THE EFFECTS OF ALCOHOL ON RECALL, RECALL CONFIDENCE, RESPONSE LATENCY, AND MOOD DURING THE ASCENDING AND DESCENDING LIMBS OF THE BLOOD ALCOHOL CURVE OF NONALCOHOLICS

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CHAPTER I

INTRODUCTION

One of the popular names assumed by the alcoholic beverages is "curse-your-memory" (Carroll, 1975, p. 32), because alcohol, being a chemical compound, is similar to ether and shares with that substance an anesthetic effect on the central nervous system. Carroll described the effects of drinking, based on blood alcohol levels, as a progressive, depressant action on the brain.

> This depressant action is one of slowing down, dulling, blunting, or impairing brain function, both perception and motor function. While undiluted alcohol can irritate the lining of the oral cavity and gastrointestinal tract, and small doses of alcohol may have no behavioral effects, the feeling and appearance of stimulation following drinking is generally symptomatic of alcohol's numbing or anesthetic quality. The talkativeness, noise, excited feelings, and increased activity are the results of lowered inhibitions and sedation. When normal restraints are removed, when the superego is dissolved, the drinker is inclined to do some rather unusual, even bizarre things. The sedative effect often masks body fatigue, thus promoting a sense of relief, a reduction of tension, and a feeling of stimulation. The drinker's self-appraisal of his "stimulated" feelings and "improved" performance is now clouded by alcohol-induced mental impairment. Such modification of mood and behavior is only a pseudostimulation (Carroll, 1975, p. 43).

The investigation of the effects of alcohol on human behavior has a long history dated back to 1940. The

voluminous systematic information documented by the Quarterly Journal of Studies on Alcohol has witnessed part of the long term effort to study the relationship between alcohol and behavioral changes (Jellinek & McFarland, 1940).

From the early attempt to understand the nature of alcohol, to the treatment of alcoholics and alcohol-related diseases such as delirium tremens and Korsakov's psychosis, there has evolved a major area of interest: the effects of alcohol on memory. Through verbal learning procedures, recent experimental studies on alcohol and memory have led to better understanding of state-dependent learning (Weingartner & Faillace, 1971; Lisman, 1974), disruption of cognitive processes (Parker, Alkana, Birnbaum, Hartley & Noble, 1974), and amnesia and short-term memory function (Tamerin, Weiner, Poppen, Steinglass & Mendelson, 1971). There is increasing evidence that, within a given blood alcohol level, alcohol would impair memory. However, in recent years there are experimental findings indicating the differential extent of impairment on memory between a rising and a falling blood alcohol level after a given dose of alcohol (Young, 1970; Jones, 1972, 1973). These findings are worth noticing.

Response Latency is a popular behavioral measure in psychology (Sternberg, 1969; Moskowitx & Roth, 1971) as well as in alcohol research. A recent study found that alcohol at blood alcohol concentrations up to 0.055% had no significant effect on choice reaction time and information

processing, although there was some indication that the accuracy of performance was impaired at blood alcohol concentrations of 0.037 and 0.055%. In contrast, alcohol had a slightly facilitating effect on accuracy and response speed when blood alcohol concentration was at 0.011% (Shillito, King & Cameron, 1974). These findings, if confirmed, would have significant bearing on the conception and practical implications of alcohol as either a stimulant or a depressant, or a combination of both with dose-response differences.

CHAPTER II

STATEMENT OF PURPOSE

There is no need to emphasize the importance and significance of investigation into the effects of alcohol on memory and response latency. A huge drinking population in this age of uncertainty, change, and controversy, together with the well-established alcohol-related psychoses and neuroses as described in the classification of mental disorders by the American Psychiatric Association, warrants any positive effort to advance systematic information on the relationship between alcohol and human behavior.

According to Mendolson (1969), alcohol is pharmacologically referred to as being sedative, narcotic, hypnotic, analgesic, and anasthetic.

> Most of the pharmacological effects of alcohol are due to its presence in the brain, since it is absorbed rapidly and enters the brain within seconds after it appears in the blood. The initial effect is on the cortex to depress the inhibitory center and the stimulatory effect commonly attributed to alcohol by laymen is due to its power to lessen inhibitions. This effect is manifested by a feeling of confidence and power, an easing of tension and anxiety, lessened self-consciousness and false euphoria (Mendolson, 1969, p. 299).

The "feeling of confidence and power" keys into an interesting aspect of the present study, i.e., the drinker's

confidence in his own action. It would seem rewarding to examine, in addition to the drinker's ability to recall and his response latency in a paired-associates learning task, his confidence in his own response.

A summary review of the results of recent studies on the effects of alcohol on the central nervous system was reported in the Second Special Report to the U. S. Congress on Alcohol and Health (Department of Health, Education, and Welfare, 1974). The report summarized the effects of alcohol on brain metabolism, nerve cell transmission, synaptic function and the biochemistry of membrane-bound processes. It was reported that alcohol had anesthetic effects on the brain. At high doses alcohol induced up to a 30% decrease in brain oxygen consumption, and reducing glucose utilization simultaneously. It was pointed out that alcohol decreased the action potential by directly interfering with the changes in ion conductance and might also decrease the resting nerve cell potential. As to effects of alcohol on synaptic function, the report went on to state that:

> Due to the structural complexity and small size of the synaptic region in nerves, no concensus exists as to the action of alcohol on synaptic functions. There is however, a large literature on the effect of alcohol on brain contents on one or another neurotransmitters such as norepinephrine, serotonin, acetylcholine and gammaaminobutyric acid. Unfortunately this extensive literature leads to no firm evidence supporting the conclusion that the various neurotransmitters are in any way related to the fundamental action of alcohol upon the brain. This unhappy state results largely from the fact that it has so far not been possible to study

> neurotransmitters within the synaptic cleft where they are functionally active (Department of Health, Education, and Welfare, 1974, p. 125).

Although there has been indication that ethyl alcohol can inhibit the active transport of potassium ions, the limited knowledge available and the technical insufficiency of the present state permitted the aforementioned report to maintain only a modest view: that there was yet no definitive answer to the kind of mechanism through which alcohol affects the central nervous system.

Within the field of psychology, studies on alcohol and memory have concentrated more on the quality and quantity of the impairment than the mechanism or process. Previous investigation on alcohol and memory centered on the dose-response aspect of alcohol. It was not until quite recent years that the nature of the blood alcohol curve within a given alcohol dose, received proper attention. It is possible that on the ascending limb of a person's blood alcohol curve, alcohol impairs memory more severely as a result of stronger inhibition. Carroll (1975, p. 43) reported that the reticular formation, a "master switchboard," might be the initial site of impairment. Thus, the person would have a subjective feeling of confidence and power, which is in fact a false euphoria. On the descending limb of the blood alcohol curve, both power and confidence feeling are absent. Because, at this point, the sedative and analgesic effect of alcohol is more pronounced, alcohol becomes becomes more a depressant. Thus, alcohol would have

a definite inhibitory effect on memory and response latency on both the ascending and descending limbs of a person's blood alcohol curve.

The possibility that alcohol may have differential effects on memory, response latency, and a host of other behavioral variables on the ascending and descending limbs of a person's blood alcohol curve, implies a new dimension in alcohol research. The purpose of the present experiment was to examine the differential effects of alcohol on short-term memory, as defined by paired-associates learning and immediate recall, on the ascending limb and on the descending limb of the subject's blood alcohol curve within a given blood alcohol level range. The subject's confidence on his own response will be studied by looking at his confidence ratings conditionalized upon the accuracy of his recall responses. Following the signal detection theory, this would yield the two signal detection parameters, response discriminability (d') and response bias (Beta). Response Latency will also be examined on each limb of the subject's blood alcohol curve.

While recent experiments on the limb effect have been shown by using high doses (Jones, 1972, 1973), the present experiment studies the limb effects under a low dose and a moderate dose respectively. It was reported in the Shillito, King & Cameron (1974) study that blood alcohol concentration up to 0.055% had no significant effect on choice reaction-time and information processing. Based on

a pilot study with four subjects who showed an average peak blood alcohol level of 0.0475% for the low dose, and 0.09% for the moderate dose, it was expected that the low dose group in the present experiment would produce a peak blood alcohol level of approximately 0.05%. It would be meaningful to compare the resulting effect on response latency. On the other hand, although the application of the signal detection paradigm to recall phenomena is becoming popular, to use the signal detection model on recall under the influence of alcohol is rare. Thus, the present study represented an explorative attempt to apply the signal detection paradigm in the alcohol and short-term memory recall area.

Independent Variables

The independent variable in the present experiment was the alcohol presented in three different levels (0.00 ml per every kg of body weight of the subject in the placebo group, 0.46 ml/kg for the low dose group, and 0.92 ml/kg for the moderate dose group). However, since the adsorption rate and elimination rate differs between individuals, it was the actual blood alcohol level which represented the percentage of alcohol in the person's blood, that affected his behavior. Blood alcohol level was, therefore, another variable. However, blood alcohol level is closely related to the amount of alcohol consumed, though not in simple direct proportion; it was conceptualized as a covariate to

the alcohol dosage. In the case of the present study, the blood alcohol level range was considered as a block. Within a given range (0.02-0.4% for the low dose group, and 0.06-0.08% for the moderate dose group), the rising blood alcohol level, which was the ascending limb of the subject's blood alcohol curve, was taken as one block, while the falling blood alcohol level, which was the descending limb of the subject's blood alcohol curve, was taken as another block. Within each block, there were two subunits of 20 paired-associates lists. These subunits were conceptualized as the third independent variable.

Dependent Variables

Through the probe paired-associates learning and recall task, three different dependent variables were measured within each treatment level, "block", and subunit.

Mean correct recall in each subunit was the total number of response words correctly recalled in every 20 trials. Partially correct recalled words were counted as incorrect.

Response discriminability (d") is an index of the subject's accuracy of awareness of correct and incorrect responses (Hochhaus, 1975). Response bias (beta) is the extent to which the subject favors one criterion over another, independent of the objective evidence available. Another measure of response bias, mean recall confidence, was simply the degree of confidence averaged across both correct and incorrect recall responses. Both d" and beta were computed

by the hit rate (HR) and the false alarm rate (FAR) according to the Hochhaus (1972) table.

Response latency was the time interval between the onset of the probe word (A-member) and the subject's verbal initiation of the response under the condition that the subject had been instructed to respond as rapidly and as accurately as possible. Response latency was measured to the nearest msec. Since any change in the subject's mood under the influence of alcohol may be related to the dependent variables (Ekman, Frankenhaeusen, Goldberg, Hagdahl, & Myosten, 1964), the Multiple Affect Adjective Check List (MAACL) was periodically administered to the subject to detect any mood change after alcohol.

CHAPTER III

REVIEW OF LITERATURE

The following review of literature focuses on three major areas, namely, the effect of alcohol on short-term memory, the effect of alcohol on response latency, and the signal detection model, especially its application to the analysis of response discriminability and confidence in a recall situation.

Alcohol and Short-term Memory

Although there is increasing evidence that alcohol impairs memory, especially short-term memory, there has been a controversy regarding the facilitating effect of alcohol. In a problem-solving experiment at doses of 0.34 ml/kg, and 0.40 ml/kg it was found that alcohol improved performance; deterioration did not appear until the dose of alcohol was increased to 1.00 ml/kg (Carpenter et al., 1961). Later, Carpenter and Ross (1964) conducted another experiment with 16 nonalcoholics at the doses of 0.0, 0.33, 0.67, and 1.00 ml/kg of body weight; the results obtained were quite dramatic:

> The effect of alcohol on total error on the RMM (Running Matching Memory) was related to the initial performance level of the subject. Subjects with the highest degree of skill (Rank 1) showed linear deteriora-

> tion with increasing doses, but subjects with less proficiency (Ranks 2, 3, and 4) showed improvement at low doses and less absolute deterioration than the best subjects. Improvement in performance was suggested at approximately 0.024% to 0.055% blood alcohol with obvious deterioration not occurring until blood alcohol exceeded 0.070%. Nevertheless, regardless of relative deterioration, the best subjects performed best, and the order of proficiency was maintained under alcohol (Carpenter & Ross, 1964, p. 578).

However, in the same year when Carpenter and Ross (1964) did their experiment, two other studies on short-term memory found that alcohol impaired short-term memory. Hutchinson, Tuchtie, Gray & Steinberg (1964) asked eight subjects to recall paired-associate tasks at a blood alcohol concentration of 0.10% and Muller, Tarpey, Giorgi, Mirone, & Rouke (1964) (in Ryback, 1971) had 10 subjects perform a pictorial recognition task at 1.15 cc/kg, both found that alcohol significantly impaired short-term memory.

Ryback (1971) reviewed 17 studies on alcohol and immediate, short-term, and remote memory, and concluded that:

> Alcohol also most severely and selectively disrupts STM. Indeed, even in normal subjects with BACS (blood alcohol concentrations) similar to those commonly produced at cocktail parties, shorter spans of STM were affected with a rise in the mean BAC from 79 to 103 mg per 100 ml. Accordingly, perhaps a disruption of STM is the specific memory dificit common to cocktail-party drinking, alcohol amnesia and the Wernicke-Korsakoff syndrome. Perhaps there is also a continuum among the latter three (Ryback, 1970, p. 1008).

It is possible that the degree of impairment under the same dose of alcohol will differ between the alcoholics and the nonalcoholics. In an attempt to examine amnesia and short-term memory function during experimentally induced intoxication, Tamerin (1971) had 13 alcoholics drink during a

free-choice drinking period of 12 to 14 days up to a quart of 100-proof beverage alcohol each day and had them perform recall tasks; he found that at moderate levels of intoxication (below 200 mg/100 ml body weight, five-second recall remained essentially normal (94%), but "percentage correct" decreased as retention interval increased. At higher levels of intoxication (above 200 mg/100 ml), it was found that the subjects forgot over 50% of normally memorable material in five minutes.

Goodwin (1972) used a placebo group and an alcohol group (8-10oz) of hospitalized male alcoholics in a free recall and recognition task. The results were that the placebo group did somewhat better on recall, but the alcohol group did somewhat better on recognition, with no significant differences in either case. It was thus concluded that relatively modest amount of alcohol had no effect on immediate, short-term or long term memory for alcoholics.

Up to 1970, studies on alcohol and memory did not distinguish between the rising and falling blood alcohol level of the subjects. This in fact posed a confounding problem in the interpretation of alcohol effects, because it was not known whether the measurement of the dependent variable was taken during the ascending or the descending limb on the blood alcohol curve of the subjects. Young (1970) found that at a given blood alcohol concentration, impairment on response latency was more pronounced when the blood alcohol concentration was rising than when it was falling.

In a review of the effects of alcohol on memory, Ryback (1971) came to the conclusion that memory impairment might be related to the acute rise in the blood alcohol level. Jones and Vega (1972) reported that the impairment on cognitive performance was less pronounced when the task was tested on the descending limb of the blood alcohol curve. Later Jones (1972) found that at 0.09% blood alcohol level (after consuming 1.32 ml/kg of 95% USP ethanol) alcohol impaired immediate, short-term, and long-term memory. Jones (1972) found that immediate memory impairment was greater on the ascending than on the descending limbs of the blood alcohol curve, while the impairment on short-term memory was invariant on the ascending and descending blood alcohol curve. He was contented that long-term memory impairment was due to the short-term memory deficit and not to state-dependent effects.

