-C subjects spent on A-C learning.

Recall of list A-B was tested one minute after the end of the second task. For the free recall test, an alphabetically arranged, typed list of the nonsense syllables from list A-B was provided. The

---

[1]See Appendix A for discussion of this criterion.

subject was asked to verbalize the adjective he had learned to each nonsense syllable. He was given three minutes in which to give all responses, about twenty seconds per response, and was allowed to give them in any order.

For the matching test, the subject was given an alphabetically-ordered, typed list of the eight adjectives used in list A-B. He indicated verbally which response had been paired with each nonsense syllable.

At the end of the experimental session, a final Breathalyzer test was given.

CHAPTER III

RESULTS

Breathalyzer Tests

For each subject, blood alcohol concentrations, determined by the Breathalyzer test, were recorded both immediately preceding (BAC_1) and following (BAC_2) the experimental tasks. In Group E, the means for BAC_1 and BAC_2 were .11 and .10, and the standard deviations were each .01. For Group C, the mean BAC_1 was .01, and the mean BAC_2 was .00, the standard deviations being .00 for both BAC_1 and BAC_2.

Acquisition

The mean number of trials to criterion for each group is presented in Table 2. One can see that all C groups reached criterion before any E group. Since all subjects learned list A-B, but not list A-C, acquisition data for list A-B were subjected to a 2x2x2 analysis of variance, the table for which can be found in Appendix D. On the average, Group E required ten more trials than Group C to reach criterion, a difference which was highly significant ($F=27.79$, $df=1$ and 32, $p < .001$). The acquisition curves shown in Figure 1 indicate that the alcohol-induced impairment of learning was present in the earliest trials, and persisted throughout the session. The analysis of variance (Appendix D) showed that in general neither the position of list A-B (first or second) nor

number of lists learned affected its acquisition in either group.

TABLE 2

AVERAGE NUMBER OF TRIALS TO CRITERION

Group	First List	Second List
E11	(A-B) 19.6 ± 9.24[a]	----
E12	(A-B) 13.6 ± 9.84	(A-C) 11.0 ± 2.83
E21	----	(A-B) 15.8 ± 5.89
E21m	----	(A-B) 15.4 ± 7.06
E22	(A-C) 13.0 ± 5.05	(A-B) 14.4 ± 4.51
C11	(A-B) 5.8 ± 2.49	----
C12	(A-B) 6.0 ± 2.35	(A-C) 8.0 ± 3.32
C21	----	(A-B) 6.4 ± 1.14
C21m	----	(A-B) 7.0 ± .71
C22	(A-C) 9.8 ± 8.23	(A-B) 7.6 ± 2.07

[a]Standard Deviation

The effect of having learned one list on the learning of a second was further assessed for both E and C subjects by comparing number of trials to acquisition of list A-B for those subjects who learned only the A-B list to the learning of A-B for subjects who acquired A-C first. Neither alcohol nor control subjects took more trials to learn A-B after having learned A-C than did subjects who learned only A-B ($t=-.89$, $p > .10$; and $t=1.42$, $p < .10$). However, an analysis of specific trial errors showed that there were effects of learning a first list on the acquisition of the second which differed for the two groups. For subjects who learned two lists, the number of first-list responses

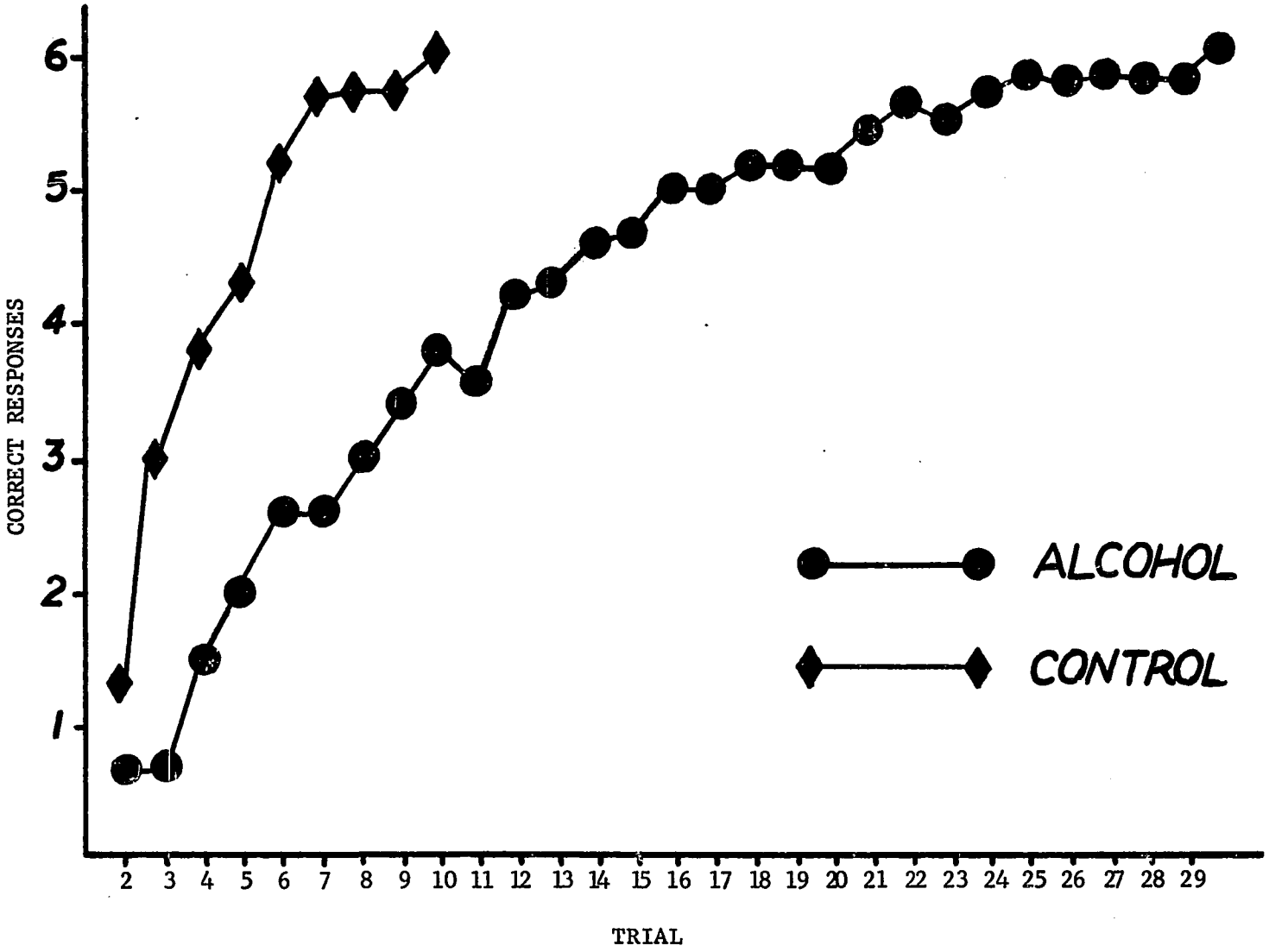


Figure 1. List A-B acquisition

appearing in second-list learning (intrustions) was counted. To be classed as an intrusion error, the response had to be exactly as given on the first list, not an approximation. The mean intrusion scores were 3.2 and 0.3 for the alcohol and control subjects respectively, a difference which is significant beyond the .05 level (t=2.36). This finding suggests that alcohol potentiated interference effects in the A-B, A-C paradigm, an interpretation which is supported by the analysis of memory which appears in a later section of this chapter.

