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## THE UNIVERSITY OF OKLAHOMA

### GRADUATE COLLEGE

# THE EFFECTS OF ALCOHOL ON MEMORY DURING THE ASCENDING AND DESCENDING LIMB OF THE BLOOD ALCOHOL CURVE

### A DISSERTATION

## SUBMITTED TO THE GRADUATE FACULTY

# in partial fulfillment of the requirements for the

## degree of

## DOCTOR OF PHILOSOPHY

٠<u>۳</u>.

BY

### BEN MORGAN JONES

## Oklahoma City, Oklahoma

THE EFFECTS OF ALCOHOL ON MEMORY DURING THE ASCENDING AND DESCENDING LIMB OF THE BLOOD ALCOHOL CURVE

APPROVED BY Ŵ

DISSERTATION COMMITTEE

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# THE EFFECTS OF ALCOHOL ON MEMORY DURING THE

### ASCENDING AND DESCENDING LIMB OF

THE BLOOD ALCOHOL CURVE

### CHAPTER I

### INTRODUCTION AND STATEMENT OF THE PROBLEM

### Introduction

Alcohol has been reported to affect a variety of psychological functions from psychomotor coordination to complex cognitive functions (Carpenter, 1962). Perhaps one of the most important findings has been that alcohol impairs memory, a basic component of many cognitive tasks (Carpenter and Ross, 1965). Memory deficits have been reported in nonalcoholic volunteers for short-term memory tasks such as digit span (Tarter, Jones, Simpson and Vega, 1971), intermediate-term memory for pictures from 1 to 9 minutes (Ryback, Weinert and Fozard, 1970) and longterm memory (24 hours) for word associations (Goodwin, Powell, Bremer, Hoine and Stern, 1969). Memory deficits have also been reported for alcoholics during withdrawal (Allen, Faillace and Reynolds, 1971), intoxicated alcoholics (Goodwin, Othmer, Halikas and Freeman, 1970) and chronically deteriorated alcoholic Korsakoff patients (Cermak, Betters and Goodglass, 1971; Talland, 1965).

Thus, memory deficits are associated with both acute and chronic use of alcohol. Several reports have indicated that memory deficits also

are related to the rise of the blood alcohol level in alcoholic (Goodwin et al., 1970) and non-alcoholic (Ryback et al., 1970) subjects. However, whether these same deficits also appear on the descending limb has not been reported. This appears important since recent evidence (Jones and Vega, 1972) has re-emphasized earlier reports (Goldberg, 1943) that cognitive functions are more affected during the rise of the blood alcohol level (ascending limb) than during its decline. The ascending limb of the blood alcohol curve may last up to one hour after a single drink depending upon dose and concentration of the drink. However if testing is begun an hour or more after ingestion, the peak blood alcohol level probably has been passed and the subject may be on the descending limb, although the blood alcohol level may register the same as if testing had been done on the ascending limb. If memory is also affected differentially on the ascending and descending limb, it may indicate that cognitive deficits are a result of impaired memory functioning and not pure "reasoning" ability (Carpenter and Ross, 1965). It therefore seems necessary to clarify the effect of alcohol on short-term, intermediate-term and long-term memory in regard to the ascending and descending limb of the blood alcohol curve.

Although differences between the ascending and descending limb have been primarily reported for psychological functions, there are also biological differences. The clearest evidence of biological differences has been reported for the electroencephalogram (EEG), a biological rhythm frequently associated with behavior changes. EEG changes are closely related to blood alcohol level on the ascending limb with an initial excitation followed by slowing (Gibbs, Gibbs and Lennox, 1937; Guerrero-

Figueroe, Rye, Gallant, and Bishop, 1970; Hadji-Dimo, Ekberg and Ingvar, 1968; Horsey and Akert, 1953; Von Hedenstrom and Schmidt, 1951). The EEG on the descending limb returns to normal rather slowly and is not related to blood alcohol level (Davis, Gibbs, Davis, Jetter and Trowbridge, 1941; Engel and Rosenbaum, 1945; Gibbs <u>et al</u>., 1937; Ungerleider, 1958; and Von Hedenstrom and Schmidt, 1951).

Alcohol also effects hormonal secretion differently on the ascending and descending limb. Alcohol diuresis occurs only on the ascending limb and is related to the duration of the increasing blood alcohol level (Bisset and Walker, 1957; Eggleton, 1942, 1949; Flynn, 1958; Haggard, Greenberg and Carroll, 1941; Kalbfleisch, Lindman, Ginn, and Smith, 1963; Murray, 1932; Nicolson and Taylor, 1938; Raiha, 1960; Strauss, Rosenbaum and Nelson, 1950; Van Dyke and Ames, 1951). Alcohol inhibits the release of the antidiuretic hormone (ADH), thus allowing for diuresis to occur. This effect has not been observed on the descending limb.

The differences described between the ascending and descending limb for psychological test performance, electroencephalographic changes and hormonal balance all indicate that these two phases of the blood alcohol curve should be taken into consideration when evaluating the effects of alcohol.

The purpose of the present investigation is to evaluate shortterm, intermediate-term and long-term memory on the ascending and descending limb of the blood alcohol curve. The following literature review will first point out several studies that have reported differences in psychological functions on the ascending and descending limb. These

studies demonstrate the deficits are more pronounced on the ascending limb than on the descending limb. The second section will be devoted to reviewing further some of the studies that have reported memory deficits following acute alcohol ingestion. The third section is a critique and evaluation of the studies presented in the first two sections. The final section will be a statement of the problem to be investigated in this study.

The effects of alcohol on memory will be reviewed in terms of short-term, intermediate-term and long-term memory tasks. This threestage classification is similar to that of Shuttleworth and Morris (1966) that has been used by several investigators to describe both acute (Ryback <u>et al</u>., 1970) and chronic (Goodwin, Crane and Guze, 1969) effects of alcohol on memory. Short-term memory will refer to memory for events that occurred a minute or less before recall. Intermediate-term memory will cover the period of recall of information from one to fifteen minutes. Long-term memory will cover recall for more than fifteen minutes. In general, these studies indicate that memory impairment is observed for all three memory periods, but no one study systematically demonstrates this. It is also clear that memory deficits are not very apparent until a blood alcohol level of 0.07% to 0.08% is reached.

The important alcohol variables that will be considered in the following studies, if available, are amount and type of ethanol, concentration of dose, drinking time, time of testing in relation to drinking, subject characteristics and nature of the psychological task.

Effects of Ethanol on Psychological Functions During the Ascending and Descending Limb of the Blood Alcohol Curve - Literature Review

Only a limited number of studies have explicitly investigated psychological differences on the ascending and descending limb of the blood alcohol curve. A repeated testing procedure is usually employed where subjects are initially tested before ethanol administration (baseline) and then repeatedly tested during the alcohol period for eight to ten hours. Results indicate that most psychological functions are impaired during the first 30 to 60 minutes after ethanol consumption (ascending limb) (Ekman, Frankenhaeuser, Goldberg, Hagdahl and Myrsten, 1964). Most functions begin to return to baseline after one to two hours (descending limb) and may be completely back to "normal" while the blood alcohol level is still relatively high on the descending limb.<sup>1</sup>

Eggleton (1941) presented evidence that the direction of change of blood alcohol level is important in determining an individual's performance on psychological tests. She reported that performance on the typewriter, dotting machine and distraction machine were more impaired if the blood alcohol level were increasing than if it were decreasing at the same blood alcohol level. However, her data were limited mainly to the performance changes of one subject over several months.

A lengthy monograph devoted to studying the effects of alcohol on sensory, motor and psychological functions was published by Goldberg (1943). He administered Swedish Potato brandy (40 vol. %) or about

<sup>&</sup>lt;sup>1</sup>Although performance may have returned to the baseline level, this does not mean that the subject on the descending limb of the blood alcohol curve is in the same psychological state as the non-alcoholized subject.

31.7 g of alcohol per 100 cc. This amounted to 0.63 to 1.42 g of absolute alcohol per kg body weight which was consumed within ten minutes. Blood samples were taken at 20 to 30 minute intervals during the postabsorptive period. Subjects were divided into abstainers, moderate drinkers and heavy drinkers. Sensory tests consisted of a flicker test and a corneal test; motor tests were the Romberg and the finger-finger test; psychological tests were a subtraction test and the Bourdon test (cancellation). The blood alcohol level for the appearance threshold (beginning of impairment) was lower than the blood alcohol level for disappearance threshold for the modified Romberg, the finger-finger test and the psychological tests. These findings indicate that psychological functions returned to baseline at a higher blood alcohol level than they first appeared to be impaired. However subjects were tested repeatedly on each test at least nine times over a nine-hour period. Although a placebo group was included for comparison, the repeated testing of the alcohol subjects may have resulted in their improved performance on the descending limb.

Gruner (1955) administered 1.2 g of absolute alcohol per kg body weight, diluted to 33%, to 10 subjects. Each subject consumed the beverage within 30 minutes. Blood samples were taken every hour. Performance was found to be impaired on the Bourdon test (measuring tenacity) and reaction times (measuring vigilance), particularly during the rise of the blood alcohol curve. The average total impairment of attention at a blood alcohol concentration of 0.10% was 75% during the absorption phase and 40% during the elimination phase.

In a recent study, Young (1970) gave 4 ml of alcohol per kg

body weight of whiskey (40% alcohol) in the ratio 85% whiskey and 15% water to 10 subjects. They consumed the beverage within 15 minutes. Reaction times and blood alcohol concentrations were measured 5, 10, 15 minutes, 3/4, 1-1/4, 2, 3, and 3-1/2 hours after drinking. Reaction times increased as blood alcohol concentration increased on the ascending limb. However, during the descending limb reaction times improved and were not related to blood alcohol concentration. Since testing was carried out at specific times after alcohol ingestion, comparisons in performance were not made at the same blood alcohol levels on the ascending and descending limb. A control period indicated little change in reaction time with repeated testing.

Sidell and Pless (1971) administered 0.5 to 2.0 ml of alcohol per kg body weight (0.4 to 1.6 g/kg) to 26 subjects in a 30% concentration in orange juice. The beverage was given in four divided doses 10 minutes apart over a 30-minute period. The tests consisted of an addition task (Number Facility Test), a time estimation task (VITA), and a perceptual motor task which required a high degree of eye-hand coordination (ZITA). Subjects were tested repeatedly for eight hours. The greatest performance decrements occurred on the ascending limb. While cognitive ability returned rather quickly on the descending limb, tests requiring hand-eye coordination remained impaired for some time on the descending limb.

In a recent study, Jones and Vega (1972) administered 1.32 ml of alcohol per kg of body weight to 20 subjects and a placebo to 20 additional subjects. Blood alcohol level was assessed by breath samples. Ten alcohol subjects were tested at 0.08% on the ascending limb and 10

subjects were tested at 0.08% on the descending limb; placebo subjects were tested at comparable times as alcohol subjects. Using independent groups comprised of subjects tested only once at a specific blood alcohol level, it was found that subjects tested on the ascending limb performed poorer than subjects tested on the descending limb on the Shipley-Hartford abstract scale. Effects of fatigue and practice were minimized in these results since independent groups were used.

In summary, most psychological functions tested appear to be more disrupted on the ascending than on the descending limb of the blood alcohol curve. Although there are many additional reports concerning the effects of alcohol on behavior, it is difficult to assess their contribution to this issue since results are not reported with reference to the limb of the blood alcohol curve. It would be important to extend these findings to determine if memory functions are also less affected on the descending limb.

Effects of Alcohol on Human Memory - Literature Review

The early studies concerned with effects of alcohol were concerned mainly with learning and not memory <u>per se</u>. Mead (1939) reviewed eight studies, six of which were German, that reported an effect of alcohol on learning. Impairment was reported for learning numbers (Kraeplin, 1892), nonsense syllables (Kurka, 1928; Gylys, 1925; Hahn, 1926), and rote material (Rudin, 1904) as well as decreased ability to memorize (Kruz and Kraeplin, 1901) and reproduce syllogistic material (Seward, 1936). No effect of alcohol was found for the ability to learn words when presented one letter at a time backwards (Dodge and Benedict, 1915). Memory, a primary component of all of these tasks, may have been

the primary variable responsible for the reported learning impairment.