In another study, Jones (1973) reported that at 0.05% blood alcohol level (after 0.66 ml/kg alcohol), and at 0.09% blood alcohol level (after 1.32 ml/kg alcohol), alcohol impaired memory more on the ascending limb for both groups, but the effect was invariant when the two groups were compared on either the ascending or the descending limbs. It was found that the high dose group was tested on the ascending limb, and the low dose group was tested on the descending limb.

Jones (1973) explained that it was because the high dose of alcohol took a longer time to reach its peak, as

compared to a lower alcohol dose; thus when the blood alcohol levels of the high dose subjects were still ascending, those of the low dose subjects were already on their descending limb.

Alcohol and Response Latency

Under the conception that alcohol is an anesthetic drug, a sedative, and a depressant, it is reasonable to assume that reaction-time will increase in direct proportion with the increment in the dose of alcohol. However, Young (1970) found that over a period of time, simple reaction-time was not directly related to blood alcohol concentration, suggesting a bodily adaptation effect. He further suggested that at a given blood alcohol concentration, more impairment on simple reaction-time occurs when the blood alcohol concentration was rising than when it was falling.

The results of the Shillito, King, & Cameron (1974) study were at variance with the Young (1970) findings. Shillito et al. (1974) used five male students at the peak BALs of 0.0, 0.03, 0.06, and 0.09% (after consuming 0.0, 0.26, 0.52, and 0.78 ml 86 USP alcohol per kg of body weight) to perform a key pressing task. They concluded that:

> (1) Choice reaction time is not affected by BALs of up to 0.055%. Tasks employing choice reaction time cannot be regarded as sensitive to alcohol effects, since measurable decrements in other psychomotor performance have been recorded at lower concentrations of alcohol in the blood. (2) There is some evidence that accuracy of performance is impaired at BALS of 0.037 and 0.055% while 0.011% had a slight facilitating effect on

> performance. It appears that tasks in which accurate performance is the main criterion are more likely to be sensitive to alcohol effects than tasks which involve information processing. (3) Different subjects employ different strategies to maintain performance. In particular, the subject's willingness to trade off accuracy against speed is an aspect of performance which should be further investigated (Shillito et al., 1974, p. 1032).

Although choice reaction time is somewhat different from the simple reaction time to be studied in the present experiment, the Shillito et al. (1974) comment on the subject's "willingness to trade off accuracy against speed" was a feature subsumed in the signal detection model in the response accuracy aspect.

Huntley (1973) found that at the blood alcohol concentration of 0, 50, 100 mg per 100 ml (after 0, .74, and 1.21 ml of 95% ethanol per kg of body weight), reaction-time became longer as blood alcohol concentrations were increased. Tharp, Rundell, Williams & Lester (1974), Tharp (1975) and Huntley (1974) found that acute alcohol intoxication slowed verbal RL more when the stimulus response relationship was newly learned than when it was highly familiar; it was also found that the degree of impairment was positively correlated with the number of choice or stimulus response alternatives available.

Signal Detection Theory and Response Discriminability

Signal detection theory is one of the recent significant contributions to traditional psychophysics. It evolved from

problems in radio and telephone communication and radar (Green & Swets, 1966) and has developed into a sophisticated methodology in studying decision and judgment. It does not rely on the usual relationship between two kinds of response, hits and false alarms. Signal detection theory instead measures two aspects of an observer's decision, sensitivity and response bias, which are the two parameters of the signal detection index. According to McNicol (1972), sensitivity, d', is:

> ...the value of the signal distribution mean, measured in SD units of the noise distribution, when the noise distribution mean is equal to zero and both distributions are Gaussian and have S. D. = 1.

$$d' = \frac{\bar{X}_s - \bar{X}_n}{\sigma_n}$$

(McNicol, 1972, pp. 56-7).

According to Hochhaus (1972), d' = ABS (HR) - ABS (FAR), and B = ORD (HR) / ORD (FAR), where HR = P_{sn} (A), the proportion of signals presented that are affirmed by the subject, and FAR = P_n (A), the proportion of times that a signal is reported when no signal was actually presented.

The d' index is a Type-1 d' which measures the sensitivity of stimulus discrimination, while the sensitivity index for the response discrimination, as in the case of giving confidence ratings to one's recall response, the Type-2 d', or d", will be used in the present experiment (Clarke, Birdsall & Tanner, 1959). The application of the Type-2 analysis to recall tasks with confidence ratings has

been explored by Pollock and Decker (1958), Bernbach (1967, 1972), Bernbach and Bower (1970), and Hochhaus and Antes (1973), for examples. Pollock (1959) differentiates between "signal discriminability" for Type-1 d', and "response discriminability" for Type-2 d' (d").

Hochhaus (1970) used a probe paired-associates paradigm to apply the Type-2 d" to a multiple-choice recognition task. He found that variations in d" matched changes in probability correct, and that frequent correct guesses lower response discriminability. To relate this observation to the effect of alcohol on response confidence, it would be reasonable to assume that subjects in the ascending blood alcohol curve would tend to guess more. However, since their probability correct will be expected to be lowered, response discriminability will probably not be lowered dramatically.

Bernbach (1967) has noted that d" is constant in most memory studies, and is unaffected by factors which strongly affect probability correct (such as serial position and repetitions). In word perception tasks, d" loses its invariance, but still appears independent of probability correct (Hochhaus and Antes, 1973). Overall, the d" index appears to measure the subject's awareness of his own prior accuracy, an ability Hochhaus and Antes (1973) called "knowing that you know" (cf. Hart, 1965).

Because the connection between feelings of confidence and alcohol consumption has been noted, though not carefully

documented empirically (e.g., Mendelson, 1969), and because recall confidence ratings are closely related to response latency (Murdock, 1968), the investigation of d" in connection with alcohol use appears deserving of further investigation.

The direct prediction of higher confidence in connection with added alcohol dosage must be tempered by the fact that alcohol may increase response latency. Since there is the possibility that a person might base his recall confidence on his own response latency, it is highly likely that increasing response latency may lead to a cancellation effect on increases in recall confidence brought about by alcohol induced feelings of confidence and euphoria.

Prior application of the signal detection paradigm to analyze the effect of alochol on recall is absent. Schneider and Carpenter (1969) studied the effect of alcohol on auditory signal detection and found that alcohol strongly affected the nonsensory aspects of performance, such as response variability, judgmental inconsistency, and task complexity. The Schneider and Carpenter (1969) study was about stimulus conditional signal discriminability (Type-1 d'), while in the present study, the response conditional response discriminability (Type-2 d') was analyzed.

Hypotheses

The present experiment attempted to test a major conceptual hypothesis, namely, that alcohol is a depressant or

anasthetic agent which has a dominating inhibitory effect on the subject's nervous system. Although the possibility of euphoria could not be completely ruled out, its nature and extent still remained to be seen. Specifically, the present experiment was designed to gather further evidence concerning questions of the effects of alcohol on recall, recall confidence, response latency, and mood.

A basic question is, does alcohol impair short-term memory in a low blood alcohol level range of 0.02% to 0.04%, and more so in a moderate blood alcohol level range of 0.06% to 0.08%? If so, is the impairment more pronounced on the ascending limb of the blood alcohol curve than on the descending limb of the blood alcohol curve in both the low and the moderate blood alcohol level ranges?

From the evidence gathered in the literature review, these doses of alcohol, hence the respective blood alcohol level ranges, have not been studied before. However, recent experimental evidence (Jones, 1972, 1973; Jones & Vega, 1972) pointed to the direction that while the low dose may or may not have any effect on memory, the moderate dose should have a higher probability of impaired short-term memory. Thus, it was hypothesized that in general the number of words corectly recalled for the placebo group would be higher than that of the low dose group, which would in turn be higher than that of the moderate dose group. Specifically, on the ascending limb of the subject's BAC, mean number of words correctly recalled for the placebo group is expected to be

higher than that of the low dose group, which would in trun be higher than that of the moderate dose group. On the descending limb of the subject's blood alcohol curve, mean correct recall of the placebo group is expected to be higher than that of the moderate dose group, while mean correct recall between the low dose group and placebo group is expected to be invariant.

A second question central to the effect of alcohol on human behavior is that of response latency. Does alcohol increase a subject's latency in general? If so, does the impairment on latency become more pronounced on the ascending limb of the subject's BAC than on the descending limb? In accordance with the notion that alcohol is an anesthetic drug, it was hypothesized that the mean response latency for the moderate dose group is longer than that of the low dose group, which in turn is longer than that of the placebo group. While it is expected that this would also be true on the ascending limb of the blood alcohol curve, it was hypothesized that on the descending limb of the subject's blood alcohol curve, mean response latency of the moderate dose group is invariant with that of the low dose group, but the mean response latency of both the moderate dose group and the low dose group is expected to be longer than that of the placebo group.

However, a fascinating question would then be in order. Might response latency, taken as an index of recall confidence, be a more accurate indicator of correct recall than

would confidence ratings themselves? To what extent would response discriminability (d") of the moderate dose and low dose groups be different from that of the placebo group? Would the effects of alcohol tend to shrink the subject's d" (an indicator of anesthetic effect) or to boost the response discriminability toward the other extreme (a sign of pseudostimulation) with dose response differences? Since there has not been any prior research in this area, the results will be thoroughly analyzed in this explorative area.

A final question of the relationship between mood and behavior must be geared into the analysis of the above major dependent variables. While there has been some indication that alcohol had dose response differences on mood change, the quality and extent of change has yet to be examined. Consistent with the conceptual hypothesis that alcohol is a depressant it was hypothesized that the subject's mean score on the depression scale of the MAACL increases as the dose increases.

CHAPTER IV

METHOD

Subjects

Eighteen male adult (age 21 or above) students (16 undergraduate and 2 graduate) at Oklahoma State University volunteered as subjects. Undergraduate students received extra credit for their participation. Students volunteering to participate in the present experiment were required to fill out a questionnaire (Appendix A) regarding their general health and drinking history. Only those students who were nonalcoholics, not abstainers, but were moderate social drinkers were used as subjects. Written informed consent statements (Appendix B) were signed by each subject. Since all subjects were willing to release personal medical information, a written consent was incorporated into the informed consent statement, the signing of the consent statement made it possible for an M.D. to access the subject's individual health record kept by the University Clinic, to ensure that each subject was physically and mentally suitable to participate in the present alcohol experiment. Only those who were drug-free, and who were free from any current or potential disease sensitive to alcohol were finally given

the option to serve as subjects in the present experiment. Subjects were requested to stop using any medication 24 hours prior to the experimental session, so as to avoid any possible drug-alcohol interaction effects.

Each subject was informed of his right to discontinue participation in the experiment at any time. They were informed about the use of alcohol in the experiment, but were not to be told the specific amount. A post-experimental debriefing was held shortly after the completion of the experiment.

Design

The 18 subjects were randomly assigned to either the placebo group, the low dose group, or the moderate dose group in sequential order, so that there were six students per group.

The design of the experiment was a split-plot, type SPF-3.22 (Kirk, 1968, p. 298). The between-subject treatment was the dose of alcohol (placebo, low dose, moderate dose). The within-subject treatment was the two limbs (ascending and descending) of blood alcohol curve of the subjects; there were two blocks of 20 paired-associates lists within each of the ascending and descending limbs of the blood alcohol curve.

Task and Apparatus

Each subject learned and recalled a total of 80 A-B paired-associate lists divided into four blocks of 20 lists

each. Two blocks were learned and recalled during the ascending blood alcohol curve, the other two during the descending blood alcohol curve. There were five A-B word pairs in each list, followed by a single probe. The A-B members of each list were randomly paired from a word pool selected from Kuchera and Francis (1967). A PL/1 computer program was used to print on white paper the instructions, two practice lists, and 80 paired-associates lists. Following each list there was one probe--an A - member generated randomly by the computer program from among the five A - members in the list. To respond, the subject had to speak the corresponding B - member, and announce his confidence in the accuracy of his response according to a four-point rating category (definitely right, possibly right, possibly wrong, definitely wrong). The material was presented by a memory drum (Model IBM, Lafayette) at the rate of 1 pair/2 sec.. The subject was seated in a separate room watching the television screen on which were shown the lists to be learned, as these were relayed via a Sony videotape recorder and camera. The television set was placed at approximately 122 cm from the subject at eye level height. The subject was instructed to respond as quickly and accurately as he could, although his actual response was self-paced.

A microphone was suspended from the subject's neck; the microphone was connected to a voice key (Gerbrands). A four bank timer (Model 52013, Lafayette) was programmed to activate the memory drum, so that the latter advanced one

line every two sec., in the order of "Trial No.", the five A-B pairs, and the probe. On the thirteenth second when the probe was presented, simultaneously the voice key was reset and the clock (Hunter) was started. The subject's voice response stopped the clock via the voice key. Then the four bank timer recycled the same sequence for the next trial. A portable tape recorder (Sony TR-50) was placed in front of the subject to record his responses. As soon as the subject responded verbally, the experimenter within two sec. pressed the manual control button connected to the four bank timer to initiate the next cycle of presentation. While the subject was learning the five paired-associates lists, the experimenter recorded the response latency as shown on the dials of the clock and then reset the clock (completed within five sec.). A breathalyzer (Stephenson Model 900) was used to measure the subject's blood alcohol level to the nearest 0.0025%. The laboratory layout is shown in Figure 1.

Administration of Alcohol

The subjects were instructed not to consume any alcoholic beverage within 24 hours of the experimental session, and to fast four hours prior to testing. Alcohol (190 USP ethanol) was mixed with commercial orange drink at a 1:4 ratio. Thus, for each alcoholic beverage, there was 20% alcohol, and 80% orange drink, plus one standard ice cube.