What accounts for the slow learning shown by the alcohol groups? Inspection of the data suggested that the alcohol subjects had acquired the response pool several trials before they were able to associate the responses to the correct stimuli, whereas it appeared that the controls had acquired the S-R associations and the response pool almost simultaneously.

In order to examine this hypothesis, a cumulative count was made for each subject of the number of different A-B responses expressed on or before each of four different trials, using as a reference, the trial immediately preceding the acquisition trial, Y-1, and the three adjacent trials preceding that trial, Y-2, Y-3, and Y-4. It was not necessary that a response be correctly paired with its stimulus in order to be counted. Table 3 shows the average number of different A-B responses accumulated on or prior to Y-1, Y-2, Y-3, and Y-4[2]. Examination

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[2]These means do not include trials on which it was impossible for a subject to score. For example, if a subject reached criterion in four trials, for him there would be no such trial as Y-4. His data would therefore be excluded from the Y-4 mean. Thus, for C, 17 subjects are included in Y-4, 22 in Y-3, and 25 in Y-2 and Y-1. For E, 25 subjects are included in all four trials.

TABLE 3

NUMBER OF DIFFERENT RESPONSES ON EACH OF THE FOUR TRIALS PRECEDING ACQUISITION TRIAL, AVERAGED FOR E AND FOR C

	Y-4	Y-3	Y-2	Y-1
E	5.5 ± 2.14 (N = 25)	6.2 ± 1.37 (N = 25)	6.7 ± 1.07 (N = 25)	6.9 ± 1.00[a] (N = 25)
C	2.4 ± 2.27 (N = 17)	3.8 ± 2.35 (N = 22)	5.1 ± 1.90 (N = 25)	6.1 ± 1.26 (N = 25)

[a]Standard Deviation

of this table shows a difference between alcohol and control groups in the trial on which they had accumulated six or more different responses. For example, on trial Y-3, the third trial preceding the acquisition trial, the Group E subjects had given an average of 6.2 different A-B responses. Group C, on the other hand, had given only 3.8 different responses on that trial, and achieved six different responses only on Y-1, the trial just preceding criterion performance. Thus, E subjects apparently required about three trials after their response pool acquisition trial to reach criterion, while C subjects needed only one additional trial.

The cumulative analysis assumes, however, that once a response was acquired, it remained in the subject's repertoire until the learning criterion was reached. A second method of analysis which does not make this assumption is to identify the precise trial on which each subject first emitted six different responses from the A-B list. This analysis, which failed to confirm the results shown in Table 3, indicated that for both the E and C subjects, six different responses were expressed only on trial Y, the acquisition trial. This result implies that it cannot

be assumed that a response, once acquired, remained in the repertoire; at least not for the alcohol groups. Instead it appeared that, for the alcohol subjects, a response, once expressed, might be forgotten on later trials.

More detailed analysis of the A-B data indicated, however, that there were at least two reasons for the slowed learning of the E subjects: (1) having once emitted a response which was a member of the A-B set, the alcohol subject was more likely to forget the response on a succeeding trial (response loss), and (2) having once associated a response with the correct A-B stimulus, the alcohol subject was more like to associate the response incorrectly on a succeeding trial. The latter error will be called equivocation.

The means for response loss and equivocation shown in Table 4

TABLE 4

MEAN RESPONSE LOSS AND EQUIVOCATION FOR ALCOHOLS AND CONTROLS

	Response Loss	Equivocation
Alcohol	.84 ± .37[a]	.11 ± .10
Control	.49 ± .19	.02 ± .00

[a]Standard Deviation

are based on scores for each subject as follows: For each successive pair of acquisition trials (2-3, 3-4, 4-5, etc.) up to and including the criterion trial, all responses which were members of the A-B set were tallied and scored correct or incorrect. Lost responses were those which were expressed on the (i)th trial and not given on the (i+1)th trial. Equivocation was scored when a response was linked to the correct stimulus

on the (i)th trial and expressed but incorrectly linked on the (i+1)th trial. Each of the two types of error was then summed for each subject, and divided by the number of trials to criterion, the resulting scores being the average response loss and the average equivocation per acquisition trial.

The results in Table 4 suggest that both groups suffered some response loss, and statistical analysis (zero-mu t-test, t_o) showed that for each group this effect was significantly different from zero (t for controls=5.69, $p < .001$). However, the average response loss per trial for the alcohol subjects was nearly twice that for the controls (.84 and .49, respectively) and this difference was also statistically significant ($t=3.09$, $p < .01$).

The average equivocation score for controls (Table 4) was essentially zero, while the score for alcohol subjects was low (.11) but significantly different from zero ($t_o=6.88$, $p < .001$) and from the control mean of .02 ($t=4.52$, $p < .001$).

Thus the data for both groups indicate that during acquisition, members of the response set could be learned, only to be forgotten on later trials. Alcohol increased the rate of this short-term forgetting, and this in part accounts for the increase in trials to criterion. However, the alcohol subjects were handicapped by a second problem which was not found for the control subjects. That is, once a control subject had correctly associated a response with a stimulus, the correct linkage was sustained on the succeeding trial, provided the response was emitted. Conversely, the alcohol subjects showed a low but significant rate of equivocation, such that a response correctly associated on one trial

might be recalled but incorrectly linked on the next.

It could be argued that the difference in equivocation scores between alcohol subjects and controls was a function of the alleged disinhibiting effects of alcohol. Thus, the control subject, when uncertain about an S-R pair, might omit the response on that trial, while the alcohol subject might express the response despite his uncertainty. If this were true, one could expect the overall response rate of the alcohol subjects to be greater than that of the controls. This hypothesis was tested by computing, for each subject, the mean number of responses per trial during acquisition. The data in Table 5 show that alcohol and

TABLE 5

NUMBER RESPONSES PER TRIAL, AVERAGED FOR E AND FOR C

	Responses Per Trial	Correct Responses Per Trial	Incorrect Responses Per Trial
E	4.27 ± 1.19	2.81 ± .64	1.46 ± .92
C	4.57 ± .93	3.76 ± .66	.81 ± .77

control subjects gave approximately the same number of responses per trial. For the alcohol group, 65.8 percent of all responses were correct, while 34.2 percent were incorrect. For controls, the corresponding percentages were 82.4 and 17.6. The t-test revealed that controls made as many responses on each trial as did alcohol subjects, ($t=1.00$, $p>.10$), but responded correctly more frequently ($t=5.28$, $p<.001$), and incorrectly less frequently ($t=-2.78$, $p<.005$).

Memory

A subject's score for free recall (hard) (FRH) was defined as

the number of A-B responses he correctly associated with A-B stimuli. The score for the matching test (M), where both the stimuli and responses were made available, was the number of responses correctly assigned to the A-B stimuli.