Ekman et al. (1964) studied short-term and intermediate-term memory after administering three doses of alcohol (0.96, 1.28, and 1.92 ml per kg body weight) to each of eight male medical students on different days. Subjects drank the whiskey in 10 to 15 minutes. Blood samples were taken from the finger tip at 10, 40, 65, 95, 140, 190, 240 and 290 minutes after drinking was begun. Mean maximum blood alcohol levels, as analyzed by the Widmark micromethod, were 0.35%, 0.45% and 0.72% for the three doses. The memory test involved presenting a list of eight pairs of nonsense syllables to the subject for 30 seconds. Memory was tested immediately (short-term memory) as well as seven minutes later (intermediate-term memory) by presenting a new list with the second member of each pair matched with four nonsense syllables. The subject was required to recognize the correct pair. This entire procedure was repeated with different lists eight times, once before alcohol and preceding each of the blood samples. Therefore testing was carried out on both the ascending and descending limbs since the study lasted about 5 hours. The results indicated that the effect of alcohol on performance was small, probably because the recognition task was fairly simple. The investigators report a "slight impairment" was found after the two large doses (0.045% and 0.072%) while an increase in performance occurred at the small dose (0.035%). However no statistical tests were reported, and the results were not presented in relation to the ascending and descending limb. These findings suggest that alcohol does not impair short-term and intermediate-term memory as measured by the recognition method at blood alcohol levels below 0.072%. The low blood alcohol levels together

with the repeated testing and simplicity of the recognition task all may have contributed to the unimpaired performance.

In a well-designed study, Carpenter and Ross (1965) evaluated the effect of alcohol on short-term memory by giving four doses of alcohol (0.0, 0.33, 0.67, 1.00 ml of absolute alcohol per kg body weight) in the form of 86.7 proof whiskey, with carbonated water, equal to half the volume of whiskey. Sixteen subjects consumed the drink within 15 minutes, and testing was begun 15 minutes later. Breath samples were taken at 10 minute intervals after drinking. The design consisted of four identical 4 X 4 latin squares with three pre-drinking and seven postdrinking subplots. The subjects performed at a running matching memory task. The numeral 1 or 2 appeared in the left tube and the symbol + or - appeared in the right tube; the subject reported whether each numeral was the same or different from the one immediately preceding it. For the right hand tube, the subject reported whether each symbol was the same or different from the one 2 symbols back. The subject performed a 1-back and a 2-back match for every stimulus pair. A linear relationship was found between dose and blood alcohol level. Performance appeared to improve at blood alcohol levels of 0.024% to 0.055%, with obvious deterioration not occurring until blood alcohol levels of 0.070% or higher. This is consistent with the previous study that found facilitation at 0.035% and no impairment below 0.072%. It also appeared that the major effect of alcohol was on 2-back and not 1-back performance, indicating a greater impairment on the more complex task. When subjects were ranked on predrinking proficiency, it was found that the best subjects (rank 1) responded to doses differently than ranks 2, 3, and 4. Since subjects

were tested for 70 minutes post-drinking, the majority of the testing was probably carried out on the ascending limb, especially for the highest dose. However subjects who were tested at lower doses may have reached peak rather quickly and started descending before testing was completed. This may explain the apparently improved performance at the two low doses.

Ryback et al. (1970) measured both short-term and intermediateterm memory after administering 1.2 cc/kg of body weight of 95% ethanol diluted with 300 ml of orange drink to 10 subjects; an equal volume of orange juice with 1 cc of 95% ethanol was given as a placebo to four control subjects. The alcohol subjects reached a mean peak of 0.103%. A series of colored pictures was presented on 15 mm film to the subjects (age 21-29) and after every two inspection pictures two recognition pictures were presented, one which had been presented earlier. The subject's task was to identify the "old" picture. This task is similar to the one reported earlier (Ekman et al., 1964) since a recognition task was used. Recognition of the initial picture was tested after 24.5, 48.5, 96.5, or 192.5 items had appeared between it and the recognition pictures. A practice period preceded the drinking period in order to establish baseline performance. Breath samples were obtained from the subjects after 30 to 45 minutes of testing. The entire experiment lasted 90 minutes. Results indicated that over a 45 minute period recognition performance declined for the alcohol group, but not the placebo group. This decline was apparently due to the increasing blood alcohol level from 30 minutes (0.079%) to 45 minutes (0.103%) since absolute blood alcohol level was not related to performance. However both of the

blood alcohol levels are higher than those reported where no impairment was found. It also was found that recognition was much poorer in the alcohol group for pictures that had been presented 192.5 items or 9 minutes earlier (intermediate-term memory) than for items presented 24.5 (1 minute) or 96.5 items (4 minutes) earlier (short-term memory). Thus, short-term and intermediate-term memory for recognition of pictures was impaired in this study when presented and measured on the ascending limb at blood alcohol levels of 0.079% or higher. It appears that memory impairment, at least for recognition tasks, does not occur until a blood alcohol level of 0.07% or higher is reached. This is in contrast to an earlier study (Goodwin <u>et al</u>., 1969) where pictures were presented under alcohol on day 1 at blood alcohol levels from 0.08% to 0.14% and subjects were tested for recognition on day 2; in this case testing on both occasions occurred about 1 hour 40 minutes after beginning to drink, probably on the descending limb, and no memory impairment was found.

Several studies have used digit span to study short-term memory following alcohol ingestion. Tarter <u>et al</u>. (1971) administered 1.10 ml of 95% ethanol per kg body weight diluted with four parts orange juice to 26 male medical students. Thirteen subjects were tested under alcohol the first day and placebo the second day and 13 subjects had the reverse order of testing. Testing was begun 30 minutes after consuming the drink when the mean blood alcohol level was 0.08%. Short-term memory was measured by digit span and dichotic listening. The results indicated that memory for digits forward was impaired following alcohol. Also, it was found that first ear recall for digits presented dichotically was poorer for the alcohol group at six digits while second ear recall was poorer

for the alcohol group only for recall of three digits, with both groups performing at chance level on 4, 5, and 6 digits from second ear. This finding that short-term memory for digits was impaired following a dose of alcohol resulting in a blood alcohol level of 0.08% is again consistent with previous reports that impairment is found above 0.07%. It is difficult to determine whether subjects were tested on the ascending or descending limb. Since digit span was given early, the subject may have been on the ascending limb. However the dichotic stimulation task was given last when blood alcohol levels were probably decreasing. This may be one reason why more striking changes were not noted.

Hutchison, Tuchtie, Gray and Steinberg (1964) investigated digit span after administering two fluid ounces of whiskey to eight hospital patients after they had completed a test battery without alcohol. When breathalyzer readings reached 0.10%, the second test battery was administered. Subjects returned in 24 hours for the retention test. Results indicated the digit span backward, but not forward, was reduced following alcohol. This finding is not consistent with the Tarter et al. (1971) study where digits forward was impaired, but the discrepancy may have been due to the practice subjects received before alcohol. Learning of noun paired associates also was impaired under alcohol. This result is not consistent with the Ekman et al. (1964) study but may be due to the different blood alcohol levels used (0.10% as compared to 0.072%). Further, recall methods differed: the anticipation method was employed in the Hutchinson et al. (1964) study as opposed to the recognition method in the Ekman et al. (1964) study. Long-term memory was measured the next day in the Hutchinson et al. (1964) study by asking subjects to

recall TAT cards seen the previous day as well as the title given to the card. Although the recognition of cards was not impaired, the recall of the titles was impaired. This again suggests alcohol does not interfere with recognition as much as with recall. It appears from the data presented concerning blood alcohol levels that there was not much change in the 20 minutes from beginning of testing to end of testing: two increased, two remained the same, two decreased and two did not have a second reading. However the blood alcohol level was rising from 0.15% to 0.20%, a much higher level than used in the previous studies.

Kalin (1964) also studied long-term memory by administering alcohol at a "stag party" by giving cocktails that each contained two shots (3 ounces, 86 proof beverage) or soft drinks to the placebo group. A record was kept of the number of drinks consumed in a 25 minute period; however this amount was not reported or were blood alcohol levels reported. Subjects wrote a set of stories to four different TAT pictures on three occasions: before alcohol, 25 minutes after beginning to drink, and after a second 25 minute period. Recall of the stories both before and after seeing the pictures the next day was evaluated in a non-alcohol condition for about half the subjects in each group. Recall the second day was impaired for both alcohol testing sessions as compared to the placebo group, with the second alcohol session being the poorest. This was apparently due to a greater amount of alcohol being consumed by the second session (13.83 ounces) as compared to the first (7.17 ounces). Exactness of recall was poorer mainly for recall in the second alcohol session. The mean number of TAT pictures recalled also decreased as more alcohol was consumed. A significant positive correlation was re-

ported between amount of alcohol consumed and recall of stories written before alcohol on the next day. These data are consistent with the previous study that found alcohol subjects impaired in ability to remember titles to TAT cards. Since all subjects were apparently drinking continuously, testing was probably carried out during the ascending limb. Memory for stories the next day with alcohol was related to amount of alcohol consumed and probably to blood alcohol level, although the latter was not measured.

### Critique and Evaluation

These studies demonstrate that short-term, intermediate-term and long-term memory are impaired by alcohol. However, memory impairment may not be detected until the blood alcohol level is above 0.07%. Although most studies do not report the limb of the blood alcohol curve that the subject is on at the time of testing, two studies report that memory is impaired on the ascending limb (Ryback <u>et al</u>., 1970; Goodwin <u>et al</u>., 1970) while there are no studies that explicitly deal with the descending limb. It may be that memory, similar to other cognitive functions, is more impaired on the ascending than the descending limb at comparable blood alcohol levels (Jones and Vega, 1972).

Another factor that needs to be considered in the evaluation of the effect of alcohol on memory, in addition to alcohol variables, is the characteristic of the memory task. Most memory tasks used in alcohol studies have involved non-verbal stimuli such as pictures, symbols, or digits; the subject is usually requested to identify or recognize material he has seen at an earlier time. Since recognition of material is not as difficult as recall, it is not surprising that small differences

are frequently reported between alcohol and placebo subjects for recognition tasks. A more rigorous test of memory would involve recall of previously presented material. However, there is some difficulty in developing recall tests for non-verbal information. One method of overcoming this difficulty would be to present verbal information and ask for verbal recall. This would serve two purposes. First, recall could be tested as opposed to recognition. Second, the effects on alcohol on short-term memory on non-verbal tasks could be extended to include verbal material.

There are several types of tasks that could be used. Peterson and Peterson (1959) have used a distractor task where trigrams are followed by a delay task. However the main variable is the effect of various delay tasks on recall of the trigrams. Although this task has been widely used, it measures the effect of distraction on memory rather than merely the ability to recall the material and therefore is not the most appropriate task to use to measure the effects of alcohol on short-term memory.

Probe tasks have also been used to evaluate memory for items imbedded within a list. For example, sequential probe tasks require that the subject recall an item that appeared immediately before or after a given item in a list. Position probe tasks ask the subject to recall the ordinal position of an item. The most common task involves paired associates where the subject is given a sequence of paired items (Murdock, 1963). Later he is then given the first item and asked to recall the second item. These tasks require that the subject "associate" pairs of words or remember the relation of one word to another or to a

given position. These tasks would not be the most appropriate since they are concerned more with relations between words than with memory per se.

The purest and simplest type of memory task is the serial recall task. This involves merely presenting a list of words and asking for recall at the end of the list. This can be done in two ways: fixed recall and free recall. The fixed order recall is similar to the probe tasks in that the subject must remember the relationship of one word to another. However the free recall task only requires that the subject remember the items presented without regard to order. This task then provides one of the clearest measures of verbal recall. It also has been extensively used by a variety of investigators and has been incorporated into a model of short-term memory (Glanzer, 1971). In a series of articles, Glanzer (1967, 1969, 1971) has described this free recall verbal task and has postulated this task can be used to demonstrate a two-storage memory system within this short-term memory task. He hypothesized and provides evidence that the last few items in a list go into short-term storage (STS) and the first few items go into long-term storage (LTS). This STS is described as "robust" (Glanzer, 1971), in that very few experimental manipulations can affect it. However LTS is sensitive to both subject variables (age and intelligence) as well as to experimental manipulations such as list length, rate of presentation, and mnemonic structure.

The effect of alcohol on memory needs to be clarified in several ways. First, a task should be used that can detect short-term memory changes as well as intermediate-term and long-term memory changes. The free recall task described above can be used in its traditional manner to evaluate short-term memory and can be extended to evaluate inter-

mediate-term and long-term memory by asking for recall at later times. Second, blood alcohol levels should be carefully controlled so that all subjects are tested at the same level, about 0.08% to 0.09%, since most studies do not report memory deficits until these levels are reached. Third, the limb of the blood alcohol curve should be controlled by testing subjects on both the ascending and descending limb to determine if there is a differential memory impairment as a function of the limb as there is for other cognitive functions. These three variables must be considered in interpreting the effect of alcohol on memory.

### Statement of the Problem

The effects of alcohol upon memory as a function of ascending and descending limb blood alcohol levels is an important problem area. The present investigation uses three approaches to study the effect of alcohol on memory: an empirical study, the application of these data to a model, and predictions derived from an extention of the model.