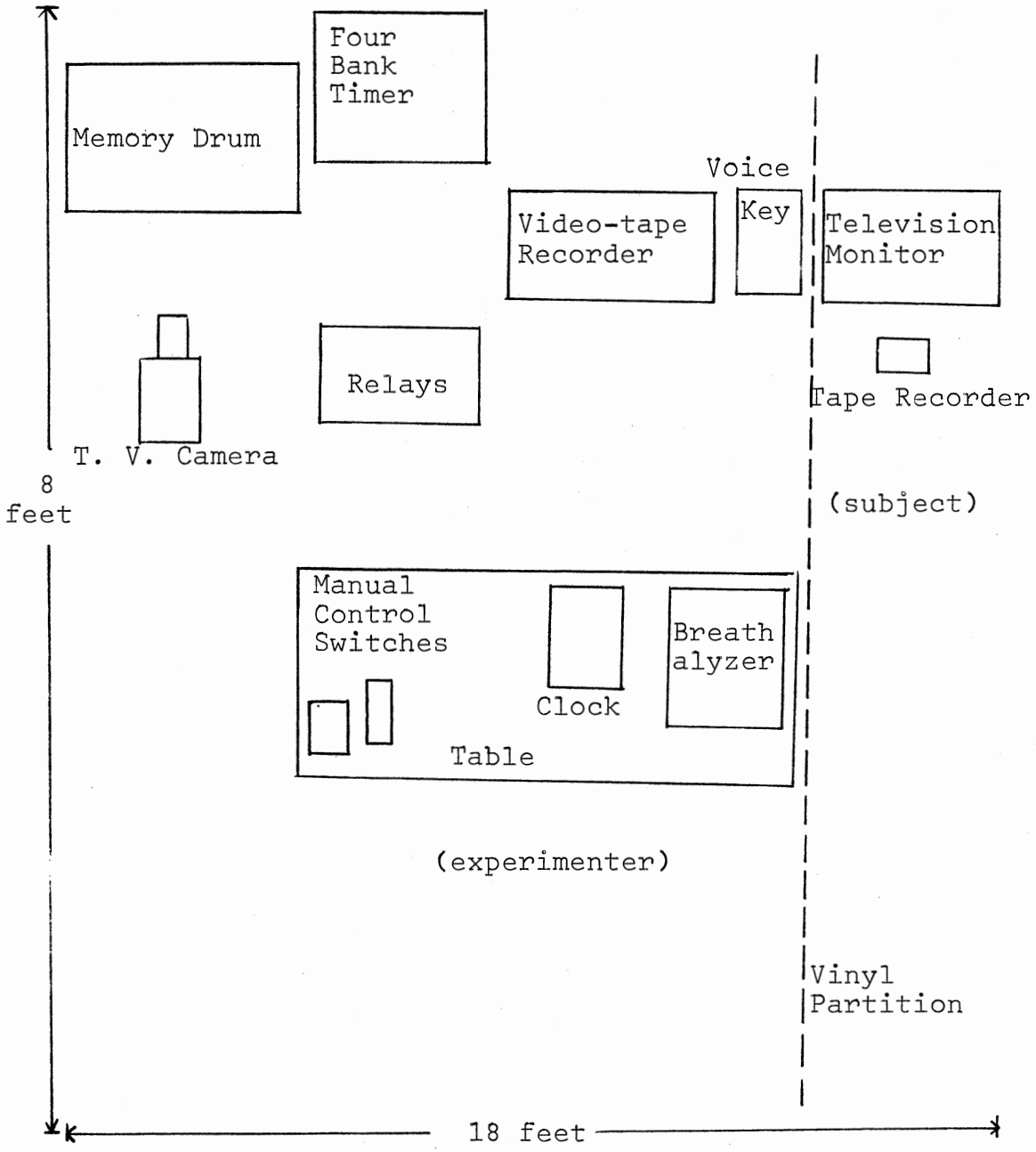

Figure 1. Laboratory Layout

Each subject in the low dose group received 0.46 ml of alcohol per kg of his body weight, those in the moderate dose group each received .92 ml/kg alcohol mixed with four times that amount in chilled orange drink. The six subjects in the placebo group were split into two subgroups of three, each placebo subject was matched with two subjects in either the low dose or the moderate dose groups, to use the base alcohol weight (0.46ml/kg or 0.92 ml/kg) times the placebo subject's own weight to get the amount of orange drink. Four ml of alcohol was floated on top of the placebo drink in order to facilitate deception regarding the subjects knowledge of his treatment as a control group member.

Subjects in the low dose group were given five min. to consume the alcoholic beverage, while those in the moderate dose were given 10 min. to consume the drink. In each case, half of the alcoholic beverage had to be consumed by 2.5/5 min. The two subgroups of placebo subjects were matched again with their corresponding counterparts in drinking times.

Procedure

Each subject was tested individually. When a subject showed up, he was requested to sign on the top of the data sheet certifying that he had not taken any medication or drug. Then he was weighed (shoes and overcoat off), took the sobriety test and was given a sheet of instructions to read. Any doubts regarding the procedures of the experimental

tasks were clarified. Then the subject went through the practice trials, and after that took a practice breath test and the MAACL. When he had completed the MAACL he was given the appropriate drink which he had to consume in front of the experimenter within the appropriate time. Then, he was asked to rinse his mouth with a standard glass of tap water to clear any residual alcohol from his mouth. At about 15 min. after the beginning of the drink, he was administered his second breath test. Subjects began their learning and recall task when their BALs reached the predetermined range. It was expected that subjects in the low dose group would reach an average peak of 0.05%, and that of the moderate dose group an average peak of 0.09%. Low dose group subjects, therefore, began their task on the ascending limb of their BAC at the range of 0.02 to 0.04%, while subjects in the moderate dose group began their task at the range of 0.06 to 0.08%. There was a variable break waiting for the expected peak blood alcohol level to appear. After the appearance of the peak blood alcohol levels, subjects in the low dose group began their task on the descending limb of their blood alcohol curve at the range of 0.04 to 0.02%, while those in the moderate dose group began their task at the range of 0.08 to 0.06%. In each case, the subject had to take another breath test and MAACL after he had finished each 20-trial block.

The subjects in the placebo group began their task immediately after they had taken their second breath test at

the completion of the drink. They also took a breath test after they had completed each 20 trials. They were again split into two groups, in a yoke control design, to match with either the low dose or the moderate dose groups in taking a break between the ascending and the descending limbs.

The experimental task took about an hour. Then a modest snack was provided immediately after the subject had completed the experimental tasks and taken the breath test and MAACL. A subject was dismissed only when his blood alcohol level returned to 0.00%. Subjects were instructed to study or rest during the waiting period, which took about three hours for subjects in an alcohol condition. Subjects were not allowed to smoke, to eat, or to drink anything before the completion of the experimental task.

CHAPTER V

RESULTS

The results were mainly analyzed by separate two-way or three-way analyses of variance. In the case of *a priori* comparisons, the *t* statistic was used regardless of the significance of *F* associated with the independent variable. However, since some of the hypotheses were exploratory in nature, and, in order to look for potential treatment differences, a liberal view was adopted regarding *a posteriori* comparisons, hence appropriate caution should be exercised in the interpretation of the results in these cases.

Correct Recall

The number of words correctly recalled was analyzed in a 3x2x2 analysis of variance (Alcohol X Trials X Task), the results of which are summarized in Appendix K. A table of means and standard deviations for the number of words correctly recalled on the ascending and descending limbs of the blood alcohol curve for each alcohol condition is presented in Appendix F. As predicted, there were significant differences in the number of words correctly recalled between the three alcohol conditions, F (2, 15) = 4.97, $p < .025$. Out of every 20 trials, the mean number of words correctly recalled

was 9.63 by the placebo group, 6.88 by the low dose group, and 7.46 by the moderate dose group. As hypothesized, the placebo group correctly recalled a greater number of words than the low dose group, t (15) = 2.99, $p < .005$ (one-tailed), and the moderate dose group, t (15) = 2.36, $p < .025$ (one-tailed). However, the finding that the low dose group recalled somewhat worse than the moderate dose group was quite unexpected, but the difference was not significant, t (15) = .64, $p > .05$. Overall, limb effect did not reach the .05 level, F (1, 15) = 3.58, $p > .05$. However, on the ascending limb of the subject's blood alcohol curve, the placebo group recalled significantly better than the low dose group, t (15) = 2.68, $p < .01$ (one-tailed), and the moderate dose group, t (15) = 2.05, $p < .05$ (one-tailed); the low dose group did worse than the moderate dose group, but the difference was not significant, t (15) = .95, $p > .05$. On the descending limb of the subject's blood alcohol curve, mean correct recall of the placebo group was significantly greater than that of the low dose group, t (15) = 2.52, $p < .025$ (one-tailed), and the moderate dose group, t (15) = 2.06, $p < .05$ (one-tailed); again, the low dose group had a nonsignificantly smaller mean correct recall than that of the moderate dose group, t (15) = .47, $p > .05$. Figure 2 shows the mean correct recall of the placebo, low, and moderate groups respectively on the ascending and descending limbs of the subject's blood alcohol curve.

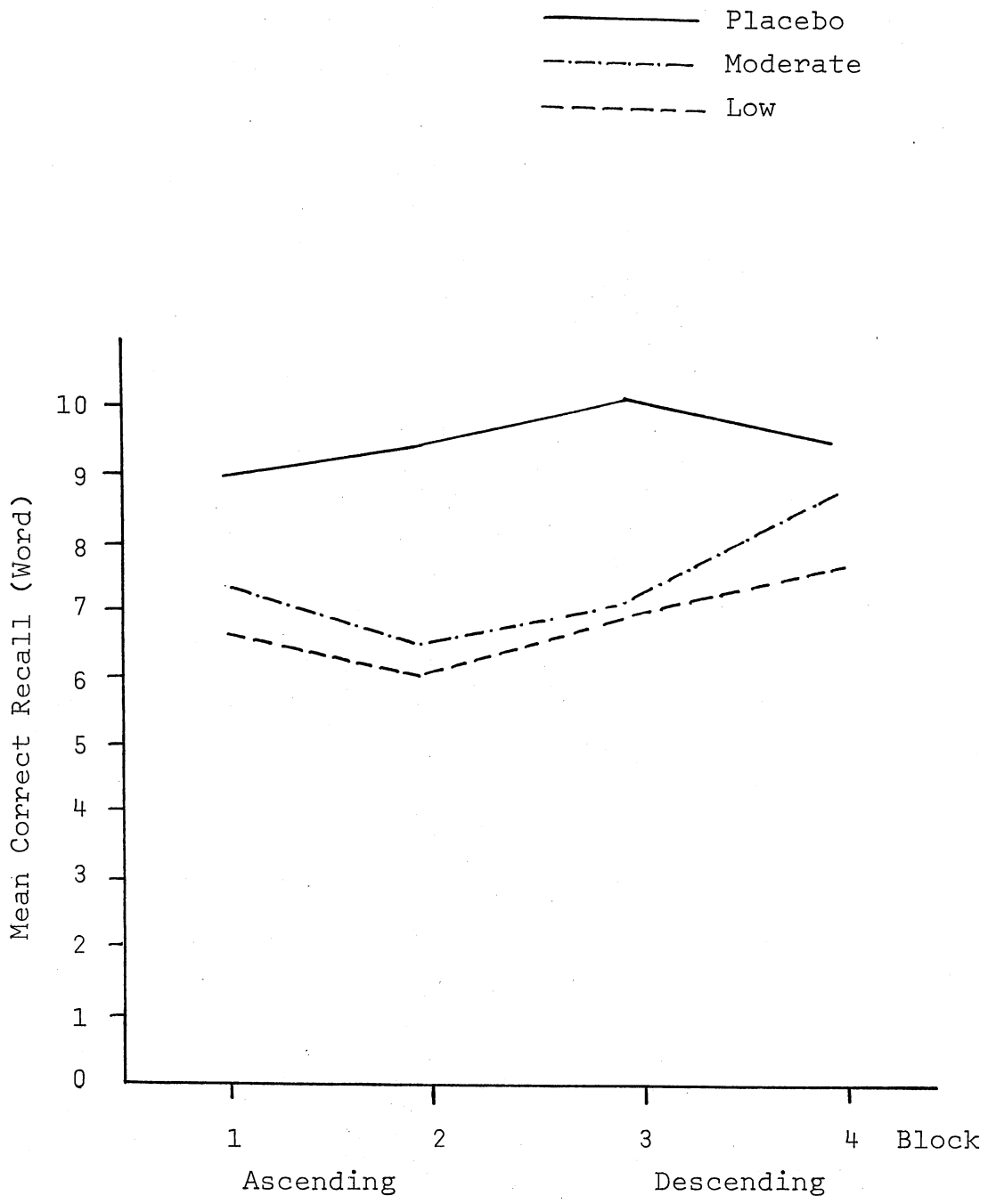

Figure 2. Mean Correct Recall on the Ascending and Descending Limbs of the Blood Alcohol Curve

Response Discriminability

Response discriminability (d") for each block of learning and recall task was computed by first collapsing over the data of all six subjects. Group hit rates (HR) and false alarm rates (FAR) were then plotted on "normal-normal" graph paper (codex no. 41,453) to obtain the "receiving-operating characteristic" (ROC) curve. The interception of the ROC curve with the minor diagonal of "normal-normal" graph paper determined the final HR and FAR which yielded the d" value from the Hochhaus (1972) table. A test for linearity of the slopes of the ROC curves showed no significant difference from 1, t (11) = 1.52, $p > .2$ (two-tailed). The mean slope for all subjects was .87. Figure 3a shows the change of d" between blocks of learning and recall tasks on the ascending and descending limbs of the subject's blood alcohol curve. To obtain a more stable value, d" was again computed by further combining the data of tasks 1 and 2, and that of 3 and 4. The d" of the ascending limb and that of the descending limb were obtained respectively through the graphical procedure described earlier. These d" by limb values are shown in Figure 3b. The G-test (Gourevitch & Galanter, 1967) was computed to provide standard deviation estimates for *a posteriori* multiple comparisons. The results showed that within the low dose group, response discriminability was significantly higher on the descending limb than on the ascending limb,

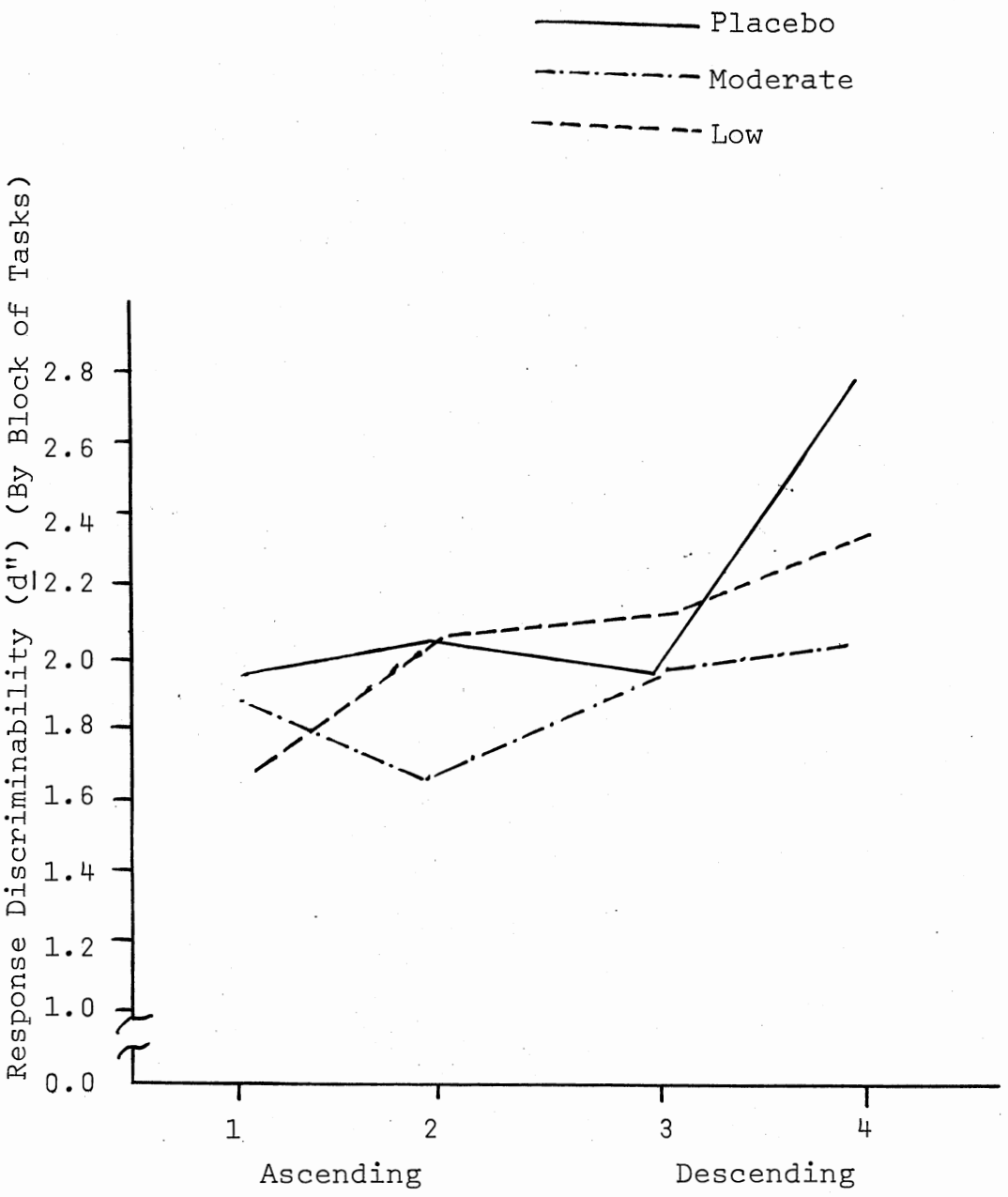

Figure 3a. Response Discriminability (d") on the Ascending and Descending Limbs of the Blood Alcohol Curve (By Block)

$G = 3.1860$, $p < .002$ (two-tailed), while it was invariant within the placebo group and the moderate dose group, respectively.