The average number of errors for each group on all three measures of memory is presented in Table 6. It can be seen that in general, the

TABLE 6

MEAN NUMBER ERRORS ON MEMORY TESTS

	Free Recall (Hard)	Free Recall (Easy)	Matching
ALCOHOL			
E11	2.6 ± 2.07[a]	2.6 ± 2.07	1.8 ± 1.30
E12	6.4 ± 1.14	5.6 ± 1.52	3.8 ± 1.48
E21	3.2 ± 1.92	2.0 ± 1.41	2.0 ± 1.58
E21m	----	----	3.0 ± 2.68
E22	3.8 ± 2.17	3.0 ± 1.87	1.6 ± 1.67
CONTROL			
C11	1.4 ± 1.67	1.2 ± 1.64	1.0 ± 1.41
C12	3.8 ± 2.39	3.6 ± 2.07	0.8 ± 1.30
C21	1.0 ± .71	0.8 ± 0.45	1.0 ± 1.00
C21m	----	----	0.8 ± 1.09
C22	1.4 ± 2.07	1.2 ± 1.64	1.6 ± 2.19

[a] Standard Deviation

memory test performance of control subjects surpassed that of alcohol

subjects. In all three tests, all control groups except C12 performed as well as or better than any alcohol group. Analysis of variance showed that the superior overall performance of control subjects was statistically reliable. For FRH and M the F's were 15.31 ($p < .01$, 1 & 32 df) and 5.00 ($p < .05$, 1 & 32 df), respectively. Results of the analysis of variance for memory are presented in Appendix E.

As expected M was easier for both groups than FRH ($F=45.96$, $p < .001$). When analyzed separately, each group showed better M than FRH performance, with $F=42.5$ ($p < .01$) and $F=9.41$ ($p < .01$) for the alcohol and control groups, respectively.

The overall effect of alcohol on retention of the response pool and on S-R pairings was examined. It will be recalled that two scores, based on "hard" and "easy" criteria were computed for the free recall test. In free recall (easy) (FRE) a response from the A-B list was counted correct even if it was paired with the wrong stimulus. If, as suggested by the acquisition data, the alcohol subject retained the response pool, but not the S-R pairings, then scores on FRE should be identical for the two groups. However, analysis showed that even with the relaxed criterion, controls were superior to alcohol subjects. For the E groups the mean number of correct responses was 4.7, while for controls, the mean was 6.3, this difference being significant beyond the .01 level (F-test). In general, FRE was not significantly easier than FRH ($t=.89$, $p > .10$), but was harder than matching ($t=1.91$, $p < .05$). Mean correct responses were 5.50 for FRE, 5.05 for FRH, and 6.28 for M. Separate analysis for alcohol and control subjects showed that FRE was not easier than FRH for either group ($t=1.00$, $p > .10$; $t=.33$, $p > .10$). For

alcohol subjects, mean errors were 3.30 for FRE and 4.00 for FRH. For controls, corresponding means were 1.70 and 1.90. Performance was no worse for FRE than for M, for either alcohol or control subjects ($t=1.52$, $p > .05$; $t=1.38$, $p > .05$). For alcohol subjects, mean errors were 3.30 for FRE and 2.40 for M. For controls, means were 1.70 and 1.04.

The question of what was forgotten by the alcohol groups can also be examined by more detailed analysis of the relative impairment on FRH and M. The fact that controls were superior to alcohol subjects on M indicates that the E groups had forgotten more of the S-R links than the C groups. However, the relative effects on FRH and M were not identical for the two groups. The significant ($p < .05$) drug by test interaction effect illustrated in Figure 2 indicates that the difference between the two groups was larger on FRH than on M. Taken together, the two analyses indicate that alcohol impaired the retention of both the response set and the S-R links, but the relatively greater improvement shown by alcohol subjects on M suggests that in general the alcohol effect was more pronounced on the response set.

Incidentally, scores on M apparently were not affected by the interpolated FRH test for either group of subjects. Comparison of E21 with E21m, and of C21 with C21m showed that neither difference was statistically significant.

Is the effect of alcohol on verbal memory due to impairment of retention mechanisms or retrieval or both? It will be seen that the answer probably depends on the specific learning requirements imposed on the subject. In the non-interference conditions where subjects learned only one list, the performance of controls was clearly superior

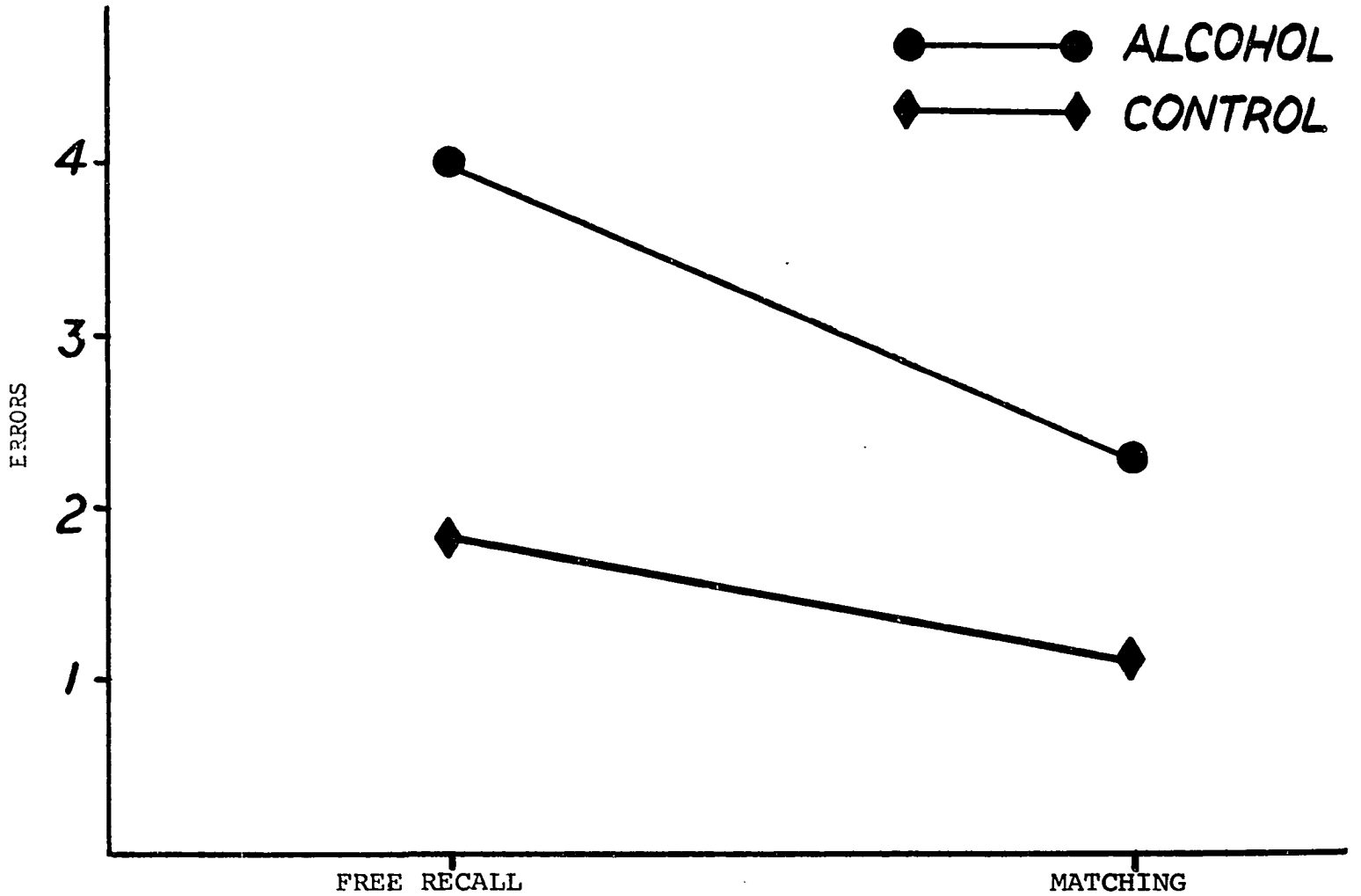


Figure 2. Drug by test interaction

to that of alcohol subjects on FRH ($F=5.02$, $p < .05$) and FRE ($F=4.0$, $p < .05$) but not on M ($F=1.41$, $p > .10$). Thus, where interference due to learning more than one list was not a factor, alcohol tended to impair response retrieval, (and/or retention), but not retention of associations. However, the drug by test interaction, illustrated in Figure 3, failed to reach significance, indicating that in the non-interference condition alcohol's differential effects on these memory phases was not pronounced.