The empirical study investigates the effects of alcohol on short-term memory during the ascending and descending limb of the blood alcohol curve. From the review of the literature, it appears that memory for non-verbal material is impaired following alcohol ingestion. Although memory impairment also has been reported for verbal material, a more systematic and rigorous approach is needed. Using a well documented free recall task, the Glanzer technique, it is hypothesized that:

 Short-term memory for verbal material in a free recall task is impaired following ingestion of a moderate dose of alcohol.

However, it is evident from the literature that cognitive differences may also be a function of on which limb of the blood alcohol

curve the behavior is sampled. Although cognitive functions are generally impaired on the ascending limb, there is evidence to suggest that they may return to baseline rather quickly on the descending limb. Therefore the above hypothesis may be expressed more specifically with regard to the limb of the blood alcohol curve at comparable blood alcohol levels (0.09%):

la) Short-term memory for verbal material in a free recall task is impaired on the ascending limb of the blood alcohol curve at a blood alcohol level of 0.09%.

lb) Short-term memory for verbal material in a free recall task is impaired on the descending limb of the blood alcohol curve at a blood alcohol level of 0.09%.

1c) Short-term memory for verbal material in a free recall task is more impaired on the ascending than on the descending limb of the blood alcohol curve at a blood alcohol level of 0.09%.

The second approach is the application of the empirical data to a theoretical model. Glanzer, using the free recall task, has divided short-term memory into short-term storage (STS) and long-term storage (LTS). Operationally defined, short-term storage consists of the last three to four items in a list while long-term storage refers to the first three to four items. The STS is described as "robust" in that very few experimental manipulations affect it. However, LTS is sensitive to both subject variables (age and intelligence) as well as to experimental manipulations such as list length, rate of presentation and mnemonic structure. A recent study has also indicated that one drug, marihuana, interferes with LTS (Abel, 1971). It would appear, using Glanzer's model and

techniques, that if there is an affect of alcohol on memory, then LTS would be impaired. This could be directly tested by plotting serial position curves and leads to the following testable hypothesis directly derived from Glanzer's model, given a deficit in short-term memory:

2) Long-term storage is more impaired than short-term storage in a free recall task following ingestion of a moderate amount of alcohol.

The third approach to the study of the effects of alcohol on memory involves extending Glanzer's model. Although Glanzer reports data only for short-term memory, it is also possible to test for intermediate-term and long-term memory for the items presented in the shortterm memory task. Intermediate-term memory could be tested by asking for recall of several lists after each list had been recalled individually in the short-term memory task. This would give an estimate of intermediate-term memory, i.e., memory for words up to 15 minutes prior to recall.<sup>2</sup> Since long-term storage (LTS) is hypothesized to be impaired during the short-term memory task, then intermediate-term memory should be impaired following alcohol ingestion. The following prediction is made from extending Glanzer's model:

3) Intermediate-term memory for verbal material in a free recall task is impaired following a moderate dose of alcohol.

Long-term memory is expected to be impaired if short-term and intermediate-term memory are impaired. Long-term memory in this case refers to recall of items that have been presented 15 minutes or more

 $<sup>^2</sup>$ This is somewhat different from traditional intermediate-term memory since the subject has already recalled the items once at the end of each list.

earlier. The following prediction is set forth:

4) Long-term memory for verbal material in a free recall task is impaired following a moderate dose of alcohol.

However, this prediction can be more specifically expressed with regard to the state in which long-term recall is asked. That is, recall could be obtained while the subject is still in the alcohol state but an hour or more after seeing the material. Assuming the subject is not able to recall items earlier in the alcohol state, the following prediction is made:

4a) Long-term recall for verbal material in a free recall task is impaired following a moderate dose of alcohol when tested one hour later in the alcohol state.

Long-term memory was also tested 23 hours later during a nonalcohol state. The following prediction was made:

4b) Long-term memory for verbal material in a free recall task is impaired following a moderate dose of alcohol 23 hours later during a non-alcohol state.

A recognition task was also given 23 hours later. The following prediction was made:

5) Recognition of items presented during an alcohol state is impaired 23 hours later during a non-alcohol state.

In summary, the empirical data evaluate the hypothesis that alcohol impairs short-term memory, and that memory impairment is greater on the ascending than on the descending limb of the blood alcohol curve. Applying these data to Glanzer's model of short-term memory, it is hypothesized that the impairment of short-term memory is due primarily to

### CHAPTER II

#### METHOD

### Subjects

Forty paid volunteer medical students ranging in age from 21 to 31 years served as subjects. All subjects were instructed to obtain a regular night's sleep before the testing day and not to drink alcoholic beverages or take medication the night prior to testing or on the day of testing. Subjects were asked to eat a light breakfast on the day of testing and not to eat, drink, or smoke from that time until reporting to the laboratory.

All subjects were tested from 1 to 5 P.M. Each subject was randomly assigned to an alcohol or placebo group with 20 subjects in the alcohol group and 20 subjects in the placebo group. The alcohol group was tested on the ascending limb  $(A_1)$  and on the descending limb  $(A_2)$  of the blood alcohol curve at blood alcohol levels of 0.09%. Since  $A_1$  testing occurred earlier in the evening than  $A_2$  testing, the placebo group was also tested early  $(A_1)$  and late  $(A_2)$  to control for temporal and practice effects. Each subject was asked to return the next day at 12 noon for a 30 minute follow-up. Recall and recognition for all words were tested at this time.

The alcohol and placebo groups did not differ significantly on

age, education or weight as described in the Appendix A.

### Procedure

The subject was given instructions on the procedure for giving breath samples using the Breathalyzer (Stephenson, Model 900). Subjects in both the alcohol and placebo groups were told that repeated breath samples would be required during the afternoon and were told that they would be asked to record test instructions on a tape recorder.

#### Alcohol Concentration

The alcohol subjects received 1.32 milliliters of 95% USP ethanol per kilogram of body weight. The alcohol was mixed with orange drink in the ratio of four parts of orange drink to one part of alcohol. A previous study in our laboratories showed this dose produces a peak blood alcohol level of about 0.11% (Jones and Vega, 1972). The placebo subjects received the same volume drink with only four milliliters of alcohol floated on top of the orange drink. This procedure proved effective in an earlier study in convincing placebo subjects that they had received alcohol (Jones and Vega, 1972). In the present study, subjects were also told that they would receive one of several doses of alcohol. These additional instructions, together with the taste of alcohol and the repeated breath samples as described below, served to convince all subjects that they had received a moderate dose of alcohol. The subjects began to drink about 2:00 P.M. and were asked to consume the beverage in fifteen minutes. Drinking time was controlled by giving one/third the total amount every five minutes.

### Breathalyzer Samples

Immediately after the subject finished drinking he was asked to rinse his mouth with water to clear it of residual alcohol. Breath samples were taken continuously throughout the afternoon at 5 to 10 minute intervals. The first two breath readings were usually invalid. Valid breath readings were obtained about 15 minutes after the subject finished drinking. Breath samples were obtained before and after each test for all subjects.

Blood alcohol levels were recorded for each subject. Testing was begun when subjects reached a blood alcohol level of 0.08% on the ascending limb of the blood alcohol curve. This usually occurred about 30 to 45 minutes after they began to drink. In order to control for temporal effects, placebo subjects were tested at a comparable time as the A<sub>1</sub> testing for the alcohol subjects. Repeated breath samples were taken for alcohol subjects and when they reached a peak (about 0.11%) and started descending, testing was begun at 0.09%. Therefore testing during the A<sub>1</sub> and A<sub>2</sub> periods was carried out in the same blood alcohol level range. Subjects in the placebo group were also tested at the same time as A<sub>2</sub> testing, about two hours after beginning to drink. The mean blood alcohol levels for the short-term memory task were 0.09% on the ascending limb and 0.09% on the descending limb. The four milliliters of alcohol given to subjects as a placebo failed to register a reading on the Breathalyzer.

### Baseline Measures (Pre-alcohol)

All subjects were administered the Shipley Institute of Living Scale to obtain baseline estimates of intellectual functioning. The com-

bined vocabulary and abstraction score was converted to WAIS-equivalent IQ score (Paulson and Lin, 1970). Subjects were also given the Eysenck Personality Inventory (Form A) to evaluate baseline levels of extraversion and neuroticism. These results provided information concerning the comparability of subjects on personality variables which conceivably could interact with alcohol and account for possible differences. There were no significant differences between groups for the Shipley or Eysenck scores as presented in the Appendix A.

All subjects were tested on the Verbal Memory Test (VMT) to obtain baseline scores prior to alcohol intoxication. The task was a free recall verbal memory test described by Glanzer (1969). A pilot study indicated that a population of students similar to the present sample performed comparably to other reports. Each S was shown 18 lists consisting of 12 successive words. The words were monosyllabic high frequency nouns drawn from the Thorndike-Lorge (1944) AA lists. The words were assigned and ordered into lists at random. Six lists were presented before drinking to obtain baseline performance for each subject. Six different lists were then presented on the ascending limb and six additional different lists on the descending limb of the blood alcohol curve. Placebo subjects also received six lists at the three time periods similar to the alcohol subjects. Each word was printed in black on a white background, one word on a slide. The words were shown on a screen with a 35 mm Carousel projector. Following Glanzer's study (1969) each slide was on for one second with a 1.1 second interval between slides. The screen was approximately 6 feet from the subject. Total time for each list was 24 seconds.

Each subject was run individually. The subject read each list of words aloud over a tape recorder and started writing the words immediately after the termination of the last word. Each subject was allowed one minute to write his responses. The next list was presented 30 seconds after responses to the previous list were obtained. After recall of the sixth list, each subject was given five minutes to recall as many of the 72 words as he could remember. Recall was tested for the control period during the control period, on the ascending and descending limb, and 23 hours later. Recall for words presented on the ascending limb was tested on the ascending limb as well as on the descending limb was tested on the descending limb as well as 23 hours later.

### Measures Administered After Alcohol

Subjects were tested twice on the Verbal Memory Test and the Eysenck Personality Inventory after consuming the drink. Reasons for readministering the Eysenck Personality Inventory will be given later. Each subject receiving alcohol was tested on the ascending limb (0.09%) and also on the descending limb (0.09%). Placebo subjects were tested at comparable times as alcohol subjects. Subjects were tested first on the ascending limb for recall of words they had seen during the control period. They were then tested on six different lists of words. Recall for all 72 words was given immediately following recall of the sixth list. Subjects were then given Form B of the Eysenck Personality Inventory.

Subjects were first asked to recall words from the control period and ascending limb period when they reached 0.09% on the descending limb. They were then tested on 6 different lists, and recall for

all of these words was obtained. Subjects were then given Form A of the Eysenck Personality Inventory. A 23-hour recall and recognition period for all words was also obtained.

Although subjects were not told they would have to recall words at a later time, it was possible that rehearsal could take place during two unfilled intervals. One interval (about 10 minutes) existed between the end of drinking and testing of short-term memory on the ascending limb. The other interval (about 60 minutes) was between testing of short-term memory on the ascending and descending limb. It was decided to fill these two time intervals with two tasks that would prevent rehearsal and also would not be likely to interfere with verbal recall. The Eysenck Personality Inventory and the Advanced Raven's Progressive Matrices (1962) were chosen to fill these intervals.

These two particular tasks were chosen since an earlier study had indicated that they might be sensitive to the direction of change of the blood alcohol level (Jones and Vega, 1972). The Eysenck Personality Inventory was included for another aspect of the study that involved looking at changes in personality occurring as a function of the ascending and descending limb. The Raven's Progressive Matrices was given for two reasons. First, the Raven's consists of two parts: Set I (5 minute time limit) and Set II (40 minute time limit). Set I was used to fill the interval on the ascending limb while Set II was used to fill the interval on the descending limb. A second reason for using the Raven's was that a previous study had indicated that performance might be impaired on the ascending limb but not on the descending limb (Jones and Vega, 1972). However, data from these two tasks will not be reported

here since the tasks were used mainly to fill the time intervals and were not related directly to the statement of the problem.

Subjects returned the next day at noon for a 23 hour recall and recognition task. Subjects were given 15 minutes to recall all the words from the previous day. They then were given a 15 minute recognition task that consisted of the 216 words, arranged in alphabetical order, that they had seen the previous day. However, subjects were told that the list consisted of words from the previous day in addition to other words. They were asked to cross out words that they had not seen the previous day. They were also asked to place a 1 (baseline), 2 (ascending limb) or 3 (descending limb) next to the words they did recognize, indicating the group from which the word came. Since the subjects had actually seen all of the words, the ones they omitted represented the number of words they actually denied seeing. The number of words correctly identified indicated the ability of the subjects to properly place the word in the correct group.