Between limb comparisons across alcohol conditions showed only significant difference in response discriminability between the placebo and the low dose groups on the ascending limb, $G = 3.57$, $p < .002$ (two-tailed), the differences between the rest of the possible meaningful comparisons were nonsignificant.

Recall Confidence

Since the small number of correctly and incorrectly recalled words were too unstable for the calculation of recall confidence in a 20-trial block, a weighted method was adopted. Mean recall confidence was computed in each 20-trial block (task) by multiplying the total number of correct and incorrect recall in each of the 4-category confidence ratings of "Definitely Right, Possibly Right, Possibly Wrong, and Definitely Wrong," with 4, 3, 2, and 1, respectively; the sum of these four products (divided by the total number of recall attempts) yielded the mean recall confidence for that block of 20-trial learning and recall task. Mean recall confidence was analyzed in a 3x2x2 analysis of variance (Alcohol X Limb X Task), the results of which are summarized in Appendix M. No significant differences were found either between alcohol conditions, or between limbs, or between tasks. Figure 4 shows the mean recall confidence between

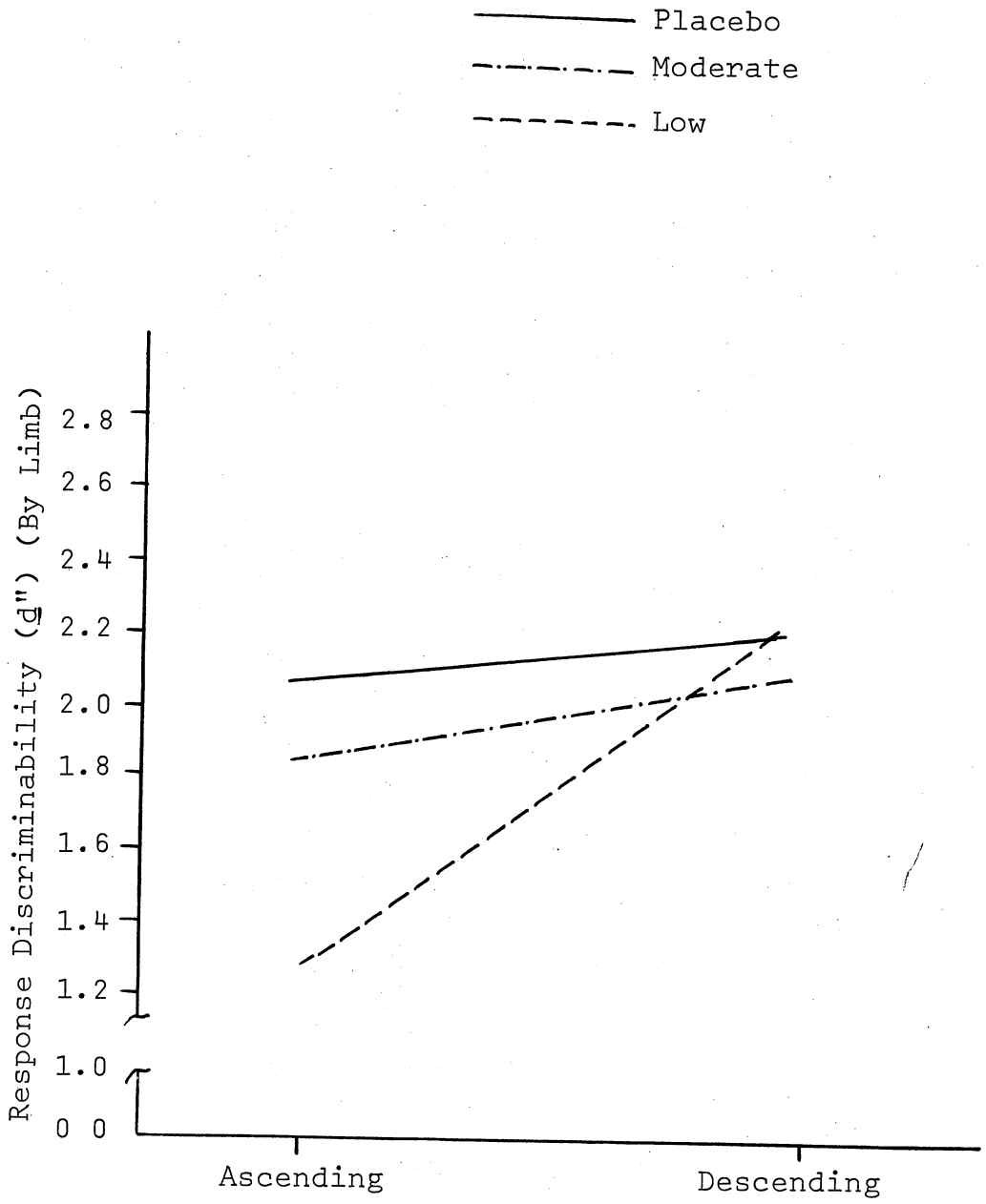

Figure 3b. Response Discriminability (d") on the Ascending and Descending Limbs of the Blood Alcohol Curve (By Limb)

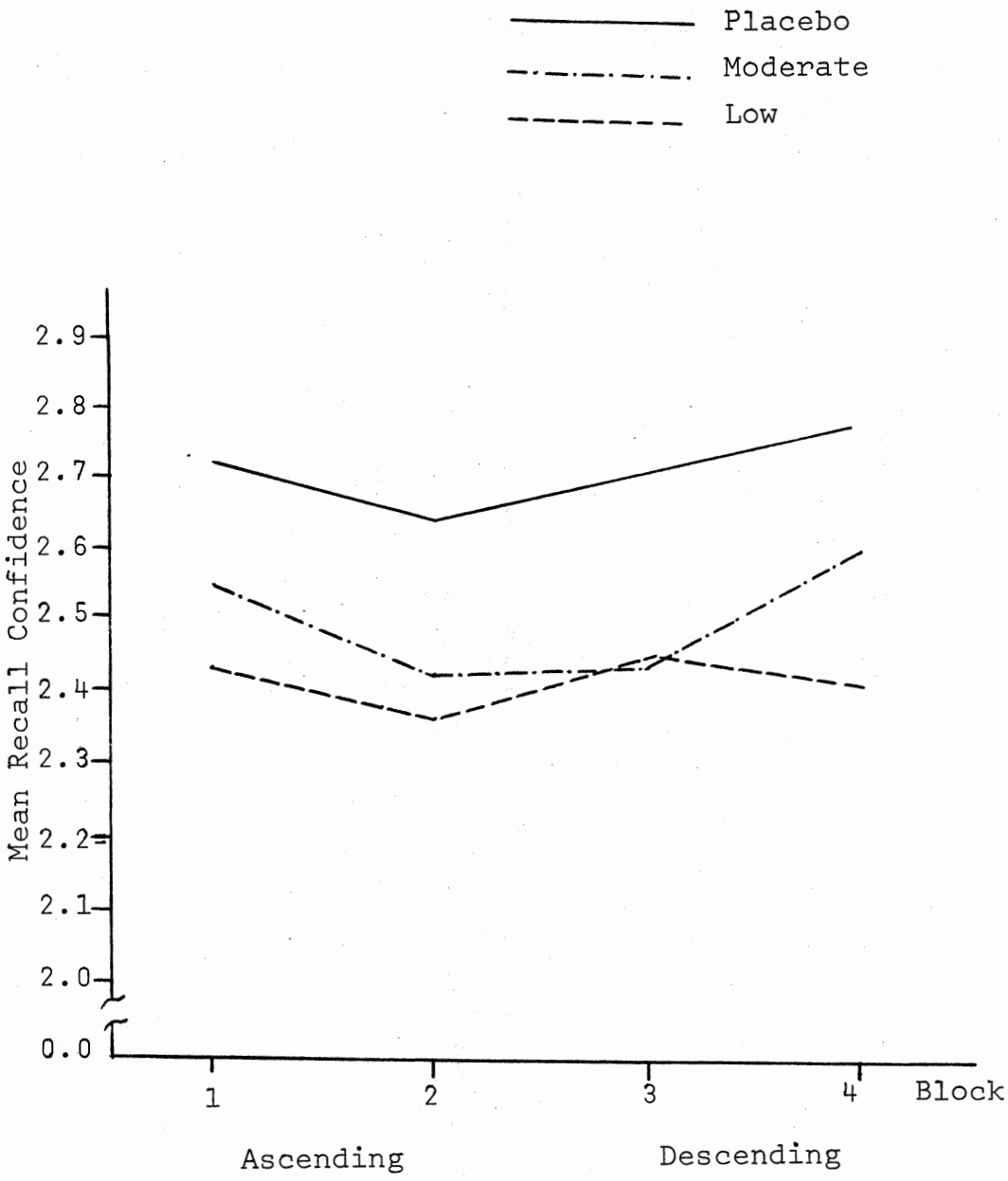

Figure 4. Mean Recall Confidence on the Ascending and Descending Limbs of the Blood Alcohol Curve

tasks on the ascending and descending limbs of the blood alcohol curve.

Since the final HR and FAR values obtained from the normal-normal graphic paper used to find the d" would, by definition, always yield a Beta = 1, it was decided to use the middle criterion (i.e., the dichotomy between "Possibly Right" and "Possibly Wrong") to obtain an appropriate index for response bias from the Hochhaus (1972) table. Beta averaged across subjects in each 20-trials of the ascending and descending limbs of the blood alcohol curve is presented in Figure 5.

Response Latency

Mean response latency in each block of 20-trials was analyzed in a 3x2x2 analysis of variance (Alcohol X Limb X Task), the results of which are summarized in Appendix L. A table of means and standard deviation of the mean response latency for each limb and alcohol condition is presented in Appendix G. The mean response latency was 2.23 sec., 2.20 sec., and 2.42 sec. for the placebo, low dose, and moderate dose groups, respectively. The nonsignificant differences in mean response latency between the alcohol conditions was unexpected, F (2, 15) = .24, $p > .05$. Thus, there was no between group comparison of limb effect on response latency. However, there was a significant difference in limb effect within the individual alcohol condition itself, F (1, 15) = 6.32, $p < .025$. In the placebo group there was no signifi-

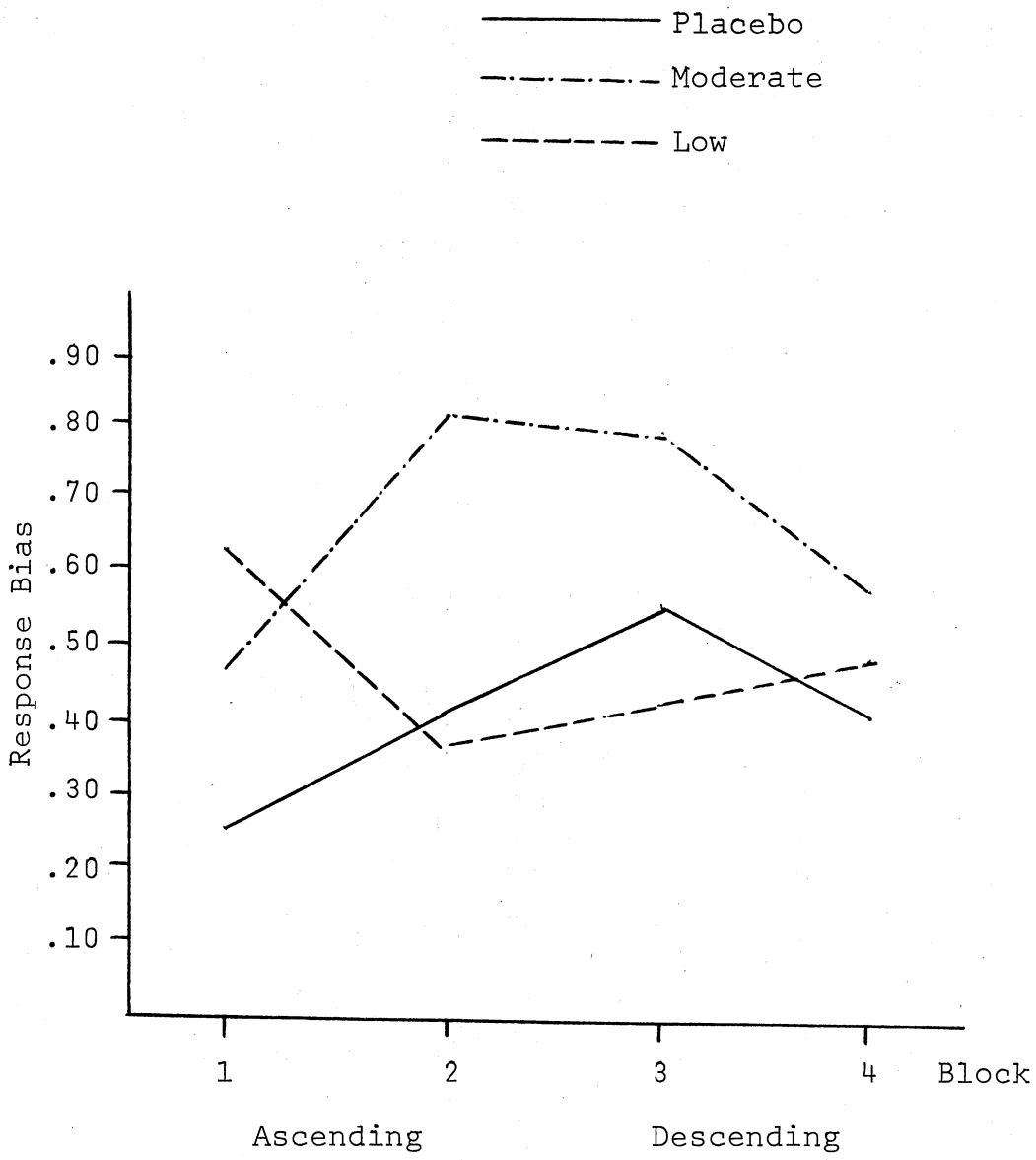

Figure 5. Response Bias (Beta) on the Ascending and Descending Limbs of the Blood Alcohol Curve

cant difference in mean response latency between limbs, t (15) = .47, $p > .05$. However, mean response latency was longer on the ascending limb than on the descending limb for both the low dose group, t (15) = 3.09, $p < .005$ (one-tailed), and the moderate dose group, t (15) = 2.60, $p < .025$ (one-tailed). Figure 6 shows the mean response latency on the ascending and descending limbs of the subject's blood alcohol curve.