The effect of interference on the alcohol subjects depended upon whether list A-B was learned first or second, i.e., PI vs RI. When A-B had been learned first, followed by A-C, FRH, FRE and M performance all were impaired by alcohol, the F's being 5.87, $p < .05$; 3.64, $.05 < p < .10$; and 7.81, $p < .01$, for FRH, FRE, and M, respectively, for E12 vs C12. Thus, the drug-test interaction displayed in Figure 4 was significant ($F=10.59$, $p < .01$). Figure 4 shows that controls performed about the same (and well) on both memory tests, while alcohol subjects showed substantial improvement on M. On the other hand, when the second list learned was the list to be remembered, the effect of alcohol was the same as that for the non-interference case. Controls were clearly superior to alcohol subjects only on FRH and FRE, suggesting that in this case alcohol impairs only response retrieval (and possibly response retention). The F's were 5.0 ($p < .05$); 2.94 ($.05 < p < .10$), and 0.0 for FRH, FRE, and M, for E22 vs C22.

It may be argued that since controls who learned one list or learned list A-B second made so few errors on FRH there was virtually no room for improvement on M. There is some evidence against this argument, since their errors on both free recall and matching were significantly

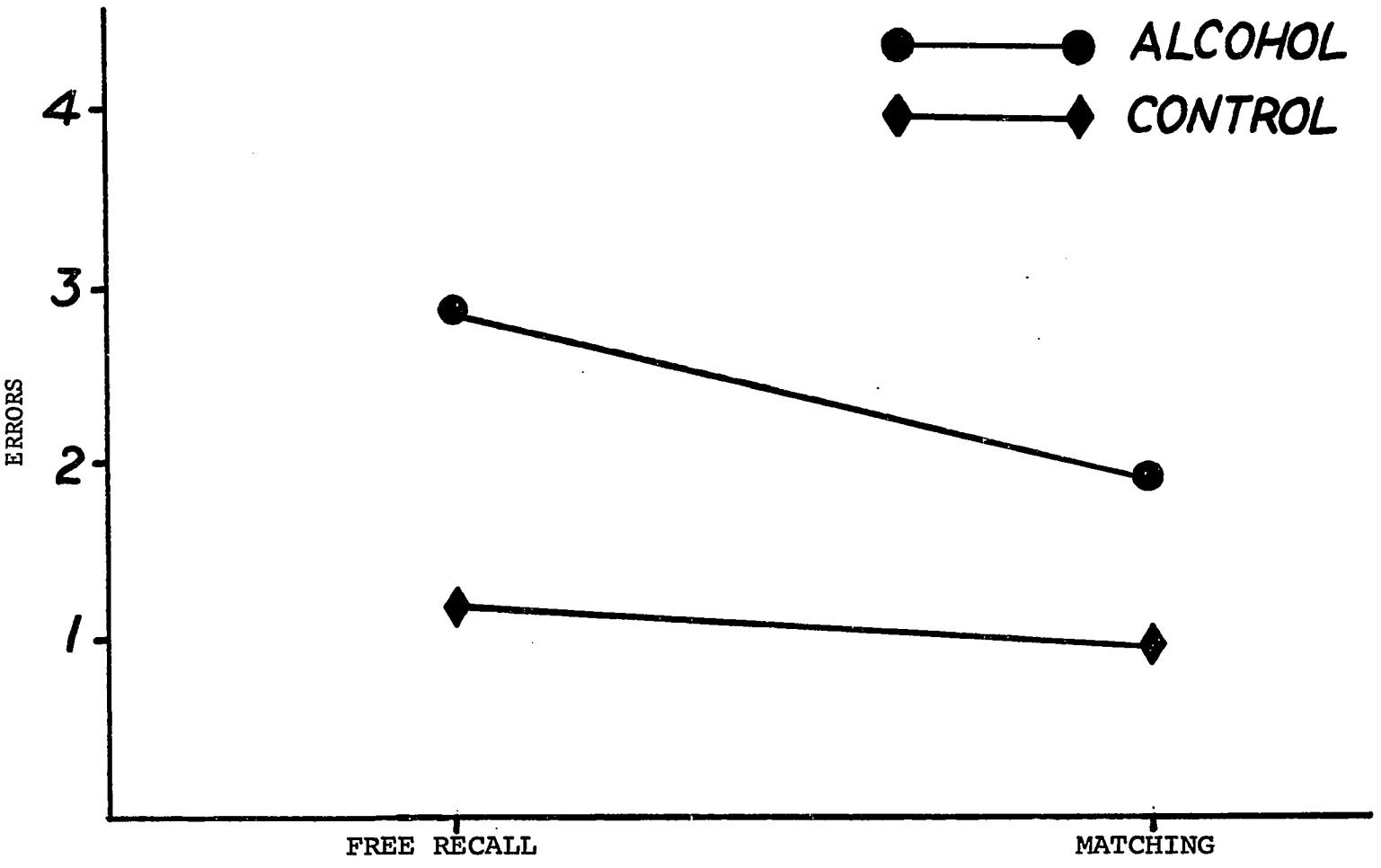


Figure 3. Drug by test interaction for subjects who learned one list

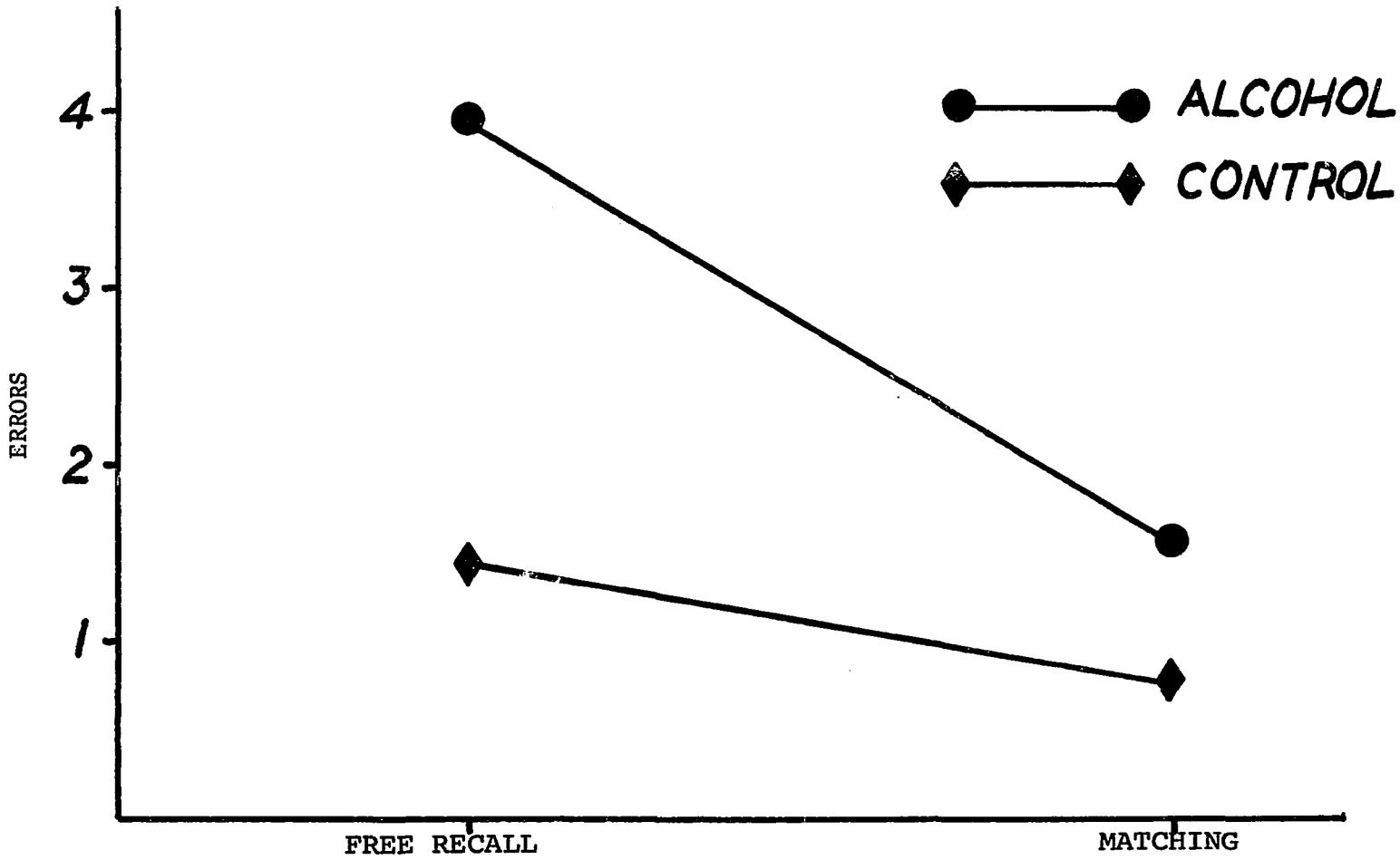


Figure 4. Drug by test interaction for subjects who learned A-B, then A-C

different from zero (t_o=3.30, $p < .01$; and t_o=3.06, $p < .01$). These results imply that when interference is generated by interpolated learning, the effect of alcohol becomes more complex.

Are the effects of proactive and retroactive inhibition altered by alcohol? Table 7 presents the data for PI and RI, for both alcohol

TABLE 7

PROACTIVE AND RETROACTIVE INHIBITION

Groups	Indicates	Test	F	t
E12 vs E11	RI for E	FRH	12.53[c]	
		FRE		2.61[b]
		M	3.47[a]	
C12 vs C11	RI for C	FRH	5.00[b]	
		FRE		2.03[b]
		M	.03	
E22 vs E21	PI for E	FRH	.31	
		FRE		.95
		M	.14	
C22 vs C21	PI for C	FRH	.14	
		FRE		.53
		M	.31	

[a] $p < .10$
[b] $p < .05$
[c] $p < .01$

and control subjects. PI was examined by comparing the FRH, M, and FRE performance of E22 with E21 and C22 with C21. Neither alcohol nor control subjects showed any evidence of PI on any memory test. However,

RI effects were found in both groups. RI was assessed by comparing performance of E12 and C12 to that of E11 and C11, respectively, for FRH, FRE, and M. Alcohol subjects showed RI on all memory tests, while controls had RI effects only on FRH and FRE. It can be seen that the combination of RI and alcohol disrupted retention of S-R bonds, as well as impairing retrieval and/or retention of responses, while RI alone was probably detrimental only to response retrieval.

It will be recalled that in Chapter I the question was asked whether interference effects can account for all forgetting under alcohol. Assuming that the allegedly non-interfering task used in the present experiment did not disrupt memory, those subjects who learned only one list should have performed nearly perfectly on the memory tests. However, FRH and M scores for both alcohol and controls who learned one list differed significantly from zero. For alcohol subjects, the zero mu t-tests were 4.80 ($p < .001$) for FRH and 4.38 ($p < .01$) for M. Corresponding t_os for controls were 3.09 ($p < .05$) and 2.74 ($p < .05$). Thus, both these memory tests showed that even in the absence of interfering material, some forgetting occurred for both alcohol and control subjects. As was shown in Table 6 and Figure 3, this deficit was greater for alcohol subjects than for controls. Is this latter result due to potentiation by alcohol of decay effects?