The following is a summary of the testing order and the approximate times of testing:

1-1

A.	Baseline (B)	Time
	1. Subject arrives at laboratory	1:00 p.m.
	2. Shipley Institute of Living Scale	1:20 p.m.
	3. Eysenck Personality Inventory (Form A)	1:40 p.m.
	4. Short-term Memory Task (B)	1:45 p.m.
	5. Intermediate-term memory task (B)	2:00 p.m.
в.	Ascending Limb (A <sub>l</sub> )	
	1. Begin drinking	2:10 p.m.

	2. Raven's Progressive Matrices - Set I	2:25 p.m.
	3. Recall Baseline words (B)	2:40 p.m.
	4. Short-term memory task (A <sub>l</sub> )	2:45 p.m.
	5. Intermediate-term memory task $(A_1)$	3:00 p.m.
	6. Eysenck Personality Inventory - Form B	3:05 p.m.
C.	Descending limb (A <sub>2</sub> )	
	1. Raven's Progressive Matrices - Set II	3:20 p.m.
	2. Recall Baseline (B) and Ascending Limb ( $A_1$ ) words	4:00 p.m.
	3. Short-term memory task (A <sub>2</sub> )	4:15 p.m.
	4. Intermediate-term memory task (A <sub>2</sub> )	4:35 p.m.
	5. Eysenck Personality Inventory (Form A)	4:45 p.m.
D.	Twenty-three hour follow-up (B <sub>2</sub> )	
· •	l. Recall words from B, $A_1$ and $A_2$	12:00 noon
	2. Recognition task for B, $A_1$ and $A_2$ words	12:15 p.m.

# Statistical Analysis

The basic data were analyzed by 2 (Group) X 3 (Testing Period) analyses of variance with repeated measures. The main effect of Testing Period (a repeated measure) was evaluated by using conservative degrees of freedom. Simple effects were evaluated by F-tests.

# CHAPTER III

# RESULTS

#### Short-term Memory

Means, standard deviations, and F-ratios for the short-term memory task are presented in Table 1. A significant main effect for Testing Period and a significant Groups (G) X Testing Period (T) interaction were obtained. Simple effects were analyzed since the G X T interaction was significant. Figure 1 illustrates the nature of the interac-The placebo group improved slightly but nonsignificantly over the tion. three testing periods. The alcohol group was comparable to the placebo group in baseline performance but showed a decline in performance from baseline on both the ascending and descending limb of the blood alcohol curve. Performance of the alcohol group on the ascending limb was signnificantly different from their own baseline performance (F = 56.23, p <.01) as well as from the second testing of the placebo group (F = 8.46, p < .01). They were also significantly different on the descending limb from their own baseline performance (F = 22.18, p < .01) as well as from the third testing of the placebo group (F = 4.42, p < .05). A comparison of performance of the alcohol group on the ascending and descending limb indicated that performance improved significantly from the ascending limb to the descending limb (F = 7.78, p < .01). These results sup-

			Testing	Period				F-ratios	
Group	В		Al		A <sub>2</sub>		Group	Testing	GXT
	Mean	S.D.	Mean	S.D.	Mean	S.D.	(G)	(T)	
Alcohol	43.45	8.82	35,25	7.84	38.30	8.80	2.33	10.75*	19.74*
Placebo	41.60	6.71	42.65	7.80	43.65	8.09			

MEANS AND STANDARD DEVIATIONS AND F-RATIOS FOR ALCOHOL AND PLACEBO GROUPS ON THE SHORT-TERM MEMORY TASK

TABLE 1

\*p < .01

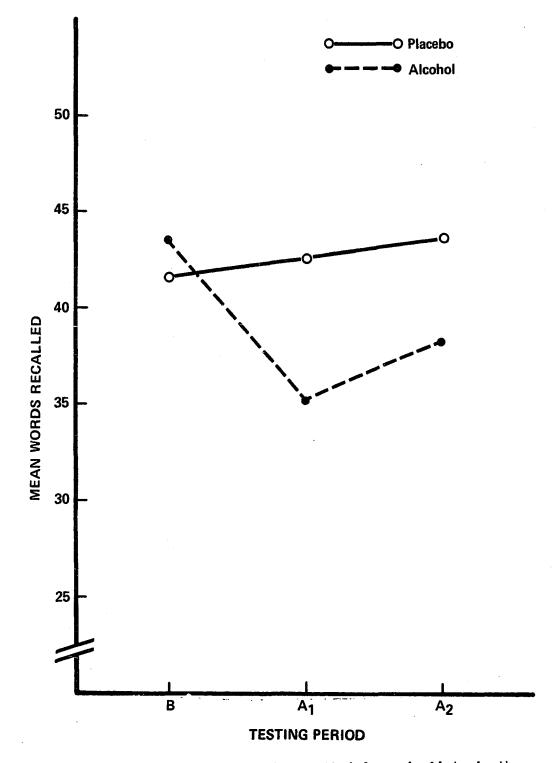


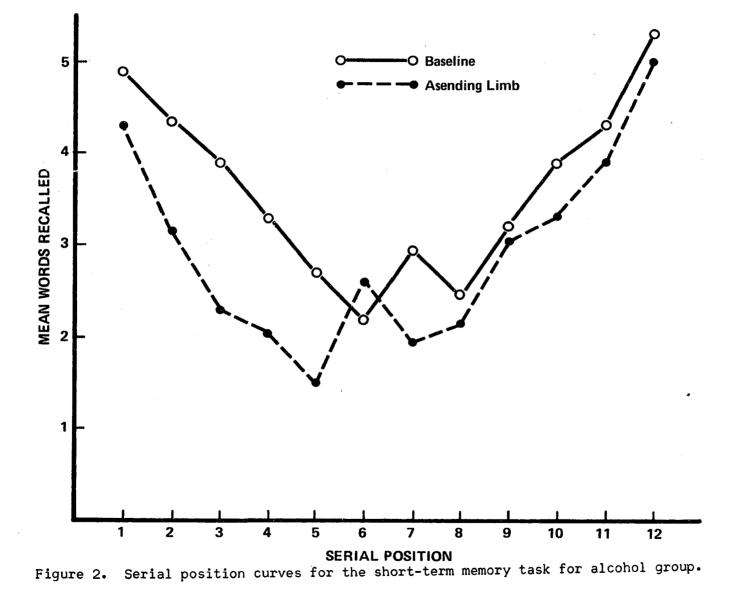
Figure 1. Mean total words recalled from six lists in the short-term memory task.

port the hypothesis (hypothesis 1) that alcohol impairs short-term memory in a free recall task, both on the ascending (la) and descending (lb) limb. The hypothesis (lc) that short-term memory is more impaired on the ascending than on the descending limb is also supported.

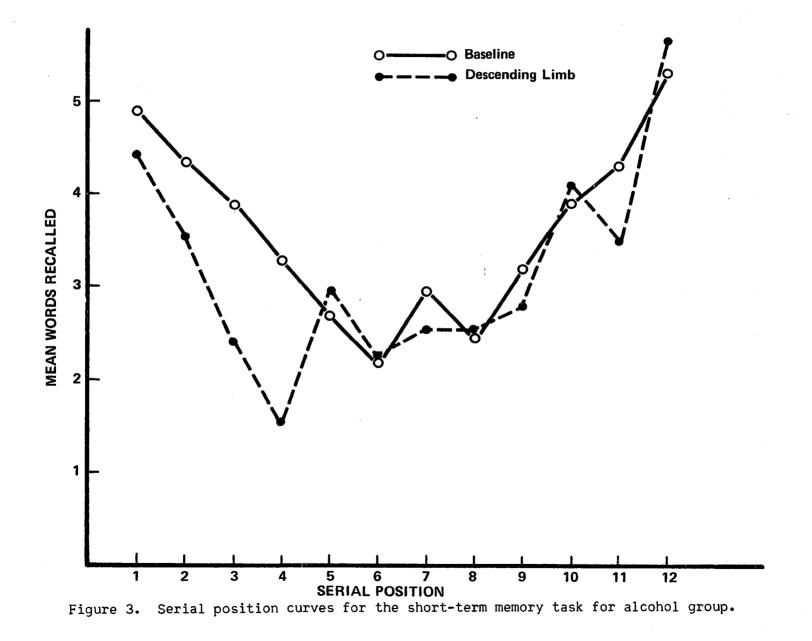
# Serial Position Curves for Short-term Memory

Serial position curves for the ascending and descending limbs for the alcohol group are presented in Figures 2 and 3. It is apparent that recall of the words in the first positions is affected on both the ascending (Figure 2) and descending (Figure 3) limbs. These differences are more clearly illustrated in Figure 4 which compares the first four, the middle four and last four positions for the three testing periods in the alcohol group. Both the ascending (t = 5.48, p < .01) and descending limb (t = 4.55, p < .01) performance differed significantly from baseline for the first four positions (hypothesis 2). However, there was no significant difference between the ascending and descending limb performance for these first four positions. Performance for the middle four positions on the ascending limb was also significantly different from baseline (t = 2.32, p < .05) while there was no difference between baseline and descending limb for these middle four positions. There were no significant differences among the testing periods for the last four items. A comparison between the ascending and descending limb performance indicated that recall of the middle four items was significantly poorer for the ascending than for the descending limb (t = 3.16, p < .05).

These results support the hypothesis that impaired short-term memory following alcohol ingestion is due to the poor recall of the first



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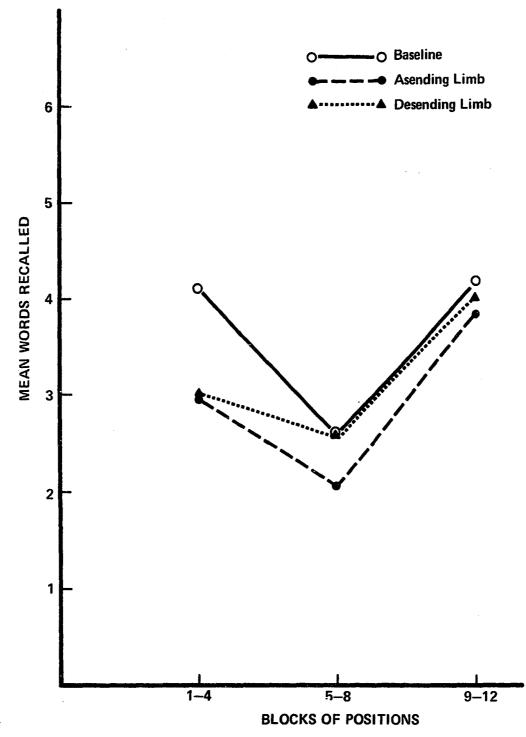


Figure 4. Mean words recalled per block of positions for the alcohol group.

four items in the free recall list, i.e., those items which Glanzer believes go into LTS. This impairment is observed on both the ascending and descending limbs. However, the difference between the ascending and descending limb is due to the poor recall of the middle four items on the ascending limb.

#### Intermediate-term Memory

Means, standard deviations, and F-ratios for intermediate-term memory are presented in Table 2. Significant main effects were obtained for Group (F = 23.47, p < .01) and Testing Period (F = 13.30, p < .01) as well as a significant Group X Testing Period interaction (F = 16.17, p < .01). The placebo group improved slightly but nonsignificantly across testing periods. Figure 5 illustrates the impaired performance of the alcohol group on both the ascending and descending limb of the blood alcohol curve. Simple effects indicated that performance of the alcohol group on the ascending limb was significantly different from baseline performance (F = 46.22, p < .01) as well as from the second testing of the placebo group (F = 27.93, p < .01). The same effect was observed on the descending limb where performance was impaired as compared to both baseline (F = 41.02, p < .01) and the third testing of the placebo group (F = 30.96, p < .01). However, there was no significant difference in intermediate-term memory between the ascending and descending limb performance. These results indicate that alcohol impairs intermediate-term memory on both the ascending and descending [imb.