In order to examine the possible relationship between response latency and recall confidence, the following two indices were computed. An index of discriminability based on latency was computed by grouping the raw scores of response latency into four distinct groups: 0.8-1.3 sec., 1.3-2.0 sec., 2.0-3.0 sec., and 3.0 sec. and up. These four groups of response latencies were taken, for the sake of analysis here, to represent the subject's bases or criteria for responding with "Definitely Right, Possibly Right, Possibly Wrong, or Definitely Wrong," respectively. The index of discriminability based on response latency (d'_L) was computed for each two blocks (40 trials) for each subject. Since the slopes of the group ROC curves for response latency was not significantly different from 1, t (5) = 1.94, $p > .1$ (two-tailed), the four distinct groups of latency data in each limb were collapsed into two categories (the sum of the correct "Definitely Right" and "Possibly Right" divided by the total number of correct recall as HR, the sum of incorrect "Definitely Right" and "Possibly Right" divided by the

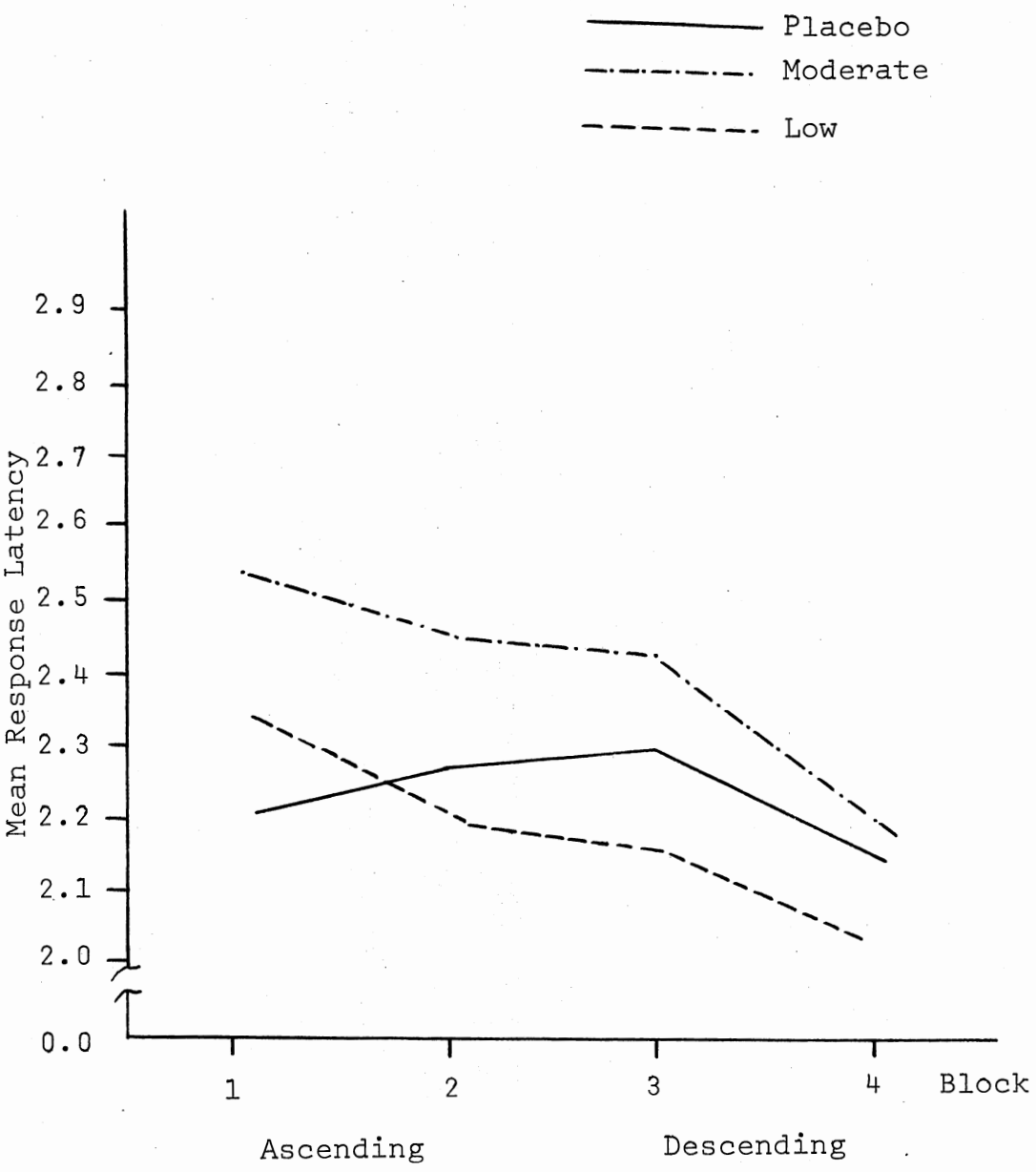

Figure 6. Mean Response Latency on the Ascending and Descending Limbs of the Blood Alcohol Curve

total number of incorrect recall as the FAR). d''_L was then obtained from the Hochhaus (1972) table. The d''_L was further analyzed in a 3x2 analysis of variance (Alcohol X Limb), the results of which are summarized in Appendix N. There was no significant difference in the index of discriminability based on response latency between the three alcohol conditions. However, the within group limb effects were significantly different, F (1, 15) = 4.97, $p < .05$. The index of discriminability based on response latency was significantly higher on the descending limb than on the ascending limb for both the placebo group, t (15) = 2.37, $p < .05$ (two-tailed), and the moderate dose group, t (15) = 3.06, $p < .01$ (two-tailed). As for the low dose group, the index of discriminability based on latency was invariant between the two limbs. Figure 7 shows the index of discriminability based on response latency, on the ascending and descending limbs of the subject's blood alcohol curve.

Because of the "0" and "1" values in the cumulative table for each 20-trial block hindered the calculation of initial HR and FAR, in other words, there were not enough data points to determine individual ROC curves, a binary confidence (d''_{bin}) was computed for each subject in each limb (40 trials). In each case the HR was obtained by dividing the sum of the correct "Definitely Right" and "Possibly Right" with the total number of correct recall; in like manner, the FAR was obtained by using the sum of the incorrect "Definitely Right" and "Possibly Right" by the total number

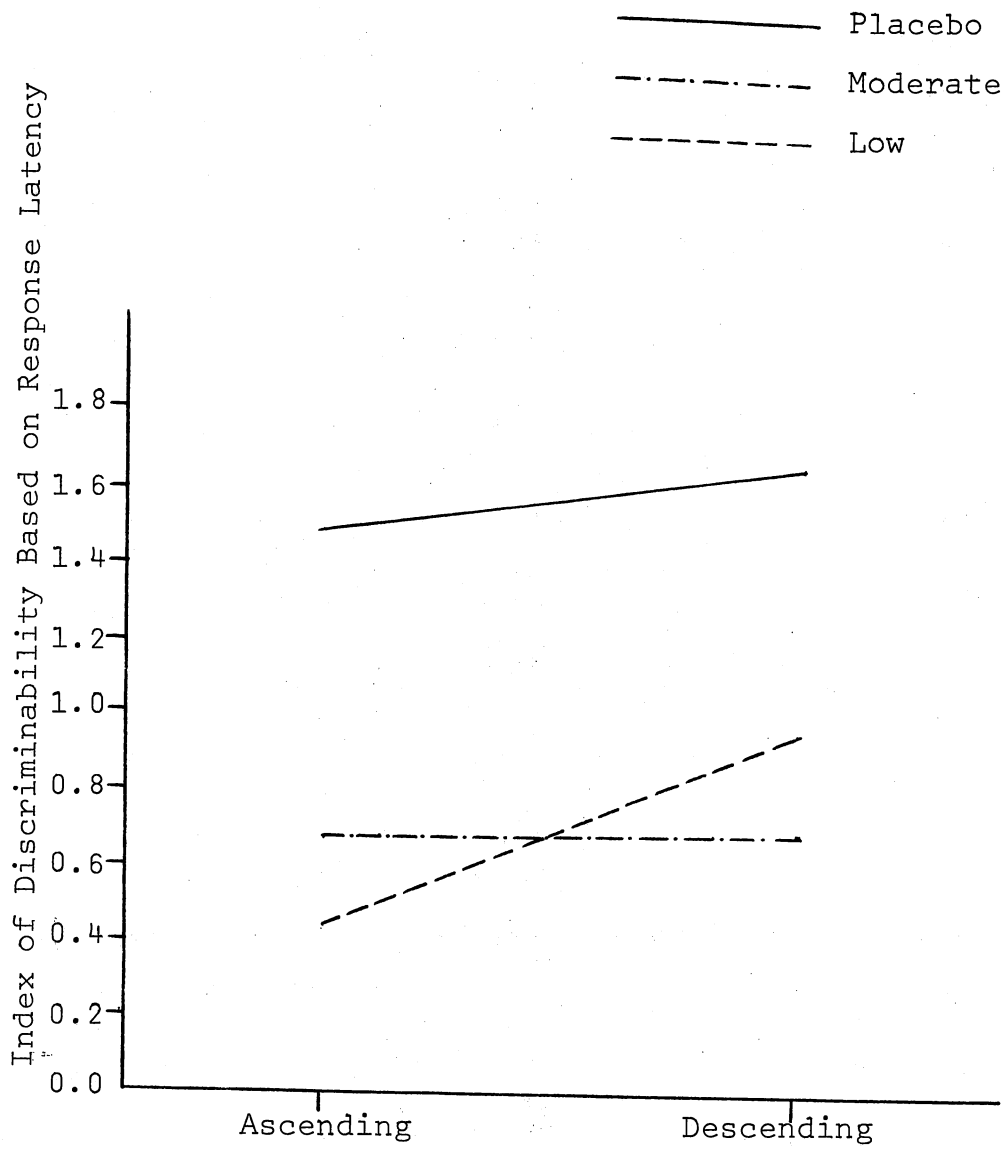

Figure 7. Index of Discriminability Based on Response Latency on the Ascending and Descending Limbs of the Blood Alcohol Curve

of incorrect recall. The $\underline{d}''_{bin}$ value was then obtained from the Hochhaus (1972) table. The $\underline{d}''_{bin}$ of the individual subjects was analyzed in a 3x2 analysis of variance (Alcohol X Limb), the results of which are summarized in Appendix O. No significant differences of between alcohol conditions or between limbs were observed. Figure 8 shows the response discriminability based on binary confidence.

$\underline{d}''_{bin}$ and $\underline{d}''_{L}$ for individual subjects were compared in all possible pairs. In general, $\underline{d}''_{bin}$ was significantly higher than $\underline{d}''_{L}$ beyond the .002 level as a whole, and on both limbs (sign test, $\underline{p} < .002$).

Mood

Only raw socres were used for the anxiety, depression, and hostility scales of the Multiple Affect Adjective Check List. Separate 3x5 analyses of variance (Alcohol X MAACL Administration) were computed in the analysis of each scale, the results of which are summarized in Appendix P (Anxiety Scale), Appendix Q (Depression Scale), and Appendix R (Hostility Scale), respectively. A table of means and standard deviations for the Anxiety, Depression, and Hostility scales are presented in Appendix H, Appendix I, and Appendix J, respectively. No significant differences were found between alcohol condition and MAACL administration for the Anxiety scale as well as for the Hostility scale. However, for the Depression scale, there was a significant difference between alcohol conditions, $\underline{F}$ (2, 15) =

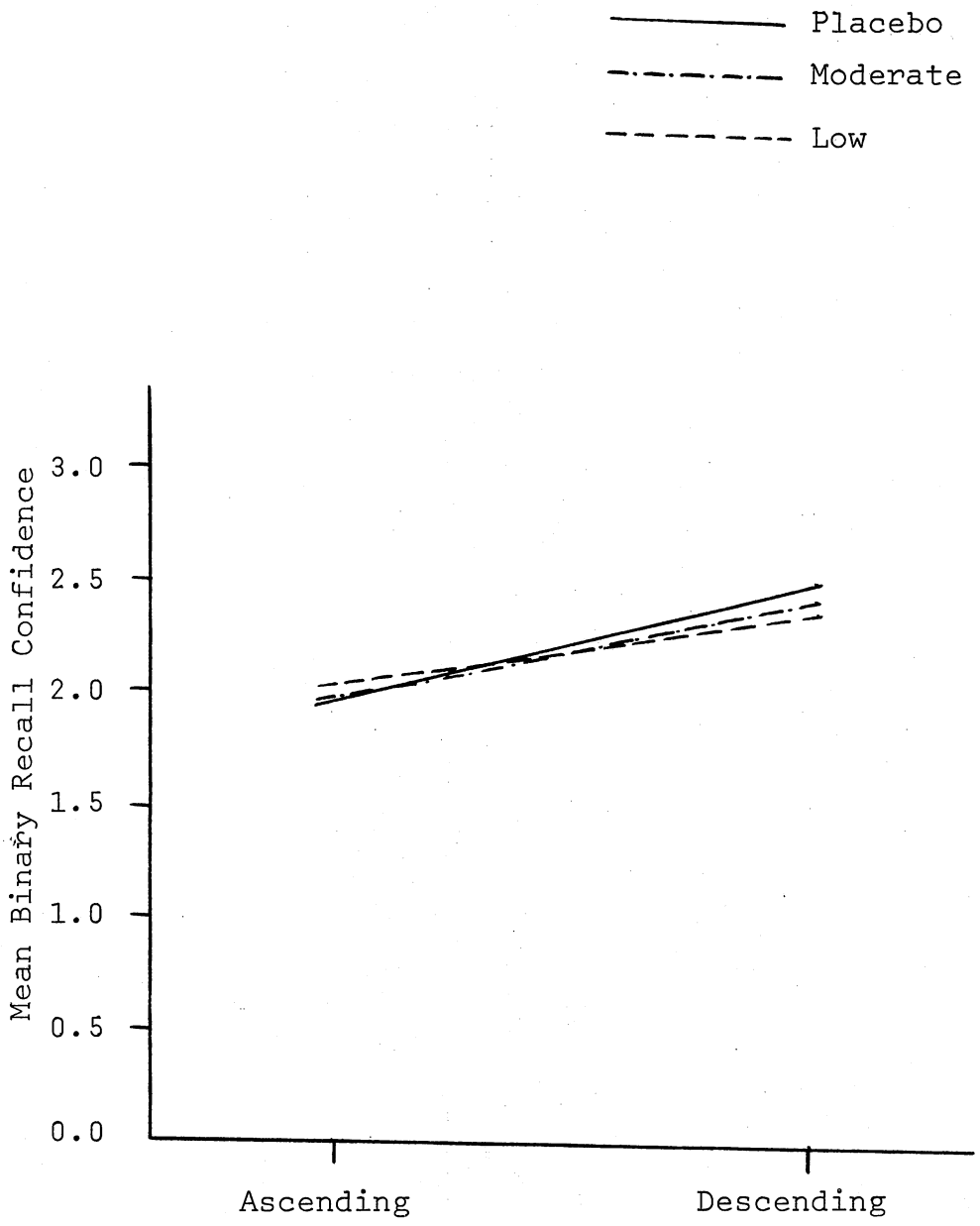

Figure 8. Mean Binary Recall Confidence on the Ascending and Descending Limbs of the Blood Alcohol Curve

4.89, $p < .025$. The Depression score was significantly higher in the low dose group than the placebo group, t (15) = 2.36, $p < .05$ (two-tailed), and was also significantly higher in the moderate dose group when compared with the placebo group, t (15) = 2.48, $p < .05$ (two-tailed). The Depression score was invariant between the low dose group and the moderate dose group, t (15) = .13, $p > .05$. Differences in depression score must be cautiously interpreted, however, in light of significant pre-treatment differences in this index between the placebo group and the alcohol groups, F (2, 15) = 5.28, $p < .05$. Compared with the placebo group, the mean depression score was higher on the low dose group, t (10) = 2.89, $p < .02$ (two-tailed), and on the moderate dose group, t (10) = 2.72, $p < .05$ (two-tailed). Figures 9, 10, and 11 show the mean raw Depression scores, Anxiety scores, and Hostility scores, respectively between administrations of the MAACL.