An assessment of A-B decay can be made by comparing the performance of those subjects who learned A-B first with those who learned A-B second, when A-B was the only list learned by either group. The ts for alcohol subjects (E11 vs E21) were as follows: FRH, -.47; FRE, .53; M, -.22. For controls (C11 vs C21), the ts were: FRH, .49; FRE, .53;

M, .00. Thus, there was no evidence of decay for either the alcohol or control subjects. In fact, the negative ts found in the alcohol group indicate that those subjects averaged slightly more errors when A-B was learned second than when it was learned first, a finding which is the reverse of that predicted by the decay hypothesis. It seems reasonable to conclude that for both groups, single-list forgetting was due to non-specific interference effects, and that these were potentiated by alcohol.

CHAPTER IV

DISCUSSION

Acquisition

The results of the present investigation agree with previous research (Korman, Knopf, & Austin, 1960; Storm, Caird, & Korbin, 1965; Storm & Caird, 1967; Tartar, 1968; and Goodwin, et al., 1969) in finding acquisition of verbal material to be impaired by alcohol. The study showed further that in general A-B learning was not affected by prior learning of list A-C or by prior performance of the copying task. In addition, alcohol and control subjects were not affected differentially by these two factors.

The nature of the learning problem of alcohol subjects was examined in several ways. It was found that with reference to the trial on which criterion was achieved, the alcohol subjects had apparently learned the six required A-B responses at least three trials before they reached the criterion of six correct S-R pairs, while the control subjects were able to express the correct S-R pairs almost simultaneously with accumulation of the six responses. On the other hand, identification of the exact trial on which the six responses were first emitted showed that for both groups, the first expression of the six required responses occurred simultaneously with achievement of the experimental criterion.

These discrepant results disconfirm an assumption which is necessary for the cumulative analysis; namely, that a response once expressed remains in the subject's repertoire throughout the remaining trials to criterion. If that had been true the results of the two analyses of response acquisition would have been identical. An examination of the trial-to-trial consistency of responding showed that for both alcohol and control subjects, a response could be expressed on a given trial and lost on the next. However, the rate of response loss from trial to trial was considerably greater for alcohol subjects than for controls.

In the analysis of trial data, a second kind of error was identified, in which the subject correctly linked a response to a stimulus on a given trial and assigned the same response to another (wrong) stimulus on the next trial. This was labeled equivocation. For control subjects, the rate of equivocation per trial was essentially zero, but for the alcohol groups this was a statistically significant, although relatively low-rate, kind of error.

These results imply that slow verbal learning by the alcohol subject results from the fact that alcohol impairs short-term retention of both the responses and the S-R links. The rate of equivocation, however, is quite low compared to the rate of response loss, so that the latter effect probably accounts for most of the learning deficit caused by the drug.