# Serial Positions Curves for Intermediate-term Memory

Serial position curves for baseline and the ascending limb of

MEANS AND	STANDARD D	DEVIATIONS	AND F-RATIOS	FOR ALCOHOL	AND PLACEBO
	GROUPS ON	I THE INTE	RMEDIATE-TERM	MEMORY TASK	

			Testing	Period				F-ratios	
Group	В		A	1	F	2	Group	Testing	GXT
	Mean	S.D.	Mean	S.D.	Mean	S.D.	(G)	(T)	
Alcohol	20.50	7.82	6.70	4.57	7.50	4.65	23.47*	13.30*	16.17*
Placebo	19.00	5.85	19.00	9.27	20.45	10.07			

\*p < .01

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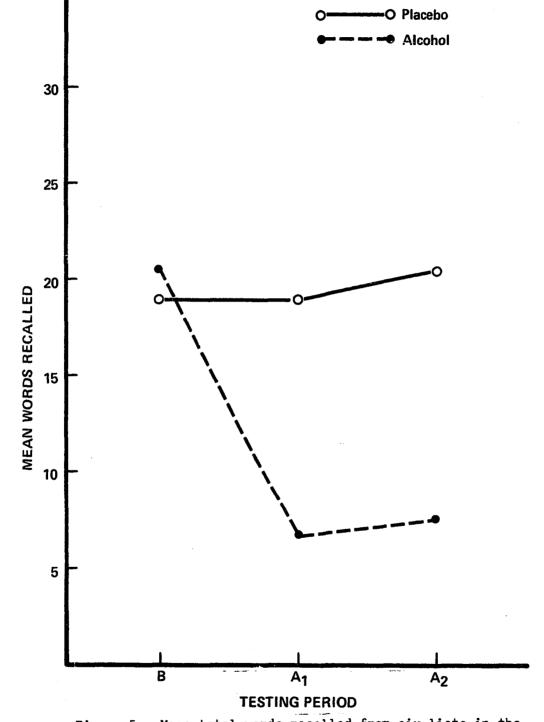
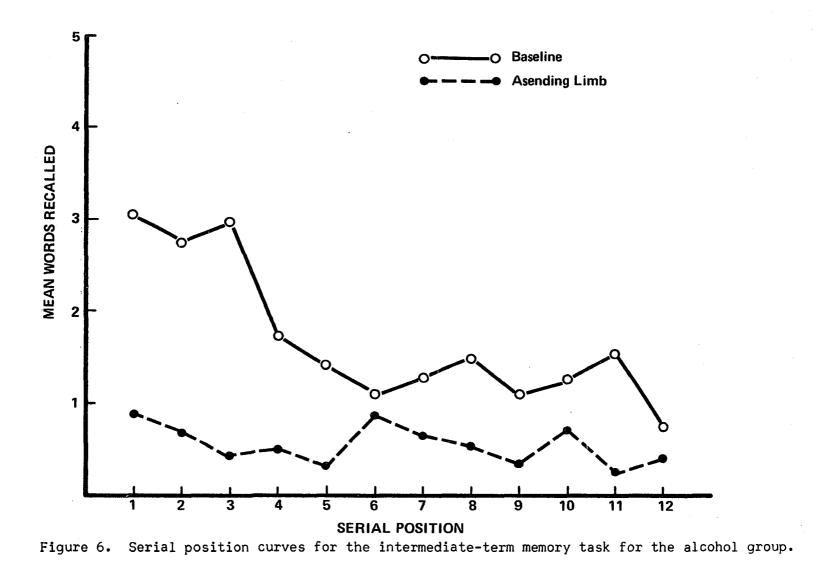


Figure 5. Mean total words recalled from six lists in the intermediate-term memory task.

the blood alcohol curve for the alcohol group are presented in Figure 6. Recall of items from the first four positions of each list are significantly impaired on the ascending limb (t = 10.67, p < .01). This is also the case for the descending limb (t = 8.10, p < .01). No significant differences for middle or last four positions were found. Alcohol apparently interferes with recall of the early items in each list but not with the last items.

# Long-term Memory

Means, standard deviations, and F-ratios for long-term memory are presented in Table 3. Significant main effects were obtained for Group (F = 13.95, p < .01) and Testing Period (F = 25.79, p < .01) as well as a significant Group X Testing Period interaction (F = 23.25, p < .01). There were no significant differences in long-term memory for the three periods in the placebo group. Figure 7 illustrates the impaired performance of the alcohol group on both the ascending and descending limb of the blood alcohol curve. Simple effects indicated that performance of the alcohol group on the ascending limb was significantly different from baseline performance (F = 61.53, p < .01) as well as from the placebo group (F = 28.13, p < .01). The same effect was observed on the descending limb where performance was impaired as compared to both baseline (F = 79.18, p < .01) and the placebo group (F = 22.28, p < .01). However, there was no significant difference in long-term memory between the ascending and descending limb performance. These results are similar to intermediate-term memory where there is an overall effect of alcohol on both the ascending and descending limb with no difference between the ascending and descending limb.



			Testing	Period				F-ratios	
Group	В		A	1	A	2	Group	Testing	GXT
	Mean	S.D.	Mean	S.D.	Mean	s.D.	(G)	(T)	
Alcohol	16.90	6.84	5.00	4.10	3.40	3.22	13.95*	25 <b>.</b> 79 <sup>*</sup>	23.25*
Placebo	15.00	5.65	16.35	8.42	13.50	9.79			

MEANS AND STANDARD DEVIATIONS AND F-RATIOS FOR ALCOHOL AND PLACEBO GROUPS ON THE LONG-TERM MEMORY TASK

TABLE 3

\*p < .01

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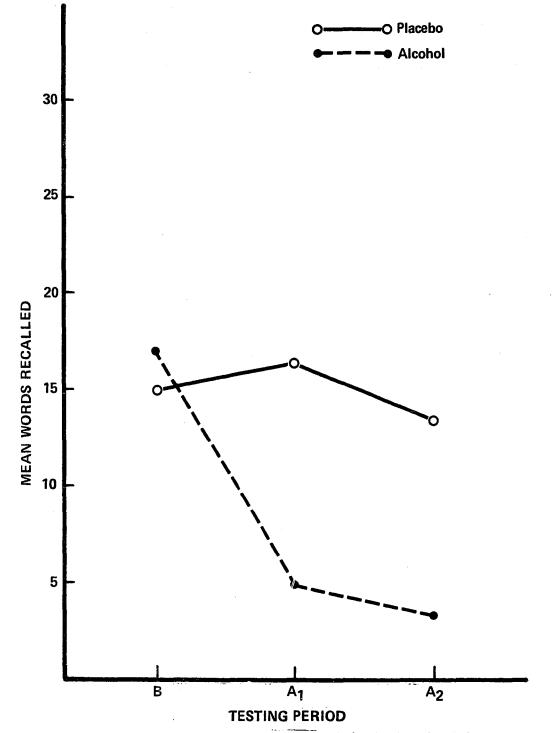


Figure 7. Mean total words recalled from six lists in the long-term memory task.

# Recall of Baseline Words on the Ascending and Descending Limb

Figure 8 illustrates the recall of baseline items (B) presented before alcohol (intermediate-term memory) and on the ascending  $(A_1)$  and descending  $(A_2)$  limb. There were no significant differences between the alcohol and placebo groups. It is clear that alcohol subjects are able to recall these baseline words adequately at a blood alcohol level of 0.09% on both the ascending and descending limb. It would appear that retrieval mechanisms are intact, at least for words seen prior to alcohol.

#### Recall of Alcohol Words During Alcohol

Figure 9 illustrates the recall of words originally presented on the ascending limb  $(A_1)$  during the ascending limb (intermediate-term memory) and the descending limb  $(A_2)$ . Although there is a significant drug effect (F = 35.05, p < .01), there is no significant testing period main effect or a significant interaction. These results indicate that there is little change in recall of words from the intermediate-term memory task on the ascending limb to recall on the descending limb.

#### Short-term Memory Impairment: Errors of "Commission"

Subjects wrote down incorrect words as well as correct words during the short-term memory task. These incorrect words were classified into four categories to determine if alcohol also affected the type or number of incorrect words. The four types of incorrect words along with the total number of incorrect words in each testing period are presented in Table 4. Means, standard deviations, and F-ratios for the in-

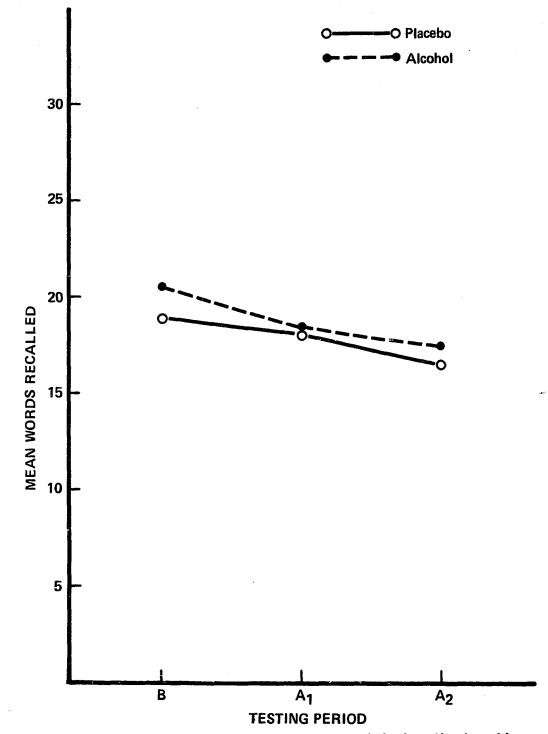


Figure 8. Mean baseline words recalled during the baseline period and on the ascending and descending limb in the intermediate-term memory task.

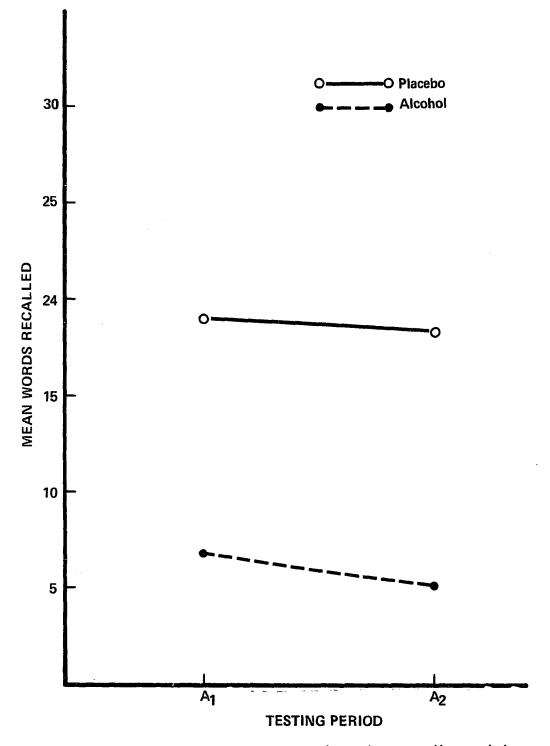


Figure 9. Mean  $A_{\underline{l}}$  words recalled on the ascending and descending limb.

Group	Testing Period	Repeated Words Within a List	Words from Prior Lists	Misspelled Words	Erroneous Words	Total Incorrect Words
Alcohol	В	1.0	31	2	22	65
	Al	26	33	16	37	112
	A <sub>2</sub>	16	29	9	31	× 85
Placebo	В	2	33	4	34	74
	A <sub>1</sub>	3	21	3	16	43
	A <sub>2</sub>	4	14	2	19	39

INCORRECT	WORDS	IN	THE	SHORI	MEMORY	TASK

TABLE 4

correct words are presented in Table 5. A significant Drug (F = 7.32, p < .05) and Drug X Testing Period interaction (F = 7.40, p < .01) were obtained. Figure 10 illustrates the interaction in that the placebo group decreased in the number of incorrect words across testing periods while the alcohol group increased. Simple effects indicated that alcohol subjects increased in the number of incorrect words from baseline to the ascending limb (F = 10.23, p < .01). However there was no significant difference between baseline and descending limb or between ascending and descending limb. A between group analysis revealed that the alcohol group had a greater number of incorrect words on the ascending (F = 14.87, p < .01) and on the descending (F = 6.61, p < .05) limb than the placebo group. Analysis of the categories of incorrect words indicated that the number of "erroneous" words decreased significantly in the placebo group from baseline to  $A_1$  (t = 2.20, p < .05), while there was no significant change in the alcohol group. The number of words from prior lists decreased significantly in the placebo group from baseline to  $A_2$  (t = 2.81, p < .05), while there was no significant change in the alcohol group. There were only a few subjects who made the other types of errors and these were not statistically analyzed.