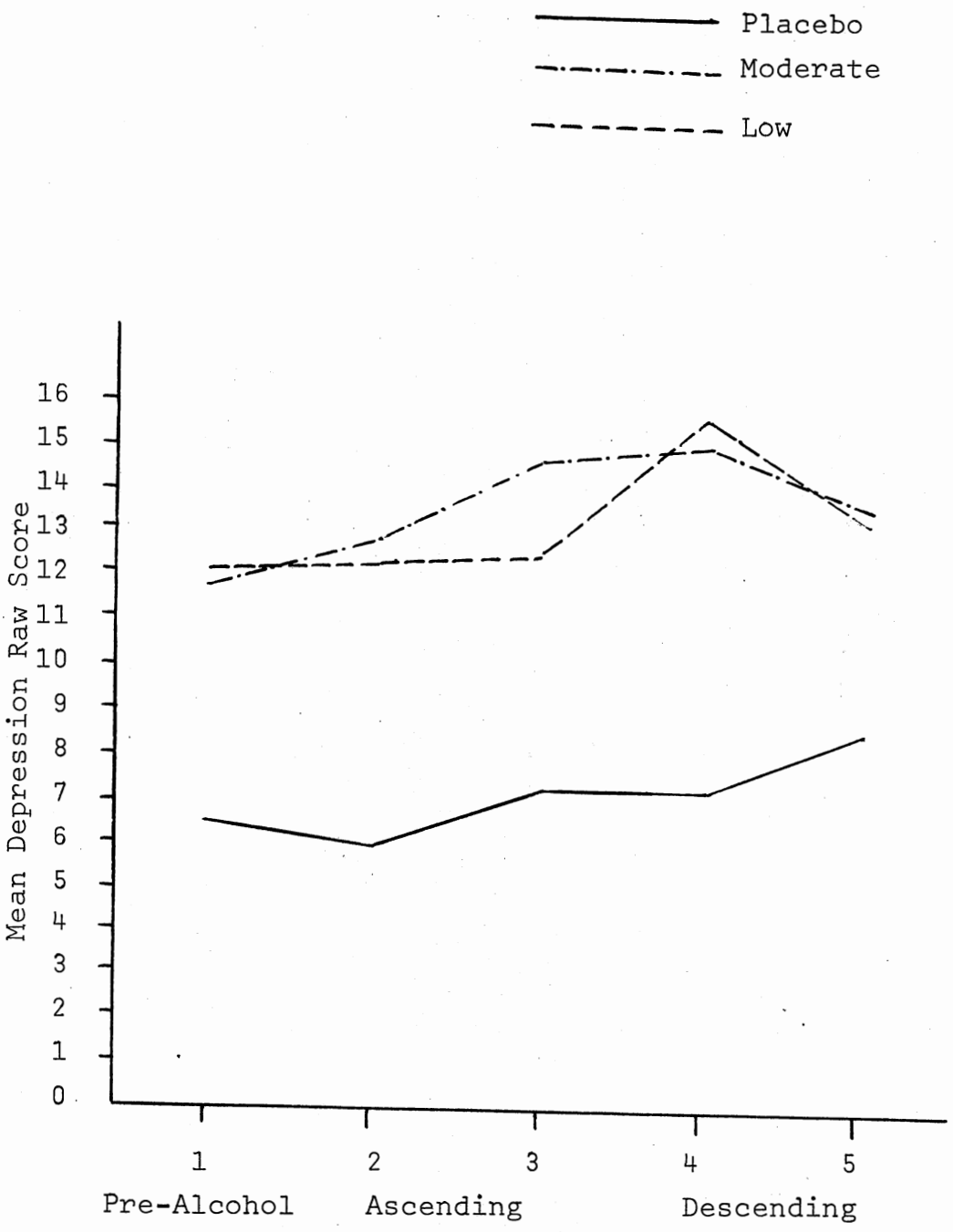

Figure 9. Mean Raw Scores of the Depression Scale of the Multiple Affect Adjective Check List

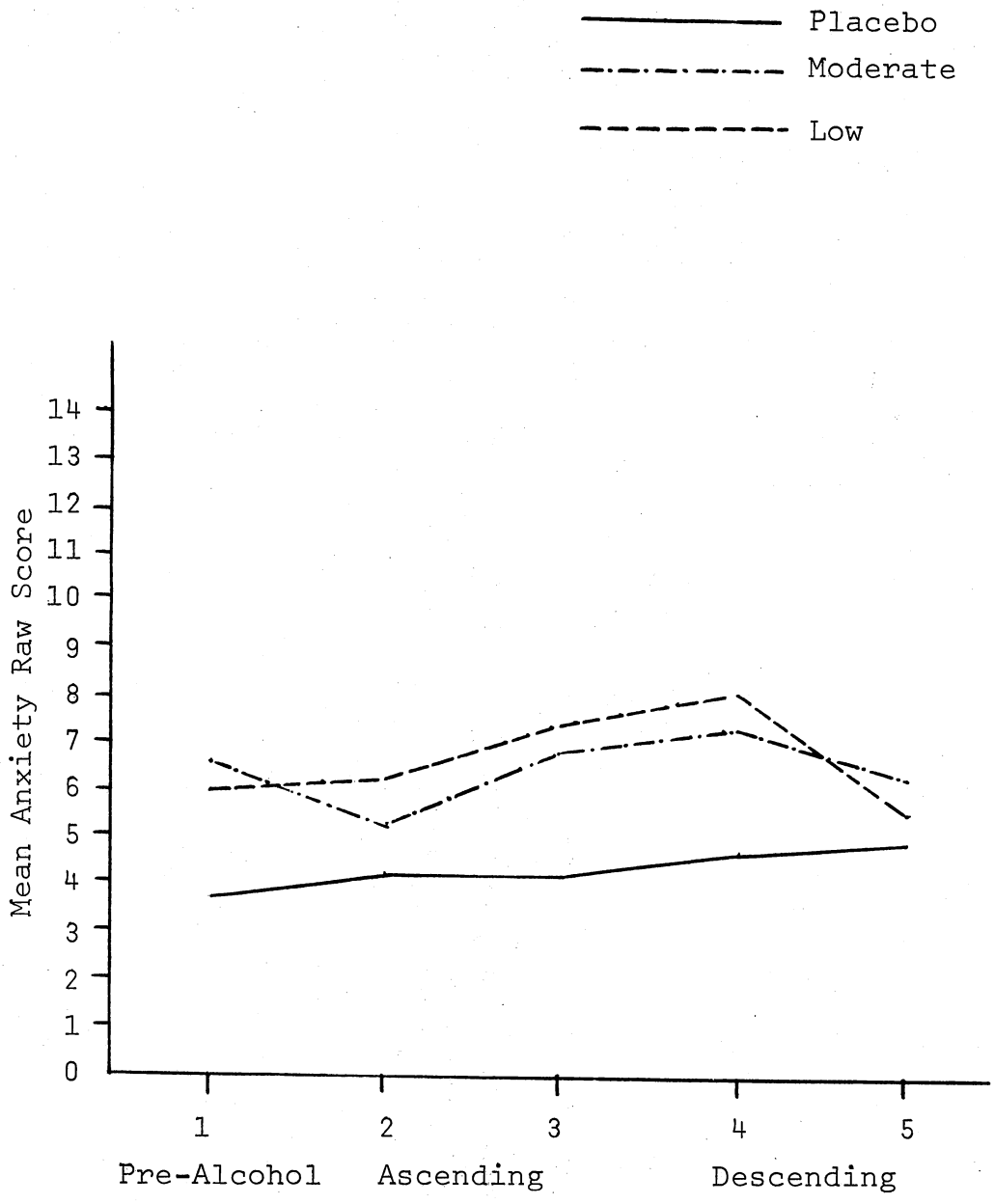

Figure 10. Mean Raw Score of the Ansiety Scale of the Multiple Affect Adjective Check List

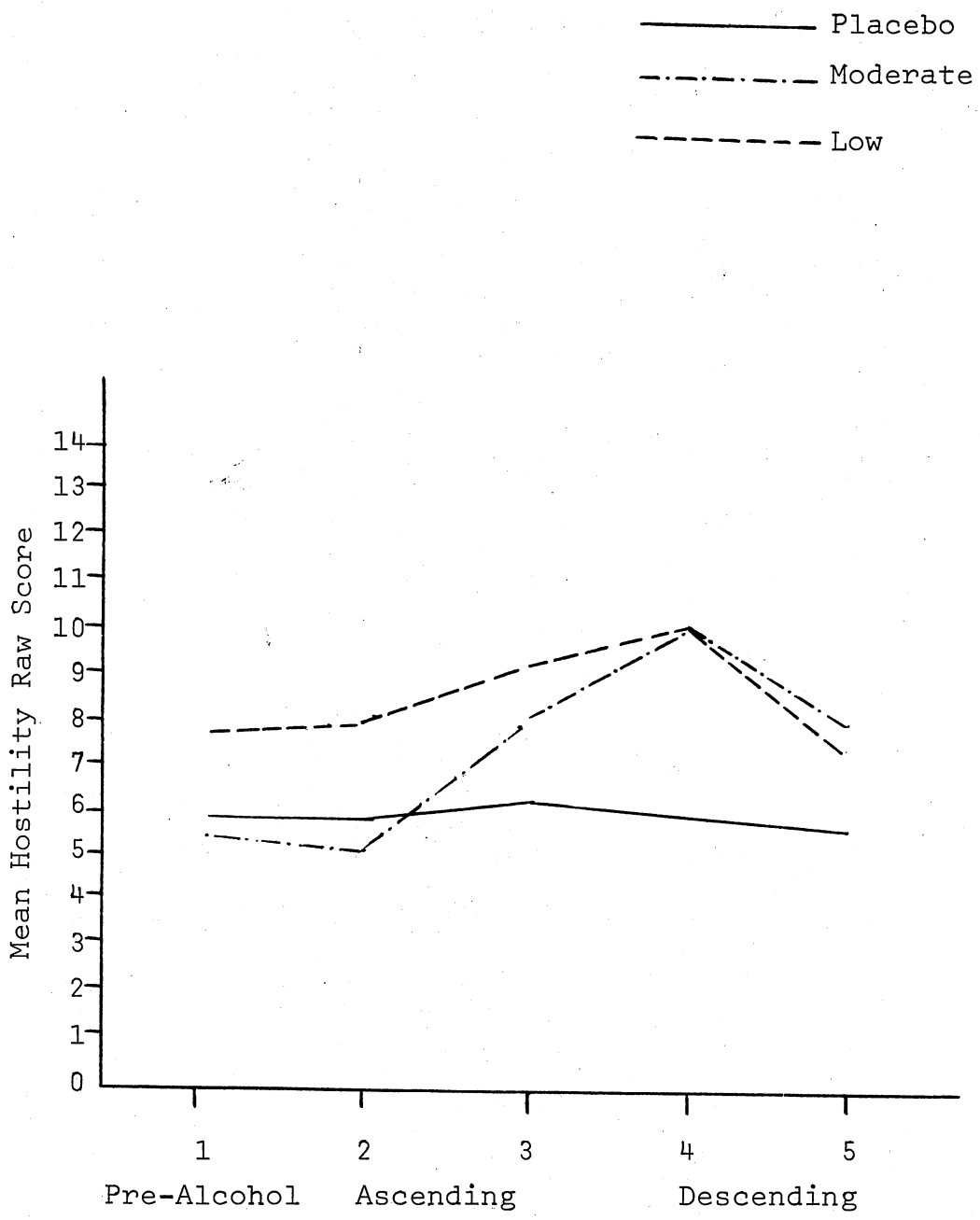

Figure 11. Mean Raw Scores of the Hostility Scale of the Multiple Affect Adjective Check List

CHAPTER VI

DISCUSSION

As expected, at a blood alcohol level of 0.06-0.08%, the moderate dose of alcohol did impair short-term memory. It is more interesting to note that alcohol also significantly impaired short-term memory at a blood alcohol level range of 0.02-0.04%. However, the fact that subjects under a blood alcohol level range of 0.02-0.04% did consistently slightly poorer in recall than those under a comparatively higher blood alcohol level range of 0.06-0.08% came as a total surprise. Although the difference was not statistically significant, it cautioned the straightforeward explanation of dose response differences. Since it is difficult to accept the notion that a lower dose of alcohol should have greater impairment on memory than a relatively higher one, in the present case a 1:2 proportion, four possible explanations are in order.

The first doubt is, of course, whether the learning and recall tasks of the low dose group were performed on the appropriate blood alcohol level range, and hence on the ascending and descending limbs as designed. A simple data check has ruled out such a possibility. A second possibility was that subjects expected a strong drink, but found it not

as strong as they expected, and, as a result, their motivation to try their best in the memory task was lowered. This was not acceptable, because in the post-experimental discussion, two of the six subjects in the low dose group rated one drink as high, two considered the drink moderate, the rest thought it was low. A third explanation was testing time. Although four out of the six low dose subjects were tested in the afternoon (2:00 to 6:00), the curve in Figure 2 did not show any sign of fatigue. Finally, it could be speculated that being in such a low dose group, the subjects were not motivated or challanged to pull themselves together, if those in the moderate dose group were thus motivated and challanged. If this was true, then it may become another dose response characteristic. It is, however, obvious that the low dose did not facilitate recall.

The results that the placebo group had consistently higher mean correct recall than both the low dose group and the moderate dose group could be more favorably explained by the overall parallel elevation of the placebo group against the other two rather than limb effect as such. This was further supported by the fact that there was no significant within group limb effect in both the low dose and the moderate dose groups. The slight decline in mean correct recall in the last 20 trials was too insignificant to be interpreted as fatigue. It, however, rendered the possibility of any practice effect less likely.

The ability to discriminate right from wrong answers was in general not affected by alcohol. With the exception of the low dose group, there were no significant limb effects between alcohol conditions as well as within each condition. That means the moderate dose subjects were able to discriminate between right and wrong responses on both the ascending and the descending limbs almost as well as those in the placebo group. Here the unique nature of the low dose group reappeared again: its ability to discriminate right from wrong answers was significantly lower than that of the placebo group on the ascending limb. Within the low dose group itself, the ability to discriminate one's responses was higher on the descending limb than on the ascending limb. Figure 3 shows the dramatic upshift of the low dose group's response discriminability. Since this within group limb effect on response discriminability could not simply be explained by the alcohol effect, as no such difference was observed in the moderate dose group, it remained another unexplained characteristic of the low dose group in this study.