One objection to this interpretation is that failure to emit a response may reflect uncertainty and caution rather than loss of the response from recent memory. The instructions to the subjects were to

verbalize a response to a given stimulus when they knew the correct response. They knew that they might guess, but may have responded only when fairly confident of the S-R pairings.

Underwood, Runquist and Schulz (1959) gave free recall (easy) tests at various points during paired associate learning to assess the degree of response learning. Their method more accurately reflects response availability than the method used here. It might also be argued that subjects under the influence of alcohol may be less cautious than sober subjects, so that for a control subject, the number of responses emitted at any point during learning would underestimate the size of his response pool. It is true that the responses given by controls were more frequently correct than those given by alcohol subjects, but alcohols and controls did not differ in the average number of responses (right plus wrong) given per trial. The averages for both groups were much lower than eight per trial, the maximum possible. There is, therefore, no basis for assuming that the responses given by alcohol subjects represented a greater percentage of their response pool than those given by controls. For the same reason, it is unlikely that the significantly greater rate of equivocation found in the alcohol groups was due to loss of response inhibition or lack of caution.

Interference effects during paired associate acquisition have been studied by a number of investigators (e.g., Bunch & Winston, 1936; McGeoch, McKinney & Peters, 1937; and Baddeley & Dale, 1966). Bunch and Winston's subjects found it harder to learn A-C if A-B had been learned a week earlier, than if only A-C was learned. However, when list A-C immediately followed A-B learning, the effect of having learned A-B on

A-C acquisition was apparently minimal (McGeoch, et al., 1937; Baddeley & Dale, 1966). The results of the present study support this latter finding. The number of trials to criterion on list A-B did not depend upon whether A-C learning had preceded A-B acquisition. This was true for both alcohol and control subjects. Even though first list learning did not retard second list learning for alcohol subjects, it is interesting to note that for them, some interference effects were evident. The number of first list intrusions during the acquisition of the second list was significantly greater for these subjects than for controls. If the notion of several investigators (e.g., Melton & Von Lackum, 1941; and Barnes & Underwood, 1961) is correct, that first list responses are unlearned during second list learning, then it can be concluded that alcohol subjects experienced difficulty in unlearning first list responses, once they had finally mastered them. This then, was an indication that alcohol potentiated interference effects.

Memory

The results of the present study reveal that alcohol disrupts memory. This finding has been reported by a number of investigators (Vogt, 1930; Jellinek & McFarland, 1940; Carpenter 1962; Kalin, 1964; Storm, Caird, & Korbin, 1965; Storm & Caird, 1967; and Goodwin, et al., 1969).

In this study, differential effects of alcohol on the retention and retrieval phases of the memorial process are difficult to identify for two reasons: (1) they depend on the conditions of learning, and (2) the three tests of memory which were used were not specific enough to rule out certain interpretations. Consider, for example, the sequence

of scores found for the alcohol subjects. They were relatively impaired on both tests of free recall, but showed significant improvement on matching. In idealized form, this pattern would indicate that they had retained the S-R bonds, but that either retention or retrieval of responses was impaired. As was pointed out in the introduction, if one assumes that retention of S-R bonds implies retention of the response set, then the pattern described above implies impairment of response retrieval mechanisms. It should be clear, however, that a more direct probe could have been made with a multiple-choice test, where the subject selected the correct response from a group of adjectives. If he were unable to recognize the correct response, we could conclude that he had failed to retain the response because in this test, retrieval would not be required.

Realizing that the argument may be tenuous, the writer has chosen to make the assumption that retention of S-R bonds implies retention of the response set. Therefore, where appropriate, inferences will be made about response retrieval, and will not include the qualifier, "and/or response retention".

The comparison of all alcohol subjects with all controls showed inferior performance of alcohol subjects on all three measures of memory, FRH, FRE and M. Thus, the overall effect of alcohol was to impair both the retention of associations and the retrieval or retention of the response pool. However, the significant drug by test interaction showed that, relative to the controls, the alcohol subjects improved on M. This implied that the greatest effect of alcohol was on retrieval of the response set.

Under two learning situations, that in which only one list was learned and that in which the last list learned was the list to be remembered, alcohol disrupted only response retrieval. But when A-C was learned between the learning of A-B and testing for A-B memory, both retention of S-R associations and retrieval of the response set suffered.

When alcohol's effects on PI and RI were explored, no evidence for PI was detected. PI increases as the length of the retention interval increases (Underwood, 1948; Goggin, 1966; Slamecka & Ceraso, 1960; and Houston, 1967) and as the degree of original learning increases (Waters, 1942). The A-B list in the present experiment was learned to a criterion of only 75 percent, and the retention interval was only one minute. Houston (1967) demonstrated that a one-minute retention interval was insufficient to produce PI, even when the criterion for original learning was 100 percent.

A number of investigators have demonstrated methods by which RI effects can be magnified. RI may be potentiated by increasing the similarity of first and second list stimuli (McGeoch & McGeoch, 1937; McGeoch, McKinney, & Peters, 1937; Melton & Von Lackum, 1941; Osgood, 1949; and Baddeley & Dale, 1966). RI also increases as a function of degree of interpolated learning. (McGeoch & McGeoch, 1937; Swenson, 1941; and Thume & Underwood, 1943), although with very high degrees of interpolated learning, RI fails to increase further (Thune & Underwood, 1943).

In the present study, alcohol subjects experienced a greater RI effect than did controls on both memory tests, a fact which implies that alcohol, too, potentiates RI. For controls, there was total absence of RI on the matching test, indicating that for them, the learning of list

A-C disrupted retrieval of the responses, but not the retention of associations. Alcohol subjects showed RI on FRE, FRH, and M. Therefore, for alcohol subjects, the deficit on list A-B brought about by learning list A-C affected both retention of associations and either response retention or retrieval or both.

According to interference theory, all memory deficit can be attributed to either proactive or retroactive influences. The results of this study showed that even under non-interference (i.e., non-RI) conditions, both alcohol and control subjects exhibited some forgetting. In this situation, alcohol exaggerated memory loss, especially impairing response retrieval. Can decay theory account for forgetting in non-interference conditions? Does alcohol accelerate trace decay? If so, those subjects learning a single list second should have performed better than those learning a single list first. The results showed absolutely no such trend. Presumably the small but significant deficit shown by the controls for single list recall was due to non-specific interference effects rather than decay, and apparently alcohol potentiated these effects. The finding that alcohol potentiated RI effects, and non-specific interference effects as well, implies that alcohol is itself a source of interference. That is, alcohol disrupted memory in a very specific way, such that the effects of this physiological agent on verbal memory were similar to if not identical with the effects of RI, on operation defined in behavioral terms.

Implications

Kalin's (1964) study suggested that the interfering effects of interpolated material are reduced by alcohol. The present study found

the opposite to be true. Interference effects increased under alcohol. One important difference between this and Kalin's study might account for the discrepancy in results. Kalin's subjects were tested for recall while sober, while subjects in the present study were tested while still intoxicated. Recent evidence from state-dependent learning studies (e.g., Storm, Caird & Korbin, 1965; Storm & Caird, 1967; and Goodwin, et al., 1969) shows that memory is better when material is both learned and recalled while the subject is in the same state (either drunk or sober) than when learning and recall occur in different states. The alcohol subjects in Kalin's study remembered their first TAT story better than they remembered more recent ones. Since the first story was written prior to drinking, while the others were written during the drinking period, it could be argued that subjects recalled the first story best, because their state was the same when they wrote and recalled that story, but different when they wrote and recalled the other stories. The present study, in which subjects remained in the same state for both learning and testing, allows a more definitive statement about the effects of alcohol on RI. This investigation clearly showed that alcohol potentiated RI.