#### Recognition Task

Results from the recognition task given one day later are presented in Table 6. Alcohol subjects crossed out more words (i.e., made more "omission" errors) that had appeared during the ascending (t = 4.71, p < .01) and descending (t = 3.50, p < .01) limb than during baseline. There was no significant difference among these periods in the placebo group. Alcohol subjects also classified baseline words significantly

Group			Testing	Period				F-ratios	
	B	}	A	1	A	2	Group	Testing	GXT
	Mean	S.D.	Mean	S.D.	Mean	S.D.	(G) <sup>-</sup>	(T)	
Alcohol	3.25	3.77	5.60	3.45	4.25	2.40	7.32*	1.11	7.44 <sup>**</sup>
Placebo	3.70	2.77	2.15	2.03	1.95	2.06			

# MEANS, STANDARD DEVIATIONS AND F-RATIOS FOR INCORRECT WORDS FROM THE SHORT-TERM MEMORY TASK

TABLE 5

\*p < .05 \*\*p < .01

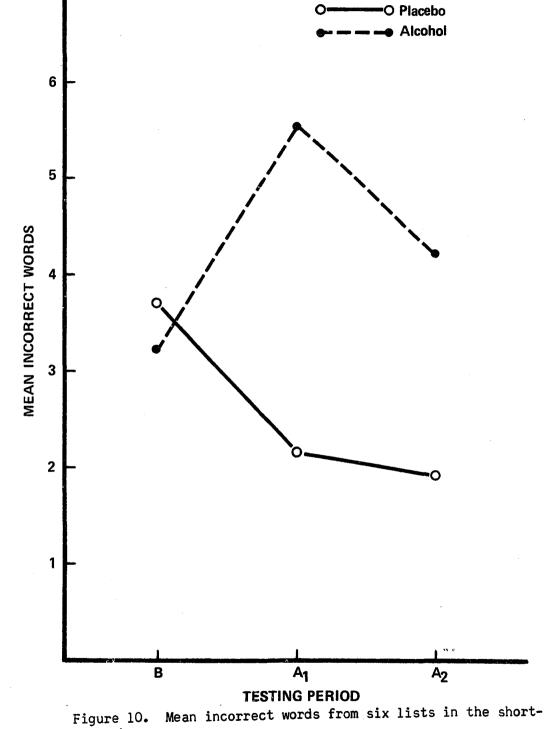


Figure 10. term memory task.

			Testing	Period		
Groups	]	3		Al	A <sub>2</sub>	
<del></del>	Mean	S.D.	Mean	S.D.	Mean	s.D.
Alcohol	26.67	16.85	36.39	19.75	34.67	18.36
Placebo	29.79	14.03	28.05	14.76	26.42	14.65

MEANS AND STANDARD DEVIATIONS FOR OMISSIONS FROM THE RECOGNITION TASK

TABLE 6

better than words presented on the ascending (t = 2.14, p < .05) and on the descending (t = 4.09, p < .01) limb (Table 7). Again placebo subjects classified correctly about the same number from each testing period. Thus alcohol subjects did not recognize words seen during the alcohol periods as well as words seen during the preceding baseline period.

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TABLE 7

#### MEANS AND STANDARD DEVI JMBER OF ITEMS CORRECT IN EACH GROUP DIVIDED BY THE TOTAL NUMBER OF ITEMS RECOGNIZED

Groups	Testing Period					
	В		Al		A <sub>2</sub>	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Alcohol	0.20	0.10	0.13	0.03	0.08	0.03
Placebo	0.15	0.04	0.16	0.04	0.14	0.04

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# CHAPTER VI

# DISCUSSION

The results of the present study demonstrate that short-term, intermediate-term and long-term memory are impaired following acute alcohol ingestion. Short-term memory is an operational definition and does not necessarily refer to a process. Thus, a short-term memory deficit refers to the task and not to a capacity. More specifically, the shortterm memory impairment is due to impaired recall of items that Glanzer believes go into long-term storage (LTS) but not to recall of items from short-term storage (STS). No significant difference was found between ascending and descending limb impairment for intermediate-term memory. Long-term memory (measured the next day) was also equally impaired for words presented on the ascending and descending limb. The memory deficit was attributed to the poor recall of the first four words in each list, those words described by Glanzer as going into long-term storage (LTS), and was especially evident in the intermediate-term and long-term memory tasks. Alcohol subjects also gave more incorrect words (commission errors) than the placebo subjects, especially on the ascending limb. Recall of baseline words during the ascending and descending limb, and 23 hours later was not impaired for the alcohol group. However recall and recognition of the alcohol words the next day were impaired for the alcohol group.

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#### Nature of the Memory Deficit

The nature of the memory deficit was explored in terms of Glanzer's model (1971) of short-term storage (STS) and long-term storage (LTS). The model has three basic elements. First, items go through preliminary processing at a "sensory" level. This processing includes storage of the input information over very short periods of time (half a second or less). Glanzer does not elaborate on this part of the processing. The item next appears in short-term storage (STS). It stays there for varying periods of time and then is displaced by the entry of subsequent items. The item can either stay in STS or drop out. Some or all the information about the item may be transferred to long-term storage (LTS). However this does not imply that the item has been removed from STS since it can be represented in both stores at the same time. Information can remain in LTS, be lost or become inaccessible.

Glanzer has demonstrated that the beginning and end of the serial position curve in a free recall task are differentially affected by a number of experimental variables. He states that these effects are due to output from two different storage mechanisms. He considers the major part of the end peak to consist of output from STS while the early sections of the curve consist solely of output from LTS. In the present study, it was found that alcohol impaired recall of items from the first part of the serial position curve. This indicates that LTS is impaired following alcohol consumption in the short-term memory task. This deficit could be due to several factors. It does not appear the deficit is due to the preliminary processing or perception of the items since words are pronounced correctly as they are presented. The deficit could be due to words dropping out of STS and therefore not being transferred to LTS. Another possibility is that items are transferred to LTS but are lost or become inaccessible.

The intermediate-term memory task provides data which bear on the above possibilities. If words were not transferred from STS to LTS, then a greater deficit for the LTS items would have been expected in the short-term memory task than was found. The other possibility is that information is transferred to LTS but is either lost (not consolidated) or becomes inacessible (retrieval is impaired). The data from the intermediate-term memory task indicate that many items from LTS recalled in the short-term memory task can no longer be recalled 15 minutes later. This differential impairment of short- and intermediate-term memory indicates that those items that were in LTS during the short-term memory task are either no longer in LTS or not retrievable during alcohol. However since words presented under alcohol are not retrievable on the ascending limb, the descending limb or in a non-alcohol state 23 hours later, it appears that for all practical purposes they are lost. The loss appears to be due to a failure of consolidation to take place during the alcohol period rather than to a failure of the subject's retrieval mechanisms. This inference further is supported by the fact that recall of baseline words during the alcohol period is not affected by alcohol.

#### Relevance of the Ascending and Descending Limb

As early as 1943 Goldberg demonstrated that the threshold for behavioral impairment was lower on the ascending than on the descending limb of the blood alcohol curve. He reported that the disappearance of

impaired performance often occurred at a higher blood alcohol level on the descending limb than its appearance on the ascending limb. Recent studies have amplified these results by demonstrating that cognitive impairment is greater on the ascending limb than on the descending limb at comparable blood alcohol levels (Jones and Vega, 1972). The present experiment extends these findings to short-term memory, an integral component of many cognitive tasks (see Appendix B for further discussion). This suggests that any task that involves short-term memory may be differentially impaired on the ascending and descending limb following alcohol ingestion. It also suggests an important methodological point for drug studies (other than alcohol) where differential limb effects may be important.

#### State-Dependent Learning Studies

The results have some relevance to studies which have reported that information learned under alcohol is recalled better in a subsequent alcohol state than in a different state, i.e., state-dependent learning (Storm, Caird, and Korbin, 1965; Storm and Caird, 1967; Madill, 1967; Goodwin <u>et al</u>., 1969; Tarter, 1970). However dissociation between drug states has been reported to be asymmetrical (in only one direction), although the direction differed in several studies. Dissociation was found in the drug-no drug condition in two studies (Goodwin <u>et al</u>., 1969; Storm and Caird, 1967) and in the opposite direction (no drug-drug) in three experiments reported by Madill (1967). Overton (1971) concludes there is no consistent evidence supporting the existence of an asymmetrical dissociation in either direction. It is evident from the present study that information presented in a no-drug state (baseline) can be

recalled in a later alcohol state. That is, dissociation was not found from the no-drug to drug state, a finding reported in several other studies (Goodwin <u>et al</u>., 1969; Storm and Caird, 1969). It was also found that information presented originally during alcohol could not be recalled very well the next day in a no-drug state. This is similar to the asymmetrical results reported by Goodwin <u>et al</u>. (1969). However it was also demonstrated in the present study that this information could not be recalled fifteen minutes after presentation while the subject was still in the alcohol state. Therefore impaired recall the next day was due to the effect of alcohol on memory <u>per se</u> and not to a change in the drug state.

Another way of examining state-dependent learning would be to determine if dissociation could occur from baseline to the ascending limb or to the descending limb (no drug-drug) since these two phases of the blood alcohol curve may be qualitatively as well as quantitatively different. The data suggest that such a dissociation does not occur. It is also apparent that dissociation does not occur from ascending limb or descending limb to the next day's performance (drug-no drug) since subjects can recall as many words the next day as they can fifteen minutes after seeing the words in the drug state.

Dissociation was also examined within the alcohol state since the ascending and descending limb may represent different "states" of the organism. Again, no evidence for state-dependent learning was found since subjects could remember about the same number of words on the descending limb as they remembered on the ascending limb in the intermediate-term memory task.

These results indicate that the poor cognitive performance re-

ported in state-dependent learning studies on day two in the A-NA group may not be due to a change in the state of the organism. Rather, it may be a result of impaired intermediate-term memory since this same deficit also appears about 15 minutes later during the alcohol state. In the present study if short-term memory during alcohol had been compared relative to the control group to recall the next day, it would have appeared that the greater deficit was due to a change in state. However if intermediate-term memory during the alcohol state is compared with recall the next day, there is no significant difference. It may be necessary to test for intermediate-term memory during the alcohol state before one can conclude that dissociation has occurred. Although there was no comparable A-A group in this study, it would be predicted that recall the next day would be similar to the A-NA group. The data from this study do not support state-dependent learning with alcohol using a verbal free recall task when intermediate-term memory deficits are taken into consideration.

### Short-term vs. Intermediate-term Memory Deficits: Biological Aspects

The differential impairment of short-term memory on the ascending and descending limb may be related to the different biological states which have been reported for these levels. Electroencephaloc aphic (EEG) changes are different during the rise and fall of the blood alcohol level (Gibbs <u>et al.</u>, 1937; Davis <u>et al.</u>, 1941; Engel and Rosenbaum, 1945; Van Henderstrom and Schmidt, 1951; Underleider, 1958). Further evidence suggests that EEG changes such as hippocampal theta are related to initial learning (Grastyan, Lissak, Madarasz and Donhoffer, 1959). It has

also been reported that theta rhythm is decreased by ethanol (Brown and Shryne, 1965). Ryback (1971) has hypothesized that a rapidly rising blood alcohol level pharmacologically depresses hippocampal function and hence the ascending portion of the limbic system by blocking theta. He does not report possible effects on the descending limb. However, such an explanation would account for the greater short-term memory deficits seen on the ascending than on the descending limb.

The intermediate-term and long-term memory deficits may be due to other biological variables since they are equally impaired on the ascending and descending limb. A series of studies by Agranoff (Agranoff, Davis, and Brink, 1965; Agranoff and Klinger, 1964; Brink and Agranoff, 1966) have indicated that some drugs which interfere with protein synthesis (but not ribosomal RNA or transfer RNA) also interfere with memory. Davis and Agranoff...(1966) demonstrated that puromycin, which interferes with protein synthesis, does not seem to interfere with shortterm memory, but blocks transfer to long-term storage as if preventing consolidation. A recent report has indicated that ethanol interferes with brain protein synthesis (Tewari and Noble, 1971). It may be that ethanol inhibits brain protein synthesis equally on the ascending and descending limb, therefore producing an impairment in intermediate-term and long-term memory due to a failure of consolidation.

# Practical Applications

To the extent that verbal and non-verbal processes are affected similarly by alcohol, this study has implications for automotive driving safety. The driver may find that his shortened memory for events occurring during alcohol intoxication may hinder his driving ability. The

blood alcohol level used in this study (0.09%) is below the level for legal intoxication in most states (0.15%). Yet, the memory impairment was very evident. While drivers may not have difficulty remembering events that occurred before drinking, they may have difficulty remembering new events during alcohol intoxication. The difficulty in evaluating driver competency may also be compounded if the limb of the blood alcohol curve is not established. That is, a driver might have a low blood alcohol level at the time of testing but may be on the ascending limb. If this is the case, then if he is allowed to drive he may be in more danger 20 to 30 minutes later as his blood alcohol level continues to rise. Thus, most traffic accidents may occur on the ascending limb of the blood alcohol curve, at lower blood alcohol levels than have been reported.

#### Future Studies

There are several limitations to the present study which suggest directions for future studies. These limitations will be divided into experimental design variables, alcohol variables, subject variables, task variables, and response variables.