Given these unique but consistent features of the low dose group, it is tempting to suggest that within a certain limit, alcohol may impair short-term memory at a low blood alcohol level range of 0.02-0.04% to an extent comparable to that produced by a higher blood alcohol level range of 0.06-0.08%. A counter suggestion would be to interpret the particular feature of the low dose group in terms of cog-

nitive dissonance: that all subjects came into the experimental laboratory expecting a strong drink, but while the placebo subjects guessed the drink was not strong immediately, the subjects given the low dose had to take it as a strong drink in order to be consistent with their expectations, and were thus subjectively "drunk," similar to the extent experienced by the subjects given the moderate dose. However, until further evidence is available, these suggestions must be considered as highly speculative. In the present study, placebo subjects were also asked to give their subjective estimation of the dose of alcohol after they had finished the experimental tasks. Three out of six placebo subjects felt that the placebo drink was either "nothing," or it was the weakest or lowest dose, while the other three thought it was moderate or between low and moderate. This, together with the rating on the alcohol dose by low dose subjects mentioned earlier, complicated the cognitive dissonance suggestion.

Unless the impairment on short-term memory shown by the low dose group is interpreted otherwise, the results of the present study did not lend support to the Shillito, King, and Cameron (1974) finding that blood alcohol concentration had no significant effect on information processing. Since the moderate dose used, and the blood alcohol level range during which the subjects were tested were not exactly comparable to other studies (Vega & Jones, 1972; Jones, 1973), it is not appropriate to make parallel comparisons.

Although there was no prior prediction regarding the change in recall confidence under the influence of alcohol, the results that there were no significant differences in mean recall confidence between the placebo, low and moderate dose groups argue clearly against the possibility of pseudostimulation, as argued by Carroll (1975, p. 43). Subjective euphoria was also not obvious, although most alcohol subjects reported the feeling of "high" sometime before they reached the peak blood alcohol level, only two subjects (moderate dose) reported feeling happier. The results on mean recall confidence suggested that alcohol did not "boost" the subjects' confidence in their recall, nor did it lower their recall confidence. However, an alternative interpretation could be that the mean averaged out the possible but not outstanding differences.

The same invariance did not take place in response bias, instead there were quite obvious criterion shifts between the placebo and the two alcohol groups, as well as within each of these. However, since there was no statistical test for any possible significance of Beta, the following interpretation should be taken with caution. From Figure 5 it seems likely that both the placebo and the moderate dose group shifted their response bias from a lower point in the first 20 trials, to a higher point by the second and third blocks of 20 trials, and then went down again, with the moderate dose group exhibiting a consistently higher Beta than that of the placebo group.

These changes in Beta were consistent with the changes in probability of correct responses in both groups with reference to Figure 3a. It could mean that the placebo group with a high d" and a moderate Beta had a balanced pattern of responding, that is, shifted evenly between the four confidence rating categories. With a moderate d" and a high Beta, it could mean that the moderate dose group shifted more to lower confidence ratings most of the time, and finally returned to a balanced distribution of confidence.

The pattern for the low dose group was somewhat different from either the placebo or the moderate dose group. Generally speaking, the low dose group had a moderate d" and a moderate Beta. From the pattern of Beta changes in Figure 5, and the changes in d" in Figure 3a, it could mean that the low dose group shifted more to the "Possibly Right" and "Possibly Wrong" categories than one extreme or the other.

The results on response latency showed that in general there were no significant differences between the three groups. While this seems to be unexpected, it could not be taken on the surface. Since there were significant limb effects, the nonsignificant difference on overall mean response latency between the three groups could be interpreted as a result of averaging out of differences.

There was no significant difference in mean response latency between the first 40 trials and the last 40 trials

for the placebo group. However, as predicted, alcohol did increase response latency, in that it took subjects in the low dose group and in the moderate dose group significantly longer before they responded on the ascending limb of their blood alcohol curves than on the descending limb. In other words, alcohol had more acute effect on response latency on the ascending limb than on the descending limb. The invariant mean response latency between limbs across alcohol groups was not expected, but it could also be interpreted as the result of averaging out, as in the overall mean response latency between groups.

The index of response discriminability based on latency was calculated for comparison with the analogus measure of response discriminability based on binary recall confidence. That $\underline{d}''_L$ was not significantly different between groups was consistent with the mean response latency results. The ability to discriminate right from wrong responses based on response latency was likewise not significantly different between limbs for the low dose group. But, the placebo group and the moderate dose group consistently discriminated better in the second 40 trials than the first 40 trials; it appears that response latencies actually represent a source of information concerning recall accuracy, and that part of the subject's knowledge concerning his recall accuracy may be based on observation of his own response speed.

The results based on the index of binary recall confidence were also consistent with that of mean recall confidence. That is, the three groups exhibited no significant difference in their binary recall confidence. A comparison between $\underline{d}''_{bin}$ and $\underline{d}''_{L}$ showed that $\underline{d}''_{bin}$ was a better indicator of correct recall than $\underline{d}''_{L}$ in all groups and in all limbs, with the exception of the placebo group in the last 40 trials. This invariance between the $\underline{d}''_{bin}$ and $\underline{d}''_{L}$ in the last 40 trials of the placebo group could mean that during the last 40 trials placebo subjects had a better index of discriminability. If so, it would be as accurate for placebo subjects to use either the confidence ratings or the latency measures as an indicator of their correct recall. While it is still impossible to reach a possible cause and effect relationship between response latency and recall confidence, it is possible to argue that subjects actually anchored their recall confidence on response latency rather than the other way around. However, post-experimental discussion with subjects revealed that in some cases it was because they subjectively felt that it was right that their response latency was shorter, in other instances when they had to ponder and mentally process the information retrieval by the cue, it took them longer to respond, and were thus inclined to have lower confidence in their responses.

The only significant change in the subject's mood was shown by the Depression scale of the Multiple Affect Adjective Check List. The mean raw score in the Depression scale was 7.2, 13.3 and 13.6 for the placebo, low and

moderate dose groups, respectively. From Figure 9 it was obvious that subjects in both the low dose group and the moderate dose group were gradually more depressed until the fourth MAACL, and began to level off slightly. However, it is dangerous to interpret this as the sole cause of alcohol effect, since both the low dose and the moderate dose groups scored higher on the Depression scale than the placebo group, even before they consumed alcohol. It could imply that alcohol depressed subjects more around their peak blood alcohol curve at the end of the ascending and at the beginning of the descending limbs. Or, it could mean that alcohol had a slight but not significant effect on the change of mood in this case. But then a question is in order: why did the subjects of both the low dose and the moderate dose groups have a higher baseline depression score than that of the placebo subjects? Since no subject knew what alcohol condition he was in before he consumed the alcohol, and for many of them, even after they had consumed the alcohol, there is no evidence to speculate on the difference in the baseline score on the Depression scale.

Since there was no significant change in mood between the placebo group and the alcohol groups, and the slight change of mood within each group itself was also insignificant, the mood of the subjects could be considered as a constant between alcohol conditions and between limbs. This implies that whatever changes in the subjects' memory impairment, prolongation of response latency and recall con-

fidence were probably independent of the slight mood fluctuations as shown by the Anxiety, Depression and Hostility scales of the MAACL.

CHAPTER VII

SUMMARY

The purpose of the present study was to examine the effects of alcohol on recall, recall confidence, response latency, and mood. Specifically, it was designed to examine the effects of the alcohol on these four dependent variables within a low and a moderate blood alcohol level on the ascending and descending limbs of the subjects' blood alcohol curve.

In a 3x2x2 split-plot design, 18 male students were randomly assigned into three groups (placebo, low and moderate), and were tested on each of the ascending and descending limbs of their blood alcohol curves for two blocks of 20 paired-associate lists. Subjects in the low dose condition were given .46 ml alcohol/kg body wieght, those in the moderate dose condition were given .92 ml/kg of alcohol, while the placebo subjects were given only 4 ml of alcohol. The paired-associate lists were presented by a memory drum at 1 pair/ 2 sec., and were relayed to a television set by a video-tape recorder. Subjects were instructed to respond as quickly and accurately as possible with the corresponding response word and one of the four-point rating categories. A microphone connected to a voice key

started and stopped a clock to measure the subject's response latency in msec. The Multiple Affect Adjective Check List (Today Form) was administered to each subject before the experiment and after each 20 trials.

The results showed that alcohol, within the blood alcohol range of 0.02-0.04% for the low dose group, and 0.06-0.08% for the moderate dose group, impaired short-term memory in general, and on both the ascending and descending limbs. However, the low dose group consistently performed slightly but not significantly poorer than the moderate dose group. While alcohol significantly increased response latency on the ascending limb compared to the descending limb in both the low dose and the moderate dose groups, there was no significant difference in mean response latency between groups. Mean response discriminability ($\underline{d}$") based on binary recall confidence was found to be a more accurate indicator of correct recall than response discriminability based on response latency. No significant change in the subjects' mood was found between alcohol conditions and limbs.

Due to the particular features of the low dose group, it was suggested that future research should also examine the particular effects of alcohol at low blood alcohol levels. While the emphasis on possible limb effects might be a new dimension in alcohol research, the potential contribution of the signal detection paradigm was stressed. In view of the unparalleled nature of some of the questions

asked in the present study, the present exploratory findings should be interpreted with appropriate caution.

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APPENDIX A

DRINKING QUESTIONNAIRE

Identification Number:

Introduction

The following questions will enable the experimenter to determine whether you are a potential subject for an alcohol study to be conducted soon. Please answer all questions as frankly and accurately as you can. All information will be kept confidential and destroyed after use.

Please note that this is not a test or examination. If, while you are completing this questionnaire, you decide that you are no longer interested in contemplating participation in the alcohol study, you may stop and leave.

PART A

In the following questions/statements please check ( ) the appropriate box to indicate your willingness or reservation.

I.	If you are going to serve as a subject in the alcohol study it is necessary, to safeguard your well-being, that you sign a statement authorizing a designated M. D. (a member of the supervising committee of this study) to get access to your health record kept by the University Hospital & Clinic to ensure that you are suitable to participate in this study in which you will be required to consume alcohol.	Willing to sign ( ) Reservation/ unwilling ( )
II.	You will be asked to stop taking any medication/drugs (if applicable) 24 hours before the experimental session, and to fast four hours prior to your appearance for the session.	Willing/ possible ( ) Unwilling/ impossible ( )
III.	The experimental tasks will take about one hour to complete, but you will not be allowed to leave the laboratory until your blood alcohol level is zero; depending on the dose you will get, it may take another 30 minutes to 3 hours. Are you willing, or is it possible to devote a maximum of about five hours for this experiment? (You may study or take a rest during the waiting period).	Willing/ possible ( ) Reservation/ Impossible ( )

PART B

DRINKING QUESTIONNAIRE

Age: _____ Weight: _____ Height: _____

1. At what age did you first drink alcohol? _____

2. At what age did you start drinking regularly? _____

3. Do you consider yourself a (an):

Abstainer _____
Light Drinker _____
Moderate Drinker _____
Heavy Drinker _____

4. What is your drinking frequency?
(Please check one in each column.)

Less than once ( )	Daily ( )
Once ( )	Weekly ( )
Twice ( )	Monthly ( )
More than twice ( )	

When do you usually drink?

Morning _____
Afternoon _____
Evening _____
Any time _____

5. When was the last time you drank alcohol? _____
How much? _____

6. How much, and what, did you drink in the last 7 days?

Beer? _____ Wine? _____ Liquors? _____

7. What and how much did you drink in the last month?

Beer? _____ Wine? _____ Liquors? _____

8. Is this an average amount? _____ More? _____ Less? _____

9. What has been your longest (in hours) period of uninterrupted drinking?

   _____ hours

   What did you drink? _____

   How much? _____

   When was that? _____

10. Have you ever been drunk? _____

11. How many times have you been drunk? _____

12. When was your last drunk? _____

13. How many times have you been sick (vomiting)? _____

    When was the last time (if applicable)? _____

14. Do you usually drink alone? _____ With others? _____

15. Have you ever worried about your drinking habits? _____

    Alcoholism in family? _____

16. To the best of your knowledge, do you have any health problems or any illness at present?

    _____

    What are they (if applicable)? _____

17. Are you currently taking any medication/drugs? _____

    For what purposes? _____

18. What and when was your last illness? _____

19. Have you ever considered yourself an alcoholic? _____

    Have you ever been so labeled? _____

    Have you ever been hospitalized as a result of drinking? _____

20. Have you ever experienced blackouts from drinking? _____

21. Have you ever been unable to attend school or work due to drinking? _____

22. Have you ever been unable to stop drinking at a given session? _____

Since the alcohol study requires only a limited number of subjects, depending on the responses, not all volunteers interested in being subjects will have the chance to participate in the study. However, all those who have completed this questionnaire will be notified within ten minutes whether they will be offered the option to serve as subjects in the alcohol study.

Thank you very much for your interest in the alcohol study!

Kwan-hung Wong
(Experimenter)

APPENDIX B

INFORMED CONSENT STATEMENT

AUTHORIZATION FOR USE OF ALCOHOL FOR

INVESTIGATIONAL PURPOSES

I, _____, voluntarily consent to
(Name of participant)
participate in the investigation entitles: "Recall, Recall Confidence, Response Latency, and Mood on the Ascending and Descending Limbs of the Blood Alcohol Curve of Nonalcoholics," the purpose of which has been explained to be by Kwan-hung Wong. I hereby authorize Kwan-hung
(Experimenter)
Wong, graduate student, Department of Psychology, Oklahoma State University, to conduct the foregoing study in which I will serve as a subject.

I understand that I will be asked to complete several learning and recall tasks after consuming one of several doses of alcohol, the highest dose used in this study will be .42 millilitre per every pound of body weight. In addition, I will have to take the Multiple Affect Adjective Check List several times.

I have been informed of the possibilities of discomforts, risks, and adverse effects, as well as the benefits, which could result as outlined below, and I have been given an opportunity to ask questions.

(Benefits: General). To allow the participant to become familiar with the effects of alcohol in a controlled experimental condition where blood alcohol levels will be recorded. In addition, extra course credit will be given.

(Risks: General). The risks are minimal since only persons with a history of social use of alcohol will be tested. The calculated maximal peak blood alcohol level for the highest dose used in this stydy will be 0.09%. Although infrequent, it is possible that some people who

absorb alcohol rapidly may be uncomfortable (dizziness or vomiting) for a short time; if the discomfort prolongs, the participant will be stopped from further participation in the experiment, and be escorted home or sent to a physician, if necessary.