Studies concerning the memory of Korsakoff patients have failed to agree on effects of this disease on memory. Only one such study (Talland, 1959) used materials similar to those of the present investigation, and it found no evidence for RI in Korsakoff patients. The pronounced effects of a single dose of alcohol on RI found here suggest that impairment of Korsakoff patients is not identical with the impairment induced by the drug. However, Korsakoff's syndrome develops not only

from alcohol, but probably also out of dietary deficiencies and brain damage. The difference between these and Talland's findings may be due to the fact that the disease is not wholly attributable to alcohol. Or, it may be that if BACs in the present study had reached higher peaks, alcohol would have eliminated RI.

How could alcohol produce interference effects on verbal recall? Posner (1966) discusses the concept of an information processing capacity in short-term memory. He believes that both previously stored material and interpolated information processing reduce this capacity. Because the same information-processing mechanisms both sustain the memory trace for the first few seconds and handle new information, increasing the interpolated information load disrupts memory.

The results of an unpublished study by Janes and Williams (1969), as well as for the acquisition phase of the present investigation, could be explained in terms of this information processing capacity. The Janes and Williams study, in which alcohol and control subjects either heard a story or read it aloud showed that alcohol was especially effective in reducing story recall when subjects read the story. It was suggested in that study that the effect of alcohol was to reduce information processing capacity so that the simultaneous requirements of reading the story aloud and committing it to memory overloaded the system in such a way that memory suffered. In the present study, the reduction of information processing capacity by alcohol may have made difficult the tasks of both retaining previously learned responses and acquiring new responses.

Posner's model may not directly apply to the memory portion of the present investigation, since it is intended to explain forgetting

over seconds rather than minutes. It becomes relevant, however, if we assume that verbal material is remembered by rehearsal, even for relatively long-term storage, and that alcohol reduces the capacity for rehearsal. In this case, Posner's model would predict the interaction between RI and alcohol effects found here. That is RI, as interpolated information processing, uses part of the channel available for rehearsal of the A-B list. Alcohol further reduces channel capacity, thus potentiating the interfering effect of RI.

It might be argued that differences between alcohol and control subjects on task performance in the present study were simply due to motivational differences. Although it is impossible to disprove that argument, the results suggest that alcohol subjects were motivated. First, all subjects reached criterion. Second, there are differential effects on response measures. If one assumed a generalized defect in motivation, differential effects on performance might be hard to explain. Furthermore, all subjects in this study were judged by the investigators to be highly motivated throughout the experiment. They appeared to be trying to conceal rather than reveal their state of inebriation. Nevertheless, the effects of alcohol found here might be quantitatively and qualitatively different in subjects working for different incentives.

If alcohol is detrimental to both learning and memory, as is shown by the results of the present study, then one would expect to find general intellectual deterioration with prolonged use. The longer the history of alcohol abuse, the greater the expected impairment. It is certainly true that Korsakoff patients, whose use of alcohol has been intense enough to cause brain damage, perform poorly on intellectual

tasks (Meissner, 1968; Victor, et al., 1959; Talland, 1959 & 1960; and Malerstein & Belden, 1968). Since these persons are brain-damaged one cannot assume that their cognitive deficits are directly attributable to alcohol abuse. Instead, they may result from CNS deterioration.

Chronic alcoholics with no evidence of gross brain damage have been studied (Fitzhugh, Fitzhugh, & Reitan, 1960; Jonsson, Cronholm, & Izikowitz, 1962; and Storm & Caird, 1967). When WAIS scores are considered, the findings of Fitzhugh, Fitzhugh, and Reitan (1960) are not in keeping with the notion that prolonged use of alcohol lowers intellectual ability. According to their study, chronic alcoholics' scores did not differ significantly from those of normals on this intelligence test. However, other tests did show differences between chronic alcoholics and normals. For example, on tests from the Halstead-Reitan battery, known to discriminate brain-damaged patients from normals, the performance of alcoholics was similar to that of brain-damaged patients. These tests are believed to measure adaptive ability.

Subjects in the Fitzhugh, Fitzhugh, and Reitan (1960) investigation had been hospitalized an average of twelve days. Pronounced intellectual impairment in alcoholics might occur only during drinking periods, so that when inebriated, the alcoholic's task performance might be much poorer than that of drinking controls. One would then expect his performance to improve as he is deprived of alcohol. Results reported by Storm and Caird (1967) and by Jonsson, Cronholm, and Izikowitz (1962) are in agreement with these hypotheses. Alcoholics in the Storm and Caird (1967) work were tested while under the influence of alcohol. The investigators were attempting to replicate an experiment they had done

with normal, drunk subjects, but had to make the tasks simpler for the chronic alcoholics. The latter simply could not reach criterion on the task that had been used for normals.

The effects over time of being hospitalized without alcoholic beverages were shown when two groups of alcoholics, the first hospitalized for an average period of 2.2 days, and the second for twenty-eight days, were compared with each other and with hospitalized controls on several tests (Jonsson, Cronholm, & Izikowitz, 1962). Large differences were found between the two alcoholic groups on memory tests, reasoning ability, and spatial ability. However, even after twenty-eight days without alcohol, chronic alcoholics were inferior to controls in eight of the twenty-four tests, including three of the five tests for verbal understanding.

Recent evidence[3] on the abstracting behavior of alcoholics indicates that this ability deteriorates as a function of duration of the drinking problem.

The above investigations lend some credence to the notion that prolonged abuse of alcohol may lower the level of intellectual functioning.

In conclusion, the effects of a single moderate dose of alcohol on paired-associate learning and recall are complex. Acquisition is slowed, apparently because of the impairment of short-term memory. The alcoholized subject loses responses once acquired, and, to a lesser degree, forgets S-R links once established. When a single list is finally learned, the main effect of alcohol (like that of RI) seems to

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[3]Personal communication, D. Jones and O. Parsons, 1970.

be on the retrieval of responses. That is, deficit appears on a test of recall but not on a matching test where both the stimuli and the responses are supplied. When there is interpolated interference, as in the A-B, A-C paradigm, alcohol affects the recall of both the responses and the S-R associations, thus potentiating the effects of retroactive interference.

A possible explanation for these effects may be that alcohol reduces the capacity for rehearsal operations, which are an active form of information processing. According to the model which was developed for short-term verbal memory by Posner, the acquisition of the A-C list in an A-B, A-C paradigm requires part of the channel normally available for rehearsal of the A-B pairs. If the capacity for such information processing is further reduced by alcohol, the interaction found here between the effect of alcohol and the effect of RI would be expected.

CHAPTER V

SUMMARY

Studies of the effects of alcohol on verbal learning and memory generally agree that alcohol impairs both functions. However, there is little information concerning how this impairment comes about, or what specific aspects of learning and retention are affected. The design of the present study permitted a closer look at the mechanisms of acquisition and memory most affected by a moderate dose of alcohol. Fifty paid male volunteers assigned to alcohol (.72 ml per pound body weight) and placebo (15 ml) conditions, learned and recalled either one (A-B) or two (A-B, A-C) lists of paired associates and retention was tested by free recall and matching.