#### Experimental Design Variables

There are several experimental design changes that would help clarify the memory impairment. The use of independent groups for the ascending and descending limb testing would eliminate the effect of practice. It would also be advisable to use a double-blind technique to help avoid experimentor bias.

# Alcohol Variables

One limitation to the present study is the single blood alcohol

level investigated. Future studies should be directed toward studying various blood alcohol levels, especially lower blood alcohol levels. This may be especially relevant since there have been reports of facilitation of cognitive performance at low blood alcohol levels (Carpenter <u>et al.</u>, 1969). It now also seems necessary to determine if such facilitation is related to the limb of the blood alcohol curve.

A variable that was not controlled in the present study was absorption rate. One report has indicated this may be an important variable (Eggleton, 1943) while another found no relationship between absorption rate and cognitive performance (Jones and Vega, 1972). However, it is possible that the differences reported on the ascending and descending limb may be related to the rapid absorption on the ascending limb as compared to the slow elimination rate on the descending limb. Absorption could be controlled by administering several small doses over a longer time period or giving subjects a standard amount of food before alcoholingestion.

# Subject Variables

The present study was limited to the effect of alcohol on young, highly educated males who reported being light to moderate social drinkers. These findings should be extended to include older subjects to determine if age or drinking experience influence the results. It would also be informative to determine if the ascending and descending limb differences are apparent in chronic alcoholics. One report has also indicated that the effect of alcohol is related to baseline performance. That is, subjects who do better in a baseline period are not as impaired under alcohol as those who perform more poorly (Carpenter and Ross, 1965).

This indicates that subjects with a lower educational level who might perform poorer in the baseline period would be even more impaired during the alcohol state.

Another subject variable that has been virtually ignored in most alcohol studies is that of sex. There is little experimental data to indicate whether there is a sex difference in response to alcohol. Females would have to be tested in relation to their menstrual cycle since many psychological and biochemical changes are related to this biological rhythm.

### Task Variables

There are a variety of experimental manipulations that could be performed with the free recall task in order to better delineate the effect of alcohol. Glanzer (1971) has reported that presentation rate, word frequency, list length, mnemonic structure and concurrent task load affect only LTS; they do not affect STS. This suggests that performance on the free recall task under alcohol could be improved by slowing the presentation rate to one word every nine seconds since this should improve recall of words from LTS (Glanzer and Cunitz, 1966). It also might be possible to improve performance under alcohol with a shorter list (less than 12 words); more dramatic differences under alcohol might be obtained with a longer list (20 words) (Murdock, 1962; Postman and Phillips, 1965). Another method of improving recall would be to use mnemonically related words since this also improves only recall of items from the first part of the list (Glanzer and Schwartz, 1971). There is also one additional way to produce more impaired performance. That is, instead of no delay between the end of list and recall as used in

the present study, a secondary task could be presented in this interval. The more difficult the task, the greater the impairment should be for recall of the first part of the list (Murdock, 1965; Silverstein and Glanzer, 1971). It would be difficult to improve performance by using words of higher normative frequency since the most frequent nouns (AA) were used in the present study.

Finally, other tasks could be used. There are a variety of other verbal memory tasks; however, a non-verbal task would be of interest as there are reports that alcoholics perform poorer on non-verbal than verbal tasks, indicating that right hemisphere functioning may be impaired (Jones, 1971). A recent study in our laboratories has indicated that non-alcoholics also perform poorly on non-verbal tasks during acute alcohol intoxication.

### Response Variables

The influence of alcohol on various psychophysiological response systems also should be examined during cognitive tasks. Several studies were reviewed earlier that indicated that alcohol may produce an EEG activation (ascending limb) followed by a slowing and return to baseline (descending limb). Alcohol has also been reported to result in an increased heart rate (Doctor and Bernal, 1964; Doctor, Naitoh, and Smith, 1966; Doctor and Perkins, 1961; Holmberg and Martens, 1955). Unpublished data in our laboratories indicate that heart rate is higher on the ascending than on the descending limb. Both the EEG (Glass, 1964) and heart rate (Lacey, Kagan, Lacey, and Moss, 1963) have been reported to be related to cognitive performance and may give useful information when studying the effects of alcohol on cognitive processes.

## CHAPTER V

### SUMMARY

Several studies have indicated that the effects of alcohol upon memory may be a function of ascending and descending limb blood alcohol levels. There is also evidence that alcohol may differentially affect short-term, intermediate-term, and long-term memory. The present investigation used three approaches to study the effect of alcohol on memory: an empirical study, the application of these data to a theoretical model, and predictions derived from an extension of the model.

The empirical study investigated the effect of alcohol on shortterm verbal memory during the ascending and descending limb of the blood alcohol curve at a blood alcohol level of 0.09%. It was hypothesized that short-term memory would be impaired both on the ascending limb (hypothesis la) and descending limb (hypothesis lb) and also that there would be a greater impairment on the ascending than on the descending limb (hypothesis lc).

The second approach involved the application of the empirical data to Glanzer's model of short-term storage and long-term storage. It was hypothesized that long-term storage would be more impaired than short-term storage in the short-term memory task (hypothesis 2).

The third approach extended Glanzer's model to include inter-

mediate-term and long-term memory. It was predicted that since long-term storage was impaired that intermediate-term memory (hypothesis 3) and long-term memory (hypotheses 4a, 4b, 5) also would be impaired.

The effect of alcohol on human memory was investigated employing a repeated measures design. Male medical students who reported being social drinkers were tested. Subjects in the alcohol group (n = 20) were tested before alcohol ingestion (baseline) and later tested on different material on the ascending and descending limb of the blood alcohol curve at a blood alcohol level of 0.09%, as measured by the Breathalyzer. Subjects in a placebo group (n = 20) were tested at comparable times as the alcohol group to control for practice and fatigue effects. Short-term verbal memory was measured by the free recall technique (no-delay) and data were interpreted in terms of Glanzer's model of short-term storage and long-term storage. Short-term and intermediate-term memory were measured during the baseline period and on the ascending and descending limb. Long-term memory was obtained for all words the following day during a non-alcohol condition. Baseline words were also recalled on the ascending and descending limb.

It was found that short-term memory was impaired on both limbs of the blood alcohol curve, but a greater deficit was found on the ascending than on the descending limb. These results support hypotheses la, lb, and lc. The memory deficit following alcohol ingestion was due to poor recall of the first four items in a twelve-item list. This was interpreted to demonstrate an impaired long-term storage following Glanzer's model, supporting hypothesis 2. The difference between ascending and descending limb performance was due mainly to poor recall of the

middle four items in the list. There was no difference between alcohol and placebo groups or between ascending and descending limb performance on the last four items, those words which according to Glanzer are in short-term storage. Overall, alcohol subjects made significantly more errors of commission (wrote more incorrect words) than placebo subjects. Alcohol subjects also made more commission errors on the ascending than on the descending limb.

Intermediate-term memory was investigated by obtaining recall of the words from the short-term memory task fifteen minutes later. In the placebo group the first four items in each list were recalled better than the other items, supporting and extending Glanzer's model, since the first four items are presumably in long-term storage and should be recalled better than the other items. In the alcohol group intermediateterm memory was impaired on both the ascending and descending limb of the blood alcohol curve. Unlike short-term memory, there was not a differential impairment on the ascending and descending limb. It was found that the intermediate-term memory deficit was due mainly to the poor recall of the first four items in each list. The results again indicate that alcohol impairs long-term storage and support hypothesis 3.

Long-term memory for baseline and alcohol words was measured 23 hours later. Recall of baseline words was not impaired for the alcohol group, suggesting intactness of the retrieval processes. However impairment was found for words presented during the alcohol conditions in both a recall and recognition task, supporting hypotheses 4a, 4b, and 5. There was no difference in recall between words presented on the ascending and descending limb. It would seem that the long-term storage defi-

cit is due to a failure of permanent consolidation.

Recall of baseline words was also measured on the ascending and descending limb. It was found that alcohol did not impair recall of these words during the alcohol period. Thus, dissociation was not found for the no-drug - drug condition. There also was no evidence for dissociation when intermediate-term memory during alcohol was compared to long-term memory the following day (drug - no-drug).

It was suggested that the short-term memory deficit may be related to the altered electroencephalographic activity that has been reported to occur on the ascending and descending limb. Intermediate-term and long-term memory deficits were related to possible biochemical alterations such as impaired brain protein synthesis that has been reported following alcohol ingestion.

Future studies were suggested that included extending these findings to lower blood alcohol levels and to additional groups such as chronic alcoholics. It also was suggested that EEG activity and heart rate changes be monitored following acute alcohol ingestion to evaluate the effects on alcohol on physiological response systems that have been related to cognitive performance.

In summary, alcohol was found to impair short-term, intermediate-term and long-term memory. Short-term memory was more impaired on the ascending than on the descending limb of the blood alcohol curve (0.0%). There was no difference in intermediate-term or long-term memory on the ascending and descending limb. The memory deficit was due to a failure of the alcohol subjects to recall adequately the first four items in a twelve-item list. This was interpreted in terms of Glanzer's

model as a deficit in long-term storage. No such deficit was found for short-term storage. The deficit was considered to be due to a failure of consolidation of items into long-term storage and not to a failure of retrieval. The findings were also related to state-dependent learning studies. It was concluded that dissociation was not demonstrated in this study for either the no-drug - drug condition or the drug - no-drug condition.

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APPENDIX A

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	Age		Weight	Height	<u>Shi</u> Vocab-	pley-Hartf	ford	Eysenck In	Persor ventory	-	Raven's Matrices	
Subjects		Education	(1bs.)	(in.)	ulary	Abstract	WAIS	E	N	L	Set I	Set Il
1	25	14	180	74	29	32	110	16	12	2	7	19
2	24	18	190	72	30	36	117	8	20	1	7	19
3	26	14	210	70	37	36	120	18	10	1	9	17
4	21	13	180	72	26	26	103	19	9	2	10	13
5	33	19	155	69	36	32	116	12	4	3	10	18
6	26	13	180	71	33	36	117	11	7	0	7	16
7	21	17	205	75	31	36	118	11	8	0	12	25
8	22	17	165	70	35	40	125	13	3	1	11	22
9	21	13	165	72	35	38	124	16	17	3	12	23
10	24	16	180	72	34	38	123	6	3	0	7	17
11	25	17	175	75	34	32	114	11	6	1	9	19
12	21	17	180	72	35	30	116	15	14	3	7	2
13	23	18	150	68	34	40	125	8	2	1	10	20
14	27	14	145	71	40	24	113	10	0	6	7	12
15	25	19	160	72	30	40	117	15	17	0	10	14
16	24	18	155	71	37	38	125	8	13	3	12	22
17	23	18	170	72	36	38	125	8	4	2	12	27
18	23	17	155	70	39	38	127	12	9	0	10	21
19	22	17	200	75	39	38	127	10	17	2	12	27
20	24	19	160	67	36	40	126	14	11	2	8	14
Mean	24.00	16.40	173.00	71.50	34.30	35.40	119.40	12.05	9.30	1.65	9.45	18.35
S.D.	2.79	2.11	18.38	2.19	3.61	4.64	6.44	3.63	5.77	1.50	1.99	5.79

## DEMOGRAPHIC AND TEST DATA FOR ALCOHOL GROUP

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		Education	Weight (lbs.)	Height (in.)	Shipley-Hartford Vocab-			Eysenck Personality Inventory			Raven's <u>Matrices</u>	
Subjects	Age				ulary	Abstract	WAIS	E	N	L	Set I	Set I
1	25	16	150	70	31	32	112	11	16	0	9	18
2	24	17	155	68	40	40	130	15	7	0	11	22
3	34	19	195	72	37	40	123	7	7	0	9	20
4	25	16	245	77	39	36	121	14	9	7	11	27
5	22	17	111	65	37	38	125	11	7	1	12	26
6	22	17	135	70	32	40	123	12	8	4	10	26
7	22	17	155	71	30	36	117	16	10	0	10	25
8	22	16	155	68	37	38	125	13	7	1	10	26
9	22	17	160	72	34	40	125	14	. 9	4	11	23
10	22	17	215	72	36	. 36	123	17	12	0	11	28
11	26	18	165	69	39	32	118	12	7	2	10	25
12	22	17	180	76	32	34	117	13	5	3	10	29
13	21	17	215	75	32	34	117	18	8	5	11	30
14	21	15	140	70	32	40	123	17	16	0	11	26
15	28	17	205	74	38	36	121	9	5	1	11	29
16	23	17	200	73	36	38	125	15	12	2	11	26
17	22	17	165	71	32	34	117	18	15	1	12	34
18	28	17	185	72	40	38	124	11	10	1	12	25
19	23	18	170	71	33	36	120	10	8	2	12	25
20	22	17	195	74	35	40	125	19	4	2	11	35
Mean	23.8	16.95	174.80	71.50	35.10	36.90	121.55	13.60	9.10	1.80	10.75	26.25
S.D.	3.19	0.83	32.24	2.89	3.21	2.71	4.20	3.28	3.51	1.94	0.91	4.05