I understand that the information derived from this study may prove to be useful in advancing knowledge on the effects of alcohol on memory, mood, and reaction-time. I also understand that:

1. Whereas no assurance can be made concerning results that may be obtained (since results from investigational studies cannot be accurately predicted), the experimenter will take every precaution consistent with the best experimental procedures.
2. By signing this consent form, I have not waived any of my legal rights or released the experimenter and the Oklahoma State University from liability for negligence. I may revoke my consent and withdraw from this study at any time.
3. I agree to stop from using any drug/medication 24 hours before I participate in the experimental session. I further agree not to smoke, eat, or drink anything before the experimental tasks are completed.
4. I agree to remain at the laboratory following the administration of the alcohol until my blood alcohol level is zero, with the understanding that a modest snack will be provided after completion of the experimental tasks.
5. I understand that any information obtained from me will be treated as confidential and will receive a code number so that they will remain anonymous throughout the study, and be destroyed after the completion of the study.

To safeguard my well-being, I hereby authorize that a designated M. D. be allowed access to my health record kept by the University Hospital & Clinic to ensure that I am physically suitable to participate in the foregoing study in which I will have to consume alcohol.

I now understand the experiment as outlined above and explained by the experimenter, and I voluntarily agree to participate in the foregoing study.

_____ _____ _____

Subject's Signature Experimenter's Signature Witnessed by

_____ _____ _____

Identification Number Date Time of Day

APPENDIX C

PERSONAL AND ALCOHOL DATA SHEET

****

Personal Information and Blood Alcohol Level (BAL) Data

Name: _____ Identification Number: _____

Age: _____ Weight: _____ Height: _____

I certify that I have not taken any medication or drugs in the last 24 hours.

_____
Subject's Signature

Date: _____ Time: _____

****

Calculation of alcohol dose:

_____ Weight(lbs) _____ ml alcohol _____ ml alcohol

_____ X 4 _____ _____ ml orange drink

_____ ml alcohol _____ ml orange _____ ml total

****

BAL Data

Difference	Time	Breath No.	BAL	Comments
				Sobriety Test
0 minutes				Begin Drinking
				Finish Drinking
				Rinse Mouth With Water
		1		
		2		
		3		
		4		
		5		
		6		
		7		
		8		
		9		
		10		
		11		
		12		

APPENDIX D

INSTRUCTIONS

You are about to participate in a memory experiment. This is not a test of your IQ or personality. Although the task may seem to be a very simple one, our research indicates that it can provide important information about the effect of alcohol on memory. Therefore, your very close cooperation is absolutely necessary for the success of the experiment. What follows is a description of your part in the experiment. Please hold your questions until the instructions are over; the experimenter will then be glad to answer any questions you have. We are interested in the way pairs of words are remembered and we also want to learn how confidence in memory develops, under the influence of alcohol.

Your task will be to first study five stimulus-response word pairs; these will be shown to you one pair at a time. You are to memorize the response word that goes with each stimulus word, so as to be able to recall the corresponding response word, when you see the stimulus word alone.

In each trial your memory of one of five pairs will be tested. For each test, as soon as the stimulus word is pre-

sented, please say the corresponding response word as quickly as possible. It is very important that you verbally respond with a word, the best of your memory at the moment. Also, it is important to speak your response clearly and distinctly into the microphone.

After you have chosen your response, you are also to verbally indicate how confident you are that your answer is correct. You should say "Definitely Right" after you have said the response word, if you think the response word given is definitely correct. Say "Possibly Right" if you think your answer is possibly correct. Say "Possibly Wrong" if you think your answer is possibly incorrect. Say "Definitely Wrong" if you think your answer is definitely incorrect.

There will be four blocks of 20 lists each in the experiment. You will take a breath test and a "Multiple Affect Adjective Check List" test, at the end of each block. Following are two example study lists followed by one stimulus word. Please practice by verbally responding with the response word, and a rating to whichever stimulus word presented after the five pairs.

APPENDIX E

MULTIPLE AFFECT ADJECTIVE CHECK LIST

Today Form

By Marvin Zuckerman & Bernard Lubin

Date _____ Age _____ Sex _____

DIRECTIONS: On this sheet you will find words which describe different kinds of moods and feelings. Mark an X in the space beside the words which describe how you feel now--this moment. Some of the words may sound alike, but we want you to check all the words that describe your feelings. Work rapidly.

1. ___ active
2. ___ adventurous
3. ___ affectionate
4. ___ afraid
5. ___ agitated
6. ___ agreeable
7. ___ aggressive
8. ___ alive
9. ___ alone
10. ___ amiable
11. ___ amused
12. ___ angry
13. ___ annoyed
14. ___ awful
15. ___ bashful
16. ___ bitter
17. ___ blue
18. ___ bored
19. ___ calm
20. ___ cautious
21. ___ cheerful
22. ___ clean
23. ___ complaining
24. ___ contented
25. ___ contrary
26. ___ cool
27. ___ cooperative
28. ___ critical
29. ___ cross
30. ___ cruel
31. ___ daring
32. ___ desperate
33. ___ destroyed
34. ___ devoted
35. ___ disagreeable
36. ___ discontented
37. ___ discouraged
38. ___ disgusted
39. ___ displeased
40. ___ energetic
41. ___ enraged
42. ___ enthusiastic
43. ___ fearful
44. ___ fine
45. ___ fit
46. ___ forlorn
47. ___ frank
48. ___ free
49. ___ friendly
50. ___ frightened
51. ___ furious
52. ___ gay
53. ___ gentle
54. ___ glad
55. ___ gloomy
56. ___ good
57. ___ good-natured
58. ___ grim
59. ___ happy
60. ___ healthy
61. ___ hopeless
62. ___ hostile
63. ___ impatient
64. ___ incensed
65. ___ indignant
66. ___ inspired
67. ___ interested
68. ___ irritated
69. ___ jealous
70. ___ joyful
71. ___ kindly
72. ___ lonely
73. ___ lost
74. ___ loving

75. ___ low
76. ___ lucky
77. ___ mad
78. ___ mean
79. ___ meek
80. ___ merry
81. ___ mild
82. ___ miserable
83. ___ nervous
84. ___ obliging
85. ___ offended
86. ___ outraged
87. ___ panicky
88. ___ patient
89. ___ peaceful
90. ___ pleased
91. ___ pleasant
92. ___ polite
93. ___ powerful
94. ___ quiet
95. ___ reckless
96. ___ rejected
97. ___ rough
98. ___ sad
99. ___ safe
100. ___ satisfied
101. ___ secure
102. ___ shaky
103. ___ shy
104. ___ soothed
105. ___ steady
106. ___ stubborn
107. ___ stormy
108. ___ strong
109. ___ suffering
110. ___ sullen
111. ___ sunk
112. ___ sympathetic
113. ___ tame
114. ___ tender
115. ___ tense
116. ___ terrible
117. ___ terrified
118. ___ thoughtful
119. ___ timid
120. ___ tormented
121. ___ understanding
122. ___ unhappy
123. ___ unsociable
124. ___ upset
125. ___ vexed
126. ___ warm
127. ___ whole
128. ___ wild
129. ___ willing
130. ___ wilted
131. ___ worrying
132. ___ young

APPENDIX F

TABLE OF MEANS AND STANDARD DEVIATIONS FOR NUMBER OF WORDS CORRECTLY RECALLED ON THE ASCENDING AND DESCENDING LIMBS OF THE BLOOD ALCOHOL CURVE

Alcohol Condition	Blood Alcohol Curve	Task	Mean	Standard Deviation
Placebo	Ascending	1	9.00	1.55
		2	9.50	1.87
	Descending	1	10.33	1.75
		2	9.67	2.42
Low	Ascending	1	6.67	.52
		2	6.17	1.17
	Descending	1	7.00	2.37
		2	7.67	1.97
Moderate	Ascending	1	7.50	2.51
		2	6.67	2.25
	Descending	1	7.17	1.83
		2	8.50	2.59

APPENDIX G

TABLE OF MEANS AND STANDARD DEVIATIONS FOR RESPONSE LATENCY ON THE ASCENDING AND DESCENDING LIMBS OF THE BLOOD ALCOHOL CURVE

Alcohol Condition	Blood Alcohol Curve	Task	Mean	Standard Deviation
Placebo	Ascending	1	2.22	.64
		2	2.27	.63
	Descending	1	2.29	.53
		2	2.14	.74
Low	Ascending	1	2.37	.72
		2	2.24	.48
	Descending	1	2.16	.66
		2	2.03	.40
Moderate	Ascending	1	2.56	.71
		2	2.46	.81
	Descending	1	2.44	.74
		2	2.22	.55

APPENDIX H

TABLE OF MEANS AND STANDARD DEVIATIONS FOR THE ANXIETY SCALE OF THE MULTIPLE AFFECT ADJECTIVE CHECK LIST (MAACL)

Alcohol Condition	MAACL Administration	Mean	Standard Deviation
Placebo	1	3.67	1.86
	2	4.17	2.32
	3	4.17	3.37
	4	4.67	2.88
	5	4.83	1.83
Low	1	6.17	2.48
	2	6.50	3.78
	3	7.83	2.56
	4	8.50	1.87
	5	5.83	2.87
Moderate	1	7.00	2.37
	2	5.50	2.43
	3	7.00	2.97
	4	7.67	4.13
	5	6.33	3.33

APPENDIX I

TABLE OF MEANS AND STANDARD DEVIATIONS FOR THE DEPRESSION SCALE OF THE MULTIPLE AFFECT ADJECTIVE CHECK LIST (MAACL)

Alcohol Condition	MAACL Administration	Mean	Standard Deviation
Placebo	1	6.50	2.17
	2	6.00	2.53
	3	7.50	2.26
	4	7.33	3.61
	5	8.67	4.46
Low	1	12.33	4.32
	2	12.50	5.32
	3	12.67	4.84
	4	15.83	3.92
	5	13.17	4.88
Moderate	1	12.00	3.63
	2	12.67	3.67
	3	14.67	5.99
	4	15.17	10.03
	5	13.67	7.39

APPENDIX J

TABLE OF MEANS AND STANDARD DEVIATIONS FOR THE HOSTILITY SCALE OF THE MULTIPLE AFFECT ADJECTIVE CHECK LIST (MAACL)

Alcohol Condition	MAACL Administration	Mean	Standard Deviation
Placebo	1	6.33	3.27
	2	6.33	2.42
	3	6.50	2.74
	4	6.33	3.08
	5	6.00	1.41
Low	1	8.08	3.35
	2	8.33	2.42
	3	9.83	2.48
	4	10.83	5.78
	5	7.83	2.88
Moderate	1	5.83	2.56
	2	5.67	3.17
	3	8.83	4.31
	4	10.83	6.05
	5	8.33	4.08

APPENDIX K

SUMMARY OF THE ANALYSIS OF VARIANCE FOR NUMBER OF WORDS CORRECTLY RECALLED

Source	df	MS	F	p <
Between Subjects				
Alcohol Condition (A)	2	50.3887	4.9740	.025
Subject w. A	15	10.1305		
Within Subjects				
BAC Limb (L)	1	11.6806	3.5788	ns
Recall Task (T)	1	.1250	.1526	ns
A x L	2	.0555	.0170	ns
A x T	2	.1666	.2034	ns
L x T	1	2.3472	1.4903	ns
A x L x T	2	4.3889	2.7865	ns
L x Subject w. A	15	3.2638		
T x Subject w. A	15	.8194		
L x T x Subject w. A	15	1.5750		

APPENDIX L

SUMMARY OF THE ANALYSIS OF VARIANCE

FOR MEAN RESPONSE LATENCY

Source	df	MS	F	p
Between Subjects				
Alcohol Condition (A)	2	.3471	.2350	ns
Subjects w. A	15	1.4773		
Within Subjects				
BAC Limb (L)	1	.3459	6.3236	.025
Recall Task (T)	1	.2248	2.9898	ns
A x L	2	.0530	.9681	ns
A x T	2	.0186	.2468	
L x T	1	.0494	.9467	ns
A x L x T	2	.0123	.2360	ns
L x Subjects w. A	15	.0547		
T x Subjects w. A	15	.0752		
L x T x Subjects w. A	15	.0522		

APPENDIX M

SUMMARY OF THE ANALYSIS OF VARIANCE

FOR MEAN RECALL CONFIDENCE

Source	df	MS	F	p<
Between Subjects				
Alcohol Condition (A)	2	.5615	2.2338	ns
Subjects w. A	15	.2514		
Within Subjects				
BAC Limb (L)	1	.0125	.1103	ns
Recall Task (T)	1	.0059	.2200	ns
A x L	2	.0038	.0333	ns
A x T	2	.0142	.5324	ns
L x T	1	.1209	2.0283	ns
A x L x T	2	.0448	.7521	ns
L x Subjects w. A	15	.1136		
T x Subjects w. A	15	.0267		
L x T x Subjects w. A	15	.0596		

APPENDIX N

SUMMARY OF THE ANALYSIS OF VARIANCE

FOR INDEX OF DISCRIMINABILITY

BASED ON RESPONSE LATENCY

Source	df	MS	F	p<
Between Subjects				
Alcohol Condition (A)	2	2.3816	2.3483	ns
Subjects w. A	15	1.0141		
Within Subjects				
BAC Limb (L)	1	1.2174	4.9653	.05
A x L	2	.3092	1.2612	
L x Subjects w. A	15	.2452		

APPENDIX O

SUMMARY OF THE ANALYSIS OF VARIANCE

FOR BINARY RECALL CONFIDENCE

Source	df	MS	F	p
Between Subjects				
Alcohol Condition (A)	2	.0213	.0260	ns
Subjects w. A	15	.8212		
Within Subjects				
BAC Limb (L)	1	1.0609	1.4369	ns
A x L	2	.1626	.2202	ns
L x Subjects w. A	15	.7383		

APPENDIX P

SUMMARY OF THE ANALYSIS OF VARIANCE FOR DEPRESSION SCALE OF THE MULTIPLE AFFECT ADJECTIVE CHECK LIST (MAACL)

Source	df	MS	F	p <
Between Subjects				
Alcohol Condition (A)	2	393.5442	4.8897	.025
Subjects w. A	15	80.4842		
Within Subjects				
MAACL Administration (M)	4	19.8444	1.7638	ns
A x M	8	4.7941	.4261	ns
M x Subjects w. A	60	11.2511		

APPENDIX Q

SUMMARY OF THE ANALYSIS OF VARIANCE FOR ANXIETY SCALE OF THE MULTIPLE AFFECT ADJECTIVE CHECK LIST (MAACL)

Source	df	MS	F	p<
Between Subjects				
Alcohol Condition (A)	2	64.7111	2.8089	ns
Subjects w. A	15	23.0377		
Within Subjects				
MAACL Administration (M)	4	7.3722	1.7889	ns
A x M	8	2.9055	.7051	ns
M x Subjects w. A	60	4.1210		

APPENDIX R

SUMMARY OF THE ANALYSIS OF VARIANCE FOR HOSTILITY SCALE OF THE MULTIPLE AFFECT ADJECTIVE CHECK LIST (MAACL)

Source	df	MS	F	p<
Between Subjects				
Alcohol Condition (A)	2	54.0444	2.1113	ns
Subjects w. A	15	25.5977		
Within Subjects				
MAACL Administration (M)	4	22.6944	2.4380	ns
A x M	8	8.0861	.8687	ns
M x Subjects w. A	60	9.3088		

VITA

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Master of Science

Thesis: THE EFFECTS OF ALCOHOL ON RECALL, RECALL CONFIDENCE, RESPONSE LATENCY, AND MOOD DURING THE ASCENDING AND DESCENDING LIMBS OF THE BLOOD ALCOHOL CURVE OF NONALCOHOLICS

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