The acquisition of the lists was slowed by alcohol, this effect being due, apparently, to impairment of short-term memory for the responses, and to a lesser extent, the S-R associations. The alcohol subjects, having once acquired a correct response, tended to forget it on a later trial.

Alcohol caused a general disruption of paired associate memory, such that both free recall and matching were affected, but there were also differential effects on the two retention tests which were related to specific conditions of learning. In the non-interference case, in

which subjects learned and recalled a single list, alcohol caused deficit in free recall but not in matching performance. It was concluded tentatively that in this simple case, alcohol had impaired the mechanisms of response retrieval but not the retention of associations. In the A-B, A-C (retroactive interference, or RI) condition. The deficit induced by alcohol extended to matching performance, and thus to retention of associations.

No evidence for proactive interference was found in either group, except that during acquisition of the second list in the A-B, A-C condition, alcohol subjects committed more intrusion errors than the placebo group. Retroactive interference alone caused deficit in free recall, but not matching performance. Thus, like alcohol, RI appeared to affect the mechanisms for response retrieval but not the retention of associations. When the two treatments were combined, impairment was extended to matching performance. Thus, alcohol potentiated the effects of RI.

Since the results did not support a decay explanation of memory loss, it was concluded that all forgetting was due to specific and nonspecific interference effects, and that alcohol exacerbated these effects. It was concluded from this and other studies that alcohol may potentiate the effects of RI by reducing the effective capacity for processing information. The results of the study were evaluated for their relevance to comparable studies of chronic alcoholics, Korsakoff patients and normals.

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APPENDIXES

APPENDIX A

DISCUSSION OF LEARNING CRITERION

In any study of memory, the experimenter must be certain all groups have equal initial mastery of the material before he can compare their retention. When the nature of the materials to be learned or the conditions under which they are to be learned vary among groups, it is likely that group learning curves will differ significantly. Some investigators of problems of this nature have assumed that if all groups master the material to the same criterion (e.g., one perfect trial), they have achieved equal levels of learning. Underwood (1964) shows that this assumption is invalid. If conditions cause one of two groups to learn at a significantly faster rate than another, then after one perfect recitation the faster group has actually learned more than the slower. One must consider the learning that occurs on the final trial. The faster learners gain more per trial than the slower ones. Therefore, after the last trial, the fast learners have actually overlearned the material to a greater degree than the slower group has.

Underwood (1964) suggests the use of a method by which one can extrapolate, from examination of performance on each item in n trials, the probable performance on the n+1 trial. This estimated performance on the n+1 trial is a more accurate measure of level of learning than n

performance. If one were to employ Underwood's method, one would have to run pilot studies, which would indicate acquisition rates for each group. The data from pilot studies could then be used to determine the number of trials that should be given each group in order to assure that all groups have learned to approximately the same degree.

As part of the present experiment, some experimental groups learned two paired associate lists. Underwood's procedure would require six pilot studies. Both alcohol and control subjects would learn list A-B, both would learn list A-B, followed by list A-C, and both would learn list A-C, followed by A-B. Even then, pilot work by the present investigator indicates, the variance in learning rates among alcohol subjects, especially, would be too great to allow accurate determination of any constant number of trials that would insure equal learning by all groups.

An alternative to the Underwood procedure is therefore proposed. Underwood (1964) has illustrated the gross differences in actual levels of learning between fast and slow learning groups, after one perfect trial. The slow group has reached asymptote, while the more rapid learners' curve still has a steep slope. At this level, therefore, more is being gained on each trial by the fast learners than by the slow. At lower levels of proficiency, however, 75 percent, for example, curves for the two groups are more similar, and amount gained per trial is thus more comparable. Because of the similarity of slow and fast learners' curves at criterion levels below 100 percent and the variability of learning rates within the alcohol group, a constant criterion, rather than a constant trials method, appears more advantageous. The criterion

chosen should be significantly less than 100 percent. At the same time, the level of learning should be great enough to insure some retention after interpolated activity. A criterion of six out of eight correct was therefore chosen for the present study.

APPENDIX B

INSTRUCTIONS TO SUBJECTS

The object of this task is to learn nonsense-syllable, word pairs. Each nonsense syllable is composed of three letters, a consonant, vowel, and consonant. For example, P-A-B, N-I-Q, and X-O-F. On the memory drum before you, you will first see a nonsense syllable alone, such as PIF. Read out loud the letters that comprise the nonsense syllables. Do not try to pronounce the nonsense syllable as a word. Simply spell the syllable out loud.

After the nonsense syllable appears alone, it will appear again, followed by a word, such as PIF BOOKCASE. Your task is to learn which words go with which nonsense syllables. After the entire list has been presented once, four asterisks will appear, and the list will appear again, in a different order. This time, and on all subsequent presentations of the list, as each nonsense syllable is presented alone, spell it out loud, then try to say the word that goes with it, before the nonsense syllable and word appear together. Keep trying to anticipate the words until I tell you to stop.

Remember to spell out the nonsense syllable when it appears alone. This tells me where you are on the list.

In each presentation of the word list, the pairs appear in a

different order, so do not attempt to memorize the order in which the pairs appear. The order will keep changing, although the pairs remain the same.

Now we will proceed with the first list.

APPENDIX C

PAIRED ASSOCIATE LISTS

Practice list #1	List A-B
POV coiled	GEP open
JOH equal	FUJ skillful
RUC perfect	RYK legal
BAF massive	VUL parted
MEZ gracious	DOH cautious
NAL sudden	TAS knightly
GEY foggy	CIY quiet
QIS wishful	NIM waxen

Practice list #2	List A-C
ZAC oblong	GEP missing
LYR tiresome	FUJ ghostly
KUN witless	RYK awkward
VEP hidden	VUL wealthy
HIX formal	DOH vocal
DYS jolly	TAS spicy
GOM clouded	CIY double
JOQ brittle	NIM famished

APPENDIX D

ANALYSIS OF VARIANCE FOR A-B ACQUISITION

Source	SS	df	MS	F
Drug (D)	883.6	1	883.6	27.79[a]
Position (P)	.4	1	.4	.13
List (L)	22.5	1	22.5	.71
DP	16.9	1	16.9	.53
DL	48.4	1	48.4	1.52
PL	19.6	1	19.6	.62
DPL	8.1	1	8.1	.25
Error	1017.6	32	31.8	

[a] $p < .01$

APPENDIX E

ANALYSIS OF VARIANCE FOR MEMORY

Source	SS	df	MS	F
Between Subjects				
Drug (D)	54.45	1	54.45	10.74[b]
Position (P)	11.25	1	11.25	2.22
List (L)	26.45	1	26.45	5.22[a]
DP	1.25	1	1.25	.25
DL	2.45	1	2.45	.48
PL	14.45	1	14.45	2.85
DPL	6.05	1	6.05	1.19
Error (between)	162.20	32	5.07	
Within Subjects				
Test (T)	31.25	1	31.25	45.96[c]
DT	4.05	1	4.05	5.96[a]
PT	4.05	1	4.05	5.96[a]
LT	8.45	1	8.45	12.43[b]
DPT	4.05	1	4.05	5.96[a]
DLT	.05	1	.05	.07
PLT	3.65	1	3.65	5.37[a]
DPLT	1.65	1	1.65	2.43
Error (within)	21.80	32	.68	

[a] $p < .05$
[b] $p < .01$
[c] $p < .001$