# DEMOGRAPHIC AND TEST DATA FOR PLACEBO GROUP

Subjects	RPM Set I	Recall B on A <sub>l</sub>	VMT Al	Recall A <u>l</u>	EPI Al	Peak BAL	RPM Set II	Recall B and A <sub>l</sub> on A <sub>2</sub>	VMT A <sub>2</sub>	Recall A <sub>2</sub>	EPI A <sub>2</sub>
1	30	40	50	70	80	95	95	148	160	175	185
2	35	45	55	67	75	75	80	125	145	163	170
3	26	32	38	50	58	67	67	103	135	149	155
4	35	45	50	65	75	65	82	145	160	170	175
5	20	35	45	58	65	120	75	120	130	150	160
6	33	40	50	60	70	97	77	133	160	170	178
7	33	38	43	58	63	93	73	108	128	143	148
8	35	40	45	60	65	75	75	125	135	150	155
9	27	32	40	53	60	105	67	105	120	130	140
10	25	40	50	65	70	85	85	130	155	170	175
11	31	63	73	88	93	93	118	158	163	178	188
12	28	34	43	58	68	68	90	126	158	176	183
13	32	38	45	58	65	72	72	124	149	156	161
14	35	50	40 75	90	100	100	110	155	165	180	190
15	43	53	63	73	88	63	98	123	133	148	153
16	35	40	50	60	70	50	135	175	197	208	212
17	25	30	45	55	65	75	80	145	160	175	180
18	40	45	50	70	80	80	100	140	160	175	185
19	47	53	60	78	85	85	95	135	167	183	190
20	78	88	98	108	133	168	148	203	218	238	248
Mean	34.65	44.05	53.40	67.20	76.40	86.55	91.10	136.30	154.90	169.35	176.55
S.D.	12.04	13.21	14.44	14.39	17.43	25.42	22.17	24.02	23.14	23.84	24.33

NUMBER OF MINUTES FROM BEGINNING OF DRINKING TO TESTING FOR THE ALCOHOL GROUP

B = Baseline,  $A_1 = Ascending Limb$ ,  $A_2 = Descending Limb$ , RPM = Raven's Progressive Matrices, EPI = Eysenck Personality Inventory, VMT = Verbal Memory Test and BAL = Blood Alcohol Level.

Subjects	RPM Set I	Recall B on Al	VMT A <sub>l</sub>	Recall Al	EPI Al	Peak BAL	RPM Set II	Recall B and A <u>1</u> on A <sub>2</sub>	VMT A <sub>2</sub>	Recall A <sub>2</sub>	EPI A2
1	25	40	45	58	65	_	77	115	130	145	150
2	15	25	33	45	52	_	63	105	120	130	136
3	25	35	45	55	65	-	80	120	135	150	155
4	35	40	40	60	68	-	80	123	135	148	155
5	24	30	37	48	56	-	65	107	117	130	137
6	27	35	45	60	65	-	75	115	130	140	150
7	35	45	50	65	75	_	85	125	140	150	155
	15	30	35	45	50	-	60	85	95	110	115
8 9	35	40	45	55	65	-	75	115	135	150	160
10	23	28	35	45	55	-	65	105	117	127	135
11	25	35	45	55	65	-	75	115	130	140	150
12	25	35	40	55	60	-	65	105	120	135	140
13	23	30	35	48	55	-	62	105	115	128	135
14	17	28	35	45	53	-	65	108	120	133	140
15	25	35	45	55	65	-	70	115	130	142	150
16	25	30	40	53	60	-	70	110	122	137	143
17	25	35	45	55	65	-	75	115	130	140	145
18	30	40	45	55	65		75	135	145	160	165
19	20	30	40	50	55	-	65	105	120	135	140
20	25	30	35	50	57	-	65	105	120	132	140
Mean S.D.	24.95 5.74	33.80 5.23	41.10 5.17	52.85 5.71	60.80 6.44		70.60 7.09	111.65 10.27	125.30 11.03	138.10	144.80 11.18

NUMBER OF MINUTES FROM BEGINNING OF DRINKING TO TESTING FOR THE PLACEBO GROUP

B = Baseline,  $A_1 = Ascending Limb$ ,  $A_2 = Descending Limb$ , RPM = Raven's Progressive Matrices, EPI = Eysenck Personality Inventory, VMT = Verbal Memory Test and BAL = Blood Alcohol Level.

Subjects	SI	Short-term Memory			Intermediate-term Memory			Long-term Memory			all eline During hol ods	Recall of A <sub>l</sub> Words on the Descending Limb	
	В	Al	A <sub>2</sub>	В	Al	A2	B	Al	A <sub>2</sub>	B on Al	B on A2	A <sub>1</sub> on A <sub>2</sub>	
1	25	21	23	10	1	4	9	3	5	9	9	1	
2	34	24	31	21	5	6	16	3	1	16	16	2	
3	34	30	32	16	6	6	13	6	2	12	12	5	
4	35	24	27	10	2	5	9	2	6	8	9	0	
5	36	32	31	14	4	2	8	2	0	14	13	3 8	
6	37	31	34	16	3	7	19	3	3	13	15	8	
7	39	38	31	18	7	2	14	8	0	13	11	6	
8	40	29	34	13	4	3	14	2	3	12	15	1	
9	43	34	42	19	11	12	19	9	7	16	18	9	
10	43	35	36	21	9	6	20	10	3	22	23	15	
11	43	41	42	17	3	3	11	0	0	,12	13	0	
12	45	37	50	23	4	16	11	0	2	18	14	0	
13	45	43	44	26	18	10	18	12	0	27	21	12	
14	46	40	37	15	10	10	11	11	10	23	17	8 3	
15	52	34	44	33	6	7	23	0	2	24	23		
16	52	35	37	26	3	7	21	3	1	24	20	4	
17	52	44	47	22	12	12	25	9	8	25	22	11	
18	52	45	51	17	3	10	15	0	3	12	11	0	
19	54	35	35	37	8	3	33	9	2	34	36	8 8	
20	62	53	58	36	15	9	29	8	10	36	30	8	
Mean	43.45	35.25	38.30	20.50	6.70	7.50	16.90	5.00	3.40	18.50	17.40	5.20	
S.D.	8.82	7.84	8.80	7.82	4.57	4.65	6.84	4.10	3.22	8.00	6.98	4.51	

## SHORT-TERM, INTERMEDIATE-TERM, AND LONG-TERM MEMORY DATA FOR THE ALCOHOL GROUP

Subjects	_	Short-term Memory			Intermediate-term Memory			Long-term Memory			ll eline During hol ods	Recall of A <sub>l</sub> Words on the Descending Limb	
	В	Al	A2	В	Al	A <sub>2</sub>	В	Al	A <sub>2</sub>	B on Al	B on A <sub>2</sub>	A <sub>1</sub> on A <sub>2</sub>	
1	30	30	30	12	11	15	8	9	10	. 11	9	12	
2	32	41	35	19	17	10	12	19	1	16	15	16	
3	33	37	33	15	8	11	8	4	1	11	8	6	
4	34	31	39	16	13	19	10	16	15	13	11	17	
5	36	35	38	21	20	16	24	17	11	22	22	22	
6	39	42	50	17	25	20	14	25	13	14	14	27	
7	40	33	46	12	13	20	11	7	12	12	11	11	
8	40	42	37	19	11	14	13	9	10	17	18	13	
9	40	43	42	11	18	15	11	14	13	9	10	16	
10	41	39	46	22	26	27	16	27	24	22	20	27	
11	43	42	40	21	19	18	21	16	19	22	22	18	
12	43	44	47	26	22	29	22	18	17	26	24	28	
13	43	46	41	20	17	19	15	7	4	19	16	8	
14	43	46	43	18	14	9	11	8	3	17	14	9	
15	44	46	41	19	11	8	12	13	· 9	18	16	10	
16	45	48	48	13	14	25	10	18	7	11	10	20	
17	49	41	47	26	26	28	28	18	18	32	33	23	
18	49	56	61	14	21	20	16	18	11	17	14	21	
19	52	49	47	24	23	36	16	24	33	22	20	27	
20	56	62	62	35	51	50	22	40	39	32	25	41	
Mean	41.60	42.65	43.65	19.00	19.00	20.45	15.00	16.35	13.50	18.15	16.60	18.60	
S.D.	6.71	7.80	8.09	5.85	9.27	10.07	5.65	8.42	9.79	6.62	6.42	8.67	

## SHORT-TERM, INTERMEDIATE-TERM AND LONG-TERM MEMORY DATA FOR THE PLACEBO GROUP

Subjects	RPM Set I	Recall B on Al	VMT Al	Recall Al	EPI A <sub>l</sub>	Peak BAL	RPM Set II	Recall B and A <sub>l</sub> on A <sub>2</sub>	VMT A <sub>2</sub>	Recall A <sub>2</sub>	EPI A <sub>2</sub>
1	0.120	0.130	0.120	0.130	0.135	0.140	0.140	0.095	0.095	0.090	0.090
2	0.090	0.090	0.095	0.100	0.133	0.140	0.115	0.110	0.095	0.090	0.090
3	0.075	0.085	0.095	0.110	0.100	0.120	0.120	0.115	0.100	0.100	0.100
4	0.110	0.120	0.130	0.140	0.130	0.140	0.130	0.080	0.090	0.080	0.090
5	0.065	0.050	0.070	0.060	0.070	0.090	0.070	0.090	0.080	0.090	0.090
6	0.090	0.090	0.115	0.120	0.120	0.135	0.130	0.110	0.090	0.095	0.100
7	0.085	0.080	0.090	0.090	0.090	0.110	0.085	0.100	0.095	0.095	0.090
8	0.100	0.090	0.090	0.095	0.090	0.110	0.110	0.095	0.095	0.095	0.090
9	0.080	0.090	0.080	0.090	0.080	0.100	0.090	0.100	0.090	0.080	0.080
10	0.060	0.075	0.085	0.090	0.100	0.110	0.110	0.090	0.080	0.080	0.080
11	0.060	0.070	0.075	0.080	0.090	0.090	0.080	0.085	0.070	0,065	0.070
12	0.075	0.080	0.090	0.095	0.100	0.100	0.090	0.100	0.075	0.080	0.080
13	0.090	0.090	0.075	0.085	0.100	0.110	0.110	0.090	0.085	0.080	0.080
14	0.075	0.095	0.100	0.115	0.120	0.120	0.110	0.090	0.095	0.085	0.095
15	0.110	0.135	0.145	0.120	0.110	0.145	0.110	0.095	0.090	0.085	0.080
16	0.090	0.090	0.100	0.090	0.080	0.100	0.075	0.050	0.050	0.060	0.050
17	0.050	0.070	0.080	0.080	0.085	0.095	0.090	0.100	0.090	0.085	0.080
18	0.095	0.090	0.100	0.110	0.120	0.120	0.120	0.085	0.080	0.080	0.075
19	0.070	0.075	0.080	0.090	0.100	0.100	0.100	0.095	0.090	0.100	0.100
20	0.060	0.070	0.070	0.080	0.080	0.090	0.080	0.070	0.070	0.070	0.070
Mean	0.083	0.088	0.094	0.099	0.101	0.112	0.103	0.092	0.085	0.084	0.084
S.D.	0.019	0.020	0.020	0.020	0.018	0.017	0.020	0.014	0.012	0.011	0.012

PERCENT BLOOD ALCOHOL LEVELS FOR EACH TASK FOR THE ALCOHOL GROUP

B = Baseline,  $A_1 = Ascending Limb$ ,  $A_2 = Descending Limb$ , RPM = Raven's Progressive Matrices, EPI = Eysenck Personality Inventory, VMT = Verbal Memory Test, BAL = Blood Alcohol Level. <u>8</u>5

APPENDIX B

The difference in short-term memory between the ascending and descending limb was due primarily to recall of the middle items of the serial position curve. Glanzer (1971) has suggested that recall of these items is related to the probability that an item will be registered in LTS. It may be that an increasing blood alcohol level then acts to reduce the probability that an item will be transferred from STS to LTS while this is not the case for a decreasing blood alcohol level. Since recall of the middle items for intermediate-term and long-term memory is not different on the ascending and descending limbs, it appears that those items that were recalled on the descending limb during the shortterm memory task were not permanently stored for later recall.